

# Congenital Tuberculosis – Late Manifestation of The Maternal Infection

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## ABSTRACT

Tuberculosis in pregnancy though not uncommon, congenital tuberculosis continues to be a rare entity. A case of congenital tuberculosis where the mother manifested the disease 3 months after it was diagnosed in the newborn is reported considering its rarity.

**Keywords:** Congenital tuberculosis, Steroids

Infection with tubercle bacilli either during the intrauterine life or before complete passage through birth canal is termed as congenital tuberculosis. Only 300 such cases have been reported so far in literature with about 10 cases from India<sup>1</sup>. Mortality rate is approximately 50%, which underscores the importance of early diagnosis and treatment.

## CASE REPORT

Twenty seven days old male baby presented with history of poor feeding and fast breathing for 2 days. Baby had normal delivery at term with birth weight 2700 grams. Early postnatal period was uneventful. Baby did not receive any vaccination at birth.

On examination, baby was drowsy with marked pallor, CRT – 6 seconds, respiratory rate –122/minute, heart rate being 164/minute. Liver and spleen were enlarged. Auscultation of the chest revealed bronchial breath sounds in the right infrascapular area with bilaterally scattered crepitations.

Chest x-ray showed diffuse miliary shadows with confluence in the right lung. Hemoglobin was 9.6 gm/dl, total count 28,700/cmm with neutrophilic predominance, band form 6% with toxic granulations in the neutrophils. CRP was 192mg/lit (normal – 6mg/lit). Serum electrolytes, renal and liver function tests were normal. Blood and urine cultures were negative.

As there was clinical and radiological deterioration even after 5 days of initiation of broad spectrum antibiotic therapy, the possibility of a tubercular infection was thought of and gastric aspirates were sent for AFB. The gastric aspirates on the 2<sup>nd</sup> and 3<sup>rd</sup> day showed plenty of AFB, and Bactec culture was positive for *M. tuberculosis* on the 20<sup>th</sup> day.

Antituberculosis therapy was initiated with 4 drugs (HRZS), and considering the miliary pattern and moribund condition of the baby IV dexamethasone was added. Within 2 days of initiation of therapy the baby showed remarkable improvement. However, attempt to withdraw the dexamethasone on the 5<sup>th</sup> day resulted in rapid deterioration and desaturation. So the steroid was reintroduced, converted to oral prednisolone with initiation of feeding; and gradually tapered off over a period of one month.

The source of infection remained a mystery as thorough contact tracing did not reveal any abnormality. However, 3 months after initiation of the baby's treatment the mother presented with a left sided pleural effusion which was subsequently proved to be of tubercular etiology.

Presently, mother and baby have both completed antituberculosis therapy and are doing well.

## DISCUSSION

Tubercular bacilleamia during pregnancy may result in infection of the placenta or the maternal genital tract. Such infection may then be transmitted to the fetus by hematogenous spread through the umbilical vein (primary complex in the liver) or by aspiration or

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ingestion of contaminated amniotic fluid (primary complex in the lungs and gut respectively). Not infrequently the infant is infected during birth when placental separation leads to admixture of maternal foetal blood, or through swallowing of infected amniotic fluid while passing down the birth canal – these infants usually present after 3 weeks and may have normal chest x-rays at birth.

Clinically, tuberculosis in the newborn infant simulates bacterial sepsis or other congenital infections like syphilis or cytomegalovirus. Congenital tuberculosis should be suspected if aggressive broad spectrum antibiotics are ineffective and tests for other congenital infections are negative, particularly if the mother is known to have tuberculosis and especially if recently diagnosed. Symptoms may be present at birth but are usually seen in the 2<sup>nd</sup> and 3<sup>rd</sup> weeks. Hepatosplenomegaly is found in 76%, respiratory distress in 72%, fever in 48% and lymphadenopathy in 38%.<sup>2</sup> Virtually all infants have an abnormal chest radiograph, with nearly half having a miliary pattern. The tuberculin skin test result is unhelpful since it is always negative initially and can take 1-3 months to become positive<sup>3</sup>.

Diagnosis rests on clinical suspicion and demonstration of acid fast bacilli in tissue or fluids, particularly on the culture of *M.tuberculosis*. Early morning gastric samples that are positive for acid fast bacilli on microscopy should be regarded as indicative of tuberculosis, although false positives can occur<sup>4</sup>. Hageman et al found positive cultures for *M.tuberculosis* in 10 of 12 gastric aspirates, all 3 liver biopsy specimens, all 3 lymph node biopsy specimens and 2 of 4 bone marrow aspirates<sup>9</sup>. Open lung biopsy has also been used to establish the diagnosis<sup>5</sup>.

The patient described here was typical with respect to his age at presentation and nonspecificity of symptoms and signs mimicking bacterial sepsis. Only a high index of suspicion following failure of broad spectrum antibiotics clinched the diagnosis.

The first diagnostic criteria used to distinguish congenital from postnatally acquired tuberculosis were laid down by Beitzke<sup>6</sup>. These required that (i) tuberculosis must be firmly established in first few days of life, (ii) a primary hepatic complex, (iii) exclusion of extrauterine infection. As this was largely based on necroscopic data, Cantwell et al<sup>7</sup> have suggested a revised criteria for the diagnosis. The modified criteria include tuberculosis lesions in the infant and one of the following – (i) lesions in first week of life, (ii) a primary hepatic complex or caseating granuloma; (iii) documented tuberculosis infection of the placenta or endometrium; (iv) exclusion of postnatal transmission by thorough contact tracing. The current case qualifies as congenital tuberculosis by exclusion of postnatal

transmission and subsequent development of a tuberculosis pleural effusion in the mother. The baby probably acquired the infection *via* dissemination during primary bacillemia in the mother in pregnancy. In some series of congenital tuberculosis, fewer than half of the mothers were known to be suffering from tuberculosis at the time of delivery, the diagnosis in the infant then leading to the maternal diagnosis<sup>7</sup>.

Baby was treated with the standard 4 drugs – isoniazid, rifampicin, pyrazinamide and streptomycin<sup>8,14</sup>. Intravenous dexamethasone was started along with the ATD's considering the miliary pattern on the chest x-ray, but attempts to withdraw it on day 5 after initiation resulted in sudden deterioration. So it was reintroduced. Recent trials have reported hemodynamic and survival benefits associated with the use of physiologic steroid replacement in patients with septic shock. These results are coupled with the observation of "relative adrenal insufficiency" in some patients with severe sepsis<sup>9,10</sup>. So far steroids have not been advocated in the treatment of congenital tuberculosis, but our patient seems to have been remarkably benefited by its use. Further studies are required to establish the role of steroids, if any, in the treatment of congenital tuberculosis.

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