Late Neonatal Respiratory Distress: A Presentation Of Congenital Tuberculosis

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Citation

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Abstract

Congenital tuberculosis is a rare condition and majority of the cases have been reported before the chemotherapeutic era. We report a baby who was born to a primigravida mother and presented at 20 days of life with dyspnoea, jaundice and hepatosplenomegaly. Chest radiograph showed miliary mottling. Acid-fast-bacilli (AFB) was demonstrated in the gastric aspirate smear and also detected by polymerase chain reaction. Despite disease induced jaundice, anti-tubercular treatment was started and patient showed complete recovery.

INTRODUCTION

Congenital tuberculosis (TB) is a rare entity with 300 cases reported so far, 1 and only 11 cases from India 2. Approximately 30 cases of congenital TB have been reported since the review in 1980 by Hageman, et al 3. The transmission of tubercular bacilli to the fetus occurs by hematogenous spread through placenta, in-utero aspiration and ingestion of infected amniotic fluid or secretions during delivery. The hematogenous route and in-utero aspiration account for approximately half of the cases. In addition, post natal infection may occur from contact with a contagious mother or carer or ingestion of infected breast milk from a mother with tuberculous breast abscess.4 Congenital TB is rare if the mother received adequate treatment during pregnancy.5 We report a neonate with congenital TB who presented to us with respiratory distress, jaundice and who showed complete recovery with anti-tuberculous therapy.

CASE REPORT

A twenty day old male neonate, weighing 2610 g, presented to us with a history of fever for ten days, fast breathing for 2 days and refusal to feed for one day. The baby was delivered as full term by spontaneous vaginal delivery with birth weight of 2500g. Physical examination revealed a febrile (102°F), icteric and dyspnoeic infant with a respiratory rate of 90/min. On examination of chest, there was marked subcostal retraction on inspection, but palpation, percussion and auscultation of chest was normal. There was no lymphadenopathy. Cardiovascular examination was normal. There was profuse nasal secretions and hepatosplenomegaly (liver span: 10 cm & spleen: 2 cm below left sub-costal margin).

Investigations revealed: haemoglobin: 13 gm/dl, total leucocyte count: 13,100/cu mm with 60% neutrophils, 36% lymphocytes and 4% monocytes. Erythrocyte sedimentation rate was 20mm after the 1st hour. Cerebrospinal fluid examination was normal. Serum electrolytes, creatinine, urea and blood sugar were normal. The liver function tests were deranged; AST: 269IU/L, ALT: 386IU/L, serum bilirubin: 5.9mg/dl, direct bilirubin: 4.3mg/dl, serum total protein: 4.6g/dl, serum albumin: 2.1g/dl, alkaline phosphatase: 254 IU, prothrombin time 27 seconds (control: 13 seconds) and activated partial thromboplastin time: 45 seconds (control: 32 seconds).

The chest radiograph showed milliary mottling in both lung fields (figure-1).

Figure 1

Figure 1: CXR of neonate showing milliary mottling in both lung fields.



Blood, C.S.F and urine cultures were sterile. Gastric aspirate showed M.tuberculosis by Polymerase Chain Reaction (amplification was done in a thermocycler- Biometra, Goettingen, Germany), but C.S.F, urine and nasal secretions were negative. Gastric aspirate smears also detected Acid Fast Bacilli (+++). Ultrasonography of abdomen showed hepatosplenomegaly with no evidence of fluid in peritoneal cavity and mesenteric and retroperitoneal lymph node. The billiary system was also normal. Ophthalmological examination was normal. VDRL was negative. Biopsy of the liver was not done because the diagnosis was confirmed by AFB staining and PCR of the gastric aspirate and patient's coagulation profile was deranged.

Anti-tubercular treatment was started with four drugs (isoniazid, rifampicin, pyrazinamide and streptomycin) and liver functions were monitored weekly for toxicity in the first month and monthly thereafter. The infant improved symptomatically over a period of 10 days and started breast feeding. There was no further elevation of hepatic enzymes and transaminases normalised after 6 weeks of antituberculous therapy.

The infant's family was screened for tuberculosis and his mother was found to be the source of infection. Her chest radiograph showed evidence of pleural effusion, which was confirmed by a diagnostic pleural tap. The pleural fluid was straw coloured with a cog-web coagulum formation. The pleural fluid ADA was 116 IU/I. The cytochemical examination of pleural fluid showed 1396 cells with 95% lymphocytes and 5% neutrophils, protein: 3.2 g/dl and sugar: 60 mg/dl. Sputum was positive for Acid Fast Bacilli (++). ELISA (3 kits test) for human immunodeficiency virus (HIV) was negative. Tuberculin skin test with 10 units PPD was 20x20 mm (positive). The endometrial biopsy showed non-caseating epitheloid cell granuloma in between endometrial glands, langerhan's giant cell & collection of epitheloid cells, suggestive of tubercular infection (figure-2). The infant's mother was then referred to department of chest and tuberculosis and treated with anti-tuberculous therapy.

Figure 2

Figure 2: Microphotograph of endometrial biopsy showing a single non-caseating epitheloid cell granuloma in between endometrial glands, Inset: 400x magnification showing a single Langhan giant cell & collection of epitheloid cells.



DISCUSSION

The diagnosis of congenital TB is often difficult. Cantwell et al ₆ have proposed revised diagnostic criteria for the diagnosis of congenital TB. The infant must have proved tuberculous lesions and at least one of the following: (i) Lesions in the first week of life; (ii) A primary hepatic complex or caseating hepatic granulomas (iii) Tuberculous infection of the placenta or the maternal genital tract or (iv) Exclusion of the possibility of postnatal transmission by a thorough investigation of contacts, including the infant's hospital attendants, and by adherence to existing recommendations for treating infants exposed to tuberculosis ₆. In our patient, the mother had pulmonary as well as endometrial tuberculosis.

The clinical features of congenital TB has been described by Hudson (1956) $_7$. The median age at presentation is 24 days (range, 1 to 84)₆. The onset of symptoms varies from the first few days of life to a few months of age, with an average of 2 - 4 weeks as in our patient. The clinical manifestations are often nonspecific and include fever, respiratory distress,

abdominal distension, lethargy, irritability, hepatosplenomegaly, lymphadenopathy, jaundice, ear discharge and skin papules. Central nervous system involvement occurs in fewer than 50% cases $_{8}$. In most infants with congenital TB chest radiographs are abnormal at presentation and include non specific parenchymal infiltrates, adenopathy and milliary mottling (50%).

Although gastric aspirate cultures are said to be a poor diagnostic tool ₉, it has been associated with a high yield of positive cultures for M tuberculosis in most of the reported cases of congenital TB. Gastric aspirate and PCR for AFB are the most expedient and reliable ways of establishing the diagnosis. The yield in CSF is usually low. Demonstration of a primary hepatic complex, which requires an open surgical procedure or autopsy, is not essential for diagnosis ₄. A percutaneous liver biopsy specimen, demonstrating caseating granulomas is sufficient evidence for diagnosis 6.

Our patient presented with respiratory distress and hepatosplenomegaly. The milliary pattern in his chest radiograph was the clue that prompted us to get a chest radiograph of the mother done. This led to further investigations that conclusively documented tuberculosis in order to start anti-tubercular therapy.

We reported this case to emphasize that an early diagnosis of congenital TB in neonate is difficult and requires a high index of suspicion and initiation of anti-tubercular therapy is critical for a favorable outcome.

KEY MESSAGE

- Early diagnosis of tuberculosis in neonate is difficult
- High index of suspicion and anti-tuberculous therapy is life saving

References

1. Cantwell M, Snider DE Jr, Cauthen G, et al. Epidemiology of tuberculosis in the United States, 1985 through 1992. JAMA 1994; 272:535-539 2. Kumar R, Gupta N, Sabharwal A, S. Congenital tuberculosis. Indian J Pediatr 2005; 72:631-633. 3. Hageman J, Shulman S, Schreiber M, Luck S, Yogev R. Congenital tuberculosis: critical reappraisal of clinical findings and diagnostic procedures. Pediatrics 1980; 66:980-984. 4. Pablo JS, Amina A. Tuberculosis in Taeusch HW, Ballard RA, and Gleason CA: Avery's Diseases of the Newborn.8th ed. Philadelphia: W.B. Saunders 2005:542-547. 5. Schaefer G, Zervoudakin IA, Fuchs FF, et al. Pregnancy and pulmonary tuberculosis. Obstet Gynaecol 1975; 46:706-715. 6. Cantwell MF, Shehab ZM, Costello AM, et al. Brief report: congenital tuberculosis. N Engl J Med 1994; 330:1051-1054. 7. Hudson FP. Clinical aspects of congenital tuberculosis. Arch Dis Child 1956; 31: 136-139. 8. Starke JR. Tuberculosis: an old disease but a new threat to the mother, fetus, and neonate. Clin Perinatol 1997;

24:107-127.9. Kendig EL Jr: Prognosis of infants born of tuberculous mothers. Pediatrics 1960; 26: 97.

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