

Evolution of non-culprit coronary plaques assessed by serial virtual histology-intravascular ultrasound in ST-segment elevation myocardial infarction and chronic total occlusion

Purpose: It remains uncertain whether natural course of coronary non-culprit plaques differ between ST-segment elevation myocardial infarction (STEMI) and chronic total occlusion (CTO) patients. The aim of this study was to compare the natural history of non-culprit coronary artery lesions in patients with STEMI and CTO.

Methods: We performed serial virtual histology (VH)-intravascular ultrasound (IVUS) studies to evaluate proximal non-culprit and untreated lesions that had a plaque burden >40% in 24 patients with STEMI and 11 patients with CTO at baseline and 6-month follow-up.

Results: A total of 26 lesions in STEMI patients and 11 lesions in CTO patients were analyzed. In STEMI patients, 20 of 26 lesions were IVUS-derived thin-cap fibroatheromas (TCFAs) at baseline. During follow-up, 10 of 20 TCFAs evolved into six pathological intimal thickenings (PITs), three thick-cap fibroatheromas (ThCFAs) and one fibrotic plaque. Two TCFA lesions were developed from a ThCFA and a PIT. In CTO patients, only two lesions were classified as TCFAs in 11 lesions (TCFAs in STEMI vs. CTO; 76.9% vs. 18.1%, $p=0.002$). All TCFAs changed into PIT, and there was no newly developed TCFA. The factors that were associated with plaque healing, defined as decreased proportion of necrotic core at the maximal necrotic core site were non-diabetes mellitus, high hs-CRP and proximal lesion locations.

Conclusions: The plaques of non-culprit lesions were more unstable in STEMI group than in CTO group at baseline. During the 6-month follow-up, changes in plaque composition were more dynamic in STEMI group than those in CTO group.