OP8 LOW SALT SLOWS THE BEAT

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INTRODUCTION

Hyponatraemia, the most common electrolyte abnormality, is usually asymptomatic. If clinical manifestations occur, usually related to central nervous system dysfunction. Hyponatraemia presenting as a cardiac conduction defect is very rare.

CASE DESCRIPTIONS

A 55-year-old female with underlying hypertension presented with a week history of failure symptoms, palpitation and poor oral intake. Upon arrival, she was alert and not in respiratory distress. Her vital signs were bradycardia with HR 47, BP 106/80 mmHg, RR 20, SpO_2 100% under room air, GCS full and capillary blood glucose 8.8 mmol/L. Physical examination was unremarkable. Point of care ultrasound showed good cardiac contractility and no regional wall motion abnormality. Her regular antihypertensives treatments are beta blocker, calcium channel blocker, ACE inhibitor and thiazide.

Initial 12-leads electrocardiogram (ECG) showed junctional bradycardia HR 50 with atrial ectopics. 4 hours later, she became hypotensive but remained alert. Intravenous atropine 1 mg was given and started on infusion adrenaline subsequently. The laboratory investigations revealed sodium 98 mmol/L, potassium 4.3 mmol/L, magnesium 0.7 mmol/L and troponin I <0.01ng/mL. Hyponatraemia was corrected by rapid hypertonic saline in the emergency department followed by maintenance of 0.9% saline. On the next day, ECG showed normal sinus rhythm with repeated sodium 106 mmol/L. In the ward, the maintenance was changed to dextrose 5% in view of rapid increment in sodium concentration. She was discharged well on day 6 with normal sinus rhythm ECG and sodium 130 mmol/L. The final diagnosis was severe hyponatraemia likely secondary to thiazide use with resolved junctional bradycardia.

DISCUSSION

80% of bradyarrhythmias are caused by factors external to the cardiac electrical system. The common causes include cardiac ischaemia, drugs and electrolyte disturbances (hyperkalaemia and hypermagnesaemia). Hyponatraemia as a sole cause of cardiac conduction defect is very rare. There are a few articles describing patients with atrioventricular blocks due to hyponatraemia but their relation is far from established.

CONCLUSION

The differential diagnoses for bradyarrhythmia are broad thus any patient with cardiac block should be carefully investigated for reversible causes. Temporizing measures such as vasoactive drugs and pacing should be considered in symptomatic patients.

KEYWORDS

Hyponatraemia, bradyarrhythmia