Nigerian Journal of Paediatrics 2011; 38(2):100-103

CASE REPORT

A. A Orogade
WN Ogala
R. Onalo

Congenital tuberculosis: a case report

Received: 28 February 2011
Accepted: 18 May 2011

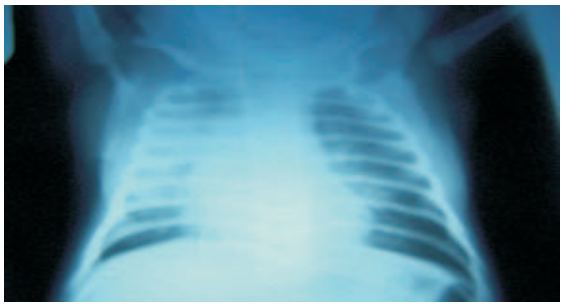
AA Orogade (✉), Ogala W.N, Onalo R.
Department of paediatrics
Ahmadu Bello University Teaching Hospital Zaria-Nigeria
Email: orogade@yahoo.com

Abstract: Congenital tuberculosis is insufficiently understood and has been rarely reported even in areas endemic for the disease. Unless a high index of suspicion is maintained, the diagnosis can be missed. A case of congenital tuberculosis is herein reported to illustrate the difficulty in identifying such infants and also to sensitize the medical practionner in TB endemic areas of the need for etiologic diagnosis in congenital pneumonias as well as for antenatal screening for TB in mothers at risk.

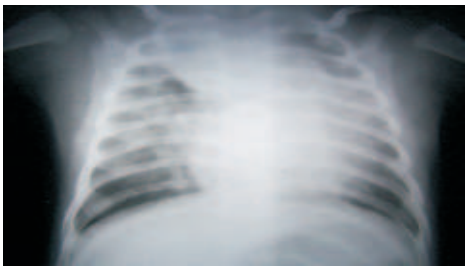
Case report

A five day old male full term infant presented with two days history of fever, noisy breathing and excessive crying. . The infant's mother, a 20 year old undergraduate university student had had a febrile illness 4 months before delivery, was treated with antibiotics and fully recovered. She had remained asymptomatic after that and denied exposure to active tuberculosis. The baby whose birth weight was 3300g now weighed 3280g. Physical examination revealed a temperature of 38°C, irritability, central cyanosis, severe respiratory distress, moderate jaundice, few bilateral small sized inguinal lymph node swellings and a papular skin eruption with an erythematous background. He was moderately tachypneic (RR=88/min), had severe subcostal and intercostal recessions, with decreased breath sound intensity and wide spread coarse crepitations. The baby had hepatomegaly of 5cm below the right costal margin and a splenomegaly of 2cm. There was no cardiomegaly or evidence of cardiac failure and examination of other systems was essentially normal. His white blood cell count was 9,900 per cubic millimeter with 44.4% polymorphonuclear cells, 47.3% lymphocytes, 6.2% monocytes, 1.8%eosinophils and basophils 0.3%. Hematocrit was 55%. Bacterial cultures of blood, cerebrospinal fluid and urine were negative. Biochemical analysis of CSF was normal and culture, negative. Serum Concentration of total bilirubin was 130μmol/L.

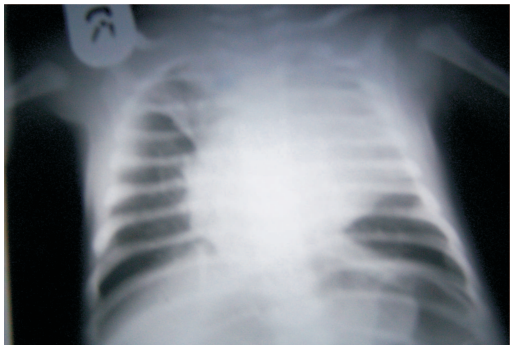
Serological testing for congenital infections and human immunodeficiency virus (HIV) were negative. Initial chest radiograph showed bilateral lung parenchymal infiltrates (Fig1).



Echocardiography study was normal
The baby received intravenous cefuroxime and intramuscular gentamycin for five days but respiratory symptoms worsened with virtually absent breath sounds in the right upper and mid lung zones. Repeat chest roentgenography then revealed collapsed right upper lung fields (Fig. 2).

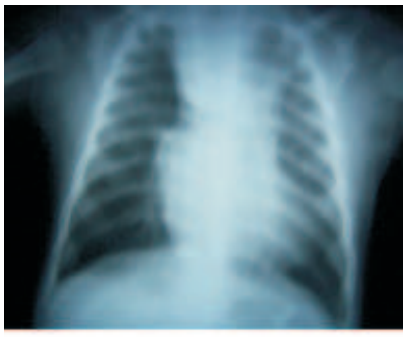


Antibiotic regime was changed to Ceftriaxone with some clinical and radiological improvement within six days. However, there was persistence of moderate respiratory distress with tachypnea, intercostal and subcostal recessions with respiratory rates ranging between 64/min and 74/min. He was discharged on parents request to the neonatal clinic and had short duration appointments for close monitoring. Chest radiography was repeated about three weeks after discharge which showed progressive lung disease with homogenous opacity of the left and right upper zones (Fig. III). By the 7th week of life, the child was failing to thrive; he weighed 3250g as compared to birth weight of 3300g.



Screening for congenital tuberculosis was initiated at this point. Tuberculin skin test (Mantoux test) was negative. The mother was also screened for tuberculosis and found to have a Tuberculin Skin Test reaction of 18mm, and an ESR of 34mm/hr. Her chest radiograph showed significant hilar lymphadenopathy. She was not coughing and so could not produce sputum. Endometrial biopsy was not done. She was found to be sero negative for HIV. The baby then had a diagnostic BCG test which yielded an accelerated reaction with formation of an induration in two days and scar formation within three weeks of administration.

The baby was then commenced on streptomycin, isoniazid and rifampicin with significant clinical response within three weeks of initiation of therapy. By the 10th week of life his weight had increased by 1150g to 4400g at an average of 383g/week. Complete resolution of clinical and radiographic features was noted at 16weeks and 24 weeks respectively.



Discussion

Congenital tuberculosis is defined as tuberculosis occurring in infants caused by *M. tuberculosis* infection during the intrauterine life or before complete passage through the birth canal[1]. Diagnostic criteria for congenital tuberculosis were proposed in 1955[2]. Cantwell[3] later made a review in which there should be proven tuberculosis lesion in the infant plus one of the following: lesions occurring in the first week of life, a primary hepatic complex, maternal genital tract or placental tuberculosis and exclusion of postnatal transmission by thorough investigation of contacts. Congenital tuberculosis is believed to be rare and fewer than 300 cases have been reported worldwide by 1989[4,5]: Blackall et al[6] found three affected patients among 100 infants of mothers with active tuberculosis, but in two other series [4] no affected patients were found among 260 and 1369 such infants. In this area of tuberculous endemicity however, there is insufficient local literature to give an accurate incidence of congenital tuberculosis. This may be due to the difficulty in fulfilling the case definition of congenital tuberculosis.

Tuberculous bacillaemia during pregnancy may result in infection of the placenta or the maternal genital tract. Such infection may then be transmitted to the fetus by hematogenous spread from the placenta to the umbilical vein or by the aspiration or ingestion of amniotic fluid contaminated by placental or genital infection either before delivery or at the time of delivery. This is as opposed to acquired tuberculosis which is contracted at any post natal age though the most usual contact of postnatal infection is also the mother. What differentiates the congenital from tuberculosis acquired in early post natal life then is that there has to be proof of such antenatal transmission. Usually an endometrial biopsy done shortly after delivery that shows the presence of acid fast bacilli is confirmatory of the diagnosis. A diagnosis of congenital tuberculosis in this patient was not initially entertained, but became a consideration after he did not respond clinically or radiologically to conventional first and second line regimes for common neonatal infections.

The median age at presentation in most reported cases was 24 days (range 1-84)[7]. The patient in this report was typical with respect to age at presentation. Respiratory distress, fever and hepatosplenomegaly are the nonspecific symptoms and signs that characterize congenital tuberculosis[8], with which our patient also presented. These signs could be present also in bacterial sepsis, other congenital infections such as cytomegalovirus, herpes simplex virus, HIV, toxoplasmosis, syphilis and malaria. Making an early diagnosis of tuberculosis in a neonate is therefore difficult and requires a high index of

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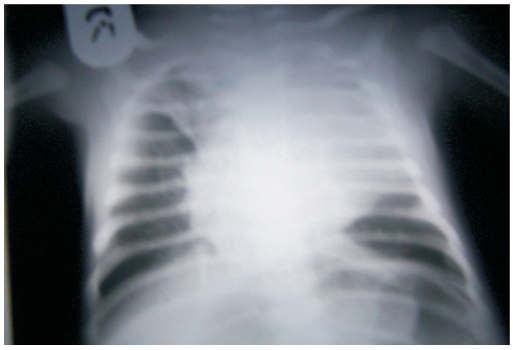

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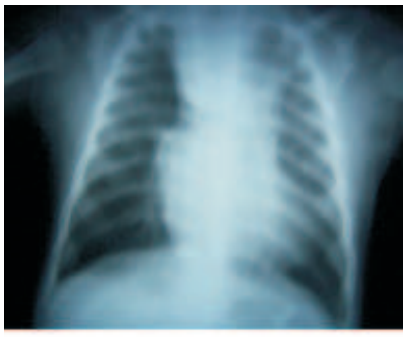

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suspicion. Tuberculosis may be suspected in a sick neonate who has clinical features of septicemia, but whose response to adequate doses of appropriate broad spectrum antibiotics and supportive therapy is poor. An important clue could also be maternal or family history of tuberculosis; however it is not unusual that the diagnosis of infection in the infant rather leads to the discovery of tuberculosis in the mother. Indeed in most series[8,9] as indeed in this index case, mothers are asymptomatic at the time of their infant's diagnosis.

Acid fast stains of smears and mycobacterial cultures from multiple sites: gastric aspirates, endotracheal aspirates, CSF, open lung biopsy and liver biopsy are necessary to make a diagnosis[2]. Many infants with congenital tuberculosis have abnormal findings on chest radiographs[10]. This patient's roentenogram progressed rapidly from bronchopneumonic changes to lung collapse and only improved significantly when anti tuberculous therapy was commenced. Military pattern is common in infants as well as Hilar/mediastinal lymphadenopathy and parenchymal infiltrates. Some infants have normal findings on chest radiographs early in the course of the disease and later rapidly develop profound radiological abnormalities. Accelerated BCG response as was observed in this patient should be considered as a diagnostic tool.

Response to anti tuberculous therapy is usually dramatic with full recovery and normal lung function thereafter as in this patient. The place of therapeutic trials as diagnostic tool may become significant in resource limited settings where diagnosis could easily be missed.

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