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Opinion Article

Is it Possible to Determine Aortic Stenosis Using a Patient's Lipid Profile?

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DESCRIPTION

In developed countries, aortic stenosis is one of the most prevalent valvular diseases requiring surgery. The risk factors for coronary artery disease and the development of aortic stenosis are identical. Diabetes Mellitus (DM), smoking, a high Body Mass Index (BMI), high blood pressure, and atherosclerosis are only a few of the many reasons for Cardiovascular Risk Factors (CVRF). Even though their cardiovascular risk is low, the number of people with atherosclerosis and cardiovascular illness is relatively large. In middle-aged asymptomatic patient groups with no other risk factors, atherosclerosis remains a major risk factor for coronary artery disease and peripheral vascular disease. Patients in the middle age group with low CVRF are identified using coronary artery classification or carotid ultrasonography; roughly 60% of them have subclinical atherosclerosis.

Atherosclerosis is a chronic inflammatory condition that causes the arterial walls to harden and thicken. Its pathogenesis is based on local vascular injury, inflammation, and oxidative stress. The initial phase in this process is endothelial damage in the blood vessels. In this injured area, platelet and leukocyte adhesion as well as lipid accumulation occur. Smooth muscle cell proliferation is triggered by the release of endothelium induced growth factor by these sticky cells. The cholesterol and fatty acid concentration of atherosclerotic plaque is high. Furthermore, lipoproteins are responsible for transporting cholesterol and fatty acids, rendering them vital for plaque formation. The inverse proportional association between triglyceride and High Density Lipoprotein (HDL) levels is well recognized. HDL is known to function by transporting cholesterol in the opposite direction. Indeed, HDL protects LDL from oxidation, which helps to prevent atherosclerotic plaque formation. In addition, Triglycerides (TG) have an effect on atherosclerosis that is not mediated by HDL or LDL. A rise in TG levels results in a drop in serum HDL and an increase in LDL. TG does not normally deposit in the vascular wall, but as TG rich lipoprotein levels in the blood elevate, it tends to flow through the damaged endothelium and into the sub-endothelial cavity. This pathologic accumulation contributes to the formation of atheroma plaques by enhancing oxidation and smooth muscle proliferation.

There is a close association between LDL-C and atherosclerotic cardiovascular disease, according to numerous researches. To control LDL levels, many clinical trials support statin medication. The rosuvastatin and simvastatin are considered to raise HDL-C levels. As a result, it's critical to find out which of these laboratory variables is involved in the development of arterial atherosclerosis. The goal of this study was to learn more about the relationship between HDL, LDL, and cholesterol levels and atherosclerosis in large arterial tissues like the ascending aorta. Aortic Stenosis (AS) is a hemodynamically significant constriction of the left ventricle outlet with multiple different etiologies, whereas aortic sclerosis is a thickening or calcification of the aortic valve that does not restrict left ventricular outflow. AS is characterized as valvular, sub-valvular, or supra-valvular depending on the level of obstruction.

At the junction of the left ventricular outflow tract and the aortic root, the normal aortic valve is a specialized structure. The leaflets are made up of three layers: fibrosa, spongiosa, and ventricularis, which stretch from the aorta to the ventricular surface. Endothelium surrounds both the ventricular and aortic sides of this leaflet structure, which is in continuity with both the ventricular endocardium and the aortic endothelium. The aortic valve is divided into layers, each with its own structure and function: The fibrosa contains circumferentially oriented collagen fibres, which provide most of the leaflet's strength; the spongiosa, which contains mucopolysaccharides and functions to resist compressive forces and facilitate movements between the fibrosa and ventricularis during leaflet motion, is found at the bases of the leaflets and contains mucopolysaccharides. As a result, in this study there is no correlation between HDL-C, LDL-C, triglyceride, or total cholesterol levels with the pathological development of aortic atherosclerosis.

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