Proceedings of Anticancer Research, 2026, Volume 10, Issue 1
http://ojs.bbwpublisher.com/index.php/PAR
ISSN Online: 2208-3553
ISSN Print: 2208-3545

# Cerebral Tuberculoma on the Left Frontal Lobe

**Zixuan Zhou[1], Shanshan Wang[2]***

[1]Graduate School of Xinjiang Medical University, Urumqi 830054, Xinjiang Uygur Autonomous Region, China
[2]Medical Imaging & Radiology Center, People's Hospital of Xinjiang Uygur Autonomous Region, Urumqi 830001, Xinjiang Uygur Autonomous Region, China

**Corresponding author: Shanshan Wang, 1549578545@qq.com*



**Abstract:** Cerebral tuberculoma, a rare extrapulmonary tuberculosis manifestation, was reported in a 70-year-old man presenting with seizure-like episodes. MRI revealed a 13.6 × 45.1 × 7.9 mm left frontal extra-axial lesion with T1-isointense/T2-hyperintense signals, marked contrast enhancement, and adjacent leptomeningeal thickening. Negative thoracic/abdominal CT and inflammatory CSF findings initially suggested a neoplasm. Histopathological examination of the resected lesion confirmed granulomatous inflammation with acid-fast bacilli, thereby leading to the diagnosis. This case report emphasizes the importance of considering tuberculous granuloma in older patients with intracranial masses and inconclusive CSF studies. Multi-model imaging combined with biopsy remains critical for definitive diagnosis. Early surgical intervention and antitubercular therapy may improve outcomes.

**Keywords:** Cerebral tuberculoma; Meningeal thickening; Leptomeningeal enhancement

**Online publication:** February 9, 2026

## 1. Introduction

Cerebral tuberculoma, a distinct form of central nervous system tuberculosis caused by *Mycobacterium tuberculosis* infection, manifests as a granulomatous lesion within the brain parenchyma [1]. In recent years, the incidence of cerebral tuberculoma has risen alongside the global resurgence of tuberculosis and the increasing prevalence of immunocompromised conditions such as HIV/AIDS [2]. Tuberculomas involving the frontal lobe, a critical functional brain region, can induce seizures, cognitive impairment, motor deficits, and other neurological dysfunction, thus severely compromising patients' quality of life. Therefore, precise diagnosis, timely treatment, and comprehensive care are essential for improving prognosis [3]. This report analyzes clinical data for a rare case of left frontal cerebral tuberculoma without clinical evidence of tuberculosis, focusing on its diagnostic and therapeutic challenges.

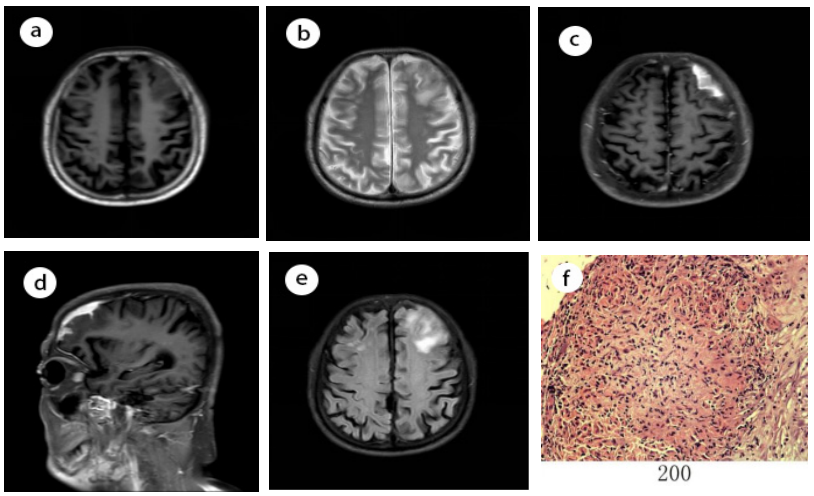
## 2. Case report

A 70-year-old man was admitted with two episodes of transient loss of consciousness accompanied by intermittent

limb convulsions. Three months prior, he had experienced a sudden loss of consciousness with limb convulsions lasting several minutes, followed by 3 hours of drowsiness, which resolved after symptomatic management at a local hospital. After recurrence, his symptoms worsened, including persistent unconsciousness and convulsions lasting 7 hours. He denied frothing at the mouth, dizziness, headache, nausea, vomiting, or incontinence. A physical examination revealed a temperature of 36.7°C, pulse of 80 bpm, respiratory rate of 21/min, blood pressure of 133/95 mmHg, and no significant abnormalities in cardiopulmonary or abdominal assessments. In neurological examination, neck stiffness, Kernig's sign, Brudzinski's sign, and focal deficits were absent. His laboratory findings included white blood cells at 8.42 × $10^9$/L, eosinophils at 0.7%, hemoglobin at 12.4 g/dL, platelets at 168 × $10^9$/L, glucose at 5.84 mmol/L, total protein at 69.7 g/L, albumin at 36.84 g/L (↓), and globulin at 32.86 g/L (↑). Lumbar puncture revealed cerebrospinal fluid (CSF) with Pandy's test positivity, glucose at 10.50 mmol/L (↑), protein at 1.96 g/L (↑), and no bacterial growth after 5-day culture.

Cranial MRI revealed an irregular left frontal subcranial lesion (13.6 × 45.1 × 7.9 mm) with an isointense T1-weighted signal (**Figure 1a**) and slightly hyperintense T2-weighted signal (**Figure 1b**), and demonstrated marked enhancement post-contrast. Adjacent frontotemporal leptomeningeal thickening and uniform enhancement were accompanied by mild swelling of the left frontal parenchyma, which showed a slightly diminished T1 signal (**Figure 1c**) and hyperintensity on T2-weighted (**Figure 1d**) and FLAIR sequences, with prominent leptomeningeal enhancement (**Figure 1e**). The left frontal subcranial lesion and adjacent frontotemporal dural thickening suggested benign extracranial pathologies, including central nervous system Rosai-Dorfman disease (CNS-RDD), hypertrophic pachymeningitis, and meningioma en plaque (MEP). Neuronavigation-assisted cerebral lesion resection and brain biopsy were performed. Histopathological analysis confirmed granulomatous inflammation with acid-fast staining positivity in the left frontal lobe—findings consistent with tuberculosis. The final diagnosis was cerebral tuberculoma (BTB) (**Figure 1f**).

After active surgical intervention, antitubercular therapy, management of underlying comorbidities, and comprehensive supportive care, our patient achieved progressive clinical improvement. Antitubercular treatment was continued post-discharge with scheduled follow-up evaluations.




**Figures 1.** a–b: An irregular left frontal subcranial lesion (13.6 × 45.1 × 7.9 mm3) showing isointense T1 and slightly hyperintense T2 signals. Figures b-c: Marked post-contrast homogeneous enhancement with adjacent frontotemporal meningeal thickening and mild left frontal parenchymal swelling. Figure e: Hyperintense T2/FLAIR signals and prominent surrounding leptomeningeal post-contrast enhancement. Figure f: Acid-fast stain positive granulomatous lesions in the left frontal lobe, consistent with tuberculosis.

## 3. Discussion

The clinical manifestations of cerebral tuberculoma include multidimensional nonspecific features, which can be broadly categorized into three patterns: (1) intracranial hypertension syndrome (progressive headache, projectile vomiting, and papilledema); (2) focal neurological deficits (motor impairment, dysarthria, and visual field defects); and (3) neuropsychiatric symptoms (cognitive decline and behavioral abnormalities) [4]. The imaging findings in our case aligned with the histopathological characteristics of immature tuberculoma [5], including dynamic clinicopathological correlations, in which the early-stage symptoms reflect predominantly vasogenic edema-induced intracranial hypertension (headache or dizziness), whereas granuloma expansion and perilesional gliosis can trigger seizure-like episodes [6–8]. Multimodal CT/MRI is critical for diagnosis and staging. Pathologically, immature tuberculomas exhibit granulomatous hypercellularity with vascular proliferation, which translates to distinct imaging patterns comprising (1) T1-weighted isointensity/hypointensity reflecting a dynamic equilibrium between cellular density and water content; (2) T2-weighted central isointensity with peripheral hyperintensity ("double-ring sign") due to inflammatory infiltration and vasogenic edema; and (3) marked post-contrast homogeneous or clustered nodular enhancement due to blood-brain barrier disruption by neocapillaries, and no mature "target sign" architecture, given the absence of caseous necrosis [9,10]. These evolving imaging signatures provide critical diagnostic clues. We therefore advocate for a tripartite decision-making framework integrating imaging staging, pathological progression, and therapeutic strategies.

The first differential diagnosis in this case, CNS-RDD, affects predominantly children and adolescents, has a slight male predilection, and is characterized by painless lymphadenopathy and histiocytic proliferation potentially involving multiple organ systems. Although most cases have favorable outcomes, central nervous system involvement can lead to severe neurological complications including headache, seizures, limb weakness, and cranial nerve dysfunction [11]. Typical MRI features include T1-weighted isointensity, T2-weighted iso-/mild hyper-/hypointensity, and marked homogeneous post-contrast enhancement. Notably, early-stage cerebral tuberculomas without caseous necrosis can also exhibit uniform enhancement because of contrast accumulation in vascularized lesions, whereas CNS-RDD typically presents as extracerebral enhancing masses with T1 isointensity and T2 iso-/mild hyperintensity [12]. These findings partially overlapped with the imaging findings in our case. Therefore, a comprehensive evaluation integrating age-sex epidemiology, clinical manifestations, multimodal imaging (e.g., MR spectroscopy), and microbiological evidence is essential for differential diagnosis.

Our second differential diagnosis, hypertrophic cranial pachymeningitis (HCP), is a rare fibroinflammatory disorder characterized by chronic, progressive thickening of the dura mater. Etiologically, HCP is classified as idiopathic (immune-mediated, with unclear pathogenesis) or secondary to infections, malignancies, or autoimmune diseases. Pathologically, it manifests as focal/diffuse fibrous dural thickening with chronic presence of inflammatory infiltrates (lymphocytes and plasma cells) [13]. Clinically, 92% of patients with HCP initially present with persistent headache, which is frequently accompanied by cranial neuropathies (e.g., optic/oculomotor nerve involvement) and ataxia, whereas epileptic seizures occur in rare cases [14,15]. Contrast-enhanced MRI demonstrates specific features of T1-weighted iso-/hypointensity, T2-weighted hypointensity, and marked dural enhancement, predominantly along the tentorium cerebelli and falx cerebri, thereby forming a characteristic "Mercedes-Benz sign" enhancement pattern [16]. Although our case exhibited meningeal thickening on MRI and inflammatory laboratory markers, the absence of typical persistent headache, elevated CSF leukocytes, or protein levels necessitated exclusion of HCP through meningeal biopsy.

The third diagnostic consideration in our report, MEP, a rare meningioma subtype, predominantly involves

the sphenoid ridge or skull base regions [17,18]. Its hallmarks include extensive dural infiltrations with adjacent skull hyperostosis (incidence: 25%–49%) [19]. MRI typically demonstrates T1-weighted isointensity, T2-weighted iso-/mild hyperintensity, iso-intensity on DWI, and homogeneous enhancement post-contrast, thus resulting in imaging overlap with immature cerebral tuberculoma. However, MEP classically exhibits a dural tail sign, with enhancement confined to the dural layer [20]. Although our case showed a left frontal subcranial irregular enhancing lesion with adjacent meningeal thickening partially matching typical MEP imaging features [21] (T1 iso-intensity, T2 iso-/mild hyperintensity, and homogeneous enhancement), key differentiating factors included prominent leptomeningeal enhancement extending beyond typical MEP patterns. Consequently, MEP was the third differential diagnosis.

In our patient, the CSF showed a positive Pandy's test, and elevated protein and glucose, probably as a result of blood-brain barrier disruption and CSF metabolic derangements caused by tuberculous inflammation. The negative CSF bacterial culture might have reflected the slow growth rate and extensive culture requirements of *Mycobacterium tuberculosis*. Although CSF analysis plays a critical diagnostic role in tuberculous meningitis, its utility in cerebral tuberculoma remains limited [22,23].

Our patient presented with seizure-like episodes (transient loss of consciousness and limb convulsions) as the predominant clinical feature, and lacked typical tuberculous toxic symptoms (e.g., low-grade fever, night sweats, or weight loss) and chest CT findings of active pulmonary tuberculosis (e.g., tree-in-bud opacities or cavitation). These atypical features substantially complicated early diagnosis. On the basis of this case, we propose that clinicians should prioritize cerebral tuberculoma in the differential diagnosis for the following high-risk groups: (1) older patients with new-onset seizures (particularly with immunosenescence); (2) patients with intracranial space-occupying lesions without identifiable infectious sources; and (3) patients with suboptimal responses to antiepileptic drugs, even in the absence of tuberculosis exposure history or classic symptoms. Clinical decision-making should follow a three-tiered strategy comprising: (1) comprehensive history-taking and systemic examination (focusing on immune status and potential TB exposure risks); (2) multimodal neuroimaging (contrast-enhanced MRI, DWI/ADC analysis, and MR spectroscopy); and (3) timely acquisition of microbiological evidence (e.g., CSF GeneXpert assay) or histopathological confirmation (stereotactic biopsy). Establishing a stepwise diagnostic pathway integrating clinical vigilance, imaging screening, and histopathological confirmation has the potential to overcome traditional diagnostic limitations, thereby enabling early initiation of standardized antitubercular therapy to optimize neurological outcomes.

## 4. Conclusion

Cerebral tuberculoma remains a challenging diagnosis in older patients, especially in the absence of systemic or pulmonary tuberculosis. This case underscores the importance of integrating clinical suspicion, multimodal imaging features (including MRI with contrast enhancement and meningeal evaluation), and histopathological confirmation to establish an accurate diagnosis. Early surgical intervention followed by antitubercular therapy can lead to favorable neurological outcomes. Clinicians should consider tuberculoma in the differential diagnosis of intracranial mass lesions, even in non-endemic regions and in patients without typical tuberculous symptoms.

## Author profile

Zhou Zixuan, Year of Birth: 2000, Gender: Female, Ethnicity: Han, Hometown: Jiangsu, Academic Degree: Bachelor's Degree, Main Research Field: Medical Imaging;
Wang Shansham, Year of Birth: 1980, Gender: Female, Ethnicity: Han, Hometown: Shandong, Title: Chief Physician, Degree: Doctoral Graduate, Main Research Field: Imaging Radiomics.

## Disclosure statement

The authors declare no conflict of interest.

## References

[1] Verma S, Salotagi S, Angadi V, et al., 2024, Complicated Neurological Pathways: Tuberculoma With Guillain–Barré Syndrome – An Uncharted Clinical Odyssey. BMJ Case Reports, 17: e262702.

[2] Indoria A, Kulanthaivelu K, Prasad C, et al., 2024, Radiomics Features for the Discrimination of Tuberculomas From High-Grade Gliomas and Metastasis: A Multimodal Study. Neuroradiology, 66: 1979–1992.

[3] Koesbandono R, Muljadi R, Sutanto S, et al., 2024, Intracranial Tuberculomas: Review of MRI Findings and Clinical Features. Clinical Radiology, 79: 354–362.

[4] Spruijt L, Mosch A, Hoffmann C, et al., 2024, Mycobacterium bovis Infection of a Deep Brain Stimulation System Following Intravesical Bacillus Calmette–Guérin Instillation. Journal of Parkinson's Disease, 14: 1061–1069.

[5] Zhu G, Mo Y, Su Y, 2014, Clinical Characteristics of Intracranial Tuberculoma. Journal of Clinical Neurology, 27: 64–66.

[6] Yamamoto M, Manabe T, Yokokawa K, et al., 2023, Cerebral Tuberculoma Diagnosed by Nested Polymerase Chain Reaction of a Formalin-Fixed Paraffin-Embedded Brain Biopsy Sample. Internal Medicine, 62: 1827–1833.

[7] de Oliveira P, Braz Corbi M, Siqueira A, et al., 2023, Brain Tuberculoma in Pediatric Heart Transplant Recipient. Pediatric Transplantation, 27: e14496.

[8] Deng L, Diao S, Zhang Y, et al., 2023, Misdiagnosis of Early Cerebral Tuberculoma as Pneumonia: A Case Report. Academic Journal of Chinese PLA Medical School, 44: 823–826.

[9] Kalita J, Shukla R, Pandey P, et al., 2024, mRNA Profiling of Cytokines to Understand Paradoxical Response in HIV-Uninfected Tuberculous Meningitis. Tuberculosis, 144: 102463.

[10] Xia A, Wan J, Li X, et al., 2024, Rv0927c Suppresses the Activation of the HIF-1α Pathway Through VHL-Mediated Ubiquitination and NF-κB/COX-2 Pathway to Enhance Mycobacteria Survival. Microbiological Research, 278: 127529.

[11] Huang B, Zong M, Zong W, et al., 2016, Intracranial Rosai–Dorfman Disease. Journal of Clinical Neuroscience, 32: 133–136.

[12] Duoji Y, Zha D, Liu H, et al., 2023, Surgical Management of Cerebral Tuberculoma in Tibet Autonomous Region: A Report of 16 Cases With Literature Review. Chinese Journal of Clinical Neurosurgery, 28: 83–85.

[13] Leng F, Xu X, Kong Y, 2021, Nursing Care for Pediatric Patients With Idiopathic Hypertrophic Pachymeningitis. Journal of Nursing Science, 36: 40–41.

[14] Abrantes F, Moraes M, Rezende F, et al., 2020, A Clinical Approach to Hypertrophic Pachymeningitis. Arquivos de Neuro-Psiquiatria, 78: 797–804.

[15] Yonekawa T, Murai H, Utsuki S, et al., 2014, A Nationwide Survey of Hypertrophic Pachymeningitis in Japan. Journal of Neurology, Neurosurgery & Psychiatry, 85: 732–739.

[16] Wu S, Lin J, Ma Y, et al., 2020, Clinical Manifestations and Imaging Features of Patients With Hypertrophic Pachymeningitis. Chinese Journal of Clinical Research, 33: 633–637.

[17] Muhsen B, Aljariri A, Hashem H, et al., 2022, En-Plaque Sphenoid Wing Grade II Meningioma: Case Report and Review of Literature. Annals of Medicine and Surgery, 74: 103322.

[18] Mingo K, Sweeney A, Thompson R, et al., 2016, Hyperostotic En Plaque Meningioma Mimicking Fibrous Dysplasia of the Temporal Bone. Otology & Neurotology, 37: e317–e318.

[19] Gupta S, Mohindra S, Radotra B, et al., 2006, Giant Calvarial Hyperostosis With Biparasagittal En Plaque Meningioma. Neurology India, 54: 210–212.

[20] Chen S, Yang B, Li Y, et al., 2017, CT and MRI Features of Temporal Bone En Plaque Meningioma. Chinese Journal of Otology, 15: 454–457.

[21] Min J, Kang S, Lee J, et al., 2005, Hyperostotic Meningioma With Minimal Tumor Invasion Into the Skull. Neurologia Medico-Chirurgica, 45: 480–483.

[22] Chen J, Zheng J, Zhou Q, 2021, Exploratory Analysis of Risk Factors for Short-Term Prognosis in Patients With Tuberculous Meningitis. Journal of Hebei Medical University, 42: 1256–1260.

[23] Mayorga-Corvacho J, Vergara-Garcia D, Riveros W, et al., 2021, Tuberculoma in the Fourth Ventricle: An Unusual Location. Turkish Neurosurgery, 31: 658–660.