Iatrogenic thrombosis on the tendinous cords of the tricuspid valve

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Abstract Iatrogenic thrombosis within the heart is rare following intracardiac operations. We undertook surgery to correct a 30-month-old girl with Fallot's tetralogy. After a period of 6 months following the operation, echocardiography revealed a thrombus measuring 16 by 12 millimetres attached to the tendinous cords supporting the antero-superior leaflet of the tricuspid valve. Medical treatment having failed, we proceeded to surgical excision so as to avoid pulmonary embolisation. Histological examination confirmed the thrombotic nature of the mass.

Keywords: Cardiac operation; intracardiac thrombus; tetralogy of Fallot

THROMBOSIS WITHIN THE HEART IS RARE. Should it occur, then despite its rarity, it has many different potential aetiologies. We report here an unusual case of iatrogenic thrombosis on the tendinous cords supporting the antero-superior leaflet of the tricuspid valve subsequent to surgical correction of tetralogy of Fallot. We review potential causes of ventricular thrombosis, and discuss them in the light of our own findings.

Case report

A 30-months-old girl underwent total correction of tetralogy of Fallot. Preoperative echocardiography had shown a perimembranous ventricular septal defect opening to the inlet of the right ventricle with a diameter of 11 millimetres. There was a gradient of 25 millimetres of mercury across the right ventricular outflow tract, with an additionally stenotic pulmonary valve. The ventricular septal defect was closed with a Dacron patch, using separate pledgetted sutures. The subpulmonary outflow tract was enlarged with a transjunctional pericardial patch. The patient

was discharged on seventh postoperative day without any complications.

After a period of 6 months, however, echocardiography revealed a mass measuring 16 by 12 millimetres attached to the tendinous cords supporting the anterosuperior leaflet of the tricuspid valve. We started thrombolytic therapy promptly. Haematological examinations remained normal, but there was no evidence of regression of the mass. We proceeded, therefore, to surgical excision so as avoid the risk of embolisation. We entered the heart via a right atriotomy under cardiopulmonary bypass. The mass, measuring 15 by 10 millimetres (Fig. 1), was attached to the tendinous cords of the antero-superior leaflet (Fig. 2). It was resected cleanly from the cords. There was no postoperative complication, and the patient was discharged on the seventh postoperative day. Histological examination revealed subacute and chronic thrombosis, including calcification, within the mass, which did not contain any myocardial tissue.

Discussion

Involvement of a prosthetic valve is the most common reason for thrombosis in the right side of the heart. Yunus et al. 1 presented 2 patients with past histories of multiple cardiac operations who were given fibrinolytic therapy for thrombosis involving right

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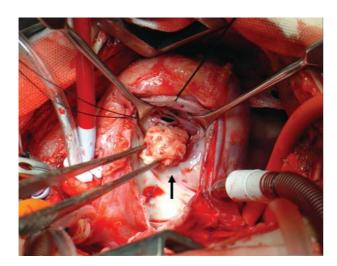


Figure 1.

The thrombotic mass (black arrow) is held with the surgical forceps.

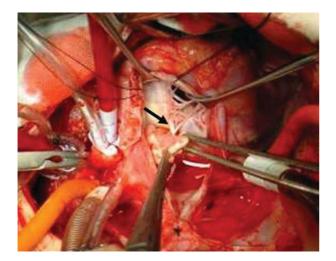


Figure 2. Pulling back the thrombotic mass with the forceps reveals its origin from the tendinous cords supporting the antero-superior leaflet of the tricuspid valve (black arrow).

heart structures. The first patient had thrombosis of a prosthetic pulmonary valve. The second patient, with tricuspid atresia, developed stenosis of the atrial pathways and right atrial thrombus subsequent to conversion to the Fontan circulation. Cases showing the benefit of thrombolytic therapy for thrombus on the right side of the heart, however, are limited.

Systemic diseases of the connective tissue, like Behcet's Syndrome, may cause thrombosis in the right atrium and ventricle. Kirali et al.² excised thrombus successfully from the right atrium and ventricle in such a setting with cardiopulmonary bypass, their patient having been unresponsive to medical treatment.²

Disruption of laminar flow, and stasis, both play a role in hypercoagulation. The entity of arrhythmogenic right ventricular cardiomyopathy, characterized pathologically by fibrofatty replacement primarily of the right ventricle, and clinically by life-threatening ventricular arrhythmias in apparently healthy young people, can also be associated with thrombosis.3 Congenital or acquired disorders of coagulation are predictors of thrombosis. In our patient, we evaluated the platelet count, proteins S and C, antithrombin III level, factor VIII, IX, XI, XII, factor V Leiden mutation, prothrombin potential genetic mutations, anticardiolipin antibodies, and levels of homocystein. All the haematological findings were normal. Blunt trauma to the chest is another rare cause of formation of thrombus in the right atrium and ventricle,⁵ but there was no suggestion of trauma in our patient.

We suppose, therefore, that iatrogenic injury of the endocardium following intracardiac surgery is the most probable reason for the formation of the thrombotic mass. Erentug et al.⁶ described formation of a right atrial thrombus subsequent to closure of an atrial septal defect that mimicked a cardiac tumour. In our patient, we presume that traction on the tendinous cords of the tricuspid valve and the subpulmonary infundibulum during exposure of ventricular septal defect may have caused endocardial destruction. Damage to the endocardial layer could then have triggered a thrombotic cascade following the corrective operation. This could well have been enhanced by the non-laminar flow through right ventricle and newly constructed subpulmonary outflow tract in the early postoperative period, exacerbated by the placement of a prosthetic Dacron patch secured with pledgetted sutures.

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