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Case Series

Atypical presentations of Neurotuberculosis.

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Abstract

Tuberculosis is considered to be one of the oldest of human diseases. It virtually involves majority of organ system in human body. Neurotuberculosis is known to involve meninges brain parenchyma, vertebral column, spinal cord either individually or in various combinations. Clinical features of neurotuberculosis depend on site of involvement. Atypical presentations are known. We present a case series of 3 patients of neurotuberculosis with atypical clinical presentation.

Key words: Neurotuberculosis, Brain parenchyma, Vertebral column.

Introduction

Tuberculosis of nervous system is caused by the human strain of Mycobacterium tuberculosis. CNS is affected secondarily from a primary focus elsewhere, most probably the lung (1). Tubercle bacilli hematogenously spread from lung to involve the central nervous system. The highly vascular choroid plexus is the common site for the tubercule formation . The burnt of the disease usually falls on the basal meninges. However secondary vascular changes and perenchymatous changes of the brain are equally characteristic and presents with different clinical manifestations. Neurotuberculosis can present with myriad of atypical features which should be kept in mind while encountering such cases.

Case Report

Case 1: A 22 year, male presented to us with complaints of headache of 15 days duration associated with intermittent jerky movement of left upper and lower limb which started in the left hand, marched to arm then to left side of the body including left lower limb. This abnormal sensation lasted for 2-3 sec and then used to subside. Patient had 15- 20 such episodes per day. There was no history of fever, diplopia, vomiting, tonic clonic seizures or urinary incontinence. No history suggestive of any cranial nerve abnormality.

Patient was conscious, cooperative and well oriented to time place and person. He was afebrile with pulse rate of 80/min, regular, RR-18/min, BP-120/70 mm of Hg. There was no pallor, icterus, oedema or lymphadenopathy. His cardiovascular, respiratory and per abdominal system exam was normal. His CNS exam revealed mild left sided weakness with left plantar extensor. Sensory system examination was normal. MRI brain with contrast revealed meningeal enhancement with ring enhancing lesion in right parietal region (fig 1&2). CSF examination revealed 300 cells/hpf, all of them were lymphocytes, protein- 160 mg/dl, sugar- 30 gm/dl and CSF, ADA was positive. Diagnosis was neurotuberculosis (tuberculoma with meningitis) and anti tubercular treatment was started with Inj. Dexamethasone 10 mg IV stat and 10 mg IV 6 hourly for 4 days followed by oral Prednisolone over 2 months in a tapering dose.

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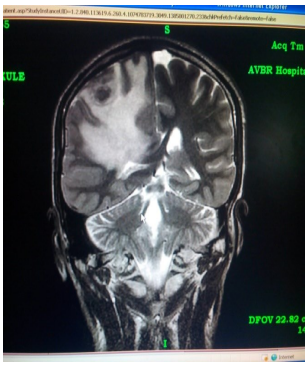
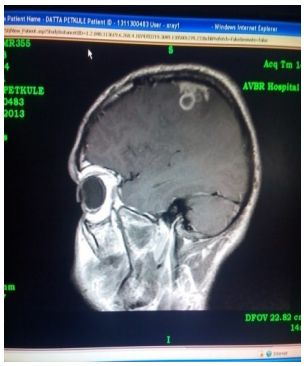

Fig -1 & 2: showing contrast enhancing MRI images of brain showing ring enhancing lesion in Right high parietal region with meningeal enhancement.

Case 2: A 35 year, male presented to us with history of 15 days of headache and intermittent abnormal behaviour as told by relatives. Headache was unilateral, severe, predominantly retro orbital lasting for 30- 40 min with 4- 5 episodes per day. As per relatives during attacks patient used to be conscious but in a bit with drawl and non interactive. He used to be coherent and there were no seizures.

There was no history of vomiting, diplopia, blurred vision, urinary continence. No history suggestive of any cranial nerve abnormality. Patient was conscious, cooperative and well oriented to time place and person. He was afebrile with pulse rate of 78/min, regular, RR-15/min, BP- 110/80 mm of Hg. There was no pallor, icterus, oedema or lymphadenopathy. His cardiovascular, respiratory, per abdominal system and CNS exam was normal. Sensory system examination was normal.

On investigations his haemoglobin was 14 gm%, TLC- 9800 cells/ cu mm, platelet- 2.21 lack/ cumm. His kidney and liver function test were normal. Patient was started on oral analgesics and observed. On 2nd day of admission it was noticed that patient suddenly became withdrawn from surrounding. He was conscious but not responding to verbal commands. He had automatism in form of lip smacking and repeated picking movements of shirt button and bed sheet. This episode lasted for 6-8 minutes. Patient was unaware of such episode when it got over. So he was provisionally diagnosed as psychomotor seizure.

His CECT brain revealed meningeal enhancement. His CSF exam revealed 500 cells/ cumm, 90% lymphocytes with protein 300 mg/dl and sugar- 40 mg/ dl. So his diagnosis was confirmed to be tubercular meningitis and anti tubercular treatment was started with Inj. Dexa 10 mg IV stat and 10 mg IV 6 hourly for 4 days followed by oral Prednisolone over 2 months in a tapering dose.

Case 3: A 35 year old female patient presented with complaints of diplopia since 7 days and inability to open right eye since 6 days. There was no history of fever, headache, vomiting or seizures.

On general examination she was conscious, cooperative oriented to time, place and person. Her pulse rate was 100/min, regular, BP- 130/80 mm of Hg. There was no pallor, icterus, oedema. Her RS, CVS, PA examination was normal. Her CNS exam revealed normal higher function with partial right 3rd cranial nerve palsy (partial ptosis, deviation of rt. eye ball down and laterally with normal papillary reaction). Motor and sensory exam were normal. Slight neck stiffness was present; Kernig's and Brudzinski's signs were absent jolt accentuation maneuver elicited headache. Her CSF revealed 900 cells/hpf with 90% of them were lymphocytes, sugar- 35 mg/dl and protein- 200 mg/dl. Her MRI brain with contrast revealed granuloma in right caudate and lentiform nucleus as shown in (Fig 3).

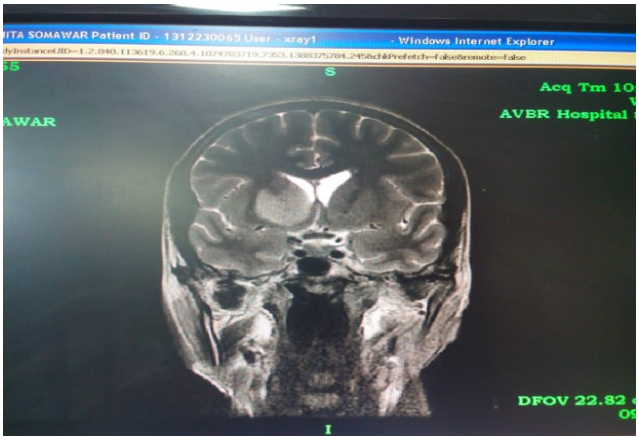

Fig –3: Coronal section of MRI brain with contrast showing tubercular granuloma at Rt. caudate and lentiform nucleus.

Her diagnosis was confirmed to be neuro tuberculosis (meningitis and granuloma) and anti tubercular treatment started with Inj. Dexamethasone 10 mg IV stat and 1o mg IV 6 hourly for 4 days followed by oral prednisolone over 2 months in a tapering dose.

Discussion

Tuberculosis affects one third of total world's population and is a leading cause of morbidity and mortality [2]. Tuberculosis of CNS constituted 5% of extra pulmonary tuberculosis patients. Late and atypical forms of neurotuberculosis are common which invariably leads to delay in diagnosis and increases mortality and morbidity. Nearly are due to mycobacterium tuberculosis. Usually neurotuberculosis occurs

via hematogenous spread from a primary focus which invades meninges and/ or brain parenchyma forming rich focus. The bacilli remain dormant for many years and neurotuberculosis occurs by reactivation when host immune response decreases. Tubercular meningitis occurs when subependymal or subpial or intracerebral tubercle ruptures.

The basic pathogenesis of presentation of neurotuberculosis is due to meningitis which leads to meningeal signs, cranial nerve palsies and hydrocephalus. Alteration of sensorium, seizures, hypothalamic and brain stem signs can occur due to extensive infiltration of brain parenchyma. Focal neuro deficit can occur due to strokes from vasculitis or from tuberculoma.

Cerebral oedema and raised ICT are due to hypersensitivity response to tubercular antigen [3]. There are many atypical presentations of neurotuberculosis which can confuse a physician where a high degree of suspicion is kept. Some of the atypical presentations of neurotuberculosis are focal neurodeficits and aphasia, multiple cranial nerve palsies, visual loss, focal and generalized seizures. Some patients may have isolated athetoid or choreoathtoid movement. Some patients may present with myoclonic seizures, atonic drop attacks, gait ataxia and hearing impairment [4]. Our cases were typical presentations of atypical manifestations of neurotuberculosis. The first case presented with cluster like headache with psychomotor epilepsy. We extensively reviewed the literature and this may be the first case of CNS tuberculosis presenting as trigeminal autonomic cephalalgia like picture and psychomotor seizure.

Our second case highlighted pure sensory motor epilepsy with Jaksonian march pattern. Third case manifested as pupillary sparing partial 3rd cranial nerve palsy. A high degree of clinical suspicion, neuroimaging and CSF exam confirmed CNS tuberculosis in these cases and treatment initiated.

The present study concludes that atypical manifestations of neurotuberculosis are not uncommon. Any atypical presentation with or without fever or signs of meningeal irritation should also be investigated keeping neurotuberculosis in mind because timely imaging and CSF studies will confirm the diagnosis and early initiation of treatment will benefit the patient.

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