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Case Report

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Third cranial nerve palsy as an initial presentation of tuberculous meningitis

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ABSTRACT

In this case report, we present a 54-year-old male patient diagnosed with tuberculous meningitis after presenting with third cranial nerve palsy. Tuberculous meningitis is a rare but potentially fatal infection in certain clinical conditions, and its diagnosis can bechallenging, especially when patients present with atypical symptoms. Early treatment is crucial,as delayed therapy may lead to a high risk of mortality. This case report highlights the importance of considering tuberculous meningitis in the differential diagnosis after excluding other common and life-threatening causes in the presence of atypical findings, such as third cranial nerve palsy. Prompt initiation of treatment is essential when there is clinical suspicion.

Keywords: Tuberculous, meningitis, abdusens

INTRODUCTION

Tuberculous meningitis (TBM) is a severe form of central nervous systemtuberculosis caused by *Mycobacterium tuberculosis*. It is characterized by a subacute progression and often presents with nonspecific symptoms such as fever, headache, and malaise, which complicates early diagnosis [1,2].

CASE

A 54-year-old male was admitted to an outside facility with complaints of headache anddiplopia that began four days ago. On neurological examination at the outside facility, hisright eye had limited movements upward, downward, and toward the midline. Patient stated severe headache that originates from the right occipital region, radiating toward the right eye. He also reported severe right leg pain, the onset of which was simultaneous with that of the other symptoms. Brain computed tomography (CT), brain diffusion magnetic resonance imaging (MRI), brain and carotid CT angiography, and contrast-enhanced MRI were performed at the same external facility without eliciting pathological findings. After excluding ischemic stroke, cerebral hemorrhage, aneurysm, and cavernous sinus thrombosis, among other differential diagnoses, he was referred to us for further investigation and treatment.

The character of headache at admission to our department remained unchanged—severe pain originating from the right occipital region and radiating to the right eye. Photophobia, phonophobia, nausea, or vomiting did not accompany headache. During the neurological exam, there was a deviation in the right eye during spontaneous gaze. The patient initially presented with limitation of upward, downward, and inward movements of the right eye, accompanied by ptosis on the right side. Unfortunately, as photographs of the patient's initial presentation were not available, they could not be included in this report. Steroid and mannitol therapy was initiated, and improvement in the patient's right eye began on the 5th day of treatment, with complete resolution observed by the 11th day.The rest of the neurological examination was normal. In the patient's left eye, ptosis and limitation of inward, upward, and downward gaze developed on the 11th day. There was no evidence of meningeal irritation (Figure).

He was diagnosed with mantle cell lymphoma four years ago but has been in remission for the last two years without other pathologies. No signs of recurrence were detected in the patient by the department of hematology. Contrast-enhanced brain MRI and MR venography performed at our facility

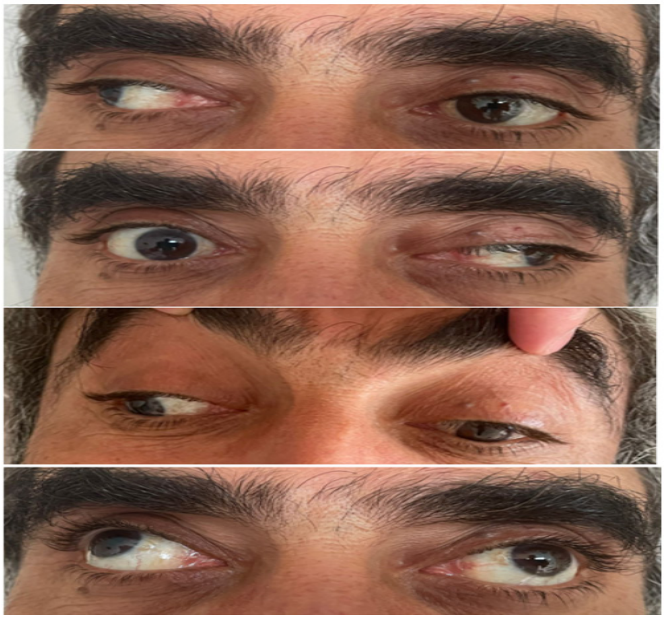

Figure. The right eye deviated outward in spontaneous gaze (1), and there was restriction of movement inward gaze (2), downward gaze (3), and upward gaze (4) in the right eye. Also, inward gaze (1)- downward gaze (3) and upward gaze (4) have been observed in the left eye. (This photo was taken on the 12th day after the first symptom appeared.)

also did not reveal pathological findings for aneurysm, or cavernous sinus thrombosis. The results of routine blood tests were in normal ranges. Fundoscopic examination was normal with no papilledema. A lumbar puncture was carried out which revealed increased cerebrosipinal fluid (CSF) pressure and protein level, with decreased CSF glucose (less than 50% of the simultaneously assayed plasma glucose). Additionally, 920 leukocytes and 70 erythrocytes per mm^3 were detected in the CSF (CSF opening pressure: 28 cmH_2O; CSF glucose: 45 mg/dl; simultaneous plasma glucose: 101 mg/dl; CSF protein: 166 mg/dl). No atypical cells were observed in the CSF pathology; it was reported as a lymphocyte-rich fluid.

The meningitis panel tests (*Cryptococcus gattii/neoformans*, *Listeria monocytogenes*, *Neisseria meningitidis, Streptococcus pneumoniae, Haemophilus influenzae, Streptococcus agalactiae, Escherichia coli* K1, Cytomegalovirus, Enterovirus, Human parechovirus, Varicella zoster virus, Herpes simplex virus 1, Herpes simplex virus 2, Human herpesvirus 7, Human herpesvirus 8, Human herpesvirus 6) performed on the CSF sample were found to be negative. Also, no growth was observed in the culture of the CSF sample and CSF histochemistry analyses showed no atypical cells. TBM was considered in the patient, and a QuantiFERON test was performed. The serum QuantiFERON-TB test resulted positive. The TBM medication was started on 1×300 mg isoniazid, 2×300 mg rifampin, 2×500 mg pyrazinamide, and 2×500 mg ethambutol. Additionally, intravenous steroid therapy was initiated at a dose of 1 mg/kg/day (total 80 mg) and was gradually tapered after 5 days.

The CSF results are presented in Table. In repeated lumbar punctures, a decrease in protein, glucose, opening pressure, and leukocyte count was observed. The patient was started on antiviral therapy (acyclovir 750 mg three times daily for 7 days)and mannitol therapy under the preliminary diagnosis of HSV. Following significant improvement in right eye adduction limitation and ptosis, the patient subsequently developed left eye ptosis and adduction limitation (complete third cranial nerve palsy) one day later. A third lumbar puncture was performed on the patient on the 11th day. Meningitis panel: *Haemophilus influenzae* (meningitis) detected as positive.

DISCUSSION

The diagnosis of TBM heavily depends on clinical presentation, imaging studies, and cerebrospinal fluid (CSF) analysis.[11] Imaging techniques, such as CT and MRI, can show basal meningeal enhancement, hydrocephalus, and tuberculomas, which are indicative of TBM.[3,4] However, in many cases, imaging may not reveal any abnormalities. In our case, no abnormal findings were observed in neuroimaging techniques.[11] On the other hand, the symptoms seen in TBM are not unique to this condition and can also occur in other types of meningitis, such as viral or fungal meningitis. These overlapping symptoms can complicate the diagnostic process.[6]

Cranial nerve involvement is quite common in TBM, and the reported prevalence varies widely depending on the patient population and the stage of the disease. Reported series indicate cranial nerve involvement rates ranging from 17% to 40%.[12-15] Among cranial nerves, the sixth cranial nerve (abducens) is most commonly involved in TBM, with reported involvement rates ranging from 20% to 40%, followed by the oculomotor (10%–30%), facial (5%–15%), and optic nerves (10%–20%).[12,13,15]

The examination of CSF is one of the important steps in diagnosing TBM. CSF analysis typically shows elevated protein levels, low glucose levels, and lymphocytic pleocytosis, which are critical for confirming the diagnosis.[4,5] In our case we also observed elevated protein levels, decreased glucose levels and lymphocytic pleocytosis.

Diagnosing TBM can pose challenges even to experienced clinicians. When patients present with headache and third cranial nerve palsy, as in our case, it is essential to first rule

Table. CSF analysis over time

Day	Opening pressure (n=6-20cmH_2O)	Leukocytes (n=0-5 mm^3)	Protein (mg/dl) (n=15-45 mg/dl)	Glucose (mg/dl) (n=50-80 mg/dl)	Albumin (n=10-30 mg/dl)	Na (n=142-150 mmol/L)	Cl (n=118-132 mmol/L)
Day 1	28	920	166.0	45	53.27	143	118
Day 5	23	250	167.31	32.6	52.57	143	119
Day 11	19	2000	261.16	28.6	44.29	141	120
Day 21	14	None	288.84	19.6	43.58	147	120

n: Normal range of variable, CSF: Cerebrospinal fluid

out other common and potentially fatal conditions such as ischemic stroke, intracranial hemorrhage, and cavernous sinus thrombosis. Once these possibilities are excluded, meningitis should be considered in the differential diagnosis. Neuroimaging should be conducted, and CSF analysis is indispensable for assessment. Besides routine CSF analysis, specific diagnostic tests for Mycobacterium tuberculosis, such as direct smear microscopy and polymerase chain reaction (PCR), should be performed.

In our case, these specific tests yielded negative results; however, other routine CSF analyses aligned with a diagnosis of TBM. Additional diagnostic tests, including the purified protein derivative (PPD) skin test and other evaluations to identify tuberculosis in other organs, can further support the diagnosis. Once TBM is confirmed, or if clinical suspicion is high, treatment should begin immediately without delay.

Treatment for TBM typically involves a prolonged course of antitubercular therapy, often paired with corticosteroids to reduce inflammation and manage complications like hydrocephalus.[4,7] Effective management of TBM is critical, as untreated cases can result in significant morbidity and mortality; studies report case fatality rates ranging from 15% to 68%.[8] Additionally, hydrocephalus, a common complication of TBM, may require surgical intervention to alleviate increased intracranial pressure.[9,10]

CONCLUSION

TBM is a rare but serious condition that may initially present with atypical neurological symptoms such as cranial nerve palsies in the absence of classical meningeal signs. This case emphasizes the vital importance of maintaining a high level of clinical suspicion for TBM in patients presenting with isolated third cranial nerve involvement, especially when neuroimaging does not reveal common vascular or structural pathologies.

In our case, the absence of radiological findings and negative CSF microbiological tests initially complicated the diagnostic process. However, the characteristic CSF findings—lymphocytic pleocytosis, elevated protein, and low glucose—when evaluated together with the clinical course and auxiliary tests such as PPD and QuantiFERON, allowed for a presumptive diagnosis of TBM and timely initiation of antitubercular treatment.

Early diagnosis and prompt treatment of TBM are crucial for reducing long-term neurological sequelae and mortality. This case highlights the necessity of initiating empirical treatment in suspected TBM cases based on clinical judgment supported by laboratory data, without waiting for definitive microbiological confirmation.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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