Pediatric Tubercular Empyema Thoracis: A Diagnostic Dilemma

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Abstract:Empyema thoracis is defined as the collection of pus in the pleural cavity. Mycobacterium tuberculosis as an etiological agent accounts for only 2% of the tuberculous pleurisy cases. History of contact and absence of Bacillus Calmette-Guerin(BCG) vaccination scar are usually seen. We are hereby reporting, a 4 year old male child with left sided tuberculous empyema who had been adequately immunized. There was no history of tuberculosis in the family, past infection or contact with an adult case.Our case was managed conservatively with a favourable outcome.

Keywords: Tuberculous Empyema, Pediatrics, hypersensitivity reaction.

I. Introduction:

The overall burden of tuberculosis in a developing country like India, is around 10.2% ^[1],where paediatric population constitutes about 34% of the total population. There is often a diagnostic challenge to differentiate tuberculous from non-tuberculous empyema. Mycobacterium tuberculosis alone as a causative organism of empyema is rare^[2]. Unilateral pleural effusion usually develops 6-12 weeks after the primary infection. It is usually a hypersensitivity reaction to the tubercular antigen. Tubercular pleural effusions are usually unilateral exudative effusions with right sided predilection^[3].

II. Case Report:

A 4 year old male child hailing from Uttar Pradesh, India, presented to a tertiary care hospital with complaints of gradual onset of cough, coryza, fever and breathlessness since 10 days. Fever was high grade, associated with dry, non-paroxysmal, non-productive cough and breathlessness, which was increasing over the past few days. He was an immunized child with no past history of tuberculosis. There was no history of tuberculosis in the family and the child belonged to a low socioeconomic status. The child had been referred to our hospital, as the symptoms progressed on injectible antibiotics in the form of amoxicillin with clavulanic acid and ceftriaxone for last three days.

On examination, child was sick looking, moderately built and nourished. Vital parameters were, a heart rate of 128/min, respiratory rate of 60/min, with subcostal and intercostal retractions. The temperature was 102°F, with bilateral, discrete, non-tender, matted cervical lymphnodes measuring 1.5cm x 1.5cm. There was pallor with no other stigmata of tuberculosis on general physical examination. Respiratory system revealed, shift of trachea on the right side, decreased movements with stony dullness, decreased vocal fremitus and diminished air entry in the left infraclavicular, inframammary, infraaxillary and infrascapular areas.

On investigations, Complete Blood Count showed, hemoglobin : 9.3 gm/dl, total leucocyte count: 16.2/cmm, with neutrophils of 65% , lymphocytes of 20% , platelets of 212×10^{3} /µL suggestive of neutrophilicleucocytosis. Chest x- ray postero-anterior view revealed complete left sided opacity with mediastinal and tracheal shift to the right side. Obliteration of the cardiophrenic and costophrenic angle was also noted(Fig.1). Ultrasonography(USG) of the chest confirmed left sided moderate to severe pleural effusion with thin septations and no loculations. An ultrasound guided thoracocentesis revealed turbid pleural fluid with ph–7.1, proteins-5gm/dl, sugar- 40gm/dl, total counts- 100 cells/cumm, with neutrophils -80% and lymphocytes-20%. On basis of the above investigations and clinico-radiological deterioration, it prompted us to step up the antibiotics which were vancomycin and ceftazidime. Insertion of intercostal drainage tube was performed which drainedpus(Fig.1). The child was also started on maintenance intravenous fluids along with symptomatic treatment.Pleural fluid and blood cultures did not show any growth.

After 72 hours of the above antibiotics, there was no symptomatic improvement, hence the child was investigated on the lines of tuberculosis. Mantoux test was positive (20mm x 20 mm) and gastric lavage samples did not demonstrate any acid fast bacilli. Erythrocyte sedimentation rate was 3mm/hour, screening test for HIV

was non-reactive and pleural fluid for Adenosine deaminase(ADA) was positive, the levels being 78 U/L.Computed-tomography (CT) thorax with contrast revealed fluid, atelectasis with no evidence of mediastinal lymphadenopathy.

The child was started on four drug anti-tubercular daily regimen comprising of Isoniazid(H)-5mg/kg/day, Rifampicin(R)-10mg/kg/day, Pyrazinamide(Z) 25mg/kg/day and Ethambutol(E) 15mg/kg/day for 6months. In addition to this, child was started on steroids i.e prednisolone (1mg/kg/day) for six weeks. Intercostal drainage tube was removed after 3 days, as there was less than 30 ml of pleural fluid drainage. The antibiotics were continued for a total of 5 days. In the subsequent days of hospital admission, the child started showing signs of improvement, in the form of decreased fever spikes, cough and breathlessness. The child started accepting orally and was advised to take a high protein diet. On follow up, the child was asymptomatic with adequate weight gain and good air entry on auscultation. A significant radiological improvement showing declining haziness with near normal clearing was noted at the end of 6 months(Fig.2).

III. Discussion:

In a developing country like India, where incidence of tuberculosis is very high among the population, the etiology of empyema can pose a diagnostic dilemma. Tuberculous pleural effusion accounts for only 15.4% of the cases in children less than 10 years of age. Due to rampant use of antibiotics and delay in seeking medical consultation, it is indeed a challenge for isolating the organism in Indian conditions. Most common symptoms include high grade fever, thoracic pain and dyspnoea.

The diagnosis of tuberculous empyema can be confirmed by presence of acid fast bacilli in 25% of the cases, however pleural biopsy has proven to be diagnostic in more than 90% of the cases. The adenosine deaminase levels of more than 70 U/L supports the diagnosis of tuberculous empyema. The suggested pathogenesis in these cases is rupture of a subpleural caseous focus which results in delayed hypersensitivity reaction to mycobacterial antigens and rapidly progresses to form tubercular empyema^[3].

Initially, in our case we suspected pyogenic etiology as the child had an acute history, was sick looking, with no history of tuberculosis in the past or in the family and being a healthy immunized child. In literature, the commonest causative organisms of empyema in pediatric age group are Staphylococcus aureus, Streptococcus pneumonia and Streptococcus pyogenes^[4]. As the patient's condition did not improve inspite of broad spectrum antibiotics, it prompted us to investigate further for finding out the causative organism. On the basis of positivemantoux test and increased level of pleural fluid ADA, we started the child on anti-tubercular chemotherapy along with steroids.

Usually, detection of acid fast bacilli in tubercular empyema is difficult as the bacilli population seen is small and pleural fluid cultures are positive only in 25-45% of the cases^[3]. Polymerase chain reaction(PCR), for the detection of mycobacterial deoxyribonucleic acid(DNA), has shown to have a sensitivity of 31.3% and specificity of 96.6%^[5]. We treated the child with anti-tubercular chemotherapy with four drug daily regimen for 6 months, in which isoniazid, rifampicin, pyrazinamide, ethambutol was given for 4 months and rifampicin along with isoniazid for 2 months. Simultaneously, corticosteroids in the form of prednisolone (1mg/kg/day) for 6 weeks was started followed by tapering of the dosage over the next two weeks. The role of steroids in pleural tuberculosis is debatable. Few trials conducted with respect to the role of adjunctive oral corticosteroids found that there was early resolution of clinical symptoms, less residual pleural fluid but no difference in the development of pleural thickening or adhesions^[6].

IV. Conclusion:

To conclude, in developing countries, tuberculosis should always be kept in mind while treating a patient of empyema. There can be varied presentation of the same and therefore isolating one particular organism can be challenging to the treating physician.

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pleural effusion with intercostal drain in-situ

Fig.2 Chest X-ray showing improvement post 6 months of anti-tubercular chemotherapy