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Case Report

Stage III tuberculous meningoencephalitis with brain abscess in a young infant: a rare case report

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ABSTRACT

Tuberculous meningitis (TBM) is the most severe form of TB in children and is associated with high mortality and related morbidities. Some of the rare complications of TBM, especially in infants, are formation of brain abscess, hydrocephalus, vasculitis, and cerebral infarction. We reported a case of a 3 ½ months old female infant who presented with fever, cough, irritability, poor feeding, and continuous convulsions. Investigations revealed basal meningeal exudates, multiple cerebral and brainstem infarcts, vasculitis, tuberculomas, hydrocephalus, and a left parietal brain abscess. CSF findings were suggestive of TBM. The patient was diagnosed with stage III tuberculous meningoencephalitis complicated by multiple deep cortical and brainstem infarcts, vasculitis, tuberculomas, hydrocephalus, brain abscess and status epilepticus. She was managed successfully with anti-tubercular therapy, corticosteroids, antiepileptics, abscess evacuation, Ommaya reservoir insertion, and ventriculoperitoneal (VP) shunting. Significant improvement has been noted, and she is in stable condition.

Keywords: Tuberculous meningitis, Infant, Brain abscess, Hydrocephalus, Vasculitis, Cerebral infarction, Tuberculoma, Status epilepticus

INTRODUCTION

In developing countries such as India, TB is a significant epidemiological threat to children worldwide.[1] India contributes substantially to the global TB burden, with children younger than 15 years accounting for approximately 10-12% of all TB cases.[2] An estimated 0.3 to 0.35 million pediatric TB cases are reported annually in India, contributing significantly to childhood morbidity and mortality.[3]

A recent epidemiological shift has been noted from classical pulmonary manifestation towards extrapulmonary forms, involving the increased intracranial pressure, abscess, vasculitis, cerebral infarctions, and hydrocephalus.[4] Further, the COVID-19 pandemic has also influenced the epidemiology of infectious diseases. The COVID-19 pandemic disrupted TB control programmes and healthcare access, potentially contributing to delayed diagnosis and increased disease severity and goals.[2] These changes have highlighted the need for greater clinical awareness.

TBM is the most severe form of extrapulmonary TB associated with high mortality and long-term neurological morbidity.[4] Young infants often present with nonspecific symptoms like fever, cough, excessive irritability, malnutrition, and seizures.[4,5] Delay in diagnosis can lead to complications and even death in certain cases. The formation of an intracranial abscess in young infants with Tuberculous meningoencephalitis makes it a rare case.[4,5]

We encountered this rare case of stage III tuberculous meningoencephalitis in a young infant complicated by multiple deep cortical and brainstem infarcts, vasculitis, tuberculomas, hydrocephalus and abscess formation.[5]

However, we successfully treated this patient by a multidisciplinary approach involving paediatricians, neurologists, radiologists by giving AKT, anti-epileptic medications, fixed dose combinations of anti-TB tablets, followed by evacuation of abscess and VP shunting.[5]

CASE REPORT

A 3-month-old female infant 3rd issue of a non-consanguineous marriage (NCM), was brought with complaints of cough for 8-7 days, fever (on and off) for 8-10 days, excessive irritability for 3 days, and continuous convulsions for 1 hour on the date of admission. The infant was apparently alright 4 days prior to admission, then started developing a cough and cold. Cough, which is dry in nature, is not associated with post-tussive vomiting. A fever that is acute in onset, mild to moderate grade, intermittent in frequency, and gradually progressive in nature, relieved on taking medication and again reappears, is associated with excessive irritability. For that child was 1st shown to the outside hospital on the OPD basis and received symptomatic treatment, but the fever did not subside.

Hence, the child was again taken to the outside hospital on 4th March 2026, and blood investigations were done and told to be abnormal and referred to our hospital for further management. There is a history of poor feeding. No history of yellowish discolouration of eyes, vomiting, loose stools, trauma, bleeding manifestations, or urine output. No significant past history. Family history was positive for TB in a household contact. Full-term LSCS, cried immediately after birth.

On examination

Comatose baby, temperature (104°F), heart rate-240 beats/min, respiratory rate-60 breaths/min, SpO_2-98% on O_2 prongs, peripheral pulses well felt, extremities warm, hydration adequate, some pallor present, no oedema, no lymphadenopathy, no icterus, no cyanosis. There was hepatomegaly on per abdominal examination.

Provisional diagnosis

Acute CNS infection with refractory status epilepsy.

Hospital course

The child was admitted to the paediatric ward with the above-mentioned complaints. On admission, the child had continuous convulsions, febrile (104 F).

Investigations were done, CBC report showed Hb-7.8 (normal range-12-20 g/dl), TLC-3400 (normal range 4000-11000/mm^3) and platelet 236000 (normal range: 150000-450000/mm^3), polymorph-10 (normal range 40-70%), Lymphocytes-86 (normal range 20-40%), QCRP: 228.

Peripheral blood smear

On 04/03/26, RBC morphology-Reduced erythron, microcytosis, hypochromic, anisopoikilocytosis, tear drop cells seen.

WBC morphology-Leukopenia, relative Lymphocytes reactive. Parasite-No parasites are seen.

Tracheal aspirate

On 24/03/26, Gram stain-pus cells>25/lpf, epithelial cells 10-25/lpf, many gram-positive cocci in pairs seen. AFB stain-no acid-fast bacilli seen. Culture and sensitivity-No pathogen isolated.

Gastric aspirate

On 07/03/26, ZN stain-AFB not seen.

Renal function test

On 04/03/26 Sr creatinine (mg/dl)-0.4, Sr urea (mg/dl)-32. On 10/03/26 Sr creatinine (mg/dl)-0.3, Sr urea (mg/dl)-17, Sr creatinine (mg/dl)-0.4, Sr urea (mg/dl)-12, Sr creatinine (mg/dl)-0.4, Sr urea (mg/dl)-19.

Sputum for AFB (mother)-acid-fast bacilli not seen. Sputum for AFB (father)-acid-fast bacilli not seen.

Coagulation profile

On 04/03/26 PT was 21.4, APTT- 48.3 and the INR was 1.93.

Serology

On 17/03/26 HIV-non-reactive, HCV-negative, HBsAg-negative.

Liver function test

SGOT was 56, SGPT was 16 and total bilirubin 0.2 mg/dl.

CSF findings

WBC-16, polymorphs-20, lymphocytes-80, sugar-51 mg/dl, protein-1797 mg /dl, Gram stain-no pus cells and no organism, AFB stain-no acid-fast bacilli seen, CBNAAT-negative, CSF culture-sterile.

MRI brain

MRI brain suggestive of meningitis with basal exudates-possibly tubercular etiology.

Secondary vasculitis and vasospasm seen with resultant bilateral acute cerebral and brainstem infracts.

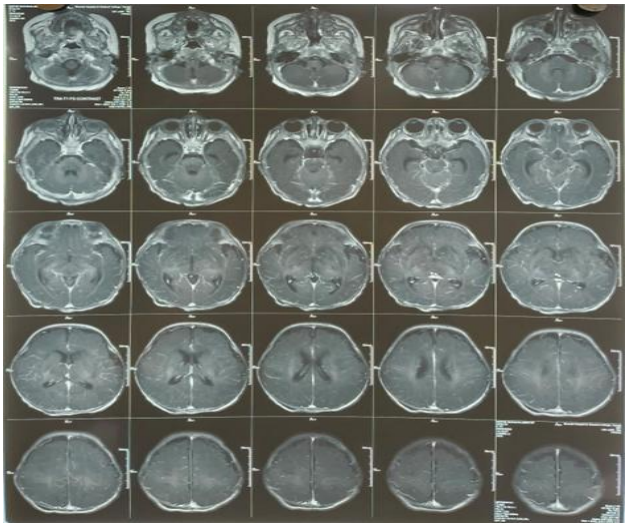

Figure 1: (Post contrast axial T1) basal meningeal enhancement.

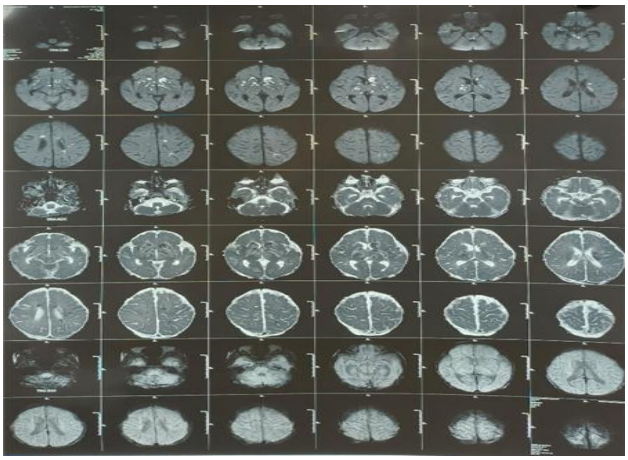

Figure 2: (DWI/ADC) bilateral basal ganglia infarcts.

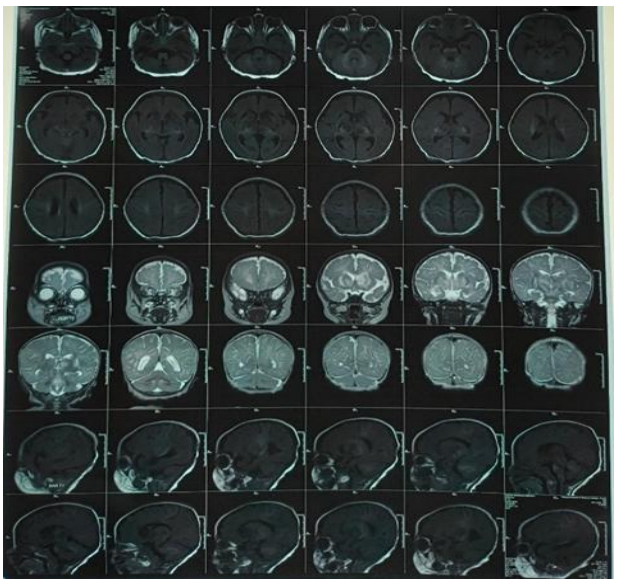

Figure 3: (Coronal T2) dilatation of ventricles suggestive of obstructive hydrocephalus.

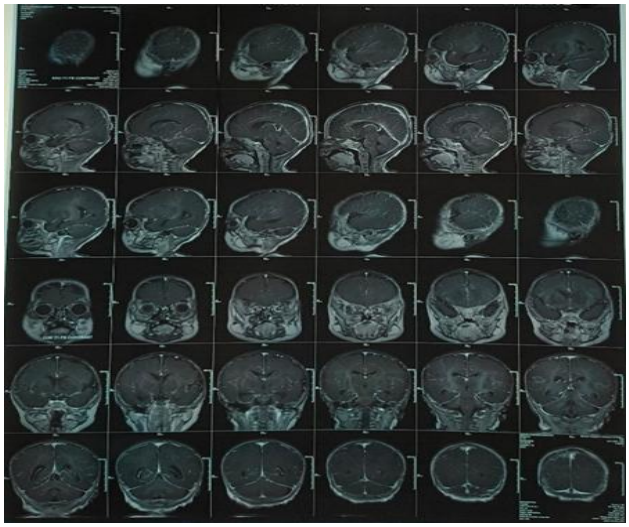

Figure 4: (Post contrast sagittal/coronal) basal leptomeningeal enhancement.

EEG

It is abnormal due to epileptiform discharges arising from the right Fronto-temporal region, diffusely showing background.

CT brain plain and contrast

On follow-up, complicated CNS TB with tubercular meningitis, multiple tuberculomas and an enlarging left parietal brain abscess, associated with hydrocephalus and diffuse cerebral oedema, with post-operative changes (Craniotomy + Ommaya reservoir) and old infarcts-overall showing disease progression.

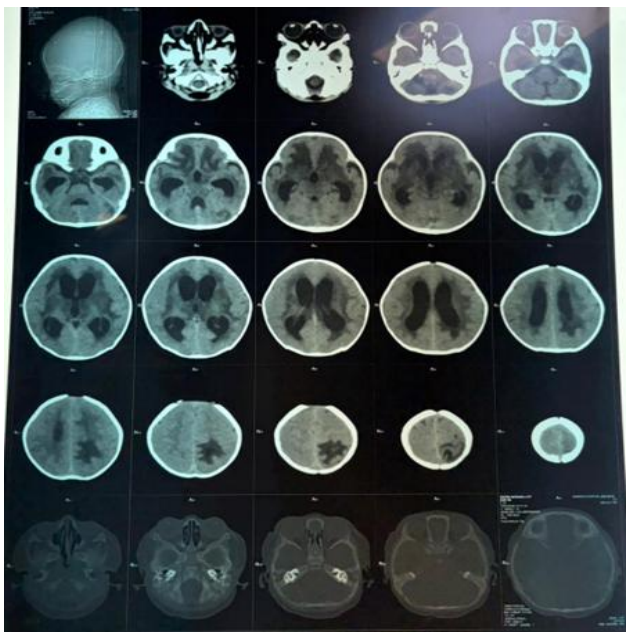

Figure 5: Axial CT brain showing marked dilatation of the lateral and third ventricles, suggestive of hydrocephalus.

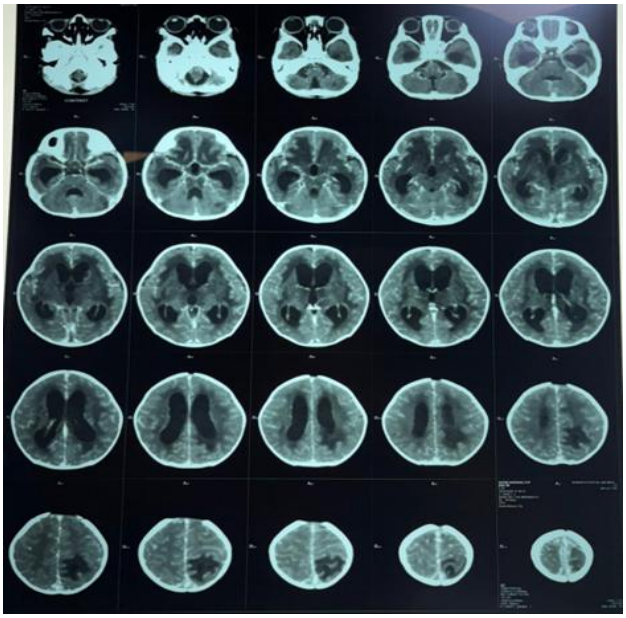

Figure 6: Contrast-enhanced axial CT brain showing basal meningeal enhancement in the basal cisterns, characteristics of tuberculous meningitis.

Treatment plan

All the investigations, i.e. CSF, MRI and CBC with contrast findings, were favouring TBM even when CSF CBNAAT was negative. So, the child was started on AKT, and started with 4 FDC tablets as per weight. Tab 3 FDC (Paediatric) Tab rifampicin, tab isoniazid, tab pyrazinamid-1 tablet plus 1 tab etambutol 100 mg, vitamin D3 sachets 60,000 IU 1 sachet for 6 weeks. After the Neurosurgery consultation, the advice; Abscess evacuation and Ommaya reservoir insertion were done. Special needs regarding care for the child were: adequate hydration, healthy diet, feeding tube care and attention. VP shunting was done after four weeks of admission, in view of gross hydrocephalus on CT brain.

Final diagnosis

Tuberculous meningoencephalitis stage III [according to BMRC staging system], brain abscess (evacuation done), multiple deep cortical and brainstem infracts, vasculitis, hydrocephalus (Ommaya reservoir inserted), VP Shunting done, tuberculomas, status epilepticus and anaemia.

DISCUSSION

We reported a case of Stage III Tuberculous meningoencephalitis (TBM) in a 3 ½ months old female infant who presented with fever, cough, excessive irritability, and continuous convulsions.[6] On neurological investigations, it showed basal meningeal enhancement, hydrocephalus, multiple cerebral and brainstem infarcts, tuberculomas, and formation of a brain abscess.[4] CSF findings show elevated protein levels and decreased glucose levels. Hence, the diagnosis was confirmed based on the above findings. So, the treatment included anti-tubercular therapy, corticosteroids, antiepileptic medications and neurosurgical interventions. During the course of the hospital stay, the infant was now clinically and haemodynamically stable, and there were no fresh complaints.

The delayed diagnosis and treatment of TBM can lead to a major setback in developing countries such as India.[3,7] As these countries have low access to healthcare services, environmental factors such as overcrowding, poor socioeconomic conditions, pollution, lack of nutrition, low ventilation, delayed control of TB programs and lack of awareness of it.[3,8] Therefore, the number of paediatric TBM cases is rising.[8]

The clinical course of TBM is typically subacute, with a median symptom duration of 10 days (range: one to nine months) before diagnosis.[7,9] The predominant symptoms are low-grade fever, malaise, headache, and hydrocephalus.[7,9] Nuchal rigidity may be absent, particularly in younger patients.[7,9] These nonspecific symptoms make early diagnosis challenging.[7,9] Thus, imaging studies, such as brain MRI, are useful for diagnosing and assessing complications.[7,9] The MRI findings of TBM include basilar meningeal enhancement and hydrocephalus with potential brainstem infarctions.[1,7] For patients presenting with prolonged neurological findings for a few weeks, clinicians should 1) investigate if family members have had prolonged fever and cough, 2) check Bacillus Calmette-Guerin vaccination status, and 3) consider imaging studies.[1,9] When basilar meningeal enhancement or brainstem infarction is observed on MRI, CSF examination, which may also give characteristic findings of TBM, and early anti-TB treatment are recommended.[7,9]

The presence of multiple complications in a young infant makes the TBM case different.[1] The most common complication is cerebral infarct, which results from the vasculitis, i.e. inflammation of the blood vessels around the basal cisterns.[4,9,10] Infarctions involving deep cerebral parts have a high mortality rate.[10] Even hydrocephalus also occurs due to obstruction of cerebrospinal fluid pathways, mainly seen in advanced cases of TBM.[7] Brain abscess formation is uncommon in TB, specifically in young infants.[4] Similar cases reported in the literature have described hydrocephalus and cerebral infarction. However, the coexistence of TBM, multiple tuberculomas, brain abscess, cerebral infarctions, vasculitis, refractory status epilepticus, hydrocephalus, and in a 3-month-old young infant makes this case exceptionally rare.[1,4] Our patient reported all the above complications. Therefore, it makes the case even more uncommon.[1]

Regarding the management of complicated TBM, a multidisciplinary approach, in which our patient was started with anti-tubercular therapy, corticosteroids, antiepileptic medications, abscess evacuation with

Ommaya reservoir placement and the insertion of the VP shunt.[1,7,9] Now the patient has achieved hemodynamic and clinical improvement on time with the medical and neurosurgical intervention.

CONCLUSION

This case highlights the young infant with a rare condition of stage III Tuberculous meningoencephalitis with brain abscess, brain stem infarcts, and tuberculomas. Early diagnosis and neuro-investigations and treatment with anti-tubercular therapy, anti-epileptic medications and some neurosurgical procedures like abscess evacuation and insertion of VP Shunt can improve and reduce the adverse neurological outcomes in paediatric cases of TBM.

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