IJC Heart & Vasculature 11 (2016) 43–48

Contents lists available at ScienceDirect

IJC Heart & Vasculature

journal homepage: http://www.journals.elsevier.com/ijc-heart-and-vasculature

Coronary plaque morphology on multi-modality imagining and periprocedural myocardial infarction after percutaneous coronary intervention☆

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ARTICLE INFO

Article history:
Received 1 November 2015
Accepted 4 March 2016
Available online 17 March 2016

Keywords:
Coronary imaging
Percutaneous coronary intervention
Periprocedural myocardial infarction
Vulnerable plaque

ABSTRACT

Percutaneous coronary intervention (PCI) may be complicated by periprocedural myocardial infarction (PMI) as manifested by elevated cardiac biomarkers such as creatine kinase (CK)-MB or troponin T. The occurrence of PMI has been shown to be associated with worse short- and long-term clinical outcome. However, recent studies suggest that PMI defined by biomarker levels alone is a marker of atherosclerosis burden and procedural complexity but in most cases does not have independent prognostic significance. Diagnostic multi-modality imaging such as intravascular ultrasound, optical coherence tomography, coronary angioscopy, near-infrared spectroscopy, multidetector computed tomography, and magnetic resonance imaging can be used to closely investigate the atherosclerotic lesion in order to detect morphological markers of unstable and vulnerable plaques in the patients undergoing PCI. With the improvement of technical aspects of multimodality coronary imaging, clinical practice and research are increasingly shifting toward defining the clinical implication of plaque morphology and patients outcomes. There were numerous published data regarding the relationship between pre-PCI lesion subsets on multi-modality imaging and post-PCI biomarker levels. In this review, we discuss the relationship between coronary plaque morphology estimated by invasive or noninvasive coronary imaging and the occurrence of PMI. Furthermore, this review underlies that the value of the multimodality coronary imaging approach will become the gold standard for invasive or noninvasive prediction of PMI in clinical practice.

1. Introduction

Invasive coronary angiography (ICA) has been used as the gold standard for the diagnosis of coronary narrowing and clinical decision making for coronary interventions. However, coronary angiography has several limitations, including the substantial interpretation variability of visual estimates and assessment of lesion severity for diffuse atherosclerotic lesions and intermediate-severity lesions [1,2]. Percutaneous coronary intervention (PCI) has been one of the standard revascularization procedures for patients with significant coronary artery disease. PCI may be complicated by periprocedural myocardial infarction (PMI) as manifested by elevated cardiac biomarkers such as creatine kinase (CK)-MB or troponin T. The occurrence of PMI has been shown to be associated with worse short- and long-term clinical outcome [3–5]. However, recent studies suggest that PMI defined by biomarker levels alone is a marker of atherosclerosis burden and procedural complexity but in most cases does not have independent prognostic significance [6–8]. With the improvement of technical aspects of multimodality coronary imaging, clinical practice and research are increasingly shifting toward defining the clinical implication of plaque morphology and patients' outcomes. In this review, we discuss the relationship between coronary plaque morphology estimated by invasive or noninvasive coronary imaging and the occurrence of PMI.

2. Periprocedural myocardial infarction after PCI

Cardiac troponin I (cTnI) and cTnT are the biomarkers of choice for the diagnosis of myocardial damage, because they are the most sensitive and cardiac-specific biomarkers currently available. The recently published third universal definition of MI attempts to provide some guidance by defining PMI in patients with normal (<99th percentile upper reference limit (URL)) baseline cTn concentrations as an elevation of >5 × URL within 48 h of the procedure together with either (i) evidence of prolonged (>20 min) ischaemia as demonstrated by chest pain; (ii) ischaemic ST changes or new pathological Q waves; (iii) angiographic evidence of a flow-limiting complication, such as of loss of patency of a side branch, persistent slow-flow or no-reflow, embolization; or (iv) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality [9]. Porto et al. found that the cause of periprocedural

☆ Grant support: None.
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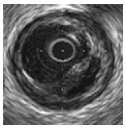
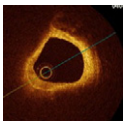
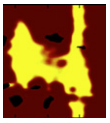
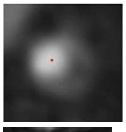
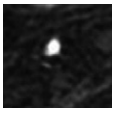
http://dx.doi.org/10.1016/j.ijcha.2016.03.009

Table 1
Characteristics of various imaging modalities for analysis of coronary plaque for the detection of PMI.

Modalities	Images	Characteristics of plaque
IVUS		Grayscale: plaque burden, ruptured plaque, attenuated plaque VH: necrotic core area or volume IB: lipid volume iMAP: necrotic tissue volume
OCT		Ruptured plaque TCFA (large lipid core and a thin fibrous cap)
Coronary angioscopy		Intensive yellow plaque Ruptured plaque
NIRS		Lipid-core plaque (LCP) with a maxLCBI (4 mm) ≧500
MDCT		Low-attenuation, positive remodeling, spotty calcification Ring-like appearance
MRI		High-intensity plaque detected in non-contrast T1WI

PMI = periprocedural myocardial infarction, IVUS = intravascular ultrasound, VH = virtual histology, IB = integrated backscatter, OCT = optical coherence tomography, TCFA = thin-cap fibroatheroma, CAS = coronary angioscopy, NIRS = near-infrared spectroscopy, LCBI = lipid core burden index, MDCT = multi-detector computed tomography, MRI = magnetic resonance imaging.

myocardial necrosis after PCI was the impairment of flow in coronary side branches and distal embolization of atheromatous or thrombotic materials [10]. Park et al. have demonstrated that the side-branch occlusion was the most common cause (57.3%) for the mechanism of PMI, and other reasons included slow- or no-reflow, flow-limiting dissection, and distal embolization [11]. The slow-flow, no re-flow phenomenon, or PMI after PCI has been associated with distal embolization, especially of plaque debris, and with unfavorable clinical outcomes [12,13]. Therefore, pre-PCI plaque composition on coronary imaging may have an impact on myocardial infarction during PCI (Table 1).

3. Invasive imaging for prediction of PMI

Diagnostic multi-modality imaging can be used to closely investigate the atherosclerotic lesion in order to detect morphological markers of unstable and vulnerable plaques in the patients undergoing invasive coronary angiography and PCI. There were numerous published data regarding the relationship between pre-PCI lesion subsets on multi-modality imaging and post-PCI biomarker levels.

3.1. Intravascular ultrasound (IVUS) (Table 2)

IVUS is widely used to assess coronary artery morphometry. Virtual histology (HV) IVUS, integrated backscatter (IB) IVUS, and iMAP IVUS are available radio frequency analysis in clinical practice. Mehran et al. [14] studied 2256 consecutive patients who underwent PCI of 2780 native coronary lesions and had pre-PCI IVUS imaging; both a greater lesion and reference segment plaque burden were associated with post-PCI creatine kinase-MB elevation. Fujii et al. [15] compared creatine kinase-MB release after stent implantation in 62 patients with IVUS-detected ruptured plaques with 62 matched control patients; patients with ruptured plaques had higher creatine kinase-MB release rates than control patients, and independent predictors of post-PCI creatine kinase-MB elevation were presence of ruptured plaque and unstable angina. Grayscale IVUS has significant limitations in assessing plaque composition, and radiofrequency-derived histopathological analysis by IVUS may provide quantitative information on plaque composition. VH-IVUS has demonstrated that large necrotic cores are related to distal embolization and post-PCI cTn elevation [16,17]. The VH-IVUS may play an important role in detecting which lesions are high risks for myocardial necrosis after PCI. Uetani et al. [18] investigated 114 consecutive patients who received elective stent implantations following IB-IVUS analysis regarding the association between quantitative analysis of the plaques of target lesions and the risk of post-procedural myocardial injury after stenting. Lipid and fibrous volumes correlated with post-procedural cardiac biomarkers, and the lipid volume fraction (lipid volume/total plaque volume) also correlated with post-procedural TnT and CK-MB. Lipid volume and volume fraction were concluded to be independent predictors of post-procedural myocardial injury. The innovative radiofrequency-based analysis of tissue characterization by IVUS (iMAP-IVUS) analyses also demonstrated that necrotic tissue volume is a potent predictor of PMI [19]. In a study of 95 consecutive patients with stable angina or ACS patients with slow flow after PCI had significantly higher absolute necrotic plaque volumes (43.3 mm^3 versus 20.1 mm^3; $P = 0.0004$), as detected by iMAP IVUS [20].

Table 2
Coronary plaque characteristics on IVUS for prediction of PMI.

Author	Number of patients	Events rate (%)	Imaging result	Odds ratio (95% CI)
Mehran et al. [14]	2256	CK-MB ≧ 1 × ULN (25.7%)	Plaque burden	1.14 (1.07–1.82)
Fujii et al. [15]	62	CK-MB > 3 × ULN (15%)	Ruptured plaque	$P = 0.03$
Kawamoto et al. [16]	44	HITS with Doppler guidewire > 12 (29.5%)	Necrotic core area (VH, mm^2)	4.41 (1.03–18.81)
Hong et al. [17]	80	TnT > 3 × ULN (47.5%)	Necrotic core area (VH, mm^2)	1.318 (1. 090–1.594)
Uetani et al. [18]	114	TnT > 3 × ULN (11.4%)	Lipid volume (IB, mm^3)	0.011 (0.004–0.016)
Higuchi et al. [19]	33	TnT > 5 × ULN (36.3%)	Necrotic tissue volume (iMAP, mm^3)	1.026 (1. 004–1.048)
Utunomiya et al. [20]	95	Slow flow (11.6%)	Necrotic plaque volume (iMAP, mm^3)	n/a

PMI = periprocedural myocardial infarction, CI = confidence interval, IVUS = intravascular ultrasound, CK-MB = creatine kinase, ULN = upper limit of normal, TnT = troponin T, VH = virtual histology, IB = integrated backscatter.