Print ISSN: 2319-2003 | Online ISSN: 2279-0780

IJBCP International Journal of Basic & Clinical Pharmacology

doi: 10.5455/2319-2003.ijbcp20140240

Case Report

Alcoholism with central pontine demyelination: a case report

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Received: 28 October 2013
Revised: 17 November 2013
Accepted: 25 November 2013

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ABSTRACT

Central pontine myelinolysis is a non-inflammatory demyelinating disease characterized by loss of myelin with relative neuron sparing, associated with rapid correction of hyponatremia and sometimes hypernatremia or chronic alcoholism. We are reporting a case of 52 year old male patient who was chronic alcoholic from past 20 years, presented to us with complaints of altered sensorium and dysarthria of 5 days duration .He was investigated and diagnosed as case of central pontine myelinosis associated with chronic alcoholism.

Keywords: Central pontine myelinolysis, Demyelinating disease, Chronic alcoholism

INTRODUCTION

Central Pontine Demyelination (CPM) is neurological disease caused by severe damage of myelin sheath of nerve cell in brain stem more precisely in Pons. It is iatrogenic in etiology characterized by acute paralysis, dysphagia, dysarthria and other neurological symptoms.[1] It most commonly presents as complication of treatment of hyponatremia although patient of chronic alcoholism can present with it. Central Pontine Demyelination was first described by Adam and college in 1959 as a disease affecting alcoholics and malnourished.[2] Many follow up studies have shown that hyponatremia or rapid correction of hyponatremia leads to CPM, still nutritional deficiency and chronic alcoholism remain important causes of the same and should be considered as a possible cause in malnourished or alcoholic patients. CPM can lead to confusion in diagnosis in many patients because of bizarre neuropsychiatric presentation. We hereby present a similar case with a diagnostic dilemma that weather it was hepatic encephalopathy, Wernicke's encephalopathy or alcohol withdrawal. However on follow up turned out to case of CPM.

CASE REPORT

A 52 year old male patient presented to us with complaints of altered sensorium and dysarthria of 5 days duration. He also had jaundice from 4 days associated with melena. His illness was insidious in onset and was rapidly progressive. He had history of chronic alcohol consumption, from 20 yrs. however there was no past history suggestive of chronic liver disease.

On examination he was in delirium and was disoriented. Pulse - 80/min, BP - 90/50 mmHg. He had icterus He was running fever of 99°F. On nervous system examination, he was in a confused and disoriented state with GCS E-3 M-4 V-5. Pupils were equal and reacting to light bilaterally with bilateral horizontal nystagmus. He was moving all 4 limbs in response to painful stimulus. He also had neck rigidity and Kernig's sign was positive.

Other system examination including abdomen was within normal limits. His lab reports revealed Hb - 10.3 gm/dl, total leukocyte count - 7900/mm3, differential leukocyte count - P67L29E4, S. bilirubin - 3.9mg/dl, ALT - 58 IU/dl, AST - 48 IU/dl , ALP - 31.9 IU/dl, Na 135meq/dl K - 2.7 meq/dl. His ultrasound abdomen was normal. As patient was in altered sensorium and had signs of

meningism, his CT Brain and CSF analysis was done, and both were normal. In view of history of chronic alcoholism, jaundice and melena, he was provisionally diagnosed as a case of hepatic encephalopathy with a possibility of Wernicke's encephalopathy and was treated with high bowel wash, lactulose, antibiotics and high dose thiamine. After 4-5 days his sensorium improved in the form that he had spontaneously eye opening and followed verbal commands. However his dysarthria and bilateral horizontal nystagmus persisted. He had muscle power of grade 3/5 in all four limbs. There was no sensory deficit clinically. In view of above clinical findings MRI brain was done which revealed changes suggestive of likely pontine and extrapontine osmotic demyelination with old ischemic insult in right cortical and subcortical occipital region.

DISCUSSION

Central pontine myelinolysis is a non-inflammatory demyelinating disease characterized by loss of myelin with relative neuron sparing. It may be associated with demyelination elsewhere in the central nervous system. The common extrapontine sites are the putamen, caudate nucleus, midbrain, thalamus and subcortical white matter (extra pontine myelinolysis).[3] In the past before MRI, these cases were only definitively identified at autopsy because CT scans are not good at visualizing CPM compared with MRI. Over 75% of cases are associated with rapid correction of hyponatremia and sometimes hypernatremia or chronic alcoholism.[4,5] A good correlation was found between development of central pontine myelinolysis and the correction of sodium more rapidly than 12 meq/day. A number of other conditions have been associated with central pontine myelinolysis such as orthotopic liver transplantation, chronic renal failure, dehydration, diabetes mellitus, sepsis, advanced malignancy and acute hemorrhagic pancreatitis.[6]

The pathophysiological mechanism of CPM is unknown. The central pons is anatomically unusual in that gray matter and white matter are intermixed, and this feature is presumed to account for the vulnerability of this area to osmotic injury. In most areas of the brain, oligodendrocytes are embedded within white matter and physically isolated from the capillary-rich gray matter. It has been proposed that, in CPM, oligodendrocytes in close proximity to gray matter are exposed to a myelinotoxic substance as a result of osmotic stress.[7]

Since both the pontine and extra pontine sites involved have a rich grey and white matter interface, it has been hypothesized that a rapid osmotic change causes an endothelial injury in the more vascular grey and white matter which induces the release of myelinolytic factors that damage the adjacent white matter.[8] The term osmotic myelinolysis is currently favored for this condition. The initial symptoms may be weakness, confusion and dysarthria. In severe cases there is spastic quadriparesis and pseudo bulbar palsy.

This may evolve within 3 - 10 days into a locked-in-state (pseudo coma).

Death within 2 - 3 months is the usual outcome in severe cases. The 6-month survival rate is 5 - 10%.[5] Mochizuki H et al showed a favorable outcome in 9 patients who had CPM following alcohol binge.[9] The imaging features are due to increased water content in the affected areas. Transverse pontine fibers are more severely affected, compared with descending corticospinal tracts. Lesions appear hypo intense on T1WI and hyperintense on T2WI and show varied enhancement following contrast administration (Figure 1 and Figure 2).

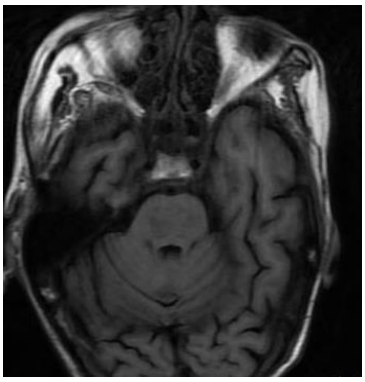

Figure 1: Lesions appear hypo intense on T1WI and hyperintense.

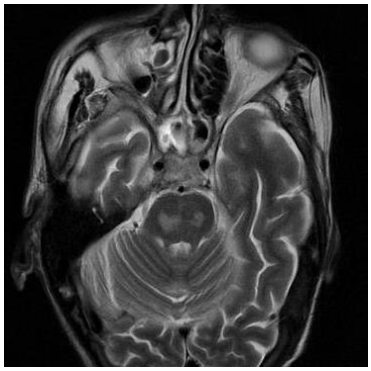

Figure 2: Lesions appear hypo intense on T2WI and hypointense.

Differential diagnosis of central pontine myelinolysis includes infarct, metastasis, glioma, multiple sclerosis, encephalitis, radiotherapy and chemotherapy. However concomitant involvement of the pons and basal ganglia is specific for osmotic myelinolysis. In such cases the imaging differential diagnosis includes hypoxia, Leigh disease and Wilson's disease. The classical history combined with imaging findings distinguishes osmotic myelinolysis from the rest of the conditions.

A number of therapeutic approaches have been tested, although no specific therapy exists. Correction of serum sodium should not exceed 12meq/24h. Recovery varies from no improvement to substantial recovery, although the outcome of this condition is frequently fatal.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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doi:10.5455/2319-2003.ijbcp20140240
Cite this article as: Arora R, Singhal R, Virmani SR, Gupta A. Alcoholism with central pontine demyelination: a case report. Int J Basic Clin Pharmacol 2014;3:230-2.