Case report

Moderate rheumatic mitral stenosis complicated by massive thrombus formation

Jana Čepelová a,*, Jan Škvařil a, Josef Bešík b, Tomáš Marek b, Eva Krčová a, Jan Pírk b, Martin Malý a

a Department of Cardiology, Internal Clinic of First Faculty of Medicine of Charles University and Military University Hospital, Prague, Czech Republic
b Cardiovascular Surgery Department, Institute for Clinical and Experimental Medicine, Prague, Czech Republic

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A B S T R A C T

Advanced rheumatic mitral stenosis with enlarged left atrium and atrial fibrillation can predispose to the formation of thrombi over time, if anticoagulation treatment is ineffective. We present the case of a 66-year-old woman with atrial fibrillation and syncope. Moderate rheumatic mitral stenosis was diagnosed by echocardiography and two huge thrombi were incidentally revealed within the enlarged left atrium. CT scan of the brain did not demonstrate cerebral ischemia. The patient underwent early mitral valve replacement surgery and extirpation of the thrombotic masses. A pacemaker was implanted because of symptomatic bradycardia.

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Case report

A 66-year-old woman with a history of rheumatic fever was admitted to the hospital for symptoms of heart failure, atrial fibrillation with ventricular bradycardia and a recent episode of syncope. The principal findings on the physical examination included harsh systolic and muffled diastolic precordial murmur, discreet pulmonary rales and edema of the lower limbs. Chest x-ray demonstrated cardiomegaly.

Transthoracic (Fig. 1) and consequently transesophageal (Fig. 2) echocardiography represented moderate mitral stenosis with thickening, sclerosis and fusion of the cusps, MVAi (PHT) was 0.71 cm²/m², Pg max/mean 8/3 mmHg (in balanced hemodynamic condition) and insignificant regurgitation was present. The left atrium was spheroidally remodeled and significantly enlarged (A4C 69×60 mm, LAVi 60.3 cm³/m²). The echocardiography revealed two huge egg-shaped formations, 26×42 mm and 22×30 mm respectively, with a wide base attached to the anterior and inferior sections of the atrial walls. Mean tricuspid regurgitation of non-rheumatic origin and systolic pulmonary hypertension (PASP 45 mmHg) were present, as were mild rheumatic aortic stenosis and regurgitation. AVAi was assessed 0.79 cm²/m², Pg max/mean 26/15 mmHg (Figs. 3 and 4).

The patient underwent early mitral valve replacement surgery using a stented bioprosthesis and extirpation of the two old, decorolated atrial thrombi. A pacemaker was
implanted because of atrial fibrillation with slow ventricular response. A CT scan of the brain did not demonstrate significant cerebral ischemia. One month after surgery, the patient’s course was uneventful, with favorable morphology and hemodynamic function of the bioprosthesis.

Discussion

The woman with incidentally revealing moderate rheumatic mitral stenosis and huge formations of thrombi, was primary hospitalised for heart failure and referenced for implantation of pacemaker because of atrial fibrillation with slow ventricular response.

Except for bradyarrhythmia, syncope could have been explained with intermittent occlusion of mitral valve orifice by thrombotic masses. Either cerebral stroke due to embolisation of thrombi should be taking into account, however CT scan of brain did not confirm significant cerebral ischemia.

Although actual state of anticoagulation was in therapeutic range (INR 2.6), we have not any long-term data of constant effective anticoagulation therapy. According to available data, only approximately 60% of patients on warfarin are effectively anticoagulated. Diuretics and ineffective warfarin therapy over a longer period of time could then facilitate the formation of thrombi in the left atrium.

Prothrombotic states had not been detected in this case.

Findings of thrombi are most likely associated with atrial fibrillation, mitral stenosis or left ventricular aneurysm. Apart from thrombi, the differential diagnosis of the masses in the

Fig. 1 – Transthoracic apical four-chamber view confirming the presence of a large thrombus within an enlarged left atrium. Note the thickening and calcification of the mitral and aortic valves.

Fig. 2 – Transesophageal image confirming the presence of a thrombus in the left atrium adjoining the aortic outflow tract, 41×27×25 mm in size.

Fig. 3 – Two perpendicular transesophageal views of the mitral valve. Note the thickening, fusion and calcification of the cusps limiting valve opening and resulting in moderate mitral stenosis. The thrombus is marked on the right side of the image.

Fig. 4 – Transesophageal color imaging of the mitral stenosis. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
cardiac chambers must take into account also vegetations and tumors. Vegetations may be seen in patients with infective endocarditis of the damaged native valves, prosthetic valves or cardiac devices. Cardiac tumors are rare (0.001–0.03%) and their clinical symptoms are often affected by location of the tumor rather than its histologic type [1,2]. However, the definitive etiology of cardiac masses can be determined only by histological analysis following after surgical extirpation.

Conclusion

Rheumatic mitral stenosis, even of moderate severity, associated with an enlarged left atrium and atrial fibrillation can be complicated by the formation of thrombi. Prevalence of left atrial thrombi in patients with ineffective anticoagulation therapy is as high as 30% [3] with possible obstruction of the outflow tract, cerebral or systemic embolism. Mitral valve replacement is necessary in most cases because of the damage incurred by the whole valvular system as a consequence of the rheumatic process [4–6].

Conflicts of interest

No conflict of interest.

Funding body

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Ethical statement

The research was done according to ethical standards.

Informed consent

The presented patient agreed with publication of her disease history.

References