CASE REPORT

Beer, hyponatraemia and cardiac conduction defects

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SUMMARY

The authors report a case of a 68-year-old man who was diagnosed with an irreversible second-degree atrioventricular (AV) Mobitz II 2:1 block temporally associated with profound hyponatraemia. The cause of the hyponatraemia was beer potomania. The co-occurrence of reversible first, second and third-degree heart blocks and hyponatraemia has been described in a few published case reports. However, this case is noteworthy as the AV block persisted, despite correction of serum sodium concentration as opposed to other published cases, which meant that the patient required a permanent pacemaker.

BACKGROUND

Hyponatraemia is frequently encountered in hospitalised patients.¹ The cardiac electrophysiological effects of sodium are rarely clinically significant²; however, cases of cardiac conduction defects temporally associated with hyponatraemia have been previously described.³ Patients with hyponatraemia, especially those with profound hyponatraemia, should be observed via cardiac monitoring to detect any cardiac conduction defects.

CASE PRESENTATION

A 68 year-old male alcohol misuser presented to the emergency department in an obtunded state. His relatives reported that he had been drinking 15 pints of beer daily for the last few months. However, lately, he had been skipping meals and also reported some episodes of vomiting. He had a medical history of hypertension well controlled on amlodipine. The patient did not have any medical history of cardiac disease.

Physical examination revealed a bradycardia of 38 beats/min. He was normotensive, afebrile and had an oxygen saturation of 94% on room air. He had a Glasgow Coma Scale score of 9. The patient was not cooperative for a full neurological examination; however, there were no pupillary abnormalities and he had bilateral flexor plantar reflexes. There were no signs of hypovolaemia.

Laboratory data revealed a hyponatraemia of 98 mmol/L (135–145 mmol/L), a hypokalaemia of 3.36 mmol/L (3.5–5.1 mmol/L) and a hypochloreaemia of 59 mmol/L (98–106 mmol/L). The hyponatraemia was true as evidenced by the low serum osmolality of 218 mOsm/kg. The urinary sodium level was 12 mmol/L (54–190 mmol/L). Hypothyroidism and adrenal insufficiency were also excluded.

Serum creatinine, urea, calcium, phosphate and magnesium were within normal limits. Complete blood count, C-reactive protein and liver function tests were unremarkable. Serial high-sensitivity troponins were also negative.

An ECG revealed a new-onset second-degree atrioventricular (AV) Mobitz II 2:1 block ([figure 1](#)). ECGs taken 1 month previously during an admission with alcohol intoxication showed sinus rhythm ([figure 2](#)).

A CT brain scan revealed mild atrophy and no pulmonary lesions were noted on a chest X-ray.

The patient was initially given 1 L of 0.9% saline at the emergency department and a temporary pacemaker was inserted in view of the bradycardia. He was subsequently admitted to the intensive therapy unit for sodium correction with 3% saline and close monitoring. After 160 mL of hypertonic saline, the serum sodium climbed up to 113 mmol/L. In view of the fast rate of change of serum sodium concentration, the hypertonic saline was stopped and the patient was given 5% dextrose. The progression of serum sodium during the first 72 hours is summarised in [figure 3](#).

In addition, the patient was also prescribed lorazepam, thiamine and vitamin B supplements to reduce the risk of alcohol withdrawals and Wernicke-Korsakoff syndrome, respectively.

OUTCOME AND FOLLOW-UP

The patient made steady improvement and after 24 hours was alert and able to communicate. He was advised to restrict fluids and was kept on 0.9% saline. After 4 days, the patient dislodged the temporary pacemaker wires and there was failure to capture and sense. In view of the risk of R on T pacing, the pacemaker was turned off. The patient remained well but the AV block persisted despite the fact that the sodium level had improved to 130 mmol/L. As a result, the patient underwent a permanent pacemaker insertion.

After 11 days in hospital, the patient was discharged fully independent with a serum sodium of 132 mmol/L. He was advised to cut down on alcohol intake and to restrict his free water intake to 1.5 L/day.

DISCUSSION

Beer potomania is a syndrome of euvoalaemic, hypotonic hyponatraemia due to excessive beer intake and poor solute intake. It was first recognised in 1971 by Demanet *et al.*⁴

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Figure 1 Rhythm strip (lead II), rate 25 mm/s, amplitude 10 mm/mV, second-degree atrioventricular (AV) Mobitz II 2:1 block.

Figure 2 Rhythm strip (lead II), rate 25 mm/s, amplitude 10 mm/mV, sinus rhythm, taken 1 month previously during an admission for alcohol intoxication.

The pathophysiology of beer potomania revolves around the fact that free water clearance via the kidneys necessitates an adequate daily intake of solutes.⁵

In patients with normal renal function, ingestion of large amounts of free water does not lead to hyponatraemia. This is possible as the kidneys are capable of excreting copious amounts of dilute urine provided there is good and adequate amount of solute intake. The intake of solutes is necessary for the kidney to excrete free water. Without proper solute intake, the normal functioning electrolyte gradient that pulls free water from the plasma into the urine is effectively destroyed and hence free water excretion is impaired. Since free water excretion is impaired, there is free water retention leading to a hypotonic hyponatraemia.⁵

Beer is very poor in solute content. Hence, in patients with excessive beer intake and poor solute intake, there is a low solute load presented to the kidneys and thus free water excretion is impaired leading to the dilutional hyponatraemia.

It is also important to note that in such patients the excessive amount of free water retained in the circulation will suppress

the antidiuretic hormone (ADH).⁶ Once these patients are administered solutes, the kidneys start to excrete large amounts of free water again as the low ADH levels limit free water uptake. This can be dangerous as the serum sodium concentration might increase rapidly over a short period of time predisposing to osmotic demyelination.⁵

In view of the latter, patients who are chronic alcoholics, undernourished or suffer from advanced liver disease are at a greater risk of osmotic demyelination. It is for this reason that such patients are best managed in an intensive therapy/high dependency unit where serial measurement of serum electrolytes occurs.

The treatment of beer potomania should be managed according to the severity of symptoms. If the patient is asymptomatic, fluid restriction and close monitoring of the clinical and biochemical parameters is safe. However, if the patient is suffering from severe symptoms, treatment with hypertonic saline is indicated.⁷ In all cases, it is advised that the serum sodium level should not increase by >10 mmol/L in a 24-hour period.

The poor dietary intake puts these patients at risk of refeeding syndrome once parenteral/enteral feeding is commenced and hence phosphate levels should also be closely monitored.

The electrophysiological effects of sodium are rarely clinically significant.² However, the co-occurrence of hyponatraemia and reversible ST segment elevations,⁸ as well as reversible first, second and third-degree heart blocks, has been documented in a few published case reports.^{3 9 10} In this case, the concurrent presentation of profound hyponatraemia and the AV block suggests that the hyponatraemia may have precipitated the AV block.

The pathogenesis of hyponatraemia-induced cardiac conduction defects is poorly understood.⁹ Sodium affects phase 0 (depolarisation) of the cardiac non-nodal cell action potential. The low extracellular sodium concentration shortens phase 0 of the cardiac action potential,² and reduces the amplitude of the action potential. Reduction of the amplitude of this action potential may potentially lead to a prolonged PR interval.⁹

This highlights the importance of cardiac monitoring in those patients with profound hyponatraemia.

Figure 3 Progression of serum sodium during the first 72 hours.

A distinctive feature in this case is the persistent AV block despite correction of the serum sodium concentration. Consequently, our patient required a permanent pacemaker.

Learning points

- ▶ Beer potomania is a syndrome of dilutional hyponatraemia that occurs in patients with a history of excessive beer consumption and poor dietary intake.
- ▶ Sodium should be corrected slowly (ie, less than 10 mmol/L increase in a 24-hour period) to reduce the risk of osmotic demyelination especially in undernourished patients and those with alcoholism and advanced liver disease.
- ▶ Patients with profound hyponatraemia should be observed via cardiac monitoring to assess for any cardiac conduction defects. Moreover, profound hyponatraemia is optimally managed in an intensive therapy/high dependency unit setting where sodium levels can be monitored closely and hypertonic saline can be administered.

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