Research Paper

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Case Report of Central Pontine Myelinolysis

Medicine

KEYWORDS :

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Introduction

Central pontine myelinolysis (CPM), also known as Osmotic demyelination syndrome, is a neurological disease caused by severe damage of the myelin sheath of nerve cells in the brainstem, more precisely in the area termed the *pons*, predominately of iatrogenic etiology.Clinical presentation of CPM is heterogeneous and depend on the regions of the brain involved. Observable immediate precursors may include

- seizures,
- disturbed consciousness,
- gait changes
- and decrease or cessation of respiratory function.

Frequently observed symptoms in this disorder are acute para- or quadraparesis, dysphagia, dysarthria, diplopia, loss of consciousness, and other neurological symptoms associated with brainstem damage. The patient may experience locked-in syndrome where cognitive function is intact, but all muscles are paralyzed with the exception of eye blinking. These result from a rapid myelinolysis of the corticobulbar and corticospinal tracts in the brainstem.

Case Report

A 50 year old male, a chronic alcoholic patient admitted at our tertiary care centre with chief complaint of severe abdominal pain at epigastric region since 1 day which was associated with nausea,vomiting. On admission vitals were normal and in systemic examination tenderness at epigastric region present. Routine investigation were normal and serum lipase was very high. Patient was admitted in ICU and treated as standard protocol of acute pancreatitis, patient gradually improved symptomatically.

On second day patient had complaint of altered sensorium in term of irrelevant talking and increase sleepiness and also complaint of bilateral lower limb weakness.His vitals were normal but in systemic examination there was epigastric pain had been improved and in CNS examination patient was drowsy,Power 4/5 in bilateral lower limb with normal response to deep tendon reflex,normal sensorium,plantar flexion. MRI Brain with whole spine screening advised.In that finding suggestive of CPM (central pontine myelinolysis) and also extrapontine myelinolysis. At that time there were no electrolyte imbalance(serum sodium level normal). Patient treated symptomatically for 2days and patient improved.

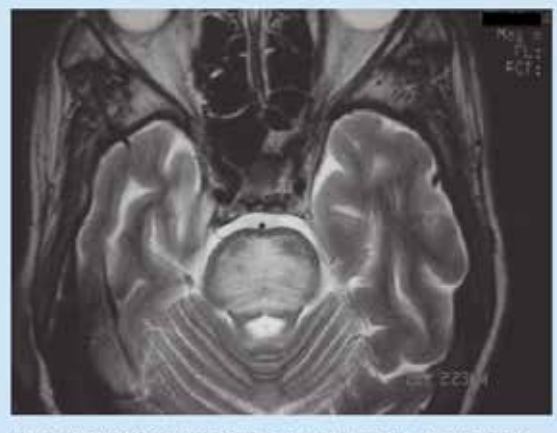

Fig. 1. Axial-T2W MR scan showing hyperintense signal in the central pons with predominant involvement of transverse pontine fibres and relative sparing of descending corticospinal tracts.

Discussion

Central pontine myelinolysis presents most commonly as a complication of treatment of patients with profound, life-threatening hyponatremia . It occurs as a consequence of a rapid rise in serum tonicity following treatment in individuals with chronic, severe hyponatraemia who have made intracellular adaptations to the prevailing hypotonicity. Hyponatremia should be corrected at a rate of no more than 12-20 mmol/L of sodium per day to prevent central pontine myelinolysis.

Although less common, it may also present in patients with a history of chronic alcoholism or other conditions related to decreased liver function. In these cases, the condition is often unrelated to correction of sodium or electrolyte imbalance.

It can be difficult to identify using conventional imaging techniques. It presents more prominently on MRI than on CT, often taking several weeks after acute onset of symptoms before it becomes identifiable. Imaging by MRI demonstrates an area of high signal return on T2 weighted images.

Prevention and treatment

To prevent CPM from its most common cause, overly rapid reversal of hyponatremia, the hyponatremia should be corrected at a rate not exceeding 10 mmol/L/24 h or 0.5 mEq/L/h; thus avoiding hypernatremia.Alcoholic patients should receive vitamin supplementation and a formal evaluation of their nutritional status.Once demyelination of the pons has begun, there is no cure or specific treatment. Care is supportive, with the goal of preventing complications like aspiration pneumonia or deep vein thrombosis. Alcoholics are usually given vitamins to correct for other deficiencies.

Prognosis

The prognosis is overall poor. However, recent data indicate that the prognosis of critically ill patients may even be better than what is generally considered, despite severe initial clinical manifestations and a tendency by the intensivists to underestimate a possible favorable evolution. While some patients die, most survive and of the survivors, approximately one-third recover; one-third are disabled but are able to live independently; one-third are severely disabled.[Permanent disabilities range from minor tremors and ataxia to signs of severe brain damage, such as spastic quadriparesis and locked-in syndrome. Some improvements may be seen over the course of the first several months after the condition stabilizes.The extent of recovery depends on how many axons were damaged.

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