Accelerated aortic allograft fibrocalcification after right ventricular outflow tract reconstruction in pediatric patients: report of two cases

Abstract  Two cases are presented of accelerated aortic allograft fibrocalcification after right ventricular outflow tract (RVOT) reconstruction occurring within 2 months after surgery in a 5-year-old and 22-month-old. Potential determinants of early calcification, clinical management after implantation and surgical alternatives are discussed. [Eur J Cardiothorac Surg (1996) 10:290–293]

Key words  Allograft · Aortic valve · Pulmonary valve · Calcification · Immunogenicity

Introduction

Cryopreserved homografts have become the conduit of choice for ventricular outflow tract reconstruction in pediatric cardiac surgery worldwide. Despite their well-known superiority over bioprostheses and mechanical valves, recent reports have focused on homograft accelerated degeneration [5, 6]. Precocious allograft tissue failure has been identified as a troublesome phenomenon with a prevalence in the intermediate-term follow-up of recipients less than 3 years of age when an aortic valve homograft is used for left ventricular outflow tract reconstruction [5]. Homograft dysfunction for conduits utilized in the right ventricular outflow tract (RVOT) position has only occasionally been reported, with a prevalence in the intermediate-term follow-up of younger recipients (<18 months at operation) [3].

We report on two cases of accelerated aortic allograft fibrocalcification after RVOT reconstruction, which occurred within only 2 months after surgery in two children of 5 years and 22 months, respectively. Potential determinants of early calcification, clinical management after implantation and surgical alternatives are discussed.

Case 1

A 5-year-old, 14.2 kg, black African girl was admitted to our Institution with the diagnosis of mesocardia, viscerointestinal situs solitus, ventricular D-loop, double-outlet right ventricle with pulmonary stenosis, subaortic ventricular septal defect, straddling tricuspid valve, left juxtaposition of the right atrial appendage and persistent left superior vena cava.

At surgery, through a right ventriculotomy, the ventricular septal defect was repaired so that flow was directed from the left ventricle to the aorta by means of a Dacron patch whose implantation was achieved by partial mobilization and reattachment of the tricuspid chordal apparatus. The main pulmonary artery was divided and the proximal stump oversewn. The RVOT was reconstructed with an AB0 blood-type non-compatible 18 mm cryopreserved aortic homograft, displaced rightward to the aorta. The right pleural space was opened wide to provide additional space for the conduit. The right ventricular pressure was half systemic coming off cardiopulmonary bypass. The postoperative period was uneventful. She was discharged home with a 12 mmHg gradient through the homograft valve.

Two months after surgery, the child was again referred to our hospital for worsening dyspnea and fatigue associated with a febrile episode. A chest roentgenogram showed cardiomegaly and severe calcification of the RVOT homograft revealed by an eggshell profile (Fig. 1). Two-dimensional echocardiography showed partial detachment of the left ventricle-to-aorta patch in the presence of a minimal
bidirectional shunt and a severe stenosis at the valve level of the aortic homograft in the RVOT position, with a measured gradient of 70 mmHg. Cardiac catheterization measured a suprasystemic pressure in the right ventricle. Cultures were negative.

At reoperation, the aortic homograft appeared grossly calcified with retracted valve leaflets (Fig. 2 a, 2b). After conduit removal, the interventricular patch, partially detached inferiorly and posteriorly, was resutured and the RVOT reconstructed by means of a blood-type (AB0) compatible cryopreserved 18 mm pulmonary homograft. The chest was left open to prevent compression of the conduit in the presence of generalized edema. Sternal closure was successfully undertaken on the 2nd postoperative day. The patient’s recovery was uneventful. She was put on steroids for 6 weeks. At 9-month follow-up the patient was in NYHA class I with a 15 mmHg gradient across the pulmonary homograft with no evidence of primary tissue failure.

The explanted AB0-incompatible aortic homograft revealed retracted valve leaflets and mural calcification. Histological examination of the excised allograft tissue showed extensive calcification, intimal thickening and minimal mononuclear cell infiltrates (hematoxylin and eosin stain).

Case 2
A 22-month-old white boy was referred to us from another institution with a diagnosis of tetralogy of Fallot with pulmonary atresia, diminished confluent pulmonary arteries (Nakata index: 320 mm²/m²) [11], and a status-post right modified Blalock-Taussig shunt with a 4 mm Gore-Tex tube inserted at age 1 month. He underwent corrective operation with shunt take-down, ventricular septal defect closure with a Dacron patch and RVOT reconstruction by means of an AB0 blood-type non-compatible 18 mm cryopreserved aortic homograft to establish continuity with the pulmonary arteries. The postoperative period was uncomplicated. A chest roentgenogram at 2 months’ follow-up showed severe calcification of the homograft (Fig. 3) with echocardiographic evidence of distal conduit mild stenosis (systolic gradient of 20 mmHg). The patient is currently under close observation with serial echocardiographic assessment.

Discussion
Cryopreserved homografts have become the most widely used conduits for ventricular outflow reconstruction in children [4, 10]. Being rarely associated with thromboembolic events or infective endocarditis and having high surgical versatility, these conduits are preferred over bioprostheses and mechanical valves with their well-known long-term limitations, which are enhanced in the pediatric
population. Events of precocious allograft degeneration in children, however, have recently been reported. Clarke et al. [5] found a significant prevalence of accelerated aortic allograft fibrocalcification and valvular insufficiency in patients less than 3 years of age at initial replacement of the left ventricular outflow tract. Among ten long-term survivors in this age group, seven (70%) had progressive allograft calcification and insufficiency, six of whom (60%) required reoperation to replace the homograft within 3.7 years (mean 1.9 years) after primary conduit implantation.

Chan et al. [3] reported a frequent and progressive dysfunction of cryopreserved homografts used for RVOT reconstruction in children with a statistically significantly more rapid deterioration for those conduits implanted in younger recipient (<18 months), with a predicted progression towards severe regurgitation for 50% of the conduits implanted by 15.3 months postoperatively.

In the present two cases, accelerated degeneration with severe calcification of the aortic homograft in the RVOT position occurred within only 2 months after implantation. The first patient required urgent reoperation for a concomitant detachment of the interventricular patch, possibly due to an endocarditic process. The explanted conduit appeared completely calcified with retracted valve leaflets. The tunnel-shape of the proximal conduit revealed an underestimated sternal compression. The second patient developed homograft calcification with a 20 mmHg gradient at the distal anastomotic site. He is currently asymptomatic, under close observation.

Several potential determinants of such an accelerated degeneration have been investigated, regarding particularly tissue viability, immunogenicity and homograft storage procedures. A potential mechanism of allograft failure possibly involved in our cases was an immunologically mediated host response to the graft. The AB0-incompatible less antigenically active non-viable conduit (cryopreservation after 72 h) might represent the option of choice for young recipients. Possible an associated treatment with anti-inflammatory agents in the postoperative period might be indicated. Based on evidence of precocious calcification demonstrated for the aortic conduit [8], a pulmonary homograft should be preferred, particularly for RVOT reconstruction and when a substantially subsystemic intrahomograft pressure is to be expected [7]. This was the policy we adopted in the second operation for our first patient, with total freedom from tissue failure at a follow-up of 9 months.

In conclusion, the advantage of homograft implantation in infants has to be balanced with evidence of potential early calcification and failure requiring reintervention. The etiology of conduit precocious degeneration might be related to immune mechanisms. Based on our experience, the use of AB0-compatible less antigenically active non-viable pulmonary homografts for RVOT reconstruction in association with anti-inflammatory therapy in the postoperative period is advisable until further data become available.

References