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Congenital Tuberculosis Proven by Liver and Lymph Node Biopsy

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Abstract: Congenital tuberculosis is defined as tuberculosis occurring in infants as a result of infection acquired from their mothers. Congenital tuberculosis maynot be rare and it is necessary to look into this condition more seriously. Tsanscutaneous liver biopsy may help to confirm it's prenatal origin and explain causes of the multi organ involvement. We report a case of congenital tuberculosis diagnosed by liver biopsy in infant with FTT and hepatosplenomegally. A 5 month old male infant developed FTT, fatty diarreha and abdominal distension. He had cough, wheezing and weight loss too. On the physical examination there was hepatosplenomegally, axillary and inguinal lymph node enlargement. All of investigations were negative except percutaneous liver and lymph node biopsy that revealed many acid fast bacilli.

Key words: Cogenital tuberculosis, liver, lymph, node biopsy, infants

INTRODUCTION

Tuberculosis can be acquired in the perinatal period. Infants may acquire Tuberculosis (TB) by Transplacental spread through the umbilical vein to the fetal liver, by aspiration or ingestion of infected amniotic fluid or via air borne inoculation from close contacts (Hageman *et al*., 1980; Cantwell *et al*., 1994).

The clinical presentation of neonatal TB is nonspecific but is usually marked by multiple organ involvement. Transcutaneous liver biopsy may help confirm it's prenatal origin (Kumar *et al*., 2005).

The neonate may look acutely or chronically ill. Fever, lethargy, respiratory distress hepatosplenomegally or failure to thrive may indicate TB in a neonate with a history of exposure.

A primary complex in the liver can be regarded as strong evidence of congenital tuberculosis (Kumar *et al*., 2005).

Diagnosis is by culture and perhaps X ray and biopsy. Biopsy of the liver, lymph nodes, lung or plural may be needed to confirm diagnosis.

In this study, we discuss about an infant with tuberculosis that have been diagnosed by percutaneus liver biopsy.

CASE REPORT

A 5-months old male infant born as the 1st child of healthy and non-related parents developed FTT, fatty diarrhea and abdominal distension 2 weeks prior to his admission. He was delivered by NVD and his birth weight was 2700 g. He was admitted for assessment of non-bloody diarrhea and he weighted 4400 g at that admission. His weight was declined 300-400 g 2 weeks before that admission. In addition, he had coughed and wheezing from 2 months prior to the admission for which he was received some antibiotics.

His past medical history was highlighted by an iron deficiency anemia and a period o f diarrhea for which he had received supplemental ferrous sulphate and cotrimoxazole and furazolidone, respectively. Until 2 months of age, he was breastfed when was started on formula in addition to breast milk (Table 1 and 2).

At the physical examination of his first admission, hepatosplenomegaly was the main finding. The liver and spleen could be palpated 2 and 4 cm below the costal margin, respectively.

Serologic tests for torch syndrome was negative. The αFP level was in normal limits. The mother and infant were checked for HIV and negative results were obtained.

Peripheral blood smear demonstrated hypo chromic microcytic anemia. Sudan test in three stages was negative for steatorrhea. Stool examination and culture, blood culture and urinary analysis and culture were all negative too. Urinary analysis for existence of reducing substances and The sweat test for cystic fibrosis were also negative. The lipid profile of his parents depicted no abnormality.

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Table 1: Laboratory findings of SGOT

Hemoglobin	12.1 g dL^{-1}	SGOT	220 u L^{-1}
ESR(1st h)	30	SGPT	220 u L^{-1}
ESR(2nd h)	90	ALP	450 u L^{-1}
WBC	24800 mm^{-3}	Triglyceride	510 mg dL^{-1}
Polymorphonuclear	32%	Cholesterol	166 mg dL^{-1}
Lymphocytes	68%		

Table 2: Laboratory findings of WBC

WBC	14.300 mm^{-3}	Serum total protein	5.4 g dL^{-1}
PMN	25%	CD4	46%
Lymphocytes	72%	CD8	8%
Eosinophiles	3%	ESR 1st h	100
Hemoglobin	7.9 g dL^{-1}		

At ultrasonography, the bowel distension with ileus was observed extra to hepato splenomegaly and portal vein congestion. There was no abnormality in other investigations such as bone X-Ray studies and ophthalmoscopic exams. By the way a diagnostic were gaucher's disease, glycogen storage disease or other inborn errors of metabolism. We had ruled out intestinal lymphangectasia too. He was discharged and advised to be visited again with the biopsy results on hand.

The results of liver biopsy was reported as necrotizing granulomatous hepatitis with presence of acid fast bacilli most possibly tuberculosis.

After the diagnosis of tuberculosis in infant ,the parents and close relatives were evaluated for TB and their CXR, gastric suction and PPD all were negative. But in evaluation of mother's genital tract, the result of endometrial biopsy was compatible with tuberculosis and there was a lot of Acid Fast Bacill (AFB). Triple anti-tuberculosis drug regimen was started with isoniazid, pyrazinamide and rifampin. At the same time, he underwent axillary and inguinal lymph nodes biopsy which illustrated chronic and non-caseating granulomatosis with many acid fast bacilli.

He was admitted for third time when he was ten month age due to tachypnea, poor feeding and respiratory distress. He had been acutely ill and feverish from 3 days before admission while it was three months that he was receving anti-tuberculosis treatment. Physical examination revealed an ill infant with high body temperature, respiratory distress with fine inspiratory crackles, tachypnea, pansystolic cardiac murmur II/VI, hepatosplenomegaly with liver 4-5 cm and spleen 8-10 cm below the costal margin.

His CXR showed the findings compatible with bacterial pneumonia with right-sided pleural effusion and maximum cardiac size. The culture of gastric aspirate was negative for acid-fast bacilli. The blood culture was positive for coagulase-negative staphylococcus. Liver function test were normal. He was immediately started on intravenous ceftriaxone and digoxin in addition to his anti-tuberculosis medications. At the seventh day of admission, his general condition begun to deteriorate with severe respiratory distress which lead to his admission to ICU(Intensive Care Unit) where treatment with some new drugs such as intravenous crystal penicillin, furosemide, cloxacillin commenced. After 24 h of admission in ICU, he underwent mechanical ventilation for respiratory failure. Treatment with digoxin was replaced by intravenous dopamine. He developed hemorrhagic secretions from endotracheal tube and bradycardia for which we started vitamin K and FFP. Despite treatments, his general condition worsened with poor peripheral perfusion and bradycardia. He deceased after weaning from mechanical ventilator on account of this parent's consent.

RESULTS AND DISCUSSION

Congenital tuberculosis is a disease with a high mortality and less than 300 cases of congenital tuberculosis have been reported so far in the literature despite high prevalence of tuberculosis in the world. A timely diagnosis is difficult because the symptoms in the infants are often nonspecific and mothers are often asymptomatic (Kumar *et al*., 2005). Tuberculosis should be suspected in infants who are unresponsive to empiric antibiotics (Yi-Hung, 2002). In the past the diagnosis was usually confirmed only by autopsy because of the high mortality rate.

Congenital tuberculosis is still a dangerous disease. In a review of 58 cases since isoniazide was introduced, 26 of the 58 patients died before receiving treatment, suggesting that delay in diagnosis may have been decisive (Jody, 2002).

There is need to have a high index of suspicious. The frequency of congenital tuberculosis is probably underestimated. It's early diagnosis is essential but often difficult as the initial manifestations are delayed.

Congenital tuberculosis is particularly difficult to diagnose. In the classic study of Hageman *et al.* (1980) only 2 of 16 infants with congenital tuberculosis who were tested had positive skin tests (Jody, 2002).

In this case the uncommon and elusive nature of the extra pulmonary tuberculosis led to delay in the diagnosis of the mothers and infants. Percutaneous liver biopsy confirmed the prenatal origin and explained the cause of the multiorgan involvement.

In our patient at first admission the initial symptoms were very complicated and we had some different differential diagnosis according to failure to thrive, bloody diarrhea, hepato splenomaegaly, abdominal distension and peripheral lymph node enlargement.

Along with we studied the status of parents and house contacts by performing of PPD, CXRay and gastric suction that there was not any evidence of tuberculosis, but simultaneously endometrial biopsy of mother revealed the presence of many acid fast bacilli so we started antituberculosis treatment along with her son. Congenital tuberculosis has high mortality rate (50%) and in spite of antituberculosis therapy we lost our patient with a pneumonia due to coagulase negative staphylococcus.

Our case was very unusuall because congenital tuberculosis with primary complex in the liver is extremely rare.

The intra uterine infection is supported by the findings of acid fast bacilli from the mother's endometria tissue.

The present case had caseating hepatic granolomas. The presence of caseating hepatic granulomas permits congenital and postnatuly acquired tuberculosis to be distinguished on the basis of liver biopsy findings alone.

Yi-Hung (2002) had a case report in which percutaneous liver biopsy revealed scattered milliary granulomas in the portal area and hepatic lobules compatible with mycobacterium infection in an infant with hepatomegally abdominal distension, respiratory distress and FTT (Yi-Hung, 2002).

CONCLUSION

It concluded that the patient had congenital tuberculosis based on the following:

- All of the patients contact were healthy and had negative screening tests.
- The patient had careating granulomtous lesions in his liver and lymph nodes.
- The mother had no respiratory symptoms and had a normal chest X-Ray,
- The mother had endometrial caseating granulomas (Popil *et al*., 1998; Canwell *et al*., 1994; Pillet *et al*., 1993).

Nuzha reported that TB was not diagnosed in 53% of mothers untill the diagnosis of the infant. Twelve (71%) of their cases were extrapulmonary and one mother (6%) had tuberculosis ostomyelitis (Popil *et al*., 1998; Machin *et al*., 1992; Matthai *et al*., 1994; Kumar *et al*., 2005).

These factors virtually exclude the possibility of post natal transmission and demonstrate in utero transmission.

Congenital tuberculosis may occur as a result of maternal tuberculosis when the illness involves the genital tract or the placenta.

Congenital tuberculosis is considered rare by many, however recently there has been an increased incidence in developing countries.

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