Chapter 18

Current Clinical Status of Vascular Non-Invasive Imaging Methodologies

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ABSTRACT

Rupture of high risk atherosclerotic plaque is responsible for acute vascular events such as myocardial infarction and sudden cardiac death. Several non-invasive vascular imaging methods have been developed to identify and characterise atherosclerotic plaques at risk of rupture. In this chapter we will discuss the background, rationale, and current state of non-invasive vascular imaging.

INTRODUCTION

Many risk factors and physiological responses contribute to the development of atherosclerotic plaques, a disease process that primarily affects large and medium sized arteries. Fatty streaks appear within the vascular intima early in adolescence and evolve over many decades into atherosclerotic plaques with the deposition of lipids, necrotic debris, migration of smooth muscle and inflammatory cells (Ross & Glomset, 1976a, 1976b; Stary, 1992, 2000). A fibrous cap produced by smooth muscle cells maintain the stability of the plaque and separates the thrombogenic core of the plaque from the blood stream. In the early stages of development the plaques do not obstruct the vessel lumen due to outward expansion of the vessel wall (positive remodeling) (Glagov, Weisenberg, Zarins, Stankunavicius, & Kolettis, 1987).

The stability of the fibrous cap and the entire plaque is regulated by the lymphocytes and macrophages cells within the plaque (Daugherty & Rateri, 2002). Lymphocytes and macrophages when activated secrete interferon, matrix degrading proteases and other cytokines that inhibit smooth muscle cells, reduce collagen production and effect thinning of the fibrous cap (Davies,
Eventual erosion and/or rupture of the fibrous cap expose the necrotic core to the blood stream leading to thrombus formation and abrupt closure of the vessel lumen. Several postmortem studies have confirmed the presence of plaque erosion (Farb et al., 1996) and rupture (Burke et al., 1997) in patients who presented with acute vascular events. Other characteristic features such as increased neovascularisation and macrophage accumulation especially in the shoulder region of the plaque have also been noted in ruptured plaques (Moreno et al., 2004; Muller, Abela, Nesto, & Tofler, 1994). Plaque components and hallmarks of a rupture prone plaque are summarized in Table 1.

Assessment of coronary atherosclerosis by coronary angiography is limited since atherosclerotic plaques do not affect the arterial lumen until late in the disease process (Kim et al., 2002; Topol & Nissen, 1995). Furthermore, non-critical coronary stenoses are more often the culprit lesion in patients presenting with acute coronary syndrome (Hackett, Davies, & Maseri, 1988). Majority of patients who present with an acute coronary syndrome do not have prior symptoms of myocardial ischaemia. It is therefore essential to measure plaque burden and function before symptoms develop (Fayad & Fuster, 2001). Intravascular ultrasound, optical coherence tomography, thermography, intravascular magnetic resonance imaging and other intravascular imaging techniques were developed to assess the structure and function of atherosclerotic plaques early in their development when they are still confined to the arterial wall. However the techniques are invasive and can be utilised only in selected patients.

Non-invasive vascular imaging modalities include magnetic resonance imaging (MRI), cardiovascular computed tomography angiography (CCTA), and nuclear imaging techniques. Until recently the primary role of non-invasive imaging has been to identify myocardial ischaemia in symptomatic patients and act as a gatekeeper for coronary revascularization. In this chapter we discuss in detail the various non-invasive methods used to image atherosclerosis.

**NON-INVASIVE VASCULAR IMAGING**

Non-invasive vascular imaging can provide information about the size and distribution of plaques, the total atherosclerotic burden and in addition, can characterize plaque morphology, composition,

### Table 1. Plaque components and factors that predispose to plaque rupture

<table>
<thead>
<tr>
<th>Plaque components</th>
<th>Atheromatous core – lipid, necrotic debris</th>
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<tbody>
<tr>
<td>Extracellular matrix</td>
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<td>Calcium</td>
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<td>Fibrous cap</td>
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<tr>
<td>Features of high risk plaque</td>
<td>Thin cap fibroatheroma</td>
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<tr>
<td></td>
<td>Large lipid/necrotic core</td>
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<td></td>
<td>Active inflammation – plaque infiltration with macrophages and lymphocytes</td>
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<td></td>
<td>Outward positive remodeling</td>
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<td>Increased plaque microneovascularisation</td>
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<td>Plaque ulceration / Plaque fissure</td>
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<td>Spotty calcification</td>
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<td>Platelet aggregation and thrombus formation</td>
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