Calcified pericardial band at the level of right ventricle causing midventricular obstruction: a case report of constrictive pericarditis

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Abstract

Constrictive pericarditis is a debilitating but potentially curable disease. Diffuse pericardial thickening and calcification is a classic feature described; localized pericardial constrictions are very rare. Here we report a case of a young female who had presented with constrictive pericarditis and had localized calcific constrictive band located at the level distal to atrioventricular groove which had caused right midventricular obstruction. In many patients the cause of the pericardial disease is undetermined, and in them an asymptomatic or forgotten bout of viral or tubercular pericarditis, acute or idiopathic, may have been the inciting event. Our patient had pulmonary tuberculosis during childhood and received antituberculous treatment. Investigations revealed the presence of thickened pericardium and a thickened calcific constrictive band around the right ventricles at midventricle level causing midventricular obstruction. She was referred to another centre for further surgical management.

Key words: Constrictive pericarditis, constrictive band, tuberculosis.

Introduction

Chronic constrictive pericarditis mostly results when the resorption of a chronic pericardial effusion or the healing of an acute fibrinous or serofibrinous pericarditis is followed by obliteration of the pericardial cavity with the formation of granulation tissue.1 The latter gradually contracts and forms a firm scar, encasing the heart and interfering with filling of the ventricles. The time interval between the episode of acute pericarditis and calcified pericardial band at the level of right ventricle causing midventricular obstruction: a case report of constrictive pericarditis

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rheumatoid arthritis, SLE, and chronic renal failure with uremia treated by chronic dialysis. Whipple’s diseases, amyloidosis, dermatomyositis and sarcoidosis are the other rare causes of constrictive pericarditis. “Mulibrey” nanism, an autosomal recessive syndrome, characterized by growth failure, muscle hypotonia, hepatomegaly, ocular changes, enlarged cerebral ventricles, mental retardation, ventricular hypertrophy, and chronic constrictive pericarditis. In many patients the cause of the pericardial disease is undetermined, and in them an asymptomatic or forgotten bout of viral pericarditis, acute or idiopathic, may have been the inciting event.

Case report

A 25-year female was admitted to College of Medical Sciences-Teaching Hospital with complaints of insidious onset and gradually progressive abdominal distension of 2 months duration. It was associated with breathlessness initially on minimal exertion and later on even at rest. For last one week, her breathlessness became persistent and she also noticed palpitation, easy fatigability, and pitting pedal edema. She gave a history of an episode of long lasting febrile illness associated with cough and weight loss in childhood for which she was empirically treated with antitubercular drugs for a period of one year. Two months ago, she noticed abdominal distension which progressed gradually and started to have exertional dyspnea and fatigue.

On examination, she appeared pale and undernourished. She had pitting pedal edema and raised JVP with prominent x and y descent. The blood pressure was 90/60 mmHg and the pulse rate was 106 beats/min, regular and of low volume. Pulsus paradoxus was absent. Apical impulse was located in 5th intercostals space 1 cm medial to left midclavicular line. There was a pericardial knock in 4th intercostals space left side. There was a pansystolic murmur in left lower sternal border which increased on inspiration. Others cardiovascular findings were normal. An examination of chest was unremarkable. Per abdomen examination revealed mild tender hepatomegaly and moderate ascites. A chest X-ray showed calcification of the Pericardium (Figure 1).

Figure 1. CXR-PA view showing calcification of pericardium, seen in lower right corner area (arrows)

A transthoracic echocardiogram showed a thickened and calcific pericardium (10 mm) and a localized calcific band at midventricle level causing constriction distal to atrioventricular groove. There was calcification in atrioventricular groove as well.
The left ventricular posterior wall showed sudden diastolic cessation with septal bounce. The inferior vena cava was dilated, with absent respiratory variation. There was midventricular obstruction in the right ventricle with ballooning in the apical area causing dumbbell shaped appearance of left ventricle (figure 2).

A ring of calcification, encircling the right and left ventricles distal to atrioventricular groove was seen at the junction of the basal and mid cavity level. An altered shape of left ventricle cavity was seen as a result the heart had a dumbbell shape because of a tight constricting band seen just around and immediately distal to the atrioventricular groove (figure 3). The calcification was also noticed at the level of atrioventricular groove better seen in figure 2.

The patient was managed conservatively with minimal diuretics and after stabilization she was referred to a cardiovascular surgical centre for further definitive treatment.

Discussion

Constrictive pericarditis is an uncommon but potentially curable pathology. Localized pericardial constrictions are very rare. Our case had features suggestive of constrictive pericarditis, with an accompanying tight constrictive band at the level of midventricle causing right midventricular obstruction resulting in a dumbbell-shaped heart with apical ballooning. In addition to the calcific constrictive band, she also had calcification of pericardium and the atrioventricular groove.
Localized pericardial constriction is reported to be a rare form of constrictive pericarditis, and if such present most of the time constriction occurs at the atrioventricular level. Depending on the location of pericardial constriction, clinical presentation of localized constriction may be variable including obstruction of right ventricular outflow tract, and pulmonary stenosis.

Among pericarditis, generalized constrictive pericarditis is a usual rule but the localized form is rare. Localized pericardial band has been reported after previous pericardiectomy and along with congenital heart disease. The most common manifestation reported with localized constrictive band involves the atrioventricular groove, along with obstruction to the right ventricular outflow tract. In contrast to these usual patterns, our patient had two unique features. Firstly it had calcified constrictive band at the midventricular level distal to atrioventricular groove along with extensive calcification in pericardium and AV groove. The midventricular obstruction was another rare conditions recorded in our case. Review of literature shows the presence of RV outflow tract obstruction in constrictive pericarditis. Usually the calcification does not necessarily cause clinical constriction. The extensive calcification is the most notable characteristic of chronic constrictive pericarditis. The patient discussed here had pulmonary tuberculosis in childhood and was successfully treated with antitubercular drugs and this time at the age of 25 years, she had presented with the features suggestive of constrictive pericarditis.

Constrictive pericarditis is a potentially curable chronic pericardial disease. Whether antituberculous treatment prevents constrictive pericarditis is debatable. Although, the reason has not been discussed, localized constrictions are most common around the atrioventricular groove posteriorly and over the infundibulum anteriorly. It is believed that the AV groove and RV body–infundibulum junction is the transition gutter-like area between the relatively fixed (and less moving) structures such as the atria and infundibulum and the dynamic ventricles. This would logically result in stasis of an organizing collection in this particular region and this collection, over years, is replaced with cicatrix fibrocalcific tissue. Total pericardiectomy is the treatment of choice for constrictive pericarditis and has good long-term results. In high risk subjects, subtotal pericardiectomy can also be done with satisfactory result. Although the detail investigation in our subjects did not reveal any evidence of active or healed tuberculosis except that from history of past illness, we believe that the primary tuberculosis that had occurred during childhood and was treated successfully had caused cicatrization that led to chronic constrictive pericarditis.

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References


