








Brain Tuberculoma as a Differential Diagnosis of Single Intracranial Lesion: Case Report

Tuberculoma cerebral como diagnóstico diferencial de lesão intracraniana única: Relato de caso

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Abstract

Tuberculosis (TB) of the central nervous system (CNS) is considered one of the most severe forms of presentation of the disease. Although only 1% of TB cases involve the CNS, these cases represent around between 5 and 15% of extrapulmonary forms.^{1,2} Tuberculous meningitis (TBM) is the most frequent form of CNS TB. The granulomas formed in the cerebral tuberculoma may cause hydrocephalus and other symptoms indicative of a CNS mass lesion. In the absence of active TB or TBM, the symptoms may be interpreted as indicative of tumors.^{3,4} The prognosis is directly related to the early diagnosis and proper treatment installation.⁵ We report the case of a patient with intracranial hypertension syndrome, expansive mass in the parieto-occipital region, accompanied by a lesion in the rib, initially thought to be a metastatic lesion, although posteriorly diagnosed as a cerebral tuberculoma.

Keywords

- ▶ brain tuberculoma
- ▶ metastasis
- ▶ diagnosis

Resumo

A tuberculose (TB) do sistema nervoso central (SNC) é considerada uma das formas mais graves de apresentação da doença. Embora apenas 1% dos casos de TB envolvam o SNC, isto representa aproximadamente entre 5 e 15% das formas extrapulmonares.^{1,2} A meningite tuberculosa é a forma mais frequente de TB do SNC. Os granulomas formados no tuberculoma cerebral podem causar hidrocefalia e outros sintomas indicativos de lesão com efeito de massa no SNC. Na ausência de TB ativa ou meningite tuberculosa, os sintomas podem ser interpretados como indicativos de tumores intracranianos.^{3,4} O prognóstico está diretamente relacionado ao diagnóstico precoce e instalação adequada do tratamento.⁵ Relatamos o caso de um paciente com síndrome de hipertensão intracraniana, formação expansiva em região parieto-occipital esquerda, acompanhada por lesão em arco costal, inicialmente tida como metástase, com posterior diagnóstico de tuberculoma cerebral.

Palavras-chave

- ▶ tuberculoma cerebral
- ▶ metástase
- ▶ diagnóstico

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Introduction

Tuberculosis (TB) of the central nervous system (CNS) has an incidence of 10 to 30%, predominating in developing countries.¹⁻³ The main route of dissemination of *Mycobacterium tuberculosis* is hematogenic, coming from a distant focus, usually the lungs.^{4,5} The usual patient is a child or young adult with headache, seizures, progressive motor deficits and/or signs and symptoms of elevated intracranial pressure. Most patients have no symptoms of systemic infection or signs of meningitis.⁶⁻⁹ In the absence of active TB or tuberculous meningitis (TBM), the symptoms of tuberculomas can be interpreted as indicative of tumors.^{10,11} Due to what has been described, we report the rare case of a patient with TB of the CNS, who presented an unusual clinical and radiological onset of symptoms.

Case Report

An 82-year-old male patient developed progressive left parieto-occipital headache, 3 months prior to admission, associated with visual agnosia and vertigo, evolving to worsening of the headache, associated with the previous symptoms. Beyond that, he did not present any alteration at

the general physical and neurological examination and was hospitalized for evaluation.

Brain magnetic resonance imaging (MRI) evidenced an expansive formation in the cortical left parieto-occipital region with apparent dural implantation and a thick and irregular peripheral impregnation by the contrast, suggesting parenchymal infiltration. The lesion measured $4.7 \times 4.1 \times 2$ cm, with extensive vasogenic edema in the adjacent parenchyma, midline shift to the right of 0.5 cm, and regular meningeal thickening (→ Fig. 1A-D). A computed tomography (CT) of the abdomen showed enlargement of the retroperitoneal lymph nodes and an unspecific renal lesion. Furthermore, a thoracic CT scan presented osteolytic lesion in one left rib. Added to swollen lymph nodes and the image findings, the primary hypothesis for the intracranial lesion was metastatic lesion, and the patient was referred for neurosurgical therapy.

The patient was submitted to resection of the brain lesion, through microsurgical technique. The postoperative CT scan confirmed complete resection of the lesion, with remaining adjacent edema at the lesion site. A follow-up MRI was performed 90 days after the surgery, confirming the excision of the lesion, as well as reduction in the preoperative edema (→ Fig. 1E-H). The histological study was negative for any neoplastic lesion, evidencing a chronic granulomatous

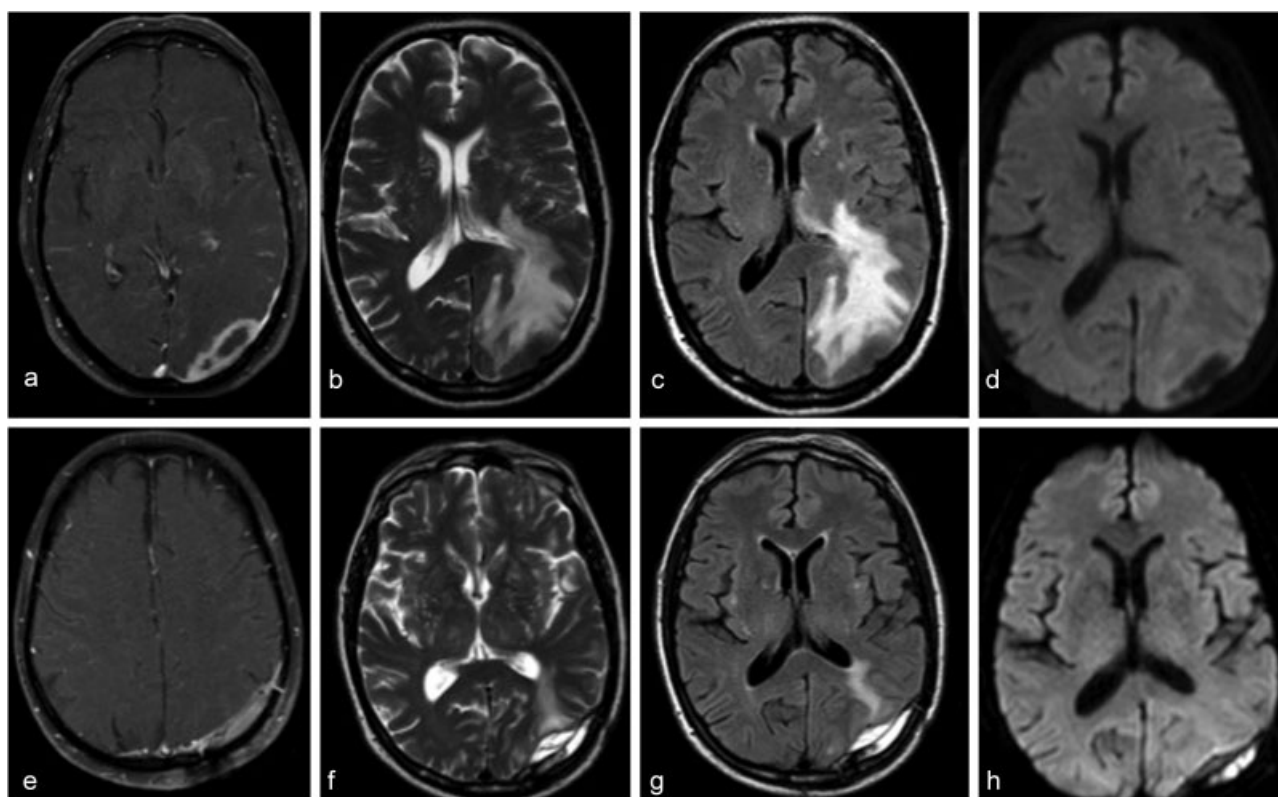


Fig. 1 (A) Preoperative T1-weighted magnetic resonance imaging (MRI) with gadolinium, axial incidence, evidencing an intradural lesion, attached to the dura mater, compressing the occipital lobe, with peripheral enhancement. (B) Preoperative T2-weighted MRI, axial incidence, confirming the important vasogenic edema of the intradural lesion. (C) Preoperative FLAIR MRI, axial incidence, highlighting the important vasogenic edema. (D) Preoperative diffusion MRI sequence, axial incidence, evidencing no restriction to water diffusion inside the lesion. (E) Postoperative T1-weighted MRI with gadolinium, axial incidence, evidencing markedly reduction in lesion size, with scar tissue in the operative field. (F) Postoperative T2-weighted MRI, axial incidence, confirming the findings on T1-weighted image. (G) FLAIR MRI, axial incidence, confirming important reduction of the vasogenic edema, when compared with the preoperative image. (H) Postoperative diffusion MRI sequence, axial incidence, evidencing only scar tissue in the side of the lesion.

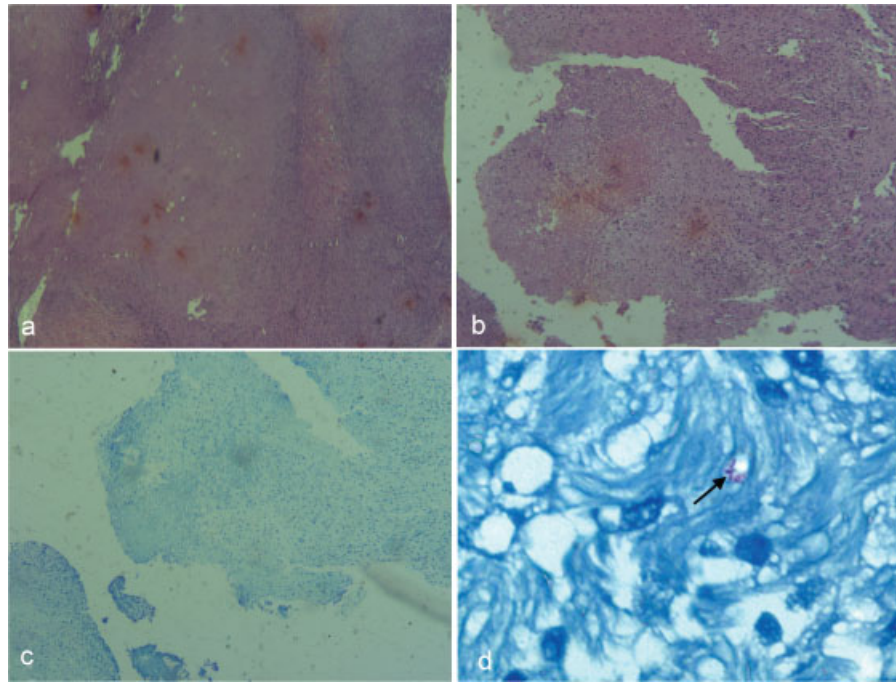


Fig. 2 (A) Hematoxylin & Eosin staining, 50x. Brain parenchyma with inflammatory process. (B) Hematoxylin & Eosin staining, 100x. Brain parenchyma confirming inflammatory process. (C) Wade staining, 50x. Brain parenchyma with granuloma. (D) Wade staining, 100x. *M. tuberculosis* bacilli evidenced in the left rib biopsy.

inflammatory process and caseous necrosis, characteristic of cerebral tuberculoma. Also, the biopsy performed in the rib lesion evidenced the presence of *M. tuberculosis* (► Fig. 2A-D).

Treatment with tuberculostatic agents (rifampicin, isoniazid and pyrazinamide) was then started. After 3 months, the patient presented a significant improvement of the whole clinical scenario, referring only occasional visual disturbance.

Discussion

Tuberculosis is among the most lethal infectious diseases worldwide. Primarily as a disease of the respiratory tract, CNS TB represents between 5 and 15% of extrapulmonary disease. These cases present the highest rates of morbidity and mortality.^{10,12-14} Central nervous system TB can be classified into three categories: tuberculous meningitis (TBM), spinal arachnoiditis, and cerebral tuberculoma, as the case presented.^{6,13}

Any recent or remote disseminated bacilleamia of TB can originate deep-seated tubercles in the brain and develop conglomerate caseous focus, known as tuberculomas. The lesions are formed by the interaction between the immune response of the host and the mycobacterial pathogen, covered with a fibrous encapsulation.⁶

These granulomas have the potential to cause all the symptoms of a CNS mass lesion, as hydrocephalus, headache, vomiting, drowsiness, papilledema, hemiparesis or seizures.^{6,10} They can present as focal neurological deficits without evidence of any systemic disease.^{15,16} Headache is among the most common symptoms, and in the case described, it was located in the left parieto-occipital region with progressive increase of its intensity, as observed in 80% of the cases.¹⁷

Regarding the lesion site, two thirds of the CNS tuberculomas are located in the cerebellum and one third in the cerebral hemispheres and, among the hemispherical locations, the predominance occurs in the frontal and parietal lobes.¹⁸ The differential diagnosis can include any mass lesion, such as malignant lesions, sarcoidosis, cysticercosis, toxoplasmosis and pyogenic abscess.^{15,16,19,20}

The correct diagnosis is important for an early start of the treatment and to achieve better outcomes. The imaging findings are not specific, which reinforces the need to consider the clinical presentation of the patient and the epidemiology of the region, which are enough to elaborate the presumptive diagnosis. On CT scan, a nodular enhancing lesion, with a central hypodense region is characteristic. On MRI, the early stage of focal cerebritis is marked by edema and poor-defined enhancement, while in the later mature stage, central hypointensity and peripheral enhancement on T2-weighted images are evidenced.^{4,6,21,22} Although the diagnosis can be made on the basis of clinical and epidemiological considerations, a needle stereotactic biopsy may be considered if necessary.⁶

It is not possible to differentiate granulomas from other brain expansive lesions on the basis of neurological symptoms, since these depend basically on the site and size of the lesion.^{11,23} Signs and symptoms of intracranial hypertension and seizures are frequent manifestations.^{24,25}

The usual presentation of a tuberculoma on a brain CT scan is a hypodense or slightly hyperdense nodule, representing the central area of caseous necrosis and an enhancing halo of epithelioid cells, surrounded by a hypodense area of edema, as shown in our case. The contrast can evidence the presence of multiple lesions and complications of tuberculous meningitis.

During the antibiotic treatment, serial CT scans show progressive regression of the size of the lesion.²⁶ None of the imaging methods, including brain MRI, can accurately differentiate tuberculomas from other intracranial masses.^{27,28} However, some authors have suggested that the presence of a central area of calcification (“target sign”) could be pathognomonic of tuberculomas.²⁹

Anti-tuberculous agents (ATTs) should be initiated in any case with important clinical suspicion, rather than delayed until the confirmed diagnosis. The four first line ATTs include isoniazid, rifampicin, pyrazinamide, and ethambutol or streptomycin. The duration of the treatment ranges from 9 to 12 months, which can be extended beyond the resolution of the symptoms and of the radiographic lesions. The Bacillus Calmette-Guérin (BCG) vaccination reduces the incidence of a new tuberculoma.¹⁰

Neurosurgical evaluation should be performed immediately in patients with elevated intracranial pressure (ICP).⁶ Tuberculomas may require surgical resection to treat the mass effect, since immediate removal offers improvement in the overall outcome, including cases of drug resistance or paradoxical worsening, an event that occurs in up to 25% of treated TB patients. The surgical approach can vary, due to the location tuberculoma of the surrounding vascular, nerve, and cortical structures, and the preference of the surgeon.¹⁰

Besides that, lumbar puncture can alleviate elevated ICP in communicating hydrocephalus, while ventriculoperitoneal shunting may be the most appropriate treatment for non-communicating hydrocephalus.¹⁰ Moreover, steroids are useful when cerebral edema produces altered mental status or focal neurologic deficits. Finally, the prognosis is better when treatment begins before the development of focal neurologic signs and altered state of consciousness.⁶

Conclusion

Cerebral tuberculoma prognosis has improved dramatically during the last several decades, from a nearly fatal diagnosis to over 80% survival with timely treatments. Awareness of tuberculoma must be maintained when assessing CNS masses, including patients with no history of tuberculosis disease elsewhere, to ensure appropriate treatment.^{10,13}

Conflict of Interests

The authors have no conflict of interests to declare.

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