

Plaque erosion is a predictable clinical entity and tailored management in patients with ST-segment elevation myocardial infarction

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Diagnostic intravascular imaging [i.e., intravascular ultrasound (IVUS), optical coherence tomography (OCT), and coronary angiography (CAS)] have been used to assess the coronary artery atherosclerosis to detect stable, unstable and vulnerable plaques in the patients with acute coronary syndrome (ACS) who undergo percutaneous coronary intervention (PCI). Vulnerable plaques tend to be rupture and subsequent to thrombosis formation. Furthermore, two types of vulnerable plaques consist of the rupture- and erosion-prone (1). The ruptured plaque has large lipid-rich necrotic core with thin and inflammatory fibrous cap (2). With improving the technology of intracoronary imaging, the relationships between plaque morphology and patient's clinical prognosis greatly interested us in clinical practice and research. The OCT is a high-resolution imaging for plaque characteristics with approximately 10–20 μm resolutions. It can measure fibrous cap thickness and lipid arch, and detect presence of thin-cap fibroatheroma (TCFA), plaque rupture and plaque erosion (3). Previously numerous reported data revealed the relationship between plaque ruptures and OCT imaging (4) (5). However, plaque erosion has remained less well understood. Furthermore, there was controversy over the difference between the real incidence of plaque erosion in living patients and autopsy data.

Recently, Dai and colleagues conducted a prospective cohort study of determining predictors of plaque erosion in patients with STEMI (6). Among 822 STEMI patients who received the pre-intervention OCT, 209 (25.4%) had

plaque erosion and 564 (68.6%) had plaque rupture. The multivariable logistic analysis demonstrated that plaque erosion was significantly related to less than 50 years old, current smoking, lower lesion severity, non-multivessel disease, larger vessel size, less dyslipidemia, hypertension, chronic kidney disease (CKD), diabetes mellitus (DM), and near a bifurcation. Plaque erosion tends to be observed in younger patients and pre-menopausal women. Especially, cigarette smoking is the important coronary risk factor of plaque erosion rather than other risk factors such as dyslipidemia, hypertension, CKD, or DM. Plaque erosion is placed most in the proximal segment and near a bifurcation of the left anterior descending artery. Near a bifurcation is anatomically the most significant predictor of plaque erosion. Thrombus involved with plaque erosion occurs in fibrous or non-lipid plaques without TCFA and macrophages with larger lumen vessel. Especially, current smoking and near a bifurcation are observed in men whereas less than 50 years old is most predictively observed in women.

These results have the important investigative implications. Based on these results, plaque erosion is a predictable clinical entity. Plaque erosion has specific role of risk factors in men and women, and furthermore should be considered regarding tailored management in STEMI patients. Pathological studies demonstrated that plaque erosion is not observed in the disruption of fibrous cap and the thrombus formed by plaque erosion does not come into contact with the lipid pool or necrotic core but

directly contacts the smooth muscle cell in the proteoglycan rich matrix (7). The definite OCT-defined plaque erosion is defined as the intraluminal thrombus on a relatively intact intima without plaque rupture. However, the OCT image cannot accurately visualize the endothelial cells on the plaque. The EROSION study demonstrated that administration of heparin and dual anti-platelet therapy (DAPT) effectively reduced volume of residual thrombus without stent implantation, and increased the blood flow without re-occlusion of the erosive culprit lesion at 1 month in selected ACS patients with plaque erosion (8). Furthermore, follow-up OCT imaging after one-year revealed a further reducing residual thrombus volume between one month and one-year follow-up (9). Major adverse cardiovascular event for ≤ 1 year did not occur in 92.5% of ACS patients with plaque erosion who received the DAPT such as aspirin and ticagrelor without stenting. Preintervention 3-vessel OCT demonstrated that ACS patients with culprit-related plaque erosion had a few nonculprit plaques and the lower levels of instability on panvascular vessels in comparison with those with culprit-related plaque rupture (10). They confirmed that there were distinctively pathophysiologic different mechanisms between in plaque erosion and plaque rupture.

On the other hand, this study has several limitations. It excluded the patients with non-STEMI and unstable angina. The plaque erosion defined by OCT is an exclusive diagnosis because the current OCT system cannot visualize individual endothelial cells. A large residual thrombus is also difficult to distinguish plaque rupture from plaque erosion by assessment of the underlying plaque characteristics. Use of routine OCT imaging has not established the clinical benefits properly.

Large-scale clinical trials in the future will reveal the potential strengths of whether these findings may herald the paradigm shift targeted for comprehensive atherothrombotic risk assessment, modification from coronary risk factors observed in plaque rupture, and tailored management in patients with plaque erosion.

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Footnote

Conflicts of Interest: The author has no conflicts of interest to declare.

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