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Case Report

Primary tuberculous pyogenic ventriculitis in an immunocompetent patient: A case report

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ABSTRACT

Background: Although tuberculosis (TB) of the central nervous system is quite common, tuberculous pyogenic ventriculitis is not only rare; it is a devastating disease in an immunocompetent patient if left untreated.

Case Description: We present the case of a 43-year-old man who underwent successful treatment for tuberculous pyogenic ventriculitis that presented with meningeal syndrome and loss of consciousness.

Conclusion: Tuberculous pyogenic ventriculitis is a rare manifestation of intracranial tuberculous infection. Despite advances in imaging techniques, the diagnosis of intraventricular TB is essentially biological.

Keywords: Abscess, Immunocompetent, Intraventricular, Primary, Tuberculosis

INTRODUCTION

The central nervous system tuberculosis (CNS-TB) is one of the most severe forms of tuberculosis (TB), with the highest morbidity and mortality.[5] The most commonly reported symptoms were TB meningitis, tuberculomas, and, more rarely, pyogenic ventriculitis.[1,5,8] In immunocompetent patients with CNS-TB, tuberculous pyogenic ventriculitis accounts for 4–8% of brain lesions.[2] It is defined as an encapsulated collection of frank pus containing viable tubercular bacilli in the cerebral ventricular system without evidence of tubercular granuloma.[1,7] However, primary tuberculous pyogenic ventriculitis is a rare event.

In this report, we describe the case of a 43-year-old man who developed tuberculous pyogenic ventriculitis and review the available literature.

CASE REPORT

Written informed consent was obtained from the patient and his family for the publication of this case report and accompanying images.

History

A 43-year-old man was admitted to the emergency department with acute onset of loss of consciousness. He denied having any pre-existing comorbidities, and his medical history was unremarkable. His family history included the death of his father 7 months ago from pulmonary TB.

Examination

On arrival to the emergency room, the patient's vital signs were as follows: blood pressure, 118/85 mmHg; pulse rate, 78 bpm; body temperature, 38.3°C; and Glasgow Coma Scale, of 8 E3V1M4 with the stiffness of the neck.

Pathological findings

The laboratory blood test revealed an increased of C-reactive protein (CRP) at 242.5 mg/L. Retroviral serology results were negative. The chest X-ray was normal. A computed tomography (CT) scan of the brain showed a lesion in the left lateral ventricle with minimal, fine, and complete rim enhancement. There was an enlargement of the left lateral ventricle with an isodense lesion, suggesting an intraventricular collection [Figure 1]**.** On brain magnetic resonance imaging (MRI), the lesion showed low signal intensity on T1-weighted (T1W) imaging and high signal on T2-weighted (T2W) imaging. Gadolinium-enhanced MRI showed thickened ependymal sheath enhancement with increasing ventricular size. In fluid-attenuated inversion recovery (FLAIR), the ventricle contains a layering of fluid and sediment. There was diffusion restriction in the posterior temporal horn of the lateral ventricle of the lesion, which was observed on diffusion-weighted imaging [Figure 2]. These radiological findings suggested the presence of suppurative fluid in the cerebral ventricles.

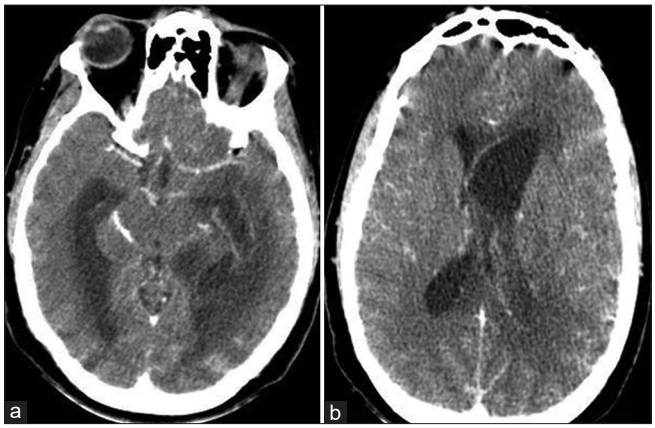

Figure 1: (a and b) Computed tomography scan of the brain showed a lesion in the left lateral ventricle with minimal, fine, and complete rim enhancement. There was an enlargement of the left lateral ventricle with an isodense lesion, suggesting an intraventricular collection.

Surgery

The patient was taken to the operating room for an emergency left frontal external ventricular drainage (EVD). The ventricular catheter was inserted through Kocher's point in the frontal horn. A thick, cloudy, pus-like fluid emerged from the drain. This was collected and sent for bacteriological examination. In the operative room, the drainage process was continued by negative pressure until clear cerebrospinal fluid (CSF) was obtained. After EVD placement, the patient was transferred to the intensive care unit (ICU) department.

Postoperative course

The monitoring of CSF analysis and CRP were performed [Table 1]. Empirical intravenous antibiotic treatment with third-generation cephalosporin (per-operative single dosage administration) was continued after surgery. The result of Ziehl-Neelsen staining to detect acid-fast bacteria in the pus sample was positive. GeneXpert was positive for *Mycobacterium tuberculosis*. Antibacillar treatment was started and then continued for a total of 12 months. Antiepileptics drugs (valproic acid) were administered for a total of 3 months. In addition, intravenous corticosteroids were administered for a total of 4 weeks. Ten days after surgery, the patient was transferred to the infectious diseases department. The patient was free from the EVD catheter on day 17th. The patient recovered completely with no complications following 6 weeks of in-hospital management. He was then discharged without any motor or sensory impairments. At the 6-month follow-up, the patient was examined as an outpatient and was in good health. The patient underwent a follow-up brain scan, which was normal [Figure 3].

DISCUSSION

Tuberculous pyogenic ventriculitis is an uncommon presentation of CNS-TB. The immunological status of the patient is crucial for the development of a latent or disseminated infection, which is much more common in patients with immunodeficiency. Based on the literature review, only four cases of tuberculous pyogenic ventriculitis have been reported in the English literature to date.[1-6] Vajramani *et al*.[7] were the first authors to report a case of tuberculous pyogenic ventriculitis in a 26-year-old woman with an unremarkable past medical history. The authors suggested that the intracranial spreading of the *Mycobacterium* may be from the enlargement of a small choroid plexus or a subependymal tubercle.[6]

Nevertheless, the origin of such lesions is not clear in the reported cases. Exposure to *M. tuberculosis* increases the risk of TB infection. This risk is higher in immunodeficient patients as they are particularly susceptible to progression

Table 1: CSF and CRP monitoring of our patient.

	Operative day	1 d postoperative	3 d postoperative	5 d postoperative	7 d postoperative
CSF (Color)	Cloudy	Clear	Clear	Clear	Clear
WBC types	600 Lymphocytes (83%)	300 Lymphocytes (80%)	100 Lymphocytes (72%)	40 Lymphocytes (60%)	20 Lymphocytes (43%)
Proteins (15–40 mg/dL)	270	213	145	92	57
Glucose (50–80 mg/dL)	0.91	0.7	0.59	0.73	0.65
Blood	Serum	Serum	Serum	Serum	Serum
CRP (mg/dL)	242.5	239	161	69	49

d: Day, CSF: Cerebrospinal fluid, WBC: White blood cells, CRP: C-reactive protein

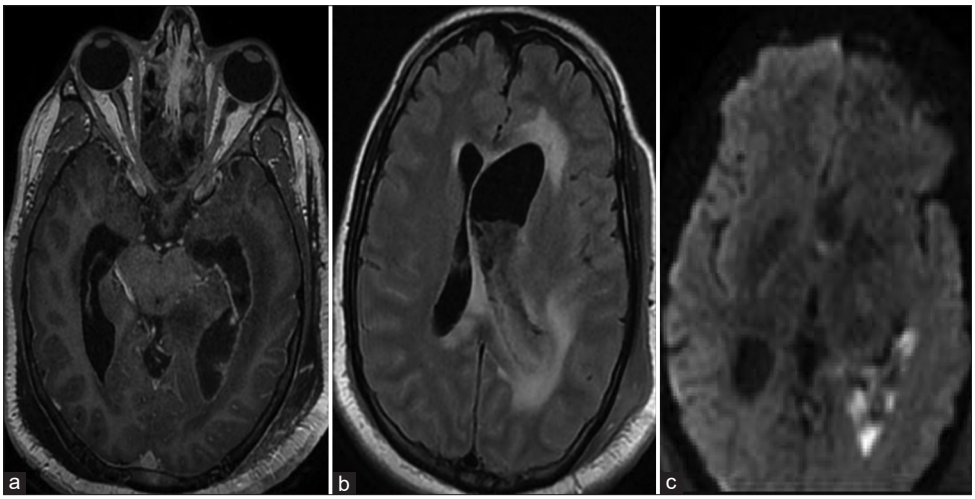

Figure 2: (a) Gadolinium-enhanced magnetic resonance imaging showed thickened ependymal sheath enhancement with increasing ventricular size. (b) In fluid-attenuated inversion recovery, the ventricle contains a layering of fluid and sediment, suggesting collection. (c) There was diffusion restriction in the posterior temporal horn of the lateral ventricle.

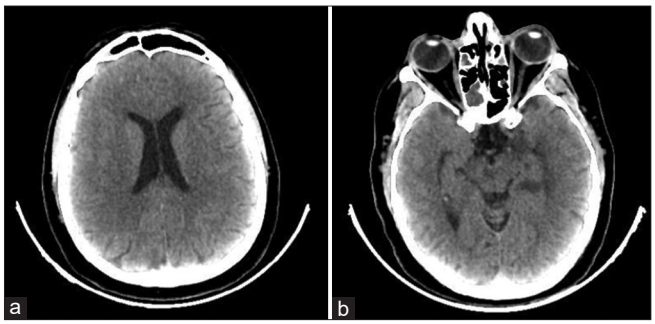

Figure 3: (a and b) Postoperative computed tomography of the head: Showing complete cleaning of the intraventricular abscess.

from latent to active disease. In our case, the father died from TB. Our patient's human immunodeficiency virus status was negative.

The tubercle bacilli could potentially enter the ventricles through the choroid plexus by hematogenous spread from a primary site of infection, usually the lung, without pulmonary manifestation. Ventricular involvement in neurotuberculosis can occur in different ways. For example, Rich's juxta-ependymal foci can cause meningitis by tearing into the subarachnoid spaces. It can also cause varying degrees of inflammation of the ventricular surface ependyma and choroid plexus with the formation of tubercles, tuberculomas, and rarely abscesses.[1,3] The development of a tuberculous abscess may be due to a large inoculum leading to an excessive exudative reaction with massive caseation. The softening of the caseum due to the influx of polymorphonuclear leukocytes leads to the formation of pus. In immunocompromised individuals, a poor inflammatory response as a result of altered cell-mediated immunity can also lead to abscess formation.[1] On MRI, the temporal and occipital choroid plexus appeared inflamed and involved by the abscess [Figure 2], indicating that a previous choroid plexitis may have been the source of the abscess formation.

The most common clinical symptoms observed were intracranial hypertension syndrome and meningeal

syndrome, with or without fever.[1,3,4,6] It was similar to our case. Although not specific, both CT and MRI are sensitive for detecting tuberculous abscesses. Tuberculous abscesses are typically characterized by central hyperintensity on T2W and rim enhancement on post-contrast T1W scans. In our case, the tuberculous pyogenic ventriculitis showed T1W hypointensity, T2W hyperintensity, and rim enhancement. T2 FLAIR MR imaging showed left periventricular edema and ventriculomegaly. The abscess lesion showed a hyperintense lesion on diffusion-weighted imaging. Our patient underwent EVD. The same procedure was used by Hanafiah *et al*.[5] This method aimed to relieve the intracranial high pressure, ensure drainage of the abscess, and confirm the diagnosis. Regardless of advances in imaging techniques, the diagnosis of intraventricular TB is essentially biological. These objectives were achieved in our patient. The patient recovered well with no neurological deficits 6 months later.

CONCLUSION

Tuberculous pyogenic ventriculitis is a rare manifestation of intracranial tuberculous infection. In the reported cases, the origin of lesions is not clear. Although immunodeficient patients are at higher risk of developing the rare forms of CNS TB, there is no understood immunity in the cases reported to date. Despite advances in imaging techniques, the diagnosis of intraventricular TB is essentially biological.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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