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*Original Research Article*

# Surgical management of posterior fossa tuberculosis

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**Abstract**
Tuberculoma of the brain is a dangerous disease that is becoming more common in developed countries as tuberculosis continues to spread. At first, antimycobacterial drugs are used to treat brain tuberculoma. But neurosurgery is the main part of treatment. Surgery is needed if the tuberculoma does not respond to medical treatment, if it is causing symptoms, or if there is no other way to find out what is wrong. In this paper, we talk about four cases of posterior fossa tuberculomas in people who had never had tuberculosis or a weak immune system before. Brain tuberculosis had to be treated with surgery in all cases for it to go away safely and effectively.

**Keywords:** Tuberculosis, cerebellar tuberculoma, surgery, brain, antimycobacterial

**Abbreviations**
**TB:** Tuberculosis.
**CNS:** Central Nervous System.
**MR:** Magnetic Resonance.
**CT:** Computed Tomography.

## Introduction

Tuberculosis (TB) is making a comeback in Western countries because people are moving there from places where it is common [1]. Hematogenous spread of TB is what causes it to spread to the central nervous system (CNS) and show up as meningitis or a tuberculoma in about 10% of cases [2]. CNS tuberculomas can be found both above and below the tentorium, but rarely in the posterior fossa. They can show up as a single lesion or as a group of lesions [3]. Most tuberculomas have a necrotic area in the middle that is surrounded by brain tissue that is swollen and has reactive gliosis [4]. Several studies have shown that even when patients are treated well, their prognosis is bad, with a death rate of between 6 and 65% [5]. In this paper, we talk about two cases of brain tuberculomas that happened in people who had never had TB before and were treated with surgery at our institution in the last four years. We talk about how hard it is to get a diagnosis before surgery and how important surgery is in dealing with difficult cases [6].

## Case Presentation

**Place of Study:** Osmania Medical College, Hyderabad, India

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## First Patient

A 35-year-old woman came to our outpatient clinic because she had been having pulsating headaches on the left side for four months, along with temporary left eye ptosis and low-grade fever. Her past medical history wasn't anything special; she didn't have a weak immune system, and she had worked for a short time in an immigration centre [7]. A Magnetic Resonance (MR) scan of the brain showed a 23 mm nodular lesion in the left superior cerebellar hemisphere and an 8 mm nodular lesion in the right inferior cerebellar hemisphere. Both of these lesions had central necrosis and ring enhancement and were surrounded by edema. The central core of the larger mass had very limited diffusion [8]. A CT scan of the whole body showed that both lungs had nodules with uneven edges and dead spots in the middle. At the time of admission, the patient seemed to be in good health overall, and the neurological exam showed nothing unusual. A biopsy was taken from the lung lesion: The histological exam showed "multifocal giant cell granulomatous inflammation with central necrosis," which is suggestive of TB granuloma [9]. Mycobacterium tuberculosis was found in bronchial alveolar lavage. So, a 4-drug treatment for tuberculosis was started: isoniazid 300 mg/day orally, which was later replaced with levofloxacin 750 mg/day orally; rifampicin 600 mg/day orally; ethambutol 800 mg/day orally; and pyrazinamide 1500 mg/day orally. The patient also had a third ventriculostomy because hydrocephalus started to happen.

The patient was then sent home, but he or she kept taking drugs to fight mycobacteria as an outpatient. One month later, the patient was taken back to the hospital because her headache and stiff neck were getting worse. A repeated MR scan of the brain showed that the left lesion, which had stayed the same size, had "grown" three satellite cystic lesions, which meant it had gotten bigger. The smaller lesion on the right, on the other hand, was stable. The medical treatment with steroids and mannitol didn't work. So, the patient had a left paramedian suboccipital craniotomy and the left cerebellar lesion was taken out completely. Without the typical caseous necrosis and Langhans giant cells, the histopathology showed a chronically inflamed area with a dead background. This picture showed how a tuberculoma changed into an abscess [11]. Mycobacterium Tuberculosis was found in the tissue by both microbiological and genomic tests. After the surgery, there were no problems, and the symptoms of intracranial hypertension got much better. After surgery, an MRI of the brain showed that the left lesion had been completely removed and that the smallest one in the right cerebellar hemisphere had shrunk in size (76 mm). The patient then finished her treatment for mycobacteria (15 months overall). At the 3-year follow-up, her health is good and the MR scan of her brain shows nothing unusual [12].

## Second patient

Tuberculosis (TB) is making a comeback in Western countries because people are moving there from places where it is common [13]. Hematogenous spread of TB is what causes it to spread to the central nervous system (CNS) and show up as meningitis or a tuberculoma in about 10% of cases [14]. CNS tuberculomas can be found both above and below the tentorium, but rarely in the posterior fossa. They can show up as a single lesion or as a group of lesions [15]. Most tuberculomas have a necrotic area in the middle that is surrounded by brain tissue that is swollen and has reactive gliosis [16]. Several studies have shown that even when patients are treated well, their prognosis is bad, with a death rate of between 6 and 65% [17]. In this paper, we talk about two cases of brain tuberculomas that happened in people who had

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never had TB before and were treated with surgery at our institution in the last four years. We talk about how hard it is to get a diagnosis before surgery and how important surgery is in dealing with difficult cases.

**Third patient**

On February 3, 2021, a 12 years old girl was taken to the infectious disease ward of Hospital. Her grandparents, who took care of her, had brought her in on a stretcher [18]. She had headaches, stomachaches, vomiting, blurred vision, weakness, and ataxia in the past. Four months before she was admitted, she started getting headaches that got worse over time, and she sometimes felt sick and threw up. A month before she was admitted, she had blurry vision, got weaker, and had trouble walking and doing other things she wanted to do. She wasn't sick or coughing. A doctor at the nearby health centre told her to go to Hospital Civil. Soon after she was sent to the hospital, she started having short, generalized tonic-clonic seizures, and then she got too weak to walk. The patient had not had any of the normal childhood vaccines, like the BCG vaccine. Her home was not a good place to live, and she ate unpasteurized cow's milk [19]. At the hospital, the patient's weight was 26 kg, his height was 150 cm, and his body mass index (BMI) was 11.6 (reference BMI for age, 18.5–24.9, by http://www.nhlbi.nih.gov/ guidelines/obesity/BMI/bmi-m.htm, accessed May 2, 2014). Her heartbeat was 121 beats per minute, her breathing rate was 60 beats per minute, her blood pressure was 70/40 mm Hg, and the temperature in her armpit was 36.3 °C. She was unconscious and couldn't understand what was being said to her. The patient's pupils were small and didn't react to light. They also had convergent esotropia on both sides, general weakness, and flexor plantar responses on both sides. Cranial axial computed tomography (CT) with contrast showed a mass in the posterior fossa and hydrocephalus. This was confirmed by cranial MRI with gadolinium. T1-weighted MRI showed a large, well-defined, heterogeneous mass in the right posterior fossa with hyperdense rim enhancement and hydrocephalus measuring 61 69 62 mm3. At first, the lesion was thought to be a primary brain tumour that was blocking the flow of CSF [20]. Right away, a ventriculo-peritoneal shunt was put in. The patient was given 2 g/kg of mannitol through an IV to reduce what was thought to be cerebral edoema. A few days later, a spinal T1-weighted MRI showed that the cervical, thoracic, and lumbar dura had a lot of gadolinium enhancement. This could be a sign of an infectious process like TB. But the TST was negative, and there were no white blood cells in the CSF. The amount of protein in the CSF was 230 mg/dL. The ratio of glucose in the CSF (37 mg/dL) to glucose in the plasma (113 mg/dL) was 0.32 (the reference range is >0.6). The Ziehl–Neelsen technique and culture did not show any acid-fast bacilli (AFB). Because it was hard to tell what was wrong, the tumour was taken out. It turned out to be a big mass that looked like cheese [21]. Hematoxylin-eosin staining and Ziehl–Neelsen staining showed granulomatous inflammation with central caseous necrosis surrounded by edematous brain tissue with reactive gliosis and many AFB. Using the new Xpert MTB/RIF assay, Rifampicin-susceptible M. tuberculosis was found in the CSF. This confirmed the diagnosis of central nervous system (CNS) TB. Ten days after being admitted, a chest CT showed consolidation in the lower left lung. However, Ziehl–Neelsen staining and TB DNA amplification of bronchial and gastric aspirates with the Xpert MTB/RIF assay were negative for M. tuberculosis. Even though her grandparents didn't have TB symptoms, her local health department was told to test other family members for TB [22]. The patient begun on 10 mg/kg/day isoniazid, 20 mg/ kg/day rifampicin, 35 mg/kg/day pyrazinamide, and 20 mg/kg/day ethambutol. For the treatment of CNS TB, dexamethasone was also added. The test for human deficiency virus infection came back negative. One month after surgery and the start of anti-TB treatment, the patient's neurological condition stayed the same: he was still unconscious and his pupils didn't react to light. He also had bilateral esotropia, general

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weakness, and flexor plantar response in both feet. She couldn't talk, so she was fed through an orogastric tube [23]. A CT scan of the head showed that the original site had no new or old lesions or contrast enhancement. The patient was cared for at Hospital Civil by a team of experts in infectious diseases and many different specialties. The medical care and medicines were free. After three months, the grandparents asked to move her to a hospital that was closer to her home. The patient died of lung problems six months after being moved [24].

## Fourth patient

A 49-year-old right-handed man with no other health problems was admitted because he had been getting worse at walking and having headaches for 3 months. The general physical exam was fine. Cerebellar signs, such as cerebellar ataxia, dysmetria, and dysdiadochokinesia, were found during a neurologic exam. The fundus of the eye was fine. The chest X-ray and routine blood tests were both fine. The patient did not have HIV. A computed tomography (CT) scan of the brain showed a midline vermian lesion that went all the way to the left cerebellum. The lesion had a mass effect on the fourth ventricle and was hypodense on its own [25]. A magnetic resonance imaging (MRI) of the brain showed that there were mass lesions in irregular patterns. On T1- and T2-weighted images, the processes looked different, and there was edema around them. After the gadolinium was given, the lesion was 3 4 cm in size and had multiple intensely enhanced nodules [26]. While our patient was lying on his back, a suboccipital craniotomy was done. After the dura mater was opened and the corticectomies were done, the lesion was white, infiltrative, firm, and not hemorrhagic. It also didn't have any planes of cleavage. A complete removal of the tumour was done. The way things went after the surgery was fine. The diagnosis of tubercu loma was confirmed by a histopathological exam. Daily chemotherapy with isoniazid, rifampicin, pyrazinamide, and ethambutol was started, and the patient was sent to the department of infectious diseases for follow-up. Two months later, the patient was sent home to finish 8 months of treatment for tuberculosis. 14 months after surgery, our patient was doing better, but he still has some very mild cerebellar ataxia. One year after surgery, a control MRI was done, and there were no signs of the cancer coming back [27].

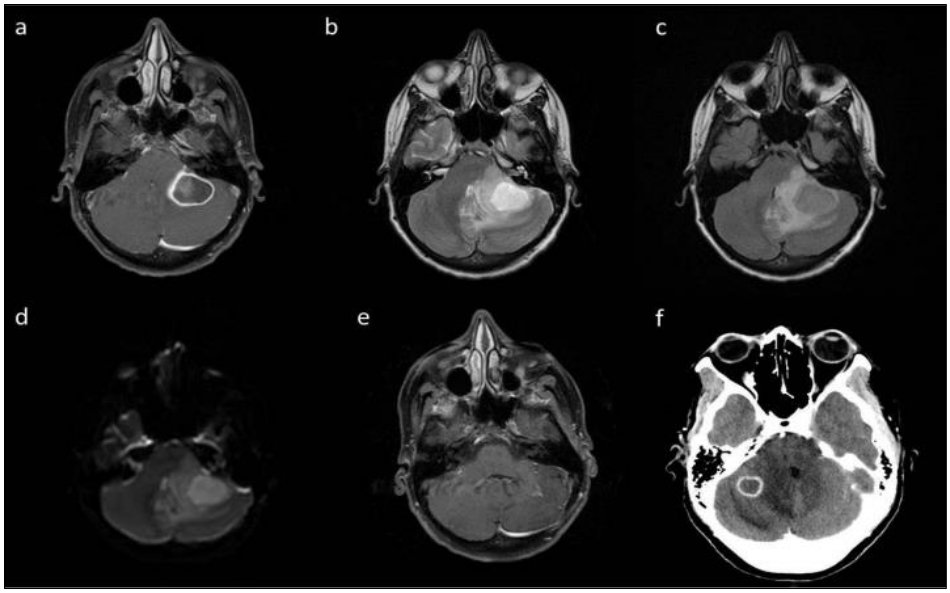


**Fig 1:** Neuroimaging of tuberculoma.

(a) While the T2-weighted; (b) FLAIR; (c) Sequences show a well-defined lesion with perifocal edema and mass effect; (d) The diffusion-weighted images show a marked

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restriction of diffusion in the necrotic central core of the larger mass; (e) The post-operative post-contrast T1-weighted axial MR scan shows the gross total removal of the lesion; (f) Pre-operative post-contrast axial CT scan of Case 2 demonstrating a round shaped lesion in the right cerebellar hemisphere characterised by a central necrotic core, a ring enhancement and perilesional edema, determining mass effect on the fourth ventricle.

## Discussion

In developing countries, TB is a big health problem. But because of immigration and a rise in the number of people with weak immune systems, TB has come back as a major health problem in developed countries as well. Tuberculomas happen when small tuberculous spots (called "Rich foci") get bigger but don't break into the subarachnoid space. This means that they can happen even if you don't have typical tuberculous meningitis [28, 29]. Histopathologically, an intracranial tuberculoma is a lesion made up of epithelioid cells, GI cells, and lymphocytes that surround a caseating necrotizing centre. It is usually a single lesion and is most often found in the frontal and parietal lobes. However, there have been reports of multiple lesions and lesions in the posterior fossa [30, 31]. In most cases of CNS TB, medical treatment is the right choice. This isn't always true for tuberculomas, which usually don't respond well to treatment with antimycobacterial drugs [32]. Surgery for tuberculoma can also be justified if a tissue biopsy is needed to start the right treatment as soon as possible or if there is a large mass effect that needs to be surgically decompressed. From a review of the literature, we found 8 case reports of posterior fossa tuberculomas, and in almost all of them, neurosurgery was done. Also, in 6 of the 8 cases, the surgery was done to relieve pressure, while in the other case, a biopsy was done only to find out what was wrong [33]. In our cases, life-threatening masses in the posterior fossa didn't respond to medical treatment or needed emergency decompression. Also, in both cases, the cerebellar tuberculoma was the first sign of TB, and in Case 2, TB had to be diagnosed through surgery. Case 1 is the first time, as far as we know, that one cerebellar tuberculoma responded to treatment while the other did not [34, 35].

There were many different combinations of focal signs and symptoms that were similar to those caused by other brain stem and cerebellum lesions. Based on our review of the literature, infratentorial tuberculoma, intracranial hypertension, and localising signs (cerebellar syndrome and ponto-cerebellar angle syndrome) can all happen. Before surgery, our patient had signs of a cerebellar syndrome. No imaging method can reliably tell the difference between tuberculoma and other intracranial mass lesions [36, 37]. On CT scans, tuberculomas can look like isodense, hyperdense, round, or lobulated masses with uneven walls that show uniform enhancement after contrast is given. Most of the time, they look like ring-enhancing lesions with vasogenic oedema around them [38]. MR imaging is a key part of diagnosis because it can find these lesions earlier than CT and is more sensitive and specific than CT. Non-caseating tuberculoma is usually isointense or hypointense on T1 images and very intense on T2 images. Homogeneous enhancement is seen with gadolinium. Solid tuberculoma is usually very low in signal on T1 images and very low in signal on T2 images. The granulation tissue and compressed glial tissue in the central core are to blame for this relative low intensity. This is because the central core has more cells than the brain parenchyma [39].

## Conclusion

In conclusion, brain tuberculomas are not rare and should always be considered when a brain lesion is found inside the skull. Neuroimaging might be able to help with the diagnosis, since diffusion-weighted images of people with TB show that their movement is very limited. Even

though medical therapy must be tried first, surgery is usually the best way to treat tuberculomas, especially when the mass effect is a big problem. Cerebellar tuberculomas aren't very common, but it's important to think about diagnosis when there are masses in the infratentorial space. They are usually hard to diagnose, and waiting too long to find and treat this rare location can lead to a higher death and illness rate.

**Conflict of Interest:** None

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