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Case Report

Congenital tuberculosis in a neonate

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Introduction

Tuberculosis (TB) is endemic, especially in developing nations. Congenital TB is rare in neonates and more so multi drug resistant TB (MDR-TB) and not many cases have been reported. About 300 cases of congenital TB have been mentioned in the literature till 1989 with subsequent figures of 58 in 1994 and 18 more cases till 2005.¹ One of the reasons for the rarity of this disease in neonates is probably the lack of clear cut differentiation between congenital and acquired cases. Though its incidence rate is low but mortality rates are high especially if infected with MDR-TB.² We report a female neonate with congenital TB, found to be Rifampicin resistant on gene Xpert and the “congenital” cause confirmed on liver biopsy.

Case report

A female neonate was born to a primigravida mother at term by cesarean section in view of non-progress of labor with birth

weight of 3.4 kg. The baby was on exclusive breast feeds since birth and was apparently asymptomatic till end of third week. She was readmitted at 28 days of life with cough, increased chest activity and fever. The mother had developed cough and breathlessness, about 2–3 days after delivery and on evaluation, CT chest was suggestive of Pulmonary Koch's. Adenine Deaminase (ADA) level was raised in her pleural fluid, and hence she was put on Anti Tubercular Treatment (ATT) at about 15 days post delivery. All this while the baby was on exclusive breast feeds and was roomed in with mother. The baby developed respiratory symptoms with cough, increased chest activity and fever from day 20 of life. The baby was on oral antibiotics and antipyretics from local medical care providers since then, but as there was no improvement, the baby got admitted at 28 days of life. On examination, she was febrile with pallor, cyanosis, tachypnoea, subcostal retractions, oxygen saturation of 85% at room air; bilateral wheeze, coarse crepitations and distended abdomen with hepatosplenomegaly. Liver span was about 8 cm and spleen was palpable about 3 cm below left costal margin. The neonate was managed with oxygen support and intravenous (IV) antibiotics initially. However, based on clinical suspicion and chest radiograph (Fig. 1), she was started on ATT namely Isoniazid, Rifampicin, Pyrazinamide and Ethambutol (HRZE) within a week of admission. Serial gastric aspirates for 3 days were negative for Acid Fast Bacilli (AFB). There was mild improvement in clinical status initially but she developed thrombocytopenia and deranged liver function tests after 2 weeks of starting ATT. There was no improvement on subsequent chest radiograph but HRZ was stopped in view of likely hepatotoxicity. The gene Xpert for gastric aspirate as well as the sputum of mother revealed Rifampicin resistance. Ethambutol was

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Fig. 1 – Multiple non-homogenous bilateral opacities at admission.

continued and Injection Streptomycin, IV Ciprofloxacin were added along with oral Prednisolone. After 3 weeks of ATT, she deteriorated further and developed seizures, respiratory distress, fall in oxygen saturation and required mechanical ventilation and anticonvulsants. Repeat chest radiograph showed worsening and extensive non-homogenous opacities (Fig. 2). The analysis of Cerebrospinal Fluid (CSF) revealed raised proteins, leucocytosis with predominant lymphocytes, but no AFB. Transcranial USG was normal. The baby had a sudden cardiac arrest on day 57 from which she could not be revived. Liver biopsy on post mortem examination revealed presence of AFB (Fig. 3) and granulomatous lesions of tubercular etiology (Fig. 4).

Discussion

Tuberculosis in a newborn is either acquired in utero (congenital) or in neonatal life from mother or other contacts and it is difficult to differentiate between the two on clinical grounds. Though congenital TB is associated with prematurity, intra uterine growth restriction (IUGR) but can be seen in term and appropriate for gestational age (AGA) babies as well. The modes of spread of tubercular bacilli in fetus in case of congenital TB have been well described in literature. The infected placenta may spread bacilli through the umbilical vein, which may develop a primary focus in the liver with involvement of the periportal lymph nodes and further passage of bacilli into the main circulation. Alternatively, the bacilli may remain dormant in the pulmonary circulation and after birth, because of increased oxygenation and circulation, lead to primary TB. The inhalation or ingestion or aspiration of infected amniotic fluid following a rupture of caseous lesion in

Fig. 2 – Extensive bilateral opacities (after 3 weeks of ATT).

Fig. 3 – Liver biopsy showing acid fast bacilli on Ziehl Neelsen stain (100× oil immersion).

placenta may also spread the bacilli. This can cause multiple primary foci in lungs, gut, middle ear.^{3–6}

The signs and symptoms of congenital TB, frequently are hepatosplenomegaly (76%) followed by respiratory distress (72%), fever (48%), abdominal distension, and lethargy.^{3,7} Based on review of cases, Cantwell et al. formulated a criteria to diagnose congenital TB and they suggested proven tuberculous lesions and any one of the following; lesions within first week of life, caseating hepatic granulomas, tuberculous infection of the maternal genital tract or placenta or both, and postnatal transmission excluded.^{3,8,9}

The dilemma in our case was whether this was a case of congenital or acquired TB as the neonate presented to us at 28 days of life. In our case, the mother was started on ATT 15 days

Fig. 4 – Linear cores of tissues from liver biopsy revealing a focus of granulomatous lesion (4×).

post delivery following symptoms soon after delivery and it suggested latent TB in her, during pregnancy. The presence of hepatosplenomegaly, respiratory distress, and fever in neonate suggested the mode of spread to be likely hematogenous via umbilical vein as it led to more fulminant course and hepatosplenomegaly. In our case, the symptoms appeared in the third week of life, whereas congenital TB usually occurs during initial weeks but some studies suggest that it may even occur within one month of life.^{8,10} The liver biopsy revealed granulomas which confirms disseminated TB. The CSF examination did not reveal AFB however PCR was not done. There are reports in literature that the CSF yield for isolation of *M. Tuberculosis* is often less than 20% and meningitis per se occurs only in one third cases of congenital TB.^{3,7,8}

To conclude, we have reported a rare case of congenital TB in a neonate, presenting with respiratory distress and hepatosplenomegaly in late neonatal period. The early diagnosis and management is crucial in congenital TB as it has a high mortality rate of about 50%. The post mortem liver biopsy clinched the diagnosis in our case and hence every effort must be made to convince the parents for at least partial/post mortem autopsy in such situations, as substantiated through this case.

Conflicts of interest

The authors have none to declare.

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