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Case Report

A silent invader: disseminated tuberculosis presenting with hepatic abscesses in early childhood

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ABSTRACT

Disseminated tuberculosis (TB) is a severe and potentially fatal form of Mycobacterium tuberculosis infection caused by haematogenous spread to two or more non-contiguous organs. Diagnosis is often challenging due to its non-specific clinical presentation and resemblance to other systemic infections. We describe a 9-month-old partially immunized male infant presenting with recurrent vomiting, fever, and poor feeding. Imaging revealed hepatic abscesses and pulmonary nodules. Cerebrospinal fluid GeneXpert detected Mycobacterium tuberculosis. The child was managed with first-line antitubercular therapy, supportive care, and nutritional rehabilitation, with marked clinical improvement. Disseminated tuberculosis in infancy is uncommon but carries high morbidity and mortality. Early clinical suspicion, prompt diagnosis, and timely initiation of therapy are crucial for favourable outcomes.

Keywords: Disseminated tuberculosis, Hepatic abscess, Infant, GeneXpert, Antitubercular therapy

INTRODUCTION

Disseminated tuberculosis (TB) occurs when Mycobacterium tuberculosis spreads haematogenously to two or more non-contiguous organs. Although rare in infancy, several studies have reported its high mortality when diagnosis is delayed.[1] Symptoms are often non-specific, mimicking bacterial or fungal infections.[2] Clinically reported cases indicate that hepatic and adrenal involvement, though uncommon, may signify systemic dissemination.[3] This report highlights the diagnostic challenges of a 9-month-old infant with disseminated TB presenting with hepatic and adrenal lesions.

CASE REPORT

A 9-month-old male infant presented with one month of recurrent non-bilious vomiting, fever, poor feeding, and lethargy. He had received empirical antibiotics at a local hospital without improvement. The child was born at term via caesarean section for non-progression of labour and required NICU admission for poor cry at birth. He was previously hospitalised at 3 months of age for respiratory distress and presumed sepsis. Developmental assessment showed mild global delay, and dietary history indicated inadequate caloric and protein intake. The child was partially immunized up to 6 weeks of age as per the National Immunisation Schedule, and the BCG scar was absent.

On examination, the child was febrile, irritable but consolable, undernourished, and had hepatomegaly (3 cm below the right costal margin). Laboratory evaluation revealed anaemia (Hb 8.3 g/dL), leukocytosis (23,370/mm^3), thrombocytosis, elevated ESR (32 mm/hr), and raised inflammatory markers. Urine microscopy showed pyuria, but culture was sterile. Empirical intravenous ceftriaxone was started.

Abdominal ultrasonography showed two well-defined hypoechoic lesions in the right hepatic lobe, the largest

measuring 4.7×3.5×2.1 cm. Antibiotics were escalated to piperacillin–tazobactam and amikacin. Contrast-enhanced CT of the abdomen revealed rim-enhancing hypodense hepatic lesions compressing adjacent vessels, necrotic abdominal lymph nodes, and bilateral pulmonary nodules. Blood culture grew methicillin-sensitive Staphylococcus aureus (MSSA). A liver biopsy was deferred due to the proximity of the lesions to the portal vein.

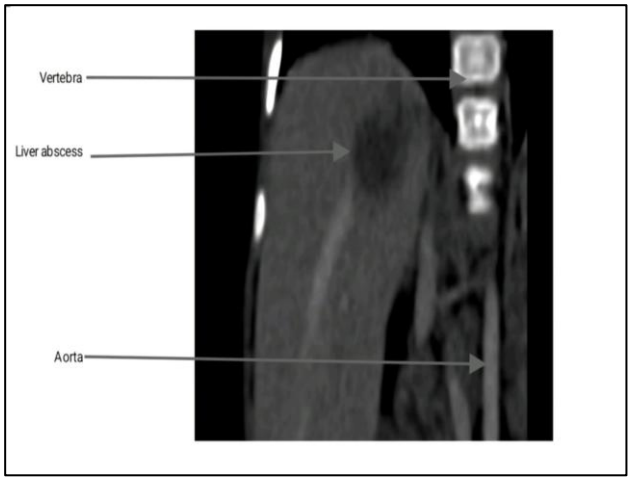

Figure 1: Contrast-enhanced CT (sagittal view) showing hepatic abscess adjacent to the aorta and vertebra

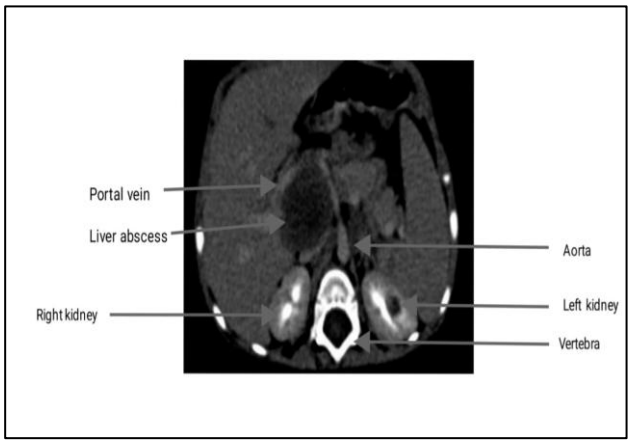

Figure 2: Contrast-enhanced CT (axial view) demonstrating hepatic abscess compressing the portal vein and adjacent structures

Given the miliary pattern on imaging, systemic involvement, and persistence of lesions, disseminated tuberculosis was suspected. Cerebrospinal fluid analysis revealed lymphocytic pleocytosis (70%), elevated protein (433 mg/dL), and low glucose (25 mg/dL); cultures were sterile. CSF GeneXpert detected Mycobacterium tuberculosis with indeterminate rifampicin resistance. Ophthalmological and ENT evaluations were normal. The child was started on first-line antitubercular therapy (isoniazid, rifampicin, pyrazinamide, ethambutol) with pyridoxine, and mannitol for suspected raised intracranial pressure. Nutritional rehabilitation for severe acute malnutrition was initiated.

During the hospital stay, the child became afebrile, gained weight, and showed significant clinical improvement. Follow-up blood cultures were sterile, and haematological parameters normalised. The patient was discharged on continued antitubercular therapy and nutritional support. Evaluation for possible inborn errors of immunity (IEI) was planned in view of disseminated tuberculosis with concurrent MSSA bacteraemia. Catch-up immunisation was scheduled after completion of therapy.

DISCUSSION

Disseminated TB in infancy is uncommon but poses significant diagnostic challenges. Its non-specific manifestations often lead to delayed recognition.[4] In the present case, prolonged fever, vomiting, and hepatomegaly closely matched patterns described in paediatric TB literature.[5]

Hepatic involvement in disseminated TB can mimic pyogenic abscesses radiologically. Rim-enhancing hypodense lesions on CT are well described as characteristic of tuberculous hepatic disease.[6] Similar imaging findings in our patient supported the diagnosis.

Coexisting MSSA bacteraemia complicated the clinical picture. Although rare, previous reports document such dual infections, particularly in children with underlying immune dysfunction.[7] This justified evaluation for possible inborn errors of immunity, especially IL-12–IFN-γ axis defects.[8]

CSF GeneXpert MTB was pivotal in confirming the diagnosis. Its high specificity and rapid turnaround time make it highly valuable in paediatric TB diagnostics.[9] Positive detection enabled early initiation of therapy.

Nutritional rehabilitation significantly contributed to recovery. Malnutrition is a well-known risk factor for severe TB disease.[10] Studies confirm that early antitubercular therapy combined with supportive care reduces mortality.[11]

Thus, each clinical parameter presentation, imaging, laboratory findings, co-infection, and therapeutic response correlated well with previously published paediatric disseminated TB research.

Unlike classical disseminated tuberculosis, where pulmonary or meningeal involvement predominates, hepatic involvement was the primary radiological finding in our patient, leading to a significant diagnostic dilemma. The coexistence of methicillin-sensitive Staphylococcus aureus bacteraemia further confounded the clinical picture and initially favoured a bacterial

etiology, delaying consideration of tuberculosis. The diagnosis was ultimately confirmed only after CSF GeneXpert testing, underscoring the challenges in establishing tuberculosis in infants with non-specific systemic manifestations and overlapping bacterial infections.

CONCLUSION

This case underscores the diagnostic complexity of disseminated tuberculosis in early childhood. A high index of suspicion is warranted in infants presenting with unexplained hepatic abscesses and systemic illness, particularly when immunization is incomplete or malnutrition is present. Early diagnosis and appropriate therapy are key to successful outcomes.

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