Case report
Successful reoperation for severe left bronchus compression after repair of persistent truncus arteriosus with interrupted aortic arch

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Abstract
Signs of the left bronchus compression, caused by aneurysmatic dilatation of the aortic root with severe aortic regurgitation, occurred 5 months after repair of the truncus arteriosus with interrupted aortic arch in an 85-day-old infant. At reoperation the dilated ascending aorta was replaced with a 14-mm Dacron tube. The aortic valve was replaced with an 18-mm Carbomedics valve. Compression of the left bronchus and the right pulmonary artery were released. The right pulmonary artery was enlarged with a pericardial patch and the original homograft was replaced with a new one. The patient remains in good clinical condition 2 years later. © 1998 Elsevier Science B.V. All rights reserved.

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1. Introduction
The left bronchus compression represents an infrequent dangerous complication occurring after correction of congenital heart lesions [1–4]. The causes are not always clear and the management may be difficult.

2. Case report
A 78-day-old infant was referred to the center with the diagnosis of persistent truncus arteriosus (PTA) for repair. On admission, the patient was tachypneic with tachycardia and had a systolic murmur. Cardiomegaly and signs of increased pulmonary blood flow were demonstrated by X-ray examination. ECHO proved the diagnosis of type II PTA but demonstrated also type B interrupted aortic arch (IAA). The truncus was huge with a dysplastic, quadricuspid and moderately regurgitant valve. Catheterization and angiocardiology confirmed the diagnosis.

The repair was done when the infant was 85 days old. It was performed from the midline sternotomy approach, in extracorporeal circulation and cardioplegia. Hypothermic circulatory arrest was used for aortic arch reconstruction [5]. The truncus was 25 mm in diameter and the bayonet-like ascending aorta was 7 mm. The right and the left pulmonary arteries arose posteriorly close to each other. Both subclavian arteries took off from the descending aorta. The repair consisted of: (a) resection of PDA and both subclavian arteries; (b) reconstruction of the aortic arch by direct anastomosis between the descending and the ascending aorta; (c) closure of VSD with a Dacron patch; and (d) reconstruction of the right ventricular-to-pulmonary artery continuity using a 14-mm pulmonary homograft (Fig. 1).
The child was discharged on the 20th postoperative day. Good ventricular function, moderate aortic regurgitation (AR) and mild right pulmonary artery stenosis (RPAS) with a gradient of 30 mmHg were found on echocardiography.

The patient was readmitted with: (a) dyspnea; (b) tachypnea; and (c) heart failure, 5 months after correction. On X-ray, cardiomegaly and signs of pulmonary congestion were seen. Echocardiographic examination proved: (i) severe AR; (ii) small residual VSD; (iii) severe RPAS; and (iv) pulmonary regurgitation. There was no gradient at the aortic anastomosis. Heart catheterization revealed small left-to-right shunt (0.20 $Q_p$), RPAS (56 mmHg) and pulmonary hypertension (mean 28 mmHg). Angiocardiology with bronchography showed extreme aortic root dilatation (35 mm in diameter) causing RPAS and the left bronchus compression, which was confirmed by tracheobronchoscopy.

Reoperation was performed from the original midline sternotomy in extracorporeal circulation, 6 months after the repair. Aneurysm of the aortic root was resected and compression of the left bronchus and the right pulmonary artery released. Dysplastic aortic valve was replaced with a Carbomedics (18-mm) bileaflet valve. The ascending aorta was substituted with a 14-mm Dacron tube. The VSD was closed with a mattress stitch. The right pulmonary artery was enlarged with a pericardial patch. The pulmonary homograft with incompetent valve was replaced with a 19-mm aortic homograft (Fig. 2). Histology of the ascending aorta did not explain its dilatation and unfortunately, the explanted pulmonary homograft was not sent for histological examination.

The postoperative course was complicated by bleeding necessitating revision. Ventilation was necessary for 8 days. Echocardiography proved a good result of reoperation and the patient was discharged 37 days after surgery. The patient remains in a very good condition, 24 months after reoperation.

3. Discussion

Compression of the left main bronchus represents a serious complication which may occur early or late after successful repair of IAA and other heart defects. Sakai and other authors observed development of the left bronchus compression after IAA repair [4,6] but most often in patients who had a combination of IAA with PTA [7,8]. Reoperation necessitated an extensive mobilization of the aortic arch with graft interposition and/or angiopexy [4,6–8].

Causes of the left bronchus obstruction may be different. Residual lesions may result in enlargement of the aortic root, pulmonary trunk or left atrium which can directly compress the bronchus. Aortic arch tension after inadequate mobilization and lower placement of the anastomosis represents the most common cause of airway obstruction after repair of IAA [4,6–8]. Adhesions can also contribute to the development of this complication. Implantation of a right-ventricular-to-pulmonary artery conduit increases the risk of the left bronchus compression by enlargement of the mediastinal space occupied by heart, great vessels and other mediastinal masses and increase of the intrathoracic pressure.

In this patient with PTA and IAA, dysplastic truncal valve caused regurgitation associated with important dilatation of PTA preoperatively, at the age of 85 days. Dilatation of the aortic root in comparison with the ascending aorta was influenced by the fact that blood
flow was predominantly shifted to the pulmonary artery and to the descending aorta across large PDA and that both subclavian arteries arose from the descending aorta. Quick progression of AR after the repair with secondary aortic aneurysm formation could be accelerated by the small ascending aorta; though no gradient was proved. Mild RPAS, which occurs sometimes after correction of PTA [9], was found by ECHO soon after the repair. With time the RPAS progressed. A 40-mm-long segment of stenotic right pulmonary artery passing behind the aortic aneurysm was found at re-catheterization. Aortic aneurysm represented evidently the most important factor in the development of both, RPAS and the left bronchus compression. This was well demonstrated by angiocardiography combined with tracheobronchography. Any role of direct aortic anastomosis was not proved.

RPAS and the left bronchus obstruction could have, however, more complex nature. The small residual subpulmonary VSD could increase the right ventricular and pulmonary artery pressure with enlargement of the pulmonary homograft—especially, when the right pulmonary artery was compressed behind the aorta. Hemodynamic changes caused cardiomegaly increasing intrathoracic pressure with subsequent progression of RPAS and the left bronchus obstruction.

In contrast to aortic homografts, enlargement of the pulmonary homografts in systemic pressure was already observed under both, laboratory and clinical conditions [9,10]. This is explained by different pulmonary and aortic wall structure and non-existent pulmonary annulus preventing dilatation. Surprisingly, however, in this patient the pulmonary homograft dilated under pressure lower than 80% of the systemic pressure.

The complex situation in this child required an extensive surgical intervention, based on: (a) resection of the aneurysm; (b) aortic valve replacement; and (c) reconstruction of the right pulmonary artery, which resolved the airway obstruction.

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References

