Water Intoxication in a 7-month Infant

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Abstract

A 7-month-old infant developed repeated hyponatraemic convulsions secondary to water intoxication after being fed with excessive free water. She was treated with 3%NaCl infusion at a rate of 2.5 mmol/kg/hr together with anticonvulsant, which successfully controlled the convulsion and corrected the hyponatraemia. The child recovered with no evidence of neurological sequelae. We reported the clinical progress and management of this patient with emphasis on the safety of rapid sodium replacement in acute hyponatraemia, and the importance of public education on avoiding unnecessary supplementation with water or diluted formula in small infants.

Key words   Hyponatraemia; Infants; Seizure; Water intoxication

Introduction

Water intoxication in infants fed with over-diluted formula or excessive fluid supplementation has been reported frequently in America.[1-6] There is no well accepted reason why normal small infants would voluntarily take in a large amount of free water. Although water supplementation besides milk is usually not recommended in young infants except in hot weather or during febrile illness, it has been found to be a common practice irrespective of the mothers' socioeconomic status or educational level.[7] Moreover, the lack of awareness by caregivers that excessive intake of free water is dangerous is found to be the most important reason of excessive free water intake.[8] The latter may cause severe hyponatraemia in infants because of their immature renal function and low glomerular filtration rate. The usual presentations are lethargy, vomiting, irritability, convulsion and hypothermia. These symptoms can be reversed by salt deficit correction. Although the neurological prognosis is usually good in those patients who are given proper and timely management,[5,9] both delayed therapy and over-rapid correction of sodium can lead to severe neurological sequelae. Severe hyponatraemia leads to cerebral oedema and the resultant increased intracranial pressure may cause tentorial herniation and respiratory arrest, with high morbidity and mortality. However, over-rapid correction is associated with osmotic demyelination syndrome. We report a case of infantile hyponatraemic convulsion secondary to water intoxication.

Case Report

A 7-month-old girl with body weight 8.5 kg was admitted into the paediatric ward after a 10 minute-generalized tonic clonic convulsion. Another similar convulsion with head and eyes deviated to right side was witnessed again shortly after admission which was terminated by intravenous diazepam at 10 minutes.

History revealed that she had recent upper respiratory tract infection symptoms and was treated with amoxicillin and paracetamol by a private general practitioner. She took only 90 ml milk but 1800 ml free water the day before

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admission. Her past medical history and perinatal history were unremarkable. Neurological examination showed bulging anterior fontanelle and brisk limbs reflexes. Pupil light reflexes were normal. She was febrile with body temperature 39°C. Physical examination of other systems was unremarkable. Her hydration status was good but there was no oedema. Brain CT scan was grossly normal. CXR, CSF and urine specimen did not show feature of infection. Blood test showed serum sodium level 116 mmol/L, with other electrolyte levels and renal function normal. Blood glucose was also of normal range.

The patient developed one more episode of seizure an hour later, which spontaneously subsided at 1 minute. Intravenous phenobarbital was then given. 90 ml 3%NaCl was given intravenously over 4 hours, which brought the sodium level to 122 mmol/L 2 hours later. The sodium level eventually became normal 7 hours after admission with further sodium supplement and fluid restriction. There was no more seizure afterward.

Investigation results returned subsequently which showed an initial serum and urine osmolarity of 243 and 252 mmol/L respectively. The initial urine sodium concentration was 90 mmol/l, which dropped to 10 mmol/l one day after admission. Adrenal function and tubular phosphate absorption were normal. Urine beta 2 microglobin: creatinine ratio was also within normal limits. EEG did not show epileptic discharge. With the history of large amount of free water intake, and after excluding the other differential diagnoses including SIADH, renal failure, tubular dysfunction and adrenal insufficiency, we concluded that the hyponatraemic convulsion was caused by water intoxication.

Phenobarbital was stopped after correction of hyponatraemia. She was discharged home 9 days later. Neurological and developmental examination shortly after the incidence and half year later did not show any abnormality.

Discussion

The diagnosis of water intoxication in our case was mainly based on the presence of severe hyponatraemia and its signs and symptoms. Moreover, her good hydration status and the initial high urinary sodium suggested normovolaemia.[10] The normal renal function test and the absence of tubular dysfunction excluded underlying renal disease. The low urinary osmolarity and the rapid increase of serum sodium after 3%NaCl infusion (2.5 mmol/kg/hr)

also made the presence of SIADH unlikely. Absence of evidence of sodium loss and the history of 1800 ml of free water intake in a single day, helped conclude that this was a case of acute water intoxication caused by inappropriate feeding practice.

Water intoxication can lead to hyponatraemia. Severe hyponatraemia causes cerebral oedema because water moves from extracellular compartment into brain cells. Three independent factors might affect the CNS function: the serum sodium concentration, the rate of change in the level and the duration of the hyponatraemia. Sterns et al. induced hyponatraemia in rats with variable rate of onset and duration of disturbance, and compared the effect of fast and slow sodium correction. They found that an acute fall in plasma sodium to 106 mmol/l within 7 hours caused seizure and coma. However, a more gradual fall in plasma sodium to 95 mmol/l in 3 days did not cause those symptoms. Moreover, they noticed that the rats with hyponatraemia for less than 24 hours recovered well regardless of the rapidity of electrolyte correction. In the rats having hyponatraemia for more than 3 days, the rate of sodium correction had determining effects on neurological outcome and mortality: animals corrected slowly by evaporative water loss fared better than those being aggressively treated with intraperitoneal hypertonic saline.[11] These factors therefore must be considered while managing infants having symptomatic water intoxication

Acute severe hyponatraemia is defined arbitrarily as a fall in sodium concentration >0.5 mmol/L/hr. It is associated with high mortality and morbidity if immediate treatment is not offered properly.[12] History of excessive water intake within 2 days with compatible symptoms, normal hydration, low sodium concentration and normal renal function can be assumed to be water intoxication. 3%NaCl 1-2 ml/kg/hr (which is expected to raise the serum sodium concentration by 1-2 mmol/L/hr) should be given. The above therapy appears not to be associated with adverse neurological outcome[11,13] and should be continued till sodium concentration has reached ≥120 mmol/L, as lower levels may cause irreversible neurological damage.[12] However, the controversy in the treatment of symptomatic hyponatraemia has not yet been settled. Experiment on rats with induced hyponatraemia in 7 hours showed that neurological outcome was not affected by the rapidity of correction.[11] An initial rapid sodium correction (1-2 mmol/L/hr) with 3%NaCl infused at rate of 1-2 ml/kg/hr has been proposed in acute hyponatraemia.[14,15] Oh et al., after thorough literature review, recommended that serum sodium level should be raised at a rate of 3 mmol/L/hr till

there was an absolute change of 4-6 mmol/L in refractory status epilepticus secondary to hyponatraemia.[16] However, Gruskin and Sarnaik suggested a 'very-rapid correction' regime which increased the sodium level by 4-6 mmol/L over 10-15 minutes in symptomatic patients.[17] A retrospective study on 41 children also found that rapid increase in serum sodium concentration by 3 to 5 mmol/L with the use of bolus intravenous hypertonic saline was safe and effective in managing acute hyponatraemia.[18] Although there is evidence that 'very rapid' correction of acute hyponatraemia is safe, it seems that there is no extra-advantage to increase the sodium level in minutes except in life threatening condition. In our case, 3%NaCl of 2.5 ml/kg/hr was given and the patient did not develop further seizure after the beginning of therapy.

Rapid correction of chronic hyponatraemia is well recognised to be associated with central pontine myelinolysis characterised by histological change of myelin loss with almost intact axon and minimal inflammation.[19] Patients with the lesion might develop a variety of complications including behavior change, pseudobulbar palsy, quadriplegia and coma. Cluitmans and Meinders reviewed 117 patients with chronic hyponatraemia (fall of serum concentration <0.5 mmol/L/hr). Seventy-one of them were treated with sodium correction rate >0.5 mmol/L/hr. Thirty patients developed central pontine myelinolysis and 13 developed other neurological sequelae. The lower the initial sodium concentration, the more likely would the patient develop neurological complications. Another 46 patients were managed with correction rate <0.5 mmol/L/hr. Only one patients developed central pontine myelinolysis. This patient also had pre-existing chronic alcoholism and hepatic failure, which might explain the neurological finding. Animal studies on brain water and electrolyte suggested that there is cerebral adaptation to osmolarity changes if sufficient time is given.[11] There is loss of intracellular organic osmolytes to prevent excessive cerebral swelling. After correction of hyponatraemia, organic osmolytes and the brain cell osmolarity return to normal over 3 to 5 days but serum electrolyte and osmolarity increase rapidly, resulting in cerebral dehydration. Therefore, slow correction is recommended on patients having hyponatraemia that has been present for more than 3 days.

In the management of a convulsing child, protection of airway and oxygen supplementation cannot be overemphasised. Intubation and positive ventilation are only indicated if there is respiratory involvement. Anticonvulsant such as diazepam should also be considered in prolonged

seizure (>10 minutes); indeed the serum sodium concentration is usually unavailable initially. It should be remembered that most hyponatraemic seizures are secondary to acute sodium level change and 3%NaCl infusion at a rate of 2 ml/kg/hr is recommended especially if the history suggests that hyponatraemia develops within 48 hours. At this rate of sodium replacement, serum sodium level usually increases by about 2 mmol/L/hr. When the patient becomes asymptomatic and the sodium concentration has reached between 120 and 125 mmol/L, slow correction together with fluid restriction in the following 24 hours is appropriate. Intravenous bolus of 4-6 ml 3%NaCl over 15 minutes in order to bring the sodium level up by about 5 mmol/L may be considered only in patients with refractory status epilepticus, repeated apnea requiring intubation or coma us a result of hyponatraemia.

In chronic hyponatraemia (>48 hours), judicious slow sodium replacement at a rate of 0.5 mmol/L/hr should be given so as to avoid central pontine myelinolysis. In case of unsure timing of electrolyte disturbance, asymptomatic hyponatraemia should always be treated as chronic adaptive status.

In conclusion, our case illustrates that dietary inquiry is crucial in the investigation of infants having unexplained hyponatraemic convulsion and immediate sodium correction should not be delayed if there is evidence of acute water intoxication. Because of the potential neurological complications of water intoxication, prevention is of paramount importance. Free water supplement, especially in large volume, should be avoided in young infants. Diluted formula during acute diarrhea is also not recommended. Accurate re-hydration solution concentration in infants having diarrhoea should be prescribed and its administration should be carefully supervised. Finally, breast-feeding should always be advocated unless really contraindicated as it avoids inadvertent overdilution of formula.

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