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Case report

Native aortic valve thrombus as a source of embolisation into the coronary artery

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ABSTRACT

Authors present the case of 66-year-old patient after repeated systemic embolisations (lower extremity, axillary artery), admitted for inferior myocardial infarction. Coronary angiography demonstrated peripheral subtotal occlusion of posterior descending artery (PDA) of embolic origin. Transoesophageal echocardiography (TOE) revealed mobile mass on aortic valve, which was subsequently extirpated surgically. Histological examination described thrombus. Case report depicts the native aortic valve thrombus as a rare source of systemic or coronary embolisation. It simultaneously supports the indication of TOE at systemic embolisations of unknown source, even if transthoracic echocardiography (TTE) finding is normal, and shows its key role in diagnostic algorithm in similar events.

Introduction

Embolisation as a mechanism of myocardial infarction is well known [1,2]. Prizel et al. found autopsy evidence of infarction caused by emboli in 5% hearts, selected for post mortem coronary angiogram because of known or suspected history of cardiovascular or pulmonary disease (that was 13% of all autopsy-confirmed infarctions) [1]. Its sources can be endocarditis (infective, nonbacterial thrombotic), thrombi from left-sided heart cavities, valves (native, prosthetic), paradoxical embolism, tumors (myxoma, fibroelastoma), or heart catheterization or surgery [3]. Nevertheless the native aortic valve thrombosis as a source of embolisation into the coronary artery is uncommon.

Case report

Sixty six-year-old male with repeated systemic embolisation in his medical history, treated in other hospital: 2 years before current admission the acute occlusion of left low leg arteries, treated with partially successful local thrombolysis, and embolic occlusion of right axillary artery before 13 months, resolved by embolectomy. Subsequent hematologic examination for possible hypercoagulable state revealed just mild

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hyperhomocysteinemia. Patient was temporarily treated with warfarin. There were still hypertension, celiac disease and mild chronic sideropenic anemia, related to celiakia, in his medical history. No thrombembolic event occurred in family history. Chronic medication included hydrochlorothiazide and amiloride in fixed-dose combination, oral iron supplement, allopurinol, betahistine. Warfarin was withdrawn 5 months before admission.

Patient was presented to our center from the emergency department of community hospital with repeated chest pain, the latest 2 days before admission, and new Q waves in leads II, III, aVF in his electrocardiogram. On admission the patient's state was stable, he was without dyspnea, his blood pressure was 120/80, heart rate 84 min^{-1} and his lungs were clear within the auscultation, abdomen and lower extremities without abnormalities. The patient was referred for cardiac catheterization, coronary angiography demonstrated peripheral subtotal obstruction of PDA of embolic origin, only slight signs of atherosclerosis (Figs. 1 and 2). With regard to peripheral localization of thrombembolus, ECG signs of definite Q-infarction, clinical stabilization and absence of symptoms, the approach was conservative.

Laboratory tests showed microcytic, sideropenic anemia (hemoglobin 109...94 g/l), troponin I 25.401 ng/ml, iron level 3.4 μmol/l, renal insufficiency (kreatinin 162...149 μmol/l), total cholesterol 5.15 mmol/l, triacylglycerides 1.78 mmol/l, LDL-cholesterol 3.22 mmol/l, HDL-cholesterol 0.91 mmol/l. Transthoracic echocardiography reveals left ventricle ejection fraction 45%, inferior wall hypokinesis, mild aortic and tricuspidal insufficiency (trace regurgitation), mitral regurgitation I/IV. Chest X-ray was normal. Within search for a source of embolisation TOE was performed and showed echogenic mobile ovale mass (Fig. 3) attached by a stalk to the right coronary leaflet of aortic valve, 27×10 mm, with trace regurgitation. There was mitral regurgitation II/IV, left atrium including the appendage without clots. Our differential diagnosis of the mass included fibroelastoma, other type of tumor or thrombus. Vegetation was considered unlikely.

Additional examinations performed within the hospitalization: abdominal ultrasonography revealed a ''bright'' liver with increased echogenicity, chronic nephropathy on the left, fibrogastroscopy described sliding hiatus hernia, suspected gastroespohageal reflux and atrophic mucosa in duodenum (''mosaic'' pattern), suspected of celiakia.

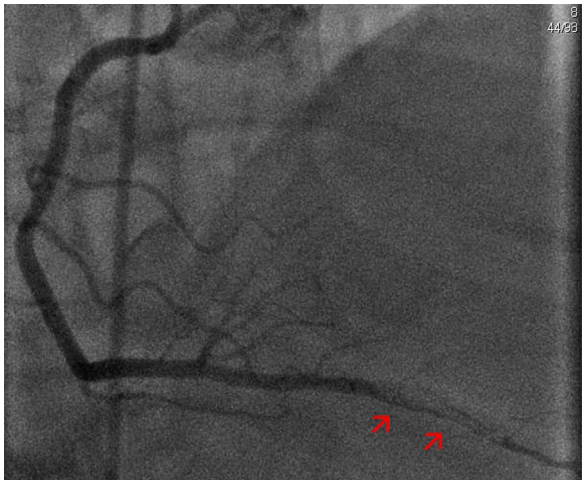

Fig. 1 – Right coronary artery with peripheral subtotal obstruction in PDA (arrows).

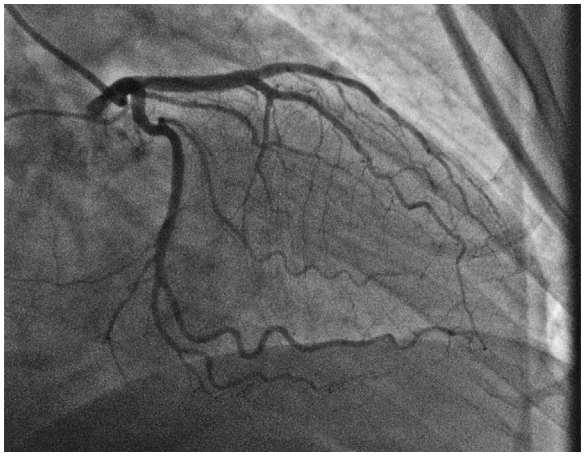

Fig. 2 – Left coronary artery.

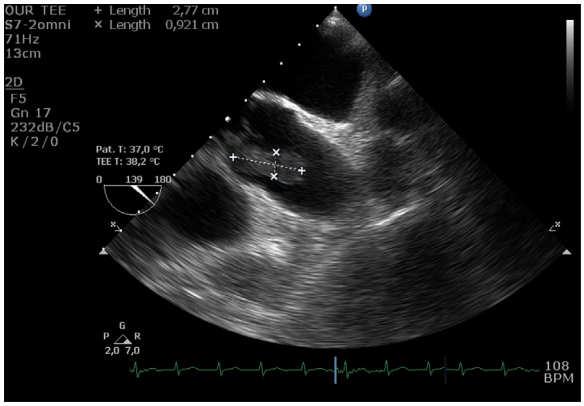

Fig. 3 – Transoesophageal finding of mass attached to the aortic valve.

The course of hospitalization, lasting 5 days, was uncomplicated, no further systemic embolisation occurred. Anticoagulation with low-molecular weight heparin was initiated, followed by warfarin in usual way.

4 weeks later patient underwent surgery: in cardiopulmonary bypass the mass of aortic valve was extirpated, macroscopically described as fragile, crumbly, jelly-like. Based on perioperative TOE, mitral valve anuloplasty was performed simultaneously. Histological finding depicts mixed, organized, mainly red thrombus (Fig. 4). Postoperative course was uneventful, patient was discharged on 10th day with warfarin, intended for lifelong. Currently (9 months after surgery) patient is doing well, managed by cardiologist and angiologist in outpatient clinics.

Discussion

The native aortic valve thrombus as a cause of peripheral embolisation, or more precisely myocardial infarction, is rare