Magnetic resonance imaging diagnostic features of giant intracranial tuberculoma

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ABSTRACT

Introduction: Central nervous system (CNS) tuberculosis (TB) is among the least common yet most devastating forms of human mycobacterium TB infection. Giant tuberculomas are often confused with brain tumors on cranial magnetic resonance imaging (MRI), emphasizing the need for more meticulous and elaborate imaging analysis. Case Report: A 38-year-old woman presented with progressive loss of vision associated with headaches. Magnetic resonance imaging of the brain showed a giant left frontal lobe mass lesion with imaging features that could prospectively suggest the diagnosis of tuberculoma. Subsequent operative and histopathological findings confirmed the diagnosis of intracranial tuberculoma. Conclusion: Parenchymal CNS TB, with or without extracerebral manifestations, may present as a space-occupying lesion with complex neuroimaging morphology suggesting benign or malignant neoplasms. Tuberculomas may be differentiated by their unique MRI pattern, including conventional and advanced MRI sequences like MR spectroscopy (MRS) and MR perfusion (MRP) parameters.

INTRODUCTION

Tuberculomas are rare and account for 1–2% of all intracranial lesions [1, 2]. Intracranial tuberculomas are potentially curable and its early differentiation from other space-occupying lesions of the brain is a clinical priority for prompt antituberculous therapy [3]. Conventional MRI sequences cannot confidently diagnose tuberculomas in most of the cases as there is substantial overlap in imaging features [4]. It is deemed that additional advanced imaging modalities, such as MRS, perfusion MRI, diffusion weighted imaging (DWI), and susceptibility weighted imaging (SWI) can improve diagnostic accuracy, thereby obviating the need for tissue biopsy [5, 6].

This report presents a case of giant intracranial tuberculoma that underwent surgical excision. Intraoperative findings and subsequent histopathological examination confirmed the diagnosis of tuberculoma. The significance of peculiar radiological findings with specific emphasis on importance of preoperative MRI with advanced MR sequences is highlighted.
CASE REPORT

A 36-year-old female patient came to the emergency room with two weeks history of worsening headaches and blurring of vision. She was noted to be completely blind in both eyes at presentation. Neurologically, she was conscious. The pupils were dilated measuring 5 mm in size, showing very sluggish response to light on both sides and the fundoscopic examination revealed bilateral papilledema. She had right-sided upper motor neuron type facial nerve palsy, with no other motor or sensory deficits. Babinski’s reflexes were extensor bilaterally.

Nonenhanced computed tomography (CT) scan (Figure 1) showed left frontal and basal ganglia lesion appearing predominantly isodense to gray matter with smaller areas of low attenuation within. There was significant perilesional edema, producing mass effect and a midline shift of 10 mm to the right. Magnetic resonance imaging of the head showed multiple left frontal, and basal ganglia mass lesions with iso- to hypointense T2/FLAIR signal (Figure 2A), and isointense central and hyperintense peripheral T1 signal, and postcontrast sequences showed thick nodular peripheral enhancement (Figure 2B). There was no diffusion restriction within the central necrosis of the lesion (excluding abscess) and there was small area of blooming in SWI at the medial aspect of the largest anteromedial lesion which could be due to minor hemorrhage/paramagnetic material (Figure 3A).

Dynamic susceptibility contrast (DSC) MR perfusion showed mildly increased relative cerebral blood volume (rCBV) mainly peripherally (Figure 3B). Magnetic resonance spectroscopy showed elevated choline peak with increased choline to N-acetylaspartate (NAA) ratio, and significant increase in lipid and inverted lactate peaks (Figure 4). Dynamic susceptibility contrast MR perfusion curve showed that there was only mild prominence of rCBV within the region of interest (ROI) drawn including the solid portions of the largest lesion. The apex of the rCBV troughs from the ROIs in the contralateral normal brain and ipsilateral M3 middle cerebral artery branch were much deeper than that from the mass lesion (Figure 5). Mass effect was noted similar to that in the CT scan and the MRI also clearly demonstrated lesion indentation and mass effect on the left side of optic chiasm/left optic tract (Figure 6A) with hyperintense abnormal T2 and hypointense T1 signal in the optic chiasm crossing over to the right. The left optic tract (Figure 6B) was thickened, and hyperintense with no enhancement noted.

The patient underwent left pterional craniotomy and complete surgical resection of the left frontal lesion. Intraoperatively, the mass lesion was pale, minimally vascular, and had variable consistency with some areas of caseation. There was a well demarcated interface between the mass and brain parenchyma. Intraoperative frozen section showed findings suggestive of a granulomatous lesion. Routine immediate postoperative MRI study showed no residual mass lesion. The final histopathology confirmed chronic granulomatous inflammation with caseation necrosis and histomorphological features suggestive of tuberculoma. Although acid fast bacillus stain was negative, the tissue culture was positive for Mycobacterium tuberculosis complex. Polymerase chain reaction (PCR) for tuberculosis was also positive. The

![Figure 1: Axial nonenhanced CT scan shows a left frontal white matter and basal ganglia lesion appearing mainly isodense to gray matter with smaller areas of low attenuation noted within.](image1)

![Figure 2: (A) Axial T2W MR shows multiple irregular left frontal lobe and basal ganglia mass lesions demonstrating iso- to hypointense T2 signal (arrow head). (B) Axial postcontrast MRI shows thick nodular peripheral enhancement (arrows), with central nonenhancement (in the area of T2 iso- to hypointense signal) due to solid caseating necrotic center.](image2)
patient was started on antituberculous medications with four-drug regimen.

On her follow-up visit in outpatient clinic, she was conscious and alert with no neurological deficits, and able to ambulate without support. She received antituberculous treatment for one year with serial imaging which showed no residual/recurrence of diseases at one year follow-up (Figure 7).

DISCUSSION

Intracranial tuberculoma manifesting as a clinically evident mass lesion of the brain is distinctly uncommon in the West [1]. However, in parts of world where tuberculosis is still endemic, such as in Indian subcontinent, parts of South East Asia and Africa, it still accounts for 20–30% of all intracranial lesions [2]. The signs and symptoms of tuberculomas resemble those of other intracranial space-occupying lesions [3]. Clinical features that may be helpful in distinguishing tuberculomas from other brain tumors are constitutional symptoms, a history of active or known TB elsewhere in the body and a past close contact with a patient who is an open case of TB [3]. In our case, the patient was not having history of past exposure and there were none of features indicating systemic tuberculosis. Her clinical features were suggestive of a

![Figure 3](image1.png)

**Figure 3**: (A) Axial SWI shows blooming hypointensity which could be due to minor hemorrhage or other paramagnetic material (arrow head). (B) Dynamic susceptibility contrast (DSC) MR perfusion relative cerebral blood volume (rCBV) image shows mild increased relative rCBV in the solid enhancing areas, mainly peripherally (small arrow heads).

![Figure 4](image2.png)

**Figure 4**: MR spectroscopy shows elevated choline peak with increased choline to NAA ratio, and significant increase in lipid with inverted high lactate.

![Figure 5](image3.png)

**Figure 5**: (A) DSC MR perfusion curve shows that there is only mild prominence of rCBV with the region of interest (ROI) drawn including the solid portions of the largest lesion (red). (B) Note that the apex of the rCBV troughs from the ROIs in contralateral normal brain (green) and ipsilateral M3 middle cerebral artery branch (yellow) are much deeper than that from the mass lesion.

![Figure 6](image4.png)

**Figure 6**: (A) Coronal T2W MRI of the head shows indentation and mass effect on the optic chiasm from the mass lesions with hyperintense abnormal T2 signal extending to the right side of the chiasm (thin arrow). (B) Axial T2W MRI shows hyperintense signal of the left optic tract (thick arrow) which appear thickened.

![Figure 7](image5.png)

**Figure 7**: Follow-up after one year, postgadolinium enhanced images show postsurgical changes with complete resolution of tuberculoma mass lesion.
slowly progressive mass lesion, causing worsening of ophthalmological and neurological deficits.

The MRI features of a tuberculoma depend on whether it is noncaseating, caseating with a solid center, or caseating with a liquefied center [5]. On T1 weighted images, tuberculomas show isointense or hypointense signal intensity whereas on T2 weighted images, tuberculomas display iso- to hypointense, or hyperintense signal. Tuberculomas with solid caseating center show central iso- to hypointense signal intensity on T2W images owing to granulation tissue in the core of the lesions [5]. Tuberculomas with central liquefactive caseation and noncaseating tuberculomas exhibit hyperintense signal on T2W images. On postcontrast study 64 (88.8%) patients with caseation (with solid center or liquid center) exhibit ring enhancement [2, 5].

Tuberculomas may be differentiated from metastases and gliomas by their metabolite pattern comprising of elevated Cho/Cr ratio, reduced NAA/Cr and NAA/Cho ratios with presence of prominent lipid peak. Markedly elevated Cho/Cr ratio, reduced NAA/Cr and NAA/Cho ratios with/without lipid or lactate peak favors a diagnosis of neoplastic brain lesion [7, 8]. Perfusion MRI has been used to differentiate ring enhancing tuberculomas from metastasis. Ring enhancing tuberculoma wall shows mildly elevated mean rCBV values (2–3.3). The entire nodular enhancing tuberculoma can also show similar mild increased rCBV. Metastasis wall shows much more elevated mean rCBV (5.43), with a cut-off value of ≥3.745 suggested to differentiate ring enhancing metastases from tuberculomas [7].

One study showed promising role of SWI in the discrimination of tuberculomas from metastatic brain lesions and gliomas with the presence of a complete and regular peripheral hypointense ring favoring the diagnosis of tuberculomas [6]. Our case did not fulfill this criterion, although there was central blooming which may be caused by minor hemorrhage or some other paramagnetic material. After reviewing our case, even though the peripheral irregular thick nodular enhancement of the lesion could represent high grade glioma with central necrosis, the T2 hypointense signal and absence of diffusion restriction in the nonenhancing solid caseating core would exclude it in favor of granulomatous infection. High grade gliomas or highly cellular metastasis can show T2 hypointensity due to the densely packed tumor cells, those areas would show solid enhancement in postcontrast T1WI and can show diffusion restriction. These high-grade tumors can show nonenhancing necrotic areas in the core, but those areas would show T2 hyperintensity of the necrotic material and no diffusion restriction. The core of an abscess will also be filled with nonenhancing pus, but this pyogenic material in the abscess will restrict diffusion.

Giant intracranial tuberculomas present a special clinical diagnostic challenge [3]. The nonspecific clinical presentation and lack of any past exposure with no obvious evidence of systemic tuberculosis put these cases in blurred zone where a preoperative diagnosis is difficult to reach with confidence [3, 5]. Therefore, these additional advanced imaging features can provide differentiating information that can guide clinicians to diagnose such lesions preferably in favor of granulomatous mass rather than a neoplastic lesion [6, 8]. Although surgical excision was an obvious indication in our case due to significant ophthalmological and neurological deficits, neuroimaging review gives us an insight for diagnostic features of tuberculoma to have a high index of suspicion for future clinical practice. In small-sized tuberculomas, these additional advanced MR modalities provide more data to diagnose such lesions with confidence [3, 4]. In parts of the world where TB is still endemic and patient lacks clinical features of systemic tuberculosis, need for invasive operative procedures for histopathological diagnosis may be avoided [2]. Additional future radiological studies are required to consolidate this approach with more focus on such cases comparing the data.

CONCLUSION

Intracranial tuberculomas have a high incidence in parts of the world where TB is still endemic and poses a specific clinical challenge with routine neuroimaging in the absence of systemic tuberculosis. Tuberculomas may be differentiated from other mass lesions such as metastases and gliomas by their unique MRI patterns, especially nonenhancing, nondiffusion restricting, T2 iso- to hypointense core, ring enhancement of the lesion, metabolite pattern with prominent lipid/lactate peaks, and only mild elevation of rCBV in perfusion imaging. A consolidated data on such imaging features may prompt early therapeutic intervention obviating need for invasive procedures for tissue diagnosis.

REFERENCES


Author Contributions
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