

Probable Congenital Tuberculosis in a 1.5-Month-Old Infant: A Rare Case Presentation

ALOK KHANNA¹, DEEPIKA YADAV², JAI BHAGWAN³, DINKAR YADAV⁴, ANJALI VERMA⁵

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ABSTRACT

Congenital Tuberculosis (TB) is a rare but serious condition transmitted from an infected mother in utero or during delivery. Early diagnosis is often challenging due to nonspecific symptoms in neonates. Delayed treatment can lead to high morbidity and mortality. The authors hereby report a rare case of congenital TB in a 1.5-month-old female child who presented with complaints of persistent fever since birth and loose stools for the past four days. She had hepatosplenomegaly, pulmonary involvement with hilar lymphadenopathy and central nervous system involvement. A strong maternal history of pulmonary TB and early postnatal maternal demise raised clinical suspicion. The child responded well to anti-tubercular therapy and corticosteroids. This case emphasises the need for high suspicion of congenital TB in neonates born to mothers with active TB and highlights the importance of early initiation of treatment to improve outcomes.

Keywords: Anti-tubercular treatment, Cantwell criteria, Congenital infection, National Tuberculosis Elimination Programme, Pulmonary tuberculosis

CASE REPORT

A 1.5-month-old female infant presented to the Paediatric emergency with a history of fever since birth and loose stools for the past four days. Fever was undocumented, intermittent and low-grade. Loose stools were 5-6 episodes per day with watery consistency and not mixed with blood. A history of one episode of abnormal body movement in the form of raised tone of bilateral upper and lower limbs with fixation of eyeballs, lasting for a few seconds, occurred four days back with no history of loss of consciousness. The patient's attendant did not seek any medical advice for the seizure. The child was born at 38 weeks of gestation via normal vaginal delivery, with a birth weight of 1.8 kg, which was less than the 10th centile and was Small for Gestational Age (SGA). The infant's mother had a history of fever for one week before delivery and was diagnosed with pulmonary Tuberculosis (TB) and was started on Anti-Tubercular Therapy (ATT). No other close contact was screened. The mother of the child also had a seizure at the time of delivery. She expired 8 days after delivery. The baby was not breastfed and was on exclusive top feeding, receiving powdered milk administered with a Katori-paladai.

On examination, the infant was pale, alert, and irritable with features of dehydration. Vital signs were: Heart Rate (HR)-130/min, Blood Pressure (BP)-72/54 mmHg, Pulse Pressure (PP)- good volume, Capillary Refill Time (CRT)<3 sec, Respiratory Rate (RR)-44/min, SpO₂-99% in room air, with the body temperature-100.8°F. The weight of the child at admission was 2.1 kg. On respiratory system examination, there were subcostal retractions. Hepatosplenomegaly was noted on per abdomen examination, as shown in [Table/Fig-1]. Further investigations were planned to evaluate the child. Ultrasonography (USG) brain was normal and USG whole abdomen revealed hepatosplenomegaly. Fundus examinations also revealed no abnormality. Chest X-ray showed consolidation in the upper and middle right lobes with bilateral hilar lymphadenopathy as shown in [Table/Fig-2].

Laboratory investigations were:

Chest X-Ray was suggestive of bilateral infiltrate with hilar lymphadenopathy. Erythrocyte Sedimentation Rate (ESR) was also raised. Cerebrospinal fluid obtained and showed proteins 58

mg/dL, sugar 36 mg/dL (serum glucose level was 89 mg/dL) and 60 cells/mm³ with lymphocytic predominance (70%). She had a positive tuberculin test with 10 mm induration at 48 hours. Catridge-Based Nucleic Acid Amplification Test (CBNAAT) of the gastric aspirate sample was positive for *Mycobacterium tuberculosis* and rifampicin resistance was not detected [Table/Fig-3]. All the above findings were suggestive of tubercular aetiology. Hence, the baby was diagnosed as a case of probable congenital TB with pulmonary involvement.



[Table/Fig-1]: showing a child with congenital Tuberculosis (TB) with hepatosplenomegaly and chest retractions.



[Table/Fig-2]: Chest X-ray showing consolidation in the right lobe.

S. No.	Investigations	Results	References
1.	Hb (g/dL)	8.3	10-17
2.	WBC (/cumm)	17000	4000-11000
3.	CRP	Positive	0.1-1 mg/dL
4.	Neutrophils/Lymphocytes	30%/67%	
5.	Platelet count (lac)	2.4	1.5-5.5
6.	Urea/Creatinine (mg/dL)	34/0.4	3.4-16.8; 0.1-0.3
7.	SGOT/SGPT (U/L)	36/34	20-67; 5-33
8.	Total bilirubin/Direct bilirubin (mg/dL)	0.4/0.1	0.5-0.68 /0.05-0.3
9.	Na/K (mEq/dL)	142/3.8	135-145; 3.5-5.5
10.	ESR	38 mm in the first hour	
11.	Chest X-ray [Table/Fig-2]	Bilateral infiltrates with hilar lymphadenopathy	
12.	USG abdomen	Hepatosplenomegaly	
13.	CBNAAT - CSF	Negative	
14.	Mantoux	10 mm	
15.	CSF analysis	60 cells/mm ³ (70% lymphocytes, 30% neutrophils), protein 58 mg/dL, glucose 36 mg/dL, culture and sensitivity did not show any growth.	
16.	PT/INR	32/1.2	
17.	CBNAAT (gastric aspirate)	Positive for Mycobacterium tuberculosis	

[Table/Fig-3]: showing laboratory investigations of the child. Hb: Haemoglobin; WBC: White blood cells; CRP: C-reactive protein; SGOT: Serum glutamate oxaloacetate transaminase; SGPT: Serum glutamate pyruvate transaminase; ESR: Erythrocyte sedimentation rate; CBNAAT: Cartridge-based nucleic acid amplification test; CSF: Cerebrospinal fluid; PT: Prothrombin time; INR: International normalised ratio

After confirmation of diagnosis, the child was initiated on first-line ATT. Patient was given HRZE (H-25 mg, R-38 mg, Z-75 mg, E-50 mg) for the initial 2 months and HR for the next 9 months with Pyridoxine throughout the ATT course. Corticosteroid (Dexamethasone 0.15 mg/kg/dose QID) was also given for 6 weeks, which was tapered over 4 weeks. Gradual clinical improvement was observed in fever and general condition. Supportive care and nutritional management

were continued. Supportive care in the form of maintaining hydration, temperature and hygiene. Measured KS feeds were given with vitamin D supplementation. IFA was also advised. Attendants were also counselled for treatment adherence. The child remained under follow-up every month with regular assessment for treatment response and developmental progress. After initiation of first-line anti-tubercular therapy and supportive care, the infant showed gradual clinical improvement, including resolution of fever. The child was last followed up on 1 month ago and adequate weight gain was present with no evidence of developmental delay.

DISCUSSION

Perinatal TB is rare, but when it does occur, it is most often due to the mother having genital tract TB, allowing the infection to spread to the foetus before birth. Transmission to the foetus can occur either through the placenta (via the bloodstream) or by the baby inhaling or swallowing infected amniotic fluid during birth [1]. The average prevalence of all forms of TB in India is estimated to be 5.05 per thousand, the prevalence of smear-positive cases is 2.27 per thousand, and the average annual incidence of smear-positive cases is 84 per 1,00,000 annually [2].

TB that spreads through the bloodstream can affect multiple organs and often remains hidden until the immune system is triggered. In such cases, the infection can reactivate and cause disease after birth. TB affecting the lungs is rare in newborns unless they aspirate infected fluid during delivery. Symptoms generally appear in the second or third week of life. Common signs include fever, difficulty breathing, an enlarged liver or spleen, poor feeding, lethargy, irritability, weight loss, abdominal distention, failure to gain weight, ear discharge, and skin lesions [3]. Perinatal TB, though rare, should be suspected in neonates with fever of unknown origin, hepatosplenomegaly, and a history of maternal TB. When mothers are diagnosed and/or treated for TB in pregnancy, their neonates should be screened for TB at birth or within the first days of life [4].

TB in newborns can look similar to infections like bacterial sepsis or other congenital infections {e.g., syphilis, toxoplasmosis, Cytomegalovirus (CMV)}. It should be suspected if a newborn does not improve with antibiotics and no clear cause is found. A key clue is a maternal history of TB, though the mother's TB is often only found after the baby's illness is diagnosed. The Tuberculin Skin Test (TST) is usually negative at first but may become positive in 1-3 months. Diagnosis may involve CSF analysis, imaging, and molecular testing such as CBNAAT. Cantwell modified the criteria diagnosis of congenital TB; it requires (a) Mycobacterium tuberculosis lesion and (b) 1 of the following secondary findings: (1) Primary hepatic complex (caseating granuloma) on biopsy, (2) lesions from any source (i.e., pulmonary, hepatic, and skin) in the first weeks after birth, (3) exclusion of postnatal transmission through a thorough investigation of contacts, or (4) TB infection of the maternal genital tract and/or placenta [5] [Table/Fig-4] [6,7] presents the comparative analysis of similar cases.

Congenital TB, in this case, has not been established because the histopathological examination of the placenta was not performed. Infants with congenital TB may be asymptomatic at birth, but symptoms can occur within days to weeks after birth. This is due to the different immune status of each newborn and the onset of disease may be slower in some children [8]. Singh M et al., [9] reported clinical and laboratory findings to investigate for TB as a) if newborn with unresponsive worsening pneumonia, particularly in those from endemic areas, b) if the mother was diagnosed to have TB and baby has non-specific symptoms, c) when the CSF revealed a high lymphocyte count in the absence of any identifiable bacterial pathogen and d) in presence of fever and hepatosplenomegaly.

Early initiation of ATT significantly improves prognosis. According to National Tuberculosis Elimination Programme India, management begins with a daily Fixed Dose Combination of Anti Tubercular

S. No.	Case Report	Year	Symptom	Investigation	Treatment	Recovery
1.	Bor Ö et al., [6]	2007	Fever, Respiratory distress, Failure to thrive	Positive Tuberculin test, GA yielded acid-fast bacilli, CSF protein 117 mg/dL, glucose 3 mg/dL, 55 polymorphonuclear leukocytes and 44 lymphocytes/mm ³	HRZ (2 months) + HR (9 months) with steroids for 3 months	Yes
2.	Yeh JJ et al., [7]	2019	Fever, abdominal distension	CT showed multiple nodules over the lung, spleen, and hepatic hilar region. Both gastric lavage and pleural effusion cultures confirmed the diagnosis of TB.	HRZE (2 months) + HR (9 months) with steroids	Yes
3.	Present case	2025	Fever, Loose stools, Seizure	Chest X-ray showed B/L infiltrate with hilar lymphadenopathy, Mantoux positive, CBNAAT for GA positive, CSF shows 60 cells with 70% lymphocytes	HRZE (2 months) + HR (9 months) with 10 weeks steroids	Yes

[Table/Fig-4]: Comparative analysis of similar cases [6,7].

Treatment containing isoniazid, rifampicin, pyrazinamide, and ethambutol for two months. It is followed by four months of isoniazid, rifampicin, and ethambutol [10]. Clinical follow-up for recurrence of TB, either relapse or reinfection, and post-TB disease is important [11]. The death rate is high if diagnosis is delayed. A study of 170 congenital TB cases reported between 1994 and 2009 discovered a high mortality rate; of these cases, a total of 68 patients and patients without treatment died [12]. However, outcomes are much

better with early diagnosis and proper treatment. In areas where TB is common, doctors should consider it even when a newborn's only symptom is persistent fever.

CONCLUSION(S)

This case highlights the importance of early recognition and treatment of congenital TB, especially in neonates born to mothers with active TB. A multidisciplinary approach involving paediatricians, microbiologists, and radiologists is essential for timely diagnosis and management.

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PARTICULARS OF CONTRIBUTORS:

- 1. Professor, Department of Paediatrics, PGIMS, Rohtak, Haryana, India.
- 2. Junior Resident, Department of Paediatrics, PGIMS, Rohtak, Haryana, India.
- 3. Junior Resident, Department of Paediatrics, PGIMS, Rohtak, Haryana, India.
- 4. Senior Resident, Department of Paediatrics, PGIMS, Rohtak, Haryana, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Dinkar Yadav,
Senior Resident, Department of Paediatrics, PGIMS, Rohtak-124001,
Haryana, India.
E-mail: dr.dinku2012@gmail.com

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Jun 18, 2025
- Manual Googling: Oct 30, 2025
- iThenticate Software: Nov 01, 2025 (20%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

Date of Submission: Jun 14, 2025
Date of Peer Review: Aug 28, 2025
Date of Acceptance: Nov 04, 2025
Date of Publishing: Mar 01, 2026