CASE REPORT

When plenty is too much: water intoxication in a patient with a simple urinary tract infection

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Accepted 25 August 2016

SUMMARY

Healthcare professionals frequently advise patients with simple infective illness to drink more fluids. Here, a 59-year-old woman with a urinary tract infection followed such advice resulting in hospital admission with symptomatic acute hyponatraemia. Water intoxication is well recognised as a cause of symptomatic hyponatraemia in endurance sports, MDMA use and psychogenic polydipsia. It has rarely been described outside of these circumstances. With normal renal function, it is difficult to overwhelm the excretory capacity for water. However, in infective illness, increased levels of antidiuretic hormones (which may be secreted both appropriately to correct volume status and inappropriately as a feature of disease) reduce renal excretion of water. In this scenario, could increased administration of oral hypotonic fluids lead to hyponatraemia, with associated morbidity and mortality, than has previously been recognised? There is a need for more research to qualify our oft-given advise to drink more fluids.

BACKGROUND

We frequently advise our patients to 'drink plenty of fluids' and 'keep well hydrated' when they are unwell. But, what do we mean by that? Are there potential risks of this apparently harmless advice? Here, we present a case of a 59-year-old woman, who drank a large volumes of fluid to 'flush out her system' when she felt the telltale signs of a urinary tract infection (UTI) recurring. Her zealous self-administration of fluids resulted in a hospital admission for symptomatic acute hyponatraemia.

Acute hyponatraemia (defined as hyponatraemia developing over <48 hours) is a medical emergency with potentially fatal neurological sequelae. It requires prompt recognition and action. Hyponatraemia (defined as Na <134 mmol/L) is a feature in 4% of presentations to the emergency department, with a case mortality rate of 17.9%.[1] Initial symptoms are non-specific, including headache, nausea, vomiting, tiredness, disorientation and speech disturbance. These can progress to seizures, coma and death due to cerebral oedema with raised intracranial pressure and consequent cerebellar herniation. The severity of symptoms relate to the overall sodium level and the rapidity of its decline.[2]

Voluntary water intoxication as a response to medical advice and illness prevention is a rarely reported cause of hyponatraemia in those with normal physiology. If renal function is normal, it is difficult to consume more water than can be excreted, unless antidiuretic hormone (ADH) levels are increased. Fatal water intoxication has been reported in psychogenic polydypsia,[3] endurance exercise,[4] after ingestion of MDMA[5] and anecdotally during university hazing activities as well as during water-based torture rituals. In the case reported here, simple medical advice about therapeutic water consumption was followed to ill effect. This mirrors a previous case report,[6] in which a young, previously healthy female drank excessive amounts of water during an episode of uncomplicated gastroenteritis, resulting in acute hyponatraemia and death. Together these two cases highlight the importance of clear history taking (including a collateral), prompt investigation and correction of electrolyte imbalance and, also the need to qualify our advice regarding water consumption in simple infective illness.

CASE PRESENTATION

A 59-year-old woman presented to the emergency department with symptoms and a urine dip consistent with a UTI and was triaged to the minors department. She was prescribed antibiotics and analgesia. During her visit to the emergency department, she became progressively shaky and muddled. She vomited several times, was tremulous and exhibited significant speech difficulties.

Taking an ABCDE approach to this evolving situation, there was no compromise of the patient's airway, her breathing was rapid and shallow with normal oxygen saturations and chest examination. She was euvolaemic, with a regular heart rate of 74 bpm and blood pressure at 126/87 mm Hg. Blood sugar was in normal range. Neurologically, the patient's Glasgow Coma Scale was 15. She displayed hesitant speech, intermittent word finding difficulties but normal comprehension. She was distractible with an attention deficit, otherwise her neurological examination was normal. The patient was afebrile.

Owing to the new onset of word finding difficulties and the attention deficit a stroke call was put out and blood tests and CT scan were ordered (see the Investigations section for results).

Her partner described that the patient had woken up that Sunday morning with dysuria and lower abdominal pain, which she had attributed to her recurrent UTI. Throughout the day she had consumed several litres of water based on medical advise she recalled from previous similar episodes. She attended the emergency department late the same day as her symptoms had not subsided.

To cite: Lee LC, Noronha M. *BMJ Case Rep* Published online: [*please include* Day Month Year] doi:10.1136/bcr-2016-216882

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The patient had no medical history other than recurrent UTI. She took no regular medications and reported no allergies. She was a non-smoker, with moderate alcohol intake and no recreational drug use.

INVESTIGATIONS

Initial venous blood gas results demonstrated hyponatreamia (123 mmol/L), alkalaemia (pH 7.5) with a reduced venous pCO_2 (3.71 kPa) and venous bicarbonate (21 mmol/L). Formal laboratory results confirmed the above bedside tests, and showed marginally raised inflammatory markers (white cell count 10.72×10^9 L, C reactive protein 3 mg/L) and reduced plasma osmolality (257 Osmol/kg). There were no other haematological or biochemical abnormalities. Thyroid function was within normal range. Unfortunately, urine was not collected for osmolality or sodium levels.

ECG revealed sinus rhythm with no acute change.

CT scan head was unremarkable, with no evidence of cerebral oedema. The following day, morning cortisol was measured and found to be within normal range.

OUTCOME AND FOLLOW-UP

The patient was fluid restricted to 1 L over the following 24 hours. By the following morning, she felt clinically much improved. Serum sodium was 135 mmol/L 13 hours after the initial sample. She was discharged that day with follow-up review and blood tests in the outpatients department.

DISCUSSION

As healthcare professionals we often advise patients to drink plenty of fluids when faced with mild infective illness. The theoretical benefit of increased oral fluid intake is mitigating any depletion in intravascular volume due to systemic vasodilation, increased vascular permeability, reduced oral intake and replacing fluids lost in fever and vomiting. In urine infection, water diuresis has been found to temporarily reduce colony count of bacteria in urine,[7] but the clinical correlation of this result is not clear. As demonstrated here, the harmful effects of increased fluid intake include confusion, vomiting and speech disturbance, and potential for catastrophic outcomes due to low blood sodium concentrations.[6] There is a paucity of research

Patient's perspective

- In 2015 the year was one of 364 days, I lost 24 hours of my life when a simple UTI, or rather my actions in response to it, took over my life.
- I have, since my 20s, had periods where cystitis and related UTIs have flared up. The cause has never been clear although some things seem to aggravate, if not contribute, to it; these include sex, water changes, travelling on planes and stress. I do not recall if this occasion could be related to a known trigger but I recognised a dull 'thumbprint' pressure in my lower abdomen on waking that Sunday morning. I know that when this occurs I must (a) drink lots of water and (b) get to the doctor or accident and emergency (A&E) department quickly to get antibiotics; otherwise 4–6 hours later I am passing blood in my urine and in severe pain. The 'drinking lots of water' advice was given many years ago and I remember that 'half a pint every half an hour' was the guideline. Usually I would have gone to Kings A&E department immediately; however, my partner was arriving a bit later and I thought there was no harm waiting but I had better make sure that I did drink a lot. I am sure I exceeded the 'half a pint every half an hour' advice because I knew how quickly the infection usually progresses and I thought I could stall it a little. I will not ever delay again!
- During our wait in the A&E department I continued consuming water and although I felt rotten I did not notice anything unusual. We had a while to wait, and then again once in the clinic, but all seemed to follow the normal routine. Although I was increasingly feeling lightheaded and sick, which is not at all normal for me, I attributed these feelings to the delay in getting treatment and thought that once I had the drugs I would be happy to go home and collapse. This was not to be, apparently my partner got concerned when I grabbed his arm rather strongly as I descended from the trolley to go home 'Not like you at all' he said afterwards —I should explain I am fit and strong, independent and almost never ask for help. He sat me down having decided I should be readmitted, and was in the process of doing so when he noticed me tottering towards the loo. His words were 'you never totter and never ask me into the loo, when you did I knew it was serious'. That is the end of any accurate memory I have of the day.
- I have patchy recollections of being asked questions I understood but seemed unable to articulate answers for and found this distressing. I remember seeing my partner looking grave and forlorn, which was more frightening than what was happening as I did not know or understand what was happening. I remember seeing my hand in front of me shaking rather violently and I wondered why I could not stop it, then realised that my whole body was shaking. At that point I became terrified and am told that on three occasions I said I was 'turning into my mother'. This refers to the fact that she suffered an unexpected and huge stroke about 18 months before, she was given hours to live—she did in fact recover to almost full functionality. What must have been hours later, in the small hours of the morning, I noticed a huge bruise on my hand; my partner said it was where a cannula had been, I do not remember that. I do not mind the not remembering, what was very unsettling was knowing and feeling things I could not express and, as someone used to managing everything and being articulate, not being in control.
- Something like 24 hours later I was bored, a sure sign I was on the mend! I was quite weak but strong enough to walk myself to a car and get home. It took about a week to feel 'normal' again and if I am honest I think I was tired for at least another week. The episode was something of a shock. I think of myself as indestructible, can cope with anything and will work through any inconvenient malaises—I know how to manage almost everything. Well not this time. My partner told me the hospitable staff were very attentive but were obviously somewhat mystified by my symptoms which, once understood, were quickly dealt with. I read the article written by Laura Lee with interest as it gives me an insight into a lost day in my life. It is also informative in terms of understanding the implications of my actions in, of me, a not uncommon situation.
- My own conclusions are: 1) follow your instincts go to get the drugs as soon as you recognise the symptoms; 2) drink half a pint every half an hour and no more; 3) make sure your will and power of attorney are sorted.
- Thank you to the staff at Kings A&E department.

weighing the risks and benefits of advice to 'drink more fluids'. Indeed, in a Cochrane review, there was found to be no evidence for or against increased oral fluid intake in acute respiratory infection.[8]

The case reported here described a 59-year-old woman who drank large volumes of fluids due to its perceived therapeutic value when suffering from a UTI. She developed disorientation, nausea, vomiting, a generalised tremor and speech production deficit. Bedside blood tests allowed a speedy identification of her serum sodium level (123 mmol/L) as the causative factor in her acute deterioration, and with simple fluid restriction, deleterious consequences were averted. A mortality rate of 28.9% has been reported for patients presenting to the emergency department with serum sodium of <125 mmol/L.[1] To reiterate a point made by Sjobolm *et al*,[6] the presentation of a previously, healthy adult with acute cerebral symptoms creates a time-critical clinical challenge; however, this must not detract from the need for a comprehensive collateral history to guide diagnostic approach. A history of excessive water intake combined with possible intravascular depletion should prompt speedy analysis of serum electrolyte levels.

Water intoxication during an episode of infective illness is rarely recognised as a cause of acute symptomatic hyponatraemia. In order to develop hyponatraemia, the capacity to renally excrete water must be impaired (except in psychogenic polydipsia where consumption of 10–15 L of fluid a day overwhelms renal function[3]). There is an excess of ADH despite low plasma osmolality. This can be either inappropriately or appropriately driven, through non-osmotic triggers.[9] Although several infections are known to induce ADH release,[9] there are no reports of an association between UTI and inappropriate secretion of ADH. In the case described here, non-osmotic factors, such as vomiting, sweating, systemic vasodilation and increased vascular permeability, most likely appropriately drove secretion of ADH to restore euvolaemia. The increased ADH levels when combined with excess fluid intake resulted in acute hyponatraemia. Indeed, in the emergency department, it has been reported that the majority of cases of hyponatraemia are associated with hypovolaemia secondary to underlying disease, most commonly of the gastrointestinal and urogenital systems.[1] If it is the case that the majority of patients presenting to the emergency department with hyponatraemia are hypovolaemic due to underlying disease, it follows that many of these individuals may have raised ADH levels as a feature of their illness. Is the common medical advice to 'drink more fluids' a potential additive factor to their electrolyte disturbance and therefore also increasing associated morbidity and mortality?

In conclusion, the above discussion adds weight to the need for more research into the basis of the frequent common advice to 'drink more fluids'. What are the true risks and benefits of increased fluid intake and how frequently do these occur? Is excess hypotonic fluid intake an under-recognised factor in the aetiology of acute hyponatraemia, at least in presentations to the emergency department? How are we better able to guide our patients about how much to drink when they are unwell? Is there a place for drinking sodium replete fluids or measuring urine osmolality at home?[10]

Learning points

- Acute symptomatic hyponatraemia requires prompt recognition and action.
- It is crucial to take a comprehensive history to guided diagnostic approach in a previously healthy adult presenting with acute cerebral symptoms. A history of excess fluid intake combined with possible intravascular depletion should warrant prompt measurement of serum electrolytes.
- There is a paucity of evidence behind the advice to 'drink plenty of fluids' in the management of mild infective illness. This needs to be addressed, especially considering the significant morbidity and mortality of acute hyponatraemia.

Contributors LCL and MN managed the case together and subsequently developed the idea for the paper. LCL performed the literature search, drafted and revised the paper. She is guarantor. MN revised the paper.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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