COMMENT ON STEGMAN ET AL.

High-Intensity Statin Therapy Alters the Natural History of Diabetic Coronary Atherosclerosis: Insights From SATURN. Diabetes Care 2014;37:3114–3120

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We read with interest the article by Stegman et al. (1), which showed that high-intensity statin treatment reversed coronary plaque in patients with diabetes. The study provided new evidence for aggressive statin therapy in such a population; further, it underscored the importance of the LDL target value in diabetic patients. However, results from the study should be interpreted with following considerations.

First, although diabetic patients with coronary artery disease (CAD) represent a special population at high risk, participants recruited in the study (1) were relatively low risk; that is to say, they had mild to moderate severity of diabetes and CAD. This is reflected by the baseline characteristics of the diabetic patients, which showed a higher percentage of oral antidiabetes drug use (73%) and a lower percentage of insulin use (27%), prior percutaneous coronary intervention (20.1%), and secondary prevention drug use (aspirin 53.5%, ACE inhibitors 39.6%, etc.) for CAD. Thus, generalization of the conclusion to all diabetic patients with CAD is not proper.

Second, patients with CAD in the study were not an identical entity—patients with stable CAD and acute coronary syndrome were analyzed together. In fact, the Integrated Biomarkers and Imaging Study-4 (IBIS-4) (2) showed that, under treatment of high-intensity statin therapy, reduction of percent atheroma volume in nonculprit vessels of patients with ST-segment elevation myocardial infarction was only observed in those without diabetes; in contrast, there was even a tendency to increased percent atheroma volume in diabetic patients. Another serial intravascular ultrasound study from Japan (3) found that presence of diabetes weakened the effect of statin on coronary plaque regression in patients with acute coronary syndrome. In this sense, the result of coronary atheroma regression in the study by Stegman et al. was largely driven by statin effect in diabetic patients with stable CAD. However, the heterogeneous study population blurred the genuine effect of statin in diabetic patients.

Third, in comparison with CAD patients without diabetes, vulnerable plaque, including large necrotic core and thin-cap fibroatheroma, was more common in those with diabetes (4). Although Stegman et al. showed comparable degree of plaque regression in patients with and without diabetes from the perspective of atheroma volume, the progression or regression of plaque composition was not observed. Further studies are needed to elucidate the effect of statin on stabilizing coronary plaque in diabetic patients.

Last, as Stegman et al. (1) state in their limitations, diabetes status and glycemic control were less clear in the included patients; however, no matter what emphasis is put on the effect of statin, it must be kept in mind that glycemic control is the cornerstone for management of diabetic patients. Further, the benefits and risks of aggressive statin therapy should be balanced.

Duality of Interest. No potential conflicts of interest relevant to this article were reported.

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