

Case Report: An 8 Yr Child Present with Seizures Due to Hyponatremia

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ABSTRACT

A previously well 8 yr (wt: 24 kg) child presented with seizures. He had history of loose motions 4-5 episodes & infusion of excess of isolyte p by a quack. The child had confusion, reduced consciousness and seizures but without a significant history of epilepsy, fever & head injury. His serum sodium was noted to be low at 117 mmol/litre. He needed anti-convulsants with subsequent intubation to stop the fitting and was commenced on a hypertonic saline infusion with frequent monitoring of serum sodium. After 10 days patient discharged from hospital with full recovery. Disturbances in sodium concentration are common in the critically ill patient and associated with increased mortality. Correction of hypernatraemia/hypertonicity is critical, but should not exceed 12 mmol/l/day in order to reduce the risk of rebounding brain oedema. There is a risk of long-term neurological damage from central pontine myelinolysis if the hyponatraemia is corrected too rapidly. He went on to make a full recovery without any long-term neurological complications.

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INTRODUCTION

Hyponatremia is defined as a decrease in the serum sodium concentration to a level below 136 mmol/L. It can be associated with low, normal, or high tonicity. Osmolality of plasma is due to the contribution to the osmolality of solutes (e.g. sodium and glucose) that cannot move across the cell membranes, thereby inducing transcellular shifts in water. Dilutional hyponatremia, by far the most common cause of hyponatremia, is caused by water retention or water intoxication. If water intake exceeds the capacity of the kidneys to excrete water, body solutes dilute, causing hypoosmolality and hypotonicity. Water intoxication in children is very common in periphery set up, specially by untrained doctors in developing countries. An accurate history and physical examination can help to determine whether the patient has hypovolemia, euvolemia, or hypervolemia and the cause of volume depletion.

Hypovolemia refers to any condition in which the extracellular fluid volume is decreased. When severe, it leads to a clinically apparent reduction in tissue perfusion.

The symptoms of hyponatremia are mainly neurological and relate to the sharply fall of serum sodium [1]. Acute hyponatraemia is defined as occurring within <48 h. There are usually no symptoms if serum sodium is 130–135 mmol/l. If serum sodium falls to 125–130 mmol/l than resulting in nausea and malaise. Restlessness, disorientation, depressed reflexes, headache muscle cramps & seizures are noticed if serum sodium falls below 125 mmol/l. When severe hyponatraemia evolves over a period of hours resulting in seizures, respiratory arrest, brain-stem herniation, permanent brain damage and death can occur [2].

On the other hand, patients with chronic hyponatraemia are often asymptomatic

irrespective of the degree of hyponatraemia. Symptoms of hyponatremia occur only if there is acute decline in the level of sodium, or if serum sodium falls below 110 mmol/l. In chronic hyponatraemia present for >48 h, adaptive action taken by brain to protect itself against cerebral edema: a rapid increase in plasma sodium can lead to a decrease in brain cell volume with resulting in demyelination [3]. It may not be noticed until 2–6 days after correction of sodium, and most patients are left with permanent neurological dysfunction including quadriplegia, pseudobulbar palsy and seizures. Coma and death may occur [2].

Case presentation

This is the case of a 8 yr child presented with seizures. The child had three to four episodes of diarrhea. After that he went to quack & was given isolyte p in excess. Thirty minutes after infusion, first generalised tonic-clonic seizure occurred, lasting about 5 minutes before spontaneously resolving. Diazepam per rectal was given to the patient. He remained confused and had a further seizure within 2 hours. After that patient had shifted to higher center, where the patient was given intravenous anti-convulsants (phenytoin) causing a decrease in consciousness requiring subsequent intubation for airway protection.

Her immediate blood tests showed a metabolic acidosis, likely to have been caused by the two fits, as well as low serum sodium of 117 mmol/liter. Another lab parameters like blood urea is 16 mg %, potassium 3.5 mmol/liter along with

calcium 9.3mg %, uric acid 5.8mg % with chloride towards low levels. Urinalysis revealed a urine osmolality of 67 mosmol/kg and urinary sodium of 10 mmol/liter. A paired serum osmolality of 228 mosmol/kg was consistent with a dilute serum and urine, suggesting water overload. Neurological examination before intubation showed normal fundi and no focal neurological abnormality other than the marked confusion.. A lumbar puncture was normal as was subsequent cranial magnetic resonance imaging (MRI) showing no venous sinus thrombosis or evidence of central pontine myelinolysis.

The child sodium was initially corrected at a rate of 1 to 2 mmol/l/hour with hypertonic saline. The correction was done because of his ongoing seizure risk and stopped when the serum sodium reached 125 to 130 mmol/liter (**Table 1**). Too rapid correction of serum sodium can trigger demyelination of pontine and extrapontine neurons to occur after one or up to several days after the correction. This causes neurological dysfunction like quadriplegia, pseudobulbar palsy, seizures and death. It has been reported that osmotic demyelination have occurred after rates of correction is more than 12 mmol/liter per day [4]. A correction rate of up to 8 mmol/liter per day is recommended to reduce the risk of osmotic demyelination but can be cautiously increased if severe symptoms remain same [5]. The child was subsequently managed on the intensive care unit. After 10 days patient discharged from hospital with full recovery.

Table 1: Serum and Urine Electrolyte/Osmolality Results During Treatment

	Day 1	Day 2	Day 3	Day 4	Day 8	Day 10
Sodium (135–145 mmol/liter)	117	122	126	132	137	138
Potassium (3.5–4.7 mmol/liter)	3.5	3.7	4.0	4.0	4.2	4.6
Chloride (98–109 mmol/liter)	87	95	103	102	104	104
Urea (10–45 mg %)	14	16	19	24	21	26
Serum osmolality (280–300 mosmol/kg)	220	240	268	272	285	287

CONCLUSION

The cause of fitting in this patient is hyponatraemia due to overhydration by

untrained doctor. There is always mistakes done by even highly qualified doctors for adjusting drug dosing in children.

Symptoms are more severe with an acute reduction in serum sodium and occur due to cerebral edema. The earliest findings are typically nausea and malaise, followed by headache, lethargy and confusion. Eventually seizures, coma and respiratory arrest will follow. Overly rapid correction may also be hazardous causing central pontine myelinolysis, seizures, paraesthesia and striatal syndrome. Hyponatraemia can be life-threatening, if not diagnosed & treated early. In children overdosing with fluids & drugs is very common. It is very important to calculate the dose of all the fluids & drugs according to the weight of the children. Simple formulae exist to calculate the amount of sodium needed and how much hypertonic intravenous fluid will be required to allow safer correction.

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