Case Report

Congenital Tuberculosis Presenting as Ascites

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Abstract
Congenital tuberculosis is a rare disease of which the most common presentations include respiratory distress, fever, and organomegaly. We report a case of congenital tuberculosis presenting with ascites.

Keywords: ascites, congenital tuberculosis

Introduction

Congenital tuberculosis (TB) is a rare disease with severe clinical presentation of *Mycobacterium tuberculosis* infection. The following is the report of a case of congenital TB with an unusual presentation.

Case Report

A two-month-old girl was admitted to Imam Reza University Hospital in Mashhad, Iran with mild fever and abdominal distention since two weeks prior with the impression of sepsis. The girl, weighing 3700 g at birth, was born vaginally after a normal pregnancy to a 25-year-old mother. She was formula fed and her vaccinations were up to date. When she was 20 days old, her mother was admitted to the neurology ward with a diagnosis of tuberculous meningitis. The mother's chest X-ray was normal and sputum smear for acid fast bacilli (AFB) was negative. Her grandmother died eight months prior to admission due to pulmonary tuberculosis. On examination, the infant was irritable, with a weight of 5800 g and axillary temperature of 38.8°C. Her pulses and blood pressure were within normal ranges. The patient had tachypnea and rales in both lungs. Heart examination was normal. She had a distended fluid-filled abdomen and no jaundice.

Examination of gastric washing was negative for AFB for three consecutive days and culture for *Mycobacterium tuberculosis* showed no growth. Ascitic and cerebrospinal fluid analyses both revealed high protein, low glucose, and raised white cell count with predominant lymphocytes; acid fast smear and culture were negative. We performed PCR assay (Bioscience Ltd., Heslington,York, England) on these specimens by two different pairs of oligonucleotide primers, targeting the gene encoding for the 85 KDa protein (common to all mycobacteria) and the insertion sequence IS6110, which was specific for *M. tuberculosis* complex. The first primers (up-stream 5'-CCT GCG AGC GTA GGC GTC GG-3'; downstream 5'-CTC GTC CAG CGC CGC TTC GG'-3') were designed to amplify a 123bp sequence of IS6110.[1] The other primer pairs (up-stream 5'-ATC AAC ACC CCG GCG TTC GAG-3'; downstream 5'-CGG CAG CTC GCT GGT CAG GA-3') were expected to amplify a 162 bp DNA fragment.[2] PCR test for *Mycobacterium tuberculosis* was negative for both ascitic and cerebrospinal fluids.

The mother and infant's HIV serostatus were negative. Additional laboratory tests revealed serum albumin 2.9 g/dL, alkaline phosphatase 142 units/L, aspartate aminotransferase 85 units/L, alanine aminotransferase 106 units/L, lactate dehydrogenase 142 units/L, international normalized ratio 1.7, partial thromboplastin time 25, urea 16 mg/dL, and creatinine 0.3 mg/dL. C-reactive protein was positive. Venous blood gas showed pH 7.47, pO_2 54.7 mmHg, pCO_2 33.1 mmHg, and base excess 1.8 mEq/L. Chest radiography showed diffuse miliary-like lesions. Monteux skin test was negative.

Considering her positive family history (TB in her mother and grandmother), the patient was kept in an isolated room and an anti-tuberculosis regime (amikacin + rifampin + isoniazid + pirazinamid) was initiated for two months, followed by rifampin and isoniazid for ten months. Cefotaxime was also administered for ten days, beginning on admission. Steroid therapy was given at 2 mg/kg of prednisolone for one month and tapered the next month. Supportive measures included intravenous albumin, fresh frozen plasma, cryoprecipitate, packed red blood cells, and platelets. Chest X-ray and tuberculin skin test of all close contacts did not reveal any abnormalities. Within one week of initiation of therapy, the infant exhibited remarkable improvement. After about two weeks, we noticed yellowish pus in her left auditory canal. Smear for AFB with Ziehl–Nielsen staining was positive from ear discharge materials. For PCR, DNA was extracted from the pus by means of a standard phenol/enzymatic digestion method and PCR for *Mycobacterium tuberculosis* was positive.[3] She was discharged and followed up regularly in our clinic for one year with complete resolution of her signs and symptoms.

Discussion

Although TB is a common infection worldwide, congenital TB is very rare, with around 300 cases reported in the literature.[4] Tuberculous bacilli may be transmitted from an infected mother to the fetus by the transplacental route, forming a primary complex in the infant's liver with secondary hematogenous spread, or by aspiration or ingestion of the infected amniotic fluid, leading to a primary focus in the infant's lung or gastrointestinal tract.[5] As demonstrated in the present case, congenital tuberculosis is particularly difficult to diagnose. In contrast to our patient, in most reported cases the mothers are often apparently healthy; as in one

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review 24 out of 32 mothers were asymptomatic.[6]

Congenital TB is difficult to distinguish from early postnatal infection. The widely accepted criteria by Cantwell et al. for congenital TB include proven tuberculosis lesions of the newborn and one of the following: (1) lesions in the first week of life; (2) hepatic granuloma or primary hepatic complex; (3) TB of the placenta or maternal genital tract; or (4) exclusion of postnatal transmission.[5] We believe that our patient developed TB in utero because her mother had TB meningitis without pulmonary involvement and negative results for TB among other close contacts.

Timely diagnosis of congenital TB is critical for prompt initiation of treatment and prevention of nosocomial transmission. Although infants lack powerful coughs, transmission may occurs secondary to suctioning or direct contact. A 0.8% transmission rate from the neonates to hospital personnel has been reported.[7]

Congenital and early postnatal infections have similar treatment and prognosis. Therefore, a more inclusive single categorization of perinatal TB has been suggested.[8]

According to Cantwell's review, hepatomegaly, fever and respiratory distress are the most frequent clinical features.[5] In our case and other recent reports, early signs and symptoms are often nonspecific and mimic more common neonatal diseases such as bacterial sepsis.[9,10]

Tuberculosis should be considered in an ill neonate with a poor response to conventional antibiotic therapy, especially in endemic areas for TB or if the mother has risk factors for TB. Symptoms may be present at birth but typical features of congenital TB usually appear after a few weeks. Our patient presented atypically with ascitis and fever as the first signs of TB. Few studies reported ascites as the initial presentation of congenital TB.[4] Other reported uncommon manifestations of congenital TB included isolated otitis, lymphadenitis, facial nerve palsy, and TB of the spine.[11–14] Our patient was finally diagnosed by a positive smear of otic discharge for AFB along with a strong family history of TB. The ear is an unusual site for primary infection. It may be speculated that transmission occurred when infected amniotic fluid lodged in the fetal ear canal or via the eustachian tube, either in utero or during birth.[15]

Our patient was treated with four anti-TB drugs for two months, followed by two drugs for ten months. Although the optimal duration of therapy has not been established, many experts treat infants with congenital or postnatal acquired tuberculosis for 9 to 12 months because of the low immunologic capability in young infants.[9]

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