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Brief Report

Replacement of the aortic valve after the arterial switch operation

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Abstract An 11-month-old infant had undergone a primary arterial switch operation, including the Lecompte maneuver, for correction of discordant ventriculo-arterial connections and closure of an accompanying ventricular septal defect.

At discharge, there were no signs of aortic valvar incompetence. Regurgitation across the aortic valve was detected first at the age of 2 years, and then increased progressively, as documented by serial echocardiographic studies. There had been no history of bacterial endocarditis. At the age of 10 years, echocardiography revealed severe aortic valvar incompetence. At operation, the aortic valve had three leaflets, all of which were short, with very restricted movement. Absence of sufficient leaflet tissue precluded a durable valvar reconstruction, so the aortic valve was replaced with a 21 mm mechanical prosthesis. Histological examination of the removed leaflets revealed nodular swelling due to mucous changes of the matrix, as well as fibrous alteration and formation of scar tissue, including areas of fibroblastic and capillary proliferation. There were no signs of calcification or acute inflammatory changes. Improvement of left ventricular function was observed both early postoperatively and later on. Our observation shows that aortic valvar incompetence after an arterial switch operation can be caused by degenerative changes of the neo-aortic leaflets, which prevent plastic reconstruction of the valve, necessitating valvar replacement.

Keywords: Neo-aortic valve; Jatene operation; valvar replacement

Repair or replacement of the new aortic valve after an arterial switch operation is rare. Only a few cases have been reported.[1–3] The mechanism for the incompetence is not clear. We report here replacement of the neoaortic valve 9 years after an arterial switch operation in a patient with severe valve incompetence and impaired ventricular function.

Case report

A 10-year-old boy was referred to our institute from abroad, where at the age of 11 months he had undergone a primary arterial switch operation, including the Lecompte maneuver, for correction of discordant ventriculo-arterial connections and closure of an accompanying ventricular septal defect. The septal defect had been closed through the right atrium using a synthetic patch. At operation, it was noted that the right and circumflex coronary arteries arose from the left hand facing sinus, while the anterior descending coronary artery took origin from the right hand facing sinus. Both aortic and pulmonary valves were normal. The coronary arteries were transferred using the trap-door technique. The neo-pulmonary arterial sinuses were reconstructed with autologous pericardium. The patient had been discharged 9 days postoperatively, without signs of aortic valvar incompetence, after an uneventful postoperative course. At the age of 2 years, however, aortic valvar incompetence was noted, which increased progressively as documented by serial echocardiographic studies. There had been no history of bacterial endocarditis.

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When seen by us, the boy was clinically asymptomatic, with a systemic arterial pressure of 110/32 mm of mercury. Echocardiography revealed severe aortic valvar incompetence, with limited movements of the valve leaflets. The orifice of the valve was seen to have a diameter of 23 mm, and the left ventricle was markedly dilated, with left ventricular end-diastolic and end-systolic dimensions of 66 and 45 mm, respectively. There was sufficient but impaired pump function, with a shortening fraction of 31%. There were no signs of obstruction within the left ventricular outflow tract, nor pulmonary arterial stenosis or incompetence. Cardiac catheterization and angiography validated the echocardiographic findings, and excluded stenosis and aneurysmal dilation of the ascending aorta. Both coronary arteries followed a regular course, without stenosis or distortion. Left ventricular end-diastolic pressure was measured at 4 mm of mercury, and left ventricular volume was calculated angiographically as 501 ml at end diastole, and 298 ml at end systole, with a resulting ejection fraction of 40%.

Because of the severe aortic valvar incompetence, the boy underwent surgery using moderate hypothermic cardiopulmonary bypass. The pulmonary trunk was transected at the previous anastomosis site. The neo-pulmonary valve, formerly the aortic valve, was tricuspid and competent, showing no macroscopic signs of degenerative change. The aorta was transected high, to prevent injury of the coronary arteries.

Crystalloid cardioplegic solution was injected directly into the orifices of the coronary arteries, which were widely patent. The aortic valve was tricuspid, but all three leaflets were short, with very restricted movement (Fig. 1). Reconstruction was attempted using ring plasty and suture reduction of the aortic sinuses, the aortotomy was closed, and the aortic clamp released. After restoration of cardiac function, transesophageal echocardiography showed significant residual aortic valve incompetence. The aortic clamp, therefore, was re-applied and the aortotomy reopened. After repeating the instillation of cardioplegia, the plasty sutures were removed and the aortic valve was replaced with a 21 mm mechanical prosthesis. The aortic incision was closed, and the pulmonary trunk was re-anastomosed using continuous suture. The aortic clamp was released and, after rewarming, cardiopulmonary bypass was discontinued without any catecholamine support. The period of aortic clamping had been 74 min, and the time spent on cardiopulmonary bypass was 142 min. The postoperative course was uneventful, ventilatory support being required for 16 h, and the patient was discharged from hospital on the 9th postoperative day on oral anticoagulation.

Histological examination of the removed leaflets of the aortic valve revealed nodular swelling due to mucous changes of the matrix, as well as fibrous alteration and formation of scar tissue, including areas of fibroblastic and capillary proliferation. There were no signs of calcification or acute inflammatory changes (Fig. 2).

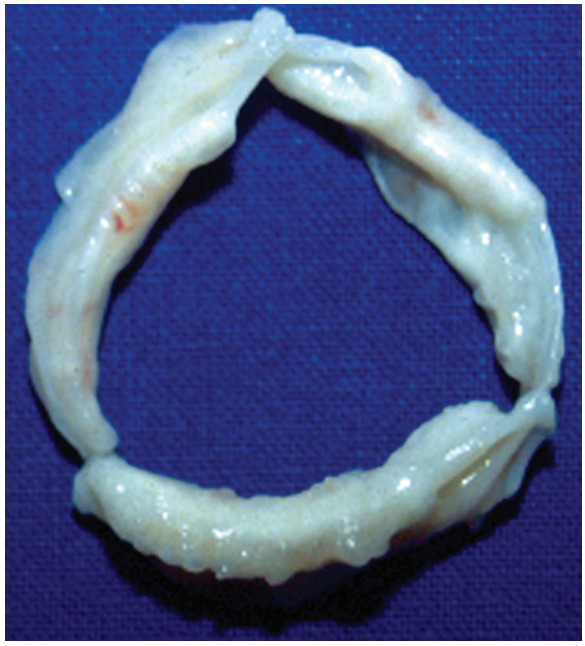

Figure 1.
Macroscopic view of the resected leaflets of the aortic valve. Note the significant shortening, and fibrotic changes, of all three leaflets.

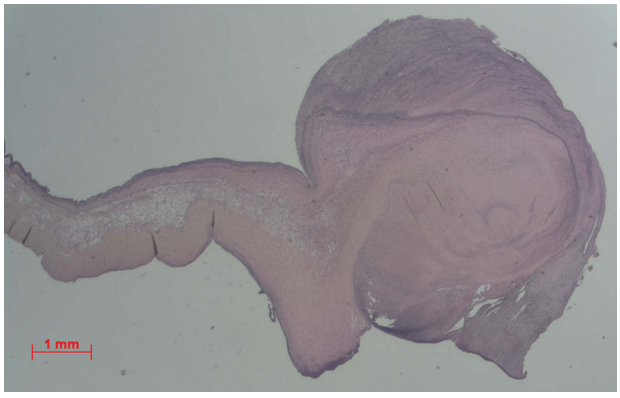

Figure 2.
Histological section of one of the leaflets, showing nodular scars and no signs of inflammation. Magnification of 12.5, with the Elastic-van Gieson stain.

Echocardiography on the 10th postoperative day showed the left ventricle to be still dilated, but smaller than preoperatively, with left ventricular end diastolic dimension of 45 mm, end systolic dimension of 38 mm, and shortening fraction of 19%.

The patient was last seen in our outpatient department 12 months after the operation. His blood pressure was then 110/70 mm of mercury, left ventricular

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end-diastolic dimension was 46 mm, left ventricular end-systolic dimension was 32 mm, with an ejection fraction of 65% and shortening fraction of 30%.

Discussion

Mild or moderate aortic valvar incompetence after an arterial switch operation is reported to occur in up to one-third of cases.[4,5] Possible explanations are previous banding of the pulmonary trunk, use of the trapdoor technique for transfer of the coronary arteries, closure of the ventricular septal defect through the pulmonary valve, iatrogenic injury of the aortic valvar leaflets, dilation of the non-coronary sinus of Valsalva of the new aortic valve with prolapse of the corresponding leaflet, turbulence on the sites of aortic and coronary arterial anastomosis, lack of subvalvular support for an overriding arterial valve, and dilation of the orifice of the new aorta.[4,6–8] It has been suggested that such incompetence can be avoided by achieving a single-stage neonatal repair, placating the noncoronary sinus of Valsalva when there is significant mismatch between the neo-artic root and the distal aorta.[2] Only a few reports have mentioned surgical treatment of severe aortic incompetence. Suggested options have included comissuroplasty combined with annuloplasty in cases of a dilated aortic orifice with poor coaptation of the leaflets,[2] repair of the valve,[9] or the Ross operation.[7] Because of the low incidence of severe incompetence, reported at less than 1%, valvar replacement is rarely necessary.[1,2,7] In our case, the exact mechanism of the severe valvar incompetence is unclear. One of the reasons may be a subtle inherent abnormality of the pulmonary valve that became significant when the valve was required to support the systemic arterial pressure load.[3] Significant shortening of all three aortic leaflets, with mycotic changes in the matrix, indicated a degenerative process without any signs of endocarditis.

Absence of sufficient valvar tissue precluded a durable valve reconstruction, so that valve replacement was inevitable. An autograft replacement was not considered a choice because of significant mismatch and the unclear prognosis of the patient. Improvement of left ventricular function was observed both early postoperatively and later on. Our case shows that aortic valvar incompetence after an arterial switch operation can be caused by degenerative changes of the neo-aortic leaflets, which prevent plastic reconstruction of the valve, necessitating valvear replacement.

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