CLINICAL VIGNETTE

Vasculitis or coronary atherosclerosis? Optical coherence tomography images in polyarteritis nodosa

Maciej Lewandowski, Jarosław Gorący, Irmina Kossuth, Małgorzata Peregud-Pogorzelska

Department of Cardiology, Pomeranian Medical University, Szczecin, Poland

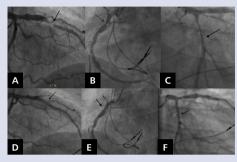


Figure 1. A–F. Endovascular treatment during acute coronary syndrome (arrows)



Figure 2. A, B. Treatment of stenosis of left anterior descending artery during follow-up (arrows)

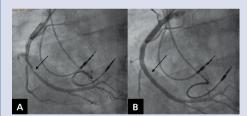


Figure 3. A, B. Treatment of stenosis of right coronary artery during follow-up (arrows)

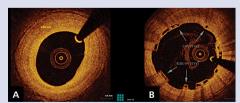


Figure 4. Optical coherence tomography images; **A**. Severe overgrowth of the intima in the mid-right coronary artery. A new lesion — not present four months earlier; **B**. Normal endothelisation of bioresorbable stent implanted four months earlier (arrows)

A 29-year-old man was admitted to hospital for assessment of the coronary artery disease and further diagnostic work-up. Medical history revealed cutaneous polyarteritis nodosa diagnosed at the age of 17 years, treated with low-dose glucocorticoids, and arterial hypertension treated with a β -blocker. He was a non-smoker and had no diabetes or hypercholesterolaemia. Eight months earlier he had a pacemaker implanted due to third-degree atrioventricular block. Four months after the implantation he was treated for acute coronary syndrome (ACS). Stenoses within the left anterior descending artery (LAD) (Fig. 1A, D) and the right coronary artery (RCA) (Fig. 1B, E) were treated with drug-eluting stents (DESs). In the subsequent two weeks a bioresorbable stent was implanted (in another hospital) to the left circumflex branch because of another ACS (Fig. 1C, F). Due to the presence of stenosis in the proximal segment of the LAD, a subsequent stage of invasive treatment was planned and intensive anti-inflammatory treatment was implemented (methylprednisolone and cyclophosphamide). During the hospital stay, the patient underwent coronary angiography and optical coherence tomography (OCT). The results of previous stent implantations were assessed as optimal. The previously described stenosis of the LAD was observed along with new stenosis in the mid-RCA (Fig. 2A, 3A). OCT confirmed significant stenosis in the LAD. Additionally, a marked intimal thickening was observed in the RCA without typical features of atherosclerotic plaque, including fibrous cap, calcifications, or lipid core (Fig. 4A). Previously implanted stents were partially covered with a thin layer of neointima (Fig. 4B). Two further DESs were implanted with optimal results (Fig. 2B, 3B). Intensive systemic therapy is currently being continued and no angina has been observed. The clinical importance of our case lies in the fact that clear criteria for a differentiation between atherosclerotic and inflammatory lesions have not been defined yet, and the images described herein have not yet been published. OCT revealed a lesion of the RCA, which is not typical of atherosclerosis. Intimal thickening reached over 500 μ m without typical features of plaques (Fig. 4A). The importance of systemic treatment in patients with all inflammatory or autoimmune disease must be emphasised. After a period of high disease activity (ACS, progression of lesions in the coronary arteries), aggressive systemic treatment slowed down the disease progression. The invasive treatment included multi-stage angioplasty (instead of bypass grafting), which is an accepted therapeutic option in such patients, although with limited experience. No restenosis or excessive numbers of uncovered struts were observed (Fig. 4B). The process of stent endothelialisation probably depends both on the appropriate stent implantation technique and on intensive systemic treatment. To summarise, the diagnosis and appropriate treatment of vasculitis and atherosclerosis remain a challenge and require the cooperation of the rheumatologist, cardiologist, and vascular medicine specialist. OCT can be a useful modality for the assessment and monitoring of the treatment process.

Address for correspondence:

Maciej Lewandowski, MD, PhD, Department of Cardiology, Pomeranian Medical University, al. Powstańców Wielkopolskich 72, 70–111 Szczecin, Poland, e-mail: malewandowski@tlen.pl

Conflict of interest: none declared

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