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Case Report

Tuberculous hemorrhagic meningoencephalitis. A rare manifestation of miliary TB. Case report and review ☆

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ABSTRACT

Tuberculosis (TB) remains a significant global health challenge, ranking as the second leading cause of death from an infectious disease. Central nervous system (CNS) TB, although rare, accounts for approximately 1% of all TB cases and can manifest as tuberculous meningitis, tuberculoma, abscess, or less commonly, hemorrhagic meningoencephalitis. We report a case of hemorrhagic meningoencephalitis secondary to TB, a rare but serious complication.

Introduction

Tuberculosis (TB) is a relatively widespread infectious disease caused by the mycobacterial species *Mycobacterium tuberculosis* (M. *tuberculosis*) [1]. It ranks as the second leading cause of death globally from a single infectious disease, surpassed only by COVID-19 [2]. It was estimated that more than 1.7 billion people are infected with M. *tuberculosis* and in 2021 alone, the global incidence of TB was 10.6 million [1].

About 90% of people infected with M. tuberculosis develop latent TB, where the immune system is able to contain the initial infection, preventing its spread. As a result, the bacteria remain dormant in the lungs, causing minimal or no symptoms [3]. In about 5% of cases, the immune system fails to control the initial infection, leading to primary TB. Another 5% successfully contain the infection initially, but over time, the dormant bacteria may reactivate due to certain risk factors, resulting in postprimary TB [3]. Miliary (disseminated) TB occurs when the uncontrolled infection spreads through the bloodstream, reaching other organs and significantly increasing the risk of mortality [3]. Risk factors for developing miliary TB and eventually CNS TB include age (with children being more susceptible than adults), being in an immunocompromised state whether from HIV co-infection or the use of immunosuppressive medications, malnutrition, alcoholism, recent measles in children, malignancies, and the general prevalence of TB in the community [4]. We present a case of miliary TB

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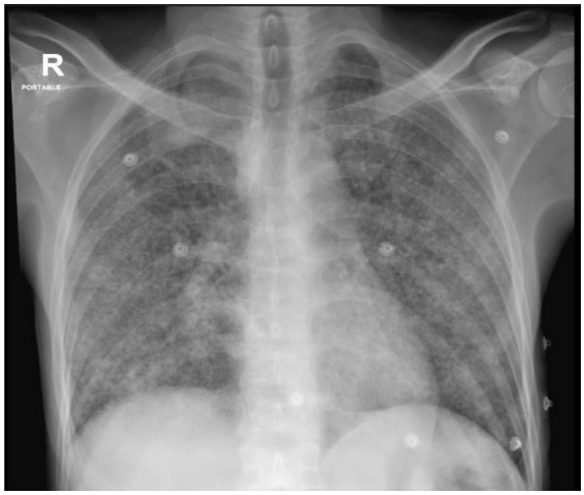

Fig. 1 – AP portable chest radiograph taken on admission displays extensive bilateral miliary nodules, suggestive of miliary TB.

in a 39-year-old male with no previous medical history who developed a rare CNS manifestation of TB hemorrhagic meningoencephalitis.

Case presentation

A 39-year-old male with no significant past medical, surgical, or family history, presented to the Emergency Department with a 2-month history of persistent fever, a 15 kg weight loss, decreased appetite, night sweats, dry cough, and shortness of breath. The patient had no history of recent travel or contact with sick individuals but lived in close quarters with 13 other inmates. Initial examination revealed tachycardia, hypotension responsive to fluids, and fine tremors. A chest radiograph showed bilateral miliary nodules, raising strong suspicion for miliary tuberculosis (TB). Laboratory results revealed hyponatremia, elevated liver enzymes (AST, ALT, ALP), and elevated inflammatory markers (Fig. 1).

Given the clinical picture, the patient was admitted under infectious disease and internal medicine teams, placed in isolation, and started on empirical antibiotics (Tazocin) while a TB workup was initiated, including sputum cultures and smears. An initial CT scan of the chest, abdomen, and pelvis revealed widespread pulmonary miliary nodules, cavitations, and mediastinal lymphadenopathy, highly suggestive of miliary TB. With his complaints of headache, an MRI brain was ordered to evaluate for CNS involvement.

Initial MRI of the brain revealed evidence of vasogenic edema, intraparenchymal hemorrhage and leptomeningeal enhancement of the left posterior insula and temporoparietal regions. These features are suggestive of hemorrhagic; possibly secondary to tuberculosis. The patient was immediately started on standard anti-TB therapy (isoniazid, rifampin, ethambutol, pyrazinamide) along with oral corticosteroids (dexamethasone) to address the CNS involvement.

During his hospital course, the patient's symptoms gradually improved, though he remained tachycardic, with persistent liver enzyme elevation and hyponatremia. His repeat imaging, including a CT thorax, demonstrated a slight reduction in cavitary lesions but persistence of diffuse miliary nodules. Serial chest radiographs showed no significant changes. Follow-up for the brain findings was scheduled.

Follow up MRI of the brain revealed progression of the leptomeningeal disease, with worsening nodular and cystic rim enhancement in the left insular fissure and adjacent areas. New area of left frontal vasogenic edema developed. However, there was partial resolution of the previously noted peri–insular edema. Despite the imaging worsening, the patient remained clinically stable, with improved headache symptoms, and was maintained on anti-TB treatment.

Over the subsequent months, the patient continued to improve clinically, with normalized liver function tests and resolution of his hyponatremia. A final MRI brain, approximately a year after admission, showed significant resolution of the left frontal-temporal leptomeningeal disease, with residual ring-enhancing lesions. Given the slow response to therapy, the patient's TB treatment was extended to a total of 15 months, with a plan for close follow-up.

Overall, the patient's clinical course highlights a gradual improvement in both systemic and CNS tuberculosis, with significant imaging improvement of the brain lesions despite early signs of progression on imaging (Figs. 2–6).

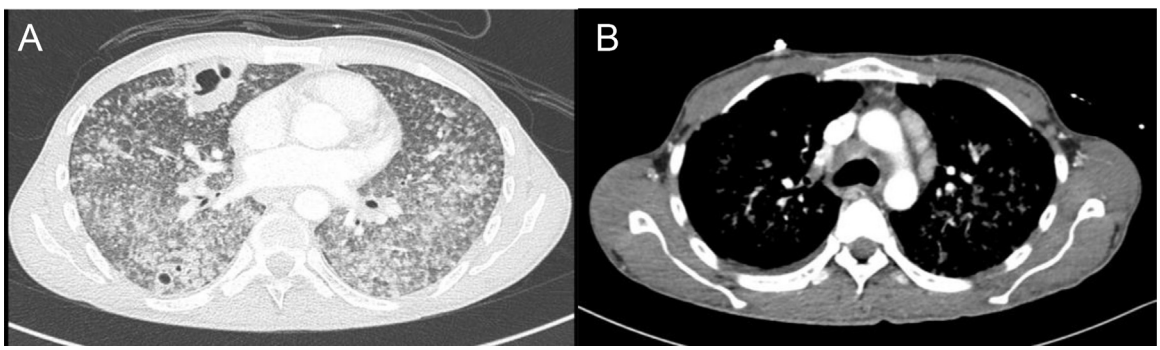

Fig. 2 – Initial CT chest shows (A) numerous, innumerable widespread tiny nodules scattered across both lung parenchyma with few thick-walled cavities bilaterally, (B) Mediastinal window shows multiple enhancing mediastinal lymph nodes.

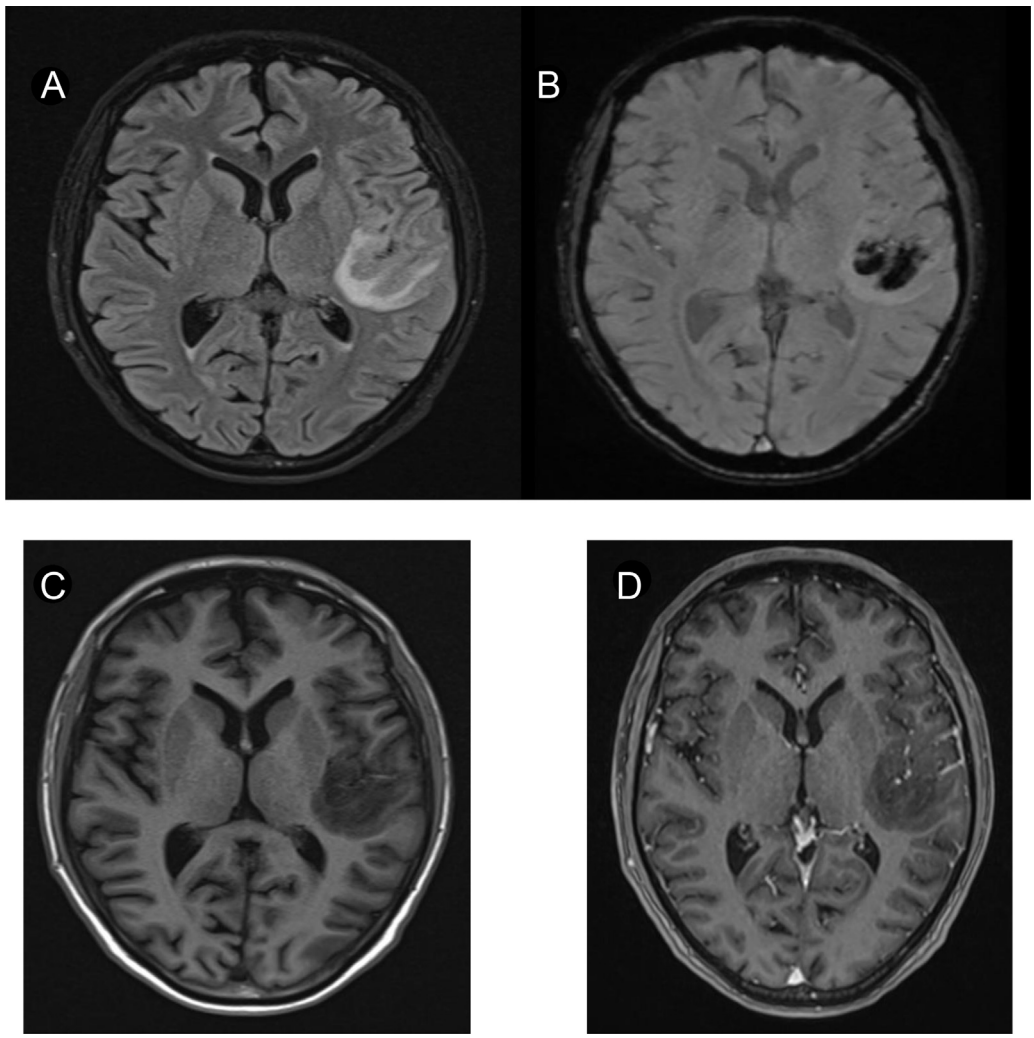

Fig. 3 – Initial MRI brain. (A) T2 FLAIR shows an area of high T2 and FLAIR signal at the subcortical white matter of the left posterior insula and temporoparietal regions; consistent with vasogenic edema. (B) The same area shows dark SWI blooming consistent with intraparenchymal hemorrhage. (C) T1 weighted image pre contrast. (D) T1 weighted image post contrast shows overlying leptomeningeal enhancement and engorged vessels within the pathological area.

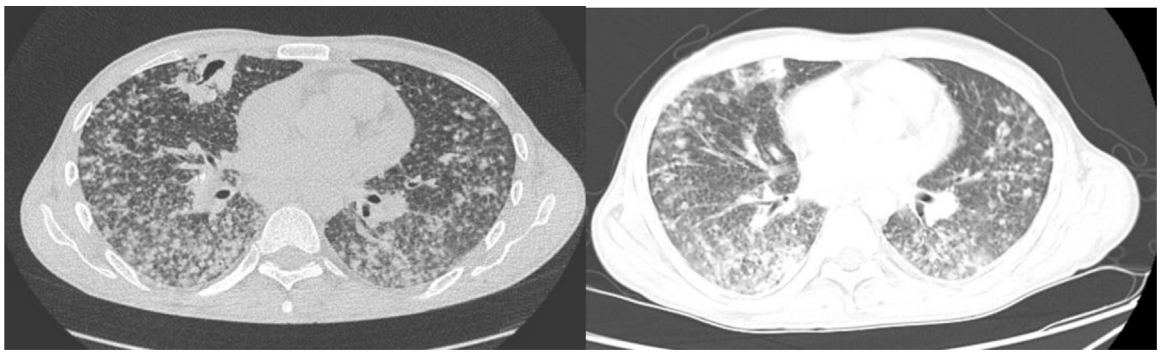

Fig. 4 – Subsequent follow-up CT scan of the chest (pulmonary window) taken within 1 month of each other show slightly improved appearance of cavitary lesion in the anterior segment of right upper lobe and persistent widespread pulmonary nodules bilaterally.

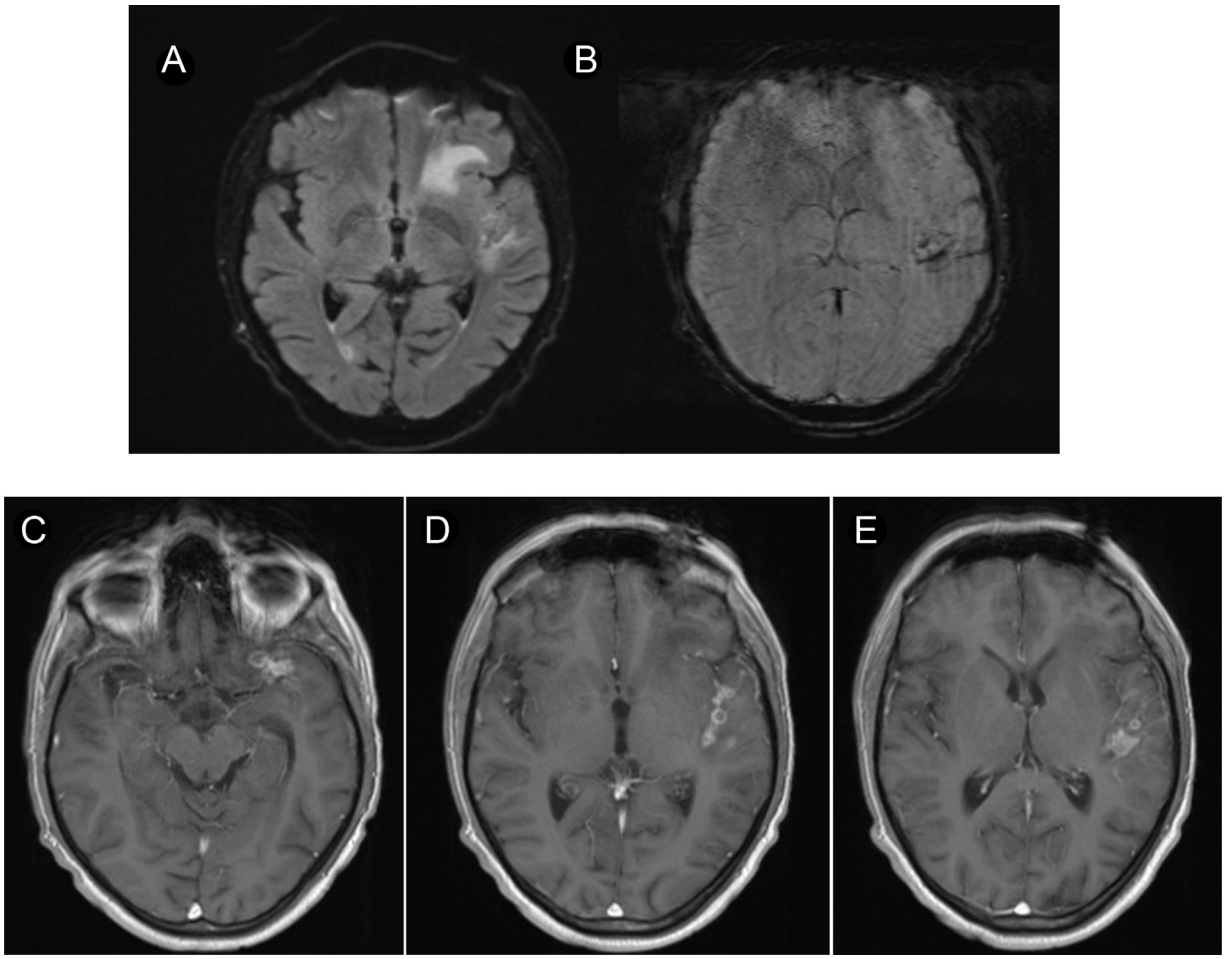

Fig. 5 – Follow-up MRI brain shows (A) T2 FLAIR images: Regression of left peri–insular temporal lobe edema and development of left frontal subcortical edema. (B) SWI: Persistent but less blood products. (C-E) Post Gad images: Nodular and cystic rim enhancing lesions in the left insular fissure and adjacent CSF spaces.

Discussion

CNS TB makes up around 1% of all TB cases [5]. CNS TB can manifest in multiple ways, including tuberculous meningitis (TBM), tuberculoma, tuberculous abscess, spinal arachnoiditis, and less commonly transverse myelitis [6,7]. Cases of hemorrhagic meningoencephalitis are less commonly reported in the literature, however, there are a few reports of cerebral vascular changes and lesions caused by TB, as well as multiple cases of both nonaneurysmal and tuberculous aneurysmal subarachnoid hemorrhage [8–10].

On further investigation, CNS TB can produce various imaging findings, including basal meningeal enhancement in up to 90% of cases, hydrocephalus in 66%, and infarction in over 50%. In cases of tuberculous meningoencephalitis, restricted diffusion of cortical and adjacent subcortical structures is typically observed on DWI [7]. Therefore, the differentials could be wide and can include malignancies, bacterial, viral, or parasitic CNS infections, and autoimmune encephalitis [7,11]. Cases of this presentation of tuberculous hemorrhagic meningoencephalitis are very difficult to come across in the literature with only one case fitting the exact diagnosis being reported in 1947 [12]. More common yet still rare manifestations of CNS TB include spontaneous subarachnoid hemorrhage rather than cerebral hemorrhage described above and cerebral hemorrhagic complications in confined tuberculous meningitis rather than meningoencephalitis as described above [8,9,13,14]. Moreover, there are 2 cases that report intraventricular hemorrhage being a rare complication in CNS TB, one as a complication of tuberculoma and the other in tuberculous meningitis [15,16].

Mortality rate in the literature was readily found for TBM reaching as high as 42%. The mortality rate for other CNS TB manifestations was not found in the literature due to the limited data available [2]. Because of the high mortality rate, sensitive diagnosing modalities along with timely interventions are crucial for the management of CNS TB. MRI was found to be the most sensitive modality for diagnosis especially if used with contrast achieving a sensitivity as high as 96.05%. It was found to be more sensitive than CSF analysis which itself was more sensitive than a CT scan [17].

Adjunct glucocorticoid therapy was found to be widely helpful in managing various CNS infections and improving

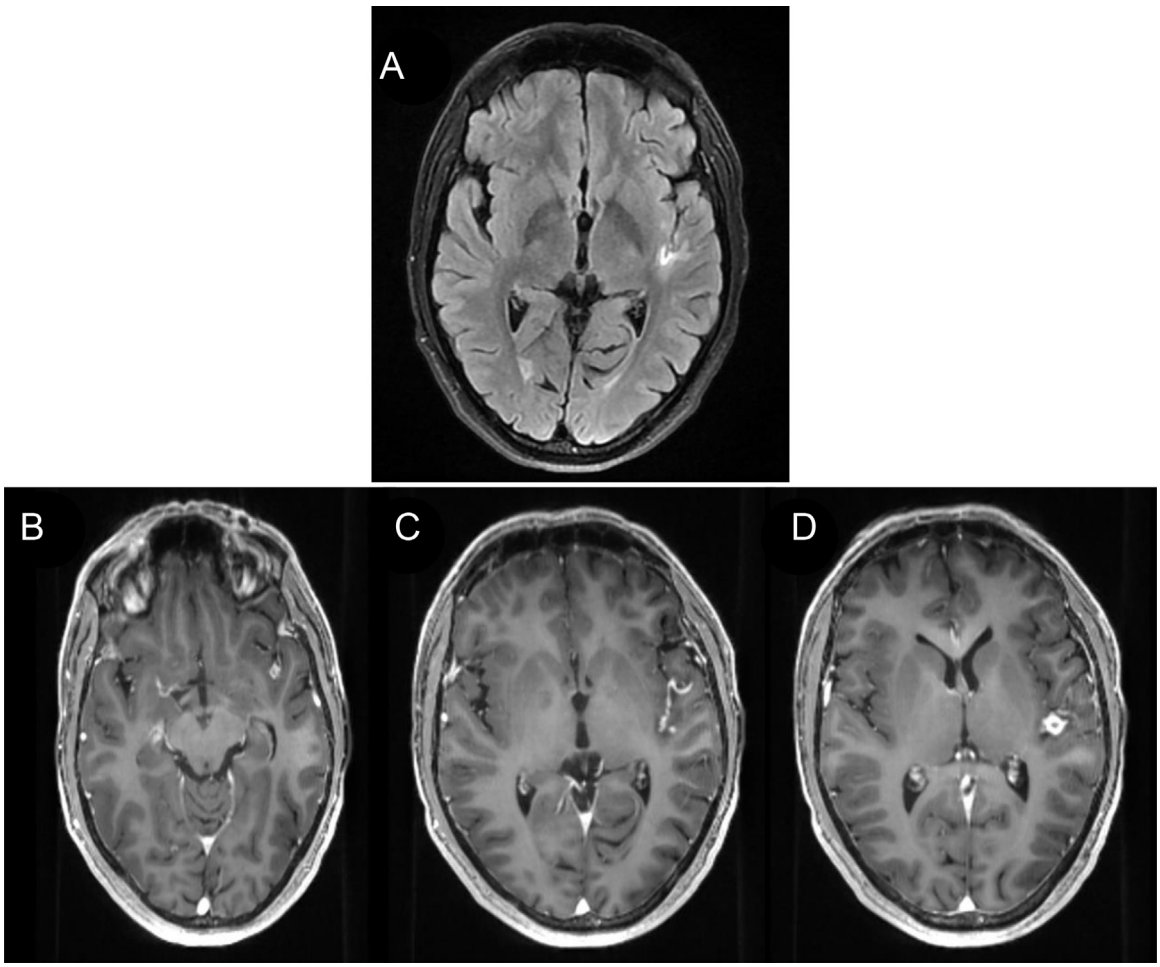

Fig. 6 – Final MRI Brain of patient taken a year after admission shows residual edema in the left peri–insular temporal lobe, as seen on (A) T2 FLAIR image. There is significant resolution of the left fronto-temporo-parietal insular subcortical leptomeningeal disease with residual ring enhancing lesions seen in the left peri–insular temporal lobe, as seen on (B-D) interval T1 postcontrast images.

their outcomes along with decreasing mortality rates including CNS TB [18]. This is most likely because steroids help in reducing inflammation that could damage and be harmful to the CNS [18]. Therefore, adjunct glucocorticoids have been largely recommended to be used in patients with CNS TB even in patients with HIV regardless of severity in order to decrease mortality and improve outcomes [4,19]. The glucocorticoid therapy should be used as an adjunct to the usual isoniazid, rifampicin, pyrazinamide, and ethambutol (RIPE) therapy used in the treatment of typical pulmonary TB for 2 months which are then followed by isoniazid and rifampicin for 10 months at least [19].

Conclusion

Tuberculous hemorrhagic meningoencephalitis is a rare and life-threatening complication of miliary TB, requiring prompt diagnosis and intervention. Timely radiological detection plays a crucial role in improving patient outcomes, but clinical observation and specialized treatment remain equally important. Comprehensive evaluation, combining both clinical and radiological approaches, is essential to identify and manage potentially severe CNS manifestations of TB.

Patient consent

The patient gave his consent for this case report to be published.

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