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Recurrent rapid homograft degeneration and extensive calcification

Tekrarlayan hızlı homogreft dejenerasyonu ve yaygın kalsifikasyonu

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Right ventricular outflow tract (RVOT) reconstruction with a homograft conduit is performed in patients with congenital heart disease such as pulmonary atresia and truncus arteriosus.[1] Despite favorable short-term results of homograft conduits, calcification in the graft wall and valvular thickening, leading to conduit degeneration, stenosis, and eventually failure, can be inevitable in the mid- and long-term.[2] In the majority of patients, early intervention is directly or indirectly associated with the mismatched homografts and pulmonary valve or with the surgical technique; however, early degeneration can occur depending on the immunohistological properties of the conduit.[3] In pediatric cases, surgical interventions using conduits for congenital heart disease have been increasingly used in the presence of conditions such as calcification which promote conduit failure.[3,4]

A three-year-old girl was admitted to our clinic with complaints of respiratory distress and dyspnea. Her medical history revealed a total surgical correction due to type 1 truncus arteriosus in the neonatal period in an external center with a 14 mm pulmonary homograft for the right ventricle-pulmonary artery continuity. One year after the initial operation, the patient underwent redo surgery for severe pulmonary valve stenosis and left pulmonary artery stenosis related to homograft degeneration. The left pulmonary artery was repaired using a pericardial xenograft patch and the right ventricle-pulmonary artery continuity was maintained using a 17 mm aortic homograft. Chest X-ray showed diffuse calcification of the pulmonary conus (Figure 1a). Echocardiography revealed a peak systolic gradient of 80 mmHg at the pulmonary valve level with Grad 3 pulmonary valve regurgitation. Contrast-enhanced computed tomography for further evaluation demonstrated an aneurysmal dilatation of the RVOT, diffuse and tubular calcification of the homograft, and severe stenosis of the pulmonary bifurcation (near-complete stenosis of the left pulmonary artery outflow tract

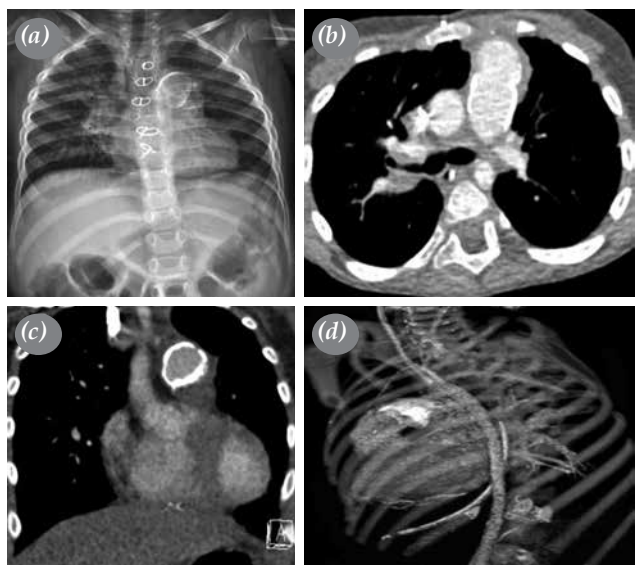

Figure 1. (a) Chest X-ray showing diffuse calcification of right ventricular outflow tract in a patient with type 1 truncus arteriosus. **(b-d)** Contrast-enhanced computed tomography showing calcification of right ventricular outflow tract in multiple slides.

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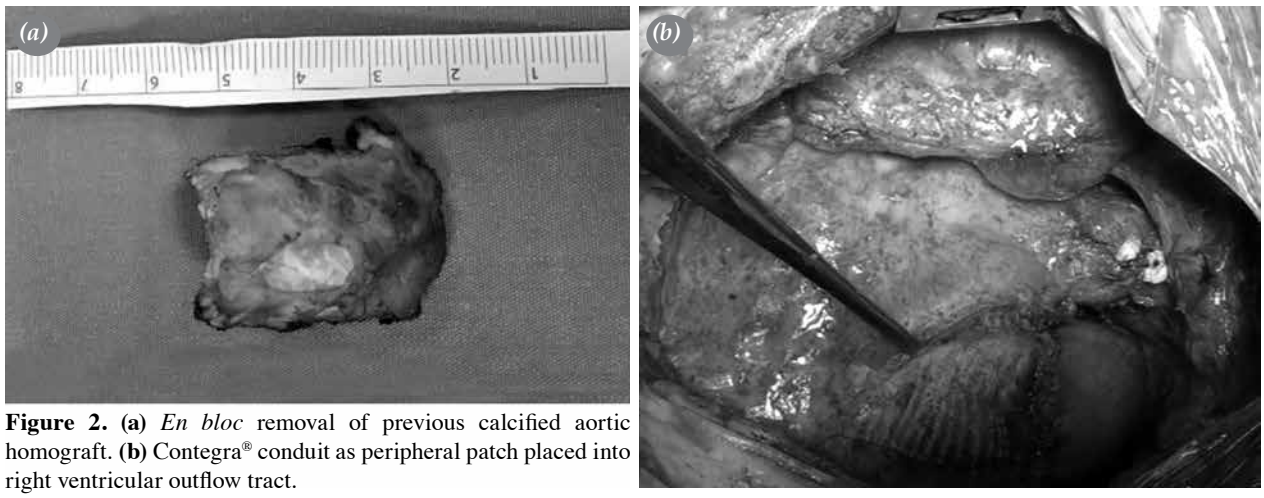

Figure 2. (a) *En bloc* removal of previous calcified aortic homograft. **(b)** Contegra® conduit as peripheral patch placed into right ventricular outflow tract.

(Figure 1b-d). No metabolic or renal pathology promoting calcification was observed. Based on these findings, the patient was scheduled for surgery. A written informed consent was obtained from each parent. The calcified homograft and the RVOT aneurysm were excised (Figure 2a) and the left pulmonary artery stenosis was treated with a pericardial patch. A 14 mm bovine jugular vein valved conduit (Contegra®, Medtronic Inc., Minneapolis, MN, USA), compatible with the pulmonary artery size of the patient, was placed into the repaired bifurcation (Figure 2b). The postoperative period was uneventful, and the patient was discharged in the postoperative fifth day with full recovery.

In conclusion, although early homograft conduit degeneration is rare, conduit replacement, particularly in the infancy and early childhood period, may lead to reduced durability of the conduit due to immunological rejection, thereby promoting the progression to conduit failure. Hence, severe and *en bloc* calcifications should be surgically treated in patients with the diagnosis of conduit failure.

Declaration of conflicting interests

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