CASE REPORT

Rapidly Progressive Effusive Constrictive Pericarditis Caused By Methicillin Sensitive Staphylococcus aureus (MSSA)

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ABSTRACT
Effusive-constrictive pericarditis is a clinical syndrome characterized by concurrent pericardial effusion and pericardial constriction, where constrictive hemodynamics are persistent after effusion is drained. It may present at any point along the clinical course, from the occurrence of an effusion to the development of chronic pericardial constriction. We refer an unusual case of effusive constrictive pericarditis developing rapidly within days, following purulent pericarditis secondary to chest trauma.

Key words: Effusive constrictive pericarditis. Pyogenic pericarditis. Methicillin sensitive. Staphylococcus aureus. Trauma.

INTRODUCTION
Effusive-constrictive pericarditis was first described in the 1960s. Ever since then, review of literature has revealed scarce information on this clinical entity, mainly isolated case reports have been cited. The largest series is of 15 patients, who were prospectively evaluated over a period of 16 years.¹ The hallmark of this clinical haemodynamic syndrome is the persistence of elevated right atrial pressure after intrapericardial pressure has been reduced to normal levels by removal of pericardial fluid.¹ Once detected, pericardial effusion must be evaluated to determine its cause and hemodynamic significance.

The etiology of effusive constrictive pericarditis can often be suspected from the clinical setting in which the effusion occurs. Cases of effusive-constrictive pericarditis of idiopathic origin or due to chest radiation, chemotherapy or various infectious agents have been reported.¹ Constrictive pericarditis is a rare but recognized sequelae of coronary bypass surgery.² It has also been reported to develop after pyogenic pericarditis, but the progression from pericardial effusion to development of constriction takes a longer time. Early constriction following pyogenic pericarditis has been reported in young children.³ To our knowledge, rapid progression to constriction after purulent pericarditis, in adults has not been reported yet. Hence, this case is unique due to the fact that constrictive pericarditis developed within one month of effusion.

CASE REPORT
A 22-year-old male with a history of left sided stab wound in chest, referred from a peripheral hospital, presented with dyspnea on exertion for the last one month. He was treated at an outside tertiary care centre and a chest tube was placed for left sided hemothorax. After initial examination, his bed side echocardiography was done in the emergency room, which showed massive pericardial effusion of 2.9 cm circumferential area, with exaggerated respiratory variation on tricuspid and mitral valve, suggestive of cardiac tamponade. Emergency pericardiocentesis was done, 1.5 litre of frank pus was drained and parenteral antibiotics were started. Pericardial fluid routine analysis showed pyogenic features and Methicillin sensitive Staphylococcus aureus (MSSA) was isolated. A pericardial window was made to facilitate the free drainage of pericardial fluid. The pericardial cavity contained frank pus and pericardium was grossly thickened. Biopsy was taken and sent for histopathology. However, the patient remained symptomatic with signs of bi-ventricular failure. A repeat echo was performed which showed good left ventricular function with an ejection fraction of 55%, thickened pericardium, dilated right heart and significant paradoxical septal motion with respiratory variation at mitral and tricuspid inflow were noted, suggestive of cardiac tamponade. Emergency pericardiocentesis was done, 1.5 litre of frank pus was drained and parenteral antibiotics were started. Pericardial fluid routine analysis showed pyogenic features and Methicillin sensitive Staphylococcus aureus (MSSA) was isolated. A pericardial window was made to facilitate the free drainage of pericardial fluid. The pericardial cavity contained frank pus and pericardium was grossly thickened. Biopsy was taken and sent for histopathology. However, the patient remained symptomatic with signs of bi-ventricular failure. A repeat echo was performed which showed good left ventricular function with an ejection fraction of 55%, thickened pericardium, dilated right heart and significant paradoxical septal motion with respiratory variation at mitral and tricuspid inflow were noted, suggestive of interventricular interdependence. These features were consistent with constrictive pericarditis.

In view of clinical implications of the diagnosis, a cardiac catheterization was performed. It showed left ventricle pressure (LV) of 110 mmHg, left ventricular end diastolic pressure (LVEDP) of 28 mmHg, right atrium (RA) pressure of 20 mmHg, right ventricle (RV) pressure of 44 mmHg, right ventricular end diastolic pressure (RVEDP) of 26 mmHg and pulmonary artery pressure (PAP) of 40/28 mmHg. A square root sign was also noted during inspiration (Figure 1). Meanwhile, his pericardial
Histopathology report arrived, which revealed surface ulceration and exudation with granulation tissue with marked acute and chronic inflammation and fibrosis, consistent with constrictive pericarditis (Figure 2). A diagnosis of “effusive constrictive pericarditis” was made and pericardiectomy was advised.

At pericardiectomy, a grossly thickened parietal and visceral pericardium, and a loculated area of pus was found. The visceral pericardium was adherent to the underlying myocardium and extensively fibrosed. Both visceral and parietal pericardium were widely excised. He was managed with antibiotics and supportive treatment was given. Postpericardiectomy echo was performed, which revealed normal left ventricular size and ejection fraction 60% with normal right heart dimensions and no sequelae of constrictive pathology was noted. Patient was discharged home in stable condition.

**DISCUSSION**

Effusive-constrictive pericarditis is a clinical syndrome characterized by concurrent pericardial effusion and pericardial constriction, where constrictive hemodynamics are persistent after effusion is drained. The visceral and parietal layers of pericardium adhere to each other or to the myocardium and impair ventricular filling, thus, decreasing stroke volume and cardiac output.

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Constrictive physiology with a co-existing pericardial effusion usually produces tamponade. Such patients may be mistakenly thought to have only tamponade, and hence diagnosis of constrictive pericarditis may be missed. However, elevation of the right atrial and pulmonary wedge pressures persisting after drainage of the pericardial fluid is a sign of constriction. Echocardiography and right heart catheterization plays an important role in differentiating constrictive pericarditis from restrictive cardiomyopathy, which incorporates the concept of ventricular interdependence.

Recognition of effusive constrictive pericarditis is of critical significance. There are case reports of constrictive pericarditis that remained clinically unsuspected until autopsy. Hence, treatment only with pericardiocentesis or pericardial window may be inadequate. Special attention also needs to be paid to the degree of involvement of both visceral and partial layer of pericardium and extensive pericardiectomy has been suggested as the procedure of choice. This patient had widespread involvement of pericardium extending up to the visceral layer and causing massive fibrosis, therefore, a wide resection of pericardium was done.

A few researchers observed that spontaneous resolution of symptoms in cases of idiopathic acute exudative pericarditis, so they were labeled with transient cardiac constriction. The tamponade and constriction may resolve after pericardiocentesis, and pericardiectomy is not needed. Therefore, it is suggested that pericardiectomy can be delayed unless constriction becomes severe and persistent.

In this patient, the course of illness was aggressive with development of extensive myocardial fibrosis within a month. In lieu of this, an extensive pericardiectomy and debridement was performed on urgent basis, with reasonable postoperative recovery and good clinical outcome.
Rapidly developing effusive constrictive pericarditis followed by acute pyogenic pericarditis is a rarely reported complication. Early and extensive pericardiectomy, paying special attention to the visceral pericardium, depending on the extent of involvement is the treatment of choice.

REFERENCES


