Constrictive pericarditis following open-heart surgery in a child

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ABSTRACT

A 6-year- old child developed constrictive pericarditis 2 years after undergoing an open-heart surgery for a congenital cardiac disorder. No other cause of pericarditis was identified. The clinical condition improved after pericardiectomy. The case is reported for its rarity.

Keywords: Constrictive pericarditis, heart surgery, postoperative

INTRODUCTION

Postoperative constrictive pericarditis (POCP) is a unique entity. It has mainly been reported in adults after coronary artery bypass graft surgery. Only four cases of POCP have been described in children. Diagnosis may be difficult and delayed, especially in the presence of an underlying cardiac lesion. Accordingly, we report a child who developed POCP after surgery for congenital heart disease.

CASE REPORT

A 3-year-old girl presented with cyanosis. On examination heart rate of 130/min, blood pressure of 100/64 mmHg, and arterial oxygen saturation of 81% were noted. Echocardiography revealed an unbalanced atrioventricular septal defect with double outlet right ventricle and left atrioventricular valve stenosis. The ostium primum atrial septal defect was tiny and restrictive. The ventricular septal defect was very large and biventricular repair was not feasible. A patent ductus arteriosus was also present. Cardiac catheterization showed mean pulmonary artery pressure of 77 mmHg with pulmonary artery wedge pressure of 37 mmHg. Atrial septectomy and ligation of the patent ductus were done along with pulmonary artery banding with 34 mm Mersilene. The postoperative course was uneventful.

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Two years later, the child presented with massive ascites with pedal edema and facial puffiness. There was no history of fever, recurrent chest pain, or any other systemic illness during this time. The ascites was gradually progressive. On physical examination, she had elevated jugular venous pressure with hepatomegaly and ascites. There was no pulsus paradoxus. Routine blood investigations were normal. Abdominal ultrasound showed free fluid, hepatomegaly, single left sided spleen, and no other abnormality. Her chest X-ray [Figure 1] showed cardiomegaly and pulmonary venous hypertension. Electrocardiography showed ectopic atrial rhythm, first-degree atrioventricular block, and poor R wave progression but was unchanged from the previous recording.

Echocardiography showed features of congestion such as dilated inferior vena cava and lack of inspiratory collapse. There was no pericardial effusion. Biatrial enlargement was also present. There was no significant atrioventricular valve regurgitation, and systemic ventricular function was normal. Respiratory variation in inflow velocities across the atrioventricular valves or septal bounce were not appreciated due to the presence of a large ventricular septal defect and atrial septectomy. Diagnosis of constrictive pericarditis

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(CP) was suspected despite cardiomegaly and atrial dilation.

CT angiogram, [Figure 2] however, did not reveal pericardial thickening or calcification. Previous records did not suggest any evidence of the development of postpericardiotomy syndrome (PPS) in the first year after surgery. There was no history of tuberculosis.

A repeat cardiac catheterization showed elevation of right atrial, ventricular diastolic, and pulmonary artery diastolic pressures all being nearly equal around 20 mmHg. Mean pulmonary artery pressure was 33 mmHg with pulmonary vascular resistance index (PVRI) of 2.9 Wood units. The right atrial pressure of 22 mmHg in the absence of a significant left to right shunt, ventricular dysfunction, or atrioventricular regurgitation was suggestive of a constrictive physiology.

The diagnosis was confirmed at operation. The pericardium was found to be adherent to the underlying epicardium over the right atrial and ventricular walls. The pericardium, however, was not significantly thickened. Histopathological examination of the excised pericardial tissue showed fibrosis, hyalinization, and focal chronic inflammation consistent with pericarditis [Figure 3].

Since the calculated PVRI at preoperative catheterization was 2.9 Wood units, and the pulmonary arteries were thin walled at pulmonary arteriotomy, bilateral bidirectional cavopulmonary shunt with antegrade flow interruption was carried out 4 days after pericardiectomy as an intermediate palliation for the future. The staged surgery was done in view of the unusual combination of POCP and the presence of an underlying condition requiring univentricular palliation and can be argued. This is, however, not the point of this communication. There was a remarkable regression in the ascites and marked symptomatic improvement. On follow-up 1-year after pericardiectomy the child is active and growing adequately [Figure 4].

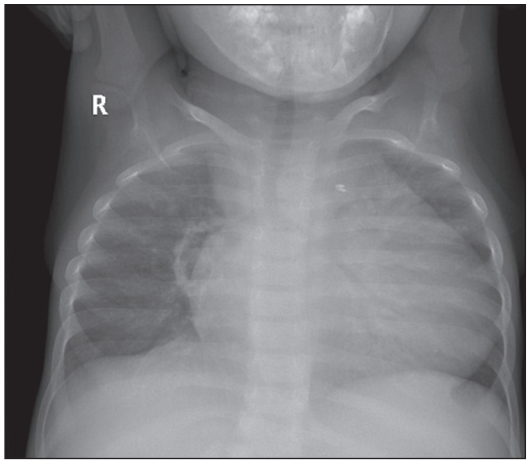

Figure 1: Chest radiograph before pericardiectomy showing cardiomegaly and pulmonary venous hypertension

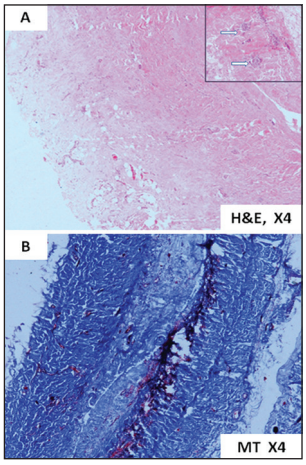

Figure 3: Histopathology of excised pericardium. Pericardium showing (a) Dense fibrosis and hyalinization with focal chronic inflammatory cell infiltration (inset, arrow) (b) Masson Trichrome stain imparts blue color to the fibrosed area

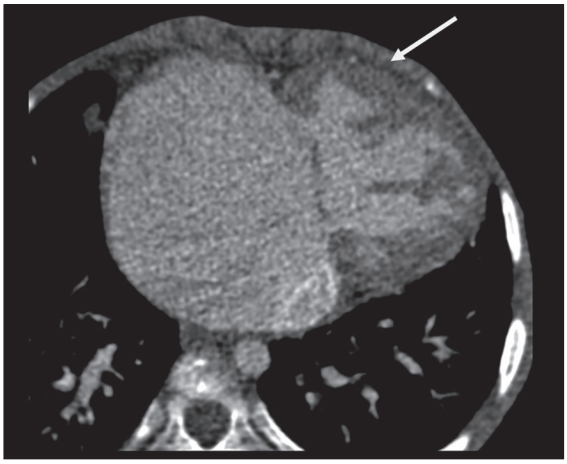

Figure 2: Computed tomography image showing normal thickness of the pericardium (arrow)

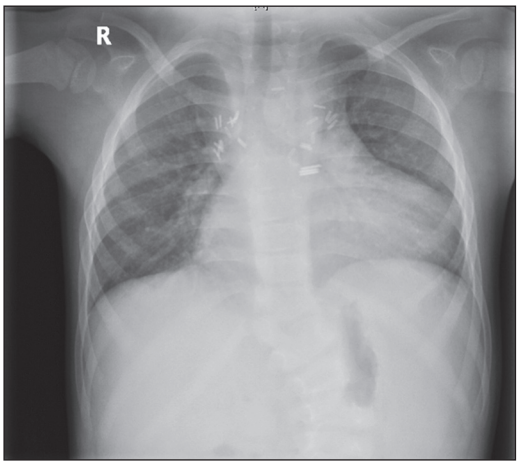

Figure 4: Chest radiograph after pericardiectomy showing some decrease in cardiomegaly

Table 1: Case reports of POCP in children after open-heart surgery

Case report	Initial operation	Age at initial operation (years)	Age at pericardiectomy (years)	Clinical presentation	Diagnostic investigation	Outcome
Saiki *et al.*[5]	VSD closure	2	18	Massive ascites	Echocardiography, catheterization	Regression of symptoms
Okamoto *et al.*[6]	VSD closure	6	12	Massive ascites	Catheterization	Regression of symptoms
Okamoto *et al.*[6]	VSD closure	8	13	Hepatomegaly	Catheterization	Regression of symptoms
Kim *et al.*[7]	ASD closure	12	14	Dyspnea	Echocardiography, catheterization	Regression of symptoms
Present case	Atrial septectomy with PAB and PDA ligation	3	6	Massive ascites	Clinical synthesis (catheterization, echocardiography and CT nondiagnostic)	Regression of symptoms

POCP: Postoperative constrictive pericarditis, CT: Computed tomography, PDA: Patent ductus arteriosus, PAB: Pulmonary artery banding, ASD: Atrial septal defect, VSD: Ventricular septal defect

DISCUSSION

CP is a well-recognized though uncommon complication of open-heart surgery. The incidence of POCP had been reported as 0.2-0.3%.[1] However, it is being increasingly recognized and is now one of the common causes of pericardial constriction in the Western world.[2,3]

The majority of cases have been reported after coronary artery bypass graft[4] or valve replacement surgery.[2-4] However, POCP may follow any form of cardiac surgery. To the best of our knowledge, only four cases of POCP have been described in children [Table 1].[5-7] In addition, one case of transient pericardial constriction after surgical correction of the subaortic stenosis has been described in a 7-year-old boy that responded to steroids.[8]

Several intra- and postoperative factors have been implicated in the occurrence of POCP. Factors such as the use of povidone iodine,[6] iced saline solution,[1] air drying, and chemical exposure, leaving open pericardium at the time of surgery,[2] and postoperative wound infection have been implicated but with no clear evidence of causality. PPS appears important in the genesis of some cases of POCP as 12% of patients with PPS in a series subsequently underwent pericardiectomy for POCP.[9]

The interval between the initial operation and presentation of pericardial constriction is variable ranging from 2 weeks to years after the initial surgery.[1] The diagnosis is often delayed as the symptoms are usually attributed to the primary heart disease and the previous operation. A high index of clinical suspicion needs to be maintained for early diagnosis. The reason for the occurrence of constriction in our case remains speculative. There was nothing unusual in the postoperative course nor was there any febrile illness. The diagnosis in our case was particularly difficult because of the rarity of CP in young children and the presence of an underlying structural lesion that might cause heart failure. Even the computed tomographic scan did not show any features suggestive of constriction, but CP with normal thickness of pericardium is well recognized.[10] Thus, a synthesis of clinical findings led to the conclusion of pericardial constriction that was confirmed at operation and by subsequent postoperative course.

Pericardiectomy remains the definitive form of therapy for POCP. Higher operative mortality and worse survival rates have been reported with POCP compared to idiopathic CP.[2]

CONCLUSION

CP can rarely occur following open-heart surgery even in young children. A high index of suspicion in the proper clinical setting is warranted.

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Conflicts of interest

There are no conflicts of interest.

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