

Chronic Type A Dissection in a Pulmonary Autograft

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A 37-year-old patient presented with severe aortic valve insufficiency due to massive dilatation of the neo-aortic root (77 mm diameter) 14 years after a Ross procedure. Intraoperatively, the dilatation appeared to be caused by a localized chronic dissection of the pulmonary autograft.

In experienced hands, the Ross operation shows minimal perioperative complications. Although both the autograft and allograft have demonstrated limited durability, this has not yet resulted in considerable reoperation rates (1). In the long term, however, dilatation of the pulmonary autograft root may occur (2,3). Histopathological investigations have identified the presence of cystic medial necrosis, elastic fragmentation and findings indicative of chronic media rupture, which may have been the cause of progressive dilatation (4). Herein is reported a rare case of chronic type A dissection in a pulmonary autograft, identified 14 years after the initial Ross procedure.

Case report

In 1991, a 23-year-old man with severe insufficiency of a tricuspid aortic valve underwent elective aortic root replacement with the pulmonary autograft and implantation of a pulmonary allograft (24 mm) in the right ventricular outflow tract (i.e., the Ross procedure). The patient had suffered from rheumatic fever as a child. The postoperative period was uncomplicated, and pre-discharge echocardiography showed trivial neo-aortic valve insufficiency and pulmonary valve stenosis, with a peak pressure gradient of 18 mmHg.

At two years after surgery, the aortic root appeared thickened on echocardiography, and the diameter had

Surgery consisted of a modified Bentall procedure with a mechanical composite valve, with an uncomplicated postoperative course.

The Journal of Heart Valve Disease 2007;16:162-164

slightly dilated (38 mm). The trivial aortic valve insufficiency persisted, and the pulmonary valve stenosis peak pressure gradient remained at 19 mmHg (mean 12 mmHg). In 1998, the patient was still symptom-free, and echocardiography revealed identical findings as described previously; consequently, the patient withdrew from further medical attention.

In April 2005, the patient reported to a local hospital with acute palpitation, dyspnea and fatigue; he was examined but, without any conclusive diagnosis, was discharged with medical therapy. One month later, transthoracic echocardiography revealed a left ventricular end-diastolic dimension of 78 mm with massive aortic insufficiency, normal myocardial thickness, and contractility. The mechanism of the aortic insufficiency was not clear on echocardiography or subsequent angiography, though the latter technique showed an asymmetrically dilated proximal ascending aorta (Fig. 1A). Under the suspicion of severe autograft degeneration, the patient was referred to the present authors' institution. A repeat echocardiography showed severe left ventricular dilatation, good left ventricular function, and massive aortic insufficiency with a severe aortic root aneurysm (maximum diameter 77 mm).

Following preoperative screening and acquisition of consent, the patient was reoperated on in August 2005. During surgery, and following redo sternotomy and extensive adhesiolysis, the asymmetrically dilated aortic root was evident. The right posterior side of the aortic root and ascending aorta was densely adherent to the superior vena cava and right atrium. Therefore, cardiopulmonary bypass was instituted through the groin. The distal ascending aorta was cross-clamped at the base of the innominate artery, and an aortotomy

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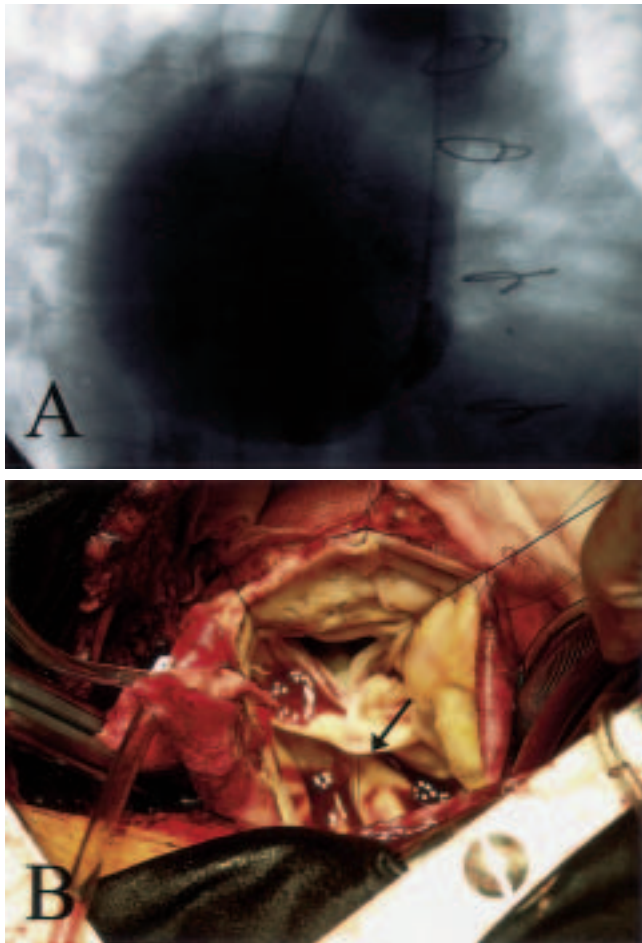


Figure 1: A) Aortic angiogram showing asymmetric root dilatation without a definite intimal flap. B) The aortic root as viewed from the operating surgeon. An intimal rim (arrow) is present in the pulmonary autograft.

performed. Myocardial arrest was achieved with antegrade Bleese cardioplegia until the septal temperature was 10°C. Upon inspection of the root, there appeared to be an intimal rim that commenced at the lateral side of the left coronary ostium and extended circumferentially approximately 1 cm distal to the non-coronary annulus (Fig. 1B). The distal anastomosis of the autograft, as well as the ascending aorta, was free of dissection and the aortic leaflets appeared normal. No valve-sparing procedure was performed as the patient had strictly requested the implantation of a mechanical aortic valve prosthesis. Therefore, a Bentall procedure was performed, re-implanting the left coronary ostium as an inlay (due to severe adhesions posteriorly) and the right coronary ostium as a button. The graft used as replacement was a mechanical composite of 29 mm diameter (Carboseal® Valsalva; CarboMedics Inc., Austin, Texas, USA). The patient was successfully and uneventfully weaned from the extracorporeal circulation.

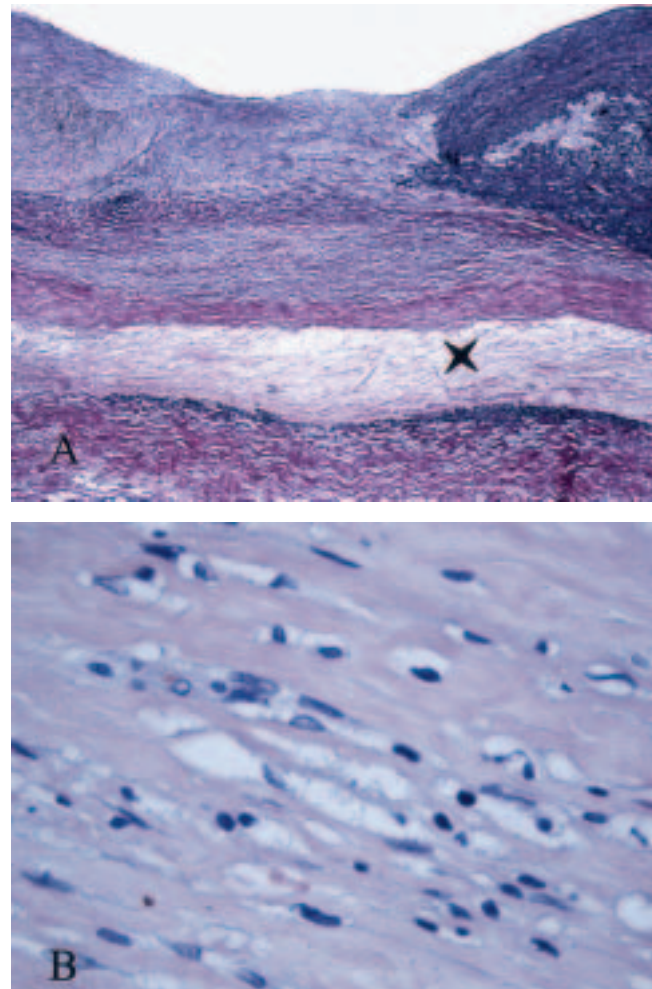


Figure 2: A) Histology of the resected pulmonary autograft root wall transition, showing a dissection (asterisk) in the lower media of the autograft. B) The autograft wall has marked degeneration of the elastic fibers of the media due to cystic medial necrosis, with deposition of mucopolysaccharide material. Staining: A) elastic van Gieson, original magnification $\times 50$; B) hematoxylin and eosin, original magnification $\times 400$.

Histologic examination of the explanted pulmonary autograft revealed degeneration of the elastic fibers of the media due to cystic medial necrosis (Fig. 2). There was a sparse chronic inflammatory infiltrate in the pulmonary autograft, and no acute infiltrate or fibrin deposition, which confirmed chronic pulmonary autograft dissection. The leaflets showed normal histological findings.

The hospital stay was uneventful and the patient discharged on day 7 after surgery. At a 16-month follow up examination the patient remained asymptomatic.

Discussion

Pulmonary autograft root dilatation is one of the most common late complications seen after the Ross procedure (2,3). Likewise, it is well known that the majority of patients with bicuspid aortic valves have histological abnormalities in the aortic and the pulmonary artery wall, such as cystic medial necrosis, elastic fragmentation and changes in smooth muscle cell orientation (4). In theory, a combination of pulmonary autograft dilatation and abnormal histology might lead to dissection within the autograft, but this long-term complication is rarely described. To the present authors' knowledge, only three reports have been made regarding dissection in a dilated pulmonary autograft with initially a bicuspid aortic valve (5-7). The present patient had a tricuspid aortic valve and had developed a chronic type A dissection in the dilated pulmonary autograft 14 years after the Ross procedure, with a histological finding of cystic medial necrosis and degeneration of the elastic fibers of the media. This proved that root dilatation and even dissection late after autograft root replacement is not only related to bicuspid aortic valve disease (which is supported by histopathologic studies (8,9)), but also to other variables such as operative technique and hemodynamic condition, which are associated factors for root dilatation following the Ross procedure.

The present case illustrates that aortic dissection may occur as a long-term complication in a pulmonary autograft, even with an initially tricuspid aortic valve. A high degree of suspicion is warranted in evaluating acute symptoms in a patient following the Ross procedure. Strict monitoring, for example with computed tomography, is advised to follow the dimensions of the autograft root. In view of the life-threatening aspect of this late complication, it is worth considering reoperation in an early dilated phase (prophylactic), as would occur in a patient with a collagen disease such as Marfan syndrome.

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