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Egyptian Journal of Neurosurgery (2023) 38:36
https://doi.org/10.1186/s41984-023-00216-x

Egyptian Journal of Neurosurgery

CASE REPORT

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Large intracranial supratentorial tuberculoma imitating as a malignant lesion: a case report

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Abstract

Background CNS TB roughly correlates to 1–10% of all intracranial tumors. The spectrum of CNS-TB is wide and can present as tuberculous meningitis, tubercular abscess, tuberculoma, or hypertrophic pachymeningitis. Tuberculomas are usually located at the corticomedullary junction and periventricular region, as expected for hematogenous dissemination. They are mostly infratentorial in children and supratentorial in adults. They may present months to years after infection.

Observations Here we have a 55-year-old female who presented with a history of headaches, sudden loss of consciousness, and seizures. Initial imaging showed a right parietal mass lesion appearing to be malignant on initial imaging which had a significant mass effect and midline shift. The patient was operated on for the same and histopathology showed chronic granulomatous lesion and tissue staining s/o tuberculosis.

Lessons So a diagnostic option for infective etiology should be kept even for an intracranial supratentorial mass lesion.

Keywords Intracranial, Tuberculoma, Malignant, Supratentorial, Case reports

Background

According to the 2017 Global Tuberculosis Report, India had an estimated 2,800,000 cases of TB or nearly 25% of all cases worldwide. Of those, 15,000 cases were multidrug-resistant, with a mortality rate of 42,000 people [1]. The incidence and prevalence of central nervous system-TB (CNS-TB) are unknown but roughly account for 1–10% of all TB cases [2–4]. It is generally acknowledged that TB bacilli transmit hematogenously to the central nervous system (CNS). To control the infection, granuloma (Rich foci) form around the bacilli; when these foci burst into the subarachnoid space, the result is a severe inflammatory response that leads to TBM [5]. Interferon-gamma (IFN-gamma) and tumor necrosis factor-alpha (TNF-alpha) are crucial mediators in these occurrences. The clinical manifestations of TBM are chiefly due to this inflammation, and hence, dexamethasone is useful as an adjuvant in TBM but not in tuberculoma. [6] Tubercular meningitis and tuberculomas are the two most important clinical manifestations, the result of the hematogenous dissemination of M. tuberculosis. The potentially fatal condition known as intracranial tuberculoma is difficult to identify clinically and radiologically, and it frequently resembles other expansive lesions. [7, 8]Hence, this case becomes important as the preoperative investigations suggested a malignant lesion, whereas post-operative histopathology confirmed a tuberculous lesion.

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Case presentation

The patient was a 65-year-old Indian woman from Rajasthan with no previous medical history. She came to us with a progressive severe headache and an episode of sudden loss of consciousness and seizures from which she recovered spontaneously. The local hospital, a referral center, ordered an MRI that showed a large right parietal mass lesion with associated vasogenic edema and mass effect, and midline shift. The patient was alert, oriented to place and person, with a Glasgow score of 15/15 isochoric and isoreactive pupils with no evidence of any focal deficit. Muscle strength of 5/5 was seen in all four limbs. She had no associated comorbidities and the blood picture was normal. A metastatic lesion or a high-grade glioma were thought to be the possibilities. No evidence of a primary lesion or primary tubercular origin was seen in the preoperative workup. A cortical-subcortical lesion was found during craniotomy and gross total excision was done. The pathology department then observed granulomatous inflammation in the excised tissue and on ZN staining Tuberculosis was confirmed. The postoperative period was uneventful and she was started on Anti tubercular treatment according to the Revised National Tuberculosis control program (Figs. 1, 2).

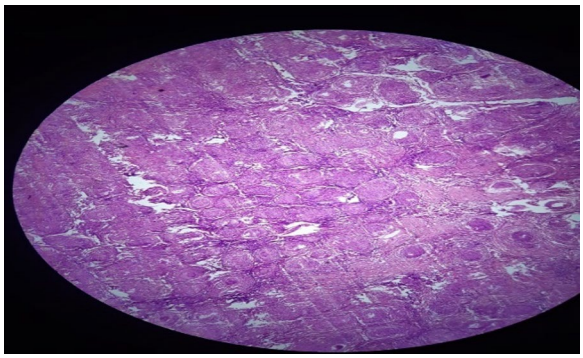

Fig. 2 Shows Langerhans cells with lymphocytic infiltration s/o tuberculosis

Discussion

CNS tuberculosis is very common in the Indian subcontinent. CNS TB need not always manifest as a sequela of pulmonary TB. Less than 5% of patients with pulmonary TB develop CNS TB and not all patients with CNS TB have pulmonary TB. Lin et al. in their series observed that only 3% of pulmonary TB patients had extrapulmonary TB, and only 19.6% of these patients had pulmonary TB [9]. Interestingly, pulmonary TB is more common among males, whereas extrapulmonary TB affects females more often. CNS TB spares no age group but immunocompromised individuals are at a higher risk. [10] Cortical lesions may present with seizures, large conglomerate lesions may present with raised ICP, and focal deficits, and Infratentorial tuberculomas may present with brainstem syndromes, cerebellar symptoms, multiple cranial nerve palsies, and long tract signs. Seizures in IT could be early (< 1 month) or delayed (> 1 month). Early seizures are usually due to meningeal irritation, whereas delayed seizures are primarily due to tuberculoma, hyponatremia, or infarct. If the tuberculoma is big and situated in a non-eloquent region of the brain, features of elevated ICP without localizing impairments may be visible. In the absence of tubercular meningitis or pulmonary tuberculosis, intracranial tuberculomas (IT) and tubercular abscesses (TA) can develop. It is difficult to distinguish tuberculomas and tubercular abscesses in these patients from other common mass lesions and pyogenic/fungal abscesses. CSF analysis and imaging findings are often equivocal in most patients with IT and TA and diagnostic

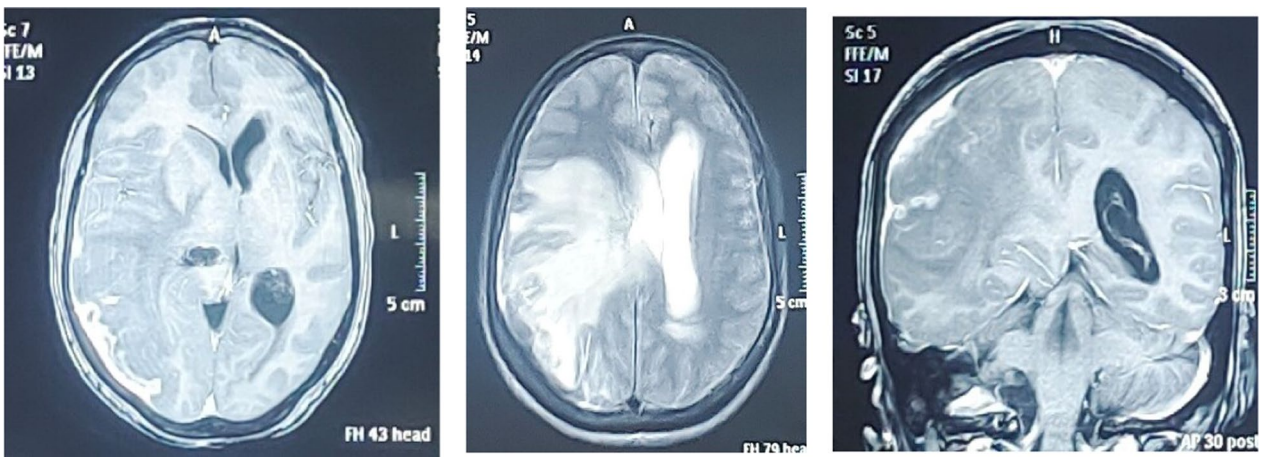

Fig. 1 Shows the right parietal diffuse lesion with a mass effect

confirmation may require histological or microbiological proof. Performing surgery for tissue diagnosis in deep-seated, eloquent area lesions and patients with other morbidities is challenging.

In this case, surgery was undertaken assuming it was a malignant tumor even though there were no clinical or radiological signs of tuberculoma and it was later identified on histopathology to be tuberculoma. This case serves as an example of the nonspecific cerebral tuberculoma presentation. To diagnose cerebral tuberculosis, one should consider this, especially in nations like India where tuberculosis incidence is high. The lesion at the cerebral parenchyma or meninges level may become active years after the infection.

Conclusion

Many times, intracranial lesions are thought to be malignant and are removed surgically. Even in the absence of pulmonary symptoms, the possibility of CNS tuberculosis should always be kept open, particularly in a country like India where tuberculosis prevalence is so high. Using ZN staining and CSF culture to commence ATT quickly and minimize morbidity is best.

Abbreviations

CNS	Central nervous system
TB	Tuberculosis
TBM	Tuberculous meningitis
ZN	Ziehl Neelsen
IT	Intracranial tuberculoma
TA	Tubercular abscess
ATT	Antitubercular treatment
CSF	Cerebro spinal fluid
MRI	Magnetic resonance Imaging

Acknowledgements
Not Applicable

Author contributions
SV collected the data and wrote the manuscript and JS did the proofreading and advised on corrections.

Funding
Not Applicable.

Availability of data and material
Not Applicable.

Declarations

Ethics approval and consent to participate
Not Applicable.

Consent for publication
Consent for publication from the patient's guardians was taken.

Competing interests
The authors declare that they have no competing interests.

Received: 30 March 2023 Accepted: 17 June 2023
Published online: 31 August 2023

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