STUDIUM PRZYPADKU / CLINICAL VIGNETTE

Spontaneous coronary artery dissection as a cause of acute coronary syndrome

Samoistna dyssekcja tętnicy wieńcowej jako przyczyna ostrego zespołu wieńcowego

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Dissection is defined either as a split in the intima or the presence of intramural haematoma. The dissection of a coronary artery can, in rare cases, lead to acute coronary syndrome (ACS) or manifest itself as cardiac tamponade or cardiogenic shock. Spontaneous dissection occurs in 0.1–0.28% of patients with ACS undergoing coronary angiography. The diagnosis should be considered in young patients without risk factors of coronary artery disease, as well as in perinatal women or women using hormonal contraceptives. It can also occur in patients with congenital connective tissue disease, e.g. Marfan syndrome or after cocaine ingestion and prolonged physical effort. A patient aged 54 years without atherosclerosis risk factors was admitted to the department due to acute retrosternal pain. The patient had not been treated cardiologically, did not smoke, had a positive family history, and led an active lifestyle. Electrocardiogram test performed after admission revealed ST segment elevation in leads II, III, aVF, and $V_5 - V_6$ (Fig. 1). The patient received acetylsalicylic acid and clopidogrel, and was immediately qualified to invasive diagnostics of the coronary arteries. Coronary angiography revealed spontaneous dissection with the presence of a blood clot in the proximal segment of the left anterior descending (LAD) artery and another blood clot in the distal segment closing the artery. No abnormalities were demonstrated in the other epicardial arteries (Fig. 2). The patient was qualified to conservative treatment: abciximab was injected intravenously and a control coronary angiography after seven days was scheduled. Laboratory tests showed elevated troponin T max levels (724 ng/mL) without an increase in CPK-MB levels. Echocardiography showed hypokinesis of apical and medial segments of anterior and lateral walls, and ejection fraction of 50%. The control coronary angiography revealed no blood clot in the

proximal segment of the LAD artery, with smooth vessel outline and with TIMI-3 flow (Fig. 3). The patient was qualified to conservative treatment, whereas double antiplatelet therapy was continued. Pathogenesis of the coronary artery dissection is not completely determined. Maehara et al. [Am J Cardiol, 2002; 89: 466-468] and Venzetto et al. [Eur J Cardiothorac Surg, 2009; 35: 250–254] reported the presence of two types of coronary artery dissection: the first one is initiated by a breach of neointimal continuity and propagation of medial dissection (recognised both angiographically and in intravascular ultrasound), whereas the latter one results from the pathology of the arterial intima without communication to the vessel lumen (recognised only in intravascular ultrasound). Optimal treatment of spontaneous coronary artery dissection (SCAD) has not been determined and may vary, depending on dissection extension and clinical signs. In the present case study SCAD occurred in a male patient without risk factors of coronary artery disease, after sudden and intense emotional distress. As a result of pharmacological treatment clinical stabilisation was achieved. During 10 months of follow-up observation no recurrent symptoms of angina were reported in the patient. Coronary fibromuscular dysplasia is also considered as an essential process in the pathogenesis of SCAD.



Figure 1. Sinusoidal rhythm 70 min ST segment elevation in leads II, III, aVF, and $V_A - V_B$



Figure 2. Dissection in the proximal segment of the left anterior descending artery



Figure 3. Control coronary angiography after seven days

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