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Case report

Ancient foe, serious weapon: Subarachnoid hemorrhage secondary to TB meningitis

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ABSTRACT

Tuberculosis is an ancient infectious disease with global distribution and a multitude of multisystem presentations. Infection of the central nervous system (CNS) is the most serious presentation manifested as tuberculous meningitis (TBM), intracranial tuberculoma, and tuberculous arachnoiditis all associated with significant morbidity and mortality. TBM is the commonest form of CNS manifestations capable of causing secondary arteritis leading to vascular complications. We report a case of a 22-year-old Indian patient diagnosed with TBM who subsequently presented with sudden onset severe headache, which was eventually diagnosed as subarachnoid hemorrhage. Radiological assessment confirmed secondary complications with cerebral aneurysmal dilatation attributed to TBM. The patient was safely managed with combined radiological and surgical interventions with uneventful outcomes. Review of the literature revealed that such complication of TBM is rare usually with serious implications. We aim to highlight to infection specialists to be aware of such association.

Introduction

Tuberculous Meningitis (TBM) accounts for about 1 % of all TB cases and up to 10 % of extrapulmonary disease in some endemic areas [1]. Although rare, the disease has significant morbidity and mortality, often lethal with no interventions hence caution and vigilance should be observed to diagnose it early to avert significant complications including permanent neurological sequelae cited as high as 50 % of cases [2]. Patients typically present insidiously with a prodrome of constitutional symptoms that peaks into meningitic phase, culminating into a serious and life-threatening paralytic sequelae [3]. In TBM, the hallmark of the pathological processes is meningeal exudative inflammation and active vasculitis leading to increased intracranial pressure with secondary hydrocephalus and subsequent neurological deficit [4,5]. Progressive CNS disease is the manifestation of basal meningitis while cerebral ischemia is commonly associated with secondary vasculitis and arteritis induced by persistent local inflammation [3,6]. Vascular phenomenon like intracranial aneurysms can rarely develop leading to ominous risks such as secondary subarachnoid hemorrhage (SAH) with its substantial implications [6].

We report a case of a young patient diagnosed with TBM then presented acutely with SAH secondary to intracranial aneurysm presumed secondary to the infectious vasculitis. There has been a limited number of reports outlining the relationship between the two conditions which worth highlighting to improve early detection.

Case report

A 22-year-old Indian male who was previously healthy, presented to the emergency department (ED) at our acute care hospital with one-week history of subjective fever, cough, and weight loss. Clinical examination was generally unremarkable, including a normal temperature, but with reduced breath sounds at the right lung base. Hematological, biochemical parameters including inflammatory markers were unremarkable, including ESR of 22 mm/h, but TB QuantiFERON assay was positive. Chest radiograph (Fig. 1) revealed faint right lower zone consolidation with minimal pleural effusion. The patient was initially assessed as a possible case of community-acquired pneumonia, but clinical deterioration with nausea, vomiting, headaches, and confusion,

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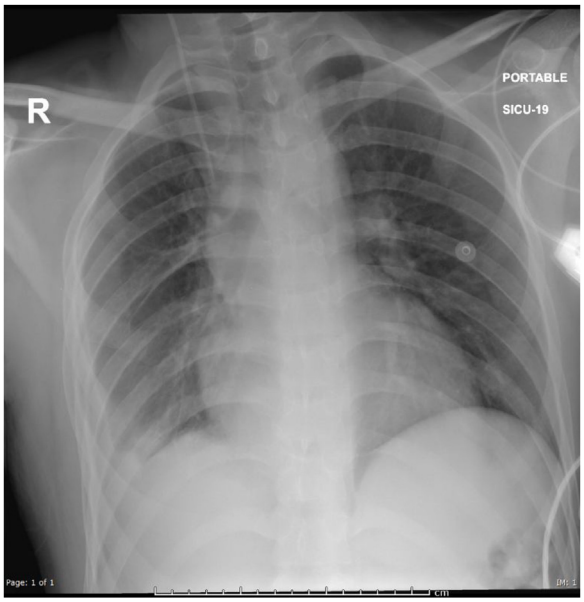

Fig. 1. AP Chest radiograph showing faint right lower zone consolidation with minimal pleural effusion.

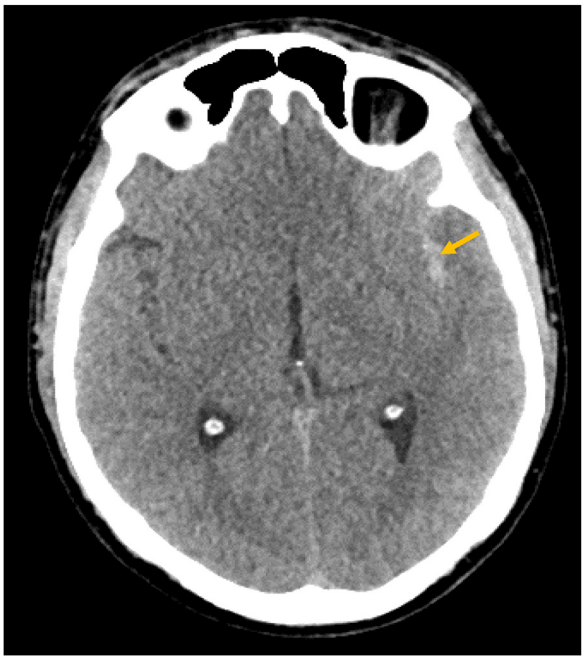

Fig. 2. Plain head CT demonstrating subarachnoid hemorrhage at left Sylvain fissure (arrow).

prompted further evaluation. Brain CT scan was unremarkable while lumbar puncture and CSF analysis demonstrated significant lymphocytic pleocytosis with 300 cells with 90 % lymphocytic predominance combined with low CFS glucose at 1.7 mmol/l (normal 2.22–3.8 mmol/L), high CSF protein at 1.98gm/dl (normal 0.15−0.45gm/dl). Microscopy showed no organisms, while bacterial cultures were negative. The diagnosis of disseminated TB with TBM was supported by a positive CSF GeneXpert MTB/RIF PCR assay from sputum and CSF showing no preliminary resistance, confirmed upon culture as a fully sensitive Mycobacterium Tuberculosis. The patient was started on conventional quadruple regimen including Rifampicin, Isoniazid, Pyrazinamide and Ethambutol with adjuvant tapering doses of steroid therapy planned for 12-month duration. The patient made a significant clinical response with reversal of all symptoms up to discharge with no residual complications.

One month following discharge, the patient presented to the ED with intense acute headache and vomiting. Physical examination was unremarkable with no neurological deficit. Urgent CT head revealed acute subarachnoid hemorrhage (Fig. 2). Further assessment with brain CT angiogram, Magnetic Resonant Imaging, and angiogram (MRI/MRA) confirmed left tempo-partial subarachnoid hemorrhage together with probable left middle cerebral artery infective aneurysm (Fig. 3). The patient underwent urgent craniotomy with warping of the left aneurysm, followed by embolization. Despite the grave implications, all the patient acute symptoms resolved, followed by a short period of rehabilitation to continue remaining therapy. Follow up showed no residual neurological sequelae.

Discussion

TBM is one of the infrequent forms of the TB spectrum yet lethal because of diffuse CNS pathology leading to basal meningitis, increased intracranial pressure, secondary hydrocephalus, vascular complications and ultimately fatal outcomes if not appropriately recognized and timely managed [1].

The pathophysiology of the disease is implicit probably starting as hematogenous spread with meningeal seeding foci followed by the development of persistent meningeal and vascular inflammation with thick gelatinous exudates leading to the obstruction of CSF flow and occasionally vascular events, occlusive ischemic infarctions being more common than hemorrhagic events [6]. The assessment and diagnosis of TBM is always challenging since typical presentation is frequently insidious with no clear directive symptoms, paucity of clinical signs as well as challengingly, nonspecific investigations including low yield CSF examinations even with advanced modalities [3,7]. It is important to establish epidemiological links of previous exposure or infection in suspected cases particularly in immigrant population from high endemic areas, previous reviews of our cohort showed the majority of TBM cases were imported with substantial complications [8]. In suspected cases, the diagnosis of TBM is supported by plausible hints of CNS infection, sterile lymphocytic CSF with divergent low CSF glucose and high proteins levels. Previous exposure to TB manifested by a positive tuberculin skins test like PPD or interferon gamma release assays has poor diagnostic predictions [3,9]. During initial clinical evaluation, it is important to stratify patients clinical condition into mild, moderate or severe based on symptoms and neurological assessment including level of consciousness since outcomes are noticeably worse with advanced disease [9,10]. Radiological imaging favors brain MRI as compared to CT in outlying the pathology, particularly leptomeningeal enhancement following contrast administration [11]. Since the disease has ominous consequences with reported death or permanent neurological disabilities in almost half of cases, it is advocated to have a high degree of suspicion for probable cases including early presumptive management to avert complications. Prompt anti-tuberculous medications together with adjuvant steroids and surgical interventions in selected cases have shown to reduce mortality as well as neurological sequelae [9].

Although infrequent, vascular complications such as intracerebral or subarachnoid hemorrhages have been previously reported [12,13]. Persistent mycobacterial infection leads to arteritis with degradation of arterial walls resulting in the development of aneurysmal dilatations and subsequent rupture [4,6]. In the reported case, the time gap between initial assessment and the second presentation with SAH reflects that phenomenon. Although

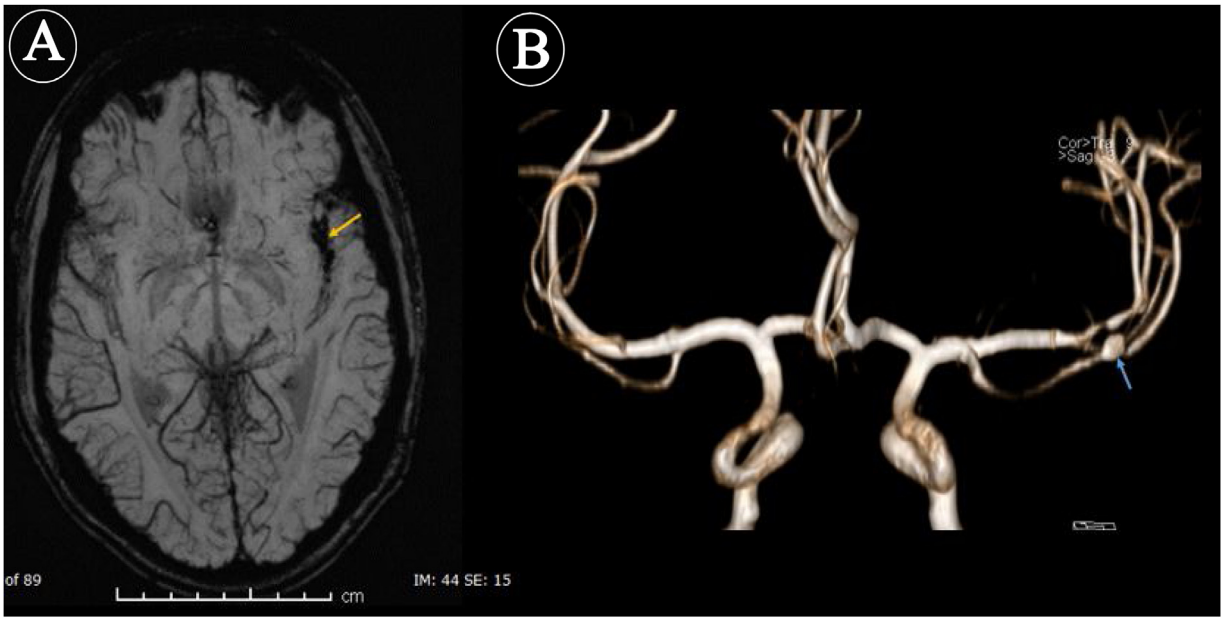

Fig. 3. Susceptibility weighted image (A) displayed dark blooming along the left sylvian fissure (orange arrow) corresponding to subarachnoid haemmorage seen in CT. 3D time of flight intracranial MRA (B) showed a small obliquely oriented saccular aneurysm arising from the anterior temporal branch of the left MCA (blue arrow).

this seems the most plausible explanation for the patient presentation taking into consideration the patient age and the absence of the other underlying diseases, we acknowledge the two conditions might be separate. There are no available reports of the prevalence nor outcomes of subarachnoid hemorrhage in the context of TBM while surgical and interventional radiological procedures remain a lifesaving approach as in our case [14].

Conclusion

Tuberculosis although historic and ancient disease, remains enigmatic with a wide range of clinical presentations. Involvement of CNS is usually difficult to recognize and frequently leads to significant morbidity and mortality. Vascular phenomena like vasculitis and secondary aneurysms leading to brain hemorrhages are rare but has serious complications which needs awareness to avoid catastrophic consequences.

Consent compliance with ethical standards

A written informed consent was obtained from the patient to include clinical presentation together with results and imaging. This was subsequently reviewed and approved by the institution ethics and research review board.

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Declaration of Competing Interest

The authors report no declarations of interest.

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