Macrophages and Neovascularization in In-Stent Neoatherosclerosis: An Accelerated Inflammatory Phenotype by OCT with Therapeutic Implications

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Short Editorial related to the article: Inflammatory Phenotype by OCT Coronary Imaging: Specific Features Among De Novo Lesions, In-Stent Neointima, and In-Stent Neo-Atherosclerosis

In this edition of *Arquivos Brasileiros de Cardiologia*, Pinheiro et al.[1] show optical coherence tomography (OCT) data with clear differences in inflammation and neovascularization among de novo atherosclerosis, in-stent restenosis due to intimal hyperplasia, and in-stent neoatherosclerosis.

Patients undergoing percutaneous coronary intervention with stent implantation may have recurrent symptoms of coronary heart disease due to in-stent restenosis due to vascular injury that triggers an intimal proliferative response,[2] minimized by current techniques and stents of new generation.[3] However, the neointimal lesion secondary to a delay in neoendothelialization can lead to symptom recurrence, usually during the first year of coronary intervention.[4]

In patients with acute coronary syndromes, pronounced inflammatory responses can be detected for weeks,[5] contributing to plaque instability[6] and infarcted mass and ventricular remodeling after myocardial infarction.[7] In addition, stent implantation also promotes systemic and local inflammation.[8]

Russel Ross[9] defined atherosclerosis as an inflammatory disease.[9] Pinheiro et al.[1] reported increased inflammatory activity and neovascularization among de novo lesions and intra-stent neoatherosclerosis. These two forms of atherosclerosis may have important differences in pathophysiology. Atherosclerosis in native arteries is related to cardiovascular risk factors, and it takes a long time to develop, but after acute coronary syndromes and/or stent implantation, systemic inflammation may accelerate its progression.[6] Intra-stent neoatherosclerosis is a new and fast form of atherosclerosis-related to vascular injury and inflammation.[4]

Complications such as atherosclerotic plaque rupture seem to be associated not only with plaque expansion[10] but also with characteristics of greater vulnerability (inflammatory content, thin fibrous cap, and higher lipid content).[11] OCT has properly addressed all these aspects. The article's findings have important implications:

The need for appropriate therapy (including highly-effective lipid-lowering therapies) to prevent the development of atherosclerosis in native coronary arteries and possibly in the intra-stent atherosclerosis.[12,13]

The study raises the debate about the relevance of residual inflammatory risk and the opportunity to use anti-inflammatory drugs.[14,15]

More studies are necessary to understand better intra-stent neoatherosclerosis and the need for more comprehensive strategies, to prevent this form of stent failure.

Keywords

Coronary Restenosis; Phenotype/inflammation; Percutaneous Coronary Intervention; Atherosclerosis; Diagnostic, Imaging/methods.

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