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Case Report

Tuberculous meningitis with stroke: A case report of diagnostic dilemma and therapeutic triumph ☆,☆☆

Shailendra Katwal, MD[a,*], *Anjila Thapa, MBBS*[b], *Aayush Adhikari, MBBS*[c], *Pratik Baral, MBBS*[d], *Mukhtar Alam Ansari, PhD*[e]

[a] *Department of Radiology, Dadeldhura Subregional Hospital, Dadeldhura, Nepal*
[b] *Kharanitar Primary Healthcare Center, Nuwakot, Nepal*
[c] *Manang Hospital, Chame, Manang, Nepal*
[d] *Institute of Medicine, Tribhuwan University Teaching Hospital, Kathmandu, Nepal*
[e] *National Medical College, Birgunj, Nepal*

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ABSTRACT

Tuberculous Meningitis (TBM) is a rare manifestation of *Mycobacterium tuberculosis* infection affecting the meninges. We present a case of a 40-year-old male initially presenting with neurological deficits mimicking ischemic stroke. Despite classic signs, including fever, headache, and neck stiffness, TBM was initially overlooked, leading to delayed treatment. Comprehensive assessment, imaging findings, and characteristic cerebrospinal fluid findings, confirming TBM with tubercular stroke. The patient responded positively to antitubercular therapy and steroids. This case underscores the diagnostic challenges of TBM, emphasizing the need for a broad differential diagnosis, particularly in regions with a high tuberculosis prevalence. Recognition of atypical presentations is crucial for timely intervention and improved outcomes.

Introduction and importance

Meningitis is characterized by inflammation of the meninges, the protective membranes that cover the brain and spinal cord. It can have various causes, including viral, bacterial, or fungal infections [1]. When it results from infection with the bacterium *Mycobacterium tuberculosis* (MTB), it is termed Tuberculous Meningitis (TBM). TBM arises from the infiltration of the meninges by Mycobacterium tuberculosis bacilli [2].

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* Corresponding author.
E-mail address: shailendrakatwal@gmail.com (S. Katwal).
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Tuberculous meningitis (TBM) constitutes approximately 5% of all cases of extrapulmonary tuberculosis [3]. *M tuberculosis* bacilli enter the host by droplet inhalation, after which the localized infection escalates within the lungs and then disseminates to the regional lymph nodes [4]. The bacilli may then seed to the central nervous system and result in 3 forms of CNS TB: tuberculous meningitis, intracranial tuberculoma, and spinal tuberculous arachnoiditis [5]. In TBM, the infection involves the seeding of the meninges by MTB, forming subependymal accumulations known as Rich foci. These foci can rupture into the subarachnoid space, triggering a robust inflammatory response that manifests as typical meningitis symptoms such as fever, headache, and neck stiffness [6]. Patients with TBM often exhibit preceding constitutional symptoms, including low-grade fever, loss of appetite, malaise, fatigue, and weight loss [7]. The resulting exudates from this inflammatory reaction can occasionally encase cranial nerves, leading to nerve palsies that resemble ischemic strokes [8].

A stroke is defined by a sudden onset of focal neurological deficits with vascular origins, either ischemic or hemorrhagic [9]. The chance of having a stroke doubles about every 10 years after age 55 [10]. Although stroke is common among older adults, many people younger than 65 years also have strokes [11]. About 1 in 7 strokes occur in adolescents and young adults ages 15-49 [12]. An ischemic stroke is a type of stroke that occurs when there is a blockage or obstruction within a blood vessel that supplies blood to the brain [11]. TBM patients may present with symptoms resembling ischemic strokes mainly due to the pressure effect of the inflammatory exudates [8]. Ischemic strokes in TBM may also result from tuberculous vasculitis and can be the cause of ischemic strokes in young individuals [13].

Case details

A 40-year-old male presented to our hospital with right-sided weakness of the body, confusion, and restlessness. The weakness was insidious in onset, gradually progressive, and accompanied by difficulty in speech production. It was preceded by a week of severe global headache and concomitant photophobia. The patient reported a continuous fever during the period with temperatures reaching a maximum of 102.8°F with associated chills and rigors. He had sought medical attention at a nearby healthcare center where he received treatment with analgesics. The headache was refractory to analgesics and the patient reported 2 episodes of projectile vomiting. The patient reported no history of abnormal body movements or history of similar episodes in the past. There was no history of calf pain/swelling, palpitations, bone pains, or heart disease in the past.

On examination, he had weakness of the right upper and lower extremities and an upgoing Babinski. Right sided power of upper and lower limbs was 1 of 5 and marked hyperreflexia (3+) was noted on performing knee and biceps jerks. Other significant examination findings included: neck rigidity, and uvula deviation to the right. The gait was normal, and facial symmetry was intact. The blood workup revealed mild leukocytosis (11,400 WBCs/cu mm) and an ESR of 72 mm/h in the first hour. Examination of the precordium and chest X-ray were both normal. The magnetic resonance imaging (MRI) results for the patient revealed high signal intensity on T2 and FLAIR sequences in the left basal ganglia, anterior and posterior limbs of the internal capsule, medial temporal lobe, and the left periventricular region (Figs. 1A and B). Areas with high signal on T1 indicated laminar necrosis (Fig. 1C). These regions exhibited restricted diffusion, accompanied by patchy enhancement in postcontrast images (Figs. 1D–F). Additionally, effacement of the basal cistern with mild hydrocephalus was observed.

Lumbar puncture findings included straw colored cerebrospinal fluid (CSF) with: a WBC count of 200 cells/cu mm, with lymphocytic predominance, glucose 10 mg/dL, protein 110 mg/dL, and adenosine deaminase (ADA) of 270 mg/L. Both bacterial culture and potassium hydroxide (KOH) mount of CSF yielded negative results. Serum serology for HIV, Hepatitis B, C, and syphilis were all negative.

The patient was admitted with a diagnosis of Tubercular stroke, a complication of tubercular meningitis. Antitubercular therapy, and dexamethasone was started along with fluid and electrolyte therapy. The condition improved over a week of in-patient therapy, and the patient was discharged on antitubercular therapy via the Direct Observed Short Course treatment program. A follow-up 2 years following the initial hospitalization has shown improvement in the patient's condition.

Clinical discussion

TBM can be a complex and challenging medical condition that often poses diagnostic difficulties [3]. In this case report, we share an intriguing example of TBM complicated by a stroke—a relatively uncommon clinical scenario. Hence, it emphasizes the importance of considering various possibilities when diagnosing patients.

The fact that our patient had symptoms of both TBM and a stroke highlights the need for thorough clinical assessments and a high level of suspicion. It's crucial to be aware that TBM can sometimes lead to the development of a stroke, especially in regions where tuberculosis is common [14]. As our case shows, not considering TBM as a possibility can lead to delays in treatment, potentially making the patient's condition worse. Additionally, our case reminds us that TBM can sometimes have unusual symptoms, making it even more challenging to diagnose correctly. While we usually look for signs like fever, headache, and neck stiffness to diagnose TBM, our patient's main issue was neurological deficits, which initially led us to consider stroke as the primary diagnosis.

The exact reasons behind strokes in TBM are still under investigation. While some suggest that pressure from inflammatory substances might play a role, there is increasing recognition that tuberculous vasculitis—a type of inflammation in blood vessels—is a contributing factor [8,13]. This understanding not only affects diagnosis but also influences treatment decisions, such as considering anti-tuberculosis medications and immune system suppression in cases where vasculitis is present. In a comparable clinical scenario, Patil et al. [14] documented a case resembling ours, where magnetic resonance

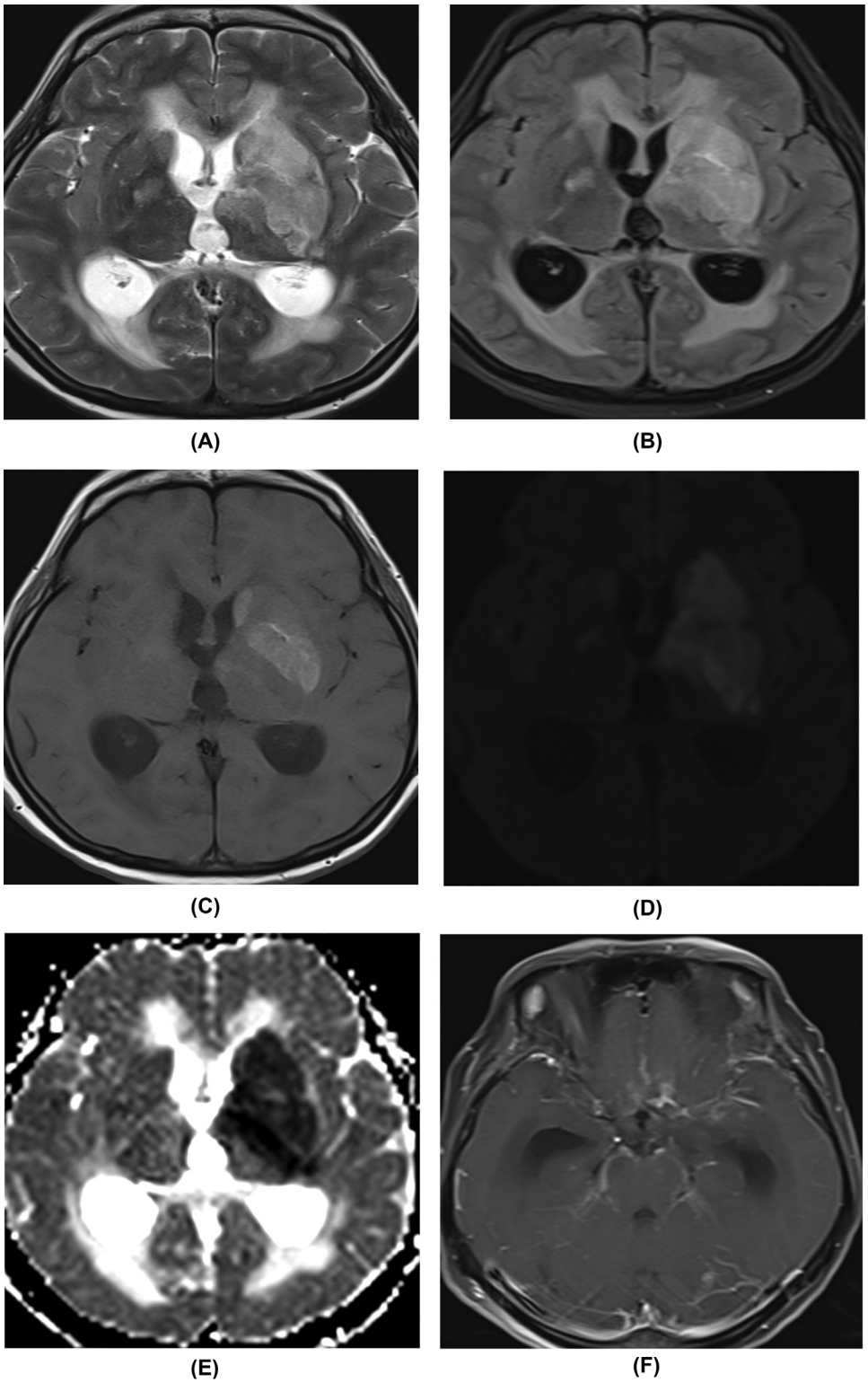

Fig. 1 – (A) T2 weighted Axial magnetic resonance Image at the level of basal ganglia showing high signal intensity in left basal ganglia, anterior and posterior limb of left internal capsule. (B) FLAIR axial magnetic resonance Image showing high signal intensity in the left basal ganglia, anterior and posterior limb of internal capsule. (C) T1 weighted axial magnetic resonance image at the level of basal ganglia showing the T1 high signal intensity within the left basal ganglia region. (D and E) DWI and ADC weighted magnetic resonance image at the level of left basal ganglia showing the diffusion restriction in left basal ganglia. (F) T1 postcontrast image at the level of basal cistern showing the patchy meningeal enhancement.

imaging (MRI) was employed to identify tuberculous vasculitis as the underlying cause of the stroke. In that instance, the patient was started with a combination of antitubercular therapy and steroid treatment. Although there have been reported cases of tubercular meningitis, presenting as a stroke in young patients due to vasculitis remains a rare occurrence.

Hence, when a patient exhibits the classic signs and symptoms of a stroke, it is essential not to rush to a hasty classical stroke diagnosis. Instead, one should maintain a broad perspective, remaining vigilant to any additional indications, such as a low-grade fever or neck pain. In such cases, TBM should be considered among the potential diagnoses, prompting a comprehensive evaluation to confirm or rule out its presence in the patient's medical condition.

Conclusion

This case highlights the diagnostic complexity of TBM complicated by a stroke, emphasizing the necessity for vigilant consideration in regions with high tuberculosis prevalence. Recognition of atypical complications of TBM such as tubercular stroke, is pivotal for timely intervention, ensuring improved outcomes for patients with this challenging neurological condition.

Ethical approval

This case report did not require review by the ethical committee.

Author contributions

Shailendra Katwal: Conceptualization, as mentor and reviewer for this case report and for data interpretation. Anjila Thapa: Contributed in performing literature review and editing. Aayush Adhikari: Contributed in writing the paper and reviewer for this case. Pratik Baral: Contributed in writing the paper. Mukhtar Alam Ansari: Contributed in writing the paper and mentoring. All authors have read and approved the manuscript

Registration of research studies

Not applicable

Provenance and peer review

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Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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