CASE REPORT

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Decompensated Liver Disease: An Unusual Manifestation of Constrictive Pericarditis in a Young Child

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ABSTRACT

Constrictive pericarditis is a rare but potentially reversible cause of cardiac cirrhosis in children. It results from a thickened and fibrotic pericardium restricting diastolic filling, leading to systemic venous congestion and secondary hepatic fibrosis. Paediatric cases are exceedingly uncommon and often misdiagnosed as primary hepatic disease due to overlapping clinical features. We report a 10-year-old female who presented with progressive abdominal distension and ascites for 25 days, with a history of intermittent abdominal distension and low-grade fever over four years. Examination revealed raised jugular venous pressure, hepatomegaly, and ascites. Investigations demonstrated hepatic fibrosis pericardial thickening with effusion on CECT thorax, and echocardiographic features consistent with effusive–constrictive pericarditis. The tuberculin skin test was strongly positive, although CBNAAT was negative. A diagnosis of probable tubercular constrictive pericarditis with secondary cardiac cirrhosis was made. The child showed significant clinical improvement with anti-tubercular therapy, corticosteroids, and diuretics. Chronic hepatic venous congestion from Constrictive pericarditis can lead to congestive hepatopathy and subsequent cirrhosis. The hepatic dysfunction is often reversible following timely pericardiectomy. However, delayed diagnosis, as in this case, may result in advanced hepatic fibrosis. This case highlights the importance of including Constrictive pericarditis in the differentials of unexplained decompensated liver disease in children, especially in tuberculosis-endemic regions. Early recognition and comprehensive cardiovascular evaluation are essential in paediatric patients presenting with hepatic dysfunction, as prompt intervention can reverse cardiac cirrhosis and prevent irreversible hepatic injury.

Keywords: *Hepatic fibrosis; Tuberculosis; Pericardiectomy; Corticosteroids; Anti Tubercular Treatment*

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INTRODUCTION

Constrictive Pericarditis is a disease process causing serious limitation to cardiac diastolic filling resulting from thickened pericardium, adhesion and even calcification due to various causes, which commonly occurs in adults. Constrictive pericarditis is an exceedingly rare disease entity in children, with limited published case reports. Specific incidence rates are not well-documented, with very few cases reported in paediatric age group which generally presents with exercise-induced dyspnoea, bipedal oedema and ascites and often diagnosed as liver or renal disease. [1] It can be caused by tuberculosis, connective tissue disease, radiotherapy, cardiac surgery, trauma, neoplasm and may also be idiopathic. Most commonly seen findings are ascites, enlarged liver, bipedal oedema and hypoalbuminemia. [2] Herein the authors report a case of a 10-year-old female with constrictive pericarditis leading to decompensated liver failure.

CASE SUMMARY

History and Examination

A 10-year-old female was referred from a primary health care centre to our tertiary care hospital with complaints of progressively increasing abdominal distension over the preceding 25 days [Figure 1].

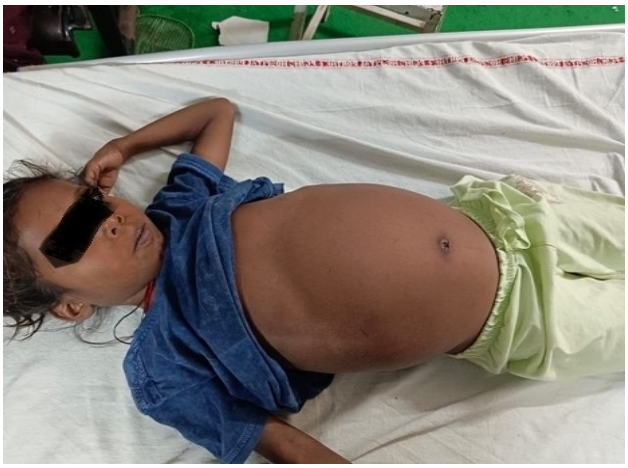

Fig 1: Distention of abdomen

The child had a history of intermittent abdominal distension associated with low-grade fever for the past four years, had taken treatment for the same, the details of which aren't known. She was developmentally normal fully immunized and was the fourth-born child of a non-consanguineous marriage. There was no history suggestive of jaundice, bleeding manifestations, dyspnea, chest pain, or altered bowel habits. There was no history of tuberculosis contact in the family or history of any chronic illness among

family members. On general examination, the child appeared undernourished and pale, with bilateral pedal edema. There was no icterus, cyanosis, clubbing, or lymphadenopathy. Vital parameters were stable. BCG scar was present. Anthropometric evaluation revealed a body mass index (BMI) of 15 kg/m², corresponding to the 25th–50th percentile for age. Abdominal examination revealed generalized distension with visible dilated superficial veins. The liver was palpable 4.5 cm below the right costal margin, spanning 12 cm, and enlargement of left lobe. The surface was firm and smooth, with well-defined margins and non-tender to palpation. There was no splenomegaly. Fluid thrill was present, indicating the presence of ascites. Cardiovascular system examination revealed raised jugular venous pressure [Figure 2], with no visible epigastric or precordial pulsations.

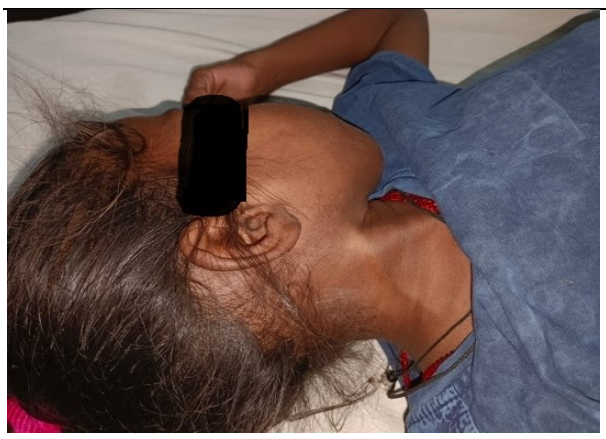

Fig 2: Raised JVP

The apical impulse was localized to the fifth intercostal space, approximately 8 cm from the midclavicular line, and was not displaced. There was no heave, thrust, or thrill. On auscultation, the first and second heart sounds were slightly muffled with no murmurs heard. Examination of the respiratory and central nervous systems did not reveal any abnormality.

INVESTIGATIONS

Laboratory investigations are summarized in Table 1.

Table 1: Summary of Investigation

Hb(g/dl)	10.6
WBC(cells/cumm)	5900
DLC(P/L/M/B)	52/46/1/1
Platelets(lacs/cumm)	1.9
AST/ALT(U/L)	40/24
Albumin(gm/dl)	2.7
ALP(U/L)	127
Ceruloplasmin(mg/dL)	32.66
Serum creatinine(mg/dL)	0.4
Blood urea(mg/dL)	24
INR	1.1
Na/K(mEq/L)	137/4.2
Hepatitis B antigen	Non reactive
Anti hepatitis C IgG	Non reactive
HIV	Non reactive
IgA tTg	Negative

Slit-lamp examination revealed a Kayser Fleischer ring [Fig. 3].

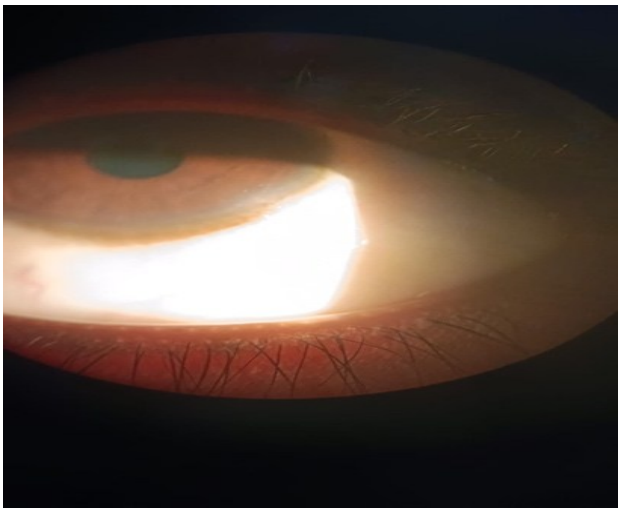

Fig 3: Kayser Fleischer ring

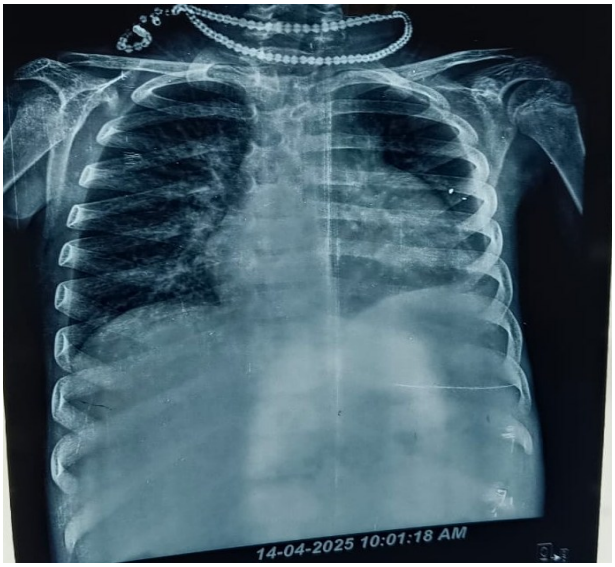

Figure 4: Chest X ray findings: Cardiomegaly with cardio-thoracic ratio 0.6 and right perihilar haziness

Chest radiograph [Fig.4] showed cardiomegaly (cardiothoracic ratio 0.6) with right perihilar haziness. The tuberculin skin test was strongly positive (18 mm), while cartridge based nucleic acid amplification test (CBNAAT) of ascitic fluid and sputum was negative for Mycobacterium tuberculosis.

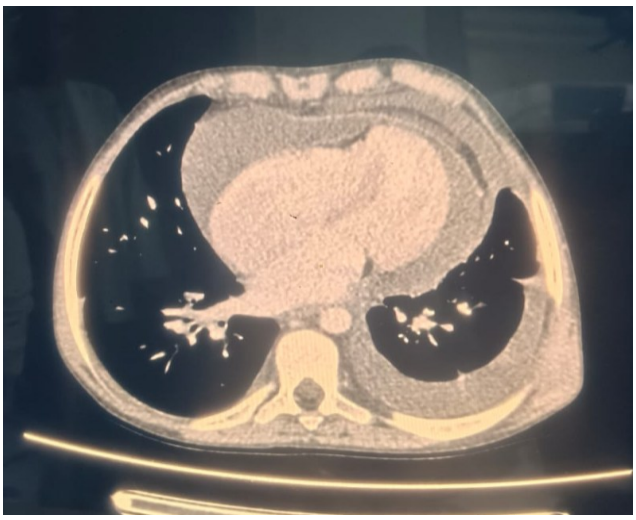

Figure 5: CECT thorax: Pericardial effusion with pericardial thickness measuring 4mm and enhancing pericardium, tubular right ventricle, apex tilted to the right side and dilated right atrium and inferior vena cava along with left side minimal pleural effusion

Ultrasonography demonstrated gross ascites, hepatomegaly (11 cm) with altered echotexture, portal vein diameter 14.5 mm. Echocardiography revealed pericardial effusion with pericarditis, mild tricuspid regurgitation, no

mitral or aortic regurgitation, diastolic flow reversal in the hepatic vein, and ejection fraction > 25%, consistent with effusive-constrictive pericarditis. Transient elastography showed LSM 18.6 kPa (IQR/median 4%), correlating with fibrosis stage F4. Upper GI endoscopy was normal, without varices. CECT thorax [Fig.5] demonstrated pericardial effusion with thickened (4 mm), enhancing pericardium, tubular right ventricle, rightward-tilted apex, dilated right atrium and IVC, and minimal left pleural effusion, suggestive of chronic inflammatory pericarditis. The patient was managed conservatively with bed rest with propped up position, salt restriction, and protein supplementation. Based on the clinical profile and high incidence of tubercular infection in the region, imaging findings, a diagnosis of tubercular pericarditis was strongly considered. Anti-tubercular therapy, corticosteroids, and diuretics were initiated, following which the patient demonstrated progressive clinical improvement with gradual resolution of oedema and ascites. She was discharged in a stable condition on oral therapy, advised regular follow-up, and planned for definitive surgical management with pericardiectomy which the parents denied.

DISCUSSION

Any cardiac condition resulting in hepatocyte damage is known as cardiac cirrhosis. It is possibly thought to occur due the following mechanism responsible: hepatic venous congestion, decreased hepatic blood flow and hypoxemia followed by atrophy, hepatocyte necrosis, thrombi resulting from cholestasis. Constrictive pericarditis is an uncommon cause of liver cirrhosis in children but is important to recognize due to its potential reversibility with timely intervention. Prolonged hepatic venous congestion from this hemodynamic disturbance can lead to congestive hepatopathy and ultimately secondary cirrhosis. The clinical overlap of symptoms of cardiovascular and hepatobiliary system often delay diagnosis. [3] Our index case is one such scenario wherein long standing constrictive pericarditis led to chronic liver disease. In paediatric patients, constrictive pericarditis may arise from infections such as

tuberculosis, prior cardiac surgery, autoimmune disorders, connective tissue diseases, radiotherapy, neoplasms or may be idiopathic. Child was previously treated by various practitioners with diuretics. Later she developed decompensated liver failure and right heart failure manifesting as ascites and engorged neck veins, hence a proposed mechanism wherein long standing right heart failure leading to liver cirrhosis was thought. In our case, cause of the pericarditis was not identified, however the child had prolonged low-grade fever, reactive mantoux, had no history of tubercular contact, hence a possibility of pericarditis of tubercular origin was thought and anti-tubercular therapy was initiated. Nahid et al, reported a case of an 11-year-old with constrictive pericarditis developing chronic right heart failure due to markedly elevated filling pressures of the ventricles, leading to congestion of hepatic vein, further leading to ischaemia and eventually fibrosis. Paediatric constrictive pericarditis is rare, with most cases, including those by Nahid et al., arising post-infection or post-surgery and showing a relatively straightforward course. In contrast, our patient presented with chronic intermittent abdominal distension over four years, advanced hepatic fibrosis, and effusive-constrictive physiology, complicating the diagnosis. [4] The combination of severe ascites, portal hypertension, and Kayser-Fleischer rings, along with a favourable response to anti-tubercular therapy and steroids, makes this case distinct from previously reported paediatric cases. This highlights the importance of a comprehensive, multidisciplinary approach in atypical presentations of paediatric constrictive pericarditis. [5] Constrictive pericarditis, though rare in children, should be considered as a differential diagnosis of unexplained decompensated liver disease, especially when signs of right-sided heart failure coexist. Our case highlights the importance of thorough cardiovascular evaluation in Paediatric patients presenting with features of hepatic dysfunction. Erdal et al, reported a similar case in a 21-year-old with constrictive pericarditis who was initially diagnosed as cirrhosis, hence early recognition and timely surgical intervention can reverse hepatic congestion and irreversible

hepatocyte damage, further preventing progression to irreversible cirrhosis, and significantly improve long-term outcomes. [6]

Liver dysfunction in constrictive pericarditis primarily results from congestive hepatopathy, where elevated central venous pressure impedes hepatic venous drainage, leading to portal hypertension, ascites, and hepatomegaly which is often reversible following pericardiectomy, the definitive surgical treatment **[Fig 7]**. [7]

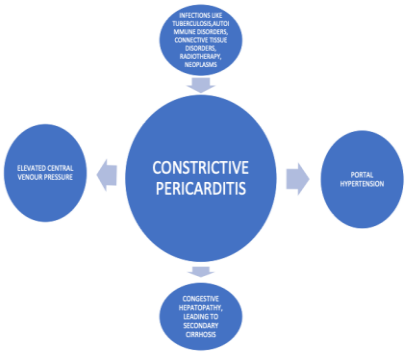

Fig 7: Liver dysfunction in constrictive pericarditis primarily results from congestive hepatopathy, where elevated central venous pressure impedes hepatic venous drainage, leading to portal hypertension, ascites, and hepatomegaly

The improvement in liver function post-pericardiectomy is attributed to the relief of elevated central venous pressure, leading to decreased hepatic venous congestion, allowing for improved hepatic blood flow, reduction in portal hypertension. The importance of early recognition lies in the fact that hepatic injury may be partially or fully reversible with surgical intervention. [8,9] Hence, constrictive pericarditis should be considered in any child with unexplained decompensated liver disease cirrhosis. Constrictive pericarditis and cardiac cirrhosis, although uncommonly seen in paediatric population, should be kept as a possibility in any child presenting with decompensated liver disease. Early identification and intervention is the key to prevent irreversible hepatocyte damage.

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