

# Left Parietal Lobe Tuberculoma in an Immunocompromised Patient Diagnosed in Europe

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## Abstract

*Tuberculomas are intracranial manifestations of tuberculosis. In endemic regions they account for a third of intracranial lesions. In patients without signs of meningitis, clinical features are indistinguishable from any other intracranial lesions. Neuroimaging can present various patterns, which can be identified on computed tomography (CT) scan and magnetic resonance imaging (MRI). Each pattern has characteristic MRI appearances and differential diagnoses on imaging.*

*We present a case of central nervous system tuberculosis (CNS TB) in an immunoincompetent host who presented new-onset seizures, a 46-year-old woman who complained of headache and consciousness disturbance. Brain CT and MRI showed lesion in left parietal lobe, ring enhancement, with mass effect and edema.*

*A left parietal craniotomy with total resection of the lesion was performed, without complications associated with the procedure. Histopathological study was suggestive of tuberculoma with multifocal granulomas, caseous necrosis and Langerhans giant cells. Early diagnosis and treatment can be lifesaving. Even, in developed countries the diagnosis is difficult and frequently delayed or missed. Histopathology is required for a definitive diagnosis. Prompt surgical resection and decompression if necessary with adequate antitubercular treatment yield better neurological outcomes. The type and the duration of the treatment depend on age, general state of health, possible resistance to the medicines and the location of the infection in the body.*

**Keywords:** Central Nervous System Tuberculosis, Tuberculoma, Mycobacterium, Craniotomy, Total resection.

## 1. Introduction

Tuberculosis (TB) an infectious disease caused by *Mycobacterium tuberculosis* is one of the leading causes of death worldwide. In 2020, 1.5 million people died from TB, of which 214,000 had HIV/AIDS. About 90% of humans who developed this disease are adults, with more cases among men than women. About a quarter of the world's population is infected with *M. tuberculosis* [1].

TB is a curable and preventable disease. About 85% of people who develop TB disease can be successfully treated with a 6-month drug regimen and regimens of 1–6 months can be used to treat TB infection [1]. The most important threats to controlling the TB epidemic is the appearance of *M. tuberculosis* strains resistant to the most effective drugs, migration and globalization, and the HIV and TB co-infection [2, 3].

The causative agent of tuberculosis is *Mycobacterium tuberculosis*, it is highly aerobic and therefore affects primarily the respiratory system. The disease typically affects the lungs (pulmonary TB) but can affect other sites (extrapulmonary TB) [1, 3]. The involvement of the CNS by this infection accounts for 1% of all the TB cases and 5% of all extrapulmonary TB cases [4, 5].

Tuberculosis of the central nervous system (CNS) is the most devastating form of the disease. The pathogen induces a granulomatous inflammatory response in the brain, resulting from hematogenous spread of the bacteria from a primary focus elsewhere in the body [6].

CNS TB includes three clinical entities: tuberculous meningitis, intracranial tuberculoma, and spinal tuberculous arachnoiditis. The most frequent form of presentation in low-prevalence countries

is meningitis. Tuberculomas are intracranial manifestations of tuberculosis. In endemic regions they account for a third of intracranial lesions [7].

In developing countries, TB has high incidence rate, however the diagnosis remains very difficult [1, 4]. Rapid diagnosis is essential to prevent its transmission to other individuals as well as to improve the prognosis of the disease [8].

Due to its complex clinical manifestations, extrapulmonary TB poses a great difficulty on early diagnosis. In patients without signs of meningitis, clinical features are indistinguishable from any other space-occupying lesion. Neuroimaging can present various patterns, which can be identified on computed tomography (CT) scan and magnetic resonance imaging (MRI). MRI is considered the modality of choice in assessment and detection of CNS TB due to its higher specificity and sensitivity. Each pattern has characteristic MRI appearances and differential diagnosis on imaging [9].

Tuberculoma may arise during or after a meningitis. This pathology usually occurs in an immunocompromised host, but cases in immunocompetent patients have also been described. An even smaller portion of the CNS tuberculomas are located in the deep

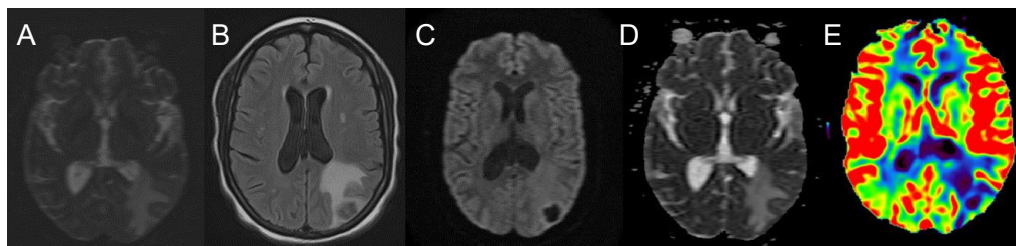
brain parenchyma, not causing meningeal irritation. These deep lesions can remain asymptomatic or eventually cause headaches, induce seizures and precipitate neurological deficits caused by the mass effect of these space-occupying lesions [10].

We reported a rare case of left parietal lobe tuberculoma in an immunocompromised woman who developed headache, altered state of consciousness and seizures without visual loss. A surgical procedure was performed, as described below, and allowed to obtain a histological result and complement the medical treatment according to current guidelines.

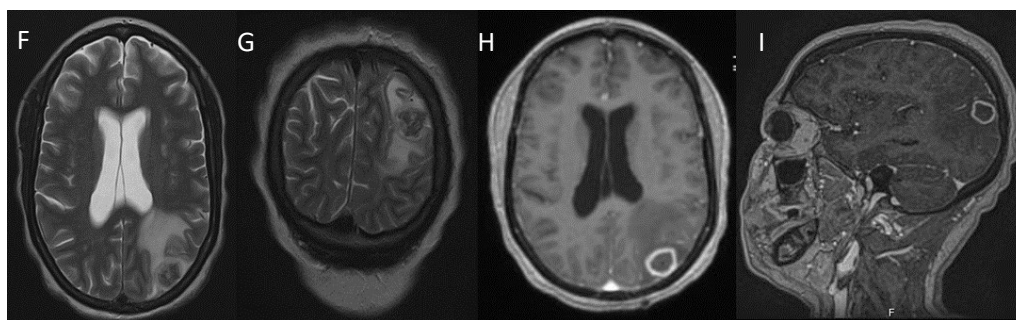
## 2. Case Report

We present a case of a 46-year-old African woman with immunodeficiency due to infection by HIV. She came to our hospital with complaints of headache, altered state of consciousness and seizures. Upon physical examination the patient showed no significant visual deficits, no fever and no episodes of nausea or vomiting.

A brain CT was carried out. Posteriorly, an MRI revealed a left parietal lobe lesion, with ring-enhancing enhancement and vasogenic edema (Figures 1 and 2).



**Figure 1:** Brain MRI revealing left parietal intracranial lesion. The lesion shows hypointensity on T2/ FLAIR (images A and B). Of note is the characteristic pattern of diffusion restriction (images C and D), as well as the absence of perfusion (image E). A. Axial T2 Sequence; B. FLAIR Axial Sequence; C. DWI; D. ADC map. ; E. Perfusion



**Figure 2:** Brain MRI (cont.) The lesion presents hypointensity on T1 and T2 sequences and enhancement with peripheral contrast uptake (images H and I). F. Axial T2 Sequence; G. Coronal T2 Sequence; H Axial T1 sequence with contrast; I. Contrast-enhanced Sagittal T1 Sequence.

At this point, our differential diagnosis was the following: neurocysticercosis, tuberculomas, toxoplasmosis, lymphoma, high grade glial serial tumors, metastatic brain cancer.

After the brain CT and MRI, and since the patient did not exhibit any focal neurological deficits, and she presented a reduced

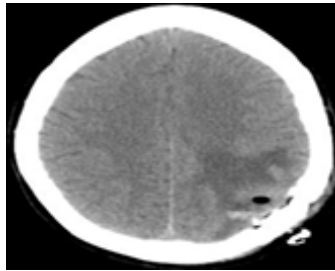
Glasgow Coma Scale, no papilledema and in this context was performed a lumbar puncture (LP).

LP showed an opening pressure of 350 mm H<sub>2</sub>O, cerebrospinal fluid (CSF) white blood cell count of  $7 \times 10^3 / \mu\text{L}$ , and CSF glucose and protein were 48 mg/dL and 87 mm/dL, respectively, with a

53% monocytes predominance. The patient was also screened for HIV with an Ab/Ag (antibody/antigen) screen, which was non-reactive.

Due to size, location and mass effect, a left parietal craniotomy

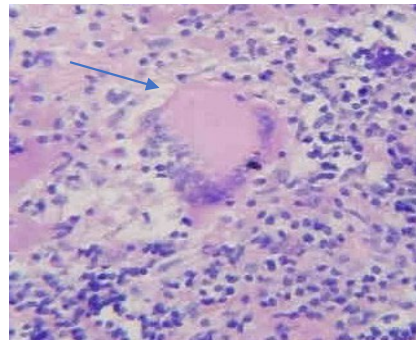
with neuronavigation support was performed. Using the support of the surgical microscope and resorting to microdissection techniques, a total resection of the lesion was obtained. There were no complications associated with the aforementioned procedure (Figure 3).



**Figure 3:** Postoperative contrast-enhanced computed-tomography (CE-CT) reveals absence of post-surgical complications with apparent complete excision of the lesion.

Towards a high index of suspicion for TB, she was empirically placed on four anti-TB medications and continued a steroid who had previously started as levetiracetam 500 mg twice a day. The

histopathological study was strongly suggestive of tuberculoma with the presence of multi-focal granulomas, caseous necrosis and giant cells of Langerhans (Figure 4).



**Figure 4:** Coloration Hematoxylin-eosine (H&E x400), Giant cells of Langerhans (arrow) with numerous lymphocytes scattered around.

It was identified a *M. tuberculosis* complex after six weeks in the laboratory and a report by the Public Health Services Department showed pan sensitivity without any resistance. The patient's drug regime consisted of isoniazid, rifampin, pyrazinamide, ethambutol, pyridoxine, and dexamethasone. All four anti-TB medications were administered for 2 months with maintenance therapy consisting of isoniazid and rifampin for an additional nine months. Dexamethasone was administered and tapered over a total of eight weeks at 0.3 to 0.4 mg/kg/day for two weeks, 0.2 mg/kg/day for week 3; 0.1 mg/kg/day for week 4, and then 4 mg per day and tapered 1 mg off the daily dose each week. The patient's symptoms rapidly improved with this drug regime, and repeat brain imaging a few weeks after starting medications revealed that some of the tuberculomas had already resolved.

Airborne isolation was cleared after three negative sputum acid-fast bacilli (AFB), and the patient was discharged home with the same 4-drug regimen and a steroid taper dose as described previously. The patient-maintained follow-up during 2 years and had no new seizures or recurrence of the lesion.

### 3. Discussion

Tuberculosis is a major health concern in developing countries where its prevalence is high. There is obviously an overall decrease in the number of people newly diagnosed with TB and notified. Comparing the data for 2019 and 2020 there is a decrease from 7,1 million to 5,8 million, respectively. Data up to June 2021 showed continued declines. The most obvious impact on TB after disruptions caused by the COVID-19 pandemic was a large global drop in the number of people newly diagnosed with TB and reported in 2020, compared to 2019 information [1].

In certain areas, late diagnosis and the difficult access to TB treatment resulted in an increase in the number of deaths related to this pathology. Best estimates for 2020 are 1.3 million TB deaths among HIV-negative people (up from 1.2 million in 2019) and an additional 214,000 among HIV-positive people (up from 209,000 in 2019). Declines in TB incidence (the number of people developing TB each year) achieved in previous years have slowed almost to a halt. These impacts are forecast to be much worse in 2021 and 2022 [1].

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When it spreads to the CNS, TB usually manifests as meningitis, the most common symptom. *M. tuberculosis* rarely infects the brain parenchyma in immunocompetent individuals [11]. Intracranial tuberculomas are uncommon forms of extrapulmonary TB. The tuberculoma is a granuloma formed by the inflammatory response to *M. Tuberculosis* infection. Macrophages induced by T-lymphocytes engulf the bacilli and form giant cells. Caseous material containing a few bacilli may appear at the center, surrounded by gliosis and lymphocytic infiltration. These lesions are slow-growing with variable perifocal edema, variable in size (up to 3-4 cm) [12].

They tend to occur at the grey-white matter junction due to arrest of the haematogenously spread microbes caused by a reduction in calibre of vessels in that region. Occasionally, lesions can develop in the brain parenchyma secondary to spread of cerebrospinal fluid (CSF) infection through the perivascular (Virchow Robin) spaces. It may involve any part of the CNS, more common in the cerebral hemispheres. They may be severe with a high risk of mortality, therefore a prompt diagnosis and treatment is necessary. The incidence of intracranial tuberculoma is 5–30% of all intracranial lesions in developing countries. The symptomatology is variable depending on the location, the size and the number of lesions. The diagnosis and treatment are late due to the clinical polymorphism and the absence of a previous history of tuberculosis in the majority of the patients [13]. Tuberculomas may be found anywhere in the brain or attached to the meninges though they are most commonly found in the parenchyma [14].

Patients with tuberculoma often present symptoms of high intracranial pressure with focal neurological deficits. The most frequent presentation of intracranial hypertension includes headache, papilledema, vomiting, seizures, visual disturbance, drowsiness, hemiparesis, paraparesis and ataxia. Fever and signs of systemic infection are rare [13]. In our case the patient presented headaches with subsequent deterioration of the state of consciousness and new-onset seizures.

The management of intracranial tuberculoma remains a big challenge due to the lack of specific diagnostic tools. Diagnosis is usually made by evaluating the clinical presentation, epidemiology, and imaging studies and sometimes fine needle biopsy. The most used techniques are often based on imaging such as CT scan and MRI. The CT scan is the imaging exam performed initially. Nevertheless, 80% of the cases diagnosed by CT alone could be false positive [12]. Lumbar punctures (LP) are sometimes avoided due to the risk of brain herniation, but when performed, they usually reveal normal and nonspecific results. MRI presents greater sensitivity and specificity than TC scan and, when achieved once suspecting an intracranial tuberculosis, should include axial precontrast T1W, T2W, FLAIR (fluid-attenuated inversion recovery sequence), DWI (diffusion-weighted imaging sequence), and GRE (gradient echo sequence)/SWI (susceptibility-weighted imaging sequence) sequences and postcontrast T1W scans in all three planes. H proton spectroscopy and magnetization transfer

imaging may be additionally performed in cases where the morphology or distribution of the lesions are atypical, presenting a diagnostic challenge. Magnetic resonance angiography (MRA) and magnetic resonance venography (MRV) may be used in cases related to vascular complications [15]. MRI is usually inconclusive and may not distinguish TB from a brain tumor. Therefore, the definitive diagnosis is established through a brain biopsy [13].

Tuberculomas within the brain are usually treated pharmacologically and not with surgery as this may seed meningitis. Surgical intervention is indicated if the lesions are at risk of causing obstructive hydrocephalus or mass effect to surrounding brain parenchyma or neuroocular structures. They are often misdiagnosed as tumors or metastatic disease [14].

Many authors emphasize the need for biopsy to obtain material for histopathological study and thus obtain a definitive diagnosis of tuberculoma [14]. Surgical removal must include decompression and excision as much as possible preventing the additional neurological morbidity followed by intensive treatment with antitubercular drugs as the biopsied or partially excised lesion may exhibit persistent lesion even after full course of antituberculostatic treatment [16].

An intensive treatment with antitubercular drugs during the adequate time (6–9 months) could be enough to eliminate tuberculoma but may need to be prolonged one year or more depending on the radiological state of the lesion [17].

#### 4. Conclusions

Intracranial tuberculosis is a unique disease with different forms and characteristics in imaging tests such as CT or MRI. Produces a wide spectrum of intracranial patterns.

Differential diagnosis can be difficult, especially in countries with fewer technological resources. Even in developed countries, given the wide presentation of this pathology, diagnosis is difficult and often delayed.

Early diagnosis and treatment are essential prognostic factors. It has been shown that early initiation of treatment is associated with a lower incidence of permanent neurological sequelae. Although the symptoms and the characteristics of the imaging tests are important for the diagnosis they are unspecific. Actually, the histological study is the technique that allows the definitive diagnosis.

Surgical resection or biopsy when indicated, should be complemented with adequate antituberculous treatment. It is currently considered the gold standard and produces better neurological results.

The type of treatment and the duration depend on several factors such as age, general health, possible drug resistance, and the infection location. In fact, as a relevant and worrying aspect for



the future, the incidence of multidrug-resistant tuberculosis has increased during the last decade in many parts of the world.

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