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Acute water intoxication in an adult man: 'dental hyponatraemia' revisited

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ABSTRACT

The differential diagnosis of hyponatraemia is notoriously wide. However, only a minority is acute, ie develops in less than 48 hours. We describe an unusual cause of water intoxication due to toothache. A 30-year-old man with no medical history of note presented in an acute confusional state. Laboratory results disclosed profound hyponatraemia. Urinary indices were consistent with overdrinking, but in the absence of a reliable history, other aetiologies had to be excluded. This case highlights the benefit of a structured approach in the assessment of electrolyte disturbances.

KEYWORDS: water intoxication, dental hyponatraemia

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Case presentation

A 30-year-old, previously healthy, man was airlifted to hospital because of acute confusion. After a planned day off work, his wife had found him standing naked in the bedroom in an agitated, disoriented state with unintelligible speech, and called emergency services. During a phone conversation 3 hours earlier, he had complained of nausea and headache.

In the emergency department, the patient was inarticulate and appeared confused and combative. He was afebrile. Pupils were equal and reactive and the neck was supple. No lateralising signs were apparent. There was no rash. He was sedated with propofol and midazolam. An urgent computed tomography of the head disclosed no abnormalities. A collateral history was provided by his wife: he was a smoker but did not take any recreational drugs. He was not on any medications. Three days earlier, while chopping wood, a small brick had hit his forehead, leaving a small scar just above his left socket. One day before his admission, he had attended a dental appointment.

Laboratory results revealed profound hypotonic hyponatraemia (sodium of 113 mmol/L); glucose, kidney and liver function were in the normal range, and inflammatory markers were not elevated. A urine toxicology screen was negative and alcohol levels were normal. Urinary sodium was <20 mmol/L, and urinary osmolality 29 mosmol/kg. The patient remained sedated on intermediate care. He received 250 mL of 3% saline, and empiric antibiotic cover with ampicillin/sulbactam. Over the following 35 hours, he voided 9,450 mL of urine, amounting to a net fluid deficit of 7,100 mL. In parallel, sodium levels increased to 138 mmol/L.

Differential diagnosis of acute hyponatraemia

Initially, the working diagnosis was acute hyponatraemia associated with the recent head trauma, causing the syndrome of inappropriate antidiuresis (SIAD). However, biochemistry results were more in keeping with a form of low-solute hyponatraemia, but the history and negative alcohol levels were not suggestive of beer potomania. Given the reported head trauma, injury of the pituitary causing secondary adrenal insufficiency was considered. In view of the acute onset of confusion and hyponatraemia, a diagnosis of limbic encephalitis was also entertained. A viral infection remained a remote possibility, specifically tick-born encephalitis, which is endemic in this part of Austria. Although acute psychosis *per se* can be a cause of SIAD, this was not deemed likely in the differentials.

Further investigations

Due to concerns of a viral or paraneoplastic encephalopathy, our patient underwent magnetic resonance imaging and a lumbar puncture, both yielding normal results. There were no focal findings on electroencephalography. Adrenal and thyroid function tests came back normal.

Case progression and outcome

Twenty-two hours after admission, the sedation was discontinued and the patient woke up and quickly became coherent. He was transferred to a normal ward the following day. He could now provide a detailed history: a bad molar had been bothering him for several weeks, before the pain became unbearable, prompting an urgent dental appointment on Monday morning when the tooth was duly extracted. Although the patient had been provided with analgesics, he found that drinking cool water eased the pain better and, therefore, began to drink large amounts of water. The next morning, the wound was still sore. Over the ensuing 5 hours, he ingested close to 10 litres of water (the patient recalled refilling his bottle (Fig 1) at least seven times). On this day, his food intake consisted of a single bread roll. His last recollection was the phone call from his wife around lunchtime.

Due to his sweaty job in a constantly hot environment, consuming a generous supply of fluids was second nature to him.

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Fig 1. Patient's water bottle (1.5 L capacity), which he recalled refilling at least seven times.

On an average working day, he would usually drink 5 to 6 litres of water.

We diagnosed acute dilutional hyponatraemia due to excessive water intake for relief of his toothache. Our patient developed rhabdomyolysis (peak creatine kinase of 43,244 U/L); however, kidney function remained normal throughout his hospital stay, and the patient was discharged after 1 week in his usual state of health. The trivial frontal injury had been a red herring.

Discussion

Acute water intoxication is mostly seen in psychiatric patients, with primary polydipsia and anorexia nervosa representing prime examples. Water intoxication without psychiatric causes has also been reported following urinary drug screening, preparation for colonoscopy, in association with exercise, or with drugs (particularly ecstasy).[1,2] Cerebral oedema is a feared consequence and symptoms can progress quickly from confusion to seizures and coma; brain herniation is the dreaded outcome. Acute symptomatic hyponatraemia is best treated with a bolus of hypertonic (3%) sodium chloride. A swift increase of sodium levels (by approximately 4 mmol/L) usually suffices to alleviate symptoms. If hyponatraemia develops within hours, the brain does not have time to fully adapt; therefore, the risk of osmotic demyelination syndrome in this setting is nil. The impressive aquaresis documented in our patient was the result of persistent, adequate antidiuretic hormone suppression, resulting in the excretion of dilute urine and quick normalisation of sodium levels.

The kidneys' capability to clear water is generally very robust. Traditional teaching is that eunatraemia is maintained even in the face of a water intake approaching 20 litres per day.[1] However, renal water excretion is also dependent on the amount of dietary solutes, mainly in the form of protein (metabolised to urea) and salt. If a diet is essentially devoid of such solutes, maximal urinary volume decreases significantly.[1] As the maximum water excretion per hour cannot exceed 1 litre, the speed of water intake is the second major mechanism for the development of dilutional hyponatraemia.[1] If too much fluid is ingested in too little time, hyponatraemia will invariably ensue.

Water intoxication in the 'dental context' is rare and seems better recognised in children and adolescents.[3,4] We could only retrieve one adult case report involving a 25-year-old woman who developed symptomatic hyponatraemia secondary to prolonged consumption of iced water for relief of dental pain.[5] The development of 'dental hyponatraemia' in a sturdy man without other health conditions appears unique and provides a caveat that even water can be too much of a good thing.

Key points

- Urinary indices are key in the assessment of dysnatraemias.
- The initial diagnosis of hyponatraemia hinges on the level of antidiuretic hormone (urine osmolality providing a viable proxy).
- Severe neurologic manifestations of hyponatraemia mandate urgent treatment with hypertonic saline.
- Excessive fluid intake can overwhelm renal capacities for water clearance.
- The perils of overdrinking deserve better recognition. ■

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