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Case Report

A case of papilledema with TB meningitis with bilateral abducent nerve palsy – A diagnostic challenge

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ABSTRACT

Tuberculous meningitis (TBM) contributes around 5% among extrapulmonary tuberculosis (TB) and it is devastating with significant morbidity and mortality. It is associated with high frequency of neurologic sequelae and mortality. TBM has a subacute presentation with non-specific clinical signs making difficulty in early diagnosis. We report a case presenting with only ocular symptoms and no typical signs of meningitis with normal brain imaging. Real time PCR and other nucleic acid amplification methods are increasingly used for detection of mycobacterium TB from cerebrospinal fluid. When recognized early and treated promptly with appropriate anti-tuberculous medication, prognosis is good.

Keywords: Tuberculosis meningitis, Papilledema, Atypical, Abducent nerve palsy

INTRODUCTION

Tuberculous meningitis (TBM) is most devastating form of tuberculosis (TB) in the central nervous system that may cause death or severe disability up to 50–60% of affected patient.[1] It is associated with high frequency of neurologic sequelae and mortality. Vision impairment and blindness in particular is a dreadful complication.[2] TBM has a subacute presentation with non-specific clinical signs making difficulty in early diagnosis.[1] We report a case presenting with only ocular symptoms and no typical signs of meningitis with normal brain imaging

CASE REPORT

A 40-year-old female was bought to emergency department with complaints of blurring of vision in both eyes for 2 days and diplopia on seeing with both eyes for 1 day with no other complaints such as headache, nausea or vomiting, fever, neck stiffness, or any neurological symptoms. On initial assessment, she is conscious and oriented and afebrile. On ophthalmological examination, 15° esotropia in the right eye with restriction of abduction in both eyes right eye greater than left eye. Her best corrected visual acuity (BCVA) in both eyes was 6/24.

Fundus examination revealed bilateral optic disc swelling with tortuous vessels and splinter hemorrhage which is depicted in Figure 1. The test for optic nerve function which included pupillary light reflexes, color vision, visual fields were normal in both eyes. Cranial nerve examination revealed initial right sixth nerve palsy which subsequently involved the left side and other cranial nerve was found to be normal. We made a provisional diagnosis of idiopathic intracranial hypertension and suggested for investigations.

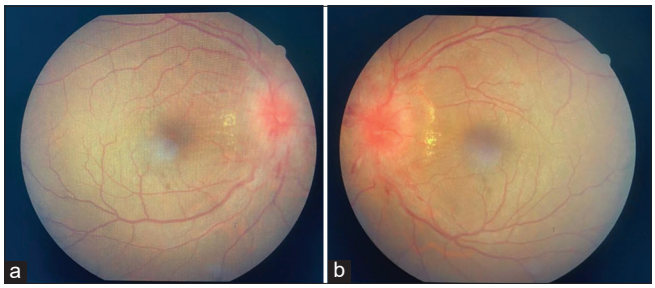

Figure 1: (a) Right eye fundus showing papilledema. (b) Left eye fundus showing papilledema.

Investigation

Laboratory investigation of complete blood count showed raised ESR of 120 mm with Mantoux reading 16 mm at 72 h. Chest x-ray was normal. Neurologist opinion was sought and suggested for contrast-enhanced CT brain which was normal with no intracranial mass, midline shift, or meningeal enhancement. MRI of brain and orbit shows normal optic nerve with no dural and meningeal enhancement, no enhancing lesion in the brain. MRI and MRV were normal with no evidence of cerebral venous sinus thrombosis. Physician carried out lumbar puncture showing clear fluid with raised opening pressure 36 cm of water with low glucose 1.4 mmol/L, protein 2.6 g/L. Cerebrospinal fluid (CSF) cytology revealed WBC count of 8×10^9/L and with lymphocyte predominance with no malignant cells. CSF for gram stain showed that no micro organisms and for acid fast bacilli were positive for *Mycobacterium tuberculosis*. Quantiferon TB Gold test was positive. CSF real-time PCR (RT PCR) came out to be positive for *M. tuberculosis* complex. She was diagnosed as TBM with papilledema with bilateral lateral rectus palsy and started on anti-TB medication. She was started with 2 months of Isoniazid (5 mg/kg/day, maximum dose 300 mg/day), Rifampicin (10 mg/kg/day, maximum dose 600 mg/day), pyrizinamide (25 mg/kg/day, maximum dose 2 g/day), and intramuscular streptomycin (20 mg/kg/day, maximum dose 1 g/day), during the initiation phase. This was followed by 7 months of continuation phase with isoniazid and rifampicin of above mentioned dose. Patient received intravenous dexamethasone (0.4 mg/kg body weight/day and tapering 0.1 mg/kg every week) and started with oral dexamethasone for next 4 weeks (4 mg/day tapering 1 mg each week).[2] Contact tracing was carried out with screening for her husband and children. One of her children was positive and was given treatment appropriately.

Outcome and follow up

On review of this patient, her BCVA improved to 6/9 with marked reduction in optic disc swelling and resolving lateral rectus palsy. There was no evidence of neurological sequelae and optic nerve function in both eyes remained normal following completion of anti-TB treatment.

DISCUSSION

TBM contributes around 5% among extrapulmonary TB and it is devastating with significant morbidity and mortality.[2] Diagnosis of TB meningitis is based on clinical features, laboratory, CSF findings, and radiological imaging. However, the diagnosis will be difficult if there is no evidence of classical clinical findings. Diagnosis will be obvious once neurological sign of advanced disease that is present but then the prognosis becomes poor.[3]

The most common presenting symptoms of TBM are headache, nausea, vomiting, fever, and anorexia. Common clinical signs include stiffness of neck, cranial nerve palsy, confusion, hemiparesis, seizures, and coma.[4] TBM is classified into three grades of severity according to the British Medical Research Council modified criteria.[5] Stage 1 disease encompasses an alert and conscious patient with no neurological deficit (Glasgow coma scale [GCS] 15). Stage 2 is manifestation of meningeal irritation with slight or no clouding of sensorium (GCS 14–11) and minor neurological deficit while Stage 3 includes major neurological deficit with severe clouding of sensorium, convulsions, and involuntary movements (GCS<10). This staging is used clinically to stratify patient and to predict prognosis.[6]

A review of 101 patients by Sinha *et al.* reported that majority of patients (80%) with TBM presented in late stages (Stage 2 and Stage 3).[7] Ocular signs such as cranial nerve palsies and papilledema were seen in last stage of TBM, 52% and 31%, respectively, with 27% of patients having visual impairment. Visual impairment of worse than 6/18, papilledema and cranial nerve palsy at initial presentation of TBM were found to be predictors of blindness and severe disability at 6 months.[7] Our patient presented atypically with ocular sign of visual impairment, papilledema, and sixth cranial nerve palsy in early stage of TBM without classical features of meningitis such as headache, vomiting, neck rigidity, and fever. However, CSF analysis typically demonstrates lymphocyte predominant pleocytosis, elevated protein, and low glucose that aids in diagnosing TBM. Adverse outcomes such as neurologic sequelae, blindness, and disability at 6 months were not seen in our patient probably as she was started with anti TB treatment early.

Optochiasmatic arachnoiditis and optochiasmal tuberculoma were the common causes for vision impairment detected with contrast MRI, 41% and 21%, respectively. Other possible causes for vision impairment in TBM include optic nerve involvement by TB lesion causes optic neuritis, tuberculous abscess, brain infarcts,

hydrocephalus, or ethambutol toxicity.[7] Our patient had normal neuroimaging findings with no obvious TB pathology causing vision impairment. There was also no evidence of optic neuritis as optic nerve function test was normal. The vision impairment in our patient could be caused by transient optic nerve head ischemia and axoplasmic stasis. The transient optic nerve head ischemia and axoplasmic stasis can happen either from direct compression or reduced perfusion of axons from an acute increase in intracranial pressure.[8,9] This may happen in fulminant cases of intracranial hypertension.

The positive Mantoux test and high ESR in our patient suggested TB infection. The gold standard for diagnosis of TBM is with culture of *M. tuberculosis* in CSF. Culture results are slow as culture with Lowenstein-Jensen media takes about 8 weeks, while semiautomated radiometric culture systems such as Bactec FX 40 take about 6 weeks to culture *M. tuberculosis*. At present, the usage of PCR for the detection of *M. tuberculosis* DNA in CSF is the widely used method as it is more rapid, sensitive, and specific.

In our patient, the quantitative RT PCR technique used for detection of *M. tuberculosis* in the CSF. This technique has been reported to have a higher sensitivity (95.8%) and specificity (100%) compared with the standard conventional PCR for patients with clinically suspected TBM.[10]

CONCLUSION

TB meningitis is often a diagnostic challenge with non-specific signs and symptoms. Delayed diagnosis can result in rapid progression of neurologic deficits and poor prognosis. RT PCR and other nucleic acid amplification methods are increasingly used for detection of *M. tuberculosis* from CSF. When recognized early and treated promptly with appropriate Anti tuberculosis medication, prognosis is good.

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.

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