Abstract:

The first case of extra pontine myelinolysis was described in 1987 and before that the concept of central pontine myelinolysis was being widely used. When extra pontine myelinolysis and central pontine myelinolysis occur simultaneously it is known as the osmotic demyelination syndrome. The etiology of extra pontine myelinolysis is most commonly a metabolic insult to the brain in the form of over or rapid correction of hyponatremia (a change in serum sodium more than 25mmol/litre within 48 hours). Moreover it is said that some predisposing factors attribute to its severity like Alcoholism, Malnourishment, Sepsis, Severe burn, SIADH, Chest infection etc. Extra pontine myelinolysis is more commonly found in chronically hyponatremic patients in whom it occurs 7-14 days after acute osmotic shift. This report describes a 57 years old male with extra pontine myelinolysis with history of rapid correction of hyponatremia who presented with acute parkinsonian symptoms.

Case Report:

A 57 years old male, known case of type 2 diabetes mellitus and hypertension had history of multiple episodes of vomiting 15 days back before coming to our hospital. He was admitted in a local hospital where on biochemical evaluation his serum sodium was found to be 104 mmol/lit which was corrected to 130 mmol/lit within 24 hours. The patient was improving and was being discharged after a 5 day stay in the hospital. Then after 2-3 days the patient developed tremors of hands and tongue, dysarthria and stiffness of limbs. After admission to our hospital, on doing laboratory investigation he was found to have dyselectrolytemia which were corrected accordingly. On neurological examination the patient followed simple commands; cog wheel rigidity was positive. GCS was found to be E4V3M5. MRI brain was done which showed features suggestive of extra pontine myelinolysis. The clinical features along with MRI findings led the patient to be diagnosed as a case of extra pontine myelinolysis with acute Parkinsonian symptoms. He was managed in our set up conservatively. Physiotherapy training was given and the patient was gradually improving and was discharged by explaining the nature of the illness to the patient party.

Discussion:

Extra pontine myelinolysis is a rare, acute, demyelinating process that involves the

areas of the brain outside the pons. It is most commonly found in association with central pontine myelinolysis which involves the pons. When osmotic demyelination occurs, it can be lethal as it is usually irreversible and has no proper management. Thus prevention is the key. It is generally suggested that the rate of correction of serum sodium should not be more than 1-2mmol/litre/hour during first few hours and not more than 8-10mmol/lit in first 24 hours.

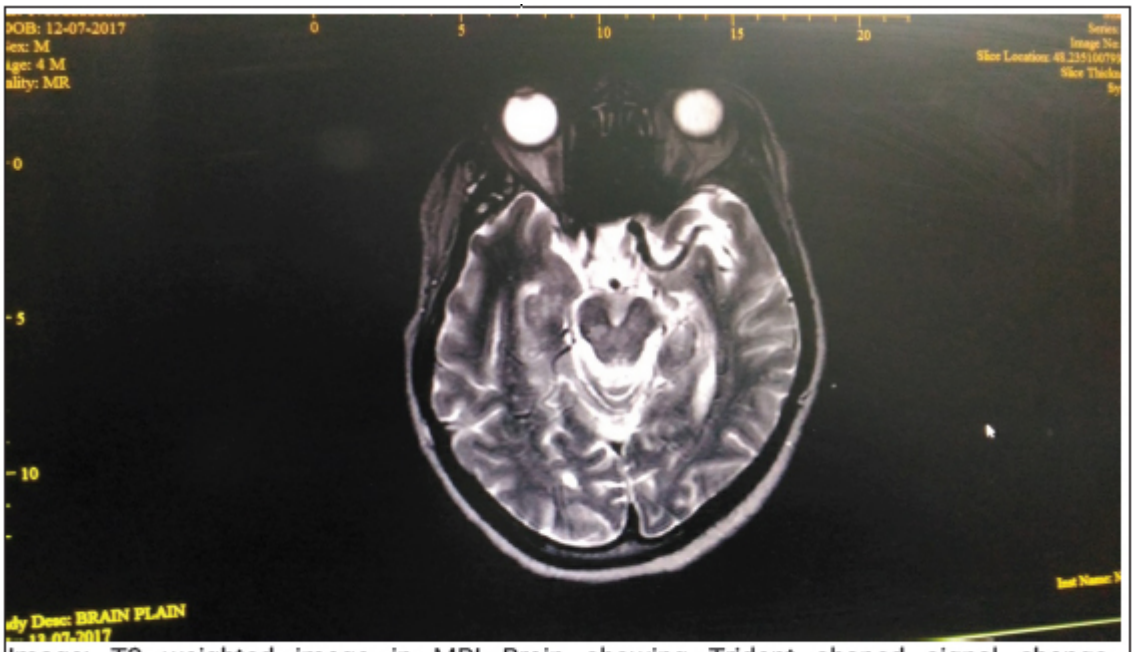

Image: T2 weighted image in MRI Brain showing Trident shaped signal change characteristic of extra pontine myelinolysis (The patient had central pontine myelinolysis as well)

Rapid correction of acute hyponatremia causes rapid osmotic shifts of fluid which leads to hypernatremia and eventually shrinkage of brain cells and demyelination. When hyponatremia develops gradually that is in chronic hyponatremia the brain cells can compensate by decreasing intracellular osmolarity through the loss of osmolytes and thereby limiting neurologic dysfunction. So during rapid correction of chronic hyponatremia, the regeneration of these osmolytes lag and cerebral dehydration occurs. Microscopically the lesion shows loss of oligodendrocytes with preservation of axons unless the lesion is highly advanced. It may involve the basal ganglia, thalamus, cerebellum and subcortical white matter. On MRI, T2 weighted image shows trident shaped signal changes. In our case, the history of low sodium with subsequent rapid correction, the clinical symptoms and the basal ganglia changes in MRI were strongly in favour of extra pontine myelinolysis. Extra pontine myelinolysis may manifest as postural limb tremors, myoclonic jerks, parkinsonian

presentation, dystonia, catatonia or pyramidal dysfunction. Whatever may be the cause of hyponatremia whether acute or chronic, it has to be corrected slowly.

Conclusion:

In conclusion, hyponatremia may be the manifestation of various disorders. However osmotic demyelination is preventable if we correct serum sodium level slowly and if the correction is with hypertonic saline we should monitor serum sodium frequently.

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