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Postpartum Hypernatremic Encephalopathy with Osmotic Extrapontine Myelinolysis and Rhabdomyolysis: A Case Report

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Abstract: A young adult female in a postpartum state was referred to our hospital with history of fever and altered sensorium. On examination patient had pallor, was febrile and stuporous. Laboratory investigations revealed hypernatremia and raised CPK levels suggestive of rhabdomyolysis and MRI showed features suggestive of hypernatremic encephalopathy. After correction of hypernatremia with oral free water supplementation and 5% dextrose and with glucocorticoids therapy, over a period of two weeks there was gradual improvement in the sensorium of the patient. At the time of discharge patient was conscious, oriented and ambulant.
Keywords: Hypernatremia, Metabolic encephalopathy, Rhabdomyolysis, Glucocorticoids, Free water

INTRODUCTION

Hypernatremia is a state wherein serum sodium exceeds 146mEq/l and is a relatively common electrolyte disturbance encountered in neurological intensive care [1–3]. Hypernatremia from diverse etiologies presents with varied neurological manifestations dominated by encephalopathy ranging from impairment of cognition to deep coma. While the hyperosmolar encephalopathy dominates the clinical status, other manifestations including seizures, parenchymal and subdural hematomas do occur [4]. Few imaging studies in patients with hypernatremia have reported reversible cerebral shrinkage [5], hyperintensities in the deep grey matter and white matter [6–9], pons [10], and linear subcortical hemorrhages [11]. Majority of these observations have been in infants and children. Experimental work in rats with induced hypernatremia showed frequent demyelination changes in thalamus, basal ganglia, tegmentum and hippocampus [12, 13]. Rhabdomyolysis with quadriparesis and raised creatine kinase (CK) levels in hypernatremia occurs due to osmotic damage to the muscle membrane [14–18]. Hypernatremia with cerebral imaging abnormalities and rhabdomyolysis has been reported separately. The clinical syndrome due to hypernatremia producing simultaneous rhabdomyolysis and cerebral imaging abnormalities in postpartum women has not been reported.

Here we report a case of 25 yr old postpartum female who presented with hypernatremic metabolic encephalopathy.

CASE REPORT

A 25 year old female patient, presented to our emergency department with history of fever of 4 days duration and altered sensorium of 1 day. Patient had a history of normal full term vaginal delivery 13 days back which was uneventful. On arrival patient was stuporous. On examination she was febrile and had pallor. Her vitals were Pulse rate 142/min, Blood pressure – 110/70mmof Hg and respiratory rate -36/min which was rapid and shallow. Systemic examination of cardiovascular and respiratory system revealed no significant abnormality. On CNS examination she was stuporous with bilateral upgoing plantars. In view of tachypnoea , tachycardia and low oxygen saturation ,patient was intubated and put onmechanical ventilator .

Laboratory investigations showed serum sodium of 189 meq/L, chloride 151meq/L, potassium 2.9meq/L. CK was 8592units/l. Total count was 13070/cumm, serum creatinine was 1.7mg/dl and CSF analysis was within normal limits. MRI brain done showed bilateral symmetrical areas of restricted diffusion involving corona radiata, centrum semiovale,

frontoparietal white matter, internal capsule, corpus callosum and middle cerebellar peduncles which was suggestive of encephalopathy. In view of hypernatremia the mentioned findings were attributed to hypernatremic encephalopathy (Fig. 1).

Patient was treated for hypernatremia with oral free water supplementation and 5% dextrose. Her serum sodium was monitored regularly. Patient was started on glucocorticoid therapy with injection dexamethasone 4mg IV tid.

Over a period of 2 weeks patients serum sodium reduced to 141meq/L. Along with reduction of serum sodium there was progressive improvement in patients sensorium. After treatment for 2 weeks CK levels returned to baseline. Patient gradually became ambulant and was discharged after about 3 weeks of hospital stay.

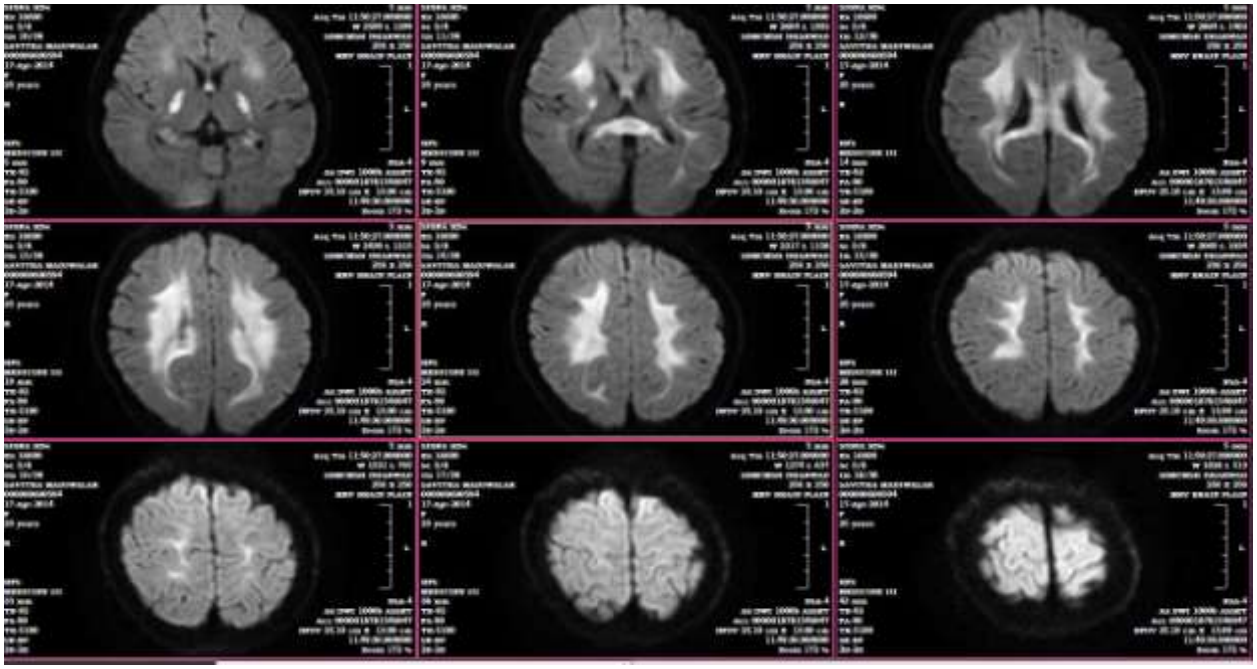

Fig. 1: MRI brain findings were attributed to hypernatremic encephalopathy

DISCUSSION

Hypernatremia is a state in which the serum level of sodium exceeds 146 mEq/l [1]. Hypernatremia due to volume loss from diuretics is a common event in the clinical practice, and is a common occurrence in the intensive care setting. Majority of these patients develop hypernatremia during hospital stay and the incidence of spontaneous hypernatremia is less common [2]. The neurological manifestations of hypernatremia consist predominantly of confusion, seizures, delirium and varying degrees of depression of consciousness culminating in deep coma. The mortality is higher in acute hypernatremia (up to 75%) than with chronic hypernatremia (about 10%) when the serum sodium exceeded 160 mEq/l [1]. Cerebral shrinkage is one of the features in imaging studies and has been shown to be reversible with correction of hypernatremia and hydration [5].

Hyperintensity of the splenium has been reported in encephalitis, epilepsy, systemic malignancy and hypernatremia. Isolated corpus callosum hyperintensities were reported from other conditions including hemolytic uremic syndrome [19]; rotavirus infection [20]; epilepsy, encephalitis, malignancies [8] and status epilepticus [21].

The MRI studies in majority of the published studies with parenchymal lesions were performed after initiation of treatment of hypernatremia. Whether these abnormalities were due to direct result of hypernatremia or from osmotic extrapontine myelinolysis secondary to infusion of hypotonic intravenous fluids is not clear. It is likely that the MRI changes seen are likely to be due to hyperosmolar state from hypernatremia in our patients and not related to treatment. The high signal intensity diffusion weighted images and low signal intensity in ADC mapping are likely to be due to cytotoxic edema which can occur with acute demyelination.

Experimental work in laboratory animals with induced hypernatremia revealed cellular damage and myelinolysis [12, 13]. With acute hypernatremia, there is cellular dehydration due to shift of water from interstitial and intracellular compartments. In the presence of sustained hypernatremia, there is an increase in the ideogenic osmolytes in the brain which

increases the cerebral osmolality and reduces the osmotic imbalance. Cerebral MR spectroscopic study in a patient with hypernatremia demonstrated increase in the osmolytes consisting of myoinositol, choline, creatine and glutamate [22]. The concentration of these osmolytes reduced with gradual reduction of serum sodium levels. Rapid correction of hypernatremia in the presence of increased cerebral organic osmolytes can cause rapid intracellular shift of water and produce cerebral edema causing clinical deterioration.

Dexamethasone had a protective role against osmotic induced demyelination in rats [23]. Our patient had improvement in sensorium once dexamethasone therapy was started. The reason for the occurrence of hypernatremia in the postpartum patients in our patient is not clear. The fever documented could have contributed to the occurrence of hypernatremia by water loss and dehydration. It is customary in some parts of Southern India to restrict water and fluid intake during puerperal state. Reduced water intake was seen our patient too and likely to have compounded the water loss due to fever and contributed to causation of hypernatremia. This poor water intake in puerperium has been linked with puerperal cerebral venous thrombosis. It is likely that combined effects of reduced water intake, fever and the high ambient temperature had contributed to the causation of hypernatremia.

Rhabdomyolysis is reported in patients with abnormal serum electrolytes including hypokalemia, hyponatremia and hypernatremia. Hypernatremia has been associated with rhabdomyolysis and profound elevation of the serum CK levels with acute renal failure secondary to the rhabdomyolysis [14–17]. The rhabdomyolysis is hypothesized to occur from osmotic insult and disruption of the muscle membrane, leading to the release of the enzymes and myoglobin. Our patient had generalized hypotonia with raised CK levels suggestive of rhabdomyolysis and associated quadriparesis. The improvements in the muscle power in our patient also coincided with reduction of the serum CK levels.

Hypernatremia being a potentially lethal condition with high mortality can probably be prevented by antepartum counseling of women about proper hydration. Corticosteroids may improve the outcome of hypernatremic demyelination.

CONCLUSION

Postpartum hypernatremia is a potentially reversible metabolic dysfunction which can produce encephalopathy, osmotic demyelination and rhabdomyolysis. Early identification and management of hypernatremia can improve the morbidity and mortality. The pathogenesis of hypernatremia in postpartum status is unresolved at present and needs further studies.

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