14 Coronary Artery Disease

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14.1 Introduction

Coronary heart disease is the leading cause of morbidity and mortality in the developed countries, and the World Health Organization estimate that it will be the leading cause of morbidity and mortality worldwide by 2020 (Reddy and Yusuf 1998). Catheter-based X-ray coronary angiography is considered the technique of choice for detecting, quantifying and treating atherosclerotic coronary lesions. This technique, however, has many limitations and, because of its invasive nature, it is not suited as a screening modality. The substantial technical progress in magnetic resonance (MR) imaging, electron beam computed tomography (EBCT) and multi-slice computed tomography (MSCT) in the last decade has opened new horizons to study the coronary arteries non-invasively, and therefore to compete with catheter-based coronary angiography. Although non-invasive coronary artery imaging with MR imaging is highly challenging and, to date (2004), should still be considered as emerging, it has the potential to provide unique information about the presence, extent and severity of coronary artery disease (CAD). Three-dimensional angiographic or “luminographic” MR imaging techniques can detect or exclude the presence of stenotic atherosclerotic le-
sions, while MR flow techniques can be applied to assess the severity of a stenotic lesion on the distal coronary flow. In addition, MR imaging techniques are under development to visualize the coronary artery wall, enabling assessment of wall remodelling and plaque characterization. The above-mentioned techniques can also be applied to study the patient with recurrent anginal symptoms after percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) surgery. Unlike EBCT or MSCT, coronary MR angiography does not require iodinated contrast agents or X-ray radiation and therefore has the potential to become a more widespread and easy-to-use screening tool to investigate subjects at high risk for developing atherosclerosis and acute coronary syndromes. Coronary MR imaging techniques can also be part of a broader cardiac investigation where, within a single MR imaging study, valuable and often unique information can be obtained with regard to ventricular function, myocardial perfusion and myocardial viability in patients with ischemic heart disease. Since most coronary MR imaging techniques are still premature for clinical use, the different approaches and strategies currently used are extensively discussed. In addition, the current and potential clinical indications, as well as the role of MR imaging compared with MSCT and EBCT, are highlighted in this chapter.

14.2 Pathogenesis and Pathophysiology of Coronary Artery Disease

Atherosclerosis, the disease process underlying CAD, is a systemic disease of the arterial vessel wall that causes distinct clinical manifestations, depending on the affected circulatory bed and the characteristics of the individual lesions (Fuster et al. 1999; Fuster and Gotto 2000). Atherosclerosis develops silently over several decades and begins as early as young adulthood (Strong et al. 1997). Since clinical complications such as myocardial infarction and stroke usually occur in asymptomatic, apparently healthy middle-aged or older people, identification of high-risk subjects, assessment of the atherosclerotic burden, and application of appropriate preventive strategies (e.g. improvement of plaque stability with lipid-lowering medication) during the pre-symptomatic detection of the disease, is considered a major challenge for the 21st century (Celermajer 1998; Grundy et al. 1999).

According to the criteria of the American Heart Association Committee on Vascular Lesions, coronary artery plaques and plaque progression can be subdivided into five phases of progression and six different lesion types (classified according to Stary; Stary et al. 1992). The pathogenesis of atherosclerosis is complex and a detailed description is beyond the scope of this chapter. In brief, the influx of lipids and/or accumulation into the vessel wall leads to remodelling of the arterial wall, which consists of fatty accumulation, and an increased number of macrophages (Fuster 1994). Type I–III lesions are mainly differentiated by their proportions of lipids, macrophages and smooth muscle cells and whether the lipid is intra- or extracellular. The potential for clinical problems, however, begins when the process of lipid influx and accumulation continues (Fig. 14.1). A type IV lesion, i.e. atheroma, has a predominance of extracellular lipid, mainly diffuse; and a type Va lesion, i.e. fibroatheroma, has a high lipid content, mainly localized, and a very thin capsule (Stary et al. 1995). Both lesions can evolve into the more fibrotic and stenotic types Vb (mainly calcified) and Vc (mainly fibrotic) lesions. However, both type IV and Va lesions also have a tendency for acute rupture with a change in their geometry, and subsequent thrombus formation may lead to the type VI, i.e. complicated, lesions (Fig. 14.1). Disruption-prone plaques in the coronary arteries, the so-called “vulnerable plaques” tend to have a thin fibrous cap (cap thickness 65–150 µm), a large lipid core (plaque type IV–Va), a high number of macrophages, and a low number of smooth muscle cells. These vulnerable plaques are often only modestly stenotic, and therefore angiographically silent.

Fig. 14.1. Evolution of coronary artery disease. (Adapted from Zaman et al. 2000). SMC, smooth muscle cells