The Infectious Cloak: How Tuberculosis Masked a Primary CNS Lymphoma

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AIMS

To present a challenging case of Primary Central Nervous System Lymphoma (PCNSL) initially misdiagnosed as central nervous system tuberculosis (CNS TB) in a patient with a history of pulmonary TB. This case aims to highlight the critical diagnostic pitfalls, underscore the limitations of empirical therapy, and emphasize the necessity of early histopathological confirmation in mass-forming brain lesions, especially in TB-endemic regions.

Case history

Here we present a case of 40-year-old male with a known history of pulmonary tuberculosis, diagnosed six months prior. He defaulted on his anti-tubercular treatment (ATT) after completing only three months. He presented with acute-onset weakness in the right upper and lower limb with deviation of angle of mouth to left side with confusion associated unable to talk and was unable to comprehend commands.
On examination Patient had right hemiparesis , right UMN facial palsy with global aphasia .

Methods

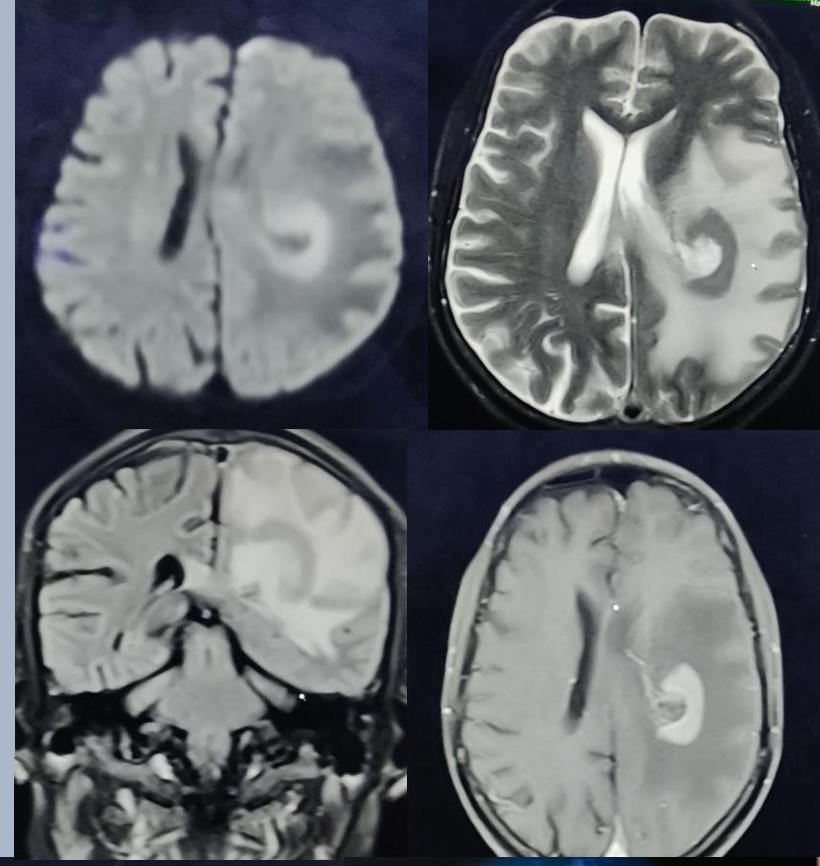
1. Initial Clinical Assessment & Working Diagnosis: The patient's acute neurological deficits and history of defaulted TB treatment led to a high suspicion of a CNS tuberculoma with vasogenic edema, as seen on initial CT brain.

2. Empirical Therapeutic Trial: Based on the working diagnosis, empirical ATT and corticosteroids were initiated.

3. Advanced Diagnostic Workup: Due to the space-occupying lesion's characteristics on MRI (thick, smooth peripheral enhancement, significant edema, mass effect) and intense metabolic activity on PET-CT, the diagnosis of PCNSL was suspected despite initial clinical improvement.

4. Definitive Diagnosis: A stereotactic brain biopsy was performed, which is the gold standard for diagnosing PCNSL .

5. Staging and Co-morbidity Management: PET-CT and bone marrow biopsy were used for staging, confirming the disease was confined to the CNS. ATT was continued for the active pulmonary TB.



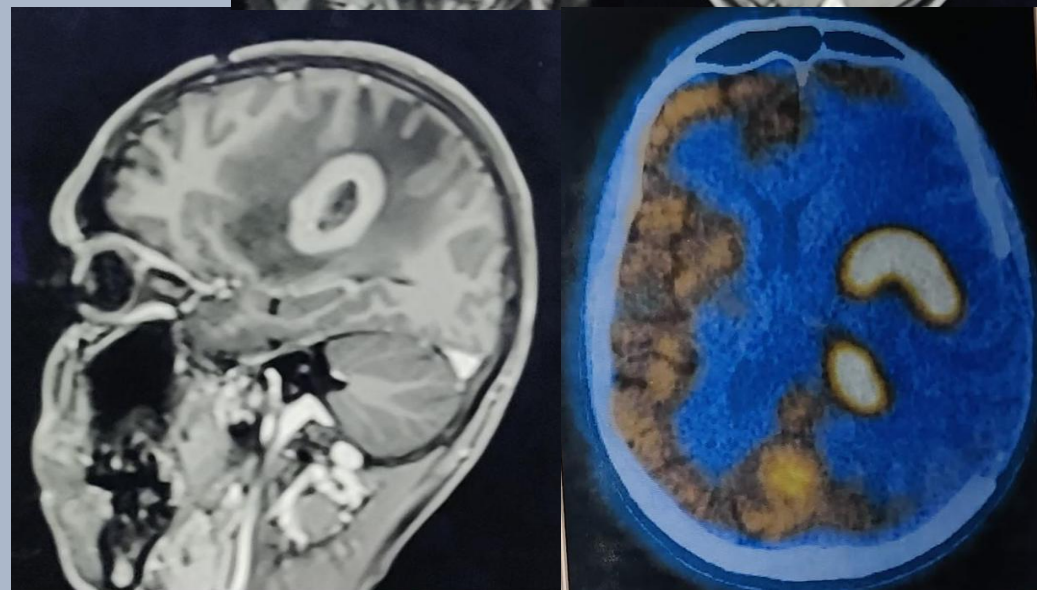
Results

Deceptive Initial Response: The patient showed significant neurological improvement within 48 hours of starting corticosteroids, a known lympholytic agent, which misleadingly supported the inflammatory/infectious hypothesis.

Definitive Histopathology: Brain biopsy confirmed Diffuse Large B-Cell Lymphoma (DLBCL), the most common subtype of PCNSL (Grommes et al., 2018). Immunohistochemistry showed an ABC/non-GC phenotype (MUM1+, Bcl6+, CD10-), typical for PCNSL, with a high proliferative index (Ki-67 90%).

Final Diagnosis: Primary CNS Lymphoma (DLBCL) with co-existing active Pulmonary Tuberculosis.

Treatment Initiated: Combination chemotherapy with R-MVP regimen (Rituximab, high-dose Methotrexate, Vincristine, Procarbazine) was initiated alongside a modified ATT regimen.



Discussion

This case epitomizes a critical diagnostic challenge in neurology: the uncanny ability of Primary Central Nervous System Lymphoma (PCNSL) to masquerade as infectious pathology. Our patient's presentation—an acute neurological deficit in a known tuberculosis defaulter with a ring-enhancing brain lesion—initially pointed strongly towards CNS tuberculoma. This diagnostic pitfall is particularly treacherous in TB-endemic regions, where clinical suspicion naturally leans towards common pathogens. The initial dramatic response to corticosteroids provided a false sense of diagnostic confirmation, as steroids cause rapid lympholysis in PCNSL, mimicking a response to anti-inflammatory therapy for infection.

This narrative of diagnostic confusion is powerfully echoed in the literature. The case by Tai et al. (2020) presents an almost identical scenario, where a patient with microbiologically proven disseminated TB subsequently progressed on anti-TB therapy, requiring a repeat brain biopsy to ultimately diagnose PCNSL. This underscores a vital lesson: the confirmation of one serious disease (TB) does not preclude the presence of another.

Finally, the work of Dres et al. (2012) reminds us that the relationship between TB and lymphoma may be more than mere mimicry; it can be one of coexistence, potentially fueled by lymphoma-induced immunosuppression. This justifies a high index of suspicion for malignancy when a "proven" TB exhibits an atypical course. The definitive diagnosis in our case, as in all literature, was achieved not through imaging alone but via histopathological examination of the brain lesion—the unequivocal gold standard.

Conclusion:

PCNSL can be a great mimicker, especially in TB-endemic areas. A history of TB should not preclude the consideration of lymphoma in patients with atypical or mass-forming CNS lesions. Early neuroimaging, a high index of suspicion, and timely biopsy are paramount. This case demonstrates that an initial response to steroids can be deceptive and should not delay definitive histopathological diagnosis, which is crucial for guiding appropriate chemotherapy and improving patient outcomes.

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