

Letter to the editor

Clinical Updates on Lipid Metabolism

Hamid Yahya Hussain¹¹ Dubai health Authority

Copyright: © 2018 Hamid Yahya Hussain, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Lipids are fats, sourced out from food or synthesized by the liver. Triglycerides (TGs), as well as cholesterol, contribute most to disease, although all lipids are physiologically important. Cardio metabolic conditions, and gradually increase in the risk for pancreatitis is closely linked to Severity of HTG, specifically if triglyceride levels are not attended reduction throughout the follow-up [1]. On the contrary, mild-to-moderate HTG recognized as a cardiovascular risk factor, yet extremely severe HTG may not increase further, the risk for MI, stroke, or mortality. The reason behind is that the very low-density lipoprotein (VLDL) particles and Large triglyceride risk chylomicron are large enough to penetrate the endothelium and forming plaque. Nevertheless, a familial HTG Is always associated with increased risk of MI and strokes and death, smaller VLDL remnant particles, may maximize the size of fatty plaques, increase inflammatory cytokines and thrombosis [2-4]. More to that, it has been evidenced based noticed that elevated TGs are significantly associated with more health costs and care utilization raising the concern of reducing TG levels has great benefits to the patient.

Increase levels of lipids (mainly cholesterol) can lead to long-term problems, such as atherosclerosis. In general, increase total cholesterol level (that includes LDL, HDL, and VLDL cholesterol), and in specific a high level of LDL (the “bad”) cholesterol, eventually lead to maximizing the risk of atherosclerosis and risk of heart attack or stroke. It’s been obvious that not all types of cholesterol increase this risk. A high level of HDL (the “good”) cholesterol recognized to decrease risk, and conversely, a low level of HDL cholesterol may increase risk. In parallel, the long-term impact of triglyceride levels to the risk of heart attack is not as clear as enough [5]. As such, the very high levels of triglycerides (exceeding 500 milligrams per deciliter of blood, or mg/dL) associated with high risk of pancreatitis. Among all above 20 years groups, the total cholesterol, triglycerides, LDL cholesterol, and HDL cholesterol after fasting should be assess minimum every 4 to 6 years by fasting lipid profile [6]. concerning younger age groups (Children) a fasting lipid profile can be screened out at age 9 and 11 if they have a family member who has severe hyperlipidemia or developed coronary artery disease at a young age) they should be assessed age of 2 years old. Recently Available data, emphasizing that elevated TGs are always associated with worse outcomes among patients with ASCVD, even when LDL-C appears well adjusted but, factors like the dietary pattern, diabetic control, and physical activity, were not tested in multivariate models, which may interfere with the relationship between TGs and healthcare utilization [7]. Physicians are highly advised to educate their patients to follow a healthy lifestyle, as it can improve TG levels as well as CV risk factors. A researcher found that “Lowering triglycerides or LDL-C appears to have the same effect on the risk of coronary heart disease per Apo B particle lowered.” Hence, the clinical benefit of any lipid-lowering therapy should be linked to the absolute change in Apo B levels, not necessarily linked to the change in plasma triglyceride or LDL-C levels.”

In relation to Coronary artery diseases, lipid metabolism disorders can play a significant role in progressing of CAD. Thus, Dyslipidemia has a central role in the treatment protocol for both

prevention and treatment of cardiovascular events associated with coronary artery disease [8]. There are many pieces of evidence generated by epidemiological studies which prove an association between lipid parameters, and the risk of developing coronary artery disease as well as a progression of a pre-existing disease [9,10]. In specific, up owing levels of low-density lipoprotein cholesterol (LDL-C), reduced levels of HDL cholesterol (HDL-C), and high levels of triglycerides as well as increased lipoprotein(a) [Lp(a)] levels which should be taken in consideration when assessing the risk stratification of patients when seeking primary prevention of coronary artery disease. Dietary changes, Lifestyle as well as intensified statin therapy, with an addition of ezetimibe stay the best interventions at primary and secondary CAD; it will defiantly improve the prognosis by lowering levels of LDL-C. Contributions by genetic studies led to an understanding of the relationship between coronary artery disease and lipid metabolism. Causal progression of CAD demonstrated for LDL-C, Lpa and triglyceride-rich lipoproteins (TRL), and not be demonstrated for HDL-C in various studies. Reduction of LDL-C by proportion affect subtilisin/kexin type 9 (PCSK9) inhibitions and by the cholesterol ester transfer protein (CETP) inhibitor anacetrapib on cardiovascular events is investigating at the time being by different research programs.

References

1. American College of Cardiology.
2. Sinning D, Leistner DM Landmasses U (2016) Impact of lipid metabolism parameters on the development and progression of coronary artery disease: An update, *Erz* 41:273-280.
3. Alberti KGMM, Zimmet P, Shaw J (2005) IDF epidemiology task force consensus group. The metabolic syndrome-a new worldwide definition. *Lancet* 366:1059-1062.
4. Grundy SM, Brewer HB, Jr, Cleeman JI, Smith SC, Jr, Lenfant C, et al. (2004) National Heart, Lung, and Blood Institute; American Heart Association. Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Arterioscler Thrombus HVACs Biol* 24: e13-e18.
5. Malik S, Wong ND, Franklin SS, Kamath TV, L'Italien GJ, et al. (2004) Impact of the metabolic syndrome on mortality from coronary heart disease, cardiovascular disease, and all causes in United States adults. *Circulation* 110: 1245-1250.
6. International Diabetes Federation: IDF Worldwide Definition of the Metabolic

*Corresponding author: Hamid Yahya Hussain, Department of Community and Family Medicine, WHO public health Medicine Consultant, P O BOX 23317, Sharjah, UAE, Tel: 00 971 502608873; Fax: 00 971 502608873; E-mail: hussainh569@gmail.com

Received: September 07, 2018; Accepted: September 18, 2018; Published: September 21, 2018.

- Syndrome. <http://www.idf.org/metabolic-syndrome> Accessed 13 Feb 2017.
7. Kaur J (2014) A comprehensive review on metabolic syndrome. *Cardiol Res Pract* 2014: 943162. [[crossref](#)]
 8. Ford ES (2005) Prevalence of the metabolic syndrome defined by the international diabetes federation among adults in the U.S. *Diabetes Care* 28: 2745-2749.
 9. Waterhouse DF, McLaughlin AM, Sheehan F, O'Shea D (2009) An examination of the prevalence of IDF- and ATPIII-defined metabolic syndrome in an Irish screening population. *Ir J Med Sci* 178:161-166.
 10. Zhao Y, Yan H, Yang R, Li Q, Dang S, et al. (2014) Prevalence and determinants of metabolic syndrome among adults in a rural area of Northwest China. *PLoS One* 9: e91578. [[crossref](#)]