Ruptured aneurysm of sinus of Valsalva – an unexpected cause of tachycardia and severe dyspnea in a young patient (RCD code: I-1B.0)

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Abstract

The presented case report illustrates a rare and unexpected cause of tachycardia and exercise tolerance reduction in a young patient with noncoronary sinus of Valsalva aneurysm rupture. The patient was referred to our hospital due to complaints of palpitation, dyspnoea and severe reduction of exercise tolerance of acute onset. Transthoracic echocardiography revealed a shunt between aorta and right atrium, moderate aortic and tricuspid regurgitation and signs of hyperkinetic circulation. Transesophageal echocardiogram confirmed the presence of ruptured aneurysm. The patient was consulted by a local multidisciplinary Heart Team and referred for cardiac surgery, after which he recovered without any complications. JRCD 2016; 3 (1): 17–19

Key words: rare disease, aortic disease, heart failure, echocardiography

Case presentation

A 37-year-old male with cardiovascular risk factors including uncontrolled arterial hypertension, smoking, family history of coronary artery disease, was admitted to the Cardiovascular Department due to severe dyspnea and palpitations. The symptoms started acutely, a day before admission and rapidly progressed to New York Heart Association (NYHA) class III. Until then, the patient had been physically active with good subjective sense of health. He denied chest pain, alcohol, caffeine and drugs abuse or chest trauma. Physical examination revealed body temperature of 36.7 Celsius degrees, regular pulse of 130 bpm, blood pressure of 130/60 mm Hg on both upper limbs, grade 3 continuous heart murmur best heard at Erb’s point, bilateral basal crepitations on lung auscultation, rest oxygen saturation of 96%, no peripheral edema or signs of bleeding. Laboratory results disclosed elevated level of high-sensitivity troponin (hsTn) up to 0.077 ng/ml (normal value <0.014 ng/ml), with creatine kinase and creatinine kinase-MB within normal limits. Hyperthyroidism or anemia were ruled out. Inflammatory parameters were not elevated. Electrocardiogram (ECG) revealed sinus tachycardia of 130 bpm, ST-segment elevation at J-point in leads V2-V3. Chest X-ray showed pulmonary venous congestion. Coronary angiography showed no stenotic lesions Transthoracic echocardiography demonstrated enlargement of the left atrium and left ventricle, left ventricular hypertrophy, moderate aortic and tricuspid regurgitations with right ventricular systolic pressure of 50 mmHg. Furthermore, the examination revealed a shunt between the ascending aorta and the right atrium (Figure 1A-C). Transesophageal echocardiogram revealed an aneurysm of the noncoronary sinus of Valsalva, 11 × 12 mm in size, with an adjacent movable structure (Figure 1D). Color Doppler imaging confirmed the presence of a fistula between ascending aorta and right atrium through the ruptured sinus of Valsalva aneurysm (Figure 1E). The patient was consulted with local Heart Team and was qualified for urgent surgical intervention. He underwent successful repair of the ruptured aneurysm with a use pericardial patch (Figure 1F). The patient recovery period was uneventful and was discharged home in good clinical condition, without dyspnea or palpitations. He remains in regular follow-up.
Sinus of Valsalva aneurysm (SVA), is a rare cardiac malformation occurring in approximately 0.09% of the general population with a male to female ratio of 4:1, accounting for 0.1% to 3.5% of all congenital cardiac defects [1]. Usually, it affects right or noncoronary sinus of Valsalva. It was first described by John Thurman in 1840 [2]. The pathomechanism of SVA formation is associated with a congenital deficiency in elastin tissue and abnormal bulbus cordis which can lead to a dissection between the aortic media and annulus fibrosus [1]. It is rarely a consequence of conditions affecting the aortic wall, such as infections (bacterial endocar-

**Discussion**

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ditis, tuberculosis or syphilis), chest trauma, atherosclerosis or connective tissue disorders [3,4]. Hemodynamic consequences of SVA can vary from asymptomatic to severe heart failure [3]. Even unruptured aneurysm, depending on the size, can lead to serious haemodynamic complications. It can obstruct left or right ventricle outflow tract and generate significant pressure gradient or complete heart block [1,5]. Consequences of ruptured SVA may include arrhythmias, aortic insufficiency, congestive heart failure or thromboembolic event. Rapture of an aneurysm often precipitates dramatic clinical complications and requires prompt diagnosis and treatment.

In our patient, presenting with acute onset and rapidly progressing heart failure, transthoracic and transesophageal echocardiography showed to play a definitive role in making correct diagnosis of a ruptured SVA. Decision regarding treatment was based on multidisciplinary Heart Team consultation. The patients was successfully operated and discharged home free of symptoms.

References