

Case Report

An Uncommon Presentation of a Common Etiology : Tubercular Cerebral Abscess in Pulmonary TB

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A patient, recently diagnosed with Pulmonary Tuberculosis and on ATT for the last 1 month, was initially admitted in a primary care setup with severe headache and altered consciousness for 5 days and then referred to our institute for further management. Extensive investigations and imaging led to the conclusion that the patient had a massive Tubercular Brain Abscess (TBA) in the background of Pulmonary TB. [J Indian Med Assoc 2022; 120(12): 64-6]

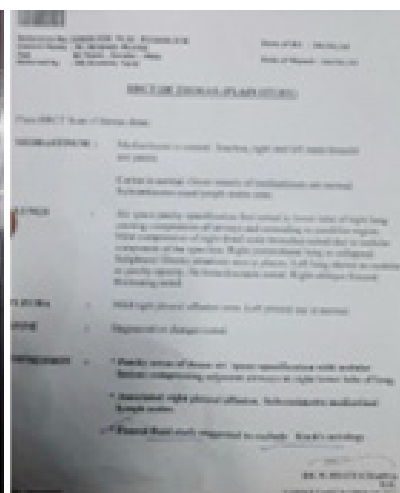
Key words : Fever, Headache, Tubercular, Cerebral abscess, NCCT Brain, MRI Brain.

Tuberculous Brain Abscess (TBA) is a rare presentation of Central Nervous System (CNS) tuberculosis. They often present with focal neurological signs, and are associated with a past history of tuberculosis. The following is a case report on one such patient encountered in our hospital setup.

CASE REPORT

A 60-year-old male from Kultali, South 24 Parganas, West Bengal, presented with the chief complaint of fever, headache and altered sensorium for last 5 days and was admitted to a primary care setup from where he was referred to our hospital for further management. On admission, he was unconscious with a GCS of 8. There was history of high-grade fever for last 2 days, recorded as 102°F on a single occasion, not associated with chill and rigor and subsided temporarily with medication. There was no history of trauma, vomiting or any episode of seizures on presentation.

One month back, patient had presented with a history of low grade temperature for 2-3 weeks, night sweats along with loss of weight (undocumented), and right sided chest pain. Initial investigations including Chest X-ray and HRCT thorax were done which was reported as- pleural effusion and right lower lobe consolidation with subcentimetric mediastinal



Figs 1 & 2 — (1) Dig CXR showing Right lower lobe consolidation and (2) HRCT thorax report

Editor's Comment :

- Tubercular Brain Abscess is a rare manifestation of CNS tuberculosis in adults, whose clinical presentation mimics that of pyogenic meningitis and poses a diagnostic challenge. It is rare even in a country like India where tuberculosis has a high prevalence and requires a high degree of clinical suspicion.
- Histological evidence may not always be available.
- ATT should be started as early as possible in all cases to limit the extent of complications before contemplating surgical intervention if TBA is considered as the diagnosis.

lymph nodes? Koch's etiology (Figs 1 & 2). Pleural fluid analysis was done and a diagnosis of 'microbiologically confirmed TB' was made based on which patient was started on ATD as per regimen (3 tabs 4 FDC) on

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He had been on regular medication till his condition started deteriorating 1 week back and he was unable to take oral feed properly.

There was a history of CVA with left sided hemiparesis 2 years back, with no residual paralysis. Patient was hypertensive, but on irregular medication for the same.

He had no known addictions, there was no history of high-risk behaviour. His family history was non-contributory as well.

Patient was unconscious at the time of admission and regained consciousness within a few hours after conservative management. Pulse rate was 100/min, respiratory rate 22/min, and BP recorded on admission was 120/80 mm Hg. SpO₂ was 96% in room air. Temperature was 101 deg F.

His higher mental function could not be assessed at the time of admission. Tone was reduced in all four limbs and plantar reflex was found to be extensor bilaterally. Signs of meningeal irritation were absent. Pupils were normal in size bilaterally and reacting sluggishly to light. When he regained consciousness, he was disoriented and restless.

An urgent NCCT Brain was done at admission which showed the following (Figs 3 & 4) :-

Immediate measures were taken to reduce the patient's intracerebral edema, ATT was continued and the patient slowly regained orientation over the next 24 hours. A Neuromedicine and Neurosurgical consult was sought and patient was advised MRI Brain (contrast) with a MR Spectroscopy.

Initial laboratory investigations showed low Haemoglobin (7 gm/dl), Total Leucocyte Count of 3800, Differential Leucocyte count – lymphocyte count of 400, neutrophil : 3200, Urea, Creatinine, Sodium, Potassium, Liver Function Tests within normal range. His ICTC report was non-reactive.

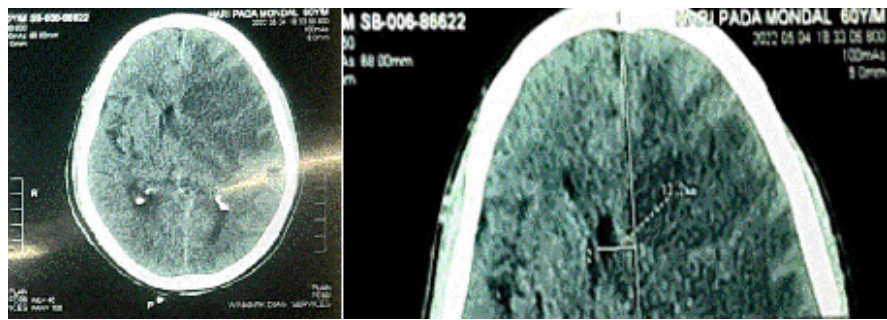
MRI Brain with contrast and MR Spectroscopy (Figs 4 & 5) was done, which revealed the following :-

“Large peripherally rim-enhancing irregular shaped cystic lesion about [(AP) 40.4 mm * (TR) 58.4 mm * (CC) 36.8 mm] showing incomplete internal septae, significant perilesional edema and mass effect at left fronto-parietal region showing MRS features-likely Tubercular Cerebral Abscess.

Microangiopathic chronic ischaemic changes in bilateral periventricular deep white matter, centrum semiovale, bilateral cerebral subcortical white matters.

Cerebrospinal Fluid (CSF) intensity chronic lacunar infarcts at bilateral basal ganglia and right paraventricular deep white matter regions.”

Patient improved gradually



Figs 3 & 4 — NCCT Brain – a large area of ? cavitation in the left fronto-parietal area with surrounding perilesional edema and midline shift

over the next 48 hours with medical management. Spontaneous eye opening was present, he was responsive to verbal commands and oriented to time, place and person. Given the infrastructural constraints, we were not able to microbiologically prove the presence of AFB or M tuberculosis from a cerebral pus aspirate. However, taking into consideration the patient's background of pulmonary TB and the MRI Brain with MRS analysis, it was considered prudent to maintain him on ATT and steroids after due consultation with the Neurology department. The trajectory of the patient's hospital stay was satisfactory.

DISCUSSION

Tuberculous Brain abscess (TBA) is a rarely reported form of CNS tuberculosis, occurring in 4-8% of non HIV patients. Despite the low frequency (only around 1% of extra-pulmonary tuberculosis cases)², it is given special importance because of the very high mortality associated with it. It is a focal collection of pus containing abundant Acid-fast Bacilli (AFB) surrounded by a dense capsule comprising vascular granulation tissue. The abscess is often single, sometimes multilocular and accompanied by greater perilesional oedema³. TBA is an uncommon clinical entity even in countries where TB is endemic⁴.

TBA always poses a diagnostic dilemma as they are difficult to differentiate from pyogenic brain abscesses,

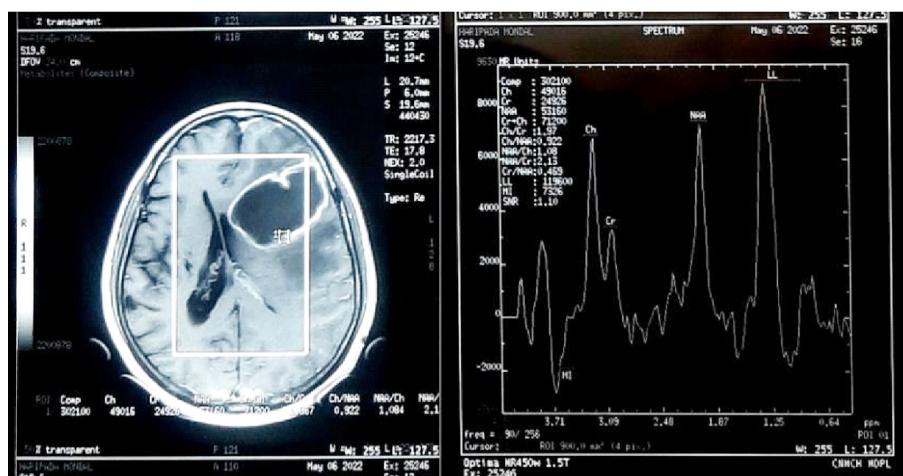


Fig 4 (B) — Choline Creatinine peak >2 , prominent lipid lactate peak

Tuberculous Meningitis and Tuberculoma on the basis of clinical, laboratory and roentgenographic information. They are more frequently encountered in patients who are immunocompromised. Patients with tuberculous abscess present non-specifically, with focal neurological deficits, seizures or signs and symptoms of raised intracranial pressure. These brain abscesses, like tuberculomas, are probably secondary to haematogenous dissemination from a primitive site, most often pulmonary⁵.

Mycobacterium Tuberculosis can be isolated from the pus (ZN stain, AFB demonstration or PCR) which is sometimes unable to be isolated from Tuberculomas.

Approach to management is medico-surgical. ATT along with corticosteroid therapy have been shown to improve mortality in multiple studies. Studies show that it reduces mortality and neurological sequelae in patients with a medium severity picture (confusion, focal signs) and also in comatose patients⁶.

Surgical drainage is often curative and the mainstay of treatment for large abscesses, though associated with its own risks and sequelae. Progression under anti-tuberculosis treatment is not discriminatory diagnostic evidence. Several observations have reported the development of Tuberculous Abscesses under well-conducted specific treatment as was also seen in our patient^{7,8}.

CONCLUSION

TBA still remains a topic of concern and intrigue due to poor prognosis despite medical and surgical intervention. Despite best efforts and management under specialist care, outcomes in majority of patients have not thus far been as promising as hoped and expected. Given the rarity of the condition, even among the CNS TB manifestations, it remains an area of research and development. One of the reasons for the morbidity and mortality could be due to the late presentation seen in majority of patients and concomitant presence of an immunocompromised state.

Patient would need integrated approach with neurosurgical intervention and Neuromedicine consultation. The following was done from our end after management with ATT and steroids. Patient was ultimately lost to follow-up.

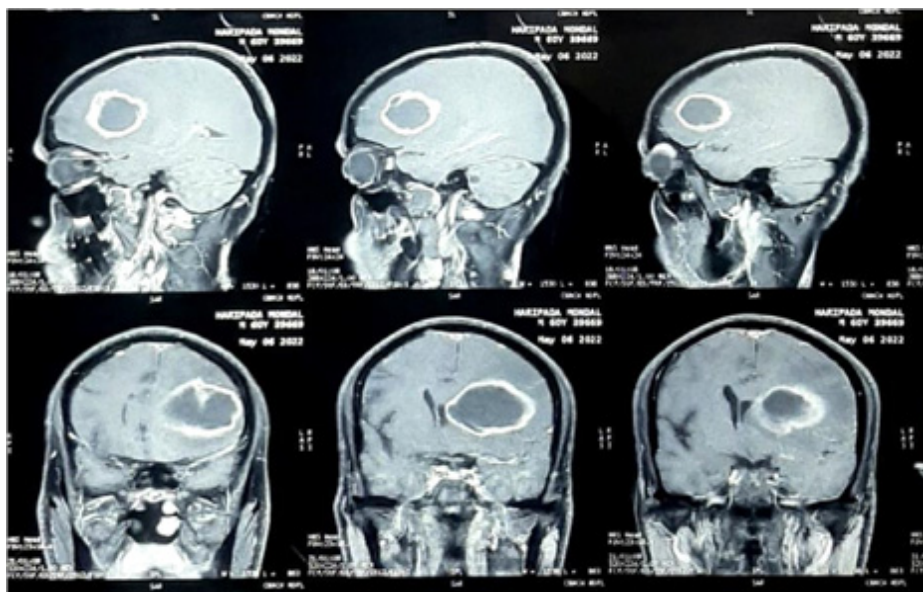


Fig 4 & 5 — Large peripherally rim-enhancing irregular shaped cystic lesion about [(AP) 40.4 mm * (TR) 58.4 mm * (CC) 36.8 mm] showing incomplete internal septae, significant perilesional edema and mass effect at left fronto-parietal region

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