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Case Report

Left cerebellar abscess - a rare presentation of tubercular meningoencephalitis in childhood

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ABSTRACT

Tuberculous meningitis (TBM) is a life-threatening manifestation of extrapulmonary tuberculosis, commonly seen in developing countries. It predominantly affects the leptomeninges and accounts for 5–15% of extrapulmonary TB cases. While tuberculomas are frequent intracranial complications, cerebellar abscesses are exceedingly rare, especially in paediatric and immunocompetent populations. We report a rare case of a 10-year-old male who presented with fever, headache, vomiting, altered sensorium, and hearing loss. Neurological examination revealed signs of raised intracranial pressure, cerebellar involvement, and meningitis. Magnetic resonance imaging (MRI) imaging demonstrated a large left cerebellar abscess with surrounding edema and obstructive hydrocephalus. Cerebrospinal fluid (CSF) analysis supported the diagnosis of TBM, and GeneXpert testing confirmed *Mycobacterium tuberculosis*. The patient was managed with anti-tubercular therapy (ATT), corticosteroids, and supportive measures, including mannitol and acetazolamide. Neurosurgical intervention involved emergency external ventricular drainage and stereotactic aspiration of the abscess. This case highlights the importance of early recognition and combined medical-surgical management of rare TB-related CNS complications to prevent permanent neurological deficits. To our knowledge, only three paediatric cases of cerebellar tubercular abscesses have been reported in the literature to date, emphasizing the rarity and clinical significance of this presentation.

Keywords: Extrapulmonary, Leptomeninges, Tuberculomas, *Mycobacterium tuberculosis*

INTRODUCTION

Tuberculous meningitis (TBM) is the most common presentation of intracranial tuberculosis, and usually refers to infection of the leptomeninges. It accounts for 5–15% of extrapulmonary tuberculosis and is the most severe form of the disease. Most common clinical manifestations are fever, headache, vomiting and neck stiffness. Cranial nerve palsies of 3rd, 4th and 6th nerves may be seen. Seizures, focal neurological deficits, stupor and coma may be seen in late stages.[1]

TBM is a life-threatening form of CNS tuberculosis, often associated with hydrocephalus and vasculitis. While tuberculomas are common in TBM, cerebellar abscesses are rarely reported.

The pathogenesis likely involves hematogenous spread or direct extension from infected meninges. Early recognition and management are crucial to prevent long-term neurological deficits.[2] We came across 21 pieces of literature out of which there were only three pediatric cases of tubercular cerebellar abscess reported till now.[3]

We present a case of a 10-year-old male diagnosed with TBM who developed a left cerebellar abscess with non-communicating hydrocephalus, necessitating neurosurgical intervention.

CASE REPORT

A 10-year-old male child product of non-consanguineous marriage, 1st in order, received in PICU of tertiary care teaching hospital with chief complaints of fever since 15 days, vomiting and headache since 7 days, altered sensorium since 1 day and gradual decrease in hearing since 3 months with right ear pain. Fever was low grade, sudden in onset and progressive in nature followed by vomiting and headache. Patient was disoriented and had incomprehensible speech. No past history of TB, immunosuppression or any other prior CNS infections and no relevant family history was present.

On neurological examination, patient was dull lethargic, not oriented to time place and person, GCS was E2V3M4. Neck stiffness with positive Kernig sign present. Speech was incomprehensible with prominent dysarthria. All cranial nerves examination was found to be normal. Motor system examination was done, no change in bulk and tone was normal. Power of b/l upper limb (4/5) and b/l lower limb (4/5). Superficial reflexes were intact with planter extensor. Knee jerk was exaggerated with absent clonus; ankle jerk reflex was normal. Sensory system was normal including pain, fine touch, crude touch and proprioception. Positive cerebellar signs including ataxia, horizontal nystagmus, dysarthria, dysmetria (finger nose finger test positive), intention tremor, dysdiadochokinesia and positive heel knee test. Pupils were b/l pin point.

Fundus examination was suggestive of mild papilledema. ENT examination suggestive of b/l ear pain with gradual hearing loss but no ear discharges present. Routine investigations were done Hb (10. 5g/dl), platelet count (282 k/microlitre), TLC (5.93 k/microlitre). Liver function test (LFT) and renal function test (RFT) was normal, C-reactive protein (CRP) was negative. Cerebrospinal fluid (CSF) examination revealed total cell count 50 cells (lymphocytes 90% neutrophils 10%), low CSF sugar (31.80 mg/dl), high CSF protein (266 mg/dl).

Contrast enhanced MRI shown evidence of altered signal intensity lesion measuring approximately 40×27×46 mm seen in left cerebellum (Figure 1) with T2W/FLAIR hyperintense edema, appearing hyperintense on TIW, hypointense on T2W sequences with diffusion restriction on DWI images and showing peripheral enhancement on post contrast study. There was evidence of dilatation of bilateral lateral ventricles/right lateral ventricle (diameter measuring approximately 21 mm) and left lateral ventricle (Figure 2) (diameter measuring approximately 17.5 mm) with subtle basal cistern enhancement on post contrast study, suggestive of left cerebellar abscess with non-communicating hydrocephalous and meningitis as described.

GeneXpert MTB was positive for *Mycobacterium tuberculosis*. Mantoux test was positive. There was no evidence of pulmonary TB on chest X-ray.

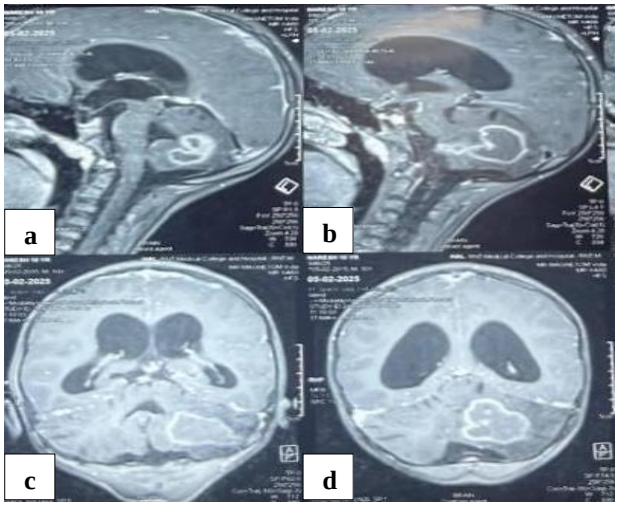

Figure 1 (a-d): CE MRI showing cerebellar abscess.

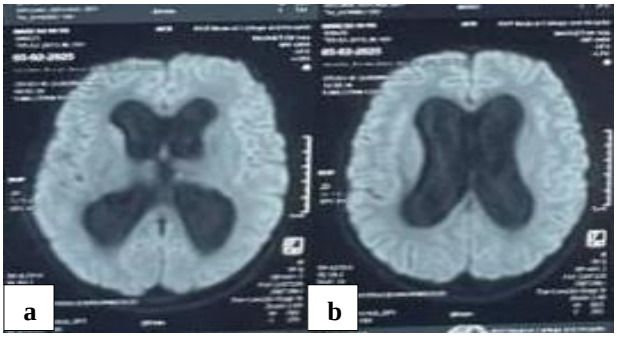

Figure 2 (a and b): CE MRI showing dilated ventricles.

The patient was started on IV fluids and started on empirical antibiotics like ceftriaxone and vancomycin along with metronidazole. Also patient was started with paediatric ATT, IV dexamethasone (0.15 mg/kg/dose 6 hourly) was given to reduce CNS inflammation and cerebral edema. IV mannitol and tab acetazolamide was used to manage intracranial pressure and IV antiepileptics were given prophylactically. Surgical intervention done external ventricular drain (EVD) which is an emergency procedure to relieve hydrocephalus and prevent brainstem compression, posterior fossa craniotomy and abscess drainage (stereotactic aspiration of the left cerebellar abscess) was also done. Patient was discharged on request and Follow up of patient could not be done.

DISCUSSION

TB is a chronic infectious disease commonly seen in developing countries. Even though it is a disease primarily affecting the lungs can affect the central nervous system (CNS) and can result in various neurological sequelae and even death. While supratentorial tubercular lesions are relatively common, the disease affecting the infratentorial compartment and cerebellum is very rare, that too in an immunocompetent paediatric patient.[3]

The organisms most frequently isolated from brain abscesses are *Staphylococcus aureus*, Gram-negative bacilli, anaerobes and *Streptococcus viridans*. Tubercular brain abscess is rather rare in places where tuberculosis is endemic, like India. Though tuberculosis in CNS occurs due to hematogenous spread of *Mycobacteria* from elsewhere; TBM may occur via lymphatic spread from cervical lymph nodes. Tubercle bacilli are immobilized in end-arteries, which leads to formation of sub meningeal tubercular foci, which may further lead to various presentations of tuberculosis.[4] Clinically patients with cerebellar tubercular abscess present with fever, headache, alteration of consciousness, seizure, cerebellar signs and or with hydrocephalus as was seen in our case.[5]

Das et al in 2021 reported a case of 5-year-old boy who presented with features of raised intracranial pressure and was diagnosed to have a cerebellar lesion causing hydrocephalus as similar to our case. An emergency surgical decompression was performed and the histopathological examination revealed that the lesion was suggestive of tubercular abscess.[3]

Aggarwal et al in 2015 reported an uncommon case 12-year-child with otogenic tubercular cerebellar abscess that was managed successfully with surgical excision and anti-tubercular treatment. A CT scan showed a well-defined cystic lesion in right cerebellar hemisphere and it was enhancing in a ring like manner after contrast administration without much mass effect or peri-lesional edema. There was associated mild ventriculomegaly.[6]

Ramesh et al reported a case of concomitant tuberculosis and pyogenic cerebellar abscess in patients with pulmonary tuberculosis, who responded to suboccipital craniotomy, antitubercular treatment and antibiotics.[7]

The differential diagnosis included infectious causes i.e. tubercular (TB) abscess/tuberculoma that can mimic pyogenic abscess, bacterial cerebellar abscess due to *Streptococcus*, *Staphylococcus*, or *Pseudomonas* (especially in immunocompromised patients), fungal abscess due to *Aspergillus* or *Cryptococcus*, particularly in immunocompromised individuals, neurocysticercosis (multiple ring-enhancing lesions that can cause obstructive hydrocephalus if near the fourth ventricle) and brucellosis (can rarely present as focal abscesses in the CNS).[8] Non-infectious causes include primary or secondary cerebellar tumors like medulloblastoma, ependymoma, or metastases causing mass effect and obstructive hydrocephalus, demyelinating disease (e.g., ADEM, MS) and paraneoplastic or autoimmune cerebellitis. Thirdly, vascular causes like cerebellar infarct with secondary liquefaction that resembles an abscess on imaging and cerebral venous sinus thrombosis (CVST).[9]

Treatment of a left cerebellar abscess in tubercular meningoencephalitis includes a combination of anti-tubercular drugs and antibiotics for secondary infections.[10] Emergency external ventricular drain (EVD) relieves hydrocephalus and prevents brainstem compression. Posterior fossa craniotomy with stereotactic aspiration enables abscess drainage as was done in this case.[11] Corticosteroids reduce inflammation.

CONCLUSION

Tuberculous meningitis with cerebellar abscess and hydrocephalus is a rare but serious CNS complication, even in immunocompetent children. Early diagnosis through clinical, imaging, and laboratory findings is crucial. This case highlights the importance of prompt anti-tubercular therapy and surgical intervention to prevent long-term neurological damage, emphasizing TB consideration in paediatric neurological cases, especially in endemic regions.

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