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BRIEF REPORT

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Fatal hyponatraemic brain oedema due to common gastroenteritis with accidental water intoxication

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Abstract Acute symptomatic hyponatraemia is a life-threatening emergency which must be diagnosed and treated promptly. The initial symptoms are often dramatic, with seizures and coma, and there is therefore a risk that the diagnosis and the urgent sodium correction therapy may be delayed by procedures such as computed tomography (CT) of the brain. As the most common aetiological factors are psychotic polydipsia and different iatrogenic causes, this condition usually develops in hospitalised patients. Water intoxication alone is very unlikely to cause severe hyponatraemia in a person with normal renal function, unless for some reason the antidiuretic hormone secretion is increased. We describe a case in which dehydration due to common gastroenteritis in combination with excessive intake of water caused the death of a young, previously healthy woman. Increased awareness of this potentially fatal condition is recommended.

Key words Acute hyponatraemia · Brain oedema · Computed tomography · Water intoxication

Introduction

Acute symptomatic hyponatraemia is a serious condition in which prompt elevation of the serum sodium concentration is imperative in order to prevent the development of fatal cerebral oedema [1]. The most common aetiological factors are psychotic polydipsia and iatrogenic administration of hypotonic fluids perioperatively [2]. Excessive intake of water alone is very unlikely to cause severe hyponatraemia in a person with normal renal function, unless for some reason the antidiuretic hormone secretion is increased [3]. Only a few reports of fatal accidental water intoxication in persons without psychiatric disease have previously been published. We describe here an additional case in which dehydration due to gastroenteritis in combination with excessive intake of water caused the death of a young, previously healthy woman.

Case report

A 27-year-old previously healthy mother of two, was brought unconscious to the emergency room at 10.20 p.m. with seizures and dilated pupils. The day before, her one-year-old son had fallen ill with diarrhoea. Our patient experienced her first symptoms 14 h before admission. Frequent diarrhoea and vomiting forced her to spend most of the day in the bathroom. In the afternoon when her husband came home he recommended her to drink lots of water. Taking his advice, she frequently drank directly from the tap during the next 3–4 h. When her husband came to check on her at 9.50 p.m. she seemed exhausted and was almost unresponsive. A few minutes later she had a seizure that lasted 2–3 min. On transportation to hospital she was unconscious but moved her arms and legs spontaneously. Upon arrival at the emergency room a new generalised seizure was observed and the patient was given 10 mg of diazepam intravenously. Her blood pressure was 110/70 mm Hg and her heart rate 60 beats/min. A bedside blood glucose test was normal. Blood samples for electrolyte analyses were not taken at this stage. The patient was intubated and immediately referred to the X-ray department for computed tomography (CT) of the brain. The CT scan was initially reported to be normal (Fig. 1 A). The patient was admitted to the medical intensive care unit at 11.30 p.m., where mechanical ventilation was instituted. By then she was in deep coma and did not respond to pain stimulus. Both pupils were fixed and dilated. Laboratory bedside test results, available at 11.45 p.m., showed a serum sodium concentration of 106 mmol/l and a serum potassium value of 2.1 mmol/l. The haemoglobin concentration was 92 g/l and the arterial blood gases were normal except for signs of hyperventilation. The body temperature was 36.5 °C and the urine production was approximately 150 ml/h. Nearly 2 h after admission to hospital, an infusion of hypertonic saline was started. The initial serum sodium correction rate was almost 3 mmol/l per h and this rate was maintained to a se-

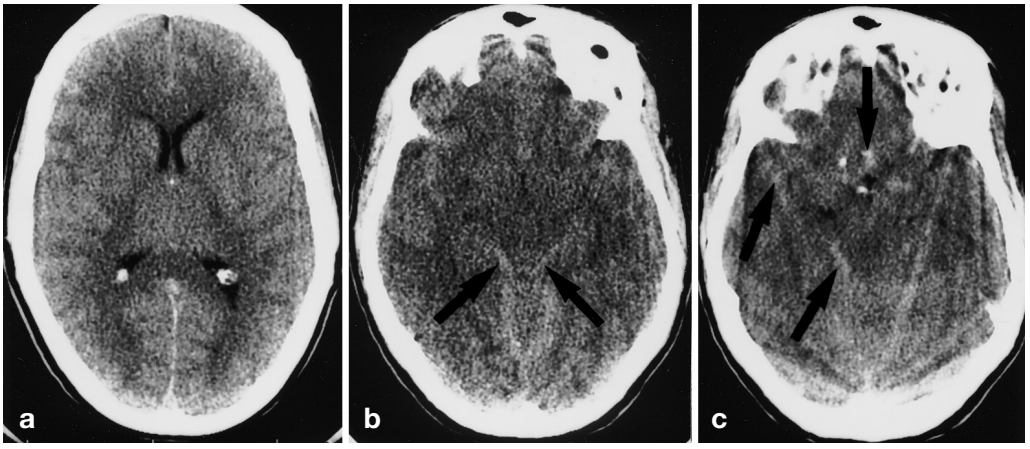

Fig. 1 A–C Three different CT sections obtained at the same examination in the reported patient **A** The CT scan was initially reported to be normal **B** At a second evaluation, compression of the cisterna ambiens was noted *(indicated by arrows)* and cerebral oedema was therefore judged to be present **C** Signs of a subarachnoid haemorrhage in the form of diffuse blood clots *(indicated by arrows)* were erroneously reported

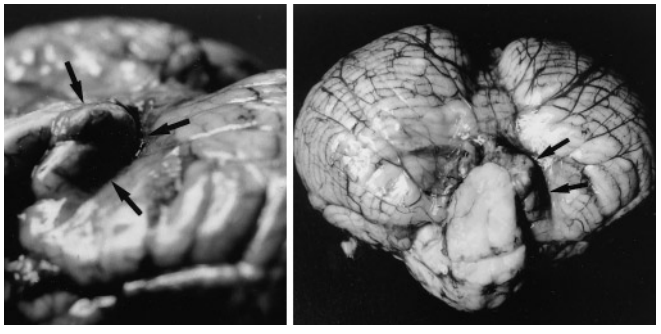

Fig. 2 At autopsy, the brain showed all signs of a pronounced cerebral oedema and a typical pressure cone *(arrows, left photograph)* and herniation of the cerebellum *(arrows, right photograph)*

rum sodium concentration of 127 mmol/l. A separate infusion of isotonic saline with 40 mmol of potassium was administered simultaneously. Furosemide 20 mg and betamethasone 8 mg were given intravenously. Further laboratory results showed C-reactive protein less than 5 mg/l, leucocyte count 12.3×10^9/l, a normal creatinine value of 49 μmol/l and a low serum osmolality of 226 mosm/kg. Eight hours after admission, a new evaluation of the CT scan gave rise to conflicting opinions among the radiologists. They now agreed that the picture was abnormal and showed cerebral oedema (Fig. 1 B). Moreover, signs of a subarachnoid haemorrhage were suspected to be present (Fig. 1 C). The patient remained unconscious and a brain death was diagnosed. Sixteen hours after admission, the mechanical ventilation was discontinued. At autopsy, pronounced cerebral oedema with cerebellar herniation was found (Fig. 2). There was no evidence of any intracranial haemorrhage.

Discussion

Hyponatraemia is reported to be the most common electrolyte disorder in hospitalised patients [4]. The serum sodium concentration normally varies between 135–147 mmol/l. If it falls below approximately 120 mmol/l there is a substantial risk of serious consequences. The major clinical symptoms of hyponatraemia are neurological and include headache, nausea, tiredness, motor impairment, speech disturbance, confusion, epileptic seizures and coma [3]. The appearance of severe symptoms correlates both with the serum sodium level and the rapidity of its fall [5]. When managing a patient with severe hyponatraemia, it is therefore of great importance to decide whether the electrolyte disturbance is of an acute or chronic nature [1].

A hyponatraemia that has developed during a period of more than 24–48 h is considered to be chronic and commonly gives rise to gradually appearing undramatic symptoms, as the brain cells have had time to adapt by reducing their contents of sodium and potassium [2]. Chronic hyponatraemia with mild-moderate symptoms should be corrected slowly, with a serum sodium correction rate of less than 0.5 mmol/l per h, to prevent therapy-induced neurological sequelae such as central pontine or extrapontine myelinolysis [1, 6]. Acute symptomatic hyponatraemia, on the other hand, is an emergency with poorer prognosis in which prompt and rapid sodium correction is imperative to prevent cerebral herniation and death [2, 7]. When the serum sodium concentration suddenly falls, the decrease in extracellular osmolality leads to a rapid redistribution of water to the intracellular space, which may result in cerebral oedema [2, 5]. Whenever the available data do not permit discrimination between chronic and acute hyponatraemia in a patient with seizures or in coma, the best strategy is probably to give rapid correction therapy for 3–4 h and then to reduce the correction rate to less than 0.5 mmol/l per h [8, 9].

The present case report illustrates several points of importance with regard to the diagnosis and management of patients with symptomatic hyponatraemia of acute onset. First, despite the fact that the initial management of a young, previously healthy individual with serious cerebral symptoms poses a severe challenge and calls for prompt decision-making, it is of great importance that a careful history be obtained before deciding on further diagnostic procedures. A history of true volume depletion (gastrointestinal or renal losses) fol-

lowed by excessive intake of water in combination with severe cerebral symptoms must necessitate immediate analysis of the electrolyte levels. Nowadays, most hospitals have laboratory equipment capable of the rapid measurement of serum sodium and potassium concentrations, often coupled with the determination of arterial blood gases, and these laboratory tests should not be delayed by other diagnostic measures such as CT scan of the brain. In our patient, the analyses of the serum sodium and potassium levels were delayed for about 85 min.

Second, the finding of a low serum sodium concentration in combination with clinical signs of cerebral oedema and a history suggestive of acute development of the hyponatraemia should lead to prompt and rapid initial correction of the electrolyte disorder. A serum sodium correction rate of 1–2 mmol/l per h to a serum sodium concentration of 120–125 mmol/l is recommended, and this is most effectively achieved by the infusion of hypertonic saline [1, 7]. An estimate of the optimal infusion rate, expressed as millimoles of sodium per hour, is roughly given by the product aimed serum sodium correction rate x total body water (TBW, estimated as $0.5 \times$ kg body weight in women and $0.6 \times$ kg body weight in men). The total amount of sodium in millimoles required to raise the serum sodium concentration from 106 to 120 mmol/l, may be approximately estimated by the formula TBW x (120 – 106). The infusion rate should be adjusted under the guidance of hourly determinations of the serum sodium concentration. As the risk of therapy-induced neurological sequelae is minimal in acute hyponatraemia [2, 10], an initial rate of correction as rapid as that in the patient reported here is acceptable in cases with severe symptoms. However, at no time should the serum sodium concentration be allowed to increase by more than 25 mmol/l during the first 48 h, nor should the normal serum level be allowed to be reached during this time [10].

Third, the prognosis in untreated acute severe hyponatraemia is serious, especially in premenopausal women [11]. The incidence of severe hyponatraemia is reported to be similar in men and women [3]. Premenopausal women and children, however, seem to be more sensitive to a rapid decrease in serum osmolality and develop permanent brain injury more often [3]. A proposed explanation for this is that oestrogen may impair and testosterone promote brain adaptation to hyponatraemia. In the present case, the treatment after the diagnosis finally had been established, with rapid sodium correction and the usual measures against cerebral oedema was correct. However, a therapy-resistant brain oedema with cerebellar herniation had developed and caused the death of this young woman.

Fourth, the interpretation of CT scans of the brain is difficult in cases of suspected brain oedema. In the present case, the CT scan was first erroneously reported to be normal. At final evaluation, however, the presence of cerebral oedema was agreed upon, but the possibility of a subarachnoid hemorrhage was also raised. Similar misjudgements by experienced radiologists have been reported previously [12].

In conclusion, a more rapid recognition of the aetiology of the severe cerebral symptoms in this young woman may have led to a more favourable outcome. This case is reported in order to increase the awareness of this potentially fatal condition and to emphasise the importance of obtaining a proper history before deciding on time-consuming, sometimes unnecessary and often inconclusive diagnostic procedures such as CT of the brain. Acute symptomatic hyponatraemia is a life-threatening emergency which must be diagnosed and treated promptly.

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