

## Two Rare Cases of Hypernatremic Haemorrhagic Encephalopathy in Children.

Dr Shital Turakhia<sup>1</sup>, Dr Jyoti Gupta<sup>2\*</sup>

<sup>1</sup> Associate Professor, <sup>2</sup> Third year resident doctor, Department of Radiodiagnosis, B. J. Medical College and Civil hospital, Ahmedabad.

### Abstract:

Hypernatremia is defined as increased serum sodium concentration more than 145mEq/L. It occurs most commonly in pediatric patients, geriatric patients and patients with debilitated conditions in ICU who suffers from severe water losses. Newborns and toddlers are at high risk as they have poor thirst control and regulatory mechanism. It occurs when patient is suffering from diarrhea and then mismanagement by over treating the patient with sodium containing fluids. CNS complications and radiological findings are rarely recognized and reported. We hereby report cases of two such pediatric patients who were referred to our hospital from primary health care centers with complain of convulsions and diarrhea.

**Key-words:** Diarrhoea, Encephalopathy, Hypernatremia, Water losses.

### Introduction:

Hypernatremic hemorrhagic encephalopathy is a rare clinical as well as radiological diagnosis predominantly found in neonates and elderly patients in whom electrolyte imbalance and incorrect or rapid correction of sodium is done. I hereby report two such rare cases that were treated in our centre.

### Case Report 1:

A 6 months old male patient suffered from fever, loose stool and vomiting for eight days. It was associated with two episodes of generalized tonic clonic convulsions (GTCS). Patient was admitted to primary care center for 5 days. The patient was given I.V. paracetamol and I.V. fluids for dehydration. Ringer lactate was administered as the suitable I.V. fluid therapy. No improvement was seen in the clinical status of patient. He was then transferred to tertiary care center- Civil hospital Ahmedabad for further management. At the time of admission, he was subconscious, tachycardiac Heart rate – 200 beats per minute, tachypnoeic- respiratory rate - 60/min with normal CVS function. Patient had features of shock with severe metabolic acidosis on blood investigation. His serum sodium levels were high- 167 mEq/L. C-reactive protein was 218, Haemoglobin- 6.0 gm%. As patient was subconscious and suffered from GTCS, MRI was done which revealed focal areas of haemorrhage [Late subacute haemorrhages in posterior part of left cerebellum (Image 1.A, 1.B, 1.C) and subdural haemorrhage in left parietal para-sagittal region (Image 2.A, 2.B)]. Oxygen support with IV fluid (normal saline), IV antibiotics



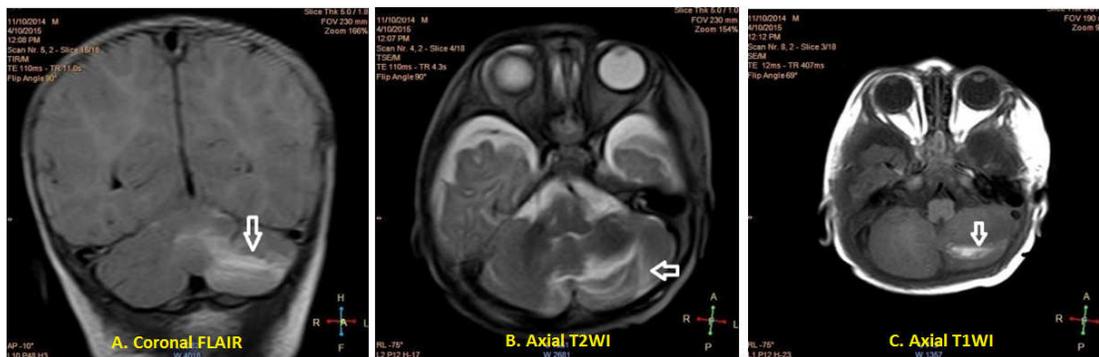
#### \* Corresponding Author:

Dr Jyoti Gupta,

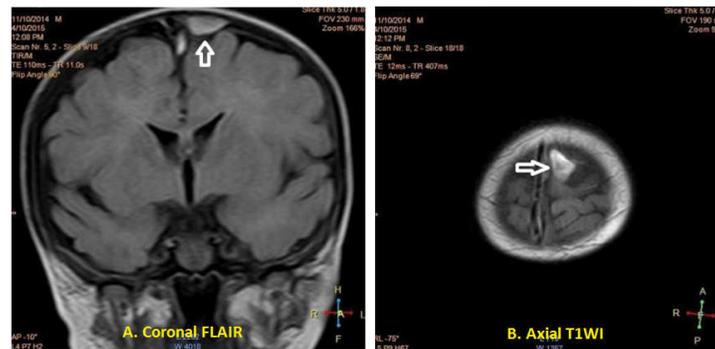
E-mail: [jyotigupta99@gmail.com](mailto:jyotigupta99@gmail.com)

and Packed Cell volume were given to the patient. Patient developed abdominal distension. So USG was done and revealed mild free fluid in peritoneal cavity. Patient developed generalized anasarca. Gradually the patient improved and became conscious with gradual increase in oral intake. Patient became afebrile and his vitals became stable. His blood investigations revealed improvement in CRP- 38.2 with normal CBC reports.

### Image-1 Hyperintensity in left cerebellar hemisphere Suggesting Intra-parenchymal Haemorrhage



### Image-2 Hyperintensity in left parietal parasagittal location Suggesting Subdural Haemorrhage

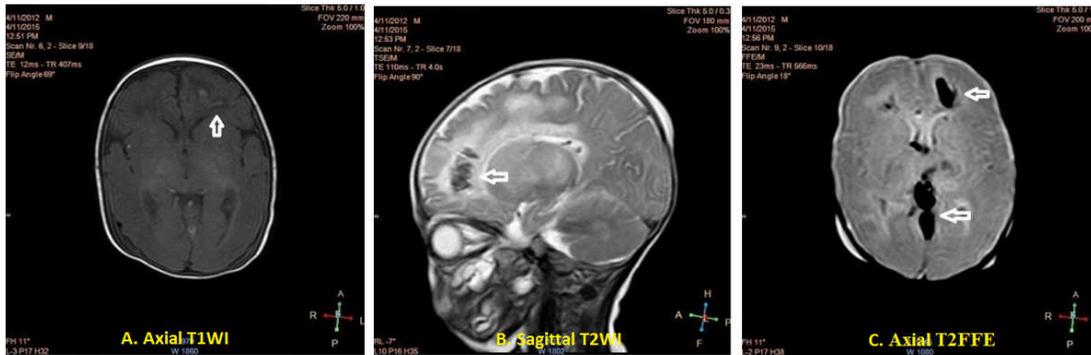


#### Case report 2:

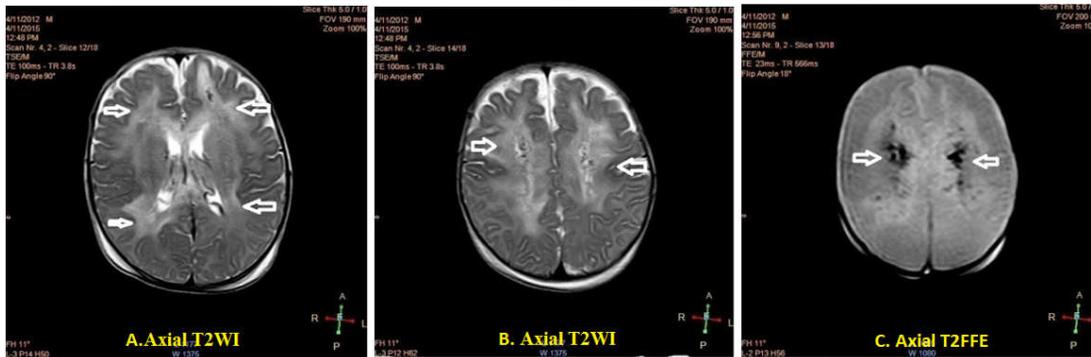
Another four month old male child developed high grade fever and increased frequency of stools for two days. He was taken to a primary health care centre. Patient's was referred to civil hospital Ahmedabad for further management after giving first line treatment. On admission, patient was drowsy, temperature was normal; heart rate was 150 bpm, respiratory rate 46 times pm with normal CVS function. His haemoglobin was 6.95 gm%. His blood investigations revealed hypernatremia (Serum sodium level- 154 mEq/L) and hypokalemia (Potassium level- 2.96 mEq/L). He was given IV antibiotics, IV paracetamol with IV fluid therapy. Injection KCl was added to the treatment in view of hypokalemia. On 2<sup>nd</sup> day of admission, patient suffered from convulsion at night with another episode next morning. Injection carbamazepine was added to the treatment. CSF examination was done which came out to be normal. Chest X ray revealed patch of para-cardiac consolidation. MRI brain revealed left frontal sub cortical white matter early sub acute haemorrhages (Image 3.A, 3.B, 3.C, 4.A, 5.A), hemorrhagic foci in bilateral high parietal deep white matter (Image 5.B,

5.C), early sub acute subdural haemorrhage in posterior interhemispheric region (Image 4.B, 4.C, 5.A, 5.B) suggestive of changes of hemorrhagic hypernatremic encephalopathy. Patient regained consciousness on day 3 of admission. Serial biochemical investigation revealed gradual improvement in serum sodium and potassium levels. After correction of salt overload and volume deficit, patient was started on maintenance fluid ISO-P. As his mother was on AKT for pulmonary Koch's INH prophylaxis was started. CBC revealed anaemia – haemoglobin was 5.61 gm%. Two units of PCV were given to child. Patient was gradually fully conscious with good oral intake and was discharged with explanation of danger signs to the patient's relative.

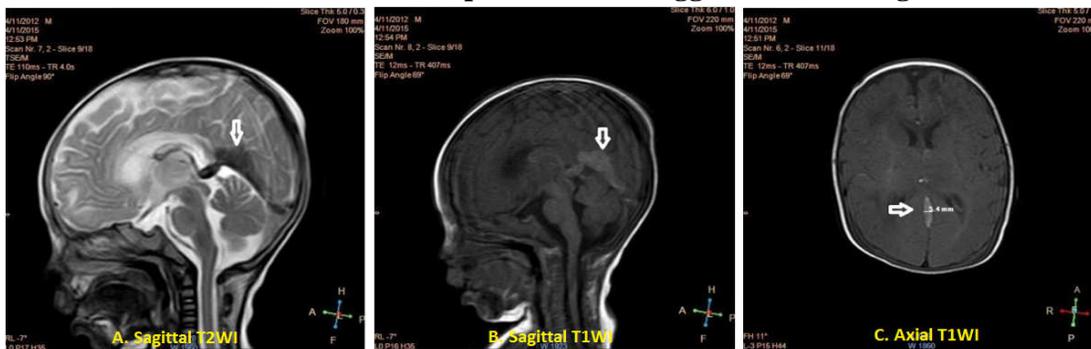
**Image-3 Hyperintensity in left frontal region with blooming in left frontal region and Posterior interhemispheric fissure (3.C) suggesting Haemorrhage**



**Image-4 Hyperintensity in bilateral parietal periventricular region with blooming on T2FFE images suggest Haemorrhage**



**Image-5 hypointensity (5.A) & Hyperintensity (5.B & 5.C) in Posterior interhemispheric fissure suggest Haemorrhage**



**Discussion:**

Hypernatremia is a condition in which there is increase in serum sodium level more than 145mEq/l. It can be caused by excessive water loss or sodium gain or combination of both. It is a state of hyperosmolarity. Plasma sodium concentration and serum osmolarity are controlled by two mechanisms: <sup>1</sup> urinary concentration which is regulated by pituitary secretions and renal effects of ADH, arginine vasopressin and <sup>2</sup> Thirst. (Medscape) In case of hypernatremia, as sodium is in extracellular space it increases osmolarity of extracellular fluid which leads to migration of intracellular fluid into extracellular space leading to cellular dehydration and ultimately cellular death.<sup>1</sup>

It occurs in children, elderly patients, patients with impaired mental status or some psychiatric illness and patients under mechanical ventilation.<sup>2</sup> Net water loss accounts for most cases of hypernatremia. Hypertonic solution administration is mostly due to clinical intervention or accidental sodium loading.<sup>1</sup> Because of some physiologic characteristic infants are predispose to dehydration as they have large surface area in relation to height and weight, so they are more prone to large evaporative water loss.<sup>1</sup>

**Hypernatremia and CNS Haemorrhage:**

Brain cells are especially vulnerable to complications resulting from cellular dehydration which results from hypernatremia.<sup>1</sup> Due to cellular dehydration shrinkage of brain occur which leads to tear of cerebral vessels resulting in hemorrhage, seizures, paralysis and encephalopathy.<sup>1</sup> Mechanism by which hypernatremia affects CNS is –cell water outflow and slowed sodium transportation between blood and CSF would lead to reduced cerebral and CSF volumes. This results in negative pressure around brain tissues, leading to venous and capillary expansion and consequent vessel rupture. Then cerebral hemorrhage would result from cerebral retraction inside skull vault which associated with negative CSF pressure would stress vascular network and can produce hemorrhage from dilated and congested capillary network.<sup>2</sup>

Luttcell established a temporal and cause and effect relationship between hypernatremia and CNS hemorrhage so it is possible to imply that CSF pressure reduction is fundamental for hemorrhage induction as it was completely prevented by fluid infusion keeping positive CSF pressure.<sup>3</sup>

In the available studies, sodium levels associated with intracranial hemorrhage are consistently above 160mmol/L in children.<sup>2</sup> As child's brain is softer than adult brain-brain tissue retracts more than adult brain-justifying more causes of hemorrhage in pediatric population than in adults.<sup>2</sup>

Patients can present with altered sensorium, headache, hyperreflexia, muscle twitches, skeletal muscle rigidity, seizures, coma and death.<sup>4</sup>

Imaging studies can reveal type of hemorrhage like SAH, ICH, IVH, subdural hemorrhage and rule out any other causes involving hypothalamus which can disturb thirst control of patients like tumor or granulomatous lesion or any other pathology.<sup>4</sup>

Imaging findings other than haemorrhage are white matter changes which show

hyperintensity on T2 weighted imaging. These findings can be due to changes in white matter due to hypernatremia.<sup>5</sup> Central pontine and extra-pontine myelinolysis are described with rapid correction of sodium. However, myelinolysis has rarely been described with hypernatremia. On MRI, extra-pontine myelinolysis can involve basal ganglia, thalami and cerebral white matter.<sup>6</sup>

Treatment of hypernatremia includes correction of hypernatremia, but in cases of chronic hypernatremia rapid correction may again cause cerebral edema, seizures and permanent neurodevelopmental sequel. Recommended rate of sodium correction is 0.5mEq/h or as much as 10-12mEq/24hrs. Dehydration should be corrected over 48-72 hours. If the sodium level is more than 200mEq/L, peritoneal dialysis should be performed using high glucose, low sodium dialysate. Serum sodium level should be monitored every 4 hours. However, in cases with rapidly developed hypernatremia, rapid correction will not cause cerebral edema.<sup>7</sup> The volume of replacement fluid needed to correct the water deficit is determined by using the concentration of sodium in the replacement fluid.<sup>1</sup>

### **Conclusion:**

Hypernatremia is a well known complication of overzealous correction of dehydration in newborn infants with improper electrolyte correction and can lead to severe neurological problems in the form of hypernatremic hemorrhagic encephalopathy. A well tailored rational approach should be implemented in treatment of infants with dehydration. MR imaging is the best available modality for early detection of such complications and may help in guiding the clinician in proper management.

### **Limitations:**

Elicitation of clinical history, good eye for suspicion and good communication between clinician and radiologist are the major limiting factors.

### **References:**

1. Ewa Elenberg MD, Muthu Kumar Vellaichamy- eMedicine Specialist, Pediatrics, Hypernatremia: Cardiac disease and critical care medicine. Published online; Feb13 2014
2. Lucas Sampaio Mata, Dimitri Gusmao, Antonio Raimundo Pinto de Almeida., Hypernatremic Haemorrhagic encephalopathy: Case report and literature Review-, Rev Bras TeIntensiva 2010; 22(3): 305-309
3. Enarles N. Luttrell, Hemorrhagic encephalopathy induced by hypernatremia: II experimental observation on hyperosmolarity in cats. AMA Arch neurol. 1959; 1 (2) : 153-160
4. P K atpathy, P M Diggikar, Harmandeep Singh Chahal, AL Kakrani, Preeti B. Rupnar, Subdural hematoma in adult due to hypernatremia, Journal of association of physician of India., April 2014- Vol 62, P- 48

5. J. Carlos Ayus, Dawna L. Armstrong and Allen I. Arieff, Effects of hypernatremia in central nervous system and its therapy in rats and rabbits: Journal of physiology(1996) 492.1,pp-243-255
6. Sonia Bhatia, Abhay Kumar Kapoor, Anurag Sharma, Rajiv Gupta, Sushil Kataria, Cerebral encephalopathy with extra-pontine myelinolysis in a case of post partum hypernatremia , Indian Journal of radiology and imaging, vol-24, p-57-60, 2014
7. Horacio J Adroque MD, Nicolaos E. Madias MD , Primary care, hypernatremia,, 2000, vol-342, No.20, p-1494-95