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Stage 2 Tuberculous Meningitis Mimicking Acute Disseminated Encephalomyelitis in a Child: A Diagnostic Challenge

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Abstract: ***Background**: Tuberculous meningitis (TBM) is one of the most severe forms of childhood tuberculosis, accounting for high morbidity and mortality. Its clinical spectrum often overlaps with other infectious and inflammatory conditions of the central nervous system (CNS), such as acute disseminated encephalomyelitis (ADEM). Rapid and accurate diagnosis is critical for timely initiation of anti-tubercular therapy (ATT). **Case Presentation**: A 9-year-old girl, born of second-degree consanguineous marriage, presented with acute progressive quadriparesis, cranial nerve palsies, headache, vomiting, fever, and altered sensorium. On admission, she was lethargic with GCS 12/15, right abducens (VI) and facial (VII) nerve palsies, hypotonia, and hyporeflexia. MRI brain revealed ill-defined, non-enhancing signal changes in the brainstem, cerebellum, and periventricular region, suggestive of demyelination. Nerve conduction studies were normal. She received intravenous methylprednisolone with initial improvement in neurological status. However, cerebrospinal fluid (CSF) analysis later tested positive for Mycobacterium tuberculosis using cartridge-based nucleic acid amplification test (CBNAAT). ATT was initiated, leading to marked clinical recovery with normalization of cranial nerve function and motor strength. **Conclusion**: TBM may mimic ADEM in pediatric patients, particularly in tuberculosis-endemic areas. CBNAAT of CSF is an essential diagnostic tool for early and accurate detection. Prompt recognition and treatment can significantly reduce morbidity.*

Keywords: Tuberculous meningitis, acute disseminated encephalomyelitis, CSF CBNAAT, pediatric neurology, case report

1. Introduction

Tuberculosis (TB) remains a major global health burden, with India contributing to the highest number of cases worldwide. Tuberculous meningitis (TBM) represents the most severe and life-threatening form of extrapulmonary TB in children. The clinical presentation is often subacute but can be acute in some cases, mimicking other neuroinflammatory disorders. Acute disseminated encephalomyelitis (ADEM) is one such differential, characterized by post-infectious or post-vaccination demyelination of the CNS. Both conditions can present with fever, headache, altered sensorium, focal neurological deficits, and radiological findings of multifocal brain lesions.

Despite advances in neuroimaging, diagnosis of TBM remains challenging due to overlapping features. While MRI may suggest demyelination, it is not definitive for TBM. Conventional microbiological methods such as smear microscopy and culture have limited sensitivity and require prolonged turnaround time. The advent of nucleic acid amplification tests such as Xpert MTB/RIF and CBNAAT has improved diagnostic accuracy, particularly in extrapulmonary TB. Here, we present a case where TBM initially masqueraded as ADEM, highlighting the importance of molecular diagnostics.

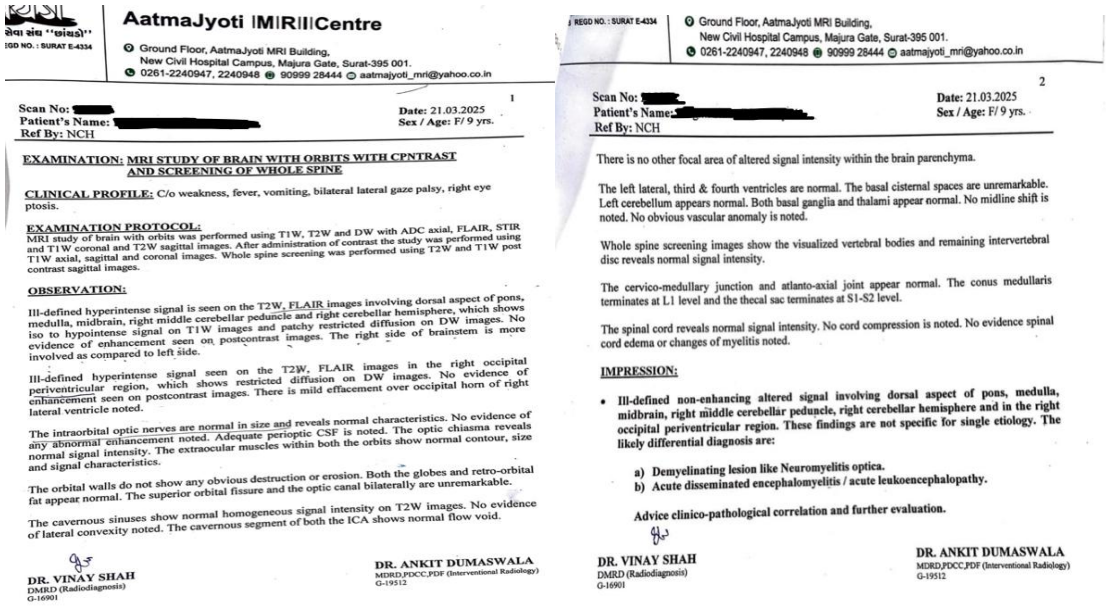
2. Case Presentation

A 9-year-old female child, the fifth born of a second-degree consanguineous marriage, residing in a city in South Gujarat. Pt was admitted with weakness of all four limbs for 6 days, recurrent vomiting for 5 days, headache for 3 days, and fever for 2 days. The weakness began in the lower limbs, was acute in onset, and progressively ascended to involve the upper limbs and trunk within 48 hours. She was unable to climb stairs, rise from the floor, or walk without support. Vomiting was frequent, non-bilious, and non-projectile. Headache was dull-aching and diffuse. Fever was high-grade without chills or rigors. On the day prior to admission, she developed decreased responsiveness.

She had no history of convulsions, trauma, recent infections, ear discharge, cough, or tuberculosis contact. Her immunization status was complete till 5 years of age. Developmental history and scholastic performance were normal. She belonged to a lower middle-class family and was on a calorie- and protein-deficient diet.

On examination, she was lethargic and disoriented. Her vitals were stable. Pallor was present but there was no icterus, clubbing, lymphadenopathy, or cyanosis. Anthropometry was appropriate for age and not severely malnourished. Neurological examination revealed cranial nerve involvement: right eye ptosis, esotropia, incomplete eyelid closure during sleep, and deviation of the mouth angle suggestive of right-sided abducens (VI) and facial (VII) nerve palsies. Motor examination showed hypotonia and decreased power (UL 3/5, LL 2/5) with hyporeflexia. Sensory system was intact. No cerebellar signs were elicited.

MRI brain demonstrated ill-defined, non-enhancing hyperintense lesions involving the dorsal pons, medulla, midbrain, right cerebellar hemisphere, and periventricular white matter, initially suggestive of demyelinating pathology such as ADEM or neuromyelitis optica. CSF analysis was performed after ruling out papilledema and showed positive results for Mycobacterium tuberculosis on CBNAAT. Nerve conduction studies were normal.



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Scan No: [redacted] Date: 21.03.2025
Patient's Name: [redacted] Sex / Age: F/ 9 yrs.
Ref By: NCH

EXAMINATION: MRI STUDY OF BRAIN WITH ORBITS WITH CPNTRAST AND SCREENING OF WHOLE SPINE

CLINICAL PROFILE: C/o weakness, fever, vomiting, bilateral lateral gaze palsy, right eye ptosis.

EXAMINATION PROTOCOL:
MRI study of brain with orbits was performed using T1W, T2W and DW with ADC axial, FLAIR, STIR and T1W coronal and T2W sagittal images. After administration of contrast the study was performed using T1W axial, sagittal and coronal images. Whole spine screening was performed using T2W and T1W post contrast sagittal images.

OBSERVATION:

Ill-defined hyperintense signal is seen on the T2W, FLAIR images involving dorsal aspect of pons, medulla, midbrain, right middle cerebellar peduncle and right cerebellar hemisphere, which shows iso to hypointense signal on T1W images and patchy restricted diffusion on DW images. No evidence of enhancement seen on postcontrast images. The right side of brainstem is more involved as compared to left side.

Ill-defined hyperintense signal seen on the T2W, FLAIR images in the right occipital periventricular region, which shows restricted diffusion on DW images. No evidence of enhancement seen on postcontrast images. There is mild effacement over occipital horn of right lateral ventricle noted.

The intraorbital optic nerves are normal in size and reveals normal characteristics. No evidence of any abnormal enhancement noted. Adequate perioptic CSF is noted. The optic chiasma reveals normal signal intensity. The extraocular muscles within both the orbits show normal contour, size and signal characteristics.

The orbital walls do not show any obvious destruction or erosion. Both the globes and retro-orbital fat appear normal. The superior orbital fissure and the optic canal bilaterally are unremarkable.

The cavernous sinuses show normal homogeneous signal intensity on T2W images. No evidence of lateral convexity noted. The cavernous segment of both the ICA shows normal flow void.

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There is no other focal area of altered signal intensity within the brain parenchyma.

The left lateral, third & fourth ventricles are normal. The basal cisternal spaces are unremarkable. Left cerebellum appears normal. Both basal ganglia and thalami appear normal. No midline shift is noted. No obvious vascular anomaly is noted.

Whole spine screening images show the visualized vertebral bodies and remaining intervertebral disc reveals normal signal intensity.

The cervico-medullary junction and atlanto-axial joint appear normal. The conus medullaris terminates at L1 level and the thecal sac terminates at S1-S2 level.

The spinal cord reveals normal signal intensity. No cord compression is noted. No evidence spinal cord edema or changes of myelitis noted.

IMPRESSION:

- **Ill-defined non-enhancing altered signal involving dorsal aspect of pons, medulla, midbrain, right middle cerebellar peduncle, right cerebellar hemisphere and in the right occipital periventricular region. These findings are not specific for single etiology. The likely differential diagnosis are:**
  - **a) Demyelinating lesion like Neuromyelitis optica.**
  - **b) Acute disseminated encephalomyelitis / acute leukoencephalopathy.**

Advice clinico-pathological correlation and further evaluation.

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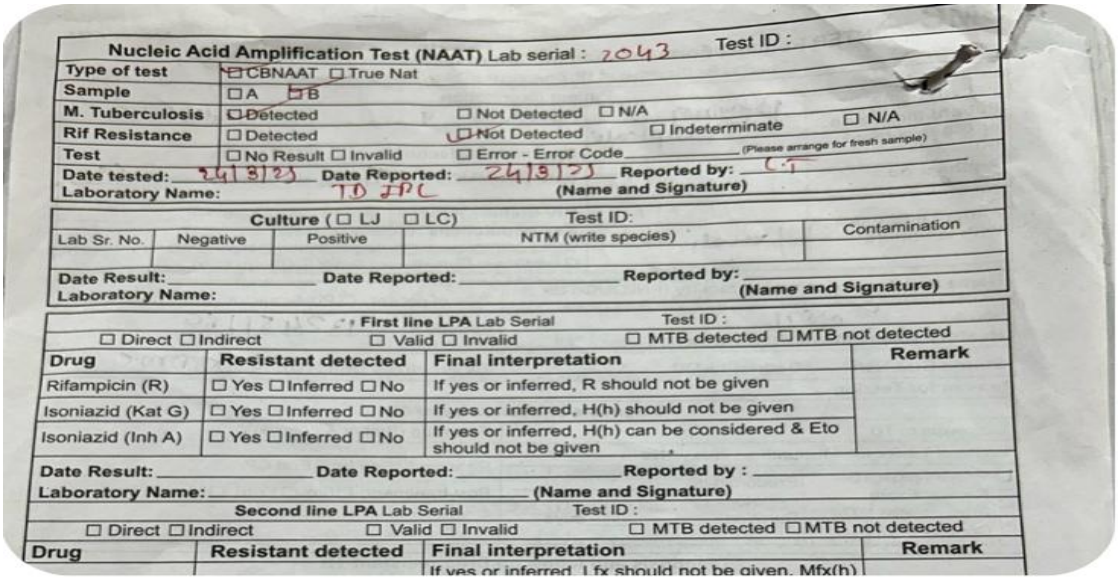
Image 1: MRI Brain with Orbit with contrast and screening of whole spine

She was initially managed with broad-spectrum antibiotics, antivirals, and intravenous methylprednisolone (30 mg/kg/day) for 5 days. Neurological improvement was noted within 24 hours, with better orientation, motor power, and speech. However, transient behavioral disturbances such as agitation and mood changes occurred, requiring psychiatric evaluation.

Upon receiving positive CBNAAT results, a diagnosis of Stage 2 TBM was made. Anti-tubercular therapy (isoniazid, rifampicin, pyrazinamide, and ethambutol) was initiated. The child showed steady improvement with complete resolution of cranial nerve palsies and regained ambulatory function. She was discharged after 9 days of hospitalization.



Nucleic Acid Amplification Test (NAAT) Lab serial : 2043 Test ID :

Type of test	☐CBNAAT ☐True Nat
Sample	☐A ☐B
M. Tuberculosis	☐Detected ☐Not Detected ☐N/A
Rif Resistance	☐Detected ☐Not Detected ☐Indeterminate ☐N/A
Test	☐No Result ☐Invalid ☐Error - Error Code _____ (Please arrange for fresh sample)

Date tested: 24/3/25 Date Reported: 24/3/25 Reported by: C.T
Laboratory Name: TD JPC (Name and Signature)

Culture (☐LJ ☐LC) Test ID:

Lab Sr. No.	Negative	Positive	NTM (write species)	Contamination

Date Result: _____ Date Reported: _____ Reported by: _____
Laboratory Name: (Name and Signature)

First line LPA Lab Serial Test ID :
☐Direct ☐Indirect ☐Valid ☐Invalid ☐MTB detected ☐MTB not detected

Drug	Resistant detected	Final interpretation	Remark
Rifampicin (R)	☐Yes ☐Inferred ☐No	If yes or inferred, R should not be given	
Isoniazid (Kat G)	☐Yes ☐Inferred ☐No	If yes or inferred, H(h) should not be given	
Isoniazid (Inh A)	☐Yes ☐Inferred ☐No	If yes or inferred, H(h) can be considered & Eto should not be given	

Date Result: _____ Date Reported: _____ Reported by : _____
Laboratory Name: _____ (Name and Signature)

Second line LPA Lab Serial Test ID :
☐Direct ☐Indirect ☐Valid ☐Invalid ☐MTB detected ☐MTB not detected

Drug	Resistant detected	Final interpretation	Remark
		If yes or inferred, Lfx should not be given, Mfx(h)	

Image 2: CBNAAT Report of Patient

3. Discussion

This case illustrates the diagnostic challenge of distinguishing TBM from ADEM in pediatric patients. Both conditions may present with acute onset neurological deficits, cranial neuropathies, and similar MRI findings. ADEM typically follows a viral illness or vaccination, presenting with multifocal neurological deficits and bilateral MRI lesions in the white matter. In contrast, TBM is characterized by basal meningeal inflammation, cranial nerve involvement, and hydrocephalus in advanced stages, but atypical presentations may mimic demyelination.

Our patient presented with acute quadriparesis and cranial nerve palsies, with MRI findings initially favoring ADEM. The transient improvement with corticosteroids further supported this impression. However, the positive CSF CBNAAT was decisive in confirming TBM. Steroids have a role in both conditions: in ADEM as the primary treatment, and in TBM as adjunctive therapy to reduce inflammatory damage.

The utility of CSF CBNAAT in TBM has been established in multiple studies, with specificity approaching 100% and sensitivity ranging from 50–70%. Its ability to detect rifampicin resistance further enhances its clinical utility. Ratageri et al. (2019) demonstrated its value in pediatric TBM, while Solomons et al. (2015) highlighted the limitations of conventional CSF analysis. Our case reinforces the recommendation to use CBNAAT in suspected TBM cases, especially when clinical and radiological features are ambiguous.

Early diagnosis and initiation of ATT are critical in improving outcomes and preventing long-term neurological complications such as cognitive impairment, seizures, and motor deficits. In endemic regions, TBM must always remain a key differential diagnosis in pediatric CNS presentations.

4. Conclusion

Tuberculous meningitis can closely mimic demyelinating disorders such as ADEM in children, leading to diagnostic dilemmas. While neuroimaging provides important clues, definitive diagnosis requires microbiological confirmation. CSF CBNAAT is a rapid and reliable tool that aids early diagnosis and initiation of therapy. Clinicians must maintain a high index of suspicion for TBM in endemic regions to ensure timely treatment and prevent neurological sequelae. Especially in children who are malnourished even if they do not have apparent TB contact history. Threshold for doing complete CSF examination including TB CBNAAT should be low.

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