Hypernatremic hemorrhagic encephalopathy as an unexpected critical complication in children: a case report from a tertiary hospital

Ricky Saunders¹, Ida Bagus Gde Suparyatha², Dyah Kanya WatiⅢ*, I Nyoman Budi Hartawan¹, Agung Bagus Sista Satyarsa³

INTRODUCTION

Hypernatremic hemorrhagic encephalopathy is an uncommon severe complication of hypernatremic dehydration which is relatively uncommon in patients with hypernatremia. Severe hypernatremia might lead to brain shrinkage resulting in intracranial bleeding which can increase morbidity and mortality, especially in children. Hypernatremic hemorrhagic encephalopathy is harmful, but only limited cases are reported. This case highlights the role of cautious management of hypernatremic dehydration and the need for intense monitoring due to the unexpected complication of HHE.

CASE PRESENTATION

A 4 months old female infant presented with a decreased consciousness. She showed signs of shock along with acute diarrhoea and severe dehydration. Laboratory findings revealed severe hypernatremia and high septic marker. Rehydration and correction of hypernatremia were done, but during the process, several episodes of general tonic-clonic seizure occurred. A Head CT scan was done and showed subdural hemorrhage in the posterior interhemispheric fissure and intraventricular hemorrhage in the posterior horn of the right lateral ventricle. Conservrative management was undertaken and the patient was clinically improved progressively. On the 11th day of hospitalization, the patient's condition had been significantly improved without neurological sequelae and was later discharged on the 11th day of hospitalization.

ABSTRACT

Introduction: Hypernatremic hemorrhagic encephalopathy (HHE) is one of the complications of hypernatremic dehydration which is relatively uncommon in patients with hypernatremia. Severe hypernatremia might lead to brain shrinkage resulting in intracranial bleeding which can increase morbidity and mortality, especially in children. Hypernatremic hemorrhagic encephalopathy is harmful, but only limited cases are reported. This case highlights the role of cautious management of hypernatremic dehydration and the need for intense monitoring due to the unexpected complication of HHE.

Case presentation: A 4 months old female infant presented with a decreased consciousness. She showed signs of shock along with acute diarrhoea and severe dehydration. Laboratory findings revealed severe hypernatremia and high septic marker. Rehydration and correction of hypernatremia were done, but during the process, several episodes of general tonic-clonic seizure occurred. A Head CT scan was done and showed subdural hemorrhage in the posterior interhemispheric fissure and intraventricular hemorrhage in the posterior horn of the right lateral ventricle. Conservative management was undertaken and the patient was clinically improved progressively. On the 11th day of hospitalization, the patient's condition had been significantly improved without neurological sequelae and was later discharged on the 11th day of hospitalization.

Conclusion: Close monitoring of clinical, neurological, and laboratory examination is essential in the management of hypernatremic dehydration due to the possibility of HHE, even when on the right procedure. Once neurological complication is suspected, an immediate imaging study should be performed and modifying therapy is required.

Keywords: Cerebral hemorrhage, hypernatremic dehydration, infants.


Received: 2023-02-16
Accepted: 2023-03-22
Published: 2023-04-01

¹Department of Pediatric, Faculty of Medicine, Universitas Udayana/Prof. dr. I Goesti Ngoerah Gde Ngoerah General Hospital, Denpasar, Bali, Indonesia; ²Faculty of Medicine, Universitas Udayana, Denpasar, Bali, Indonesia;

ⅢCorresponding author: Dyah Kanya Wati;
Department of Pediatric, Faculty of Medicine, Universitas Udayana/Prof. dr. I Goesti Ngoerah Gde Ngoerah General Hospital, Denpasar, Bali, Indonesia; dyahkanyawati@unud.ac.id

*Corresponding author: Dyah Kanya Wati;
Department of Pediatric, Faculty of Medicine, Universitas Udayana/Prof. dr. I Goesti Ngoerah Gde Ngoerah General Hospital, Denpasar, Bali, Indonesia; dyahkanyawati@unud.ac.id

Published by Intisari Sains Medis

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acidosis (pH 7.33, pCO$_2$ 19.9, pO$_2$ 197, BE -15.4, HCO$_3$ - 10.6, SO$_2$C 99.7, anion gap 25.4). After the shock improved, rehydration was started for 24 hours with a quarter normal saline of 5% dextrose 40 ml/hour. During rehydration, the fluid was switched to half normal saline 5% dextrose 40 ml/hour to avoid a rapid fall of plasma sodium level. First-line antimicrobial cefotaxime 50 mg/kgBW/dose 3 times/day intravenously was started. After 12 hours of adrenaline infusion, she was hemodynamically stable, thus the adrenaline infusion was discontinued.

After 15 hours of rehydration, she suddenly experienced a general tonic-clonic seizure for 15 minutes. The seizure was resolved after the administration of a loading dose of phenobarbital (20 mg/kgBW) intravenously. The serial electrolyte evaluation showed plasma sodium level decreased from 175 mmol/L to 160 mmol/L (13 mmol/L) in 24 hours. The second general tonic-clonic seizure occurred and lasted for 10 minutes. The patient was referred to our tertiary hospital due to suspected intracranial hemorrhage as a complication of severe hypernatremia.

At our tertiary hospital, she appeared severely ill, under sedation of an anticonvulsant drug with normal vital signs. Notable physical findings were the bulging anterior fontanelle and pinpoint pupil. She showed no neurological deficit. Head CT-scan result revealed subdural hemorrhage on the posterior interhemispheric fissure with a hyperdense lesion on the posterior corn right lateral ventricle (intraventricular hemorrhage). The head CT scan was shown in Figure 1. The hemostatic function was normal. Laboratory evaluation at our hospital revealed an average plasma sodium level (of 147 mmol/L). Complete blood count evaluation revealed moderate normochromic normocytic anaemia (Hemoglobin 8.4 g/dL, MCV 76.9 fl., MCH 29.1 pg).

The patient was treated conservatively by the neurosurgeon. She was given maintenance fluid with 5% dextrose quarter saline in 950 ml/day. The antibiotics were switched to cefoperazone sulbactam and amikacin. The intravenous phenobarbital maintenance dose was continued (5 mg/kg/day). Transfusion of packed red cells (PRC) was added.

After approximately 24 hours of hospitalization, the patient still had 2 episodes of focal seizure that lasted for 1 minute. Diarrhea, level of consciousness, and bulging anterior fontanelle were improved (Glasgow coma scale 11). On the 6th day of hospitalization, she was already alert (Glasgow coma scale 15) and had no seizures. General condition was improved without neurological deficit. Sodium level was normal (sodium 141 mmol/L). The monitoring of serum electrolytes was shown in Figure 2. Blood culture results were negative. Oral formula intake given was increased gradually. Antibiotics were completed for 10 days and the patient was discharged on day 11th with no sequelae.

**DISCUSSION**

Hypernatremic dehydration in infancy is a potentially lethal condition. Three basic mechanisms of hypernatremia are pure water loss (diabetes insipidus), hypotonic fluid loss (vomiting or diarrhea), or hypertonic sodium gain (hypertonic feeding preparations such as improperly prepared infant formula). Early recognition of fluid loss and hypernatremia in infants is extremely difficult due to the subtle of classical
CASE REPORT

Newborns and toddlers are at risk of hypernatremia due to immature kidneys and immature thirst mechanisms, resulting in excessive water loss and decreased ability to increase urine output when dehydrated. The patient may have seizures, paralysis, and encephalopathy. Sodium levels associated with intracranial hemorrhage reported are above 160 mmol/L in children.

A previous report from Leung et al stated that concentrated formula as a traditional practice to relieve constipation symptoms in Taiwan resulted in hypernatremic dehydration. The babies came with sepsis-like symptoms. The baby appeared lethargic with a high fever and diarrhea, and a convulsion within 17 hours of hospital admission. In the Hariram study, 92% of patients with severe neurological complications were in shock on presentation. The degree of dehydration is often underestimated in hypernatremic patients due to relatively well-preserved intravascular volume. The signs of severe dehydration and shock occur late and are probably missed by caregivers. Symptoms of hypernatremia in infants are tachypnea, muscle weakness, restlessness, a high-pitched cry, insomnia, lethargy, coma, increased tone, nuchal rigidity, brisk reflexes, myoclonus, asterixis, chorea, myoclonic seizure, absence seizure, or rhabdomyolysis. In our case, the patient presented late with a decrease of consciousness, septic shock presentation, and signs of severe dehydration.

Management of hypernatremia is challenging. The therapy includes the correction of hypernatremia cautiously to avoid a rapid fall in plasma sodium level. Rapid fall in plasma sodium level may cause cerebral edema, seizures, and permanent neurodevelopmental sequelae. Recommend rate of sodium correction is 0.5 meq/hour or as much as 10-12 meq/24 hours. Dehydration should be corrected over 48-72 hours. If the sodium level is more than 200 meq/L, peritoneal dialysis should be performed using high glucose, low sodium dialysate. Serum sodium levels should be monitored every 4 hours. In our case, initial fluid resuscitation was performed and a vasoactive drug was initiated due to unstable haemodynamic status. Fluid loading to expand the intravascular volume and slow correction with hypotonic intravenous fluid put this patient at higher risk of cerebral edema due to the rapid fall of plasma sodium level. She was given dextrose 5% quarter saline then switched to dextrose 5% half saline.

Hypotonic fluid can induce hyponatremia and worsen central nervous system injury. Our patient sodium level decreased and exceeded the maximum correction target of hypernatremia (10-12 meq/24 hours) even though slowly cautious correction was performed. Frequent monitoring of serum electrolytes (4-6 hourly) along with adjustment of the rate of infusion or composition of IV fluid is essential.

Imaging studies can reveal the neurological complication of hypernatremia such as intracranial hemorrhage types like subarachnoid hemorrhage (SAH), intracranial hemorrhage (ICH), intraventricular hemorrhage (IVH), and subdural hemorrhage (SDH). Several previous case reports published hypernatremic hemorrhagic encephalopathy in a pediatric population. Shital reported a hypernatremic hemorrhagic encephalopathy in a 6-month-old patient who suffered from diarrhea. The patient experienced convulsion at plasma sodium level 154 mmol/L before rehydration. MRI brain revealed sub-acute hemorrhage on the left frontal subcortical white matter and subacute subdural hemorrhage in the posterior interhemispheric region. The patient regained consciousness on 3rd day of hospitalization. Our patient experienced a serial general tonic-clonic seizure at the higher plasma sodium level during rehydration. Instead of having cerebral edema due to a rapid fall in plasma sodium level, our patient had a subdural hemorrhage on the posterior interhemispheric fissure, which marked the hypernatremic hemorrhagic encephalopathy as the complication of severe hypernatremia. However, the intracranial hemorrhage was not extensive but our patient took a longer time to regain her consciousness on day 6th of hospitalization. Hypernatremic hemorrhagic encephalopathy should be considered even after the hypernatremia correction has already started.

CONCLUSION

This case highlights the role of immediate and cautious management of hypernatremic dehydration simultaneously with intense monitoring due to the unexpected complication of hypernatremic hemorrhagic encephalopathy. Immediate rehydration does not necessarily prevent the neurological complication of severe hypernatremia. Cautious administration of intravenous hypotonic fluid could still decrease the plasma sodium level unexpectedly. Rapid recognition of hypernatremia complications by monitoring clinical, neurological, and laboratory evaluation to detect fatal complications must be undertaken. An imaging study could be the method of choice to detect fatal neurological complications.

CONFLICT OF INTEREST

There is no potential conflict of interest relevant to this article reported.

FUNDING

None.
AUTHOR CONTRIBUTION
All authors took part in this report, review, and manuscript.

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