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Stanowisko Polskiego Towarzystwa Diabetologicznego

Clinical Recommendations on the Management of Individuals with Diabetes – 2026 Position Statement of Diabetes Poland

Recommendations Committee for 2026

Professor Aleksandra Araszkiewicz, MD, PhD
Department of Internal Medicine and Diabetology,
Poznań University of Medical Sciences, Poznań, Poland

Sebastian Borys, MD, PhD
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Professor Marlena Broncel, MD, PhD
Department of Internal Medicine and Clinical Pharmacology,
Medical University of Łódź, Łódź, Poland

Professor Andrzej Budzyński, MD, PhD
Department of General and Oncological Surgery,
Ludwik Rydygier Memorial Specialist Hospital, Kraków, Poland

Katarzyna Cyganek, MD, PhD
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Professor Katarzyna Cypryk, MD, PhD
Department of Internal Medicine and Diabetology,
Medical University of Łódź, Łódź, Poland

Katarzyna Cyranka, MD, PhD
Department of Psychiatry,
Jagiellonian University Medical College, Kraków, Poland

Professor Leszek Czupryniak, MD, PhD
Department of Diabetology and Internal Medicine,
Medical University of Warsaw, Warsaw, Poland

Mariusz Dąbrowski, MD, PhD, Professor at the University of Rzeszów
Department of Pathophysiology and Human Physiology,
University of Rzeszów, Rzeszów, Poland

Professor Grzegorz Dzida, MD, PhD
Department of Internal Medicine,
Medical University of Lublin, Lublin, Poland

Professor Tomasz Dziedzic, MD, PhD
Department of Neurology,
Jagiellonian University Medical College, Kraków, Poland

Professor Edward Franek, MD, PhD
Mossakowski Medical Research Institute, Polish Academy of Sciences, Warsaw, Poland
Department of Internal Medicine, Endocrinology and Diabetology,
National Medical Institute of the Ministry of the Interior and Administration, Warsaw, Poland

Danuta Gajewska, PhD, DSc (Eng.)
Department of Dietetics, Faculty of Human Nutrition and Consumer Sciences,
Warsaw University of Life Sciences (SGGW), Warsaw, Poland

Andrzej Gawrecki, MD, PhD
Department of Internal Medicine and Diabetology,
Poznań University of Medical Sciences, Poznań, Poland

Professor Maria Górska, MD, PhD
Department of Endocrinology, Diabetology and Internal Medicine,
Medical University of Białystok, Białystok, Poland

Professor Janusz Gumprecht, MD, PhD
Department of Internal Medicine, Diabetology and Nephrology,
Medical University of Silesia, Katowice, Poland

Professor Barbara Idzior-Waluś, MD, PhD
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Professor Przemysława Jarosz-Chobot, MD, PhD
Department of Paediatric Diabetology,
Medical University of Silesia, Katowice, Poland

Professor Zbigniew Kalarus, MD, PhD
Department of Cardiology, Congenital Heart Diseases and Electrotherapy,
Medical University of Silesia, Katowice, Poland
Silesian Centre for Heart Diseases, Zabrze, Poland

Professor Monika Karczewska-Kupczewska, MD, PhD
Department of Internal Medicine and Metabolic Diseases,
Medical University of Białystok, Białystok, Poland

Professor Tomasz Klupa, MD, PhD
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Professor Andrzej Kokoszka, MD, PhD
Second Department of Psychiatry,
Medical University of Warsaw, Warsaw, Poland

Professor Anna Korzon-Burakowska, MD, PhD
Department of Hypertension and Diabetology,
Medical University of Gdańsk, Gdańsk, Poland

Professor Irina Kowalska, MD, PhD
Department of Internal Medicine and Metabolic Diseases,
Medical University of Białystok, Białystok, Poland

Professor Adam Krętowski, MD, PhD
Department of Endocrinology, Diabetology and Internal Medicine,
Medical University of Białystok, Białystok, Poland

Hanna Kwiendacz, MD, PhD, Professor at the Medical University of Silesia
Department of Internal Medicine, Diabetology and Nephrology,
Medical University of Silesia, Katowice, Poland

Professor Lilianna Majkowska, MD, PhD
Department of Diabetology and Internal Medicine,
Pomeranian Medical University, Szczecin, Poland

Professor Maciej Małecki, MD, PhD
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Professor Artur Mamcarz, MD, PhD
Third Department of Internal Medicine and Cardiology,
Medical University of Warsaw, Warsaw, Poland

Bartłomiej Matejko, MD, PhD, DSc (Eng.), Professor at Jagiellonian University
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Professor Beata Matyjaszek-Matuszek, MD, PhD
Department of Endocrinology, Diabetology and Metabolic Diseases,
Medical University of Lublin, Lublin, Poland

Beata Mianowska, MD, PhD, Professor at the Medical University of Łódź
Department of Paediatrics, Diabetology, Endocrinology and Nephrology,
Medical University of Łódź, Łódź, Poland

Beata Mrozikiewicz-Rakowska, MD, PhD, Professor at CMKP
Centre of Postgraduate Medical Education, Warsaw, Poland

Professor Małgorzata Myśliwiec, MD, PhD
Department of Paediatrics, Diabetology and Endocrinology,
Medical University of Gdańsk, Gdańsk, Poland

Professor Katarzyna Nabrdalik, MD, PhD, DSc (Health Sciences)
Department of Internal Medicine, Diabetology and Nephrology,
Medical University of Silesia, Katowice, Poland

Professor Krzysztof Narkiewicz, MD, PhD
Department of Hypertension and Diabetology,
Medical University of Gdańsk, Gdańsk, Poland

Professor Jacek Sieradzki, MD, PhD
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Jan Skupień, MD, PhD, Professor at Jagiellonian University
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Professor Bogdan Solnica, MD, PhD
Department of Clinical Biochemistry,
Jagiellonian University Medical College, Kraków, Poland

Professor Tomasz Stompór, MD, PhD
Department of Internal Medicine,
University of Warmia and Mazury in Olsztyn, Olsztyn, Poland

Professor Krzysztof Strojek, MD, PhD
Department of Internal Medicine, Diabetology and Cardiometabolic Diseases,
Medical University of Silesia, Katowice, Poland
Silesian Centre for Heart Diseases, Zabrze, Poland

Professor Agnieszka Szadkowska, MD, PhD
Department of Paediatrics, Diabetology, Endocrinology and Nephrology,
Medical University of Łódź, Łódź, Poland

Professor Agnieszka Szypowska, MD, PhD
Department of Paediatric Diabetology and Paediatrics,
Medical University of Warsaw, Warsaw, Poland

Aleksandra Uruska, MD, PhD
Department of Internal Medicine and Diabetology,
Poznań University of Medical Sciences, Poznań, Poland

Professor Ewa Wender-Ożegowska, MD, PhD
Department of Reproduction, Chair of Fetomaternal Medicine,
Poznań University of Medical Sciences, Poznań, Poland

Przemysław Witek, MD, PhD
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Bogumił Wolnik, MD, PhD
Department of Hypertension and Diabetology,
Medical University of Gdańsk, Gdańsk, Poland

Professor Mariusz Wyleżoł, MD, PhD
Second Department of General, Vascular and Oncological Surgery,
Medical University of Warsaw, Warsaw, Poland

Professor Edward Wylęgała, MD, PhD
Department of Ophthalmology,
Medical University of Silesia, Katowice, Poland

Professor Agnieszka Zmysłowska, MD, PhD
Department of Clinical Genetics,
Medical University of Łódź, Łódź, Poland

Professor Dorota Zozulińska-Ziółkiewicz, MD, PhD
Department of Internal Medicine and Diabetology,
Poznań University of Medical Sciences, Poznań, Poland

Chapter 13 was developed in collaboration with **Dr. Jacek Wolf, MD, PhD**; **Chapter 26** was developed in collaboration with **Prof. Wojciech Szczeklik, MD, PhD**, and **Dorota Studzińska, MD, PhD**; **Chapter 28** was developed in collaboration with **Andrzej Marcinkiewicz, MD, PhD**, and **Prof. Jolanta Walusiak-Skorupa, MD, PhD**; **Chapter 31** was developed in collaboration with **Prof. Renata Górska, MD, PhD, Prof. Nadia Sawicka-Gutaj, MD, PhD, Prof. Jolanta Słowikowska-Hilczer, MD, PhD, Prof. Michał Rabijewski, MD, PhD**, and **Prof. Marek Ruchała, MD, PhD**.

Declaration of members of the committee for recommendations on potential conflicts of interest is available at: ptdiab.pl

Special acknowledgment to those who have contributed to clinical recommendations for the management of people with diabetes in previous years:

Prof. Elżbieta Bandurska-Stankiewicz, MD, PhD
Department of Endicronology, Diabetology and Internal Medicine,
University of Warmia and Mazury, Olsztyn, Poland

Prof. Anna Czech, MD, PhD
Department of Internal Medicine and Diabetology,
Medical University of Warsaw, Poland

Prof. Józef Drzewoski, MD, PhD
Department of Internal Medicine, Diabetology and Clinical Pharmacology,
Medical University of Lodz, Poland

Prof. Maria Górska, MD, PhD
Department of Endocrinology, Diabetology and Internal Medicine,
Medical University of Bialystok, Poland

Prof. Władysław Grzeszczak, MD, PhD
Department of Internal Medicine, Diabetology and Nephrology,
Medical University of Silesia in Katowice, Poland

Teresa Koblik, MD, PhD
Department of Metabolic Diseases,
Jagiellonian University Medical College, Kraków, Poland

Prof. Barbara Mirkiewicz-Sieradzka, MD, PhD
Department of Metabolic Diseases,
University Hospital in Krakow, Poland

Prof. Wojciech Młynarski, MD, PhD
Department of Pediatrics, Oncology and Hematology,
Medical University of Lodz, Poland

Prof. Dariusz Moczulski, MD, PhD
Department of Internal Medicine and Nephrodiabetology,
Medical University of Lodz, Poland

Prof. Anna Noczyńska, MD, PhD
Department of Endocrinology and Diabetology of the Developmental Age,
Medical University of Piastów Śląskich in Wrocław, Poland

Prof. Joanna Rymaszewska, MD, PhD
Department of Psychiatry,
Medical University of Piastów Śląskich in Wrocław, Poland

Prof. Marek Strączkowski, MD, PhD
Department of Metabolic Disease Prevention,
Institute of Animal Reproduction and Food Research,
Polish Academy of Sciences in Olsztyn, Poland

Prof. Małgorzata Szelachowska, MD, PhD
Department of Endocrinology, Diabetology and Internal Medicine,
Medical University of Bialystok, Poland

Dr. Beata Telejko, MD, PhD
Department of Endocrinology, Diabetology and Internal Medicine,
Medical University of Bialystok, Poland

Prof. Bogna Wierusz-Wysocka, MD, PhD
Department of Internal Medicine and Diabetology,
K. Marcinkowski Medical University of Poznan, Poland

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Diabetes Poland Clinical Recommendations 2026: Summary of Revisions

Dear Colleagues,
In line with a long-standing tradition, we are pleased to present another edition of the clinical recommendations of the Diabetes Poland (PTD) concerning the management of individuals with diabetes. The team responsible for preparing the 2026 Recommendations sincerely hopes that, as in previous years, they will contribute to further improvement in the quality of diabetological care, which will directly translate into an improved quality of life for this particular population of people living with diabetes. For many years, we have been witnessing rapid developments in available technologies and pharmacotherapeutic options; however, diabetes – alongside obesity – continues to remain one of the key challenges of the 21st century.
Diabetes treatment must be individually tailored for each patient. It should take into account the various mechanisms leading to the development of the disease, require close and ongoing cooperation between therapeutic teams and individuals with diabetes, and involve an interdisciplinary, multi-pronged approach. When adjusting a treatment regimen to individual therapeutic goals, one should not overlook the quality of life mentioned above. Striving for optimal glycaemic control, weight reduction, and cardiovascular and renal protection, while ensuring the highest possible level of safety, is of fundamental importance in preventing and slowing the progression of diabetes-related complications. We are all aware that diabetes complications remain a central issue in contemporary diabetology, particularly from the patient's perspective.
Since 2005, Diabetes Poland has been preparing and publishing clinical recommendations on the management of people with diabetes. Updated annually, they are intended to follow the results of the latest clinical trials, indicate optimal management strategies, and support physicians in their daily clinical practice.
The initiative to develop those guidelines date back to 2004 and was introduced by Professor Jacek Sieradzki, who was President of Diabetes Poland at that time. The first Chair of the Diabetes Poland Recommendations Committee in 2005 was the late Professor Władysław Grzeszczak, who held this position until 2011. Subsequent representatives of the Main Board of Diabetes Poland for the Recommendations included Professor Leszek Czupryniak (2011–2015), Professor Dorota Zozulińska-Ziółkiewicz (2015–2019), and Professor Irina Kowalska (2019–2023). The current Recommendations, like previous editions, are the result of intensive teamwork by a large group of experts representing various fields of medicine and – importantly – also reflect the contributions of those who participated in the development of diabetes clinical recommendations in past years.

Summary of the most important changes in the Diabetes Poland Recommendations for 2026

In **CHAPTER 1**, an HbA_{1c} value in the range of 5.7–6.4% was introduced as a basis for the diagnosis of prediabetes. The chapter was also expanded to include a description of the clinical stages of type 1 diabetes.

In **CHAPTER 2**, information was provided on the approval of teplizumab, a drug inhibiting the progression of type 1 diabetes at stage 2. In addition, attention was drawn to the fact that GLP-1 receptor agonists (liraglutide, semaglutide) and the dual GIP/GLP-1 receptor agonist (tirzepatide) reduce the risk of developing type 2 diabetes in individuals with BMI ≥ 27 kg/m² and prediabetes. A recommendation was also added that treatment of obesity in individuals with prediabetes and in individuals without glucose metabolism disorders but with at least high cardiovascular risk should be provided under the care of a diabetology outpatient clinic.

In **CHAPTER 3**, attention was drawn to the need to consider the use of CGM systems in individuals treated with insulin using less frequent injection regimens, as well as in individuals not treated with insulin. In addition, minimum requirements for

quality parameters that CGM systems used in clinical practice should meet were presented. The table of example substances and clinical conditions that may interfere with glucose readings obtained using blood glucose meters was also updated.

In **CHAPTER 4**, the target triglyceride concentration was modified.

In **CHAPTER 5**, an updated and expanded model for organising medical care for people with diabetes and in diabetes prevention was presented.

In **CHAPTER 6**, the recommended duration of folic acid supplementation in the preconception period was modified.

In **CHAPTER 7**, the section addressing the health benefits of increased, regular physical activity was expanded. Attention was also drawn to the fact that the use of wearable activity trackers (e.g. smartwatches/fitness bands) constitutes a motivating factor for increasing physical activity. Recommended daily step counts for various patient groups were also defined.

In **CHAPTER 8**, assessment and monitoring of diabetes-related distress at least once a year as part of diabetology care were recommended. Suggested questionnaires, together with dedicated instructions for their use, are available in a designated section on the Diabetes Poland website.

CHAPTER 9 was developed in a new concept and format.

In **CHAPTER 10**, attention was drawn to the need to use only CGM systems meeting the required quality and safety standards. It was also indicated that AID systems should be offered to every person with type 1 diabetes, and that the decision on their potential implementation should be made through shared decision-making, taking reimbursement and financial issues into account.

In **CHAPTER 11**, the indications for the use of the dual GIP/GLP-1 receptor agonist were expanded based on its proven cardioprotective effect.

In **CHAPTER 12**, a recommendation was included that every change in the insulin therapy model should be accompanied by therapeutic re-education. The figure illustrating the insulin therapy algorithm was also modified.

CHAPTER 13 presents new content regarding the treatment of arterial hypertension. Two figures illustrating the presented recommendations were also prepared.

In **CHAPTER 14**, information was introduced regarding the current lack of specific therapies lowering Lp(a) concentration and the recommendation for early treatment of cardiovascular risk factors and more intensive lowering of LDL cholesterol levels. A link to a calculator enabling estimation of cardiovascular risk using Lp(a) was also provided. In the table concerning cardiovascular risk categorisation, a category of extremely high risk was added, including patients with atherosclerotic cardiovascular disease who experienced a recurrent cardiovascular event despite receiving the maximum tolerated statin dose, as well as patients with polyvascular atherosclerosis. For this group, a target LDL-C value of < 40 mg/dl was proposed. A table summarising types and causes of hypertriglyceridaemia was also introduced, and management of hypertriglyceridaemia was updated.

In **CHAPTER 15**, it was emphasised that glucose (or its isomer, dextrose) should be the first-line treatment for hypoglycaemia, and only in the absence of glucose should other simple carbohydrates be used. It was also added that physicians should regularly assess patients' cognitive function; in the event of deterioration, patients should be monitored more closely for hypoglycaemic episodes, preferably using CGM.

In **CHAPTER 16**, information was added to highlight the potential risk of diabetic ketoacidosis in individuals with type 1 diabetes who significantly reduce insulin doses as a result of treatment with GLP-1 receptor agonists or GIP/GLP-1 receptor agonists. The chapter was also supplemented with information that treatment of uncomplicated, mild diabetic ketoacidosis may be conducted using rapid-acting insulin analogues administered subcutaneously every 1–2 hours under glucose monitoring and continuation of basal insulin or using continuous subcutaneous insulin infusion with glucose monitoring. The level of scientific evidence for two of the most important recommendations was upgraded.

In **CHAPTER 18**, information was included stating that GLP-1 receptor agonists are recommended therapy for primary and secondary prevention of stroke in individuals with type 2 diabetes and should be recommended regardless of HbA_{1c} values. The chapter was also enriched with information on the epidemiology of stroke among individuals with diabetes. It was also emphasised that during the treatment of acute stroke, hyperglycaemia should be avoided, while recognising minimisation of hypoglycaemia risk as a priority.

In **CHAPTER 19**, situations in which nephrology consultation should be considered were clarified. In addition, in the table concerning dosing of non-insulin antihyperglycaemic agents, the row regarding sulfonylureas was expanded, taking into

account different available preparations in the context of their metabolism.

In **CHAPTER 20**, it was clarified that there is no need to perform screening ophthalmological examinations in women with gestational diabetes. It was also noted that if screening using a fundus camera and image assessment by qualified personnel or with the use of artificial intelligence-based image analysis software detects diabetic retinopathy, immediate referral to an ophthalmologist is recommended. The chapter also included a mention of non-arteritic anterior ischaemic optic neuropathy (NAION), with individual cases reported in individuals treated with GLP-1 receptor agonists, while noting that no causal association between GLP-1 receptor agonist therapy and the risk of developing retinopathy has been demonstrated.

In **CHAPTER 21,** recommendations were included regarding the selection of treatment for chronic neuropathic pain, assessment of its effectiveness, and setting therapeutic goals. Treatment algorithms for neuropathic pain were also updated.

In **CHAPTER 22**, it was indicated that efforts should be made to establish specialist wards or subunits for the treatment of diabetic foot disease. It was also clarified in which cases blood cultures should be performed in individuals with diabetic foot disease, and attention was drawn to the fact that a positive probe-to-bone test result suggests suspicion of osteomyelitis.

In **CHAPTER 23**, a recommendation was added that in children with type 1 diabetes during disease remission or when using AID systems, a narrow target range (TITR) of 70–140 mg/dl should be assessed. In patients treated with multiple daily insulin injections, the use of technologies supporting recording of administered insulin doses was recommended. In children with type 1 diabetes and coexisting obesity, the addition of a GLP-1 receptor agonist to therapy was recommended. The age at which screening tests for detection of preclinical stages of type 1 diabetes in children should be performed was proposed. It was recommended that CGM be used periodically in patients at stage 2 of type 1 diabetes. It was emphasised that therapeutic education in preclinical stages should cover topics relevant to this stage of the disease. The age above which screening for dysglycaemia should be performed in children with BMI > 85th percentile was lowered to 8 years, with repeat testing every 2 years. In children with type 2 diabetes, consideration of initiating therapy with a GLP-1 receptor agonist was recommended if body weight does not normalise despite stabilisation of glycaemic values. Recommendations concerning inpatient and outpatient diabetology care for children and adolescents with diabetes were modified.

In **CHAPTER 24**, a provision was added that standard preconception care should include, in addition to achieving glycaemic targets, nutrition, physical activity, education on self-monitoring, and screening for comorbidities and diabetes complications. A recommendation was also introduced that in women with pregestational diabetes, regardless of diabetes type, ophthalmological examination should be performed before conception or at the latest in the first trimester and repeated in each trimester. It was also indicated that GLP-1 receptor agonists and GIP/GLP-1 receptor agonists lack studies allowing their use during pregnancy and therefore should not be used during pregnancy planning or the reproductive period if effective contraception is not applied. Administration of these agents should be discontinued 4–8 weeks before planned pregnancy, and immediately after confirmation of an unplanned pregnancy. In addition, recommendations for the treatment of arterial hypertension in pregnancy were unified with those in Chapter 13, and, similarly to Chapter 6, the recommended duration of folic acid supplementation in the preconception period was modified.

In **CHAPTER 25**, a recommendation was added that individuals aged over 65 years should be assessed at least once a year for geriatric syndromes, hypoglycaemia, and polypharmacy, as these may affect diabetes treatment and reduce quality of life. Attention was also drawn to ensuring adequate protein intake in the diet. It was added that in individuals over 65 years of age with type 1 diabetes, the use of AID systems may be considered.

In **CHAPTER 26**, it was indicated that before gastrointestinal endoscopic procedures in individuals treated with GLP-1 receptor agonists or GIP/GLP-1 receptor agonists, omission of one dose of the drug before the planned procedure should be considered (a minimum of 7 days without drug activity). Additionally, the table presenting perioperative recommendations for non-insulin antihyperglycaemic agents was expanded with information regarding incretin-based therapies.

In **CHAPTER 27**, information was updated in accordance with the latest recommendations regarding preventive vaccinations.

In **CHAPTER 31**, it was emphasised that MASLD constitutes a non-classical complication of diabetes. The subsection on the treatment of MASH was updated, indicating resmetirom as the only drug

registered in Europe and mentioning semaglutide (registered in the USA). Attention was also drawn to the necessity of behavioural modification, including diet, physical activity, weight reduction, smoking cessation, and avoidance of alcohol consumption, as one of the pillars of MASLD treatment. A new subsection concerning hypogonadism in men with diabetes was also added.

In all chapters, the cited literature was updated.

In **APPENDIX 1** (formerly Appendix 7), significant modifications were introduced, presenting current organisational requirements for diabetology care in adults.

In **APPENDIX 5,** it was emphasised that providing care for individuals treated with PIP or AID within coordinated care models lacking an appropriate therapeutic team is not recommended. Indications and contraindications for reimbursement of PIP and for continuation of PIP therapy and reimbursement of equipment by the National Health Fund were modified, as well as the scope of activities involved in initiation of PIP or AID therapy in the initiating centre. Tables presenting mandatory and recommended specifications for PIP were also updated.

In **APPENDIX 7** (formerly Appendix 1), the need to assess mental health and psychosocial problems in individuals with type 1 diabetes transferred from paediatric diabetology care to adult care or in the event of a change of diabetology clinic was highlighted. The Diabetology Care Information Card was also simplified.

We would like to sincerely thank everyone who contributed to the preparation of this new edition of the Diabetes Poland Recommendations.

Professor Janusz Gumprecht, MD, PhD
Plenipotentiary of the Main Board of Diabetes Poland for Clinical Recommendations

Professor Irina Kowalska, MD, PhD
President of Diabetes Poland

List of Abbreviations

ABI – Ankle–Brachial Index
ABPM – Ambulatory Blood Pressure Monitoring
ACE – Angiotensin-Converting Enzyme
ACEI – Angiotensin-Converting Enzyme Inhibitors
ACS – Acute Coronary Syndrome
ACR – Albumin-to-Creatinine Ratio
ADA – American Diabetes Association
AER – Albumin Excretion Rate
AF – Atrial Fibrillation
AID – Automated Insulin Delivery
ALT – Alanine Aminotransferase
anti-GAD – Glutamic Acid Decarboxylase Antibodies
anti-HBs – Hepatitis B Surface Antibody
apoB – Apolipoprotein B
APTT – Activated Partial Thromboplastin Time
APS – Artificial Pancreas System
ARB – Angiotensin Receptor Blockers
ARNI – Angiotensin Receptor–Neprilysin Inhibitors
ASA – Acetylsalicylic Acid
ASCVD – Atherosclerotic Cardiovascular Disease
AST – Aspartate Aminotransferase
AT1 – Angiotensin II Type 1 Receptor
ATP – Adenosine Triphosphate
BCVA – Best-Corrected Visual Acuity
BE – Base Excess
BGM – Blood Glucose Monitoring
BMI – Body Mass Index
BP – Blood Pressure
CAD – Coronary Artery Disease
CCB – Calcium Channel Blockers
CCS – Chronic Coronary Syndrome
CFRD – Cystic Fibrosis-Related Diabetes
CGM – Continuous Glucose Monitoring
CK – Creatine Kinase
CKD – Chronic Kidney Disease
CME – Cystoid Diabetic Macular Oedema
CRT – Cardiac Resynchronisation Therapy
CSII – Continuous Subcutaneous Insulin Infusion
CV – Coefficient of Variation
CVOT – Cardiovascular Outcome Trial
DAPT – Dual Antiplatelet Therapy
DASH – Dietary Approaches to Stop Hypertension
DBP – Diastolic Blood Pressure
DFS – Diabetic Foot Syndrome
DIY – Do-It-Yourself (insulin delivery systems)
DKA – Diabetic Ketoacidosis
DME – Diabetic Macular Oedema
DN4 – Douleur Neuropathique en 4 Questions
DPP-4 – Dipeptidyl Peptidase-4
DPP-4i – Dipeptidyl Peptidase-4 Inhibitor
DRIL – Disorganisation of the Retinal Inner Layers
DRT – Diffuse Diabetic Macular Oedema
DSMES – Diabetes Self-Management Education and Support
EASD – European Association for the Study of Diabetes
ECG – Electrocardiogram
ED-NOS – Eating Disorders Not Otherwise Specified
eGFR – Estimated Glomerular Filtration Rate
EPA – Eicosapentaenoic Acid
ESC – European Society of Cardiology
ESH – European Society of Hypertension
ETDRS – Early Treatment Diabetic Retinopathy Study
FGM – Flash Glucose Monitoring
FIB-4 – Fibrosis-4 Index
FRC – Fixed-Ratio Combination
GDM – Gestational Diabetes Mellitus
GI – Glycaemic Index
GIK – Glucose–Insulin–Potassium
GIP – Glucose-Dependent Insulinotropic Polypeptide
GL – Glycaemic Load
GLP-1 – Glucagon-Like Peptide-1
GLP-1 RA – Glucagon-Like Peptide-1 Receptor Agonist
GMI – Glucose Management Indicator
HAV – Hepatitis A Virus
HbA_{1c} – Glycated Haemoglobin
HCL – Hybrid Closed Loop
HDL-C – High-Density Lipoprotein Cholesterol
HF – Heart Failure
HFmEF – Heart Failure with Mildly Reduced Ejection Fraction
HFpEF – Heart Failure with Preserved Ejection Fraction
HFrEF – Heart Failure with Reduced Ejection Fraction
HHS – Hyperosmolar Hyperglycaemic State
HTG – Hypertriglyceridaemia
HTN – Hypertension
Hz – Hertz
IA-2 – Insulinoma-Associated Autoantigen 2 Antibodies
IAA – Insulin Autoantibodies
ICA – Islet Cell Autoantibodies
ICD – Implantable Cardioverter-Defibrillator
IFG – Impaired Fasting Glucose
IgA – Immunoglobulin A
IGT – Impaired Glucose Tolerance
IIT – Intensive Insulin Therapy

INR – International Normalised Ratio
ISPAD – International Society for Pediatric and Adolescent Diabetes
KDIGO – Kidney Disease: Improving Global Outcomes
LADA – Latent Autoimmune Diabetes in Adults
LDL-C – Low-Density Lipoprotein Cholesterol
Lp(a) – Lipoprotein(a)
LVEF – Left Ventricular Ejection Fraction
LVH – Left Ventricular Hypertrophy
MASLD – Metabolic Dysfunction–Associated Steatotic Liver Disease
MASH – Metabolic Dysfunction–Associated Steatohepatitis
MDCT – Multidetector Computed Tomography
MDI – Multiple Daily Injections
MELAS – Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like Episodes
MIDD – Maternally Inherited Diabetes and Deafness
MODY – Maturity-Onset Diabetes of the Young
MRA – Mineralocorticoid Receptor Antagonist
MRI – Magnetic Resonance Imaging
MRSA – Methicillin-Resistant *Staphylococcus aureus*
Na – Sodium
NaCl – Sodium Chloride
$NaHCO_3$ – Sodium Bicarbonate
NAION – Non-Arteritic Anterior Ischemic Optic Neuropathy
NFG – Normal Fasting Glucose
NGSP – National Glycohemoglobin Standardization Program
non-HDL-C – Non–High-Density Lipoprotein Cholesterol
NPDR – Non-Proliferative Diabetic Retinopathy
ns-MRA – Non-Steroidal Mineralocorticoid Receptor Antagonist
OCT – Optical Coherence Tomography
OGTT – Oral Glucose Tolerance Test
PAD – Peripheral Arterial Disease
PCI – Percutaneous Coronary Intervention
PCOS – Polycystic Ovary Syndrome
PDR – Proliferative Diabetic Retinopathy
PGDM – Pregestational Diabetes Mellitus
PHQ-9 – Patient Health Questionnaire-9
POCT – Point-of-Care Testing
PPAR-γ – Peroxisome Proliferator-Activated Receptor Gamma
RAAS – Renin–Angiotensin–Aldosterone System
RAS – Renin–Angiotensin System
rTMS – Repetitive Transcranial Magnetic Stimulation
SADI – Single Anastomosis Duodeno–Ileal Bypass
SBP – Systolic Blood Pressure
SCS – Spinal Cord Stimulation
SGLT2 – Sodium–Glucose Cotransporter 2
SGLT2i – Sodium–Glucose Cotransporter 2 Inhibitor
SI – International System of Units
SINBAD – Site, Ischaemia, Neuropathy, Bacterial Infection, Area, Depth
SMBG – Self-Monitoring of Blood Glucose
SNRI – Serotonin–Noradrenaline Reuptake Inhibitor
SPC – Single-Pill Combination
SPECT – Single-Photon Emission Computed Tomography
SU – Sulfonylureas
T1DM – Type 1 Diabetes Mellitus
T2DM – Type 2 Diabetes Mellitus
TAR – Time Above Range
TBR – Time Below Range
TCA – Tricyclic Antidepressants
$TcPO_2$ – Transcutaneous Oxygen Pressure
TG – Triglycerides
TIR – Time in Range
TITR – Time in Tight Range
TOD – Target Organ Damage
UACR – Urine Albumin-to-Creatinine Ratio
ULN – Upper Limit of Normal
USG – Ultrasonography
VAS – Visual Analogue Scale
VEGF – Vascular Endothelial Growth Factor
WHO – World Health Organization
ZnT8 – Zinc Transporter 8 Autoantibodies

1. Diagnosing Glucose Tolerance Disorders

CHAPTER HIGHLIGHTS
• Glucose tolerance disorders are diagnosed based on glycaemia: fasting plasma glucose, plasma glucose at 120 minutes of the oral glucose tolerance test, random plasma glucose, as well as HbA_{1c} results. [**A**]
• Screening for the early diagnosis of prediabetes/type 2 diabetes should be conducted in all individuals aged over 45 years and, irrespective of age, in all individuals in whom at least one risk factor for diabetes is present. [**B**]
• In all pregnant women in whom diabetes has not previously been diagnosed, diagnostic testing should be performed to detect glucose tolerance disorders. [**A**]
• The diagnosis of diabetes in children within the first 6–12 months of life requires genetic testing for permanent neonatal diabetes. [**A**]
• In individuals with cystic fibrosis, an oral glucose tolerance test (OGTT) should be performed annually after the age of 10 years to diagnose diabetes. [**A**]

Diabetes is a group of metabolic diseases characterised by hyperglycaemia resulting from defects in insulin secretion and/or insulin action. Chronic hyperglycaemia is associated with damage to, dysfunction of, and failure of various organs, particularly the eyes, kidneys, nerves, heart, and blood vessels.

I. Symptoms of hyperglycaemia indicating the possibility of diabetes:

- polyuria (increased diuresis),
- increased thirst,
- unintentional weight loss,
- other, less typical symptoms: weakness and increased sleepiness, purulent skin lesions, and inflammation of the genitourinary tract.

II. Terminology and diagnostic criteria for glucose tolerance disorders:

In the presence of symptoms of diabetes, random plasma glucose should be measured. A result of ≥ 200 mg/dl (≥ 11.1 mmol/l) is diagnostic of diabetes.

Fasting plasma glucose, plasma glucose at 120 minutes of the OGTT, and measurement of glycated haemoglobin (HbA_{1c}) may be considered equally valid diagnostic criteria, even though they identify diabetes in different individuals. Compared with fasting plasma glucose and HbA_{1c}, measurement of plasma glucose at 120 minutes of the OGTT allows diabetes and prediabetes to be detected in a greater number of individuals.

The principles of diagnostics and diagnostic criteria for hyperglycaemic states in pregnancy are discussed in Chapter 24.

In individuals receiving antihyperglycaemic medications for reasons other than type 2 diabetes, the basis for the diagnosis of the disease is an HbA_{1c} result ≥ 6.5% (≥ 48 mmol/mol).

III. Principles of performing diagnostic tests

- The OGTT should be performed without prior restriction of carbohydrate intake, in the morning, in a fasting individual (at least 8 hours without food and fluids other than water), who is well rested after a night's sleep. Between

Table 1.1. Terminology of hyperglycaemic states (according to WHO 1999, 2019; modified)

Glycaemic parameter	Normal values	Prediabetes		Diabetes
		Impaired fasting glucose (IFG)	Impaired glucose tolerance (IGT)	
Venous plasma glucose concentration, fasting*	< 100 mg/dl (5.6 mmol/l)	100–125 mg/dl (5.6–6.9 mmol/l)		≥ 126 mg/dl (≥ 7.0 mmol/l)
Venous plasma glucose concentration at 120 minutes of the OGTT	< 140 mg/dl (7.8 mmol/l)		140–199 mg/dl (7.8–11.0 mmol/l)	≥ 200 mg/dl (≥ 11.1 mmol/l)
Glycated haemoglobin (HbA_{1c})	< 5.7% (39 mmol/mol)	5.7–6.4% (39–47 mmol/mol)		≥ 6.5% (≥ 48 mmol/mol)

OGTT – oral glucose tolerance test

**For the diagnosis of diabetes, it is necessary to identify one abnormality, with the exception of fasting plasma glucose, for which confirmation of the abnormal result on two separate occasions is required.*

ingestion of a solution containing 75 g of glucose and blood sampling, the tested individual should remain at rest at the testing site for 2 hours; all measurements of plasma glucose concentration should be performed in the laboratory using venous plasma.

- If it is necessary to perform an OGTT in an individual with glucose intolerance (i.e. prediabetes) who is receiving metformin for this reason, metformin should be discontinued for at least one week prior to the day on which the OGTT is performed:
  - » glycaemia measurements used for diagnostic purposes should be performed in the laboratory using venous plasma; the use of glucometers or continuous glucose monitoring systems for this purpose is not permitted;
  - » HbA_{1c} measurements should be performed in the laboratory using methods certified by the National Glycohemoglobin Standardization Program (NGSP); HbA_{1c} measurements should not be performed for diagnostic purposes using point-of-care testing (POCT) analysers, even if certified by the NGSP;
- HbA_{1c} should not be used for the diagnosis of diabetes in individuals with conditions that interfere with the relationship between HbA_{1c} values and mean glycaemia, such as anaemias, certain haemoglobinopathies, and HIV infection, as well as in individuals undergoing haemodialysis, receiving erythropoietin or antiretroviral therapy, and in women during pregnancy and the postpartum period; in such individuals, diagnostic criteria based on plasma glucose concentration should be used.

Table 1.2. Risk factors for type 2 diabetes

• overweight or obesity (BMI ≥ 25 kg/m² and/or waist circumference ≥ 80 cm in women or ≥ 94 cm in men)
• cardiovascular disease
• diabetes in the family history (parents or siblings)
• prediabetes
• previous gestational diabetes
• arterial hypertension
• dyslipidaemia
• fatty liver disease
• low level of physical activity
• in women who have given birth to a child weighing > 4000 g or weight of a newborn > 4000 g
• polycystic ovary syndrome (PCOS)
• belonging to an environmental or ethnic group at increased risk of diabetes

IV. Screening for type 2 diabetes:

- screening for diabetes should be performed once every 3 years in all individuals aged over 45 years;
- irrespective of age, such screening should be performed annually in all individuals in whom at least one risk factor for type 2 diabetes is present (Table 1.2).

V. Aetiological classification of diabetes

The classification of diabetes according to the World Health Organization (WHO) is presented in Table 1.3.

Type 1 diabetes can currently be detected in the preclinical phase (Table 1.4). Diagnostic evaluation includes the measurement of autoantibodies: antibodies against glutamic acid decarboxylase (glutamic acid decarboxylase antibodies – anti-GAD), antibodies against insulin (insulin autoantibodies – IAA), antibodies against tyrosine phosphatase 2 (insulinoma-associated autoantigen 2 – IA-2), antibodies against zinc transporter 8 (zinc transporter family member 8 – ZnT8), and, optionally, antibodies against undefined islet antigens (islet cell antibodies – ICA). These tests should be performed in a reference laboratory.

If a positive titre of at least two autoantibodies is identified, an assessment of the degree of impairment of glucose tolerance should be performed, including HbA_{1c} and an OGTT. Table 1.4

Table 1.3. Aetiological classification of diabetes (WHO 1999, 2019)

Type 1 diabetes – autoimmune destruction of pancreatic β cells, usually leading to absolute insulin deficiency (including the former entity LADA – latent autoimmune diabetes in adults).
Type 2 diabetes – progressive loss of pancreatic β-cell capacity for adequate insulin secretion, accompanied by insulin resistance.
Other specific types of diabetes: • genetic defects of β-cell function • genetic defects of insulin action • diseases of the exocrine pancreas (pancreatitis, following pancreatic resection, pancreatic neoplasms) • endocrinopathies • drugs and other chemical substances • infections • genetic syndromes sometimes associated with diabetes • rare forms of immune-mediated diabetes
Hyperglycaemia first identified during pregnancy: • diabetes in pregnancy • gestational diabetes mellitus

Table 1.4. Clinical stages of type 1 diabetes

	Stage 1	Stage 2	Stage 3
Characteristics:	• autoimmunity • normoglycaemia • asymptomatic	• autoimmunity • dysglycaemia • asymptomatic	• autoimmunity • diagnostic criteria for diabetes fulfilled
Diagnostic criteria:	• presence of at least two positive autoantibodies against pancreatic islet β cells • absence of IFG and IGT • normal HbA_{1c}	• presence of at least two positive autoantibodies against pancreatic islet β cells • dysglycaemia: IFG and/or IGT, and/or HbA_{1c} 5.7–6.4%, and/or an increase in HbA_{1c} of ≥ 10% compared with baseline	• autoantibodies present, although they may disappear over time • diagnosis of diabetes according to established diagnostic criteria

presents the diagnostic criteria for the individual stages of type 1 diabetes.

In an adult with diagnosed diabetes, if there are doubts regarding classification, the diagnosis of type 1 diabetes requires demonstration of the presence of autoantibodies and/or a C-peptide concentration < 200 mmol/l measured 1–6 hours after a meal. As a first step, antibodies against glutamic acid decarboxylase (anti-GAD) should be measured. If the result is negative, antibodies against tyrosine phosphatase 2 (anti-IA2) and/or insulin (IAA) and/or zinc transporter 8 (anti-ZnT8) should be measured next, provided these tests are available.

Monogenic diabetes

Monogenic diabetes accounts for up to 6% of all cases of diabetes. It results from mutations in a single gene. Most of its forms are associated with defects in insulin secretion, and the most common include:

- MODY diabetes (maturity onset diabetes of the young),
- permanent neonatal diabetes mellitus (PNDM) and transient neonatal diabetes mellitus (TNDM),
- mitochondrial diabetes.

Including monogenic forms of diabetes in the differential diagnosis may contribute to optimisation of treatment and to establishing an appropriate prognosis for the patient and their family members. A definitive diagnosis of monogenic diabetes is established based on the results of genetic testing in conjunction with the patient's phenotype. Given that more than 40 different types of monogenic diabetes have been identified to date, next-generation sequencing (NGS) is currently regarded as the "gold standard" for such diagnostics, as it enables simultaneous analysis of a panel of approximately 40 associated genes. However, analysis of genetic variants in genes associated with monogenic diabetes should be performed exclusively in certified laboratories with experience in conducting this type of testing.

Qualification for and interpretation of genetic testing for monogenic diabetes, as well as any therapeutic decisions resulting from such a diagnosis, should be undertaken by centres with appropriate expertise and experience in this field, confirmed by certification from Diabetes Poland.

Syndromic forms of monogenic diabetes require extensive experience in the interpretation of multiple clinical and laboratory features present in the patient in relation to the presence of a causative variant in a specific gene; therefore, they should be analysed exclusively in centres experienced in diabetes genetics, in cooperation with a clinical geneticist.

MODY diabetes

In families in which autosomal dominant, early-onset diabetes resulting from impaired insulin secretion is present and is not accompanied by obesity in the majority of cases, MODY diabetes should be considered in the differential diagnosis, and mutations in the genes responsible for its development should be sought. The most common forms of MODY diabetes are associated with mutations in the *HNF1A* gene (HNF1A-MODY; formerly MODY3) and the glucokinase gene (GCK-MODY; formerly MODY2), which together with mutations in the *HNF4A* gene (HNF4A-MODY; formerly MODY1) account for approximately 70–80% of all forms of monogenic diabetes. In order to identify patients whose clinical and laboratory features suggest a high probability of MODY diabetes, the use of the MODY probability calculator (www.diabetesgenes.org/mody-probability-calculator/) may also be helpful.

The typical clinical picture of patients with **HNF1A-MODY** includes:

- early onset of diabetes (typically before the age of 25–35 years),

- lack of insulin dependence and lack of a tendency to ketoacidosis, low insulin requirements, and detectable C-peptide despite several years or even longer duration of the disease,
- a family history of diabetes involving at least two generations (early-onset diabetes in at least two family members, usually without obesity); an OGTT performed at an early stage of diabetes usually shows a marked increase in glycaemia, often with normal fasting values,
- absence of autoantibodies typical of type 1 diabetes,
- glycosuria greater than would be expected based on glycaemic values.

In a significant proportion of patients with HNF1A-MODY, chronic diabetic complications develop; therefore, optimal glycaemic control should be pursued from the onset of the disease. The treatment of choice (except during pregnancy or in the presence of typical contraindications) is the initiation of sulfonylurea derivatives. Once their effectiveness is exhausted, combination therapy with insulin and/or other antihyperglycaemic agents should be considered.

Confirmation of **GCK-MODY** should be considered in the following situations:

- fasting hyperglycaemia,
- an increase in glycaemia during the OGTT that in most cases is below 54 mg/dl (3 mmol/l),
- HbA_{1c} values typically just above the upper limit of normal, only occasionally exceeding 7.5% (58 mmol/mol),
- one parent with diagnosed hyperglycaemia or diabetes; however, the absence of a positive family history does not exclude this form of the disease.

In the case of confirmed GCK-MODY, the treatment of choice is a low glycaemic index diet and physical activity; pharmacotherapy is usually not required. Insulin therapy may be required in women with GCK-MODY during pregnancy.

Permanent neonatal diabetes mellitus is defined as diabetes developing before the age of 6–12 months of age. All patients with neonatal diabetes should undergo genetic testing. This should include testing for mutations in the *KCNJ11* and *ABCC8* genes, in which causative variants are the most common cause of permanent neonatal diabetes, and their confirmation constitutes an indication for treatment with sulfonylurea derivatives. In the case of transient neonatal diabetes mellitus, in which most patients have abnormalities in the 6q24 region (6q24-TNDM), the method of choice is MS-MLPA (methylation-specific multiplex ligation-dependent probe amplification). Confirmation of a diagnosis of 6q24-TNDM is associated with a risk of recurrence of diabetes after several years of complete clinical remission and then constitutes an indication to attempt treatment with sulfonylurea derivatives.

The most common cause of **mitochondrial diabetes** is the *m.3243A>G* mutation of the leucine tRNA gene. Testing for this mutation in mitochondrial DNA should be performed in cases of maternally inherited, early-onset diabetes. This condition is currently classified as a syndromic form of monogenic diabetes due to the presence of numerous additional manifestations that may occur alongside diabetes. This form of diabetes is often accompanied by progressive hearing impairment (MIDD – maternally inherited diabetes with deafness), as well as short stature and visual disturbances. The same mitochondrial DNA mutation may also cause MELAS syndrome (mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes), in the course of which diabetes may occur alongside myopathy, hearing impairment, encephalopathy, cardiomyopathy, and visual disturbances. Therapeutic management of mitochondrial diabetes may include dietary measures and the use of sulfonylurea derivatives or insulin, depending on the degree of insulin secretion defect. Metformin therapy should be avoided in mitochondrial diabetes.

Diabetes in individuals with cystic fibrosis

Diabetes occurs in approximately 20% of adolescents and 40–50% of adults with cystic fibrosis and represents the most common comorbidity. Cystic fibrosis–related diabetes (CFRD) belongs to other specific types of diabetes associated with diseases of the exocrine pancreas; it develops slowly and is usually asymptomatic for many years. Diabetic ketoacidosis occurs rarely, most likely due to preserved endogenous insulin secretion. Initially, hyperglycaemia is usually observed in situations that exacerbate insulin resistance, such as acute and chronic infections, glucocorticosteroid therapy, or the intake of large amounts of carbohydrates (oral, intravenous, via a gastric tube, or via percutaneous gastrostomy). Insulin therapy is the treatment of choice.

In individuals with cystic fibrosis aged over 10 years, an OGTT should be performed annually during periods of good health in order to detect

diabetes; it is recommended that glycaemia be measured at least in the fasting state and 2 hours after a glucose load of 1.75 g/kg body weight.

REFERENCES

1. Chung WK, Erion K, Florez JC, et al. Precision medicine in diabetes: a Consensus Report from the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetologia 2020; 63: 1671–1693.
2. Colclough K, Ellard S, Hattersley A, Patel K. Syndromic Monogenic Diabetes Genes Should Be Tested in Patients With a Clinical Suspicion of Maturity-Onset Diabetes of the Young. Diabetes 2022; 71: 530–537.
3. De Franco E, Flanagan SE, Houghton JAL, et al. The effect of early, comprehensive genomic testing on clinical care in neonatal diabetes: an international cohort study. Lancet 2015; 386: 957–963.
4. Genuth S, Alberti KG, Bennett P, et al. Expert Committee on the diagnosis and classification of diabetes mellitus. Follow-up report on the diagnosis of diabetes mellitus. Diabetes Care 2003; 26: 3160–3167.
5. Greeley SAW, Polak M, Njølstad PR, et al. ISPAD Clinical Practice Consensus Guidelines 2022: The diagnosis and management of monogenic diabetes in children and adolescents. Pediatr Diabetes 2022; 23: 1188–1211.
6. Holt RIG, de Vries JH, Hess-Fischl A, et al. The management of type 1 diabetes in adults. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetologia 2021; 64: 2609–2652.
7. International Expert Committee. International Expert Committee report on the role of the A1C assay in the diagnosis of diabetes. Diabetes Care 2009; 32: 1327–1334.
8. Little RR, Rohlfing C, Sacks DB. The NGSP: over 20 years of improving HbA1c measurement. Clin Chem 2019; 65: 839–848.
9. Pietrusiński M, Grzybowska-Adamowicz J, Płoszaj T, et al. The Clinical and Diagnostic Characterization of 6q24-Related Transient Neonatal Diabetes Mellitus: A Polish Pediatric Cohort Study. Biomedicines 2025; 13: 2492. DOI: https://doi.org/10.3390/biomedicines13102492.
10. Saint-Martin C, Bouvet D, Bastide M, Bellanné-Chantelot C. Gene Panel Sequencing of Patients With Monogenic Diabetes Brings to Light Genes Typically Associated With Syndromic Presentations. Diabetes 2022; 71: 578–584.
11. World Health Organization. Classification of diabetes mellitus 2019. https://www.who.int/publications/i/item/classification-of-diabetes-mellitus.
12. Zieleniewska NA, Szum-Jakubowska A, Chlabicz M, et al. The prevalence of diabetes and prediabetes: a population-based study. Pol Arch Intern Med 2023; 133: 16407. DOI: 10.20452/pamw.16407.

2. Prevention and Delay of Diabetes Development

CHAPTER HIGHLIGHTS
• In patients with overweight or obesity, active screening for prediabetes should be undertaken, particularly in individuals with other features of the metabolic syndrome, allowing earlier implementation of preventive and therapeutic interventions aimed at reducing the risk of type 2 diabetes and cardiovascular disease. [**A**]
• Patients with prediabetes should receive recommendations regarding a healthy lifestyle (physical activity of at least 150 minutes per week; in individuals with overweight or obesity, body weight reduction of at least 7% over one year and its maintenance), as well as information on the effectiveness of such measures in preventing progression to diabetes. [**A**]
• GLP-1 receptor agonists (liraglutide, semaglutide) or the dual GIP/GLP-1 receptor agonist (tirzepatide) reduce the risk of developing type 2 diabetes in individuals with BMI ≥ 27 kg/m² and prediabetes. [**B**]
• In individuals with prediabetes, particularly those with concomitant impaired fasting glucose and impaired glucose tolerance and/or a body mass index ≥ 35 kg/m² and/or age below 60 years, as well as in women with a history of gestational diabetes, pharmacological prevention of diabetes in the form of metformin should be considered in parallel with lifestyle modification. [**A**]
• Screening should be performed by measuring fasting plasma glucose, performing an oral glucose tolerance test (OGTT), or measuring HbA_{1c}. [**C**]

Type 1 diabetes

Currently, there is no clinically available method for preventing type 1 diabetes, either in the general population or in individuals at increased risk. Teplizumab is the only approved medication that inhibits the progression of type 1 diabetes at stage 2.

This therapy is not available in Poland.

Type 2 diabetes

1. Screening may be performed by measuring random plasma glucose, fasting plasma glucose, performing an OGTT, or measuring HbA_{1c}. The OGTT remains the most sensitive method for detecting prediabetes.
2. Risk factors for type 2 diabetes: see Chapter 1.
3. Overview of recommendations for the prevention or delay of the onset of diabetes:

- individuals at high risk should receive appropriate education on the role of healthy lifestyle principles in the prevention of type 2 diabetes;
- all individuals benefit from increased physical activity regardless of age; however, the highest effectiveness of such intervention should be emphasised in individuals aged over 60 years;
- individuals with prediabetes should receive recommendations regarding a healthy lifestyle – body weight reduction of at least 7% over one year in individuals with overweight or obesity and its maintenance through physical activity adapted to the patient's abilities (at least 150 minutes per week), as well as adherence to an appropriate diet, together with information on the effectiveness of such measures in reducing the risk of developing diabetes;
- in individuals with prediabetes, particularly those with concomitant impaired fasting glucose and impaired glucose tolerance and/or a body mass index ≥ 35 kg/m² and/or age below 60 years, as well as in women with a history of gestational diabetes mellitus (GDM), pharmacological prevention of type 2 diabetes in the form of metformin should be considered in parallel with lifestyle modification;
- in individuals with BMI ≥ 27 kg/m² and prediabetes, in addition to behavioural treatment, pharmacotherapy with GLP-1 receptor agonists (liraglutide, semaglutide) or the dual GIP/GLP-1 receptor agonist (tirzepatide), with proven efficacy in reducing the risk of type 2 diabetes in this group, should be considered;
- when non-pharmacological management of obesity does not result in sufficient weight reduction, initiation of pharmacological treatment or referral for metabolic surgery should not be delayed;
- metabolic surgery is effective in the prevention of type 2 diabetes, particularly in individuals with prediabetes;
- due to the shared pathogenesis of obesity, type 2 diabetes, and cardiovascular disease, in individuals with obesity without a diagnosis of diabetes but with cardiovascular disease, including heart failure with preserved ejection fraction, and with cardiovascular risk factors, including prediabetes, pharmacotherapy for obesity with a GLP-1 receptor agonist (semaglutide) or the dual GIP/GLP-1 receptor agonist (tirzepatide), with proven cardiovascular risk reduction or improvement in heart failure symptoms in this group, should be considered;
- it is recommended that treatment of obesity in individuals with prediabetes and in individuals without disturbances of glucose metabolism but with very high or high cardiovascular risk be **provided within a diabetology outpatient clinic**;
- repeated lifestyle counselling at every patient visit is of key importance for the effectiveness of prevention of disorders of glucose metabolism;
- regular assessment in individuals with prediabetes for the presence of other cardiovascular risk factors (e.g. obesity, tobacco smoking, arterial hypertension, lipid disorders) is recommended, and if present, appropriate treatment should be initiated; treatment targets for comorbid conditions in individuals with prediabetes are the same as for the general population;
- the use of diabetogenic medications should be avoided.

REFERENCES

1. Butler J, Shah SJ, Petrie MC, et al. Semaglutide versus placebo in people with obesity-related heart failure with preserved ejection fraction: a pooled analysis of the STEP-HFpEF and STEP-HFpEF DM randomised trials. Lancet 2024; 403: 1635–1648.
2. Carlsson LMS, Sjöholm K, Jacobson P, et al. Life expectancy after bariatric surgery in the Swedish Obese Subjects Study. N Engl J Med 2020; 383: 1535–1543.
3. Deanfield J, Verma S, Scirica BM, et al.; SELECT Trial Investigators. Semaglutide and cardiovascular outcomes in patients with obesity and prevalent heart failure: a prespecified analysis of the SELECT trial. Lancet. 2024; 404: 773–786.
4. Diabetes Prevention Program Research Group. Long-term effects of metformin on diabetes prevention: identification of subgroups that benefited most in the diabetes prevention program and diabetes prevention program outcomes study. Diabetes Care 2019; 42: 601–608.
5. Diabetes Prevention Program Research Group. The 10-year cost-effectiveness of lifestyle intervention or metformin for diabetes prevention: an intent-to-treat analysis of the DPP/DPPOS. Diabetes Care 2012; 35: 723–730.

6. Hankosky ER, Wang H, Neff LM, et al. Tirzepatide reduces the predicted risk of atherosclerotic cardiovascular disease and improves cardiometabolic risk factors in adults with obesity or overweight: SURMOUNT-1 post hoc analysis. Diabetes Obes Metab 2024; 26: 319–328.
7. Haw JS, Galaviz KI, Straus AN, et al. Long-term sustainability of diabetes prevention approaches: a systematic review and meta-analysis of randomized clinical trials. JAMA Intern Med 2017; 177: 1808–1817.
8. Herold KC, Gitelman SE, Gottlieb PA, et al. Teplizumab: a disease-modifying therapy for type 1 diabetes that preserves β-cell function. Diabetes Care 2023; 46: 1848–1856.
9. Karczewska-Kupczewska M, Nikołajuk A, Kondraciuk M, et al. The relationships between FLAIS, a novel insulin sensitivity index, and cardiovascular risk factors in a population-based study. Cardiovasc Diabetol 2022; 21: 55.
10. Knowler WC, Barrett-Connor E, Fowler SE, et al.; Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med 2002; 346: 393–403.
11. Le Roux CW, Astrup A, Fujioka K, et al. SCALE Obesity prediabetes NN8022-1839 study group. 3 years of liraglutide versus placebo for type 2 diabetes risk reduction and weight management in individuals with prediabetes: a randomised, double-blind trial. Lancet 2017; 389: 1399–1409.
12. Packer M, Zile MR, Kramer CM, et al.; SUMMIT Trial Study Group. Tirzepatide for heart failure with preserved ejection fraction and obesity. N Engl J Med 2025; 392: 427–437.
13. Perreault L, Davies M, Frias JP, et al. Changes in glucose metabolism and glycemic status with once-weekly subcutaneous semaglutide 2.4 mg among participants with prediabetes in the STEP Program. Diabetes Care 2022; 45: 2396–2405.
14. Ratner RE, Christophi CA, Metzger BE, et al.; Diabetes Prevention Program Research Group. Prevention of diabetes in women with a history of gestational diabetes: effects of metformin and lifestyle interventions. J Clin Endocrinol Metab 2008; 93: 4774–4779.
15. Sigal RJ, Alberga AS, Goldfield GS, et al. Effects of aerobic training, resistance training, or both on percentage body fat and cardiometabolic risk markers in obese adolescents: the healthy eating aerobic and resistance training in youth randomized clinical trial. JAMA Pediatr 2014; 168: 1006–1014.
16. Zieleniewska NA, Szum-Jakubowska A, Chlabicz M, et al. The prevalence of diabetes and prediabetes: a population-based study. Pol Arch Intern Med 2023; 133: 16407.

3. Glycaemic Monitoring

CHAPTER HIGHLIGHTS:
• Most individuals treated with insulin using multiple daily injections should use continuous glucose monitoring (CGM) or perform self-monitoring of blood glucose (SMBG) both before and after meals, during the night, before planned physical activity, when hypoglycaemia is suspected, and before activities during which hypoglycaemia is particularly dangerous (e.g. driving). [**B**]
• As part of an educational programme, SMBG may help individuals treated with insulin using less intensive injection regimens to make independent therapeutic decisions [**B**], as well as individuals with diabetes not treated with insulin. [**E**]
• When prescribing SMBG, continuous education of individuals with diabetes should be ensured, together with periodic assessment of the correctness of self-monitoring technique, interpretation of results, and the impact of SMBG on therapeutic decision-making. [**E**]
• CGM, when used in combination with intensive insulin therapy, is a useful tool for reducing HbA_{1c} results and for achieving and maintaining glycaemic treatment targets in individuals with type 1 diabetes. [**A**]
• CGM may be a useful tool for individuals with impaired awareness of hypoglycaemia and for those with recurrent episodes of hypoglycaemia. [**B**]
• The use of CGM systems should also be considered in individuals treated with insulin using less intensive injection regimens [**A**], as well as in individuals not treated with insulin. [**C**]
• Individuals with diabetes using CGM should also always have access to SMBG. [**A**]

Ongoing glucose monitoring and retrospective assessment of glycaemia are integral components of appropriate diabetes management. Proper implementation of self-monitoring of glycaemia requires systematic education of individuals with diabetes, with particular emphasis on developing and maintaining skills in the use of blood glucose meters and continuous glucose monitoring (CGM) systems, as well as on interpretation of self-monitoring results. This includes using the obtained data for day-to-day adjustment of diet, physical activity, and doses of antihyperglycaemic medi-

cations. Another essential element of monitoring diabetes management is regular measurement of glycated haemoglobin (HbA_{1c}), and in individuals using CGM systems, analysis of glycaemic reports.

I. Self-monitoring of glycaemia

Self-monitoring of glycaemia is an integral component of diabetes management.

Individuals with diabetes treated with multiple daily insulin injections or continuous subcutaneous insulin infusion should use CGM systems, which increase the safety and effectiveness of insulin therapy and improve quality of life, as well as the quality of diabetes care.

The use of glucose monitoring systems is particularly recommended in individuals with type 1 diabetes with unstable disease course, frequent episodes of hypoglycaemia, and impaired awareness of hypoglycaemia, as it improves the safety and effectiveness of treatment.

Self-monitoring of glycaemia, including the use of CGM systems, is also recommended to achieve therapeutic targets in individuals treated with single daily insulin injections, oral antihyperglycaemic medications and/or GLP-1 receptor agonists and/or GIP/GLP-1 receptor agonists (Table 3.1). All individuals with diabetes, regardless of the method of treatment, should monitor glycaemia more frequently in situations of malaise or sudden deterioration in health status.

To ensure appropriate self-monitoring of glycaemia, individuals with diabetes should receive training and education in the use of blood glucose meters and CGM systems, interpretation of results, and subsequent management (Figure 3.1). The scope of educational counselling is discussed in detail in Chapter 9 and Annex 1.

For glycaemic monitoring, the use of blood glucose meters that display results as plasma glucose concentration is recommended. These devices should have a declared measurement error, confirmed in publications and manufacturer materials, not exceeding 15% for glucose concentrations ≥ 100 mg/dl (5.6 mmol/l) and 15 mg/dl (0.8 mmol/l) for glucose concentrations < 100 mg/dl (5.6 mmol/l). In patients performing ≥ 4 measurements per day, analysis of results using dedicated computer software may be helpful. Verification of the accuracy of blood glucose meters, together with assessment of correct device use, should be performed whenever measurement inaccuracy is suspected and at least once a year at the outpatient facility where the individual with diabetes receives care. This verification should involve measurement of glucose concentration in the same sample using the blood glucose meter and a comparative method – either a laboratory method or point-of-care testing (POCT) consistent with the laboratory method. Differences between results should not exceed the above-mentioned acceptable error limits.

The minimum quality performance requirements that CGM systems used in clinical practice should meet are presented in the Diabetes Poland expert statement (Table 3.2).

In addition to the specified minimum requirements, fulfilment of the following criterion is recommended: at each anatomical insertion site, the number of paired reference readings should be sufficient to allow precise determination of the declared accuracy. For younger children, the minimum number of paired readings should be at least 2,500, whereas for adults it should be at least 10,000.

Examples of substances that may interfere with glucose readings obtained using blood glucose meters and CGM systems are presented in Tables 3.3 and 3.4.

II. Glycated haemoglobin

The HbA_{1c} result reflects the average blood glucose concentration over approximately the 3 months preceding the measurement; however, around 50% of circulating HbA_{1c} is formed during the last month before testing.

Table 3.1. Recommended frequency of self-monitoring of glycaemia using a blood glucose meter

Method of diabetes treatment	Frequency of glycaemia measurements
Multiple daily insulin injections (i.e. at least 3 injections per day), intensive functional insulin therapy, regardless of the type of diabetes	Multiple measurements throughout the day (i.e. at least 4 times daily; 8 measurements per day recommended), according to established treatment principles and individual patient needs
Individuals with type 2 diabetes treated with fixed insulin doses.	According to the individual patient's clinical needs
Individuals treated with non-insulin antihyperglycaemic medications.	According to the individual patient's clinical needs

HbA_{1c} should be measured once a year in individuals with a stable course of the disease who are achieving treatment goals. In individuals who do not achieve treatment goals, or in whom a change in therapy has been introduced, HbA_{1c} measurements should be performed at least once every quarter.

HbA_{1c} measurements should be carried out using analytical methods certified by the National Glycohemoglobin Standardization Program (NGSP) (http://www.ngsp.org). HbA_{1c} testing may also be performed outside the laboratory setting using point-of-care testing (POCT), provided that both the method and the analyser are certified by the NGSP. Diagnostic laboratories report HbA_{1c} results as a percentage (%) and in SI units.

When interpreting HbA_{1c} results, interfering factors should be taken into account, such as alterations in erythrocyte lifespan, haemoglobinopathies, and chemical modifications of haemoglobin, which may limit or preclude the clinical usefulness of the measurement.

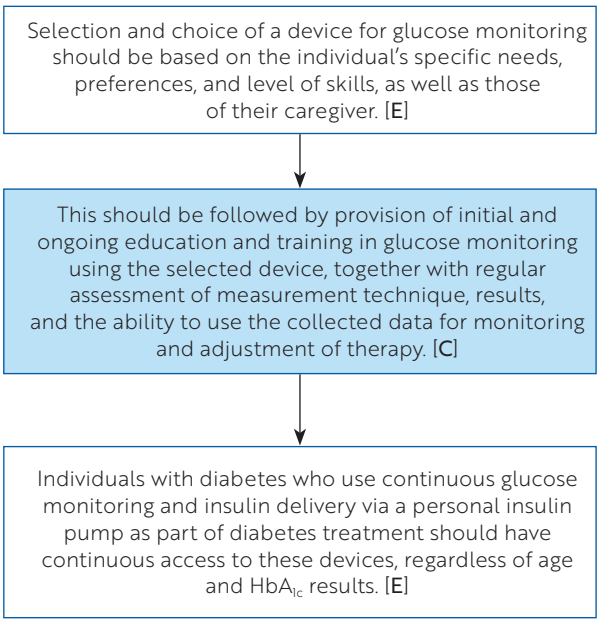


Figure 3.1. Recommendations regarding individualisation of the approach to glucose monitoring [10]

Table 3.2. Key factors determining fulfilment of minimum quality criteria for CGM systems used in clinical practice [9]

1. System performance should be tested at all anatomical sites of sensor wear. Data should include results of meal and insulin challenges, which assess rapid changes in glucose concentration and time spent at extreme glucose values. At least 8% of paired readings should be within the range < 70 mg/dl (< 3.9 mmol/l), and no more than 5% of readings should exceed 300 mg/dl (> 16.7 mmol/l).
2. Results of clinical studies should demonstrate stable system performance, both analytically and clinically, throughout the entire sensor wear period. The sensor should be tested at least at the beginning (days 1–3), midway, and at the end of the wear period, as well as during periods of rapid fluctuations in glucose concentration.
3. Clinical studies should reflect changes in glucose concentration in the target population under real-world conditions, taking into account glucose variability. Differences in performance at various rates of glucose change (trend accuracy) should be reported alongside other performance data*.
4. CGM sensors should demonstrate clinically acceptable accuracy even in the presence of significant concentrations of interfering substances that may occur in target users. This includes, among others, endogenous substances and their metabolites, food products, dietary supplements, and medications.
5. The device should incorporate system safeguards that prevent the use of single-use sensors beyond their declared wear period.
6. Verification and validation of the CGM system should provide evidence of safe and reliable transmission of glucose concentration data at clinically relevant time intervals to other connected devices that are intended to receive these data and safely perform their functions
7. Performance data should demonstrate the absence of clinically significant interruptions in sensor data availability throughout the entire wear period, in accordance with conformity assessment testing. Such interruptions could prevent the safe and effective use of other digitally connected devices**.
8. System specifications and data management processes should ensure privacy protection and cybersecurity in accordance with European data protection regulations.
9. All clinical study data should be included in product labelling for each intended user group at the time of placing the product on the market.
10. CGM system performance should be evaluated using at least three independent production batches of sensors, which should be verified for regulatory purpose

**Including populations in which at least 70–75% of participants have type 1 diabetes, to ensure accuracy and reliability across glucose variability. The selected reference method should be a laboratory method (e.g. Yellow Springs Instruments or equivalent) with a clearly documented source of the sample (venous blood, arterialised venous blood, or capillary blood).*

***This means that CGM data are unavailable at the moment when treatment decisions need to be made—both by the user and by a connected device. An example may be a situation related to insulin administration, insulin dose adjustment, or an approaching episode of hypoglycaemia or hyperglycaemia.*

Table 3.3. Examples of substances and clinical conditions that may interfere with glucose readings obtained using glucometers [1]

Substance or clinical condition	Effect on glucometer measurement
Maltose	Falsely elevated result
Galactose	Falsely elevated result
Xylose	Falsely elevated result
N-acetylcysteine	Falsely elevated result
Acetaminophen	Falsely elevated result in the presence of hypoglycaemia
Dopamine	Falsely elevated result in the presence of hypoglycaemia
Furosemide	Falsely reduced result
Vitamin C	Falsely elevated or reduced result
Uric acid	Falsely elevated result in the presence of marked hypoglycaemia or marked hyperglycaemia
Haematocrit (high)	Falsely reduced result
Haematocrit (low)	Falsely elevated result

Table 3.4. Examples of substances that may interfere with glucose readings obtained using CGM systems [1]

Substance	System	Effect
Acetaminophen > 4 g/day Any dose	Dexcom G6, Dexcom G7 Medtronic Guardian	Sensor readings higher than actual glucose concentration
Ascorbic acid > 500 mg/day (vit. C)	Freestyle Libre	Sensor readings higher than actual glucose concentration
Hydroxyurea	Dexcom G6, Dexcom G7, Medtronic Guardian	Sensor readings higher than actual glucose concentration
Mannitol	Senseonics Eversense	Sensor readings higher than actual glucose concentration
Sorbitol	Senseonics Eversense	Sensor readings higher than actual glucose concentration

REFERENCES

1. American Diabetes Association. Diabetes technology: Standards of Care in Diabetes – 2025. Diabetes Care 2025; 48 (Suppl 1): S146–S166.
2. Battelino T, Phillip M, Bratina N, et al. Effect of continuous glucose monitoring on hypoglycemia in type 1 diabetes. Diabetes Care 2011; 34: 795–800.
3. Elgart JF, Gonzalez L, Prestes M, et al. Frequency of self-monitoring blood glucose and attainment of HbA1c target values. Acta Diabetol 2016; 53: 57–62.
4. Farmer A, Wade A, Goyder E, et al. Impact of self-monitoring of blood glucose in the management of patients with non-insulin treated diabetes: open parallel group randomised trial. BMJ 2007; 335: 132.
5. Grant RW, Huang ES, Wexler DJ, et al. Patients who self-monitor blood glucose and their unused testing results. Am J Manag Care 2015; 21: e119–e129.
6. Juvenile Diabetes Research Foundation Continuous Glucose Monitoring Study Group; Beck RW, Hirsch I, Laffel L, et al. The effect of continuous glucose monitoring in well-controlled type 1 diabetes. Diabetes Care 2009; 32: 1378–1383.
7. Juvenile Diabetes Research Foundation Continuous Glucose Monitoring Study Group. Sustained benefit of continuous glucose monitoring on A1C, glucose profiles, and hypoglycemia in adults with type 1 diabetes. Diabetes Care 2009; 32: 2047–2049.
8. Malanda UL, Welschen LM, Riphagen II, et al. Self-monitoring of blood glucose in patients with type 2 diabetes mellitus who are not using insulin. Cochrane Database Syst Rev 2012; 1: CD005060.
9. Mathieu C, Irace C, Wilmot EG, et al. Minimum expectations for market authorization of continuous glucose monitoring devices in Europe-'eCGM' compliance status. Diabetes Obes Metab 2025; 27: 1025–1031.
10. Miller KM, Beck RW, Bergenstal RM, et al. T1D Exchange Clinic Network. Evidence of a strong association between frequency of self-monitoring of blood glucose and hemoglobin A1c levels in T1D Exchange clinic registry participants. Diabetes Care 2013; 36: 2009–2014.
11. Yeh HC, Brown TT, Maruthur N, et al. Comparative effectiveness and safety of methods of insulin delivery and glucose monitoring for diabetes mellitus: a systematic review and meta-analysis. Ann Intern Med 2012; 157: 336–347.

4. Setting Treatment Targets in Diabetes

CHAPTER HIGHLIGHTS:
• In individuals with diabetes, the general target for glycaemic control, expressed as an HbA_{1c} result, is ≤ 7.0% (53 mmol/mol). [**A**]
• An LDL-C concentration < 55 mg/dl (< 1.4 mmol/l) and a reduction of at least 50% from baseline in individuals with diabetes at very high cardiovascular risk, with reduction of non-HDL-C to < 85 mg/dl (< 2.2 mmol/l) as a secondary target. [**A**]
• An LDL-C concentration < 70 mg/dl (1.8 mmol/l) and a reduction of at least 50% from baseline in individuals with diabetes at high cardiovascular risk, with reduction of non-HDL-C to < 100 mg/dl (< 2.6 mmol/l) as a secondary target. [**A**]
• An LDL-C concentration < 100 mg/dl (< 2.6 mmol/l) in individuals with diabetes at moderate cardiovascular risk, with reduction of non-HDL-C to < 130 mg/dl (< 3.4 mmol/l) as a secondary target. [**A**]
• The recommended blood pressure target is 120–129/70–79 mm Hg. [**A**]

I. General remarks

1. Treatment targets in diabetes should be understood as achieving target values for glycaemia, blood pressure, lipid profile, and body weight.
2. In older individuals and in the presence of comorbid conditions, if the estimated life expectancy is not longer than 10 years, treatment targets should be relaxed to a degree that does not impair the patient's quality of life.
3. Contemporary diabetology is based on the principle of advanced individualisation of treatment targets and therapy intensification. In every person with diabetes, particularly those with type 2 diabetes, when defining treatment targets and selecting a therapeutic strategy, the patient's attitude and expected engagement in treatment (including support from people in their environment), the risk of hypoglycaemia and its potential consequences (which are more serious in older individuals and in those with cardiovascular and/or nervous system disease), duration of diabetes, expected life expectancy, presence of severe vascular complications of diabetes and significant comorbidities, the individual's level of diabetes education, as well as the balance between benefits and risks of achieving specific therapeutic targets should all be taken into account. In certain situations (e.g. advanced complications, older age), the defined treatment targets should be achieved gradually.

II. Glycaemic control targets

General targets: HbA_{1c} ≤ 7% (≤ 53 mmol/mol).

Individual targets:

1. HbA_{1c} ≤ 6.5% (≤ 48 mmol/mol):
   - in type 1 diabetes, when pursuing this target is not associated with an increased risk of hypoglycaemia or deterioration in quality of life – fasting and preprandial glycaemia, including self-monitoring, 70–110 mg/dl (3.9–6.1 mmol/l), and 2 hours after the start of a meal, as assessed by self-monitoring: 140 mg/dl (7.8 mmol/l);
   - in short-duration type 2 diabetes (disease duration < 5 years);
   - in children and adolescents – irrespective of diabetes type – when pursuing this target is not associated with an increased risk of hypoglycaemia or deterioration in quality of life.

   When assessing glycaemic profiles in relation to target HbA_{1c} results, reference should be made to the conversion presented in Table 4.1, which relates HbA_{1c} results to mean daily glucose concentration and the range of blood glucose concentrations.
2. HbA_{1c} 8.0–8.5% (64–69 mmol/mol):
   - in individuals of advanced age with long-standing diabetes and significant macrovascular complications (previous myocardial infarction and/or stroke) and/or multiple comorbidities, when the expected life expectancy is shorter than 10 years;
   - if, in an individual with diabetes aged > 65 years, life expectancy is anticipated to exceed 10 years, while implementing general treatment targets, gradual improvement of glycaemic control should be pursued, with HbA_{1c} ≤ 7% adopted as the target value;
3. HbA_{1c} < 6.5% (48 mmol/mol) in women with pre-existing diabetes planning pregnancy, and < 6.0% (42 mmol/mol) in the second and third trimesters of pregnancy, provided this is not associated with an increased frequency of hypoglycaemia.

In patients using CGM systems, one of the key parameters for assessment of glycaemic control should be time in range (TIR). Detailed recommen-

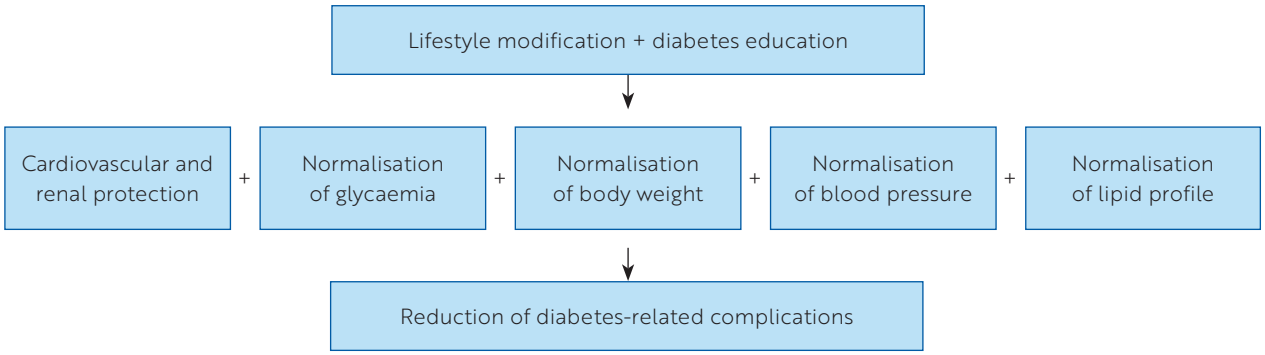


Figure 4.1. Holistic approach to strategies leading to reduction of the risk of diabetes-related complications

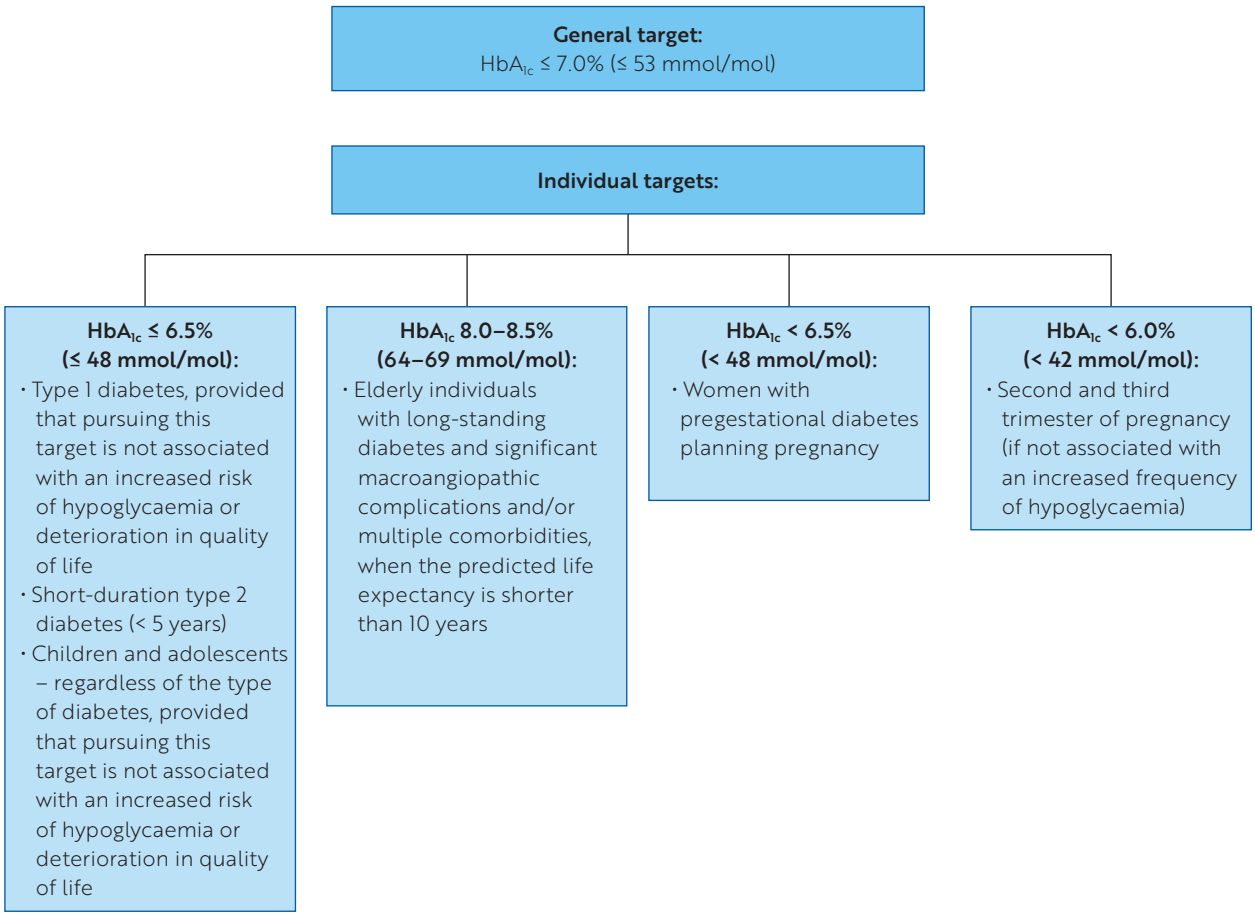


Figure 4.2. Glycaemic control targets

dations regarding TIR according to diabetes type are presented in Table 4.2.

III. Lipid control targets

- an LDL-C concentration of < 55 mg/dl (< 1.4 mmol/l) and a reduction of at least 50% from baseline in individuals with diabetes at very high cardiovascular risk;
- an LDL-C concentration of < 70 mg/dl (1.8 mmol/l) and a reduction of at least 50% from baseline in individuals with diabetes at high cardiovascular risk;
- an LDL-C concentration of < 100 mg/dl (2.6 mmol/l) in individuals at moderate cardiovascular risk (young individuals < 35 years of age with type 1 diabetes without chronic complications and without other cardiovascular risk factors, or individuals with type 2 diabetes aged < 50 years, with diabetes duration < 10 years, and without other risk factors);
- a non-HDL cholesterol concentration of < 85 mg/dl (2.2 mmol/l) in individuals with diabetes at very high cardiovascular risk;

Table 4.1. Relationship between HbA_{1c} value and mean plasma glucose concentration [7]

HbA_{1c}	Mean glucose concentration [mg/dl]	Mean glucose concentration [mmol/l]	Mean fasting glucose concentration [mg/dl]	Mean pre-meal glucose concentration [mg/dl]	Mean postprandial glucose concentration [mg/dl]
6.0	126	7.0			
< 6.5			122	118	144
6.5–6.99			142	139	164
7.0	154	8.6			
7.0–7.49			152	152	176
7.5–7.99			167	155	189
8.0	183	10.2			
8.0–8.5			178	179	206
9.0	212	11.8			
10.0	240	13.4			
11.0	269	14.9			
12.0	298	16.5			

Correlation between HbA_{1c} values and mean glycaemia: 0.92

Table 4.2. Target glycaemic parameters in individuals with type 1 and type 2 diabetes and in pregnant women using continuous glucose monitoring [1]

	TIR		TBR		TAR	
	% of readings; time per day	Target values	% of readings; time per day	Values below target	% of readings; time per day	Values above target
Type 1 diabetes / Type 2 diabetes	> 70%; > 16 hours, 48 minutes	70–180 mg/dl (3.9–10.0 mmol/l)	< 4%; < 1 hour < 1%; < 15 minutes	< 70 mg/dl (< 3.9 mmol/l) < 54 mg/dl (< 3.0 mmol/l)	< 25%; < 6 hours < 5%; < 1 hour, 12 minutes	> 180 mg/dl (> 10.0 mmol/l) > 250 mg/dl (> 13.9 mmol/l)
Elderly/ individuals at high risk of hypoglycemia	> 50%; > 12 hours	70–180 mg/dl (3.9–10.0 mmol/l)	< 1%; < 15 minutes	< 70 mg/dl (< 3.9 mmol/l)	< 50%; < 12 hours < 10%; < 2 hours, 24 minutes	> 180 mg/dl (> 10.0 mmol/l) > 250 mg/dl (> 13.9 mmol/l)
Pregnant women with type 1 diabetes	> 70%; > 16 hours, 48 minutes	63–140 mg/dl (3.5–7.8 mmol/l)	< 4% < 1 hour < 1% < 15 minutes	< 63 mg/dl (< 3.5 mmol/l) < 54 mg/dl (< 3.0 mmol/l)	< 25% < 6 hours	> 140 mg/dl (> 7.8 mmol/l)

TAR – time above range: hyperglycaemia, TBR – time below range: hypoglycaemia, TIR – time in range.

- a non-HDL cholesterol concentration of < 100 mg/dl (2.6 mmol/l) in individuals with diabetes at high cardiovascular risk;
- a non-HDL cholesterol concentration of < 130 mg/dl (3.4 mmol/l) in individuals with diabetes at moderate cardiovascular risk;
- a triglyceride concentration of < 135 mg/dl (< 1.52 mmol/l).

IV. Blood pressure targets:

- systolic blood pressure 120–129 mm Hg. [IA]
- diastolic blood pressure 70–79 mm Hg. [IA]

For detailed criteria: see Chapter 13.

REFERENCES

1. Battelino T, Danne T, Bergenstal RM, et al. Clinical targets for continuous glucose monitoring data interpretation: recommendations from the international consensus on time in range. Diabetes Care 2019; 42: 1593–1603.
2. Cosentino F, Grant PJ, Aboyans V, et al. ESC Scientific Document Group. 2019 ESC Guidelines on diabetes, prediabetes, and cardiovascular diseases developed in collaboration with the EASD. Eur Heart J 2020; 7: 255–323.
3. Garber AJ. Treat-to-target trials: uses, interpretation and review of concepts. Diabetes Obes Metab 2014; 16: 193–205.

4. Inzucchi SE, Bergenstal RM, Buse JB, et al. Management of hyperglycemia in type 2 diabetes, 2015: a patient-centered approach: update to a position statement of the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care 2015; 38: 140–149.
5. Lipska KJ, Ross JS, Miao Y, et al. Potential overtreatment of diabetes mellitus in older adults with tight glycemic control. JAMA Intern Med 2015; 175: 356–362.
6. Marx N, Federici M, Schütt K, et al. 2023 ESC Guidelines for the management of cardiovascular disease in patients with diabetes. Eur Heart J 2023; 44: 4043–4140.
7. Nathan DM, Kuenen J, Borg R, et al. A1c-Derived Average Glucose (ADAG) Study Group. Translating the A1C assay into estimated average glucose values. Diabetes Care 2008; 31: 1473–1478.
8. Stratton IM, Adler AI, Neil HAW, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. BMJ 2000; 321: 405–412.
9. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 329: 977–986.
10. Vijan S, Sussman JB, Yudkin JS, et al. Effect of patients' risks and preferences on health gains with plasma glucose level lowering in type 2 diabetes mellitus. JAMA Intern Med 2014; 174: 1227–1234.
11. Wei N, Zheng H, Nathan DM. Empirically establishing blood glucose targets to achieve HbA1c goals. Diabetes Care 2014; 37: 1048–1051.

5. Organisation of Medical Care for People with Diabetes and for Diabetes Prevention

CHAPTER HIGHLIGHTS:

- Contemporary diabetes care requires collaboration within a multidisciplinary therapeutic team, including physicians, nurses providing education or diabetes educators, dietitians, and psychologists. Care should be person-centred, taking into account the individual situation, needs, and preferences of the person with diabetes. Collaboration with specialists from related fields is also necessary due to the multidisciplinary nature of chronic diabetes-related complications and comorbid conditions. [**B**]

I. Outpatient Care

In outpatient care for a person with diabetes, cooperation between primary health care (PHC) and the diabetology therapeutic team is essential.

I.1. Responsibilities of primary health care

1. Health promotion, identification of risk factors, prevention of disorders of glucose metabolism, and education regarding prediabetes and diabetes.
2. Diagnosis of disorders of glucose metabolism.
3. Referral to a diabetology outpatient clinic for long-term treatment in the case of:

- type 1 diabetes,
- other specific types of diabetes,
- difficulty in establishing the type of diabetes,
- any type of diabetes in children and adolescents, including preclinical stages of type 1 diabetes,
- any type of diabetes in women planning pregnancy and in pregnant women.

4. Treatment of prediabetes,
5. Treatment of type 2 diabetes, including simple insulin therapy.
6. Referral for diabetology consultation (less frequently for long-term specialist care) in the case of:

- failure to achieve therapeutic targets; referral primarily for intensification of insulin therapy,
- occurrence of comorbid conditions complicating treatment,
- occurrence of diabetic complications,
- occurrence of complications related to pharmacotherapy,
- other special circumstances.

7. Implementation, in subsequent management, of therapeutic decisions and recommendations resulting from specialist consultation.
8. Issuing prescriptions for medications in use and orders for reimbursed medical devices on prescription (Polish eZWM national electronic reimbursement system).

I.2. Tasks of specialist care

Outpatient specialist care should be available on most working days of the week (optimally on every working day).

1. Verification of treatment outcomes and setting treatment targets in individuals with diabetes managed in primary care, as part of annual follow-up.
2. Management of individuals with diabetes treated with injectable therapies (insulin, GLP-1 receptor agonists, GIP/GLP-1 receptor agonists).
3. Initiation of therapy using continuous subcutaneous insulin infusion (CSII), including automated insulin delivery (AID) systems.
4. Ongoing management of individuals with diabetes treated with continuous subcutaneous insulin infusion (CSII), including AID systems.
5. Implementation of continuous glucose monitoring (CGM) systems and education on the correct interpretation of glycaemic data.
6. Management of children and adolescents with all types of diabetes, including preclinical stages of type 1 diabetes.
7. Differential diagnosis of diabetes types, including monogenic diabetes and diabetes associated with other diseases.
8. Diagnosis, prevention, and management of individuals with diabetes with respect to chronic complications.
9. Diabetes-specific educational counselling.
10. Dietary counselling in the diabetes outpatient clinic for individuals with diabetes or obesity.
11. Psychological counselling in the diabetes outpatient clinic.
12. Diagnosis and management of diabetes in pregnant women.
13. Diagnosis and treatment of conditions affecting the course and management of diabetes.
14. Monitoring of individuals with diabetes in accordance with current recommendations of Diabetes Poland (Table 5.1).
15. Management of obesity in individuals with diabetes or without disorders of glucose tolerance, but with very high or high cardiovascular risk, or with MASLD and high or intermediate risk of fibrosis.
16. Management of diabetic foot disease – see Chapter 22.

II. Specialist inpatient care

II.1. Conditions requiring urgent hospitalisation

1. Acute complications of diabetes (severe, recurrent hypoglycaemia and acute hyperglycaemic emergencies).
2. Diabetic foot disease – indications: see Chapter 22, Section VII.

Table 5.1. Recommendations for monitoring adult individuals with diabetes

Parameter	Remarks
Dietary and therapeutic education	At every visit
Physical examination, blood pressure measurement, and body weight measurement	At every visit
HbA_{1c}*	Once a year; more frequently if there are doubts about maintenance of normoglycaemia or a need to verify treatment effectiveness after modification
Total cholesterol, LDL-C, HDL-C, triglycerides (TG) in serum	Once a year; more frequently in the presence of dyslipidaemia
Albuminuria (ACR)	Once a year in individuals with type 1 diabetes from the 5th year of disease duration; in individuals with type 2 diabetes from the time of diagnosis; and in all individuals with diabetes and concomitant arterial hypertension
Serum creatinine and estimated glomerular filtration rate (eGFR)	Once a year (in type 1 diabetes after 5 years of disease duration)
Complete blood count, TSH, ALT, AST, sodium, potassium, calcium, phosphorus in serum, and urinalysis with sediment	According to clinical needs
Fundus examination	In individuals with type 1 diabetes after 5 years of disease duration; in individuals with type 2 diabetes – from the time of diagnosis (details: see Chapter 20)
Assessment for diabetic neuropathy and diabetic foot disease	Once a year in individuals at very low risk of ulceration (details: see Chapters 21 and 22)

** More frequently in individuals with uncontrolled diabetes after adjustment of antihyperglycaemic treatment.*

II.2. Conditions requiring hospitalisation due to inability to achieve therapeutic goals in the outpatient setting

1. Newly diagnosed type 1 diabetes requiring initiation of insulin therapy and structured education.
2. Newly diagnosed or metabolically decompensated type 2 diabetes with clinical features of hyperglycaemia requiring insulin therapy.
3. Diabetes diagnosed at a stage of advanced microangiopathic or macroangiopathic complications requiring insulin therapy.
4. Progression of chronic microangiopathic complications requiring modification of therapy (e.g. severe non-proliferative diabetic retinopathy and more advanced stages of retinopathy; stage 3b–5 chronic kidney disease).
5. Painful neuropathy and autonomic neuropathy.
6. Advanced macroangiopathic complications.
7. Modification of therapeutic regimens in individuals who fail to achieve treatment targets and require initiation or adjustment of insulin therapy.
8. Initiation of intensive insulin therapy using a personal insulin pump, including AID systems.
9. Initiation of insulin therapy in gestational diabetes or pregestational diabetes not previously treated with insulin.
10. Difficulty achieving normoglycaemia in pregnant women with pregestational diabetes.
11. Impaired awareness of hypoglycaemia resulting in severe hypoglycaemic episodes.
12. Fear of hypoglycaemia/hyperglycaemia requiring intensive education and round-the-clock support from the therapeutic team.
13. Mental disorders leading to discontinuation of antihyperglycaemic therapy.

REFERENCES

1. Tchero H, Kangambega P, Briatte C, et al. Clinical effectiveness of telemedicine in diabetes mellitus: a meta-analysis of 42 randomized controlled trials. Telemed J E Health 2019; 25: 569–583.
2. TRIAD Study Group. Health systems, patients factors, and quality of care for diabetes: a synthesis of findings from the TRIAD study. Diabetes Care 2010; 33: 940–947.
3. Tricco AC, Ivers NM, Grimshaw JM, et al. Effectiveness of quality improvement strategies on the management of diabetes: a systematic review and meta-analysis. Lancet 2012; 379: 2252–2261.

6. Behavioural Therapy

Chapter highlights:
· All individuals with diabetes should receive education on the general principles of healthy nutrition in diabetes, provided by appropriately qualified professionals (physicians, dietitians, diabetes specialist nurses, diabetes educators), using a variety of methods and techniques, including telemedicine. Detailed dietary recommendations should be individualised according to the patient's needs and capabilities. [**A**]
· Continuous glucose monitoring (CGM) systems play a very important educational role in optimising diet, particularly with regard to the glycaemic impact of meals, across all types of diabetes. [**B**]
· Carbohydrates are the primary macronutrient determining prandial insulin requirements. A key element of dietary education in individuals with type 1 diabetes should be training in recognising and estimating carbohydrate content in meals in order to optimise insulin dosing. Individuals with type 2 diabetes should be educated on portion size control and on the distribution of carbohydrates across individual meals and the overall diet. [**A**]
· There is no single universal diet suitable for all individuals with diabetes. Optimal macronutrient proportions for this group of patients should be determined individually, taking into account age, level of physical activity, presence of diabetes-related complications, comorbid conditions, and personal preferences of the individual with diabetes. [**E**]
· Physical activity, due to its multidirectional benefits, is an integral component of appropriate, comprehensive diabetes management. To achieve optimal effects, physical activity should be regular, undertaken at least every 2–3 days, and preferably daily. [**A**]
· Individuals with diabetes should limit uninterrupted sedentary time. [**B**]

Behavioural therapy is an essential component of the management of all individuals with diagnosed diabetes (both type 1 and type 2), as well as those with prediabetes (including preclinical stages of type 1 diabetes), at all ages. Appropriate nutrition and physical activity play a crucial role in

improving overall health and in the prevention and treatment of chronic diabetes-related complications. All individuals with diabetes should receive education on the general principles of healthy nutrition in diabetes, delivered by appropriately qualified professionals (physicians, dietitians, diabetes specialist nurses, diabetes educators), using a variety of methods and techniques, including interactive approaches and telemedicine. Management of people with diabetes should incorporate a therapeutic lifestyle that includes a varied diet, regular physical activity, avoidance of tobacco smoking and alcohol use, optimal sleep duration, and stress reduction. Education on a therapeutic lifestyle, tailored to the needs and capabilities of individuals with diabetes, enables achievement of intended therapeutic goals and reduces costs associated with the treatment of diabetes-related complications.

It should be emphasised that one of the key priorities of behavioural therapy in diabetes, irrespective of diabetes type, is the maintenance of a healthy body weight.

Dietary recommendations

I. General recommendations

The aim of dietary treatment in individuals with diabetes is to achieve and maintain:

- normal (or near-normal) plasma glucose concentrations in order to prevent diabetes-related complications,
- optimal serum lipid and lipoprotein concentrations,
- optimal blood pressure values to reduce the risk of vascular disease,
- a desirable body weight.

Dietary treatment includes guidance on:

- individually calculated dietary energy intake,
- distribution of calories across meals throughout the day,
- sources of foods providing energy, vitamins, minerals, and phytochemicals,
- foods whose intake should be limited.

When planning a diet, individual dietary and cultural preferences, age, sex, level of physical activity, and economic status should be taken into account. An important element of dietary education should be the provision of information in a practical manner, enabling direct application of the acquired knowledge in everyday life. Meal consumption is an important component of quality of life for people with diabetes. Recommendations provided to patients should emphasise the wide range of possibilities for individual choice and composition of the diet. Information regarding the need to limit or eliminate specific food products should be communicated to patients only in justified situations and based on scientific evidence. Dietary education should be provided at the time of diagnosis. Verification of the patient's knowledge regarding nutritional management of diabetes should be carried out annually, with re-education as needed.

The dietary management strategy in individuals with diabetes should include:

- assessment of habitual dietary intake,
- nutritional diagnosis,
- definition of the goals and assumptions of dietary intervention,
- nutritional intervention (individual and/or group counselling),
- monitoring of dietary intake and evaluation of treatment outcomes,
- modification of dietary assumptions if the intended therapeutic goal is not achieved.

Individuals with diabetes should be encouraged to follow the principles of healthy eating recommended for the general population and, additionally, to:

- control portion sizes of foods habitually consumed,
- control the amount of carbohydrates consumed in the overall diet and in individual meals,
- limit foods containing rapidly absorbable carbohydrates, including added sugars and free sugars,
- consume meals regularly, including breakfast,
- eat meals at a slow pace.

There is no universal diet suitable for all individuals with diabetes. Various dietary strategies may be used in the management of diabetes, such as the Mediterranean diet, the DASH diet, the flexitarian diet, and plant-based diets. These dietary patterns assume a substantial intake of non-starchy vegetables, maximal limitation of added sugars and refined grains, and an increased proportion of minimally processed foods.

Individuals with type 1 diabetes should avoid consumption of rapidly absorbable carbohydrates and adhere to the general principles of a well-balanced diet. In every case, both dietary assumptions and the insulin treatment regimen should be individualised. Insulin therapy should be adapted to the dietary habits of the person with diabetes, the composition of consumed meals (carbo-

hydrate, protein, and fat content), as well as their lifestyle and level of physical activity.

When developing dietary recommendations, priority should be given to recognising and estimating the content of absorbable carbohydrates in meals, for example using the carbohydrate exchange system. When selecting foods, glycaemic index (GI) and glycaemic load (GL) values may also be helpful. Considerable interindividual variability is observed in glycaemic responses after consumption of the same meal or food product (e.g. dairy products). The order in which foods from different food groups are consumed within a single meal may significantly influence postprandial glycaemia. It is beneficial to consume vegetables and protein-containing products (meat, fish) before starchy foods.

In individuals with diabetes in the oldest age groups, dietary education should be simple and easy to understand. The diet should be individualised while ensuring an adequate protein intake. Although carbohydrates are the primary macronutrient determining prandial insulin requirements, individuals with type 1 diabetes should also be educated about the glycaemic effects of proteins and fats.

CGM systems are an effective tool facilitating assessment of the impact of the quantity and quality of macronutrients and their mutual proportions on glycaemia. Dedicated applications may also support periprandial glycaemic control. When selecting such applications, guidance and recommendations from leading diabetes societies should be followed.

In type 2 diabetes, the main goals of therapy are maintenance of optimal metabolic control of the disease, reduction of excess body weight, and maintenance of a desirable body weight. Therefore, in addition to the recommendations outlined above, total dietary energy intake adjusted to age, current body weight, and level of physical activity is of fundamental importance. The energy deficit should be determined individually to allow slow but systematic weight reduction (approximately 0.5–1 kg per week). A reduction in body weight of at least 5% per year compared with baseline weight results in a measurable improvement in glycaemic control; however, optimally, weight reduction should reach at least 7% per year. A daily caloric deficit of 500–750 kcal is considered safe.

Weight reduction may be achieved by using energy-restricted diets with various macronutrient proportions (proteins, fats, carbohydrates); however, long-term use of diets with markedly reduced carbohydrate intake and fasting regimens is not recommended. Portion size control is recommended for all individuals with diabetes and overweight or obesity.

II. Specific recommendations

Diet composition

1. Carbohydrates:

- there is insufficient scientific evidence to define a single optimal amount of carbohydrates in the diet for individuals with diabetes;
- the proportion of carbohydrates in the diet should account for approximately 45% of total energy intake; if carbohydrates are derived from products with a low glycaemic index (GI) and high fibre content, their contribution to total dietary energy may be higher (up to 60%); a high proportion of energy derived from carbohydrates should be recommended for individuals with very high levels of physical activity, whereas a lower proportion of energy from carbohydrates (25–44%) may be temporarily recommended for individuals with low physical activity whose ability to increase activity is limited, for example due to comorbid conditions;
- the main sources of carbohydrates should be wholegrain cereal products, particularly those with a low GI (< 55);
- the primary restriction should concern simple carbohydrates (mono- and disaccharides), the intake of which should be reduced to a minimum by individuals with diabetes; limitation of added sugars (used during food production and meal preparation) and free sugars is also recommended; their main sources include sugar and confectionery, as well as honey, fruit juices, and fruit drinks;
- sweetening agents (sweeteners) should be consumed in moderation and in amounts not exceeding the manufacturer's recommendations; during the registration process of low-calorie sweeteners, an acceptable daily intake (ADI) expressed in mg/kg body weight/day is established, defined as the amount that can be safely consumed daily over a lifetime without adverse health effects; ADI values for selected low-calorie sweeteners are presented in Table 6.1;
- daily fructose intake should not exceed 50 g; the use of fructose as a sugar substitute is not recommended;

Table 6.1. Acceptable daily intake (ADI) values for selected low-calorie sweeteners

Substance	Labelling on food products	ADI [mg/kg body weight/day]
Acesulfame potassium	E950	0–15
Aspartame	E951	0–40
Cyclamate	E952	0–7
Saccharin	E954	0–5
Sucralose	E955	0–15
Neotame	E961	0–2
Steviol glycosides	E960	0–4

- the minimum daily intake of dietary fibre should be 25 g or 15 g per 1000 kcal of the diet; efforts should be made to increase fibre intake by including at least two portions of wholegrain cereal products and three portions of fibre-rich vegetables; if it is not possible to achieve the recommended fibre intake, supplementation with dietary fibre, particularly water-soluble fractions, should be considered; increasing the intake of resistant starch (a fibre fraction) in the diet is advisable.

2. Fats:
- in dietary management of diabetes, the proportion of fat in the diet should be similar to that recommended for individuals without diabetes and may range from 25% to 40% of total dietary energy;
- the quality of dietary fat is more important than total fat intake; with a high fat intake, the proportion of individual fatty acid types is particularly important;
- saturated fats should provide less than 10% of total dietary energy;
- monounsaturated fats should provide up to 20% of total dietary energy, while polyunsaturated fats should account for approximately 6–10% of total dietary energy;
- dietary cholesterol intake should not exceed 300 mg/day, and in individuals with dyslipidaemia, less than 200 mg/day;
- to reduce LDL-C levels, the proportion of saturated fats in the diet should be reduced and/or replaced with low-GI carbohydrates and/or monounsaturated fats;
- in individuals with hypercholesterolaemia, inclusion of foods containing plant sterols/stanols in amounts of 2–3 g/day may be beneficial;
- intake of trans fatty acid isomers should be minimised, particularly those derived from processed foods;
- plant-based fats are recommended, with the exception of palm oil and coconut oil.

3. Proteins:
- protein intake should be determined individually; there is no evidence of adverse effects associated with the use of high-protein diets in the dietary management of individuals with diabetes; in most individuals with diabetes, as in the general population, protein should provide 15–20% of total dietary energy (approximately 1–1.5 g/kg body weight/day); in individuals with type 2 diabetes and excess body weight, an energy-restricted diet containing 20–30% protein increases satiety and facilitates weight reduction and maintenance; individuals with chronic kidney disease should maintain protein intake at approximately 0.8–1 g/kg body weight/day;
- there is no need to restrict animal protein, although in some individuals it may be beneficial to replace animal protein with plant protein (e.g. legumes).

4. Vitamins and micronutrients:
- supplementation with vitamins or micronutrients in individuals without confirmed deficiencies is not recommended;
- exceptions include vitamin D_3 (supplementation according to recommendations for the general population), folic acid (supplementation at a dose of 400 µg for at least 12 weeks prior to conception and continuation until the 12^{th} week of pregnancy), and vitamin B_{12} in individuals treated long-term with metformin in whom deficiency has been confirmed;
- multivitamin supplementation may be necessary in older individuals, vegetarians, vegans, and individuals following very low-calorie diets.

5. Alcohol:
- alcohol use by individuals with diabetes is not recommended (there is no safe dose);

- individuals with diabetes should be informed that alcohol inhibits hepatic glucose release and, therefore, its use(especially without food) may predispose to hypoglycaemia.

6. Salt:

- total salt intake (from all sources, including foods and added salt) should not exceed 5 g/day (2300 mg sodium/day);
- in justified cases (e.g. in individuals with salt-sensitive arterial hypertension), greater restrictions on salt intake are recommended in accordance with DASH diet principles; however, data on reducing sodium intake below 1500 mg/day in individuals with diabetes are inconclusive.

Dietary recommendations for individuals with diabetes in special situations (e.g. during pregnancy, in children and adolescents, in individuals with advanced nephropathy) are presented in the relevant chapters. Detailed practical guidance on dietary management of diabetes is provided in the recommendations of the Polish Society of Dietetics (www.ptd.org.pl).

Physical activity

Physical activity, due to its multidirectional benefits, is an integral component of appropriate, comprehensive management of diabetes. It increases insulin sensitivity, improves glycaemic control and lipid profile, supports body weight reduction, and has a beneficial effect on mood, even in individuals with depression.

1. Principles of undertaking physical activity:

- initial recommendations regarding physical activity should be moderate and tailored to the individual's capacity to exercise;
- to achieve optimal effects, physical activity should be regular, undertaken at least every 2–3 days and preferably daily;
- when initiating intensive physical activity, warm-up exercises lasting 5–10 minutes should be performed, followed by cool-down exercises at the end;
- physical activity may increase the risk of acute or delayed hypoglycaemia;
- alcohol use may increase the risk of post-exercise hypoglycaemia;
- attention should be paid to preventing dehydration in conditions of high ambient temperature;
- the risk of foot injury during physical activity should be considered (particularly in the presence of peripheral neuropathy and reduced pain sensation), together with the need for appropriate foot care and comfortable footwear.

2. Intensity of physical activity.

The physician, based on the full clinical picture, determines the appropriate intensity of physical activity.

An appropriate form of physical activity for individuals with diabetes (with coexisting overweight or obesity) at any age is Nordic walking.

The most suitable form of physical activity in individuals with type 2 diabetes aged over 65 years and/or with overweight is brisk walking (to the point of mild breathlessness), performed 3–5 times per week (approximately 150 minutes per week).

Individuals without significant contraindications, particularly in younger age groups, should be encouraged to increase physical activity, including participation in sports. Such individuals require additional education regarding the glycaemic effects associated with different types of physical activity (e.g. aerobic, resistance, and interval exercise).

CGM systems are an excellent tool facilitating peri-exercise glycaemic control, both in real time and for retrospective assessment of the effects of physical activity and implemented therapeutic interventions on glycaemia. Dedicated applications may also support peri-exercise glycaemic control. When selecting such applications, as with those used to optimise periprandial glycaemic control, guidance and recommendations from leading diabetes societies should be followed.

A simple and effective recommendation is to limit uninterrupted sedentary time, particularly in adults with type 2 diabetes. Glycaemic benefits may be achieved by avoiding sitting without breaks for longer than 30 minutes.

3. Risks associated with physical activity in individuals with diabetes.

Physical activity undertaken without appropriate preventive measures may result in hypoglycaemia or, less commonly, hyperglycaemia and metabolic decompensation. Preventive measures should include the pre-exercise period, the post-exercise period (up to 12 hours after exercise), and long-term management (taking into account gradual improvement in physical fitness over time). Detailed management strategies for the periexercise period aimed at avoiding extreme glycaemic values are presented in Chapter 7.

Moderate and intensive physical activity may have an adverse impact on general health status in certain clinical situations:

- proliferative diabetic retinopathy – risk of vitreous haemorrhage and retinal detachment;

- diabetic kidney disease – increased albumin excretion and proteinuria;
- autonomic neuropathy – presence of orthostatic hypotension;
- risk of myocardial ischaemia.

4. Physical activity during the COVID-19 pandemic.

It should be emphasised that individuals with diabetes should maintain the recommended level of physical activity regardless of the epidemiological situation. In the event of restrictions imposed by epidemiological circumstances that limit mobility or access to sports facilities, efforts should be made to replace previous forms of physical activity with alternatives that can be performed despite such restrictions, for example in home settings. As this may involve changes in the type of exercise and, consequently, its glycaemic effects and required precautionary measures, each such situation requires consultation with the treating physician.

Smoking cessation

In every individual who currently smokes tobacco or has smoked in the past, the following should be established:

- age at smoking initiation;
- duration of smoking;
- number of cigarettes smoked;
- any previous attempts to quit smoking and their duration;
- time since smoking cessation in individuals who have stopped smoking.

Counselling should include:

- informing individuals with diabetes who have not previously smoked about the risks associated with tobacco smoking and use of electronic cigarettes;
- encouraging complete cessation of smoking and use of electronic cigarettes;
- supporting individuals with diabetes in the decision to quit smoking;
- providing psychological support and, if necessary, pharmacological support;
- discussion of smoking status at every medical visit;
- a written note in the medical records if an individual with diabetes refuses to quit smoking.

Sleep

Proper sleep hygiene is an important element of a healthy lifestyle. In individuals with diabetes, sleep duration and quality may be impaired due to the pathophysiology of the disease, behavioural factors, and factors related to the applied treatment. Conversely, poor sleep quality and inappropriate sleep duration may lead to deterioration of metabolic control. Moreover, an evening chronotype (a natural tendency to function better in the evening and at night) is an independent risk factor for the development of type 2 diabetes, regardless of sleep quality and duration. Ensuring adequate sleep duration and quality should be an important component of diabetes management. This may be supported by recommendations regarding the timing and quality of the last meal, self-monitoring of glycaemia (with appropriate alarm settings in individuals using CGM), avoidance of factors leading to hyperglycaemia or hypoglycaemia, and preference for treatment strategies that ensure stabilisation and optimisation of nocturnal glycaemia.

Individuals with diabetes or prediabetes should be assessed for sleep disorders. To facilitate self-management of sleep, patients may consider the use of sleep monitoring devices such as watches, wristbands, specialised mats, and similar devices, or self-completed questionnaires assessing sleep duration and quality.

REFERENCES

1. Bell KJ, Smart CE, Steil GM, et al. Impact of fat, protein, and glycemic index on postprandial glucose control in type 1 diabetes: implications for intensive diabetes management in the continuous glucose monitoring era. Diabetes Care 2015; 38: 1008–1015.
2. Debras C, Chazelas E, Sellem L, et al. Artificial sweeteners and risk of cardiovascular diseases: results from the prospective NutriNet-Santé cohort. BMJ 2022; 378: e071204.
3. Evert AB, Dennison M, Gardner CD, et al. Nutrition therapy for adults with diabetes or prediabetes: a consensus report. Diabetes Care 2019; 42: 731–754.
4. Fleming GA, Petrie JR, Bergenstal RM, et al. Diabetes digital app technology: benefits, challenges, and recommendations. A consensus report by the European Association for the Study of Diabetes (EASD) and the American Diabetes Association (ADA) Diabetes Technology Working Group. Diabetologia 2020; 63: 229–241.
5. Franz MJ, Boucher JL, Rutten-Ramos S, et al. Lifestyle weight-loss intervention outcomes in overweight and obese adults with type 2 diabetes: a systematic review and meta-analysis of randomized clinical trials. J Acad Nutr Diet 2015; 115: 1447–1463.
6. Fu S, Li L, Deng S, et al. Effectiveness of advanced carbohydrate counting in type 1 diabetes mellitus: a systematic review and meta-analysis. Sci Rep 2016; 6: 37067.

7. Greenwood DA, Gee PM, Fatkin KJ, et al. A systematic review of reviews evaluating technology-enabled diabetes self-management education and support. J Diabetes Sci Technol 2017; 11: 1015–1027.
8. Hallberg SJ, Jake ED, Kushner A, Athinarayanan SJ. Improving the scientific rigour of nutritional recommendations for adults with type 2 diabetes: a comprehensive review of the American Diabetes Association guideline-recommended eating patterns. Diabetes Obes Metab 2019; 21: 1769–1779.
9. Hamdy O, Mottalib A , Morsi A , et al. Longterm effect of intensive lifestyle intervention on cardiovascular risk factors in patients with diabetes in real-world clinical practice: a 5-year longitudinal study. BMJ Open Diabetes Res Care 2017; 5: e000259.
10. Henson J, Covenant A, Hall AP, et al. Waking up to the importance of sleep in type 2 diabetes management: a narrative review. Diabetes Care 2024; 47: 331-343.
11. Klupa T, Benbenek-Klupa T, Matejko B, et al. The impact of a pure protein load on the glucose levels in type 1 diabetes patients treated with insulin pumps. Int J Endocrinol 2015; 2015: 216918.
12. Lean ME, Leslie WS, Barnes AC, et al. Primary careled weight management for remission of type 2 diabetes (DiRECT): an open-label, cluster-randomised trial. Lancet 2018; 391: 541–551.
13. Lewgood J, Oliveira B, Korzepa M, et al. Efficacy of dietary and supplementation interventions for individuals with type 2 diabetes. Nutrients 2021; 13: 2378.
14. Matejko B, Gawrecki A, Wrobel M, et al. Physiological characteristics of type 1 diabetes patients during high mountain trekking. J Diabetes Res 2020: 8068710.
15. Papamichoua D, Panagiotakosb DB, Itsiopoulosa C. Dietary patterns and management of type 2 diabetes: a systematic review of randomised clinical trials. Nutr Metab Cardiovasc Dis 2019; 29: 531–543.
16. Pawlak R. Vegetarian diets in the prevention and management of diabetes and its complications. Diabetes Spectr 2017; 30: 82–88.
17. Perkins BA, Turner LV, Riddell MC. Applying technologies to simplify strategies for exercise in type 1 diabetes. Diabetologia 2024; 67: 2045–2058.
18. Riddell MC, Gallen IW, Smart CE, et al. Exercise management in type 1 diabetes: a consensus statement. Exercise management in type 1 diabetes: a consensus statement. Lancet Diabetes Endocrinol 2017; 5: 377–390.
19. Schwingshackl L, Chaimani A,Hoffmann G, et al. A network meta-analysis on the comparative efficacy of different dietary approaches on glycaemic control in patients with type 2 diabetes mellitus. Eur J Epidemiol 2018; 33: 157–170.
20. Scientific Advisory Committee on Nutrition (SACN). Lower carbohydrate diets for adults with type 2 diabetes. May 2021. Available from: www.gov.uk/government/groups/scientific-advisory-committee-on-nutrition.
21. Shapira N. The metabolic concept of meal sequence vs. satiety: glycemic and oxidative responses with reference to inflammation risk, protective principles and mediterranean diet. Nutrients 2019; 11: 2373.
22. Shukla AP, Andono J, Touhamy SH, et al. Carbohydrate – last meal pattern lowers postprandial glucose and insulin excursions in type 2 diabetes. BMJ Open Diab Res Care 2017: 5: e000440.
23. The Members of The Joslin Clinical Oversight Committee. Evidence-based diabetes management. Am J Manag Care 2018; 4: SP204–SP262.
24. Thorsen IK, Johansen MY, Pilmark NS, et al. The effect of frequency of activity interruptions in prolonged sitting on postprandial glucose metabolism: a randomized crossover trial. Metabolism 2019; 96: 1–7.
25. Williams PG. The benefits of breakfast cereal consumption: a systematic review of the evidence base. Adv Nutr 2014; 5: 636S–673S.

7. Principles of Physical Activity and Sports Participation for Individuals with Diabetes

CHAPTER HIGHLIGHTS:
• Individuals with type 1 diabetes without clinically significant chronic diabetes-related complications may engage in all forms of physical activity, including high-intensity exercise. [**C**]
• Aerobic exercise performed up to the point of moderate breathlessness is safe and may be recommended for all individuals with diabetes who have no contraindications. [**B**]
• In individuals with type 2 diabetes, the addition of resistance exercise to aerobic training is recommended. [**B**]
• Severe hypoglycaemia is a contraindication to physical activity for 24 hours. [**E**]
• Delayed hypoglycaemia may occur up to 24 hours after completing physical activity. [**B**]
• Proliferative diabetic retinopathy is a contraindication to physical activity until stabilisation of retinal findings is achieved. [**E**]
• Hyperglycaemia > 250 mg/dl without confirmed ketonaemia and/or ketonuria is not a contraindication to physical activity after provided that the patient feels well and understands the cause of hyperglycaemia. [**E**]
• Principles of physical activity in competitive sports and competitions setting differ significantly from those in recreational sport and require individually tailored approaches. [**E**]

I. Recommended duration and intensity of physical activity.

Engagement in physical activity by an individual with diabetes should be assessed by a diabetologist based on evaluation of the individual's level of physical activity (type, duration, and intensity of exercise), potential contraindications, as well as expectations, knowledge, skills related to hypoglycaemia prevention, and current level of physical conditioning. In individuals with type 2 diabetes aged over 65 years and/or with obesity, as well as in patients after a cardiovascular event and those with cardiovascular disease, monitoring of heart rate and assessment of exercise intensity using the Borg scale are recommended. Target heart rate ranges and exercise intensity may be determined during an electrocardiographic exercise test and/or a cardiopulmonary exercise (spiroergometric) test. In this group of patients, aerobic exercise performed up to the point of breathlessness is safe and should be recommended for at least 150 minutes per week. In adult individuals with diabetes, increasing physical activity to up to 300 minutes per week may be associated with additional health benefits without a significant increase in the risk of adverse events. These recommendations may also be implemented in a seated position in patients with mobility limitations. In this group, replacing sedentary time with physical activity of any intensity, including low-intensity activity, is recommended.

A target of 10,000 steps per day may be considered for more physically active individuals with diabetes. However, a range of 6,000–8,000 steps per day, taking into account individuals aged over 60 years, is associated with a reduction in cardiovascular and all-cause mortality.

Individuals with diabetes (without significant contraindications) are recommended to engage in daily vigorous physical activity, including active participation in sports.

A motivating factor for increasing physical activity and improving safety is the use of physical activity monitoring devices, wearable activity trackers (e.g. smartwatches or fitness bands).

II. Contraindications to physical activity

Contraindications to recreational sports participation are discussed in Chapter 6. Decisions made by the diabetologist may require consultation with other specialists, including an ophthalmologist, cardiologist, nephrologist, and neurologist.

Annex 6 of the Diabetes Poland recommendations presents contraindications to participation in sports training and competitive events.

III. Glycaemic monitoring during physical activity

In individuals treated with insulin who are physically active, the use of continuous glucose monitoring (CGM) systems is recommended. The role of the therapeutic team includes assistance in selecting an appropriate CGM system, education on interpretation of glucose data, individual programming of higher hypoglycaemia alert thresholds, and guidance on securing the CGM device during sports activities. When self-monitoring of blood glucose is

performed using a glucometer, blood glucose levels should be measured up to 15 minutes before the start of physical activity, during exercise, and after completion of physical activity. Informing accompanying persons during physical activity about the presence of diabetes significantly facilitates effective glycaemic self-monitoring and management.

IV. Hypoglycaemia and hyperglycaemia related to physical activity

Changes in glycaemia during physical activity are illustrated in Figure 7.1.

Severe hypoglycaemia is a contraindication to undertaking physical activity for 24 hours.

In the event of a hypoglycaemia alert ≤ 70 mg/dl, rapidly absorbable carbohydrates should be consumed, preferably in liquid form, and physical activity may be resumed after resolution of hypoglycaemic symptoms.

In the case of severe hypoglycaemia in individuals with type 1 diabetes, the effectiveness of glucagon after intensive physical activity may be reduced; nevertheless, an attempt to administer glucagon should always be made.

Late-onset hypoglycaemia may occur up to 24 hours after completion of physical activity, and the risk is higher in untrained individuals and those engaging in physical activity irregularly. This group should pay particular attention to prevention of nocturnal hypoglycaemia.

Anaerobic exercise may lead to hyperglycaemia. Correction with rapid-acting insulin should be undertaken cautiously due to the risk of hypoglycaemia occurring several hours after completion of physical activity.

If hyperglycaemia > 250 mg/dl is present and accompanied by ketonuria and/or ketonaemia ≥ 1.5 mmol/l, physical activity is contraindicated.

If hyperglycaemia > 250 mg/dl is not accompanied by ketonuria and/or ketonaemia and/or the cause of hyperglycaemia is known, light to moderate physical activity may be undertaken.

V. Principles of physical activity in individuals with type 2 diabetes not requiring insulin therapy

In individuals with diabetes who do not use insulin or sulfonylurea derivatives, the risk of hypo-

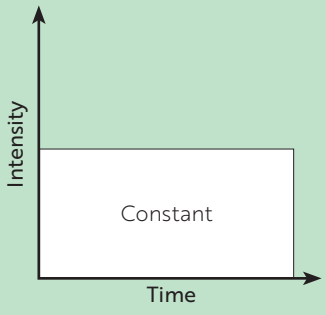
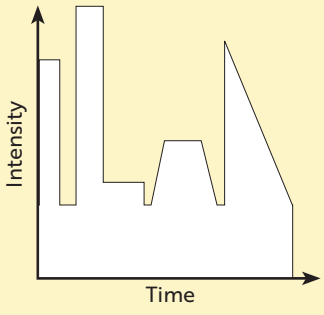
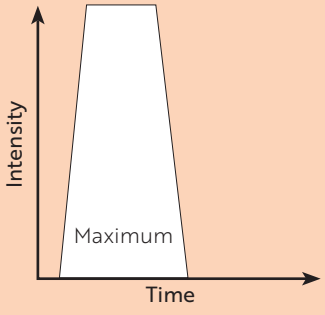
Type of exercise	**Aerobic** Examples: walking, Nordic walking, light cycling, jogging	**Mixed (aerobic-anaerobic)** Examples: team sports, fast running, swimming, interval cycling	**Anaerobic** Examples: sprints, resistance training with maximum load, explosive exercises
Intensity			
Heart rate range	< 55 (60)% HR_{max}	60–75 (80)% HR_{max}	> 75 (80)% HR_{max}
Borg scale	7–11	12–15	16–20
Expected glycaemic response	Decrease	Decrease and/or increase	Increase
Risk of hypoglycaemia	High	Increased	Low
Reduction of prandial insulin bolus for activity lasting > 30 minutes (not applicable to automated insulin delivery systems)	25% for low-intensity exercise 50% for higher-intensity exercise lasting > 45 minutes 75% for prolonged, intensive aerobic exercise lasting several hours	Reduction may be required 25% if a slight decrease in glycaemia is expected 50% for moderate-intensity exercise with an expected rapid decrease in glycaemia	Dependent on the athlete's experience No reduction of prandial bolus Administration of a small insulin bolus before or during physical activity

Figure 7.1. Types of physical activity and their impact on glycaemia changes

glycaemia is very low. Blood glucose values below 100 mg/dl do not require consumption of additional carbohydrate portions. Self-monitoring of blood glucose in relation to physical activity should be performed only periodically.

Regular physical activity improves insulin sensitivity and thereby increases the likelihood of delaying the initiation of insulin therapy. An important complement to aerobic training is the inclusion of resistance (strength) exercises. Training should involve large muscle groups, with 8–12 repetitions performed 2–3 times per week.

VI. Principles of physical activity in individuals treated with insulin

Regardless of the type of exercise, the target glycaemic range should be 126–180 mg/dl, or slightly higher in individuals at increased risk of hypoglycaemia and/or with impaired hypoglycaemia awareness. Initiation of physical activity is not recommended when blood glucose is below 90 mg/dl. In the case of resistance exercise and expected hyperglycaemia, there are no contraindications to starting physical activity at a glucose level of 90 mg/dl.

Physical activity undertaken within 2 hours of administration of a rapid-acting insulin analogue requires a reduction of the insulin dose if the activity lasts at least 30 minutes.

Reduction of the prandial insulin bolus may range from 25% to 75% and depends on the duration and intensity of physical activity.

Physical activity requires consumption of additional carbohydrates in the following amounts:

- 1.0–1.5 g/kg body weight per hour of intensive physical activity during the peak action of a prandial insulin bolus that has not been reduced;
- 0.2–0.5 g/kg body weight per hour of intensive physical activity during the peak action of a prandial insulin bolus that has been reduced or that was administered more than 2 hours before the start of physical activity.

The maximum duration of insulin pump disconnection during physical activity should not exceed 3 hours. A prerequisite for insulin pump disconnection is the presence of active insulin, the amount of which should be monitored using a bolus calculator.

Reduction of basal insulin administered as NPH (Neutral Protamine Hagedorn), or long-acting insulin analogues should be considered in the case of prolonged or all-day endurance exercise. A preceding reduction of the dose of ultra-long-acting insulin analogues by 25% may be considered before all-day or multi-day physical activities.

During insulin pump therapy, a reduction of the basal insulin infusion rate by 20–80% is recommended, depending on the intensity and duration of physical activity, preferably starting 2 hours before the onset of exercise.

VII. Principles of undertaking physical activity in individuals treated with automated insulin delivery systems

The use of a hybrid closed-loop system during physical activity still requires the patient to plan exercise and modify therapy. Separate education in this area is required.

Recommended therapy modifications include:

- setting a higher target glucose value 90 minutes (1–2 hours) before starting physical activity lasting longer than 30 minutes, particularly in the case of aerobic exercise,
- activating a function that reduces insulin delivery and is dedicated to physical activity ("Ease-off" for CamAPS FX; "Temporary target" for 780G) 90 minutes (1–2 hours) before planned physical activity,
- meals consumed up to 2 hours before exercise require a reduction in the insulin dose by 25–33%,
- intake of additional portions of carbohydrates:
  - » they should not be entered into the system,
  - » consumption of carbohydrates is recommended 5–10 minutes before the start of exercise, with amounts usually smaller than in the case of therapy using a conventional insulin pump,
  - » consumption of carbohydrates earlier than 20 minutes before physical activity will result in increased insulin delivery by the hybrid closed-loop system, which may lead to hypoglycaemia;
- suspension of insulin delivery:
  - » if the insulin pump is disconnected during physical activity, pump operation must be suspended,
  - » the maximum duration of insulin pump disconnection or suspension during physical activity should not exceed 3 hours.

VIII. Principles of undertaking physical activity in women with hyperglycaemia during pregnancy

It is recommended that all women with hyperglycaemia during pregnancy and after delivery (in the absence of medical contraindications) undertake physical activity during this period. Moderate physical activity contributes to a reduction in the incidence of gestational diabetes mellitus (GDM), gestational hypertension, preterm delivery, and caesarean section.

Moderate-intensity aerobic physical activity is recommended for at least 150 minutes per week (3–4 times per week, with exercise sessions lasting 30–60 minutes). Exercise intensity should be lower than 60–80% of the maximum heart rate for maternal age, most commonly not exceeding 140 beats per minute. Static aerobic and muscle-strengthening exercises may also be performed. The addition of stretching exercises may also be beneficial.

Preferred forms of physical activity include walking, stationary cycling, dancing, water aerobics, stretching exercises, and lifting light weights. Lifestyle modification and physical activity are essential components of management in GDM and may constitute sufficient therapy or delay initiation of insulin therapy in many women.

In patients treated with insulin for pregestational diabetes mellitus (PGDM), physical activity requires reduction of insulin doses in the basal infusion and/or insulin boluses, in accordance with the principles applied prior to pregnancy.

Undertaking additional physical activity (beyond daily activity) requires consultation with a gynaecologist.

REFERENCES

1. Adolfsson P, Taplin CE, Zaharieva DP, et al. ISPAD Clinical Practice Consensus Guidelines 2022: exercise in children and adolescents with diabetes. Pediatr Diabetes 2022; 23: 1341-1372.
2. Paluch AE, Bajpai S, Bassett DR, et al.; Steps for Health Collaborative. Daily steps and all-cause mortality: a meta-analysis of 15 international cohorts. Lancet Public Health 2022; 7: e219–e228. DOI: 10.1016/S2468-2667(21)00302-9.
3. Berghella V, Saccone G. Exercise in pregnancy! Am J Obstet Gynecol 2017; 216: 335–337.
4. de Oliveira VLP, de Paula TP, Viana LV. Pedometer- and accelerometer-based physical activity interventions in type 2 diabetes: a systematic review and meta-analysis. Nutr Metab Cardiovasc Dis 2024; 34: 548–558.
5. Ding D, Nguyen B, Nau T, et al. Daily steps and health outcomes in adults: a systematic review and dose-response meta-analysis. Lancet Public Health 2025; 10: e668–e681. DOI: 10.1016/S2468-2667(25)00164-1.
6. Jelleyman C, Yates T, O'Donovan G, et al. The effects of high-intensity interval training on glucose regulation and insulin resistance: a meta-analysis. Obes Rev 2015; 16: 942–961.
7. Laredo-Aguilera JA, Gallardo-Bravo M, Rabanales-Sotos JA, et al. Physical activity programs during pregnancy are effective for the control of gestational diabetes mellitus. Int J Environ Res Public Health 2020; 17: 6151. DOI: 10.3390/ijerph17176151.
8. Moser O, Müller A, Aberer F, et al. Comparison of insulin glargine 300 U/ml and insulin degludec 100 U/ml around spontaneous exercise sessions in adults with type 1 diabetes: a randomized cross-over trial (ULTRAFLEXI-1 Study). Diabetes Technol Ther 2023; 25: 161–168.
9. Moser O, Riddell MC, Eckstein ML, et al. Glucose management for exercise using continuous glucose monitoring (CGM) and intermittently scanned CGM (isCGM) systems in type 1 diabetes: position statement of the European Association for the Study of Diabetes (EASD) and of the International Society for Pediatric and Adolescent Diabetes (ISPAD) endorsed by JDRF and supported by the American Diabetes Association (ADA). Diabetol 2020; 63: 2501–2520.
10. Moser O, Zaharieva D, Adolfsson P, at al. The use of automated insulin delivery around physical activity and exercise in type 1 diabetes: a position statement of the European Association for the Study of Diabetes (EASD) and the International Society for Pediatric and Adolescent Diabetes (ISPAD). Horm Res Paediatr 2024. DOI: 10.1159/000542287.
11. Ostman C, Jewiss D, King N, et al. Clinical outcomes to exercise training in type 1 diabetes: a systematic review and meta-analysis. Diabetes Res Clin Pract 2018; 139: 380–391.
12. Paldus B, Morrison D, Lee M, et al. Strengths and challenges of closed-loop insulin delivery during exercise in people with type 1 diabetes: potential future directions. J Diabetes Sci Technol 2023; 17: 1077–1084.
13. Peng P, Zhang N, Huang J, et al. Effectiveness of wearable activity monitors on metabolic outcomes in patients with type 2 diabetes: a systematic review and meta-analysis. Endocr Pract 2023; 29: 368–378.
14. Perkins BA, Turner LV, Riddell MC. Applying technologies to simplify strategies for exercise in type 1 diabetes. Diabetologia 2024; 67: 2045–2058.
15. Riddell MC, Gallen IW, Smart CE, et al. Exercise management in type 1 diabetes: a consensus statement. Lancet Diabetes Endocrinol 2017; 5: 377–396.
16. Riddell MC, Scott SN, Fournier PA, et al. The competitive athlete with type 1 diabetes. Diabetologia 2020; 63: 1475–1490.

17. Sluik D, Buijsse B, Muckelbauer R, et al. Physical activity and mortality in individuals with diabetes mellitus: a prospective study and meta-analysis. Arch Intern Med 2012; 172: 1285–1295.
18. Stanford KI, Goodyear LJ. Exercise and type 2 diabetes: molecular mechanisms regulating glucose uptake in skeletal muscle. Adv Physiol Educ 2014; 38: 308–314.
19. Tikkanen-Dolenc H, Wadén J, Forsblom C, et al.; FinnDiane Study Group. Physical activity reduces risk of premature mortality in patients with type 1 diabetes with and without kidney disease. Diabetes Care 2017; 40: 1727–1732.
20. WHO guidelines on physical activity and sedentary behaviour. Available from: https://www.who.int/publications/i/item/9789240015128.
21. Wilson LM, Jacobs PG, Riddell MC, et al. Opportunities and challenges in closed-loop systems in type 1 diabetes. Lancet Diabetes Endocrinol 2022; 10: 6–8.

8. Psychological Care in Diabetes

CHAPTER HIGHLIGHTS
• Psychosocial factors have a significant impact on the clinical and psychological status of individuals with diabetes. [**B**]
• Whenever possible, assessment of the patient's mental state should be performed at the initiation of diabetes treatment and subsequently at every medical visit. [**B**]
• Specific mental disorders frequently coexist with diabetes and significantly increase the risk of developing diabetes-related complications. [**A**]
• In individuals with diabetes, the presence of anxiety symptoms, diabetes-related distress, addictions, depression, eating disorders, and impairment of cognitive functioning should be assessed. These conditions may substantially impair adaptation to the disease. [**B**]
• Psychological and social care should be integrated with a collaborative, patient-centred approach and be available to all individuals with diabetes in order to optimise treatment outcomes and quality of life. [**A**]

The patient's mental state (sense of well-being) affects almost all aspects of therapeutic management. Poor adherence to recommendations is very often associated with psychological problems that require diagnosis and appropriate psychotherapeutic interventions. For this reason, education based solely on the provision of information about prescribed treatment and recommended management is of limited effectiveness. The patient's mental state should be assessed at the initiation of diabetes treatment and subsequently at every medical visit. The use of appropriately designed questionnaires and tests is recommended for this purpose. Under optimal conditions, this assessment is performed by a psychologist who is a member of the therapeutic team. In the absence of access to a psychologist, such an assessment should be carried out by the attending physician.

I. The most common emotional problems occurring in individuals with diabetes

- Diabetes distress (DD), defined as chronic negative emotions related to diabetes, including the following dimensions:
  - » emotional burnout scale,
  - » patient–physician relationship-related stress scale,
  - » treatment regimen-related stress scale,
  - » diabetes-related social stress scale,
- depression,
- fear of hypoglycaemia (FoH),
- fear of complications,
- specific eating disorders, so-called diabulimia,
- alarm fatigue,
- fear of stigmatisation,
- sleep disorders,
- cognitive disorders.

II. Optimal management should include:

- appropriate communication,
- continuous assessment (monitoring) of the patient's mental state and adherence to medical recommendations,
- when necessary, referral for psychological support, psychiatric care, or psychotherapy.

III. An individualised approach to a person with diabetes aims to:

- jointly establish with the patient an optimal treatment strategy whose implementation, in the opinion of the person with diabetes, is

feasible in their current life (psychosocial) situation – shared decision making; the possibility of using modern technologies in diabetes treatment should be taken into account,
- fostering motivation for optimal self-management,
- avoiding frightening the patient with the consequences of non-adherence to medical recommendations, which in most cases is ineffective and harmful,
- applying an optimal educational approach based on psychological assessment,
- using appropriate, non-judgemental language, e.g. "glucose levels outside the TIR range" instead of "bad sugars", "high glycaemic variability" instead of "poor control", etc.

IV. Assessment of the mental state (psychological diagnosis) of a person with diabetes in clinical practice includes:

- social and psychological (life) situation,
- patient's quality of life,
- attitudes, beliefs, difficulties, and responsibilities related to diabetes (unjustified fears and worries may impair the ability to cope with the disease),
- sense of influence over the course of the disease (an inadequate sense of control over diabetes leads to coping styles characterised by avoidance of thinking about the disease and/or reduction of emotions triggered by the disease),
- assessment of coping styles related to the disease (a reduced tendency to seek optimal coping strategies and a decline in problem-focused coping styles related to the disease are observed),
- assessment of the severity of diabetes-specific disorders: diabetes distress, depression, fear of hypoglycaemia (FoH), fear of complications, eating disorders,
- assessment of general anxiety symptoms, addictions, and impairment of cognitive processes (which may significantly hinder adaptation to diabetes).

V. Basic methods for verifying the emotional state of a patient with diabetes

1. To assess the risk of depression, use screening tools freely available online:

The World Health Organization-Five Well-Being Index (WHO-5; www.who-5.org) – a score < 13 indicates the need for further assessment for depression, and a score < 7 indicates a high risk of depression,
- Patient Health Questionnaire-9 (PHQ-9; www.phqscreeners.com/overview.aspx) – a score < 5 indicates normal mood, 5–9 mild depression, 10–14 moderate depression, 15–19 moderately severe depression, and 20–27 severe depression; in the Polish version, a score > 12 indicates a high risk of depression,

and/or ask the following two questions:
- during the past month, have you often been bothered by feeling down, depressed, or hopeless?
- during the past month, have you often been bothered by little interest or pleasure in doing things?

A positive answer to at least one of these questions has a sensitivity of 97% and a specificity of 67% for the diagnosis of depression. In such cases, the patient should be referred for psychiatric consultation.

2. Diabetes distress should be assessed and monitored systematically, at least once a year, as part of diabetology care. Regular monitoring of diabetes distress supports early identification of problems and enables implementation of appropriate psychological support. Assessment should be performed using validated tools that allow identification of sources of diabetes-related emotional burden and monitoring of their changes over time. The Polish versions of the DDS-17, PAID, and WHO-5 questionnaires, together with instructions for use, are available in a dedicated section on the website of Diabetes Poland (PTD).
3. To assess fear of late complications, ask the following question: To what extent are you worried about the future and the possibility of developing serious complications?
- 0: this is not a problem;
- 1: this is a minor problem;
- 2: this is a moderate problem;
- 3: this is quite a serious problem;
- 4: this is a serious problem.

A score of three points or more indicates a significant risk of developing psychosocial problems. In case of any doubts, a psychological assessment by a qualified psychologist should be requested.

VI. Psychological interventions in individuals with diabetes include:

- developing a sense of influence over the course of the disease through:

  - » providing the patient with information about the disease and its treatment in a manner that is understandable to them,
  - » jointly formulating therapeutic goals and plans that, in the patient's opinion, are realistic,
  - » gradual progression towards an optimal level of adherence to recommendations (the "small steps" strategy),
  - » offering support in the event of failure to implement previously agreed plans (so that the person with diabetes knows that the physician will help identify the cause of failure and will not adopt a negative attitude),
  - » offering access to psychological self-help applications,
- shaping and maintaining a problem-oriented coping style focused on managing diabetes-related challenges,
- individualised use of modern technologies as support in daily functioning, improvement of quality of life, reduction of diabetes distress, and building a sense of safety.

VII. Treatment of mental disorders in patients with diabetes

The presence of clinically significant depression (depressive episode, dysthymia) and other mental disorders requires psychiatric consultation. In the case of adjustment disorders related to adaptation to the disease, psychotherapeutic interventions may be undertaken by a primary care physician or a specialist. In more complex cases, assistance from a clinical psychologist and, if necessary, a psychotherapist is required.

VIII. Team-based care

A key condition for effective therapy is a consistent approach across the entire therapeutic team. Effective communication among team members is essential. In diabetology outpatient clinics, a psychologist is an indispensable member of the specialist treatment team.

REFERENCES

1. Anderson RJ, Freedland KE, Clouse RE, et al. The prevalence of comorbid depression in adults with diabetes: a meta-analysis. Diabetes Care 2001; 24: 1069–1078.
2. Anderson RJ, Grigsby AB, Freedland KE, et al. Anxiety and poor glycemic control: a meta-analytic review of the literature. Int J Psych Med 2002; 32: 235–247.
3. Aquino JA, Baldoni NR, Flor CR, et al. Effectiveness of individual strategies for the empowerment of patients with diabetes mellitus: a systematic review with meta-analysis. Prim Care Diabetes 2018; 12: 97–110.
4. Baldoni NR, Aquino JA, Sanches-Giraud C, et al. Collective empowerment strategies for patients with diabetes mellitus: a systematic review and meta-analysis. Prim Care Diabetes 2017; 11: 201–211.
5. Bombaci B, Passanisi S, Longo A, et al. The interplay between psychological well-being, diabetes-related distress, and glycemic control: a continuous glucose monitoring analysis from a population of adolescents with type 1 diabetes. J Diabetes Complications 2025; 39: 109142. DOI: 10.1016/j.jdiacomp.2025.109142.
6. Cichoń E, Kiejna A, Kokoszka A, et al. People with diabetes need a lower cut-off than others for depression screening with PHQ-9. PLoS One 2020; 15: e0240209. DOI: doi: 10.1371/journal.pone.0240209.
7. Cichoń E, Kiejna A, Kokoszka A, et al. Validation of the Polish version of WHO-5 as a screening instrument for depression in adults with diabetes. Diabetes Res Clin Pract 2020; 159: 107970. DOI: doi: 10.1016/j.diabres.2019.107970.
8. https://www.easd.org/guidelines/statements-guidelines/diabetes-distress/
9. Kokoszka A, Jastrzębski A, Obrębski M. Ocena psychometrycznych właściwości polskiej wersji Kwestionariusza Zdrowia Pacjenta-9 dla osób dorosłych. Psychiatria 2016; 13: 187–193.
10. Kovacs Burns K, Nicolucci A, Holt RIG, et al.; DAWN2 Study Group. Diabetes Attitudes, Wishes and Needs second study (DAWN2): cross-national benchmarking indicators for family members living with people with diabetes. Diabet Med 2013; 30: 778–788.
11. Lloyd CE, Nouwen A, Sartorius N, et al. Prevalence and correlates of depressive disorders in people with type 2 diabetes: results from the International Prevalence and Treatment of Diabetes and Depression study, a collaborative study carried out in 14 countries. Diabet Med 2018; 35: 760–769.
12. Nouwen A, Adriaanse M, van Dam K, et al.; European Depression in Diabetes (EDID) Research Consortium. Longitudinal associations between depression and diabetes complications: a systematic review and meta-analysis. Diabet Med 2019; 36: 1562–1572.
13. Snoek FJ, Anarte-Ortiz MT, Anderbro T, et al. Roles and competencies of the clinical psychologist in adult diabetes care – a consensus report. Diabet Med 2024; 41: e15312. DOI: 10.1111/dme.15312.
14. Young-Hyman D, de Groot M, Hill-Briggs F, et al. Psychosocial care for people with diabetes: a position statement of the American Diabetes Association. Diabetes Care 2016; 39: 2126–2140.

9. Therapeutic Education

CHAPTER HIGHLIGHTS:
• Therapeutic education (diabetological and dietary), as well as re-education of all individuals with diabetes and those at risk of developing diabetes and with preclinical stages of type 1 diabetes, are key elements of effective disease management and prevention of its complications. [**A**]
• Patients, as well as their caregivers, should be provided with guaranteed access to education in order to acquire the knowledge and skills necessary for independent disease self-management. [**A**]
• The key objectives of education include achievement of therapeutic targets, optimisation of metabolic control, and improvement of the quality of life of the patient and their family. The implementation of these objectives should be regularly reviewed during follow-up visits. [**B**]
• Assessment of needs in the areas of self-care, education, and support should be carried out in the following situations: at diagnosis; annually when therapeutic targets are not achieved; after significant modification of therapy; in the event of factors influencing the course of the disease (clinical, physical, or psychosocial); and during periods of change in the life of a person with diabetes or in the organisation of care. The aim of such assessment is to support and develop skills related to implementation of the treatment plan, nutritional management, and maintenance of well-being. [**E**]
• A consistent position adopted by members of the therapeutic team (physician, nurse, diabetes educator, dietitian, psychologist) increases the effectiveness of education and improves psychological aspects of treatment. The therapeutic team is led by a physician. [**B**]
• The therapeutic team should possess the knowledge and skills required to deliver personalised education. [**B**]
• Tele-education is an effective method of delivering knowledge and support in the area of diabetes self-management. [**B**]
• The creation and use of structured educational programmes, including at a nationwide level, are recommended. [**A**]

I. General recommendations

- Education constitutes an integral part of diabetes treatment. In specialist diabetology care, it should be addressed to individuals with diabetes, including those in preclinical stages of type 1 diabetes. Individuals with prediabetes should receive education within primary health care. Caregivers and family members of people with diabetes may also be included in educational activities.
- Education should be delivered by an interdisciplinary therapeutic team under the supervision of the physician responsible for treatment. The team includes a physician, nurse, diabetes educator, dietitian, and psychologist. Education should be provided systematically during medical, educational, and dietary consultations, and its content should be regularly updated according to the individual needs of the patient.
- The educational programme, coordinated with therapy, should be developed with the active participation of the patient, their caregivers, and family members. Establishing individual therapeutic goals is essential.
- The key objectives of education are the transfer of knowledge and support of patients in independent disease self-management, lifestyle modification, maintenance of healthy dietary habits, and regular physical activity, with the aim of improving health status, minimising the risk of complications, and supporting overall patient well-being.
- Behavioural strategies should support diabetes self-management and patient engagement in health-promoting behaviours (e.g. medication adherence, physical activity, healthy nutrition, use of modern technologies) in order to achieve optimal treatment outcomes.
- The most effective educational programmes are personalised and adapted to the individual needs, experiences, and capabilities of participants. Diabetes education should take into account the age, cognitive and developmental level, and cognitive abilities of the person receiving education, and in the case of minors or individuals with intellectual limitations, also the needs and competencies of their caregivers.
- Programmes dedicated to individuals in preclinical stages of type 1 diabetes, pregnant women, older adults, and their caregivers or family members should address their specific needs.
- The therapeutic team cooperates with the patient's environment, ensuring continuity of care, confidentiality of information, and communication of up-to-date recommendations.
- Responsibility for assessing the extent to which planned educational outcomes have

been achieved lies with the person delivering the education.
- Education may be provided individually or in groups.

II. Framework programme of therapeutic education

- Emotional support: assistance in accepting the diagnosis, strengthening motivation, and developing independence and a sense of responsibility, including responsibility for self-education.
- Medical fundamentals: information on the disease, treatment, mechanisms of action of medications and self-monitoring techniques; criteria for the diagnosis of glucose metabolism disorders, including diabetes; classification of diabetes; pathomechanisms; physiology of glucose homeostasis; ranges of normal glycaemic and ketone values (appropriate to the type and stage of diabetes).
- Individual therapeutic goals: establishment of goals taking into account the conditions and needs of the person receiving education. Monitoring of health indicators, including target and individual values and glycaemic variability, time in range, glycated haemoglobin, lipid levels, blood pressure, body weight, and patient well-being; additionally, in children and adolescents, harmonious physical development and pubertal maturation. The monitoring process should also consider the presence of comorbidities and quality of life.
- Monitoring and methods of glucose and ketone measurement using glucometers: measurement techniques and interpretation of results.
- Continuous glucose monitoring systems: discussion of principles of operation and benefits of CGM use, selection of an appropriate system, and development of skills for interpreting the obtained data.
- Pharmacotherapy:
  - provision of knowledge regarding oral and injectable antihyperglycaemic medications and insulin therapy – mechanisms of action, dosing, potential adverse effects, and principles of medication storage,
  - training in correct administration of insulin and other injectable medications using pens or syringes,
  - principles of self-adjustment of medication doses,
  - principles of intensive functional insulin therapy,
  - treatment using personal insulin pumps, including automated insulin delivery (AID) systems (the scope of education is discussed in Annex 5).
- Principles of a healthy lifestyle: behavioural management, with particular emphasis on dietary treatment (the scope of education is discussed in Chapter 6).
- Acute (hypoglycaemia and hyperglycaemia with diabetic ketoacidosis) and chronic diabetes complications (cutaneous complications, including post-insulin lipohypertrophy and lipoatrophy, microvascular and macrovascular complications, neuropathy, and diabetic foot syndrome): recognition, treatment, and prevention.
- Management in clinically significant situations such as disease progression, failure to achieve therapeutic goals, occurrence of additional factors affecting glycaemia (including infections, comorbidities, or risk behaviours), and significant life changes of the patient, including pregnancy planning, pregnancy, contraception use, travel, and changes in time zones.
- Psychological support: at disease onset, in diabetes-related burnout, difficulties with therapeutic adherence, diabetes-related stress, depression, eating disorders, quality of life, sleep and sexual functioning, in relation to the risk of diabetes in family members, and expectations regarding treatment and its outcomes.
- Vaccinations: their role and importance, as well as recommended vaccinations in specific age groups.
- Social rights: information on the rights of individuals with diabetes, including employment, driving licences, insurance, disability status, and reimbursement policies.
- Use of specialist health care: principles of preparing a person with diabetes for a medical visit (including data downloads and transmission from device memory, basic interpretation of reports, and formulation of the health problem), the importance of regular visits and examinations, and adherence to medical recommendations. Development of an individual model for transition of adolescents from paediatric to adult diabetology care: see Annex 7.
- Current information on ongoing research and clinical interventions slowing disease progression, new medications or new technologies,

as well as associations and foundations for people with diabetes: opportunities to obtain support from patient organisations and membership in such organisations.
- In individuals with preclinical stages of type 1 diabetes, it is important to provide them and their families with basic knowledge about the autoimmune process, stages of the disease, risk and prevention of ketoacidosis at the diagnosis of stage 3 type 1 diabetes, principles and habits of healthy nutrition, and support in coping with the new situation.

III. Organisation of therapeutic education

Therapeutic education is provided in specialised diabetology hospital wards and within outpatient specialist care (OSC). In OSC, the required education and re-education should be delivered during separate educational visits, upon referral by a physician.

- Individual outpatient educational visits address:
  - general knowledge about diabetes, principles of therapy and disease monitoring, and information on acute and chronic diabetes complications,
  - self-monitoring of glycaemia using continuous glucose monitoring systems,
  - initiation and management of functional intensive insulin therapy using injection devices,
  - implementation of the functional intensive insulin therapy method using an insulin pump, including automated insulin delivery (AID),
  - behavioural management, with particular emphasis on dietary treatment (dietetic consultation).
- The duration of education depends on the clinical stage of diabetes and the implemented treatment model:
  - oral and injectable antihyperglycaemic medications and simple insulin therapy: 1–2 hours,
  - multiple daily insulin injection therapy: 1–3 hours,
  - functional intensive insulin therapy: 3–5 hours,
  - functional intensive insulin therapy using an insulin pump, including AID: 9 hours.
- Financing: therapeutic education (diabetological and dietary) should be financed separately, independently of medical visits.

IV. Standards for centres providing education

- Educational infrastructure: separate educational rooms for group or individual education, equipped with educational tools, including multimedia resources.
- Use of digital methods in education and self-education, including tele-education, webinars, mobile applications, reliable websites, and digital coaching. Remote methods should complement and support traditional forms of education and be adapted to patient preferences.
- Medical documentation related to education should include: the scope of education, taking into account the tools used, the patient's current needs, the outcomes of the educational process, and therapeutic recommendations.
- Assessment of education quality by patients and their caregivers: feedback collected after completion of the educational process.
- The employer should create conditions enabling continuous professional development and improvement of qualifications.

REFERENCES

1. Ammmentorp J, Thomsen J, Kofoed PE, et al. Understanding how different mechanism of life coaching offered to young adults with type 1 diabetes can improve their ability to see opportunities and overcome barriers. Patient Educ Couns 2020; 103: 544–548.
2. Brorsson AL, Leksell J, Andersson Franko M, et al. A person-centered education for adolescents with type 1 diabetes – a randomized controlled trial. Pediatr Diabetes 2019; 20: 986–996.
3. Buysse H, Coremans P, Pouwer F, et al. Sustainable improvement of HbA1c and satisfaction with diabetes care after adding telemedicine in patients on adaptable insulin regimens: results of the TeleDiabetes randomized controlled trial. Health Informatics J 2020; 26: 628–641.
4. Chrvala CA, Sherr D, Lipman RD. Diabetes self-management education for adults with type 2 diabetes mellitus: a systematic review of the effect on glycemic control. Patient Educ Couns 2016; 99: 926–943.
5. D'Souza RS, Ryan M, Hawkes E, et al. Questionnaire-based service evaluation of the efficacy and usefulness of SEREN: a structured education programme for children and young people diagnosed with type 1 diabetes mellitus. BMJ Open Qual 2021; e001337. DOI: 10.1136/bmjoq-2021-001337.
6. Davis J, Hess Fischl A, Beck J, et al. 2022 national standards for diabetes self-management educations and support. Diabetes Care 2022; 45: 484–494.

7. Dickinson JK, Maryniuk MD. Building therapeutic relationships: choosing words that put people first. Clin Diabetes 2017; 35: 51–54.
8. ElSayed NA, Aleppo G, Aroda VR, et al. Facilitating positive health behaviors and well-being to improve health outcomes: standards of care in diabetes – 2023. Diabetes Care 2023; 46 (Suppl 1): S68–S96.
9. ElSayed NA, Aleppo G, Aroda VR, et al. Improving care and promoting health in populations: standards of care in diabetes – 2023. Diabetes Care 2023; 46 (Suppl 1): S10–S18.
10. Hatipoglu B, Pronovost PJ. Role of diabetes self-management education for our health systems and economy. J Clin Endocrinol Metab 2025; 110 (Supplement 2): S91–S99. DOI: 10.1210/clinem/dgae913.
11. He X, Li J, Wang B, et al. Diabetes self-management education reduces risk of all-cause mortality in type 2 diabetes patients: a systematic review and meta-analysis. Endocrine 2017; 55: 712–773.
12. Heller SR, Gianfrancesco C, Taylor C, et al. What are the characteristics of the best type 1 diabetes patient education programmes (from diagnosis to long-term care), do they improve outcomes and what is required to make them more effective? Diabet Med 2020; 37: 545–554.
13. Kim JY, Jin SM, Sim KH, et al. Continuous glucose monitoring with structured education in adults with type 2 diabetes managed by multiple daily insulin injections: a multicentre randomised controlled trial. Diabetologia 2024; 67: 1223–1234.
14. Kolb L. An effective model of diabetes care and education: the ADCES7 self-care behaviors™. Sci Diabetes Self Manag Care 2021; 47: 30–53.
15. Mauri A, Schmidt S, Sosero V, et al. A structured therapeutic education program for children and adolescents with type 1 diabetes: an analysis of the efficacy of the „Pediatric Education for Diabetes" project. Minerva Pediatr (Torino) 2021; 73: 159–166.
16. Olinder A, DeAbreu M, Greene S, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Diabetes education in children and adolescents. Pediatr Diabetes 2022; 23: 1229–1242.
17. Phillip M, Achenbach P, Addala A, et al. Consensus guidance for monitoring individuals with islet autoantibody-positive pre-stage 3 type 1 diabetes. Diabetes Care 2024; 47: 1276–1298.
18. Phillip M, Bergenstal RM, Close KL, et al. The digital/virtual diabetes clinic: the future is now – recommendations from an international panel on diabetes digital technologies introduction. Diabetes Technol Ther 2021; 23: 146–154.
19. Schlüter S, Freckmann G, Heinemann L, et al. Evaluation of the SPECTRUM training programme for real-time continuous glucose monitoring: a real-world multicentre prospective study in 120 adults with type 1 diabetes. Diabet Med 2021; 38: e14467. DOI: 10.1111/dme.14467.
20. Sharma V, Feldman M, Sharma RJ. Telehealth technologies in diabetes self-management and education. Diabetes Sci Technol 2024; 18: 148–158.
21. Speight J, Holmes-Truscott E, Garza M, et al. Bringing an end to diabetes stigma and discrimination: an international consensus statement on evidence and recommendations. Lancet Diabetes Endocrinol 2024; 12: 61–82.
22. Szewczyk A, Tobiasz-Kałkun N, Stefanowicz-Bielska A, et al. Practical guidelines for nursing and midwifery diabetes care – 2026. A position of the Polish Federation for Education in Diabetology. Pielęgniarstwo XXI wieku 2025; 24: 267–320.
23. Wang W, Samadbeik M, Puri G, et al. A scoping review of digital solutions in diabetes outpatient care: functionalities and outcomes. Int J Med Inform 2025; 202: 105967. DOI: 10.1016/j.ijmedinf.2025.105967.

10. Type 1 Diabetes – Standards of Care 2026

CHAPTER HIGHLIGHTS
· The recommended treatment model is intensive functional insulin therapy using multiple daily subcutaneous insulin injections or continuous subcutaneous insulin infusion (CSII) delivered via a personal insulin pump; systems of automated insulin delivery (AID) are characterised by the highest effectiveness in optimising metabolic control and improving quality of life. [**A**]
· A key element of type 1 diabetes therapy is the patient's acquisition of skills to modify insulin doses depending on the carbohydrate content of meals, current glycaemia, and planned physical activity. For optimisation of insulin dosing, knowledge of the impact of protein and fat on glycaemia is also important. [**E**]
· In individuals with type 1 diabetes, the use of insulin analogues is preferred due to a lower risk of hypoglycaemia and greater quality of life. [**A**]
· CGM systems should constitute the most important element of glycaemic self-monitoring in type 1 diabetes from the moment of diagnosis; only devices meeting the required standards of quality and safety should be used. [**A**]
· In individuals using CGM systems, one of the basic parameters for assessing diabetes control should be time spent in the target glycaemic range (time in range, TIR), optimally exceeding 70%. [**E**]
· All therapeutic decisions regarding the treatment of type 1 diabetes should be made in agreement with the patient and after obtaining their acceptance. [**E**]

I. Type 1 diabetes treatment

1. Individuals with type 1 diabetes absolutely require insulin therapy. Insulin treatment should be maintained even during periods of partial remission of the disease.
2. The recommended treatment model is intensive functional insulin therapy using multiple daily subcutaneous insulin injections or continuous subcutaneous insulin infusion (CSII) delivered via a personal insulin pump. A prerequisite for effective treatment is appropriately delivered education (according to the principles outlined in Chapter 9), enabling the person with diabetes to independently adjust insulin doses based on systematically performed self-monitoring of blood glucose using a glucometer or other devices registered for this purpose (in accordance with the principles described in Chapter 3). Treatment of adult patients with type 1 diabetes using a personal insulin pump should be conducted by physicians experienced in pump therapy. Possession of a certificate from the Insulin Pump Training Programme of Diabetes Poland is recommended. In individuals with type 1 diabetes, the use of insulin analogues is preferred due to a lower risk of hypoglycaemia and greater quality of life.
3. Optimisation of insulin dosing is an important aspect of insulin therapy in type 1 diabetes. Long-term use of supraphysiological doses of insulin without diagnostic evaluation of the causes of increased insulin requirements and without attempts at causal treatment – except in justified cases (additional illness, medications increasing insulin demand, stress) – may lead to adverse metabolic consequences as well as excessive weight gain.
4. A key element of type 1 diabetes therapy is the patient's acquisition of skills to modify insulin doses depending on the carbohydrate content of meals, baseline glycaemia, and planned physical activity. Knowledge of the effects of proteins and fats on glycaemia is also important for optimising insulin dosing; however, this is of lesser importance when automated insulin delivery (AID) systems are used.
5. The use of CGM systems is the preferred method of self-monitoring in type 1 diabetes; only devices meeting the required quality and safety standards should be used.
6. Particularly effective is the combination of CSII and CGM technologies in devices that automatically suspend insulin delivery during hypoglycaemia or imminent hypoglycaemia (predictive low glucose suspend), as well as in AID systems, which can also autonomously correct hyperglycaemia.
7. AID systems may be used at any stage of diabetes treatment, regardless of the patient's prior technological experience. Although there is a positive correlation between baseline metabolic control and outcomes achieved with AID systems, it should be emphasised that significant improvement in glycaemic control can be expected in every individual with type 1 diabetes, irrespective of pre-implementation results. It should be stressed that for many individuals with type 1 diabetes who, for various

reasons, have been unable for years to achieve good or even acceptable glycaemic control, the use of AID may be the only way to improve metabolic control and reduce the risk of diabetes-related complications. AID systems should be offered to every person with type 1 diabetes. The decision on their potential implementation should be made jointly with the patient, taking reimbursement and financial aspects into account.

8. Devices operating on principles similar to AID include pumps based on open-source APS (artificial pancreas system) applications, known as DIY (do-it-yourself) pumps. Although such systems may significantly improve metabolic control in many individuals, it must be emphasised that they are not certified systems and that responsibility rests with the patient.
9. Patients treated with semi-automated pumps (predictive insulin suspension), AID systems, or DIY systems require appropriately modified education that takes into account the specific characteristics of these devices. One clinically important difference concerns the management of hypoglycaemia, where smaller amounts of glucose (5–15 g) are usually sufficient to normalise glycaemia. When initiating AID systems in individuals without prior CSII experience, education should focus on issues related to pump operation in hybrid mode, principles of switching to manual mode in emergency situations, and technical aspects. Training on other topics important in traditional insulin pump therapy, which are not applicable in AID systems, should be minimised. From the perspective of patient education for AID use, it should be emphasised that prandial bolus administration is based on estimation of carbohydrate content of meals. There is no need for precise estimation of protein, fat, or caloric content.
10. Telemedicine is an important tool for optimising diabetes control. For all individuals with type 1 diabetes, the therapeutic team, in agreement and cooperation with the patient, should aim to develop a system enabling effective remote medical consultations. Development of such a system should be based on patient education and encouragement to use appropriate technological solutions. Remote medical visits for patients with type 1 diabetes may constitute both an element of routine diabetology care and a solution used during epidemiological threats.
11. Medications that, when combined with insulin therapy, may improve glycaemic control and reduce body weight in type 1 diabetes include SGLT-2 inhibitors and GLP-1 receptor agonists; however, these drugs are not currently approved for adjunctive treatment of type 1 diabetes. If the use of medications from these groups is necessary in individuals with type 1 diabetes for indications other than direct antihyperglycaemic effects (cardiological or renal indications, obesity treatment), close and ongoing adjustment of insulin therapy is required, optimally based on CGM or with the use of automated insulin pumps. The importance of monitoring ketonuria/ketonaemia should be emphasised to prevent euglycaemic diabetic ketoacidosis when SGLT-2 inhibitors are used, particularly in individuals using AID systems. In some individuals with type 1 diabetes and features of insulin resistance, the addition of metformin may be associated with certain clinical benefits. General principles of antihyperglycaemic management in individuals with type 1 diabetes are presented in Figure 10.1.
12. At every stage of treatment, individuals with type 1 diabetes must have access to psychological support. The diabetologist managing the treatment should refer the patient for psychological consultation both at the patient's request and proactively when necessary, bearing in mind that psychological problems may affect up to half of individuals with type 1 diabetes.

II. Organization of care for individuals with type 1 diabetes

1. From the time of diagnosis and throughout the further course of the disease, a person with type 1 diabetes should remain under the care of a diabetologist. This approach ensures continuous cooperation with the educational team (in accordance with the principles described in Annex 7) and access to necessary consultations. Treatment using personal insulin pumps should preferably be provided in centres where at least one member of the therapeutic team holds a valid certificate from the Diabetes Poland Insulin Pump School or is undergoing such training.
2. Newly diagnosed cases of type 1 diabetes, as well as acute diabetes complications that are difficult to manage, require hospitalisation in a reference centre.

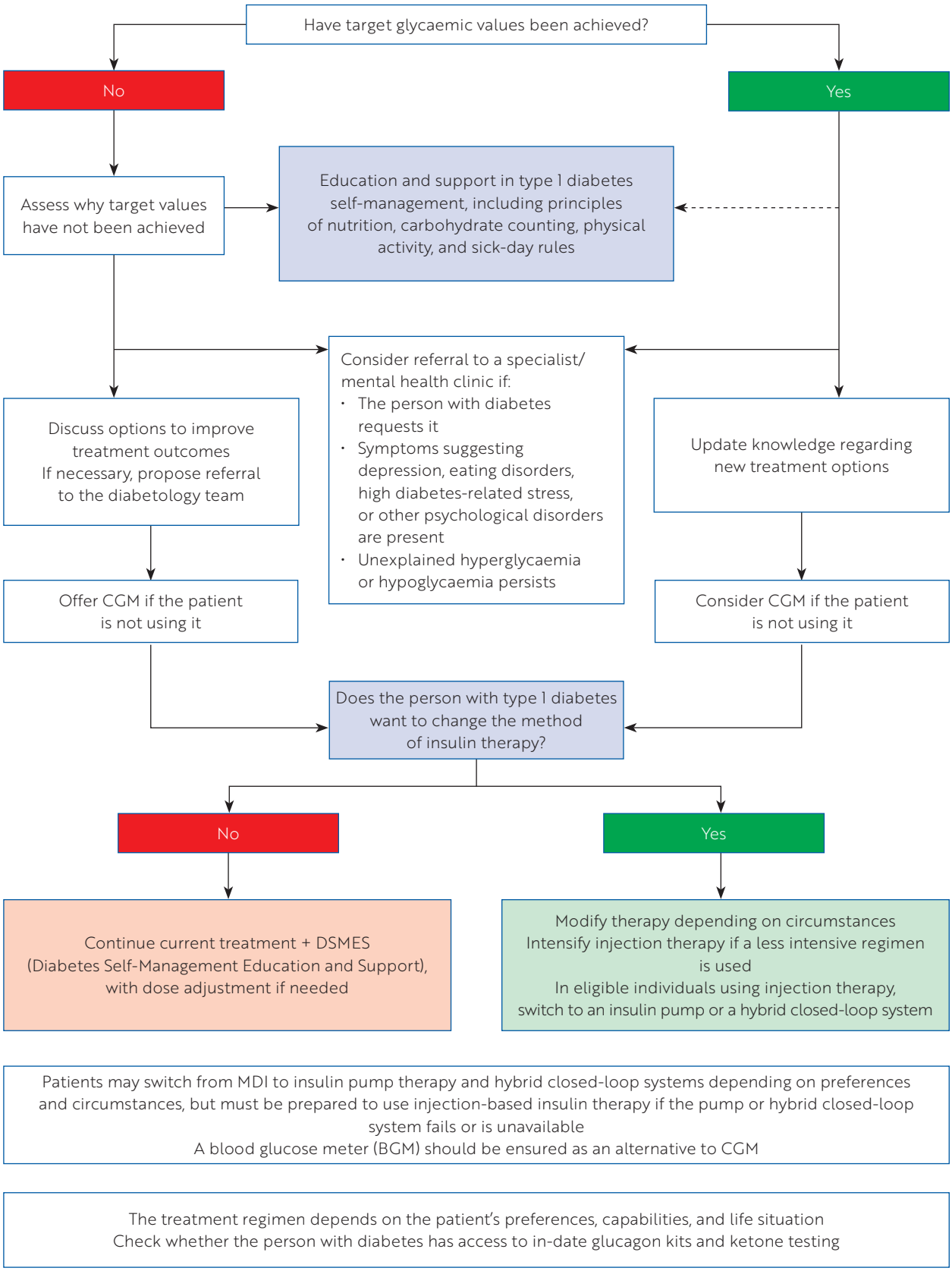


Figure 10.1. General principles of antihyperglycaemic management in individuals with type 1 diabetes

III. Treatment goals in type 1 diabetes

1. Metabolic control and maintenance of blood glucose levels as close to the normal range as possible. The primary therapeutic goal is to achieve an HbA_{1c} value ≤ 7%. Striving for lower HbA_{1c} values (≤ 6.5%) is justified provided that it is not associated with an increased risk of hypoglycaemia or deterioration of the quality of life of the person with diabetes.
2. Achieving treatment goals from the onset of the disease may prevent the occurrence of acute and chronic complications and enable a normal, active family, professional, and social life.
3. In individuals who systematically use CGM, the primary therapeutic goal is to achieve a high proportion (over 70%) of time spent within the therapeutic target range, defined as glycaemic values between 70 and 180 mg/dl. It should be emphasised that one of the treatment priorities should be avoidance of hypoglycaemia (the acceptable time spent below 70 mg/dl, and 54 mg/dl should not exceed 4% and 1% of time, respectively). Target glycaemic parameters for individuals using CGM are presented in Table 4.2.
4. In selected individuals, therapeutic goals may be defined on the basis of time in tight range (TITR) of 70–140 mg/dl. The decision to assess glycaemic control using TITR as a complement to TIR should be made cautiously, taking into account the patient's level of education, engagement in treatment, available technologies, and psychological profile. At present, the use of TITR should be limited to a selected group of patients using AID systems and individuals in the remission phase of type 1 diabetes.
5. It should be emphasised that one of the key goals of type 1 diabetes treatment is also to maintain the highest possible quality of life for the patient.

IV. Early detection of chronic complications of diabetes

1. This is possible through screening for diabetic nephropathy, retinopathy, and neuropathy. The principles of conducting these examinations in individuals with type 1 diabetes are discussed in Chapters 19, 20, and 21. In individuals with type 1 diabetes and long disease duration, particularly those diagnosed at a young age, large-vessel disease (diabetic macroangiopathy) may manifest earlier than in the general population, presenting as ischaemic heart disease, cerebrovascular disease, or peripheral arterial disease of the lower limbs. The principles of diagnosis and treatment of ischaemic heart disease are discussed in Chapter 17, while the management of stroke and acute coronary syndromes follows the recommendations presented in Chapters 18 and 17.1.
2. Recognition and management of acute complications. A properly educated person with type 1 diabetes must be familiar with the principles of management of acute, moderate, and mild hyperglycaemia and hypoglycaemia and should be able to manage such situations independently. More severe conditions require medical assistance in accordance with the principles presented in Chapters 15 and 16. Special situations in individuals with type 1 diabetes:
   a) A person with type 1 diabetes (with good metabolic control) treated with intensive insulin therapy may undergo "day surgery" procedures (minor surgical interventions).
   b) In hospital settings, a patient with type 1 diabetes who has previously used advanced technologies effectively, such as CGM systems or personal insulin pumps, should be allowed to continue self-management based on these systems, provided that appropriate supervision is ensured and the patient's general condition allows it.
   c) A well-educated patient with type 1 diabetes who achieved satisfactory treatment outcomes prior to hospitalisation should participate in therapeutic decision-making regarding diabetes management during hospitalisation; in selected cases, the patient may manage treatment independently, provided that glycaemic therapeutic targets are achieved. Perioperative management principles in individuals with type 1 diabetes are presented in Chapter 26.
   d) Type 1 diabetes is more frequently accompanied than in the general population by endocrinopathies, particularly autoimmune thyroid diseases (Hashimoto's disease, Graves' disease), adrenal insufficiency (Addison's disease), as well as coeliac disease, vitamin B12 deficiency anaemia, and connective tissue diseases; coexistence of these conditions may significantly worsen the course of type 1 diabetes.
   e) Obesity with features of insulin resistance may occur in individuals with type 1 diabetes, resulting in increased daily insulin requirements and deterioration of metabolic

control; diagnosis and management in such cases require specialist assessment and treatment.

f) Eating disorders such as bulimia or anorexia, as well as fear of hypoglycaemia, represent a growing problem among young people with type 1 diabetes; diagnosis and treatment of these conditions require specialist psychiatric care with close cooperation with a diabetologist.

g) A growing problem among young people, which in most cases remains undiagnosed and causes significant glycaemic variability, is the use of illicit drugs and psychoactive substances.

h) Some older patients with type 1 diabetes may require liberalisation of therapeutic goals; decisions should be guided primarily by biological rather than chronological age. In older patients with type 1 diabetes who are in good biological condition, continuation or initiation of treatment using advanced technologies should not be ruled out a priori. A well-educated person with type 1 diabetes, treated with intensive insulin therapy and achieving good metabolic control, is capable of undertaking the same level of physical activity and achieving similar professional goals as individuals of comparable age without diabetes.

REFERENCES

1. Anderson SM, Buckingham BA, Breton MD, et al. Hybrid closed-loop control is safe and effective for people with type 1 diabetes who are at moderate to high risk for hypoglycemia. Diabetes Technol Ther 2019; 21: 356–363.
2. Battelino T, Danne T, Bergenstal RM, et al. Clinical targets for continuous glucose monitoring data interpretation: recommendations from the international consensus on time in range. Diabetes Care 2019; 42: 1593–1603.
3. Beck RW, Raghinaru D, Calhoun P, Bergenstal RM. A comparison of continuous glucose monitoring-measured time-in-range 70-180 mg/dL versus time-in-tight-range 70-140 mg/dL. Diabetes Technol Ther 2024; 26: 151–155.
4. Bell KJ, Barclay AW, Petocz P, et al. Efficacy of carbohydrate counting in type 1 diabetes: a systematic review and meta-analysis. Lancet Diabetes Endocrinol 2014; 2: 133–140.
5. Bergenstal RM, Klonoff DC, Garg SK, et al. Threshold-based insulin-pump interruption for reduction of hypoglycemia. N Engl J Med 2013; 369: 224–232.
6. Bolinder J, Antuna R, Geelhoed-Duijvestijn P, et al. Novel glucose-sensing technology and hypoglycaemia in type 1 diabetes: a multicentre, non-masked, randomised controlled trial. Lancet 2016; 388: 2254–2263.
7. Breton MD, Patek SD, Lv D, et al. Continuous glucose monitoring and insulin informed advisory system with automated titration and dosing of insulin reduces glucose variability in type 1 diabetes mellitus. Diabetes Technol Ther 2018; 20: 531–540.
8. Castañeda J, Mathieu C, Aanstoot HJ, et al. Predictors of time in target glucose range in real-world users of the MiniMed 780G system. Diabetes Obes Metab 2022; 24: 2212–2221.
9. Cnop M, Klupa T, Tentolouris N, et al. Europe has to step up its efforts to produce innovative and safe diabetes technology. Diabetologia 2017; 60: 2532–2533.
10. Cyranka K, Matejko B, Klupa T, et al. Type 1 diabetes and COVID-19: the level of anxiety, stress and the general mental health in comparison to healthy control. Psychiatr Pol 2021; 55: 511–523.
11. Danne T, Cariou B, Banks P, et al. HbA1c and hypoglycemia reductions at 24 and 52 weeks with sotagliflozin in combination with insulin in adults with type 1 diabetes: the European inTandem2 study. Diabetes Care 2018; 41: 1981–1990.
12. Gawrecki A, Klupa T, Araszkiewicz A, et al. Utilization of do-it-yourself artificial pancreas systems in the management of patients with type 1 diabetes: a position statement of the Pump School Education Initiative by Diabetes Poland. Pol Arch Intern Med 2019; 129: 141–142.
13. Holt R, DeVries H, Hess-Fischl A, et al. The management of type 1 diabetes in adults. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes Care 2021; 44: 2589–2625.
14. Knox ECL, Quirk H, Glazebrook C, et al. Impact of technology-based interventions for children and young people with type 1 diabetes on key diabetes self-management behaviours and prerequisites: a systematic review. BMC Endocr Disord 2019; 19: 7. DOI: 10.1186/s12902-018-0331-6.
15. Lepore G, Rossini A, Bellante R, et al. Switching to the Minimed™ 780G system achieves clinical targets for CGM in adults with type 1 diabetes regardless of previous insulin strategy and baseline glucose control. Acta Diabetol 2022; 59: 1309–1315.
16. Matejko B, Juza A, Kieć-Wilk B, et al. One-year follow-up of advanced hybrid closed-loop system in adults with type 1 diabetes previously naive to diabetes technology: the effect of switching to a calibration-free sensor. Diabetes Technol Ther 2023; 25: 554–558.
17. Matejko B, Juza A, Kieć-Wilk B, et al. Transitioning of people with T1D from multiple daily injections and self-monitoring of blood glucose directly to minimed 780 g

advanced hybrid closed loop system: a two-center, randomized, controlled study. Diabetes Care 2022; 45: 2628–2635.
18. Mathieu C, Irace C, Wilmot EG, et al. Minimum expectations for market authorization of continuous glucose monitoring devices in Europe-'eCGM' compliance status. Diabetes Obes Metab 2025; 27: 1025–1031.
19. Miller KM, Beck RW, Bergenstal RM, et al. Evidence of a strong association between frequency of self-monitoring of blood glucose and hemoglobin A1c levels in T1D exchange clinic registry participants. Diabetes Care 2013; 36: 2009–2014.
20. Pinsker JE, Bartee A, Katz M, et al. Predictive low-glucose suspend necessitates less carbohydrate supplementation to rescue hypoglycemia: need to revisit current hypoglycemia treatment guidelines. Diabetes Technol Ther 2021; 23: 512–516.
21. Pratley RE, Kanapka LG, Rickels MR, et al. Effect of continuous glucose monitoring on hypoglycemia in older adults with type 1 diabetes: a randomized clinical trial. JAMA 2020; 323: 2397–2406.
22. Rawshani A, Sattar N, Franzén S, et al. Excess mortality and cardiovascular disease in young adults with type 1 diabetes in relation to age at onset: a nationwide, register-based cohort study. Lancet 2018; 392: 477–486.
23. Riddell MC, Gallen IW, Smart CE, et al. Exercise management in type 1 diabetes: a consensus statement. Lancet Diabetes Endocrinol 2017; 5: 377–390.
24. Tauschmann M, Hovorka R. Technology in the management of type 1 diabetes mellitus – current status and future prospects. Nat Rev Endocrinol 2018; 14: 464–475.
25. The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 329: 977–986.
26. The Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Research Group. Retinopathy and nephropathy in patients with type 1 diabetes four years after a trial of intensive therapy. N Engl J Med 2000; 342: 381–388.
27. Triolo TM, Armstrong TK, McFann K, et al. Additional autoimmune disease found in 33% of patients at type 1 diabetes onset. Diabetes Care 2011; 34: 1211–1121.

11. Oral Antihyperglycaemic Agents, Injectable GLP-1 Receptor Agonists, and GIP/GLP-1 Receptor Agonist in the Treatment of Type 2 Diabetes

CHAPTER HIGHLIGHTS:
· Pharmacotherapy of type 2 diabetes should be accompanied by comprehensive, structured education, appropriate nutritional management, psychological support, and planned physical activity. The choice of medications should take into account all individual patient characteristics, including priorities, lifestyle and health behaviours, comorbidities, motivation, cognitive impairment, and social factors. [**E**]
· As first-line therapy when initiating pharmacological treatment of type 2 diabetes, sodium–glucose cotransporter 2 inhibitors (SGLT2 inhibitors; gliflozins), GLP-1 receptor agonists, dual GIP/GLP-1 receptor agonist, or metformin, should be considered as the preferred options. In patients with documented atherosclerotic cardiovascular disease, heart failure, chronic kidney disease, or multiple cardiovascular risk factors, the choice should primarily take into account the cardiovascular and nephroprotective effects of these agents. [**A**]
· Combination therapy at the time of diagnosis of type 2 diabetes should be considered in patients belonging to the above-mentioned high-risk groups and in those with marked hyperglycaemia (HbA_{1c} ≥ 8.5%). [**A**]
· If treatment is initiated with monotherapy that becomes insufficient to achieve or maintain the target HbA_{1c} level, a second antihyperglycaemic agent should be added. This decision should not be delayed beyond 3–4 months. [**A**]
· In patients with atherosclerotic cardiovascular disease, heart failure, chronic kidney disease, or multiple cardiovascular risk factors, treatment intensification should prioritise agents with proven beneficial effects on disease progression and on total and cardiovascular mortality. [**A**]
· In patients with heart failure, SGLT2 inhibitors should be preferred; in cases of contraindications to their use, GLP-1 receptor agonists or GIP/GLP-1 receptor agonists should be used. [**A**]
· In patients with established atherosclerotic cardiovascular disease, multiple cardiovascular risk factors, or chronic kidney disease, the use of both drug classes (SGLT2 inhibitors and GLP-1 receptor agonists or GIP/GLP-1 receptor agonist) should be considered. Early combination therapy with metformin and/or SGLT2 inhibitors and/or GLP-1 receptor agonists and/or GIP/GLP-1 receptor agonist should be considered in all such patients, regardless of achievement of glycaemic targets. [**A**]
· In all patients with type 2 diabetes, efforts should be made to achieve individually defined therapeutic goals, including glycaemic targets, body weight reduction, and other components of multifactorial therapy. [**B**]
· Due to the progressive nature of type 2 diabetes, insulin therapy in individually tailored regimens is indicated in many individuals with this condition. [**B**]
· All therapeutic decisions regarding the treatment of type 2 diabetes should be made in agreement with the patient and after obtaining their acceptance. [**E**]

Individually tailored pharmacological reduction of hyperglycaemia, which in the multifactorial management of type 2 diabetes (alongside treatment of obesity, arterial hypertension, and dyslipidaemia) is accompanied by behavioural interventions, plays a key role in the prevention and slowing of progression of chronic diabetes complications (both macrovascular and microvascular).

I. Hyperglycaemia reduction

Hyperglycaemia is reduced by correcting the pathogenic mechanisms of type 2 diabetes, namely insulin resistance, impaired insulin secretion, and impaired incretin effect. Another therapeutic mechanism of antihyperglycaemic agents is glycosuria induction.

Treatment of type 2 diabetes must be progressive and adjusted stepwise to the progressive nature of the disease, while also taking comorbid conditions into account. If the therapy used at a given stage becomes ineffective (i.e. the individual target HbA_{1c} value is not achieved), escalation to the next stage should be undertaken after 3–4 months. Avoidance of therapeutic inertia is one of the fundamental principles of effective treatment of type 2 diabetes.

II. Stages of type 2 diabetes treatment

1. Initiation of therapy:
   - Lifestyle modification consisting of reducing the caloric content of meals and increasing physical activity to a minimum of 30–45 mi-

nutes per day in order to achieve weight reduction.

- Pharmacological treatment may be initiated as monotherapy or combination therapy; as first-line agents when initiating pharmacological treatment of type 2 diabetes, SGLT2 inhibitors, GLP-1 receptor agonists, GIP/GLP-1 receptor agonist, and metformin should be considered first.
- GLP-1 receptor agonists or GIP/GLP-1 receptor agonist and SGLT2 inhibitors with proven clinical benefits should be preferred in individuals with cardiovascular disease, multiple cardiovascular risk factors, or chronic kidney disease; in patients with heart failure, SGLT2 inhibitors should be preferred, and if contraindicated, GLP-1 receptor agonists or GIP/GLP-1 receptor agonist should be used; in patients with established atherosclerotic cardiovascular disease, multiple cardiovascular risk factors, or chronic kidney disease, the use of both drug classes should be considered; PPAR-γ agonists and saxagliptin should not be used in patients with heart failure.
- To date, no data have been published confirming a nephroprotective effect of GIP/GLP-1 receptor agonist in trials with primary renal endpoints.
- The therapeutic effectiveness of initiated treatment can be assessed only after several weeks of use.
- The decision to initiate combination therapy in newly diagnosed diabetes should be particularly considered in patients at very high cardiovascular risk and in those with marked hyperglycaemia (HbA_{1c} > 8.5%). In patients from the above risk groups, a combination regimen should include an SGLT2 inhibitor and/or a GLP-1 receptor agonist or GIP/GLP-1 receptor agonist.

2. Intensification of therapy with oral agents or GLP-1 receptor agonists, or a dual GIP/GLP-1 receptor agonist:

- Lifestyle modification and addition to monotherapy or dual therapy of a drug from a class not previously used: metformin, or an SGLT2 inhibitor, or an incretin-based drug (DPP-4 inhibitor, GLP-1 receptor agonist, or dual GIP/GLP-1 receptor agonist), or a sulfonylurea, or a PPAR-γ agonist. The choice of medication at each stage should take into account comorbidities – primarily diagnosed cardiovascular disease and chronic kidney disease – as well as the presence of obesity, risk of hypoglycaemia, and the patient's financial capabilities. In patients with atherosclerotic cardiovascular

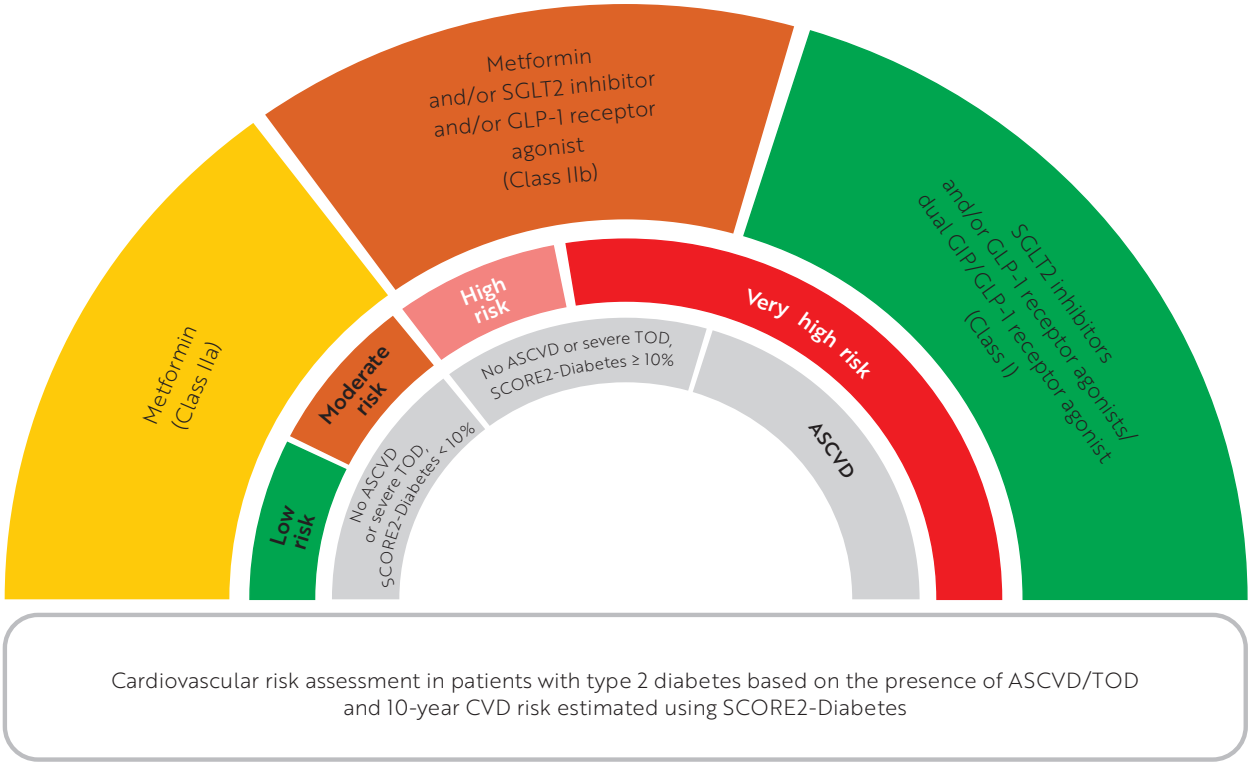


Figure 11.1. Cardiovascular risk categories and recommended antihyperglycaemic therapy in patients with type 2 diabetes according to the ESC [13]; modified based on SUPRASS-CVOT

disease, heart failure, chronic kidney disease, or multiple cardiovascular risk factors, agents with proven beneficial effects on disease progression and on all-cause and cardiovascular mortality should be used as first-line therapy. Such effects have been demonstrated for certain SGLT2 inhibitors and selected GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonist. Early combination therapy with SGLT2 inhibitors and/or selected GLP-1 receptor agonists or a dual GIP/GLP-1 receptor agonist and/or metformin should be considered in the above-mentioned cases for every patient, regardless of achievement of glycaemic targets. In the presence of obesity, preference should also be given to a GLP-1 receptor agonist or a dual GIP/GLP-1 receptor agonist. In patients at high risk of hypoglycaemia, the same drug classes should be considered, as well as DPP-4 inhibitors or PPAR-γ agonists.

- Lifestyle modification and triple or quadruple therapy using agents with different mechanisms of action from the following groups: metformin, SGLT2 inhibitors, GLP-1 receptor agonists, dual GIP/GLP-1 receptor agonist, sulfonylureas, DPP-4 inhibitors, and PPAR-γ agonists. Drug selection at this stage is based on the same principles as at earlier stages, as well as on general rules for combining antihyperglycaemic agents.

3. Intensification of treatment with insulin:

- Lifestyle modification and simple insulin therapy, primarily using basal insulin (NPH insulin, long-acting analogue, ultra-long-acting analogue; various regimens—see Chapter 12), with continuation of metformin and other oral agents or injectable GLP-1 receptor agonists or a dual GIP/GLP-1 receptor agonist, particularly in the presence of obesity. In patients receiving their first injectable therapy (e.g. basal insulin or a GLP-1 receptor agonist), intensification may involve the use of fixed-ratio combination products containing basal insulin and a GLP-1 receptor agonist. These preparations may also be used as first injectable therapy.
- Lifestyle modification and complex insulin therapy with recommended continuation of metformin and other oral agents (metformin, incretin-based drugs, PPAR-γ agonists, SGLT2 inhibitors) or injectable GLP-1 receptor agonists or a dual GIP/GLP-1 receptor agonist, especially in the presence of persistent excess body weight (see Chapter 12).

At every stage of treatment, efforts should be made to achieve individually defined glycaemic targets, body weight goals, and other objectives of multifactorial therapy.

4. Simplification of the antihyperglycaemic treatment model (simplification):

- Many patients with type 2 diabetes require reduction of treatment complexity and burden, particularly insulin therapy, as well as consideration of liberalisation of glycaemic targets; this applies, for example, to patients at high risk of hypoglycaemia, with cognitive impairment, poor adherence to medical recommendations, limited life expectancy, or when complex treatment regimens negatively affect quality of life.
- A key tool in this approach is reducing the number of insulin injections and the insulin dose by using individually tailored combinations with non-insulin antihyperglycaemic agents.

Failure to simplify antihyperglycaemic therapy in patients with indications for such an approach constitutes a form of therapeutic inertia**.**

III. List of medications

Medications used in the treatment of type 2 diabetes are presented in Table 11.1.

When selecting therapy and combining medications, their effects on non-glycaemic parameters should be taken into account (risk of death, cardiovascular disease, chronic kidney disease, body weight, risk of hypoglycaemia, lipid metabolism, etc.), and therapy should be individualised (see subsection 4.1.3). Results of randomised clinical trials indicate benefits in terms of reduction in all-cause and cardiovascular mortality, as well as cardiovascular and renal endpoints, with the use of certain agents from the groups of GLP-1 receptor agonists, dual GIP/GLP-1 receptor agonists, and SGLT2 inhibitors.

IV. Practical algorithm for pharmacotherapy of type 2 diabetes

The algorithm is presented in Figures 11.2 and 11.3.

Table 11.1. Antihyperglycaemic agents used in the treatment of type 2 diabetes

	Metformin	SGLT2 inhibitors	GLP-1 receptor agonist	Dual GIP/GLP-1 receptor agonist	DPP-4 inhibitors	Sulfonylureas	PPAR-γ agonists
Main effect/ mechanism	Reduction of hepatic glucose production, increase in peripheral insulin sensitivity	Induction of glycosuria	Glucose-dependent increase in insulin secretion, appetite suppression	Glucose-dependent increase in insulin secretion, appetite suppression	Increase in insulin levels depending on the degree of hyperglycaemia	Increased insulin secretion	Increase in peripheral insulin sensitivity
Glucose-lowering efficacy	High	High	High	High	Moderate	High	High
Cardiovascular benefit		Yes[#,A]	Yes[#]	Yes			
Renal benefit		Yes	Yes[#]				↓
Plasma insulin	↓	↓	↑↑	↑↑	↑	↑↑	↓
LDL cholesterol	↓	↔ or ↑	↓	↓	↓ or ↔	↔	↔
HDL cholesterol	↑	↑	↑	↑	↑	↔	↑
Triglycerides	↓	↔	↓	↓	↔	↔	↓
Body weight	↓ or ↔	↓	↓↓	↓↓	↔	↑	↑
Risk of hypoglycaemia	↔	↔	↔	↔	↔	↑	↔
Adverse effects	Gastrointestinal disorders	Genital infections, dehydration (especially in older adults)	Gastrointestinal disorders (nausea, vomiting)	Gastrointestinal disorders (nausea, vomiting)	No significant adverse effects	Hypoglycemia, weight gain	Fluid retention (oedema), weight gain, increased risk of long bone fractures
Contraindications	According to the current SmPC (Summary of Product Characteristics).						

**Insulin: see Chapter 12.*

***See Table 19.4.*

[#]Demonstrated for selected agents within the class, in accordance with currently published results of randomised clinical trials.

[A]In the case of empagliflozin and canagliflozin, no differences were observed in CVOT studies between doses of 10 and 25 mg and 100 and 300 mg, respectively.

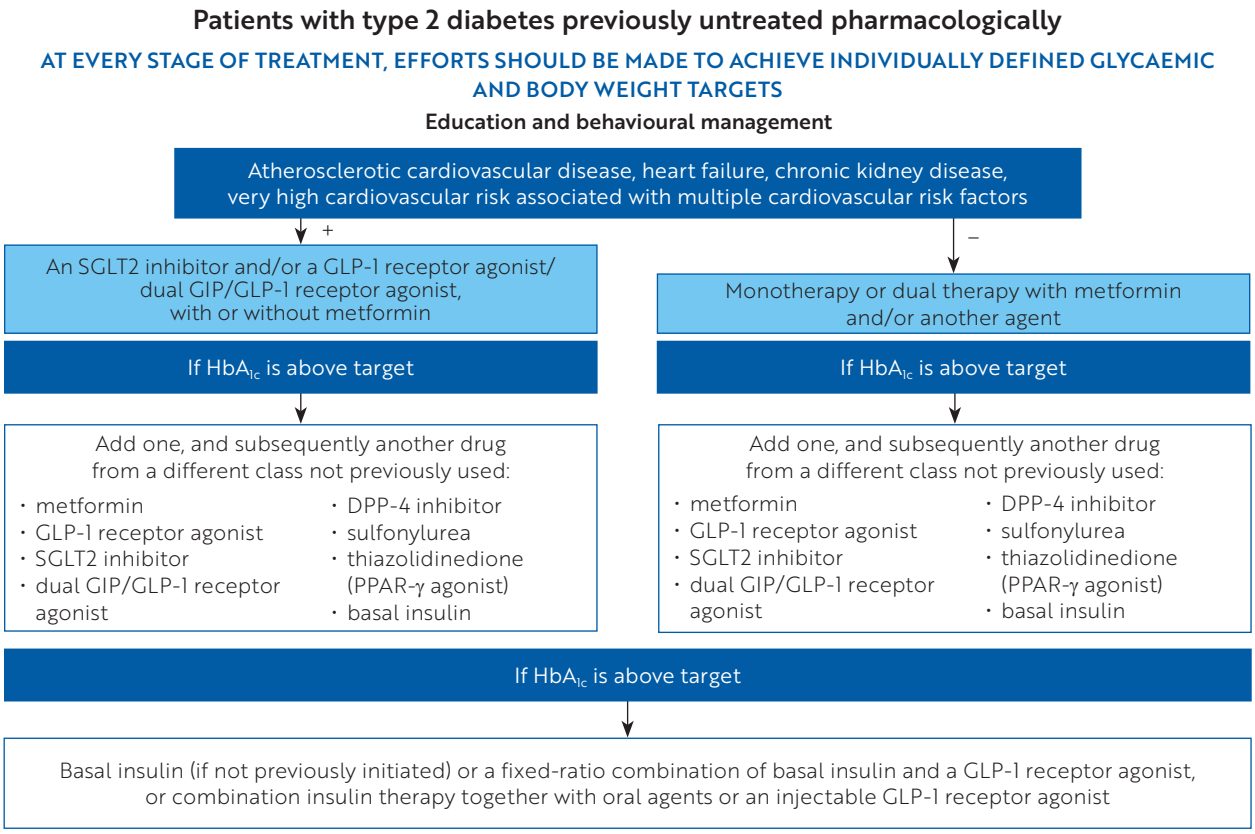


Figure 11.2. Therapeutic management algorithm for individuals with type 2 diabetes previously untreated pharmacologically

CV – cardiovascular

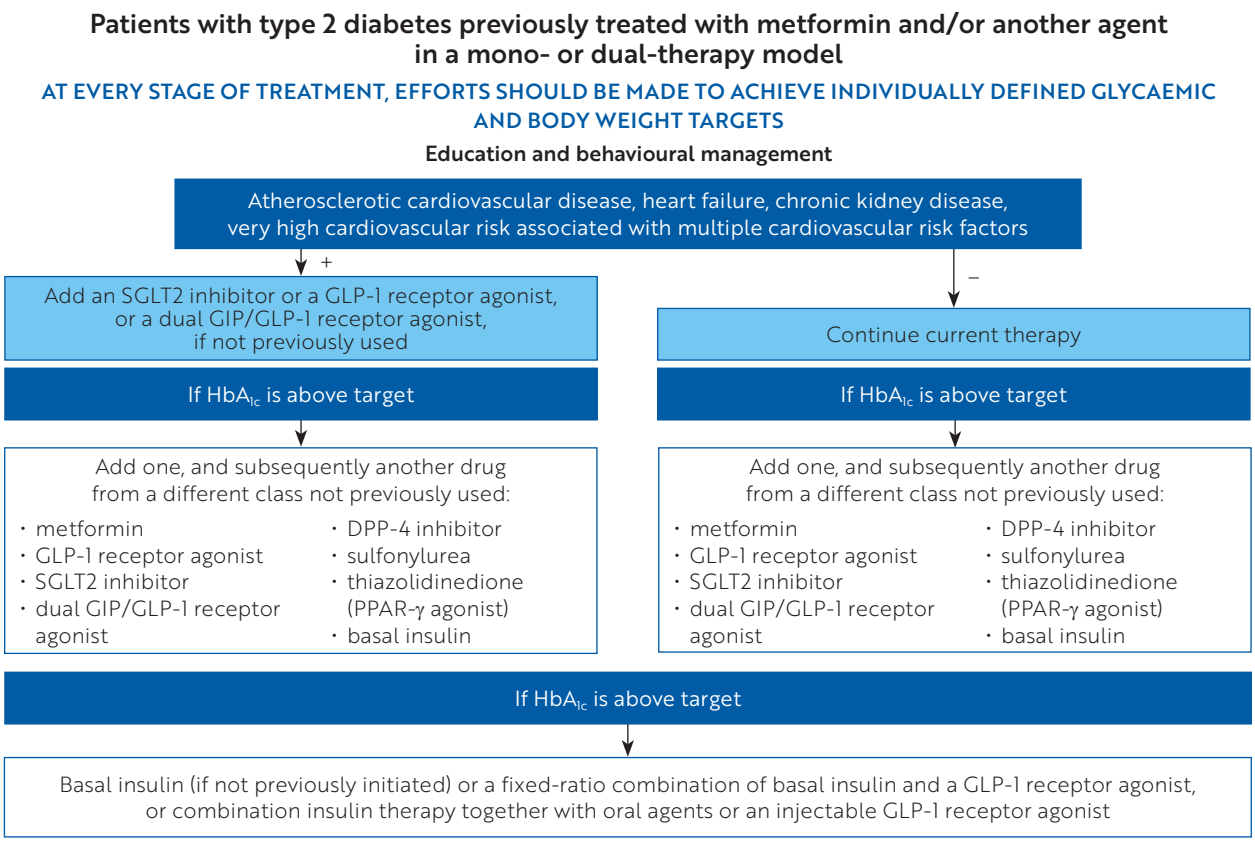


Figure 11.3. Therapeutic management algorithm for individuals with type 2 diabetes previously treated with metformin and/or another agent in a mono- or dual-therapy model

CV – cardiovascular

**An SGLT2 inhibitor or a GLP-1 receptor agonist, or a dual GIP/GLP-1 receptor agonist should be added regardless of the complexity of the treatment regimen (e.g. triple or quadruple therapy, complex insulin regimens) in every patient at very high cardiovascular risk who has not previously received agents from these drug classes.*

REFERENCES

1. Davies MJ, Aroda VR, Collins BS, et al. Management of hyperglycaemia in type 2 diabetes, 2022. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetologia 2022; 65: 1925–1966.
2. Frías JP, Davies MJ, Rosenstock J, et al.; SURPASS-2 Investigators. Tirzepatide versus semaglutide once weekly in patients with type 2 diabetes. N Engl J Med 2021; 385: 503–515.
3. Gaede P, Lund-Andersen H, Parving HH, et al. Effect of a multifactorial intervention on mortality in type 2 diabetes. N Engl J Med 2008; 358: 580–591.
4. Gerstein HC, Bosch J, Dagenais GR, et al. Basal insulin and cardiovascular and other outcomes in dysglycemia. N Engl J Med 2012; 367: 319–328.
5. Gerstein HC, Colhoun HM, Dagenais GR, et al. Dulaglutide and cardiovascular outcomes in type 2 diabetes (REWIND): a double-blind, randomised placebo-controlled trial. Lancet 2019; 394: 121–130.
6. Holman RR, Bethel MA, Mentz RJ, et al. Effects of once-weekly exenatide on cardiovascular outcomes in type 2 diabetes. N Engl J Med 2017; 377: 1228–1239.
7. Holman RR, Farmer AJ, Davies MJ, et al. Three-year efficacy of complex insulin regimens in type 2 diabetes. N Engl J Med 2009; 361: 1736–1747.
8. Holman RR, Thorne KI, Farmer AJ, et al. Addition of biphasic, prandial, or basal insulin to oral therapy in type 2 diabetes. N Engl J Med 2007; 357: 1716–1730.
9. Husain M, Birkenfeld AL, Donsmark M, et al. PIONEER 6 investigators. Oral semaglutide and cardiovascular outcomes in patients with type 2 diabetes. N Engl J Med 2019; 381: 841–851.
10. Kahn SE, Haffner SM, Heise MA, et al. Glycemic durability of rosiglitazone, metfor min, or glyburide monotherapy. N Engl J Med 2006; 355: 2427–2443.
11. Marso SP, Bain SC, Consoli A, et al. Semaglutide and cardiovascular outcomes in patients with type 2 diabetes. N Engl J Med 2016; 375: 1834–1844.
12. Marso SP, Daniels GH, Brown-Frandsen K, et. al. Liraglutide and cardiovascular outcomes in type 2 diabetes. N Engl J Med 2016; 375: 311–322.
13. Marx N, Federici M, Schütt K, et al. 2023 ESC Guidelines for the management of cardiovascular disease in patients with diabetes: developed by the task force on the management of cardiovascular disease in patients with diabetes of the European Society of Cardiology (ESC). Eur Heart J 2023; 44: 4043–4140.
14. Matthews DR, Paldanius PM, Proot P, et al. Glycaemic durability of an early combination therapy with vildagliptin and metformin versus sequential metformin monotherapy in newly diagnosed type 2 diabetes (VERIFY): a 5-year, multicentre, randomised, double-blind trial. Lancet 2019; 394: 1519–1529.
15. McGuire DK, Marx N, Mulvagh SL, et al.; SOUL Study Group. Oral semaglutide and cardiovascular outcomes in high-risk type 2 diabetes. N Engl J Med 2025; 392: 2001–2012.
16. McMurray JJV, Solomon SD, Inzucchi SE, et al. Dapagliflozin in patients with heart failure and reduced ejection fraction. N Engl J Med 2019; 381: 1995–2008.
17. Neal B, Perkovic V, Mahaffey KW, et al. Canagliflozin and cardiovascular and renal events in type 2 diabetes. N Engl J Med 2017; 377: 644–657.
18. Nicholls SJ, Pavo I, Bhatt DL, et al.; SURPASS-CVOT Investigators. Cardiovascular outcomes with tirzepatide versus dulaglutide in type 2 diabetes. N Engl J Med 2025; 393: 2409-2420.
19. Packer M, Anker SD, Butler J, et al. Cardiovascular and renal outcomes with empagliflozin in heart failure. N Engl J Med 2020; 383: 1413–1424.
20. Packer M, Zile MR, Kramer CM, et al.; SUMMIT Trial Study Group. Tirzepatide for heart failure with preserved ejection fraction and obesity. N Engl J Med 2025; 392: 427–437.
21. Perkovic V, Jardine MJ, Neal B, et al. Canagliflozin and renal outcomes in type 2 diabetes and nephropathy. N Engl J Med 2019; 380: 2295–2306.
22. Perkovic V, Tuttle KR, Rossing P, et al. ; FLOW Trial Committees and Investigators. Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes. N Engl J Med 2024; 391: 109–121.
23. Rosenstock J, Emral R, Sauque-Reyna L, et al. Advancing therapy in suboptimally controlled basal insulin-treated type 2 diabetes: clinical outcomes with iGlarLixi versus premix BIAsp 30 in the SoliMix randomized controlled trial. Diabetes Care 2021; 44: 2361–2370.
24. UK Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). Lancet 1998; 352: 854–865.
25. Wiviott SD, Raz I, Bonaca MP, et al. Dapagliflozin and cardiovascular outcomes in type 2 diabetes. N Engl J Med 2019; 380: 347–357.
26. Zinman B, Wanner C, Lachin JM, et al. Empagliflozin, cardiovascular outcomes, and mortality in type 2 diabetes. N Engl J Med 2015; 373: 2117–2128.

12. Insulin Therapy in Type 2 Diabetes and Other Specific Types of Diabetes

CHAPTER HIGHLIGHTS
· In individuals with type 2 diabetes, insulin analogues are preferred due to a lower risk of hypoglycaemia. [**A**]
· Type 2 diabetes is a progressive condition. The progression of underlying pathophysiological disturbances, particularly β-cell dysfunction, necessitates stepwise intensification of treatment, including the initiation of insulin therapy. [**B**]
· The use of CGM systems improves the effectiveness and safety of insulin therapy. [**B**]

I. Indications for initiation of insulin therapy in type 2 diabetes

- Newly diagnosed diabetes (with the possibility of returning to the standard treatment algorithm and discontinuing insulin): blood glucose ≥ 300 mg/dl (16.7 mmol/l) accompanied by clinical symptoms of hyperglycaemia.
- Failure of non-insulin therapy (HbA_{1c} exceeding individual target values despite treatment intensification) (Figure 12.1).

II. Indications for modification of the current antihyperglycaemic treatment strategy

Switching from oral antihyperglycaemic therapy to insulin-based combination treatment in the presence of inadequate glycaemic control:

- confirmation of persistent hyperglycaemia,
- poor tolerance of oral medications,
- ineffective attempts to correct potentially reversible causes of hyperglycaemia, such as:
  - » dietary errors,
  - » insufficient physical activity,
  - » irregular intake of oral antihyperglycaemic agents (non-adherence),
  - » infections,
  - » inadequate dosing of oral medications,
- prior use of the antihyperglycaemic potential of GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists, which should constitute the first injectable therapy in type 2 diabetes (after taking into account the patient's financial possibilities and medication tolerance).

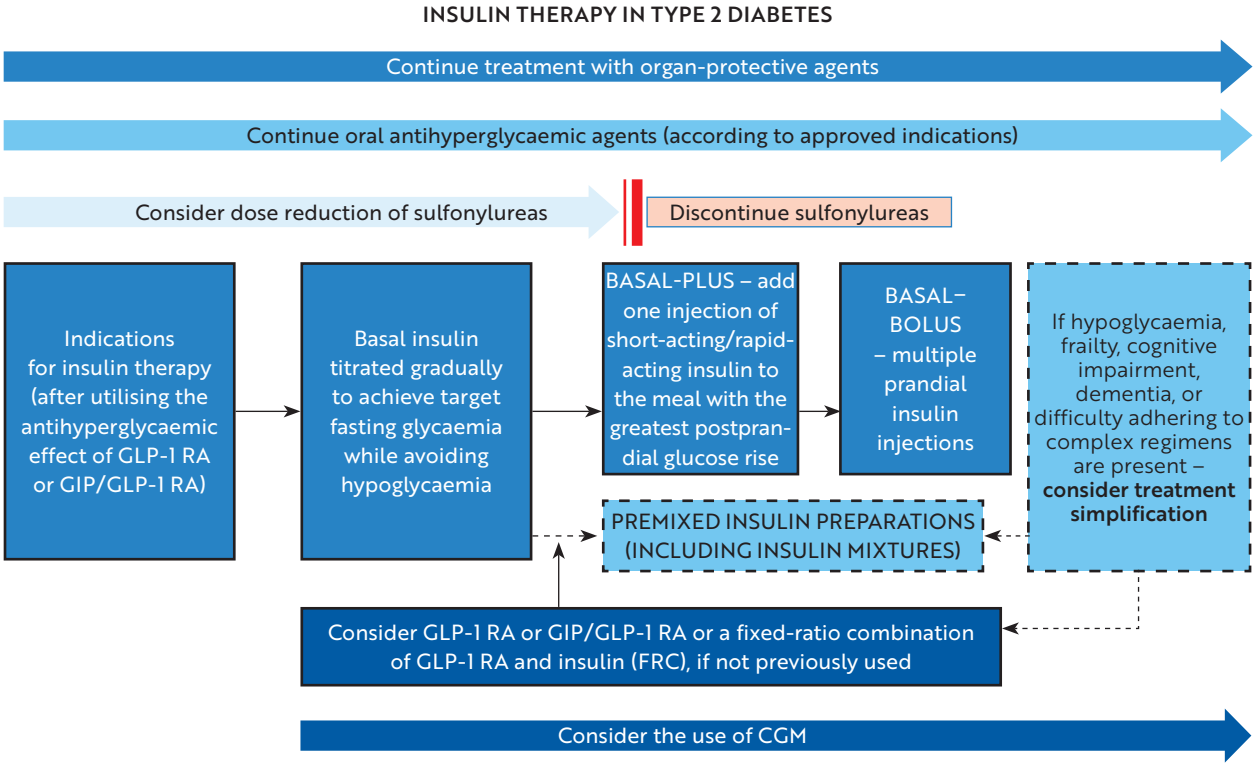


Figure 12.1. Practical algorithm for insulin therapy in type 2 diabetes. Models for initiation and intensification of insulin therapy

III. Indications for initiation of insulin therapy irrespective of glycaemic values

- cystic fibrosis–related diabetes,
- carriers of insulin gene mutations and individuals with compound mutations in the glucokinase gene,
- a justified request by the patient.

IV. Indications for temporary insulin therapy

- decompensation of diabetes caused by transient factors (infection, trauma, glucocorticoid therapy, etc.),
- surgical procedures (see Chapter 26),
- stroke (see Chapter 18),
- percutaneous transluminal coronary angioplasty (PTCA),
- acute coronary syndrome,
- other acute conditions requiring hospitalisation in an intensive care unit.

V. Insulin therapy algorithm in type 2 diabetes

1. Long-acting insulin analogue (administered once daily or once weekly) or NPH (isophane) insulin given as a single injection:
   - in the presence of fasting (morning) hyperglycaemia — administered in the evening; the use of long-acting insulin analogues reduces the risk of nocturnal and severe hypoglycaemia,
   - in the presence of normal fasting glycaemia and daytime hyperglycaemia — administered in the morning (multiple injections of short-acting insulin or rapid-acting insulin analogues may also be considered in cases of postprandial hyperglycaemia).
2. The recommended starting dose of basal insulin is 0.1–0.2 units/kg body weight, or 10 units, or 70 units in the case of once-weekly insulin.
3. In selected cases, when initiation of insulin therapy has been excessively delayed, resulting in marked hyperglycaemia and HbA_{1c} values substantially exceeding the therapeutic target (usually by 1.5% or more), immediate introduction of **more intensive insulin regimens may be considered (see point 6).** Such approaches should be particularly considered in relatively young patients with a long-expected life expectancy. The final choice of insulin preparation should be individualised, taking into account patient preferences, daily activity patterns, number of meals, and treatment costs.
4. Oral antihyperglycaemic agents and injectable incretin-based therapies may be used in accordance with their approved indications in individuals treated with insulin:
   - continuation of agents with proven cardiovascular risk–reducing effects is recommended,
   - in the presence of overweight or obesity, combination therapy with an SGLT-2 inhibitor or an incretin-based agent (**GLP-1 receptor agonist, dual GIP/GLP-1 receptor agonist**, or DPP-4 inhibitor) together with metformin should be preferred,
   - in all patients, continuation of metformin therapy should be pursued whenever it is tolerated and not contraindicated.
5. Assessment of glycaemic control should be performed after 4–5 days, with gradual dose increases of 2–4 units, based on self-monitoring results, until optimal glycaemic control is achieved.
6. If the required basal insulin dose exceeds 0.3–0.5 units/kg/day without achieving glycaemic targets, **or in situations described in point 3**, treatment intensification may be considered through:
   a) gradual addition of short-acting insulin or rapid-acting insulin analogue injections to basal insulin, initially before the meal associated with the greatest postprandial glucose rise, and subsequently before additional meals ("basal-plus", intensive insulin therapy). The recommended initial prandial insulin dose is 4 units or 10% of the total daily basal insulin dose;
   b) addition of an injectable **GLP-1 receptor agonist or a dual GIP/GLP-1 receptor agonist, if not previously used, including a fixed-ratio combination (FRC) preparation consisting of basal insulin and a GLP-1 receptor agonist** (this preparation may be used as the first injectable therapy).
   c) use of premixed insulin formulations, including analogue mixtures.

   At this stage of therapy, discontinuation of insulin secretagogues should be considered.
7. When high insulin doses are required, exceeding 100 units per day (suggestive of insulin resistance), the underlying causes should be identified and the potential for adverse effects considered. An attempt to reduce insulin resistance is recommended through the use of continuous subcutaneous or intravenous insulin infusion for 72–96 hours.

VI. Intensive insulin therapy

Intensive insulin therapy is implemented according to similar principles across all types of diabetes, using either multiple daily insulin injections or a personal insulin pump for continuous subcutaneous insulin infusion.

1. Principles of intensive insulin therapy:
- use of CGM systems or multiple daily blood glucose measurements,
- patient-led decision-making regarding insulin dose adjustments and possible supplemental doses, depending on measured glycaemic values, energy requirements, and physical activity,
- precise definition of individualised target glycaemic control parameters,
- appropriate therapeutic and nutritional education, as well as patient motivation,
- availability of rapid contact between the patient and the treating therapeutic team,
- in type 2 diabetes, continuous subcutaneous insulin infusion using a personal insulin pump is not a routine treatment modality.

2. Multiple daily injection algorithms:
- short-acting insulin or rapid-acting insulin analogue administered before meals, and
- a long-acting insulin analogue (administered daily or once weekly) or NPH (isophane) insulin to provide a stable basal insulin level overnight and/or in the early morning hours.

In selected cases of type 2 diabetes, when fasting glycaemia is within the normal range, the use of prandial insulin alone may be sufficient.

3. Insulin pump therapy

Treatment with personal insulin pumps should be conducted in centres with appropriate experience in this form of therapy and responsible for patient qualification for such treatment.

Any change in the insulin therapy model (optimisation, intensification, or simplification) should be accompanied by appropriate therapeutic education or re-education.

VII. Simplification of therapeutic regimens

In individuals who have achieved satisfactory glycaemic control but in whom implementation of the insulin therapy algorithm is challenging (e.g. older adults, patients with frailty syndrome, cognitive impairment, or those requiring care from third parties), or in whom complex regimens significantly increase the risk of hypoglycaemia, efforts should be made to simplify the insulin therapy regimen (see Figure 12.1).

Failure to implement such simplification is currently considered a manifestation of therapeutic inertia.

REFERENCES

1. Bergenstal RM, Klonoff DC, Garg SK, et al.; ASPIRE In-Home Study Group. Threshold-based insulin-pump interruption for reduction of hypoglycemia. N Engl J Med 2013; 369: 224–232.
2. Diabetes Control and Complications Trial (DCCT)/Epidemiology of Diabetes Interventions and Complications (EDIC) Study Research Group. Mortality in type 1 diabetes in the DCCT/EDIC versus the general population. Diabetes Care 2016; 39: 1378–1383.
3. Philis-Tsimikas A, Asong M, Franek E, et al. Switching to once-weekly insulin icodec versus once-daily insulin degludec in individuals with basal insulin-treated type 2 diabetes (ONWARDS 2): a phase 3a, randomised, open label, multicentre, treat-to-target trial. Lancet Diabetes Endocrinol 2023; 11: 414–425.
4. Rosenstock J, Frías J, Rodbard H, et al. Tirzepatide vs Insulin Lispro Added to Basal Insulin in Type 2 Diabetes: The SURPASS-6 Randomized Clinical Trial. JAMA 2023; 3: e2320294. DOI: 10.1001/jama.2023.20294.
5. The Diabetes Control and Complications Trial Research Group. The effect of intensive reatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 329: 977–986.
6. Yeh HC, Brown TT, Maruthur N, et al. Comparative effectiveness and safety of methods of insulin delivery and glucose monitoring for diabetes mellitus: a systematic review and meta-analysis. Ann Intern Med 2012; 157: 336–347.

13. Arterial Hypertension: Standards of Care 2026

Developed in collaboration with dr hab. n. med. Jacek Wolf.

CHAPTER HIGHLIGHTS
• The general goal of blood pressure control in individuals with diabetes is to achieve values of 120–129/70–79 mm Hg. [**A**]
• Non-pharmacological and pharmacological treatment of arterial hypertension should be initiated simultaneously when blood pressure is ≥ 130/80 mm Hg. [**A**]
• Pharmacotherapy should be initiated with the use of a combination of two drugs, optimally as a single-pill combination (SPC): an angiotensin-converting enzyme inhibitor (ACEI) or an angiotensin II receptor blocker (ARB) combined with a dihydropyridine calcium channel blocker or a thiazide or thiazide-like diuretic. [**A**]
• Pharmacotherapy of arterial hypertension should be continued uninterrupted, as only then is cardiovascular risk reduction achieved. [**A**]
• In the treatment of arterial hypertension in individuals with diabetes, efforts should be made not only to achieve target blood pressure values, but also to maintain or restore normal circadian blood pressure variability assessed using 24-hour monitoring, particularly in women with diabetes during pregnancy. [**B**]

In Poland, 11–12 million adult Poles suffer from arterial hypertension (AH), of whom 20–25% remain unaware of the disease, and only approximately 40% of patients with AH are treated in a satisfactory manner (according to previous standards). In the treatment of AH, reduction of blood pressure is the most important factor determining a reduction in the risk of target-organ complications, regardless of the therapy used. Most patients with AH and diabetes should achieve blood pressure control within the range of 120–129/70–79 mm Hg.

Blood pressure measurement technique

- Office and home blood pressure measurements should be performed after a short period of rest, in a seated position with the back supported and both feet flat on the floor, eliminating all possible distracting stimuli.
- The blood pressure value to be interpreted in the office setting is the mean of two consecutive measurements performed at rest in the seated position, or the mean of the second and third measurements if the difference between the first and second exceeds 10 mm Hg for systolic blood pressure.
- Out-of-office measurements should be routinely pursued, in particular proper home blood pressure monitoring performed by the patient. Based on out-of-office measurements, cardiovascular risk can be determined more precisely, and this approach is currently preferred for the diagnosis of AH.

II. Screening and diagnostics (Figure 13.1):

- Blood pressure should be measured at every routine diabetology visit, but no less frequently than every 6 months.
- Blood pressure values < 120/70 mm Hg are considered optimal (both office and home measurements).
- Blood pressure values in the range of 120–139/70–89 mm Hg are defined as elevated blood pressure; in this group, lifestyle modification should be implemented, similarly to patients with diagnosed AH.
- Arterial hypertension is diagnosed when blood pressure during a visit is ≥ 180/110 mm Hg or ≥ 140/90 mm Hg at two independent visits. Currently, diagnosis of AH based on ambulatory blood pressure monitoring (ABPM) or home monitoring is preferred, with attention paid to the correctness of home measurements.
- The diagnosis of AH should be re-evaluated in the presence of persistently low blood pressure values despite de-escalation of therapy (remission). This situation most often results from significant weight reduction (pharmacotherapy, metabolic surgery).

III. Principles of antihypertensive therapy

- Non-pharmacological treatment and pharmacotherapy of AH should be initiated simultaneously when blood pressure exceeds 130/80 mm Hg.

IV. Non-pharmacological treatment

- In every case of diagnosed arterial hypertension and elevated blood pressure (120–139/70–89 mm Hg), management should include intensive lifestyle modification aimed at treatment of obesity, reduction of salt intake, optimisation of diet, alcohol abstinence, smoking cessation, appropriate physical acti-

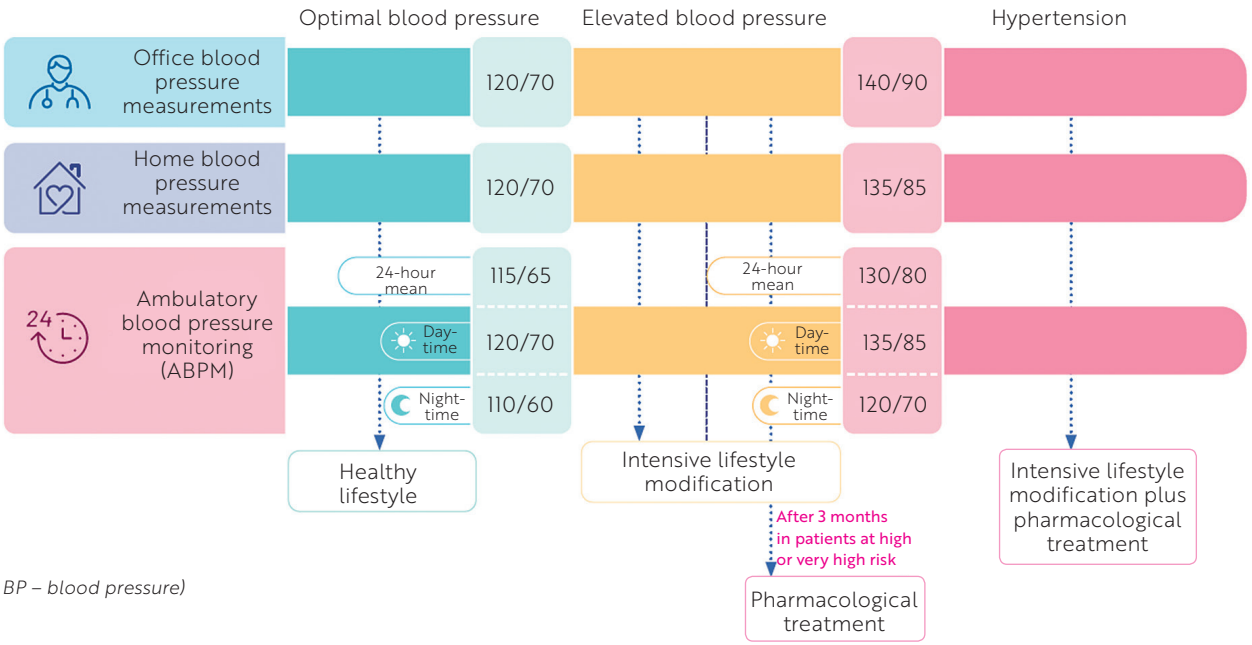


Figure 13.1. Threshold blood pressure values and initiation of antihypertensive therapy [Prejbisz et al. Arterial Hypertension 2024; 28: 113–168. DOI: 10.5603/ah.10391612]

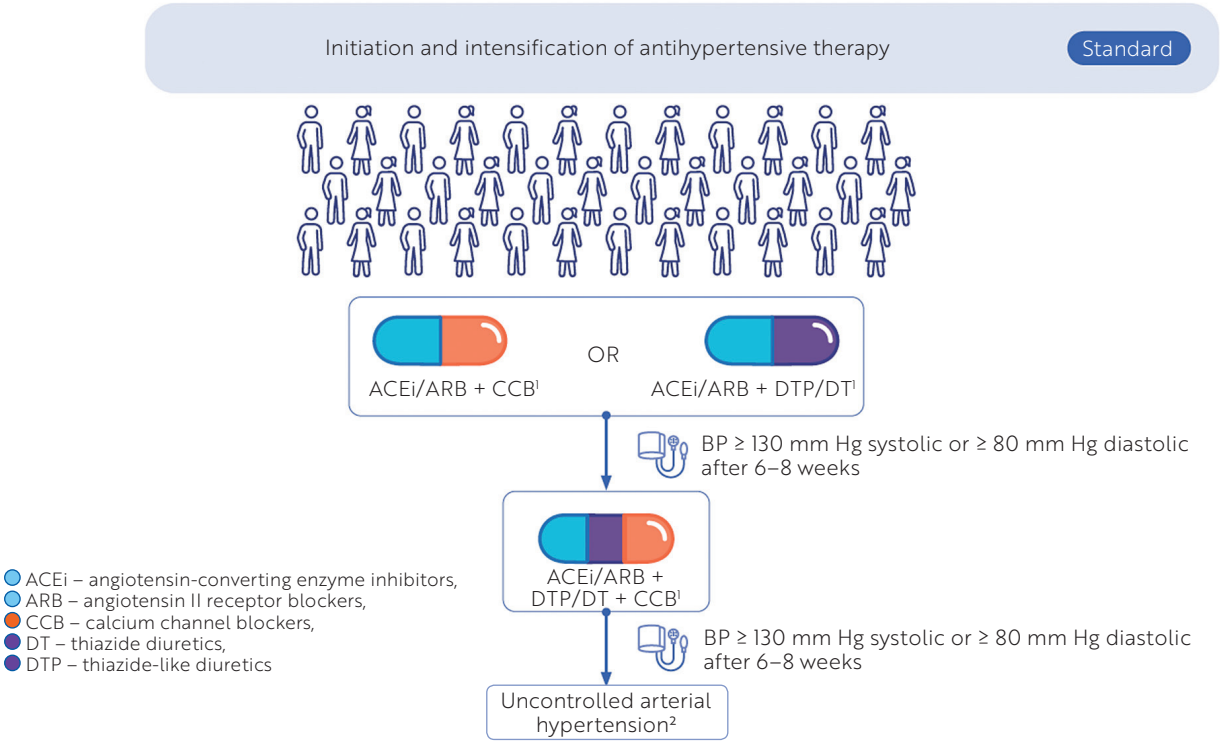
vity, maintenance of proper sleep hygiene with 7–8 hours of nocturnal sleep, and treatment of sleep disorders.

V. Pharmacological treatment (Figure 13.2)

- In most patients, once arterial hypertension has been correctly diagnosed, pharmacotherapy should be initiated according to general principles, i.e. with a single pill combination (SPC). SPC medications are classified as long-acting agents and are administered once daily.
- The majority of SPC preparations contain: 1) a drug from group A, i.e. an angiotensin-converting enzyme inhibitor (ACEI) or an angiotensin II type 1 receptor antagonist (ARB, sartans), combined with 2) a drug from group C, i.e. a dihydropyridine calcium channel blocker (dhp CCB), and/or with 3) a drug from group D, i.e. thiazide-like or thiazide diuretics (DTP/DT). In addition, SPC combinations containing β-adrenolytics (BB) are available. Triple fixed-dose combinations (3-SPC) contain agents from groups A + C + D.
- Considering the complex pathomechanisms of arterial hypertension in diabetes (obesity, fluid retention associated with high sodium sensitivity), initiation of antihypertensive therapy with a 2-SPC containing a diuretic (A + D) should be considered.
- In rare cases – such as systolic blood pressure < 140 mm Hg, situational hypotension, age > 80 years, or isolated systolic hypertension – monotherapy may be considered when initiating antihypertensive treatment; in such cases, long-acting agents should be preferred. If blood pressure control remains suboptimal after initiation of 2-SPC therapy, treatment effectiveness should be increased by recommending a triple fixed-dose combination (3-SPC).
- β-adrenolytics may be used in the treatment of arterial hypertension at any stage of therapy, provided that (numerous) indications for their use are present (e.g. cardiological complications, other comorbid conditions).
- Among antihyperglycaemic agents, SGLT-2 inhibitors and GLP-1 receptor agonists exert a hypotensive effect.

VI. Uncontrolled and resistant hypertension

- If target blood pressure values are not achieved despite therapy with a triple fixed-dose combination (3-SPC), the current therapeutic approach should be reviewed, taking into account the most common causes of treatment failure, i.e. (1) non-adherence to medical recommendations, (2) excessive or regular alcohol consumption, (3) untreated obstructive sleep apnoea, or (4) untreated or inadequately treated obesity, as well as considering diagnostics for other secondary forms of hypertension (hyperaldosteronism, renal artery stenosis, others).
- In cases of truly resistant hypertension, a fourth antihypertensive agent should be added; in the absence of contraindications, this should be a mineralocorticoid receptor



[1]Doses adjusted to blood pressure values.
[2]Further management presented in Figure 13.1.
BP – blood pressure, AH – arterial hypertension

Figure 13.2. General principles of initiation and intensification of antihypertensive therapy

antagonist (spironolactone) or a β-adrenolytic if it has not been previously used in therapy.
- The choice of the fifth and sixth agent should be made between loop diuretics (torasemide), an α-adrenolytic (doxazosin XR/XL), or a centrally acting agent (clonidine).
- Patients with truly resistant hypertension require specialist consultation and extended diagnostic evaluation in centres with an appropriate level of referral.

VII. Target values of antihypertensive therapy

- Blood pressure should be brought within the therapeutic range within 6–8 weeks from initiation of treatment.
- For most individuals in whom pharmacological treatment of hypertension has been initiated, target values are defined as 120–129 mm Hg for systolic blood pressure and 70–79 mm Hg for diastolic blood pressure, or corresponding values obtained in out-of-office measurements. Reduction of systolic blood pressure to < 130 mm Hg is associated with benefits related to a reduced risk of stroke.
- Pharmacological reduction of blood pressure to values < 120/65–60 mm Hg should be avoided.
- If achievement of the standard therapeutic target is not possible, efforts should be made to reduce blood pressure to the maximum level tolerated by the patient.
- In some patients, i.e. those aged > 80 years, with frailty syndrome, risk of hypotension, or isolated systolic hypertension, more liberal therapeutic targets should be applied, i.e. < 140/90 mm Hg.

VIII. Management of hypertension in pregnancy

Management of hypertension during pregnancy includes:
- maintaining a normal diet without significant restriction of salt intake; women with chronic hypertension should continue to follow the principles of a low-sodium diet,
- recommending physical activity: at least 150 minutes of moderate-intensity exercise per week (30–60 minutes, 3–4 times per week),
- initiation of pharmacological treatment of hypertension when blood pressure is ≥ 140/90 mm Hg,
- maintaining blood pressure values during therapy within the range of 110–140/80–85 mm Hg,
- use of acetylsalicylic acid at a dose of 150 mg/day administered in the evening, initiated

before the 16th week of pregnancy for the prevention of pre-eclampsia,
- mandatory hospitalisation of a pregnant woman when blood pressure is ≥ 160/110 mm Hg or when symptoms indicative of pre-eclampsia are present,
- use of a sequential therapeutic regimen, starting with oral labetalol or methyldopa or sustained-release nifedipine (alternatively amlodipine); if therapy is ineffective, two of the above-mentioned drugs should be combined, and in the case of further lack of effectiveness, three of the listed drugs; pregnant women may also use extended-release metoprolol according to indications, however metoprolol should not be combined with labetalol.

IX. Additional remarks

- Concern regarding the use of thiazide diuretics in individuals with diabetes and hypertension in the context of glycaemic control should not constitute a barrier to prescribing these agents. Antihypertensive therapy in such patients may be associated with an increase in HbA_{1c} (~0.2%); however, this effect is dose dependent. In fixed-dose combinations, the lowest effective therapeutic doses are used, which exert a minimal or negligible metabolic effect.
- Thiazide-like diuretics (according to the Summary of Product Characteristics, used at GFR > 30 ml/min) have a pronounced direct vasodilatory effect and are effective antihypertensive agents. Premature discontinuation of thiazide or thiazide-like diuretics when GFR > 30 ml/min significantly increases the risk of uncontrolled hypertension.
- Combination of drugs from the same class, e.g. ACEI + ACEI or ACEI + ARB, is absolutely contraindicated. An exception is the combination of different classes of diuretics, e.g. a thiazide-like diuretic with a loop diuretic or with an MRA.

REFERENCES

1. Arguedas JA, Leiva V, Wright JM. Blood pressure targets for hypertension in people with diabetes mellitus. Cochrane Database Syst Rev 2013; 10: CD008277. DOI: 10.1002/14651858.CD008277.pub2.
2. Brunstrom M, Carlberg B. Effect of antihypertensive treatment at different blood pressure levels in patients with diabetes mellitus: systematic review and meta-analyses. BMJ 2016; 352: i717. DOI: 10.1136/bmj.i717.
3. Cushman WC, Evans GW, Byington RP, et al. Effects of intensive blood-pressure control in type 2 diabetes mellitus. N Engl J Med 2010; 362: 1575–1585.
4. ElSayed NA, Aleppo G, Aroda VR, et al. Erratum. 10. Cardiovascular disease and risk management: standards of care in diabetes – 2023. Diabetes Care 2023;46 (Suppl 1): S158–S190. Diabetes Care 2023; 46: 898. DOI: 10.2337/dc23-er04.
5. Emdin CA, Rahimi K, Neal B, et al. Blood pressure lowering in type 2 diabetes: a systematic review and meta-analysis. JAMA 2015; 313: 603–615.
6. Mancia G, Kreutz R, Brunstrom M, et al. 2023 ESH Guidelines for the management of arterial hypertension. The Task Force for the management of arterial hypertension of the European Society of Hypertension: endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). J Hypertens 2023; 41: 1874–2071.
7. Mann JF, Schmieder RE, McQueen M, et al. Renal outcomes with telmisartan, ramipril, or both, in people at high vascular risk (the ONTARGET study): a multicentre, randomised, double-blind, controlled trial. Lancet 2008; 372: 547–553.
8. Marx N, Federici M, Schütt K, et al. 2023 ESC Guidelines for the management of cardiovascular disease in patients with diabetes: developed by the task force on the management of cardiovascular disease in patients with diabetes of the European Society of Cardiology (ESC). Eur Heart J 2023; 44: 4043–4140.
9. McEvoy JW, McCarthy CP, Bruno RM, et al. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. Eur Heart J 2024; 45: 3912–4018.
10. Palmer SC, Mavridis D, Navarese E. Comparative efficacy and safety of blood pressurelowering agents in adults with diabetes and kidney disease: a network meta-analysis. Lancet 2015; 385: 2047–2056.
11. Postępowanie w nadciśnieniu tętniczym u kobiet w ciąży. Zapobieganie, diagnostyka, leczenie i odległe rokowanie. Stanowisko Polskiego Towarzystwa Nadciśnienia Tętniczego, Polskiego Towarzystwa Kardiologicznego oraz Polskiego Towarzystwa Ginekologów i Położników. Ginekol Perinatol Prakt 2019; 4: 43–111.
12. Prejbisz A, Dobrowolski P, Doroszko A, et al. Guidelines for the management of hypertension in Poland 2024 – the position of the Polish Society of Hypertension/Polish Cardiac Society Experts. Arterial Hypertens 2024; 28: 113–168.
13. Thomopoulos C, Parati G, Zanchetti A. Effects of blood-pressure-lowering treatment on outcome incidence in hypertension: 10 – Should blood pressure management differ in hypertensive patients with and without diabetes mellitus? Overview and meta-analyses of randomized trials. J Hypertens 2017; 35: 922–944.
14. Wright JT Jr, Williamson JD, Whelton PK, et al. A randomized trial of intensive versus standard blood-pressure control. N Engl J Med 2015; 373: 2103–2116.
15. Zoungas S, Chalmers J, Neal B, et al. Follow-up of blood-pressure lowering and glucose control in type 2 diabetes. N Engl J Med 2014; 371: 1392–1406.

14. Dyslipidemia: Standards of Care 2026

CHAPTER HIGHLIGHTS – THERAPEUTIC GOALS:
• LDL-C concentration < 55 mg/dl (< 1.4 mmol/l) and a reduction of at least 50% from baseline values in individuals with diabetes at very high cardiovascular risk, and as a secondary target reduction of non-HDL-C concentration to < 85 mg/dl (< 2.2 mmol/l). [**A**]
• LDL-C levels < 70 mg/dl (< 1.8 mmol/l) with at least a 50% reduction from baseline values in individuals with diabetes at high cardiovascular risk. As a secondary target, non-HDL-C levels should be lowered to < 100 mg/dl (< 2.6 mmol/l). [**A**]
• LDL-C levels < 100 mg/dl (< 2.6 mmol/l) in individuals with diabetes at moderate cardiovascular risk. As a secondary target, non-HDL-C levels should be lowered to < 130 mg/dl (< 3.4 mmol/l). [**A**]

CHAPTER HIGHLIGHTS – TREATMENT
• Statins are the first-choice lipid-lowering medications. In individuals at very high and high cardiovascular risk, it is recommended to use statins at the highest recommended or tolerated doses to achieve therapeutic goals. [**A**]
• If the treatment goal is not achieved, combining a statin with ezetimibe is recommended. [**A**]
• In individuals at very high cardiovascular risk who do not reach the target LDL-C level despite optimal statin and ezetimibe therapy or who cannot tolerate statins, the addition of PCSK9 inhibitors is recommended. [**A**]
• In cases of complete statin intolerance, consider ezetimibe as monotherapy [**C**] or in combination with PCSK9 inhibitors. [**B**] Alternatively, bempedoic acid may be used. [**B**]
• For individuals with hypertriglyceridaemia (TG: 135–499 mg/dl; 1.52–5.6 mmol/l), the use of high-dose eicosapentaenoic acid (EPA) (2 g twice daily) in combination with a statin may be considered. [**B**]

LDL-C is the main primary target of lipid-lowering therapy. In patients with diabetes and high triglyceride (TG) concentrations, obesity, and low LDL-C levels, non-HDL-C or apoB should be measured. Treatment targets differ in patients with type 2 diabetes depending on cardiovascular risk (Table 14.1). Non-HDL-C should be considered a secondary treatment target.

I. Diagnosing lipid disorders

1. Medical history should include:
   a) presence of atherosclerotic cardiovascular diseases: chronic coronary syndrome, ischaemic stroke, peripheral arterial disease (PAD),
   b) assessment of secondary causes of hyperlipidaemia – diet, comorbid conditions, medications (Table 14.2),
   c) occurrence of baseline LDL-C concentration > 190 mg/dl in the family – identification of lipid disorders and premature cardiovascular disease (assessment of the probability of familial hypercholesterolaemia using the Dutch Lipid Clinic Network [DLCN] score and referral of patients with a score ≥ 6 points to specialist centres participating in drug programme B.101) (Table 14.3).
2. Lipid profile testing may be performed in non-fasting patients; however, in patients with hypertriglyceridaemia (TG > 150 mg/dl, > 1.7 mmol/l), testing should be performed after 8–12 hours since the last meal.
3. Measurement of lipoprotein(a) [Lp(a)] should be considered at least once in the lifetime of every adult patient. Lp(a) levels > 50 mg/dl (105 nmol/l) should be regarded in all adults as a factor increasing cardiovascular risk, with higher Lp(a) concentrations associated with a greater increase in this risk. In individuals with very high Lp(a) levels > 430 nmol/l (> 180 mg/dl), cardiovascular risk is comparable to that observed in patients with familial hypercholesterolaemia. Measurement of Lp(a) should be considered in all patients with premature onset of cardiovascular disease (myocardial infarction, ischaemic stroke), with aortic stenosis, familial hypercholesterolaemia, and for risk refinement in individuals at the borderline between moderate and high cardiovascular risk.

Due to the lack of specific therapies lowering Lp(a) levels, **early treatment of cardiovascular risk factors** and **more intensive reduction of LDL-C concentrations** are recommended, taking into account both **total cardiovascular (CV) risk and Lp(a) concentration**.

Lp(a) calculator – www.lpaclinicalguidance.com

Table 14.1 Cardiovascular risk categories in people with type 2 diabetes and recommended target lipid levels according to ESC 2023 guidelines

Cardiovascular risk	Criteria	Recommended target lipid concentrations
Extremely high	Patients with atherosclerotic cardiovascular disease who experience a recurrent cardiovascular event despite treatment with the maximum tolerated statin dose. Patients with multivessel atherosclerosis (coronary and peripheral vessels).	Primary target: LDL-C < 40 mg/dl (< 1.0 mmol/l) Secondary target: non-HDL-C < 70 mg/dl (< 1.8 mmol/l) apoB – no defined therapeutic target in this group
Very high	Type 2 diabetes and atherosclerotic cardiovascular disease* or target-organ damage**, or 10-year cardiovascular risk ≥ 20% according to the SCORE2-Diabetes calculator.	Primary target: LDL-C < 55 mg/dl (< 1.4 mmol/l) and LDL-C reduction ≥ 50% Secondary target: non-HDL-C < 85 mg/dl (< 2.2 mmol/l) apoB < 65 mg/dl
High	Type 2 diabetes and no criteria for very high risk, and 10-year cardiovascular risk from 10% to < 20% according to the SCORE2-Diabetes calculator.	LDL-C < 70 mg/dl (< 1.8 mmol/l) Non-HDL-C < 100 mg/dl (< 2.6 mmol/l) apoB < 80 mg/dl
Moderate	Type 2 diabetes and no criteria for very high risk, and 10-year cardiovascular risk from 5% to < 10% according to the SCORE2-Diabetes calculator	LDL-C < 100 mg/dl (< 2.6 mmol/l) Non-HDL-C < 130 mg/dl (< 3.4 mmol/l) apoB < 100 mg/dl
Low	Type 2 diabetes and no criteria for very high risk, and 10-year cardiovascular risk < 5% according to the SCORE2-Diabetes calculator	No target lipid concentrations established due to insufficient evidence

** Atherosclerotic cardiovascular disease – coronary artery disease, myocardial infarction, lower extremity arterial disease (LEAD), carotid artery disease, aortic aneurysm, ischaemic stroke, or revascularisation of arteries due to atherosclerosis.*

*** Severe target-organ damage: eGFR < 45 ml/min/1.73 m² irrespective of albuminuria, or eGFR 45–59 ml/min/1.73 m² with albuminuria [urinary albumin-to-creatinine ratio (UACR) 30–300 mg/g; stage A2], or proteinuria (UACR > 300 mg/g; stage A3), or presence of microvascular disease in at least three different sites, e.g. albuminuria (stage A2) plus retinopathy plus neuropathy.*

SCORE2-Diabetes applies to patients aged 40–69 years with type 2 diabetes, without atherosclerotic vascular disease and/or severe target-organ damage.

Table 14.2 Secondary causes of hyperlipidaemia

Diet	Comorbid conditions	Medications
High-fat diet High-carbohydrates diet Alcohol	Hypothyroidism Diabetes Obesity Lipid storage disorders Kidney diseases – nephrotic syndrome, uraemia Liver diseases – primary biliary cholangitis, primary sclerosing cholangitis, alcoholic liver cirrhosis, MASLD Anorexia nervosa Cushing syndrome Systemic lupus erythematosus Psoriasis HIV infection Pregnancy – third trimester: increase in TG	Glucocorticosteroids (GCS) Oral oestrogens Oral retinoids Ion exchange resins Protease inhibitors (lopinavir with ritonavir, saquinavir, fosamprenavir) Nucleoside reverse transcriptase inhibitors (stavudine, zidovudine) Non-nucleoside reverse transcriptase inhibitors (efavirenz) Thiazide diuretics Non-selective β-adrenolytics (except carvedilol) Tamoxifen Cyclophosphamide Cyclosporine Sirolimus Everolimus L-asparaginase Second-generation antipsychotics (clozapine, olanzapine)

MASLD – metabolic dysfunction associated steatotic liver disease

II. Monitoring and follow-up of lipid levels in diabetes

Lipid levels should be measured at the time of diabetes diagnosis, and thereafter their concentrations should be monitored once a year or more frequently, depending on their values.

If lipid concentrations are above therapeutic targets, monitoring every 8–12 weeks from the initiation of therapy is recommended until target levels are achieved; with the exception of patients after an acute coronary syndrome, in whom lipid measurement after treatment initiation should be performed within 4–6 weeks.

Before initiating lipid-lowering therapy, ALT, CK, and TSH should be measured.

If lipid concentrations are within the desired range, follow-up lipid profile testing should be performed once a year, and ALT and CK only if symptoms occur.

III. Non-pharmacological treatment of dyslipidaemia in individuals with diabetes

1. Lifestyle modification includes:
- increasing physical activity,
- reduction of body weight in individuals with overweight or obesity,
- smoking cessation.

2. A diet with restriction of saturated fat intake to < 10% of total energy intake, cholesterol intake < 300 mg/day or < 200 mg/day in the presence of elevated LDL-C, and maximal limitation of trans-unsaturated fats; intake of n-6 polyunsaturated fatty acids should constitute 4–8% of energy intake, and intake of n-3 polyunsaturated fatty acids should include 2 g of linolenic acid and 200 mg/day of very-long-chain fatty acids.

IV. Pharmacological treatment of dyslipidaemia in individuals with diabetes

1. **Statins:**
   a) statins remain first-line therapy for lowering LDL-C concentrations in patients with type 1 diabetes, type 2 diabetes, and dyslipidaemia due to their effectiveness in preventing cardiovascular events and reducing cardiovascular mortality, regardless of sex,
   b) statins with strong lipid-lowering effects (rosuvastatin and atorvastatin) are indicated in patients with diabetes at high or very high cardiovascular risk, as they reduce LDL-C concentrations by 40–63% and significantly reduce the incidence of major cerebrovascular and coronary events; in these patients, high doses of statins are recommended: rosuvastatin 20–40 mg/day and atorvastatin 40–80 mg/day,
   c) the beneficial effect of statins outweighs their potential diabetogenic effect, estimated as a 9% increase in the risk of developing diabetes, particularly in older patients and those at risk of diabetes,
   d) statins are safe and generally well tolerated; subjective adverse effects (such as fatigue, muscle pain, and neurological symptoms) are more common than objective adverse effects due to the nocebo effect, and women experience adverse effects more frequently than men; in most cases of myopathy or rhabdomyolysis, drug interactions occur in the context of higher statin doses or combination with gemfibrozil. Factors increasing the risk of myopathy include older age, intense physical exertion, hypothyroidism, and drug interactions.

Table 14.3 Criteria for the diagnosis of familial hypercholesterolaemia (FH) – point-based scale (according to the Dutch Lipid Clinic Network)

Physical examination	Points
Tendon xanthomas	6
Corneal arcus	4
LDL-C	
> 330 mg/dl (> 8.5 mmol/l)	8
250–329 mg/dl (6.5–8.4 mmol/l)	5
190–249 mg/dl (5.0–6.4 mmol/l)	3
155–189 mg/dl (4.0–4.9 mmol/l)	1
Genetic testing	
Mutation in *LDLR, APOB, PCSK9* genes	8
Family history	
First-degree relatives with premature coronary or vascular disease	1
First-degree relatives with LDL-C > 190 mg/dl	1
First-degree relatives with tendon xanthomas and/or corneal arcus	2
Children and adolescents < 18 years of age with LDL-C > 155 mg/dl	2
Clinical history	
Premature coronary artery disease*	2
Premature cerebrovascular or peripheral vascular disease*	1

Certain diagnosis: > 8 points, probable diagnosis: 6–8 points, possible diagnosis: 3–5 points, unlikely diagnosis: < 3 points

**Premature cardiovascular disease: age < 55 years in men, < 60 years in women.*

e) contraindications to statin therapy:
- pregnancy and breastfeeding,
- reproductive age if a woman does not use effective contraception,
- baseline ALT values three times above the upper limit of normal (of unknown cause),
- baseline creatine kinase (CK) values four times above the upper limit of normal,
- active viral hepatitis,
- liver failure (Child–Pugh class B and C),
- choroideraemia,

f) assessment of statin therapy safety – ALT should be measured before treatment initiation, after 8–12 weeks of therapy, and after each statin dose increase or in the event of symptoms; CK is recommended to be measured before treatment initiation and does not require routine monitoring, only in the presence of myalgia during therapy,

g) statin intolerance:
- **complete intolerance:** statin intolerance is defined as the inability to use the drug due to the occurrence of clinically significant adverse effects and/or a marked increase in biomarkers (ALT/AST > 3 × ULN or CK > 5 × ULN) in the absence of concomitant factors increasing the risk of adverse effects, such as hypothyroidism, drug interactions, physical exertion, or muscle diseases.

Complete statin intolerance should refer to at least two different statins, including one irrespective of the dose used and another at the lowest dose (rosuvastatin 5 mg/day, atorvastatin 10 mg/day, simvastatin 10 mg/day, pitavastatin 2 mg/day).

In the case of complete statin intolerance, ezetimibe should be considered as monotherapy or in combination with a PCSK9 inhibitor. Alternatively, bempedoic acid may be used; this is a prodrug that reduces cholesterol synthesis by inhibiting adenosine triphosphate (ATP) citrate lyase, with very limited musculoskeletal adverse effects. In patients at high cardiovascular risk who are intolerant to statins, the use of bempedoic acid (180 mg once daily) was associated with a reduction in cardiovascular events, while a higher incidence of gout and cholelithiasis was observed.
- intolerance to high-dose statins – if a patient reports adverse effects during treatment with high statin doses, the following strategies may be undertaken:
  - » discontinuation of the statin and re-challenge after symptom resolution with the same statin at a lower dose in combination with ezetimibe, or
  - » switching to another statin at a lower dose in combination with ezetimibe.

There is evidence of a beneficial lipid-lowering effect of statins (atorvastatin or rosuvastatin) even at low doses administered every 2–3 days.

Approximately 70–90% of patients reporting statin intolerance (fatigue, muscle pain, neurological symptoms) are able to tolerate statin therapy upon re-administration.

2. Combination lipid-lowering therapy

a) **statin + ezetimibe:** if target LDL-C levels are not achieved with statin therapy, combination treatment with a statin and ezetimibe is recommended; in patients after acute coronary syndrome, combination therapy with a high-dose statin (atorvastatin 80 mg or rosuvastatin 40 mg) plus ezetimibe 10 mg may be considered from the outset,

b) **statin + ezetimibe + PCSK9 inhibitor (alirocumab, evolocumab) or statin + ezetimibe + inclisiran:** in patients at very high cardiovascular risk who do not achieve target LDL-C levels despite optimal statin and ezetimibe therapy, addition of a PCSK9 inhibitor or inclisiran is recommended.

Evolocumab and alirocumab are monoclonal antibodies that bind PCSK9, leading to a marked reduction in LDL-C levels by approximately 60% in monotherapy and up to 85% when combined with high-dose statin therapy and ezetimibe. They significantly reduced cardiovascular events (cardiovascular death, myocardial infarction, stroke, hospitalisation for unstable angina, or coronary revascularisation) in subgroups of patients with diabetes and atherosclerotic cardiovascular disease. In addition to LDL-C, they significantly reduce other atherogenic lipids (TG, non-HDL-C, apoB-containing particles) in patients with diabetes and mixed dyslipidaemia. No significant association was demonstrated between antibody use and the risk of developing diabetes. In patients with impaired renal function (including those on haemodialysis), dose adjustment is not required. Evolocumab is recommended subcutaneously at a dose of 140 mg every 2 weeks, and alirocumab subcutaneously at a dose of 150 mg every 2 weeks or 300 mg once monthly.

Inclisiran-siRNA – a small interfering RNA molecule – inhibits hepatic PCSK9 synthesis. When added to high-dose statin therapy, it reduces LDL-C by a further 50–55% in patients with and without diabetes. It is recommended for

use in patients with eGFR > 15 ml/min/1.73 m², administered subcutaneously at a dose of 284 mg every 6 months. Studies evaluating the effect of inclisiran on cardiovascular events are ongoing.

In Poland, treatment with PCSK9 inhibitors and inclisiran is available within the B.101 drug programme in specialist outpatient clinics. Patients with suspected familial hypercholesterolaemia (according to the DLCN score of at least 6 points) and patients after acute coronary syndrome within the last 5 years may be referred to the programme.

c) **statin + fibrate:** in patients at high cardiovascular risk who have achieved target LDL-C levels but continue to have TG concentrations > 200 mg/dl (> 2.3 mmol/l), fenofibrate in combination with a statin may be considered. Combination therapy with a statin and a fibrate (particularly gemfibrozil) is associated with an increased risk of abnormal liver enzyme values, myopathy, and rhabdomyolysis, especially in the presence of chronic kidney disease and when high drug doses are used. Renal function should be assessed before therapy, after 3 months, and after 6 months. Combination therapy must not be recommended when eGFR < 30 ml/min/1.73 m². If a decline in eGFR to < 30 ml/min/1.73 m² is observed during fenofibrate therapy, treatment should be discontinued.

d) **statin + ethyl eicosapentaenoate (EPA):** in patients with hypertriglyceridaemia (TG 135–499 mg/dl; 1.52–5.6 mmol/l), the use of ethyl eicosapentaenoate (eicosapentaenoic acid ester) at high doses, i.e. 2 × 2 g, in combination with a statin should be considered. EPA lowers TG concentrations and favourably affects cardiovascular outcomes; however, at the highest dose (4 g/day), it increases the risk of atrial fibrillation.

V. Hypertriglyceridaemia

The risk of acute pancreatitis occurs at TG concentrations > 750 mg/dl (> 8.5 mmol/l) and depends on the presence of chylomicrons in serum. Hypertriglyceridaemia accounts for approximately 10% of cases of acute pancreatitis.

The classification of hypertriglyceridaemia according to TG values and its causes is presented in Table 14.4.

Experts from the American Diabetes Association recommend intensification of lifestyle modification and optimisation of glycaemic control in individuals with diabetes and elevated TG concentrations (> 150 mg/dl; 1.7 mmol/l) and/or low HDL-C concentrations (< 40 mg/dl; < 1.0 mmol/l for men and < 50 mg/dl; < 1.3 mmol/l for women).

Management of hypertriglyceridaemia is presented in Table 14.5.

VI. Type 1 diabetes

In type 1 diabetes, elevated LDL-C values are observed in patients with poorly controlled glycaemia. High HDL-C concentrations may be pro-inflammatory and therefore atherogenic.

Cardiovascular risk assessment in patients with type 1 diabetes has been less well studied than in patients with type 2 diabetes. Based on the Scottish–Swedish diabetes registry, a tool was developed to predict 10-year cardiovascular risk in patients with type 1 diabetes: https://diabepi.shinyapps.io/cvdrisk/.

Statins constitute the cornerstone of lipid-lowering therapy in type 1 diabetes. In patients with type 1 diabetes aged over 40 years, without atherosclerotic cardiovascular disease, statin therapy should be considered in order to reduce cardiovascular risk.

In younger patients with type 1 diabetes, below 40 years of age, early initiation of statin therapy may be justified in the presence of other cardiovascular risk factors, microvascular complications, or a 10-year cardiovascular risk > 10% calculated using the Scottish–Swedish model.

Table 14.4. Types and causes of hypertriglyceridaemia

Type of hypertriglyceridaemia	TG values, mg/dl (mmol/l)	Causes
Optimal	< 135 (< 1.52)	–
Elevated	135–199 (1.52–2.29)	Diet Lack of physical activity Type 2 diabetes Alcohol Obesity Metabolic syndrome
Hypertriglyceridaemia	200–749 (2.3–8.5)	
Severe hypertriglyceridaemia	> 750 (> 8.5)	Monogenic Polygenic

Table 14.5. Management of hypertriglyceridaemia according to TG concentration

TG concentration	TG: 200–749 mg/dl (2.3–8.5 mmol/l) Increased VLDL-TG Increased cardiovascular risk	Severe > 750 mg/dl (> 8.5 mmol/l) Presence of chylomicrons and VLDL-TG Increased risk of acute pancreatitis
Primary therapeutic target	· Target LDL-C concentration	· TG reduction
Secondary therapeutic target	· Target non-HDL-C concentration	· Target LDL-C and non-HDL-C concentration, if the risk of acute pancreatitis is reduced
Non-pharmacological treatment	· Strict diabetes control, correction of secondary causes of hypertriglyceridaemia (diabetes, chronic liver or kidney disease and/or nephrotic syndrome, hypothyroidism) · Replacement of drugs increasing TG levels with neutral agents · Reduction of alcohol intake or alcohol abstinence · Body weight reduction in the presence of overweight or obesity · Reduction of carbohydrate intake, particularly fructose and sucrose · Increased physical activity · Replacement of saturated fats with polyunsaturated fats	· Alcohol abstinence · Restrictive low-fat diet: fat intake < 20% of total calories, 10–20 g of fat per day (2000 kcal diet) · Use of medium-chain triglycerides (MCT, C6–12) · Reduction of total carbohydrate intake, particularly fructose and sucrose · Body weight reduction in the presence of overweight or obesity · Increased physical activity
Pharmacological treatment	· Statin (atorvastatin, rosuvastatin) · High doses of ethyl eicosapentaenoate (2 × 2 g/day) should be considered in combination with a statin in patients at high or very high cardiovascular risk and increased TG levels (135–499 mg/dl or 1.52–5.63 mmol/l) in order to reduce the risk of cardiovascular events · Addition of a fibrate may be considered if target LDL-C has been achieved and TG concentration remains > 200 mg/dl (> 2.3 mmol/l) in patients at high cardiovascular risk	· Fenofibrate + omega-3 fatty acids (4 g/day) · Volanesorsen (300 mg subcutaneously once weekly) should be considered in patients with severe hypertriglyceridaemia (> 750 mg/dl or > 8.5 mmol/l) caused by familial chylomicronaemia syndrome (FCS), in order to reduce TG concentration and the risk of pancreatitis · In acute conditions, rapid reduction of TG levels can be achieved using plasmapheresis · In individuals with diabetes not treated with insulin, insulin therapy should be initiated, most often via intravenous infusion using an infusion pump, to achieve optimal glycaemic control – this approach allows reduction of hypertriglyceridaemia within 2–5 days; in addition to insulin, the use of low-molecular-weight or unfractionated heparin should be considered
Genetic testing	· No indication for genetic testing in most cases, except for suspected familial dysbetalipoproteinaemia*	· Genetic diagnostics for FCS or MCS

FCS – familial chylomicronaemia syndrome
MCS – multifactorial chylomicronaemia syndrome
**Familial dysbetalipoproteinaemia (remnant lipoprotein disease rich in TG) should be suspected at TG concentrations of 300–1000 mg/dl, apoB < 120 mg/dl, non-HDL-C/apoB > 3.69 mmol/g (1.43 mg/mg) in patients with a positive family history of premature atherosclerotic cardiovascular disease (myocardial infarction, stroke, PAD).*

REFERENCES

1. Banach M, Burchardt P, Chlebus K, et al. Wytyczne PTL/KLRWP/PTK/PTDL/PTD/PTNT diagnostyki i leczenia zaburzeń lipidowych w Polsce 2021. Nadciśn Tętn Prakt 2021; 7: 113–222.
2. Bebu I, Braffett BH, Orchard TJ, et al.; DCCT/EDIC Research Group. Mediation of the effect of glycemia on the risk of CVD outcomes in type I diabetes: the DCCT/EDIC study. Diabetes Care 2019; 42: 1284–1289. DOI: 10.2337/dc18-1613.
3. Bhatt DL, Steg PG, Miller M, et al. Cardiovascular risk reduction with icosapent ethyl for hypertriglicerydemia. N Engl J Med 2019; 380: 11–22. DOI: 10.1056/NEJMoa1812792.
4. Charlton-Menys V, Betteridge DJ, Colhoun H, et al. Targets of statin therapy: LDL-cholesterol, non-HDL-cholesterol and apolipoprotein B in type 2 diabetes in the Collaborative Atorvastatin Diabetes Study (CARDS). Clin Chem 2009; 55: 473–480.

5. Cholesterol Treatment Trialists' (CTT) Collaboration. Fulcher J, O'Connell R, Voysey M, et al. Efficacy and safety of LDL-lowering therapy among men and women: meta-analysis of individual data from 174 000 participants in 27 randomised trials. Lancet 2015; 385: 1397–1405.
6. Ginsberg HN, Elam MB, Lovato LC, et al. Effects of combination lipid therapy in type 2 diabetes mellitus. N Engl J Med 2010; 362: 1563–1574.
7. Giugliano RP, Cannon CP, Blazing MA, et al. Benefit of adding ezetimibe to statin therapy on cardiovascular outcomes and safety in patients with versus without diabetes mellitus:results from IMPROVE-IT (Improved Reduction of Outcomes: Vytorin Efficacy International Trial. Circulation 2018; 137: 1571–1582.
8. Hero C, Rawshani A, Svensson AM, et al. Association between use of lipid-lowering therapy and cardiovascular diseases and death in individuals with type I diabetes. Diabetes Care 2016; 39: 996–1003.
9. Johannesen CDL, Mortensen MB, Landsted A, et al. Apolipoprotein B and non-HDL-cholesterol better reflect residual risk than LDL-cholesterol in statin-treated patients. J Am College Cardiol 2021; 77: 1439–1450.
10. Jones P, Kafonek S, Hunninghake D. Comparative dose efficacy study of atorvastatin vesrus simvastatin, pravastatin, lovastatin, and fluvastatin in patients with hypercholesterolemia (the CURVES study). Am J Cardiol 1998; 81: 582–587.
11. Kearney P, Blackwell L, Collins R, et al. Cholesterole Treatment Trialists' (CTT) Collaboration. Efficacy of cholesterol-lowering therapy in 18 686 people with diabetes in 14 randomised trials of statins: a meta-analysis. Lancet 2008; 371: 117–125.
12. Lorenzatti AJ, Monsalvo ML, Lopez JAG, et al. Effects of evolocumab in individuals with type 2 diabetes with and without atherogenic dyslipidenia: an analysis from BANTING and BERSON. Cardiovasc Diabetol 2021: 20: 94. DOI: 10.1186/s12933-021-01287-6.
13. Mach F, Baigent C, Catapano AL, et al. 2019 ESC/EAS guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS). Eur Heart J 2020; 41: 111–188.
14. Mach F, Koskinas KC, Roeters van Lennen JE, et al. 2025 Focused Update of the ESC/EAS Guidelines for the management of dyslipidaemias. Eur Heart J 2025. https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Focused-Update-on-Dyslipidaemias.
15. Marcovecchio ML, Chiesa ST, Bond S, et al. ACE inhibitors and statins in adolescents with type I diabetes. N Engl J Med 2017; 377: 1733–1745.
16. Marx N, Federici M, Schütt K, et al. 2023 ESC Guidelines for the Management of Cardiovascular Disease in Patients With Diabetes: Developed by the Task Force on the Management of Cardiovascular Disease in Patients With Diabetes of the European Society of Cardiology (ESC). Eur Heart J 2023; 44: 4043–4140.
17. McGurnaghan SJ, McKeigue PM, Read SH, et al. Development and validation of a cardiovascular risk prediction model in type 1 diabetes. Diabetologia 2021; 64: 2001–2011.
18. Moriarty PM, Thompson PD, Cannon CP, et al. Efficacy and safety of alirocumab vs ezetimibe in statin-intolerant patients with a statin rechallenge arm: the ODYSSEY ALTERNATIVE randomized trial. J Clin Lipidol 2015; 9: 758–769.
19. Nissen SE, Lincoff AM, Brennan D, et al. Bempedoic acid and cardiovascular outcome in statin intolerant patients. N Engl J Med 2023; 388: 1353–1364.
20. Nissen SE, Dent-Acosta RE, Rosenson RS, et al. Comparison of PCSK9 inhibitor evolocumab vs ezetimibe in statin intolerant patients:design of the goal achievement after utilizing an anti-PCSK9 antibody in statin-intolerant subjects 3 (GAUSS-3) trial. Clin Cardiol 2016; 39: 137–144.
21. Rawshani A, Sattar N, Franzen S, et al. Relative prognostic importance and optimal levels of the risk factors for mortality and cardiovascular outcomes in type 1 diabetes mellitus. Circulation 2019; 139: 1900–1912.
22. Ray KK, Colhoun HM, Szarek M, et al. Effects of alirocumab on cardiovascular and metabolic outcomes after acute coronary syndrome in patients with or without diabetes: a prespecified analysis of the ODYSSEY OUTCOMES randomised controlled trial. Lancet Diabetes Endocrinol 2019; 7: 618–628.
23. Ray KK, Wright RS, Kallend D, et al. Two phase 3 trials of inclisiran in patients with elevated LDL cholesterol. N Engl J Med 2020; 382: 1507–1519.
24. Reston JT, Buelt A, Donahue MP, et al. Interventions to improve statin tolerance and adherence in patients at risk for cardiovascular disease: a systematic review for the 2020 US Department of Veterens Affairs and US Department of Defense Guidelis for Management of Dyslipidemia. Ann Intern Med 2020; 173: 806–812.
25. Rosenson RS, Daviglus ML, Handelsman Y, et al. Efficacy and safety of evolocumab in individuals with type 2 diabetes mellitus:primary results of randomized controlled BANTING study. Diabetologia 2019; 62: 948–958.
26. Sabatine MS, Giugliano RP, Keech AC, et al. FOURIER Steering Committee and Investigators. Evolocumab and clinical outcomes in patients with cardiovascular disease. N Engl J Med 2017; 376: 1713–1722.
27. Sattar N, Preiss D, Murray HM, et al. Statins and risk of incident diabetes: a collaborative meta-analysis of randomized statin trials. Lancet 2010; 375: 735–742.
28. Taskinen MR, del Prato S, Bujas-Bobanovic M, et al. Efficacy and safety of alirocumab in individuals with type 2

diabetes mellitus with or without mixed dyslipidaemia: analysis of the ODYSSEY LONG TERM trial. Atherosclerosis 2018; 276: 124–130.
29. Thanassoulis G, Williams K, Ye K, et al. Relations of change in plasma levels of LDL-C, non-HDL-C and apo-B with risk reduction from statin therapy: a meta-analysis of randomized trials. J Am Heart Assoc 2014; 3: e000759. DOI: 10.1161/JAHA.113.000759.
30. Visseren FLJ, Mach F, Smulders YM, et al. 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J 2021; 42: 3227–3337.
31. Warden BA, Duell PB. Inklisiran: a novel agent for lowering apolipoprotein B-containing lipoproteins. J Cardiovasc Pharmacol 2021; 78: e157–e174.
32. Zhang XL, Zhu QQ, Zhu L, et al. Safety and efficacy of anti-PCSK9 antibodies: a meta-analysis of 25 randomized, controlled trials. BMC Med 2015; 13: 123.

15. Hypoglycaemia

CHAPTER HIGHLIGHTS
• A person with diabetes should be asked about symptoms and frequency of hypoglycaemia at every visit. [**C**]
• Every person at high risk of clinically significant hypoglycaemia (< 54 mg/dl (3.0 mmol/l)) should have access to glucagon. Family members and caregivers of a person with diabetes, as well as teachers of children and adolescents with diabetes, should be familiar with the method of glucagon administration. [**E**]
• A change in the method of diabetes treatment should be considered in the presence of episodes of severe hypoglycaemia and hypoglycaemia unawareness. [**B**]
• In the treatment of hypoglycaemia in conscious individuals (at a glucose concentration ≤ 70 mg/dl (3.9 mmol/l)), the administration of 15 g of glucose is crucial; alternatively, other simple carbohydrates may be given orally. If glucose measurement after 15 minutes still indicates hypoglycaemia, glucose/carbohydrate administration should be repeated. After resolution of hypoglycaemia, if recurrence is possible, the person with diabetes should eat a snack/meal. [**C**]
• In individuals with diabetes treated with insulin who have hypoglycaemia unawareness or have experienced an episode of severe hypoglycaemia, the therapeutic goal should be a slightly higher glucose concentration for a period of at least several weeks, in order to at least partially restore hypoglycaemia symptom awareness and prevent its occurrence in the future. [**A**]
• In individuals with diabetes treated with insulin who have hypoglycaemia unawareness or frequent episodes of hypoglycaemia, the use of CGM is strongly recommended. [**A**]

I. Definition

Hypoglycaemia is diagnosed when blood glucose concentration falls below 70 mg/dl (3.9 mmol/l), regardless of the presence of clinical symptoms, which in some individuals, especially those with long-standing type 1 diabetes, may occur only at lower glycaemic values. The value of 70 mg/dl (3.9 mmol/l) should be regarded as an alert level requiring the intake of glucose (or its isomer – dextrose), or alternatively other simple carbohydrates, as well as adjustment of doses of glucose-lowering medications, irrespective of the presence or absence of symptoms, in order to prevent a further decline in glucose levels. This constitutes the rationale for defining the threshold value for impending hypoglycaemia at 70 mg/dl (3.9 mmol/l).

Clinically significant hypoglycaemia should be defined as a value below 54 mg/dl (3.0 mmol/l). Symptoms of hypoglycaemia may also occur at higher glycaemic values, even > 100 mg/dl (5.6 mmol/l), when glucose levels decline rapidly. So-called hypoglycaemia unawareness, defined as the lack of perception of pathologically low glycaemic values (≤ 70 mg/dl, i.e. ≤ 3.9 mmol/l), is an important complication of frequent hypoglycaemic episodes. It may also be a manifestation of diabetic neuropathy.

The classification of hypoglycaemia according to the International Hypoglycemia Study Group (2017) is presented in Table 15.1. Severe hypoglycaemia is an episode requiring the assistance of another person in order to administer carbohydrates, glucagon, or undertake other actions. Blood glucose values during the episode may be unavailable; however, resolution of symptoms after administration of glucose and/or glucagon is considered sufficient evidence that the episode was caused by low blood glucose concentration.

Recurrent severe hypoglycaemia is defined as two or more episodes of severe hypoglycaemia within the previous 12 months.

Table 15.1. Classification of hypoglycaemia according to the International Hypoglycemia Study Group [6]

Blood glucose levels	Criterion	Comment
Alert glucose level (level 1)	≤ 70 mg/dl ≤ 3.9 mmol/l	Glucose concentration requiring treatment with simple carbohydrates – adjustment of doses of glucose-lowering medications is indicated
Clinically significant hypoglycaemia (level 2)	< 54 mg/dl < 3.0 mmol/l	Sufficiently low glucose concentration indicating clinically significant hypoglycaemia
Severe hypoglycaemia (level 3)	No specific blood glucose threshold	Hypoglycaemia associated with severe impairment of cognitive function requiring assistance of third parties to terminate the episode

II. General remarks

1. A person with diabetes should not be automatically regarded as being at risk of hypoglycaemia and burdened with consequences related to employment or social functioning resulting from this assumption.
2. The physician should perform regular assessment of patients' cognitive function, and in the case of its deterioration, patients should be monitored more closely for hypoglycaemia, optimally using CGM.
3. The risk of hypoglycaemia increases in the following situations:

- use of insulin as monotherapy or in combination with other antihyperglycaemic agents,
- use of sulfonylurea derivatives as monotherapy or in combination with other antihyperglycaemic agents,
- inappropriate dosing of the above-mentioned drugs in situations of increased physical activity, reduced caloric intake, or alcohol consumption,
- striving for rapid normalisation of HbA_{1c} values,
- coexistence of other conditions predisposing to hypoglycaemia (including chronic kidney disease, hypothyroidism, adrenal insufficiency, eating disorders, diseases associated with impaired intestinal absorption, cognitive disorders),
- hypoglycaemia unawareness,
- an episode of severe hypoglycaemia in recent weeks,
- occurrence of high glycaemic variability.

In certain situations (older adults, individuals with ischaemic heart disease), hypoglycaemia may constitute an immediate life-threatening condition.

III. Management of recurrent hypoglycaemia includes:

- thorough analysis of the person's habits and the treatment of diabetes and other comorbid conditions,
- education of people with diabetes regarding prevention of hypoglycaemia (e.g. recommendation to reduce insulin dose before planned physical activity),
- modification of diabetes therapy in order to reduce the risk of hypoglycaemia, e.g. replacement of sulfonylureas with drugs associated with a lower risk of hypoglycaemia, change of the insulin therapy regimen, use of insulin preparations with a lower risk of hypoglycaemia, use of insulin pumps, preferably automated insulin delivery (AID) systems, or systems with automatic suspension of insulin delivery in the event of impending hypoglycaemia,
- frequent self-monitoring and use of continuous glucose monitoring systems (CGM), if available to the patient.

IV. Management of hypoglycaemia unawareness

Management as in recurrent hypoglycaemia should be implemented, and additionally:

- education of people with diabetes and their relatives regarding recognition of subtle and atypical warning symptoms of hypoglycaemia (hypoglycaemia awareness training),
- consideration of this condition in occupational activities and driving,
- modification of therapy aimed at a significant reduction in the frequency of hypoglycaemia as the only method to improve hypoglycaemia awareness.

V. Acute management of hypoglycaemia

1. In a conscious person:

- consumption of 15 g of glucose (or, if unavailable, other simple carbohydrates) is recommended, followed by blood glucose measurement after 15 minutes; if hypoglycaemia persists, repeated intake of 15 g of glucose/simple carbohydrates and reassessment after 15 minutes is recommended; if there is a risk of recurrent hypoglycaemia, e.g. after erroneous administration of an excessive insulin dose, alcohol consumption, or prolonged physical activity, in addition to the above intervention,

consumption of complex carbohydrates and glucose monitoring are recommended.

2. In an unconscious person or a person with impaired consciousness who is unable to swallow:
- intravenous administration of 10% or 20% glucose solution (initial bolus dose 0.2–0.5 g of glucose/kg body weight), followed, if necessary, by continuous intravenous infusion of 10% glucose under glycaemic control,
- if there is a risk of recurrent hypoglycaemia, maintenance of a 10% glucose infusion under glycaemic control is recommended,
- in the case of difficult venous access, 1 mg of glucagon should be administered intramuscularly or subcutaneously (0.5 mg in children with body weight < 25 kg and 1 mg in children with body weight ≥ 25 kg); glucagon may also be administered intranasally at a dose of 3 mg in people with diabetes aged over 4 years, regardless of body weight,
- after regaining consciousness, if there is a risk of recurrence of hypoglycaemia, oral administration of 10–20 g of carbohydrates and glucose monitoring are recommended,
- in individuals with diabetes treated with insulin or sulfonylureas, prolonged episodes of hypoglycaemia may occur and may sometimes require many hours of glucose infusion,
- in the event of severe hypoglycaemia, hospitalisation of the patient should be considered due to the life-threatening condition associated with the risk of irreversible damage to the central nervous system, especially when recurrence of severe hypoglycaemia is possible,
- in outpatient settings, trained persons from the patient's surroundings should be advised to administer injectable glucagon intramuscularly or subcutaneously, or intranasal glucagon.

3. In individuals treated with intensive insulin therapy using insulin analogues or personal insulin pumps, management of hypoglycaemia usually includes only oral administration of 15 g of glucose and blood glucose measurement after 15 minutes. If low glucose values persist, glucose administration should be repeated, and glucose concentration reassessed after another 15 minutes (the 15/15 rule). In personal insulin pump therapy, in the event of hypoglycaemia or impending hypoglycaemia, suspension of the basal insulin infusion and reassessment of glucose is indicated. In individuals using hybrid closed-loop (HCL) systems, in the case of hypoglycaemia or risk thereof, 5–8 g of oral glucose may be administered, and glucose reassessed after 15 minutes.

4. In the case of administration of an excessive dose of long-acting insulin (human or analogue): the possibility of delayed recurrence of hypoglycaemia after initial recovery from the hypoglycaemic episode should be taken into account.

REFERENCES

1. Abraham SB, Arunachalam S, Zhong A, et al. Improved real-world glycemic control with continuous glucose monitoring system predictive alerts. J Diabetes Sci Technol 2021; 15: 91–97.
2. Beck SE, Kelly C, Price DA. Non-adjunctive continuous glucose monitoring for control of hypoglycaemia (COACH): results of a post-approval observational study. COACH Study Group. Diabet Med 2022; 39: e14739.
3. Griffin TP, Gallen G, Hartnell S, et al. UK's Association of British Clinical Diabetologist's Diabetes Technology Network (ABCD-DTN): best practice guide for hybrid closed-loop therapy. Diabet Med 2023; 40: e15078.
4. Heinemann L, Freckmann G, Ehrmann D, et al. Real-time continuous glucose monitoring in adults with type 1 diabetes and impaired hypoglycemia awareness or severe hypoglycaemia treated with multiple daily insulin injections (HypoDE): a multicentre, randomised controlled trial. Lancet 2018; 391: 1367–1377.
5. Hermanns N, Heinemann L, Freckmann G, et al. Impact of CGM on the management of hypoglycemia problems: overview and secondary analysis of the HypoDE study. J Diabetes Sci Technol 2019; 13: 636–644.
6. International Hypoglycemia Study Group. Glucose concentrations of less than 3 mmol/L (54 mg/dL) should be reported in clinical trials: a joint position statement of the American Diabetes Association and European Association for the Study of Diabetes. Diabetes Care 2017; 40: 155–157.
7. Mellor J, Kuznetsov D, Heller S, et al. Risk factors and prediction of hypoglycaemia using the Hypo-RESOLVE cohort: a secondary analysis of pooled data from insulin clinical trials. Diabetologia 2024; 67: 1588–1601.
8. Seaquist ER, Anderson J, Childs B, et al. Hypoglycemia and diabetes: a report of a work group of the American Diabetes Association and the Endocrine Society. Diabetes Care 2013; 36: 1384–1395.

16. Management of Acute Diabetic Complications in Hyperglycaemic States

CHAPTER HIGHLIGHTS:
• Treatment of acute hyperglycaemic states should be conducted in accordance with established standards of care regarding fluid and electrolyte replacement and insulin therapy. Management according to a diabetic ketoacidosis protocol shortens the duration of treatment. [**B**]
• In diabetic ketoacidosis, crystalloids are preferred for the replacement of water deficit in the body, as they have an advantage over colloid solutions. [**B**]
• In acute hyperglycaemic states, continuous intravenous insulin infusion is preferred; the initial dose should be calculated based on the current body weight rather than the blood glucose level. [**C**]
• In acute hyperglycaemic states, particularly in diabetic ketoacidosis, potassium supplementation should be provided with monitoring of its serum concentration. [**B**]
• Administration of bicarbonates is not recommended in diabetic ketoacidosis when pH > 6.9. [**B**]

I. Classification

1. Diabetic ketoacidosis (DKA) – mortality: when treated according to standards < 1%; the risk of death is increased in individuals with recurrent episodes and coexisting hyperosmolality.
2. Hyperglycaemic hyperosmolar state – mortality 5–10%.
3. Lactic acidosis (mortality, according to historical data, approximately 50%; however, currently it largely depends on the experience of the treating centre, the severity of the underlying disease, and the presence of comorbid conditions).

II. Diabetic ketoacidosis (DKA)

1. Causes:

- interruption of insulin therapy or errors in insulin administration,
- delayed diagnosis of type 1 diabetes,
- alcohol abuse, cigarette smoking,
- acute inflammatory conditions (e.g. bacterial, viral, or fungal infections),
- pregnancy,
- acute kidney injury or chronic kidney disease (especially in advanced stages),
- use of flozins in individuals with diabetes requiring insulin therapy and at increased risk of diabetic ketoacidosis,
- others.

In individuals with type 1 diabetes who have significantly reduced insulin doses while using GLP-1 receptor agonists or GIP/GLP-1 receptor agonists, attention should be paid to the potential risk of diabetic ketoacidosis (see Chapter 10).

2. Diagnosis: see Table 16.1.
3. Differential diagnosis:

- starvation ketosis,
- alcoholic ketoacidosis [blood glucose rarely > 250 mg/dl (13.9 mmol/l), bicarbonate concentration usually ≥ 18 mmol/l],
- metabolic acidosis with an anion gap > 20 mEq/l (ethylene glycol, methanol, paraldehyde, or salicylate poisoning),
- lactic acidosis (it should be remembered that in ketoacidosis an increase in blood lactate concentration may occur),
- other comatose states leading to hyperglycaemia and ketosis, or situations in which they coexist with, for example, stroke or uraemic coma.

4. Monitoring of ketoacidosis:

- assessment of blood pressure, heart rate, respiratory rate, and level of consciousness: every 1–2 hours,
- fluid balance: every 1–2 hours,
- body temperature assessment: every 8 hours,
- blood glucose measurement: every 1 hour,
- measurement of serum or plasma sodium and potassium concentrations every 4 hours [corrected serum sodium concentration should be calculated according to the formula: for every 100 mg/dl (5.6 mmol/l) of blood glucose above 100 mg/dl (5.6 mmol/l), 2 mmol/l should be added to the measured serum Na^+ value],
- in the case of hyperkalaemia > 5.5 mmol/l, when potassium is not administered: recheck after 2 hours; with serum potassium < 5.5 mmol/l and potassium supplementation: every 4 hours,
- blood gas analysis: every 4 hours,
- baseline measurement of blood and/or urine ketones.

Table 16.1. Diagnostic criteria with assessment of the severity of diabetic ketoacidosis

Parameters	Diabetic ketoacidosis		
	Mild	Moderate	Severe
Plasma glucose concentration*	≥ 200 mg/dl (11.1 mmol/l)	≥ 200 mg/dl (11.1 mmol/l)	≥ 200 mg/dl (11.1 mmol/l)
Blood pH	7.25–7.30	7.00–7.25	< 7.00
Blood bicarbonate concentration [mEq/l]	15–18	10–15	< 10
Urine ketone bodies**	Present	Present	Present
Ketonemia (β-hydroxybutyrate) [mmol/l]	3–6	3–6	> 6
Serum osmolality [mOsm/kg/H_2O]	Variable	Variable	Variable
Anion gap***	> 10	> 12	> 12
Level of consciousness	Alert	Alert/disorientation	Stupor/coma

**Does not apply to individuals treated with SGLT2 inhibitors (flozins), in which blood glucose may be lower (euglycaemic diabetic ketoacidosis).*
***Method using nitroprusside.*
****Following the formula: Na^+ (mEq/l) – [Cl^- (mEq/l) + HCO_3^- (mEq/l)].*

5. Treatment

In adults with moderate and severe diabetic ketoacidosis (DKA), management according to the following protocol is recommended:

A. Patient hydration

The water deficit (on average 100 ml/kg body weight) should be replaced intravenously over 24–48 hours, under cardiovascular monitoring:

- 1000 ml of 0.9% NaCl during the first hour, followed by:
- 500 ml/hour of 0.9% NaCl for the next 4 hours, followed by:
- 250 ml/hour of 0.9% NaCl until restoration of acid-base balance,
- after blood glucose decreases below 250 mg/dl (13.9 mmol/l), a 5% glucose infusion should be added at a rate of 100 ml/hour; if glucose is added after 24 hours of fluid therapy, the rate of 0.9% NaCl should be reduced to 150 ml/hour,
- in states of increased energy demand (e.g. infection accompanying DKA, hyperthyroidism, pregnancy), administration of 10% glucose instead of 5% is recommended at an infusion rate of 70 ml/hour,
- in patients with body weight < 50 kg, intravenous hydration should follow paediatric recommendations (Figure 23.1),
- in euglycaemic DKA (blood glucose < 200 mg/dl; 11.1 mmol/l), from the beginning of treatment, 5% glucose should be administered in addition to 0.9% NaCl at 100 ml/hour, or 10% glucose at 70 ml/hour in the case of increased energy demand.

B. Reduction of hyperglycaemia

Intravenous insulin therapy

- the initial insulin bolus dose is 0.1 IU/kg body weight; the bolus is administered in adults with severe DKA who have not previously received subcutaneous insulin (no subcutaneous insulin depot),
- this is followed by a continuous intravenous insulin infusion at 0.1 IU/kg body weight/hour, under blood glucose monitoring; in patients who have a subcutaneous insulin depot from prior injections, intravenous insulin therapy should be initiated without a bolus, directly with an infusion of 0.1 IU/kg/hour,
- the infusion rate should be adjusted according to current blood glucose values, monitored hourly,
- the decrease in blood glucose should not exceed 100 mg/dl (5.6 mmol/l) per hour,
- if during the first hour plasma glucose does not decrease by 50–70 mg/dl (2.8–3.9 mmol/l) from baseline, the rate of intravenous insulin infusion should be increased (usually doubled) hourly until a stable glucose decline of 50–70 mg/dl/hour (2.8–3.9 mmol/l/hour) is achieved.

NOTE: In DKA patients previously treated with ultra-long-acting insulin analogues, continuation of basal insulin therapy during DKA treatment is recommended. Basal insulin administration or reconnection of a personal insulin pump should be appropriately timed so that after discontinuation of intravenous insulin infusion, the patient is not exposed to insulin deficiency.

C. Correction of electrolyte disturbances

- the potassium deficit in DKA is approximately 3–5 mmol/kg body weight,
- potassium supplementation should follow the principles below, assuming hydration with isotonic saline.

Serum potassium concentration:
K^+ > 5.5 mmol/l → do not administer KCl
K^+ 5.0–5.5 mmol/l → 5–10 mmol/hour KCl

K^+ 4.0–5.0 mmol/l → 10–15 mmol/hour KCl
K^+ 3.0–4.0 mmol/l → 15–20 mmol/hour KCl
K^+ < 3.0 mmol/l → suspend insulin therapy, administer intravenous KCl at 25 mmol/hour

Potassium supplementation exceeding 15 mmol/hour should be administered via a central venous line or through two peripheral veins.

In severe DKA, potassium replacement should be intravenous and ensure stable normokalaemia. In patients whose clinical condition allows oral intake, potassium deficits may also be corrected orally.

D. Bicarbonates therapy should be considered only if arterial pH < 6.9, administered in small doses (not exceeding 1 mmol/kg body weight). Elevated lactate concentrations during DKA (often mildly increased due to tissue hypoxia) do not constitute an indication for bicarbonate administration.

E. Prophylactic dosing of low-molecular-weight heparin should be considered in patients with severe DKA.

6. Adverse effects of treatment
- hypokalaemia related to insulin therapy and bicarbonate administration,
- hypernatraemia, mainly associated with unjustified administration of sodium bicarbonate (e.g. pulmonary oedema, cerebral oedema; in cerebral oedema, treatment with intravenous mannitol 1–2 g/kg body weight over 20 minutes is recommended),
- hyperglycaemia due to discontinuation of intravenous insulin after improvement without timely initiation of subcutaneous insulin,
- hypoglycaemia caused by excessively intensive insulin therapy,
- hyperchloraemia resulting from excessive administration of normal saline.

7. Complications of DKA:
- hypovolaemic shock,
- acute kidney injury,
- cerebral oedema, more common in children.

8. Specific aspects of managing acute DKA in children are presented in Figure 23.1.

9. Mild uncomplicated DKA

In patients with mild, uncomplicated DKA, as an alternative to intravenous insulin infusion, treatment may include subcutaneous administration of rapid-acting insulin analogues every 1–2 hours, with blood glucose monitoring and continuation of basal insulin therapy, or continuous subcutaneous insulin infusion, with glucose monitoring.

III. Hyperglycaemic hyperosmolar state (HHS)

1. Causes:
- most commonly develops as a result of delayed diagnosis or inadequate treatment of type 2 diabetes,
- in the course of stroke or myocardial infarction,
- after consumption of large amounts of alcohol,
- as a result of the use of certain diuretic drugs,
- in individuals with chronic kidney disease,
- in individuals with psychiatric disorders and concomitant infection symptoms.

2. Diagnosis

Laboratory diagnostic criteria of hyperglycaemic hyperosmolar state:
- blood glucose > 600 mg/dl (> 33.3 mmol/l),
- pH > 7.30,
- serum bicarbonate concentration > 15.0 mmol/l,
- corrected hypernatraemia (calculated according to the formula) ≥ 150 mmol/l,
- serum ketone bodies: absent/trace,
- effective osmolality > 320 mOsm/kg H_2O.

Effective osmolality (mOsm/kg H_2O) =
= 2 × [Na^+ (mmol/l)] + glucose (mmol/l)
{2 × [measured Na (mEq/l)] + [glucose (mg/dl)]/18}

Normal plasma osmolality is 280–300 mOsm/kg H_2O.

3. Differential diagnosis:
- ketoacidotic coma,
- coma in the course of central nervous system diseases,
- uraemic coma,
- coma due to intoxications.

4. Treatment

Principles of treatment are similar to those used in DKA:
- reduction of hyperglycaemia (similar insulin doses as in DKA treatment),
- normalisation of plasma osmolality — gradual reduction of osmolality (not exceeding 3 mOsm/kg H_2O/hour),
- subcutaneous administration of low-molecular-weight heparin,
- correction of water and electrolyte deficits: water loss is significantly greater than in DKA,
- use of hypotonic solutions (0.45% NaCl or an emergency rehydration fluid), and after achieving normal plasma osmolality, 0.9% NaCl administered intravenously under monitoring of the cardiovascular system; the rate of NaCl infusion should be determined according to

serum sodium concentration and plasma osmolality,
- blood glucose monitoring hourly and electrolyte monitoring every 4–6 hours.

IV. Lactic acidosis

1. Causes:
- Type A lactic acidosis develops as a result of cardiogenic shock, severe haemorrhage, septic shock, acute or chronic respiratory failure; this condition may occur in patients with diabetes,
- Type B lactic acidosis occurs due to causes other than hypoxia and is observed in individuals with diabetes, in patients with liver disease, neoplastic diseases, after ingestion of ethanol, biguanides, salicylates, or methanol.

2. Laboratory diagnostic criteria:
- blood glucose moderately elevated, but may be normal,
- decreased blood pH (< 7.30), serum bicarbonate concentration < 10 mmol/l, anion gap > 16 mmol/l,
- lactate concentration > 5 mmol/l,
- serum sodium concentration unchanged (may be decreased in alcohol-dependent individuals),
- usually increased serum potassium concentration.

3. Treatment includes the following measures:
- prevention and treatment of shock (correction of dehydration and hypovolaemia, moderate use of peripheral vasoconstrictor agents),
- prevention and treatment of hypoxaemia and hypoxia,
- reduction of excessive lactic acid production (glucose and insulin infusion under glycaemic control),
- alkalisation with sodium bicarbonate administration
- (requirement: BE × 0.3 × body weight [kg]),
- in justified cases (biochemical and/or clinical indications), renal replacement therapy is required.

REFERENCES

1. Chua HR, Schneider A, Bellomo R. Bicarbonate in diabetic ketoacidosis – a systematic review. Ann Intensive Care 2011; 6: 23. DOI: 10.1186/2110-5820-1-23.
2. Cieluch A, Uruska A, Falkowski B, et al. Nonadherence to potassium replacement protocol leads to prolonged management of diabetic ketoacidosis. Pol Arch Intern Med 2018; 128: 416–420.
3. Edwards K, Uruska A, Duda-Sobczak A, et al. Patient-perceived benefits and risks of off-label use of SGLT2 inhibitors and GLP-1 receptor agonists in type 1 diabetes: a structured qualitative assessment. Ther Adv Endocrinol Metab 2023; 14: 20420188231180987. DOI: 10.1177/20420188231180987.
4. Pasquel FJ, Tsegka K, Wang H, et al. Clinical outcomes in patients with isolated or combined diabetic ketoacidosis and hyperosmolar hyperglycemic state: a retrospective, hospital based cohort study. Diabetes Care 2020; 43: 349–357.
5. Perel P, Roberts I, Ker K. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev 2013; 2: CD000567. DOI: 10.1002/14651858.CD000567.pub7.
6. Thomas M, Harjutsalo V, Feodoroff M, et al. The long-term incidence of hospitalization for ketoacidosis in adults with established T1D – a prospective cohort study. J Clin Endocrinol Metab 2020; 105: 231–241.
7. Umpierrez GE, Davis GM, ElSayed NA, et al. Hyperglycaemic crises in adults with diabetes: a consensus report. Diabetologia 2024; 67: 1455–1479.
8. Umpierrez GE, Jones S, Smiley D, et al. Insulin analogs versus human insulin in the treatment of patients with diabetic keto-acidosis: a randomized controlled trial. Diabetes Care 2009; 32: 1164–1169.
9. Zheng Y, Meng Y, Mei M, et al. The association between GLP-1 receptor agonist and diabetic ketoacidosis in the FDA adverse event reporting system. Nutr Metab Cardiovasc Dis 2022; 32: 504–510.

17. Principles of Diagnosis and Treatment of Individuals with Chronic Coronary Syndrome and Heart Failure Coexisting with Diabetes

CHAPTER HIGHLIGHTS:
• In individuals with diabetes and chronic coronary syndrome (CCS), in the absence of contraindications, acetylsalicylic acid and statins should be used [A], and treatment with drugs blocking the renin–angiotensin–aldosterone system (RAAS) should be considered. [**B**]
• After myocardial infarction, long-term (indefinite) use of a β-blocker is recommended. [**A**]
• After myocardial infarction, drugs with documented cardioprotective effects (SGLT2 inhibitors, GLP-1 receptor agonists) should be added and continued long-term (indefinitely). [**A**]

Coronary artery disease (CAD) is a pathological process involving the formation of atherosclerotic plaques in the epicardial coronary arteries, which may, but do not have to, lead to their narrowing and/or occlusion. It is a chronic and most often progressive disease; therefore, it is regarded as serious even during apparently silent periods. The dynamic nature of CAD is associated with the diversity of its clinical manifestations, which can be broadly divided into acute coronary syndromes (ACS) and chronic coronary syndromes (CCS).

When CCS is suspected or diagnosed, the following clinical situations are most commonly encountered:

- individuals with suspected CCS and "stable" angina and/or dyspnoea,
- individuals with newly diagnosed heart failure or left ventricular dysfunction and suspected CCS,
- asymptomatic individuals or those with stable symptoms within the first year after diagnosis of the disease or after revascularisation,
- individuals with angina symptoms and suspected vasospastic or microvascular disease,
- asymptomatic individuals in whom CCS is diagnosed during screening examinations.

All of the above situations are classified as CCS; however, each of them is associated with a different risk of future cardiovascular events, which may change over time.

I. Clinical course

Differences in the clinical course of chronic coronary syndromes in individuals with diabetes indicate the need for follow-up assessments evaluating the presence of risk factors for this disease to be performed at least once a year.

II. Indications for investigations aimed at diagnosing chronic coronary syndromes

Indications for performing diagnostic, functional and anatomical investigations aimed at the diagnosis of CCS and risk stratification in individuals with diabetes (cardiology consultation):

1. Presence of typical or atypical cardiovascular symptoms.
2. Abnormal resting ECG.
3. Coexistence of atherosclerotic lesions in peripheral arteries, including the carotid arteries.
4. Planned initiation of intensive physical exercise in individuals aged > 35 years who have previously led a sedentary lifestyle.
5. Type 1 diabetes of duration > 15 years.
6. Presence, in addition to diabetes, of two or more CCS risk factors:

- abnormal lipid parameters,
- arterial hypertension,
- tobacco smoking,
- family history of premature atherosclerosis,
- presence of albuminuria,
- presence of autonomic neuropathy (Figure 17.1).

III. Treatment of individuals with diabetes and chronic coronary syndrome according to the new terminology of the European Society of Cardiology

1. Implementation of a health-promoting lifestyle (see Chapter 6).
2. Antihyperglycaemic treatment aimed at achieving therapeutic targets (see Chapter 4).
3. Reduction or normalisation of coronary artery disease risk factors:

- normalisation of arterial blood pressure (see Chapter 13),
- treatment of lipid disorders (see Chapter 14).

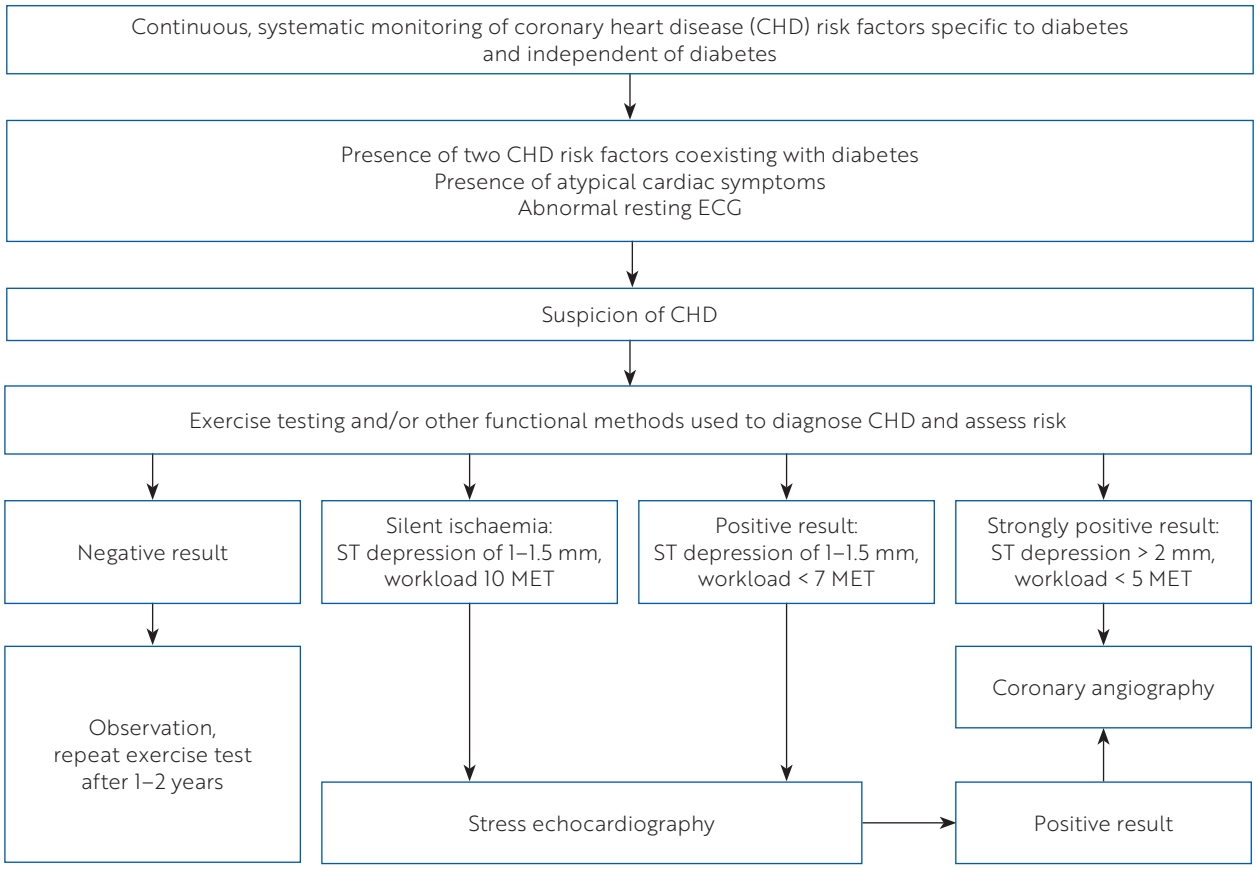


Figure 17.1. Diagnostic algorithm for confirmation of the diagnosis and risk stratification of ischaemic heart disease in individuals with diabetes in settings with limited access to modern diagnostic methods

CHD – coronary heart disease

4. Specific aspects of pharmacotherapy for CCS in diabetes:

- antiplatelet therapy – acetylsalicylic acid (ASA); ASA should also be used in individuals with type 2 diabetes and with type 1 diabetes aged > 40 years who are at increased risk of cardiovascular events (> 5% 10-year risk of developing ischaemic heart disease); the effectiveness of ASA in primary prevention has not been confirmed in individuals with diabetes at low cardiovascular risk:
  - » the recommended dose of ASA is 75–100 mg/day,
  - » in the presence of contraindications to ASA, the use of clopidogrel at a dose of 75 mg/day may be appropriate and justified,
  - » after percutaneous coronary intervention (PCI), the use of ASA at a dose of 75–100 mg/day and clopidogrel at a dose of 75 mg/day for 1–6 months is recommended; in cases of increased bleeding risk, shortening the duration of treatment to 1–3 months is indicated; in situations of high risk of coronary complications, such as a high risk related to planned stent implantation (e.g. suboptimal stent deployment or other procedural situations associated with a high risk of stent thrombosis, complex anatomical stenosis of the left main coronary artery, or implantation of stents in multiple vessels), or when dual antiplatelet therapy (DAPT) cannot be used due to ASA intolerance, prasugrel or ticagrelor may be considered as the second agent instead of clopidogrel,
- use of cardioselective β-blockers or multifunctional β-blockers with α1- and β1-receptor blockade,
- drugs blocking the renin–angiotensin–aldosterone system: ACE inhibitors or ARBs.

In cases of ineffective pharmacotherapy, revascularisation therapy should be considered.

Exercise testing and other functional methods are used in order to confirm the diagnosis, document myocardial ischaemia, stratify risk, and facilitate the choice of treatment methods and assessment of their effectiveness. Due to its continued wide availability, the exercise test is the most fre-

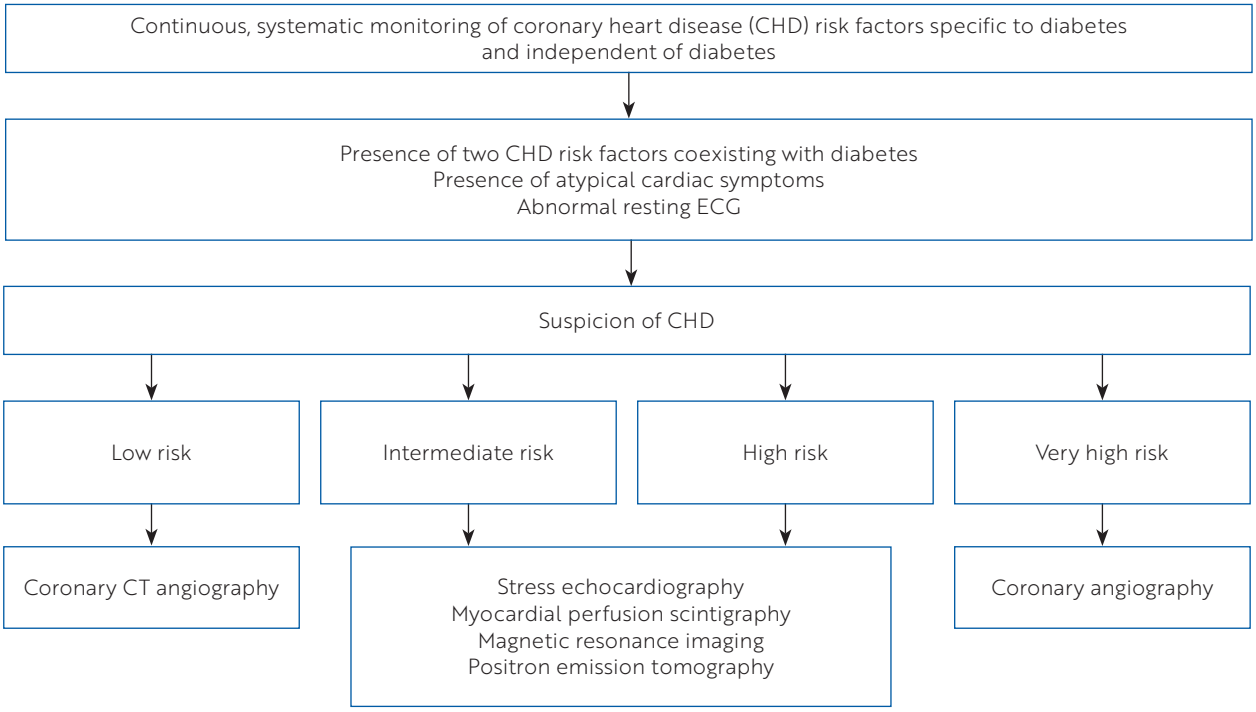


Figure 17.2. Diagnostic algorithm for confirmation of the diagnosis and risk stratification of ischaemic heart disease in individuals with diabetes with access to modern diagnostic methods

CHD – coronary heart disease

quently performed investigation; however, its sensitivity and specificity in detecting ischaemia are limited, particularly in women. It is acceptable in settings with limited access to modern diagnostic methods (Figure 17.1) and is more useful for excluding significant coronary artery stenoses. Other functional methods include stress echocardiography, myocardial perfusion scintigraphy, magnetic resonance imaging, and positron emission tomography. An algorithm of management in the presence of access to modern diagnostic methods is presented in Figure 17.2. Among anatomical methods, invasive coronary angiography remains the gold standard, while coronary CT angiography may also be useful. It should be emphasised that individuals with diabetes are most often classified as being at high or very high risk of coronary artery disease. In the high-risk group, functional testing is recommended as the first-line approach, whereas in individuals at very high risk, coronary angiography constitutes the basis of diagnostic assessment already at the initial stage. Coronary CT angiography is characterised by a high negative predictive value and is therefore more useful for excluding significant coronary artery stenoses. Its use is not recommended in individuals at high risk, as it represents an unnecessary burden related to contrast administration and exposure to ionising radiation.

REFERENCES

1. Baigent C, Blackwell L, Collins R, et al. Aspirin in the primary and secondary prevention of vascular disease: collaborative meta-analysis of individual participant data from randomised trials. Lancet 2009; 373: 1849–1860.
2. Gaede P, Lund-Andersen H, Parving HH, et al. Effect of a multifactorial intervention on mortality in type 2 diabetes. N Engl J Med 2008; 358: 580–591.
3. Kearney PM, Blackwell L, Collins R, et al. Efficacy of cholesterol-lowering therapy in 18,686 people with diabetes in 14 randomised trials of statins: a meta-analysis. Lancet 2008; 371: 117–125.
4. Knuuti J, Wijns W, Saraste A, et al. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes. Eur Heart J 2020; 41: 407–477.
5. Neumann FJ, Sousa-Uva M, Ahlsson A, et al. 2018 ESC/EACTS guidelines on myocardial revascularization. Eur Heart J 2019; 40: 87–165.
6. Shepherd J, Barter P, Carmena R, et al. Effect of lowering LDL cholesterol substantially below currently recommended levels in patients with coronary heart disease and diabetes: the Treating to New Targets (TNT) study. Diabetes Care 2006; 29: 1220–1226.

17.1. Acute Coronary Syndrome in Individuals with Diabetes – Antihyperglycaemic Treatment

CHAPTER HIGHLIGHTS:

- On hospital admission of an individual with acute coronary syndrome, blood glucose should be measured, and in individuals with diabetes, if a recent result is not available, HbA_{1c} should also be determined. [**A**]
- During the first 24 hours of acute coronary syndrome, administration of an intravenous insulin infusion with blood glucose monitoring is recommended, with target glycaemic values of 100–180 mg/dl. [**C**]

In acute coronary syndrome, in states of relative hyperglycaemia, normalisation of blood glucose using an intravenous insulin infusion is recommended. Relative hyperglycaemia is defined as blood glucose levels > 140 mg/dl (7.8 mmol/l) in individuals with previously diagnosed diabetes or > 180 mg/dl (10.0 mmol/l) in individuals without previously diagnosed diabetes. Intravenous administration of insulin is the only method that allows rapid normalisation of glycaemia and improvement of prognosis after acute coronary syndrome. Treatment of ischaemic heart disease in individuals with disturbances of carbohydrate metabolism should, whenever possible, be carried out with the involvement of a diabetologist.

I. First 24 hours of acute coronary syndrome

1. Oral antihyperglycaemic agents should be discontinued.
2. In every case of acute coronary syndrome, blood glucose should be measured on admission.
3. When blood glucose exceeds 140 mg/dl (7.8 mmol/l) in individuals with previously diagnosed diabetes or 180 mg/dl (10.0 mmol/l) in individuals without previously diagnosed diabetes, an intravenous insulin infusion should be initiated at a rate specified in Table 17.1.1. The recommended frequency of blood glucose monitoring during the day is every hour, and after stabilisation every 2 hours. Blood glucose should be maintained within the range of 100–180 mg/dl (5.6–10.0 mmol/l) by appropriate adjustment of the insulin infusion.
4. During insulin infusion, serum potassium concentration should be monitored.
   If blood glucose exceeds 180 mg/dl (10.0 mmol/l), the intravenous glucose infusion should be temporarily discontinued and restarted after blood glucose has decreased to 180 mg/dl (10.0 mmol/l), with a simultaneous increase in the rate of intravenous insulin infusion.
5. In the event of food intake, additional short-acting or rapid-acting insulin should be administered intravenously.
6. In the case of diabetic ketoacidosis, management should follow the recommendations for the treatment of ketoacidosis (see Chapter 16).

II. From the second day of acute coronary syndrome until the end of hospitalisation

1. Antihyperglycaemic treatment must ensure blood glucose values within the range of 100–180 mg/dl (5.6–10.0 mmol/l) throughout the entire day. Therefore, treatment must be individualised and preferably conducted in cooperation with a diabetologist.
2. In individuals without features of acidosis, with disturbances of carbohydrate metabolism diagnosed on the first day of acute coronary syndrome, or previously well controlled on metformin, adequate metabolic control during this period may be achieved with an appropriate diet (see Chapter 6). In all other cases, insulin therapy using a multiple daily injection regimen should be implemented according to previously described principles (see Chapter 12).
3. In individuals with type 2 diabetes who are overweight or obese, immediately before completion of hospitalisation, even as early as the third day after intervention, metformin, an SGLT2 inhibitor and/or a GLP-1 receptor agonist may additionally be introduced, provided there are no contraindications.

III. After the hospitalisation

In every individual with type 2 diabetes after acute coronary syndrome, an SGLT2 inhibitor and/or a GLP-1 receptor agonist and/or metformin should be initiated, unless contraindications or intolerance are present.

In individuals with type 2 diabetes who have achieved good metabolic control on the day of discharge, with a total daily insulin requirement

Table 17.1.1 Approximate insulin infusion rate according to blood glucose concentration.

Blood glucose	10% glucose solution [ml/hr]	Insulin [units/hr]
< 100 mg/dl < 5.5 mmol/l	50	Stop the infusion for 15–30 minutes
100–140 mg/dl 5.5–7.8 mmol/l	50	0.5–1.0
140–180 mg/dl 6.7–10.0 mmol/l	50	1.0–2.0
180–250 mg/dl 10–13.9 mmol/l	Withhold the infusion until blood glucose decreases. < 180 mg/dl (10.0 mmol/l)/hour, then 50	2.0–4.0
250–300 mg/dl 13.9–17.4 mmol/l	Withhold the infusion until blood glucose decreases. < 180 mg/dl (10.0 mmol/l)/hour, then 50	4.0–6.0

not exceeding 30 units, a return to the therapy used prior to the occurrence of acute coronary syndrome may be considered. In individuals in whom diabetes was diagnosed during hospitalisation and who achieved good metabolic control on the day of discharge, with a total daily insulin requirement not exceeding 30 units, but who are characterised by overweight or obesity, oral therapy and/or injectable GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists with beneficial effects on body weight may be used. If adequate metabolic control of diabetes cannot be achieved or the total daily insulin requirement exceeds 30 units, insulin therapy should be continued and the possibility of combination with oral therapy and/or injectable GLP-1 receptor agonists or GIP/GLP-1 receptor agonists should be considered. Every individual with disturbances of carbohydrate metabolism after acute coronary syndrome should be urgently referred to a diabetologist.

NOTE 1: In every individual with acute coronary syndrome, apart from those with previously diagnosed diabetes, HbA_{1c} should be measured before discharge from hospital, or an oral glucose tolerance test (OGTT) should be performed after discharge (Chapter 1, section III, Table 17.1.1). If impaired glucose tolerance or diabetes is diagnosed, a diabetology consultation is indicated.

NOTE 2: Before planned coronary angiography performed for diagnostic or therapeutic purposes, metformin should be discontinued at least 48 hours prior to the procedure. Metformin may be restarted 24 hours after coronary angiography.

NOTE 3: Results from randomised studies indicate an additional cardioprotective effect of SGLT2 inhibitors and GLP-1 receptor agonists. Their addition to therapy should be considered in individuals at high or very high cardiovascular risk.

REFERENCES

1. Kristensen SL. Cardiovascular, mortality, and kidney outcomes with GLP-1 receptor agonists in patients with type 2 diabetes: a systematic review and meta-analysis of cardiovascular outcome trials. Lancet Diabetes Endocrinol 2019; 7: 776–785.
2. McGuire DK, Shih WJ, Cosentino F, et al. Association of SGLT2 inhibitors with cardiovascular and kidney outcomes in patients with type 2 diabetes: a meta-analysis. JAMA Cardiol 2021; 6: 148–158.
3. NICE-SUGAR Study Investigators; Finfer S, Chittock DR, Yu-Shuo Su S, et al. Intensive versus conventional glucose control in critically ill patients. N Engl J Med 2009; 360: 1283–1297.
4. Ritsinger V, Malmberg K, Mårtensson A, et al. Intensified insulin-based glycaemic control after myocardial infarction: mortality during 20 year follow-up of the randomised Diabetes Mellitus Insulin Glucose Infusion in Acute Myocardial Infarction (DIGAMI 1) trial. Lancet Diabetes Endocrinol 2014; 2: 627–633.
5. Umpierrez GE, Reyes D, Smiley D, et al. Hospital discharge algorithm based on admission HbA1c for the management of patients with type 2 diabetes. Diabetes Care 2014; 37: 2934–2939.

17.2. Chronic Heart Failure

I. Introduction

Heart failure (HF) is a clinical syndrome manifested by several main symptoms reported by individuals (e.g. dyspnoea, fatigue), which may be accompanied by physical signs (e.g. signs of elevated jugular venous pressure, pulmonary crackles, and peripheral oedema). This syndrome is caused by structural and/or functional abnormalities of the heart that result in increased intracardiac pressures and/or inadequate cardiac output at rest and/or during exertion.

II. Classification of heart failure according to left ventricular ejection fraction

Chronic HF is classified according to the measured left ventricular ejection fraction (LVEF). The European Society of Cardiology has introduced the following HF classification:

- HF with reduced LVEF is defined as ≤ 40%, i.e., significant impairment of left ventricular (LV) systolic function – this condition is referred to as HFrEF,
- HF with mildly reduced LVEF (41–49%) – this condition is referred to as HFmEF,
- HF with preserved LVEF ≥ 50% – HFpEF; in individuals with this form, symptoms and signs of HF are present, abnormalities in heart structure and/or function are identified, and/or elevated levels of natriuretic peptides are observed.

III. Main principles of heart failure diagnostics

In the diagnostic work-up of individuals with HF, in addition to clinical history and physical examination, performance of a standard 12-lead ECG and measurement of natriuretic peptide concentrations (BNP or NT-proBNP) are recommended. In cases of strong clinical suspicion of HF or lack of availability of natriuretic peptide testing, echocardiographic examination is indicated. When the diagnosis is confirmed, echocardiography constitutes the basis for classification into individual HF phenotypes.

A normal ECG tracing makes the diagnosis of HF unlikely. ECG may reveal abnormalities such as atrial fibrillation (AF), Q waves, left ventricular hypertrophy (LVH), or widened QRS complexes, which increase the likelihood of HF and may also provide therapeutic guidance.

Normal concentrations of natriuretic peptides make the diagnosis of HF unlikely.

Echocardiography is recommended as the primary investigation for assessment of cardiac function. In addition to determining LVEF, echocardiography also provides information on other parameters, such as cardiac chamber size, eccentric or concentric LVH, regional wall motion abnormalities (which may suggest coronary artery disease, takotsubo cardiomyopathy, or myocarditis), right ventricular function, pulmonary hypertension, valvular function, and indices of diastolic function.

Furthermore, in the diagnostic evaluation of HF, basic laboratory blood tests are recommended, including serum creatinine and electrolyte concentrations, complete blood count, and liver and thyroid function tests, in order to differentiate HF from other conditions, as well as to obtain prognostic information and guidance that may help direct treatment.

Chest X-ray is recommended to assess other potential causes of dyspnoea (e.g. pulmonary disease). It may also provide findings supportive of HF, such as pulmonary congestion or cardiomegaly.

At a subsequent stage, determination of HF aetiology and initiation of treatment, including causal therapy, are recommended.

IV. General principles of pharmacotherapy for heart failure with reduced ejection fraction

It has been demonstrated that modulation of the renin–angiotensin–aldosterone system and the sympathetic nervous system using angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor–neprilysin inhibitors (ARNIs), β-blockers, and mineralocorticoid receptor antagonists (MRAs) increase survival, reduce the rate of hospitalisation due to HF, and alleviate symptoms in individuals with HFrEF. If not contraindicated or poorly tolerated, these drugs constitute the foundation of pharmacotherapy in HFrEF and should be used as the cornerstone of therapy.

An important prognostic factor is that doses of ACEIs or ARNIs and β-blockers should be systematically uptitrated to the maximum tolerated doses. ARNIs can and should be considered as first-line treatment instead of ACEIs.

SGLT2 inhibitors, i.e. dapagliflozin and empagliflozin, when added to treatment with an ACEI/ARNI, a β-blocker, and an MRA, reduce the risk of cardiovascular death and worsening HF in individuals with HFrEF. Provided these agents are not contraindicated or poorly tolerated, their use is recommended in all individuals with HFrEF already treated with an ACEI/ARNI, a β-blocker, and an MRA, regardless of the presence or absence of diabetes.

Diuretics, with loop diuretics being preferred, are recommended to reduce subjective and/or objective symptoms of congestion in individuals with HFrEF.

Angiotensin receptor blockers (ARBs) are currently recommended in individuals who do not tolerate ACEIs or ARNIs. The If-channel inhibitor ivabradine reduces heart rate and its use should be considered in individuals with HFrEF in sinus rhythm with a heart rate ≥ 75 beats per minute and LVEF ≤ 35%. Before considering ivabradine therapy, every effort should be made to initiate β-blocker treatment and to uptitrate its dose to the guideline-recommended or maximum tolerated dose.

V. Implantable cardioverter-defibrillator in the prevention of sudden death in individuals with HFrEF

An important component of therapy in individuals with HFrEF is the use of implantable cardioverter-defibrillators (ICDs) for the primary prevention of sudden cardiac death due to malignant ventricular arrhythmias. In the population in whom HF has an ischaemic aetiology, **ICD implantation is recommended** to reduce the risk of sudden death and all-cause mortality in individuals with symptomatic HF (NYHA class II–III) and LVEF ≤ 35% despite ≥ 3 months of optimal conservative (pharmacological) treatment, provided that survival longer than one year in good functional status can be expected.

In individuals after myocardial infarction, eligibility for ICD implantation should be assessed no earlier than 40 days after the infarction. In contrast, in individuals with HF of non-ischaemic aetiology, ICD therapy has a lower class of recommendation; **implantation should be considered** to reduce the risk of sudden death and all-cause mortality in individuals with symptomatic HF (NYHA class II–III) of non-ischaemic origin and LVEF ≤ 35% despite ≥ 3 months of optimal conservative treatment, provided that survival longer than one year in good functional status can be expected.

VI. Cardiac resynchronisation therapy in individuals with HFrEF (CRT/CRT-D)

Cardiac resynchronisation therapy (CRT), when used in appropriately selected individuals, reduces the need for hospitalisation due to progression of HF symptoms, improves cardiac function and quality of life, and, most importantly, reduces all-cause mortality. This form of pacing is used in individuals with HF in whom delayed ventricular activation occurs, resulting in delayed contraction of the left ventricle relative to the right ventricle, which leads to a significant reduction in left ventricular stroke volume. This is of particular importance in individuals with markedly reduced LVEF, in whom left ventricular stroke volume is already impaired at baseline. A clinical and electrocardiographic marker of delayed left ventricular activation and contraction relative to the right ventricle is prolongation of the QRS complex duration on ECG, particularly the presence of left bundle branch block (LBBB) morphology.

Current guidelines specify which individuals with HFrEF and LVEF ≤ 35% should be considered for CRT. A prerequisite for this form of therapy is prior use of causal treatment and optimal pharmacotherapy for a minimum of 3 months.

Assessment of indications for CRT with or without defibrillator function (CRT-D vs CRT-P) is also essential. Indications for CRT apply to individuals with significantly reduced LVEF, who therefore also meet criteria for ICD therapy. The final decision regarding the type of device used, CRT-P or CRT-D, is made by the cardiology centre ultimately qualifying the individual with HFrEF for this form of therapy.

Current indications for CRT include:

1. CRT is recommended in symptomatic individuals with HF, sinus rhythm, QRS duration ≥ 150 ms, and LBBB morphology.
2. CRT should be considered in symptomatic individuals with HF, sinus rhythm, QRS duration ≥ 150 ms, and non-LBBB morphology.
3. CRT should be considered in symptomatic individuals with HF, sinus rhythm, QRS duration 130–149 ms, and LBBB morphology.
4. CRT may be considered in symptomatic individuals with HF, sinus rhythm, QRS duration 130–149 ms, and non-LBBB morphology.

Individuals with HFrEF after CRT implantation require close cardiology follow-up conducted in specialised cardiology centres.

VII. Chronic heart failure in individuals with diabetes

Individuals with diabetes are at particularly high risk of developing HF. Diabetes predisposes to HF through associated macroangiopathic complications, including atherosclerosis of the coronary arteries.

Optimal pharmacotherapy for HF in individuals with and without diabetes is similar. However, antihyperglycaemic agents differ in their effects in individuals with HF; therefore, preference should be given to agents that are both safe and reduce the incidence of HF-related events.

VIII. Heart failure with preserved left ventricular ejection fraction

HFpEF differs from HFrEF in many respects. Individuals with HFpEF are often older, the proportion of women is higher, and multiple comorbidities are more common, including atrial fibrillation, chronic kidney disease, and other non-cardiovascular conditions.

The criteria and principles for the diagnosis of this HF phenotype have been presented above. The basic diagnostic criteria for HFpEF include:

1. Symptoms and signs of HF.
2. LVEF ≥ 50%.
3. Objective evidence of structural and/or functional cardiac abnormalities consistent with left ventricular diastolic dysfunction and/or increased left ventricular filling pressures, including elevated natriuretic peptide concentrations.

Until 2021, no treatment had been shown to unequivocally reduce mortality and morbidity in this population. Following publication of the 2021 ESC guidelines on the diagnosis and treatment of HF, studies evaluating the effects of dapagliflozin and empagliflozin on prognosis in individuals with HFpEF were published, demonstrating reductions in hospitalisation for HF and cardiovascular mortality. These studies showed a clear beneficial effect of these agents on outcomes in individuals with HFpEF regardless of the presence of diabetes (DELIVER and EMPEROR-Preserved). The results were incorporated into the 2023 update of the European Society of Cardiology guidelines, changing therapeutic principles in the HFpEF population.

Dapagliflozin and empagliflozin are recommended and should be used (if no contraindications are present) in the treatment of individuals with HFpEF. Equally strong recommendations apply to the management of comorbid conditions that predispose to this HF phenotype. This includes modulation of the renin–angiotensin–aldosterone system and the sympathetic nervous system using ACE inhibitors or ARNIs, β-blockers, and mineralocorticoid receptor antagonists.

Diuretics, with loop diuretics being preferred, should be used in cases associated with fluid retention.

REFERENCES

1. McDonagh TA, Metra M, Adamo M, et al. Wytyczne ESC 2021 dotyczące diagnostyki i leczenia ostrej i przewlekłej niewydolności serca. Zeszyty Edukacyjne. Kardiol Pol 2022; 80, Suppl 1.
2. McDonagh TA, Metra M, Adamo M, et al.; ESC Scientific Document Group. 2023 Focused Update of the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J 2023; 44: 3627–3639.

18. Stroke in Individuals with Diabetes

CHAPTER HIGHLIGHTS
• Hyperglycaemia identified on hospital admission in the acute phase of stroke is associated with higher mortality, a more severe course of stroke, and greater neurological deficits in both individuals with and without diabetes. [**A**]
• Interventional studies have not provided evidence that maintaining normoglycaemia in the acute phase of stroke using intravenous insulin therapy improves prognosis. Such therapy is, however, associated with an increased risk of hypoglycaemia. [**A**]
• Current guidelines regarding the correction of hyperglycaemia during stroke are based solely on expert recommendations/opinions. [**E**]
• GLP-1 receptor agonists reduce the risk of stroke in individuals with type 2 diabetes and should be considered from the very beginning of treatment, irrespective of metformin use and HbA_{1c} levels. They are also recommended for secondary prevention of stroke in individuals with type 2 diabetes. [**A**]

Diabetes is a strong risk factor for stroke, both ischaemic and haemorrhagic. The risk of ischaemic stroke in individuals with diabetes is more than twofold higher, while the risk of haemorrhagic stroke is approximately 1.5-fold higher. Diabetes is present in 28% of individuals with stroke. In the case of ischaemic stroke, this proportion is 33%, while in haemorrhagic stroke it is 26%. Elevated blood glucose levels are observed in more than 60% of individuals hospitalised due to acute stroke. In 20% of cases, hyperglycaemia occurs in individuals with previously diagnosed diabetes; 16–24% of cases involve individuals in whom diabetes had not been previously diagnosed, while in the remaining cases hyperglycaemia is transient (stress-related).

Hyperglycaemia identified in the acute phase of stroke is an unfavourable prognostic factor in individuals both with and without diabetes. Its presence is associated with a higher risk of a larger ischaemic lesion and haemorrhagic transformation, a more severe clinical course, and poorer prognosis (lower functional independence and higher early and late mortality). It remains unclear whether hyperglycaemia observed in individuals with stroke is a cause of a more severe course and worse prognosis, or rather a consequence of a larger, more severe stroke lesion.

The limited number of randomised interventional studies conducted in the acute phase of stroke (up to 72 hours) do not provide evidence that maintaining normoglycaemia achieved with intravenous insulin therapy reduces mortality or improves neurological deficit. In individuals with acute stroke, hyperglycaemia should be avoided, while prioritising the avoidance of hypoglycaemia. This justifies maintaining blood glucose concentrations in the range of 140–180 mg/dl (7.8–10.0 mmol/l). Blood glucose levels should be closely monitored, and hypoglycaemia should be strictly avoided. Target glycaemic values in the acute phase of stroke are therefore similar to those recommended in other severe acute conditions.

Insulin should be administered intravenously in 0.9% sodium chloride solution using a syringe pump, under strict glycaemic monitoring. The rate of insulin infusion should be adjusted according to blood glucose values measured at the bedside every hour or by means of continuous glucose monitoring (CGM), and after stable values are achieved, every 2 hours. An approximate scheme for adjusting the rate of intravenous insulin infusion according to blood glucose levels is presented in Table 26.1. During insulin infusion, serum potassium concentration should be monitored 2–3 times per day.

Administration of insulin in the form of an intravenous GIK (glucose–insulin–potassium) infusion is not recommended. In the first days after stroke, as well as in individuals who remain unconscious for a prolonged period, insulin should not be administered subcutaneously.

In stroke units, a defined algorithm for dosing insulin administered via intravenous infusion should be in place, taking into account changes in infusion rate according to blood glucose values. Medical and nursing staff should be trained in the management of hyperglycaemia.

When the individual's clinical condition improves and oral intake is resumed, intravenous insulin infusion should be discontinued and subcutaneous insulin therapy initiated. Discontinuation of intravenous insulin infusion should be preceded by subcutaneous administration of short-acting insulin or a rapid-acting analogue approximately one hour before stopping the infusion. The recommended subcutaneous insulin regimen consists of short-acting insulin or a rapid-acting analogue

administered before meals, together with a long-acting insulin administered once or twice daily. In some cases, administration of short-acting or rapid-acting insulin alone before meals is sufficient. Pre-meal insulin doses should be based on blood glucose measurements performed immediately before food intake.

Given the high likelihood of previously unrecognised diabetes in individuals with acute stroke, diagnostic evaluation for diabetes should be performed after stabilisation of the clinical condition in those without a prior diagnosis.

Recommendations regarding the management of arterial blood pressure and other aspects of stroke care are the same as in individuals without diabetes, as there are no data indicating benefits from different or specific approaches in individuals with diabetes.

Secondary prevention after stroke should follow generally accepted principles.

Prevention of stroke in individuals with diabetes

The risk of both first and recurrent stroke in individuals with diabetes can be significantly reduced by applying a multifactorial approach to modifiable risk factors, including lifestyle modification, correction of hyperglycaemia, treatment of arterial hypertension, and management of lipid disorders. A meta-analysis of randomised clinical trials involving GLP-1 receptor agonists indicates a beneficial effect of this drug class in reducing cardiovascular risk, including a reduction in the risk of both non-fatal and fatal stroke. GLP-1 receptor agonists, as agents that reduce the risk of cardiovascular events in individuals with type 2 diabetes and increased cardiovascular risk, should be considered from the very beginning of treatment, regardless of metformin use and HbA_{1c} levels. These agents are also recommended in individuals with type 2 diabetes for secondary prevention of stroke.

REFERENCES

1. Bellolio MF, Gilmore RM, Ganti L. Insulin for glycaemic control in acute ischaemic stroke. Cochrane Database Syst Rev 2014; 1: CD005346. DOI: 10.1002/14651858.CD005346.pub4.
2. GBD 2021 Stroke Risk Factor Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990-2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol 2024; 23: 973–1003.
3. Johnston KC, Bruno A, Pauls Q, et al.; Neurological Emergencies Treatment Trials Network and the SHINE Trial Investigators. Intensive vs standard treatment of hyperglycemia and functional outcome in patients with acute ischemic stroke: the SHINE randomized clinical trial. JAMA 2019; 322: 326–335.
4. Kleindorfer DO, Towfighi A, Chaturvedi S, et al. 2021 Guideline for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline from the American Heart Association/American Stroke Association. Stroke 2021; 52: e364–e467. DOI: 10.1161/STR.0000000000000375.
5. Lau LH, Lew J, Borschmann K, et al. Prevalence of diabetes and its effects on stroke outcomes: a meta-analysis and literature review. J Diabetes Investig 2019; 10: 780–792.
6. Sattar N, Lee MMY, Kristensen SL, et al. Cardiovascular, mortality, and kidney outcomes with GLP-1 receptor agonists in patients with type 2 diabetes: a systematic review and meta-analysis of randomised trials. Lancet Diabetes Endocrinol 2021; 9: 653–662.

19. Diabetic Kidney Disease: Prevention, Diagnosis, and Treatment

CHAPTER HIGHLIGHTS:
• Screening for increased urinary albumin excretion should be performed once a year in individuals with type 1 diabetes from the 5th year of disease duration, in individuals with type 2 diabetes from the time of diagnosis, and in all individuals with diabetes and coexisting arterial hypertension. [**B**]
• In order to reduce the risk of developing diabetic kidney disease and/or to slow its progression, optimisation of glycaemic control, blood pressure control and lipid management is required. [**A**]
• In the presence of increased urinary albumin excretion, treatment with angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) should be used, as these agents reduce the risk of progression of diabetic kidney disease (taking into account contraindications to their use). [**A**]
• SGLT2 inhibitors, GLP-1 receptor agonists and non-steroidal mineralocorticoid receptor antagonists (ns-MRAs) reduce the risk of progression of chronic kidney disease and cardiovascular complications in individuals with type 2 diabetes and chronic kidney disease. [**A**]
• When ACEIs, ARBs, ns-MRAs and/or diuretics are used, serum creatinine, sodium and potassium concentrations should be monitored. [**E**]

In individuals with diabetes, in order to detect diabetic kidney disease or assess its stage, urinary albumin excretion, serum creatinine concentration, and the estimated glomerular filtration rate (eGFR) should be determined.

Albuminuria and eGFR are independent predictors of cardiovascular and renal risk in individuals with diabetes. A reduction in eGFR without preceding or concomitant albuminuria occurs in approximately half of individuals with diabetes, regardless of diabetes type.

I. Screening for albuminuria

Screening should be performed once a year in:

- individuals with type 1 diabetes from the 5th year of disease duration,
- individuals with type 2 diabetes from the time of diagnosis.

Assessment of urinary albumin excretion should be based on determination of the albumin-to-creatinine ratio (ACR) using quantitative measurements obtained from a single urine sample, preferably a morning sample (for interpretation of results see Table 19.1). A diagnosis of increased urinary albumin excretion requires two positive ACR results.

II. Serum creatinine concentration

In individuals with diabetes, serum creatinine concentration should be measured at least once a year, irrespective of ACR values. Serum creatinine concentration should be used to calculate eGFR.

III. Glomerular filtration rate (eGFR)

To determine the estimated glomerular filtration rate (eGFR), the CKD-EPI formula should be used.

$$eGFR = 141 \times \min(Scr/k, 1)^{a} \times \max(Scr/k, 1)^{-1.209} \times \times 0.993^{Age} \times 1.018 \text{ [for women]}$$

where: Scr – serum creatinine concentration, k – 0.7 for women and 0.9 for men, a = –0.329 for women and –0.411 for men, min – the minimum of Scr/k or 1, max – the maximum of Scr/k or 1.

If use of the CKD-EPI equation is not possible, efforts should be made to monitor trends in eGFR using other estimation methods (e.g. the MDRD equation).

IV. Chronic kidney disease

The stages of chronic kidney disease are defined in Table 19.2.

V. Nephrology consultation

A nephrology consultation should be considered:

- when eGFR decreases to < 60 mL/min/1.73 m^2 and non-diabetic kidney disease is suspected,
- in the case of a rapid decline in eGFR (with a cut-off value defined as ≥ 5 mL/min/1.73 m^2/year),
- in the situations described in Table 19.3.

VI. Preventive recommendations

1. In order to reduce the risk of developing diabetic kidney disease and/or to slow its progression, optimisation of glycaemic control,

Table 19.1 Definition of abnormal urinary albumin excretion*.

Category	AER [mg/day]	ACR (spot urine sample) [mg/day or mg/g of creatinine]*,**
A1: normal albuminuria or mildly increased albuminuria	< 30	< 30
A2: moderately increased albuminuria	30–299	30–299
A3: overt proteinuria	≥ 300	≥ 300

ACR – albumin-to-creatinine ratio, AER – albumin excretion rate

**The amount of albumin excreted in urine expressed per gram of creatinine approximately corresponds to daily albuminuria, allowing avoidance of inaccuracies associated with 24-hour urine collection.*

***If ACR is reported by the laboratory in mg/mmol creatinine, the result should be multiplied by 8.85 to obtain the value in mg/g creatinine.*

Table 19.2 Stages of chronic kidney disease

Category	Description	eGFR [ml/min/1.73 m²]
G1	Kidney damage* with normal or increased eGFR	≥ 90
G2	Kidney damage* with mildly reduced eGFR	60–89
G3a	Moderately reduced eGFR	45–59
G3b	Moderately to severely reduced eGFR	30–44
G4	Severely reduced eGFR	15–29
G5	End-stage kidney disease	< 15

**Kidney damage is defined as the presence of abnormalities in biochemical tests and/or urine sediment, and/or abnormal markers of kidney damage in blood tests, and/or abnormalities detected on imaging of the kidneys or urinary tract, persisting for longer than 3 months.*

Tabela 19.3. Ryzyko progresji przewlekłej choroby nerek, częstotliwość wizyt i konieczność konsultacji nefrologicznej według eGFR i albuminurii [3]

	A1: normal or mildly increased albuminuria < 30 mg/g < 3 mg/mmol	A2: moderately increased albuminuria 30–299 mg/g 3–29 mg/mmol	A3: overt proteinuria ≥ 300 mg/g ≥ 30 mg/mmol
G1: ≥ 90 ml/min/1,73 m²	Follow-up 1	Treatment 1	Treatment and referral to a nephrologist 3
G2: 60–89 ml/min/1,73 m²	Follow-up 1	Treatment 1	Treatment and referral to a nephrologist 3
G3a: 45–59 ml/min/1,73 m²	Treatment 1	Treatment 2	Treatment and referral to a nephrologist 3
G3b: 30–44 ml/min/1,73 m²	Treatment 2	Treatment and referral to a nephrologist 3	Treatment and referral to a nephrologist 3
G4: 15–29 ml/min/1,73 m²	Treatment and referral to a nephrologist 3	Treatment and referral to a nephrologist 3	Treatment and referral to a nephrologist 4+
G5: < 15 ml/min/1,73 m²	Treatment and referral to a nephrologist 4+	Treatment and referral to a nephrologist 4+	Treatment and referral to a nephrologist 4+

Risk categories (colour coding in the original table): Low risk (if no other markers of kidney disease are present; no CKD), moderate risk, high risk, very high risk.

Notes: Numbers in the cells indicate guideline-recommended frequency of screening or monitoring per year. Suggested monitoring frequency ranges from once per year (1) ( ) to four or more times per year (4+, i.e. every 1–3 months), ( )) depending on the risk of CKD progression and CKD-related complications.

arterial blood pressure control and lipid management is required.
2. Individuals should be encouraged to stop tobacco smoking, as smoking is an independent risk factor for the development and progression of diabetic kidney disease.

VII. Treatment

1. In order to slow the progression of diabetic kidney disease, efforts should be made to achieve therapeutic targets for glycaemia, lipid profile and arterial blood pressure as presented in Chapter 4.

2. In the presence of albuminuria, therapy with an angiotensin-converting enzyme inhibitor (ACEI) or an angiotensin receptor blocker (ARB) should be used, as these agents reduce the risk of progression of chronic kidney disease (taking into account contraindications to their use). These drugs should be administered at the maximum tolerated doses.

Table 19.4. Recommendations for dosing of oral antihyperglycaemic agents, GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists depending on the stage of chronic kidney disease in type 2 diabetes [3]

	Chronic kidney disease (CKD) categories (stages) according to KDIGO (eGFR)				
	Stage G1 and G2 (eGFR > 60 ml/min/1.73 m²)	Stage G3a (eGFR 45–59 ml/min/1.73 m²)	Stage G3b (eGFR 30–44 ml/min/1.73 m²)	Stage G4 (eGFR 15–30 ml/min/1.73 m²)	Stage G5 (eGFR < 15 ml/min/1.73 m²) for the initiation of renal replacement therapy
Metformin		Dose reduction to a maximum of 2000 mg/day	More frequent eGFR monitoring dose reduction to a maximum of 1000 mg/day		
Sulfonylurea derivatives (gliclazide and glimepiride)		Increased risk of hypoglycaemia if eGFR < 60; consider dose reduction; gliclazide is the preferred drug as it is metabolised by the liver			
Sulfonylurea derivatives (glividone)					
Pioglitazone					
Linagliptin					
Saxagliptin	No need for dose modification.				
Sitagliptin			Dose reduction to 50 mg/day	Dose reduction to 25 mg/day	
Vildagliptin		Dose reduction to 50 mg/day if eGFR < 50			
Canagliflozin	Initial dose 100 mg, gradual dose increase up to 300 mg if required	Initiation or continuation at 100 mg/day		Continuation at 100 mg/day; do not initiate treatment; discontinue in patients on dialysis; continuation possible if well tolerated (cardio-renal protection)	
Dapagliflozin	Treatment initiation possible down to eGFR = 25				May be continued if well tolerated (cardio-renal protection)
Empagliflozin	Treatment initiation possible down to eGFR = 20				May be continued if well tolerated (cardio-renal protection)
Dulaglutide	Treatment initiation possible down to eGFR = 15				
Exenatide (administered twice daily)		Gradual dose escalation at eGFR 30–50			
Exenatide (administered once weekly)	Treatment initiation possible down to eGFR = 30				
Liraglutide	No need for dose modification				
Lixisenatide	Treatment initiation possible down to eGFR = 15				
Semaglutide (administered subcutaneously and orally)	No need for dose modification				
Tirzepatide	No need for dose modification				

no need for dose adjustment depending on eGFR, dose adjustment recommended depending on eGFR, use of the drug not recommended at the given eGFR

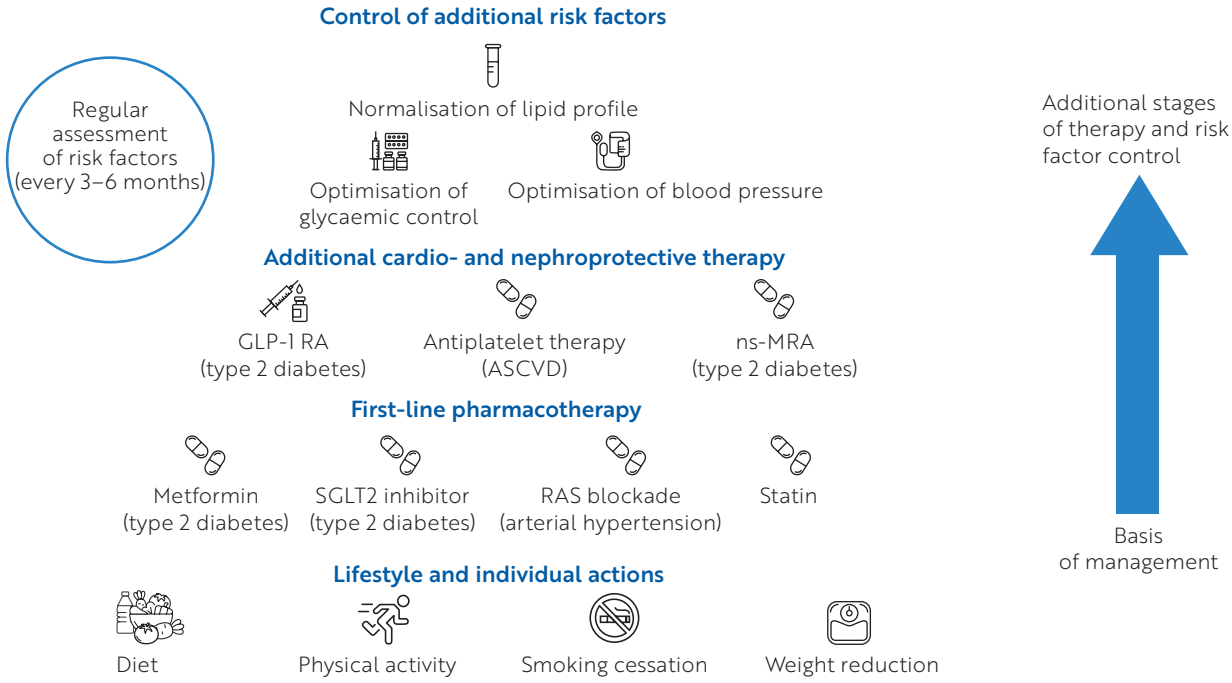


Figure 19.1. Holistic approach to cardio-renal risk [2]

3. When ACEIs or ARBs and/or non-steroidal mineralocorticoid receptor antagonists (ns-MRAs) and/or diuretics are used, serum creatinine, sodium and potassium concentrations should be monitored. An increase in serum creatinine concentration or a reduction in eGFR of ≤ 30% within 4 weeks, both after initiation of therapy and after dose escalation of drugs from the above groups, does not constitute an indication for treatment discontinuation, but rather for closer monitoring of renal function and serum potassium concentration, as well as identification of other causes of eGFR reduction or hyperkalaemia (e.g. dehydration, diet, other factors). In order to continue ACEI or ARB and/or ns-MRA therapy or to maintain appropriate dosing in individuals with elevated serum potassium concentration, the use of agents reducing gastrointestinal potassium absorption (patiromer, sodium zirconium cyclosilicate) may be considered.
4. Combined use of an ACEI and an ARB is contraindicated.
5. Metformin should not be used in individuals with eGFR < 30 ml/min/1.73 m². The dose of metformin should be reduced to 1000 mg/day in individuals with eGFR 30–44 ml/min/1.73 m² and in individuals with eGFR 45–59 ml/min/1.73 m² who are at high risk of lactic acidosis.
6. In individuals with type 2 diabetes and chronic kidney disease with eGFR ≥ 20 ml/min/1.73 m², use of an SGLT2 inhibitor with proven efficacy in reducing the risk of progression of diabetic kidney disease and cardiovascular complications is recommended, irrespective of HbA_{1c} levels, in order to reduce progression of chronic kidney disease and cardiovascular events. After initiation of SGLT2 inhibitor therapy, no change in the principles of monitoring serum creatinine concentration is required.
7. Use of a glucagon-like peptide-1 receptor agonist (GLP-1 RA) with proven cardiovascular and renal benefits is recommended in individuals with type 2 diabetes and chronic kidney disease with eGFR ≥ 25 ml/min/1.73 m² and ACR ≥ 100 mg/g. Semaglutide is the first GLP-1 receptor agonist (FLOW trial) for which a reduction in the rate of GFR decline and a reduction in the risk of renal-related mortality, as well as all-cause mortality, have been demonstrated across a wide range of eGFR values (25–75 ml/min/1.73 m²).
8. Use of a non-steroidal mineralocorticoid receptor antagonist (ns-MRA) with proven cardiovascular and nephroprotective effects should be considered in individuals with type 2 diabetes and eGFR ≥ 25 ml/min/1.73 m², normal serum potassium concentration, and persistent albuminuria (≥ 30 mg/g) despite treatment

with the maximum tolerated dose of a renin–angiotensin–aldosterone system inhibitor.

9. ACEIs, ARBs, SGLT2 inhibitors, GLP-1 receptor agonists and ns-MRAs reduce the risk of progression of chronic kidney disease and cardiovascular complications in individuals with type 2 diabetes and chronic kidney disease.

REFERENCES

1. Caudel SE, Verma A. Albuminuria in cardiovascular, kidney, and metabolic disorders: a state-of-the-art review. Circulation 2025; 151: 716–732.
2. DCCT/EDIC Research Group. Effect of intensive diabetes treatment on albuminuria in type 1 diabetes: long-term follow-up of the Diabetes Control and Comlications Trial and Epidemiology of Diabetes Interwentions and Complications Study. Lancet Diabetes Endocrinol 2014; 2: 793–800.
3. De Boer IH, Khunti K, Sadusky T, et al. Diabetes management in chronic kidney disease: a consensus report by the American Diabetes Association (ADA) and Kidney Disease: Improving Global Outcomes (KDIGO). Diabetes Care 2022; 45: 3075–3090.
4. De Boer IH, Sun W, Cleasry PA, et al. Intensive diabetes therapy and glomerular filtration rate in type 1 diabetes. N Engl J Med 2011; 365: 2366–2376.
5. Delanaye P, Glassock RJ, Pottel H, et al. An age-calibrated definition of chronic kidney disease: rationale and benefits. Clin Biochem Rev 2016; 37: 17–26.
6. Ismail-Beigi F, Craven T, Banerji MA, et al.; ACCORD trial group. Effect of intensive treatment of hyperglycemia on microvascular outcomes in type 2 diabetes: an analysis of the ACCORD randomized trial. Lancet 2010; 376: 419–430.
7. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group; KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int 2024; 105 (4S): S117–S314.
8. Mann JFE, Rossing P, Bakris G, et al. Effects of semaglutide with and without concomitant SGLT2 inhibitor use in participants with type 2 diabetes and chronic kidney disease in the FLOW trial. Nature Med 2024; 30: 2849–2856.
9. Molitch ME, Steffes M, Sun W, et al. Development and progression of renal insufficiency with and without albuminuria in adults with type 1 diabetes in the Diabetes Control Complications Trial and the Emidemiology of Diabetes Interventions and Complications Study. Diabetes Care 2010; 33: 1536–1543.
10. Patel A, MacMahon S, Chalmers J, et al. Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. N Engl J Med 2008; 358: 2560–2572.
11. Percovic V, Heerspink HL, Chalmers J, et al. Intensive glucose control improves kidney outcomes in patients with type 2 diabetes. Kidney Int 2013; 83: 517–523.
12. Perkovic V, Tuttle KR, Rossing P, et al.; FLOW Trial Committees and Investigators. Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes. N Engl J Med 2024; 391: 109–121.
13. Tuttle KR, Bakirs GL, Bilous RW, et al. Diabetic kidney disease: a raport from an ADA Consensus Conference. Diabetes Care 2014; 37: 2864–2883.
14. UK Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). Lancet 1998; 352: 854–865.
15. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet 1998; 352: 837–853.
16. Wong MG, Percovic V, Chalmers J, et al. Long-term benefits of intensive glucose control for preventing end-stage kidney disease: ADVANCE-ON. Diabetes Care 2016; 39: 694–700.
17. Zoungas S, Chalmers J, Neal B, et al. Follow-up of blood pressure lowering and glucose control in type 2 diabetes. N Engl J Med 2014; 371: 1392–1406.

20. Diabetic Eye Disease

CHAPTER HIGHLIGHTS:
· Optimisation of glycaemic control, arterial blood pressure and lipid management reduces the risk of development and progression of diabetic retinopathy. [**A**]
· Fundus examination after pupil dilation should be performed no later than 5 years after disease onset in adults with type 1 diabetes and immediately after diagnosis of type 2 diabetes. [**B**]
· Laser photocoagulation reduces the risk of vision loss in individuals with proliferative retinopathy. [**A**]
· Intravitreal anti-VEGF injections in individuals with macular oedema may improve vision. [**A**]
· Treatment with acetylsalicylic acid for cardioprotection is not contraindicated in individuals with retinopathy and does not increase the risk of vitreous haemorrhage. [**A**]

Diabetes-related complications affect virtually all anatomical structures of the visual system. The most common and the most serious complication threatening vision is diabetic retinopathy and the associated diabetic macular oedema. Diabetic retinopathy is a highly specific neurovascular complication of both type 1 and type 2 diabetes. Among non-ocular diabetic complications, cataract and secondary glaucoma are of the greatest clinical importance. The recommendations below take into account the new classification of diabetic retinopathy.

I. Natural history and classification of diabetic retinopathy

1. No signs of diabetic retinopathy.
2. Mild non-proliferative diabetic retinopathy (NPDR) – presence of microaneurysms only.
3. Moderate non-proliferative diabetic retinopathy – more lesions than in mild NPDR, but fewer than in severe NPDR.
4. Severe non-proliferative diabetic retinopathy:
   - haemorrhages (> 20) in all four retinal quadrants and/or
   - venous beading in at least two quadrants and/or
   - intraretinal microvascular abnormalities in at least one quadrant.
5. Proliferative diabetic retinopathy (PDR) – neovascularisation and growth of fibrous tissue in the retina, leading to vision loss through:
   - recurrent vitreous haemorrhage from newly formed vessels,
   - retinal detachment due to traction from proliferative tissue,
   - development of glaucoma.

II. Natural history and classification of diabetic macular oedema

1. No diabetic macular oedema.
2. Mild diabetic macular oedema – changes distant from the macular centre.
3. Moderate diabetic macular oedema – changes close to the macular centre.
4. Severe diabetic macular oedema – changes involving the macular centre.

III. Risk factors for development and progression of diabetic retinopathy

1. Duration of diabetes – the strongest prognostic factor for development and progression of diabetic retinopathy.
2. Poor metabolic control of diabetes:
   - intensive treatment reduces the risk of development and progression of retinopathy in individuals with type 1 diabetes,
   - intensive treatment of type 2 diabetes reduces the risk of retinopathy.
3. Arterial hypertension.
4. Lipid metabolism disorders.
5. Diabetic kidney disease.
6. Pregnancy in women with diabetes.
7. Puberty.
8. Cataract surgery.
9. Status post kidney and pancreas transplantation or kidney transplantation alone.

IV. Diagnosis of diabetic retinopathy

1. Visual acuity assessment.
2. Colour vision testing.
3. Fundus examination (ophthalmoscopy, always after pupil dilation).
4. Digital colour fundus photography, mainly used for screening purposes (does not replace a full ophthalmological examination).
5. Fluorescein angiography of the fundus – indications:
   - detection of changes in moderate and severe NPDR,

- detection of early neovascularisation in PDR,
- assessment of laser photocoagulation effectiveness,
- clarification of causes of unexplained visual acuity deterioration.

6. Wide-field fluorescein angiography.
7. Optical coherence tomography (OCT) – the basic method for diagnosis and monitoring of macular oedema.
8. Ultrasonography, particularly in individuals with vitreous haemorrhage.
9. Confocal microscopy (assessment of corneal nerve fibre density as an early marker of neuropathy).

V. Indications for ophthalmological examinations in individuals with diabetes

1. Initial examination:

- in type 1 diabetes – within the first 5 years from disease onset,
- in type 2 diabetes – at the time of diagnosis or shortly thereafter.

2. Follow-up examinations and possible treatment:

- indicated due to the initially asymptomatic nature of retinopathy,
- examination frequency depends on the stage of diabetic retinopathy:
  - » no retinopathy: every 1–2 years,
  - » mild and moderate NPDR: every 6–12 months,
  - » severe NPDR: laser treatment, follow-up at least every 3–6 months,
  - » proliferative retinopathy: urgent laser treatment or other ophthalmic surgery (e.g. vitrectomy),
  - » diabetic macular oedema:
  - » in non-centre-involving form – laser treatment,
  - » in centre-involving form – intravitreal anti-VEGF injections, which may be combined with laser treatment,
  - » after retinal laser treatment – 1 month after the procedure,
  - » after vitrectomy – follow-up scheduled individually depending on fundus status,
  - » in pregnant women with diabetes – every 1–3 months throughout pregnancy depending on ocular status,
  - » in women planning pregnancy – examination before conception; if necessary, laser treatment of the retina is performed.
  - » screening examinations are not required in women with gestational diabetes.

3. Clinical situations requiring urgent and/or extended ophthalmological diagnostics:

- risk of vision loss:
  - » presence of proliferative retinopathy,
  - » presence of advanced ocular complications (neovascularisation of the iris, vitreous haemorrhage, recent retinal detachment),
- presence of lesions potentially threatening vision loss:
  - » severe non-proliferative diabetic retinopathy,
  - » non-proliferative diabetic retinopathy with diabetic macular oedema,
  - » other abnormalities present in the ocular fundus that are difficult to interpret or unexplained deterioration of visual acuity.

The recommended frequency of ophthalmological examinations in individual groups is presented in Table 20.1.

VI. Screening

Screening for diabetic retinopathy is performed by an ophthalmologist or a trained professional using an ophthalmoscope (with pharmacological pupil dilation) or on the basis of colour fundus photography. Screening may also be carried out using telemedicine, with fundus cameras and image assessment by qualified personnel, or with the use of appropriate software employing artificial intelligence–based image analysis algorithms. Colour fundus photography has substantial potential for the provision of screening services in areas where access to qualified specialists is limited. If screening reveals diabetic retinopathy, prompt referral to an ophthalmologist is recommended. Retinal photography may therefore serve as a screening tool in the diagnosis of retinopathy; however, it does not replace a comprehensive ophthalmological examination, which should be performed no later than 5 years after diagnosis in adults with type 1 diabetes and at the time of diagnosis of type 2 diabetes. Subsequent examinations should be performed at intervals recommended by the ophthalmologist.

In individuals with type 1 diabetes, if no retinal changes are detected in two consecutive annual follow-up examinations, fundus examination may be performed every 2 years. In individuals with type 2 diabetes with good metabolic control and no retinal changes, examinations may be performed every 2–3 years.

In women with type 1 and type 2 diabetes, ophthalmological examinations should be performed before pregnancy or in the first trimester

Table 20.1. Recommended frequency of ophthalmological examinations in specific groups of individuals

First-time examination	
Type 1 diabetes	Within the first 5 years after disease onset (when diagnosed during puberty – shortly after diagnosis)
Type 2 diabetes	At the time of diagnosis
Follow-up examinations and possible treatment	
Stage of ocular complications	**Frequency of follow-up and treatment**
No retinopathy	Every 1–2 years
Mild and moderate NPDR	Every 6–12 months
Severe NPDR	At least every 3–6 months
Proliferative retinopathy	Urgent laser therapy
Non-centre-involving diabetic macular oedema	Laser therapy
Centre-involving diabetic macular oedema	Intravitreal injections (anti-VEGF + laser therapy)
Follow-up after ophthalmic procedures in special situations	
After laser treatment	Depending on ocular status
After vitrectomy	Depending on ocular status
Pregnancy	Depending on ocular status, every 1–3 months
Women planning pregnancy	Prior to conception – laser therapy if required

of pregnancy and subsequently repeated in each trimester and within one year after delivery, with assessment of the severity of retinopathy.

Regular fundus examinations and treatment enable prevention of vision loss due to diabetic retinopathy in up to 98% of cases.

Established screening strategies allow a substantial, even several-fold, reduction in the risk of blindness and a decrease in the costs of treatment of diabetes-related ocular complications.

VII. Treatment of diabetic retinopathy

1. Optimisation of glycaemic control, arterial blood pressure and lipid abnormalities (fenofibrate, statins). Before intensification of antihyperglycaemic therapy, assessment of the presence of retinopathy and its stage is required, as a rapid improvement in metabolic control may lead to progression of retinopathy. Although available data indicate pleiotropic effects of newer antihyperglycaemic agents, there are still too few clinical studies to determine their impact on the prevention and progression of diabetic retinopathy. Acetylsalicylic acid, when administered for cardioprotective purposes, is not contraindicated in individuals with retinopathy and does not increase the risk of intraretinal haemorrhage.
2. No association has been demonstrated between treatment with GLP-1 receptor agonists and the risk of development of retinopathy. Worsening of retinopathy may be related to a rapid improvement in glycaemic control. Diabetes and obesity are risk factors for non-arteritic anterior ischaemic optic neuropathy (NAION), a rare condition leading to sudden vision loss. Isolated cases of NAION in individuals with diabetes treated with GLP-1 receptor agonists require further investigation.
3. Treatment of diabetic macular oedema includes intravitreal administration of anti-VEGF agents (aflibercept 2 mg, aflibercept 8 mg, bevacizumab, faricimab, brolucizumab and ranibizumab) and dexamethasone in the form of an implant. Anti-VEGF injections are administered within drug programmes, qualification for which is based on ophthalmological and diabetological criteria. An important parameter for qualification is the HbA_{1c} value.

Treatment of individuals receiving therapy for the first time or those previously treated unsuccessfully is initiated with five doses of bevacizumab.

4. Retinal laser therapy (possible if the optical media of the eye are clear):

- appropriately timed retinal laser therapy inhibits progression of advanced diabetic retinopathy.
- types of retinal laser therapy:
  - » subthreshold (mainly micropulse) – without tissue coagulation, used in macular oedema without significant thickening and without deterioration of visual acuity,

   - » focal – recommended in the presence of early lesions in diabetic macular oedema without involvement of the fovea,
   - » grid-type – in diffuse macular oedema when first-line treatment has not been effective,
   - » panretinal photocoagulation – recommended in severe non-proliferative and proliferative retinopathy.
5. Intravitreal or periocular injections of steroids with antiangiogenic and anti-oedematous effects, such as triamcinolone, dexamethasone or fluocinolone acetonide in sustained-release formulations, may be considered as first-line therapy when contraindications to anti-VEGF antibodies are present or when monthly visit frequency cannot be maintained.
6. Vitrectomy – indications:
   - vitreous haemorrhages that do not resolve despite other treatment methods,
   - vitreoretinal traction running vertically towards the macula,
   - advanced proliferative retinopathy with complications.
7. In cases of irreversible visual impairment, consultation and/or rehabilitation for individuals with low vision or blindness is required.
8. In mild and moderate retinopathy with the presence of hard exudates, sulodexide may be used at a dose of 250 LSU twice daily.

REFERENCES

1. Ahmed J, Ward TP, Bursell SE, et al. The sensitivity and specificity of nonmydriatic digital stereoscopicretinal imaging in detecting diabetic retinopathy. Diabetes Care 2006; 29: 2205–2209.
2. Bignamini AA, Chebil A, Gambaro G, Matuška J. Sulodexide for diabetic-induced disabilities: a systematic review and meta-analysis. Adv Ther 2021; 38: 1483–1513.
3. Bragge P, Gruen RL, Chau M, et al. Screening for presence or absence of diabetic retinopathy: a meta-analysis. Arch Ophthalmol 2011; 129: 435–444.
4. Brown DM, Boyer DS, Do DV, et al.; PHOTON Investigators. Intravitreal aflibercept 8 mg in diabetic macular oedema (PHOTON): 48-week results from a randomised, double-masked, non-inferiority, phase 2/3 trial. Lancet 2024; 403: 1153–1163.
5. Cai CX, Hribar M, Baxter S, et al. Semaglutide and nonarteritic anterior ischemic optic neuropathy. JAMA Ophthalmol 2025; 143: 304–314.
6. Chew EY, Davis MD, Danis RP, et al.; Action to Control Cardiovascular Risk in Diabetes Eye Study Research Group. The effects of medical management on the progression of diabetic retinopathy in persons with type 2 diabetes: the action to Control Cardiovascular Risk in Diabetes (ACCORD) Eye Study. Ophthalmology 2014; 121: 2443–2451.
7. Daskivich LP, Vasquez C, Martinez C Jr, et al. Implementation and evaluation ot a large-scale teleretinal dlabetic retinopathy screening program in the Los Angeles County Department ot Health Services. JAMA Intern Med 2017; 177: 642–649.
8. Diabetes Control and Complications Trial Research Group; Nathan DM, Genuth S, Lachin J, et al. The effect of intensive treatment of diabetes on the development and progression of longerm complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 329: 977–986.
9. Do DV, Wang X, Vedula SS, et al. Blood pressurecontrol for diabetic retinopathy. Cochrane Database Syst Rev 2015; 1: CD006127. DOI: 10.1002/14651858.CD006127.pub3.
10. Gross JG, Glassman AR, Jampol LM, et al. Panretinal photocoagulation vs. intravitreous ranibizumab for proliferative diabetic retinopathy: a randomized clinical trial. JAMA 2015; 314: 2137–2146.
11. Gubitosi-Klug RA, Sun W, Cleary PA, et al. Effects of prior intensive insulin therapy and risk factors on patient-reported visual function outcomes in the Diabetes Control and Complications Trial/ Epidemiology of Diabetes Interventions and Complications (DCCT/EDIC) cohort. JAMA Ophthalmol 2016; 134: 137–145.
12. Gunderson EP, Lewis CE, Tsai AL, et al. A 20-year prospective study of childbearing andincidence of diabetes in young women, controlling for glycemia before conception: the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Diabetes 2007; 56: 2990–2996.
13. Kunutsor SK, Balasubramanian VG, Zaccardi F, et al. Glycaemic control and macrovascular and microvascular outcomes: a systematic review and meta-analysis of trials investigating intensive glucose-lowering strategies in people with type 2 diabetes. Diabetes Obes Metab 2024; 26: 2069–2081.
14. Ma Y, Lin C, Cai X, et al. The association between the use of sodium glucose cotransporter 2 inhibitor and the risk of diabetic retinopathy and other eye disorders: a systematic review and meta-analysis. Expert Rev Clin Pharmacol 2022; 15: 877–886.
15. Preiss D, Logue J, Sammons E, et al. Effect of fenofibrate on progression of diabetic retinopathy. NEJM Evid 2024; 3: EVIDoa2400179. DOI: 10.1056/EVIDoa2400179.
16. Program lekowy NFZ o numerze PL B70. Leczenie pacjentów z chorobami siatkówki (ICD-10: H35.3, H36.0). Program NFZ obowiązuje od 01.07.2022 i jest połączony razem z AMD; A: leczenie pacjentów z wysiękowym zwyrodnieniem plamki związanym z wiekiem (AMD) oraz B: leczenie pacjentów z cukrzycowym obrzękiem plamki (DME).
17. Shi R, Zhao L, Wang F, et al. Effects of lipid-lowering agents on diabetic retinopathy: a meta-analysis and systematic review. Int J Ophthalmol 2018; 11: 287–295.

18. The Diabetic Retinopathy Study Research Group. Preliminary report on effects of photocoagulation therapy. Am J Ophthalmol 1976; 81: 383–396.
19. Wells JA, Glassman AR, Ayala AR, et al. Aflibercept, bevacizumab, or ranibizumab for diabetic macular edema. N Engl J Med 2015; 372: 1193–1203.
20. Wong TY, Haskova Z, Asik K, et. al. Faricimab treat-and-extend for diabetic macular edema: two-year results from the randomized phase 3 YOSEMITE and RHINE trials. Ophthalmology 2024; 131: 708–723.

21. Diabetic Neuropathy: Prevention, Diagnosis, and Treatment

CHAPTER HIGHLIGHTS
• In individuals with type 1 diabetes, maintaining optimal glycaemic control from the time of diagnosis is of key importance in the primary and secondary prevention of peripheral diabetic polyneuropathy and autonomic neuropathy of the cardiovascular system. [**A**]
• Diagnosis of diabetic neuropathy, in addition to a detailed medical history, should include assessment of both small-fibre function (pain and/or temperature sensation) and large-fibre function (vibration sensation). Each individual should undergo annual assessment of protective sensation using a 10 g monofilament in order to evaluate the risk of diabetic foot ulceration. Examinations should be performed for the first time after 5 years in individuals with type 1 diabetes and at the time of diagnosis in individuals with type 2 diabetes. [**B**]
• Pregabalin, gabapentin, duloxetine or amitriptyline should be considered as first-line agents in the treatment of neuropathic pain in individuals with diabetes. [**A**]

Distal symmetric diabetic polyneuropathy is a cause of severe symptoms, significantly impairs quality of life, and is a recognised risk factor for the development of diabetic foot disease in the form of ulceration and Charcot neuroarthropathy. Neuropathy increases the risk of amputations, fractures and falls, increases treatment costs, and is a predictor of increased mortality. Autonomic neuropathy of the cardiovascular system constitutes an independent risk factor for increased mortality in diabetes. Neuropathy may develop already at the stage of prediabetes; therefore, in individuals with prediabetes and symptoms of peripheral neuropathy, diagnostic evaluation should also be considered.

I. Clinical classification of neuropathy:

- generalised symmetrical polyneuropathies:
  - » chronic peripheral sensorimotor,
  - » autonomic,
  - » acute sensory,
- focal and multifocal neuropathies:
  - » cranial nerve neuropathies,
  - » spinal nerve neuropathies (thoracic and lumbar),
  - » focal limb neuropathies, including compression syndromes,
  - » proximal motor neuropathy (amyotrophy).

II. Principles of neuropathy assessment:

- frequency of examinations:
  - » assessment for the presence of symptoms of diabetic neuropathy should be performed at least once a year, for the first time:
  - » in type 1 diabetes – 5 years after disease onset, unless symptoms suggestive of neuropathy are present earlier,
  - » in type 2 diabetes – at the time of diagnosis,
- other, non-diabetic aetiologies of peripheral nervous system damage should be considered and, if necessary, excluded, particularly in cases with a predominance of motor neuropathy,
- consideration should be given to periodic measurement of vitamin B_{12} concentration in individuals treated with metformin when symptoms of neuropathy occur or worsen, especially during long-term therapy and at high doses,
- in doubtful cases, neurological consultation is recommended.

III. Diagnostic criteria for diabetic neuropathy

Distal symmetric polyneuropathy

A. Diagnostic methods:

- assessment of light touch sensation using a 10 g monofilament (Semmes-Weinstein 5.07),
- assessment of vibration perception threshold – using a neurothesiometer or a calibrated 128 Hz tuning fork,
- assessment of pain sensation (sterile needle),
- assessment of temperature sensation (dual-ended testing device with a metal and a plastic tip),
- assessment of tendon reflexes,
- assessment of muscle strength,
- electroneurography (nerve conduction studies).

B. Principles of diagnosis:

- subjective symptoms: sensory disturbances, numbness, burning, tingling, stinging, shooting sensations, spontaneous pain, muscle cramps, mainly in the feet and lower legs, persisting for several months (symptoms intensify or occur predominantly at night; physical exertion neither induces nor exacerbates symptoms),
- objective signs: reduced or absent sensation to touch, vibration, pain and temperature, reduced muscle strength, reduced or absent tendon reflexes (knee and ankle),
- peripheral diabetic neuropathy is considered probable based on the presence of 2 of the following 3 elements of clinical assessment: subjective symptoms, reduced or absent sensation (touch, vibration, pain and/or temperature), and/or reduced or absent tendon reflexes,
- standardised questionnaires may be used for the assessment of subjective and objective symptoms of neuropathy. For assessment of subjective symptoms, the NSS (Neuropathy Symptom Score) may be used, as well as the simple 4-point DNS (Diabetic Neuropathy Symptom) scale, which correlates well with the NSS. For assessment of both subjective and objective symptoms, the Michigan Neuropathy Screening Instrument (MNSI) may be used due to its documented accuracy and established cut-off points for the Polish population,
- in selected individuals, additional nerve conduction studies (electroneurography) may be required to establish a definite diagnosis of neuropathy and/or to perform differential diagnosis of aetiology; this is particularly indicated in cases of rapid progression of symptoms, asymmetry, predominance of motor neuropathy, or suspicion of a non-diabetic cause,
- in the diagnosis of small fibre neuropathy, in cases of diagnostic uncertainty based on the clinical picture, additional assessment of corneal nerve fibre density using confocal microscopy or skin biopsy may be utilised,
- painful polyneuropathy is diagnosed when clinical features allowing the diagnosis of neuropathy and pain are located in the same anatomical area. The DN4 questionnaire (Douleur Neuropathique en 4 Questions), scored on a 0–10 scale, may be used as a screening tool for neuropathic pain, with a threshold of ≥ 4 suggesting neuropathic pain. Pain intensity may be assessed using a visual analogue scale (VAS) from 0 to 10. In the painful form, characteristic findings on physical examination may be normal; in the presence of typical symptoms, neuropathy may therefore be diagnosed even in the absence of abnormalities on physical examination.

Autonomic neuropathy

Autonomic nervous system function is assessed indirectly on the basis of analysis of changes in effector organ function in response to specific stimuli. Due to the non-specific nature of clinical symptoms, the diagnosis should be supported by specific tests. Other diseases of the effector organs should be excluded, organic and functional disorders of different origin should be considered, and the effects of current treatment should be ruled out.

Clinically, autonomic neuropathy most commonly manifests as hypoglycaemia unawareness, resting tachycardia, orthostatic hypotension, gastroparesis, constipation or diarrhoea, erectile dysfunction, neurogenic bladder, or disturbances of sweating.

1. Cardiovascular system

Cardiovascular autonomic neuropathy is diagnosed on the basis of tests assessing heart rate variability.

Autonomic neuropathy is considered probable or early when one heart rate variability test result is abnormal, and confirmed when two of the heart rate variability tests listed below are abnormal.

Severe (advanced) cardiovascular autonomic neuropathy is diagnosed when abnormal heart rate variability test results are accompanied by an abnormal arterial blood pressure response to standing (orthostatic challenge):

- tests assessing parasympathetic function:
  - » change in heart rate during deep breathing,
  - » change in heart rate in response to standing,

» change in heart rate in response to the Valsalva manoeuvre,
- tests assessing sympathetic function: change in arterial blood pressure in response to standing.

2. Gastrointestinal system:
- gastric dysfunction – X-ray examination, radionuclide scintigraphy, breath tests, electrogastrography (EGG), manometry, ultrasonography,
- small intestinal dysfunction – no specific diagnostic tests; exclusion of other causes, manometry, wireless diagnostic capsule for assessment of small bowel motility disorders,
- large bowel dysfunction – exclusion of other causes (endoscopy), intestinal transit after oral administration of contrast medium, manometry, wireless diagnostic capsule,
- gallbladder dysfunction – functional ultrasonography.

3. Genitourinary system:
- urinary bladder dysfunction – cystometry (assessment of bladder filling before and after micturition), electromyography of the bladder sphincter, uroflowmetry and urethral pressure profile,
- erectile impotence – questionnaires (International Index of Erectile Function – IIEF, and its abbreviated 5-item version – IIEF-5), vascular studies (Doppler ultrasonography), cavernosography, hormonal testing, psychological tests, regional assessment of vibration perception threshold, functional tests including monitoring of nocturnal erections.

4. Disorders of sweating – simple indicators of sudomotor function (e.g. Neuropad), tests requiring specialised equipment (assessment of sweat gland function using devices such as Sudoscan).
5. Pupillary function disorders – pupillometry.

IV. Treatment

Diabetic neuropathy is asymptomatic in approximately 50% of cases. Causal treatment consists of glycaemic control. Optimisation of glycaemic control should be implemented as early as possible in individuals with type 1 and type 2 diabetes in order to prevent and/or delay the development of neuropathy. In individuals with sensory disturbances, education regarding foot care and risk factors for the development of diabetic foot syndrome is required. In individuals with neuropathic pain, treatment is mandatory, as pain significantly worsens quality of life and daily functioning and may lead to depression. Various therapeutic options are available for symptomatic pain management. Treatment of autonomic neuropathy alleviates symptoms, improves quality of life and prognosis; however, it is often challenging, and effectiveness varies between individuals. The management algorithm for peripheral diabetic neuropathy is presented in Figure 21.1.

1. Treatment targeting the pathomechanisms of diabetic neuropathy:
- optimal glycaemic control is of key importance in the treatment of diabetic neuropathy, with particular attention to avoidance of hypoglycaemia and large daily glycaemic fluctuations,
- control of arterial blood pressure and lipid metabolism, smoking cessation and avoidance of alcohol consumption,
- adjunctive pharmacotherapy: α-lipoic acid, benfotiamine, angiotensin-converting enzyme inhibitors.

2. Pharmacological symptomatic treatment of neuropathic pain in peripheral diabetic neuropathy (the analgesic effect of treatment is individual-specific) is presented in Figure 21.2 and Table 21.1.

In the treatment of chronic neuropathic pain, drug doses should be individualised, aiming for the lowest effective dose, without exceeding the maximum recommended dose. Treatment effectiveness should be assessed after at least 2 weeks of use at a therapeutic dose. A realistic goal of pharmacotherapy is a 30–50% reduction in pain intensity on the visual analogue scale (VAS). The aim of treatment is not only pain reduction but also improvement in sleep quality, mobility and overall quality of life, maintenance of social activity and work ability.

Neuromodulatory methods may have potential relevance in the treatment of neuropathic pain, including transcutaneous electrical nerve stimulation (TENS), frequency-modulated electrical stimulation therapy (FREMS), repetitive transcranial magnetic stimulation (rTMS) and spinal cord stimulation (SCS). Reduction of pain intensity and improvement in quality of life have been demonstrated with the use of these methods in painful neuropathy.

3. Symptomatic treatment of autonomic diabetic neuropathy:
- cardiovascular system:
  » disturbances of heart rhythm control – controlled, graded physical exercise, angiotensin-converting enzyme inhibitors, β-adrenoreceptor blockers without intrinsic sympathomimetic activity,

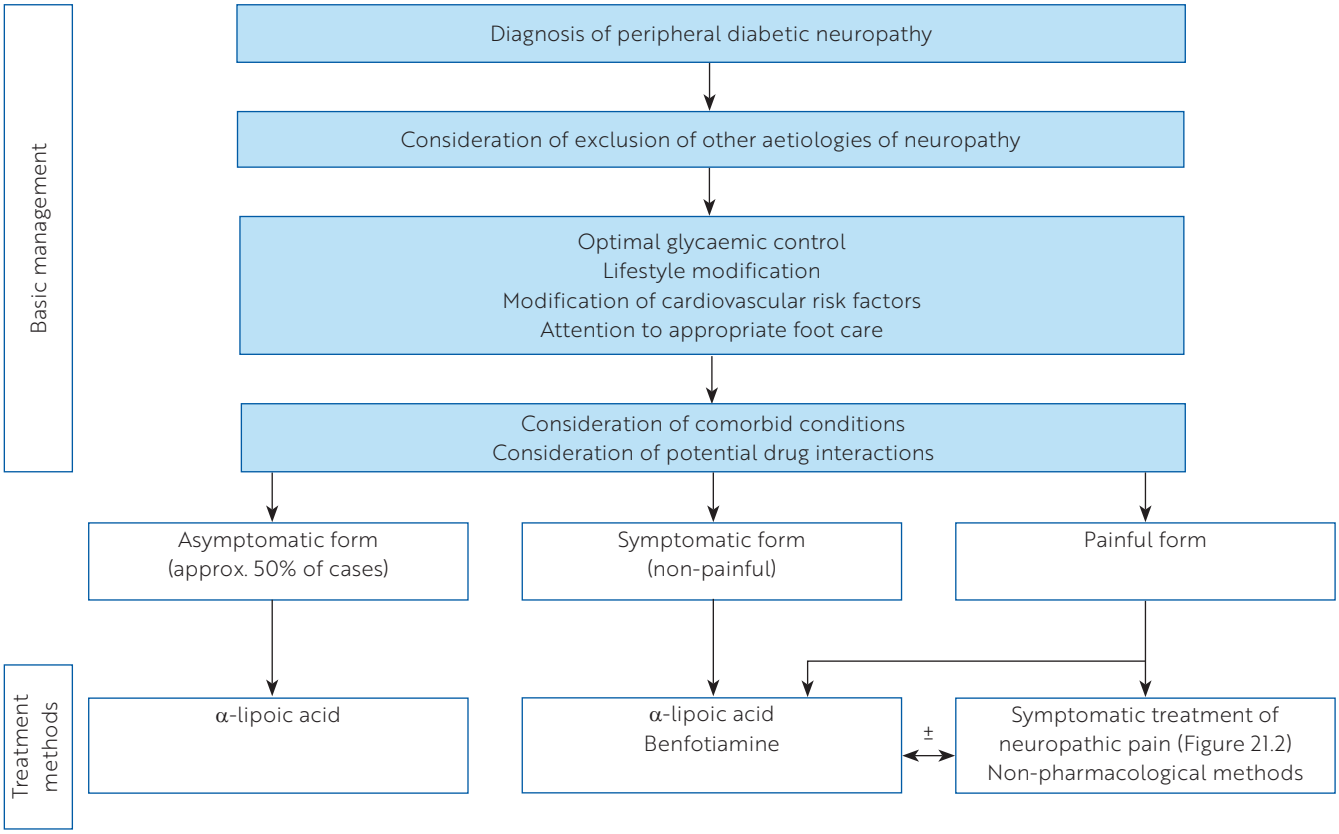
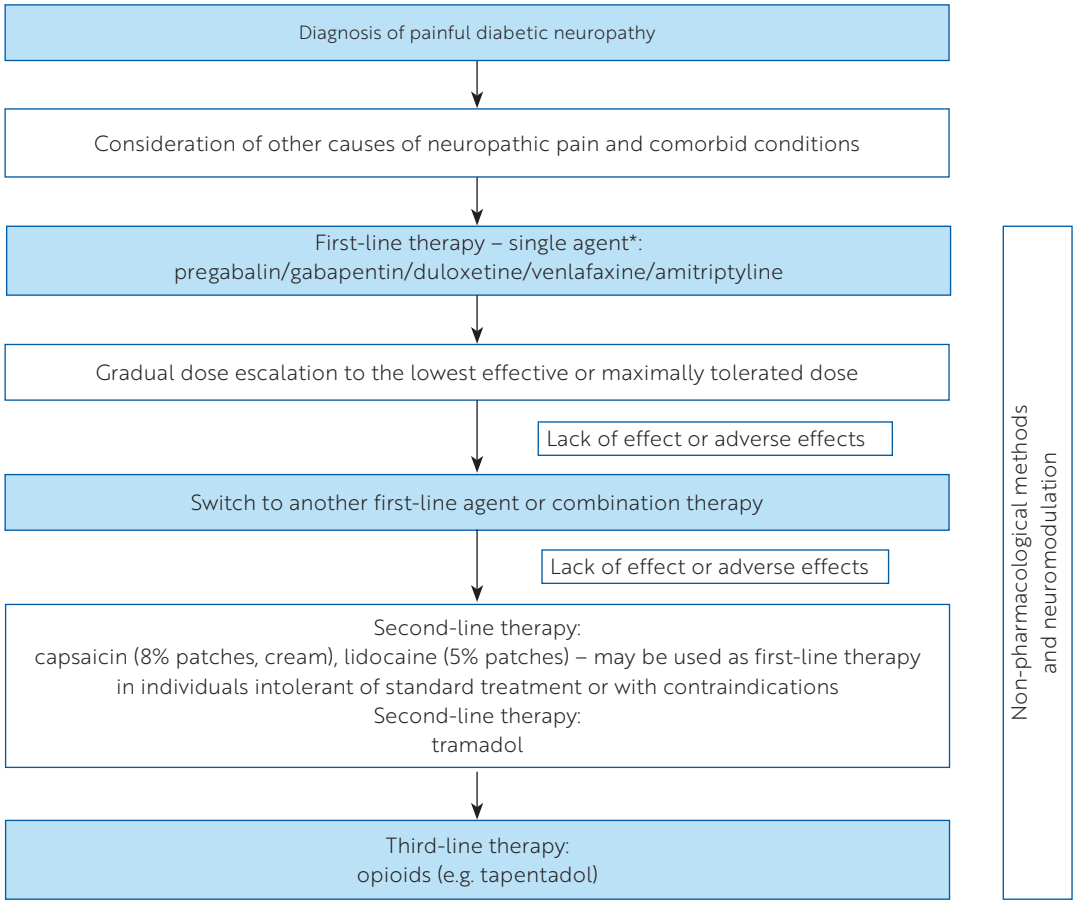


Figure 21.1. Management algorithm for peripheral diabetic neuropathy [13]

  - » orthostatic hypotension – compression garments for the lower limbs and abdomen, increased dietary salt intake, isometric exercises, mineralocorticoids (fludrocortisone), α1-adrenomimetics (midodrine),
- gastrointestinal system:
  - » gastroparesis – dietary modification (frequent small meals; in severe cases, semi-liquid or liquid diet), prokinetic agents (cisapride, itopride, erythromycin, trimebutine), agents reducing gastric secretion (H2-receptor antagonists, proton pump inhibitors), antiemetic drugs, surgical treatment, gastric electrical stimulation,
  - » intestinal dysfunction – dietary modification (consideration of a gluten-free diet, lactose restriction), cholestyramine, clonidine, octreotide, antidiarrhoeal agents (loperamide), pancreatic enzymes, antibiotics,
- genitourinary system:
  - » urinary bladder dysfunction – avoidance of urinary retention, scheduled and regular voiding, cholinergic receptor agonists (bethanechol), external bladder massage before micturition, bladder catheterisation (intermittent or permanent),
  - » sexual dysfunction in men – psychotherapy, phosphodiesterase type 5 inhibitors (sildenafil, vardenafil, tadalafil), vacuum erection devices, intracavernosal injections (prostaglandin E1), penile prostheses,
  - » sexual dysfunction in women – psychotherapy, mechanical genital stimulators, topical lubricants, flibanserin,
- disorders of sweating – botulinum toxin, vasodilatory agents, emollient creams.

REFERENCES

1. Diabetes Control and Complications Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 329: 977–986.
2. Finnerup NB, Attal N, Haroutounian S, et al. Pharmacotherapy for neuropathic pain in adults: a systematic review and metaanalysis. Lancet Neurol 2015; 14: 162–173.
3. Martin CL, Albers JW, Pop-Busui R. Neuropathy and related findings in the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications study. Diabetes Care 2014; 37: 31–38.



**Choice of drug depending on the patient's risk profile.*

Figure 21.2. Algorithm for symptomatic treatment of neuropathic pain in peripheral diabetic neuropathy [7, 10, 13]

Table 21.1. Algorithm for pharmacological symptomatic treatment of neuropathic pain in peripheral diabetic neuropathy

First-line treatment – one of the following drugs	Effective doses of drugs
Anticonvulsant drugs	
Pregabalin	300–600 mg/day
Gabapentin	900–3600 mg/day
Serotonin–noradrenaline reuptake inhibitors (SNRIs)	
Duloxetine	60–120 mg/day
Venlafaxine	75–225 mg/day
Tricyclic antidepressants	
Amitriptyline*	25–100 mg/day
Second- and third-line treatment	**Effective doses of drugs**
Opioid drugs	
Tramadol	200–400 mg/day
Tapentadol	From 50 mg twice daily, max. 500 mg/day
Topical agents	
Capsaicin, lidocaine	

Effective doses of drugs are provided. Gradual dose titration to the maximally tolerated dose is required, if necessary. In the absence of efficacy of one of the first-line drugs, switching to another agent or combination therapy is recommended. Chronic use of opioids is not recommended. At each stage, non-pharmacological methods may be used (psychotherapy, physiotherapy, acupuncture, neuromodulation techniques).

**In older individuals and in those with cardiovascular disease, amitriptyline should be initiated at a dose of 10 mg and titrated cautiously depending on response and tolerability.*

4. Meijer JW, Smit AJ, Sonderen EV, et al. Symptom scoring systems to diagnose distal polyneuropathy in diabetes: the Diabetic Neuropathy Symptom score. Diabet Med 2002; 19: 962–965.
5. Mittal R, McKenna K, Keith G, et al. Diabetic peripheral neuropathy and neuromodulation techniques: a systematic review of progress and prospects. Neural Regen Res 2025; 20: 2218–2230.
6. Perkins BA, Olaleye D, Zinman B, et al. Simple screening tests for peripheral neuropathy in the diabetes clinic. Diabetes Care 2001; 24: 250–256.
7. Soliman N, Moisset X, Ferraro MC, et al.; NeuPSIG Review Update Study Group. Pharmacotherapy and non-invasive neuromodulation for neuropathic pain: a systematic review and meta-analysis. Lancet Neurol 2025; 24: 413–428.
8. Spallone V, Morganti R, D'Amato C, et al. Validation of DN4 as a screening tool for neuropathic pain in painful diabetic polyneuropathy. Diabet Med 2012; 29: 578–585.
9. Sutkowska E, Marciniak D, Koszewicz M, ey al. Validity and reliability of the Polish version of the Michigan Neuropathy Screening Instrument. World J Diabetes 2023; 14: 435–446.
10. Tesfaye S, Sloan G, Petrie J, et al.; OPTION-DM trial group. Comparison of amitriptyline supplemented with pregabalin, pregabalin supplemented with amitriptyline, and duloxetine supplemented with pregabalin for the treatment of diabetic peripheral neuropathic pain (OPTION-DM): a multicentre, double-blind, randomised crossover trial. Lancet 2022; 27: 680–690.
11. Überall MA, Kender Z, Quandel T, et al. Progressive improvements in patient-reported outcomes with the high-concentration capsaicin patch: a retrospective cohort study in patients with painful diabetic peripheral neuropathy (CASPAR study). J Diabetes Complications 2025; 39: 109085. DOI: 10.1016/j.jdiacomp.2025.109085.
12. Ziegler D, Rathmann W, Dickhaus T, et al. Prevalence of polyneuropathy in pre-diabetes and diabetes is associated with abdominal obesity and macroangiopathy: the MONICA/KORA Augsburg Surveys S2 and S3. Diabetes Care 2008; 31: 464–469
13. Ziegler D, Tesfaye S, Spallone V, et al. Screening, diagnosis and management of diabetic sensorimotor polyneuropathy in clinical practice: International expert consensus recommendations. Diabetes Res Clin Pract 2022; 186: 109063.

22. Diabetic Foot Disease

CHAPTER HIGHLIGHTS
• Maintaining optimal control of glycaemia, lipid levels and arterial blood pressure reduces the risk of development of diabetic foot disease. [**A**]
• Management of diabetic foot disease should be provided within specialised, multidisciplinary clinics. [**B**]
• The gold standard for offloading a non-infected plantar ulcer of the forefoot or midfoot in neuropathic foot disease is a total contact cast or another non-removable knee-high offloading device. [**A**]
• Key elements in the management of diabetic foot disease, in addition to maintaining optimal glycaemic control, include surgical wound management, offloading, systemic antibiotic therapy in the presence of infection, and vascular interventions in ischaemic foot disease. [**A**]

Diabetic foot disease includes the presence, in an individual with diagnosed diabetes, of at least one of the following pathologies: peripheral polyneuropathy, peripheral arterial disease (lower limb ischaemia), infection, ulceration, Charcot neuro-osteoarthropathy, or amputation.

In regional (provincial, university) diabetes centres, multidisciplinary diabetic foot clinics (reference clinics) should be established. In addition, basic diabetic foot clinics should operate within diabetes outpatient clinics, responsible for continuation of treatment initiated in the multidisciplinary clinic. The structure and scope of activities of these clinics should be consistent with the Programme for Support of Outpatient Management of Diabetic Foot Disease of the Ministry of Health. (http://www.mz.gov.pl/zdrowie-i-profilaktyka/programy-zdrowotne/wykaz-programow/program-wsparcia-ambulatoryjnego-leczenia-zespolu-stopy-cukrzycowej/).

With regard to inpatient care of individuals with diabetic foot disease, efforts should be made to establish specialised wards or sub-wards dedicated to diabetic foot disease, capable of implementing a comprehensive treatment model for individuals requiring hospitalisation. In particular, this model should include simultaneous surgical intervention and comprehensive diabetological and internal medicine management, based primarily on the provisions set out in the Regulation of the Minister of Health of 10 July 2023 on the pilot programme for care of individuals with diabetic

foot disease, together with subsequent amendments (Regulation of the Minister of Health of 16 January 2025 amending the regulation on the pilot programme for care of individuals with diabetic foot disease) – see Annex 1.

I. Definition

Diabetic foot disease is defined as infection and/or ulceration and/or destruction of deep tissues of the foot (e.g. bone) resulting from damage to peripheral nerves and/or blood vessels of the foot, with varying degrees of severity. This definition implies a classification into neuropathic, ischaemic and mixed (neuro-ischaemic) diabetic foot disease. Diagnosis of diabetic foot disease includes assessment of the presence of peripheral polyneuropathy, disturbances of lower limb perfusion, deformities, and other risk factors for foot injury. If loss of protective pain sensation is identified, inspection of the individual's feet by a physician at every visit is recommended.

II. Risk factors for the development of diabetic foot disease

The most important risk factors for the development of diabetic foot disease are:

- peripheral neuropathy and/or features of lower limb ischaemia,
- lack of knowledge on the part of the individual,
- long-standing, poorly controlled diabetes,
- inadequate foot hygiene,
- improperly fitted footwear,
- presence of calluses,
- foot deformities,
- increased plantar pressure,
- tobacco smoking.

Factors predisposing to ulcer recurrence:

- previous amputations,
- history of foot ulceration,
- Charcot neuro-osteoarthropathy.

III. Prevention

- systematic foot examination; once a year assessment for sensory disturbances (physical examination using a 10 g monofilament and a 128 Hz tuning fork; if a monofilament or tuning fork is unavailable, assessment of sensory loss may be performed by lightly touching the toes with the examiner's index finger for 2–3 seconds) and ischaemia (assessment of pulses in the dorsalis pedis and posterior tibial arteries; if pulses in the dorsalis pedis/posterior tibial artery are not palpable, ankle–brachial index measurement is recommended); further diagnostic evaluation of ischaemia should also be considered, in consultation with a vascular surgeon or angiologist, in all individuals; the frequency of foot examinations depending on ulceration risk assessment is presented in Table 22.1,
- regular podiatric care (removal of calluses and hyperkeratosis),
- assessment of footwear and existing deformities, as well as the individual's knowledge regarding ulcer risk,
- systematic education of individuals and their families or caregivers regarding principles of ulcer prevention, with particular emphasis on appropriate footwear selection,
- treatment and/or elimination of other risk factors such as tobacco smoking, overweight, arterial hypertension and lipid disorders; regular monitoring of metabolic control with treatment adjustment according to glycaemia and glycated haemoglobin (HbA_{1c}) values,
- early detection and treatment of lower limb ischaemia,
- walking exercise may be recommended exclusively in individuals without plantar foot ulceration, provided that appropriately fitted footwear is used.

Individuals with plantar ulceration should perform exercises with offloading.

IV. Clinical classification of foot ulceration

The PEDIS classification is recommended [P – perfusion, E – extent, D – depth, I – infection, S – sensation], taking into account both infection and the ischaemic component (Table 22.2), as well as the SINBAD classification (Table 22.3).

V. Infections in the course of diabetic foot disease

1. Diagnosis of infection is based primarily on the clinical picture (presence of at least 2 of the classical subjective and objective signs of infection), and not solely on microbiological or laboratory test results.
2. Assessment of infection severity (see PEDIS classification).
3. Microbiological examination (including antibiogram) and its interpretation (colonisation, contamination, infection):

- collection of tissue fragments or aspirate after wound debridement and irrigation with ste-

Table 22.1. Frequency of preventive foot examinations according to risk category (risk stratification system of the International Working Group on the Diabetic Foot)

Category	Risk of ulceration	Characteristics	Examination frequency
0	Very low	Preserved protective sensation, no ischaemia	Once a year
1	Low	Sensory loss or ischaemia present	Once every 6–12 months
2	Moderate	Sensory loss and ischaemia present, or sensory loss with foot deformity, or ischaemia with foot deformity	Once every 3–6 months
3	High	Sensory loss or ischaemia present and one of the following: · previous ulceration · previous amputation · end-stage renal disease	Once every 1–3 months

rile solution is recommended; swab sampling should be performed only if no other option is available – it is not recommended because results are less reliable,
- required in the presence of a clinically infected wound,
- interpretation of culture results in the assessment of infection is difficult – clinical presentation should be the primary determinant,
- blood cultures are recommended only in cases of severe clinical infection with systemic signs of infection (fever > 38.6°C or hypothermia < 36°C) and erythrocyte sedimentation rate (ESR) > 70 mm/h or C-reactive protein (CRP) > 100 mg/l or leukocytosis > 20 × 10^3/µl,
- in the presence of clinically non-infected wounds, microbiological testing is not indicated; in mildly infected wounds, if antibiotics have not been used previously, omission of culture and initiation of empirical antibiotic therapy is acceptable.

4. Investigations for osteomyelitis (should be performed in every case of infected ulceration, especially of long duration):
- assessment using the probe-to-bone test; a positive test (bone palpable at the base of the ulcer) raises suspicion of osteomyelitis,
- radiography of foot bones (every 3–6 weeks); the preferred subsequent imaging modality is magnetic resonance imaging; SPECT, PET or

Table 22.2. PEDIS classification

	Grade			
	1	2	3	4
Perfusion	Features of normal perfusion: palpable pedal pulses or ABI > 0.9	Clinical signs of impaired circulation: presence of intermittent claudication, ABI < 0.9, $TcpO_2$ 30–60 mm Hg	Critical limb ischaemia: rest pain, ABI < 0.4, $TcpO_2$ < 30 mm Hg	–
Extent (size)	The dimension of the wound is determined in square centimetres			
Depth (tissue loss)	Superficial ulceration not extending beyond the dermis	Wound may involve all soft tissues	Penetration of infection to bone: visible on X-ray, features of osteolysis or bone palpable with a probe	–
Infection severity	No clinical signs of infection	Infection involving the skin and subcutaneous tissue. Area of inflammation does not extend more than 2 cm beyond the ulcer margin	Local intensification of inflammatory signs. Area extends beyond 2 cm, but no systemic signs of infection	Signs of systemic infection: fever > 38°C, heart rate > 90/min, respiratory rate > 20/min, leukocytosis > 12 × 10^3/µl or < 4 × 10^3/µl
Sensory neuropathy	No evidence of sensory neuropathy on basic testing: monofilament and tuning fork or neurotip	Presence of sensory neuropathy		

Table 22.3. SINBAD classifications

Category	Definition	Score
Site	Forefoot Midfoot/rearfoot (heel region)	0 1
Ischaemia	Normal blood supply to the foot – palpable pulse in at least one artery Clinical signs of ischaemia	0 1
Neuropathy	Preserved sensation Loss of sensation	0 1
Infection	Absent Present	0 1
Area	< 1 cm^2 ≥ 1 cm^2	0 1
Depth	Ulcer limited to skin and subcutaneous tissue Ulcer involving muscle, tendon or deeper structures	0 1
Total		6

labelled leukocyte scintigraphy may also be used,
- bone biopsy or culture from a bone fragment and histopathological examination (recommended); bone biopsy is essential if the diagnosis of osteomyelitis is uncertain or if identification of the pathogen is required,
- laboratory tests – ESR > 70 mm/h increases the likelihood of osteomyelitis; lower values indicate lower risk; CRP and leukocyte count may also be helpful; normal laboratory results do not fully exclude the presence of bone inflammation.

5. The primary criterion for dressing selection is wound character – dry or exudative.
6. Principles of antibiotic therapy:
- use only in confirmed infections (not prophylactically),
- do not delay initiation of therapy,
- initial therapy should target the most common flora (staphylococci and streptococci),
- duration of antibiotic therapy – until resolution of clinical signs of infection (not until wound healing):
  - » soft tissue infection (without bone involvement): 1–2 weeks,
  - » extended to 3–4 weeks if the infection is extensive and improves more slowly or if ischaemia coexists,
  - » in osteomyelitis, antibiotic duration is the same as for soft tissue infection if infected bone has been radically removed; 3 weeks if minor amputation was performed with residual infected bone; and 6 weeks in osteomyelitis without bone resection or amputation. To confirm remission of osteomyelitis, assessment should be performed after a minimum 6-month follow-up period following completion of antibiotic therapy. Prolongation of antibiotic therapy should also be considered when signs of infection decrease but more slowly than expected due to the extent of foot surgery,
- route of administration:
  - » intravenous – PEDIS grade 4 infections and justified cases of PEDIS grade 3 infections (MRSA infection, *Pseudomonas aeruginosa*), intolerance of oral antibiotics,
  - » oral – PEDIS grade 2 and 3 infections and after improvement in PEDIS grade 4 infections,
  - » topical – use of a gentamicin-impregnated collagen sponge (gentamicin sponge) may be considered as adjunctive therapy to systemic antibiotics if pathogen susceptibility to gentamicin has been demonstrated,
  - » intra-arterial – not recommended.

7. Empirical antibiotic therapy based on clinical presentation and microbiological data

A. Mild infections – usually Gram-positive cocci:
- MSSA staphylococci – cloxacillin,
- streptococci – amoxicillin, cephalexin.

In case of intolerance to β-lactam antibiotics – clindamycin, trimethoprim–sulfamethoxazole, doxycycline.

B. Recent exposure to antibiotics.

Infection may be caused by Gram-positive and Gram-negative bacteria – amoxicillin/clavulanate, ampicillin/sulbactam or trimethoprim–sulfamethoxazole.

C. High risk of MRSA infection (previous MRSA infection or colonisation, prolonged hospitalisation, intensive care admission, recent hospitalisation, recent antibiotic use, procedures, invasive HIV infection, admission from long-term care facilities, open wounds, haemodialysis, current central venous access).

Glycopeptides – only in the case of concurrent isolation of MRSA from peripheral blood.

In local infection with negative blood cultures, glycopeptides – vancomycin, teicoplanin (due to poor penetration into the foot, approx. 3% of the therapeutic dose) – should be replaced with antibiotics penetrating skin and subcutaneous tissue:

- ceftaroline,
- doxycycline,
- trimethoprim–sulfamethoxazole,
- linezolid,
- clindamycin,
- dalbavancin,
- daptomycin,
- fusidic acid (not available orally or intravenously in Poland).

D. Moderate or severe infection of mixed Gram-positive and Gram-negative aetiology without prior antibiotic therapy:
- amoxicillin/clavulanate,
- ampicillin/sulbactam,
- second- and third-generation cephalosporins (cefuroxime, cefotaxime, ceftriaxone).

E. Moderate or severe infection of mixed Gram-positive and Gram-negative aetiology with prior antibiotic therapy:
- ticarcillin/clavulanate,
- piperacillin/tazobactam,
- second- and third-generation cephalosporins (cefuroxime, cefotaxime, ceftriaxone),
- carbapenems (ertapenem).

F. Refractory infection of mixed Gram-positive, Gram-negative and *Pseudomonas aeruginosa* aetiology:
- ticarcillin/clavulanate,
- piperacillin/tazobactam,
- semi-synthetic penicillin resistant to penicillinase – cloxacillin + ceftazidime,
- carbapenems (meropenem, imipenem),
- fosfomycin i.v.

G. Ischaemic limb/necrosis/gas gangrene formation:
- amoxicillin/clavulanate,
- ampicillin/sulbactam,
- ticarcillin/clavulanate,
- piperacillin/tazobactam,
- carbapenems (ertapenem, meropenem, imipenem),
- third-generation cephalosporin (cefuroxime, cefotaxime, ceftriaxone) + clindamycin or metronidazole.

H. Risk of resistant Gram-negative bacilli:
- carbapenem (ertapenem, meropenem, imipenem) + aminoglycoside (amikacin) or + colistin,
- carbapenem + fosfomycin i.v.,
- colistin + fosfomycin i.v.

I. *Enterococcus faecalis* infection with preserved susceptibility:
- ampicillin i.v.,
- amoxicillin orally or i.v.,

and in case of β-lactam intolerance or resistance:
- vancomycin,
- teicoplanin,
- daptomycin,
- linezolid orally or i.v.

J. *Enterococcus faecium* infection:
- vancomycin,
- teicoplanin,
- daptomycin,
- linezolid orally or i.v.

Note!

According to the most recent recommendations of the IDSA, FDA, the National Medicines Institute and the announcement of the President of the Office dated 26 May 2023 regarding the European Medicines Agency communication on fluoroquinolone antibiotics and agents used to reduce the risk of long-lasting and potentially irreversible adverse effects, fluoroquinolone antibiotics (ciprofloxacin, levofloxacin, moxifloxacin) should not be used in empirical therapy.

VI. Multidisciplinary management of diabetic foot disease

Management of diabetic foot disease should be provided within specialised multidisciplinary clinics. This concept includes an organisational structure that enables the individual to access consultations with the required specialists who have knowledge and experience in the management of diabetic foot disease and who form a team that remains in continuous communication.

Management of diabetic foot disease includes:
- optimisation of metabolic control of diabetes in accordance with general principles of its treatment.
- Offloading is a key component of therapy. The preferred method of offloading for a neuropathic foot with a non-infected ulcer located on the plantar surface of the forefoot or midfoot is non-removable offloading. Such offloading may be recommended and applied only by appropriately trained personnel and should preferably extend to the knee; however, if this is not possible or not accepted by the individual, ankle-level devices may be used. Other offloading methods include temporary offloading footwear for the forefoot or heel, therapeutic insoles, crutches, wheelchairs, specialist footwear and limitation of walking, including in the home environment. In the presence of infection and/or limb ischaemia,

and in situations where experienced personnel are unavailable, removable offloading devices (knee-high ankle–foot orthoses) are the first and subsequent choice. When deciding on the method of limb offloading, the individual's condition and functional capacity, the presence of comorbidities, patient preference and the level of team training should be taken into account. In many individuals, particularly those with loss of protective pain sensation, ischaemia and existing deformities, the use of appropriate, individually fitted footwear insoles is recommended to correct excessive plantar pressure forces and to prevent ulceration or ulcer recurrence.

- In the presence of infection, antibiotic therapy (oral or intravenous) should be instituted as described above.
- Surgical procedures include removal of necrotic tissue, drainage and incision. Flexor tenotomy may be considered when toe deformities cause recurrent ulceration. Achilles tendon lengthening and metatarsal head resection may also be considered if an ulcer does not heal despite appropriate offloading.
- Endovascular procedures, conventional vascular surgery and hybrid procedures should be considered in diabetic foot disease with a predominant ischaemic component. Individuals with a low ankle–brachial index (< 0.5), transcutaneous oxygen pressure ($TcPO_2$) < 25 mm Hg and/or a history suggestive of intermittent claudication should be referred for urgent further vascular assessment and subsequently to a vascular surgeon or angiologist. Imaging diagnostics and revascularisation should also be considered, even when the above test results are normal, if there is no progress in wound healing within 4 weeks despite standard management. It should be emphasised that in many individuals with diabetes, lower limb ischaemia may occur without typical pain symptoms. The aim of revascularisation should be restoration of blood flow to at least one artery, preferably the artery supplying the anatomical region of the ulcer.
- Regular wound debridement should be performed, with frequency depending on local wound conditions.
- Conventional dressings and therapies providing an appropriate wound environment should be used. The use of dressings employing TLC-NOSF technology should be considered in non-infected ulcers of neuropathic–ischaemic aetiology (without features of critical or significant ischaemia) that fail to heal despite optimal standard treatment.
- Other therapeutic options include hyperbaric oxygen therapy delivered in chambers meeting required standards for ischaemic and neuro-ischaemic ulcers that do not heal despite standard management; negative pressure wound therapy, particularly for postoperative wounds in individuals in whom ischaemia and infection do not constitute significant contraindications, used alongside standard care; and agents improving perfusion in ischaemic foot disease or disease with a predominant vascular component, such as low-molecular-weight heparins (acute ischaemic states, critical limb ischaemia), acetylsalicylic acid and supervised walking exercise (should be used only in the absence of a plantar ulcer). The use of preparations derived from autologous leukocytes, platelet-rich fibrin (for ulcers without ischaemia or with mild ischaemia), topical oxygen therapy and dressings derived from human placental tissue may be considered as adjunctive therapies for the treatment of non-infected ulcers. Treatment with sulodexide may also be considered.

Transplantation of "artificial skin", growth factors, ozone therapy and autologous platelet gel are not recommended. In justified cases, wound debridement may include the use of larvae of *Lucilia sericata* bred under sterile conditions in specialised laboratories.

Every individual with diabetic foot disease should receive education on ulcer prevention.

VII. Charcot neuro-osteoarthropathy (Charcot foot)

1. Diagnosis

The diagnosis is established on the basis of medical history and clinical presentation (most commonly unilateral swelling, erythema and increased temperature of the foot, particularly in the absence of ulceration, in an individual with features of diabetic polyneuropathy), after exclusion of other causes, especially deep vein thrombosis, gout and other abnormalities detectable on imaging studies. If these abnormalities are not visible on standard radiography (weight-bearing radiograph in the standing position), magnetic resonance imaging should be performed; if MRI is unavailable, computed tomography should be used. When

these imaging modalities are not available, the individual should be managed and treated as having probable neuro-osteoarthropathy.

2. Treatment

- Active phase – continuous offloading extending to the knee is required (shorter devices are not recommended). Treatment with bisphosphonates or calcitonin is not recommended. Supplementation with vitamin D and calcium should follow general recommendations. Offloading should be maintained until stabilisation of the disease process and transition to the inactive phase. Monitoring of disease activity may be considered using skin temperature measurements on the foot surface with comparison between both feet (no clearly established cut-off point) and radiological imaging indicating consolidation. Return to full weight-bearing should be very gradual.
- Inactive phase – education, foot hygiene, specialist orthopaedic footwear with therapeutic insoles correcting existing deformities, and orthopaedic surgical procedures to correct deformities (exostectomy, arthrodesis). Management by a multidisciplinary specialist team is recommended.

VIII. Hospitalisation – indications

Emergency admission:

- infection graded as PEDIS grade 4,
- infection graded as PEDIS grade 3 when intravenous antibiotic therapy is required,
- any case of critical limb ischaemia – admission to an interventional unit capable of urgent revascularisation,
- admission to a unit with the ability to perform urgent vascular imaging (CT angiography or MR angiography), followed by urgent specialist consultation to determine eligibility for revascularisation.

Planned admission:

- lack of improvement after two months of outpatient treatment,
- preparation for planned surgical procedures (minor amputation, skin grafting, limited bone resections, revascularisation procedures),
- orthopaedic correction of deformities in the course of Charcot neuro-osteoarthropathy.

IX. Amputation

Before any amputation, assessment of limb perfusion is mandatory.

1. "Major" amputation (above the ankle) – should be considered when the following occur:

- life-threatening condition caused by infection or extensive necrosis (absolute indication),
- severe, debilitating pain refractory to treatment, particularly as a consequence of ischaemia (relative indication),
- loss of the weight-bearing function of the foot (relative indication).

2. "Minor" amputation (below the ankle) – should be considered when the following occur:

- wet necrosis,
- osteomyelitis of the distal phalanges of the toes (avoidance of prolonged antibiotic therapy – accelerates healing),
- inflammation involving the metatarsophalangeal joints, in order to perform their resection as a limb-sparing bone procedure,
- in the case of dry necrosis, a wait-and-see approach with expectation of autoamputation is recommended, with continuous monitoring for signs of infection.

The choice of the level of amputation depends on the state of limb perfusion and reconstructive and rehabilitation possibilities. The most tissue-sparing amputation possible is recommended.

3. An indication for amputation may also be a chronically treated wound in the case of deterioration of the individual's condition and lack of treatment progress, taking into account the individual's preferences.
4. The choice of the level of amputation depends on the state of limb perfusion and reconstructive and rehabilitation possibilities. The most tissue-sparing amputation possible is recommended.

REFERENCES

1. Blume PA, Walters J, Payne W, et al. Comparison of negative pressure wound therapy using vacuum-assisted closure with advanced moist wound therapy in the treatment of diabetic foot ulcers: a multicenter randomized controlled trial. Diabetes Care 2008; 31: 631–636.
2. Bonnet E, Maulin L, Senneville E, et al.; individual members of the "Review group". Clinical practice recommendations for infectious disease management of diabetic foot infection (DFI) – 2023 SPILF. Infect Dis Now 2024; 54: 104832. DOI: 10.1016/j.idnow.2023.104832.
3. Bus SA, Waaijman R, Arts M, et al. Effect of custom-made footwear on foot ulcer recurrence in diabetes: a multicenter randomized controlled trial. Diabetes Care 2013; 36: 4109–4116.

4. Chen P, Vilorio NC, Dhatariya K, et al. Guidelines on interventions to enhance healing of foot ulcers in people with diabetes (IWGDF 2023 update). Diabetes Metab Res Rev 2023; e3644.
5. Cohen M, Cerniglia B, Gorbachova T, et al. Added value of MRI to X-ray in guiding the extent of surgical resection in diabetic forefoot osteomyelitis: a review of pathologically proven, surgically treated cases. Skeletal Radiol 2019; 48: 405–411.
6. Edmonds M, Lazaro-Martinez JL, Alfayate-Garcia JM, et al. Sucrose octasulfate dressing versus control dressing in patients with neuroischaemic diabetic foot ulcers (Explorer): an international, multicentre, double-blind, randomised, controlled trial. Lancet Diabet Endocrinol 2018; 6: 186–196.
7. Frykberg RG. Topical wound oxygen therapy in the treatment of chronic diabetic foot ulcers. Medicina (Kaunas) 2021; 57: 917.
8. Frykberg RG, Franks PJ, Edmonds M, et al. A multinational, multicenter, randomized, double-blinded, placebo-controlled trial to evaluate the efficacy of cyclical topical wound oxygen (TWO2) therapy in the treatment of chronic diabetic foot ulcers: the TWO2 Study. Diabetes Care 2020; 43: 616–624.
9. Gariani K, Lebowitz D, von Dach E, et al. Remission in diabetic foot infections: duration of antibiotic therapy and other possible associated factors. Diabetes Obes Metab 2019; 21: 244–251.
10. Ince P, Abbas ZG, Lutale JK, et al. Use of the SINBAD classification system and score in comparing outcome of foot ulcer management on three continents. Diabetes Care 2008; 31: 964–967.
11. Jeon BJ, Choi HJ, Kang JS, et al. Comparison of five systems of classification of diabetic foot ulcers and predictive factors for amputation. Int Wound 2017; 14: 537–545.
12. Lauri C, Tamminga M, Glaudemans AWJM, et al. Detection of osteomyelitis in the diabetic foot by imaging techniques: a systematic review and meta-analysis comparing mri, white blood cell scintigraphy, and FDG-PET. Diabetes Care 2017; 40: 1111–1120.
13. Lipsky BA, Berendt AR, Cornia PB, et al. Infectious Diseases Society of America clinical practice guideline for the diagnosis and treatment of diabetic foot infections. Clin Infect Dis 2012; 54: e132–173.
14. Lipsky BA, Senneville É, Abbas ZG, et al.; International Working Group on the Diabetic Foot (IWGDF). Guidelines on the diagnosis and treatment of foot infection in persons with diabetes (IWGDF 2019 update). Diabetes Metab Res Rev 2020; 36 Suppl 1: e3280. DOI: 10.1002/dmrr.3280.
15. Lo ZJ, Lin Z, Pua U, et al. Diabetic foot limb salvage – a series of 809 attempts and predictors for endovascular limb salvage failure. Ann Vasc Surg 2018; 49: 9.
16. Löndal M. Hyperbaric oxygen therapy as adjunctive treatment of diabetic foot ulcers. Med Clin North Am 2013; 97: 957.
17. Prutsky G, Domecq JP, Tsapas A, et al. A systematic review and meta-analysis of off-loading methods for diabetic foot ulcers. FJ Vasc Surg 2016; 63: 59S–68S.
18. Rizzo L, Tedeschi A, Fallani E, et al. Custom-made orthesis and shoes in a structured follow-up program reduces the incidence of neuropathic ulcers in high-risk diabetic foot patients. Int J Low Extrem Wounds 2012; 11: 59–64.
19. Senneville É, Albalawi Z, van Asten SA, et al. Diagnosis of infection in the foot of patients with diabetes: a systematic review. Diabetes Metab Res Rev 2023; e3723.
20. Sheehan P, Jones P, Caselli A, et al. Percent change in wound area of diabetic foot ulcers over a 4-week period is a robust predictor of complete healing in a 12-week prospective trial. Diabetes Care 2003; 26: 1879–1882.
21. Ulbrecht JS, Hurley T, Mauger DT, et al. Prevention of recurrent foot ulcers with plantar pressure-based in-shoe orthoses: the CareFUL prevention multicenter randomized controlled trial. Diabetes Care 2014; 37: 1982–1989.
22. Wukich DK, Schaper NC, Gooday C, et al. Guidelines on the diagnosis and treatment of active charcot neuro-osteoarthropathy in persons with diabetes mellitus (IWGDF 2023). Diab Metab Res Rev 2023; e3646.
23. https://www.urpl.gov.pl/pl/informacja-prezesa-urz%C4%99du-z-dnia-26-maja-2023-r-w-sprawie-publikacji-europejskiej-agencji-lek%C3%B3w-dot

23. Diabetes in Children and Adolescents: Standards of Care

CHAPTER HIGHLIGHTS
• From the moment of diabetes diagnosis, children and adolescents as well as their caregivers/family members should receive holistic care provided by a multidisciplinary specialised care team (specialist physician – team leader, nurse – diabetes educator, dietitian, psychologist). [**A**]
• From the moment of diabetes diagnosis in children and adolescents, a care plan should be implemented using appropriate educational and digital tools in order to achieve individual therapeutic goals. [**A**]
• In children and adolescents with diabetes, the aim should be to achieve TIR ≥ 80% and CV < 36%, as well as HbA_{1c}/GMI ≤ 6.5%, while minimising episodes of hypoglycaemia and maintaining good quality of life. [**B**]
• During the remission phase of the disease or when using automated insulin delivery systems, the time in tight range (TITR; 70–140 mg/dl) should be assessed. The recommended targets are TITR > 55% and CV < 33%. [**E**]
• Children and adolescents with diabetes should use continuous glucose monitoring systems from the time of diagnosis. [**A**]
• Glucose levels should be assessed at least 8 times per day: fasting and before meals, before sleep, before, during and after physical activity, and in situations of poor well-being; according to need, also 1–2 hours after meals and at night. [**A**]
• The use of continuous glucose monitoring systems in children and adolescents improves metabolic control of diabetes (increases TIR, reduces CV, lowers HbA_{1c}), reduces the risk of acute and chronic complications, and prolongs survival. [**A**]
• Children and adolescents with type 1 diabetes, from the initiation of insulin therapy (except during the remission phase), should be treated with functional intensive insulin therapy; therapy using automated insulin delivery systems is preferred. If their use is not possible, systems with predictive low-glucose insulin suspension are preferred. [**A**]

This chapter outlines modifications to the general recommendations that reflect the specific characteristics of childhood and adolescence.

I. Diagnosis and forms of diabetes in the developmental age

1. In children and adolescents, the same tests as in adults are used to diagnose diabetes. In the case of asymptomatic hyperglycaemia, the diagnosis of diabetes requires two abnormal test results from the same sample or from two separate samples.
   An HbA_{1c} value < 6.5% does not exclude diabetes diagnosed using glucose-based tests. The role of HbA_{1c} alone in the diagnosis of type 1 diabetes in children is unclear.
2. The most common form is type 1 diabetes of autoimmune pathogenesis.
3. Screening tests (OGTT with glucose measurements at 30, 60, 90 and 120 minutes or HbA_{1c}) to detect type 2 diabetes should be performed in children after the onset of puberty or after 8 years of age who have a BMI ≥ the 85th percentile for age and sex and present risk factors for the development of type 2 diabetes. If test results are normal, screening should be repeated at intervals of no less than every 2 years. Annual screening is required if BMI increases, the cardiometabolic risk profile worsens, there is a positive family history of type 2 diabetes and/or evidence of prediabetes. Type 2 diabetes in children and adolescents is characterised by earlier onset and more rapid progression of chronic complications compared with adults.
4. It should be noted that in Poland monogenic diabetes is the second most common form of diabetes in the paediatric population. Indications for diagnostic evaluation for monogenic diabetes are presented in Chapter 1.
5. Cystic fibrosis–related diabetes (CFRD) is usually asymptomatic. In children over 10 years of age with cystic fibrosis, an OGTT with glucose measurements fasting and at 30, 60, 90 and 120 minutes should be performed annually.
6. Initial evaluation of hyperglycaemia or revision of the diabetes diagnosis includes measurement of glutamic acid decarboxylase (GAD) autoantibodies, insulinoma-associated autoantigen-2 (IA-2) autoantibodies, zinc transporter 8 (ZnT8) autoantibodies, and islet cell antibodies (ICA); these tests should be performed in a reference laboratory.

For the detection of preclinical stage of type 1 diabetes, measurement of the above antibodies is recommended in the general population (screen-

ing), particularly in individuals with a family history of type 1 diabetes.

Detection of diabetes at the preclinical stage, combined with monitoring for disease progression, allows for:

- a significant reduction in the risk of consequences of hyperglycaemia (including diabetic ketoacidosis and central nervous system injury),
- a milder introduction of insulin therapy,
- prolongation of clinical remission,
- participation in research projects aimed at preventing progression to clinically overt diabetes.

The highest sensitivity is achieved with three testing time points (at ages 2 and 6, and 10–15 years). In the case of a single screening test, the optimal age is 3 years.

Chapter 1 presents the criteria for the diagnosis of preclinical stages of type 1 diabetes according to the American Diabetes Association (ADA). In children, dysglycaemia (stage 2) may also be diagnosed in the case of glucose levels > 200 mg/dl at 30, 60 or 90 minutes of the OGTT.

Due to the 100% lifetime risk of progression to stage 3 type 1 diabetes, patients in preclinical stages require therapeutic education on a healthy lifestyle (the preclinical period is prolonged by maintaining normal body weight, regular physical activity and vitamin D_3 supplementation) and assessment of glucose values. Patients and their families should also be provided with psychological support.

In children in the preclinical phase of type 1 diabetes, to monitor disease progression, an OGTT (with glucose measurements also at 30, 60 and 90 minutes) and HbA_{1c} should be performed at least once a year. In patients with stage 2 type 1 diabetes, continuous glucose monitoring (CGM) should be used periodically (at least 4 times per year).

7. It should be remembered that a patient may have a complex aetiology of diabetes.

II. Goals of diabetes treatment

1. Prevention of acute and chronic complications of diabetes.
2. Achievement and maintenance of normal physical development, including growth, body weight and body composition (centile charts), as well as age- and sex-appropriate pubertal development, while ensuring quality of life for the child and the family.
3. Target values of parameters to reduce the risk of vascular complications:

- efforts should be made to achieve TIR ≥ 80%, CV < 36% and HbA_{1c} ≤ 6.5%, with minimisation of hypoglycaemic episodes and maintenance of good quality of life; during the remission period and when using automated insulin delivery systems, a time in tight range (TITR) of 70–140 mg/dl should be assessed; the recommended TITR is > 55% and CV < 33%,
- total cholesterol < 170 mg/dl (4.4 mmol/l), LDL cholesterol < 100 mg/dl (2.6 mmol/l), triglycerides < 100 mg/dl (1.1 mmol/l),
- blood pressure < the 90th percentile for age, sex and height (from 13 years of age < 120/80 mm Hg),
- BMI < the 85th percentile for age and sex,
- moderate- or high-intensity physical activity for at least 1 hour daily,
- sleep duration: children aged 5–13 years at least 9 hours, aged 14–17 years at least 8 hours,
- non-smoking.

III. Treatment of diabetes

1. Pharmacotherapy:

Type 1 diabetes — insulin therapy:

- the method of insulin therapy should be tailored to the individual needs of the patient and accepted by both the patient and their parents or caregivers,
- the method of choice is functional intensive insulin therapy (IIT), based on continuous adjustment of insulin doses to: the current glucose concentration and its trend, the amount of consumed carbohydrates taking into account fat and protein content of meals, as well as physical activity and emotional factors; IIT should be delivered using one of the following methods:
  - » continuous subcutaneous insulin infusion using a personal insulin pump (CSII),
  - » multiple daily injections of insulin (MDI) using pen injectors with needles ≤ 6 mm in length; the use of technologies supporting recording of administered insulin doses is beneficial,
- indications and contraindications for CSII – see the relevant chapter,
- in patients requiring intensive insulin therapy, the use of automated insulin delivery systems (AID) should be considered the standard of care; if their use is not possible, systems that automatically suspend insulin delivery in anticipation of hypoglycaemia should be considered, provided these therapeutic methods are accepted by the patient and their parents or

caregivers and there are no contraindications (guidelines of the Polish Society of Endocrinology and Diabetology for Children and Adolescents and the Pediatric Section of Diabetes Poland concerning insulin therapy using hybrid closed-loop systems in children and adolescents with diabetes in Poland – https://doi.org/10.5114/pedm.2024.144041),

- in both CSII and MDI, the use of a bolus calculator function is recommended from the initiation of therapy, as it increases glycaemic stability and reduces the risk of hypoglycaemia and hyperglycaemia; regular verification and adjustment of bolus calculator settings is required,
- the choice of rapid-acting or ultra-rapid-acting insulin analogues and long-acting or ultra-long-acting insulin analogues should be individualised, taking into account pharmacological differences between preparations and their registered indications; when using AID systems, ultra-rapid-acting analogues increase time in range (TIR); in MDI therapy, ultra-long-acting analogues reduce the risk of hypoglycaemia and allow for less rigid timing of insulin administration and usually do not require dose reduction during physical activity (see Chapter 7),
- in the paediatric population, daily insulin requirements show considerable variability; they are highest during puberty but should not exceed 1.5 units/kg body weight/day; high insulin requirements are often associated with lack of physical activity, excessive carbohydrate intake, obesity or other coexisting diseases; in the presence of obesity, the addition of a GLP-1 receptor agonist is recommended,
- in functional IIT, the basal insulin dose (20–50% of the total daily dose) and its profile depend on the child's age and the type of personal insulin pump used; when AID systems are applied, the basal dose depends on the algorithm used,
- short-acting and rapid-acting insulin should preferably be administered before meals, respectively 20–30 minutes and 15–20 minutes prior to eating, whereas ultra-rapid-acting insulin analogues should be administered 2–10 minutes before a meal; in the youngest children, due to the inability to plan meal timing and size, splitting the dose and administering half before the meal and half during or after the meal should be considered; administration of the entire dose after the meal should be used only exceptionally,
- insulin injection or infusion sites should be systematically rotated; insulin must not be administered into areas of lipohypertrophy or lipoatrophy,
- in patients treated with CSII and with low insulin requirements, dilution of insulin is acceptable.

Type 2 diabetes – in this age group the following may be used: metformin, insulin, GLP-1 receptor agonists and SGLT2 inhibitors (with age restrictions according to the Summary of Product Characteristics).

In newly diagnosed type 2 diabetes and:

- absence of symptoms, HbA_{1c} < 8.5% and no ketosis/acidosis, pharmacotherapy in children may be initiated with lifestyle modification and metformin (with gradual dose escalation up to 2 g/day or the maximum tolerated dose); in the case of metformin intolerance, initiation of a GLP-1 receptor agonist or an SGLT2 inhibitor should be considered,
- presence of symptoms and/or HbA_{1c} ≥ 8.5% without diabetic ketoacidosis, treatment should be initiated with metformin and basal insulin (at a dose of 0.25–0.5 units/kg body weight/day),
- presence of diabetic ketoacidosis – initial treatment should follow the principles used in type 1 diabetes (intravenous insulin administration).

After correction of acute metabolic disturbances and initial normalisation of glycaemia, insulin therapy should be withdrawn (usually within 2–6 weeks).

In patients with a longer disease duration, if adequate glycaemic control is not achieved (HbA_{1c} ≥ 6.5%) despite metformin therapy, a second glucose-lowering agent should be added (a GLP-1 receptor agonist – preferred in the presence of obesity – and/or an SGLT2 inhibitor). If the therapy is ineffective or the use of a GLP-1 receptor agonist and/or an SGLT2 inhibitor is not possible, basal insulin should be initiated. If body weight normalisation is not achieved despite stabilisation of glycaemic control (HbA_{1c} < 6.5%), initiation of a GLP-1 receptor agonist should be considered.

In selected cases, bariatric treatment may be considered.

Monogenic diabetes or diabetes associated with genetic syndromes – treatment depends on the type of disease (see Chapter 1).

Diabetes related to cystic fibrosis – see Chapter 1.

2. Nutrition in children and adolescents with diabetes

The basic principles of healthy nutrition in children with diabetes are the same as in their peers without diabetes. Maintenance of an appropriate energy balance is recommended, with carbohydrate intake optimally at 45% of total daily energy intake. Simple sugars should be limited to 10% of total daily energy intake, and vegetable portions should be included in every meal.

It is necessary to estimate the amount of carbohydrates consumed in meals, among others for appropriate insulin dose adjustment and preparation for physical activity (currently, counting grams of carbohydrates is preferred over carbohydrate exchange units).

Attention should be paid to adequate fluid intake.

Dietary education is required from the time of diabetes diagnosis.

3. Self-monitoring:

- glucose monitoring should be performed using continuous glucose monitoring systems (CGM) and interpretation of their data; CGM is recommended for every child and adolescent from the onset of the disease,
- if CGM cannot be used or is not accepted, capillary blood glucose measurements should be performed using a glucometer,
- the frequency of glucose data interpretation should be individualised; in functional IIT, it should be no less than 8 times per day; glucose levels should be assessed fasting and before meals, 1–2 hours after meals, at bedtime, before, during and after physical activity; nocturnal glucose profiles should also be assessed; in the event of feeling unwell, glucose concentration should be measured immediately.

The use of CGM systems requires diabetes education covering correct interpretation of current readings, therapy modification based on glucose dynamics (trend arrows), and retrospective analysis of results according to time in range (TIR) recommendations (see Chapter 4). When CGM is used, education should be expanded to include correct sensor calibration (if required) and appropriate selection and programming of alarm and notification thresholds. If CGM glucose values are inconsistent with clinical symptoms, blood glucose should be measured using a glucometer; in the case of hyperglycaemia, ketonaemia/ketonuria should also be assessed.

The use of CGM systems enables more effective adjustment of insulin doses to glucose trends, thereby increasing glycaemic stability, reducing the number of hypoglycaemic episodes, improving metabolic control and quality of life of patients and their caregivers, and reducing cardiovascular risk.

Only continuous use of CGM is therapeutically effective (at least 70% of the time).

Hypoglycaemia unawareness or frequent hypoglycaemic episodes additionally indicate the need for CGM and AID systems (recommended), or alternatively insulin pumps integrated with CGM featuring automatic insulin suspension in anticipation of hypoglycaemia.

Measurement of blood β-hydroxybutyrate using test strips is a more sensitive indicator of ketonaemia than urine ketone testing.

4. Therapeutic education:

- is a key component of diabetes management and should always involve the child and their caregivers,
- the patient and/or their parents or caregivers require education covering principles of diabetes self-management, including modern technologies used in diabetes care, as well as regular re-education according to individual needs; all individuals providing permanent or temporary care for the child must be educated,
- therapeutic education in the preclinical stages of type 1 diabetes should be appropriately adapted and focused on issues relevant to this stage of the disease,
- educational methods and programmes should be diversified and adapted to the child's age, intellectual abilities and the caregiving role of parents or caregivers,
- in adolescents and young adults, particular attention should be paid to prevention of acute and chronic diabetes complications, contraception, pregnancy, risk behaviours and addictions; acquisition of self-management skills should be gradual, as both premature and delayed transfer of responsibility to children and adolescents with diabetes is associated with treatment failure,
- workshops and camps for children, adolescents and young adults with diabetes are a beneficial and effective educational tool,
- members of the diabetes care team responsible for patients under 18 years of age attending camps or holidays without parents provide

intensive medical care on site, including night duty; legal and organisational support from administrative bodies responsible for child care is expected,
- provision and continuation of diabetes education is the responsibility of the entire diabetes care team led by a specialist physician, with a particularly important role of the diabetes educator.

5. Psychological care:
- continuous psychological care should be provided to children and adolescents with diabetes and their families from the time of diagnosis,
- care should be delivered by an experienced psychologist specialising in diabetes in childhood and adolescence, in cooperation with a child psychiatrist,
- screening for depressive disorders, eating disorders and diabetes-related distress should be performed in all patients over 12 years of age every 1–2 years and in every patient with unsatisfactory metabolic control; diabetes-related distress should also be assessed in parents or caregivers,
- periodic access to a child psychiatrist should be considered, as subclinical and clinical depressive syndromes and eating disorders are frequently observed, including anorexia nervosa (particularly in pubertal girls) and other non-specific eating disorders (eating disorders not otherwise specified – ED-NOS).

6. Additional remarks:
- involvement of the entire family in the diabetes treatment process of children and adolescents is essential; joint goal-setting is recommended, and self-education of patients and caregivers under the supervision of the therapeutic team is encouraged,
- patients should be encouraged to develop independence and gradually assume responsibility for their treatment in a manner appropriate to their age, intellectual development and emotional maturity,
- normally developing children over 10 years of age should be able to independently measure glucose levels using CGM and a glucometer, interpret results, administer insulin using a pen, change insulin pump infusion sets and CGM sensors,
- children over 13 years of age should independently perform daily diabetes self-management with ongoing support and periodic supervision from parents or caregivers.

In the presence or suspicion of social problems, cooperation with a social worker is required.

IV. Conditions coexisting with type 1 diabetes

The most common conditions include:
- autoimmune thyroiditis and coeliac disease; their course is usually oligosymptomatic or asymptomatic (e.g. glycaemic variability, disturbances in growth dynamics and pubertal development),
- IgA deficiency.

Some coexisting chronic diseases (e.g. conditions requiring systemic steroid therapy, epilepsy, Asperger syndrome, mental or intellectual disorders, obesity) may pose additional challenges in diabetes management.

V. Acute and chronic complications of diabetes (see relevant chapters):

1. Acute complications:
- in the case of blood glucose ≤ 70 mg/dl (3.9 mmol/l) or when typical clinical symptoms of hypoglycaemia occur (without severe impairment of consciousness), oral glucose should be administered at a dose of approximately 0.3 g/kg body weight; the dose depends on the current glucose level and active insulin (the maximum dose usually does not exceed 15 g of glucose in a child weighing ≥ 50 kg); blood glucose should be rechecked after 15 minutes; when using AID systems, treatment of hypoglycaemia with a smaller amount of glucose (0.15 g/kg body weight, usually up to 8 g per 15 minutes) should be considered, taking active insulin into account,
- blood glucose < 54 mg/dl (3.0 mmol/l) indicates clinically significant hypoglycaemia,
- when using CGM, clinically significant hypoglycaemia is diagnosed when sensor glucose values < 54 mg/dl persist for longer than 15 minutes,
- severe hypoglycaemia in young children is diagnosed in the presence of impaired consciousness and/or seizures (as these situations require assistance from another person, including for treatment of stage 1 and 2 hypoglycaemia),
- management of severe hypoglycaemia is described in Chapter 15,
- biochemical criteria for the diagnosis of acute hyperglycaemic states in children and adolescents are presented in Table 23.1,
- Figure 23.1 presents principles of management of diabetic ketoacidosis (DKA) in children; it

Table 23.1. Biochemical criteria for the diagnosis of acute hyperglycaemic states in children and adolescents with diabetes

Parameter	DKA*			Hyperosmolar hyperglycaemic state	Hyperosmolar DKA
	Mild	Moderate	Severe		
Plasma glucose concentration [mg/dl]	≥ 200	≥ 200	≥ 200	> 600	> 600
Venous blood pH	< 7.3	< 7.2	< 7.1	> 7.25 arterial > 7.3	< 7.3
Bicarbonate concentration [mmol/l]	< 18	< 10	< 5	> 15	< 18
Ketonemia β-hydroxybutyrate [mmol/l]	≥ 3	≥ 3	≥ 3	Absent or mild	≥ 3
Ketonuria	Moderate or severe	Moderate or severe	Moderate or severe	Absent or mild	Moderate or severe
Effective serum osmolality [mOsm/kg H_2O]	≤ 320	≤ 320	≤ 320	> 320	> 320

DKA – diabetic ketoacidosis

**For the diagnosis of DKA, all three biochemical criteria are required: 1) hyperglycaemia ≥ 200 mg/dl, 2) venous blood pH < 7.3 or bicarbonate concentration < 18 mmol/l, 3) ketonemia or ketonuria.*

is emphasised that rehydration may be carried out using 0.45% to 0.9% NaCl, or at a later stage, if appropriate, a multi-electrolyte solution,

- management of the hyperosmolar hyperglycaemic state (HHS):
  - **fluid therapy:** rapid infusion of 0.9% NaCl at a dose ≥ 20 ml/kg body weight/hour; subsequent fluid boluses should be administered until improvement in peripheral perfusion is achieved, followed by correction of fluid deficits over 24–48 hours using 0.45–0.75% NaCl; optimal rates of reduction are: serum sodium 0.5 mmol/l/hour, blood glucose 75–100 mg/dl/hour; if blood glucose decreases by > 100 mg/dl/hour during the first hours of rehydration, addition of a 2.5–5% glucose solution should be considered,
  - **insulin therapy:** insulin should be initiated when blood glucose does not decrease by at least 50 mg/dl/hour despite appropriately conducted fluid therapy alone; the initial insulin dose is 0.025–0.05 units/kg body weight/hour, then adjusted to achieve a glucose reduction rate of 50–75 mg/dl/hour,
  - **electrolytes:** deficits of sodium, potassium, phosphate and magnesium are greater than in DKA; potassium supplementation should be started as soon as renal function and diuresis are stabilised; intravenous administration of potassium phosphate and potassium chloride in a 1:1 ratio allows adequate phosphate supplementation; phosphate administration may cause hypocalcaemia; if hypomagnesaemia is identified, magnesium supplementation should be considered,
- every centre treating children with diabetes should have a written protocol for the management of DKA, specifying local indications for hospitalisation in intensive care units (ICU), taking into account staffing resources of the diabetes ward, team training and ICU availability,
- indications for care in a high-dependency unit within diabetes wards or in an ICU:
  - severe DKA (pH < 7.1) with prolonged symptom duration, circulatory disturbances and reduced level of consciousness,
  - increased risk of cerebral oedema (age < 5 years, rapidly developing acidosis, low pCO_2, high blood urea nitrogen),
  - hyperosmolar DKA.

In every case of severe DKA, an anaesthesiology consultation is required to assess eligibility for ICU treatment.

2. Chronic complications:

- regular follow-up examinations are necessary to prevent complications (Table 23.2),
- if any chronic complication is diagnosed, screening for other disorders is required (e.g. diabetic kidney disease, retinopathy, neuropathy and macroangiopathy),
- in the case of persistent albuminuria above normal values, use of an ACE inhibitor or an angiotensin II type 1 (AT1) receptor antagonist is recommended to slow its progression; treatment effectiveness requires monitoring of albuminuria,

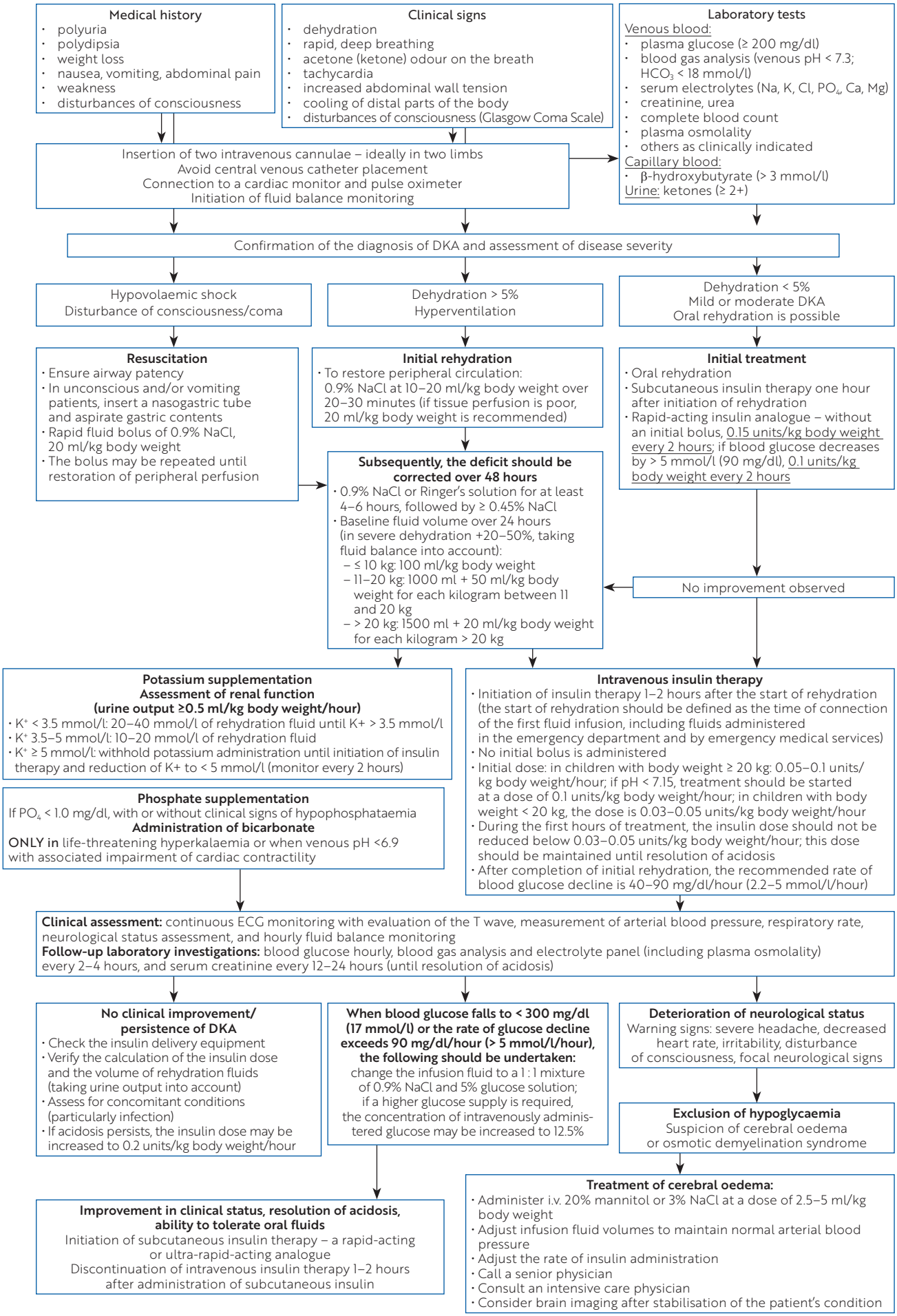


Figure 23.1. Management of diabetic ketoacidosis (DKA) in children

Table 23.2 Recommendations for diabetes care for children and adolescents with diabetes

Therapeutic education of the child with diabetes and their parents/guardians	At diagnosis and during the course of the disease; depending on the assessment of the physician or diabetes nurse educator
Education on nutritional principles for the child with diabetes and their parents/guardians	At diagnosis and during the course of the disease; depending on the assessment of the physician or diabetes nurse educator/dietitian
Psychological care for the child with diabetes and their parents/guardians	At diagnosis and during the course of the disease; depending on the assessment of the physician or diabetes nurse educator, or a psychologist as required by the patient
Diagnostics to determine the type of diabetes	At diagnosis and in the event of diagnostic revision: clinical presentation, family history, assessment of insulin secretion, measurement of pancreatic autoantibodies, assessment of insulin sensitivity*, genetic testing*
HbA_{1c}	3–4 times per year; may be measured less frequently in patients using continuous glucose monitoring (CGM) regularly
Total cholesterol, HDL-C, LDL-C, non-HDL-C, triglycerides in serum	First assessment after stabilisation of glycaemic control following diagnosis of diabetes, and subsequently: – in type 1 diabetes after the age of 10 years, with normal values, no family history of cardiovascular disease, and no other cardiovascular risk factors apart from diabetes – at least every 3 years – in type 2 diabetes – annually
Abdominal ultrasound	At diagnosis of diabetes
Monitoring of body weight and height	At each visit, using age- and sex-appropriate centile charts
Monitoring of pubertal development according to Tanner staging	According to the physician's decision, at least once per year; assessment of menstrual regularity
Blood pressure measurement	At each visit; in children <7 years of age at least twice per year; in children > 10 years of age, 24-hour ambulatory blood pressure monitoring (ABPM) every 2 years, or in the case of elevated blood pressure values on office measurements
Screening for coeliac disease	According to ESPGHAN guidelines for the diagnosis of coeliac disease – screening tests at disease onset, and subsequently every 2 years in the absence of symptoms
Assessment of thyroid function/ diagnostics of thyroid disorders	At disease onset: TSH, fT4, anti-TPO and anti-TG antibodies (thyroid ultrasound in the presence of positive antibodies and/or thyroid dysfunction); subsequently every 2 years (depending on physician's decision): TSH, anti-TPO, anti-TG
Screening for chronic complications: serum creatinine (eGFR calculation), albuminuria, urinalysis, ophthalmological consultation	To be performed after stabilisation of glycaemic control, and subsequently: – in type 1 diabetes after the age of 10 years or after >5 years' duration of diabetes – every 2 years – in type 2 diabetes – annually In the case of abnormal results, the frequency of further investigations should be individualised as required
Screening for hepatic steatosis: ALT, AST, GGTP, abdominal ultrasound	In type 2 diabetes after stabilisation of glycaemic control, and subsequently annually; also applies to children with other types of diabetes and coexisting obesity
Specialist consultations	According to general paediatric indications and in the event of diagnostic revision

**As required.*

- to normalise blood pressure, ACE inhibitors or an AT1 receptor antagonist are recommended; treatment effectiveness should be continuously monitored, and it is advisable to achieve nocturnal blood pressure dipping documented by ambulatory blood pressure monitoring (ABPM),
- in lipid metabolism disorders, i.e. when LDL-C > 100 mg/dl (2.6 mmol/l), improvement of glycaemic control and lifestyle modification are required,
- in children older than 8 years, if previous attempts at lifestyle modification and improvement of diabetes metabolic control have not had a beneficial effect on the plasma lipid profile, or if other atherosclerosis risk factors coexist with persistent LDL > 130 mg/dl, statin therapy should be considered. In the case of

LDL cholesterol >159 mg/dl (4.1 mmol/l) and/or a positive family history of cardiovascular disease, genetic testing for familial hypercholesterolaemia should be considered.

VI. Management during procedures performed under sedation or general anaesthesia in children with diabetes

1. Cooperation between the anaesthesia, surgical and diabetes teams is essential. The hospital should have perioperative management guidelines for children and adolescents with diabetes available.
2. Before elective surgery, assessment of the degree of diabetes metabolic control is recommended. If HbA_{1c} > 8.5% and/or TIR < 40%, postponement of the elective procedure until glycaemic control is achieved is recommended (in urgent cases, this should be done in a paediatric diabetes ward in the period immediately preceding the procedure).
3. Management depends mainly on the type of procedure:
- minor procedure – lasting up to 2 hours, with the possibility of returning to usual oral intake within 2–4 hours after completion,
- major procedure – lasting more than 2 hours, with inability to return to normal oral intake within 4 hours, and planned postoperative hospital stay longer than 24 hours.

4. During the procedure:
- blood glucose monitoring is required at least hourly until 4 hours after the end of the procedure,
- if there are no contraindications, a CGM system may be used,
- the target glucose level is 90–180 mg/dl (5–10 mmol/l); if postoperative stay in an intensive care unit is necessary, maintaining glucose levels within 140–180 mg/dl (7.8–10 mmol/l) is recommended.

5. **Major procedure under general anaesthesia:**
- admission on the day before the procedure: laboratory tests [including blood glucose, serum sodium and potassium, blood gas analysis, and if blood glucose > 250 mg/dl (14 mmol/l), assessment of ketonaemia or ketonuria]; consider starting intravenous insulin therapy in the evening or modifying the basal insulin dose,
- the procedure should be scheduled for the morning,
- on the morning of the procedure: start intravenous insulin therapy at least 2 hours before the procedure, in an amount dependent on glucose level (Table 23.3),
- about 2 hours before the procedure, start intravenous fluids with 0.9% NaCl and 5% glucose [if blood glucose > 250 mg/dl (14 mmol/l): 0.9% NaCl] at the following rates:
  - » body weight < 10 kg: 4 ml/kg body weight/hour,
  - » 10–20 kg: 40 ml/hour + 2 ml/hour for each kilogram between 10–20 kg,
  - » body weight > 20 kg: 60 ml/hour + 1 ml/hour for each kilogram over 20 kg; it is recommended not to exceed 80–100 ml/hour,
- in the case of marked glucose fluctuations and during longer surgical procedures, monitoring of serum sodium and potassium levels in the perioperative period is recommended,

Table 23.3. Intravenous insulin therapy (solution: 1 unit of human soluble insulin/1 ml 0.9% NaCl) during major surgical procedures in children and adolescents with diabetes (based on ISPAD Clinical Practice Consensus Guidelines, 2022)

Blood glucose level mg/dl [mmol/l] *	Insulin solution infusion rate at a concentration of 1 unit/ml (ml/kg/hour)
≤ 70** [≤ 3.9]	0 (+ intravenous 10% glucose)
71–89 [4.0–5.0]	0–0.01
90–109 [5.0–6.0]	up to 0.02 (approximately 0.01–0.02)
110–143 [6.1–7.9]	0.025 (“baseline infusion”)
144–215 [8.0–11.9]	0.05
216–270 [12.0–14.9]	0.075
> 270 [> 15.0]	0.1

**Capillary blood glucose monitoring: hourly; after a change in insulin dose every 30 minutes; if blood glucose <80 mg/dl (5 mmol/l) – every 15 minutes; when blood glucose decreases to 90–110 mg/dl (5–6.0 mmol/l), the insulin infusion rate should be reduced by 50%.*

***If blood glucose is ≤ 70 mg/dl (3.9 mmol/l), an intravenous bolus of 10% glucose should be administered (2 ml/kg body weight) and blood glucose rechecked after 15 minutes; if hypoglycaemia persists, the 10% glucose bolus should be repeated; if after the subsequent glucose bolus blood glucose remains ≤70 mg/dl (3.9 mmol/l), the intravenous insulin infusion may be temporarily discontinued for 10–15 minutes.*

- intravenous insulin therapy and fluid therapy (with added glucose) should be continued until the patient awakens and is able to resume oral intake,
- for procedures associated with a high risk of infectious complications or ICU admission, intravenous insulin therapy should be continued for a longer period postoperatively.

6. **Minor procedure under general anaesthesia:**

- admission to hospital on the day of the procedure is acceptable, with required laboratory tests performed then,
- subcutaneous insulin therapy may be continued or intravenous insulin therapy may be used (protocol as for major procedures),
- if the procedure is planned for the morning, on the day before the procedure it may be considered to reduce the evening dose of a long-acting analogue by 20–30%, or to reduce the basal rate to 70–90% in CSII without predictive low-glucose suspend, if overnight/morning glucose levels in preceding days were < 100 mg/dl,
- if using subcutaneous insulin therapy on the day of the procedure:
  - » MDI: administer the full dose of a long-acting analogue (or reduce by 20–30% if pre-noon glucose has recently been low) or an ultra-long-acting analogue (without dose reduction),
  - » CSII: maintain the usual basal insulin delivery; if there is a tendency towards low pre-noon glucose, reduce to 70–90%,
- if:
  - » blood glucose ≤ 70 mg/dl (3.9 mmol/l), administer an intravenous bolus of 10% glucose (2 ml/kg body weight) and recheck glucose after 15 minutes,
  - » blood glucose > 180 mg/dl (10 mmol/l), consider a correction bolus calculated to a target glucose of 150 mg/dl,
  - » blood glucose > 250 mg/dl (14 mmol/l), administer a correction bolus calculated to a target glucose of 150 mg/dl,
  - » blood glucose > 250 mg/dl persists for more than 2 hours, in addition to giving a subcutaneous correction bolus, assess ketonaemia or ketonuria and consider starting intravenous insulin therapy,
- 1–2 hours before the procedure, start intravenous administration of 0.9% NaCl with 5% glucose [or, if blood glucose > 250 mg/dl (14 mmol/l): 0.9% NaCl].

7. **Procedures in patients treated with continuous subcutaneous insulin infusion (CSII) using a personal insulin pump (PIP) – general notes:**

- when switching to intravenous insulin therapy, after disconnecting the infusion set from the body, remember to stop insulin delivery from the PIP,
- use of a PIP during a minor procedure is possible, provided the infusion set is not located in the surgical/examination field and the anaesthetist accepts CSII and knows basic operation of the PIP,
- the PIP subcutaneous cannula should be protected from damage during the procedure,
- interruption of subcutaneous insulin infusion (in the event of hypoglycaemia) should not exceed 30 minutes,
- a subcutaneous correction bolus should not be administered more frequently than every 2 hours,
- when using CGM systems during procedures, the following should be ensured:
  - » check whether devices used during the procedure will interfere with CGM readings,
  - » do not place the sensor in the surgical/examination field,
  - » secure the sensor against dislodgement during the procedure,
  - » ensure the procedure team has continuous access to glucose readings from CGM,
- currently, there is no evidence that AID systems can be used safely in the perioperative period. Some paediatric diabetes centres, based on their own experience, continue use of these systems with full functionality during certain minor procedures, while adhering to recommendations for use of PIP and CGM during the procedure.

When using metformin, GLP-1 receptor agonists or SGLT2 inhibitors, management should follow the approach described in the chapter on procedures in adults.

VII. Recommendations for diabetes care for children and adolescents with diabetes (Table 22.1)

1. General recommendations:

- in every new case of diabetes, the child should be hospitalised in a specialist paediatric diabetes ward; however, in the case of type 1 diabetes diagnosed at the preclinical stage, therapy and holistic diabetes care may be initiated in the outpatient setting,

- subsequently, every child with diabetes, including those in the preclinical stages of type 1 diabetes, should remain under regular specialist care in a diabetes outpatient clinic for children and adolescents until transfer to an adult diabetes clinic (transition principles are presented in Annex 1),
  - » 24-hour access to diabetes information for children and adolescents with diabetes and their caregivers must be ensured,
  - » hospitalisation in a diabetes ward should always be considered in the case of disease decompensation (persistent hyperglycaemia, glucose variability, recurrent hypoglycaemia),
  - » in diabetes care, during each hospitalisation and each diabetes consultation, analysis and interpretation of data from the memory of insulin delivery devices and glucose monitoring devices are required, in close cooperation with the patient and their caregivers.

2. Therapeutic team:

- inpatient care — per 10 paediatric diabetes beds: physicians (specialist in paediatric endocrinology and diabetology or specialist paediatric diabetologist, and if unavailable: a specialist in paediatrics or diabetology, or endocrinology with experience in paediatric diabetology) — 2 full-time posts, diabetes educators (nurses working exclusively in diabetes education) — working hours equivalent to 2 full-time posts, dietitian (involved in diabetes education) — working hours equivalent to 1 full-time post, psychologist (with knowledge of diabetes-related issues) — working hours equivalent to 1 full-time post, physiotherapist — working hours equivalent to 1/4 of a full-time post; in diabetes wards providing high-dependency care, a nurse dedicated to this care is required, with permanent access to psychiatric consultations and IT support,
- outpatient care — a therapeutic team providing care for 500 children and adolescents: a physician specialist in paediatric endocrinology and diabetology or a specialist paediatric diabetologist (if unavailable: a specialist in paediatrics or diabetology, or endocrinology with experience in paediatric diabetology), a physician in the 2nd year of specialist training in paediatric endocrinology and diabetology — working hours equivalent to 1 full-time post (up to 50% of the working time for a physician in the 2nd year of specialist training in paediatric endocrinology and diabetology), nurses — diabetes educators — working hours equivalent to 1–2 full-time posts, dietitian — working hours equivalent to 1/2 of a full-time post, psychologist — working hours equivalent to 1/2 of a full-time post, optionally a child psychiatrist — working hours equivalent to 1/4 of a full-time post. The therapeutic team must have ensured close cooperation with a child psychiatrist, physiotherapist, social worker and IT specialist, who may form part of the diabetes team.

3. Outpatient consultations:

- frequency of diabetes visits is not limited; recommended every 8–12 weeks, no fewer than 3 times per year,
- up to 50% of outpatient visits may be replaced with teleconsultations provided that it is possible to remotely read and transmit to the clinic data:
  - » from glucose monitoring devices,
  - » from insulin delivery devices or from applications serving as electronic self-monitoring diaries,
- despite using teleconsultations, in-person visits to the clinic must take place at least once every 6–8 months,
- in patients with poor metabolic control of diabetes or with additional health problems, in-person visits should be recommended every 2–3 months,
- recommended average visit duration: 30 minutes (need to analyse data from glucose monitoring and insulin delivery devices); in the case of using automated insulin delivery systems without additional health burdens: 20–30 minutes,
- educational, dietetic and psychological consultations should constitute a separate outpatient visit independent of the medical consultation and may take place on the same day as the medical consultation; they may also be provided electronically,
- additionally, the tasks of the therapeutic team include substantive supervision of education concerning care for children with diabetes in educational institutions, camps/educational workshops, and preparation of information materials.

4. Equipment of the outpatient clinic and ward:

- equipment: syringe pumps, intravenous infusion pumps, personal insulin pumps, gluco-

meters, continuous glucose monitoring devices, blood pressure Holter monitor (ABPM), ophthalmoscope, monofilament, food scale, computer set for reading and printing data from the memory of therapeutic systems, equipment for body composition assessment, stadiometer,
- access to imaging tests (including ultrasound with the possibility of assessing vascular flow, densitometry),
- rooms and necessary teaching aids for diabetes and diet education and for psychological care,
- ward additionally: at least 1 high-dependency care station per 10 diabetes beds, equipped with a pulse oximeter, ECG monitor, blood pressure monitor, access to oxygen therapy, and an ultrasound machine with the possibility of assessing vascular flow.

VIII. A child with diabetes in an educational or care institution

1. Cooperation between the diabetes care team, teaching staff, school nurse and the family aims to ensure the child's safety at school and prevent stigmatisation of children with diabetes:
- after diabetes diagnosis, teaching staff should be provided with written information about diabetes and how to provide assistance in life-threatening situations, as well as contact telephone numbers for parents, the physician and the diabetes nurse educator,
- school staff should be informed of the need for the child to have a mobile device (mobile phone, smartwatch) with applications that act as receivers and transmitters of data from CGM systems, insulin pumps, integrated systems, as well as therapy-supporting applications (e.g. for calculating carbohydrate content in meals),
- appropriate training of teaching staff in diabetes self-care is necessary,
- the nurse/staff responsible for care of the child with diabetes at school should be trained in the use of a glucometer, CGM system, insulin pen or personal insulin pump, and administration of glucagon,
- caregivers are required to ensure the institution has a continuous supply of glucose and glucagon,
- diabetes is not an indication for an individualised educational pathway or exemption from any activities (e.g. physical education classes, school trips/excursions).

2. Tasks of teaching staff:
- immediate provision of first diabetes aid in life-threatening situations,
- comprehensive support aimed at a rapid and safe return of a child with newly diagnosed diabetes to the institution and full integration with peers,
- knowledge of the basic scope of diabetes self-care,
- enabling self-monitoring in educational and care institutions for all age groups; in younger children – under staff supervision,
- enabling children to use glucose monitoring and insulin delivery devices during school activities, including tests and examinations,
- enabling full participation in school activities without the constant presence of the child's legal guardians,
- close cooperation with the diabetes care team and the child's caregivers.

IX. Travel:

- the responsibilities of the child's caregivers include informing the trip organiser about the disease, treatment method, meals, first-aid principles, and providing contact telephone numbers for the diabetes therapeutic team,
- in the case of travel abroad, an English-language certificate of the disease issued by the diabetes centre caring for the child should be prepared,
- insulin, glucagon, glucose, a glucometer with test strips, insulin pens, spare insulin pump and CGM supplies, and other oral or subcutaneous medicines (according to the therapy used) should be secured for the travel period and kept in hand luggage.

X. Physical activity and sport

1. Children and adolescents with diabetes:
- should be encouraged and supported to achieve daily moderate or vigorous physical activity lasting at least 60 minutes,
- should participate regularly in physical education classes,
- may practise sport, including competitive sport, in the same way as children without diabetes.

2. The glucose level at which physical activity can be started is 90–250 mg/dl (according to ISPAD up to 270 mg/dl), and during physical activity the aim should be to maintain glucose at 90–180 mg/dl.

3. Management depends on:
- type, intensity and duration of the planned physical activity,
- therapy method and glucose self-monitoring method,
- the possibility of planning physical activity in advance.
4. Guidance on physical activity and sport has been presented in Chapter 7 and in Annex 6.

XI. Choice of profession:

- particular attention should be paid to the education of adolescents with diabetes — they should receive the best possible education,
- the task of the diabetes team is to assist the young person with diabetes in choosing a profession by assessing their health status, presence of complications, and intellectual and psychological capabilities.

XII. Cooperation with patient organisations and informal self-help groups supporting people with diabetes

REFERENCES

1. American Diabetes Association Professional Practice Committee. 14. Children and Adolescents: Standards of Care in Diabetes – 2025. Diabetes Care 2025; 48 (1 Suppl 1): S283–S305. DOI: 10.2337/dc25-S0141.
2. Adolfsson P, Hanas R, Zaharieva DP, et al. Automated insulin delivery systems in pediatric type 1 diabetes: a narrative review. J Diabetes Sci Technol 2024; 18: 1324–1333.
3. Builes-Montaño CE, Ortiz-Cano NA, Ramirez-Rincón A, Rojas-Henao NA. Efficacy and safety of carbohydrate counting versus other forms of dietary advice in patients with type 1 diabetes mellitus: a systematic review and meta-analysis of randomised clinical trials J Hum Nutr Diet 2022; 35: 1030–1042.
4. Carlsson A, Shepherd M, Ellard S, et al. Absence of Islet Autoantibodies and Modestly Raised Glucose Values at Diabetes Diagnosis Should Lead to Testing for MODY: Lessons From a 5-Year Pediatric Swedish National Cohort Study Diabetes Care 2020; 43: 82–89.
5. Diabetologia wieku rozwojowego. Myśliwiec M, Jarosz-Chobot P., Szadkowska A (red.) Wydawnictwo Lekarskie PZWL, Warszawa 2024.
6. Elbalshy M, Haszard J, Smith H, et al. Effect of divergent continuous glucose monitoring technologies on glycaemic control in type 1 diabetes mellitus: a systematic review and meta-analysis of randomised controlled trials. Diabet Med 2022; 39: e14854. DOI: 10.1111/dme.14854.
7. Howard Dicks J, McCann LJ, Tolley A et al. Equity of Continuous Glucose Monitoring in Children and Young People With Type 1 Diabetes: A Systematic Review. Pediatr Diabetes 2025; 2025: 8875203. DOI: 10.1155/pedi/8875203.
8. ISPAD Clinical Practice Consensus Guidelines 2022. https://www.ispad.org/page/ISPADGuidelines2022.
9. ISPAD Clinical Practice Consensus Guidelines 2024. https://www.ispad.org/resources/ispad-clinical-practice-consensus-guidelines/2024-cpcg.html
10. Mallone R, Sims E, Achenbach P, et al. Emerging concepts and success stories in type 1 diabetes research: a road map for a bright future. Diabetes 2025; 74: 12–21.
11. Di Molfetta S, Di Gioia L, Caruso I, et al. Efficacy and safety of different hybrid closed loop systems for automated insulin delivery in people with type 1 diabetes: a systematic review and network meta-analysis. Diabetes Metab Res Rev 2024; 40: e3842. DOI: 10.1002/dmrr.3842.
12. Phillip M, Achenbach P, Addala A, et al. Consensus guidance for monitoring individuals with islet autoantibody-positive pre-stage 3 type 1 diabetes. Diabetologia 2024; 67: 1731–1759.
13. Szadkowska A, Chobot A, Głowińska-Olszewska B, et al. Guidelines of the Polish Society of Pediatric Endocrinology and Diabetology and Pediatric Section of Diabetes Poland on insulin therapy using hybrid closed-loop systems in children and adolescents with diabetes in Poland. Pediatr Endocrinol Diabetes Metab 2024; 30: 132–147.
14. de Visser HS, Waraich S, Chhabra M, et al.; TEAM Trial Patient Coresearchers. Automated insulin delivery systems and glucose management in children and adolescents with type 1 diabetes: a systematic review and meta-analysis. JAMA Pediatr 2025; 179: 1162–1171.

24. Diabetes and Pregnancy

CHAPTER HIGHLIGHTS
• Pregnancy planning in women with diabetes reduces the risk of adverse outcomes for both mother and child and should be an integral part of standard diabetes care in women of reproductive age. [**A**]
• Barrier methods or hormonal contraceptives should be used for contraception when planning pregnancy in women with diabetes, and education on its role in pregnancy planning and prevention of congenital malformations should be provided from adolescence. [**A**]
• In Poland, universal screening for hyperglycaemia in pregnancy is mandatory, and classification and diagnostic criteria follow WHO guidelines. [**A**] Screening tests are recommended at the first antenatal visit and between 24 and 28 weeks of gestation.
• In many women with gestational diabetes, satisfactory glycaemic control can be achieved with lifestyle and behavioural interventions; insulin pharmacotherapy should be initiated if therapeutic targets are not met. [**A**]
• General principles of diabetes management in pregnancy: – Hyperglycaemia during pregnancy increases the risk of maternal and fetal complications; therefore, both in pre-existing diabetes and in hyperglycaemia first diagnosed during pregnancy, optimal glycaemic control should be pursued throughout treatment. [**A**] – Self-monitoring of blood glucose is recommended as the primary method for assessing metabolic control in all types of diabetes complicating pregnancy. The following capillary glucose targets are recommended: fasting and pre-meal 70–90 mg/dl (3.9–5.0 mmol/l); 1 hour after the start of a meal 110–140 mg/dl (6.1–7.8 mmol/l); 2 hours after the start of a meal 100–120 mg/dl (5.5–6.7 mmol/l); between 2:00 and 4:00 a.m. 70–90 mg/dl (3.9–5.0 mmol/l). [**A**] – Continuous glucose monitoring and achievement of CGM-derived glycaemic targets (TIR, TAR) may support attainment of treatment goals in women with pre-existing diabetes. [**B**] – In all pregnant women with pre-existing diabetes, CGM use is recommended; in women with type 1 diabetes, systems integrated with insulin pump therapy and with the highest possible level of automation are preferred. [**A**] – The use of CGM should be considered in women with hyperglycaemia first diagnosed during pregnancy, when it may be helpful in managing gestational diabetes. [**E**] – Measurement of HbA_{1c} is a tool for assessing glycaemic control in women with pre-existing diabetes. An HbA_{1c} value < 6.5% (48 mmol/mol) is recommended during pregnancy planning and in the first trimester, and < 6.0% (42 mmol/mol) in subsequent trimesters. [**B**] – In addition to achieving glycaemic targets, standard preconception care should be broadened, with particular attention to nutrition, physical activity, education on self-monitoring, and screening for comorbidities and diabetic complications. [**B**] – In women with pre-existing diabetes, regardless of diabetes type, ophthalmological examination should be performed before conception and no later than in the first trimester of pregnancy, and then repeated in each trimester; routine fundus examination is not recommended in women with gestational diabetes (GDM). – Insulin is the only antihyperglycaemic agent recommended in pregnancy. Based on current evidence, the use of other glucose-lowering medications, both oral and injectable, is not recommended. [**A**] – Metformin use is permitted only in women with type 2 diabetes and polycystic ovary syndrome (PCOS) with insulin resistance, if it provides metabolic benefit in the preconception period; it should be discontinued no later than by the end of the first trimester. [**B**]
• Women with a history of GDM should be monitored for the future development of diabetes and cardiovascular disease. [**E**]

Pregnancy planning in all women with diabetes has a significant impact on pregnancy outcomes, reducing the risk of adverse events in both the mother and the fetus/newborn. In addition to focusing on the achievement of glycaemic targets, standard preconception care should place additional emphasis on appropriate nutrition, diabetes education, and screening for coexisting conditions and diabetes-related complications. Early detection of impaired glucose tolerance is recommended in women of reproductive age with obesity. A 75 g oral glucose tolerance test (OGTT) should be performed before a planned pregnancy in women without a prior diagnosis of diabetes. In women with obesity, pregnancy should be planned.

Diabetes in pregnancy may occur as:

1) pregestational diabetes mellitus (PGDM) – when pregnancy occurs in a woman with previously diagnosed diabetes, regardless of diabetes type;
2) hyperglycaemia first diagnosed during pregnancy.

I. Contraception

Women planning pregnancy should be informed that the risk of pregnancy complications

increases in the presence of poor metabolic control, diabetes-related complications, and long lasting diabetes.

Pregnancy in a woman with poorly controlled diabetes carries a higher risk for pregnancy than that associated with any currently used method of contraception.

Patients should be informed that diabetes per se is not a contraindication to hormonal contraception. Women should be assessed for standard contraindications to hormonal contraceptive use and should be able to choose an effective contraceptive method according to their preference, with full awareness of the risks associated with an unplanned pregnancy. In women with diabetes of more than 20 years' duration or with microvascular complications (nephropathy, retinopathy, neuropathy), the use of intrauterine devices or progestogen-only contraceptives is recommended.

Combined oestrogen–progestagen preparations containing less than 35 µg of ethinylestradiol, preferably 15 or 20 µg, are recommended, as they have only a minimal effect on carbohydrate and lipid metabolism. The preferred progestogen components are levonorgestrel or norethisterone due to their lowest prothrombotic potential.

A levonorgestrel-releasing intrauterine device is a particularly recommended method of contraception for obese women over 35 years of age and in the presence of vascular complications.

II. Model of care for pregnant women with diabetes

1. All women with diabetes during pregnancy planning, throughout pregnancy, and during the postpartum period should remain under the care of a multidisciplinary diabetes–obstetric team (including a perinatologist) with experience in this field. Women with type 2 diabetes treated with oral glucose-lowering agents require initiation of insulin therapy already during the preconception period in order to achieve adequate glycaemic control. The use of metformin is permitted only in women with type 2 diabetes in the preconception period, at therapeutic doses, when it ensures the recommended level of metabolic control. After conception, metformin should be discontinued no later than by the end of the first trimester.
2. Currently available GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists have no evidence supporting their safety in pregnancy and should not be used during pregnancy planning or during the reproductive period if effective contraception is not used. These agents should be discontinued 4–8 weeks before a planned pregnancy, and in the case of an unplanned pregnancy, immediately after pregnancy confirmation.
3. Every physician caring for a woman with type 2 diabetes or obesity should regularly discuss reproductive plans and inform the patient about the need for pregnancy planning, due to the presence of multiple risk factors for adverse obstetric outcomes in this population, as well as the frequent use of antihypertensive agents and statins (necessitating treatment modification). The aims of such management are:

- optimisation of diabetes treatment,
- assessment and, if necessary, treatment of chronic diabetes complications,
- diabetes education, including nutritional counselling,
- recommendation to stop smoking,
- assessment of thyroid function (exclusion of hypothyroidism); upper reference limits for TSH should be considered as follows: 2.5 µIU/ml in the first trimester and 3 µIU/ml in the second and third trimesters,
- women with previously diagnosed diabetes who are planning pregnancy or are pregnant should be informed about the risk of development and/or progression of diabetic retinopathy. Fundus examination with pupil dilation should be performed before pregnancy and in the first trimester. Pregnant women should be monitored in each trimester and for one year postpartum, according to the severity of retinopathy and ophthalmological recommendations; routine ophthalmological screening is not recommended in women with gestational diabetes mellitus (GDM),
- during pregnancy, diabetology visits should take place at least monthly and, in justified cases, every 2–3 weeks; this is related, among other factors, to changing insulin requirements and the need to monitor body weight, renal function, visual status and blood pressure; teleconsultations are acceptable,
- in the case of gestational hypertension, antihypertensive treatment should be initiated at blood pressure values ≥ 140/90 mm Hg; the therapeutic target is < 140/90 mm Hg; lowering diastolic blood pressure below 80 mm Hg is not recommended,

- in women with diabetes and pre-existing chronic hypertension or renal complications, efforts should be made to maintain systolic blood pressure <135 mm Hg and diastolic blood pressure <85 mm Hg,
- during pregnancy, a sequential antihypertensive treatment regimen is recommended, starting with oral labetalol or methyldopa, or modified-release nifedipine (alternatively amlodipine); if ineffective, two of the listed agents should be combined, and if still ineffective, three agents may be used; prolonged-release metoprolol may also be used in pregnancy according to indications, however metoprolol should not be combined with labetalol,
- in women with pregestational diabetes, acetylsalicylic acid at a dose of 150 mg/day is recommended from 12 to 36 weeks of gestation (prevention of pre-eclampsia; initiation between 12 and 16 weeks), with the decision to initiate therapy made by the obstetrician.

4. Pregnancy is discouraged in women with diabetes in the following clinical situations:

a) nephropathy with creatinine clearance <40 ml/min,
b) proliferative retinopathy refractory to treatment,
c) advanced ischaemic heart disease refractory to treatment,
d) hypertrophic cardiomyopathy or severe left ventricular dysfunction (LVEF <30%, NYHA class III/IV),
e) a history of peripartum cardiomyopathy with any residual left ventricular dysfunction,
f) autonomic neuropathy involving the cardiac conduction system or the gastrointestinal tract.

The final decision regarding reproduction belongs to the patient; however, she must be informed by relevant specialists about the risks to her health and life associated with pregnancy in these situations.

Pregnancy does not appear to be associated with postpartum worsening of chronic diabetes complications. A woman with diabetes may freely plan the number of pregnancies, provided that none of the above contraindications are present.

III. Diagnostic criteria and classification of hyperglycaemia first detected during pregnancy

All pregnant women should be screened for disorders of glucose tolerance as early as possible after pregnancy is confirmed. In women at increased risk, a 75 g oral glucose tolerance test

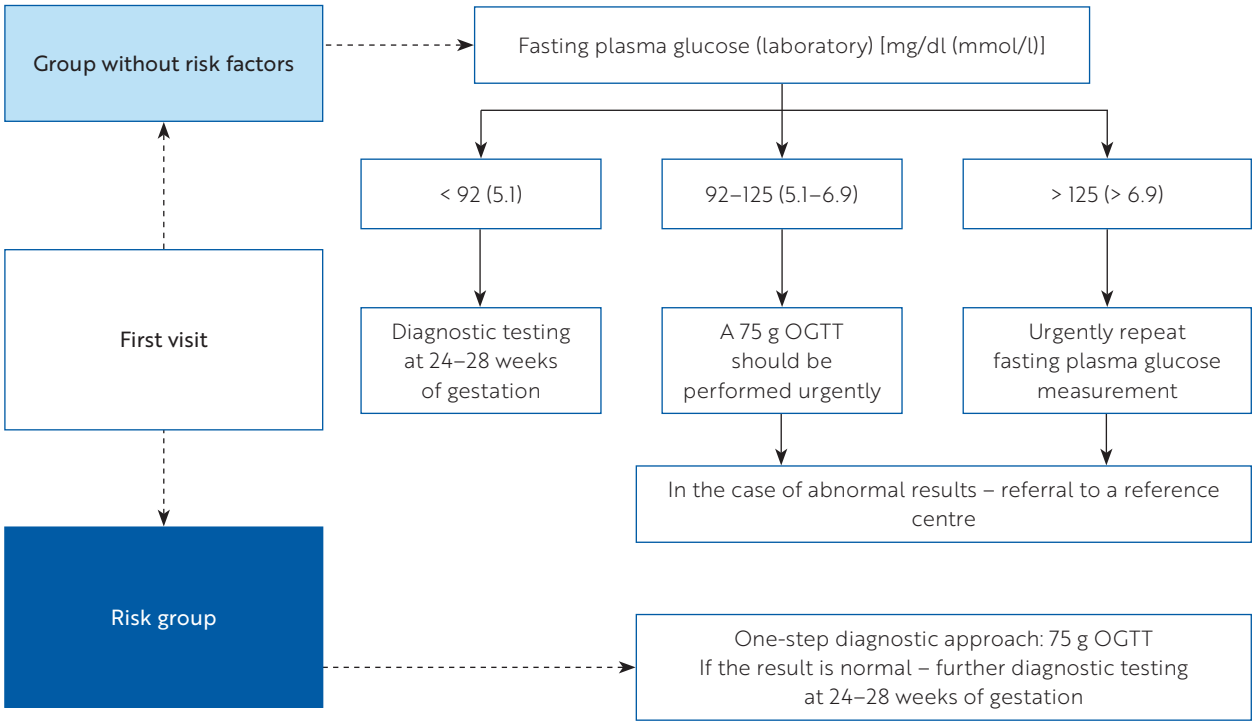


Figure 24.1. Diagnostic algorithm for disorders of carbohydrate metabolism in pregnant women

OGTT – oral glucose tolerance test
NOTE: A single fasting plasma glucose measurement in the first trimester of pregnancy between 92 mg/dl and 125 mg/dl cannot be used as a basis for the diagnosis of hyperglycaemia in pregnancy.

(OGTT) (as described in Chapter 1) should be performed at the first antenatal visit, while in the remaining women fasting plasma glucose should be measured. If no abnormal glucose values are identified (see Figure 24.1), diagnostic testing should be repeated between 24 and 28 weeks of gestation or earlier if symptoms suggestive of diabetes occur. In women without risk factors and with normal glucose values at the first examination during pregnancy, diagnostic testing should be performed between 24 and 28 weeks of gestation and should follow a one-step approach, consisting of a 75 g OGTT.

Hyperglycaemia first detected during pregnancy should be diagnosed and classified as follows:

- Diabetes in pregnancy — when any of the following criteria are met:
  - fasting plasma glucose ≥126 mg/dl (7.0 mmol/l),
  - or 2-hour plasma glucose during OGTT ≥200 mg/dl (11.1 mmol/l),
  - or random plasma glucose ≥200 mg/dl (11.1 mmol/l) in the presence of clinical symptoms of hyperglycaemia.
- Gestational diabetes mellitus (GDM) — when at least one of the criteria listed in Table 24.1 is met.

After delivery, plasma glucose levels normalise in most women; however, all women with a history of hyperglycaemia during pregnancy should undergo evaluation for disorders of glucose tolerance. A 75 g OGTT is recommended 6–12 weeks postpartum, followed by annual screening thereafter. Before planning a subsequent pregnancy, a 75 g OGTT should be performed.

Women with a history of gestational diabetes mellitus should be regarded as a high-risk group for the development of diabetes and cardiovascular disease (management: see Chapter 2).

IV. Multidisciplinary, integrated management of pregestational diabetes and hyperglycaemia during pregnancy

Hyperglycaemia during pregnancy increases the risk of obstetric complications in the mother and the developing fetus and has long-term consequences for the child's development. Therefore, treatment should aim to achieve glycaemic values comparable to those observed in healthy pregnant women. Based on current evidence, the following target capillary blood glucose values in self-monitoring during pregnancy are recommended, regardless of the type of diabetes:

- fasting and preprandial: 70–90 mg/dl (3.9–5.0 mmol/l),
- 1 hour after the start of a meal: 110–140 mg/dl (6.1–7.8 mmol/l),
- 2 hours after the start of a meal: 100–120 mg/dl (5.5–6.7 mmol/l),
- between 02:00 and 04:00: 70–90 mg/dl (3.9–5.0 mmol/l).

Pregnant women should perform self-monitoring of blood glucose after appropriate training by a nurse experienced in diabetes care. The number and timing of glucose measurements should depend on the severity of carbohydrate metabolism disturbances and the treatment used.

In women with type 1 diabetes, and in women with type 2 diabetes or gestational diabetes treated with insulin, the use of continuous glucose monitoring (CGM) is recommended. CGM may also be considered in women with hyperglycaemia first diagnosed during pregnancy if it may improve glycaemic control or at the woman's request.

For pregnant women with type 1 diabetes using CGM, glucose values:

- 140 mg/dl (7.8 mmol/l) should account for < 25% of readings,
- 63–140 mg/dl (3.5–7.8 mmol/l) for > 70%,
- < 63 mg/dl (3.5 mmol/l) for < 4%,
- < 54 mg/dl (3.0 mmol/l) for < 1% of readings.

In pregnant women with type 2 diabetes and gestational diabetes, > 90% of CGM readings should fall within the target range of 63–140 mg/dl (3.5–7.8 mmol/l).

During labour, blood glucose levels should be maintained between 100 and 130 mg/dl (5.6–7.2 mmol/l).

The efficacy and safety of automated insulin delivery (AID) systems during pregnancy have been demonstrated for CamAPS Fx and MiniMed 780G. Recommended target glucose settings are:

- preconception period:
  - MiniMed 780G: 100 mg/dl (5.5 mmol/l),
  - CamAPS Fx: 90 mg/dl (5.0 mmol/l);

Table 24.1. Diagnostic criteria for gestational diabetes mellitus based on the oral glucose tolerance test according to IADPSG 2010 and WHO 2013

Plasma glucose levels		
	[mg/dl]	[mmol/l]
Fasting	92–125	5.1–6.9
60th minute	≥ 180	≥ 10.0
120th minute	153–199	8.5–11

- during pregnancy:
  - MiniMed 780G: 100 mg/dl (5.5 mmol/l),
  - CamAPS Fx: 80–90 mg/dl (4.4–5.0 mmol/l);
- during labour and breastfeeding:
  - MiniMed 780G: 120 mg/dl (6.7 mmol/l),
  - CamAPS Fx: 110–120 mg/dl (6.1–6.7 mmol/l).

In women with pregestational diabetes, HbA_{1c} should be measured every 6 weeks, with target values of < 6.5% (< 48 mmol/mol) in the first trimester and < 6.0% (< 42 mmol/mol) in subsequent trimesters. If these targets cannot be achieved without significant hypoglycaemia, less stringent goals may be considered based on clinical judgement and individualised care; however, HbA_{1c} values >7% should be avoided. At the diagnosis of diabetes during pregnancy, HbA_{1c} should also be measured, although routine HbA_{1c} monitoring is not recommended in gestational diabetes.

1. Nutritional recommendations during pregnancy

- 40–50% carbohydrates (approximately 180 g/day), preferably with a low glycaemic index,
- 30% protein (1.3 g/kg body weight/day),
- 20–30% fat (including <10% saturated fat),
- total energy intake should depend on body weight, height, physical activity and age; average daily requirements are approximately 30 kcal/kg of ideal body weight (about 1500–2400 kcal),
- in overweight women, a diet providing 25–30 kcal/kg body weight/day is recommended,
- due to strict glycaemic targets, meals should contain consistent amounts of carbohydrates at relatively fixed times to allow appropriate insulin dose adjustment and to avoid both hyper- and hypoglycaemia,
- gestational weight gain should be monitored, as excessive gain in women with diabetes is associated with excessive fetal growth (Table 24.2),

Table 24.2. Recommendations for weight gain during pregnancy

Pre-pregnancy body mass index BMI [kg/m^2].	Recommended total weight gain [kg]	Recommended weight gain in the 2nd and 3rd trimester [kg/week]
< 18.5	12.5–18.0	0.51 (0.44–0.58)
18.5–24.8	11.5–16.0	0.42 (0.35–0.50)
25.0–29.9	7.0–11.5	0.28 (0.23–0.33)
≥ 30	5.0–9.0	0.22 (0.17–0.27)
Assuming a weight gain of 0.5-2.0 kg in the first trimester of pregnancy.		

- fasting urine ketone testing may be useful to identify women who excessively restrict carbohydrate intake to control blood glucose,
- artificial sweeteners are permitted, except for saccharin, which crosses the placenta and whose effects on the fetus are not fully understood (see Chapter 6),
- folic acid supplementation (at least 0.4 mg/day) is recommended for at least 12 weeks before conception and continued until the 12th week of pregnancy.

2. Physical activity

Provided there are no contraindications, moderate-intensity aerobic physical activity is recommended.

3. Insulin therapy

In pregestational diabetes:

- human insulins have long been used in pregnancy and are proven to be safe; observational studies have demonstrated the safety of insulin analogues lispro and glargine, as well as ultra-rapid-acting insulin aspart; insulin aspart, detemir and degludec have also been shown to be safe in randomised trials; none of these studies demonstrated placental transfer of insulin analogues,
- intensive insulin therapy using multiple daily injections or continuous subcutaneous insulin infusion (CSII) is recommended, preferably integrated with CGM and automated insulin delivery systems; such treatment should be conducted in specialised diabetes centres experienced in managing diabetes during pregnancy,
- pump therapy should ideally be initiated during preconception planning or early pregnancy (up to 12 weeks); later initiation may be considered only if satisfactory metabolic control cannot be achieved with multiple daily injections,
- insulin sensitivity may increase slightly in early pregnancy, increasing the risk of hypoglycaemia at previously adequate insulin doses; from around 16 weeks' gestation, insulin resistance increases, requiring regular and appropriate dose adjustments.

In hyperglycaemia first diagnosed during pregnancy:

- insulin therapy is recommended after failure of behavioural interventions,
- intensive insulin therapy with multiple daily injections is recommended; CSII may also be used,
- insulin requirements decrease rapidly after delivery, and in most women insulin can be

discontinued while maintaining adequate glycaemic control.

4. Oral antihyperglycaemic agents

Oral antihyperglycaemic drugs are currently not recommended for the treatment of diabetes during pregnancy. Women using these agents should discontinue them during pregnancy planning or as soon as possible after confirmation of an unplanned pregnancy.

Metformin is not teratogenic. Despite demonstrated benefits in women with GDM and PCOS (less gestational weight gain and a lower rate of large-for-gestational-age infants), **its routine use in pregnancy is not recommended**. Metformin crosses the placenta, and long-term follow-up data in exposed offspring are inconclusive. A recent meta-analysis showed lower birth weight with accelerated postnatal growth and higher childhood BMI in some children exposed to metformin in utero. Therefore, insulin remains the pharmacological treatment of choice for hyperglycaemia during pregnancy when dietary treatment is ineffective. Metformin should be discontinued no later than the end of the first trimester.

If a woman refuses insulin therapy and wishes to use metformin, its use may be considered except in cases of coexisting hypertension or preeclampsia, or in fetuses with intrauterine growth restriction, due to the risk of further growth impairment or acidosis associated with placental insufficiency.

5. Education system

- clinical education provided by a physician, nurse and dietitian experienced in insulin pump therapy,
- technical training in insulin pump use provided by a certified nurse or physician, or by a representative of the pump manufacturer,
- CGM use education,
  - » training is delivered according to a structured education record, including digital tools,
  - » therapy may be initiated once the patient has acquired basic clinical and technical knowledge of CSII (understanding therapy principles and operation of key pump functions).

6. Breastfeeding

Breastfeeding should be strongly encouraged and recommended in women with pregestational diabetes and hyperglycaemia during pregnancy, unless other contraindications exist.

7. Oral agents and lactation

Available literature and clinical data indicate that metformin is transferred into breast milk in very small amounts (<1% of maternal concentration); therefore, women with type 2 diabetes may safely use metformin during breastfeeding. The safety of other antihyperglycaemic agents during lactation has not been sufficiently studied.

REFERENCES

1. American Diabetes Association Professional Practice Committee. Management of diabetes in pregnancy: standards of care in diabetes — 2025. Diabetes Care 2025; 48 (1 Suppl 1): S306–S320. DOI: 10.2337/dc25-S015.
2. Aroda VR, Christophi CA, Edelstein SL, et al. Diabetes Prevention Program Research Group.The effect of lifestyle intervention and metformin on preventing or delaying diabetes among women with and without gestational diabetes: the Diabetes Prevention Program outcome sstudy 10-year follow-up. J Clin Endocrinol Metab 2015; 100: 1646–1653.
3. Bateman BT, Hernandez-Diaz S, Fischer MA, et al. Statins and congenital malformations: cohort study. BMJ 2015; 350: h1035. DOI: 10.1136/bmj.h1035.
4. Benhalima K, Yamamoto JM. Use of continuous glucose monitoring and hybrid closed-loop therapy in pregnancy. Diabetes Obes Metab 2024; 26 Suppl 7: 74–91. DOI: 10.1111/dom.15999.
5. Beunen K, Van Wilder N, Ballaux D, et al. Closed-loop insulin delivery in pregnant women with type 1 diabetes (CRISTAL): a multicentre randomized controlled trial – study protocol. BMC Pregnancy Childbirth 2023; 23: 180. DOI: 10.1186/s12884-023-05481-0.
6. Bolte E, Dean T, Garcia B, et al. Initiation of metformin in early pregnancy results in fetal bioaccumulation, growth restriction, and renal dysmorphology in a primate model. Am J Obstet Gynecol 2024; 231: 352.e1-352.e16. DOI: 10.1016/j.ajog.2024.06.002.
7. Bullo M, Tschumi S, Bucher BS, et al. Pregnancy outcome following exposure to angiotensin-converting enzyme inhibitors or angiotensin receptor antagonists: a systematic review. Hypertension 2012; 60: 444–450.
8. De Backer J, Haugaa KH, Hasselberg NE, et al. ESC Guidelines for the management of cardiovascular disease and pregnancy. Eur Heart J 2025; 46: 4462–4568.
9. ElSayed NA, Aleppo G, Aroda VR, et al. Management of diabetes in pregnancy; standards of care in diabetes – 2023. Diabetes Care 2023; 46 (Suppl 1): S254–S266. DOI: 10.2337/dc23-S015.
10. Farrar D, Tuffnell DJ, West J, West HM. Continuous subcutaneous insulin infusion versus multiple daily injections of insulin for pregnant women with diabetes. Cochrane Database Syst Rev 2016; 6: CD005542. DOI: 10.1002/14651858.CD005542.pub3.

11. Feig DS, Donovan LE, Corcoy R, et al. Continuous glucose monitoring in pregnant women with type 1 diabetes (CONCEPTT): a multicentre international randomized controlled trial. Lancet 2017; 390: P2347–P2359. DOI: 10.1016/S0140-6736(17)32400-5.
12. Feig DS, Sanchez JJ, Murphy KE, et al. Outcomes in children of women with type 2 diabetes exposed to metformin versus placebo during pregnancy (MiTy 740 Kids): a 24-month follow-up of the MiTy randomised controlled trial. Lancet Diabetes Endocrinol 2023; 11: 191–202.
13. Feig DS, Zinman B, Asztalos E, et al. Determinants of 733 Small for Gestational Age in Women With Type 2 Diabetes in Pregnancy: Who Should Receive Metformin? Diabetes Care 2022; 45: 1532–1539.
14. Filippi-Arriaga F, Agarwal N, Rodrigues-Martins D, et al. EASO Position Statement: Women with obesity across the reproductive life – fertility, preconception, pregnancy, postpartum, and breastfeeding. Obes Facts 2025; 18: 625–639.
15. Glatstein MM, Djokanovic N, Garcia-Bournissen F, et al. Use of hypoglycemic drugs during lactation. Can Fam Physician 2009; 55: 371–373.
16. HAPO Study Cooperative Research Group; Metzger BE, Lowe LP, Dyer AR, et al. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med 2008; 358: 1991–2002.
17. Hartling L, Dryden DM, Guthrie A, et al. Benefits and harms of treating gestational diabetes mellitus: a systematic review and meta-analysis for the U.S. Preventive Services Task Force and the National Institutes of Health Office of Medical Applications of Research. Ann Intern Med 2013; 159: 123–129.
18. Institute of Medicine (US) and National Research Council (US) Committee to Reexamine IOM Pregnancy Weight Guidelines. Weight Gain During Pregnancy: Reexamining the Guidelines. Rasmussen KM, Yaktine AL (eds.). Washington (DC): National Academies Press (US); 2009.
19. Jensen DM, Korsholm L, Ovesen P, et al. Periconceptional A1C and risk of serious adverse pregnancy outcome in 933 women with type 1 diabetes. Diabetes Care 2009; 32: 1046–1048.
20. Kusinski LC, Meek CL. Big babies, small babies: metformin exposure in pregnancy. Lancet Diabetes Endocrinol 2023; 11: 145–146.
21. Landon MB, Spong CY, Thom E, et al. A multicenter, randomized trial of treatment for mild gestational diabetes. N Engl J Med 2009; 361: 1339–1348.
22. Lee TTM, Collett C, Bergford S; AiDAPT Collaborative Group. Automated insulin delivery in women with pregnancy complicated by type 1 diabetes. N Engl J Med 2023; 389: 1566–1578.
23. Lee TTM, Collett C, Bergford S, et al. Automated closed-loop insulin delivery for the management of type 1 diabetes during pregnancy: the AiDAPT RCT. Efficacy Mech Eval 2024; 11.
24. Middleton P, Crowther CA, Simmonds L. Different intensities of glycaemic control for pregnant women with pre-existing diabetes. Cochrane Database Syst Rev 2016; 5: CD008540. DOI: 10.1002/14651858.CD008540.pub4.
25. Poolsup N, Suksomboon N, Amin M. Efficacy and safety of oral antidiabetic drugs in comparison to insulin in treating gestational diabetes mellitus: a meta-analysis. PLoS One 2014; 9: e109985. DOI: 10.1371/journal.pone.0109985.
26. Priya G, Kalra S. Metformin in the management of diabetes during pregnancy and lactation. Drugs Context 2018; 7: 212523. DOI: 10.7573/dic.212523.
27. Ratner RE, Christophi CA, Metzger BE, et al.; Diabetes Prevention Program Research Group. Prevention of diabetes in women with a history of gestational diabetes: effects of metformin and lifestyle interventions. J Clin Endocrinol Metab 2008; 93: 4774–4779.
28. Tarry-Adkins JL, Aiken CE, Ozanne SE. Neonatal, infant, and childhood growth following metformin versus insulin treatment for gestational diabetes: a systematic review and meta-analysis. PLoS Med 2019; 16: e1002848. DOI: 10.1371/journal.pmed.1002848.
29. Tolcher MC, Chu DM, Hollier LM, et al. Impact of USPSTF recommendations for aspirin for prevention of recurrent preeclampsia. Am J Obstet Gynecol 2017; 217: 365.e1–365.e8. DOI: 10.1016/j.ajog.2017.04.035.
30. de Veciana M, Major CA, Morgan MA, et al. Postprandial versus preprandial blood glucose monitoring in women with gestational diabetes mellitus requiring insulin therapy. N Engl J Med 1995; 333: 1237–1241.
31. Wotherspoon AC, Young IS, Patterson CC, et al. Diabetes and Pre-eclampsia Intervention Trial (DAPIT) Study Group. Effect of pregnancy planning on maternal and neonatal outcomes in women with type 1 diabetes. Diabet Med 2017; 34: 1303–1308.
32. Wyckoff JA, Lapolla A, Asias-Dinh BD, et al. Preexisting diabetes and pregnancy: an Endocrine Society and European Society of Endocrinology Joint Clinical Practice Guideline. Eur J Endocrinol 2025; 193: G1–G48. DOI: 10.1093/ejendo/lvaf116.

25. Diabetes in Individuals Aged Over 65 Years

CHAPTER HIGHLIGHTS
When initiating diabetes therapy in individuals aged over 65 years, therapeutic goals should be individually assessed depending on the patient's health status, diabetes-related complications, life expectancy and social and living conditions. [**B**]
One of the main objectives of diabetes treatment in individuals aged over 65 years is achieving good metabolic control while preventing hypoglycaemia, through individualisation of therapeutic targets and avoidance of medications associated with an increased risk of hypoglycaemia. [**B**]
In individuals aged over 65 years without significant complications, therapeutic targets may be similar to those applied in the younger adult population. [**C**]
When intensifying treatment, target values for glycaemia, blood pressure and lipid levels should be considered, taking into account the specific characteristics of the older age group and the presence of comorbidities. [**B**]

I. The prevalence of diabetes in the population aged over 65 years reaches 25–30%.

II. Symptoms of hyperglycaemia in individuals aged over 65 years may be less intensified than in younger individuals, which may result in delayed diagnosis of the disease.

III. In individuals with diabetes of advanced age, life expectancy is significantly shorter; therefore, when determining the treatment strategy, it should be borne in mind that prevention of complications developing after several, or more years of disease is less important than in younger individuals.

IV. Treatment goals in individuals aged over 65 years

- The overarching goal of diabetes management in older individuals is to improve or at least maintain the current quality of life, with particular emphasis on avoiding hypoglycaemia while simultaneously reducing symptoms of hyperglycaemia.
- If no significant complications or comorbidities are present in an individual with diabetes aged over 65 years, general treatment goals should be pursued, aiming for gradual glycaemic control with a target HbA_{1c} ≤ 7%, provided this does not entail an increased risk of hypoglycaemia.
- In patients of advanced age with long-standing diabetes and significant macroangiopathic complications (previous myocardial infarction or stroke), the target HbA_{1c} value is 8.0–8.5%.
- At least once a year, patients should be assessed for geriatric syndromes (e.g. cognitive impairment, depression, urinary incontinence, falls, chronic pain and frailty), hypoglycaemia and polypharmacy, as these may affect diabetes treatment and reduce quality of life.
- Diagnostic evaluation for diabetes complications should be undertaken, their progression prevented, and appropriate treatment recommended.
- Comorbid conditions should be treated in order to reduce functional impairment and improve quality of life.

V. Physical activity

After an initial assessment of the patient's individual risk and physical capacity, physical activity in the open air should be recommended, characterised by a slow onset and gradual cessation, avoidance of straining exercises and breath-holding, with particular attention to the risk of injury, especially the risk of developing diabetic foot disease.

VI. Dietary recommendations

There are no age-specific dietary recommendations; dietary modification is often of limited effectiveness due to established eating habits. However, attention should be paid to ensuring an adequate protein intake.

VII. Oral antihyperglycaemic agents, injectable GLP-1 receptor agonists and GIP/GLP-1 receptor agonists

The use of antihyperglycaemic medications in individuals aged over 65 years should follow the principles outlined in Chapter 11. Drug classes associated with a low risk of hypoglycaemia should be preferred.

- Metformin – particular caution is required in patients with an eGFR < 45 ml/min/1.73 m².
- DPP-4 inhibitors, SGLT-2 inhibitors, GLP-1 receptor agonists, dual GIP/GLP-1 receptor

agonists and PPAR-γ agonists – there are no specific contraindications to their use in individuals aged over 65 years; these agents may be particularly beneficial in this age group due to the minimal risk of hypoglycaemia; PPAR-γ agonists should not be used in patients with heart failure or a high risk of fractures.
- Sulfonylureas – use of this drug class should be particularly cautious in older individuals with diabetes due to the risk of hypoglycaemia; these agents should not be used in patients with frailty syndrome.

VIII. Insulin therapy

- There are no specific indications or contraindications for insulin therapy based solely on older age.
- Initiation of insulin should not be delayed when indicated; simple insulin regimens should be preferred (see Chapter 12).
- When initiating or modifying insulin therapy, preparations associated with the lowest possible risk of hypoglycaemia should be selected.
- Age over 65 years is not a contraindication to intensive insulin therapy.
- In individuals with type 1 diabetes and in individuals with type 2 diabetes treated with insulin, continuous glucose monitoring (CGM) systems are the preferred method of glucose monitoring.
- In individuals with type 1 diabetes, the use of automated insulin delivery (AID) systems may be considered to improve glycaemic control and reduce the risk of hypoglycaemia, depending on individual skills and available support.
- In individuals aged over 65 years with frailty, cognitive impairment or difficulties in implementing insulin regimens, treatment should be simplified, using non-insulin therapies where possible (see Chapters 11 and 12).
- In some patients of advanced age (> 80 years), effective glycaemic control may be achieved using small doses of short-acting insulin or rapid-acting insulin analogues administered before main meals, without concomitant use of long-acting (basal) insulin.
- In situations where meal size is unpredictable (e.g. patients with poor appetite or advanced dementia), administration of a rapid-acting insulin analogue immediately after the meal, at a dose adjusted to the amount consumed, may be appropriate.

IX. Diabetes education

Diabetes education should include both patients and their caregivers.

X. Antihypertensive treatment

- Age alone is not a criterion for selecting a specific class of antihypertensive agents.
- The benefits of antihypertensive therapy in individuals aged over 65 years are comparable to those observed in younger individuals.

REFERNCES

1. Barnett AH, Huisman H, Jones R, et al. Linagliptin for patients aged 70 years or older with type 2 diabetes inadequately controlled with common antidiabetes treatment: a randomized, double-blind, placebo-controlled trial. Lancet 2013; 382: 1412–1424.
2. Davies MJ, Aroda VR, Collins BS, et al. Management of hyperglycaemia in type 2 diabetes, 2022. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetologia 2022; 65: 1925–1966.
3. ElSayed NA, Aleppo G, Aroda VR, et al.; on behalf of the American Diabetes Association. Older adults: Standards of care in diabetes –2023. Diabetes Care 2023, 46 (Suppl 1): S216–S229. DOI: 10.2337/dc23-S013.
4. Kahkoska AR, Ortega J, Blilal A, Pratley RE. Clin-STAR Corner: Practice changing advances in diabetes care. J Am Geriatr Soc 2025; 73: 3245–3257.
5. Kirkman MS, Briscoe VJ, Clark N, et al. Diabetes in older adults: a consensus report. Diabetes Care 2012; 35: 2650–2664.
6. Kudva YC, Henderson RJ, Kanapka LG, et al. Automated insulin delivery in older adults with type 1 diabetes. NEJM Evid 2025; 4: EVIDoa2400200.
7. Lipska K, Ross JS, Miao Y, et al. Potential overtreatment of diabetes mellitus in older adults with tight glycemic control. JAMA 2015; 175: 356–362.
8. Maltese G, McAuley SA, Trawley S, Sinclair AJ. Aging well with diabetes: the role of diabetes technology. Diabetologia 2024; 67: 2087–2102.
9. Matter JB, Musi N, McFarland Horne F, et al. Diabetes and cardiovascular diseases in older adults. Current status and future directions. Diabetes 2014; 63: 2578–2589.
10. Ruedy KJ, Parkin CG, Riddlesworth TD; DIAMOND Study Group. Continuous glucose monitoring in older adults with type 1 and type 2 diabetes using multiple daily injections of insulin: results from the DIAMOND trial. J Diabetes Sci Technol 2017; 11: 1138–1146.

26. Perioperative Management of People with Diabetes

Prepared in collaboration with Professor Wojciech Szczeklik, MD, PhD, and Dorota Studzińska, MD, PhD.

CHAPTER HIGHLIGHTS
· Elective surgical procedures in people with diabetes should be postponed if the HbA_{1c} value exceeds 8.5%. [**B**]
· In individuals treated with insulin, insulin therapy must not be discontinued before surgery. In most people with type 2 diabetes who have good glycaemic control, non-insulin glucose-lowering agents may be continued until the day of surgery, with the exception of SGLT2 inhibitors, which should be discontinued 3 days before surgery. [**B**]
· In people with diabetes in a critical condition and those receiving parenteral nutrition, intravenous insulin therapy at a dose adjusted to blood glucose levels is recommended. [**C**]
· Perioperative blood glucose monitoring in people with diabetes reduces the risk of complications and mortality. [**B**]
· Recommended target blood glucose values in the perioperative period are 100–180 mg/dl. [**C**]

I. Examinations to be performed before a planned surgical procedure:

- a 24-hour glycaemic profile – 7 measurements during the day and an additional measurement at approximately 03:00 in patients treated with insulin; this is not required in patients using a CGM system,
- HbA_{1c} level,
- complete blood count,
- serum concentrations of creatinine, electrolytes (Na^+, K^+), and aminotransferase activity (AST, ALT),
- INR and APTT,
- assessment of acid–base balance (blood gas analysis) if disturbances are suspected,
- urinalysis,
- resting ECG (see Note 1).

NOTE 1: In patients at high or very high cardiovascular risk, as well as when extensive procedures are planned (e.g. abdominal or iliac vascular surgery, cardiac surgery), extended non-invasive cardiovascular diagnostics should be performed (exercise testing, echocardiography, Holter ECG monitoring). Extended diagnostics in accordance with current ESC recommendations mainly apply to patients undergoing high-risk procedures or those with multiple severe comorbidities.

NOTE 2: Day-case surgery may be performed in people with diabetes who have good metabolic control, including those treated with intensive insulin therapy, as well as in people with type 2 diabetes who do not require temporary insulin therapy in the perioperative period. Withholding antihyperglycaemic medication on the day of surgery should not result in blood glucose levels exceeding 180 mg/dl (10 mmol/l).

NOTE 3: For diagnostic and therapeutic gastrointestinal endoscopy in patients treated with GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists, omission of one dose prior to the planned procedure should be considered (a minimum of 7 days without drug activity).

II. Management in the preoperative period before a planned surgical procedure:

1. A patient with diabetes requiring temporary insulin therapy should be admitted to hospital, depending on the clinical situation, 1–2 days before the planned surgical procedure.
2. Elective surgery should be postponed in patients with inadequate metabolic control, defined as persistently elevated blood glucose values > 250 mg/dl (13.9 mmol/l) on a daily profile, HbA_{1c} > 8.5%, and/or the presence of glycosuria with concomitant acetonuria and/or ketonaemia.
3. If a patient with type 2 diabetes treated with two or three antihyperglycaemic agents will not consume meals on the day of surgery or is scheduled for major surgery with an increased risk of haemodynamic instability, current therapy should be discontinued and temporary insulin therapy initiated.
4. When temporary insulin therapy is required, a multiple daily injection (basal bolus) regimen is recommended.
5. Total daily insulin dose: 0.3–0.7 units/kg body weight; 50–60% of the total daily dose should be short-acting (rapid-acting) insulin administered before main meals according to the following distribution: 50–20–30% of the total daily dose of short-acting (rapid-acting) insulin.

6. The remaining 40–50% of the total daily dose should be basal insulin:
- a long-acting analogue administered once daily, most commonly in the evening, or
- intermediate-acting insulin (NPH) administered in two injections at 07:00–08:00 (40%) and 22:00–23:00 (60%). A well-trained patient with diabetes who is metabolically well controlled and treated with intensive insulin therapy adjusts insulin doses independently according to current needs; therefore, during hospitalisation, this autonomy should be preserved and rigid, non-modifiable dosing regimens should be avoided.

7. Patients treated with a personal insulin pump, including automated insulin delivery (AID) systems, should continue their current therapy until the day of surgery and, in justified cases, perioperatively and throughout hospitalisation.
8. If preoperative preparation requires a strict diet on the day(s) preceding surgery, intravenous infusion of 500 ml of 10% glucose solution with 12 units of short-acting (rapid-acting) insulin and 10 mmol of KCl is recommended instead of a meal. The potassium dose should be adjusted according to serum potassium levels.
9. Achievement of glycaemic control: during the perioperative period, blood glucose should be monitored and maintained within safe limits of 100–180 mg/dl (5.6–10.0 mmol/l).
10. The surgical and anaesthetic team should be informed of diabetes-related complications that increase operative risk (cardiac or renal disease, neuropathy, proliferative retinopathy).

NOTE 4: Temporary insulin therapy is not required in patients undergoing low-risk procedures of short duration (e.g. tooth extraction, incision of an abscess, minor outpatient amputation, cataract surgery, arthroscopy, or colonoscopy performed under anaesthesia), provided that preoperative preparation does not require modification of usual dietary intake. If fasting for more than 12 hours is necessary due to the surgical procedure, an intravenous infusion of glucose with insulin and potassium is recommended [500 ml of 10% glucose solution with 12 units of short-acting (rapid-acting) insulin and 10 mmol KCl], administered at a rate of 100–150 ml/hour. Insulin and potassium doses should be adjusted according to blood glucose and serum potassium levels.

III. Management on the day of surgery

1. An intravenous infusion of glucose, insulin and potassium should be administered under blood glucose monitoring:
- Algorithm 1 – in individuals with absolute insulin deficiency, a separate continuous intravenous insulin infusion (solution concentration: 1 unit of short-acting/rapid-acting insulin in 1 ml of 0.9% NaCl) and a glucose solution (5–10%) administered via infusion pumps is recommended. To balance 1 g of exogenous glucose, 0.2–0.3 units of insulin are required (Table 26.1). If, during surgery, blood glucose increases by 30–50 mg/dl above 180 mg/dl, the insulin infusion rate should be increased by 1–2 units/hour. If blood glucose exceeds 250 mg/dl (13.9 mmol/l), the intravenous glucose infusion should be discontinued and resumed only after blood glucose has decreased to 180 mg/dl (10 mmol/l); a simultaneous increase in the intravenous insulin infusion rate is recommended. This management should be continued until oral intake is resumed. During intravenous insulin infusion, blood glucose should be monitored hourly, and after stabilisation (three consecutive stable measurements) every 2 hours.
- Algorithm 2 – in individuals with type 2 diabetes with preserved insulin secretion, a combined glucose–insulin–potassium solution may be used: 500 ml of 10% glucose containing 12–16 units of short-acting/rapid-acting insulin and 10–20 mmol of potassium chloride. The potas-

Table 26.1. Principles of infusion of 10% and 5% glucose solutions with insulin depending on blood glucose level

Blood glucose	10% glucose solution [ml/h]	5% glucose solution* [ml/h].	Insulin [units/h]
< 90 mg/dl	50	100	Stop infusion for 15–30 minutes
< 5.0 mmol/l			
90–119 mg/dl	50	100	0.5–2
5.0–6.7 mmol/l			
120–180 mg/dl	50	100	2–3
6.7–10 mmol/l			

**Note: The 5% glucose solution is preferred when better hydration and/or higher plasma osmolality is required.*

sium dose should be adjusted according to serum potassium levels:
   - » a higher insulin dose (≥ 16 units) should be considered in individuals with obesity, in the presence of severe infection, during cardiac surgery, in patients operated on under hypothermia, or when baseline blood glucose is > 180 mg/dl (10.0 mmol/l);
   - » a lower insulin dose (< 12 units) should be considered in lean individuals, as well as in those previously treated with low insulin doses or non-insulin antihyperglycaemic agents.
2. The intravenous infusion of glucose, insulin and potassium should be started in the morning at 08:00 and continued continuously at a rate of 80 ml/hour until normal oral intake is resumed.
3. During administration of the intravenous glucose–insulin–potassium infusion, blood glucose should be monitored at least every 2 hours and maintained within the range of 100–180 mg/dl (5.6–10.0 mmol/l):

- if plasma glucose decreases or remains at the lower limit of the recommended range, the insulin dose should be reduced by 2–4 units,
- it is recommended to increase the insulin dose in the infusion by 2 units for each 30 mg/dl (1.6 mmol/l) increase in plasma glucose above 180 mg/dl (> 10 mmol/l).

4. If continuous supervision of the operated patient with diabetes is feasible, Algorithm 1 should be preferred.

IV. Postoperative management

1. Insulin therapy using multiple daily injections or a personal insulin pump should be initiated when the patient resumes oral intake and continued (in the case of temporary insulin therapy) until discharge from hospital. If a long-acting or ultra-long-acting insulin analogue is used, administration should be continued on the day before surgery and resumed after surgery at a dose reduced to 80% of the preoperative dose.
2. In individuals with type 2 diabetes who were previously treated with non-insulin agents with good glycaemic control, these medications may be resumed once normal oral intake is restarted, provided there are no clinical contraindications.

Note 5: In individuals with diabetes previously treated with insulin who undergo surgery for an acute or chronic inflammatory condition, the possibility of a gradual daily reduction in insulin requirements should be considered.

Note 6: The use of CGM systems for perioperative glycaemic control reduces the risk of hypoglycaemia.

Table 26.2. Subcutaneous insulin therapy for minor procedures performed under general anaesthesia or sedation

Basal–bolus therapy	Basal: NPH insulin – 50% of the usual morning dose; long-acting insulin analogue – 100% of the usual morning dose
	Intravenous fluids should be initiated. In patients with normal glycaemia, glucose-free fluids may be administered initially. Subsequently, fluids containing 5–10% glucose should be given in amounts sufficient to prevent hypoglycaemia
	Morning procedures: · bolus insulin – only as a corrective dose if required · initiation of intravenous fluid therapy
	Afternoon procedures: · bolus insulin – if the patient can eat breakfast, administer the usual dose of a rapid-acting insulin analogue or 50% of the short-acting insulin dose; an additional corrective dose may be given if necessary · intravenous fluids should be started 2 hours before the procedure or no later than midday
Therapy using a personal insulin pump (CSII)	May be continued only if the anaesthesiologist accepts this method of therapy and is experienced in its use
	Continuation of insulin therapy at the pre-programmed basal rate appropriate for the time of day (basal rate modification is usually not necessary)
	Hypoglycaemia: suspend basal insulin delivery (maximum 30 minutes)
	Hyperglycaemia: corrective bolus
	Intravenous fluids should be initiated 2 hours before the procedure
Hybrid closed-loop system therapy	Continuation of therapy is recommended

Table 26.3. Perioperative management recommendations for non-insulin antihyperglycaemic agents

Medication used before planned surgery	Day before surgery	Day of surgery	After surgery	Comments
Sulfonylurea derivative	Administer	Withhold	Administer when oral intake is resumed	Withhold during temporary insulin therapy
Metformin	Administer	Withhold	Administer when oral intake is resumed	If iodinated contrast is required; metformin should be withheld for at least 24 hours before the procedure. Do not administer if eGFR < 45 ml/min/1.73 m²
Pioglitazone	Withhold	Withhold	Administer when oral intake is resumed	
DPP-4 inhibitor (gliptin)	Administer	Withhold or administer	Administer when oral intake is resumed	
SGLT2 inhibitor	Withhold	Withhold	Administer when oral intake is resumed	Elimination half-life is 12–14 hours, which justifies withholding the drug for 3 days before surgery
Oral GLP-1 receptor agonist or daily injectable GLP-1 receptor agonist	Administer	Withhold	Administer when oral intake is resumed	
Weekly injectable GLP-1 receptor agonist	Withhold	Withhold	Administer when oral intake is resumed	Omission of one dose of weekly GLP-1 RA (7-day drug-free period)*
Dual GIP/GLP-1 receptor agonist	Withhold	Withhold	Administer when oral intake is resumed	Omission of one dose before planned surgery (7-day drug-free period)*

**Withholding GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists is due to the risk of aspiration of gastric contents during intubation.*

Note 7: Recommendations for perioperative management of non-insulin antihyperglycaemic agents are presented in Table 26.3.

V. Management of low-risk procedures with an expected duration of less than 2 hours

For low-risk procedures with an expected duration of less than 2 hours, performed under general anaesthesia or sedation, in metabolically well-controlled patients, hospital admission should take place in the morning of the procedure or in the afternoon of the preceding day. Subcutaneous insulin therapy may be continued, or alternatively an algorithm as for major procedures may be applied (Table 26.2).

VI. Emergency and urgent surgery

In people with diabetes, urgent or emergency surgical intervention may sometimes be required.

In such cases, the presence of peritoneal signs resulting from diabetic ketoacidosis associated with metabolic decompensation should be excluded. Therefore, in patients presenting with symptoms of an acute abdomen accompanied by diabetic ketoacidosis (urinary acetone and markers of metabolic acidosis on blood gas analysis), immediate measures should be undertaken to correct acid–base disturbances.

In the presence of diabetic ketoacidosis (pH < 7.3; blood bicarbonate concentration <18 mEq/l) or hyperglycaemic hyperosmolar state, prior metabolic stabilisation in accordance with generally accepted principles is required. If surgical intervention cannot be postponed, correction of metabolic disturbances should be carried out concurrently with surgical management.

If there are no features of acute diabetes complications and the patient has taken the morning insulin dose, intravenous insulin infusion according to the above scheme should be used during the procedure.

REFERENCES

1. Boreland L, Scott-Hudson M, Hetherington K, et al. The effectiveness of tight glycemic control on decreasing surgical site infections and readmission rates in adult patients with diabetes undergoing cardiac surgery: a systematic review. Heart Lung 2015; 44: 430e–440e.

2. Cruz P, McKee AM, Chiang H-H, McGill, et al. Perioperative care of patients using wearable diabetes devices. Anesth Analg 2025; 140: 2–12.
3. Ehrenfeld JM, Wanderer JP, Terekhov M, et al. A perioperative systems design to improve intraoperative glucose monitoring is associated with a reduction in surgical site infections in a diabetic patient population. Anesthesiol 2017; 126: 431–440.
4. Galindo R, Migdal A, Davis G, et al. Comparison of the freestyle libre pro flash continuous glucose monitoring (CGM). System and Point-of-Care Capillary Glucose Testing in hospitalized patients with type 2 diabetes treated with basal-bolus insulin regimen. Diabetes Care 2020; 43: 2730–2735.
5. Hagerman A, Schorer R, Putzu A, et al. Cardioprotective effects of glucose-insulin-potassium infusion in patients undergoing cardiac surgery: a systematic review and meta-analysis. Semin Thoracic Surg 2022; 36: 167–181.
6. Panchal S, Mahmud N, Atkins JH, et al. Endoscopy and anesthesia outcomes associated with glucagon-like peptide-1 receptor agonist use in patients undergoing outpatient upper endoscopy. Gastrointest Endosc 2025; 102: 216–222.
7. Seki H, Ideno S, Shiga T, et al. Sodium-glucose cotransporter 2 inhibitor-associated perioperative ketoacidosis: a systematic review of case reports. J Anesth 2023; 37: 465–473.
8. Simha V, Shah P. Perioperative glucose control in patients with diabetes undergoing elective surgery. JAMA 2019; 321: 399–400.
9. Studzińska D, Szczeklik W. Praktyka kliniczna – opieka okołooperacyjna: opieka okołooperacyjna nad pacjentem z cukrzycą. Med Prakt 2019; 9: 110–119.

27. Vaccinations in Individuals with Diabetes

CHAPTER HIGHLIGHTS
• Every child with diabetes should be vaccinated in accordance with the current national immunisation programme (PSO, Polish National Immunisation Programme). [**C**]
• The adult vaccination schedule includes age-specific recommended vaccinations. [**C**]
• Annual influenza vaccination is recommended for children from the age of 6 months and for adults. [**C**] Influenza vaccination is a form of cardiovascular prevention. [**A**]
• Vaccination against hepatitis B virus (HBV) and COVID-19 is recommended for all individuals with diabetes. [**C**]

I. Introduction

Diabetes is associated with an increased susceptibility to infectious diseases, a more severe course of infections leading to more frequent hospitalisations, and a higher risk of death compared with the population without diabetes. All-cause mortality among individuals with diabetes who have received vaccinations is significantly lower than among unvaccinated individuals. In Poland, vaccination coverage against infectious diseases among individuals with diabetes remains below expectations and needs; therefore, increasing awareness among both individuals with diabetes and the physicians caring for them is essential to translate into measurable health benefits.

II. Children and adolescents

Every child with diabetes should be vaccinated in accordance with the current national immunisation programme (PSO). The vaccination schedule for children and adolescents is updated annually and published on the website of the Ministry of Health. It includes mandatory, recommended, and recommended free-of-charge vaccinations. All children born after 1 January 2017 should be routinely vaccinated against invasive *Streptococcus pneumoniae* disease (pneumococci). In children who have not received mandatory vaccinations according to the schedule, catch-up vaccination should be performed as soon as possible and is mandatory up to the age of 5 years. Children with diabetes born before 1 February 2017, as unvaccinated individuals belonging to a risk group, are subject to mandatory vaccination up to the age of 19 years. In children and adolescents, prevention of human papillomavirus (HPV) infection is also important, particularly in the context of reducing the future risk of malignancies, including cancers of the genital organs, vulva and anus, as well as head and neck cancers. Currently, a Universal HPV Vaccination Programme is implemented in Poland for children and adolescents aged 9–18 years.

Other recommended vaccinations include those against COVID-19, meningococci, influenza,

varicella, hepatitis A virus (HAV), and tick-borne encephalitis.

III. Adults

The vaccination schedule for adults, including recommended vaccinations according to age, is also available on the website of the Ministry of Health. In individuals with diabetes, particular emphasis should be placed on immunisation against hepatitis B virus (HBV), pneumococcal disease, and HPV. It should also be noted that diabetes increases the risk of infection and a severe course of respiratory infections such as influenza, pneumococcal pneumonia (including invasive pneumococcal disease), pertussis, COVID-19, and respiratory syncytial virus (RSV) infection. Influenza vaccination constitutes a form of cardiovascular prevention. Conversely, acute infection increases the risk of metabolic decompensation of diabetes. In individuals with diabetes, infection with varicella zoster virus (VZV) is also hazardous, as herpes zoster is associated with the risk of post-herpetic neuralgia.

IV. Hepatitis B virus infection

For hepatitis B virus, identification of unvaccinated individuals at any age and vaccination according to the 0, 1, and 6-month schedule are recommended. If, in previously vaccinated individuals, anti-HBs antibody titres <10 IU/l are detected, revaccination with 1–3 doses of the vaccine is recommended. If a protective antibody level is not achieved after administration of 3 doses (4–12 weeks after the last dose), further vaccination should be discontinued.

V. Pneumococcal disease

Vaccination against pneumococcal disease is recommended for all adults with diabetes, particularly those aged over 65 years.

VI. Human papillomavirus (HPV)

Vaccination against HPV is recommended in young adults aged 19–26 years. In individuals aged 27–49 years, the decision to vaccinate should be made on an individual basis after consultation with a physician and consideration of the potential benefits of vaccination.

VII. Influenza

In the case of influenza virus infection, an increased risk of hospitalisation and death has been demonstrated among individuals with diabetes. Available data indicate that influenza vaccination results in a significant reduction in mortality and hospitalisation risk in this group. Therefore, annual influenza vaccination is recommended for

Table 27.1. Adult Vaccination Schedule (based on current recommendations of the Ministry of Health)

Type of vaccination	Age (years)				
	19–26	27–49	50–59	60–64	≥ 65
Influenza	Annually during the influenza season (optimally at the beginning of the season)				
Diphtheria, tetanus, pertussis	Every 10 years				
Varicella (chickenpox)	2 doses – in individuals who have not had varicella and have not been vaccinated				
Measles, mumps, rubella (MMR)	2 doses – in individuals who have not had measles or rubella and have not been vaccinated				
COVID-19	According to vaccination history and current recommendations				
Hepatitis B (HBV)	3 doses in previously unvaccinated individuals				
Human papillomavirus (HPV)	3 doses	3 doses (individual decision)			
Pneumococcal disease			1 dose	1 dose	1 dose
Herpes zoster			2 doses administered 2–6 months apart		
Respiratory syncytial virus (RSV)				1 dose	
Vaccinations recommended in the presence of additional risk factors (medical, occupational, or lifestyle-related)					
Tick-borne encephalitis	3 doses + booster doses every 3–5 years				
Hepatitis A	2 doses – in previously unvaccinated individuals				
Meningococcal disease	1–2 doses				

Routine, mandatory, and recommended vaccinations prior to travel to endemic regions should be administered in accordance with the recommendations of the Ministry of Health of 16 September 2010 (Journal of Laws 2010, No. 180, item 1215), the CDC (US Centres for Disease Control and Prevention), WHO, and NHS (National Health Service, UK). A medical assessment is required prior to each vaccination.

children from the age of 6 months and for adults. Both standard-dose vaccines and high-dose (HD) vaccines are available and are recommended as the vaccine of first choice in individuals aged ≥ 60 years.

VIII. COVID-19

Due to the risk of a severe course of SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) infection in individuals with diabetes, including an increased risk of death, booster doses of COVID-19 vaccines are recommended, adapted to the current epidemiological situation.

IX. Diphtheria, tetanus, and pertussis

Vaccination against diphtheria, tetanus, and pertussis is recommended for all adults, including those with diabetes. Booster doses should be administered every 10 years. If vaccination history is unknown, vaccination should be performed according to the 0, 1, and 6-month schedule.

X. Respiratory syncytial virus (RSV)

The US Centers for Disease Control and Prevention (CDC) recommend a single dose of RSV vaccine for adults with diabetes aged 60 years and older.

XI. Varicella zoster virus (VZV) – varicella and herpes zoster

Non-immune individuals should be vaccinated against varicella (two doses administered 6 weeks apart). For individuals with chronic diseases, including diabetes, vaccination against herpes zoster consisting of two doses is recommended. This vaccination is particularly recommended for individuals aged ≥ 50 years and for those aged ≥ 18 years who are at increased risk of developing herpes zoster and a severe disease course.

XII. Other vaccinations

Non-immune individuals are also recommended to receive vaccinations against rubella, mumps, and measles, as infection with any of these diseases may result in severe metabolic decompensation of diabetes.

REFERENCES

1. Centers for Disease Control and Prevention. CDC recommends RSV vaccine for older adults. 2023. Dostępne na: https://www.cdc.gov/media/releases/2023/s0629-rsv.html.
2. D'Addio F, Lazzaroni E, Lunati ME, et al. Vaccinome Landscape in Nearly 620 000 Patients With Diabetes. J Clin Endocrinol Metab 2025; 110: e1590–e1597. DOI: 10.1210/clinem/dgae476.
3. Goeijenbier M, van Sloten TT, Slobbe L, et al. Benefits of flu vaccination for persons with diabetes mellitus: a review. Vaccine 2017; 35: 5095–5101.
4. Górska-Ciebiada M, Saryusz-Wolska M, Ciebiada M, et al. Pneumococcal and seasonal influenza vaccination among elderly patients with diabetes. Post Hig Doswiad 2015; 69: 1182–1189.
5. Heidecker B, Libby P, Vassiliou VS, et al. Vaccination as a new form of cardiovascular prevention: an ESC Clinical Consensus Statement. Eur Heart J 2025; 46 : 3518–3531.
6. https://szczepienia.pzh.gov.pl/kalendarz-szczepien-2025/
7. https://szczepienia.pzh.gov.pl/kalendarz-szczepien-doroslych/
8. Kuchar E, Antczak A, Skoczyńska A, et al. Szczepienia przeciw pneumokokom osób dorosłych – uaktualnione rekomendacje polskie. Fam Med Prim Care Rev 2022; 24. DOI: https://doi.org/10.5114/fmpcr.2022.119420.
9. Lina B, Georges A, Burtseva E, et al. Complicated hospitalization due to influenza: results from the Global Hospital Influenza Network for the 2017-2018 season. BMC Infect Dis 2020; 20: 465.
10. Modin D, Claggett B, Kober L, et al. Influenza vaccination is associated with reduced cardiovascular mortality in adults with diabetes: a nationwide cohort study. Diabetes Care 2020; 43: 2226–2233.
11. Nitsch-Osuch A, Jankowski P, Kokoszka-Paszkot J, et al. W stronę lepszej ochrony przed grypą osób starszych. Polskie rekomendacje dotyczące wysokodawkowej szczepionki przeciw grypie. Lekarz POZ 2024; 10: 77–87.
12. Phadke VK, Bednarczyk RA, Salmon DA, et al. Association between vaccine refusal and vaccine-preventable diseases in the United States review of measles and pertussis. JAMA 2016; 315: 1149–1158.
13. Polack FP, Thomas SJ, Kitchin N, et al. Safety and efficacy of the BNT162b2 mRNA COVID-19 vaccine. N Engl J Med 2020; 383: 2603–2615.
14. Szczepienia w podróżach międzynarodowych. W: Mrożek-Bucyn D. Wakcynologia Praktyczna, wyd. III. Medica Press, Bielsko-Biała 2012, 97–103.
15. Tomczyk S, Lynfield R, Schaffner W, et al. Prevention of antibiotic-nonsusceptible invasive pneumococcal disease with the 13-valent pneumococcal conjugate vaccine. Clin Infect Dis 2016; 62: 1119–1125.
16. Wolf J, Kist LF, Brangel Pereira S, et al. Human papilloma virus infection. Epidemiology, biology, host interactions, cancer development, prevention and therapeutics. Rev Med Virol 2024; 34: e2537.
17. Wysocki J, Siewiert B, Mastalerz-Migas A, et al. Szczepienia przeciwko COVID-19 u osób dorosłych w sezonie 2023/2024. Zalecenia Polskiego Towarzystwa Wakcynologii, Polskiego Towarzystwa Medycyny Rodzinnej, Polskiego Towarzystwa Epidemiologów i Lekarzy Chorób Zakaźnych oraz Polskiego Towarzystwa Ginekologów i Położników. Lekarz POZ 2024; 10: 23–34.

28. Recommendations on Occupational Activity in Individuals with Diabetes

Prepared in collaboration with Andrzej Marcinkiewicz, MD, PhD and Professor Jolanta Walusiak-Skorupa, MD, PhD (Nofer Institute of Occupational Medicine, Łódź).

The diagnosis of diabetes must not constitute grounds for discrimination or unequal treatment. Occupational restrictions should be imposed only after careful analysis of the individual situation and health status of the person concerned.

In addition to providing effective therapy, the role of the diabetologist in maintaining occupational activity in individuals with diabetes includes:

- health education aimed at developing health awareness and understanding of limitations resulting from potential diabetes-related complications,
- supporting the issuance of an objective opinion on health-related fitness for work by providing relevant information to the physician authorised to make certification decisions.

In the assessment of health status for occupational purposes, the attitude of the individual with diabetes should be a key factor guiding the physician's decision. Every individual with diabetes, regardless of diabetes type or treatment modality, must actively participate in the management of their condition.

The assessment of health fitness for occupational tasks or for driving vehicles is carried out by a physician authorised to perform occupational health examinations or driver medical assessments. Due to the often-incidental nature of contact with the patient (frequently limited to a single visit), it is advisable, in order to enable an individualised and well-founded decision, that the individual with diabetes presents an opinion from their treating physician.

As part of a diabetology consultation for certification purposes, the diabetologist should:

- assess the individual's knowledge of their disease, treatment, and potential complications on a scale of: high, adequate, or inadequate,
- assess glycaemic self-management skills on a scale of: good, acceptable, or poor,
- assess hypoglycaemia awareness and the ability to prevent and manage hypoglycaemia on a scale of: adequate or inadequate,
- confirm the presence or absence of awareness of prodromal symptoms of hypoglycaemia,
- determine the risk of hypoglycaemia as: low, acceptable, or high,
- identify the presence of chronic diabetes complications affecting the visual system, nervous system, or cardiovascular system,
- take into account the skills and ability to consistently use continuous glucose monitoring (CGM) systems,
- include additional remarks regarding chronic diabetes complications and the current health status of the individual that are relevant to the assessment of risk to public safety.

The justification for occupational restrictions in individuals with diabetes is twofold and results from:

- the possibility of hypoglycaemic episodes and associated disturbances of consciousness,
- the potential development of late diabetes complications that may impair the ability to perform specific work tasks.

Contraindications to driving vehicles by category, as well as contraindications to employment in specific occupational positions, are presented in Appendix 2.

Individuals with advanced chronic diabetes complications must not perform tasks in which organ damage related to diabetes complications could affect workplace safety. However, this should not preclude employment in other types of work for which the given complication does not pose a risk. At the same time, the nature and burden of the work should not hinder the achievement of adequate metabolic control of diabetes and, consequently, protection against the development and progression of chronic complications.

A diabetology consultation for driver or occupational assessments should conclude with the issuance of a clear opinion in the form of structured consultation forms, templates of which are provided in Appendix 2.

Health requirements for individuals with diabetes should be divided into two categories, depending on the occupational tasks performed or the position held.

The first (higher) category includes tasks and positions requiring full psychomotor fitness and

involving exposure to adverse psychosocial factors, where performance is associated with the safety of the worker themselves and their surroundings (co-workers and other individuals not directly involved in the work but located in close proximity or affected by the performed activities, such as road users or customers in large retail environments). More restrictive health requirements apply particularly in the context of potential disturbances of consciousness, which in individuals with diabetes may result from severe hypoglycaemia.

Occupations requiring higher-category health requirements, in which the presence of diabetes must be carefully considered, include those related to public safety, such as:

- professional driving of vehicles (passenger transport, freight transport, operation of surface and underground railways, taxi services),
- uniformed and emergency services: armed forces (land forces, navy, air force), police, fire service, municipal guards, emergency medical services, maritime navigation, prison service, licensed security personnel,
- civil aviation professions: pilots and aviation engineers, cabin crew, air traffic controllers,
- particularly hazardous occupations (work at heights, work with moving machinery, furnaces, high-temperature environments, incineration plants, steelworks, mining, areas with heavy traffic, and other activities associated with a high risk of accidents).

The second (lower) category of health requirements includes tasks, positions, and associated harmful or burdensome factors that may adversely affect the course of diabetes. In this category, it is more appropriate to refer to occupations or positions that are not recommended rather than to absolute contraindications. Therefore, decisions regarding the initiation or continuation of employment in the following positions require particular caution and individual assessment:

- occupations requiring increased physical exertion, especially of a static nature (e.g. miner, steelworker),
- shift work and night work,
- exposure to carbon disulphide and pesticides, including dichlorophenoxyacetic acid derivatives (e.g. dichlorprop, mecoprop).

The diabetologist should fulfil an advisory role for young individuals with diabetes, for whom the choice of profession requires particular care. In such cases, not only the current health status but, above all, the natural history of diabetes should be taken into account, as at different stages of disease duration health limitations may preclude not only vocational training but, in the longer term, the performance of professional work.

Appendix 3 contains the document Charter of Rights and Responsibilities of the Employer and the Employee, the purpose of which is, on the one hand, to strengthen the sense of responsibility and professional position of individuals with diabetes and, on the other, to counteract their exclusion from the labour market.

29. Diabetological Care in Penitentiary Institutions

Individuals with diabetes who are detained in penitentiary institutions (prisons, remand centres, correctional facilities) should be guaranteed access to the same standard of medical care, including diabetological care, as that provided to the general patient population.

Personnel of the institution should be informed about the individual's diabetes and adequately trained in the recognition of hyperglycaemia and hypoglycaemia, as well as in appropriate management in the event of these conditions or other medical emergencies.

30. Management of Obesity in Individuals with Diabetes

Type 2 diabetes is, in the vast majority of cases, a direct consequence of overweight or obesity, which are present in approximately 80% of individuals with this type of diabetes. Obesity is also observed in around 10–15% of individuals with type 1 diabetes.

Effective management of obesity leads to improved metabolic control of diabetes and better outcomes in the treatment of many other conditions associated with excess body weight, most notably arterial hypertension, dyslipidaemia, MASLD, heart failure, musculoskeletal disorders, mental health conditions, and various degrees of eating disorders. While weight reduction is the primary goal of obesity management, treatment should address the individual's overall health status. From a comprehensive perspective, the goals of obesity management include:

- improvement in quality of life and overall health (both physical and mental),
- prevention of the development of obesity-related diseases (complications) or mitigation of their course, improvement in disease control, and, in some cases, achievement of remission.

In every individual with diabetes, body weight, waist circumference (according to IDF criteria, abdominal obesity in Europeans is defined as a waist circumference ≥ 94 cm in men and ≥ 80 cm in women), body mass index (BMI), and, where appropriate, the category of overweight or obesity should be assessed at every clinical visit. If overweight is identified (BMI 25.0–29.9 kg/m²), preventive measures should be implemented to avoid further weight gain. In the presence of obesity, appropriate therapeutic interventions should be initiated.

30.1 Non-Pharmacological Treatment and Pharmacotherapy

CHAPTER HIGHLIGHTS:

- In every individual with diabetes and obesity, therapeutic management of a non-pharmacological and pharmacological nature should be recommended. [**A**]
- In every individual with diabetes with a BMI ≥ 27 kg/m², pharmacotherapy using GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists should be recommended, taking into account the antihyperglycaemic effects of these agents. [**A**]

Behavioural intervention is a permanent component of obesity management. From the beginning of therapy, the individual should receive recommendations regarding optimal nutrition and physical activity, tailored to age, level of physical fitness, and individual preferences. The presence of potential mental health disorders should also be taken into account and assessed to determine whether they require specific treatment. Therapeutic education in this area should be continuous and delivered at every medical visit, optimally with the involvement of the entire multidisciplinary care team. Detailed recommendations regarding behavioural therapy are presented in Chapters 6–8.

Pharmacological treatment of obesity in individuals with diabetes consists of the use of GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists. In the majority of individuals, these agents enable a reduction in body weight of more than 10%, while simultaneously improving metabolic control of diabetes and outcomes of comorbid conditions. This may necessitate modification of concomitant pharmacotherapy, such as reduction of insulin doses or antihypertensive medications.

It should be emphasised that in individuals with type 2 diabetes, dulaglutide, liraglutide and semaglutide reduce cardiovascular and renal risk, while for tirzepatide a beneficial effect on cardiovascular risk has been demonstrated. Semaglutide exerts a similar effect in individuals with obesity and high cardiovascular risk, both in those with prediabetes and in those with normal glucose tolerance.

With regard to body weight reduction, the mechanism of action of agents stimulating GLP-1 and GIP receptors primarily involves increased satiety, reduced appetite, and consequently a decrease in caloric intake. For this reason, individuals should be made aware of the need to adhere to dietary composition recommendations in order to prevent loss of fat-free (muscle) mass. Accordingly, adequate intake of protein-rich foods and maintenance of appropriate physical activity, including resistance exercise, are recommended during incretin-based pharmacotherapy for obesity. The most effective method for assessing muscle mass is body composition analysis.

Effective treatment of obesity in women of reproductive age has a beneficial impact on the likelihood of conception. During pharmacological treatment of obesity, women should be advised to use contraception. GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists have not been approved for use during pregnancy and should not be used during pregnancy planning or during the reproductive period if contraception is not used. These agents should be discontinued 4–8 weeks prior to a planned pregnancy, and in the case of an unplanned pregnancy, immediately after confirmation.

Dietetic consultation should be performed in all individuals initiating or continuing treatment with GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists. This is particularly recommended in individuals who experience significant gastrointestinal adverse effects, who do not achieve clinically meaningful weight reduction, or who experience rapid weight loss that increases the risk of muscle mass loss and nutritional deficiencies.

In the case of effective obesity treatment, defined as prevention of further progressive weight gain, pharmacotherapy should not be discontinued and should be regarded as long-term treatment.

30.2 Metabolic Surgery

CHAPTER HIGHLIGHTS:

- Surgical treatment of obesity should be considered in individuals with type 2 diabetes and a body mass index greater than 35 kg/m², particularly when conservative obesity management has been ineffective, and especially in the presence of comorbid conditions and inadequate glycaemic control despite behavioural therapy and the use of antihyperglycaemic medications. [**A**]
- Every individual who has undergone surgical treatment for diabetes should remain under the long-term care of a diabetologist and a general surgeon and should receive continuous supplementation with vitamins and micronutrients in order to prevent deficiencies. [**C**]

Metabolic surgery is an effective method for the treatment of obesity and associated conditions, particularly type 2 diabetes. A multidisciplinary approach enables appropriate patient selection for metabolic surgery and the choice of the most suitable surgical technique.

I. Criteria for qualification for metabolic surgery

1. Metabolic surgery should be considered in every individual with type 2 diabetes and a body mass index greater than 35 kg/m², particularly in the presence of additional comorbidities such as arterial hypertension and lipid disorders. Qualification for metabolic surgery should be especially considered when type 2 diabetes and obesity respond inadequately to pharmacological and behavioural therapy.
2. Qualification for metabolic surgery is recommended in every individual with type 2 diabetes and a body mass index greater than 40 kg/m².
3. After taking into account the preferences of the individual with type 2 diabetes, surgical treatment of obesity may also be considered in individuals with class I obesity (body mass index 30.0–34.9 kg/m²) in whom non-surgical methods, primarily pharmacotherapy using GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists, fail to achieve sustained weight reduction and improvement in the control of comorbid conditions.
4. Individuals with type 2 diabetes aged between 18 and 65 years are eligible for metabolic surgery. In justified cases, the upper age limit may be extended to 70 years, provided that the individually assessed surgical risk is lower than

the potential benefits achievable through the procedure.

5. Qualification for bariatric surgery should be performed by a medical team including at least a diabetologist and a general surgeon with extensive experience in metabolic surgery. It is recommended that the multidisciplinary team involved in the qualification process also includes a cardiologist, pulmonologist, psychologist or psychiatrist, anaesthesiologist, and dietitian, as well as a nurse and a social worker (Figure 30.1, Table 30.1).

II. Types of metabolic surgical procedures

1. It is recommended that individuals are qualified for surgical procedures performed using minimally invasive techniques (laparoscopy).
2. In light of available evidence, individuals with type 2 diabetes should primarily be considered for the following laparoscopic procedures: Roux-en-Y gastric bypass, loop gastric bypass (mini gastric bypass), sleeve gastrectomy, biliopancreatic diversion, and single anastomosis duodeno-ileal bypass (SADI).
3. The choice of surgical procedure should be made following surgical consultation and an individual assessment of the advantages and disadvantages of each of the aforementioned metabolic surgery techniques.
4. Prior to making a decision regarding metabolic surgery, individuals should be familiarised with the informed consent forms prepared by the Polish Society of Surgeons.

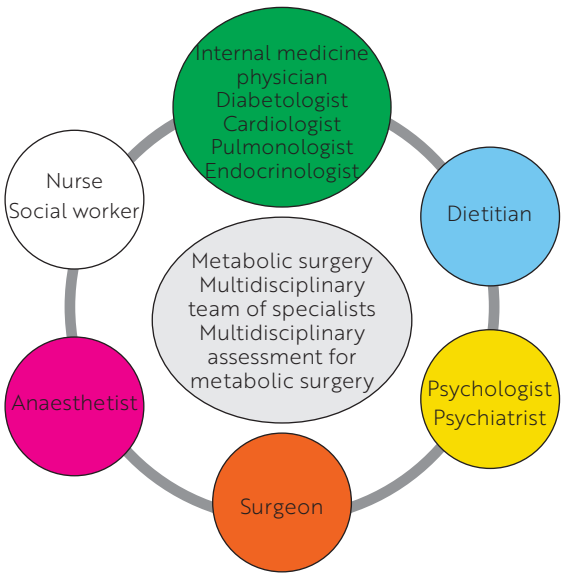


Figure 30.1. Multidisciplinary qualification for metabolic surgery

III. Complications associated with metabolic surgery in type 2 diabetes

Within 30 days after surgery, mortality associated with metabolic surgery is estimated at 0.1–0.3%, which is comparable to the mortality risk associated with laparoscopic cholecystectomy and is considered low. The most common complications following metabolic surgery include anastomotic leakage (3.1%), surgical site infection (2.3%), respiratory complications (2.3%), and gastrointestinal bleeding (1.7%).

IV. Evaluation of outcomes of metabolic surgery in type 2 diabetes

Type 2 diabetes resolves in 40–95% of individuals, depending on disease duration, baseline degree of obesity, and the type of surgical procedure performed. The following approach to evaluating outcomes of surgical treatment of type 2 diabetes is recommended:

Resolution of diabetes and comorbid conditions. Resolution of disease may be defined as discontinuation of pharmacotherapy with the following criteria fulfilled:

- HbA_{1c} level < 6.5%,
- absence of hypoglycaemic episodes,
- total cholesterol concentration < 4 mmol/l and LDL cholesterol < 2 mmol/l,
- triglyceride concentration < 2.2 mmol/l,
- blood pressure values < 140/90 mm Hg,
- body weight reduction > 15% compared with body weight at the time of surgical qualification.

Improvement in disease course. Improvement following metabolic surgery may be defined as, after reduction of preoperative medication doses:

- a decrease in HbA_{1c} of > 20%,
- LDL cholesterol concentration < 2.6 mmol/l,
- blood pressure values < 140/90 mm Hg.

V. Recommendations following surgical treatment of type 2 diabetes

1. Every individual after surgical treatment of diabetes should remain under permanent care of a diabetologist and a general surgeon.
2. Lifelong supplementation with vitamins and micronutrients is required to prevent deficiencies.

VI. Pregnancy and metabolic surgery

1. There are no contraindications to pregnancy in women after metabolic surgery once 24 months have elapsed since bariatric surgery.

Table 30.1. Additional investigations and consultations prior to planned metabolic surgery

Laboratory investigations	Blood group Complete blood count, serum sodium, potassium, urea, creatinine, TSH and cortisol Coagulation parameters Fasting plasma glucose and glycated haemoglobin (HbA_{1c})
Endoscopic and imaging investigations	Gastroscopy Abdominal ultrasound
Specialist consultations	Cardiology consultation (including ECG and echocardiography) Pulmonology consultation (including chest X-ray, spirometry, and polysomnography if indicated) Otolaryngology consultation Endocrinology consultation (in cases of abnormal serum TSH or cortisol levels)

2. Prior to conception and throughout pregnancy, regular follow-up with the treating diabetologist is recommended.

VII. Contraindications to qualification of individuals with type 2 diabetes for metabolic surgery

1. Absolute contraindications:
- lack of acceptance of surgical treatment of type 2 diabetes by the individual; alcohol or substance dependence (qualification for surgical treatment of obesity may be considered after at least one year of documented abstinence),
- psychiatric disorders that remain uncontrolled despite treatment and pharmacotherapy,
- high cardiovascular risk associated with surgery,
- endocrine disorders underlying obesity (e.g. Cushing's syndrome),
- inability to participate in long-term postoperative follow-up,
- the 24-month period preceding a planned pregnancy and breastfeeding.

2. Relative contraindications:
- weight gain in the period immediately preceding surgery, indicating lack of cooperation,
- active peptic ulcer disease, which requires treatment prior to surgery; in individuals with asymptomatic Helicobacter pylori infection, eradication before surgery is recommended but not mandatory,
- in individuals with a history of oncological treatment, oncological consultation confirming effective cancer treatment is required.

REFERENCES

1. Adams TD, Gress RE, Smith SC, et al. Longterm mortality after gastric bypass surgery. N Engl J Med 2007; 357: 753–761.
2. Alberti KG, Zimmet P, Shaw J; IDF Epidemiology Task Force Consensus Group. The metabolic syndrome – a new worldwide definition. Lancet 2005; 366: 1059–1062.
3. Arterburn DE, Olsen MK, Smith VA, et al Association between bariatric surgery and long-term survival. JAMA 2015; 313: 62–70.
4. Bliddal H, Bays H, Czernichow S, et al. Once-weekly semaglutide in persons with obesity and knee osteoarthritis. N Engl J Med 2024; 391: 1573–1583.
5. Budzyński A, Major P, Głuszek S, et al. Polskie rekomendacje w zakresie chirurgii bariatrycznej i metabolicznej. Medycyna Praktyczna Chirurgia 2016; 6: 13–26.
6. De Luca M, Shikora S, Eisenberg D, et al. Scientific evidence for the updated guidelines on indications for metabolic and bariatric surgery (IFSO/ASMBS). Surg Obes Relat Dis 2024; 20: 991–1025.
7. Eisenberg D, Shikora SA, Aarts E, et al. 2022 American Society for Metabolic and Bariatric Surgery (ASMBS) and International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO): Indications for Metabolic and Bariatric Surgery. Surg Obes Relat Dis 2022; 18: 1345–1356.
8. ElSayed NA, Aleppo G, Aroda VR, et al.; on behalf of the American Diabetes Association. 8. Obesity and weight management for the prevention and treatment of type 2 diabetes: standards of care in diabetes — 2023. Diabetes Care 2023; 46 (Suppl 1): S128–S139.
9. Frías JP, Davies MJ, Rosenstock J, et al. Tirzepatide versus semaglutide once weekly in patients with type 2 diabetes. N Engl J Med 2021; 385: 503–515.
10. Janez A, Muzurovic E, Bogdanski P, et al. Modern management of cardiometabolic continuum: from overweight/obesity to prediabetes/type 2 diabetes mellitus. Recommendations from the Eastern and Southern Europe Diabetes and Obesity Expert Group. Diabetes Ther 2024; 15: 1865–1892.
11. Kosiborod MN, Petrie MC, Borlaug BA, et al. Semaglutide in patients with obesity-related heart failure and type 2 diabetes. N Engl J Med 2024; 390: 1394–1407.

12. Lincoff AM, Brown-Frandsen K, Colhoun HM, et al. Semaglutide and Cardiovascular Outcomes in Obesity without Diabetes. N Engl J Med 2023; 389: 2221–2232.
13. Mingrone G, Panunzi S, De Gaetano A, et al. Bariatric-metabolic surgery versus conventional medical treatment in obese patients with type 2 diabetes: 5 year follow-up of an open-label, single-centre, randomised controlled trial. Lancet 2015; 386: 964–973.
14. Perkovic V, Tuttle KR, Rossing P, et al. Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes. N Engl J Med 2024; 391: 109–121.
15. Rubino F, Nathan DM, Eckel RH, et al. Metabolic surgery in the treatment algorithm for type 2 diabetes: a joint statement by international diabetes organizations. Diabetes Care 2016; 39: 861–877.
16. Schauer PR, Bhatt DL, Kirwan JP, et al.; STAMPEDE Investigators. Bariatric surgery versus intensive medical therapy for diabetes – 5-year outcomes. N Engl J Med 2017; 376: 641–651.
17. Sjostrom L, Lindroos AK, Peltonen M, et al.; Swedish Obese Subjects Study Scientific Group. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med 2004; 351: 2683–2693.
18. Sjostrom L, Narbro K, Sjostrom CD, et al.; Swedish Obese Subjects Study. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med 2007; 357: 741–752.
19. Zaveri H, Surve A, Cottam D, et al. Mid-term 4-year outcomes with single anastomosis duodenal-ileal bypass with sleeve gastrectomy surgery at a single US center. Obes Surg 2018; 28: 3062–3072.

31. Selected Special Situations and Conditions in Individuals with Diabetes

This chapter was prepared in collaboration with Professor Renata Górska, Professor Nadia Sawicka-Gutaj, Professor Jolanta Słowikowska-Hilczer, Professor Michał Rabijewski, and Professor Marek Ruchała.

I. Shift work

Shift work may be associated both with an increased risk of developing diabetes and with poorer glycaemic control. It necessitates periodic changes in the timing of administration of oral antihyperglycaemic agents or insulin.

1. In individuals with diabetes working shifts, intensive self-monitoring of blood glucose is required, particularly during periods of changes in working hours [E].
2. In individuals with diabetes working shifts, antihyperglycaemic agents associated with a low risk of hypoglycaemia and allowing greater dosing flexibility are preferred. In individuals treated with long-acting insulin, preparations providing a stable basal insulin level throughout the entire day should be selected [E].
3. Individuals with diabetes, particularly type 1 diabetes, treated with insulin should be capable of adjusting insulin doses (intensive functional insulin therapy).

II. Crossing time zones

Travel is not contraindicated in individuals with diabetes. Individuals with diabetes, particularly those with type 1 diabetes and those with type 2 diabetes treated with insulin, should prepare for travel by taking into account, among other factors, travel duration, mode of transport, changes in time zones (including the direction of travel – eastward or westward), and the climate of the destination country. Rapid changes in time zones (air travel) may pose particular challenges.

1. Individuals with diabetes, particularly those with type 1 diabetes treated with insulin, should exercise special caution during the period of adaptation to a new time zone (assumed to last as many days as the number of hours of time difference). During this period, frequent blood glucose monitoring is required.
2. Long-acting insulin analogues that provide a stable basal insulin level throughout the day may be administered at the same dose and at the same clock time according to the new local time. These preparations should be preferred, particularly in individuals who travel frequently [E].
3. In individuals treated with NPH insulin or long-acting insulin analogues that do not provide a stable basal level throughout the entire day, westward air travel (day prolongation) requires administration of the usual evening dose of long-acting insulin according to the new local time. Any hyperglycaemia resulting, for example, from in-flight meals may be corrected with additional doses of short-acting

insulin or rapid-acting insulin analogues. In the case of eastward travel (day shortening), a reduction in the evening dose of long-acting insulin may be necessary [E].

4. Individuals treated with a personal insulin pump do not need to adjust the pump clock or insulin doses if the time change does not exceed 2 hours. If the time change is greater and the planned stay in the new time zone is prolonged, gradual shifting of the basal infusion time frame by 2 hours per day is recommended.

III. Glucocorticosteroid therapy

Many medications have diabetogenic effects. Of particular importance is the diabetogenic effect of glucocorticosteroids, both because of the strength of this effect and the frequency of their use. Glucocorticosteroids primarily cause an increase in postprandial glycaemia.

1. Replacement doses of glucocorticosteroids (hydrocortisone up to 20 mg/day) and inhaled glucocorticosteroids do not have a significant impact on carbohydrate metabolism.
2. Factors increasing the risk of steroid-induced diabetes include older age, obesity, impaired glucose tolerance, high doses of glucocorticosteroids, and concomitant use of other diabetogenic drugs.
3. In the treatment of glucocorticosteroid-induced diabetes, insulin administered in an intensive insulin therapy regimen is preferred. Alternatively, only short-acting or rapid-acting insulin preparations may be administered before meals if fasting and preprandial glycaemia are acceptable. No insulin preparation or analogue has been shown to be superior to others in steroid-induced diabetes.
4. In individuals with type 2 diabetes treated with oral antihyperglycaemic agents who require temporary glucocorticosteroid therapy, particularly at high doses, temporary insulin therapy using an intensive insulin regimen is recommended.
5. In individuals with type 2 diabetes treated with basal insulin only (NPH insulin or long-acting insulin analogues), it is usually necessary to add short-acting or rapid-acting insulin before meals.
6. In individuals with diabetes treated with insulin, the use of glucocorticosteroids is associated with an increased insulin requirement, predominantly during the daytime.

IV. Periodontal disease

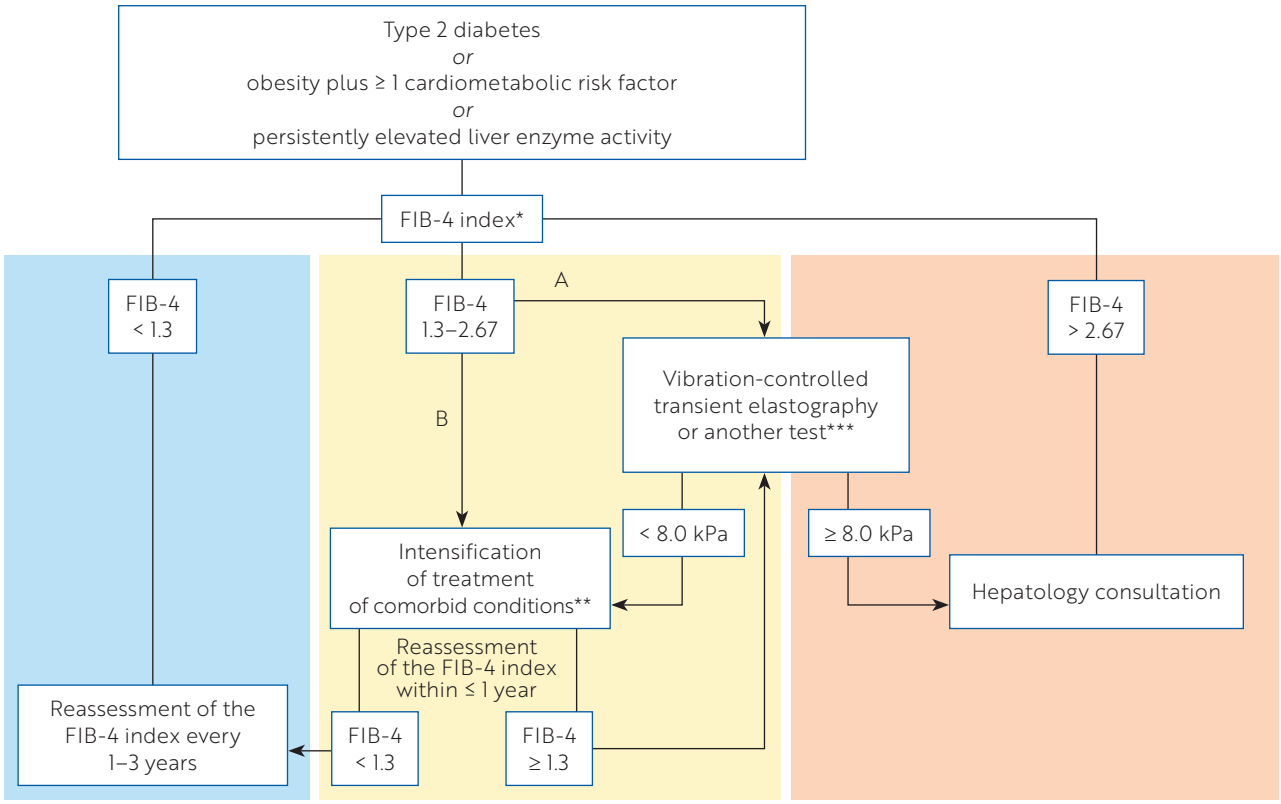
Periodontal diseases and other oral conditions occur more frequently in individuals with diabetes. Periodontal disease adversely affects metabolic control of diabetes and increases the risk of its complications. Treatment of periodontal disease improves glycaemic control.

1. In every individual with diabetes, history taking and physical examination should include assessment focused on oral diseases.
2. Every individual with diabetes should undergo a dental examination once a year.

V. Metabolic dysfunction-associated steatotic liver disease (MASLD)

MASLD, a non-classical complication of diabetes, is diagnosed in women consuming less than 140 g of alcohol per week and in men consuming less than 210 g per week, provided that at least one of the criteria listed below is met. Alcohol consumption of 140–350 g/week in women or 210–420 g/week in men allows for the diagnosis of metabolic and alcohol-associated liver disease (MetALD) in individuals meeting at least one of these criteria. Higher alcohol intake warrants the diagnosis of alcohol-related liver disease (ALD).

1. MASLD may be diagnosed in an individual with hepatic steatosis (identified by imaging or biopsy) who meets at least one of the following criteria [E]:
   - in individuals of White ethnicity: BMI ≥ 25 kg/m² or waist circumference > 94 cm in men (80 cm in women),
   - presence of diabetes or prediabetes,
   - blood pressure ≥ 130/85 mm Hg or use of antihypertensive medication,
   - triglyceride concentration ≥ 150 mg/dl (1.7 mmol/l) or use of lipid-lowering medication,
   - HDL cholesterol < 40 mg/dl (1.0 mmol/l) in men or < 50 mg/dl (1.3 mmol/l) in women, or use of lipid-lowering medication.
2. MASLD can be expected in the majority of individuals with diabetes. Approximately half of these individuals have liver fibrosis, which is associated with an increased risk of liver cirrhosis, gastrointestinal malignancies (including hepatocellular carcinoma), cardio–reno–metabolic complications, and death. The FIB-4 index is recommended as a screening tool (calculators are available, for example, on the website of the Polish Society of Gastroenterology). A FIB-4 value < 1.3 indicates low risk of liver fibrosis, while > 2.67 indicates high risk.



**FIB-4 thresholds apply to individuals aged ≤ 65 years (in those aged > 65 years, the lower FIB-4 cut-off is 2.0).*

***For example, lifestyle modification, treatment of comorbidities (e.g. GLP-1 receptor agonists), bariatric surgery.*

****For example, MRE (magnetic resonance elastography), SWE (shear wave elastography), ELF (enhanced liver fibrosis), using adapted cut-off values.*

A and B – options depending on disease history, clinical context, and local resources.

Figure 31.1. Proposed strategy for assessing the risk of advanced liver fibrosis and liver-related endpoints in individuals with MASLD according to EASL–EASD–EASO [3]

In individuals with intermediate (1.3–2.67) or high risk, confirmation using fibroelastography or other methods, such as magnetic resonance imaging or liver biopsy, may be indicated [B] (Figure 31.1).

3. In Europe, the only drug registered for the treatment of metabolic dysfunction-associated steatohepatitis (MASH) is resmetirom, a thyroid hormone receptor beta (THR-β) agonist acting in the liver (in the USA, semaglutide is also registered). Behavioural interventions (diet, physical activity, weight reduction, smoking cessation, and abstinence from alcohol) as well as pharmacological treatment (GLP-1 receptor agonists or dual GIP/GLP-1 receptor agonists, SGLT2 inhibitors, pioglitazone, and metformin) are also indicated, together with assessment and modification of other cardiovascular risk factors, including statin therapy.

VI. Hypogonadism in men with diabetes

1. Partial hypogonadism occurs in 20–50% of men with type 2 diabetes. During diabetology visits, attention should be paid to clinical symptoms suggestive of hypogonadism, including erectile dysfunction (particularly reduced frequency of morning erections), reduced libido, chronic fatigue, depressive symptoms, difficulty reducing fat mass despite lifestyle modification and pharmacotherapy, and decreased muscle mass and strength. Routine measurement of serum testosterone concentration is not recommended in men with type 2 diabetes who do not present with clinical symptoms of hypogonadism.
2. In symptomatic individuals in whom total testosterone concentration confirmed on a second measurement performed at least 4 weeks apart is < 12 nmol/l (350 ng/dl) and/

or calculated free testosterone (cfT) is < 220 pmol/l (65 pg/ml), hypogonadism should be diagnosed and referral to an endocrinologist for consideration of testosterone therapy is recommended.

REFERENCES

1. Benbenek-Klupa T. Chory na cukrzycę w podróży. W: Franek E, Walicka W (red.). Leczenie cukrzycy w praktyce klinicznej. Tom 1. Wydawnictwo PZWL, Warszawa 2018.
2. Corona G, Goulis DG, Huhtaniemi I, et al. European Academy of Andrology (EAA) guidelines on investigation, treatment and monitoring of functional hypogonadism in males: Endorsing organization: European Society of Endocrinology. Andrology 2020; 8: 970–987.
3. Cusi K, Orsak B, Bril F, et al. Long-term pioglitazone treatment for patients with nonalcoholic steatohepatitis and prediabetes or type 2 diabetes mellitus: a randomized trial. Ann Intern Med 2016; 165: 305–315.
4. EASL–EASD–EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD). J Hepatol 2024; 81: 492–542.
5. Fang L, Li J, Zeng H, Liu J. Effects of GLP-1 receptor agonists on the degree of liver fibrosis and CRP in non-alcoholic fatty liver disease and non-alcoholic steatohepatitis: a systematic review and meta-analysis. Prim Care Diabetes 2024; 18: 268–276.
6. Knutsson A, Kempe A. Shift work and diabetes – a systematic review. Chronobiol Int 2014; 31: 1146–1151.
7. Lassailly G, Caiazzo R, Ntandja-Wandji LC, et al. Bariatric surgery provides long-term resolution of nonalcoholic steatohepatitis and regression of fibrosis. Gastroenterology 2020; 159: 1290-1301.e5. DOI: 10.1053/j.gastro.2020.06.006.
8. Loomba R , Hartman ML, Lawitz EJ, et al. Tirzepatide for metabolic dysfunction-associated steatohepatitis with liver fibrosis. N Engl J Med 2024; 391: 299–310.
9. Rinella ME, Lazarus JV, Ratziu V, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. Hepatology 2023; 78: 1966–1986.
10. Targher G, Byrne CD, Tilg H. MASLD: a systemic metabolic disorder with cardiovascular and malignant complications. Gut 2024; 73: 691–702.
11. Wallace MD, Metzger NL. Optimizing the treatment of steroid-induced hyperglycemia. Ann Pharmacother 2018; 52: 86–90.

Appendix 1.

Organisational Requirements for Diabetology Care for Adults. Recommendations regarding diabetology care for children and adolescents with diabetes – see Chapter 23, Section VII

Specialist diabetology wards

1. Medical staff:
- two full-time diabetology specialists.
2. Nursing staff:
- a nurse specialising in diabetology nursing, general (medical) nursing, or internal medicine nursing, or a nurse undergoing specialisation in internal medicine nursing, or having completed a qualifying course in diabetology nursing, or undergoing such a qualifying course, or a nurse who has completed a specialist course in diabetes education,
- in the treatment of adults, in accordance with current legal regulations (Act or Regulation of the Ministry of Health), a staffing equivalent to at least 0.6 full-time posts per bed, and additionally one nurse whose duties are limited exclusively to diabetes education and care, at a ratio of one nurse per 15–20 diabetology beds, working exclusively during daytime hours.
3. Dietitian:
- at least 1 full-time post.
4. Psychologist:
- at least 0.5 full-time post.
5. Access to specialist consultations.
6. Equipment:
- at least two stations dedicated to the management of patients in acute metabolic conditions, equipped with ECG monitoring, blood pressure monitoring, an infusion pump, a pulse oximeter, access to oxygen therapy, and ECG,
- intravenous infusion pumps (minimum of two),
- a separate education room for group or individual education, equipped with educational tools, including multimedia resources, with the ability to read and analyse data from glucose meters, insulin pumps, continuous glucose monitoring systems, and automated insulin delivery (AID) systems using information technology systems,
- a body composition analyser,
- equipment for the diagnosis of neuropathy and for the diagnosis and treatment of diabetic foot syndrome (thermal tip, tuning fork – 128 Hz, 10 g monofilament, reflex hammer),
- devices for local negative pressure wound therapy,
- access to a room functioning as a wound care clinic with a full range of dressings and a set of surgical instruments (haemostatic forceps, needle holders, bone curettes, surgical scissors),
- access to cardiological diagnostics (exercise testing, ECG, echocardiography (ECHO), Holter ECG monitoring, ambulatory blood pressure monitoring) and vascular diagnostics (Doppler ultrasound of arteries).

Diabetes Poland takes the position that, within inpatient diabetology services, hospitalisations should include those that may be conducted in internal medicine wards and specialist diabetology wards, as well as hospitalisations that should be carried out exclusively in specialist diabetology wards. Specialist hospitalisations require additional diagnostic tests and procedures dictated by specific clinical situations, as well as several hours of daily education, and dietary and psychological counselling when indicated. Furthermore, in order to improve the quality of diabetology care and specialist training, the operation of a diabetology outpatient clinic within the hospital is required.

Within inpatient diabetology care for adults, the following types of hospitalisation may be distinguished:
- diabetes with inadequate metabolic control – hospitalisation in an internal medicine ward or a specialist diabetology ward,
- acute hyperglycaemic emergencies – hospitalisation in an internal medicine ward or a specialist diabetology ward,
- diabetes with diagnosis and treatment of advanced chronic complications – hospitalisation in a specialist diabetology ward,
- hypoglycaemia, hypoglycaemia unawareness, and diagnostic evaluation of the causes of hypoglycaemia, fear of hypoglycaemia – hospitalisation in a specialist diabetology ward.

Specialist diabetology outpatient clinics

1. The multidisciplinary team providing care for an individual with diabetes within specialist outpatient care (AOS) should include:
- a specialist in diabetology or a specialist in internal medicine with at least two years of

professional experience in a diabetology ward or clinic, or a physician in the second year of specialist training in diabetology or in the second year of the specialist module,
- a nurse with a specialisation in diabetology nursing or who has completed the "Diabetes Educator" course, or a nurse with a specialisation in internal medicine nursing, or who has completed a qualifying course in diabetology nursing, or who has at least two years of experience working in a diabetology ward or a specialist diabetology clinic; **diabetes education consultations should be provided by a nurse dedicated exclusively to education,**
- a dietitian employed for at least 0.5 of the physician's working time,
- access to psychological care in justified, individual clinical cases.

Educational, dietary, and psychological consultations, when ordered by a physician, should constitute separate services within the diabetology outpatient clinic. If a patient is unable to attend the clinic in person and at their request, such consultations may also be provided in the form of teleconsultations. Medical visits and educational, dietary, and psychological consultations may take place on the same day.

2. Equipment of specialist outpatient clinics:
- medical consulting rooms,
- a treatment room with a designated area for sample collection and analyses,
- a therapeutic education room,
- the ability to read and analyse data from blood glucose meters, insulin pumps, continuous glucose monitoring (CGM) devices, and automated insulin delivery (AID) systems using information technology systems,
- a set of equipment for assessment of diabetic foot syndrome (thermal tip, tuning fork – 128 Hz, 10 g monofilament, neurological hammer),
- access to imaging studies.

Optional equipment:
- fundus camera.

Teleconsultations

Teleconsultations constitute an integral element of care for individuals with diabetes. Every diabetology outpatient clinic should be equipped with the necessary hardware and software to conduct effective teleconsultations. Clinics must have a computer with appropriate software, and staff should be adequately trained. Individuals with diabetes should be encouraged to use modern technologies and applications that facilitate remote medical consultations. It should be emphasised that the effectiveness of teleconsultations increases with the availability of comprehensive source data regarding the individual's treatment (e.g. data from the memory of a blood glucose meter, CGM system, or personal insulin pump) provided to the members of the therapeutic team conducting the teleconsultation.

In individuals with diabetes, remote medical visits may form part of both routine diabetology care and care provided during situations of epidemiological threat.

Organisation of care for individuals with diabetic foot disease

I. Inpatient care – specialist diabetology wards or subunits for the treatment of diabetic foot disease

Diabetes Poland takes the position that, within the Polish healthcare system, formal and legal solutions should be established to enable the organisation of specialist diabetology wards or subunits dedicated to diabetic foot disease. These units should be able to implement a comprehensive model of care for individuals with diabetic foot disease requiring hospitalisation. In particular, this model should include simultaneous surgical intervention and broadly defined diabetological and internal medicine management. It should be based primarily on the principles set out in the Regulation of the Minister of Health of 10 July 2023 on the pilot programme of care for patients with diabetic foot syndrome, together with subsequent amendments (Regulation of the Minister of Health of 16 January 2025 amending the regulation on the pilot programme of care for patients with diabetic foot syndrome), hereinafter referred to as the Ministry of Health Regulation. In some clinical situations, the above model of care must be extended to include vascular intervention (urgent revascularisation of lower limb arteries) or orthopaedic intervention (surgical stabilisation of selected deformities in the course of Charcot neuro-osteoarthropathy).

Taking into account the complexity of clinical situations and, consequently, the complexity of hospital care needs of individuals with diabetic foot disease, the following types of hospitalisations can be distinguished. Their implementation may significantly increase treatment effectiveness and result in:

a) prevention of major amputation, and
b) complete healing of foot wounds.

1. Hospitalisation of individuals with moderate or severe infection involving bone and/or joints, i.e. hospitalisation including surgical intervention within the foot according to the model implemented under the Ministry of Health Regulation (the so-called "Szamotuły model"), lasting a minimum of 11 days and including limb-sparing bone resection, postoperative negative pressure wound therapy, targeted intravenous antibiotic therapy, and combined diabetological and internal medicine management.
2. Hospitalisation of individuals with moderate or severe infection without bone or joint involvement, i.e. hospitalisation without bone surgery, including surgical incisions with at least two negative pressure wound dressings and targeted intravenous antibiotic therapy, lasting 6–10 days.
3. Urgent short-term hospitalisation lasting 2–4 days, enabling rapid diagnostics of acute Charcot arthropathy or rapid/urgent diagnostics of ischaemia (for clinical reasons such as renal failure or rapid progression of necrosis within the foot in the course of chronic critical limb ischaemia).

As noted above, this model of care, when clinically indicated, must be extended to include vascular intervention (urgent lower limb arterial revascularisation) or orthopaedic intervention (surgical stabilisation of selected deformities in the course of Charcot neuro-osteoarthropathy).

Additional equipment required for a ward or subunit for the treatment of diabetic foot disease:

- a treatment room within the ward/subunit or with direct access, equipped with a podiatric chair,
- a set of surgical instruments (haemostats, bone rongeurs, bone curettes, surgical scissors),
- devices for local negative pressure wound therapy,
- a set for assessment of diabetic neuropathy (Semmes–Weinstein monofilament, tuning fork, thermal tip),
- a set for ankle–brachial index (ABI) assessment,
- access to Doppler ultrasound,
- a full range of wound dressings.

II. Outpatient care

Reference outpatient clinics for the treatment of diabetic foot disease

1. Staffing requirements:

- physicians: the equivalent of at least one full-time position – a specialist in diabetology with documented experience of at least one year in the treatment of individuals with diabetic foot disease,
- nurses: the equivalent of one full-time position, with documented experience of at least one year in the treatment and care of individuals with diabetic foot disease or in the treatment and care of patients with chronic wounds.

2. Access to hospitalisation in a ward (or clinic) operating under a contract with the National Health Fund (NFZ).
3. Access to multidisciplinary care, including consultations with a general surgeon, vascular surgeon or angiologist, and orthopaedic specialist.
4. Provision of the possibility of intravenous antibiotic therapy.
5. Access to basic imaging diagnostics, i.e. X-ray, ultrasound (including Doppler ultrasound), and computed tomography and/or magnetic resonance imaging.
6. Access to laboratory and microbiological tests performed in a medical diagnostic laboratory registered with the National Council of Laboratory Diagnosticians.

Basic outpatient clinics

In basic outpatient clinics, diagnostics, treatment, and prevention of ulcerations, infections, and Charcot neuro-osteoarthropathy in the course of diabetic foot disease should be provided. Basic clinics should cooperate with reference clinics, where more severe clinical cases are consulted and, if necessary, referred for further treatment.

Diabetes Poland takes the position that, within outpatient care in the Polish healthcare system, formal and legal solutions should be established to enable the implementation of the following additional procedures:

- provision of off-loading with plaster casts, including short casts (Scotch cast) and long casts (total contact cast – TCC),
- provision of local negative pressure wound therapy,
- provision of specialised dressings, including biological dressings (as part of larval therapy) and antibiotic-impregnated dressings with an appropriate carrier (e.g. calcium sulphate or calcium sulphate combined with hydroxyapatite).

Appendix 2.

Medical Certification Procedures for Drivers and Employees with Disorders of Carbohydrate Tolerance and Diabetes

I. Medical assessment of drivers

1. The medical certification procedure for drivers with diabetes is regulated by Annex No. 8 to the Regulation of the Minister of Health of 5 December 2022 on medical examinations of persons applying for driving licences and drivers (Journal of Laws 2022, item 2503), entitled "Detailed conditions for medical examination in the scope of diabetes".
2. On the basis of the medical examination performed, results of additional investigations, and conclusions from specialist consultations, the physician authorised to examine drivers assesses the risk to road traffic safety and takes this risk into account in the medical certificate.
3. In accordance with points 4.1, 6.a and 8 of the above-mentioned Annex to the Regulation, **obtaining an opinion from a specialist in diabetology or another physician managing diabetes treatment**, including confirmation of the absence of other diabetes-related health contraindications to driving, is mandatory for individuals:
   - applying for or holding driving licences of categories C1, C1+E, C, C+E, D1, D1+E, D, D+E, or a tram driving permit,
   - performing road transport within the meaning of the Road Transport Act,
   - driving emergency vehicles or vehicles transporting cash or valuables,
   - driving instructors and examiners,
   - applying for or holding driving licences of categories AM, A1, A2, A, B1, B, B+E, or T in the case of recurrent severe hypoglycaemia.
4. The physician authorised to examine drivers may also request a diabetology consultation in cases of diagnostic or certification-related doubts.
5. For a **diabetology consultation** to be taken into account by the certifying physician, it **must conclude with the issuance of an opinion in the form of a diabetology consultation form**, in accordance with the template specified in Annex No. 8 to the Regulation of the Minister of Health of 5 December 2022 referred to above.
6. When completing the consultation form, the diabetologist or other physician managing diabetes treatment should assess the individual's fitness to drive, which may consequently influence the final certification decision issued by the authorised examining physician, as follows:
   - **no health contraindications** to driving motor vehicles:
     - » **without time limitations** resulting from diagnostic evaluation of carbohydrate metabolism disorders,
     - » **with specified time limitations** resulting from identified carbohydrate metabolism disorders (corresponding to low or increased risk to road traffic safety);
   - **health contraindications** to driving motor vehicles resulting from identified disorders of carbohydrate metabolism:
     - » **relative contraindications** – with specification of a subsequent date after which the individual may reapply for medical certification (corresponding to high risk to road traffic safety with the possibility of requalification),
     - » **absolute contraindications** to driving motor vehicles (corresponding to high risk to road traffic safety without indication of a date for requalification).
7. In drivers applying for or holding driving licences of categories AM, A1, A2, A, B1, B, B+E, or T:
   - an absolute contraindication to driving is **insufficient hypoglycaemia awareness**, defined as failure to perceive pathologically low blood glucose levels and lack of appropriate response, or failure to respond to alerts provided by an external continuous glucose monitoring (CGM) device, which may result in severe hypoglycaemia and impaired consciousness,
   - a relative health contraindication is recurrent severe hypoglycaemia, defined as at least two episodes of severe hypoglycaemia within the preceding 12 months.
8. In individuals with diabetes using CGM, the physician authorised to examine drivers may certify the absence of contraindications to driving vehicles of categories AM, A1, A2, A, B1, B, B+E, or T provided that the following conditions are met:
   - continuous use of CGM for diabetes self-monitoring,
   - at least adequate patient knowledge regarding diabetes self-management, including interpretation of CGM readings,

- appropriate response to CGM alerts,
- regular diabetology care (at least two diabetology visits per year at regular 6-month intervals).

9. In the case of recurrent severe hypoglycaemia in individuals applying for or holding driving licences of categories AM, A1, A2, A, B1, B, B+E, or T, the authorised examining physician may certify the absence of health contraindications to driving provided that a diabetology opinion confirms that:
- at least 3 months have elapsed since the last episode of severe hypoglycaemia during wakefulness,
- diabetes is controlled at a level ensuring road traffic safety,
- CGM is used continuously for diabetes self-monitoring, including mandatory use while driving,
- the patient's knowledge of diabetes self-management, including interpretation of CGM readings, is at least adequate,
- appropriate responses to CGM alerts are documented,
- regular follow-up medical examinations are conducted at least twice a year at 6-month intervals, subject to point 14.

10. In drivers applying for or holding driving licences of categories C1, C1+E, C, C+E, D1, D1+E, D, D+E, or a tram driving permit, performing road transport, driving emergency vehicles or vehicles transporting valuables, as well as driving instructors and examiners, absolute contraindications to driving include:
- any episode of severe hypoglycaemia during wakefulness,
- hypoglycaemia unawareness during wakefulness, defined in Annex No. 8 to the above-mentioned Regulation of the Minister of Health of 5 December 2022 as failure to perceive pathologically low blood glucose levels, constituting a significant complication of frequent hypoglycaemic episodes,
- other diabetes-related complications precluding the ability to drive vehicles.

A prerequisite for a positive opinion regarding fitness to drive is:
  - » regular blood glucose monitoring, at least four times daily in individuals receiving insulin more than once daily; in other treatment regimens at least once daily and at times related to driving – documented in a manner allowing assessment of diabetes control,
  - » documented confirmation of adequate diabetes control by the physician managing diabetes treatment,
  - » demonstration by the examined individual of full awareness of the risk of hypoglycaemia during wakefulness.

11. The completed consultation form should be provided by the diabetologist (or other physician managing diabetes treatment) to the physician authorised to examine drivers via the patient. In the case of a negative opinion regarding fitness to drive, it is recommended that the consulting physician communicates this information directly to the certifying physician who requested the consultation.
12. During the consultation, the driver should be informed of the absolute obligation to undergo re-evaluation of medical fitness to drive in the event of any episode of severe hypoglycaemia during wakefulness, including episodes unrelated to driving.
13. The physician authorised to conduct a diabetology consultation is a physician specialised in diabetology or another specialist physician who manages diabetes treatment in the consulted individual.
14. In the following situations, consideration should be given to notifying the territorially competent department of transport or local government authority of the need for repeat medical examination to verify fitness to drive, using a form referring to Article 75(1)(5) of the Act of 5 January 2011 on Drivers (Journal of Laws 2025, item 1226, page 147):
- when there are reasonable grounds to believe that the individual is driving within less than 3 months of the last episode of severe hypoglycaemia,
- when an individual with diabetes declaring continuous CGM use fails to attend scheduled follow-up medical examinations (particularly in the presence of severe hypoglycaemia during wakefulness), and all available forms of notification have proven ineffective,
- after each episode of severe hypoglycaemia.

15. Every individual treated with insulin who has received a medical certificate indicating no diabetological contraindications to driving motor vehicles should be required to measure blood glucose (using a glucometer or CGM) immediately before starting to drive. Driving should not be commenced with blood glucose < 100 mg/dl (5.6 mmol/l), unless the diabetologist has individually determined

a different glycaemic threshold permitting driving.

16. During driving, blood glucose monitoring should be performed at least every 2 hours. If blood glucose falls below 100 mg/dl, driving should be interrupted and an appropriate amount of carbohydrates consumed. Driving may be resumed only after normalisation of blood glucose concentration and reversal of the downward trend. In cases of increased risk of hypoglycaemia (as assessed by the diabetologist or another physician managing diabetes treatment), the use of a CGM system while driving should be recommended.

II. Medical examinations of employees

1. The certification procedure for employees and individuals starting employment is regulated by the Regulation of the Minister of Health of 30 May 1996 on conducting medical examinations of employees, the scope of preventive healthcare for employees, and medical certificates issued for purposes provided for in the Labour Code (Journal of Laws 2023, item 607).
2. The physician performing a preventive medical examination of employees may extend its scope to include a diabetology consultation and additional investigations if they consider this necessary for an appropriate assessment of the health status of the individual starting work or the employee.
3. **A diabetology consultation for the purposes of preventive medical examinations**, in order to constitute a valuable opinion enabling an objective decision based on an individual assessment, **should include information key to evaluating health fitness for work under specific conditions and occupational requirements**. For this purpose, use of a diabetology consultation form according to the template is recommended.
4. On the basis of the medical examination, results of additional investigations received, and conclusions from consultations, the physician authorised to perform preventive examinations of employees issues a medical certificate stating the absence or presence of medical contraindications to performing or taking up work in a specific position.
5. **Absolute contraindications to work in positions associated with higher health requirements include:**

- recurrent severe hypoglycaemia, or even a single medically unexplained episode of severe hypoglycaemia in the past during wakefulness (a fall in blood glucose leading to impaired consciousness and requiring professional medical assistance),
- hypoglycaemia unawareness during wakefulness not expected to improve, resulting from chronic diabetes complications in the form of autonomic neuropathy, which impairs the ability to perceive progressive hypoglycaemia and consequently leads to a lack of response to falling glucose levels,
- advanced complications affecting vision, most commonly diabetic retinopathy or cataract with visual impairment,
- other advanced chronic diabetes complications,
- an opinion from a diabetologist or another specialist physician managing diabetes treatment in the consulted individual indicating a high risk of hypoglycaemia and lack of perception of prodromal symptoms of hypoglycaemia during wakefulness.

6. **Relative health contraindications to work requiring higher health requirements may be certified by the physician authorised to perform preventive examinations of employees in cases of conditions with a prospect of improvement, including:**

- inadequate metabolic control of the disease ($HbA_{1c} \geq 8\%$),
- lack of blood glucose self-monitoring or poor ability to control blood glucose,
- inadequate patient knowledge regarding diabetes, hypoglycaemia, and methods of preventing hypoglycaemia,
- non-adherence to medical recommendations.

In such cases, the next examination should take place within 1–3 months.

Prepared in collaboration with Andrzej Marcinkiewicz, MD, PhD, and Professor Jolanta Walusiak-Skorupa, MD, PhD (Nofer Institute of Occupational Medicine, Łódź).

Identification of the healthcare entity/physician's professional practice

DIABETOLOGY CONSULTATION CARD FOR DRIVER EXAMINATIONS

(Annex No. 8 to the Regulation of the Minister of Health of 5 December 2022 on medical examinations for individuals applying for driving licences and drivers, Official Gazette of 2022, item 2503)

Personal data of the patient

Full name of the patient: .....

PESEL number (National ID number): ☐☐☐☐☐☐☐☐☐☐☐

If no PESEL assigned, ID Document Name and Number: ☐

Address of residence: City: ..... Postal code: ☐☐-☐☐☐

Street: ..... House/apartment number: .....

Driver applicant ☐ Driver ☐

Diabetes Information:

Date of diagnosis: ☐ Type of diabetes: ☐ Attending physician: ☐

Healthcare Entity Conducting Medical Activities: ☐ Diabetes Clinic: ☐

Patient's knowledge of their disease, treatment, and complications: high ☐ adequate ☐ inadequate ☐

Ability to control blood glucose levels: good ☐ acceptable ☐ poor ☐

Hypoglycaemia awareness and ability to prevent and manage hypoglycaemia: adequate ☐ inadequate ☐

Presence of prodromal symptoms of hypoglycaemia: yes ☐ no ☐

Risk of hypoglycaemia: low ☐ acceptable ☐ high ☐

Presence of chronic complications of diabetes ☐ no chronic complications of diabetes ☐
If present, specify affected systems: Visual system ☐ nervous system ☐ cardiovascular system ☐

Remarks regarding chronic diabetes complications

.....

Assessment of fitness to drive:

.....

Other remarks:
For individuals using Continuous Glucose Monitoring (CGM), 3 questions must be answered:
1. Regular use of CGM: Yes ☐ No ☐
2. Adequate understanding of and response to CGM data: Yes ☐ No ☐
3. Regular clinic visits (at least every 6 months) with data readings from the pump and CGM: Yes ☐ No ☐

Additional remarks:

.....

..... (Date of the opinion)

..... (Signature, name and surname, medical license number of specialist in the field of diabetology or other physician treating diabetes)

Stamp of the health care facility or medical practice

DIABETOLOGY CONSULTATION FORM FOR PREVENTIVE EXAMINATIONS – Documentation of diabetes control by the treating physician for the purpose of assessing fitness to work

(prepared by A. Marcinkiewicz, D. Szosland)

PERSONAL DATA OF THE PATIENT

Full name of the examined person: .....

PESEL number (National ID number): ☐☐☐☐☐☐☐☐☐☐☐

If no PESEL assigned, ID Document Name and Number: ☐

Diabetes Information:

Date of diagnosis: ☐ Type of diabetes: ☐

Treating physician: ☐ Primary healthcare physician ☐ Diabetes outpatient clinic

Patient's knowledge of their disease, treatment, and complications: high ☐ adequate ☐ inadequate ☐

Ability to control blood glucose levels: good ☐ acceptable ☐ poor ☐

Hypoglycaemia awareness and ability to prevent and manage hypoglycaemia: adequate ☐ inadequate ☐

Presence of prodromal symptoms of hypoglycaemia: yes ☐ no ☐

Risk of hypoglycaemia: low ☐ acceptable ☐ high ☐

Presence of chronic complications of diabetes ☐ no chronic complications of diabetes ☐
If present, specify affected systems: Visual system ☐ nervous system ☐ cardiovascular system ☐

Comments on chronic complications of diabetes:

.....

Additional remarks:

.....

For individuals using Continuous Glucose Monitoring (CGM), 3 questions must be answered:

1. Consistent use of CGM: Yes ☐ No ☐
2. Adequate knowledge of and response to CGM alerts: Yes ☐ No ☐
3. Regular follow-up visits (at least every 6 months) with data readings from the pump and CGM: Yes ☐ No ☐

Other remarks:

.....

..... (Date of the opinion)

..... (Signature, name and surname, medical license number of specialist in the field of diabetology or other physician treating diabetes)

....., on .....
(location) (date)

Name and address of the referring entity:

.....

.....

.....

.....

Full name, address, and identification details
of the person to whom the notification relates:

.....

.....

.....

.....

Name of the territorially competent vehicle registration authority or local government body*:

.....

.....

.....

.....

NOTIFICATION

Pursuant to Article 75(1)(5) of the Act of 5 January 2011 on Driving Licences (Journal of Laws of 2025, item 1226,) we hereby notify that in respect of Mr/Ms:

.....

there are justified and serious concerns regarding his/her state of health which, in the event that he/she holds a driving licence or a permit to operate a tram, necessitate an essential and urgent assessment of medical fitness to drive the above-mentioned vehicles and verification of the existing medical certificate.

.....
(Signature of the person submitting the notification)

Notes:
Territorial jurisdiction refers to the person being reported.

Appendix 3.

Charter of Rights and Responsibilities of Employers and Employees

Diabetes is a chronic metabolic disease affecting an ever-growing number of individuals. It is estimated that approximately 3 million people in Poland live with diabetes, of whom about 60% have been diagnosed and are receiving treatment. The current scale of the disease and the increasing incidence of both type 1 and type 2 diabetes result in substantial consequences not only of a medical nature but also socio-economic. Issues related to the prevention and effective treatment of diabetes and its complications extend beyond the responsibility of the medical community and individuals with diabetes alone.

According to estimates by the World Bank, diabetes represents the second greatest economic burden on society after ischaemic heart disease. These expenditures include not only the costs of diagnosis and treatment of diabetes, including its complications, but also costs resulting from premature withdrawal from professional activity, such as incapacity for work and the associated disability benefits, as well as unemployment, which affects individuals with diabetes particularly severely.

Given that:

- the unemployment rate among individuals with diabetes is more than twice as high as among individuals without diabetes, and the resulting poorer economic situation may hinder adequate disease control,
- the workplace constitutes an important element in the prevention of non-communicable diseases,

and being convinced that:

- contemporary diabetes therapies, together with increasing patient awareness in the area of self-monitoring, allow for sustained good health and continued occupational activity,

the fact of having diabetes does not automatically make an individual a less valuable employee.

With reference to numerous initiatives undertaken at the European level, aimed at prevention, early detection, appropriate treatment, and improvement of quality of life of individuals with diabetes, including the Resolution of the European Parliament of 13 March 2012 on stepping up the fight against the diabetes epidemic in the EU and the Copenhagen Declaration adopted at the European Diabetes Forum in Copenhagen on 25–26 April 2012, on the eve of World Diabetes Day 2012, the signatories of this document, representing the medical community, the community of individuals with diabetes, and employers, call for the formulation of a charter of rights and obligations of individuals with diabetes and their potential employers. The aim is, on the one hand, to strengthen patients' sense of responsibility and their position as employees, and on the other, to prevent the exclusion of individuals with diabetes from the labour market.

Rights and obligations of employees with diabetes

1. Every individual with diabetes should be aware that effective diabetes control takes place both at home and in the workplace.
2. An employee with diabetes is obliged to follow at work the same principles used to control the disease at home, including regular blood glucose monitoring, taking medications as prescribed, adhering to meal timing and dietary principles appropriate for diabetes, and maintaining a healthy lifestyle.
3. An employee with diabetes should inform their employer about the disease and, where possible, independently adjust the mode and working hours in order to enable proper disease control.
4. An individual with diabetes should be aware that there are contraindications to performing certain occupations (including, but not limited to, pilot, public transport driver, work at heights, or work requiring exceptionally intense physical exertion) and, if employed in any such position, should notify their employer accordingly.
5. An employee with diabetes should inform their closest co-workers about the disease so that, in the event of hyperglycaemia or hypoglycaemia, colleagues are able to provide appropriate assistance and ensure continuity of work.

Rights and obligations of employers

1. Every employer should be aware that diabetes does not disqualify individuals with the condition from undertaking professional activity, and that any discrimination against an employee due to the presence or occurrence of diabetes is unacceptable. A key element in understanding the situation of an individual with diabetes is the employer's possession of basic knowledge about the disease.
2. In order to fulfil their obligations, including the duty to ensure safe and hygienic working conditions, the employer has the right and should be aware of which employees have diabetes.
3. The employer should enable an employee with diabetes to adhere to principles of disease control in the workplace and motivate them towards responsible behaviour that ensures occupational safety for the individual with diabetes as well as for co-workers.
4. Where possible, the employer should provide an employee with diabetes with a position that allows optimal disease control (including, for example, the possibility of avoiding shift work or taking short breaks for additional meals).
5. Where possible, the employer should provide an employee with newly diagnosed diabetes with an alternative or equivalent position if the current position could pose a safety risk in the workplace or make disease control more difficult.
6. Where possible, the employer should promote healthy lifestyle principles in the workplace by encouraging physical activity, a balanced diet, and participation in preventive health examinations.

On behalf of the signatories
Professor Leszek Czupryniak
President of the Polish Diabetes Association, 2011–2015

Appendix 4.

Recommendations of the Polish Endocrinological Society and Diabetes Poland on Screening for Thyroid Dysfunction in Type 1 and Type 2 Diabetes

Type 1 diabetes

1. At every diabetology visit, a clinical examination for thyroid disease should be performed. If thyroid dysfunction is suspected, serum thyroid-stimulating hormone (TSH) concentration should be measured.
2. Measurement of serum TSH and titres of thyroid peroxidase antibodies (TPOAb) and thyroglobulin antibodies (TgAb) is recommended in every patient with newly diagnosed type 1 diabetes and in patients with established disease in whom thyroid function has not previously been assessed.
3. TPOAb and TgAb titres should be measured once for the diagnosis of autoimmune thyroid disease. They are not useful for monitoring thyroid disorders.
4. In patients with TPOAb and/or TgAb titres above the reference range and TSH concentrations between 2.5 mIU/l and the upper reference limit, measurement of free thyroxine (fT4) is recommended, and TSH should be reassessed annually.
5. In patients with TPOAb titres within the reference range and TSH concentrations between 2.5 mIU/l and the upper reference limit, TSH measurement should be repeated every 2 years.
6. In patients with TPOAb titres within the reference range and TSH concentrations between the lower reference limit and 2.49 mIU/l, TSH measurement should be repeated every 5 years.
7. In patients with a positive family history of hypothyroidism due to chronic autoimmune thyroiditis, annual TSH measurement is recommended.
8. In individuals with diabetes and uncontrolled lipid metabolism, TSH measurement is recommended.
9. In every woman planning pregnancy, particularly in the case of an adverse obstetric history, measurement of TSH and TPOAb titres is recommended.
10. In every woman at 4–8 weeks of pregnancy (first obstetric visit), measurement of TSH and TPOAb titres is recommended.
11. In every pregnant woman with a history of Graves' disease, measurement of TSH and thyrotropin receptor antibodies (TRAb) is recommended at 4–8 weeks of pregnancy (first obstetric visit). Repeat measurement of TRAb is additionally recommended at the end of the second trimester (before 22 weeks of gestation).

Type 2 diabetes

1. At every diabetology visit, a clinical examination for thyroid disease should be performed, and in the presence of abnormalities on physical examination, serum TSH concentration should be measured.
2. In every patient with newly diagnosed type 2 diabetes and in patients with established disease in whom thyroid function has not previously been assessed, measurement of serum TSH is recommended.
3. In patients with TSH concentrations between 2.5 mIU/l and the upper reference limit, measurement of TPOAb titres is recommended.
4. If TPOAb titres above the reference range are detected, verification of diabetes type is recommended, primarily through measurement of glutamic acid decarboxylase antibodies (anti-GAD).
5. In patients with TPOAb titres above the reference range and TSH concentrations between 2.5 mIU/l and the upper reference limit, measurement of fT4 is recommended, and TSH should be reassessed annually.
6. In patients with TPOAb titres within the reference range and TSH concentrations between 2.5 mIU/l and the upper reference limit, TSH measurement should be repeated every 2 years.
7. In patients with TPOAb titres within the reference range and TSH concentrations between the lower reference limit and 2.49 mIU/l, TSH measurement is recommended every 5 years.
8. In individuals with diabetes and uncontrolled lipid metabolism, TSH measurement is recommended.
9. In every woman planning pregnancy, measurement of TSH is recommended.

10. In every woman at 4–8 weeks of pregnancy (first obstetric visit), measurement of TSH and TPOAb titres is recommended.
11. In every pregnant woman with a history of Graves' disease, measurement of TSH and TRAb is recommended at 4–8 weeks of pregnancy (first obstetric visit). Repeat measurement of TRAb is additionally recommended at the end of the second trimester (before 22 weeks of gestation).

In patients with diabetes and chronic kidney disease, assessment of serum TSH, free triiodothyronine (fT3), free thyroxine (fT4), and TPOAb is recommended at least once a year. In cases where TSH, fT3, and fT4 concentrations are within the reference range but TPOAb are positive, thyroid function tests should be repeated at least every 6 months.

Thyroid ultrasound examination is recommended in patients treated with GLP-1 receptor agonists who present with additional risk factors for thyroid cancer, including:

- nodular goitre or a palpable thyroid nodule,
- cervical lymphadenopathy not associated with infection,
- thyroid enlargement without a palpable nodule,
- focal thyroid lesions detected on ultrasound performed for other indications or on other imaging studies,
- a history of neck exposure to ionising radiation,
- a family history of thyroid cancer,
- carrier status of RET mutations,
- obesity.

Prepared based on the recommendations of:

Marek Ruchała, Leszek Czupryniak, Alicja Hubalewska-Dydejczyk, Andrzej Lewiński, Małgorzata Karbownik-Lewińska, Małgorzata Szelachowska, Monika Karczewska-Kupczewska, Ewa Wender-Ożegowska, Dorota Zozulińska-Ziółkiewicz, Maria Górska, Roman Junik, Katarzyna Siewko, Beata Kos-Kudła, Irina Kowalska, Nadia Sawicka-Gutaj, Paweł Gutaj, Andrzej Milewicz, Jerzy Sowiński.

Recommendations of the Polish Endocrine Society and Diabetes Poland on the diagnosis and management of thyroid dysfunction in type 1 and type 2 diabetes.

Appendix 5.

Specification of Personal Insulin Pumps – Diabetes Poland Recommendation 2026: Required Technical Standards

I. Requirements for centres initiating and/or providing diabetes treatment using a personal insulin pump (PIP) or an automated insulin delivery (AID) system

The place of service provision should be a diabetology outpatient clinic or a diabetology ward equipped with computers, appropriate software, and access to cloud-based platforms enabling the reading and analysis of data from personal insulin pumps, continuous glucose monitoring systems (CGM), and AID systems. Care provided within coordinated care models that do not have a dedicated therapeutic team is not recommended.

The centre's staff should have experience in diabetes treatment using personal insulin pumps and include:

- physicians specialised in paediatric endocrinology and diabetology,
- physicians specialised in diabetology with competence in pump therapy (possession of a certificate from the Insulin Pump Training School of Diabetes Poland is recommended),
- nurses/educators trained in pump therapy, and
- trainers responsible for technical pump support.

During follow-up visits, regular downloading and analysis of data from personal insulin pumps, CGM systems and/or glucose meters, and AID systems are required.

II. Initiation of therapy includes patient qualification for personal insulin pump therapy, training in continuous subcutaneous insulin infusion, connection of the insulin pump, a follow-up visit to verify patient skills, and achievement of adequate metabolic control of diabetes.

Patients opting for therapy using a personal insulin pump or an AID system should be aware of the functionality and technical parameters of individual pump models. This includes:

- pump type (tethered or tubeless/patch pump),
- type of bolus calculator,
- options for integration with continuous glucose monitoring systems (sensor type),
- integration with pump-dedicated applications, including automated insulin delivery functions, and
- choice of infusion set type.

III. Indications and contraindications for therapy using a personal insulin pump or AID system reimbursed by the National Health Fund (NFZ)

A. Indications for reimbursement of PIP or AID

Indications apply to individuals with diabetes who require continuous treatment using functional intensive insulin therapy with basal insulin.

Any individual with diabetes who prefers and accepts this method of treatment and has no contraindications preventing its safe use is eligible.

Currently, the preferred form of therapy using personal insulin pumps is automated insulin delivery (AID) systems.

B. Contraindications to reimbursement of personal insulin pump therapy by the NFZ

1. Mental illness or psychiatric disorders, as well as alcohol or psychoactive substance dependence, which in the treating physician's judgement prevent safe use of a personal insulin pump.
2. Intellectual disability preventing understanding of the principles of intensive insulin therapy and pump operation.

Points 1 and 2 also apply to parents or caregivers of:

- children under 16 years of age,
- individuals over 16 years of age with intellectual disability.

In children aged 13–16 years, the decision to use an AID system should always be made individually, depending on the child's ability to operate the insulin pump and manage therapy, with enhanced supervision by the diabetology therapeutic team and family assistant.

3. Certain eating disorders which, in the treating physician's judgement, prevent safe use of a personal insulin pump.
4. Unjustified absence from medical appointments (attendance at only one visit per year or no visits) in a diabetology clinic.
5. Failure to adhere to or understand the principles of intensive functional insulin therapy (lack of adequate glucose self-monitoring, omission of meal boluses, regular omission of basal insulin, lack of ketone testing during prolonged hyperglycaemia).
6. More than one episode of diabetic ketoacidosis within one year.

7. Rapidly progressive severe non-proliferative retinopathy, proliferative retinopathy before or during laser therapy.
8. Lack of acceptance of the disease despite comprehensive diabetological care and psychological support (written opinion from a psychologist experienced in diabetes care).
9. Failure to adhere to personal hygiene principles.
10. Regular exposure to strong magnetic fields.

In individuals with chronically poor metabolic control (HbA_{1c} ≥ 9.0% as an average of the last year or time in range [TIR] < 30% based on at least 90 days of data across two consecutive visits), the decision to initiate personal insulin pump therapy should be made by the therapeutic team on an individual basis.

For these patients, the preferred and often the only therapeutic option allowing rapid optimisation of glycaemic control is an automated insulin delivery (AID) system. However, in the context of limited availability of AID systems, prioritisation should depend on the overall clinical picture and an individual assessment of patient needs and capabilities.

C. Contraindications to continuation of personal insulin pump therapy and reimbursement of pump supplies

1. More than one episode of diabetic ketoacidosis within one year (including during AID therapy).
2. A higher number of episodes of severe hypoglycaemia compared with treatment using multiple daily insulin injections.
3. Failure to adhere to the principles of intensive functional insulin therapy: lack of proper glucose self-monitoring, failure to administer insulin boluses (fewer than 1–2 boluses per day).
4. Severe skin reactions at infusion set insertion sites despite use of all available preventive measures and infusion set types.
5. Irregular replacement of infusion sets (not in accordance with manufacturer recommendations), resulting in deterioration of diabetes control, particularly diabetic ketoacidosis.
6. Unjustified absence from medical appointments (attendance at only one visit in the last year or no visits).

In individuals with chronically poor metabolic control (HbA_{1c} ≥ 9.0% as an average of the last year or TIR < 30% based on at least 90 days across two consecutive visits), the decision to continue personal insulin pump therapy should be made by the therapeutic team on an individual basis.

In patients previously using conventional pumps, switching to an AID system may be indicated.

IV. Qualification for initiation or continuation of reimbursed PIP or AID therapy

A patient presenting to a centre providing reimbursed therapy should submit:

- an application with preliminary qualification from a physician working in a diabetology clinic or ward, or from a diabetologist within coordinated care,
- a glucose meter report from the last 4 weeks or a CGM report from the last 4 weeks,
- in selected patients with unsatisfactory metabolic control, additional information regarding carbohydrate intake and insulin doses may be required; sources may include a self-monitoring diary or a relevant electronic application.

In children with newly diagnosed diabetes, qualification is performed by a diabetologist or a paediatric endocrinologist–diabetologist employed in a paediatric diabetology ward.

The diabetologist or paediatric endocrinologist–diabetologist making decisions regarding reimbursement of PIP or AID therapy should take into account specific clinical and life circumstances of the patient.

V. Patient training in the principles of therapy using a personal insulin pump (PIP) or an automated insulin delivery (AID) system

Education of the patient and/or their family or caregivers should be provided at a level enabling independent use of the insulin pump and related equipment. Completion of training should be confirmed by a medical certificate or an information card.

Initial training should include technical operation of the personal insulin pump and discussion of:

- principles of programming and modifying basal insulin delivery,
- use of temporary basal rate adjustments,
- use of bolus insulin delivery,
- principles of infusion set insertion (selection and choice of insertion sites),
- interpretation of pump messages and alarms,
- preparation and interpretation of data reports generated from the personal insulin pump.

If a pump integrated with a continuous glucose monitoring (CGM) system is used, training should additionally cover the principles of system operation, interpretation of messages and alarms, and sensor insertion.

During education, particular attention should be paid to:

- management in the event of personal insulin pump failure, including principles of returning to insulin pen therapy,
- management of early symptoms of diabetic ketoacidosis,
- principles for disconnecting the insulin pump in specific situations (e.g. during physical activity or infection).

Organisational requirements when initiating personal insulin pump therapy include a minimum training duration of 9 hours, spread over at least three sessions. Additional training in automated mode operation is required when using AID systems. Training sessions should be conducted in groups of no more than 6–8 participants. In the training of children, adolescents, and individuals with intellectual disabilities, the participation of parents or legal guardians is mandatory. Patients should have the opportunity to practise using infusion sets on mannequins. It is also recommended that an infusion set be inserted into subcutaneous tissue prior to initiation of continuous subcutaneous insulin infusion therapy.

Training should continue until the patient or caregiver demonstrates proficiency in the practical aspects of using a personal insulin pump or a pump integrated with a CGM system. Responsibility for the correct delivery of training lies with the centre issuing the reimbursed personal insulin pump or AID system, while responsibility for continuation of therapy lies with the referring centre. Patient knowledge should be verified by the education team. Development of a knowledge assessment test based on the centre's own educational materials is recommended.

Patients should be trained in the use of computer-based or mobile applications for downloading and reviewing data from personal insulin pumps, CGM systems, glucose meters, and AID systems, enabling the provision of clinically effective teleconsultations.

In the case of continuation of continuous subcutaneous insulin infusion therapy with a change of personal insulin pump or AID model, re-education in functional intensive insulin therapy (FIIT) and technical training in pump operation (and CGM, if applicable) are required. Additional training in automated mode operation is required when switching to an AID system.

VI. Provision of a personal insulin pump (PIP) or an automated insulin delivery (AID) system

Provision of a personal insulin pump or an automated insulin delivery system must take into account the patient's preferences and perceptual abilities. These factors should, in turn, be considered during the education process and in the individualisation of therapy.

The table accompanying Appendix 5 presents recommendations regarding the requirements for PIP and AID systems in centres providing therapy using these technologies. It is recommended that different types of insulin pumps be available in such centres, enabling patients to choose the device best suited to their individual needs.

VII. Initiation of PIP or AID therapy in the initiating centre

Initiation of PIP or AID therapy includes:

- issuing reimbursement orders for PIP accessories and integrated CGM systems,
- establishing initial insulin pump settings,
- initiation of continuous glucose monitoring.

A trained patient, under the supervision of an educator:

- inserts the infusion set when initiating PIP therapy,
- inserts the infusion set and CGM sensor when initiating therapy with a PIP integrated with a CGM system,
- activates the automated mode when using an AID system.

For Appendix 5.

Specification of Personal Insulin Pumps – Recommendation of Diabetes Poland 2026. Mandatory minimum requirements

Parameter		Description
Bolus programming	Standard bolus	delivery accuracy not less than 0.1 IU/bolus
	Extended bolus	delivery accuracy not less than 0.1 IU/bolus; maximum bolus duration not less than 7 hours
	Dual/combination bolus	delivery accuracy not less than 0.1 IU/bolus
Temporary basal rate adjustment	Settings	Percentage or unit-based increase or decrease of the basal rate, at least every 30 minutes, with automatic return to the programmed basal rate after the set time
	Information about the active basal delivery (basal insulin)	Information on the active basal rate available from the main pump screen or pump/application on a smartphone or PDA
	Time	Duration up to 24 hours
Basal delivery programming	Entering hourly rates (number of units per hour)	Accuracy not less than 0.1, and in children < 6 years of age not less than 0.05 IU/hr.
"Pump memory"	History of boluses, alarms, basal dose, daily dose, temporary basal change, infusion set filling; the program to read data from the pump should have the ability to simultaneously read data the CGM system with which it is integrated	A minimum of 30 days through a computer program via a reader. The company provides free access to the software (available as a locally operating version or an online cloud version) and the device necessary for reading data via a computer to the diabetes centre conducting the therapy (links) and to the pump user — the requirements for the computer program are detailed in Annex 1.
Bolus calculator that is an integral part of the insulin delivery system (feature available on the insulin pump or a device that communicates wirelessly with the insulin pump or in an app on a smartphone)		**ENABLING:** to program settings in several time intervals user input of carbohydrate grams or carbohydrate exchanges active insulin calculation with user-set insulin duration, which only reduces the correction dose of the insulin bolus. manual entry of glucose levels into the bolus calculator or communication with the CGM system
Automatic infusion set filling		Yes – unlimited number of infusions set fillings per day directly using only the function in the pump
Infusion sets		**INSERTION SETS** Insertions: metal (rigid) or plastic (flexible)-all types of insertions within the reimbursement amount Length of the drain – at least 2 lengths Length of cannula – at least 2 lengths **DRAINLESS SETS FOR PATCH PUMPS.** Cannula length – at least 2 lengths
Service		Providing telephone contact 24 hours a day with an authorized hotline (knowing exactly how the pump works, all possible alarms, errors), subject to customer review. Website with the information contained in Attachment 2. Replacement of the pump within 24 hours (working days) Shipping of the pump at the expense of the company
Batteries – powering the pump		Batteries commonly available in retail outlets, petrol stations, and consumer electronics (household appliances and RTV) stores Rechargeable batteries – if the use of chargers is required, the pump user must be provided with the charger Audible signal and on-screen notification on the pump and in the smartphone/PDA (Personal Digital Assistant) application indicating impending battery depletion or battery discharge

Parameter	Description
Additional accessories needed to use a personal insulin pump	Additional accessories for the personal insulin pump, which must be systematically replaced according to the instruction manual, are provided by the manufacturer free of charge for the life of the pump (does not apply to infusion sets, insulin reservoirs, batteries, insulin pump case).
Guarantee	At least 4 years, in case of malfunction replacement with a new pump In the case of replacement of the equipment with a new one, the total warranty period is not shorter than the one proposed in the offer, the warranty calculated from the date of implementation of the NFZ (NHF) procedure, not from the date of purchase from the manufacturer
Menu	Entirely Polish language or icons or symbols
User's Manual	Entirely Polish language, all messages displayed by the pump must be described in the instructions

Insulin pump systems functionally integrated with continuous glucose monitoring (CGM) systems and automated insulin delivery (AID) systems must meet the following requirements:

- the CGM system is either an integral component of the insulin pump or is directly connected to the application operating the personal insulin pump (PIP) or AID system,
- CGM sensors and, where applicable, the required transmitter are reimbursed at the time of announcement of the tender procedure,
- the integrated system must include an automated insulin suspension function based on predicted hypoglycaemia using CGM-derived glucose values,
- the safety of the AID system must be confirmed in randomised clinical trials conducted in specific age groups,
- automated insulin delivery (AID) systems operating as hybrid closed-loop (HCL) systems must include the following functions:
  - » automatic basal insulin delivery,
  - » automatic delivery of correction doses,
  - » additional functions or settings for hypoglycaemia prevention in situations associated with increased hypoglycaemia risk,
  - » automatic switch to manual mode in the event of CGM system and/or AID application failure,
- in the case of application-based systems, the patient must receive free access to the application together with the PIP for a minimum period of 4 years and, prior to selecting an integrated or AID system, must be informed of the requirement to own a smartphone model compatible with the application,
- data from integrated systems and AID systems must be automatically transmitted to computer software with which both the patient and the treating centre must be equipped, enabling the therapeutic team to have ongoing access to the patient's data.

Specification of personal insulin pumps – Standards of care of Diabetes Poland 2026. Recommended additional requirements.

Parameter	Description
Reminder to replace the infusion set	Alarm indicating that the infusion set needs to be replaced
History of infusion set fillings	Ability to check directly in the pump's memory the history of infusion set fills
IPX 8 standard	IPX 8
Additional: a device for users to read the pump memory at home with the ability to send data to the doctor	Access to the software (either a locally running version or an online version in the cloud) and the device needed for the computer to read the data
Bolus calculator	With user-selectable mg/dl or mmol/l (glycaemic determination) settings
Continuous glucose levels monitoring system	System integrated with insulin pump or additional CGM device to support therapy with personal insulin pump
Computer software for reading pump data	Enables simultaneous reading of data from the CGM system and integration of both data sets
Basal programming	More than two additional basal profiles available for advance preparation, with the option to recall from memory and app

For selected groups of individuals, it is permissible to modify the specifications of personal insulin pumps considering the patient's educational opportunities and personalization/individualization of therapeutic.

Attachment 1.

Requirements for computer software used to read insulin pump memory:

- current basal profiles (all basal profiles available from a single pump data download, presented in charts or tables, including the exact dose and time, with precision reflecting the pump's basal delivery resolution),
- applied insulin-to-carbohydrate ratios with defined time intervals in the bolus calculator,
- bolus history (all administered boluses, differentiated by type and with time of administration specified, including extended boluses),
- history of infusion set priming/filling,
- daily graphs presenting:
  - the basal insulin delivery applied on a given day,
  - temporary basal rate changes,
  - marking of pump suspension and restart times,
  - glucose values transmitted from the integrated CGM system and/or from a blood glucose meter,
- alarm history,
- in the case of AID (HCL) systems, data regarding system settings required to activate automated mode,
- free-of-charge access to the software for patients,
- the pump data-reading software should also allow simultaneous reading of data from the integrated CGM system,
- free provision to the patient of both the software and the device required to download pump memory data in the home setting, with the possibility of transmitting these data to the therapeutic team.

Attachment 2.

Required information to be provided on the manufacturer's website:

- a helpline number through which pump users can obtain 24-hour information regarding technical problems related to the use of personal insulin pumps (PIP),
- contact details of local representatives together with their working hours,
- information on pump accessories (types of infusion sets, syringes, batteries, prices, etc.).

RECOMMENDED ADDITIONAL OPTIONS:

1. Reminder alarms for bolus administration or blood glucose measurements at user-defined times.
2. A price of infusion sets not exceeding the monthly reimbursement limit for individuals under 26 years of age and 30% of this limit for individuals over 26 years of age.

ADDITIONAL NOTE:

The contracting authority may specify additional parameters according to the needs of specific patient groups. In addition, the offer should include accessories necessary for initiating therapy and conducting patient education, including: inserters, various types of infusion sets, insulin reservoirs, pump batteries, and protective cases.

In tender evaluation, the cost of the pump should account for 60% of the total score, and additional functions for 40% of the total score.

Appendix 6.

Recommendations of Diabetes Poland and the Polish Society of Sports Medicine on Eligibility for Sports Participation in Individuals with Type 1 Diabetes

People with type 1 diabetes who have obtained a positive opinion from a diabetologist may be qualified by a sports medicine specialist to participate in any sports discipline.

One of the conditions for clearance for sports participation is treatment with functional intensive insulin therapy (FIIT) and understanding its principles. Treatment may be delivered using insulin pens or a personal insulin pump. For athletes with diabetes, the preferred method is insulin pump therapy, which allows more precise adjustment of insulin dosing to current requirements during physical exertion. Athletes with diabetes are obliged to perform systematic blood glucose monitoring using a glucometer, at least 6 times per day, with additional measurements during training sessions and competitions. The use of continuous glucose monitoring systems (CGM) is recommended, as these further support treatment and improve athlete safety.

Type 1 diabetes should not constitute a contraindication to participation in physical education classes at any educational level or in school sport (school sports associations, student sports clubs, school competitions, etc.).

Optimal glucose levels at the start of and during sport are as follows:

- aerobic exercise: 126–180 mg/dl (7–10 mmol/l),
- anaerobic exercise: 90–180 mg/dl (5–10 mmol/l).

It should be emphasised that maintaining high physical fitness and performance in people with type 1 diabetes who practise sport intensively over the long term requires > 70% time in range (TIR) 70–180 mg/dl and < 4% time below 70 mg/dl; on competition days, time below 70 mg/dl should be < 1%.

I. Contraindications to sports participation in children and adults with type 1 diabetes requiring certification by a sports medicine specialist

1. HbA_{1c}: mean value over the last 12 months > 8.5%, or a current result ≥ 9.0%.
2. More than one episode of diabetic ketoacidosis within the last 12 months.
3. More than one episode of severe hypoglycaemia within the last 12 months.
4. Blood glucose self-monitoring: < 6 measurements/day by glucometer in athletes not using CGM.
5. Visits to a diabetology clinic: < 4/year in children, < 2/year in adults.
6. Hypoglycaemia unawareness during wakefulness – a relative contraindication; it may be waived depending on the sports discipline and CGM use.
7. Chronic diabetes complications, depending on stage and sports discipline:

- proliferative retinopathy until completion of laser therapy – an absolute contraindication to all sporting activity,
- clinically overt autonomic neuropathy – a contraindication to high-intensity physical exertion,
- any microangiopathic complication or type 1 diabetes duration > 15 years in a person > 35 years of age: high and very high intensity exercise requires qualification after cardiology assessment including resting ECG, echocardiography, exercise testing, and 24-hour Holter ECG monitoring,
- macrovascular complications – qualification after cardiology assessment including resting ECG, echocardiography, exercise testing, and 24-hour Holter ECG monitoring,
- significant proteinuria 0.2–0.5 g/day (UACR 30–100 mg/g) – a relative contraindication to competitive sport; temporary exclusion; repeat testing required under rest conditions (no physical exertion) for 48 hours before and during testing, and follow-up monitoring: UACR every 3–6 months, systematic blood pressure and kidney function monitoring,
- proteinuria > 0.5 g/day (UACR > 100 mg/g) – absolute (temporary or permanent) disqualification from sport; nephrology consultation required,
- eGFR 45–60 ml/min/1.73 m² (G3a) – creatinine and eGFR monitoring at least every 3 months,
- eGFR 30–45 ml/min/1.73 m² (G3b) – a relative contraindication to competitive sport; temporary exclusion; creatinine and eGFR monitoring every 4–6 weeks,
- eGFR < 30 ml/min/1.73 m² (G4) – absolute prohibition of sports participation.

*eGFR calculation: Schwartz formula up to 15 years of age; CKD-EPI from 16 years of age.

II. Examinations required during qualification of an athlete with type 1 diabetes

Initial qualification: current investigations in accordance with the recommendations of Diabetes Poland.

HbA_{1c} values from the last 3 months, a glucometer and/or CGM report, and an insulin pump report (if used).

III. High-risk sports disciplines: motor, water, aviation, climbing

Participation in sports in which hypoglycaemia poses a particularly high risk to the safety of the individual and others is not recommended for people with type 1 diabetes.

Participation may be permitted if the following conditions are met:

- the individual is very well educated and achieves treatment targets,
- blood glucose measurement within 15 minutes before starting the activity, with a value ≥ 120 mg/dl (6.7 mmol/l**)**; glucose monitoring by glucometer every 30–60 minutes, or less frequently when CGM is used.

For high-risk sports disciplines, CGM use is recommended.

IV. Contraindications to participation in training sessions and sports competitions

1. Severe hypoglycaemia within the last 24 hours.
2. Hyperglycaemia > 250 mg/dl (13.9 mmol/l) with accompanying ketonaemia/ketonuria resulting from insulin deficiency, not carbohydrate deficiency.
3. Ketonaemia ≥ 1.5 mmol/l is an absolute contraindication to starting or continuing physical exertion.
4. Hyperglycaemia > 300 mg/dl (16.7 mmol/l) persisting for more than 2 hours.
5. Any acute event requiring medical assistance (e.g. visual disturbance, chest pain, syncope, acute infection, etc.).

Prepared by the Diabetes Poland Working Group:
Leszek Czupryniak, Andrzej Gawrecki, Przemysława Jarosz-Chobot, Tomasz Klupa, Bartłomiej Matejko, Krzysztof Pawlaczyk, Marek Postuła, Agnieszka Szadkowska, Agnieszka Szypowska, Bogumił Wolnik, Dorota Zozulińska-Ziółkiewicz
and the Polish Society of Sports Medicine Working Group:
Grzegorz Biegański, Andrzej Bugajski, Anna Jegier, Jarosław Krzywański, Marek Pietruszewski, Katarzyna Szmigielska, Wiesław Tomaszewski, Andrzej Ziemba

Appendix 7.

Recommendations on the Transition of Individuals with Type 1 Diabetes from Paediatric Diabetological Care to Adult Diabetological Care or in the Event of a Change of Diabetes Clinic

A. Transition from paediatric to adult diabetology care. The period of transition from paediatric diabetology care to adult (internal medicine-based) diabetology care is a particularly important stage in the life of a young person with type 1 diabetes. Transition is a continuous process comprising three stages: the pre-transition period (in the paediatric centre), the transfer, and the post-transition period (in the adult care centre). The fundamental principle guiding the transfer of a patient to an adult diabetology clinic should be the maintenance of continuity of medical care, without any significant gap between leaving the paediatric clinic and commencing treatment in the adult clinic.

1. To ensure a smooth transition process, the following recommendations should be observed: The timing of transfer from a paediatric diabetology clinic to an adult diabetology clinic should be determined individually, in order not to disrupt the course of therapy. Depending on the patient's emotional development, family and educational situation, and other relevant factors, the optimal age for transfer is between 18 and 21 years.
2. The patient should be prepared for transition to adult care by the paediatric diabetologist for at least one year prior to transfer. During this period, follow-up examinations of chronic diabetes complications and comorbid conditions should be performed. The assessment should also include evaluation of mental health and psychosocial issues, and appropriate interventions should be initiated if problems are identified.
3. At the final visit to the paediatric diabetology clinic, which should take place no later than 6 months before transfer, the patient should be referred to an adult diabetology clinic in a coordinated manner. This includes, in particular:

- determining the adult diabetology clinic to which the patient will be transferred,
- providing the individual with diabetes with a Diabetology Care Information Card,
- issuing a referral to the adult diabetology clinic.

4. The patient should commence care in an adult diabetology clinic no later than 6 months after completion of paediatric care.
5. During the initial visits in the adult care centre, the adaptation process of the young person to the principles of adult outpatient care should be taken into account. During the first year of adult care, clinic visits should take place at least every 3–4 months.
6. Psychological support for young people with diabetes is particularly recommended during the transition process.
7. The establishment of regional networks of cooperating paediatric and adult diabetology clinics is recommended, with agreed principles of regular communication and patient transfer.
8. In centres transferring a large number of patients, it is advisable to establish – both in paediatric and adult clinics – the role of a transition care coordinator. The coordinator's responsibilities should include managing referrals and transfers, scheduling appointments, and ensuring efficient information flow.
9. The creation of dedicated clinic days for patients transitioning to adult care is not mandatory but may be helpful, particularly for organisational reasons. Planning of adult clinic workflows should take into account the significantly greater time required for visits of patients transitioning from paediatric care, especially those treated with personal insulin pumps (PIP), including automated insulin delivery systems (AID).
10. The Diabetes Poland website provides a list of adult diabetology clinics dedicated to young adults with diabetes: https://ptdiab.pl/wiecej/lista-poradni-dla-mlodych-doroslych-z-cukrzyca-typu-1

B. Change of diabetology clinic. In the event that a change of diabetology clinic (paediatric or adult) is required, for example due to a change of place of residence, it is recommended that the patient be directed to another diabetology clinic where appropriate care can be provided. The Diabetology Care Information Card should be completed at the final visit in the current clinic.

Prepared by the Working Group: Aleksandra Araszkiewicz, Leszek Czupryniak, Andrzej Gawrecki, Przemysława Jarosz-Chobot, Tomasz Klupa, Małgorzata Myśliwiec, Agnieszka Szadkowska, Bogumił Wolnik, Dorota Zozulińska-Ziółkiewicz

DIABETES CARE INFORMATION CARD

PATIENT'S PERSONAL INFORMATION:

Nazwisko i imię:.....

PESEL (National ID number): | | | | | | | | | | | |

Diagnosis: Type 1/Type 2 diabetes..... Date of diagnosis (MM/YYYY): .....

1. Current diabetes therapy

A. Insulin therapy: YES/NO

CSII – Personal Insulin Pump (PIP): .....

(Date of last reimbursed pump issuance: DD/MM/YYYY) .....)

Prescription validity (until which month):

- Infusion sets: .....
- Reservoirs: .....

Insulin: .....

HCL: YES/NO: .....

B. Other antihyperglycemic medications (name, dosage):

.....

2. Glucose monitoring

Continuous glucose monitoring system (CGM): YES/NO (**CGM system name):**

.....

Prescription validity:

- Sensors (valid until which month): .....
- Transmitter (issue date): .....

Blood glucose meter: YES/NO

3. HbA_{1c} values

Over the last two years: ..... %

Most recent HbA_{1c} result (date: .....): .....%

4. Number of visits to the diabetology clinic in the last 12 months:.....

5. Acute diabetes complications over the last 5 Years

Number of hospitalizations due to:

- Diabetic ketoacidosis: .....
- Severe hypoglycaemia: .....

Severe hypoglycaemia in the last 12 months without hospitalisation (dates): .....

6. Chronic diabetes complications

Diagnosis		Stage/Remarks/Treatment
Diabetic eye disease (date of last examination)	YES/NO	
Diabetic kidney disease	YES/NO	
Somatic neuropathy	YES/NO	
Autonomic neuropathy	YES/NO	
Subcutaneous tissue atrophy	YES/NO	
Subcutaneous tissue hypertrophy	YES/NO	

7. Comorbid conditions

Diagnosis		Date of diagnosis	Current treatment/Comments
Autoimmune thyroiditis	YES/NO		
Coeliac disease	YES/NO		
Arterial hypertension	YES/NO		
Hyperlipidaemia	YES/NO		
Obesity	YES/NO		

8. Psychological consultation in the last 12 months: YES/NO
Outcome:

9. Education Level: Very good/Satisfactory/Requires further education

Attachments:
Hospital discharge summary: YES/NO
Results of follow-up investigations from the last 12 months: YES/NO

Patient Declaration
I hereby declare that I have received the diabetes treatment information card from the diabetology clinic.

Date of receipt .....

Patient's signature ..... Physician's signature:.....