

Editorial

Neoatherosclerosis

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Atherosclerosis is a complex process with long natural history that culminates in obstructive coronary artery disease [1]. Percutaneous coronary intervention with drug eluting stent (DES) has revolutionized the management of varying forms of acute coronary syndrome and stable coronary artery disease (CAD) [2]. Although the problem of in-stent restenosis (ISR) due to neointimal hyperplasia following bare metal stent (BMS) implantation was overcome by DES, its use has been plagued by late stent thrombosis and late ISR. In this context, autopsy studies by Nakazawa et al has shown onset of neoatherosclerosis within neointimal tissue following stenting and this finding provides novel insights into the mechanism of these complications [3].

Atherosclerosis in native coronary vessels is characterized by sub-intimal accumulation of smooth muscle cells, foamy lipid laden macrophages, extracellular matrix formation and angiogenesis. The plaques are capable of mineralization and are enclosed by fibrous cap. The histologic hallmark of atherosclerosis is presence of lipid laden foamy macrophages. The vulnerability of the plaque to rupture and initiate thrombosis is determined by several factors including lipid content and thickness of fibrous cap. Following coronary angioplasty and stent implantation, the stented segment which is exposed to vessel lumen undergoes endothelialization and formation of new intimal layer. Neoatherosclerosis is the development of atherosclerotic changes within the neointimal tissue that forms inside the stented segment of coronary artery. This process is more common and occurs as early as 4 months following DES implantation compared to BMS. Chronic and continuous neoatherosclerosis probably leads to late ISR and this explains the "late catch-up" phenomenon observed with DES. Interestingly, these neoatherosclerotic plaques are prone for rupture and thrombus formation that may manifest as late stent thrombosis. Such unstable neoatherosclerotic plaques are observed more commonly and occur at much earlier time interval following DES implantation than BMS. Evidence for these pathologic processes in-vivo has been provided by angiography, grey scale and virtual histology intra vascular ultrasound and optical coherence tomographic studies in acute coronary syndromes [4-6].

Unlike atherosclerosis in native coronary arteries which takes several decades to develop, in-stent neoatherosclerosis can occur as early as 4 months following implantation of sirolimus eluting stent [3]. The exact mechanism by which atherosclerosis occurs in accelerated manner following stenting is not known. It has been postulated that endothelialization of stented segment which is not only delayed but dysfunctional could be central to

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development of neoatherosclerosis [3]. The normal endothelial cells are resistant to entry of lipids and inflammatory mediators. Since the stented segment endothelium is dysfunctional, there is increased uptake of circulating lipids and possible upregulation of platelet endothelial cell adhesion molecule 1 (PECAM 1) which in turn facilitates uptake of inflammatory cell migration to subendothelial space. If endothelial dysfunction is the key step in pathogenesis of neoatherosclerosis, the advent of bioabsorbable scaffolds where the neoendothelium has intact vasomotor response and preserved function is likely to prevent onset of neoatherosclerosis and its complications [7].

CONFLICT OF INTEREST

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