

Disseminated Congenital Tuberculosis Unmasking Maternal Neurotuberculosis: A Case Report

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Abstract

Case Report

Background. Congenital tuberculosis (TB) is a rare and frequently fatal form of TB. Its nonspecific presentation and a low index of suspicion lead to frequent misdiagnosis and diagnostic delay. Diagnosis relies on the Cantwell criteria, and the maternal source is often unrecognised at the time the infant presents. **Case presentation.** A one-month-old infant, born at term after an uneventful pregnancy and exclusively breastfed, presented with a five-day history of fever and progressive abdominal distension. Examination showed gross abdominal distension with an everted umbilicus and marked hepatosplenomegaly, while the general condition was preserved. Laboratory work-up revealed a markedly elevated C-reactive protein with repeatedly sterile blood and urine cultures, and negative TORCH, HIV, metabolic and tumoral screening. Thoraco-abdomino-pelvic computed tomography demonstrated a miliary pulmonary pattern, necrotic mediastinal and abdominal lymphadenopathy, and micronodular hepatic, splenic and renal lesions. Percutaneous liver biopsy showed epithelioid and giant-cell granulomas with caseous necrosis, establishing caseating tuberculosis. As caseating hepatic granulomas satisfy the Cantwell criteria, congenital TB was diagnosed. Antituberculous quadritherapy was started; cerebral CT and cerebrospinal fluid analysis were normal. Maternal evaluation was initially unremarkable but the QuantiFERON test was positive; one month later the mother developed tuberculous meningitis. The infant's illness thus revealed previously latent maternal infection. **Conclusions.** Congenital TB should be considered in any neonate or young infant with unexplained fever, hepatosplenomegaly and an antibiotic-unresponsive sepsis-like illness. Liver biopsy demonstrating caseating granulomas is diagnostic under the Cantwell criteria, and systematic maternal investigation is essential: the infant may be the sentinel case that reveals maternal disease.

Keywords: Congenital tuberculosis; Caseating granuloma; Liver biopsy; Cantwell criteria; Maternal tuberculosis.

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INTRODUCTION

Tuberculosis (TB) remains a major cause of morbidity and mortality worldwide, and disease in pregnancy carries a risk of transmission to the fetus or newborn. Congenital TB acquired in utero by the haematogenous route through the umbilical vein or by aspiration/ingestion of infected amniotic fluid is exceedingly rare, with fewer than a few hundred cases reported in the literature, yet it is associated with a high case-fatality rate that reaches roughly 40–50% overall and falls substantially when treatment is started early [1–3].

The clinical and radiographic features of congenital TB are nonspecific, overlapping with neonatal sepsis and other congenital infections; consequently, misdiagnosis is reported in more than half of cases and outcome is closely linked to the promptness of treatment [2]. The diagnosis is framed by the criteria

first proposed by Beitzke and revised by Cantwell *et al.*, in 1994, which require proven tuberculous lesions in the infant plus at least one of: lesions in the first week of life; a primary hepatic complex or caseating hepatic granulomas; tuberculous infection of the placenta or maternal genital tract; or exclusion of postnatal transmission after thorough investigation of contacts [1,6]. Because the liver is a frequent site of the primary focus, caseating hepatic granulomas obtained by liver biopsy are of particular diagnostic value [1,2].

A distinctive feature of congenital TB is that maternal disease is often clinically silent and undiagnosed when the infant presents, so that the newborn may be the first identified case in the family [2,4]. We report a case of disseminated congenital TB in a one-month-old infant in which the infant's diagnosis unmasked latent maternal infection that subsequently progressed to tuberculous meningitis [7].

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CASE PRESENTATION

Patient information and clinical findings

A one-month-old infant was admitted on the 31st day of life for a five-day history of fever and progressive abdominal distension. The infant was born at term by caesarean section (performed for a scarred uterus) after a well-followed, uncomplicated pregnancy; the amniotic fluid was meconium-stained and adaptation to extrauterine life was good. Birth weight was 4000 g, the infant was exclusively breastfed, and a control weight on day 12 was 4380 g. There was no known family history of tuberculosis at presentation.

On admission the general condition was preserved and the infant was pink; axillary temperature was 38 °C and weight 4500 g. The infant was haemodynamically stable, with a normal neurological examination. The respiratory rate was 60–80 breaths/min with an oxygen saturation of 98% in room air and a normal auscultation. Abdominal examination revealed gross distension with an everted umbilicus and marked hepatosplenomegaly.

Diagnostic assessment

Initial differential diagnoses included congenital infection (fetopathy), a tumoral process (neuroblastoma with Pepper syndrome, hepatoblastoma) and metabolic disease (galactosaemia, tyrosinaemia, Gaucher disease). The full blood count showed haemoglobin 12.7 g/dL and a white-cell count of 13 400/mm³ (neutrophils 6600/mm³, lymphocytes 7000/mm³); C-reactive protein was markedly elevated at 143 mg/L. The liver panel (AST, ALT, total bilirubin, GGT) and coagulation (PT, aPTT) were normal, as was the serum ionogram. Blood and urine cultures were repeatedly sterile.

TORCH and HIV serologies were negative. The tumoral and metabolic work-up was unremarkable: alpha-fetoprotein was normal, the neonatal spot test was negative, and plasma amino-acid chromatography with succinylacetone was normal. Abdominal ultrasound showed heterogeneous hepatosplenomegaly, a thin layer of ascites, and normal kidneys and adrenal glands. Despite empirical treatment with a third-generation cephalosporin and gentamicin, fever persisted with several daily peaks, cultures remained sterile, C-reactive protein stayed elevated and the liver panel remained normal.

Thoraco-abdomino-pelvic computed tomography (CT) was therefore performed (Figures 2–3). It demonstrated multiple scattered pulmonary parenchymal nodules with a random/miliary distribution coalescing focally into areas of consolidation, together with several

mediastinal and subphrenic lymph nodes showing central necrosis (short-axis 9 mm) and a thin pleural effusion. In the abdomen, the liver was enlarged with regular contours and a heterogeneous echostructure containing multiple micronodular and nodular lesions; the spleen was enlarged and heterogeneous with nodular lesions showing partial enhancement after contrast; there was micronodular renal involvement, hilar, coeliomesenteric and lumbo-aortic lymphadenopathy with central necrosis grouped into a magma (largest 11 mm), and a small-volume intraperitoneal effusion.

Given the disseminated micronodular pattern with caseating (necrotic) lymphadenopathy, a mycobacterial cause was suspected and a percutaneous liver biopsy was performed. Histopathological examination showed hepatic tissue with epithelioid and giant-cell granulomatous lesions with foci of caseous necrosis and an associated lymphocytic inflammatory infiltrate, consistent with caseating (caseo-follicular) tuberculosis (Figure 4). Cerebral CT and lumbar puncture were normal, excluding central nervous system involvement in the infant.

Diagnosis

The infant had histologically proven tuberculous lesions together with caseating hepatic granulomas. As caseating hepatic granulomas constitute one of the Cantwell criteria, the diagnosis of *congenital tuberculosis* was retained [1]. The timing of the maternal work-up (below) further supported acquisition from the mother rather than from an identified postnatal contact.

Therapeutic intervention and follow-up

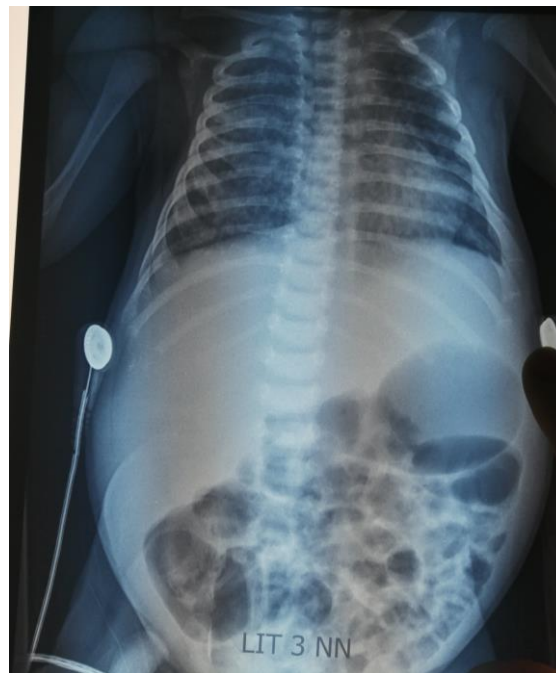
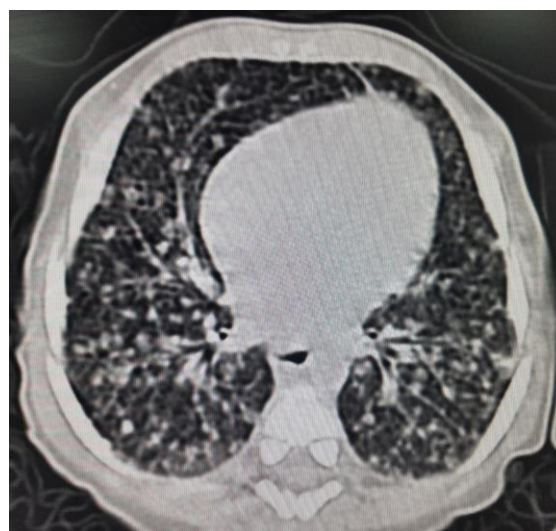
Antituberculous quadritherapy was started with isoniazid, rifampicin, pyrazinamide and ethambutol, in line with the standard two-month intensive phase followed by isoniazid and rifampicin for a prolonged continuation phase [3,4].

Maternal and family investigation

Because congenital TB implies a maternal source, the family was systematically evaluated. Clinical examination and chest radiographs of the family members were normal. Maternal assessment including gynaecological examination and pelvic ultrasound was initially normal, but the QuantiFERON-TB assay was positive; the pneumology consultation was unremarkable at that stage. One month after the infant's treatment had been started, the mother developed headache and fever; cerebral CT revealed tuberculomas and cerebrospinal fluid analysis confirmed tuberculous meningitis. She was treated with a good clinical course, and her thoraco-abdomino-pelvic CT was normal. The infant's presentation had therefore acted as the sentinel event revealing latent, then active, maternal tuberculosis.

Table 1: Timeline of key clinical events

Time point	Event
Birth (term, caesarean)	Birth weight 4000 g; good adaptation; meconium-stained amniotic fluid; exclusive breastfeeding.
Day 12	Routine control; weight 4380 g; asymptomatic.
Day ~25	Onset of fever and progressive abdominal distension.
Day 31 (admission)	Fever (38 °C), gross abdominal distension, marked hepatosplenomegaly; CRP 143 mg/L; sterile cultures.
Days 31~40	Persistent fever despite broad-spectrum antibiotics; sterile cultures; CT shows miliary/disseminated disease.
Diagnosis	Liver biopsy: caseating granulomas → congenital TB (Cantwell criteria).
Treatment	Antituberculous quadritherapy started; normal cerebral CT and lumbar puncture.
+1 month (mother)	Maternal QuantiFERON positive; subsequent tuberculous meningitis, treated with good outcome.

**Figure 1: Thoraco-abdominal radiograph****Figure 2: Chest computed tomography (lung window) showing multiple scattered parenchymal micronodules in a random/miliary distribution, coalescing focally**

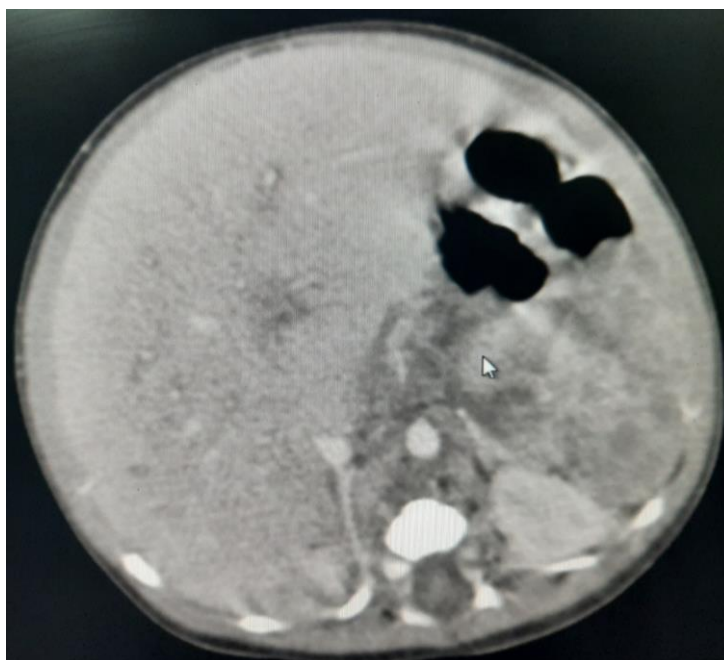


Figure 3: Abdominal computed tomography after contrast showing hepatosplenomegaly with micronodular/nodular lesions and necrotic abdominal lymphadenopathy grouped into a magma

DISCUSSION

Congenital TB is rare and difficult to recognise: its manifestations are nonspecific, and a large systematic review reported an overall mortality of about 43% that fell to roughly 15% among treated infants, with a misdiagnosis rate close to 60% [2]. The most frequent presenting features are fever, respiratory distress and hepatosplenomegaly, each described in around two-thirds of cases [3]. Our patient reproduced this pattern with fever, hepatosplenomegaly and a disseminated miliary process, but with a preserved general condition that could have falsely reassured illustrating why a high index of suspicion is essential.

The liver holds a central place in both the pathophysiology and the diagnosis of congenital TB: organisms reaching the fetus through the umbilical vein seed the liver first, so that a primary hepatic complex or caseating hepatic granulomas are characteristic and are enshrined as a Cantwell criterion [1]. In our patient, percutaneous liver biopsy provided the diagnosis by demonstrating caseating granulomas, avoiding more invasive procedures. This underscores the value of early histological sampling of an accessible lesion when disseminated mycobacterial disease is suspected and bacteriological confirmation is awaited.

A striking feature of this case is the reverse contact tracing. Maternal TB is frequently paucisymptomatic or latent during pregnancy and is undiagnosed at the time the infant presents; in large series only a minority of mothers had clinical manifestations during pregnancy [2]. Here, maternal evaluation prompted by the infant's diagnosis revealed a positive QuantiFERON test and, one month later,

tuberculous meningitis. The newborn was therefore the sentinel case that led to the identification and treatment of maternal disease a sequence that carries clear implications for maternal survival and for the prevention of further transmission.

Management follows the principle that congenital and postnatally acquired TB are treated identically: a two-month intensive phase with isoniazid, rifampicin, pyrazinamide and ethambutol, followed by isoniazid and rifampicin for a continuation phase of several months, with corticosteroids added in the presence of meningitis [3,4]. In neonates born to a mother with TB, BCG vaccination is deferred until active disease has been excluded and appropriate prophylaxis or treatment given [4]. Early initiation of therapy is the main determinant of a favourable outcome [2].

This report has the limitations inherent to a single observation, including the absence of bacteriological confirmation by culture or nucleic-acid amplification and incomplete placental histology. Nonetheless, the histological demonstration of caseating hepatic granulomas is sufficient for the diagnosis under the Cantwell criteria, and the case offers a didactic illustration of disseminated congenital TB and of the pivotal role of maternal investigation.

CONCLUSION

Congenital tuberculosis should be considered in any neonate or young infant with unexplained, antibiotic-unresponsive fever, hepatosplenomegaly and a sepsis-like illness, particularly when imaging reveals a disseminated micronodular or miliary pattern. Liver biopsy demonstrating caseating granulomas is diagnostic

under the Cantwell criteria. Equally important, systematic maternal and family investigation must accompany the diagnosis, because the infant may be the first sign of otherwise silent maternal tuberculosis.

Declarations

Ethics approval and consent to participate. This case report was approved by the Institutional Ethics Committee of the Faculty of Medicine and Pharmacy of Fez.

Consent for publication. Written informed consent for publication of this case and any accompanying images was obtained from the infant's parent/legal guardian.

Competing interests. The authors declare that they have no competing interests.

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Authors' contributions. All authors were involved in the clinical management of the patient, contributed to data collection and interpretation, and participated in drafting and critically revising the manuscript for important intellectual content. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work, in accordance with ICMJE authorship criteria.

Abbreviations. TB, tuberculosis; CT, computed tomography; CRP, C-reactive protein; BCG, Bacillus Calmette–Guérin.

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