REGRESSION OF LUMINAL STENOSIS AT THE SITE OF SILENT PLAQUE DISRUPTION AND HIGH-DENSITY LIPOPROTEIN CHOLESTEROL AS A POTENTIAL RISK OF ITS PROGRESSION IN THE ERA OF OPTIMAL MEDICAL THERAPY

Poster Contributions
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Authors: Takayoshi Nemoto, Kazunori Kashiwase, Akio Hirata, Mayu Nishio, Koshi Matsuo, Yasunori Ueda, Osaka Police Hospital, Osaka, Japan

Background: Plaque disruption followed by its healing process is supposed one of major mechanisms for atherosclerosis progression. Indeed, there is a pathological evidence of repeated plaque rupture and healing at the site of significant stenosis. We examine the change in the luminal stenosis at the site of silent plaque disruption and analyze its associated factors in the era of optimal medical therapy.

Methods: Among 144 consecutive patients who received coronary angiography and angioscopy that identified silent plaque disruption from August 2007 to December 2010 (baseline), 36 patients who had repeated coronary angiography later (follow-up) were included for analysis. Silent plaque disruption was defined as the plaque with thrombus detected by angioscopy in the non-culprit segments of coronary arteries. Diameter stenosis of the site with silent plaque disruption was angiographically measured at baseline and that of the same site at follow-up was also measured; and their difference was defined as stenosis change.

Results: Statin was used in 89% of study patients, and serum low-density lipoprotein cholesterol level was 91±21mg/dL. The diameter stenosis at silent plaque disruption decreased significantly from baseline to follow-up (32±14 vs. 27±14, p<0.001); and the stenosis change was −5.6±7.9%. High-density lipoprotein cholesterol was significantly associated with stenosis change (r=−0.51, p=0.001) and was the only factor significantly associated with stenosis change by multivariate linear regression analysis (B=−0.34, p=0.001).

Conclusions: In the era of optimal medical therapy with statin, the site of silent plaque disruption showed significant regression of luminal stenosis. However, the serum level of high-density lipoprotein cholesterol was negatively associated with the stenosis change at the site of silent plaque disruption.