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Pathophysiology and Significant Relation between Diabetic Dyslipidemia Cardiovascular Diseases

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Introduction

Diabetes can induce both microvascular (retinopathy, neuropathy, and nephropathy) and macrovascular (ASCVD, which manifests as coronary artery disease, stroke, and peripheral arterial disease) complications. Hypertriglyceridemia (HTG) with reduced levels of high-density lipoprotein (HDL)-cholesterol is a frequent complication of diabetes. While low-density lipoprotein (LDL) cholesterol levels are typically not increased, tiny dense LDL particles appear to be more atherogenic. Furthermore, increasing apolipoprotein B levels and non-HDL-cholesterol levels indicate a rise in particle number. Premature ASCVD from increased apolipoprotein B carrying particles and pancreatitis with severe HTG > 1000 mg/dL are the two primary complications of diabetic dyslipidemia [1].

Pathophysiology

The pathogenesis of atherogenic dyslipidemia in diabetes is complicated by changes in the metabolism of triglyceride-rich lipoproteins. Increased hepatic VLDL production and poor clearance of VLDL and intestinally generated chylomicrons are among the changes. Prolonged plasma retention of both VLDL and postprandial chylomicrons as partly lipolyzed residual particles is a significant consequence of delayed clearance [2]. Increased hepatic synthesis of big VLDL and/or delayed clearance of large VLDL from plasma leads to an increase in precursors of tiny dense LDL particles. There are at least seven different LDL subspecies, each with its own metabolic activity and pathogenic functions. VLDL levels in the blood are linked to LDL density and size reduction. Furthermore, plasma HDL levels, particularly the HDL2 subtype, are negatively linked to LDL size and density. Small dense LDL particles appear to result from a sequence of intravascular processing stages, including lipolysis, of particular bigger VLDL precursors [3]. Additional triglyceride enrichment of lipolytic products via cholesteryl ester transfer protein, along with hepatic lipase hydrolysis of triglyceride and phospholipids, results in enhanced formation of tiny dense LDL particles. Because of their lower affinity for LDL receptors, the plasma residence period of these LDL particles may be extended. Multiple subclasses of HDL particles have been discovered, ranging in diameter and density from the small dense HDL3c, HDL3b, and HDL3a to the larger HDL2a and HDL2b. Increased cholesterol transfer from HDL to triglyceride-rich lipoproteins, with reciprocal triglyceride transfer to HDL, appears to be a significant role in the decreases in HDL associated with type 2 diabetes and insulin resistance. Hepatic lipase hydrolyzes triglyceride-rich HDL particles, which are then quickly catabolized and removed from plasma. Reduced HDL levels in the plasma of type 2 diabetes patients are often seen as reductions in the HDL2b subspecies and relative or absolute increases in the smaller denser HDL3b and HDL3c subspecies. Insulin resistance may influence numerous variables that contribute to the development of diabetic dyslipidemia. Increased efflux of free fatty acids from adipose tissue and decreased insulin-mediated skeletal muscle absorption of free fatty acids enhance fatty acid flow to the liver in insulin resistance and type 2 diabetes. The presence of increased free fatty acid levels in those with impaired glucose tolerance implies that insulin resistance associated with elevated free fatty acid levels begins before hyperglycemia develops. Reduced glucose consumption in muscle was linked to an immediate increase of free fatty acids in one investigation of people without diabetes [4]. There is apparently a link between plasma free fatty acid levels and insulin resistance, according to epidemiological research. Free fatty acids in the form of triglycerides are accumulated in muscle, liver, heart, and pancreas in the presence of insulin resistance. Agents that reduce high levels of free fatty acids, such as thiazolidinediones (TZDs), have been found to enhance insulin sensitivity in muscle, liver, and adipose tissues. Insulin resistance enhances hepatic lipase activity, which, as previously mentioned, is responsible for the hydrolysis of phospholipids in LDL and HDL particles, resulting in smaller and denser LDL particles and a reduction in HDL2.

Diabetic dyslipidemia and cardiovascular disease

Studies have established a cause and effect relationship between TG rich lipoproteins and CVD via mutations in apolipoprotein C3. Epidemiological studies have shown a link between increased TG levels and cardiovascular disease (CVD), and previous findings have established a cause and effect relationship between TG rich lipoproteins and CVD via mutations in apolipoprotein C3. The major predictor of CVD has been LDL cholesterol. Several investigations have discovered a significant link between LDL and CVD. LDL levels may or may not rise as a result of diabetes, however there is a rise in the concentration of tiny dense LDL particles, which are more atherogenic than big LDL particles. High levels of HDL cholesterol are linked to an increased risk of coronary heart disease (CHD). A multitude of activities of HDL particles, including stimulation of cellular cholesterol efflux and direct antioxidative and anti-inflammatory characteristics, may contribute to direct cardioprotective benefits. Furthermore, low HDL cholesterol levels are frequently accompanied with high triglyceride levels, and the two have been linked to an increased risk of CHD. People with type 2 diabetes and coronary artery disease have little HDL particles. Furthermore, both hyperinsulinemia and hypertriglyceridemia are linked to low HDL2 levels and small HDL particle size. Reduced LDL receptor affinity, greater tendency for transport into the subendothelial region, enhanced binding to artery wall proteoglycans, and sensitivity to oxidative alterations appear to be connected to small dense LDL's increased atherogenic potential. Despite the fact that these are in vitro

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data, they support the idea that tiny dense LDL leads to arterial damage in individuals with diabetes-related dyslipidemia. According to the findings, the dyslipidemia associated with insulin resistance and type 2 diabetes is strongly linked to an elevated risk of cardiovascular disease [5].

References

1. Heron M (2018) Deaths: Leading Causes for 2016. Natl Vital Stat Rep 67:1-77.
2. Benjamin EJ, Virani SS, Callaway CW, Chamberlain AM, Chang AR, et al. (2018) American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2018 Update: A Report from the Am Heart Asso Cir 137:e67-e492.
3. Jialal I, Bajaj M (2009) Therapy and clinical trials: management of diabetic dyslipidemia. Curr Opin Lipidol 20:85-86.
4. Berneis KK, Krauss RM (2002) Metabolic origins and clinical significance of LDL heterogeneity. J Lipid Res 43:1363-137.
5. McNamara JR, Campos H, Ordovas JM, Peterson J, Wilson PW, et al. (1987) Effect of gender, age, and lipid status on low density lipoprotein sub fraction distribution: results from the Framingham Offspring Study. Arteriosclerosis 7:483-490.