Coronary stent infection can present as mycotic aneurysm or pseudaneurysm in as this case [1], myocardial abscess, and pericarditis with pericardial effusion [2]. Angiography and angioplasty-related bacteremia occur in <1% of patients [3]. However, in a prospective study assessing the frequency of bacteremia after cardiac catheterization, Banai and colleagues [4] found an incidence of 7.3% immediately after diagnostic catheterization and 4.6% immediately after percutaneous interventions (PCI). Four hours later, positive blood cultures occurred in 3.9% after diagnostic catheterization and in 4.1% after PCI. Drug-eluting stents (DES) may predispose more to infection because of their immunomodulating and antiproliferative effects. Impairment of local host defense mechanism and endothelialization of the stent struts might increase the susceptibility to infection [3].

Coronary aneurysms have been reported from three to four years after DES implantation. DES stents inhibit neo-intimal growth by eluting the drug locally, delay re-endothelialization [2], and further may influence the remodeling process and lead to late incomplete stent apposition. Although coronary aneurysms may develop as a result of exaggerated positive remodeling of the vessel wall, the underlying pathophysiology remains unknown. In some patients, this phenomenon has been linked to bacterial arthritis, or other rare predisposing factors such as Kawasaki disease [5]. Moreover, DES stents may aggravate inflammation and elicit hypersensitivity reactions leading to aneurysm formation. Thus, mechanical factors, such as residual dissections, arterial wall injury caused by oversized balloons and stents, high-pressure inflations, and atherectomies, complicated procedures, contained perforations, or even vessel ruptures have all been associated with early aneurysm formation (pseudaneurysms may actually develop) after PCI [2, 5]. Finally, it has also been suggested that DES related coronary aneurysms might predispose to DES thrombosis [5]. Of note, stent malapposition seems to be more frequent after DES than after bare-metal stent implantation, while factors such as DES implantation in acute coronary syndromes, long lesions, and chronic occlusions predispose to malapposition [3, 5]. Most studies suggest that late malapposition represents a pure IVUS finding without clinical symptoms, although incomplete apposition may indeed constitute a risk factor for late DES thrombosis [5].

After DES implantation, continuous clinical surveillance under prolonged dual antiplatelet therapy might be indicated, further supported by the possibility of spontaneous aneurysm resolution. Patients with larger total vessel areas and larger areas of malapposition have poorer prognosis. Therefore, the fate of the coronary aneurysm seems to depend on its size, and in this regard, IVUS appears to be superior to angiography. In cases of aneurysm thrombosis, aggressive approaches such as balloon over-inflation, coiling, covered stents, or further surgical excision might prevent further devastating complications. Regarding infected aneurysms, antibiotic therapy remains the mainstay of treatment, while it remains to be proven if early surgical intervention may result in an improved outcome in these patients. A radiolabeled leukocyte scan could be used early to confirm the diagnosis, if conventional imaging fails to identify the source of infection [3].

References


eComment: Coronary artery aneurysms after drug-eluting stent implantation

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