Case Report

A Ball-Shaped Thrombus of the Tricuspid Valve after VSD Closure

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We report successful surgical management of a 26-year-old man with a ball-shaped thrombus of the tricuspid valve. He had been treated with prednisolone for IgA nephropathy and undergone surgical closure of an isolated ventricular septal defect (VSD). No symptoms, coagulative disorders, or pulmonary embolisms were found. Preoperative echocardiography showed a ball-shaped mass that had originated from the anterior leaflet of the tricuspid valve; it also revealed a small residual VSD. A histological examination revealed the mass to be an organized thrombus with no tumor components. This was a rare case of excision of an organized thrombus of the tricuspid valve. The findings suggest that the thrombus formation may have been associated with the small shunt and/or prednisolone. (Ann Thorac Cardiovasc Surg 2009; 15: 346–349)

Key words: tricuspid valve, thrombus, ventricular septal defect, prednisolone

Introduction

Cardiac valve tumors are rare, and most are asymptomatic and diagnosed incidentally. The most common histological type is papillary fibroelastoma. A tricuspid valve thrombus can mimic a tumor. We encountered an organized thrombus of the tricuspid valve mimicking a valve tumor after patch closure of a ventricular septal defect (VSD). A tricuspid valve thrombus is extremely rare without an associated coagulative disorder. We treated a patient with a ball-shaped thrombus of the tricuspid valve without any symptoms.

Case Report

A 26-year-old man was admitted to our hospital for follow-up after patch closure of a VSD with no symptoms. He had been treated with prednisolone for IgA nephropathy since he was 18 years of age. The renal biopsy revealed diffuse mesangial proliferation and advanced sclerosis. Therefore he was diagnosed as poor prognosis group. Two years previously he had undergone surgical closure of an isolated VSD, using standardized surgical techniques and postoperative care management. The postoperative course after the VSD closure was uneventful, hemodynamically insignificant, and required no medication; no endocarditis was noted. Postoperative periodic echocardiography showed a ball-shaped mass that originated from the anterior leaflet of the tricuspid valve and moved from the right atrium to the right ventricle with grade 1/4 tricuspid regurgitation (Fig. 1A). It also revealed a small residual VSD, but no other abnormal structures (Fig. 1B). The last echocardiography at 1 year after VSD closure showed only a small residual shunt and no intracardiac mass. Coagulability examinations showed the following normal levels: activated partial thromboplastin time 25.2 sec (normal range, 20–40), prothrombin time 13.6 sec (normal range, 10–14), and international normalized ratio (INR) 1.1. Further examination revealed a negative lupus anticoagulant (LA) and a low anticardiolipin antibody titer. There was no hypercoagulative state and no history

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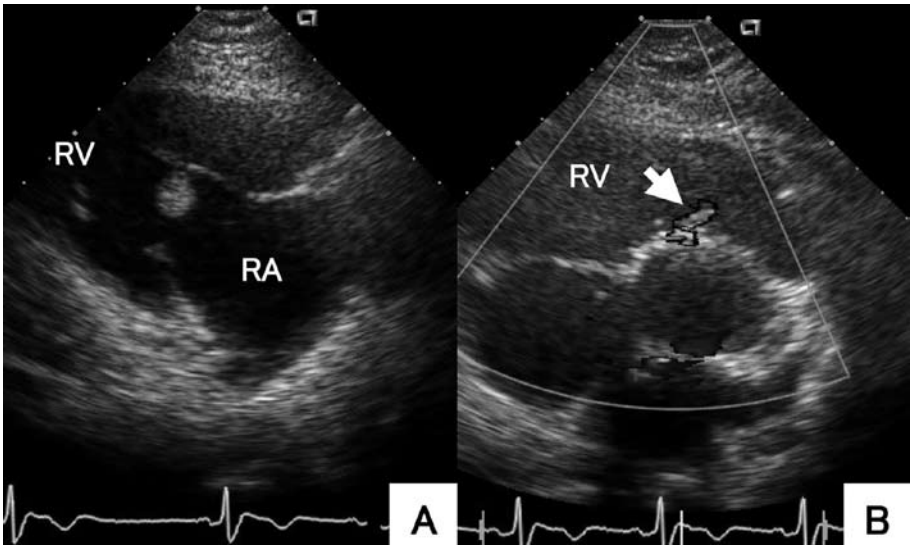

Fig. 1. Preoperative transthoracic echocardiography imaging.
A: A ball-shaped mass (15 mm in diameter) is attached to the anterior leaflet of the tricuspid valve.
B: A small residual ventricular septal defect (arrow) is present.
RA, right atrium; RV, right ventricle.

of deep vein thrombosis (DVT). We decided that surgical exploration and excision were indicated to rule out the diagnosis of a tumor and to prevent the occurrence of embolic complications. He underwent excision of the mass by use of a cardiopulmonary bypass. The pedunculated ball-shaped mass was attached by a stem to the anterior leaflet of the tricuspid valve (Fig. 2A). The mass was 15 mm in diameter, had a smooth reddish surface, and was elastically hard (Fig. 2B). It was easily resected completely from the anterior leaflet. There were no other abnormalities of the tricuspid valve. The leaflet of the tricuspid valve was not damaged, and repairing it was not necessary. Upon removal of the mass, a small residual VSD was apparent and was repaired. A histopathological examination revealed the mass to be an organized thrombus with no evidence of tumor cell components (Fig. 3). There was also no evidence of endocarditis in the specimen. Gram staining of the excised tissue and blood cultures showed no bacterial infection, and the patient's postoperative course was uneventful. Postoperative echocardiography demonstrated no tricuspid valve regurgitation, no residual shunt flow, and no recurrent thrombus formation.

Discussion

Thrombus formation on the tricuspid valve is rare. It can be categorized into three groups: that caused by coagulative and fibrinolysis disorders such as antiphospholipid syndrome (APS); that caused by a cardiac structural abnormality; and idiopathic thrombus formation in a structurally normal heart with no evidence of a coagulative disorder.[1] A tricuspid valve thrombus is most likely to occur with APS. Previous reports have described tricuspid valve thrombi in patients with APS, including one patient who presented with signs and symptoms of tricuspid stenosis.[2] Pierangeli et al.[3] reported that antiphospholipid antibodies in APS-activated endothelial cells create a hypercoagulative state. Our patient had a low anticardiolipin antibody titer and no clinical episodes of vascular thrombosis. However, he had been treated with prednisolone for IgA nephropathy, and the prednisolone may have contributed to the thrombus formation. There was no definitive evidence in our case, however, that steroids contributed to thrombus formation on the valve leaflet. Abnormalities of coagulation factors can promote thrombus formation on tricuspid valves in neonates.[4] Thrombus formation caused by a cardiac structural abnormality is exceedingly rare, and to the best of our knowledge only one case has been reported to date. Konishi et al. reported an organized thrombus attached to the tricuspid valve leaflet accompanied by a VSD and a tricuspid pouch. They suspected that stagnation of the blood flow around the pouch of a septal leaflet had caused the thrombus formation.[5] Although a tricuspid pouch was not revealed in our case, there may have been trivial stagnation of the blood flow because of the small residual shunt. Previous reports have

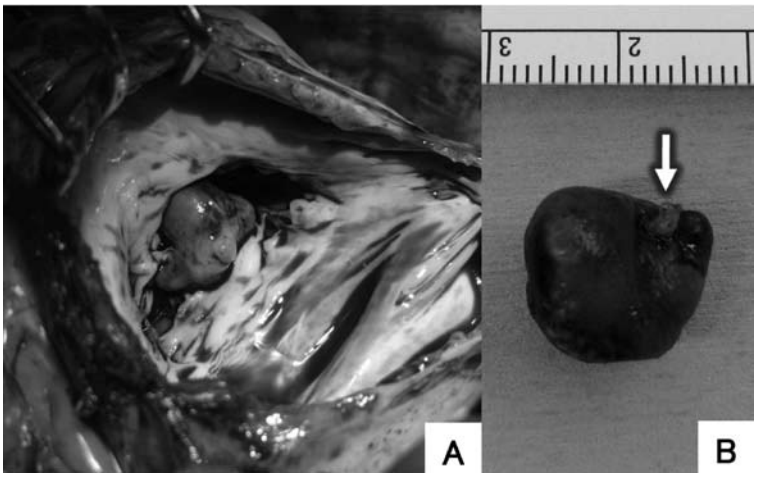

Fig. 2.
A: Intraoperative view showing the thrombus. The arrow marks the mass attached to the anterior leaflet of the tricuspid valve.
B: Surgical specimen of the ball-shaped thrombus, which was attached by a stem to the tricuspid valve (arrow).

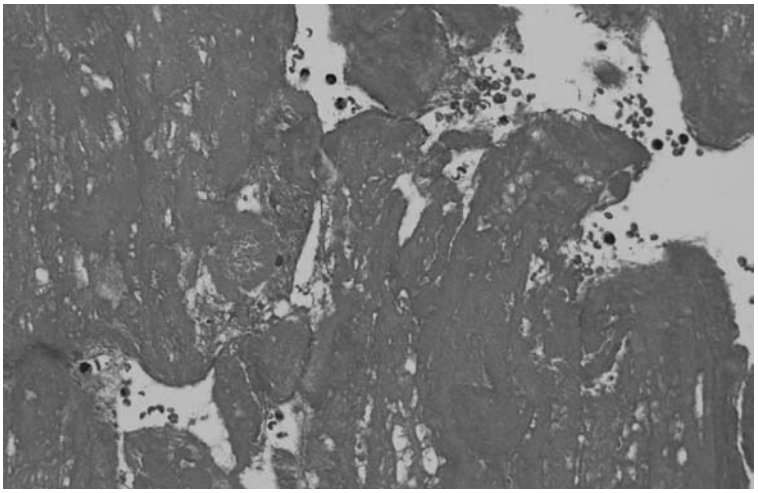

Fig. 3. Histopathological examination of the mass reveals an organized thrombus with no evidence of tumor cell components. (hematoxylin and eosin staining; magnification: ×40)

been made of a tricuspid valve thrombus in a structurally normal heart without an associated coagulative disorder.[1,6,7] All the authors of these reports assumed that the origin of the thrombus was a DVT. Preoperative computed tomography of the entire abdomen of our patient revealed no thrombus along the inferior vena cava or any tributaries. The reason for the attachment of the migrating thrombus to the tricuspid valve remains unclear.

The thrombus formation of our patient may be attributable to both the first and second groups. Additional conditions causing endothelial injury or a hypercoagulative state on the tricuspid valve surface would be necessary to trigger the attachment of a thrombus to the tricuspid valve. It seems that the natural fibrinolysis mechanisms failed to lyse the thromboemboli in our case for some unknown reason. A contributing factor may have been minor endothelial injury to the endocardium of the tricuspid valve. We can assume that the small residual shunt and prednisolone, together with transient tricuspid regurgitation, may have caused right atrial turbulence and

damage to the tricuspid valve, consequently helping the thrombus to grow bigger before being covered by the endothelium. Although thrombus on the tricuspid valve is exceptionally rare, it must certainly be added to the list of masses that can be found attached to the tricuspid valve. In conclusion, this report is the first case of a thrombus on the tricuspid valve involving a small residual VSD in a patient treated with prednisolone without coagulative disorder. However, the long-term outcome of this treatment remains unknown, and careful follow-up is required.

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