

# Multiple Parameter MRI Findings of Cerebral Tubercular Abscess: A Case Report

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## Abstract

Tubercular cerebral abscess (TBA) is an extremely rare entity. Although tuberculosis is a common infectious disease occurring worldwide, with meningitis and tuberculoma being the commonest CNS manifestations, TBA, has been infrequently described in the literature and its MRI features have also been rarely reported. Its clinical and radiological features may simulate other infectious and noninfectious neurological diseases and cystic tumors. Hence, it is essential for every radiologist to be familiar with the imaging presentations of various forms of CNS tuberculosis for timely diagnosis and treatment, thereby reducing the morbidity and mortality of this disease. Here, we report to you a case of tubercular cerebral abscess of a 60-year-old male who presented with headache and seizures for one month with no history of pulmonary tuberculosis and negative HIV serology. We further discuss about the MRI, DWI and MR spectroscopy findings of this disease and compare with other cases reported in the literature.

**Keywords:** Tubercular brain abscess; Magnetic resonance imaging; Diffusion weighted imaging; Magnetic resonance spectroscopy

**Abbreviations:** TBA: Tubercular Abscess; MRI: Magnetic Resonance Imaging; CNS: Central Nervous System; FLAIR: Fluid-Attenuated Inversion Recovery; BP: Blood Pressure; RR: Respiratory Rate; ELISA: Enzyme-linked Immunosorbent Assay; PCR: Polymerase Chain Reaction; HIV: Human Immunodeficiency Virus; MRS: Magnetic Resonance Spectroscopy; RBC: Red Blood Cells; HCT: Hematocrit; DWI: Diffusion Weighted Imaging; TB: Tuberculosis; NAA: N-acetyl aspartate; Cho: Choline; MDR-TB: Multidrug Resistance Tuberculosis; ADC: Apparent Diffusion Coefficient; CSF: Cerebrospinal Fluid

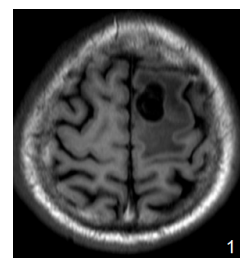
## Case Report

### Clinical history

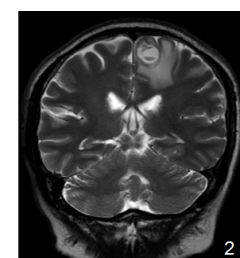
A 60-year-old man, with no significant history of past medical illness presented to our hospital with complaints of recurrent headache and seizures for one month. During the seizure episode, he experienced abnormal movement of limbs lasting for one minute, with headache and confusion in the post-ictal phase. He had history of smoking of 30 pack years. There is no history of alcohol intake. No history of pulmonary tuberculosis. No history of weight loss. On examination, he was non-febrile with normal blood pressure and respiratory rate. There was no papilledema and nuchal rigidity. Babinski sign, Kernig's sign and Brudzinski's sign were all negative. There was no gross neurological deficit. His RBC ( $4.17 \times 10^9/L$ ) and HCT (0.394 L/L) level were on lower side, whereas other routine full blood count with differentials, liver function and renal function tests were all normal. HIV serology was negative. Preoperative cranial MRI found solitary ring-enhancing lesion with marked edema and was diagnosed as metastasis. After admission, surgical resection of left frontal lobe was performed. However, biopsy and histopathological report of the specimen and culture of the pus confirmed presence of *Mycobacterium tuberculosis*. And post-operative tuberculosis antibody test and T-SPOT.TB were positive. The patient had an uneventful hospital stay and antitubercular therapy which includes rifampicin, isoniazid, and ethambutol were started immediately after surgery.

### Radiological findings

The MRI (Magnetic Resonance Imaging) scan of the head on T1WI and T2WI revealed solitary cystic encapsulated mass with surrounding prominent edema in the left frontal lobe approximately  $2.19 \text{ cm} \times 2.19 \text{ cm} \times 2.08 \text{ cm}$  in size (Figures 1 and 2), On FLAIR (Figure 3) it



**Figure 1:** T1-weighted axial MRI showing an oval, well encapsulated hypointense mass in the left frontal lobe.



**Figure 2:** T2-weighted axial MRI showing the hyperintense mass lesion with hypointense rim and surrounding oedema in the left frontal lobe.

showed isointensity. There was no restricted diffusion in DWI (Figures 4 and 5). After gadolinium contrast, a smooth and homogeneous ring enhancement was seen with no enhancement in other brain parenchyma (Figure 6). The ventricular system was symmetrical and no midline shift. Proton single voxel MRS showed marked lipid and lactate peaks at the center of the lesion. The Cho/NAA ratio was 0.167, NAA/Cr was

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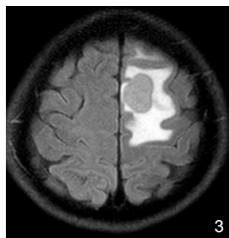
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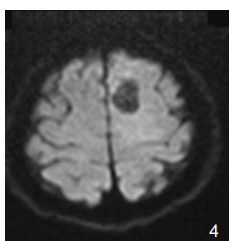
3.48 and Cho/Cr ratio 0.54 (Figure 7). There was no significant decrease of NAA and no increase of Cho compared to normal brain parenchyma.

### Histopathological findings

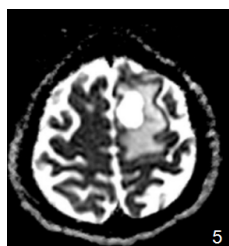
The specimen resected from the part of the frontal lobe showed a grayish white mass of tissue, approximately 2.5 cm × 2.5 cm × 0.8 cm, which contained ash-white thick pus. The hemotoxylin and eosin stain (Figures 8 and 9) exhibited extensive chronic inflammatory lymphocytes infiltration, degeneration and necrosis of cystic contents, epithelioid cells and multinucleated giant cells whereas in acid fast stain, the Ziehl-Neelsen stain, (Figure 10) showed presence of acid fast bacilli (AFB), which were stained red and clearly stood out against the blue tissue background. Hence, histopathologic evaluation of the biopsy specimen, Ziehl-Neelsen stain for acid fast bacilli, laboratory investigation (T-SPOT.TB) and tuberculosis antibody test confirmed the presence of *Mycobacterium tuberculosis*.



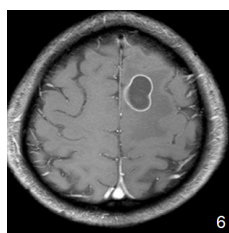
**Figure 3:** T2WI FLAIR showing the isointense mass with extensive perilesional oedema in the left frontal lobe.



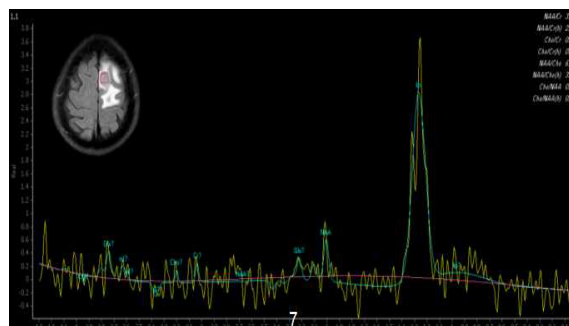
**Figure 4:** DWI images show no restricted diffusion within the mass in DWI.



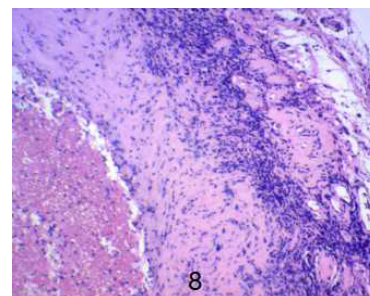
**Figure 5:** ADC images show no restricted diffusion within the mass in DWI.



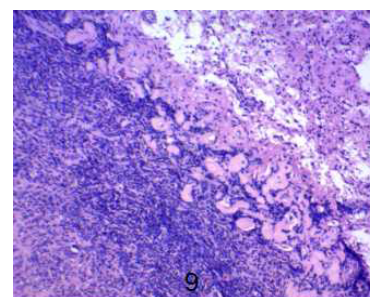
**Figure 6:** Post contrast T1-weighted axial MRI showing the well-circumscribed thin ring-enhancing mass in the left frontal lobe.



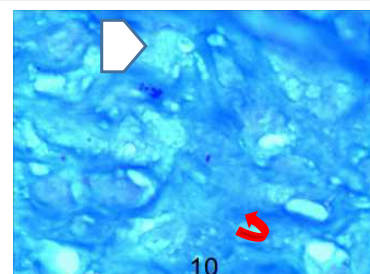
**Figure 7:** Proton single voxel MRS showed marked lipid and lactate peaks at 1.3 ppm and 1.35 ppm respectively at central part of the mass. No significant decrease of NAA and no increase of choline seen.



**Figure 8:** Histopathology of abscess showing necrotic tissue (white arrow) with fibrocystic wall (white arrow head), (H and E, × 100).



**Figure 9:** Histopathology of abscess showing the wall showing large amount of hyaline degeneration (star) and plenty inflammatory cell (curved arrow) including epithelioid cell and multinucleated giant cells. (H and E, × 200).



**Figure 10:** *Mycobacterium tuberculosis* visualization using Ziehl-Neelsen stain (red curved arrow pointing to acid fast bacilli) in blue background. (ZN stain, × 1000).

### Background

Tuberculosis is among the top 10 causes of death worldwide. According to the recent WHO data, in 2015, 10.4 million people fell ill

with TB and 1.8 million died from the disease (including 0.4 million people with HIV). Six countries were included in the list, with India leading and followed by Indonesia, China, Nigeria, Pakistan and South Africa. Almost one-third of the people with HIV were infected with TB bacteria in 2015 globally [1,2]. CNS TB present with high rate of morbidity and mortality and in nearly 1% of individuals with TB, CNS is involved which includes meningitis, tuberculomas, abscess or other manifestations. TB abscess occur more frequently (20%) in HIV infected persons compared to 4% to 7.5% of HIV negative patients with CNS TB [2,3]. Infection of the CNS with *Mycobacterium tuberculosis* is almost always secondary to a primary focus, the most common being pulmonary. The common manifestations of CNS TB are meningitis and tuberculoma. Tubercular abscess is a rare form of CNS TB occurring in only 4.8% of patients with CNS TB. These lesions occur mostly supratentorially although there are few cases reported to be found in the cerebellum [3-5]. They are associated mainly with immunocompromised individuals but occur in immunocompetent individuals as well. Here, we describe a case of central nervous system tuberculosis manifesting as brain abscess in a 60-year-old male without HIV infection and pulmonary TB.

## Discussion

According to the recent WHO data, in 2015, 1.8 million died from TB. It is a leading killer of HIV-positive people (35%). An estimated 49 million lives were saved through TB diagnosis and treatment between 2000 and 2015 [1]. Despite tuberculosis (TB) being a very common infectious disease, with largest number of new TB cases (61%) occurring in Asia in 2015, tubercular cerebral abscess remains a rare entity and occasionally described in the literature [1,2].

The criteria proposed by Whitener [6] based on a review of 57 cases in world literature, has been used for identifying the tuberculous brain abscess cases which includes: 1) macroscopic evidence of abscess with central pus, 2) abscess wall composed predominantly of vascular granulation tissue containing acute and chronic inflammatory cells and 3) evidence of presence of tubercle bacilli demonstrated on culture or by positive acid-fast stain. Our patient fulfilled all the three enlisted criteria.

The source of tuberculous brain abscess is hematogenous dissemination from a primary source, frequently from the lungs. It can also occur via spread of tuberculous foci from meninges to the brain parenchyma or from tuberculous granuloma. The most frequent clinical manifestations include headache, seizures, altered consciousness, fever, paresis, focal neurological deficits [7]. It is commonly seen in immunocompromised patients, mainly AIDS and patients with history of pulmonary TB. Our patient was neither HIV serology positive nor had history of pulmonary TB or other forms of TB.

Magnetic resonance imaging with gadolinium enhancement is the preferred method of initial investigation as it is the most sensitive test for detecting the leptomeningeal disease and parenchymal abnormalities. It is a multiplanar modality, displays the extent and location of the lesion and provides high contrast resolution. The typical feature of TBA in T1WI is a solitary or multiple, well-capsulated hypo intense lesion whereas in T2WI and FLAIR it appears to be a hyperintense lesion with significant peripheral edema. Our case displays these imaging features. According to the published literature, TBA usually shows restricted diffusion on DWI, but only one case was reported without restricted diffusion [8]. The case presented here has no restricted diffusion. The exact underlying mechanism for this in our case is unknown. However, the possible reason may be that, in our case there is no hyper viscosity

in the abscess. In our case, TBA shows significant lipids and lactate peaks on MRS which is in accordance with the reviewed literature of Gupta and Dusak [9-13]. Hence, Magnetic Resonance Spectroscopy (MRS) can provide useful information to diagnose TBA [10,11]. Although multiple parameter MRI is a useful imaging tool, TBA can be confused with various other lesions and neuroimaging study is usually nonspecific for diagnosis.

The differential diagnosis of tubercular abscess is wide and includes pyogenic and cryptococcal abscess, metastatic disease and cystic tumors.

## Pyogenic abscess

The imaging findings of tuberculous abscess on T1WI, T2WI and enhanced images are similar to pyogenic abscess. But on MR spectroscopy pyogenic abscess always show presence of amino acids whereas in case of TBA, only lipids and lactate peaks are observed with no evidence of amino acids [10,13]. Pathologically, the pyogenic brain abscess contains large number of neutrophils and proteins. The breakdown of neutrophils causes release of large number of hydrolytic enzymes that hydrolyzes proteins into amino acids [10,13]. Whereas, TBA mostly contains mycobacteria and lymphocytes with less number of neutrophils and necrotic brain tissue. The reason behind high peak of lipid in the spectrum is the lipid rich structure of mycobacteria [10,13]. Similarly, bacterial fermentation leads to occurrence of lactate and necrosis of cerebral tissue results in lactate and lipids [8]. Hence, lack of proteolytic enzymes or amino acids in tuberculous exudates compared to pyogenic inflammation is regarded significant for diagnosis of TBA by MRS. Differentiation of TBA from pyogenic abscess is important for the management.

## Fungal abscess

Fungal infections of the CNS are rare in the general population, mainly occurring in immunocompromised patients. They are most commonly located in the middle fossa, basal ganglia and mesencephalon. Neuroimaging in patients with fungal abscess reveals multiloculated thick wall with a necrotic core. On non-contrast MRI, the ring of the fungal abscess is irregular and of low signal on T2-weighted MRI images. The abscess cavity shows intracavitary projections which are considered characteristic of fungal abscesses. These intracavitary projections are confirmed to be fungal hyphae on histopathologic examination. These features vary from TBA. One study of MRS showed increased lipid peaks [13,14].

## Metastasis/tumors

Tubercular abscess can simulate metastasis in the CNS. Metastatic lesions are typically subcortical, occurring in or near the gray-white matter junction. MRI is the initial investigation of choice. But conventional MRI cannot provide an accurate diagnosis. Hence, DWI is a useful sequence to differentiate abscess from primary brain tumors or metastasis. Gupta et al. in his literature describes that in DWI, the tubercular abscess cavity is hyperintense while the tumor cavity appears hypo intense [10,13]. However, in our case there was no restricted diffusion in DWI, creating a diagnostic dilemma of metastatic tumor. Another case reported by Karki et al. explains corticosteroid regime and an organized abscess cavity may be the possible cause of no restricted diffusion in DWI in his patient [8]. In such circumstances, MRS can provide differential diagnostic information. TBA shows marked lipid and lactate peaks with no significant decrease of NAA and slightly increase of Cho whereas metastatic lesion shows markedly increased choline peak and no NAA peak in MRS. Perfusion weighted MRI can



demonstrate vascularity of the lesion's wall, differentiating it from the hyper vascular tumor capsule and abscess capsule [8,13]. Primary brain tumors, such as glioblastoma multiforme or anaplastic astrocytoma can have ring-enhancement with surrounded vasogenic edema similar to TBA, but they are large in size, located deep in the white matter and frequently cross the midline, and the enhanced wall is always irregular showing restricted diffusion on DWI. In addition, the MRS findings is different with markedly increased choline peak and decrease NAA peak and the ratio of Cho/NAA is often above 2.5, whereas in TBA it is not.

## Neurocysticercosis

Neurocysticercosis is an infectious parasitic disease. The imaging characteristics of neurocysticercosis depend on the stage of the infection. MRI is the imaging modality of choice. During the colloidal stage, the wall of the cyst appears thick and hypo intense with marked perilesional edema on T2-weighted images. After contrasting medium administration, it shows a ring like peripheral enhancement. Usually, the lesions are <20 mm in diameter and multiple in nature compared to TBA which is often solitary [15]. MRS shows decreased NAA and creatine levels and elevated lactate and metabolites such as alanine and succinate [16,17]. In addition, the various laboratory examinations available such as CSF analysis, stool test and ELISA are the mode to confirm neurocysticercosis.

## Conclusion

TBA is a rare entity which is often misdiagnosed. The conventional MRI cannot provide sufficient information for the diagnosis. The advanced imaging modality such as DWI and MRS can provide useful information for diagnosis.

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