

Isolated Oculomotor Nerve Palsy as Initial Presentation Of Tuberculous Meningitis

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Abstracts: Tuberculous meningitis (TB meningitis) is a subacute or chronic meningitis with diverse manifestations. Isolated oculomotor nerve palsy as initial manifestation is rare, which often makes early diagnosis difficult. The present case report demonstrates a patient with TB meningitis, who presented with isolated left oculomotor nerve palsy. A 40 year-old male presented with gradual onset left side ptosis. Neurological examination revealed isolated left oculomotor nerve palsy. Brain magnetic resonance imaging and cerebral angiography showed no diagnostic finding. Cerebrospinal fluid (CSF) study and the polymerase chain reaction (PCR) on CSF confirmed the diagnosis of TB meningitis. This report is to highlight the fact that TB meningitis should be kept in differential diagnosis for isolated third cranial nerve palsy especially in endemic countries like India. [Chakrabarti S NJIRM 2014; 5(2) :136-138]

Key Words: Tuberculous Meningitis, Oculomotor Nerve Palsy

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Introduction: Meningitis is a common manifestation of CNS tuberculosis. Early manifestations are often atypical and myriad leading to other possible diagnosis and unnecessary investigations and ultimately causing delay in diagnosis. Isolated cranial nerve palsy is one such rare manifestation of meningeal TB. MRI brain and CSF study including PCR usually point to the diagnosis. Final confirmation is response to anti tuberculous therapy. The treating physician should be alert to this uncommon etiology of third nerve palsy as it is potentially curable.

Case Report: A 40 year old non diabetic .non hypertensive, non smoker male presented with gradually increasing left sided drooping of eyelid along with binocular diplopia for last 3 months. His diplopia increased on right gaze and near vision .Ptosis or diplopia did not have any fatigability or diurnal variation. He did not have any fever, headache, neck pain, neck rigidity, vomiting or any altered mental status or other constitutional symptoms. No history was present which could suggest the presence of any underlying immunocompromised state. General examination revealed only mild pallor with no lymphadenopathy or thyroid enlargement. He had a Blood Pressure of 132/84 mmHg and a regular pulse rate of 76 beats/min. His nutritional status was average with BMI of 22.3 kg/m² .Neurological examination determined complete left sided ptosis with slight deviation of left eye to left in primary position. Pupillary dilatation (6 mm) with loss of light reflex, and restriction of all extraocular

movements in left eye except lateral deviation with eye in adducted position was noted. All these findings were attributed to oculomotor nerve palsy in the left eye. Extraocular movements in right eye as well as pupillary size and reflex were normal. Fundoscopy was normal bilaterally. No features of any other cranial nerve or bulbar or meningeal or cortical or spinal or sphincter or thyroid involvement were noted. Chest examination showed normal S1 and S2 with no added heart sounds. Bilateral vesicular breath sounds were audible with no added sounds. Abdominal examination revealed no organomegaly or free fluid. Routine laboratory parameters including blood counts, renal and liver function tests were within normal limits. Antinuclear antibody and ANA profile was negative; C3, C4, Rheumatoid Factor , Thyroid Function Test, C-Reactive Protein were within normal range. Chest Xray, Ultrasonography abdomen revealed normal study. HIV serology was nonreactive and Mantoux test revealed an induration of 7mm. MRI Brain and MR Angiography did not reveal any abnormal finding including any midbrain infarct or aneurysm. Cerebral arteriography was also normal. Examinations of the cerebrospinal fluid showed raised opening pressure with cloudy appearance. Cell count was 210/cmm with 70% mononuclear leukocytes with protein level of 182 mg/dl (normal 20-40 mg/dl), and glucose level of 28mg/dl (blood glucose level at the time of CSF study was 132 mg/dl). Gram stain and Zeihl Neilsen stain and fungal stain were negative. TB-PCR came out to be positive. Based upon positive TB PCR and supportive CSF study ,4

drug AntiTuberculousTherapy (ATT) with appropriate dosage according to Revised National Tuberculosis Control Programme (RNTCP) guidelines and his body weight which was 60 kg (comprising of Isoniazid 300mg,Rifampicin 600mg,Ethambutol 900mg,Pyrazinamide 1500mg daily) was started. Adjunctive corticosteroids in the form of oral Dexamethasone was started ,initially with 8 mg thrice daily with gradual tapering course with a view to stop steroids completely within 8 weeks.During hospital stay,his ptosis diminished and pupillary size decreased to 4 mm within 13 days of institution of ATT. Repeat CSF study done on 25th day revealed favourable CSF picture-protein 68mg/dl, glucose 46 mg/dl(blood glucose level at the time of CSF study was 112 mg/dl), cell count 11/cmm with all lymphocytes. Thus both clinical profile and laboratory results showed response to ATT. He was discharged on 28th day of admission in a stable condition with advice to followup every 15 days to assess his response to ATT and note the development of any hepatotoxicity if any. Prior to discharge, written permission was obtained from the patient regarding the reporting of his unique case.

Fig 1 Showing Patient With Isolated Oculomotor Nerve Palsy



Discussion : Initial clinical presentations of TBM include headache , fever, neck rigidity , mental status changes including coma, motor weakness and cranial nerve palsies[1-2]. Unilateral isolated oculomotor nerve palsy is found to be quite uncommon (2.2% only)[1].

Fig 2 Showing Normal Mri Brain (T2 Flair Sequence)



The presentation of unilateral oculomotor nerve palsy may be due to different diseases, including midbrain infarcts, tumor, aneurysm, temporal lobe herniation, infection, cavernous sinus thrombosis, diabetes mellitus, Tolosa-Hunt syndrome, migraine , multiple sclerosis, myasthenia gravis etc.History, clinical examination and appropriate tests including MRI brain,angiography usually point to the diagnosis.

TB meningitis is a rare but not unusual cause of oculomotor palsy[3-4].TB meningitis as a cause of third nerve palsy is first suspected from history and clinical examination especially if headache and features of meningeal irritation are found. CSF study can be very useful and positive TB PCR can clinch the diagnosis. ZN stain is rarely positive and a negative result should not be used in ruling out this possibility[5]. The prognosis of TB meningitis is related to the rapidity of initiating appropriate therapy. Delays in diagnosis and treatment result in severe neurological deficit. Early diagnosis of TBM is often difficult and the differentiation of cases from other conditions is problematic. In the present case, the lack of headache, neck stiffness, disturbance of consciousness or general malaise complicated the diagnosis. Empiric ATT may be instituted in some cases when TBM is of high clinical suspicion[6]. The case report highlights the fact that TBM should be in the list of differential diagnosis in a case of isolated third cranial nerve palsy as the timing of treatment is crucial for the prognosis

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