Effect of High-intensity Statin Therapy on High-density Lipoprotein (HDL) Subfractions and Regression of Coronary Atheroma: The SATURN Trial

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Aim: Statin therapy can slow the progression of coronary atherosclerosis. However, the clinical factors underlying the beneficial effects of statin therapy on disease progression remain poorly understood. We examined the relationship of circulating HDL subfractions with measures of coronary atheroma following long-term high-intensity statin therapy. Methods: Serial coronary intravascular ultrasound (IVUS) was utilized in SATURN to monitor changes in atheroma burden [percent atheroma volume (PAV)] in 915 patients with coronary artery disease, treated with rosuvastatin (40 mg) or atorvastatin (80 mg) daily for 24 months. Results: Baseline levels of total HDL-C, apo E-containing HDL-C (apoE HDL-C), HDL₃-C and HDL₂-C did not differ significantly between treatment groups. Compared with the atorvastatin-treated group, rosuvastatin-treated patients demonstrated greater increases in levels of total HDL-C (4.4% vs. -1.8%, p<0.001), apoE HDL-C (8.5% vs. -3.3%, p<0.001), HDL₃-C (3.3% vs. -2.7%, p<0.001) and HDL₂-C (7.0% vs. -0.7%, p<0.001). The alterations in apo E HDL-C and HDL₃-C levels were associated with PAV regression in rosuvastatin-treated patients (β =-0.84, p=0.018 and β =-1.10, p=0.056, respectively) but not in the atorvastatin-treated group (β =0.41, p=0.22 and β=0.71, p=0.22 for apo E HDL-C and HDL₃-C, respectively). **Conclusions:** Rosuvastatin therapy resulted in favorable changes in HDL subfractions and on-treatment levels of apoE HDL-C and HDL₃-C were associated with greater disease regression.

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