Case Report

Perinatal Tuberculosis a Case Series

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Summary

Perinatal tuberculosis is insufficiently understood and has been rarely reported even in areas endemic for the disease, and unless a high index of suspicion is maintained the diagnosis can be missed. Differentiation of congenital from early postnatally acquired tuberculosis is only of epidemiological importance. We hereby report one case of congenital tuberculosis and three cases of perinatal tuberculosis, and problems faced during investigation and management and emphasize need for improved screening of women at risk and sensitization of the medical community about this entity.

Key words: tuberculosis, perinatal, congenital tuberculosis, intrauterine infection.

Introduction

Regarding current situation of tuberculosis, WHO estimates that one-third of the world population is infected with tuberculosis and the infection rate increases ~1% per year [1]. In spite of this, fewer than 300 cases of congenital tuberculosis have been reported in the medical literature. Though incidence of TB in women of child bearing age is high especially in developing countries like India, so far only about 13 cases of congenital tuberculosis were reported from India, which suggests under diagnosis and gross under reporting [2]. More than half of these infants die if left untreated, which emphasizes the importance of early diagnosis and treatment.

This report describes the history and findings of four patients with perinatal (one congenital) tuberculosis admitted in pulmonology unit, advanced pediatric center, PGIMER, Chandigarh, India, and discusses the problems faced in the diagnosis and management of perinatal tuberculosis and reviews the literature briefly.

Case Reports

Clinical profile of all four patients has been depicted in (Table 1). There was a diagnostic problem in first two cases, which were treated initially with broad-spectrum intravenous antibiotics for pneumonia but with no improvement. In view of poor response, other etiologies like intrauterine infection, atypical or mycobacterial pneumonia, aspiration syndrome or cystic fibrosis were considered and investigated accordingly. Endotracheal aspirate was negative for bacteria, fungus and AFB. Intrauterine infection work up, reflux work up and sweat chloride test were negative. Family screening for tuberculosis was negative in case 1. In case 2, mother had tuberculosis 1.5 years back, which was treated adequately. Mother’s CXR revealed old healed tuberculosis and her sputum examination was negative for AFB. In spite of our best efforts, we could not find source of infection. However, investigations (as shown in table) suggested possibility of tuberculosis in both cases (Fig. 1). The index case 1, mother was admitted around 1 week later with headache, vomiting, and altered sensorium in neurology ward. CT of brain had shown multiple tuberculomas and was started on ATT and steroids. Her CXR and Mantoux were negative. Endometrial biopsy could not be done in view of poor general condition of mother. Diagnosis was not a problem in last two cases in view of positive family history. In case 3, mother was diagnosed as pulmonary tuberculosis 3 months prior to conception and received ATT irregularly for 7 months. She was symptomatic during pregnancy, but took the treatment regularly only after 1 month postpartum. In case 4, mother is symptomatic since 6 months of pregnancy, but was investigated only after delivery and was found to be sputum AFB positive and was started on ATT.

Case 2 was started on four drug ATT with steroids (in view of meningitis), and while others were started
on three drug ATT. All improved symptomatically in follow up with regression of organomegaly. HIV was non-reactive in all four cases.

Discussion

Perinatal tuberculosis in the neonate can be either congenital or neonatal. Making an early diagnosis of tuberculosis in an infant is difficult as symptoms usually begin early (2–4 weeks), which mimic bacterial sepsis and other congenital infections [1, 3]. Tuberculosis should be suspected and investigated for: (i) if newborn with unresponsive worsening pneumonia, particularly in those from endemic areas, (ii) if the mother was diagnosed to have tuberculosis and baby has non-specific symptoms, (iii) when their cerebrospinal fluid revealed a high lymphocyte count in the absence of any identifiable bacterial pathogen on culture as in case 2 and (iv) in presence of fever and hepatosplenomegaly.

The criteria for distinguishing congenital tuberculosis from postnatally acquired tuberculosis were established initially by Beitzke in 1935, later revised by Cantwell, et al. [4] in 1994. Case 1 possibly qualifies for diagnosis of congenital tuberculosis and other three cases are better termed as perinatal tuberculosis. As such, differentiation is only of epidemiological importance and the terminology of perinatal tuberculosis should be used instead, since postnatal type is more common, and modes of presentation, treatment, and immediate prognosis do not differ and may be difficult to distinguish at times [5]. Mother is most common source in both cases.

Regarding investigations, virtually all infants have pulmonary infiltrates on chest radiograph [6]. The tuberculin skin test may take 1–3 months to become positive. Acid-fast stains of smears and mycobacterial cultures from multiple sites (Gastric aspirates, endotracheal aspirates, CSF, or biopsy tissue liver or lung biopsy, placenta) are necessary to make a diagnosis [7]. An important clue in the diagnosis is a maternal or family history of tuberculosis. However, it is not rare that

<table>
<thead>
<tr>
<th>S. no</th>
<th>Age, sex</th>
<th>Symptoms</th>
<th>Examination findings</th>
<th>Chest X-ray</th>
<th>Mantoux test</th>
<th>G/A for AFB</th>
<th>CSF</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1½ mo, M</td>
<td>Fever, tachypnea—3 days, wheeze, HSM</td>
<td>Bil crepts + wheeze, HSM</td>
<td>CXR-miliary mottling (Fig. 1)</td>
<td>−ve</td>
<td>+ve</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>3.5 mo, M</td>
<td>Cough, tachypnea fever—2 mo and one episode of seizure</td>
<td>HSM, Tachypnea,</td>
<td>Interstitial infiltrates with hyperinflation</td>
<td>+ve</td>
<td>+ve</td>
<td>40 cells with lymphocytic predominance and hypoglycorrachia</td>
</tr>
<tr>
<td>3</td>
<td>6 mo, M</td>
<td>On and off fever, cough, poor weight gain—45 days of life</td>
<td>HSM, tachypnea with decreased air entry on left side</td>
<td>Loculated pleural effusion with (Fig. 2a and b) consolidation and mediastinal lymphadenopathy</td>
<td>−ve</td>
<td>−ve, pleural fluid</td>
<td>AFB +ve</td>
</tr>
<tr>
<td>4</td>
<td>6 mo, M</td>
<td>On and off fever, cough poor weight gain—first week of life</td>
<td>Tachypnea with decreased air entry and hyper resonant note on left side, HSM</td>
<td>Obstructive emphysema on left side (Fig. 3a–c). Mediastinal lymphadenopathy</td>
<td>+ve</td>
<td>−ve</td>
<td>Normal</td>
</tr>
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Mo, months; HSM, hepatosplenomegaly; Mantoux, tuberculin test; G/A for AFB, gastric aspirate for acid-fast bacillus; CSF, cerebrospinal fluid.

Fig. 1. CXR showing bilateral consolidation with miliary pattern.

Table 1
Clinical profile and investigations of patients
Fig. 2. (a) CXR and (b) CT scan showing consolidation of left upper lobe with loculated pleural effusion.

Fig. 3. (a) CXR, and (b), (c) CT scan showing mediastinal lymphadenopathy with obstructive emphysema on left side and right lung infiltrates.
the diagnosis of infection in the infant leads to the discovery of tuberculosis in the mother, as in our index case 1. Approach to baby born to mother with active tuberculosis has been highlighted in the Flowchart 1 [8].

In summary, there is a need to improve screening to identify and treat active tuberculosis in prenatal period, which not only decreases perinatal mortality but also prevents occurrence of this serious disease in neonate and a coherent system of cooperation between the hospital and community services, and between pediatricians and adult physicians is essential.

**References**


