International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Central Pontine Myelinosis - A Case Report

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ABSTRACT
Central pontine myelinosis(CPM) is a rare neurologic condition most frequently caused by the rapid correction of hyponatremia due to demyelination which is non inflammatory. Here we report a case of 55 years old male presented with sudden onset of abnormal movements of the whole body with reduced alertness and difficulty in swallowing. On evaluation MRI brain shows lesions in the pons which is suggestive of central pontine myelinosis. It is characterized by damage to regions of the brain, most commonly pontine white matter infarcts, after correction of metabolic disturbances such as hyponatremia. The condition is rarely seen due to under-diagnosis.

KEYWORDS: Central Pontine myelinosis, demyelination, hyponatremia

INTRODUCTION

Central pontine myelinosis (CPM) is a component of Osmotic demyelinating Syndrome (ODS). Central pontine myelinosis was first described in 1959 by Adams and his colleague in a report of four patients with pseudobulbar palsy and quadriplegia.The initial cases were seen in patients with alcohol use disorder and malnutrition; however, by the 1970s, subsequent cases showed a link with rapid sodium correction [1].

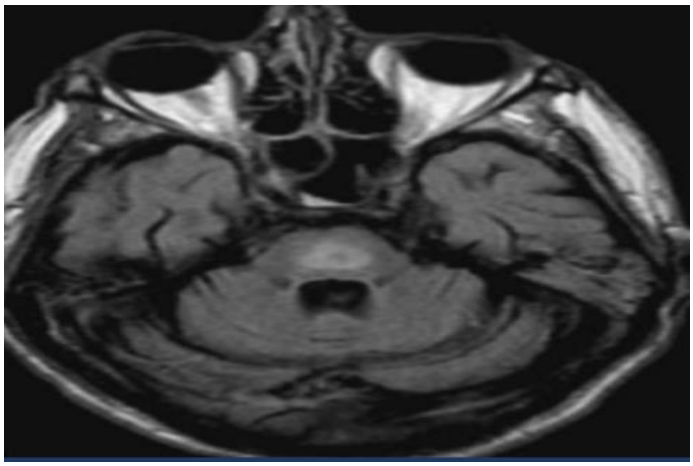
The clinical features of CPM typically begin to appear within several days after rapid correction of hyponatremia.The clinical manifestations may vary and can range from encephalopathy to coma and death .The incidence is very rare due to under diagnosis. Studies have reported neurologic complications in 25% of severely hyponatremic patients after rapid sodium correction. Patients with increased chronicity of hyponatremia and rapid rates of correction in 48 hours were found to have higher rates of associated neurologic complications [2].

Hyponatremia is defined as serum sodium below 135 mEq/L. In response to decreased serum tonicity, extracellular water shift into cells where there is higher tonicity via the process of osmosis, in an attempt to normalize the gradient, thereby causing cerebral edema. Extra pontine myelinosis is seen in up to 10% cases of ODS, and mostly involves the basal ganglia and thalamus [3].

CASE REPORT

A case of 55 years old male who is non- Diabetic and non- Hypertensive came to the emergency room with complaints of sudden onset of abnormal movements of the whole body with reduced alertness and

difficulty in swallowing for 1 day. The patient had a history of several episodes of vomiting and loose stool 4 days back for which he was referred from PHC and was managed in private hospital and discharged as the general condition was better. On day 2 of discharge while he was doing some paper work in the evening he suddenly had an abnormal movements of the whole body which lasted for approx 10 mins with associated drooling of saliva followed by reduced alertness and difficulty in swallowing. On examination patient is disoriented to time, place and person with normal vitals and GCS 11/15(E4V3M4). Chest, CVS, Per abdominal examination was unremarkable. Speech is dysarthric with increased tonicity and reduced power of the muscles in both upper limb and lower limb along with exaggerated deep tendon reflexes. Plantar shows extensor on both sides. There were no meningeal signs and fundus examination show normal study. All the laboratory parameters shows normal study. Previous discharge certificate of private hospital shows Na^+ 112 mEq/L and was corrected with hypertonic saline. NCCT brain Shows normal study. Since the patient's condition was not improving MRI brain was suggested and report shows hyperdense lesion on T-2weighted image over the pons which is suggestive of central pontine myelinosis.



Discussion

CPM has traditionally been associated with rapid correction of hyponatremia, but the etiology has not been clearly established. Diagnosis of CPM is based on clinical neurologic examination and confirmed by imaging studies. MRI is the primary modality for diagnosis and is superior to CT scan.[4]

Rapid correction of hyponatremia (> 12 mmol/L/day) poses a higher risk of developing CPM. In this patient, sodium level rapidly corrected to 137 mEq/L from 112 mEq/L leading to development of neurological symptoms that includes dysphagia, dysarthria and quadriparesis. A cutoff of 48 hours is used to differentiate acute from chronic hyponatremia[5]. This classification is useful for management and prevention of CPM. The brain is able to adapt to a decrease in serum tonicity by several proposed mechanisms. One protective mechanism is through the displacement of water from the cells into the cerebrospinal fluid.Another mechanism called volume regulatory decrease includes removal of intracellular solutes and water via ion channels to reduce swelling and normalize brain volume. With chronic hyponatremia (greater than 48 hours in duration), other adaptive mechanisms include the efflux of organic osmolytes (glutamate, taurine, and glycine) with water, which also reduces cellular swelling[6]. Therefore, patients with chronic hyponatremia have already developed compensatory mechanisms involving solute losses, which puts them at higher risk for the development of CPM. In this case due to rapid correction of hyponatremia the patient might have developed CPM. The most susceptible patients

are those with chronic hyponatremia(>48 hours) or those with severe hyponatremia (Na^+< 120 mEq/L). The patients may present with dysarthria, dysphagia, spastic quadriparesis, pseudobulbar palsy, ataxia, lethargy, tremors, dizziness, catatonia,and in the most severe cases, locked-in syndrome and coma[7].So, careful monitoring of serum sodium and slow correction can help prevent CPM.

Conclusion

CPM is a secondary neurological illness resulting from a foregoing primary disease. Though rare overall, it occurs with greater frequency in certain groups of patients. Clinicians must be aware avoid preventing the development of CPM. The treatment of CPM is still experimental at present, as no evidence-based treatment is yet available[8].

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