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Biologic significance of healed culprit plaques in stable angina versus acute coronary syndromes

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Topic(s):
Optical Coherence Tomography

Citation:
Background: Healed plaques, the signature of prior subclinical plaque destabilization, are frequently found in autopsy studies and have recently been described in patients with acute coronary syndromes (ACS).
Objectives: To compare the prevalence and features of plaque vulnerability of healed culprit lesions in stable angina pectoris (SAP) versus ACS patients by using Optical Coherence Tomography (OCT).
Methods: A total of 752 patients were included: 376 patients with SAP were selected using propensity score matching, comparable to 376 patients with ACS. Healed plaques were identified using established criteria, defined as layers of different optical density on OCT. Healed plaque prevalence along with angiographic and OCT findings were compared between the two groups.
Results: Healed plaques were more frequent in SAP than in ACS patients (42.0% vs 28.7%, p<0.001). LDL-cholesterol and high sensitive C-reactive protein (hs-CRP) levels were significantly lower in SAP patients with layered plaque as compared to ACS patients with layered plaque [97.9±36.9 mg/dL vs 116.7±39.2 mg/dL, p<0.001; 0.20 (0.10-0.83) mg/L vs 4.98 (1.00-11.32) mg/L, p<0.001, respectively]. Thin-cap fibroatheroma, macrophage accumulation and microvessels were significantly less frequent in layered plaques in SAP patients as compared to those in ACS patients (12.7% vs 56.5%, p<0.001, 7.0% vs 79.6%, p<0.001, and 20.3% vs 43.5%, p<0.001, respectively). Calcifications were found more frequently among layered plaques in SAP patients than in ACS patients (51.3% vs 33.6%, p=0.006).
Conclusions: Healed plaques, detected more frequently in SAP than in ACS patients, portend different atherosclerotic backgrounds. In SAP patients, plaque destabilization frequently does not lead to occlusive thrombosis, possibly due to low level of local vulnerability and systemic inflammation. In ACS patients, the presence of high level of local vulnerability and systemic inflammation may play an important role in occlusive thrombus formation, resulting in terminating the cycles of subclinical thrombosis and healing.
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