

Cerebral Tuberculoma: A Report of 12 Cases

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How to cite this paper: El Wavi, S.A.T., Sidi-Mohamed, M., Tolba, E., Koné, N., Eleitt, A.-E., Kleib, A.-S., Soumaré, O. and Salihiy, S.M. (2026) Cerebral Tuberculoma: A Report of 12 Cases. *Open Journal of Modern Neurosurgery*, 16, 229-233.
<https://doi.org/10.4236/ojmn.2026.163021>

Received: April 4, 2026

Accepted: June 22, 2026

Published: June 25, 2026

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Abstract

Despite their long history, cerebral tuberculomas remain a diagnostic and therapeutic challenge. This retrospective, two-center study reports the management of 12 patients treated at our institution. Diagnosis was based on clinical and radiological (MRI/CT scan) and/or histological criteria. Management combined antituberculosis treatment (100% of cases) and surgical intervention (excision) for large or compressive lesions. The results showed a favorable outcome in the majority of patients, with a significant reduction in neurological sequelae and mortality thanks to a multidisciplinary approach. These results confirm that early diagnosis and therapeutic combination are crucial for the prognosis of this disease, which affects an active population.

Keywords

Tuberculous Meningitis, Meningoencephalitis, Epileptic Seizure, Tuberculoma, Multidrug-Resistant Tuberculosis

1. Introduction

Cerebral tuberculosis remains a major cause of morbidity, primarily affecting poor and vulnerable populations. It constitutes a major public health concern in developing countries, where it accounts for 10% - 30% of intracranial space-occupying lesions in underdeveloped countries, compared to 0.2% in some Western countries [1]. Central nervous system (CNS) involvement is a severe form of tuberculosis, occurring mainly through hematogenous dissemination from a pulmonary focus [2]. Data on this pathology are limited, characterized by clinical polymorphism and diagnostic and therapeutic challenges. MRI with various spectroscopic sequences contributes to diagnosis and avoids the need for a steroid biopsy. Treatment relies on anti-tuberculosis drugs and surgery, depending on the severity of the disease.

2. Materials and Methods

This is a retrospective study of 12 cases of cerebral tuberculoma treated at the Neurosurgery Department of the Specialized Hospital Center (CHS) and the Nouadhibou Hospital Center (CH NDB) between July 1, 2022, and June 30, 2025. The mean age of the patients was 36.7 years. Computed tomography (CT) and magnetic resonance imaging (MRI) of the brain were performed on the majority of our patients. Patient consent was obtained and patient confidentiality was protected in this retrospective study.

3. Results

In our series, the average age was 36.7 years, with a female predominance (7 women and 5 men). The clinical presentation was primarily dominated by a decline in general health, headaches, and intracranial hypertension in 9 patients, sometimes associated with other symptoms such as seizures in 2 patients, neurological deficits in 2 patients, and varying degrees of altered consciousness. Tuberculomas were located in different lobes, with 7 cases supratentorial and 5 infratentorial. CT scans were performed in 10 patients and MRI in 11 patients. The miliary pattern of the neuraxis, found in 2 patients (16.6%), is highly characteristic of the disease. Lumbar puncture was performed in 6 patients. All patients underwent hospitalization and medical treatment with anti-tuberculosis drugs. Two patients underwent hospitalization following a positive lumbar puncture (LP) with lymphocytosis, elevated protein, and low glucose levels. Two patients underwent stereotactic biopsy, and one patient presented with hydrocephalus, for which surgical treatment was performed via external ventricular shunt placement. Postoperative outcomes were favorable in 90% of cases, with regression of clinical symptoms and resolution of paraclinical abnormalities. Follow-up was regular, with an average interval of one year.

4. Discussion

The origin of tuberculoma is attributed to bacterial infection by *Mycobacterium tuberculosis*, also known as Koch's bacillus, composed of tuberculous granulomatous tissue that has been contained and limited by the host's immune defenses. TNF- α appears to play a decisive role in granuloma formation [3]. Tuberculomas are firm, spherical, and can reach 10 cm in size. The frequency of cerebral and meningeal tuberculosis is variable; in some studies it is <10% [4], while the rate reported in our series is 12.2%. Early onset is frequently reported [5]; with a female predominance found in our series, with a sex ratio of 1.4. The clinical manifestations of tuberculomas are characterized by polymorphism, often with a subacute onset (as in all our patients) associated with headaches, Intracranial hypertension syndrome and/or altered consciousness ranging from confusion to coma were observed in 9 patients (75%). Seizures were noted in 2 patients, related to the preferential cortico-subcortical location of the tuberculomas. Neurological deficits were also observed in 2 patients. These results are consistent with figures reported in the literature. Brain imaging plays a significant role in the diagnosis and monitoring of tuberculoma pro-

gression. The absence of human immunodeficiency virus infection is noted despite immunosuppression being a vulnerability factor for tuberculosis. However, cerebral CT scans are not specific to tuberculomas. Without contrast injection, the lesion may be isodense, hyperdense, or of mixed density. After injection, the appearance is more typical: a hypodense lesion with peripheral rim enhancement, sometimes associated with central calcifications creating a “target image” (**Figure 1**). This appearance is not specific and can also suggest infectious or other pathologies: cysticercosis, pyogenic abscesses, sarcoidosis, metastases, gliomas, and lymphomas [6]. Brain CT scans do not necessarily detect small or infratentorial tuberculomas. On MRI, the tuberculoma appears on T1-weighted images as a moderate hypointense signal in the white matter, more pronounced at the periphery due to the edematous rim. On T2-weighted sequences, the lesion is negatively contrasted within the hyperintense edematous area. The center of the lesion is hypointense or hyperintense depending on the stage of development. The capsule is isointense or slightly hyperintense on T1-weighted images, and hypointense on T2-weighted images, taking on a target-like appearance. After gadolinium injection, very intense nodular contrast enhancement is observed, of varying sizes and often irregular, homogeneous or heterogeneous, with isointense or hypointense central necrosis (**Figure 2**). MRI is particularly useful for differentiating tuberculomas from pyogenic abscesses, neoplasms, and cysticercoses.

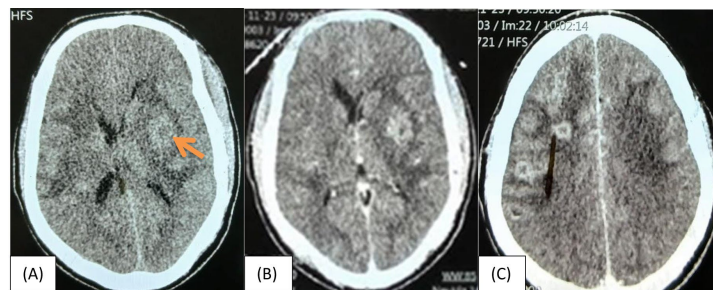


Figure 1. (A), a CT scan without contrast injection, at the arrow, the lesion has mixed density. (B), After injection, the appearance is more typical: a hypodense lesion with peripheral contrast enhancement in a ring, hypodense in the center creating a “target image”. In (C), multiple nodular lesions with the same appearance.

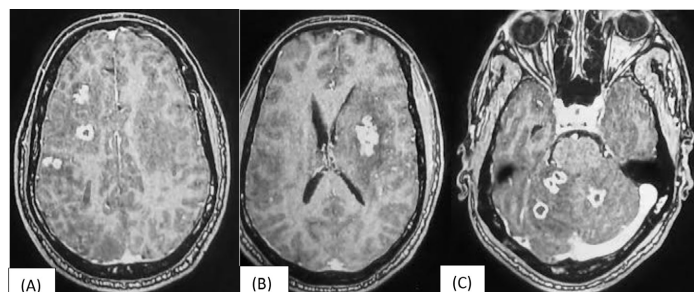


Figure 2. T1-weighted MRI with gadolinium; the capsule is hyperintense (target sign). Intense, large, irregular, homogeneous or heterogeneous nodular contrast enhancement with iso- or hypointense central necrosis. (A), (B) Supratentorial, (C) Multiple infratentorial lesions.

Tuberculomas show an elevated lipid peak (highly specific), an increase in choline, and a decrease in N-acetylaspartate (NAA) and creatinine. The choline/creatinine ratio is greater than 1 in all tuberculomas. In pyogenic abscesses, a lipid peak is found associated with a NAA peak. In neoplasms, such as metastases and gliomas, a lipid peak is found associated with a significant elevation of the choline/NAA ratio (38, 14). The miliary pattern of the neuraxis, found in 2 patients (16.6%), is very characteristic of the disease. The coalescence of several tuberculomas forming a multilocular, clustered lesion is another suggestive feature [7]. Tuberculomas can also be located in the subarachnoid, subdural, or epidural space. They are frequently supratentorial (as in our study, 58, 33%). In cases of associated meningeal involvement, CSF examination reveals lymphocytosis often below 400/mm³, variable elevated protein levels (up to 4 g/L), and low glucose levels [8]. Isolation of the tubercle bacillus from the CSF confirms the diagnosis. However, this diagnosis is challenging; the CSF may be normal, indicating the absence of meningeal involvement [9]. Sometimes, lumbar puncture cannot be performed due to a contraindication. Microbiologically, the sensitivity of isolating the bacterium from the CSF by direct examination using Ziehl-Neelsen staining and by culture on specific media is low: 11% to 44% [10]. Detection of the tubercle bacillus in the CSF by gene amplification, such as polymerase chain reaction (PCR), is very useful for rapid diagnosis, with a specificity of 90%. The diagnostic yield of stereotactic biopsy of tuberculomas varies across studies, ranging from 31% to 86%. The histological study of the samples taken confirmed the diagnosis. Early treatment is a major prognostic factor [11]. A good clinical response to antituberculosis treatment is another reason for rapid diagnosis and treatment. The treatment of cerebral tuberculomas is primarily medical, involving a combination of antibiotics (isoniazid, rifampicin, pyrazinamide, ethambutol) in two phases: an initial two-month course of four antibiotics followed by maintenance therapy of varying duration. Corticosteroids are used as adjunctive therapy. In countries with high tuberculosis endemicity, surgical excision or biopsy is deferred until there is no response to antituberculosis treatment or intolerable signs of intracranial hypertension [12]. Most international guidelines (American, British, and those of the Centers for Disease Control and Prevention) and case studies recommend a duration of 9 to 12 months or more in cases of persistent minimal lesions on imaging. In our series as a whole, the outcome is favorable with no relapse, neurological sequelae such as epileptic seizures, and no death during follow-up.

5. Conclusion

The difficulty in obtaining a definitive diagnosis of cerebral tuberculoma necessitates careful analysis of epidemiological and clinical data. The use of cerebral MRI with various sequences, particularly spectroscopic ones, greatly contributes to the diagnosis of tuberculosis and allows for early treatment of patients, avoiding stereotactic biopsy. Given the curable nature and severity of the disease, the possibility of a tuberculous origin of a cerebral mass should be considered as a matter of

principle in endemic areas. Therefore, recommendations on the management of cerebral tuberculomas, specifying in particular the cases requiring surgical intervention and the duration of anti-tuberculosis treatment, should be developed.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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