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Uncovered Culprit Plaque Ruptures in Patients With ST-Segment Elevation Myocardial Infarction Assessed by Optical Coherence Tomography and Intravascular Ultrasound With iMap

Mikkel Hougaard, MD, PhD, Henrik Steen Hansen, MD, DMSci, Per Thayssen, MD, DMSci, Lisbeth Antonsen, MD, PhD, Lisette Okkels Jensen, MD, PhD, DMSci

ABSTRACT

OBJECTIVES This study assessed the incidence and course of healing of uncovered plaque ruptures (PR) following primary percutaneous coronary intervention.

BACKGROUND The infarct-related occlusion is frequently located at the lesion site with maximum thrombus burden, whereas the culprit PR may be situated more proximally or distally.

METHODS Uncovered PR in segments adjacent to the stent were identified by optical coherence tomography and intravascular ultrasound using iMap (Boston Scientific, Marlborough, Massachusetts) within 48 h and after 12 months. The percentages of necrotic core, fibrotic tissue, lipid tissue, and calcific tissue were determined.

RESULTS Eleven uncovered PR were found in 10 of 77 patients (13.0%). Eight of these ruptures (10.4%) were identified as culprit and were located proximal to the stent. Two patients were treated before follow-up due to recurrent symptoms. After 12 months, 3 PR had healed incompletely without causing symptoms. The lumen area at the PR site was reduced (7.5 mm^2 [interquartile range (IQR): 4.8 to 9.3 mm^2] to 3.6 mm^2 [IQR: 2.8 to 8.0 mm^2]; $p = 0.012$). Proximal segments with uncovered PR had greater plaque volumes (62.1 mm^3 [IQR: 50.2 to 83.6 mm^3] vs. 38.7 mm^3 [IQR: 29.6 to 47.6 mm^3], respectively; $p < 0.001$), vessel volumes (110.7 mm^3 [IQR: 92.3 to 128.1 mm^3] vs. 76.0 mm^3 [IQR: 63.8 to 100.3 mm^3], respectively; $p < 0.001$), and greater percentages of necrotic core (34.0% [IQR: 29.0% to 44.5%] vs. 20.5% (IQR: 10.0% to 29.0%]; $p < 0.001$). Conversely, percentages of fibrotic tissue were lower (44.0% [IQR: 32.0% to 47.0%] vs. 56.0% [IQR: 46.0% to 66.0%]; $p = 0.001$), whereas no differences were found for lipid tissue and calcific tissue.

CONCLUSIONS Uncovered culprit ruptures detected by optical coherence tomography were common following primary percutaneous coronary intervention and were found to be associated with significant lumen reduction during the healing process. (J Am Coll Cardiol Img 2017;■:■-■) © 2017 by the American College of Cardiology Foundation.

Coronary plaque ruptures are regarded as the preceding cause of myocardial infarctions and are in many cases located in lesions with a high plaque burden together with a necrotic core covered by a thin fibrous cap (1-3). These features make up what is known as thin-cap fibroatheromas (TCFA). In the setting of ST-segment elevation myocardial infarction (STEMI), an occlusion of the vessel occurs due to thrombus formation, plaque hemorrhage, and vessel spasm (4). The angiographic

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ABBREVIATIONS AND ACRONYMS

CT = calcific tissue characterized by iMap

EEM = external elastic membrane

FT = fibrotic tissue characterized by iMap

IVUS = intravascular ultrasound

LT = lipid tissue characterized by iMap

NC = necrotic tissue characterized by iMap

OCT = optical coherence tomography

TCFA = thin cap fibroatheroma (thickness: <65-μm separation of lipid plaque from the vessel lumen)

VH = virtual histology

presentation is a disruption of the luminal continuity that has been caused by either an occlusion or a severe stenosis of the coronary vessel. The site with the angiographic culprit lesion is the target for percutaneous coronary intervention (PCI), but the true culprit lesion may not be lumen compromising and may be located proximal or distal to the target of intervention (5). For this reason, there will be a risk of incomplete stent coverage when intervention is guided by angiography alone.

Intravascular imaging with optical coherence tomography (OCT) or intravascular ultrasound (IVUS) can assist in the identification of the site of the true culprit and may lead to better stent coverage of these lesions. Due to the high resolution of OCT, a direct assessment of the TCFA is possible (6,7), and with IVUS, a further remodeling assessment and characterization of the plaque composition can be achieved by spectral analysis of the ultrasound scatter, known as iMap (Boston Scientific, Marlborough, Massachusetts), which enables differentiation of plaque composition into necrotic core (NC), fibrotic tissue (FT), calcific tissue (CT), and lipid tissue (LT) (8).

In the present study, blinded OCT and IVUS with iMap was performed in a first-time STEMI population within 48 h after their primary PCI procedure and was repeated after 12 months. We identified the uncovered plaque ruptures by OCT and subcategorized those showing signs of culprit features. The associated plaque volume and composition was assessed in the proximal and distal stent edge segments by using IVUS with iMap.

METHODS

SETTING AND DESIGN. The present study was a substudy of the OCTIVUS (Plaque Composition in Patients with acute ST Segment Elevation Myocardial Infarction assessed by Optical Coherence Tomography and IntraVascular UltraSound with iMap) trial (9) (U.S. Clinical Trials identifier: NCT01385631), which was a single-center double-blinded randomized trial that enrolled statin-naïve patients with first-time STEMI.

From June 2011 to June 2013, a total of 1,062 patients with STEMI were admitted, and of these, 87 patients were included. The main inclusion criteria were: first-time STEMI, no prior treatment with statins or other lipid-lowering drugs, and a nonsignificant lesion in one of the 2 nonculprit coronary arteries (angiographic diameter stenosis >20% and <50%). The main exclusion criteria were: 1) age <18 years or >81 years; 2) impaired kidney function; 3) women with child-bearing potential who were not using chemical or mechanical contraception; 4) history of malignancy, unless a disease-free period of more than 5 years was present; 5) participation in another randomized trial; and 6) treatment with cyclosporine or fibrates.

Patients were examined using OCT and IVUS with iMap within 48 h after undergoing their primary PCI. IVUS acquisition with tissue characterization and OCT of the implanted stent in the infarcted artery were performed together with a plaque study in a nonculprit artery. After 12 months, the IVUS with tissue characterization and OCT were repeated.

INTRACORONARY IMAGING ACQUISITION. Prior to the procedure, unfractionated heparin (5,000 IU) was administered. Nitroglycerin, 200 μg, was administered by intracoronary route prior to the pullback.

OCT was performed using the Lightlab model Cx7 (St. Jude Medical, Little Canada, Minnesota) and later the Ilumien System, both using a Dragonfly OCT catheter (St. Jude). The catheters were initially flushed with contrast (Visipaque, GE Healthcare, Chicago, Illinois) and wiped with heparinized saline water, activating the hydrophilic coating. Catheter placement was guided angiographically by visualization of the placed coronary stent, and the catheter was placed at least 5 mm distal to the distal stent edge. Pullback was initiated automatically by manually flushing the vessel with 20 ml of contrast (Visipaque). Pullback speed was 20 mm/s, and the total pullback distance of the system was 55 mm. Repeated pullback was performed in case of insufficient image quality or incomplete acquisition of the segment of interest.

The IVUS pullback was performed using the iLab system with a mechanical 40 MHz Atlantis SR Pro IVUS catheter (both, Boston Scientific). An automatic pullback was performed with a standard pullback speed of 0.5 mm/s. The IVUS catheter was advanced ≥5 mm distal to the stented segment, and imaging was performed throughout the stent to ≥5 mm of the proximal reference segment site. iMap data were obtained at every 30th frame (0.5 mm).

All intravascular image acquisition was documentary and not shared with the operator and did not influence the clinical decision making. During off-line review, the examiner was blinded to the patient data.

OFF-LINE ANALYSIS. OCT pullbacks were analyzed using proprietary software (St. Jude Medical). The stent and its adjacent 5-mm distal and proximal reference segments were marked and processed with the automatic lumen contour with supplementary