Case Report

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# Cerebral Tuberculoma of Right Frontal Lobe in an Unconscious Patient

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## ABSTRACT

Cerebral tuberculoma of the brain are uncommon presentation of tuberculosis (1%). A 17-year female presented to the emergency with a Glasgow Coma Score of (eye-1, verbal-1 and motor-3), 5/15 bilateral fixed pupils with laborious breathing. The radiological features were suggestive of aintracerebral abscess/ glioma. Intraoperatively the brain was very tense and a large, vascular, mass was present that was excised completely. Her histopathology revealed features of tuberculoma and she was started on anti-tubercular treatment. She is discharged home with regular follow-up for the last 10 months. Cerebral tuberculoma although uncommon should be thought of in developing countries as differential of cystic enhancing lesions of the brain.

**Keywords:** Abscess; central nervous system; glioma; tuberculoma; tuberculosis

## INTRODUCTION

Cerebral tuberculoma of the brain are uncommon presentation of tuberculosis (around 1%).[1] The bacteria spreads by hematogenous route and can involve the brain, meninges, pituitary gland, cranial nerves and the spinal cord. Tuberculomas can present with multiple cranial nerve palsies, headache, vomiting, seizure, diabetes insipidus, altered sensorium, visual loss or meningitis.[1]They can occasionally present like pyogenic abscess, metastasis, glioma or neurocysticercosis. Most cerebral tuberculomacases are small and managed with empirical anti-tubercular therapy (ATT) while larger ones amenable to surgery are excised followed by ATT. We report an interesting case of cerebral tuberculomaaffecting the right frontal lobe in a child which was managed with surgery and ATT.

## CASE REPORT

A 17-year female presented to the emergency with loss of consciousness for the past 2 hours. She had a Glasgow Coma Score (GCS) of (Eye-1, Verbal-1 and Motor-3) 5, bilateral fixed 5 mm pupils with laborious breathing. She had history of seizure disorder for the past 3 years with irregular treatment with anti-epileptics. An initial diagnosis of status epilepticus was thought and hence managed accordingly. In view of her dilated pupils she was taken for Computed Tomogram (CT) scan which showed a large hyperdense lesion in the right parietal lobe with severe perilesional edema, mass effect and midline shift and large calcifications in the right temporal and occipital lobes (Figure 1A). She was started on intracranial pressure reduction medications with 8 mg dexamethasone stat and 4 mg in 8 hourly doses along with intravenous mannitol 100 ml 8 hourly. Antiepileptics with leveteracetam 500 mg 12 hourly along with intravenous pantaprazole was also started. Once she was stablised an MRI was done. The latter showed a large homogenously contrast enhancing single lesion in the right parietal lobe with interdigitating edema, midline shift of more than 5mm and compression of the ipsilateral ventricle (Figure 1B). The MRI plain showed a T1 isointense and T2 hyperintense lesion which on contrast showed a large ring enhancing right frontal lesion with central T1 isointensity cavity with surrounding edema features were suggestive of an intracerebral abscess with differential of a glioma.

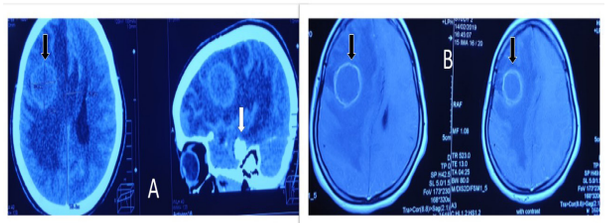


Figure 1. (A) CT scan plain showing a large hyperdense lesion in right frontal lobe with edema (black arrow) and old calcification (White arrow). (B) MRI with contrast showing a large ring enhancing right frontal lesion with central T1 isointensity cavity with surrounding edema (Black arrow).

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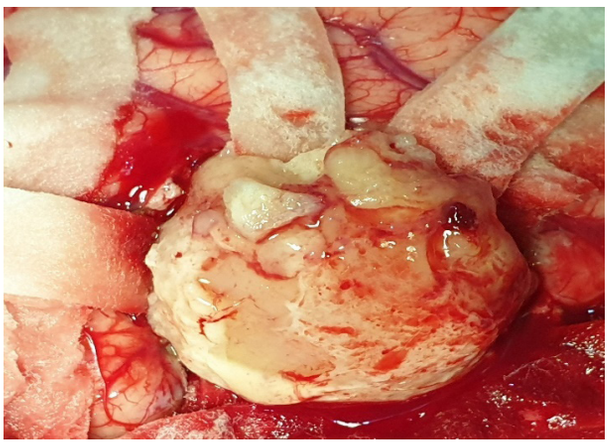

Figure 2. Intraoperative picture showing a large homogenous subcortical lesion with moderate vascularity with tense swollen brain.

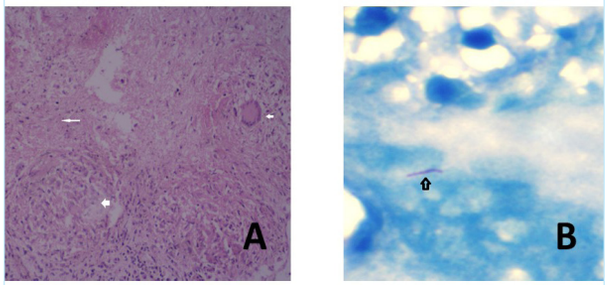


Figure 3. A. H&E stain- 40X Microscopic picture showing Langhan's giant cell (short thin arrow), areas of caseous necrosis (thin long arrow) and epitheloid cell granulomas (short thick arrow). B. H &E stain 100X showing Acid fast bacilli (arrow).

Further counselling was done and the patient taken for surgery. Intraoperatively the brain was very tense and a large, firm, vascular, ill-defined mass was present which was excised totally (Figure 2). In view of the large solid tissue for pathology any study with cerebrospinal fluid was not deemed necessary. Due to the swollen brain, decompressive craniectomy was done followed by wide expanding duroplasty by synthetic dural graft. Postoperatively, she was managed in the neurointensive care on a ventilator. The imprint cytology was suggestive of a granulomatous lesion and hence ATT along with ICP reduction medications, antiepileptics, steroids and analgesics was started. There was no past history of tuberculosis in the patient or any family member and the the chest X-Ray, abdomen ultrasound, mountox test did not reveal/suggest any features of tuberculosis. She was gradually weaned off the ventilator in 2 weeks, tracheostomy performed and was shifted to the ward with GCS of 10. Her histopathology revealed features of tuberculoma (Figure 3 A, B) with AFB seen in the tissue.

The biopsy culture for tuberculosis was negative. She was started on ATT and presently she is discharged home with regular follow-up for the last 10 months with GCS of 12. Her last CT scan at 10 months follow-up showed complete resolution of the intracranial tuberculoma (Figure 4).

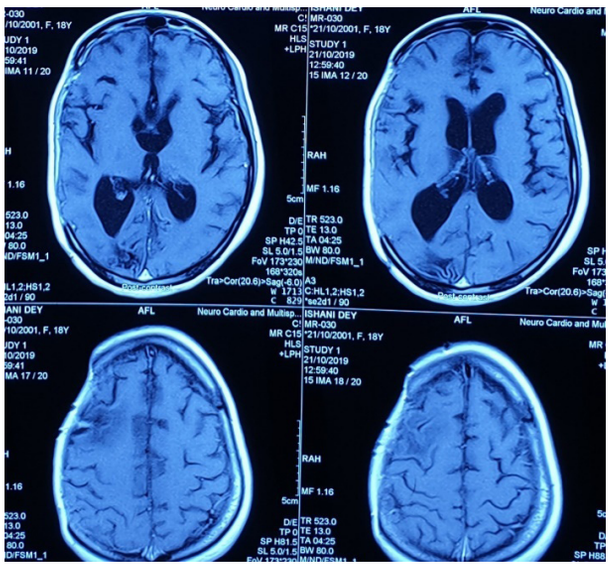

Figure 4. Repeat MRI with contrast showing the complete absence of the previous tuberculoma.

## DISCUSSION

Tuberculosis of the central nervous system (CNS) is a rare manifestation and mostly prevalent in developing countries where *Mycobacterium tuberculosis* pulmonary infection is common. It accounts for around 1 % of the CNS infections. The bacteria are an aerobic, nonspore forming acid fast bacilli taking weeks to grow in culture. Infection is airborne and spreads to the CNS via hematogenous route forming Rich foci which can then clinically manifest as meningitis, encephalitis, tuberculoma or tubercular abscess.[2,3] This detailed study in CNS tuberculosis was done by Rich and McCordock by infecting guinea pigs and rabbits by the bacilli in 1933.[4,5] In the subarachnoid space mainly around the sellar region it forms a dense exudate which can spread inferiorly to the spinal cord. Localized infection and growth of the tubercles can lead to tuberculoma formation which may be single or multiple. Histopathologically these are granulomatous lesion with central caseating necrosis containing giant epithelioid cells.

Clinically they present with features of meningism, headache, seizure, focal neurological deficits, loss of consciousness, hormonal abnormalities, vision loss, nausea and vomiting. History of tubercular exposure

is usually present in around 10% and positive Mantoux in only 30 to 50% of cases.[6] The symptoms may present according to the location of the cerebral tuberculoma and can include cranial nerve palsies, mono or quadriparesis, visual defects, severe headache or lobar syndromes. This case had history of seizure in the past but had not been investigated in a neurological centre. The presence of multiple calcifications in the right temporal and occipital lobes suggest possible previous healed tuberculomas. Her presentation with loss of consciousness, with past history of seizure was suggestive of status epilepticus. Any further delay in getting a CT head could have worsened the outcome. This suggest that a CT head after patient stabilization is a must for all cases with loss of consciousness. Urgent surgical excision of any lesion with removal of the bone can benefit similar cases. All the other test for a primary lesion outside the central nervous system were negative and hence this was labeled as acerebral tuberculoma. Due to the slow growth of thecerebral tuberculoma the symptoms may take many months to present.[6] Diagnosis is usually difficult and remains a challenge to the attending physicians. Cerebrospinal fluid (CSF) studies may show lymphocytosis and moderately elevated protein levels with low glucose level.[7] Culture and staining for the bacilli are mostly negative. Adenosine deaminase, antibody (ELISPOT), antigen detection and polymerase chain reaction are other ways to detect tubercular infection in the CSF.[8] Radiological investigations are mainly CT and the MRI of the brain. These, especially the latter will show the size, number, site, surrounding structures and edema or mass effect. Cerebral tuberculomaon MRI are T1 and T2 isointense with central hypointensity and with contrast show a peripheral ring enhancing lesion with central isointensity. Cerebral tuberculoma are shown as homogenous or ring enhancing lesion on contrast, with liquefied centre. MR spectroscopy further helps by showing decrease in NAA/Cr ratio, slight reduction of NAA/cho ratio and lipid lactate peak elevation.[9] MRI is also useful in follow-up to study the regression and change in the cerebral tuberculoma following chemotherapy.

Although helpful, the differential of pyogenic abscess, metastasis, sarcoidosis, other infective lesions (neurocysticercosis, cryptococcosis, toxoplasmosis) and glioma/lymphoma have to be considered. Culture of CSF and postoperative specimen culture is mostly negative. Treatment consists of 2-month initiation by isoniazid, rifampin, pyrazinamide, and ethambutol and followed by 6 to 12 months (range 6-36 months) of isoniazid and rifampicin as maintenance. Multidrug resistant TB is a major problem today and will need start of second line medications. ATT is supplemented with steroids, anti-epileptics and analgesics. Surgery plays an important role in the management of CNS tubercular infection by treatment of hydrocephalus, removal of the cerebral tuberculoma, ventriculo-peritoneal shunt, burr hole drainage of abscess, stereotactic biopsy of deep lesions and placement of lumbar or external ventricular drain.[10]

## CONCLUSIONS

Cerebral tuberculomas are rare disease mimicking several other space occupying lesions in brain. They should always be considered as a differential diagnosis in an individual residing in developing countries presenting with a space occupying lesion in brain. They can present acutely (as in this case) and is life-threatening. Surgery not only establishes the diagnosis but also saves life when combined with antitubercular therapy. Cerebral tuberculoma although uncommon should be thought of in developing countries as differential of cystic enhancing lesions of the brain.

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