EDITORIAL

Thinking outside the box to lower the risk of atherosclerosis

Atherosclerosis is the major cause of morbidities and mortalities worldwide,[1] it lead to transient ischemic attacks, ischemic stroke, loss of vision, myocardial infarction, angina, sudden death, Nephropathy, Mesenteric ischemia, Impotence, Claudication, limb ischemia and gangrene.[2]

I have no doubt that clinical evidences are not all authored, to the extent that most medical professionals were misled and prevented from thinking outside the box for the best diet to lower the risk of atherosclerosis.

In order to have a dietary strategy to manipulate risk factors of atherosclerosis we have to understand the atherosclerosis pathophysiology; what is behind the problem? Is it fat or carbohydrate? Are there better guidelines?

According to conventional teaching; the fatty streaks are the first sign of atherosclerosis[1] as a result of high plasma LDL cholesterol that build up plaque[3] which makes the arteries narrow and less flexible, atherosclerosis is correlated to many risk factors that can be manipulated, such as; diabetis mellitus, hypertension, obesity, high LDL, low HDL, high triglycerides, smoking and the inactivity.[2,4]

The present dietary recommendation to reduce the risk of atherosclerosis is limiting intake of saturated fat.[5]

Recent systematic reviews and meta-analyses have started to question the validity of the lipid hypothesis, as there is lack of an association or an inverse association between LDL cholesterol and both all-cause and CVD mortality in the elderly. It is important to understand the atherosclerosis pathophysiology to get oriented towards the best dietary guidelines.[6]

The combination of excess caloric intake and relatively light physical activity, with the likely consequence of obesity, can induce a state of resistance to the action of insulin, insulin resistance may be considered a compensatory mechanism that protects the cells against further insulin-stimulated glucose and fatty acid uptake and therefore result in oxidative stress which lead to intimal damage.[7,8] Followed by subintimal chronic inflammation.[9] LDL modified by oxidation and then taken up by macrophages in the arterial intima resulting in the formation of foam cells[10] which acts as a bandage that help to heal the Inflammation. Calcium deposit[11] in the atheromatous plaque and arterial stenosis results from collagen accumulation.[12]

Furthermore; Evidence indicates that postprandial hyperglycemia is associated with oxidative stress generation and directly implicated in the development of cardiovascular disease;[13] this atherogenic effect appears to be independent of other cardiovascular risk factors such as hyperlipidemia.[14] The key question is; what to target to reduce the risk of atherosclerosis?

The key to reduce the incidence of atherosclerosis is to control the cause of the inflammatory process via appropriate diet, exercise, and healthy lifestyle choices,[15] because the dietary cholesterol has not been shown to contribute significantly to LDL plasma level.[16-19]

What is behind the problem? Is it fat or carbohydrate?

Surprisingly; we can live without eating carbohydrate, because the body have the ability to produce glucose through glycolysis orgluconeogenesis; This process takes place primarily in the liver of persons practicing low carbohydrate diets.[20]

All fats, including saturated fatty acids, have important roles in the body. However, the most important fats are essential fatty acids; those the body cannot synthesize and thus must come from the food we eat, the body requires fatfor the proper structure and function of every cell in the body, promote proper nerve functioning; help produce hormones; ensure normal growth and development and facilitate the absorption.

of fat soluble vitamins[21] such as vitamin D3 which can significantly restore the damage to the cardiovascular system caused by several diseases, including hypertension, diabetes and atherosclerosis,[22] and vitamin K2 which slow progression of atheromatous calcification and decreased arterial stiffness based on the results of a comprehensive review and analysis of both observational studies and randomized, double-blind clinical trials.[23]

Personally, I have most of the risk factors of atherosclerosis. I practiced the low-carb (5%) high fat diet (70%) and (25%) protein over eight months and continuing with excellent compliance and no adverse effects, which resulted in dramatic improvement on the atherosclerosis risk factors. The BMI reduced from 38 to 27, diabetes cured and all anti-diabetic medications stopped after 15-years of insulin therapy, with Hba1C returned to normal value. Blood pressure improved and medications was cut off to half, HDL significantly raised, dramatic reductions in triglycerides and LDL slightly reduced. However, LDL does not act alone.[3]

A meta-analysis of prospective epidemiologic studies showed that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of CHD or CVD.[24]

Saturated fat does not clog the arteries: coronary heart disease is a chronic inflammatory condition, the risk of which can be effectively reduced from healthy life style interventions.[25]

In humans, ketogenic diet have been associated with significant reductions in total cholesterol, increases in HDL cholesterol levels, decreases in triglycerides levels and an increase in size and volume of LDL cholesterol particles, which is considered to reduce cardiovascular risk by decreasing atherogenicity, ketogenic diet have demonstrated an improvement of type-2 diabetes status, inflammation and blood pressure while decreasing use of anti-hypertensive mediations.[26,27]

Finally, it is the time to retire the low-fat diet and introduce the carbohydrated restricted high fat diet as a preventive and therapeutic approach to oppose the increasing insulin resistance, and keep the risk factors of atherosclerosis within a healthy range.

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