CASE REPORTS:

HYPERNATREMIC DEHYDRATION IN INFANCY:

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SUMMARY

Hypernatremic dehydration in infancy is a medical emergency, which constitutes a potential threat to life. Diagnosis is quite often difficult and the fluid deficit is commonly underestimated. This happens because the intra-cellular fluid comes into the extracellular compartment due to hypertonicity. Complications are frequent and occur not only from the primary process of hypernatremia and loss of water, but more often because of rapid rehydration. Overzealous treatment is as dangerous as delayed or suboptimal treatment. The dramatic drop in serum sodium concentration leads to rebound cerebral oedema with resultant seizures and neurological complications. The mortality in acute cases in infants can be as high as 70%. Here we report a 5-month-old baby with severe hypovolemic hypernatremic dehydration and seizures due to rotavirus gastroenteritis and usage of formula milk and improperly diluted oral rehydration solution. The infant was ventilated and successfully rehydrated with slow sodium correction and ultimately discharged without any neurological deficit. The clinical presentation and complications of hypernatremia are reviewed, and the essentials

of management are discussed.

Key Words: Cerebral edema, hypovolemia, hypertonicity, seizures.

INTRODUCTION

Early recognition of fluid loss and hypernatremia is extremely difficult in infants since the classical features of dehydration are subtle [1]. Although the total amount of water deficit is great, the dehydration is most often underestimated. Due to hypertonicity, water moves from the intra-cellular into the extra-cellular compartment maintaining intra-vascular volume and leading to intracellular volume contraction. Treatment is also difficult because plasma sodium may drop very rapidly even with cautious administration of intravenous fluid therapy. Both the hypernatremia and the dramatic changes in plasma osmolality that may follow overzealous fluid management herald grave neurological complications. Hypernatremia is more common in bottle fed babies particularly those erroneously using concentrated formula feeds and undiluted cow milk [2]. The vast majority of cases of hypernatremic dehydration (90%) are reported in children less than two years of age with the worst outcome in infants less than 6 months of age [3]. The case fatality rate in this age group is as high as 70% with conventional treatment. Here we report our experience with a 5-month-old infant who presented in severe hypovolemic hypernatremic dehydration and later developed seizures and coma, requiring intensive care treatment including ventilation, but eventually had a normal recovery. The clinical course of hypernatremia, its complications and treatment are also discussed.

Case Report:

A 5-month-old Omani child was brought to the accident and emergency department of Sultan Qaboos University Hospital in a moribund state with history of loose stools 10-12 times per day

for the last 4 days and lethargy of one day duration. The baby was on breast feeds till the age of three months, but stopped when the mother developed scabies and the local people advised her to discontinue breast feeding because her milk is contaminated and can transmit the disease to her newborn!! She started giving bottle feeds using formula milk after that. No wonder, the child soon developed gastroenteritis, for which a doctor in a nearby health clinic prescribed Oral Rehydration Solution (ORS). Despite taking the ORS the child was deteriorating and when he became lethargic, mother got concerned and brought him to the university hospital. Further inquiry revealed that the baby was using over concentrated formula feeds and the ORS used was improperly diluted (one sachet in 4 litres of water instead of one litre). When seen in the emergency room the baby was drowsy with reduced response to painful stimuli, pale with cold extremities and dehydrated with dry lips and mucous membranes. Respiration was acidotic with a respiratory rate of 40/min, heart rate of 170/min and rectal temperature of 38.4^{O}C. Abdomen was soft and the liver was palpable 2 cm below the costal margin. Lungs were clear and the heart sounds were normal. The blood gases showed a pH of 7.02, pCo2 of 2.5 kPa, pO2 of 30 kPa, Bicarbonate of 7.3 mmol/l and base excess of minus 23.7 in 100% Oxygen. Serum electrolytes concentrations were as follows: sodium 191 mmol/l, potassium 3.6 mmol/l, Creatinine 185 ▯ mol/l, and Urea 24 mmol/l. The infant was given immediate fluid resuscitation with intravenous Ringer-Lactate solution 20ml/kg over 30 minutes. This was followed by a second fluid challenge of 10ml/kg of sodium chloride 0.9% infusion over the next 30 minutes. In the ward the infant was rehydrated with intravenous solution of 0.45% sodium chloride with added 4.3% dextrose. The fluid requirement was calculated as maintenance plus 10% deficit, which is to be replaced in 72 hours to guard against rapid decline of serum sodium concentration. On this regimen plasma sodium level was not expected to be lowered by >0.5 mmol/kg/hour. However,

despite these precautions, 18 hours after admission to the ward the patient's serum sodium dropped to 164 mmol/l (rate of 1.5 mmol/hour). The patient developed generalized seizures and apnoea and was shifted to the PICU for ventilation and close monitoring. In the PICU, phenytoin and mannitol were given and the intravenous fluid was changed to 0.9% sodium chloride infusion to stop any further drop in plasma osmolality. At 24 hours the serum sodium level was 163 mmol/l. The fluid therapy was then continued with 0.45% sodium chloride infusion and followed later on with 0.3% saline and finally 0.18% saline concentrations, based on the serum sodium levels. Dehydration was corrected and serum sodium level returned to normal by the end of the third day. The infant became alert and was extubated on day 7 and anti-convulsants were slowly tapered and stopped. CT examination of the brain showed no hemorrhages or oedema and Rota virus was detected in the stool. The association between rotavirus gastroenteritis and hypernatremic dehydration with or without neurologic complications has been described before [4-6]. The baby was discharged home with proper advice on feeding and the use of ORS. Subsequent follow-up examinations of the child up to the age of two years have been normal with no evidence of any neuro-developmental problem.

DISCUSSION

Hypernatremia is a state of relative water deficiency and excessive solute concentration in all body fluids. It is said to be present when plasma sodium level is more than 150 mmol/l. Depending on the amount of deficit in total body fluids, hypernatremia is described as either hypovolemic, euvolemic or hypervolemic. Hypovolemic hypernatremia is a common problem in the paediatric practice. It usually follows acute gastroenteritis where the water loss is far greater than the salt loss [7]. The majority of affected children are below the age of 2 years and the worst outcome is observed in infancy [8]. Infants are worst affected, because of (a) immaturity of the kidney that hinders its

ability to excrete an excess sodium load (b) babies have limited ability to express thirst and (c) infants can't feed themselves and depend on their caregivers to provide adequate and appropriate fluids and feeds.

In moderate to severe hypernatremic dehydration, though water deficit is 100-120 ml/kg body weight, the sodium deficit is only 2-4 mmol/kg body weight. Early recognition is extremely difficult and dehydration is often under estimated, as water shifts from the intracellular to the extra-cellular compartment keeping normal skin turgor. There may be fever, tachycardia with poor perfusion and hypotension with hypovolemia. The skin is thick, doughy and may even feel moist due to perspiration. The mucous membrane is dry, with depressed anterior fontanel and sunken eyes. An important observation is intense thirst and craving for water. Physiologically, when the serum sodium concentration rises to >145mmol/L or the plasma osmolality level becomes >300mOsm/kg, the thirst center in the hypothalamus is stimulated and the need to drink water is intensified. Another mechanism, which tightly controls the serum osmolality is the release of Anti-Diuretic Hormone (ADH) from the neurohypophysis, which acts on the kidney to concentrate the urine, thus conserving free water. The diagnostic approach in such cases should include a careful history, assessment of circulation status, full neurological examination and determination of serum urea and electrolytes levels. Simultaneous determination of the levels of serum osmolality, plasma glucose, urinary electrolytes and urine osmolality is also important.

Complications:

The most hazardous effect of hypernatremia is on the brain. Plasma hypertonicity and the subsequent intracellular water loss causes the brain cells to shrink, leading to rupture of bridging vessels with hemorrhages (subarachnoid and parenchymal) and

thrombosis [9]. The brain respond, over a period of several hours, by acquiring new intracellular solutes known as "idiogenic osmoles" such as Taurine, Myo-inositol, Glutamine and Glycerophosphorylcholine, to protect the intracranial volume [10]. During rapid rehydration with relatively hypotonic intravenous fluids, excess water enters the cerebral cells leading to rebound cerebral oedema. Permanent cognitive impairment, cerebral dysfunction, spastic paralysis, and seizure disorders have been described [11]. Extensive lateral and central pontine and extra-pontine myelinolysis have also been reported [12,13]. Children with early onset of seizures or impairment of consciousness have a 50% chance for neurologic sequel [14]. Other recognized complications of hypernatremia include renal vein thrombosis, hyperglycemia, hypocalcemia and renal tubular injury [15]. Mortality in acute cases with serum sodium >160 mmol/l is around 45% (15-70%), while it is around 10% in chronic hypernatremia [16].

Treatment

The first priority in a dehydrated child is restoration of the intravascular volume. Immediate rehydration with intravenous infusion of 0.9% sodium chloride solution or Ringer lactate solution 20ml/kg should be given over 30 minutes. If the response is poor, another bolus of 10ml/kg over 30 to 45 minutes may be given. Once the patient is stable, serum sodium level should be gradually reduced to normal range in 48-72 hours using 0.45% sodium chloride solution. The speed of correction of serum sodium level should be dictated by the severity of symptoms and the initial serum sodium concentration. However, the rate of reduction of serum sodium should not exceed 0.5 mmol/hour [17]. If extracellular osmolality is decreased rapidly, an osmotic gradient may develop between the brain and plasma resulting in a net movement of water into the brain cells producing cerebral oedema, seizures and even permanent neurological deficit and death [18]. Maintenance fluids and

electrolytes must be given in addition to calculated water deficit in quantities sufficient to replace urinary output and insensible losses. When sodium level has decreased to 150 mmol/l, oral fluids can be substituted. Appropriate and slow reduction in serum sodium level has been attained with the classic WHO ORS (90 mmol of sodium) as well as with the low sodium (60 mmol) rice starch ORS with fewer incidences of complications [19-20]. If seizures occur, anticonvulsant drugs and mannitol should be tried together with slower reduction of serum sodium concentration. Mechanical ventilation may be needed if the child developed apnoea or evidence of carbon dioxide retention [21]. In acute severe hypernatremia with serum sodium >190 mmol/l, peritoneal & hemodialysis or hemofiltration may be needed [22].

Conclusion:

Hypernatremic dehydration in infancy is a medical emergency with high rates of mortality and morbidity. Early diagnosis and prompt and appropriate treatment are crucial for survival and prognosis. However, diagnosis is often difficult and dehydration is underestimated. Complications occur because of both, hypernatremia per se and the overzealous intravenous fluid therapy. Several textbooks and articles recommend the use of intravenous fluids that contain 25 mmol/l of Sodium in 5% dextrose for rehydrating the infants and children with hypernatremia [23]. What we have learnt from the present case and many other cases we had seen before, that even using 0.45% sodium chloride solution (sodium of 75 mmol/l) in infants with severe Hypernatremic dehydration may not be save. We can't overemphasize that the serum sodium should be lowered slowly and cautiously using the fluid that will not drop the serum sodium level by more than 10 mmol/24hours. Our patient though initially had a stormy course with severe dehydration, acidosis and seizures is doing well with normal neuro-development, after 2 years of follow-up. This may indicate that aggressive treatment of cerebral oedema including mechanical hyperventilation may

improve the prognosis in this condition.

REFERENSES

1. Gorelick MH, Shaw KN, Murphy KO. Validity and reliability of clinical signs in the diagnosis of dehydration in children. Pediatrics. 1997; 99:61.
2. Mansir T, Sarlangue J, Fayon M, Babin JP, Demarquez JL. Severe hypernatremia due to feeding error. Arch Pediatr. 2000; 7:430-432.
3. Adrogue HJ, Madias NE. Hypernatremia. N Eng J Med. 2000; 342:1493-1499.
4. Jacobson J, Bohn D. Severe hypernatremic dehydration and hyperkalemia in an infant with gastroenteritis secondary to rotavirus. Ann Emerg Med. 1993; 22:1630-1632.
5. Schumacher RF, Forster J. The CNS symptoms of rotavirus infections under the age of two. Klin Padiatr. 1999; 211: 61-64.
6. Ho L, Bradford BJ. Hypernatremic dehydration and rotavirus enteritis. Clin Pediatr (Phila) 1995; 34:440-441.
7. Ramadas DJ, Moyes CD. Hypernatremia: still seen as a problem in paediatric practice. N Z Med J. 1994; 107:311-313.
8. Mishkin MB, Simonet M, Lawrence C, Van Why SK. Hypernatremia in infancy. Curr Opin Pediatr. 1998; 10:156-160.
9. Hilliard TN, Marsh MJ, Malcolm P, Murdoch IA, Wood BP. Sagittal sinus thrombosis in hypernatremic dehydration. Arch Pediatr Adolesc Med. 1998; 152:1147-1149.
10. Schulman M. Organic osmolytes in the brain of an infant with hypernatremia. N Engl J Med. 1994; 331:1776-1777.

11. Conley SB. Hypernatremia. Pediatr Clin North Am. 1990; 37:365-372.

12. Brown WD, Caruso JM. Extrapontine myelinolysis with involvement of the hippocampus in three children with severe hypernatremia. J Child Neurol. 1999; 14:428-433.

13. AlOrainy IA, O'Gorman AM, Decell MK. Cerebral bleeding, infarcts, and presumed extrapontine myelinolysis in hypernatremic dehydration.

Neuroradiology. 1999; 41:144-146.

14. Eke F, Nate A. A prospective clinical study of patients with hypernatremic dehydration. Afr J Med Sci. 1996; 25:209-212.

15. Palevsky PM. Hypernatremia. Semin Nephrol. 1998; 18:20-30.

16. Mocharla R, Schexnayder SM, Glasier CM. Fatal cerebral edema and intracranial hemorrhage associated with hypernatremic dehydration. Pediatr Radiol. 1997; 27:785-787.

17. Hogan GR. Hypernatremia: problems in management. Pediatr Clin North Am. 1976; 23: 569-574.

18. Fiordalisi I. Central nervous system complications during hypernatremia and its repair. Arch Pediatr Adolesc Med. 1994; 148:539-540.

19. Farthing MJ. Hypernatraemia, acute diarrhoea, and oral rehydration therapy. Lancet. 1992; 339: 936.

20. Iyngkaran N, Yadav M. Rice starch low sodium oral

rehydration solution (ORS) in infantile diarrhoea. Med J Malaysia. 1995; 50:141-144.

21. Ross O. The management of extreme hypernatraemia secondary to salt poisoning in an infant. Paediatr Anaesth. 2000; 10:110-111.

22. McGraw ME, Chambers TL. Correction of hypernatremia with continuous arteriovenous haemofiltration. Arch Dis Child 1990; 65:628-630.

23. Nelson Textbook of Pediatrics. Hypernatremic dehydration. (Behrman RE, Editor) 15th edition, W.B.Saunders, Philadelphia, 1996 page 203.