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Review article – *Special issue: Acute Coronary Syndromes*

Vulnerable plaque imaging and acute coronary syndrome

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ARTICLE INFO

Article history:
Received 13 March 2014
Received in revised form
8 May 2014
Accepted 10 May 2014
Available online 3 July 2014

Keywords:
Lipid-core plaque
Vulnerable plaque
Thin-cap fibroatheroma
Near-infrared spectroscopy
Intravascular ultrasound
Optical coherence tomography

ABSTRACT

The term vulnerable plaque has been established to describe an atherosclerotic lesion with a high probability of causing a future coronary event [1,2]. The identification and stabilization of the lesion before its rupture may reduce the morbidity and mortality caused by coronary artery disease. Modern imaging modalities such as computer tomography coronary angiography, intravascular ultrasound, optical coherence tomography and near-infrared spectroscopy have a potential in finding these vulnerable plaques. This raises opportunities in the primary and secondary prevention of coronary artery disease. This review summarizes the current knowledge with an emphasis put on the research advances in the field of near-infrared spectroscopy a modality that has been intentionally developed for the detection of lipid-core plaques.

Contents

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http://dx.doi.org/10.1016/j.crvasa.2014.05.002

Introduction

In the past decades it has been established that the development of a vulnerable atherosclerotic plaque (VP) is the initial step in the process leading directly to coronary thrombosis. Plaque vulnerability was defined functionally as the susceptibility of a plaque to rupture [1]. It has been revealed in autopsy studies that the non-thrombosed lesion that most resembles the acute plaque rupture is the thin cap fibroatheroma (TCFA), which is characterized by a large necrotic core of lipid and cellular debris and with an overlying fibrous cap measuring <65 μm, containing only rarely smooth muscle cells but numerous macrophages [3]. Based on these pathological findings it seems crucial to identify such high-risk plaques in stable patients, with the ultimate goal of preventing its rupture, thereby averting myocardial infarction and sudden cardiac death [4]. The prospective clinical multicenter study PROSPECT showed that lesions responsible for unanticipated events are frequently angiographically mild. However, most of them are thin-cap fibroatheromas and can be characterized by a large plaque burden, a small luminal area, or some combination of these characteristics, as determined by gray-scale and radiofrequency intravascular ultrasonography [5]. The results of the PREDICTION trial carried out by Stone et al. proofed that not only large plaque burden, but also low local endothelial shear stress can influence plaque progression [6]. The above-mentioned characteristics identified by some non-invasive or invasive imaging methods might help to detect VP in high-risk patients. Near-infrared spectroscopy (NIRS) imaging was developed explicitly for detection of a lipid-core plaque (LCP) a believed clinical correlate of VP [4]. The hybrid catheter that contains near-infrared spectroscopy and intravascular ultrasound (NIRS-IVUS) can assess plaque burden while simultaneously identifying LRP with NIRS and therefore offers a unique possibility of combined information about chemical composition and morphology of the plaque.

Non-invasive methods – possibly an important tool in primary prevention

Although non-invasive modalities are obviously the less precise method of VP detection, a research in this field is particularly important as these techniques may help to identify high-risk plaques in asymptomatic subjects without the risks associated with an invasive procedure. Amongst the non-invasive methods CT coronary angiography (CTA) seems to be the most notable one. The presence of positive remodeling, low attenuation plaque (LAP) and spotty calcification were significantly more frequent in the culprit acute coronary syndrome (ACS) lesions in the study by Motoyama et al. [7]. Furthermore, in asymptomatic patients, presence of positive remodeling and LAP portends a greater risk for development of acute coronary events. However, CTA is not indicated in the evaluation of most asymptomatic patients given the risks of radiation burden. Primary prevention studies are needed to evaluate the merit of plaque characterization and to define the population that will benefit the most from such a strategy [4].

Invasive methods in vulnerable plaque imaging

The angiogram is still considered the ''gold standard'' for the definition of coronary anatomy; although it is well recognized that angiography significantly underestimates the extent of atherosclerosis [8]. Therefore, numerous invasive methods have been evaluated to increase sensitivity and specificity of atherosclerotic plaque detection. Some of them focus on the microanatomy of a plaque (intravascular ultrasound – IVUS, intravascular optical coherence tomography – OCT), some are directed at measuring the metabolic activity of a plaque to predict the risk of its disruption (intravascular thermography and elastography), and finally some techniques rely on measuring the chemical composition of a plaque (NIRS) [9–11]. The most promising methods include IVUS, OCT and NIRS [2].

Intravascular ultrasound

Between the above-mentioned methods, IVUS has a particular role in the VP issue. It can visualize the external elastic lamina of the vessel wall, allowing the determination of vessel size, plaque burden and morphology [12]. Radiofrequency (RF) analysis of the ultrasonic signal (virtual histology RF – IVUS) permits further characterization of plaque composition, which correlates well with histological analysis [13]. It is currently the only method with a proven capability of identifying lesions responsible for future major adverse cardiac events (MACE). This has been confirmed by prospective natural history studies.

The most important of these studies is certainly the pioneering PROSPECT trial. In this prospective study, 697 patients with ACS underwent three-vessel coronary angiography, gray-scale and radiofrequency IVUS after percutaneous coronary intervention (PCI). Subsequent MACE (death from cardiac causes, cardiac arrest, myocardial infarction, or re-hospitalization due to unstable or progressive angina) were adjudicated to be related to either originally treated (culprit) lesions or untreated (non-culprit) lesions. The median follow-up period was 3.4 years. About half of the subsequent MACE were related to non-culprit lesions. The interesting fact was that most non-culprit lesions responsible for follow-up events were angiographically mild at baseline, but were more likely than those not associated with recurrent events to be characterized by a plaque burden of 70% or greater or a small minimal luminal area or to be classified on the basis of RF-IVUS