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Case Report

Syphilitic coronary artery ostial stenosis resulting in acute myocardial infarction

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ABSTRACT

Cardiovascular abnormalities are well-known manifestations of a late form of syphilis – tertiary syphilis. Since the era of antibiotics, the incidence of late manifestations of syphilis has declined almost to a rare entity. The injury of aorta (the aortitis with a dilatation of aortic root and its associated complications) is the most common between all the cardiovascular lesions. A less common manifestation of syphilitic aortitis is coronary artery ostial narrowing related to aortic wall thickening. We present the case of a 37-year-old male who was treated for an acute myocardial infarction due to bilateral coronary artery ostial stenosis secondary to syphilitic aortitis. According to the multidisciplinary decision, surgical revascularization (coronary artery bypass grafting, CABG) was performed. According to dermatologist recommendation, patient postoperative cardiovascular treatment was supplemented with intramuscular doses of benzathine penicillin recommended for tertiary syphilis. Further follow-up visits were also planned to detect possible changes of the aortic wall, dynamics of aortic regurgitation or potential anastomotic restenosis due to progression of aortitis.

1. Introduction

Syphilis is mainly sexually transmitted infection that may present with a variety of symptoms at various stages. In recent decades the incidence of late form of syphilis has declined owing to the early recognition of the disease and the sensitivity of the pathogen to antibiotics. Cardiovascular manifestation of tertiary syphilis predominantly involves the root of aorta, leading to the formation of aneurysm and aortic valve insufficiency [1]. In rare cases syphilitic aortitis can cause coronary artery ostial narrowing related to aortic wall thickening [2]. We present the case of a young male treated for an acute myocardial infarction with ST-segment elevation due to severe bilateral coronary ostial stenosis induced by syphilitic aortitis.

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2. Case presentation

2.1. Clinical presentation

An unemployed 37-year-old patient without previous medical history was admitted to the emergency department due to acute severe chest pain, shortness of breath and numbness of both hands. The patient complained of having exertional chest pain few times in a day for 1.5 months. These episodes were short and relatively not intense, so he did not visit any doctor and did not undergo any diagnostic procedures. The patient denied other comorbidities, usage of any drugs or previous cardiovascular diseases in his family. He was a long-term smoker, but had no other risk factors for coronary artery disease. Despite the adequate conservative treatment in the Department of General Internal medicine of regional hospital, chest pain lasted for 3 days and the level of troponin was increasing from 1.17 to 1.9 μg/mL (reference value, <0.04 μg/mL). Due to suspected acute coronary syndrome, the patient was transferred to the Cardiology Intensive Care Unit for coronary angiography and interventional treatment.

During the first physical examination, general condition of the patient was stable. The heart rhythm was regular with 76 beats/min. The blood pressure was 118/70 mmHg. Cardiac auscultation revealed gentle diastolic murmur, predominantly in the aortic area. Breathing sounds were clear. No other objective significant changes were found during physical examination.

2.2. Diagnostic tests

Total blood count, creatinine level, electrolytes, glycaemia, and coagulation parameters were within reference limits. Mild dyslipidemia was found (total cholesterol, 4.36 mmol/L; low-density cholesterol lipoprotein cholesterol (LDL-C), 2.6 mmol/L; high-density lipoprotein cholesterol (HDL-C), 1.22 mmol/L; triglyceride, 1.63 mmol/L; atherogenic coefficient, 2.57).

The electrocardiogram (ECG) showed sinus rhythm, slight ST-segment elevation in III, aVF, V1–V2 leads, ST-segment depression in aVL, V4–V6 leads and negative T waves in aVL lead (Fig. 1).

Chest X-ray showed an increased size of the heart and elongated aorta without lung infiltration or venostasis.

2D echocardiography revealed evidence of mildly dilatated proximal part of ascending aorta (43 mm) with a moderate regurgitation of aortic valve. The thickness of aortic walls was noted as a marker of inflammatory process. LV systolic function was normal (EF, 55%) with concentric LV remodeling (Fig. 2).

Coronary angiography clarified causes of chest pain: subocclusions of right and left main coronary arteries were found (Figs. 3 and 4).

2.3. Treatment

Clinical data, echocardiography and angiography results were evaluated by the heart team. The options of different revascularization strategies and optimal medical therapy

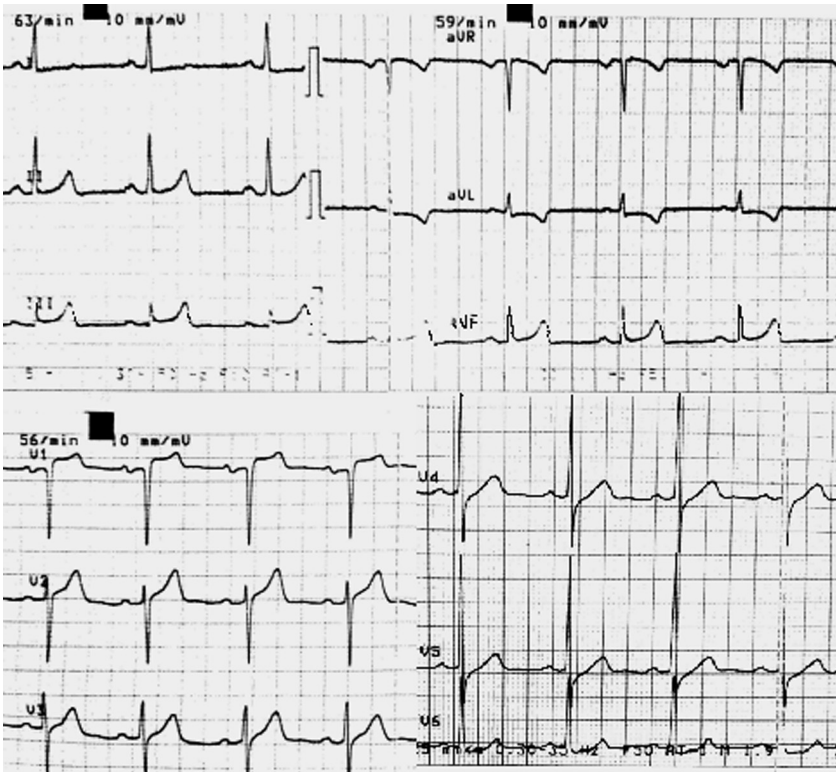

Fig. 1 – ECG with slight ST-segment elevation in III, aVF, V1–V2 leads, ST-segment depression in aVL, V4–V6 leads and negative T waves in aVL lead.

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