Cardiac Complication of Extrapulmonary Tuberculosis

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Abstract

Among communicable diseases, tuberculosis (TB) is the major cluster causing mortality in young patients. WHO reported a global estimation of 10 million people fell ill with tuberculosis (1.5 million fatalities in 2020), and India alone contributed about26%.The incidence of Extra pulmonary Tuberculosis (ETB) is about 15% of all TB cases in India. Central nervous system (CNS) tuberculosis accounted for approximately 1% of total tuberculosis. TB is a rapidly progressive and fatal disease if left untreated. The progression of the disease and the occurrence of complications vary from patient to patient. Here the authors present a case of a young adult who presented with acute onset of fever and headache and she expired within a span of 31 days after the symptoms began. The novelty in the case is that an undiagnosed rare cardiac complication of extra pulmonary tuberculosis was diagnosed at autopsy. The investigations prior to her death didn't indicate TB, but the treating physician had suspected ETB. The autopsy examination and histopathological examination supported the physician's diagnosis. The authors had deliberated the pathogenesis of the cardiac complication of extra pulmonary tuberculosis contributing to the death of the deceased.

Keywords: Extra pulmonary tuberculosis, Tuberculous meningitis, Tuberculous pericarditis, Anti tubercular therapy, Mediastinal lymphadenopathy.

INTRODUCTION

India contributes about one-fourth of the total population of tuberculosis cases worldwide. The most common type of tuberculosis is pulmonary tuberculosis. The incidence of ETB is about 15% of all TB cases in India while central nervous system tuberculosis (CNS-TB) is considered a rare presentation in young adults.[1-3] CNS-TB is a type of extra pulmonary tuberculosis that accounts for approximately 1% of TB cases.[3] Tuberculosisis commonly caused by Mycobacterium Tuberculosis that remains latent inside the body for a long period. This latent or inactive mycobacterium remains silent and highly stabilized. Even though the organism had colonized the extra pulmonary organs, the affected patients exhibit minimal symptoms. In the event of immunosuppression, latent tuberculosis is triggered thereby exhibiting nonspecific symptoms. This creates confusion for the treating physicians to initiate the appropriate treatment considering the side effects of the anti-tuberculosis drugs since the clinical investigations are highly unreliable in the initial changes.[4] This is one of the reasons for less reporting of TB complications even though it has a high mortality rate due to delay in arriving at a diagnosis. In autopsies, residual changes like scars and limited small calcified foci or abscesses are observed. Such findings are seen when a case is registered as a medico legal case (MLC) due to the sudden death of the patient. The authors report a case that presented with sudden onset altered sensorium and expired within a span of 31 days. The novelty in the case is to highlight the mismatch between the lab investigation (biochemical, microbiological) and the clinical presentation. At

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autopsy, the gross findings and histopathological examination supported the treating physician's diagnosis. The present case reinforced the fact that rapid progression, delayed presentation, and confusion in arriving at the diagnosis of tuberculosis could lead to mortality in patients.

CASE REPORT

A recently married 23 years old female, presented with a history of weight loss for a few months. Later, the patient got admitted to a private hospital after sustaining a fall due to a sudden collapse due to an altered sensorium while working at her house. General examination and systemic examination were normal. Hence, symptomatic treatment was started along with clinical evaluation by sending samples for relevant tests.

CLINICAL FINDINGS

On clinical evaluation; the serological parameter was within normal limit except for mild anemia (Hb 11.4 gm/dl), lymphopenia (16.6%; Normal 20-40%), and elevated ESR level(30 mm in 1st hour). Dengue NS1 antigen assay, malaria parasite, Antinuclear antibody (ANA), and Anti Double-Stranded DNA (Anti Ds DNA) were negative. She was positive for antibody to S. Paratyphi A on the Widal test. RT-PCR for SARS-COV-2 RNA was negative from the nasopharyngeal swab. Urine showed normal routine and negative culture and sensitivity test. The blood smear showed no acid-fast bacilli on Zein Nielsen staining. The Cerebrospinal Fluid analysis was negative for Cryptococcus with India Ink staining and Gene XPERT (NAAT) was negative for Mycobacterium Tuberculosis.

The Cerebrospinal Fluid was turbid with mild neutropenia and lymphocytopenia, high protein (192 mg/dl; Normal 15 – 45 mg/dl), low glucose (17.93 mg/dl; Normal 40-70 mg/dl), elevated LDH (43 U/L; Normal less than 40) and normal ADA (5.5 U/L; Normal less than 9 U/L) level. The CSF culture for bacterial growth was negative and no resistance was detected for rifampicin. The chest X-ray was normal. MRI brain showed areas of meningeal inflammation likely meningoencephalitis. There were focal areas of diffusion restriction seen in bilateral basal ganglia suggestive of acute patchy infarcts. EEG showedan abnormal awake study suggestive of diffuse cerebral dysfunction with intermittent generalized epileptic form discharges.

Antiviral and empirical anti tubercular therapy (ATT) was started four days after admission and a high dose of pulse steroid was given simultaneously based on the clinical evaluation. Neurological status improved marginally following two days of treatment. She was discharged on advised to take Anti Tubercular drugs regularly. After 11 days of discharge, she was brought dead to the hospital and hence a medico-legal autopsy was conducted as per the relevant provisions of CrPC and IPC.

AUTOPSY FINDINGS

The deceased was a poorly nourished and thin-built adult female weighing 42kg. There was multiple yellow-colored purulent collections present involving the interpeduncular fossa and periventricular region of the base of the brain (fig 1a, 1b & 1c). The costal surface of the right lung was partially adherent to the chest wall. A white-colored mass firm in consistency measuring 5 cm X 3 cm X 2 cm was present in the hilar region under the bifurcation of the trachea (fig 2). The pericardial sac was adherent to the anterior surface of the left ventricle and showed flakes of white-colored pus (fig 3a). The histopathological examination of the pericardium and heart showed thickened pericardium with caseous necrosis of the myocardium (fig 3b). The cause of death was given as extra pulmonary tuberculosis and its complications.

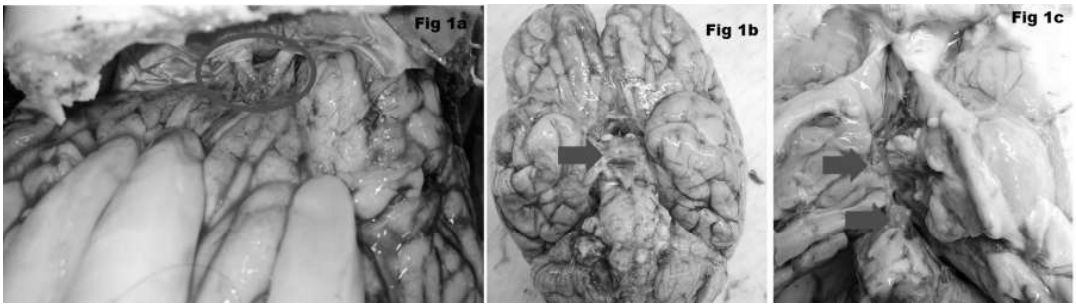


Fig 1a: Yellow-colored pus in the interpeduncular fossa surrounding the optic chiasma.

Fig 1b: Yellow-colored pus over the mamillary body.

Fig 1c: Yellow-colored pus in the fourth ventricle.

Fig 2: Hilar Lymphadenopathy at the level of bifurcation of the trachea.

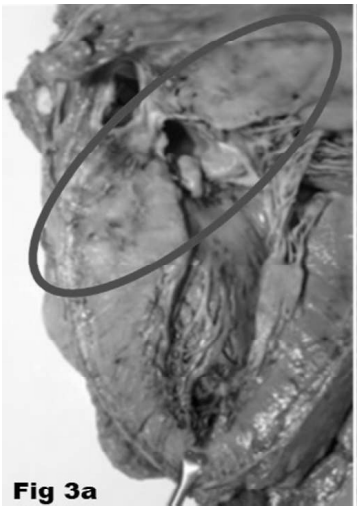


Fig 3a: 10% formalin-fixed specimen of the heart showing the affected myocardium along the entire length.

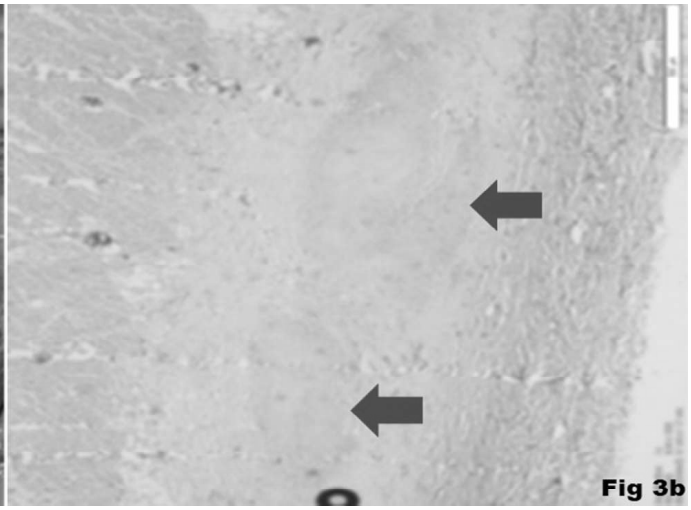


Fig 3b: Inflamed and Thickened pericardium with underlying granulomatous inflammation of the myocardium (red arrows). [H & E staining, 10x]

DISCUSSION

TB is a multisystem disease with innumerable 'presentations varying' from individual to individual and high mortality among young age individuals. It can affect almost any organ or tissue and less frequently involves hair and nails.[5] The survival period is variable and various factors influence the same. Immunosuppression is considered a triggering factor to supervene latent TB. The mean survival days of patients who died due to TB was 53.6 days (range 1–229) and about 50% of the patients died within the first 32 days as concluded by Erbes *et al*.[4] A study conducted in North Taiwan concluded most of the TB-related deaths occurred within a median survival of 20 days.[5] The survival time in the present case was 16 days since the first day of her altered sensorium. The diagnosis of Tuberculous Meningitis (TBM) clinically could be made after considering the following factors as stated by Abdelmalek et al.[6] a

1 Signs and symptoms suggestive of subacute meningitis where CSF glucose levels are low with a good response to specific anti-tuberculous treatment;

2 A positive result after evaluation of the smear for acid-fast bacilli and/or positive CSF or sputum culture for Mycobacterium tuberculosis

3 The radiological findings support TBM on cerebral MRI and/or cerebral CT in a case with lymphocytic subacute meningitis.

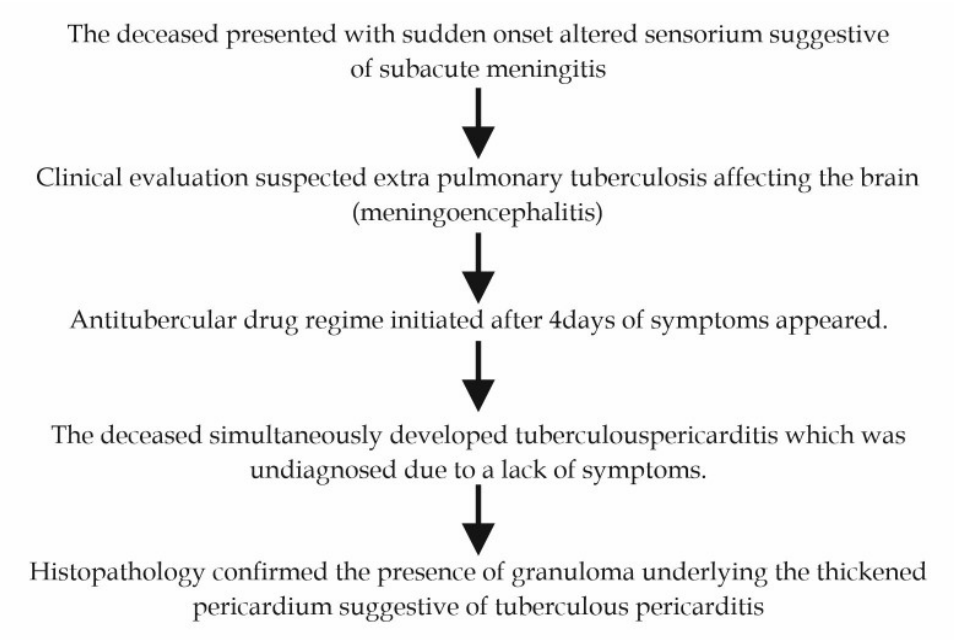
Kent SJ et al. concluded that the clinical presentation mostly is subacute with cranial nerve palsies, hemiparesis, and impaired consciousness.[7] In the present case, there were nonspecific presenting complaints like weight loss for months and sudden onset of altered sensorium, and the clinical evaluation suggested a scenario similar to subacute meningitis. The MRI brain confirmed the presence of meningeal inflammation and thereby, diagnosing meningoencephalitis. Thus, the present case abided by criteria 1 and 3 mentioned by Abdelmalek et al. except for the smear-positive.

Pericardial infection with Mycobacterium tuberculosis occurs as an extension of infection from the lung or tracheobronchial tree, adjacent lymph nodes, spine, sternum, or via the miliary spread. The primary focus of infection is rarely found as it occurs due to the reactivation of the disease. There are four pathological stages of tuberculous pericarditis according to studies.[8,9]

1. Fibrinous exudation with polymorphonuclear leucocytosis, abundant mycobacteria, and early granuloma formation with the loose organization of macrophages and T cells
2. Serosanguineous effusion with lymphocytic exudate and high protein concentration; tubercle bacilli present in low concentrations
3. Absorption of effusion with granulomatous caseation and pericardial thickening with subsequent fibrosis
4. Constrictive scarring; fibrosing visceral and parietal pericardium contracts on the cardiac chambers and may become calcified, leading to constrictive pericarditis, which impedes diastolic filling.

Tuberculous pericarditis progresses from one phase to the next. The initial phase is identified by biopsy or autopsy. The biopsy findings are isolated granulomas in the pericardium as seen in the present case. The visceral pericardial thickening is due to healing with fibrosis and calcification, as a result of treatment with anti-tubercular drugs. This can cause constriction of pericardium, and the collection of the pericardial fluid inside the pericardial cavity leads to cardiac tamponade.[10,11] However, extrapulmonary complications of TB involving the heart are less commonly observed with only 0.5%. The common finding observed is tuberculous pericarditis which manifestsas pericardial thickening (>3mm), often associated with mediastinal lymphadenopathy (LAN).[12] The present case depicted hilar lymphadenopathy and pericardial thickening which was later confirmed as pericarditis in the histopathology examination. This finding is less commonly reported and is of academic interest.

FLOW CHART OF PATHOGENESIS IN THE PRESENT CASE



CONCLUSION

This case report highlights the atypical presentation of extra pulmonary tuberculosis. Since the disease doesn't have a unique presentation, the initiation of treatment is purely based on suspicion. This will help the treating physicianto better understandthe various presentations of the disease so as to initiate the appropriate treatment avoiding the progress of the disease. The administration of appropriate treatment subsequently helps in reducing the mortality rate due to the tuberculosis.

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