Recurrent Constriction Five Years after Partial Pericardiectomy

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Abstract
Constrictive pericarditis is a relatively rare condition and an inflammatory process that involves both the parietal and visceral layers of the pericardium, eventually resulting in heart failure due to impaired ventricular filling. A 28-year-old man was referred to our hospital with a one year history of progressive right heart failure. In his history, the patient underwent a pericardiectomy, limited anteriorly though median sternotomy without the use of cardiopulmonary bypass in 2008. Herein is a case of proven recurrent pericardial constriction following pericardiectomy.

Keywords: Constrictive pericarditis; Pericardiectomy; Reoperation

Introduction
Constrictive pericarditis (CP) is an inflammatory process that involves both the parietal and visceral layers of the pericardium. Due to an inflammatory process, pericardial thickening and constriction of the ventricles are the mainstays of the disease. Pericardiectomy is the only curative treatment for chronic constrictive pericarditis [1]. Different surgical approaches (with or without cardiopulmonary bypass) and different extents of pericardiectomy procedures (total or partial) have nearly comparable and good results [2]. Persistent symptoms may occur during follow up. The cause of symptoms may be due to incomplete resection, myocardial atrophy, and intrinsic myocardial dysfunction [3]. We present a case with recurrent constriction after partial pericardiectomy.

Case Report
A 28-year-old man was referred to our hospital with a one year history of progressive right heart failure. In his history, the patient underwent a pericardiectomy, limited anteriorly though median sternotomy without use of cardiopulmonary bypass in 2008. Before and after the operation, all diagnostic tests were negative and classified as idiopathic. He reported a one year history of progressive shortness of breath, edema in the lower extremities, and enlargement of abdominal volume. On admission he presented with cardiac decompression NYHA class 3 accompanied by edema and ascites. Physical examination revealed a venous congestion with distended neck veins, lower extremity edema, ascites, and icterus. Crepitations were heard on auscultation of his lungs. On the ECG, he had permanent atrial fibrillation, to which the ventricle response was 80-85 beats per minute. Chest X ray showed a thickened and calcified pericardium (Figure 1). Trans-thoracic echocardiography demonstrated pericardial thickening, preserved biventricular ejection fraction with decreased end diastolic and end systolic volumes. Additionally, there was significant dilation of the SVC, IVC, and hepatic veins. Cardiac catheterization was performed one week post admission after the patient received intravenous diuretics, which was non-responsive to fluid balance. Superior vena cava pressure was 23 mmHg, right atrium pressure was 25 mmHg, right ventricle end diastolic pressure was 27 mmHg and left ventricle end diastolic pressure was 30 mmHg. Simultaneous pressure measurements of the right ventricle and left ventricle revealed an early diastolic ‘dip and plateau’ and equalization of end diastolic pressures. Plasma tests were negative for antinuclear antibodies, anticardiolipins, and rheumatoid factor, complement (C3, C4) levels were normal. Chest x-ray, echocardiography and catheterization demonstrated findings consistent with recurrent pericardial constriction.

Re-median sternotomy performed for entering the mediastinum. There was an extensive fibrotic and calcified pericardial tissue all around the heart chambers (Figure 2).

Figure 1: Chest x-ray. Lateral view showed calcified mass along diaphragmatic surface of the heart(*) and pericardial calcification (arrows). A. before stripping, B. after stripping

Figure 2: Intraoperative finding in pericardiectomy. A. The arrows indicate the calcified and thickened pericardium. Parietal pericardium is thickened (6-7 mm). B. Wide areas of calcification and constricting layers of epicardium were removed.
A total pericardiectomy of the entire residual pericardium was performed by cautious dissection of the phrenic nerves, which were left on the pedicles (Figure 2).

The histopathological analysis of the resected specimen demonstrated fibroblasts and chronic inflammatory foci. No normal pericardium was seen. There was no evidence of malignancy. All stains for micro-organisms were negative. The patient showed marked improvement in the intensive care unit as evidenced by the decrease in central venous pressure from 23 mmHg to 8 mmHg. The patient did well after surgery and was discharged on postoperative day 8. His NYHA score was 1 at first month follow up.

**Discussion**

In constrictive pericarditis there are changes in the volume elasticity slopes of the both ventricles. Secondary to constriction there is an increase in left and right ventricular end-diastolic pressures and a decrease in output. Additionally thickened and calcified pericardium frequently comes into direct contact with the myocardium decreasing the contractility and disrupting the coordination of diastolic filling of the ventricles [4].

Diagnosis of constrictive pericarditis can be made by echocardiography. Identification of constriction in patients who have recurrent symptoms after partial pericardiectomy is a challenging problem. Characteristic constrictions symptoms may not be present because of some portion of ventricles were freed from constricting pericardium and all patients received medical therapy with diuretics. For this reason most of the patients receive adjunctive diagnostic methods like cardiac catheterization, CT, and MRI scan.

The aim of surgery for constricting pericardium is removal of the constricting pericardium overlying both ventricles. Some surgeons prefer limited pericardiectomy to the anterior portion because of the technical difficulty of exposing the diaphragmatic surface of the heart. However, the left lateral aspect posterior to the left phrenic nerve is sufficient to relieve constructive hemodynamics. Additionally, a large portion of diseased pericardium overlying the ventricles remains after anterior or incomplete pericardiectomy [5]. Although complete pericardiectomy may be more difficult than anterior pericardiectomy, the authors did not find any increase in operative risk of the complete procedure. In this study, 30 day mortality for completion pericardiectomy was 7%, which was higher than expected risk for a primary operation [2]. Older age, poor functional status, right ventricular dysfunction, and history of mediastinal radiation therapy are the known risk factors of late death after completion pericardiectomy [6].

In a study by Cho et al., 41 patients were evaluated who underwent repeat pericardiectomy. They divided patients into two groups according to the interval between first and second operations (one year or less and more than one year). The authors concluded that the significant early mortality of repeat pericardiectomy emphasized the importance of complete pericardial resection at the first operation and accurate diagnosis of recurrent constriction. The poor clinical outcome of late (more than 1 year) reoperation suggests that many of these patients may have unrecognized diastolic dysfunction or recurrent mediastinal scarring as the cause of right-side heart failure rather than incomplete initial pericardiectomy. This supports performing a resection as complete as possible at the time of initial pericardiectomy to prevent recurrence and risk of reoperation [7].

Pericardiectomy for CP is relatively uncommon and repeat surgery is even less frequent. Furthermore, there are no guidelines for recurrent CP. The present researchers recommend complete pericardiectomy or as much as possible and special care should be given and confirming diagnostic studies should be planned to patients who present recurrent symptoms after pericardiectomy.

**References**