ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
Index Copernicus Value: 49. 23
PubMed - National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 3, Issue – 6, November - 2018, Page No. :103 - 107

Post Partum CPM: A Case Report

Dr Omar Farooq, Associate Professor, Dr Azhar Hafiz, Dr Shahnawaz Hassan, Dr Mahpara Andrabi, PG Scholars

Corresponding Author: Dr Omar Farooq, Government Medical College, Srinagar, Jammu and Kashmir, India

Type of Publication: Case Report

Conflicts of Interest: Nil

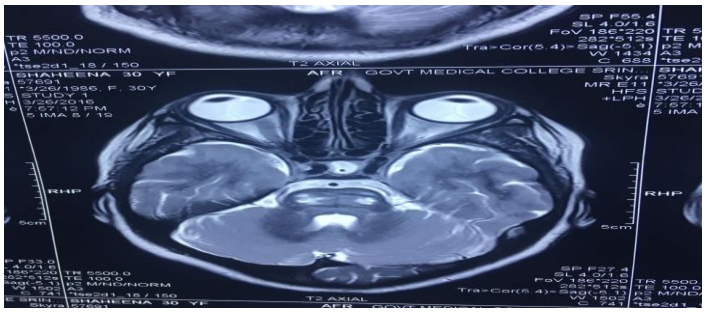
Abstract

Hypernatremia is a fatal condition, and can cause encephalopathy, rhabdomyolysis, and osmotic demyelination [1]. Electrolyte imbalance with hypernatremia can take place secondary to hidden post partum complication or can take place denovo. Hypernatremia causes demyelination same as hyponatremia but predominantly include extra-pontine structures [2]. Extrapontine myelinolysis, a moderately common metabolic disorder, is associated with neurological complications. Central pontine and extrapontine myelinolysis are commonly recognized with rapid correction of sodium [1]. Myelinolysis, however, has rarely been described with hypernatremia. We reported a case of post-partum hypernatremic encephalopathy associated with serum sodium levels as high as 187 mEq/l on presentation. However previously no hypernatremia was documented, although the serum sodium levels were brought down gradually, subsequent imaging showed progression of demyelination and deterioration of the neurological status.

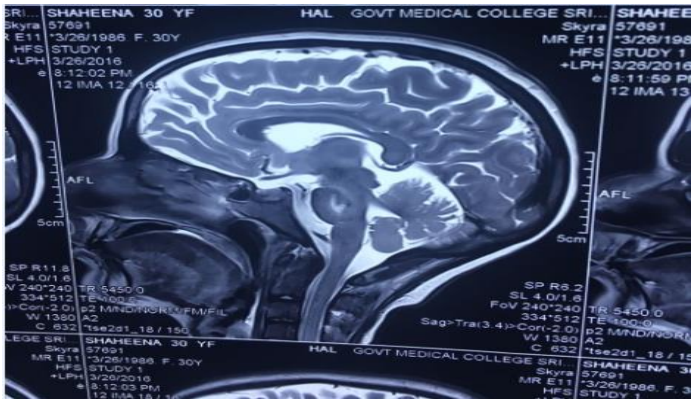
Keywords: Hypernatremia , Extrapontine myelinolysis, post-partum, Central pontine, demyelination.

Case Report

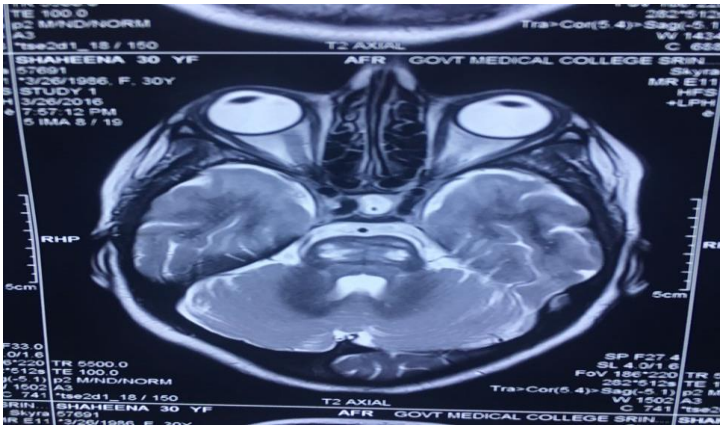
A 26-year-old female delivered a healthy child by LSCS. Postpartum, she was in good health for 1 week, when she started having complaints of generalized weakness, inability to walk, and altered sensorium with no neuro deficit which persisted for 2-3 days and gradually progressed till she was presented to the hospital. On admission, she was drowsy, agitated. A possibility of post-partum sepsis related encephalopathy was thought till her serum sodium level in arterial blood gas (ABG) was 187 mEq/l. ABG analysis was repeated, which again showed serum sodium of 187 mEq/l. Subsequently, magnetic resonance imaging (MRI) brain was advised.



a. Image 1



b. Image 2



c. Image 3

Patient was put on ventilator for one month and was weaned off gradually. Patient also developed critical illness neuropathy and ventilator associated pneumonia. However patient showed improvement over a period of time and repeat sodium was 147 mEq/l and repeat MRI was normal. Resulting in discharging of patient in heamodynamically stable condition.

Introduction

Central pontine myelinolysis (**CPM**) is a neurological disorder caused by severe damage of the myelin sheath of nerve cells in the area of the brainstem termed the pons, predominately of iatrogenic, treatment-induced cause. It is characterized by acute paralysis, dysphagia (difficulty swallowing), and dysarthria (difficulty speaking), and other neurological symptoms [3]. Osmotic demyelination due to rapid correction of the reduced sodium levels (hyponatremia) is a known entity. This includes both pontine and extra-pontine demyelination of which extra-pontine regions predominantly being basal ganglia and thalamic regions.[4] However, even high sodium levels can cause demyelination.

Discussion

Hypernatremia is quite uncommon in the post-partum period. Hypernatremia, a potentially lethal condition, can cause encephalopathy, rhabdomyolysis, and osmotic demyelination.[1] A case series of 11 patients has previously been reported, where serum sodium levels were high. An otherwise healthy female developing such a high sodium level is unusual. Even lowering the sodium at 8-10 mEq/day did not help improve her sensorium. It is known that sudden lowering of sodium (i.e., more than 10 mEq/day) may affect brain function and myelinolysis is expected, but such a high level of sodium once achieved is always detrimental to the brain tissue.[5,6,7] Even lowering sodium at a slow rate would not helpthe neurological status to improve.[8] As in our report, MRI brain revealed progressive lesions with cortical and subcortical demyelination even after the hypernatremic state was corrected. The prognosis remains poor for such patients. EEG findings are suggestive of generalized slowing and decreased amplitude of the waves.

Neurological complaints secondary to extrapontine myelinolysis (EPM) and rhabdomyolysis caused by hypernatremia are infrequently reported. Only a case series with 11 patients has been reported previously in postpartum period.[1] Although the exact etiological factor is unknown, a ritual in some communities in North India, that is, to restrict the water intake for females in post-partum period, may exacerbate dehydration.[1] This is considered as the cause of hypernatremia. Another case has been reported in literature where hypernatremia secondary to hunger strike resulted in neurological deterioration.[9] Patients present with seizures and altered sensorium. Prompt identification and appropriate management can improve outcome in these patients. We acknowledge that information concerning imaging features of EPM remains scarce and prevents accurate diagnosis. Here, we discuss the imaging features of this rare case of extrapontine demyelination caused secondary to postpartum hypernatremia. MR imaging plays the most important role in the initial diagnosis. We also discuss the differential diagnosis based on the imaging features.

MR imaging findings of osmotic demyelination syndrome consist of abnormal hyperintensity involving pons and extrapontine sites including basal ganglia, thalami, and cerebral white matter. Symmetric trident-shaped hyperintensity in the central pons is a characteristic finding with sparing of ventrolateral pons and the pontine portion of corticospinal tracts.[10] Typical sites of involvement of EPM associated with rapid correction of hyponatremia include caudate nucleus, lentiform nucleus, and thalami. The lesions exhibit minimal to no mass effect and show no enhancement in most of the cases. Restricted diffusion is seen in most of the cases. Atypical sites of involvement include grey matter, white matter, corpus callosum, splenium, cerebellum, hippocampus, and external capsule. Other rare sites include midbrain, subthalamic nuclei, claustrum, hypothalamus, medulla, and amygdala.

One previous study done on postpartum hypernatremic patients showed involvement of corpus callosum in all patients.[1] Ten patients went through a MRI which revealed hyperintensitiy of corpus callosum in all the patients in T2, FLAIR, and diffusion weighted sequences (DWI). Symmetrical hyperintensities were also seen in internal capsule, corona radiata, cerebellar peduncles, and hippocampus in various combinations. Our patient also showed involvement of corpus callosum.

“Wine-glass”-appearance has been described in patients with hypernatremic myelinolysis which consists of extensive symmetrical T2, FLAIR, and DWI hyperintensities of white matter, internal capsule through midbrain and pons to middle cerebellar peduncle.[11] It depicts the involvement of corticospinal tract. Symmetrical hyperintensities of internal capsule, crus cerebri, and pons on coronal T2W images are seen. Similar appearance has also been described in entities like primary lateral sclerosis, amyotrophic lateral sclerosis (ALS), and leukodystrophies.

Metrogyl poisoning, an entity described with similar imaging findings, manifests after metronidazole intake for more than 2 weeks. MR imaging of brain demonstrates abnormal symmetrical hyperintensity within cerebral white matter, corpus callosum, and cerebellum.[12] Near total resolution of findings is seen on discontinuation of medication.

In contrast, acute disseminated encephalomyelitis [ADEM] presents with lesions predominantly in cerebral or cerebellar cortices, subcortical and parieto-occipital white matter, centrum semiovale, cerebellar peduncles, and the brainstem.[13] The lesions are large, asymmetric, irregular in morphology, and regress dramatically with short course steroids, immunoglobulins, or plasmapheresis. Associated involvement of spinal cord is also seen. Sometimes, a “fried-egg” appearance with central rounded hyperintensity on T2W images corresponding to “egg yolk” is seen.[13]

Posterior reversible encephalopathy syndrome (PRES) is a remarkably heterogeneous group of disorders seen in patients with hypertensive encephalopathy, pregnant patients with eclampsia, and post-transplant population on cyclosporin A and tacrolimus.[14] It is also seen in patients with uremia, thrombotic thrombocytopenia purpura, and hemolytic-uremic syndrome. CT and MR imaging typically show symmetrically distributed areas of vasogenic edema predominantly within posterior circulation territories involving occipital and parietal lobes; however, involvement of anterior circulation structures is also common.

Conversion to irreversible cytotoxic edema has also been described. The abnormalities primarily affect white matter, but cortex may also get involved.[14] Localized mass effect and mild enhancement may be associated.

Cerebral venous thrombosis, another common condition associated with peripartum state, has been described as a differential.[15] The absence of a flow void and the presence of altered signal intensity in the sinus is a primary finding of sinus thrombosis on MR images. The signal intensity of venous thrombi on MR imaging varies according to the interval between onset of thrombus formation and time of imaging. Parenchymal lesions, focal edema, venous infarcts, and hemorrhages are better depicted and more commonly identified at MR than at CT.

In summary, this rare case of EPM is instructive from the perspective of imaging based primary diagnosis of demyelination.

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