

Postprandial Hyperlipemia is an Indication for Additional Risk in Sitosterolemia

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Postprandial hyperlipidemia is a condition in which triglyceride (TG)-rich lipoproteins, including exogenous chylomicron and endogenous VLDL and their remnants, accumulate after meals. Although approximately four decades ago, Zilversmit¹⁾ proposed, for the first time, that atherogenesis is a postprandial phenomenon, little attention has been paid to this notion for a while afterward. However, recent epidemiological studies have demonstrated that non-fasting, but not fasting, TG levels are associated with increased coronary heart disease (CHD)^{2, 3)}. In addition, it is noteworthy that most of the day is considered as a non-fasting state.

After meals, exogenous chylomicron are introduced into the circulation and cleared through mechanisms which are, to some degree, in common to those for clearing endogenous VLDL⁴⁾. This is the reason why chylomicron/VLDL and their remnants accumulate postprandially in the circulation. Under postprandial hyperlipemia, peak of TG is higher in magnitude and later in time. It is also well-known that postprandial hyperlipemia is positively associated with fasting TG levels.

Sitosterolemia is an autosomal recessive disorder characterized by increased plant sterol levels, xanthomas, and accelerated atherosclerosis due to mutations in ABCG5/ABCG8 genes⁵⁾. Because some patients with sitosterolemia develop severe hypercholesterolemia⁶⁾, familial hypercholesterolemia (FH) should be considered during diagnosis.

Postprandial hyperlipemia under a high LDL-cholesterol (C) condition, such as sitosterolemia and FH, is an important issue because postprandial hyper-

lipemia, if exists, can further accelerate atherosclerosis. In fact, postprandial hyperlipemia has been reported in patients with heterozygous FH with some inconsistency⁷⁾. However, such studies on sitosterolemia are completely lacking.

In this issue of the Journal of Atherosclerosis and Thrombosis, Tada et al presented, for the first time, that patients with sitosterolemia, but not those with heterozygous FH, have postprandial hyperlipemia as assessed by incremental area under curves of TG, apoB48, RLP-C, and RLP-TG⁸⁾. Further, an increased postprandial sitosterol (iAUC) is another novel finding. These findings indicate that we should pay more attention to postprandial hyperlipemia to manage the vascular risk in patients with sitosterolemia.

Although the above findings are extremely provocative, several limitations should be considered. First, sample size is too small to reach convincing conclusions. Second, as mentioned above, patients with sitosterolemia have increased fasting TG levels; therefore, postprandial hyperlipemia is well expected. In this regard, assessing postprandial hyperlipemia between patients with sitosterolemia and controls with matched fasting TG levels should be an interesting study in future. Finally, it is surprising that peaks of TG and apoB48 are earlier, i.e., contradictory findings to “typical” postprandial hyperlipemia; however, elucidation of the mechanism or a discussion of these findings are lacking.

In summary, Tada et al presented that clearance of the postprandial RLP fraction was impaired in patients with sitosterolemia compared with that in those with heterozygous FH. These findings provide new insights into the roles of ABCG5/ABCG8 in lipoprotein metabolism during the postprandial period and a reason for a dietary counselling to pre-

vent future CHD.

COI

No COI to disclose.

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