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Review

Dyslipidemia in Diabetes: Understanding the Role of Insulin in Lipid Metabolism

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Abstract

Diabetes Mellitus (DM) is one of the most prevalent chronic conditions worldwide, with an estimated incidence of 592 million globally by 2035. DM can lead to various serious complications, including diabetic nephropathy, peripheral arterial disease, central and peripheral neuropathy, coronary artery disease, diabetic retinopathy, stroke, and cardiomyopathy. One of the most important macrovascular complications of DM is diabetic dyslipidemia. It is more prevalent in type 2 diabetes (T2D) than in type 1 diabetes (T1D), as T2D is associated with insulin resistance. In normal states, lipid metabolism is mainly regulated by insulin; thus, IR linked to T2D leads to multiple lipid metabolism aberrations. Under normal conditions, insulin promotes hepatic lipid storage and synthesis while suppressing fatty acid oxidation. It also induces de novo lipogenesis through stimulating the expression of lipogenic genes. In healthy conditions, insulin suppresses very-low-density lipoprotein (VLDL) production, preventing excessive lipid export into circulation. However, insulin resistance is associated with increased VLDL secretion, contributing to hypertriglyceridemia. Although a correlation between hyperinsulinemia and hypertriglyceridemia was observed, the exact mechanisms of how insulin affects lipid metabolism, especially in diabetes dyslipidemia, are still not fully understood. The aim of this review is to discuss dyslipidemia in diabetes, its pathophysiology and treatment, as well as the role of insulin in lipid metabolism.

Keywords: *Dyslipidemia, Insulin, Insulin Resistance, Lipid Metabolism, Diabetes Mellitus, Diabetic Dyslipidemia*

Introduction

The number of diabetic patients, particularly type 2 (T2D), has elevated to 350 million globally over the last few decades (1). It is estimated to reach 592 million globally by 2035 (2). In the following 20 years, it is predicted that the number of adults with diabetes will elevate by 70% in developing countries and by 20% in developed countries (3). Diabetes Mellitus (DM) is considered a major and significant risk factor for diabetic nephropathy, peripheral arterial disease, central and peripheral neuropathy coronary artery disease, diabetic retinopathy, stroke, and cardiomyopathy (4, 5). In fact, it has been established that DM is a cardiovascular disease (CVD) equivalent (5). Notably, the rising prevalence of DM and its strong association with CVD represents a long-term disease burden for populations and their health care systems. Diabetic dyslipidemia (DD) is one of the essential macrovascular complications of DM (6, 7).

If type 1 diabetes (T1D) is well controlled with insulin, no or only a few aberrations of lipid metabolism will occur (8). A cohort study on T1D patients found that only 15.8% of patients have high low-density lipoprotein cholesterol (LDL-C) and 12.9% have high triglyceride-rich lipoproteins (TRLs) (8). DD only manifests, in a way similar to that associated with T2D, in patients with poorly controlled T1D and in those with high risk to develop obesity or metabolic syndrome (8). While in T2D patients, insulin resistance (IR), β cell failure, and hyperinsulinemia are associated with DD; there are increased plasma levels of small-dense LDL-C particles and fasting TRLs (the underlying etiologies are delayed clearance of TRLs and hepatic overproduction), as well as decreased levels of high-density lipoprotein (HDL) cholesterol (1, 9).

A correlation between hypertriglyceridemia and hyperinsulinemia was observed in multiple previous studies, indicating that insulin plays a key role in lipid metabolism (10, 11). In 1974, a study demonstrated insulin participates in the regulation of triglycerides (TGs) synthesis and its secretion from the liver (12). However, the exact mechanisms of how insulin affects lipid metabolism, especially in diabetes dyslipidemia, are still not fully understood. The aim of this review is to discuss dyslipidemia in diabetes, its pathophysiology and treatment, as well as the role of insulin in lipid metabolism.

Methods

Medline (PubMed), Scopus, and Web of Science were used in systematic research till March 11, 2025. To retrieve the synonyms of the search strategy, the MeSH database was used. Boolean operators according to the Cochrane Handbook for Systematic Reviews of Interventions were used to combine search terms by “AND” and “OR” as follows: “Dyslipidemia” OR “Dyslipoproteinemia” AND “Diabetes Mellitus” OR “Type 1 Diabetes” OR “Type 2 Diabetes” AND “Insulin” AND “Lipid Metabolism” OR “Lipogenesis” OR “Lipolysis.” Summaries and duplicates of the found studies were exported and removed by EndNoteX8. Any study that discusses dyslipidemia in diabetes and the role of insulin in lipid metabolism and is published in peer-reviewed journals was included. All languages are included. Full-text articles, case series, and abstracts with the related topics are included. Case reports, comments, animal studies, and letters were excluded.

Discussion

Role of insulin in lipid metabolism

Insulin plays a central role in lipid metabolism by regulating the balance between lipid synthesis and breakdown in the liver. Under normal conditions, insulin promotes hepatic lipid storage and synthesis while suppressing fatty acid oxidation (FAO). Insulin can induce de novo lipogenesis through stimulating the expression of lipogenic genes, including sterol regulatory element-binding protein 1c, fatty acid synthase, and acetyl-CoA carboxylase. This leads to increased conversion of acetyl-CoA into fatty acids and TGs. Additionally, insulin inhibits FAO by increasing malonyl-CoA levels, which allosterically inhibits carnitine palmitoyltransferase 1 (CPT1), a key enzyme for mitochondrial fatty acid transport and oxidation. It also regulates triglyceride secretion through

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modulating the secretion of very low-density lipoprotein (VLDL) particles. In healthy conditions, insulin suppresses VLDL production, preventing excessive lipid export into circulation. However, insulin resistance is associated with increased VLDL secretion, contributing to hypertriglyceridemia. Furthermore, insulin promotes cholesterol metabolism by enhancing low-density lipoprotein receptor (LDLR) activity, thereby increasing LDL clearance. Insulin resistance disrupts this process, contributing to dyslipidemia characterized by elevated LDL and reduced HDL. Insulin resistance also has an impact on lipid metabolism. In conditions such as type 2 diabetes mellitus and nonalcoholic fatty liver disease (NAFLD), hepatic insulin signaling remains active for lipid synthesis but fails to suppress glucose production. This results in increased hepatic lipid accumulation and systemic hypertriglyceridemia. Overall, insulin plays a crucial role in coordinating lipid metabolism, and its dysregulation leads to metabolic disorders such as NAFLD, atherosclerosis, and cardiovascular disease (10).

Pathophysiology of diabetes dyslipidemia

Type 1 diabetes is marked by autoimmune impairment of pancreatic β cells that are responsible for insulin production through CD4+ and CD8+ T cells and macrophages that infiltrate the pancreatic islets (5). It occurs in about 10% of all cases of DM (13). Ten years ago, about two million people lived with T1D in Europe and North America, and the incidence is rising due to environmental and/or lifestyle changes, resulting in an autoimmune response to islet antigens (14).

Patients with T2D consistently develop DD, its prevalence is 72–85% (15, 16). DD substantially increases the risk of developing CVD in diabetic patients compared to those without DM due to its central role in genesis and the progression of atherosclerosis (15). Diabetic dyslipidemia is characterized by quantitative, qualitative, and kinetic lipid aberrations (17, 18). The main abnormalities of quantitative lipoprotein in DD are raised TGs and reduced HDL-C (19). Notably, TRLs and their remnants are atherogenic and their increase can be attributed to reduced clearance and overproduction (17). On the other hand, qualitative lipoprotein abnormalities are mainly glycation of apolipoproteins, rise in large VLDL and small-dense LDL-C particles that are susceptible to oxidation, and elevated TGs content both in HDL and LDL-C particles (5, 16, 17).

The kinetic lipoprotein abnormalities of DD include increased HDL catabolism, increased VLDL1 production, and decreased VLDL catabolism (16). All these abnormalities are linked to each other and are considered significant risk factors for the development and progression of atherosclerosis (20). Although LDL-C levels are consistently normal in DM, their particles demonstrate a decreased turnover that is potentially atherogenic (16). These lipoprotein abnormalities are usually linked to IR, which can influence the activity of lipoprotein lipase (LPL), hepatic lipase (HL), phospholipid transfer protein (PTP), cholesterol ester transfer protein (CETP), and endothelial lipase (EL) (21). Diabetic dyslipidemia is significantly associated with NAFLD, visceral obesity, and insulin resistance (1).

Furthermore, IR is linked to excessive fatty acid flux to the liver, resulting in VLDL overproduction (1). Insulin is unable to suppress lipolysis and FoxO1—a transcription factor crucial for regulating gluconeogenesis and glycogenolysis through insulin signaling while also inhibiting adipogenesis—but it remains capable of activating rapamycin complex 1 (mTORC1) (1). FoxO1 leads to elevated expression of microsomal triglyceride transfer protein and apoCIII that induce VLDL overproduction and decrease their clearance (1). In addition, insulin inhibits the secretion of chylomicrons and the production rate of apoB48 (22), whereas IR results in chronic intestinal overproduction of apoB48 that precipitates to both NAFLD and postprandial lipemia, increasing CVD risk (5, 23, 24). Thus, recent genetic studies support the hypothesis that high TRLs or their remnants are contributing factors to CVD and mortality and that low HDL-C has no significant effect (1, 25).

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Dyslipidemia and insulin resistance

As mentioned, insulin resistance in T2D is associated with dyslipidemia and this dyslipidemia, is usually an early manifestation that may occur even before overt diabetes appears. The first atherogenic dyslipidemia caused by IR is usually hypertriglyceridemia (12), and the predominant lipoprotein triglyceride is VLDL. VLDL is synthesized by the liver and its formation is stimulated by raised delivery of free fatty acids (FFA), also called non-esterified fatty acids, to the liver (26). When insulin sensitivity is patent, insulin is able to inhibit VLDL liver secretion. While in IR, insulin levels are chronically elevated, making the liver resistant to insulin inhibitory effects on VLDL secretion; therefore VLDL secretion remains elevated even with high insulin levels (27). Furthermore, in insulin-resistant states, clearance of VLDL is insufficient, mainly due to a reduction in activity of tissue lipases, which most of them are regulated by insulin (26). One of the most crucial tissue lipases that regulate lipoprotein levels is LPL. A reduction in the activity of LPL leads to reduced clearance of VLDL (28). Additionally, a decrease in FFA absorption and an increase in lipolysis by adipocytes occur due to IR, both elevating circulating FFA levels. The increased delivery of FFA to peripheral tissues (such as the intestine and liver) and insulin resistance results in TRLs overproduction by the liver and the intestine. Moreover, elevated levels of free fatty acids themselves can induce insulin resistance (29). This results in a vicious cycle of FFAs and insulin resistance triggering each other.

When VLDL plasma levels are elevated, CETP, a plasma protein, induces the exchange of triglycerides in VLDL for cholesterol in HDL. Namely, a VLDL particle releases a molecule of triglyceride, giving it to HDL as a replacement for one of the cholesteryl ester molecules from HDL (30). Thus, two lipid changes occur: a triglyceride-rich cholesterol-depleted HDL particle, which is considered a less protective particle, and a cholesterol-enriched VLDL particle, which is significantly an atherogenic particle. A hydrolysis of the triglyceride portion of the triglyceride-rich cholesterol-depleted HDL particle and a dissociation of its protein component, apo A-I, may occur. Typically, the free apo A-I undergoes a rapid clearance. Therefore, the number of HDL particles decreases as a result of a reduction in HDL cholesterol as well as a reduction in the amount of circulating apo A-I (26). In insulin resistance states, the LDL level is typically in the normal range or only mildly elevated, whereas the composition of the LDL particle is often abnormal (31). In addition, IR results in the formation of smaller and denser, cholesterol-depleted, LDL particles. Dense, small LDL particles have been observed to be linked to elevated risk of coronary heart disease (32).

These particles exert their atherogenicity effect, at least in part, through their elevated uptake by macrophages, their considerable susceptibility to oxidation, and their ability to enter vessel walls more rapidly than larger LDL particles (33). As mentioned, hypertriglyceridemia is the cause of small, dense LDL particles in insulin resistance and the mechanism is similar to that which causes low HDL levels.

Increased levels of VLDL triglyceride in the presence of CETP can promote the transfer of triglyceride into LDL in exchange for LDL cholesteryl ester. This triglyceride-rich LDL particle undergoes hydrolysis to remove triglyceride, which leads to a smaller, denser, lipid-depleted LDL particle. The dyslipidemia associated with type 2 diabetes mellitus is shown in. The diabetic dyslipidemia composed of increased VLDL, triglycerides, and small dense LDL paired with low HDL is common in patients with type 2 diabetes and almost certainly increases the risk of atherosclerosis in this population (26).

Treatment of diabetes dyslipidemia

The treatment of DD primarily focuses on achieving optimal LDL-C levels based on cardiovascular risk. Various guidelines have evolved to define LDL-C targets and therapeutic strategies (5). The 2004 updated National Cholesterol Education Program Adult Treatment Panel III guidelines classified diabetes mellitus as a CVD equivalent, recommending an LDL-C goal of <70 mg/dl,

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guidelines aligned with these recommendations, also considering non-HDL-C and apolipoprotein B as alternative targets (34). The 2013 American College of Cardiology (ACC)/American Heart Association (AHA) guidelines from LDL-C targets to statin intensity, leading to their limited adoption outside the U.S. (35). The 2015 ACC/AHA report recommended moder diabetic adults aged 40–75 and high-intensity statins for those with ≥7.5% 10-year CV risk (36). The 2017 American Diabetes Association guidelines emphasized statin therapy for all patients and ezetimibe in certain high-risk cases (37). The 2017 American Association of Clinical Endocrinologists and the American College of Endocrinology guidelines classified T2D patients into high, very high, or extreme LDL-C targets at <100, <70, and <55 mg/dl, respectively (38). Statin-Fibrate combination is an effective strategy for atherogenic dyslipidemia (AD), as seen in the ACCORD and FIELD trials, which demonstrated CVD event reduction in select patient subgroups (39, 40). Furthermore, Statin/Omega-3 combination is recommended for AD in patients with CKD, though evidence on CVD risk reduction remains limited (41). Niacin-laropiprant was used in this setting; however, the HPS2-THRIVE study led to the withdrawal of niacin-laropiprant due to lack of efficacy and increased adverse events (42). Notably, ezetimibe moderately reduces small-dense LDL-C, while fibrates and niacin are more effective in this regard (5). Additionally, fibrates and ezetimibe improve insulin sensitivity and omega-3 fatty acids show potential benefits in insulin resistance (5). Statins, particularly pravastatin, have mixed effects on glucose metabolism, with pravastatin showing protective properties (5).

Conclusion

Diabetic dyslipidemia, driven by insulin resistance and altered lipid metabolism, significantly increases cardiovascular risk. It is characterized by elevated triglycerides, reduced HDL, and small, dense LDL particles. Effective management requires lifestyle changes, glycemic control, and lipid-lowering therapies, with statins as the primary treatment. Future research should focus on optimizing strategies to improve lipid regulation and reduce complications in diabetes.

Disclosures

Author contributions

The authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Ethics statement

Not applicable.

Conflict of interest

The authors declare no competing interest.

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