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CLINICAL CASE

A case of infective endocarditis after coronary stenting in myocardial infarction patients

Alekperov E.Z.*

Author:
Elman Zaur oglu Alekperov, MD
Senior Researcher of the Department of Myocardial Infarction of the Scientific-Research Institute of Cardiology named after J.Abdullayev, Baku, Azerbaijan

Summary

A clinical analysis was conducted of a patient who developed infective endocarditis after percutaneous coronary intervention with stenting of the right coronary artery. Despite a large amount of vegetation on the aortic valve (23 mm), it underwent a complete regression within 22 days. In addition, an interesting fact was the absence of prior aortic valve lesions, suggesting iatrogenic aortic insufficiency developed in this patient, and infective endocarditis.

Keywords

Coronary artery stenting, infective endocarditis, aortic valve

Infective endocarditis is a dangerous and severe inflammatory disease of the endocardium with a septic course. The disease most commonly affects the endocardium of modified valves and intracardiac defects of the heart. In addition, in the literature there are quite a lot of data on inflammatory changes of the endocardium in the area adjoining implantable endocardial electrodes [1]. Some interventional cardiologists believe that there is a possibility of developing bacterial endocarditis immediately after an invasive procedure, although there are limited conclusive data on this assumption in the available sources of literature [2]. There is some evidence that bacterial endocarditis involves unaffected heart valves [3]. In this regard, this case may be of some clinical and scientific interest.

Patient M, 61 years old, was admitted to the *Cardiology Department of the Central Hospital of Oilmen* (CHO) on 4 August 2012, complaining of fever, malaise, general weakness, pain in the lower back and left knee. Twenty years ago, during prophylactic examination in the *Scientific-Research Institute of Cardiology named after J.Abdullayev*, Baku, Azerbaijan, pathological changes in electrocardiogram (ECG) in the form of negative T waves (v1-v4) were found. The veloergometry conducted at the time had negative results, negative T waves in the ECG during exercise, which underwent reversion and became positive. The tolerance to the load was high, no subjective sensations during the test were observed. There were no echocardiographic signs of structural changes in the heart and valves. All that time, the patient main-

* Corresponding author. Tel: +99412 4653401 (work); +99450 3282456 (mobile). E-mail: azelman56@mail.ru

tained daily activity. His work had often been associated with physical exercise, without any discomfort in the heart. Two years ago, he first experienced a rise in blood pressure (BP) up to 180/100 mmHg. He was prescribed a fixed-dose maintenance therapy with lisinopril and hypothiazide after consultation at the *Scientific-Research Institute of Cardiology*. After this prescription, his condition was stable for a long time and BP readings remained within the target values. Once a year, the patient had a medical check-up in the *Outpatient Department of the CHO*.

Three months earlier, 11 May 2012, after emotional stress, the patient felt a searing pain in the chest, weakness, periodical cold sweat with an episode of short-term loss of consciousness. Due to the progressive deterioration, he was admitted to the *Cardiology Department of the CHO* with a diagnosis of developing acute lower myocardial infarction. X-ray examination showed a moderate expansion of the heart's shadow to the left. Echocardiogram revealed that systolic and diastolic dimensions and function of the left ventricle (LV) were normal; there was a moderate concentric LV hypertrophy. Laboratory tests detected the dynamics of troponin I going from 0.44 ng/mL to 10.19 ng/mL. Other indicators of the laboratory and instrumental methods were normal. During coronary angiography he was diagnosed with coronary subtotal occlusion of the right coronary artery, and stenting of infarct-related coronary artery was conducted. Three days later, he had a sub-febrile temperature, which normalized on the fourth day after intramuscular injection of ceftriaxone 1.0g per day. The patient was discharged from hospital in satisfactory condition a week after admission.

In spite of regular check-ups in the *Outpatient Department of the CHO*, after 1.5 months, 4 July 2012, his temperature started to rise, increasing to 38.8º C accompanied by strong chills. A total blood test showed increased erythrocyte sedimentation rate to 66 mm/h, C-protein levels to 39.8 mg/L (normal <3.1 mg/L), moderate monocytosis and anemia. Repeat blood cultures for sterility were negative. An ECG showed signs of myocardial infarction in the form of scarring of the lower wall of the LV myocardium. X-ray examination of the chest did not show any pathology, there was a slight enlargement of the heart to the left. An ultrasound examination of the internal organs: the liver, gallbladder, pancreas, urinary bladder and prostate showed that everything was in normal range; the kidneys had visible salt crystals. Echocardiography: systolic and diastolic dimensions and systolic function of LV were normal; ejection fraction - 60%; segmental function of LV walls – normal; aortic valve cusps had visible small single vegetation. The patient was re-admitted to the *CHO* with a diagnosis of subacute infective endocarditis. Despite the conducted forced comprehensive treatment with adequate doses of antibiotics, the vegetation on the aortic valve was progressively increasing and, by the 12th day, it reached 23 mm. This size of vegetation persisted up to 20 days, but then regression to 17 mm was recorded on day 25, 14 mm on day 28, and 7.5 mm on day 34 of the treatment (Figure 1). Contractile function of the myocardium and the size of the heart chambers during inpatient treatment remained within normal limits, although the echocardiography had signs of moderate aortic valve insufficiency of II degree. Echocardiography, performed on the 42nd day of inpatient treatment, showed a dense calcified area on the site of vegetation. The patient was discharged home in a satisfactory condition. Over two weeks he continued to take rifampicin and fluconazole. Currently, his condition is satisfactory and his ability to work has been completely restored. Ventricular extrasystoles of II graduation (according to Lown classification) are periodically observed. Hemodynamic signs of aortic valve insufficiency of II degree can still be seen on echocardiography. He takes regularly statins, aspirin, clopidogrel, ACE inhibitors, amiodarone, and has periodical check-ups in the Outpatient Departments of the *CHO* and *Scientific-Research Institute of Cardiology named after J.Abdullayev*.

The above data suggest that the patient has no previous signs of aortic insufficiency, as he repeatedly underwent complete preventive examination prior to the disease. According to the patient's history, he suffered from rheumatism in his youth, but the previous echocardiographic studies had not detected the presence of valvular heart disease, including aortic insufficiency. An interesting fact is that the LV dimensions remained in normal range, which is not typical for the long-term pre-aortic regurgitation; in other words, the problems emerged immediately after percutaneous coronary intervention (PCI). We can assume that PCI has contributed to traumatic injury of the aortic valve cusps and the appearance of moderate regurgitation of blood from the aorta, which was seen on echocardiography as aortic valve insufficiency of II degree. The genesis for the temperature (37-38°C), which appeared on the third day after PCI and lasted for 5 days, is not clear. The temperature became normal only after parenteral administration of the anti-

biotic. It is possible that during the procedure infection was primarily localized on the injured aortic valve cusp.

Thus, the analysis of this clinical case suggests the development of iatrogenic infective endocarditis. Perhaps such clinical situations require much closer analysis. If there is a need for an invasive intervention, interventional cardiologists should consider the potential for development of such complications.

References

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