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EDITORIAL

Serum lipids .. again, and always!

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Reports on the clinical importance of serum lipids appeared early in the 1970s. Both hypercholesterolemia and hypertriglyceridemia (the main two components of serum lipids) were implicated in the risk of coronary artery disease.[1] Blood cholesterol fractions carry different functions in different body tissues. However, the low and very-low-density lipoprotein cholesterols (LDL-C & VLDL-C) enhance the process of atherosclerosis. In contrast, high-density lipoprotein (HDL-C) is beneficial to stand against atherosclerosis of the coronary arteries.[2,3]

Cholesterol phenotyping is of importance in the evaluation of the risk of atherosclerosis, especially when the value of cholesterol is on borderline.[4] The use of the atherogenic index LDL-C/HDL-C ratio was found to be more informative than absolute cholesterol values.[5]

Causes of the familial type of hypercholesterolemia have been referred to as a mutation in the LDL receptor gene, apoprotein B, and the proprotein convertase subtilisin/Kexin type 9 gene (PCSK9). Such patients are at double risk for the development of cardiovascular disease and need early identification and treatment with the screening of all first-degree relatives. The use of inhibitors to the synthesis of apolipoprotein-100 has been recommended in the treatment of familial hypercholesterolemia along with other lipid-lowering drugs.[2,4]

In the other atherogenic familial type mixed (combined) hyperlipidemia, which occurs at a frequency of 1-5/5000, there is a decrease in high-density lipoprotein (HDL) cholesterol and an increase in non-HDL cholesterols (LDL and VLDL) showing hypercholesterolemia and hypertriglyceridemia. It is also termed Type III- hyperlipidemia, dysbetalipoproteinemia, or broad beta disease. It is a genetic disorder characterized by accumulation, in the plasma, of remnant chylomicrons from intestinal lipoproteins and VLDL remnants derived from hepatic lipoproteins resulting in the development of premature atherosclerosis.[5,6] The cause could be referred to as the expression of different isoforms of Apoprotein E (ApoE) that do not bind to the receptor or ApoE deficiency.[6,7] The molecular cause of this type is associated with the presence of ApoE2, which increases triglyceride and cholesterol levels by delaying clearance of hepatic and intestinal remnant lipoproteins.[7]

A recent review by Yao et al. (2020)[8] mentioned the mechanisms of the direct action of hypercholesterolemia on the myocardium. One of those mechanisms was attributed to the change of membrane lipid bilayer, another to the regulation of intracellular calcium ions, or the isoform expression patterns of myosin heavy chain. Any or all of these will sensitize the myocardium to exogenous damage caused by hemodynamic overload, ischemia, diabetes, or probably other pathological stimuli.[9]

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Changes in myocardial structure may, also, be caused by lipid peroxidation as a result of increased levels of fatty acids induced by raised serum cholesterol[10] or by activation or dysregulation of mast cells.[11,12] Another possibility is the aggravation of heart damage by producing autoantibodies for G protein-coupled receptors by hypercholesterolemia.[13]

Metabolism of dietary lipids starts in the intestines by the production of chylomicrons and fatty acid — albumin complex, which is then transferred to the liver and muscles for further metabolism and production of other lipoproteins.[14]

All protocols for serum lipid study recommend 12-14 hour -fast, which was found essential to stabilize lipids, especially the triglycerides,[15] however; some reports considered the postprandial surge of triglycerides a potent atherogenic factor and cause of CVD[16–18], taking into account that people spend most of the day in a non-fasting state. They may get their next meal while serum triglyceride has not returned to the fasting value; on the other hand, reports claimed that the postprandial state is a dynamic, non-steady-state condition, with the rapid remodeling of lipoproteins compared with the relatively stable fasting condition. They recommended the inclusion of postprandial or non-fasting serum lipids in the study of their atherogenic effects.[16,17,19–21]

However, until serum lipids are considered blood glucose, time is needed to establish reference values for postprandial lipids and develop standardized methods for assessing postprandial dyslipidemia in routine clinical work and, probably, will achieve early detection of CVD risk.

DECLARATIONS

Conflict of interest

The author declare that there is no conflict of interest.

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