REVIEW PAPER

Management of lipid disorders in diabetes: an overview of current recommendations

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ABSTRACT

Lipid disorders are highly prevalent among patients with diabetes. Despite their prevalence and well-established role in progression of cardiovascular (CV) diseases, dyslipidemia still remains poorly controlled. Dyslipidemia in diabetes is characterized by a distinct lipid profile: in type 2 diabetes, moderately elevated triglycerides and triglyceride-rich lipoproteins, normal or slightly elevated low-density lipoprotein cholesterol, and reduced high-density lipoprotein cholesterol (HDL-C) levels. In type 1 diabetes, hypertriglyceridemia is a common disorder, while HDL-C levels are often normal or elevated, especially in patients with poor glycemic control. Regular monitoring of the lipid profile in diabetic patients, along with effective treatment, is essential. The most important factor is determining the appropriate CV risk category and therapeutic target. Several classes of lipid-lowering agents are available, with statins forming the cornerstone of therapy. Other agents include ezetimibe, PCSK9 inhibitors or fibrates. The aim of this article is to discuss the treatment of dyslipidemia in Poland according to the current recommendations.

KEY WORDS: diabetes, dyslipidemias, cardiovascular risk.

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Epidemiology and pathogenesis

Lipid disorders are highly prevalent and constitute the most significant modifiable cardiovascular (CV) risk factor. They affect approximately 60–80% of the adult population. The WOBASZ study estimated the prevalence of hypercholesterolemia in the Polish population at 67% in men and 64% in women, while hypertriglyceridemia was diagnosed in 30% of participants. In the NATPOL study, hypercholesterolemia affected 59.5% of men and 62% of women [1, 2].

Despite the high prevalence and established role of dyslipidemia as a major modifiable risk factor for CV diseases, the control of hypercholesterolemia and attainment of target cholesterol concentrations remain poor. For instance, in the LIPIDOGRAM 2015 study, which assessed the prevalence of CV risk factors in primary care in Poland, only 11–14% of treated patients with established CV disease achieved the target low-density lipoprotein cholesterol (LDL-C) < 70 mg/dl. When applying the stricter targets introduced by the 2019 European Society of Cardiology (ESC)/European Atherosclerosis Society guidelines, fewer than 5% of patients met the treatment goals [3].

In a 2016 study conducted as part of the Cracow Secondary Prevention Program for Ischemic Heart Disease, 28.1% of post-hospitalized patients achieved the LDL-C target [4]. According to the DA VINCI study, approximately one-third of patients in Poland reach their LDL-C targets based on risk category, with only 13% achieving the target for very high-risk patients (LDL-C < 55 mg/dl or < 1.4 mmol/l) [5].

Among diabetic patients, the reported prevalence of dyslipidemia varies depending on the study from 30–60% up to 90% [6, 7].

Diabetes is a major contributor to the premature development of CV diseases. Cardiovascular complications are the leading cause of mortality in the diabetic population. In type 2 diabetes, increased CV risk may occur years before manifestation of hyperglycemia, often preceded by obesity, hypertension, dyslipidemia, and insulin resistance – components of metabolic syndrome. In type 1 diabetes, the increased CV risk is strictly associated with the development of nephropathy and hypertension.

Despite irrefutable evidence that glycemic control reduces microvascular complications, several large clinical trials have failed to demonstrate a relevant reduction in macrovascular outcomes related to atherosclerosis. Only long-term benefits have been demonstrated in type 1 diabetes and when treatment of type 2 diabetes was started early, especially in patients without diagnosed atherosclerosis. By far the most effective strategy in reducing CV risk is the treatment of dyslipidemia. Although new groups of antihyperglycemic drugs also have a beneficial effect on the CV risk, the treatment of lipid disorders remains one of the cornerstones of diabetes treatment [8, 9].

Dyslipidemia in type 2 diabetes is characterized by a distinct lipid profile: moderately elevated triglycerides (TG) and triglyceride-rich lipoproteins (TRL), normal or slightly elevated LDL-C, and reduced high-density lipoprotein cholesterol (HDL-C) levels. Triglyceride-rich lipoproteins include chylomicrons, very low-density lipoproteins (VLDL), and their remnants – metabolites of lipoproteins. In addition to quantitative abnormalities seen in lipid panels, structural and functional changes in lipoproteins are also present. These include the appearance of small dense LDL particles and dysfunctional HDL. In poorly controlled diabetes, LDL-C levels may rise and HDL-C can act as pro-inflammatory and pro-atherogenic agents [10].

In type 1 diabetes, hypertriglyceridemia may also occur, while HDL-C levels are often normal or elevated, especially in patients with poor glycemic control and diabetic nephropathy [9].

An important lipid abnormality contributing to accelerated atherosclerosis is hypertriglyceridemia. The atherogenic mechanism of TG in diabetes can be outlined as follows:

Insulin resistance and insulin deficiency → intracellular lipase activation → ↑ plasma free fatty acids → ↑ hepatic TG production → ↑ hepatic production of apoB and TG-rich VLDL → ↓ lipoprotein lipase (LPL) activity due to insulin resistance → ↓ VLDL catabolism → ↑ plasma concentrations of VLDL, VLDL remnants, and apoB.

Patients with diabetes exhibit increased activity of cholesterol ester transfer protein, which transfers TG from TG-rich lipoproteins to HDL-C and LDL-C in exchange for cholesterol esters. Subsequent hydrolysis of TGs from HDL and LDL by LPL results in the formation of small dense LDL and HDL particles with enhanced atherogenic potential.

Triglyceride-rich lipoprotein remnants, due to their small size and high apoE content, easily penetrate the endothelium and promote inflammation, contributing to atherogenesis. Moreover, nonenzymatic glycation of apoB in uncontrolled diabetes enhances the atherogenicity of LDL by making it more susceptible to oxidation.

It is noteworthy that in patients with diabetes, lipid panel parameters within or only slightly above reference ranges may mask underlying qualitative lipoprotein dysfunction. Furthermore, measuring only LDL-C does not reflect the full atherogenic particle burden or the residual risk associated with TRL. In such cases, the measurement of non-HDL-C provides valuable information about the total concentration of atherogenic lipoproteins [9, 11].

Given their elevated atherosclerotic CV risk, diabetic patients should be proactively screened for lipid abnormalities and receive prompt and effective lipid-lowering therapy.

Monitoring and risk stratification

In patients with diabetes, a lipid panel should be assessed at the time of diagnosis and then every 8–12 weeks after treatment initiation until the target levels are achieved. An exception applies to patients after an acute coronary syndrome, for whom lipid levels should be checked 4–6 weeks after therapy initiation.

Before starting treatment, glutamic pyruvic transferase (GPT), creatine phosphokinase (CPK) and thyroid-stimulating hormone levels should be measured. If lipid levels remain within target ranges, follow-up lipid profile assessments should be performed annually, and GPT or CPK should be reassessed only in the presence of symptoms – most commonly muscle pain [12].

The aim of lipid-lowering therapy, regardless of the type of diabetes, is to achieve LDL-C levels appropriate for the patient's CV risk category. After obtaining a thorough medical history and analyzing comorbidities, CV risk stratification should be carried out. A significant challenge in clinical practice is the inconsistency between national and international guidelines, notably those from the Polish Diabetes Association (PTD) and the Polish Lipid Association (PTL).

Polish Diabetes Guidelines, based on the 2023 ESC recommendations, define four categories of CV risk using the SCORE2-Diabetes calculator. This tool, an extension of the locally calibrated 10-year SCORE2 model, was developed to assess 10-year risk of fatal and non-fatal CV events in patients aged 40–69 years with type 2 diabetes mellitus (T2DM) without atherosclerotic CV disease or severe target organ damage. Cardiovascular risk categories in people with type 2 diabetes according to the PTD is shown in Table 1.

According to the 2019 guidelines of the PTL and five other societies, the CV risk categories for patients with diabetes are presented in Table 2.

It is worth noting that guidelines of the Polish Diabetes Association address only T2DM patients, whereas PTL recommendations include both T1DM and T2DM.

These discrepancies can significantly complicate treatment decisions and determination of LDL-C or non-HDL-C target values. A consensus

Table 1. Cardiovascular risk categories in people with type 2 diabetes according to the Polish Diabetes Association

Cardiovascular risk	Criteria	Target LDL-C level
Very high	Type 2 diabetes and atherosclerotic cardiovascular disease[1] or target organ damage[2], or 10-year cardiovascular risk > 20% according to the SCORE2-Diabetes calculator	LDL-C < 55 mg/dl (< 1.4 mmol/l) and reduction of LDL-C by ≥ 50%
High	Type 2 diabetes, no very high risk criteria and 10-year cardiovascular risk of 10% to < 20% according to the SCORE2-Diabetes calculator	LDL-C < 70 mg/dl (< 1.8 mmol/l) and reduction of LDL-C by ≥ 50%
Moderate	Type 2 diabetes, no very high risk criteria and 10-year cardiovascular risk of 5% to < 10% according to the SCORE2-Diabetes calculator	LDL-C < 100 mg/dl (< 2.6 mmol/l)
Low	Type 2 diabetes, no very high risk criteria and 10-year cardiovascular risk < 5% according to the SCORE2-Diabetes calculator	Target lipid levels have not been established due to lack of sufficient data

LDL-C – low-density lipoprotein cholesterol

[1] Atherosclerotic cardiovascular disease – coronary artery disease, myocardial infarction, lower extremity artery disease (LEAD), carotid artery atherosclerosis, aortic aneurysms, ischemic stroke, transient ischemic attack (TIA), and arterial revascularizations due to atherosclerosis.

[2] Severe target organ damage: eGFR < 45 ml/min/1.73 m² regardless of albuminuria, or eGFR of 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 the 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 established atherosclerotic vascular disease and/or severe organ damage.

Table 2. Cardiovascular risk categories in people with diabetes according to the Polish Lipid Association

Cardiovascular risk	Criteria	Target LDL-C level
Extreme	Post-acute coronary syndrome in a patient with diabetes and at least one additional risk factor (elevated Lp(a) > 50 mg/dl, or hsCRP > 3 mg/l, or chronic kidney disease [eGFR < 60 ml/min/1.73 m²])	< 40 mg/dl (< 1.0 mmol/l)
Very high	Type 2 diabetes with target organ damage[1] or other major risk factors[2,3], or early-onset type 1 diabetes lasting more than 20 years	LDL-C < 55 mg/dl (< 1.4 mmol/l) and reduction of LDL-C by ≥ 50%
High	Diabetes without organ damage (regardless of duration)[4]	LDL-C < 70 mg/dl (< 1.8 mmol/l) and reduction of LDL-C by ≥ 50%

eGFR – epidermal growth factor receptor, hsCRP – high-sensitivity C-reactive protein, LDL-C – low-density lipoprotein cholesterol, Lp(a) – lipoprotein (a)

[1]Organ damage is defined as the presence of microalbuminuria, retinopathy, neuropathy, and/or left ventricular myocardial damage.

[2]Other refers to the presence of at least two or more additional risk factors.

[3]Major risk factors include age ≥ 65 years, hypertension, dyslipidemia, smoking, and obesity.

[4]Does not apply to type 1 diabetes in young adults (< 35 years of age) with a diabetes duration of less than 10 years.

on CV risk calculation between societies seems essential.

Treatment

The cornerstone of dyslipidemia treatment – as with diabetes – is lifestyle modification: a proper diet and regular physical activity. Guidelines of the Polish Diabetes Association recommend reducing saturated fat intake to < 10% of total energy requirements and cholesterol intake to < 200 mg/day in patients with elevated LDL-C [12].

Glycemic control plays a crucial role in lipid management for diabetic patients, as it favorably influences lipoprotein metabolism by lowering VLDL levels, increasing LDL catabolism, reducing glycation, and enhancing LDL receptor upregulation [9].

Several drug classes are available for lipid disorder management. These agents vary significantly in their lipid-lowering potential, side-effect profiles, and indications for addition to therapy.

Statins

Statins form the basis of treatment and should be considered first-line therapy. The two most potent statins – atorvastatin and rosuvastatin – are most used. These agents have the strongest evidence supporting their role in reducing LDL-C and preventing both primary and secondary CV events. The low-density lipoprotein cholesterol lowering potency varies by drug, so the percentage reduction required to reach the therapeutic target should be calculated when selecting the appropriate agent [13]. Low-density lipoprotein cholesterol and TG-lowering potential of atorvastatin and rosuvastatin are shown in Table 3 [14].

Contraindications to statin therapy include:

- pregnancy and breastfeeding, or reproductive age without effective contraception,
- baseline GPT > 3× upper limit of the norm (ULN),
- baseline CPK > 4× ULN,
- active viral hepatitis,
- liver failure (Child-Pugh class B and C),
- choroideremia.

The most common side effects are muscle-related symptoms – myalgia, muscle weakness, and muscle cramps. More severe complications include myopathy and rhabdomyolysis [15, 16].

Table 3. Low-density lipoprotein cholesterol and triglycerides-lowering potential of atorvastatin and rosuvastatin

Parameters	Atorvastatin (%)	Rosuvastatin (%)
Decrease of LDL-C [mg]		
10	36.8	45.8
20	42	52.4
40	47.8	55
Decrease of TG [mg]		
10	20	19.8
20	22.6	23.7
40	26.8	26.1

LDL-C – low-density lipoprotein cholesterol, TG – triglycerides

Although statins have been associated with a diabetogenic effect, this should not limit their use in patients with diabetes or prediabetes, as the CV risk reduction outweighs the estimated 9–12% increased diabetes risk [12, 17].

Pitavastatin deserves mention in the context of diabetes. Among the three statins discussed, it has the weakest lipid-lowering potential, with 4 mg of pitavastatin roughly equivalent to 20–40 mg atorvastatin or 10–20 mg rosuvastatin. But what is important, pitavastatin carries a low risk of drug interactions and muscle-related side effects. Due to its high bioavailability, it is used at lower doses, potentially improving adherence. Pitavastatin does not increase diabetes risk [18].

Statin intolerance and patient adherence

A common challenge is patient reluctance to take statins or alleged intolerance. The nocebo effect contributes significantly to therapy discontinuation. According to the 3ST-POL study, 25% of patients discontinued statins within the first three months [19]. While patients often report symptoms suggestive of intolerance, a meta-analysis involving over 4 million participants found the true global prevalence of statin intolerance to be only 9.1% [20, 21]. It is worth mentioning that 75% of intolerance symptoms occur within the first 12 weeks of treatment, and 90% within the first 6 months [22].

The diversity of symptoms and patient resistance may lead to premature discontinuation by both patients and physicians. Proper identification of muscle-related statin intolerance is aided using the SAMS-CI (statin-associated muscle symptom clinical index) which is presented in Table 4 [13, 20].

The International Lipid Expert Panel definition of statin intolerance includes:

- inability to tolerate at least two different statins – one at the lowest starting dose and another at any available dose,
- intolerance related to confirmed adverse events and/or significant CPK elevation,
- symptom/biomarker improvement after dose reduction or discontinuation,
- symptoms not attributable to other conditions or drug interactions.

Ezetimibe

Ezetimibe monotherapy at 10 mg daily lowers LDL-C by approximately 15–25%, with significant inter-individual variability [23]. It is safe in patients with chronic kidney or liver disease, as it is primarily excreted *via* feces (78%) and minimally in urine (11%). However, it is contraindicated in severe hepatic impairment [24].

PCSK9 inhibitors

PCSK9 inhibitors (evolocumab, alirocumab) are monoclonal antibodies that block PCSK9 protein, preventing degradation of hepatic LDL receptors. Increased receptor expression leads to enhanced LDL-C clearance, reducing LDL-C levels by up to 60% in monotherapy and up to 85% when combined with statins and ezetimibe [15, 16, 25]. No association has been found between PCSK9 inhibitor use and new-onset diabetes [26].

Inclisiran

Inclisiran is a small interfering RNA molecule that inhibits hepatic PCSK9 synthesis, lowering LDL-C by 50–55% [27]. Currently in Poland, PCSK9 inhibitors and inclisiran are available only through the drug program B.101 or *via* emergency drug access. B.101 program covers specific familial hypercholesterolemia types and post-MI patients at very high CV risk who meet strict criteria [13].

Table 4. Modified statin-associated muscle symptom clinical index

Nature and location of muscle symptoms	
Symmetrical, hip flexors or thigh muscles	3
Symmetrical, calf muscles	2
Symmetrical, proximal upper limb muscles (coracobrachialis, biceps brachii, brachialis)	2
Asymmetrical, intermittent, nonspecific to any area	1
Time of symptom onset relative to therapy initiation or dose increase	
4–12 weeks	3
4–12 weeks	2
> 12 weeks	1
Dechallenge – time to muscle symptom improvement after statin discontinuation	
< 2 weeks	2
2–4 weeks	1
No improvement after 4 weeks	0
Rechallenge – time to recurrence of the same muscle symptoms after statin reintroduction	
< 4 weeks	3
4–12 weeks	1
> 12 weeks or no muscle symptoms	0

Probability that reported muscle pain is related to statin use:
Probable 9–11
Possible 7–8
Unlikely 2–6

Hypertriglyceridemia

Hypertriglyceridemia is defined as fasting TG level ≥ 100 mg/dl (≥ 1.1 mmol/l) and non-fasting TG ≥ 125 mg/dl (≥ 1.4 mmol/l). Extremely high TG levels (≥ 880 mg/dl or ≥ 10 mmol/l) increase the risk of acute pancreatitis.

In patients with TG < 880 mg/dl, therapy should start with statins. If LDL-C targets are not met, maximum-tolerated statin doses should be used. If TG remains > 200 mg/dl despite treatment, fenofibrate should be considered. Combination therapy is contraindicated in epidermal growth factor receptor (eGFR) < 30 ml/min/1.73 m². Creatinine and eGFR should be monitored before initiating therapy, and after 3 and 6 months. Combined statin-fibrate therapy carries an increased risk of myopathy and rhabdomyolysis [12].

Alternatively, patients may be treated with 4 g/day of eicosapentaenoic acid (EPA), which also reduces CV risk, though some data suggest a higher risk of atrial fibrillation [28].

In cases of TG > 880 mg/dl, the immediate goal is to lower TG levels and reduce pancreatitis risk. Fenofibrate should be the first drug to start therapy with, potentially combined with EPA. In urgent cases, plasmapheresis may be used for rapid TG reduction.

Very high TG levels are often seen in poorly controlled diabetes, especially in insulin-naive patients. In such cases, insulin therapy (if necessary *beta* continuous IV infusion) can substantially reduce TG levels within a few days [12].

Conclusions

If no contraindications to statins exist, they should be the first-line agents. If LDL-C goals are not achieved, ezetimibe should be added – preferably as a single-pill combination. In patients at very high CV risk who fail to meet LDL-C targets, PCSK9 inhibitors or inclisiran are recommended. If statins are contraindicated or completely intolerable, ezetimibe monotherapy should be considered.

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