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Case Report

Central nervous system tuberculomata presenting as internuclear ophthalmoplegia

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ABSTRACT: Central nervous system (CNS) tuberculoma can have variable presentation depending upon the site and number of tuberculomata. We are reporting a rare case of a 15 years old girl who presented to our hospital with binocular diplopia on right gaze. Clinical examination revealed left sided internuclear ophthalmoplegia and dysdiadochokinesia and ataxia on left side. Magnetic Resonance Imaging (MRI) of brain revealed multiple tuberculomata in both cerebral hemispheres, cerebellum, left half of medulla and pons. This case highlights the need for a high degree of suspicion for CNS tuberculosis in patients presenting with internuclear ophthalmoplegia.

KEY WORDS: ***Brainstem; Tuberculoma; Internuclear; Ophthalmoplegia; Syndrome***

INTRODUCTION

Central nervous system (CNS) tuberculoma account for 5-10% of intracranial space occupying lesions in the developing world.[1] We hereby report a case of a 15 years old girl who presented with fever and binocular diplopia due to left sided internuclear ophthalmoplegia (INO). Brain MRI revealed multiple tuberculomata in both cerebral hemispheres, cerebellum, left half of medulla and pons. INO is relatively uncommon presentation of tuberculoma.

CASE REPORT

A 15 years old, female student, resident of Maharashtra, India, presented to us with history of fever for 7 days and binocular diplopia for 5 days. Diplopia occurred only with right gaze and the patient's relatives also noticed failure of the left eye to move medially while looking to the right. She also had history of mild headache and decreased appetite. She denied history of convulsions, loss of vision, imbalance while walking or any other focal neurological deficit.

Clinical examination revealed features suggestive of left sided internuclear ophthalmoplegia i.e. failure to adduct on ipsilateral side and nystagmus on contralateral side (**Figure 1**). No other cranial nerve involvement was noted. No weakness was found in any of the limbs. Deep tendon jerks were normal bilaterally and plantars showed flexor response. Dysdiadochokinesia and ataxia was noted on left side.

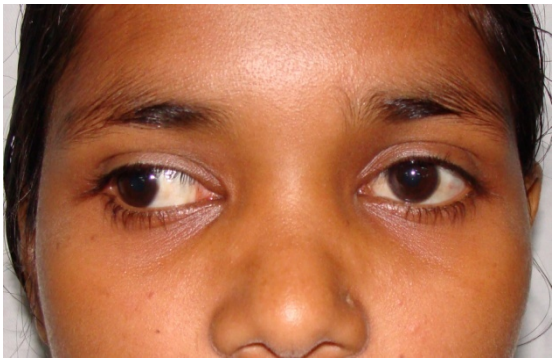

Figure 1: Demonstrating failure of left eye to adduct and nystagmus occurring in the right eye while looking right.

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Investigations revealed normal blood counts and raised erythrocyte sedimentation rate. Mantoux test was non reactive and chest radiograph was normal. MRI brain revealed multiple ring enhancing lesions in both cerebral and cerebellar hemispheres and also lesions in anterior medulla and pons. (**Figure 2 and 3a & b**)

Cerebrospinal fluid (CSF) analysis showed total cells 240, lymphocytes 80% and polymorphs 20%, proteins 120 mg% and glucose 50 mg%. CSF adenosine deaminase levels were 20 (Normal: 0 to 10). Patient was started on daily weight based 4 drug anti tuberculosis treatment (ATT), phenytoin 100mg thrice a day orally along with intravenous Dexamethasone 8mg 8 hourly. Later Dexamethasone was switched over to oral prednisolone 1mg/kg which was tapered after 6 weeks. Fever and headache responded immediately after starting the treatment. No worsening of symptoms was noted. However, adduction of left eye improved gradually after 4 weeks of steroids and ATT and diplopia disappeared by 6 weeks of treatment. Currently, patient is on 4 drug ATT and is being followed up on outpatient basis.

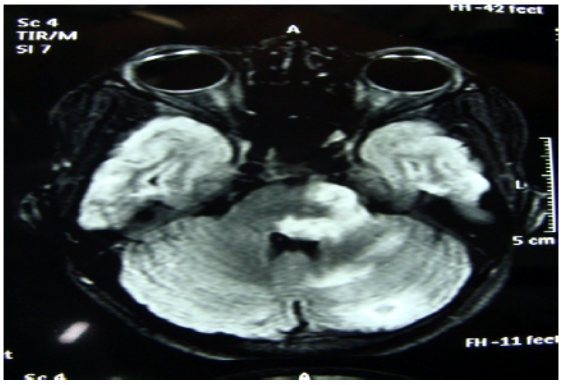
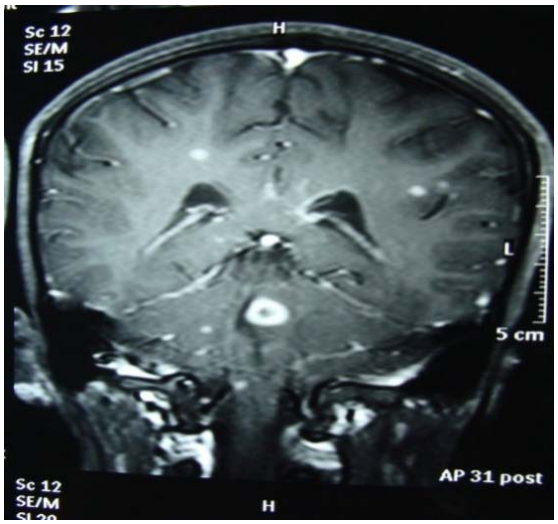


Figure 2: T2W images with FLAIR showing perilesional oedema along tuberculomata in left half of dorsal pons and left cerebellar hemisphere



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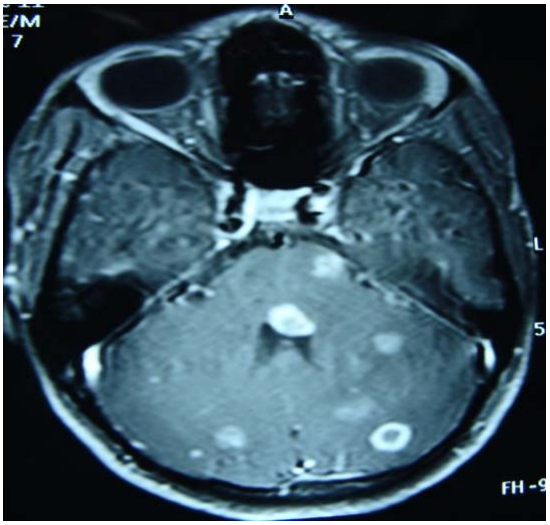

3b

Figure 3a & b: Post contrast ring like enhancement seen with multiple lesions seen in bilateral cerebral and cerebellar hemisphere and pons and medulla

DISCUSSION

Neurological tuberculosis comprises of 10-15% cases of extra pulmonary tuberculosis and occurs frequently in children. CNS tuberculoma can have varied clinical presentation ranging from mild headache, convulsions, hemiplegia, cranial nerve palsies and also rarely with panhypopituitarism[2], bilateral internuclear ophthalmoplegia[3], one and half syndrome[4] & eight and half syndrome. Infratentorial tuberculomas are more common in children and may present with brainstem syndromes, cerebellar manifestations and multiple cranial nerve palsies.[3] The MRI features of tuberculoma may differ based on presence or absence of caseation within the granuloma. The non-caseating granulomas appear hypointense on T1-weighted images, hyperintense on T2-weighted images and show homogenous enhancement after contrast administration. Caseating granulomas appear hypo- or isointense on T1 & T2 weighted images and show ring enhancement after gadolinium contrast with variable degree of perilesional oedema. The cytotoxic oedema appears hyperintense on T2 weighted images high intensity on diffusion weighted imaging (DWI) and low apparent diffusion coefficient (ADC). If central liquefaction of the caseous material occurs, tuberculoma appears centrally hypointense on T1- and hyperintense on T2-weighted images with a peripheral hypointense ring which represents its capsule. These lesions too show ring enhancement on contrast administration.[5]

Internuclear opthmaloplegia (INO) is caused by lesion in the medial longitudinal fasciculus (MLF) in the pons. Due to lesion in the MLF, ipsilateral

medial rectus fails to receive the signal to contract when paramedian pontine reticular formation (PPRF) and sixth nerve nucleus act to intiate lateral gaze. This leads to characteristic presentation of INO i.e. failure to adduct of ipsilateral eye and nystagmus of the contralateral eye. However the earliest detectable sign of INO is slowing of adducting saccades compared to abducting saccades demonstrated by rapid refixation.[6]

The lesion in the medial pons involves fibers of PPRF as well as crossing fibers of contralateral MLF. Due to involvement of PPRF and MLF, patient develops ipsilateral gaze palsy and ipsilateral INO. The net result of this lesion is complete horizontal gaze palsy of ipsilateral eye and preservation of abduction of contralateral eye. Hence the name "One and half syndrome" as there is complete gaze palsy of ipsilateral eye and half gaze palsy of contralateral eye. Combination of one and half syndrome with Facial nerve palsy is called Eight and half syndrome (1½ + 7 = 8½).[6] In a review of 410 cases of INO, it was found that commonest etiology of INO was infarction followed by multiple sclerosis and other unusual causes like trauma, tentorial herniation, vasculitis, tumor, infections like brucellosis, neurocysticercosis, tuberculosis, syphilis, lymphoma, basilar and vertebral artery aneurysm, A-V malformations, etc.[7]

CONCLUSION

Internuclear ophthalmoplegia, although rare, can be the presenting feature of CNS tuberculosis due to tuberculoma in the brainstem. Brain MRI is a good diagnostic tool to ascertain the etiology of INO. Early treatment with steroids along with ATT results in a good outcome and rapid recovery from symptoms.

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