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Constrictive Pericarditis after Surgical Closure of Atrial Septal Defect in a Child

Constrictive pericarditis following an open cardiac surgery for congenital heart disease is very rarely encountered in children. We report a case of constrictive pericarditis, diagnosed at 22 months after surgical closure of an atrial septal defect, in a 14-year-old boy who presented postpericardiotomy syndrome. Cardiac catheterization revealed uniformity of elevated diastolic pressures in all chambers, prominent x and y descents in the right atrial pressure tracing, and typical "dip and plateau" contour of ventricular end-diastolic pressures. Pericardiectomy improved the hemodymanic status of the patient. He remains symptom-free during the follow-up period of 12 months.

Key Words: *Pericarditis, constrictive; Thoracic surgery; Heart septal defect, atrial; Child*

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INTRODUCTION

Constrictive pericarditis following open cardiac surgery is a rare complication in adults who undergo myocardial revascular surgery or prosthetic valve replacement. Since the first case of this sequela reported in 1972 (1), about 200 cases have been reported in the literature with an incidence of 0.2% to 0.63% (2, 3). However, only a few cases have been reported following cardiac surgery for congenital heart disease (4-8).

Recently, we experienced a case of constrictive pericarditis after surgical closure of an atrial septal defect in a 14-year-old boy who initially presented postpericardiotomy syndrome. We report this case with the review of the literature.

CASE REPORT

A 12-year-old child underwent surgical closure of an atrial septal defect (ostium secundum type) using a Dacron patch graft on December 31, 1994. The patient's initial postoperative course was uncomplicated. Six months after the operation, he began to experience exertional dyspnea, intermittent chest tightness, and facial edema in the morning. At admission, the pulse rate was 82/min, the blood pressure 120/80 mmHg, the respiration rate 22/min and the body temperature 36.5℃. Cardiovascular examination was normal. The liver was palpable 2 cm below the right costal margin without tenderness. Mild facial edema was present. The chest roentgenogram revealed cardiac enlargement and mild pulmonary vascular congestion. The electrocardiogram showed diminished QRS voltage, by comparing the preoperative tracing. An echocardiography demonstrated echo-free space between the pericardium and epicardium, but cardiac contractility was normal. Complete blood cell counts, erythrocyte sedimentation rate and liver function tests were normal. Although the patient was afebrile and had a normal erythrocyte sedimentation rate, a postpericardiotomy syndrome was suspected. The patient was treated with furosemide, aspirin and prednisone with gradual improvement.

Twelve months later, he began to experience intermittent chest pain and dyspnea on exertion. He was hospitalized for cardiac catheterization in October 1996 (22 months after surgical repair of an atrial septal defect). Examination revealed slight jugular vein distension and distant heart sounds. The liver was palpable 3 cm below the right lower costal margin. An increased cardiac silhouette (cardiothoracic ratio=0.59) and pulmonary vascular congestion on the chest x-ray was noted (Fig. 1) and the electrocardiogram showed diminished QRS voltage. The echocardiography suggested pericardial thickening. Cardiac catheterization revealed marked elevation and near equalization of diastolic pressures in all chambers. A prominent x and y descents were seen in the right atrial pressure tracing, and the ventricular pressure recording showed a typical "square root" diastolic pres-

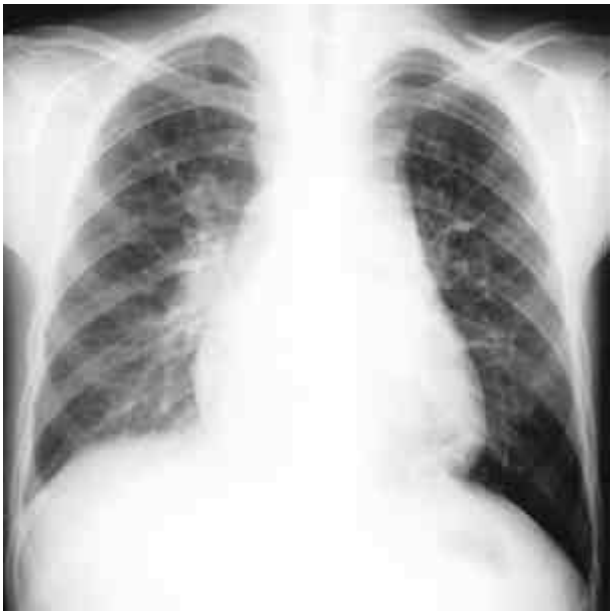

Fig. 1. Chest roentgenogram at cardiac catheterization reveals cardiomegaly and pulmonary vascular congestion.

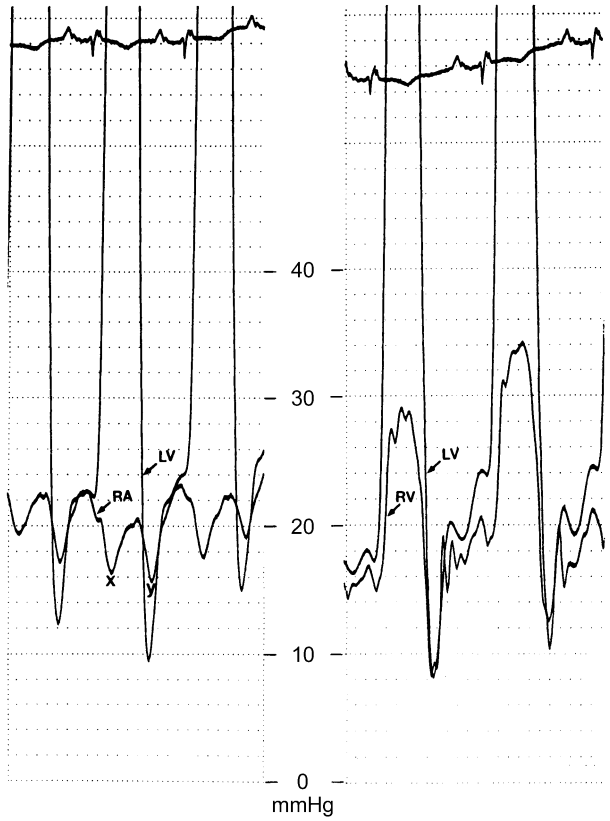

Fig. 2. Pressure recordings show markedly elevated and virtually equal diastolic pressures of right atrial (RA), right ventricle (RV) and left ventricular (LV) measuring about 20 mmHg. A prominent x and y descents is seen in RA pressure tracing. The ventricular pressure recordings show a typical "square root" diastolic pressure contour.

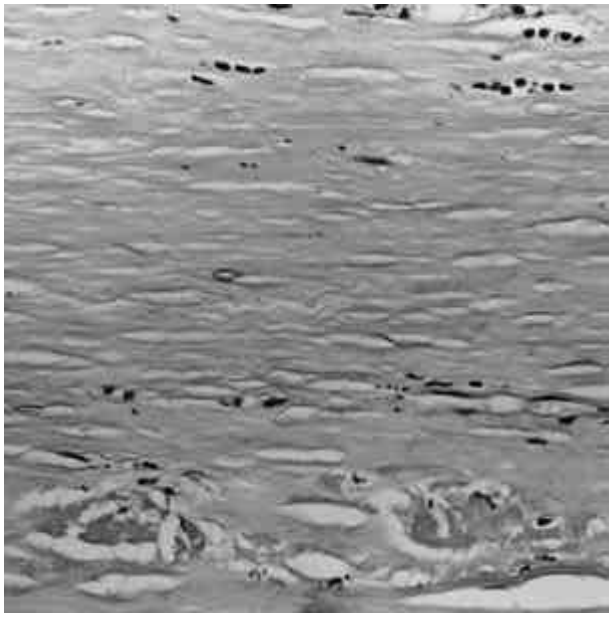

Fig. 3. The resected pericardium shows a few chronic inflammatory cell infiltrations and dense collagenous fibrous tissue (H&E, ×200).

sure contour, with about 20 mmHg of late ventricular diastolic pressures (Fig. 2).

After constrictive pericarditis was diagnosed, he underwent pericardiectomy on November 11, 1996. The pericardium had thickened (0.8 cm) and tightly adhered to the epicardium. Large portions of the pericardium was resected. Subsequent histopathologic examination of excised pericardium disclosed chronic inflammation with dense collagenous fibrous tissue (Fig. 3). Bacterial, acid-fast bacillus, and fungal cultures were negative. Pressure data of follow-up cardiac catheterization performed at 3 months after pericardiectomy revealed hemodynamical improvement (Table 1). Over a 12-month period after pericardiectomy, the patient is in good condition.

Table 1. Data of cardiac catheterization before and after pericardiectomy

Pressure (mmHg)	Before	After
RA (a/v/m)	24 / 21 / 19	14 / 12 / 11
RV (s/ed)	38 / 22	39 / 12
PA (s/ed/m)	38 / 25 / 28	36 / 16 / 12
PCW (a/v/m)	25 / 22 / 21	20 / 18 / 14
LV (s/ed)	110 / 25	140 / 8
Ao (s/ed/m)	110 / 65 / 80	135 / 75 / 99

RA, right atrium; RV, right ventricle; PA, pulmonary artery; PCW, pulmonary capillary wedge; LV, left ventricle; Ao, aorta; a, a wave; v, v wave; m, mean pressure; s, systolic pressure; ed, end diastolic pressure.

DISCUSSION

Constrictive pericarditis, characterized by a thickened, adherent pericardium that restricts ventricular filling, is caused by a wide variety of causes. Open cardiac surgery has been implicated as one of the causes. However, the exact pathogenetic mechanism has not been found, although many factors, such as air drying and chemical exposure (7), iced saline solution (2), povidone iodine (9), open pericardium at the time of surgery (4), previous chest irradiation (10), postoperative wound infection, and osteomyelitis (6) have been suggested. The combination of these factors may cause inflammation, adhesions and fibrosis, resulting in a certain degree of pericardial constriction.

Postpericardiotomy syndrome was identified as a possible factor for late development of postoperative pericardial constriction (9, 11, 12). Forty-four percent of patients who developed postoperative constrictive pericarditis was reported to be preceded by postpericardiotomy syndrome (13). In these patients, medical therapy such as steroids, nonsteroidal antiinflammatory drugs or both did not prevent the late occurrence of pericardial constriction.

The time between cardiac surgery and the development of constrictive features varied over a long period of time (12), 22 months in this case. The main symptoms and signs of constrictive pericarditis after cardiac surgery were not different from those associated with other causes.

Several echocardiographic abnormalities have been described in patients with constrictive pericarditis after cardiac surgery (2, 13). These included abnormal ventricular septal motion, flat posterior ventricular wall in diastole, variable degrees of posterior pericardial effusion, and dilatation of inferior vena cava and hepatic vein, but these findings are nonspecific. Marked pericardial thickening (greater than 5 mm) is a most helpful echocardiographic diagnostic clues (2, 14), and it is well visualized by computed tomography or magnetic resonance imaging, also (13).

Once constrictive pericarditis was suspected, right and left cardiac catheterization must be performed. It revealed near equalization of elevated diastolic pressures in all chambers (usually above 20 mmHg), prominent x and y descents with an M or W pattern in the right atrial pressure tracing, typical diastolic "dip and plateau" or "square root" contour in the ventricular pressure tracings, and paradoxic pulse in the aortic pressure tracing (2, 12, 13). A fluid challenge by rapid volume expansion may be helpful to bring out above typical findings in cases of occult constrictive pericardial disease or early pericardial constriction (15).

Medical therapy such as corticosteroids has been effective in reversing pericardial constriction only in some cases when it is given within the first 2 months of the original surgery (7). However, once irreversible pericardial fibrosis has occurred, surgical removal of the thickened pericardium is the only definitive form of therapy. It improves cardiac function and relieves symptoms in majority of patients. The operative mortality rates varied from 5.5% to 14.5% (13). Our patient showed marked improvement of symptoms after pericardiectomy and he has continued to do well during the 12 month-follow up.

In summary, constrictive pericarditis should be considered as a possible complication in patients with congenital heart disease who underwent open heart surgery, especially when a patient has a history of previous postpericardiotomy syndrome and whose condition deteriorates clinically after initial recovery.

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