



Case Report

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## A Rare Case Presentation: Tubercular Empyema Necessitans with Poliomyelitis

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### **Abstract**

*54 years old male; non-smoker; non-alcoholic; known case of seizure disorder, left hemiplegia, bronchial asthma presented with decreased appetite since 2-3 months, chest pain since 2 months, breathlessness since 5-7 days. The patient has had a history of bronchial asthma since childhood – on irregular treatment – no documents available with the patient. The patient had a history of seizures and left hemiplegia - 5 years back – on irregular treatment, no documents available. The patient also had a history of rib fractures due to trauma 2 months back. On detailed examination - the patient had scoliosis, multiple chest wall swellings, and rib fracture. The patient was diagnosed with a case of tubercular empyema with rib osteomyelitis with scoliosis. Empyema was drained with an intercostal drainage tube and was started on anti-tubercular therapy. Lung expanded and the patient was discharged on anti-tubercular therapy.*

### **Introduction**

Extrapulmonary tuberculosis (EPTB) carries a high burden which ranges from 15– 20% of all HIV-negative TB cases. The burden is still high in HIV-positive people and accounts for 40-50% of new TB cases [1]. Pleural disease is one of the most common extra-pulmonary involvements in TB in developing countries [1, 2]. Tuberculosis empyema is an entity distinct from and much less common than, TB pleural effusion. Tuberculosis empyema represents nearly 20 percent of all cases of empyema seen in high prevalence countries like India [3].

Empyema necessitates (EN) is a kind of empyema that diffuses to extrapleural space and can involve chest pain. TB is the most common cause of EN. EN usually presents as a single mass with or without pain on the chest wall; diagnosis is based on clinical view and radiologic imaging and confirmation are by smear, culture, and PCR from fluid aspiration. The treatment is a combination of drainage and standard anti-TB treatment.

### **Case Report**

54 years old male who is a non-diabetic; normotensive; non-smoker; non-alcoholic; known case of bronchial asthma and seizure disorder with left hemiplegia presented to Pulmonary Medicine OPD with complaints of decreased appetite since 2-3 months, chest pain since 2 months and breathlessness since 5-7 days. There is no associated history of fever, hemoptysis, orthopnea, and paroxysmal nocturnal dyspnea. The patient has had a history of bronchial asthma since childhood and the patient is taking

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irregular treatment but no documents are available with the patient. The patient has a history of seizures and left hemiplegia 5 years back for which he was taking treatment irregularly and no documents are available with the patient. The patient also has a history of rib fracture 2 months back due to trauma. On general physical examination, the patient was lean-built and had pallor and clubbing. Bilateral pitting pedal edema was also present along with bedsore grade-2.

On systemic examination, the patient had scoliosis with three swelling presents in the anterior chest wall which were of variable sizes, the round, well-defined, tender, local temperature was raised, mobile, not attached to the overlying skin or underlying structures. The patient had absent breath sounds on the left side. Digital chest x-ray was suggestive of pleural effusion. Diagnostic pleural fluid aspiration was done which revealed pus. An intercostal drainage tube was inserted on the left side in the mid-axillary line in the 5th intercostal space and the patient was started on a double I.V antibiotic regime. The pus aspirated was sent for culture and sensitivity, acid-fast bacilli staining, and cartridge-based nucleic acid amplification test (CBNAAT). Mycobacterium tuberculosis (Mtb) was detected in pus CBNAAT and was found to be rifampicin sensitive. Mtb was also confirmed by culture. The patient was started on anti-tubercular therapy (HRZE), a fixed-dose combination under the directly observed treatment short-course (DOTS). The patient tolerated ATT well. A total of 2130 ml of pus was drained from the left pleural cavity. All three chest wall swellings were drained separately for pus. The intercostal drainage tube was removed after 19 days. The patient was discharged in stable condition with anti-tubercular therapy for 6 months. The patient has been advised a regular follow-up after 1 month.



Figure 1: Left sided Hemiplegia



Figure 2: Chest swelling with scoliosis

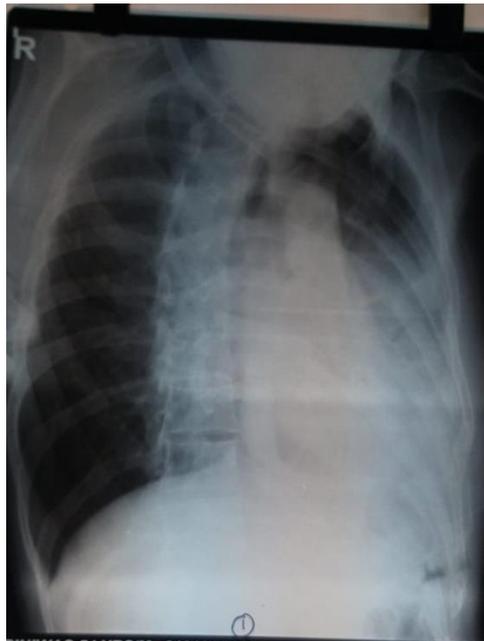


Figure 3: Left sided Empyema with scoliosis



Figure 4: Left sided Empyema with Inter costal drainage tube in situ

### Discussion

Chest wall abscesses are a rare manifestation of extrapulmonary tuberculosis [4,5]. Abscess arises due to chronic empyema which does not resolve and leaks into the chest wall. When the empyema leaks into the chest wall which is a rare complication, it is called Empyema Necessitans [6]. Usually, it presents with a single swelling in the chest wall but sometimes the patient can present with multiple masses involving bones, rib cage, and muscles [7, 8, 9, 10, 11]. Computed tomography (CT) of the thorax is pathognomonic for diagnosis of Empyema Necessitans. The sign is the connection of pleural effusion with an extrapleural mass of the chest wall [12]. Underlying lung parenchymal pathologies can also be diagnosed with the help of CT. However, histopathological confirmation is indicated [13]. Elucidation of etiological factors can be challenging especially in the case of mycobacterium tuberculosis as acid-fast smear, fine needle aspiration (FNA), and PCR can yield false-positive results [14].

However, PCR can be considered a rapid and reliable diagnostic modality but its limited availability is another roadblock in establishing the etiological factor. Surgical histologic samples can also have false-negative results, and only 20% of cases yield definite diagnoses [15].

Pneumothorax, empyema, and pyopneumothorax are complications of pulmonary tuberculosis (TB), whilst infrequent but leading to significant morbidity and mortality [21, 22]. Tuberculous pleural effusion originates from infection of the pleura by Mycobacterium tuberculosis which is characterized by fluid accumulation and the presence of chronic inflammatory cells in the pleural cavity [23]. Tuberculous empyema occurs through the same mechanism but is often accompanied by more severe lung parenchymal lesions [21]. Empyema could be related to ineffective treatment as a result of anti-

tuberculosis drugs (ATD) inadequately penetrating the thickened pleura due to fibrin deposits. Other rare complications include purulent pericarditis, peritonitis, pulmonary abscess.

The other differential diagnosis that can be considered is infection from *Staphylococcus aureus*, *Klebsiella*, noninfectious diseases like primary lung neoplasm should also be considered. A tissue biopsy can give the ultimate diagnosis [16, 17]. The pus should be completed from every involved tissue. Removal of all involved tissues such as bones and cartilages is an assured and safe approach but sometimes spreads the infection [9, 18]. The mortality rate for decortication to improve lung function is about 5%. Relapse of infection is due to incomplete excision of ribs or infected pleura, which can take place 10 years after the surgery. These patients should be closely followed-up [19, 20]. Antitubercular therapy along with surgery wherever indicated forms the cornerstone of the treatment. This prevents recurrence and reduces morbidity and mortality associated with the disease.

### **Conclusion**

High clinical suspicion of tuberculosis presenting as anterior chest wall swellings is required especially in high burden countries like India. Early diagnosis and complete treatment completely cure the patient and prevent relapses.

Knowledge of such cases is crucial since a high index of suspicion is key to adequate management. The absence of respiratory and constitutional symptoms does not rule out TB. EN is indeed a possible complication of TB-related empyema in all ages. Surgical drainage along with ATT is the most effective method of treatment for EN. The most challenging part is to identify disease EN.

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