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Dyslipidemia is a Primary Cause of Cardiovascular Disease

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Introduction

Dyslipidemia is a condition in which the amount of lipids in the blood is abnormally high (such as triglycerides, cholesterol, and fat phospholipids [1]. Dyslipidemia is a risk factor for the development of atherosclerotic cardiovascular disease (ASCVD). ASCVD includes coronary artery disease, cerebrovascular disease, and peripheral artery disease. Despite the fact that dyslipidemia is a risk factor for ASCVD, high levels do not necessitate the use of lipid-lowering drugs. In a cardiovascular risk assessment, other factors such as concurrent conditions and lifestyle are considered in addition to dyslipidemia. Hyperlipidemias, or blood lipid increases, account for the bulk of dyslipidemias in affluent countries. Dietary and lifestyle variables are commonly blamed for this. Insulin resistance can lead to dyslipidemia if it lasts for a long time. O-GlcNAc transferase (OGT) levels can also be elevated.

Description

Classification

Doctors and fundamental scientists classify dyslipidemias in two ways. The look of the body is one method (including the specific type of lipid that is increased). The alternative is due to the underlying cause of the ailment (genetic, or secondary to another condition) [2]. This classification can be difficult because most illnesses are caused by a combination of genetics and lifestyle factors. However, there are a few well-defined genetic disorders that are typically easy to identify.

The three major blood values used to measure dyslipidemia are triglycerides (TG), high density lipoprotein cholesterol (HDL-C), and low density lipoprotein cholesterol (LDL-C) (LDL-C). Dyslipidemia is defined by high triglyceride levels (fasting >1.7 mmol/L). VLDLs (very low density lipoproteins) are triglyceride transporters in the bloodstream. It's vital to remember that when measuring triglyceride levels, you'll need to fast for 8–12 hours to get an accurate result, as non-fasting TG results can be misleading. Because severe hypertriglyceridemia increases the risk of acute pancreatitis, TG levels greater than 10 mmol/L should be treated. Another blood test used to identify dyslipidemia is HDL-C. HDL cholesterol is mostly protein with a small amount of lipids. Because it works by getting to the tissues and removing excess cholesterol and fat, it has a positive effect on the body [3,4]. Because of its favourable effects in preventing plaque formation, HDL-C is characterised as "good cholesterol." Antioxidation, thrombosis protection, endothelial function maintenance, and maintaining low blood viscosity are some of HDL-other C's functions. Due to its beneficial actions, a low level of HDL cholesterol suggests dyslipidemia and is a risk factor for issues. Another commonly investigated diagnostic test is LDL cholesterol. Low density lipoproteins are made up of cholesterol, TG, phospholipids, and apolipoproteins. When LDL-C molecules bind to the endothelium of blood arteries, plaque forms. LDL-C in the bloodstream might cling to plaques once they've formed, causing further accumulation. In addition to plaque development, LDL-C molecules can be oxidised. Increased cholesterol levels and the production of inflammatory cytokines, both of which damage blood arteries, can be caused by oxidation [5]. Because of its negative implications, high levels of LDL-C increase the risk of cardiovascular disease and indicate dyslipidemia.

Dyslipidemias can also be classified based on whether the underlying cause is primary, secondary, or a mix of both. Primary dyslipidemias are genetic defects that cause increased lipid levels without any other obvious risk factors. Dyslipidemia-related issues, such as atherosclerotic cardiovascular disease, are more likely to occur at a younger age in people with primary dyslipidemias. Primary dyslipidemias are genetic defects that cause increased lipid levels without any other obvious risk factors. Dyslipidemia-related issues, such as atherosclerotic cardiovascular disease, are more likely to occur at a younger age in people with primary dyslipidemias. Some of the most common genetic disorders connected to primary dyslipidemias are homozygous or heterozygous hypercholesterolemia, familial hypertriglyceridemia, mixed hyperlipidemia, and HDL-C metabolism anomalies. Familial hypercholesterolemia is typically caused by mutations in the LDLR, PCSK9, or APOB genes, which result in high LDL cholesterol. The liver produces too much apoB-100 in patients with concurrent hyperlipidemia. This leads to the formation of a large number of LDL and VLDL molecules. This leads to the formation of a large number of LDL and VLDL molecules. Acute pancreatitis or xanthomas on the skin, eyelids, or around the cornea are common symptoms in patients with primary dyslipidemias [1]. Secondary dyslipidemias are caused by modifiable environmental or lifestyle variables, as opposed to primary dyslipidemias. Diabetes mellitus that is uncontrolled, cholestatic liver disease, chronic renal illness, hypothyroidism, and polycystic ovarian syndrome are all connected to a higher risk of dyslipidemia.

Diagnosis

When it comes to dyslipidemia screening, there is no universal agreement. Males between the ages of 25 and 30 and females between the ages of 30 and 35 should be screened at a younger age if they have a high risk of cardiovascular disease. It is unknown whether screening people under the age of 40 who have no symptoms are beneficial. Males and females who are not at risk of cardiovascular disease should be screened at the ages of 35 and 45, respectively, according to Up to Date. You should get screened regardless of your age if you have any of the risk factors listed below. The Framingham Risk Score (FRS) can be used to assess cardiovascular risk and should be updated on a regular basis.

References

1. Goulooze, Sebastiaan C., Adam F. Cohen, and Robert Rissmann. "Lomitapide." *Br J Clin Pharmacol* 80 (2015): 179.
2. Bhatt, Deepak L., P. Gabriel Steg, Michael Miller, and Eliot A. Brinton, et al. "Cardiovascular risk reduction with icosapent ethyl for hypertriglyceridemia." *NEJM* 380 (2019): 11-22.
3. Sabatine, Marc S. "PCSK9 inhibitors: Clinical evidence and implementation." *Nat*

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Rev Cardiol 16 (2019): 155-165.

4. Staels, Bart, Jean Dallongeville, Johan Auwerx, Kristina Schoonjans, Eran Leitersdorf, and Jean-Charles Fruchart. "Mechanism of action of fibrates on lipid and lipoprotein metabolism." *Circulation* 98 (1998): 2088-2093.
5. Istvan, Eva S., and Johann Deisenhofer. "*Structural mechanism for statin inhibition of HMG-CoA reductase*." *Sci* 292 (2001): 1160-1164.

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