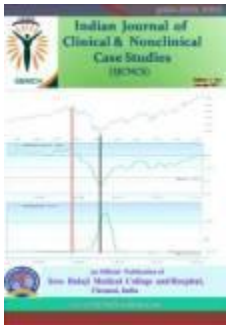

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CENTRAL PONTINE MYELINOSIS WITH HYPOKALEMIA–A RARE CASE REPORT

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ABSTRACT:

Central pontine and extra pontine myelinosis is commonly related with a rapid correction of sodium. We describe a patient with acute confusional state, dysarthria and quadriparesis with predominant hypokalemia showing mild hyponatremia. MRI imaging revealed the occurrences of pontine and extrapontine lesions which disappeared with symptomatic treatment. Hypokalemia was the predisposing factor in the pathogenesis for Central pontine myelinolysis (CPM).

Key Words: Central Pontine Myelinosis, Hypokalemia, Hyponatremia

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INTRODUCTION:

Central pontine myelinolysis (CPM) is a rare demyelination syndrome involving the centre of the basispontis. Very rarely similar lesions are seen involving extra-pontine structures called as extra-pontinemyelinolysis (EPM). These two constitute 'osmotic demyelination syndrome' (ODS) characterizedpathologically by non-inflammatory demyelination of various brain structures with sparing of axons[1]. Common predisposing factors in a clinical setting of CPM are alcoholism, malnutrition, liver disease and hyponatremia. CPM in a hyponatremic patient can develop only if it is less than 120meq/L hrs for more than 48 hrs or it is corrected with hypertonic salines[1]. We report a rare case of central pontine and extra-pontine myelinolysis associated with hypokalemia and mild hyponatremia wherein no rapid correction of sodium was done.

CASE REPORT:

A 73year old male presented with acute confusional state. It was preceded by episode of high grade fever and associated with multiple episodes of projectile vomiting. There was no history of diabetes/hypertension/ seizure/uncosciouness/drug intake/alcoholism/ salt estriction. On examination the patient was found to be well nourished. He was afebrile with a blood pressure of 140/88 mmHg. He was drowsy and talking irrelevantly. Fundus was unremarkable. Cranial nerves were normal and he was moving all four limbs. He had mild limb incoordination which he had recovered soon. Biochemical parameters and blood counts were normal. Blood sugar was within normal limits. Blood urea was 21mg/dl, Serum creatine was 1.1mgm/dl, and Vit B12 level was 523 pg/dl. Thyroid and liver profile were normal. Mild hyponatremia (124-129 meq/l) but hypokalemia (2.3- 2.4 meq/l) were found at repeated occasions. Plasma osmolality was 288-290mmol/L throughout. CSF parameters were normal. MRI revealed the occurrence of a relatively well defined hyperintense lesion on T2W and FLAIR sequences in central pons (Figure 1a). Similar lesions were seen at periphery of left cerebellar hemisphere and a small lesion at right cerebellar hemisphere inferomedial to cerebellar tonsil (Figure 1a). Post contrast these lesions showed no enhancement (Figure 1b). Patient was treated conservatively with gradual correction of potassium and sodium as per