1145 Two Distinct Characteristics of Plaque Erosion by Multimodality Intracoronary Imaging

Background

Pathological studies have reported that patients with acute coronary syndrome (ACS) may have different plaque morphologies at culprit lesions and one of the underlying mechanisms for ACS is plaque erosion. However, the morphological features of plaque erosion obtained by multiple intracoronary imaging modalities have not been fully elucidated.

Case presentation

We experienced two ACS cases of plaque erosion with two distinct characteristics assessed by multiple intracoronary imaging modalities including optical coherence tomography (OCT), near-infrared spectroscopy intravascular ultrasound (NIRS-IVUS) and coronary angioscopy (CAS).

Case1. A 66-year-old female with a history of hypertension, dyslipidemia, and diabetes mellitus presented to the emergency department with acute onset of substernal chest pain lasting 60 min. The electrocardiogram was remarkable for ST-segment elevation in leads V1–4 and reciprocal ST-segment depression in leads II, III, and aVF. She was diagnosed with anterior ST-segment elevation myocardial infarction (STEMI). Emergent coronary angiography showed severe stenosis in the proximal left anterior descending artery (LAD). Pre-procedural OCT images revealed that a smooth luminal surface with thrombus overlying a lipid-rich plaque with IFC at the culprit lesion, categorized as OCT-erosion and no evidence of plaque rupture in multiple adjacent frames. NIRS-IVUS showed an echo-attenuated plaque and max lipid core burden index of a 4-mm segment (max LCBI4mm) was 628. CAS revealed the presence of yellow plaque with superimposing red thrombus. We recognized the culprit lesion as yellow eroded lipid-rich plaque.

Case2. A 53-year-old male with a history of smoking presented to the emergency department with an episode of chest pain lasting 30 min. The electrocardiogram was remarkable for ST-segment elevation in leads V1–6 and reciprocal ST-segment depression in leads II, III, and aVF. His initial cardiac enzymes were elevated. He was diagnosed with anterior STEMI. Emergent coronary angiography showed severe stenosis in the proximal LAD. Pre-procedural OCT imaging revealed that a smooth luminal surface with thrombus overlying a fibrous plaque with IFC at the culprit lesion, categorized as OCT-erosion and no evidence of plaque rupture in multiple adjacent frames. NIRS-IVUS showed a fibrous plaque and max LCBI4mm was 0. CAS revealed the presence of white plaque with superimposing red thrombus. We considered the culprit lesion as white eroded plaque without lipid.

Discussion

OCT-defined plaque erosion may not be the unique entity but have at least two distinct plaque morphologies, and NIRS and/or CAS may provide incremental ability of discriminating these plaque phenotypes classified as plaque erosion by OCT.