Cardiovascular diseases and role of diabetic dyslipidemia and its pathophysiology.

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Introduction

Diabetes can cause both microvascular (retinopathy, neuropathy, and nephropathy) and macrovascular complications (ASCVD, which manifests as coronary artery disease, stroke, and peripheral arterial disease). Hypertriglyceridemia (HTG) with low levels of High-Density Lipoprotein (HDL) cholesterol is a common diabetic complication. While Low-Density Lipoprotein (LDL) cholesterol levels are rarely elevated, tiny dense LDL particles appear to be more atherogenic [1]. Furthermore, rising levels of apolipoprotein B and non-HDL cholesterol indicate an increase in particle number.

The two primary complications of diabetic dyslipidemia are premature ASCVD caused by increased apolipoprotein B carrying particles and pancreatitis caused by severe HTG > 1000 mg/dL.

Pathophysiology

Changes in the metabolism of triglyceride-rich lipoproteins complicate the pathogenesis of atherogenic dyslipidemia in diabetes. Among the changes are increased hepatic VLDL production and poor clearance of VLDL and intestinally generated chylomicrons. Delay in clearance results in prolonged plasma retention of VLDL and postprandial chylomicrons as partially lipolyzed residual particles [2].

Increased hepatic synthesis of big VLDL and/or delayed clearance of big VLDL from plasma results in an increase in tiny dense LDL particle precursors. There are at least seven distinct subspecies of LDL, each with its own metabolic activity and pathogenic functions. VLDL levels in the blood have been linked to a reduction in LDL density and size. Furthermore, plasma HDL levels, particularly the HDL2 subtype, are inversely related to LDL size and composition.

There are several subclasses of HDL particles, 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 play an important role in the HDL decreases associated with type 2 diabetes and insulin resistance. Hepatic lipase hydrolyzes triglyceride-rich HDL particles, which are then rapidly catabolized and removed from the bloodstream. Reduced HDL levels in type 2 diabetes patients' plasma are frequently observed as decreases in the HDL2b subspecies and relative or absolute increases in the smaller denser HDL3b and HDL3c subspecies [3].

Insulin resistance can affect a variety of factors that contribute to the development of diabetic dyslipidemia. a greater efflux of In insulin resistance and type 2 diabetes, free fatty acids from adipose tissue and decreased insulin-mediated skeletal muscle absorption of free fatty acids increase fatty acid flow to the liver [4]. The presence of elevated free fatty acid levels in those with impaired glucose tolerance suggests that insulin resistance precedes hyperglycemia. In one study of people without diabetes, decreased glucose consumption in muscle was linked to an immediate increase in free fatty acids. According to epidemiological research, there appears to be a link between plasma free fatty acid levels and insulin resistance. In the presence of insulin resistance, free fatty acids in the form of triglycerides accumulate in the muscle, liver, heart, and pancreas. Agents that lower high Free fatty acid levels, such as thiazolidinediones (TZDs), have been shown to improve insulin sensitivity in muscle, liver, and adipose tissues.

Insulin resistance increases the activity of hepatic lipase, which, as previously stated, is responsible for the hydrolysis of phospholipids in LDL and HDL particles, resulting in smaller, denser LDL particles and a decrease in HDL2.

Cardiovascular disease and diabetic dyslipidemia

Through mutations in apolipoprotein C3, studies have proven a cause and effect association between TG rich lipoproteins and CVD. Increased TG levels have been linked to cardiovascular disease (CVD) in epidemiological research, and prior discoveries have demonstrated a cause and effect relationship between TG rich lipoproteins and CVD via mutations in apolipoprotein C3. LDL cholesterol has long been regarded as the most important predictor of CVD. A substantial relationship between LDL and CVD has been established in several studies. Diabetes may or may not cause an increase in LDL values.

High HDL cholesterol levels have been related to a higher risk of coronary heart disease (CHD). HDL particles may provide direct cardioprotective advantages due to a variety of actions, including encouragement of cellular cholesterol efflux and

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direct antioxidative and anti-inflammatory properties. Low HDL cholesterol levels are usually associated with high triglyceride levels, and the two have been related to an increased risk of coronary heart disease [5].

HDL particles are low in people with type 2 diabetes and coronary artery disease. Furthermore, low HDL2 levels and small HDL particle size are associated to both hyperinsulinemia and hypertriglyceridemia. Small dense LDLs appear to be linked to lower LDL receptor affinity, a stronger inclination for transport into the subendothelial zone, increased binding to artery wall proteoglycans, and oxidative sensitivity.

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