Rare disease

Hyponatraemia associated with cardiac tamponade and chronic fluid excess

M P Weekes,¹ B A J Reddi,² S Wharton,³ A Gazis⁴¹ Department of Infectious Diseases, University of Cambridge Clinical School, Cambridge, UK² Emergency Department, Royal Adelaide Hospital, Adelaide, Australia³ Department of Respiratory Medicine, Nottingham University Hospitals NHS Trust, Nottingham, UK⁴ Department of Endocrinology and Diabetes, Nottingham University Hospitals NHS Trust, Nottingham, UK

Correspondence to M P Weekes, weekes@doctors.net.uk

Summary

We describe a complex case of hyponatraemia with two aetiologies. A 49-year-old man who drank 6 litres of dilute alcohol per day presented confused and oedematous with a serum sodium of 95 mmol/litre. Urine sodium was < 10 mmol/litre and urine osmolality 440 mOsmol/kg. Chest x-ray demonstrated a globular heart. ECG showed saddle-shaped ST elevation. ECHO demonstrated a large pericardial effusion causing marked tamponade. Following pericardiocentesis there was a marked diuresis; serum sodium returned to normal after 2 weeks. A full recovery ensued. Cardiac tamponade is associated with antidiuresis via release of antidiuretic hormone (ADH). Tamponade is also associated with antinatriuresis. Antidiuresis and antinatriuresis usually balance in cardiac tamponade; excessive fluid intake may have caused an imbalance in this case.

BACKGROUND

This case informs the reader both of a rare cause of hyponatraemia, cardiac tamponade, and provides a reminder of a more common cause, polydipsia. There are only three case reports in the literature of hyponatraemia in association with cardiac tamponade. One report in 2007 describes a similar patient with chronic excessive fluid intake who developed an idiopathic pericardial effusion and subsequently became hyponatraemic with high urine osmolality. Excessive fluid intake in the context of cardiac tamponade might predispose to the development of hyponatraemia.

CASE PRESENTATION

A 49-year-old unemployed man presented to the emergency department with a 2-week history of fatigue, confusion and falls. He had smoked heavily for 30 years, drank eight cans of 0.2% beer and four pints of 4% beer daily and ate at most one small meal per day. He used no medications. He had no significant past medical history or family history.

On examination, he was drowsy with Glasgow Coma Score 14/15. His speech was slurred and he exhibited psychomotor slowing. His blood pressure was 112/84 mm Hg, his pulse was 150 bpm and irregularly irregular, SaO₂ was 94% on air and his temperature 36.4 °C. Heart sounds were quiet, jugular venous pressure was raised at 9 cm and he had clinical evidence of a small left basal pleural effusion. He was oedematous in his hands and to mid-shins. There was generalised limb weakness but no focal neurological deficit.

INVESTIGATIONS

See table 1.

Free T₄ was 13.6 pmol/litre and thyroid-stimulating hormone was 0.6 mIU/litre. A 250 µg short synacthen test

demonstrated a rise in serum cortisol from 959 to >1400 nmol/litre, which was interpreted as normal.

C3 was 1.34 g/litre, C4 0.25 g/litre, anti-double-stranded DNA antibodies were negative, anti-nuclear antibody titre was positive at 1 in 800; anti-PR3, myeloperoxidase, mitochondrial, smooth muscle and reticulin antibodies were negative.

Chest x-ray demonstrated a globular heart and a small left pleural effusion.

ECG showed atrial fibrillation with widespread saddle-shaped ST elevation.

CT of head, chest, abdomen and pelvis were normal apart from cerebral atrophy. ECG demonstrated a large pericardial effusion causing right ventricular diastolic collapse and marked tamponade. Further physical examination demonstrated a 24 mm Hg paradox.

TREATMENT

The patient was initially empirically treated with 500 ml of 1.8% saline over 10 h then 500 ml of 2.7% saline over 18 h for life-threatening hyponatraemia. Oral fluid restriction was instigated. Chlordiazepoxide, pabrinex and intravenous co-amoxiclav were administered to treat alcohol withdrawal, possible thiamine deficiency and possible bacterial infection. Serum Na rose from 95 to 100 mmol/litre over the course of his infusion of hypertonic saline.

Following ECG, pericardiocentesis was performed because of tamponade. There was 2.2 litres of blood-stained fluid removed. Over the next 24 h, there was a diuresis of 3.7 l (figure 1). Fluid balance remained negative for some days. Oral fluid restriction was relaxed and serum sodium rose steadily to normal by day 15 (figure 1).

Table 1 Initial investigations

Haemoglobin	13.6 g/dl	Plasma Na ⁺	95 mmol/l
White cell count	21.5 × 10 ⁹ /l	Plasma K ⁺	4.6 mmol/l
Neutrophil count	19.2 × 10 ⁹ /l	Plasma urea	2.2 mmol/l
Platelet count	307 × 10 ⁹ /l	Plasma creatinine	49 µmol/l
		Plasma osmolality	198 mOsmol/kg
C reactive protein	60 mg/l		
Alcohol	10 mg/dl	Urine Na ⁺	<10 mmol/l
Lipase	44 U/l	Urine osmolality	440 mOsmol/kg
Creatine kinase	1556 U/l		
		Gamma-glutamyl transpeptidase	130 U/l
Corrected Ca ²⁺	2.07 mmol/l	Alkaline phosphatase	94 U/l
PO ₄ ²⁺	0.55 mmol/l	Alanine transaminase	72 U/l
Mg ²⁺	0.95 mmol/l	Bilirubin	17 µmol/l
		Albumin	34 g/l

Figure 1 Response to pericardiocentesis: gradual and sustained rise in serum sodium (left axis; diamonds) with significant diuresis following pericardiocentesis (urine output in millilitres per 12 h shown on right axis; black squares). Prior to pericardiocentesis, treatment with hypertonic saline and oral fluid restriction had been instigated as detailed in the text; fluid restriction was relaxed shortly after pericardiocentesis.

OUTCOME AND FOLLOW-UP

The pericardial fluid contained degenerative inflammatory cells. Cytology for malignant cells and microbial culture was negative. Repeat ECG 1 day and 4 weeks after pericardiocentesis showed no re-accumulation of fluid.

The patient improved clinically. Following pericardiocentesis, his peripheral oedema resolved and he reverted to sinus rhythm. In clinic follow-up he remained abstinent from alcohol with an improved diet; his serum sodium continued to be normal. He suffered no long-term neurological sequelae.

DISCUSSION

Hyponatraemia was initially thought to be at least partly due to low solute intake and excessive fluid intake. The patient's diet contained a daily solute intake of approximately 200 mOsmol/24 h (normal is approximately 750 mOsmol/24 h) and he drank approximately 6 litres of dilute alcohol and other fluid per day. As maximally dilute urine

has an osmolality of around 50 mOsmol/litre, the patient's maximum possible daily free water clearance was approximately 200/50 = 4 litre. The remaining free water excess of approximately 2 litre/day could have led to significant hyponatraemia—a pattern recognised in both primary polydipsia and excessive beer drinking. However, the patient had a surprisingly high urine osmolality and was oedematous; neither of these findings would have been expected in polydipsic hyponatraemia.

Hypothalamic osmoreceptors detect plasma osmolality and regulate ADH secretion by the posterior pituitary. Dilute plasma would, under normal circumstances, inhibit ADH secretion, increasing free water clearance by the kidney leading to restoration of normal osmolality.

Atrial and carotid baroreceptors detect intravascular volume status. Significant circulating volume depletion causes these receptors to override hypothalamic osmoreceptors and stimulate release of ADH. The net effect is to protect the circulation against further volume loss.

True volume depletion may occur with conditions such as gastrointestinal fluid losses, burns or haemorrhage. Apparent volume depletion can occur in states of overall volume overload, such as congestive cardiac failure and cirrhosis.

Cardiac tamponade is associated with antidiuresis via release of ADH.¹ Though the mechanism is poorly understood, decreased effective circulating volume due to the effect of tamponade on cardiac chamber filling and diastolic compliance may lead to stimulation of atrial and carotid baroreceptors.² There are a variety of other mechanisms responsible for control of ADH secretion³ that may also be involved. An elevation of ADH might not only have contributed to the hyponatraemia, but also the relatively high urine osmolality. Further impairment of urine dilution may have been caused by other ADH-independent mechanisms in the presence of a habitually high fluid intake.⁴

Cardiac tamponade is also associated with antinatriuresis partly via activation of the sympathetic nervous system and partly via activation of renin-angiotensin-aldosterone (due to combined renal and extrarenal effects of decreased cardiac output). There is evidence that in tamponade, although atrial pressure rises, there is no increased release of atrial natriuretic peptide (ANP) as atrial stretch rather than atrial pressure is thought to be more important in stimulating ANP release.^{2, 5} A study of 10 patients with pericardial tamponade found that the level of ANP increased approximately fivefold after pericardial fluid was evacuated with a corresponding rise in urine output. These findings correspond to the patient's initially low urine sodium and rapid diuresis following pericardiocentesis.

Antidiuresis and antinatriuresis usually balance in cardiac tamponade resulting in normal serum sodium. The patient's excessive fluid intake may have caused these responses to become imbalanced.

Two case reports in the English literature associate hyponatraemia with cardiac tamponade caused by a slowly accumulating haemorrhagic effusion due to warfarin toxicity⁶ and adenocarcinoma of the lung.⁷ One case report published in 2007 by Shafique et al⁴ described a patient strikingly similar to our patient. Their patient was 59 y, previously healthy, who, as an electrician, habitually drank 1–2 gallons of water daily. He presented with a 2-week history of productive cough and fever. Examination revealed paradox of 15 mm Hg and peripheral oedema. Sodium on presentation was 104 mmol/litre. Urine sodium and urine osmolality showed a similar pattern to our patient: 5 mmol/litre (low) and 641 mOsm/kg (high), respectively. ECHO demonstrated large pericardial effusion with evidence of tamponade. Following pericardiocentesis, there was a similar rapid diuresis but with a much faster correction of serum

sodium (from 105 to 123 mmol/litre within 18 h). Chest x-ray and CT showed evidence of right-sided consolidation. Despite extensive analysis of pericardial fluid, no cause was found for the accumulation of the effusion. Both their patient and our patient had idiopathic pericardial effusions, which might have related to infection at another site or another systemic disorder.

In conclusion, we report an unusual case of hyponatraemia, associated with chronic excess fluid intake and tamponade secondary to an idiopathic pericardial effusion. Why an imbalance between antinatriuresis and antidiuresis develops in some rare cases of tamponade is unclear—it is possible that excessive fluid intake in the context of tamponade might predispose to the development of hyponatraemia. The true incidence of hyponatraemia associated with cardiac tamponade is unknown.

Learning points

- ▶ In patients with hyponatraemia and dilute urine, consider water intoxication.
- ▶ Hyponatraemia with a high urine osmolality suggests water intoxication is not the cause or not the only cause.
- ▶ Cardiac tamponade is usually associated with normal serum sodium.
- ▶ The combination of chronic excessive fluid intake with cardiac tamponade may lead in some cases to hyponatraemia with high urine osmolality.

Competing interests None.

Patient consent Obtained.

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