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CASE REPORT

Water Intoxication Induced Status Epilepticus in Two Children

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This report describes two children who presented with status epilepticus. Both patients had hyponatremia, hypochloremia, low serum osmolality, and low urine osmolality caused by voluntary drinking of excessive quantities of water or diluted formula. Seizures were controlled by the administration of benzodiazepines, antiepileptic drugs, and hypertonic saline. Educating parents regarding hazards of excessive ingestion of fluid could reduce the incidence of this preventable and life-threatening condition.

Key words: Hyponatremia, water intoxication, status epilepticus, children

INTRODUCTION

Status epilepticus (SE) is continuous generalized tonic-clonic seizure activity with loss of consciousness lasting >30 min.[1] The annual incidence of SE in children is approximately 10-73 episodes per 100,000 children; the incidence is highest in children younger than 2 years of age.[2] SE is a medical emergency that mandates immediate recognition and vigorous treatment because permanent neurological deficit and/or death may ensue.[3] SE has numerous etiologies. However, water intoxication (WI) induced hyponatremia that results in SE is extremely rare.

Water intoxication, a dangerous situation, can occur in various clinical settings involving excessive water intake and impaired renal excretion of free water. Clinical presentations of WI are variable and nonspecific; if untreated, they may progress from mild symptoms such as nausea, vomiting, or confusion to acute delirium, seizures, respiratory arrest, coma, or death.[4-8] WI is common in patients with psychiatric disorders, in patients taking drugs with antidiuretic effects, in endurance athletes with forced overconsumption of water, in military training, in gastroenteritis, in cases of child abuse, in cases of swallowing excessive quantities of water during swimming lessons, and in postoperative intravenous (i.v.) hyperhydration.[9-15] In children, even the reports of WI are infrequently; however, feeding improperly diluted formula or inappropriately large quantities of free water may also lead to this serious problem.[16-21]

In this report, we describe the clinical presentations, evaluation, and treatment of two children who presented with SE. The causes of this lethal situation were not clarified until their histories and laboratory results were obtained.

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CASE REPORTS

Case 1

A previously healthy, 19-month-old girl was taken to the emergency department of a medical center because of persistent tonic-clonic convulsions, upward gaze, and loss of consciousness for 30 min. Upon admission, her seizures ceased after administration of lorazepam (0.1 mg/kg, i.v.) and phenytoin (20 mg/kg, i.v.). She was admitted to the intensive care unit for further treatment.

On physical examination, her body temperature was 35.6°C, pulse rate was 105 beats per min (bpm), respiratory rate was 28 breaths/min, blood pressure was 96/54 mmHg, and body weight was 10.7 kg. In addition, developmental milestones were normal, skin turgor was normal without edema, and her pupils were equal and reactive to light. Examination of the cranial nerve was unremarkable. Results of primary laboratory studies revealed serum levels of sodium and chloride to be 124 mmol/L, 90 mmol/L, respectively, and osmolality to be 259 mOsm/kg. History taking showed that the girl was fed more than 3 L water within 2 days by her mother to counteract

gastroenteritis and water loss. Therefore, after infusion of 3% hypertonic saline (72 ml in the initial 4 h and 72 ml in the following 20 h), her electrolyte levels returned to normal after 1 day. Consciousness was completely recovered and further seizures were not observed. Her urine amount was 4.08 ml/kg/h on Day 1 after hospital admission. She was discharged 5 days after admission. At discharge, her body weight was 9.8 kg, which was 8.4% decrease of her body weight compared with that at admission (10.7 kg). Virus isolation and identification of stool were negative. No neurological sequelae were noted after 1 year follow-up.

Case 2

An 8-month-old male infant presented with sudden-onset upward gazing, cyanosis, tonic-clonic seizures, and loss of consciousness lasting more than 1 h. He was taken to the emergency department of a medical center. After two doses of lorazepam (0.1 mg/kg), one dose of midazolam (0.2 mg/kg), and one dose of phenobarbital (20 mg/kg), his seizures stopped. His mother revealed that, to hydrate her son, she fed him excessive diluted formula (one scoop of formula powder in 250 ml water every 4 h) 2 days before admission. His past medical and perinatal histories were unremarkable. On physical examination, his body temperature was 35.9°C, heart rate was 159 bpm, respiratory rate was 32 breaths/min, blood pressure was 117/68 mmHg, and body weight was 8.5 kg. His anterior fontanel was soft and mildly bulging. His pupils were equal and reactive to light. The capillary refilling time was lesser than 2 s. Pathological reflexes were not recorded. His deep tendon reflexes were symmetrically hyperactive. Results of primary laboratory studies revealed serum levels of sodium and chloride to be 122 mmol/L, 87 mmol/L, respectively, and osmolality to be 263 mOsm/kg.

After admission, 3% hypertonic saline was administered at 17 ml/h during the initial 4 h, and then 2.5 ml/h for the following 20 h. His consciousness completely recovered after 1 day of admission. Serum levels of sodium returned to 134 mmol/L, urine output was 4.1 ml/kg/h, and a weight loss of 650 g was recorded on Day 2. The boy was discharged on Day 4 without any sequelae.

In these two cases, further investigation revealed that serum levels of glucose, albumin, triglyceride, ammonia, cortisol, thyroid-stimulating hormone, and free T4 were within normal ranges. Electroencephalography showed a pattern of postictal slowness. Brain computed tomography and renal sonography failed to reveal significant findings.

The following table is a comparison of serum and urine biochemistries, urine output, and variation in body weight before and after recovery from SE between the two cases.

	Case 1		Case 2	
	Upon admission	After treatment	Upon admission	After treatment
Hematocrit (%)	33.0	—	32.6	—
Serum sodium (mmol/L)	124	137	122	136
Serum potassium (mmol/L)	4.5	4	4.2	4.3
Serum chloride (mmol/L)	90	101	87	104
Blood urea nitrogen (mg/dL)	4	—	5	
Serum creatinine (mg/dL)	0.3	—	0.4	—
Serum osmolality (mOsm/kg H_2O)	259	293	263	295
Urine specific gravity	1.001	1.005	1.002	1.006
Urine osmolality (mOsm/kg H_2O)	59	—	78	—
Urine sodium concentration (mEq/L)	<10	—	12	—
Body weight (kg)	10.7	9.8	8.5	7.8
Urine amount (ml/kg/h)	—	4.08	—	4.1

DISCUSSION

Status epilepticus, a life-threatening medical emergency, must be recognized immediately and treated properly.[1,2] Numerous causes are known to be associated with SE.[22] In our cases, brain computed tomography showed no obvious abnormalities. Results of laboratory studies were all within normal ranges, with the exception of hyponatremia, hypochloremia, low serum osmolality, and low urine osmolality. Information garnered from their families supported that excessive intake of fluid, either by feeding too much water or diluted formula, induced WI and electrolyte imbalance, thereby causing SE.

Data regarding the incidence of WI in children are lacking. Moreover, most data are from case reports. In such reports, the clinical presentations include low core temperature, normal or excessive hydration status, and generalized or focal seizures.[11,23] Such presentations are compatible with our cases. Thus, if the first line physicians face children with focal, generalized, or status seizures; low body temperature; and hyponatremia, WI related neurological illness should be ruled out.

Some reports have targeted excessive secretion of antidiuretic hormone (ADH) as a possible underlying mechanism in children with WI.[24] In our cases, though the ADH levels were not measured, their clinical presentations and laboratory data (including a large output of dilute urine, low urine osmolality, and low urinary sodium levels) after seizure attacks did not support the diagnosis of a syndrome of inappropriate ADH secretion. In fact, a possible cause of WI in our cases was massive fluid load within a short

period jeopardizing the water balance. A reduction in serum osmolality in WI can generate an osmotic gradient between brain cells and serum. The gradient pressure cascades an influx of water into brain cells, causing progressive cerebral edema and encephalopathy.[5-8] Young children are vulnerable to this situation because of their relatively lower glomerular filtration rates and higher brain-to-skull size ratio that limits the expansion of brain cells.[7,11,25] The subtle but functional intracellular edema in the central nervous system could have explained their normal brain computed tomography and transient encephalopathy.[6]

Correction of hyponatremia is the primary component of WI management plans. If onset is lesser than 48 h and allied with symptomatic hyponatremia, 3% hypertonic saline 1-2 ml/kg/h may raise serum levels of sodium by 1-2 mmol/L/h and be maintained until symptoms relief.[7,15,20,26] If onset is more than 48 h, the rate of correction should be relatively slow to avoid central pontine myelinolysis.[7,10,12,26] Serum levels of sodium should be monitored frequently until symptoms resolve. The onset of our cases were both lesser than 48 h and they were completely recovered without any sequlae after treatment.

Available reports depicted that children with WI were severe ill. They may have a greater risk of intubation and assisted ventilation.[11,13,14,23] However, compared with such cases, the initial presentation of our cases was SE, and they recovered soon after the administration of benzodiazepines, antiepileptic drugs, and hypertonic saline. In our cases, serum levels of sodium were approximately 122-124 mmol/L, whereas in other reports it was <120 mmol/L. Because the serum levels of sodium may link to the severity of WI, the lower serum levels of the sodium are, the more severe the patient may be. In addition, the alertness of families and rapid transport of patients to the hospital without delay may explain their good prognoses.

CONCLUSION

The clinical presentations of WI are variable and nonspecific. WI induced SE in children is very rare and dangerous. The causes of this life-threatening condition are not always clarified until their histories and laboratory results are obtained. Early recognition of the condition and appropriate treatment can save the patient's life. Education primary caregivers regarding proper quantities of fluid intake in ill children may prevent the disaster condition.

DISCLOSURE

All authors declare no competing financial interests.

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