CENTRAL PONTINE MYELINOSIS WITH HYPOKALEMIA–A RARE CASE REPORT

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ABSTRACT

Central pontine and extra pontine myelinosis is commonly associated rapid correction of sodium. We describe a patient with acute confusional state, dysarthria and quadriparesis with predominant hypokalemia with mild hyponatremia. MRI imaging revealed the pontine and extrapontine lesions which disappeared on repeat study with symptomatic treatment. Hypokalemia as the predisposing factor in the pathogenesis for CPM is highlighted.

Key Words: Central Pontine Myelination, Hypokalemia, Hyponatremia

INTRODUCTION

Central pontine myelinolysis (CPM) is a rare demyelination syndrome involving the centre of the basis pontis. Very rarely similar lesions are seen involving extra-pontine structures called as extra-pontine myelinolysis (EPM). These two constitute ‘osmotic demyelination syndrome’ (ODS) characterized pathologically by non-inflammatory demyelination of various brain structures with sparing of axons (Martin 2004). 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 120 meq/L hrs for more than 48 hrs or it is corrected with hypertonic salines aggressively (Martin 2004). We report a case of central pontine and extra-pontine myelinolysis associated with hypokalemia and mild hyponatremia wherein no rapid correction of sodium was done.

CASES

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/uncosciousness/drug intake/alcohol ism/ salt estriction. On examination patient was 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 recovered soon. Biochemical parameters Blood counts were normal. Blood sugar within normal limits. Blood urea 21mg/dl, Serum creatine 1.1mgm/dl, Vit B12 level 523 pg/dl. Thyroid and liver profile normal. Mild hyponatremia (124-129 meq/l) but hypokalemia(2.3-2.4 meq/l) were found at repeated occasions. Plasma osmolality 288-290mmol/L throughout. CSF parameters were normal. MRI revealed 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 protocols. The patient recovered clinically Repeat MRI done after two weeks after correction of electrolytes showed almost complete resolution of the lesions (Figure 2).

DISCUSSION

Extra-pontine myelinolysis is a uncommon variety of osmotic demyelination syndrome involving extrapontine structures with or without involvement of the pons. A variety of sites may be involved e.g.,

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Figure 1a: MRI show foci of bright signals in left inferior cerebellar hemisphere, right cerebellar hemisphere and central pons in FLAIR sequence

Figure 1b: Same lesion showing nil enhancement on gadolinium

Figure 2: MRI taken a week after show almost complete resolution of bright signals of cerebellum in flair sequence

pons, cerebellum, lateral geniculate body, external capsule, hippocampus, putamen, cerebral cortex, thalamus, caudate nucleus, claustrum, internal capsule, midbrain, etc (Gocht, 1987). Microscopically, the lesions show degeneration and loss of oligodendrocytes with preservation of axons and nerve cells without any evidence of inflammation (Ropper, 2005). It has been shown that myelinoysis can follow a rise in serum sodium from a hyponatremic levels regardless of the cause of hyponatremia or method of
correction. The patients usually go through a biphasic clinical course: initial encephalopathic illness due to hyponatremia then recovering rapidly with correction of hyponatremia only to deteriorate several days later with dysarthria and dysphagia along with flaccid quadriplegia which later becomes spastic (Martin 2004). Our case showed changes of pontine and extrapontine myelinosis on imaging studies with significant hypokalemia and mild hyponatremia, with no history of correction with hypertonic saline in the recent past. Hypokalaemia has been reported as a possible trigger in osmotic central pontine and extrapontine myelinosis. It is seen that CPM tends to occur in hyponatraemia complicated by hypocalcaemia, because a decreased concentration of Na, K-ATPase in endothelial cell membrane during hypokalaemia predisposes the cell susceptible to injury by osmotic stress associated with the rapid rise in the serum sodium (Sugimoto, 2003). A published reviewed cases in which the values of both Na+ and K+ were given, found that 66 of the 74 cases reported were hypokalaemic (Lohr, 1994). We propose to highlight with this index case that even hypokalemia can also be an important predisposing factor in central and extrapontine myelinosis. Rapid clinical and radiological recovery in our patient and its association with hypokalemia makes it unusual.

REFERENCES


