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Case report

Transient seizure-induced sodium increase camouflaging a symptomatic hyponatremia

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SUMMARY

Hyponatremia is the most commonly observed electrolyte disturbance in clinical medicine. Occasionally the initial presentation of a patient with a symptomatic hyponatremia is a seizure or coma. This life-threatening complication needs early diagnosis and immediate treatment. Here, we report a case of a 27-year-old man who presented with an epileptic seizure, lactate acidosis and sulcal effacement on CT in which a transient sodium increase masked a clinically relevant hyponatremia thereby delaying diagnosis. This phenomenon is caused by an extracellular water shift and can occur when blood analysis is performed shortly after vigorous exercise or a seizure. This case provides awareness for a less well-known cause of plasma sodium increase and offers recommendations to prevent misinterpretation and help clinicians in decision making.

BACKGROUND

Hyponatremia is the most commonly observed electrolyte disturbance in clinical medicine.[1] Occasionally the initial presentation of a patient with a symptomatic hyponatremia is a seizure or coma. This life-threatening complication needs early diagnosis and immediate treatment.[2] Here we report a case of a patient who presented with an epileptic seizure, lactate acidosis and sulcal effacement on CT in which diagnosis was delayed due to a misinterpretation of the laboratory results. This case provides awareness for a less well-known cause of plasma sodium increase and offers recommendations to prevent misinterpretation and help clinicians in decision making.

CASE PRESENTATION

A 27-year-old man with no medical history presented with headache since the morning and increasing nausea and drowsiness during the day. He became progressively confused in the late afternoon after which his partner consulted a doctor. On arrival at the emergency department he had an epileptic seizure with spontaneous recovery. Neurological and physical examination were unremarkable. Clinically he was euvolemic. CT head showed diffuse sulcal effacement without signs of haemorrhage, basilar artery thrombosis, cerebral venous sinus thrombosis or an intracranial tumour. Venous blood gas analysis, taken minutes after the seizure, showed a pH of 6.89, Pco_2 10.8 kPa, bicarbonate 15.4 mmol/L, base excess −19.4 mmol/L and a lactate of 18.0 mmol/L. Blood chemistry was remarkable for sodium 131 mmol/L, chloride 82 mmol/L, albumin 51 g/L and an osmolality of 278 mOsmol/kg. The urine toxicology test was positive for acetaminophen but negative for opiates, amphetamines and cocaine. He was then treated with ceftriaxone, amoxicillin, acyclovir and dexamethasone awaiting the results of the lumbar puncture but the examination of the cerebrospinal fluid was normal.

This prompted us to reconsider the laboratory results. Patients relatively high haematocrit (0.48 L/L), together with the increased albumin level, were suggestive for haemoconcentration. However, we realised that measurements were done in a venous blood sample, which may have been 'haemoconcentrated' by water influx into the muscles during rhabdomyolysis. Therefore, we suggested that his arterial sodium concentration could well be lower, possibly low enough to explain the seizure. And indeed, in an arterial blood gas analysis taken 45 min after the seizure, the plasma sodium level was 118 mmol/L. Analysis of this hyponatremia revealed a urine sodium level of <20 mmol/L and an osmolality of 92 mOsmol/kg. He was diagnosed with primary polydipsia (the polyuria explaining the relatively low urine sodium concentration) and was admitted to the intensive care unit. In the first hours after admission his urine production was over 1 L/hour and the plasma sodium level rapidly increased to 128 mmol/L.

After regaining consciousness he told he had been drinking an excessive amount of water extra throughout the day (4–5 L) because he thought that his headache was caused by dehydration. He further explained that he was living a healthy lifestyle in which drinking ample amounts of water played a central role.

OUTCOME AND FOLLOW-UP

The following day he was transferred to the internal medicine ward where he was instructed to drink a maximum of 2 L of water a day. In the next 2 days his urine production normalised and the serum sodium level gradually increased to 141 mmol/L after which he was discharged from the hospital. He fully recovered and has not experienced any neurological symptoms after 4 months of follow-up.

DISCUSSION

Hyponatremia is a disturbance of fluid and electrolytes in which there is a relative excess of body fluid water compared with body sodium content.

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Reminder of important clinical lesson

It is generally defined as a sodium concentration of less than 135 mmol/L and is the most commonly observed electrolyte disturbance in clinical medicine with a reported prevalence of 4% in the adult internal medicine patient population treated in the emergency department and up to 30% in hospitalised patients.[3 4] Hyponatremia has a variety of etiologies and is most commonly caused by the syndrome of inappropriate antidiuretic hormone (ADH) secretion, the use of thiazide diuretics, congestive heart failure, severe vomiting and diarrhoea or polydipsia.[5] Symptoms are often mild and non-specific like headache, nausea and confusion but can be severe and life-threatening with cardiorespiratory distress, seizures and coma.[6] Severe symptoms are more commonly seen in cases of acute hyponatremia but also chronic hyponatremia in apparently asymptomatic patients must be considered a serious illness as it is associated with an marked increase in the risk of all-cause mortality.[7] Although rare, encephalopathy is the most serious complication of acute hyponatremia. It is the result of cerebral oedema and increased intracranial pressure caused by the osmotically driven movement of water into brain cells after the extracellular compartment has become hypoosmolar in a relative short period of time (<48 hours).[8] Left untreated hyponatremic encephalopathy could result in neurological deterioration or death.[2 9]

This case describes a man with an epileptic seizure as the initial presentation of acute hyponatremic encephalopathy for whom the diagnosis was made with a significant delay and that eventually lead to an unnecessary lumbar puncture. This delay can partly be explained by the absence of a good medical history as a result of postictal drowsiness which made it not directly clear that the patient had been drinking excessive amount of water in a short period of time. A clue for the diagnosis of an acute onset of the hyponatremia could have been his recent emerged headache and the finding on the CT head that showed signs of diffuse sulcal effacement. Although the latter is suggestive for cerebral edema, it is not specific for hyponatremia.[10] However, hyponatremic encephalopathy was mainly discarded as an initial diagnosis due to the fact that the first (venous) blood analysis taken shortly after the seizure showed a plasma sodium level of 131 mmol/L. Only after the second (arterial) blood test, which was performed 45 min later, a clinical significant hyponatremia of 118 mmol/L was found. This discrepancy cannot be explained by (pre)analytical errors as also a venous blood gas analysis, which was simultaneously taken with the initial blood drawing and which was measured on a different analyser, showed a comparable sodium level. As this sample was measured on a point-of-care blood gas analyser with a direct ion-selective electrode, pseudohyponatremia could be discarded. Interestingly, the first blood analysis also showed elevated haematocrit and albumin levels suggestive for haemoconcentration. The explanation for this relative hypernatremia is therefore water loss caused by a water shift from the extracellular compartment into the cells, presumably muscle cells considering his epileptic seizure. Rise of plasma sodium concentration has been described in subjects shortly after severe exercise or, as in this case, after seizures.[11–13] The shift of water from the extracellular compartment is driven by a rise in the effective number of osmoles in the skeletal muscle cells. During exercise, macromolecules, like glycogen, are quickly converted to many smaller molecules of L-lactate. The L-lactate ions that accumulate slowly diffuse from the cells thereby increasing intracellular osmolality relative to the interstitial tissues, leading to osmosis which drives water into the muscle cells. More importantly however, new osmoles created by the degradation of phosphocreatine into creatine and inorganic phosphates contribute to this process.[14] This intravascular water loss can rise plasma sodium concentration 10–15 mmol/L for 10–15 min after which the sodium level slowly restores to what it was before the seizure.[12 15] This effect would be most pronounced in blood taken from an affected limb (brachial or antecubital vein). Directly after the seizure venous blood (efferent) may show a much higher sodium concentration compared with an arterial sample (afferent), since the latter is a reflection of water homeostasis of the whole body and not merely from the affected limb.[14]

In case of hyponatremia, as described in our case, the sodium increase can lead to near normal or even entirely normalised plasma sodium levels causing misinterpretation. After strenuous exercise, a seizure or manifest rhabdomyolysis, we would therefore advise two strategies to correctly interpret plasma sodium concentration. First, repeated blood testing could possibly prevent misinterpretation as transient sodium increase may disappear rapidly. Second, an arterial blood sample may more accurately reflect true plasma sodium level since the effect of haemoconcentration due to rhabdomyolysis and thereby the increase in arterial plasma sodium is less prominent than in venous-derived blood taken from an affected limb vein.[14]

Learning points

- Symptomatic hyponatremia is a diagnosis that could potentially be falsely discarded when it is masked by a transient sodium increase after a seizure.
- Especially in severe cases with hyponatremic encephalopathy, where the initial presentation can be a seizure or a postictal patient with an unclear case history, this can lead to unwanted delay and unnecessary additional diagnostic tests.
- Next to awareness, also arterial blood gas analysis and repeated testing after a seizure with unknown cause, could prevent misinterpretation.

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