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Case Report

An unusual cause of refractory ascites with cirrhosis

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ABSTRACT

Reports of chronic constrictive pericarditis presenting with hepatomegaly, cirrhosis and ascites but no cardiac symptoms are rare. A 10 year old child presented with refractory ascites and firm hepatomegaly of 2 years duration which partially responded to diuretics. Doppler study showed dilated hepatic veins and inferior vena cava and ruled out hepatic venous outflow tract obstruction. Chest X ray showed pericardial calcification and 2D Echo was suggestive of chronic constrictive pericarditis. A diagnosis of chronic calcified constrictive pericarditis with cirrhosis and refractory ascites was made. Based on previous series which showed that tuberculosis is the most common cause of constrictive pericarditis, tuberculosis as the cause of constrictive pericarditis was kept. Anti tuberculous treatment as per Revised National Tuberculosis Control Program guidelines was started along with oral steroids and diuretics. Child showed response to the treatment and was later referred to cardiac surgery department for further management (pericardectomy).

Keywords: Chronic constrictive pericarditis, Pericardial calcification, Refractory ascites, Tuberculosis

INTRODUCTION

Refractory ascites is a difficult diagnostic challenge. Chronic constrictive pericarditis as a cause of refractory ascites is still underdiagnosed, and consequently undertreated. Reports of chronic constrictive pericarditis presenting with hepatomegaly, cirrhosis and ascites but no cardiac symptoms are rare.[1,2] Here we present a case of chronic constrictive pericarditis presenting with hepatic manifestations.

CASE REPORT

A 10 year old child presented with generalized distension of abdomen associated with reduced urine output and mild swelling over the face since 2 years. The distension progressed slowly over a period of time till the abdomen became tense and veins became visible over the abdomen. He passed urine 1-2 times per day. He had no swelling over feet.

The distension decreased after oral diuretics but recurred again after stopping them. During this period, he also developed fullness in upper abdomen which remained static. He had reduced appetite, poor weight gain and thinning of limbs after the onset of illness. There was no history of swelling over feet, red colored urine, jaundice, bleeding manifestations, altered sensorium, pain in abdomen, breathing difficulty, pallor, previous blood transfusion, chronic cough, prolonged fever, significant Koch's contact or any operative procedure.

The vital parameters were normal except for mild tachypnea. Weight, height and body mass index (BMI) were <3rd centile as per centers for disease control (CDC) growth charts.

He had mild periorbital puffiness, bilateral conjunctival xerosis. He had no pallor, icterus, pedal edema, clubbing, signs of chronic liver disease. There was non-tender, generalized abdominal distension, fullness in epigastric and right hypochondriac region, visible veins over upper abdomen, chest and back. There was firm, non-tender hepatomegaly but no splenomegaly. Hepatojugular reflux and fluid thrill were appreciated. Cardiac, chest and neurological examination was unremarkable.

Table 1: Relevant laboratory investigations.

Parameter		Value
Bilirubin-total/direct (mg/dL)		1.92/0.72
AST (U/L)		29
ALT (U/L)		21
Protein (gm/L)		8.0
Albumin (gm/L)		4.7
INR		1.43
Ascitic fluid	Appearance	Clear, serous
	Protein (mg/dL)	5.0
	Sugar (mg/dL)	87
	Cytology	Mesothelial cells, few lymphocytes
Urine analysis		Normal
ESR		32mm/hour
Serum creatinine (mg/dL)		0.9
HBsAg		Non-reactive
Anti HCV		Non-reactive
ANA		Negative
Mantoux test		Negative
Gastric aspirate for AFB		Negative
Cartridge based Nucleic acid amplification test		Negative

Relevant laboratory investigations are outlined in Table 1. A possibility of hepatic venous outflow tract obstruction (HVOTO) or constrictive pericarditis was kept. Ultrasonography of abdomen with Doppler showed enlarged liver with altered echotexture. Hepatic veins and inferior vena cava (IVC) were dilated showing altered spectral waveform with loss of normal triphasic waveform, however no evidence of thrombosis was seen. Portal vein, biliary system and spleen were normal. Ascites was present. Chest X ray revealed pericardial calcification. Electrocardiogram showed low voltage activity and sinus rhythm. Contrast enhanced computed tomography of abdomen showed heterogenous enhancement of liver, dilated hepatic veins and IVC and ascites. Multiple enlarged discrete mesenteric lymph nodes were seen, the largest measuring 16X12 mm. Visualised sections of chest also showed pericardial calcification. Based on above radiological findings HVOTO was ruled out. Two-D echocardiography showed increased pericardial echogenicity suggestive of chronic constrictive pericarditis, mild tricuspid regurgitation and mild pulmonary artery hypertension.

A diagnosis of chronic constrictive pericarditis with cirrhosis and refractory ascites was made. Even though work up for tuberculosis was negative, based on clinical features and previous series which showed that tuberculosis is the most common cause of constrictive pericarditis, tuberculosis as the cause of constrictive pericarditis was kept.[3,4] Anti tuberculous treatment (ATT) as per Revised National Tuberculosis Control Program (RNTCP) guidelines was started along with oral steroids and diuretics. Child showed response to the treatment and was later referred to cardiac surgery department for further management (pericardectomy).

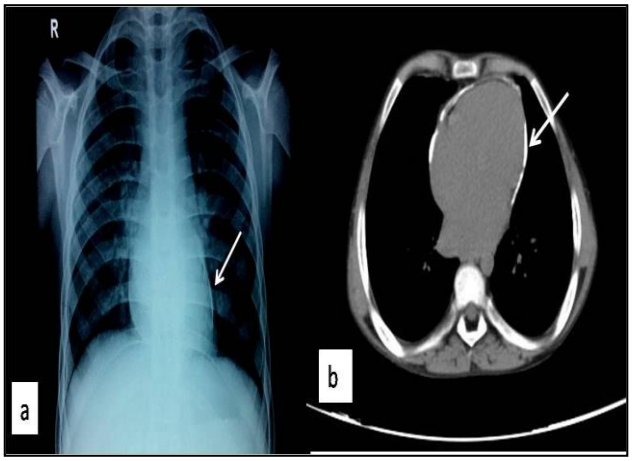

Figure 1: Chest X-ray PA view a) Computed tomography chest b) demonstrating the pericardial calcification (arrow in a and b).

DISCUSSION

Constrictive pericarditis occurs when a scarred, thickened and frequently calcified pericardium impairs cardiac filling, limiting the total cardiac volume. The most frequent causes are chronic idiopathic pericarditis, mediastinal radiation, after cardiac surgery and tuberculous pericarditis.[3,4] Tuberculosis remains an important cause of constrictive pericarditis in developing countries till date.[5]

It commonly presents with cardiac manifestations which involve features of elevated systemic venous pressures and low cardiac output. Rarely, in long standing cases, extra cardiac manifestations like refractory ascites, hepatic manifestations may be the presenting features which lead to a delay in the diagnosis of constrictive pericarditis. The present case also presented with extra cardiac manifestations and no cardiac features. HVOTO was kept as the first possibility because of refractory ascites and cirrhotic liver but was ruled out after ultrasonography and Doppler study of the liver and hepatic vessels.

Pericardial calcification seen on chest X-ray and CT scan along with 2D Echo led to the diagnosis of chronic constrictive pericarditis. Similar cases of chronic

constrictive pericarditis presenting with refractory ascites have been reported previously.[1,2] Yuan SM has reported a similar case of chronic calcified constrictive pericarditis presenting with refractory ascites in a young female.[1]

CONCLUSION

Chronic constrictive pericarditis can sometimes present with hepatic manifestations but no cardiac manifestations. A high index of suspicion should be kept for the diagnosis of chronic constrictive pericarditis in a case of refractory ascites.

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