

Case Report

Electrocardiogram manifestations of hyponatraemia

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Abstract

Electrolytes play a vital role in myocardial electrophysiological activities in the human body. Electrolyte disturbances can affect depolarisation and repolarisation of myocardial cells and thus result in arrhythmia. The most common electrolyte disturbance among hospitalised patients is hyponatraemia. We report on a case of an acquired immune deficiency syndrome patient with decompensated cirrhosis, who developed sinus arrest from hyponatraemia. The electrocardiogram manifestations at different sodium concentrations were also recorded in subsequent therapeutic processes.

Keywords: hyponatraemia, ECG, cirrhosis, sinus arrest

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Hyponatraemia is generally defined as a serum sodium concentration lower than 135 mmol/l, which is associated with increased rates of mortality and morbidity.^{1,2} The clinical presentation may be highly variable, ranging from no symptoms to neurological symptoms such as confusion and coma, or even death.³

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Although serum sodium concentration is known to be associated with arrhythmia,^{4,5} as far as we know, no study on the various manifestations of arrhythmia caused by different degrees of hyponatraemia has been published. In this case report, we recorded the case of an acquired immune deficiency syndrome (AIDS) patient with decompensated cirrhosis who developed sinus arrest due to hyponatraemia. The changes on electrocardiogram (ECG) at different serum sodium concentrations in subsequent therapeutic process were also documented.

Case report

A 50-year-old woman was admitted to our hospital on 2 December 2011 with complaints of abdominal distention for two weeks. She had a history of AIDS and chronic hepatitis C for seven years, and had no history of heart disease or hyponatraemia. The patient had been receiving antiretroviral therapy (combination of lamivudine, tenofovir and lopinavir/ritonavir) for one year. She also received diuretic treatment (furosemide) for cirrhotic ascites.

On admission, the patient was alert and orientated, and co-operated with the medical staff. On physical examination, the patient had mild sclerotic jaundice of the skin and sclera, and palmar erythema with grade 2 cirrhotic ascites. Cardiac and other system examinations revealed no abnormalities.

Laboratory tests revealed a potassium concentration of 3.68 mmol/l (normal range: 3.5–5.5 mmol/l) and a sodium level of 137.4 mmol/l (normal: 132–146 mmol/l). Total bilirubin concentration was elevated to 83.4 µmol/l (normal: 5–21 µmol/l) and albumin level was reduced to 28.7 g/l (normal: 40–55 g/l). Blood urea nitrogen concentration was 8.97 mmol/l (normal: 2.8–7.6 mmol/l), creatinine level was 156.3 µmol/l (normal: 64–104 µmol/l) and the estimated glomerular filtration rate (eGFR) was 38 ml/min. Myocardial enzymes were normal.

Abdominal ultrasound showed liver cirrhosis, portal hypertension, ascites and cholecystolithiasis. The ECG showed normal sinus rhythm on admission. After admission, diuretics (furosemide, spiro lactone and hydrochlorothiazide) were started.

On 14 December 2011, the patient showed symptoms including chest tightness, palpitation, headache, fatigue and drowsiness, and an ECG showed sinus arrest (the longest R-R interval was 3.8 seconds) and junctional escape beats (Fig. 1A). Electrolyte tests revealed severe hyponatraemia (102 mmol/l).

The hyponatraemia was gradually corrected by intravenous 3% hypertonic saline.⁶ The patient's symptoms were improved and the ECG showed various manifestations at different serum sodium concentrations (Fig. 1, Table 1). In the next few days, a 24-hour Holter recording showed sinus rhythm.

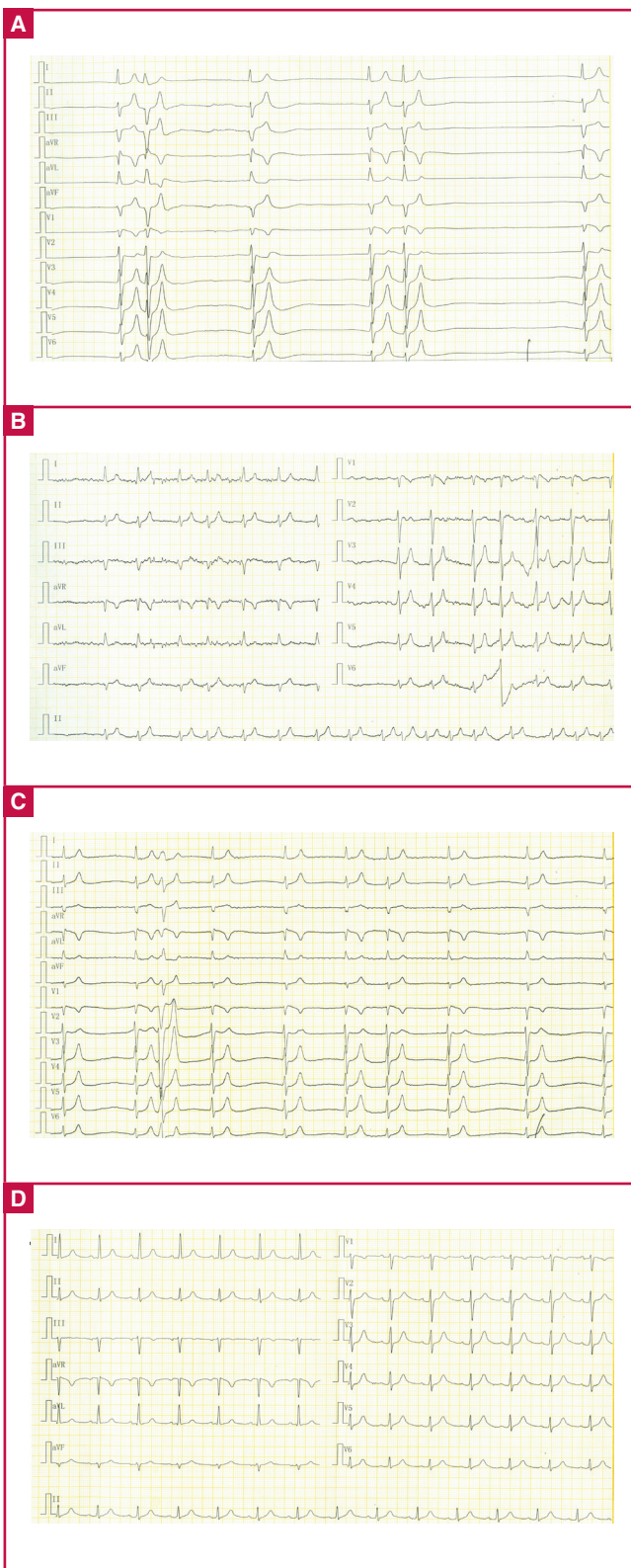


Fig. 1. Manifestations of the patient's ECG. (A) Sinus arrest (the longest R-R interval was 3.8 s) and junctional escape beat with a sodium level of 102 mmol/l. (B) Atrial fibrillation (AF) when the sodium level reached 114.8 mmol/l. (C) AF with second-degree AV block and ventricular premature beat when the sodium level reached 118.9 mmol/l. (D) Normal sinus rhythm when the sodium level reached 132 mmol/l.

Table 1. Changes in electrolyte levels and ECG manifestations

Time	Serum sodium (mmol/l)	Serum potassium (mmol/l)	Serum calcium (mmol/l)	ECG
9 December	131	5.6	1.69	Normal sinus rhythm
14 December, 11:20	102 (↓)	5.14	1.71	Sinus arrest; junctional escape beat (Fig. 1A)
14 December, 16:40	114.8 (↓)	6.01 (↑)	1.75	Atrial fibrillation (Fig. 1B)
15 December, 10:38	118.9 (↓)	6.14 (↑)	1.72	Atrial fibrillation with second-degree AV block; ventricular premature beat (Fig. 1C)
15 December, 18:00	132	5.5	1.72	Normal sinus rhythm (Fig. 1D)
15 December, 23:00	127.9 (↓)	5.14	1.75	Normal sinus rhythm

Discussion

To our knowledge, sinus arrest associated with hyponatraemia has not been reported in the literature. The pathogenesis of hyponatraemia-induced sinus arrest or cardiac conduction defects is yet to be elucidated, and data in the literature are scarce.

According to the clinical observation, low extracellular sodium concentration may shorten the depolarisation of the cardiac action potential and reduce the amplitude of the action potential in the atrioventricular (AV) node.^{7,8} It has also been shown that extremely low levels of sodium in the fluid perfusing isolated heart muscle can reduce the number of contractions as well as the excitability and conduction velocity.⁹

Remarkably, our patient developed various cardiac arrhythmias at different serum sodium concentrations. Clinical studies have suggested that hyponatraemia may cause atrial fibrillation (AF) episodes.^{10,11} Hyponatraemia due to water retention increases atrial wall stretch, which can increase vulnerability to atrial arrhythmia.¹² A study on rabbit hearts demonstrated that hyponatraemia induced genesis of pulmonary vein burst firing, which may contribute to the high occurrence of AF.¹³ AF might also be due to a combined effect of electrolyte disturbance or induced by bradycardia during sinus arrest. However, AF induced by transient bradycardia is rare and its underlying mechanisms remain unclear.¹⁴

The patient was found to have peaked T waves on ECG during severe hyponatraemia (Fig. 1A). T-wave shape reverted to normal after the hyponatraemia was corrected. Although T-wave shape changes in hyperkalaemia are well known, peaked T waves are rare in hyponatraemia. It is recommended that ECG manifestations of peaked T waves should be clinically contextualised and not attributed only to hyperkalaemia.

In the case presented here, the patient was admitted to hospital on foot and had no previous history of heart disease. Treatment of the patient with sinus arrest, AV block and AF was to stop diuresis and supply sodium only. As the patient's sodium levels returned to normal, the clinical symptoms and ECG also improved and no further arrhythmia was identified at follow ups, which made us attribute the temporary changes in the ECG to low sodium levels.

Our patient experienced hyperkalaemia during the treatment of hyponatraemia. After treatment with hypertonic saline on the first day, her potassium level reached 6.01 mmol/l. Three possible mechanisms could explain this phenomenon. Firstly,

when the patient received a high dosage of sodium after severe hyponatraemia, there was a reduction in aldosterone secretion, which would tend to decrease the rate of potassium secretion and may consequently reduce urinary excretion of potassium.¹⁵

Secondly, the patient's glomerular filtration rate was low, reducing distal tubular flow rates. The potassium concentration may have built up relatively early in the tubule and thus progressively decreased the electrochemical gradient principal cells and the secretion of potassium.¹⁶ Thirdly, considering that the period of discontinuation of spironolactone was too short and the patient's renal function was also abnormal, there may also have been remaining spironolactone, which contributed to the hyperkalaemia.

Conclusion

Serious hyponatraemia can induce sinus arrest. Various arrhythmias shown on ECG may result from different serum sodium concentrations. It is important to raise awareness to give special attention to the ECG and electrolytes in the therapeutic process for a patient with hyponatraemia.

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