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CASE REPORTS

DISSEMINATED TUBERCULOSIS AT ONE MONTH OF AGE - A CASE SERIES

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ABSTRACT

Disseminated tuberculosis (TB) is rare in neonates. We present here two cases of infants with disseminated TB who had onset of the disease at 1 month of age. The first is of a boy who had left ear discharge at one month of age with swelling behind the right ear. The second infant was a four month old boy who presented with swelling in the cervical and in inguinal region since one month of age and cough fever and vomiting since fifteen days. After various investigations, both were diagnosed with disseminated TB. Also, the mothers were detected to have pulmonary TB. The infants were started on ATT. They were asymptomatic on follow up.

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Introduction

Disseminated tuberculosis (TB), results from massive lymphohematogenous dispersion of mycobacterium tuberculosis (MTB) in the body.[1] Infants are more susceptible to developing this disease, which is associated with high morbidity and mortality.[2] Although neonatal BCG vaccination has been shown to provide higher than 80% protection against the development of disseminated TB[3], it continues to be a rare but important cause of illness in the pediatric population.[4] In the pre-antibiotic era, disseminated TB was considered to be predominantly a disease of children less than 3 years of age. However, it is now increasingly being encountered in adults as well.[1] Disseminated TB is rarely reported in infants.[5] We present 2 children with disseminated TB with onset at 1 month of age.

Case 1: A 1½ year old boy was referred for management of disseminated tuberculosis which had started at 1 month of age. He was born full term by normal vaginal delivery and received BCG vaccine. He had left ear discharge at 1 month of age with swelling behind the right ear. Aspiration of posterior auricular abscess showed acid fast bacilli (AFB) and culture grew MTB that showed sensitivity to all the first line drugs. His Chest X-Ray showed right sided upper lobe pneumonia with mediastinal adenopathy and CT chest showed right upper lobe consolidation with enlarged necrotic prevascular, paratracheal, pretracheal and subcarinal lymphnodes with miliary shadows. He also had hepatosplenomegaly. He was diagnosed to have disseminated TB and was treated with anti-tuberculous therapy (ATT). Mother was detected to have pulmonary TB during screening for TB. The child had normal CD3, CD4, CD8, CD19, CD16 and CD56, normal serum immunoglobulins and normal nitro blue tetrazolium (NBT) test. HIV ELISA was negative. He was given ATT for 1 ½ years and then stopped. Child is alright on follow up.

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Case 2: A 4 month old boy presented with swelling in the cervical and inguinal region since 1 month of age and cough, fever and vomiting for 15 days. He was on ATT consisting of Isoniazid (H) & Rifampicin (R) since past 2½ months. Mother was suffering from TB and was on ATT since past 3 months. His birth history and development was normal. Birth weight was 2.5 kg. He was on exclusive breast feeds and had only received BCG and OPV vaccine. On examination, height was 58 cms, weight was 3.8 kg and head circumference was 37.5 cm. He had multiple cervical and inguinal lymph nodes (largest measuring 2.5 cm x 2 cm) which were non tender and not matted. On systemic examination, he had hepatosplenomegaly. There were no meningeal signs. Other systems were normal. Chest X-Ray showed right sided primary complex. Ultrasound (USG) abdomen showed hepatosplenomegaly with right iliac fossa enlarged nodes measuring 2.5 cm. USG skull showed mild prominence of lateral ventricles. Cerebrospinal fluid (CSF) examination showed 35 mg/dl of proteins, sugar of 62 mg/dl and 20 cells/cumm (100% lymphocytes). HIV ELISA was negative. SGPT was 111 IU/L. In view of CNS TB, abdominal and cervical adenopathy and primary complex in lungs, he was diagnosed to have disseminated TB and started on non-hepatotoxic ATT consisting of Ofloxacin (O), Streptomycin (S), Ethionamide (E) and Ethambutol (E). At 6 months of age, his SGPT decreased to 77 IU/L. Streptomycin was omitted (after receiving 2 months therapy) and Isoniazid was added. At 7 months of age, his SGPT was 71 IU/L and rifampicin was added. Ofloxacin was omitted at 8 months of age. At 14 months of age, ATT was stopped. At 16 months of age, he weighed 10.6 kg and was asymptomatic.

Discussion

Infants' immune system is susceptible to various infections and TB is arguably foremost among those. Macrophages play a central role in phagocytosis, growth arrest and intracellular killing of MTB. Specific T-cells help in activation and equipment of macrophages to control the bacteria. However, it seems likely that infants have a greater delay in the initiation of their adaptive immune response than older children and

adults. Moreover, infant TB is due to an inadequate inflammatory response. Macrophages, neutrophils and dendritic cells in infants are present in low numbers and have chemotactic deficits that impair their recruitment to sites of inflammation. Their capacity to produce cytokines, such as tumour necrosis factor, IL-1 and IL-12, is decreased, while their capacity to produce anti-inflammatory cytokines, including IL-10, is increased. Because of these alterations in the immune system, infants are 5-10 times more likely to progress to active TB after infection and are also more likely to manifest disseminated forms of disease.[2]

Disseminated TB in infants may be congenital, due to postnatal transmission from an infected source or may have other causes. Intrauterine infection is most commonly due to the spread of the organism to the fetus via the umbilical vein causing placental seeding. Rupture of placental lesion into the amniotic cavity followed by aspiration of the infected fluid is another potential causative factor.[6] Both our patients had onset of TB at 1 month of age and both the mothers had TB, suggestive that the TB could have been acquired from the mother.

The clinical manifestations of disseminated TB are usually non-specific, often delaying the diagnosis.[1] The most frequently seen symptoms and clinical features are fever, rales, loss of appetite and weight and hepatosplenomegaly. Tuberculous meningitis (TBM) may also be seen in some cases.[7] Our second patient had TBM though he had no meningeal signs.

Life-threatening primary TB in childhood can result from known inherited or acquired immunodeficiencies, although majority of the cases remain unexplained. HIV infection does predispose subjects to severe forms of childhood TB but such infection is observed in a small fraction of affected children. The most common genetic defect identified in patients with severe tuberculosis to date is the complete deficiency of IL-12Rβ1. Children on immunosuppressive drugs for a variety of reasons such a solid organ transplantation or bone marrow transplant (aplastic anaemia, myeloma), are also at an increased risk of mycobacterium diseases.[8] In both our patients, preliminary immunodeficiency did not pick up any immunodeficiency.

Before the availability of ATT, mortality varied from 30% in infants 1-2 years of age to 55% in infants aged less than 6 months.[2] Disseminated tuberculosis is uniformly fatal if left untreated,[1] hence treatment must be started as soon as possible and therapy should be monitored. Both our patients responded to ATT.

Compliance with Ethical Standards

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Conflict of Interest None

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