

Case Reports

A 17 day old neonate with tuberculosis: Is it congenital or acquired?

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Introduction

The occurrence of transmission of tuberculosis (TB) from mother to fetus or newborn is well established, but rare, with less than 400 documented cases in the literature worldwide up to 1998¹. There are four modes of perinatal transmission of the tubercle bacilli to the fetus or the newborn². Firstly, tuberculous bacillaemia can spread the bacteria to the placenta and then to the fetus and this may lead to fetal death secondary to severe placental involvement. Secondly, placental TB can spread to the fetus via the umbilical vein to the liver and to the lymph nodes of the porta hepatis or via rupture of a placental tubercle causing amnionitis and subsequent fetal aspiration, thus resulting in primary disease in the liver, gastrointestinal tract and mesenteric lymph nodes. Thirdly, endocervical TB can spread to the newborn by aspiration during the birth process leading to primary pulmonary disease and fourthly, early postnatal exposure can occur secondary to care by the infected mother, another family member or an infected healthcare worker with active pulmonary TB. We report a case of perinatally acquired TB.

Case report

A 17 day old baby girl presented to General Hospital, Anuradhapura with a one day history of fever, poor feeding, abdominal distension and breathing difficulty. She was born at peripheral unit Kahatagasdigiliya, weighing 2840g. On examination, the baby was tachypnoeic with a respiratory rate of 66 breaths per minute. The cervical, axillary and inguinal lymph nodes were enlarged; a firm liver was palpable 4 cm below the right costal margin and an enlarged spleen was palpable 5 cm below the left

costal margin. BCG nodule was present on the outer aspect of left upper arm.

ESR was 20mm in first hour and the haemoglobin 14g/dl. The white blood cell count was $12.6 \times 10^9/L$ with 80% neutrophils and the platelet count $70 \times 10^9/L$. Chest x ray showed multiple 0.5-2mm opacities in all three zones of both lung fields (Figure 1).



Figure 1

The CSF was turbid with protein 110mg/100ml, 23 cells (all lymphocytes) and glucose 72mg/100ml with a random blood glucose of 196mg/100ml. Blood culture grew *staphylococcus aureus* while urine and CSF cultures were negative. Gastric aspirate was negative for acid fast bacilli but blood and CSF polymerase chain reaction (PCR) and DNA amplification test for *Mycobacterium tuberculosis* were positive.

Her 29 year old mother had been treated for two episodes of right lower lobe pneumonia and pleural effusion with intravenous cefuroxime, cefotaxime and erythromycin successfully at 19 and 23 weeks of gestation but had defaulted medical clinic follow up. Mother developed fever during labour which persisted for two weeks after delivery. She was transferred from District Hospital Kahatagasdigiliya with the baby at this stage. Her ESR was 130mm in first hour and chest x ray showed numerous tiny soft tissue density nodules in all three zones of both lung fields (Figure 2).

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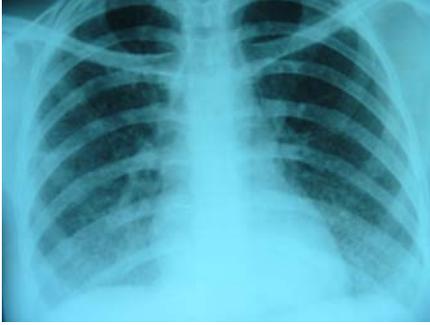


Figure 2

Although sputum for acid fast bacilli and Mantoux test were negative, blood for PCR and DNA amplification test for *Mycobacterium tuberculosis* were positive. Mother was HIV negative. Placenta was not available for examination as the delivery occurred in a peripheral unit and maternal genital tract was not investigated for TB before starting drugs as the mother was seriously ill.

Baby was diagnosed to have miliary TB and tuberculous meningitis and was treated successfully, initially with isoniazid, rifampicin and pyrazinamide for 2 months followed by isoniazid and rifampicin for 7 months. Follow up of the child showed normal growth and development.

Mother was diagnosed to have miliary TB and treated initially with isoniazid, ethambutol, pyrazinamide and rifampicin for 2 months followed by rifampicin and isoniazid for 7 months. Screening of other members of the family for tuberculosis was negative.

Discussion

In Sri Lanka TB incidence rate in the 0-14 age group has remained fairly constant from 1996-2000. It was 3.7, 4.3 and 3.7 cases per 100,000 population in 1996, 1998 and 2000 respectively³. Perinatal TB is hardly ever reported in Sri Lanka and this is probably the first documented case.

The only standardised criteria for distinguishing congenital from postnatally acquired tuberculosis were established in 1935 by Bietzke⁴. These criteria require that the infant has proven tuberculosis and one of the following; lesions in the first few days of life: a primary complex in the liver confirmation of which often requires autopsy or the exclusion of post-natal transmission by separation of the infant from the mother and other sources of infection. Patients meeting these criteria almost certainly have true congenital tuberculosis. These criteria have become increasingly difficult to be applied in current practice

especially if the infant is surviving. Recently a new set of criteria has been proposed⁵. In these criteria the infant must have proven tuberculosis and at least one of the following; lesions in the first week of life, a primary hepatic complex or caseating hepatic granuloma, tuberculous infection of the placenta or the maternal genital tract and exclusion of the possibility of postnatal transmission by a thorough investigation of contacts of the child.

In our newborn patient, the diagnosis of tuberculosis was established with certainty. Although tubercle bacilli were probably present in maternal blood stream making transplacental transmission possible, lack of evidence of a primary hepatic complex of tuberculous infection, non-availability of placenta for examination and non-investigation of maternal genital tract did not allow us to establish a firm diagnosis of congenital tuberculosis. Presentation in the third week of life and possible post-natal transmission from the mother made a diagnosis of postnatally acquired tuberculosis probable.

The most frequent signs and symptoms of congenital tuberculosis are hepato-splenomegaly (76%), respiratory distress (72%), fever (48%), lymphadenopathy (38%), abdominal distension (24%) and lethargy (21%)⁶. All these features were present in our patient.

The treatment of congenital and post-natal tuberculosis is the same². It should initially include isoniazid, rifampicin and pyrazinamide until drug susceptibility of infant's or mother's isolate is known. If there is a possibility that the organism is drug resistant (prior therapy to mother, high prevalence of such bacteria in the region) four drugs should be given adding streptomycin. If there is no drug resistance treatment should be continued with two bactericidal drugs (isoniazid and rifampicin) for 9 months. In infants it is best to avoid ethambutol or streptomycin because of the inability to test for visual acuity and ototoxicity respectively. Our patient was treated with the above regime.

Early suspicion, diagnosis and appropriate treatment were imperative for the survival of our neonate. Prevention of TB could have been possible if mother was diagnosed in early pregnancy. This case reiterates the importance of having a high degree of suspicion of TB in a high risk neonate and early appropriate treatment. It also highlights the importance of examination of placenta and investigation of genital tract for presence of TB to confirm a diagnosis of congenital tuberculosis.

References

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