

Case Report

Diagnostic dilemma in a child with bilateral empyema and pyopericardium: a case report

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ABSTRACT

Tubercular pericardial effusion in children is a rare, severe form of extrapulmonary TB, often presenting with fever, cough, tachycardia, and dyspnea, with a high incidence of cardiac tamponade. Diagnosis relies on echocardiography and pericardial fluid analysis, with treatment involving anti-tubercular therapy (ATT) and pericardiocentesis. Here we present the case of a child with bilateral pleural effusion and pericardial effusion, which was clinico-radiologically proven to be tubercular in origin and required pericardiocentesis to manage the impending cardiac tamponade.

Keywords: Pericardial effusion, Tuberculosis, Tamponade, Pericardiocentesis

INTRODUCTION

Pericardial effusion is defined as the evidence of fluid accumulation more than the normal physiological state in the fibroelastic sac of pericardium.¹ Purulent pericarditis with cardiac tamponade in children, though rare, constitutes a potentially lethal disease warranting prompt recognition and aggressive management. Pyopericardium increases intra-pericardial pressure, causing compression of all cardiac chambers, limiting cardiac inflow, and a resultant marked fall in cardiac output and culminating in cardiac tamponade, cardiogenic shock and death.² Etiologies include infectious (bacterial, tubercular, viral), non-infectious (autoimmune, malignancy, traumatic, post-operative) and idiopathic.³⁻⁷ Pericardial effusion, coupled with pleural effusion, represents a diagnostic challenge for paediatricians, considering that the typical clinical features may not correlate with the severity of the effusion and radiological diagnosis is sometimes necessary. We hereby report the successful management of a rare case of bilateral pleural effusion with purulent pericardial effusion and impending cardiac tamponade in an 8-year-old child with multidisciplinary management.

CASE REPORT

An 8-year-old female child, previously well, was brought to the paediatric emergency with chief complaint of fever for 15 days, abdominal distension noticed by parents since last 6 days and chest pain and breathlessness for past 3 days. As per the parents, the child had history of fall 15 days back after which she developed swelling and pain in the right shoulder joint, associated with restriction of movements of right upper limb.

The swelling increased over next 2 days and child also developed fever (moderate grade). She was taken to a local practitioner where pus was aspirated from the right shoulder joint but no investigations were done for the aspirated purulent fluid. She was started on oral antibiotics and analgesics. 5 days later, patient developed abdominal distension which was gradual in onset and progressive. She also developed chest pain and breathlessness, which was gradual in onset, initially limiting her routine physical activity but later progressed to orthopnea, after which she was brought to our emergency.

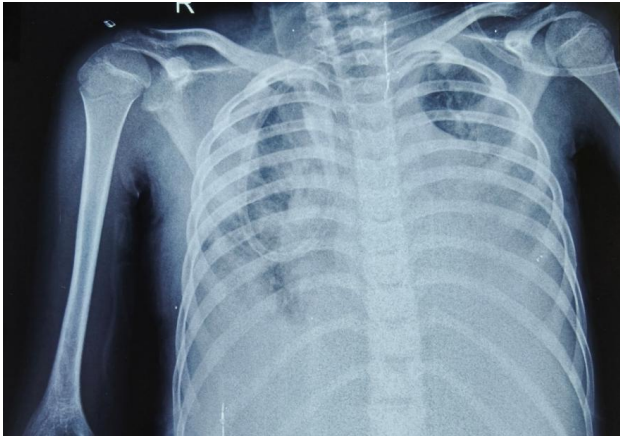


Figure 1: Chest X-ray on admission shows blunting of bilateral costo-phrenic angles along with cardiomegaly.

Table 1: Routine investigations.

S. no.	Investigations	Results
1	Hb	7.1
2	TLC	23400
3	DLC	82% neutrophils, 13% lymphocytes
4	Platelets	4.8 lac
5	TSB/DSB	0.8/0.2
6	SGOT	49
7	SGPT	31
8	ALP	215
9	HBSAG	Non-reactive
10	HCV	Non-reactive
11	HIV	Non-reactive
12	CRP	46
13	Blood C/S	No growth
14	Montaux	Induration 12 mm (positive)
15	Gastric lavage CBNAAT	No MTB detected

There was history of tuberculosis contact in the neighbourhood, who was currently on anti-tuberculosis therapy. On admission, the child preferred sitting to lying down position. She was a moderately built child with weight and height corresponding to 50th centile. Vitals on admission showed tachycardia (HR=118/min), tachypnea (RR=42/min), fever (101°F), normal BP (108/68 mm Hg) and saturation was 92% on room air. There was respiratory distress in form of nasal flaring and intercostals and subcostal retractions.

On physical examination, she had pallor, no icterus, cyanosis, clubbing, lymphadenopathy or edema over any body part. Jugular venous pressure was normal. On respiratory examination, there was decreased air entry and dull percussion note in bilateral infra-mammary, inferior axillary and infra-scapular areas. On CVS

examination, heart sounds were normally heard with no pericardial rub. Per abdomen examination revealed a non-tender hepatomegaly with a liver span of 16 cm. Patient also had restriction of movements of right shoulder joint beyond 30° abduction, although there was no obvious swelling.

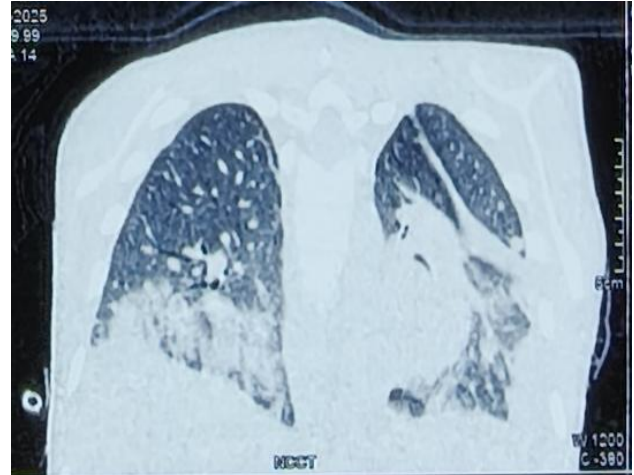


Figure 2: CECT chest shows consolidation in lower lobes of both lungs with cavitary lesions in left upper lobe.

Table 2: Pleural fluid analysis.

S. no.	Investigations	Results
1	Cytology	300 cells (85% lymphocytes, 15% neutrophils, many mesothelial and reactive mesothelial cells)
2	Glucose	71 mg/dl
3	Proteins	3.3 gm/dl
4	Gram stain	No gram-positive cocci or gram-negative bacilli seen
5	ZN stain for AFB	No AFB seen
6	C/S	No growth of pyogenic organisms
7	ADA	75 IU/l
8	CBNAAT	No MTB detected

She was started on supplemental oxygen via HFOM and empirical antibiotics ceftriaxone and vancomycin (suspecting septic arthritis leading to complicated pneumonia), after sending septic workup and other routine investigations.

ECG was done which showed low voltage QRS complexes. X-ray and USG of right shoulder joint did not show any joint space widening or bony destruction. Orthopedic consult was taken and conservative management with limb physiotherapy was advised. Chest X-ray showed bilateral pleural effusion with cardiomegaly. Pleural tap was done which revealed straw coloured free flowing fluid.

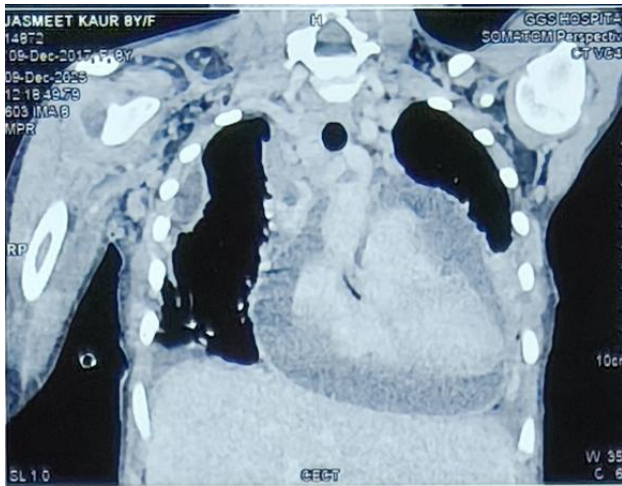


Figure 3: CECT chest showing large pericardial effusion.

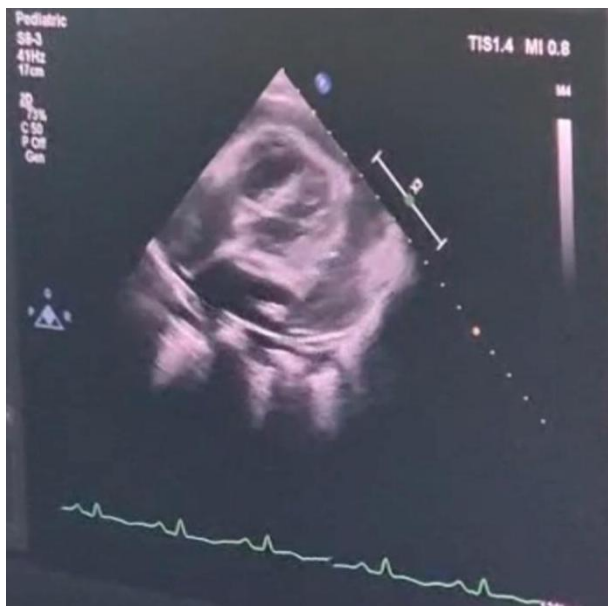


Figure 4: 2D echocardiography shows a large circumferential pericardial effusion (ECHO free space around heart in diastole=2.1 cm) with normal ejection fraction.

In view of severe respiratory distress, bilateral tube thoracostomy was done after consulting paediatric surgeon and 300-400 ml sero-sanguinous fluid was drained bilaterally and sent for analysis.

Post tube thoracostomy, patient's distress improved slightly but orthopnea persisted. CECT chest was done which revealed consolidation in lower lobes of both lungs, along with thick walled cavitary lesions in left upper lobe, significant pericardial effusion and multiple enlarged (largest 10x9 mm) lymph nodes in mediastinum. Chest and TB consult was taken and patient was started on first line anti-tuberculosis therapy as clinico-radiologically diagnosed disseminated tuberculosis.



Figure 5: Fibrin strands seen in pericardial effusion.

Table 3: Pericardial fluid analysis.

S. no.	Investigations	Results
1	Cytology	Dense inflammatory cell infiltrate comprising of degenerated polymorphs, lymphocytes and macrophages, no atypical cells
2	Malignant cells	Negative
3	Glucose	32 mg/dl
4	Proteins	5.3 gm/dl
5	Gram stain	Few pus cells, no gram positive or negative organism seen
6	ZN stain	Negative
7	C/S	No growth of pyogenic bacteria
8	CBNAAT	MTB detected

2D echocardiography was done in M-mode which showed a large circumferential exudative pericardial effusion with fibrin strands. There was also collapse of right atrium during early diastole and distended inferior vena cava, showing signs of impending cardiac tamponade. Urgent pericardiocentesis was planned in view of cardiac tamponade. Multi-hole soft 7F pigtail catheter was advanced into the pericardial sac under local anaesthesia by subxiphoid approach. Around 300 ml of cloudy, non-bloody pericardial fluid was suctioned from catheter tip. The drained pericardial fluid was sent for analysis. After pericardiocentesis, patient showed dramatic improvement in respiratory distress and orthopnea and was weaned off oxygen support in next 48 hours. Repeat ECHO was done after 3 days which showed mild pericardial effusion (anterior rim=0.8 cm,

posterior rim=0.7 cm) with non-distended inferior vena cava.

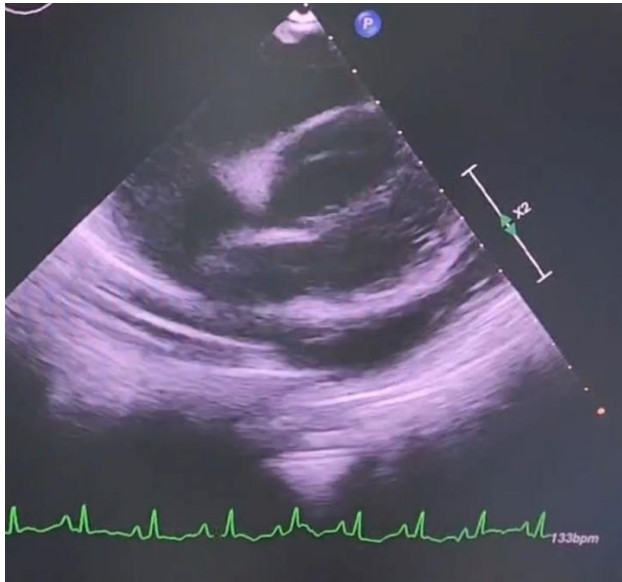


Figure 6: Repeat ECHO after 3 days shows mild pericardial effusion.

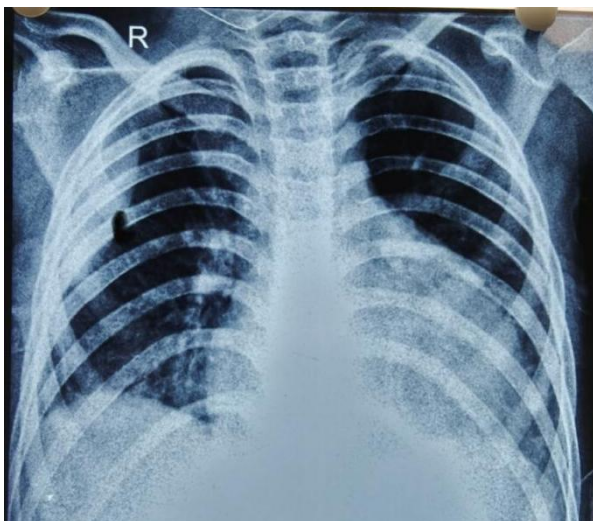


Figure 7: Chest X-ray on discharge shows resolution of pleural effusion with mild cardiomegaly.

Antituberculosis therapy along with steroids was continued and patient was discharged after completing 14 days of intravenous antibiotics. At the time of discharge, the air entry was improved bilaterally and decrease in liver span was noticed to 12 cm.

DISCUSSION

Pericardial effusion is a rare presentation of extra-pulmonary tuberculosis.⁸ In the study conducted by Bagri et al, tuberculosis was the most common cause of pericardial effusion seen in 52% of the cases, followed by bacterial in 23% of the cases. With the advent of

antibiotic therapy, there is a decline in bacterial etiology and most frequent causes are presumed to be viral in developed and tuberculosis in developing countries.

Tuberculosis with pericardial effusion is caused by spread of mycobacterium tuberculosis infection in the pericardium hematogenously, through lymphatics or direct spread occurring even without primary pulmonary infection.⁹ In our patient, there were cavitary lesions in the lungs along with presence of significant mediastinal lymphadenopathy, which maybe the primary focus of tuberculosis. The simultaneous presence of several sites of extra-pulmonary tuberculosis is an uncommon finding in the paediatric population, with few case studies of pleural and pericardial effusion in children under 15 years of age.^{10,11}

Clinical presentation of tuberculous pleural and pericardial effusion is severe in the form of fever, cough, chest pain, tachycardia, tachypnea and signs of heart failure and cardiac tamponade.¹² Although the diagnosis of cardiac tamponade maybe suspected clinically in a child with classic Beck's triad (muffled heart sounds, distended neck veins and hypotension), but these findings are both late and inconsistent, with fewer than 30% patients exhibiting full triad on diagnosis.¹³ Our patient did not have any of the classic features of Beck's triad.

Diagnosis of tuberculosis is suspected based on the high endemicity in our country, the typical clinical presentation, the ancillary tests like Montoux and microbiological and radiological confirmation. Mediastinal lymphadenopathy is the hallmark of primary pulmonary tuberculosis in children, but it may be missed on routine X-ray chest, thus necessitating the need for contrast enhanced CT chest.¹⁴ In our patient also, microbiological confirmation could not be achieved but CECT chest showed evidence of tuberculosis.

Due to its paucibacillary nature, tuberculous pleural effusion is difficult to diagnose with only laboratory tests and requires invasive procedures like thoracentesis. There is a 10% yield of MTB from pleural fluid smears and 25-85% yield from pleural fluid cultures.¹⁵ Exudative lymphocyte predominant pleural fluid, combined with increased ADA levels, strongly suggests tuberculous etiology.

Echocardiography has both diagnostic and therapeutic roles in suspected tuberculous pericardial effusion. It typically shows large effusions with fibrin strands, thickened pericardium and increased fibrinous exudative coatings.¹⁶ In our case, there was a large pericardial effusion with fibrin strands which goes in favour of tuberculous etiology. Echo guided pericardiocentesis has a well-established therapeutic role in preventing cardiac tamponade and evolution of constrictive pericarditis.

Management includes first line ATT to be started as soon as the diagnosis is confirmed clinically or radiologically,

along with steroids for a period of two months to prevent the occurrence of constrictive pericarditis.

CONCLUSION

This case study highlights the need for having a low threshold of clinical suspicion for tuberculosis in a child presenting with concomitant bilateral pleural and pericardial effusion. Microbiological confirmation cannot be achieved in all cases, thus necessitating the role of radiology to clinch the diagnosis. Both surgical (pericardiocentesis) and medical management (antibiotics, ATT and steroids) are needed to prevent long term morbidity and mortality in cases of cardiac tamponade.

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