Intracranial tuberculomas. A case report

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Background:
The increasing incidence of human immunodeficiency virus and drug-resistant variants has led to an increase in the incidence of tuberculosis (TB) globally. Annually, approximately 9 million cases of tuberculosis are identified globally. Among all manifestations of tuberculosis, the central nervous system constitutes about 10% of all situations and represents the highest mortality rate (1). Tuberculoma is the most common lesion in tuberculosis of the central nervous system, which could be located in any area of the intracranial space. The lesion can be unilateral or multifocal and can be observed with or without manifestation of meningitis (2).

Keywords: Tuberculoma, tuberculosis, central nervous system.

Case Report

Radiology

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The increasing incidence of human immunodeficiency virus and drug-resistant variants has led to an increase in the incidence of tuberculosis (TB) globally. Annually, approximately 9 million cases of tuberculosis are identified globally. Among all manifestations of tuberculosis, the central nervous system constitutes about 10% of all situations and represents the highest mortality rate (1). Tuberculoma is the most common lesion in tuberculosis of the central nervous system, which could be located in any area of the intracranial space. The lesion can be unilateral or multifocal and can be observed with or without manifestation of meningitis (2).

Case report

We present the case of a 57-year-old Guatemalan male patient with a medical history of being HIV positive for 20 years with weight loss of 1 year of evolution accompanied by fever, productive cough, headache, and behavioral changes for three weeks, as well disorientation and later seizure status. Upon physical examination, a disoriented patient was observed with deep tendon reflexes II/IV, muscle strength IV/V, Glasgow of 13 points and positive Kernig sign. Subsequently, a contrast-enhanced computed tomography of the brain was performed, showing ill-defined hypodensities predominantly on the right hemisphere, associated with poor differentiation of gray-white matter at this level, as well, the presence of two oval isodense images in simple phase, which presented slight enhancement in a ring and perilesional edema after the administration of intravenous contrast medium.

A brain MRI was performed in T1, T2, FLAIR, DWI, ADC and T1 C+ (Gd) sequences, showing multiple nodules in the cerebral, cerebellar and brainstem parenchyma, of diffuse distribution, involving cortical and subcortical gray matter, corpus callosum and basal ganglia. The imaging findings described above are associated with CD4 lymphocyte count less than 100 cells/mm3 and positive Ziehl Neelsen staining of the cerebrospinal fluid (CSF); these findings guide us to the diagnosis of emerging tuberculomas due to intracranial tuberculosis.

Discussion

Tuberculosis is prevalent globally. A devastating expression of extrapulmonary tuberculosis manifests in the central nervous system (CNS), as tuberculous meningitis (TBM), CNS tuberculoma, or spinal arachnoiditis. Central nervous system tuberculosis (CNS-TB) can arise through blood dissemination or elimination of subependymal or meningeal tuberculous foci. This disease represents the highest morbidity and mortality among all variants of tuberculosis (3). The most prevalent type of cerebral parenchymal TB are parenchymal

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tuberculomas. They are generated by the aggregation and coalescence of tubercular microgranulomas, which typically arise at the grey-white matrix junction as a result of the haematogenously disseminated microorganisms being arrested by a decrease in the caliber of arteries in that area. On occasion, lesions may form in the brain's parenchyma as a result of a CSF infection spreading through the perivascular (Virchow Robin) gaps. Nonetheless, they can arise in nearly every part of the brain, including the ventricular system, brainstem, cerebellar hemispheres, sulcal spaces, and basal cisterns (4). On unenhanced CT scans, tuberculoma may present as isodense, hyperdense, or combined density. On the other hand, an enhanced CT scan may show a ring-shaped or less likely enhancement pattern, such as a nodular or irregular area with uniform enhancement. The diagnosis indicates a central nidus of calcification with an enhancement in the surrounding annulus, also known as the objective sign. Non-enhanced MR investigations show a varied lesion, mostly of low signal intensity, with a central area of high signal intensity and high signal intensity edema on T2-weighted or FLAIR images (2).

Conclusion

For a prompt and accurate diagnosis of suspected CNS-TB patients, neuroimaging is essential. It is optimal for neuroimaging to encompass the complete neuroaxis. Both magnetic resonance imaging (MRI) and brain computed tomography (CT) are used as evaluation modalities; contrast-enhanced MRI is better at drawing boundaries. More recent methods significantly improve the diagnostic accuracy of MRI in unusual or challenging situations (3). This disease can present with non-typical radiologic signs that can occasionally be confused with other lesions, like brain tumors. To reduce the morbidity and mortality of this potentially fatal illness, radiologists and infectious disease specialists must be familiar with the varied imaging presentations of CNS tuberculosis in order to diagnose the condition promptly (2).

Conflicts of interests

There was no conflict of interest during the study, and it was not funded by any organization.

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- To the memory of the patient because his case serves as teaching through this publication.
- To Hospital Regional de Occidente for giving us the necessary supplies in the radiology department to make the diagnosis.

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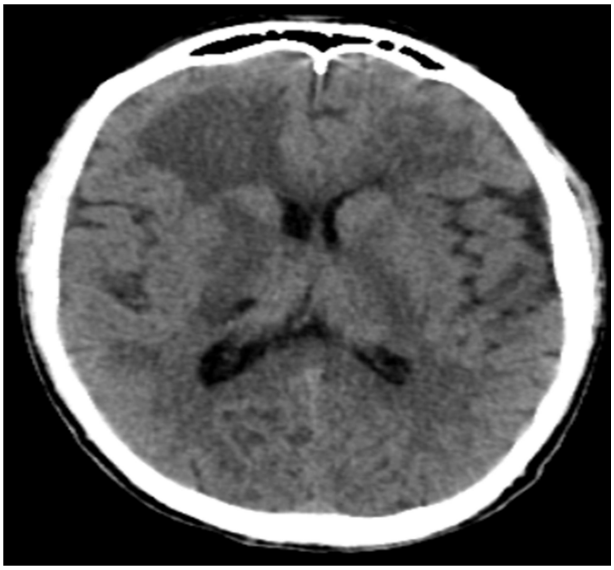

Figure 1. At the level of both frontal lobes, ill-defined hypodensities are observed with attenuation coefficients of up to +22 uH with a predominance on the right.

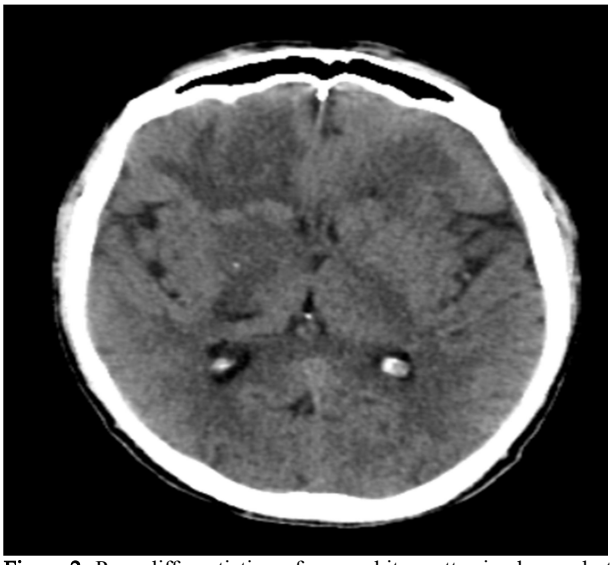

Figure 2. Poor differentiation of gray-white matter is observed at this level, inside the presence of two oval images, isodense in simple phase, with attenuation coefficients of up to +32 uH.

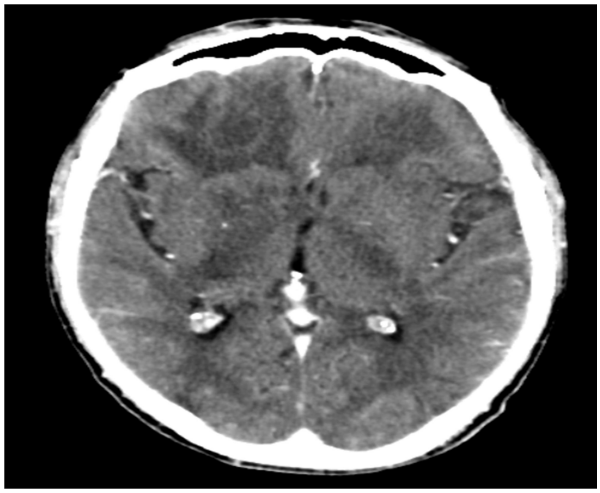

Figure 3. The contrast phase demonstrates slight ring enhancement after the administration of intravenous contrast medium up to +38 uH, associated with perilesional edema; The one on the right side measures 2.2 x 2.2 cm and the one on the left side measures 1.4 x 1.2 cm. Likewise, hypodensity is observed at the level of the right basal ganglia and thalamus.}

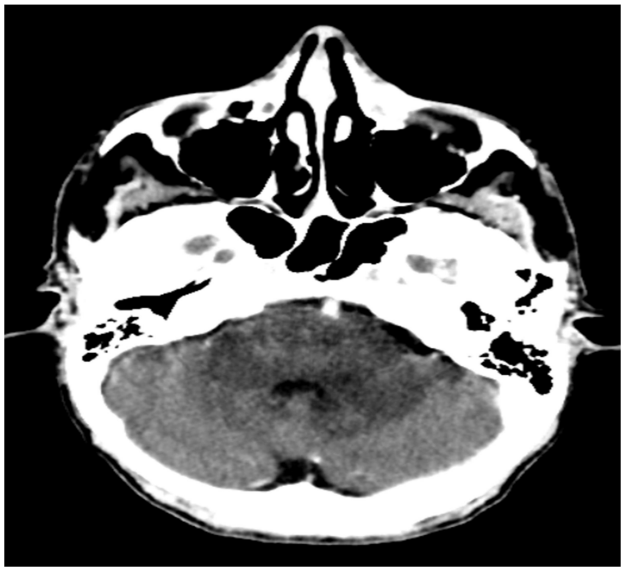

Figure 4. Hypodensity of the brain stem and cerebellar hemisphere.

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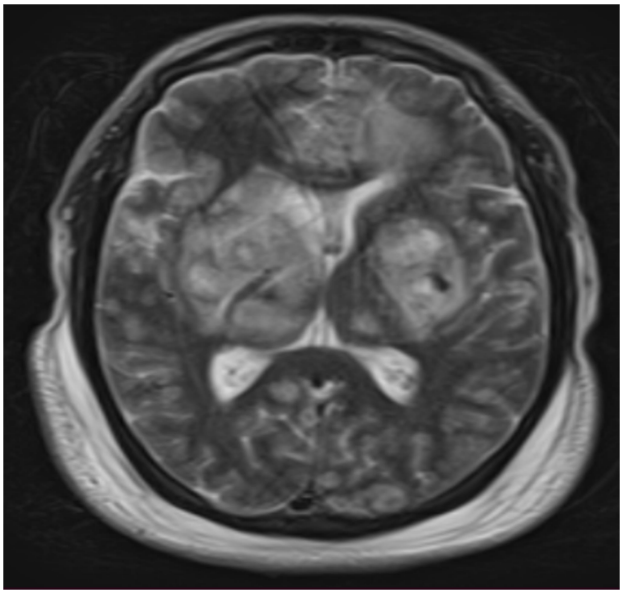

Figure 5. In the T2 sequence, hypointense foci are observed in the basal ganglia that may correspond to thick calcifications: in the basal ganglia we see two hyperintense foci on the right side and one on the left side that suggest a petechial hemorrhagic component, since they measure an average of 5.0 mm.

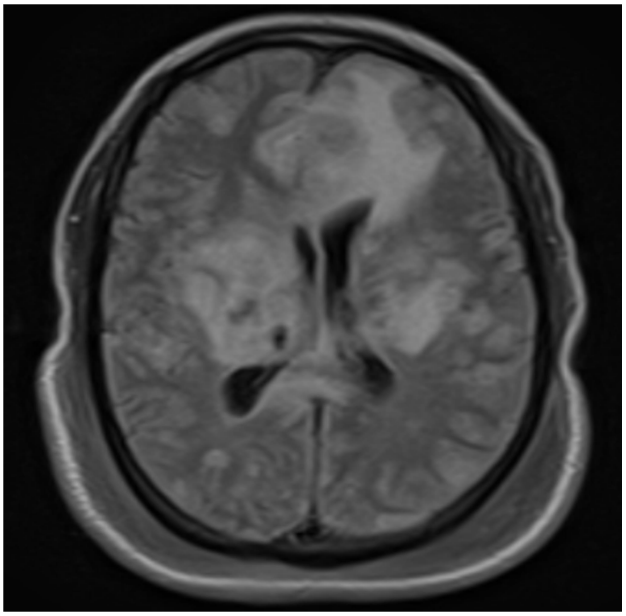

Figure 6. In the FLAIR sequence, swelling of the basal ganglia is observed, predominantly on the right, causing compression on the lateral ventricles and especially the third ventricle, which is causing dilation of the occipital horns. These nodules vary in size between 6 and 15 mm.

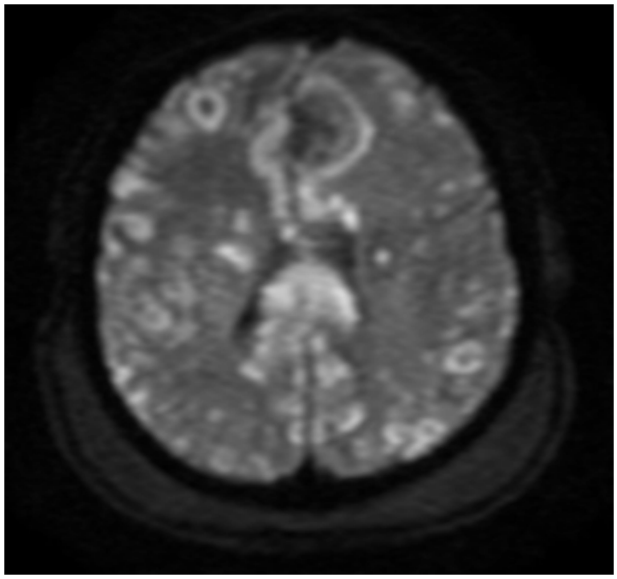

Figure 7. In the DWI phases, restriction to ring diffusion is observed, with a larger one measuring 3.4 x 1.8 cm, which generates a mass effect, located in the left frontal lobe on the midline, which moves 8 mm.

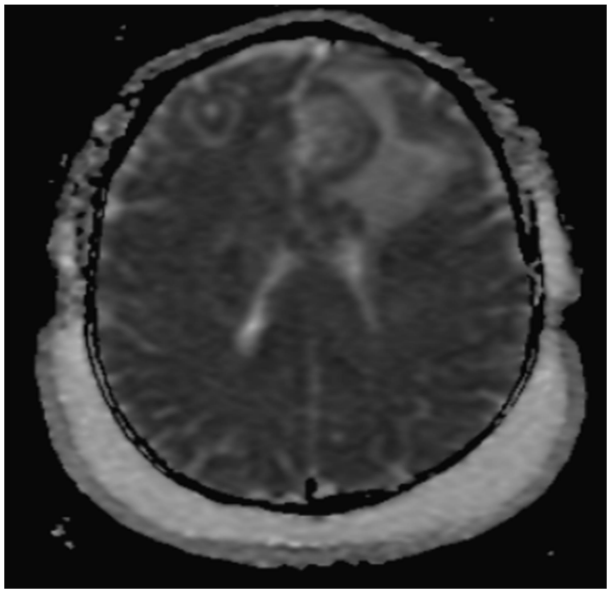

Figure 8. In the ADC sequence, a mass effect is observed, located in the left frontal lobe on the midline, which moves 8 mm, associated with vasogenic edema, with approximate measurements of 5.3 x 4.9 cm.

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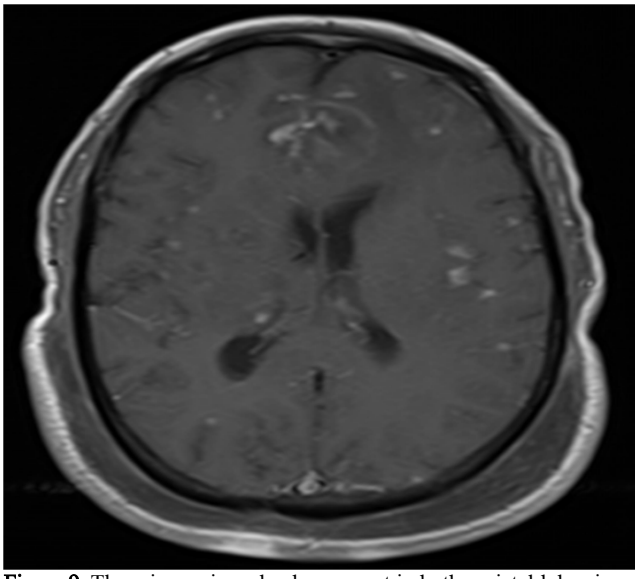

Figure 9. There is meningeal enhancement in both parietal lobes in the contrast phase, although no thickening is observed.

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