CASE REPORT

A Rare Presentation of a Common Disorder : Severe Hyponatremia Presenting as Reversible Unstable Bradyarrythmias

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ABSTRACT

Severe hyponatraemia is defined as a sodium level of less than 120 mEq/L, and it is frequently accompanied by neurological symptoms like coma, convulsions, respiratory arrest, and death. Clinical cardiac toxicity from hyponatremia, such as bradyarrhythmia, is extremely rare. In this article, we present a case of acute severe hyponatraemia that induced unstable bradyarrhythmia and led to refractory bradycardia, which did not improve despite receiving treatment in accordance with the standard Advanced Cardiovascular Life Support protocol. The patient's bradyarrhythmia has completely resolved with the administration of 3% hypertonic saline, which restored her sodium levels. Due to the possibility that severe hyponatremia may contribute to the aetiology of cardiac malfunction, this case raises awareness about the significance of closely monitoring electrocardiograms and telemetry in patients with severe hyponatremia.
Malaysian Journal of Medicine and Health Sciences (2024) 20(1):395-397. doi:10.47836/mjmhs.20.1.51

Keywords: Hyponatremia, low sodium, bradycardia, bradyarrythmia, heart block

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INTRODUCTION

Hyponatremia is the most common electrolyte abnormality in patients seen in the emergency department (ED). When clinical signs of hyponatremia do appear, they are typically caused by problems with the central nervous system. The signs and symptoms were varied and broad, and half of the patients experienced neurological symptoms such as decreased consciousness level, seizures, or muscle weakness. Some patients can present with other neurological symptoms that can resemble an ischemic stroke, such as aphasia (1).

However, cases of clinical cardiac toxicity linked to severe hyponatremia are uncommon, and it is typically challenging to identify hyponatremia as the root cause of conduction abnormalities. In this report, we described an elderly patient without pre-existing heart disease who experienced unstable bradyarrhythmia that was accompanied by severe hyponatremia. It is crucial to spread awareness about the importance of paying close attention to ECGs and telemetry in patients with abnormally low blood sodium, as hyponatremia might play a role in the pathogenesis of heart dysfunction in this type of patient.

CASE REPORT

A 73-year-old lady with multiple comorbidities of hypertension, dyslipidemia, and chronic kidney disease (CKD) stage 4 presented with generalised body weakness associated with poor oral intake and diarrhoea episodes 2 to 3 times a day for the last 1 week. She denies a history of fever, vomiting, upper respiratory symptoms, or urinary symptoms. The patient was on multiple medications such as perindopril 8 mg OD, amlodipine 10mg OD, metoprolol 50mg BD, moxonidine 0.4mg OD, simvastatin 40mg ON, frusemide 40 mg OD, and cardiprin 100mg OD. Upon arrival, she was triaged to the red zone as she was noted to be confused, hypotensive, and bradycardic. Clinically, she was pink with a Glascow Coma Scale (GCS) of 14 (disoriented),

and the peripheral was cold with prolonged capillary perfusion. There was mild to moderate dehydration, but otherwise the patient is pink with no jaundice. Her blood pressure was 85/60 mmHg, heart rate was 40 beats per minute (bpm), the oxygen saturation was 99% under room air, and she had a normal temperature. Other systemic examinations were normal.

Her bedside electrocardiogram (ECG) reveals atrial fibrillation with bradycardia, and no heart block or ischaemia changes were noted (Figure 1). As per the Advanced Cardiovascular Life Support (ACLS) protocol, she was treated for unstable bradycardia and was initially given intravenous (IV) atropine 1 mg up to 3 times. Subsequently, she was given an intravenous infusion (IVI) of adrenaline for chemical pacing, as there was no improvement with atropine. The patient initially responded to the treatment, but it was not sustained. A peripheral blood examination showed a normal full blood count, but the serum electrolytes revealed a sodium level of less than 100 mmol/L. The other electrolytes were within the normal range; however, she has an acute kidney injury as her urea and creatinine were raised from baseline 11.3/250 to 17/290 respectively on presentation. Her C-reactive protein (CRP) was also not raised and other sepsis parameters like a chest X-ray, stool, and urine examination were normal. The initial troponin I was normal and the echocardiography of the patient reveals no regional wall abnormalities with an ejection fraction of more than 60%.

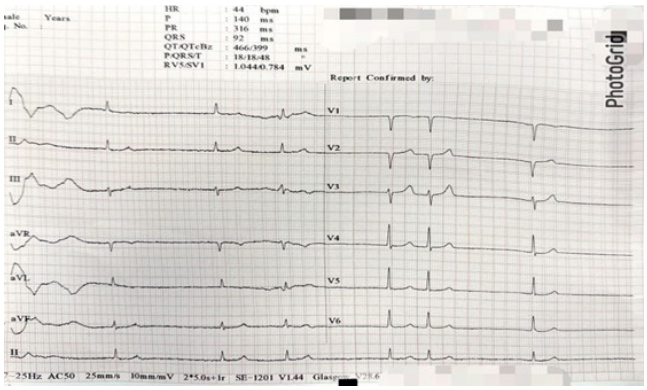

Figure 1: Initial Electrocardiogram on admission – Atrial fibrillation with Bradycardia with a heart rate of 35 to 40 beats per minute

She was started on hypertonic saline 3% solution (2 ml/kg) per hour IVI for rapid correction in view of symptomatic severe hyponatremia. Within a few hours in the General Intensive Care Unit (GICU), the patient has regained full consciousness, and her hemodynamic status has become stable, with her heart rate improving to 70 to 80 bpm (Figure 2). The repeat ECG a few days later reveals a reverted AF to sinus rhythm (Figure 3). The patient was transferred out of the GICU in a few days and discharged home a week later in a stable and conscious condition.

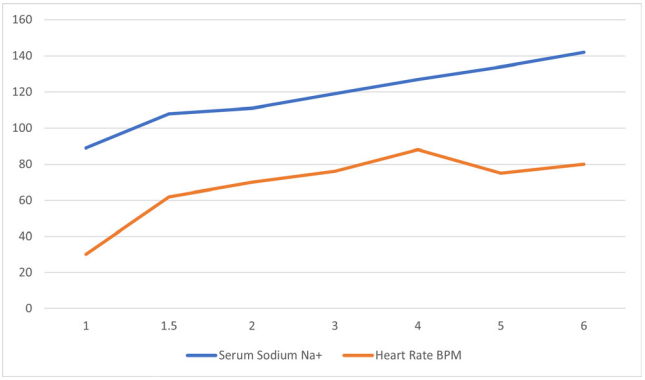

Figure 2 Course of Sodium level and changes in heart rate during initial presentation and during correction of sodium

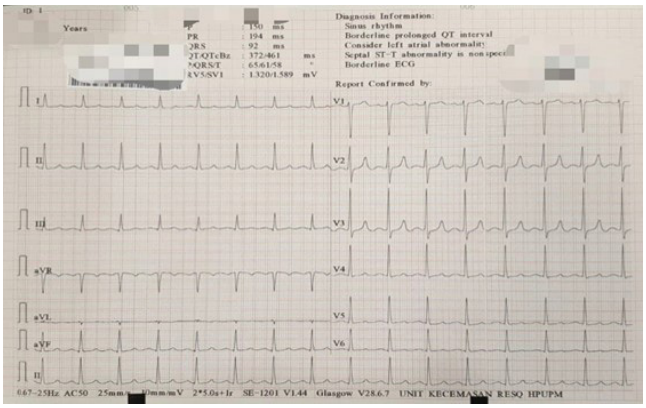

Figure 3: Electrocardiogram of patient in GICU (post Sodium correction) – Sinus rhythm with a Heart rate of 80 Beats per Minute. The Atrial Fibrillation has been reverted.

DISCUSSION

It is uncommon for hyponatremia patients to present with cardiac electrophysiological manifestations of bradyarrythmias, although neural electrophysiological alterations usually cause central nervous system manifestations such as altered sensorium or seizures. In the literature, there isn't much research that deals with hyponatremia causing heart abnormalities. Conduction problems, which are predominantly linked to hyponatremia, may be accompanied by several clinical circumstances such as an underlying cardiac illness, the use of anti-arrhythmic medications or other drugs and electrolyte abnormalities. The low sodium levels affect the AV node's action potential in several ways. Some patients develop a first-degree AV block while others have a second- or third-degree AV nodal block (2). In our patient, the hyponatremia was due to multiple causes as she has underlying CKD stage 4, a history of gastrointestinal loss with inadequate oral intake causing the patient to be in a hypovolaemic state during the presentation and the use of diuretics.

There are various explanations for how hyponatremia affects the heart. Theoretically, a decrease in sodium concentration extracellularly should result in a slowing of cardiac pacemaker activity. According to some

research, reducing extracellular sodium concentrations can reduce inward sodium flow during the initial stages of action potential formation (2). Furthermore, when the fluid perfusing an isolated cardiac muscle contained unusually low salt levels, the muscle's excitability and conduction velocity as well as the number of contractions were all reduced. Physiologically, the cardiac cell's action potential has five phases from 0 to 4. Phase 0 also known as the depolarization phase, is when the sodium channels in the membrane open. Because of the lower extracellular sodium concentration in hyponatraemia, sodium channels close early, shortening phase 0 and reducing the action potential's amplitude. Initially, this amplitude reduction prolongs the PR interval, which could lead to an AV conduction delay. However, a full AV block will manifest on the ECG when hyponatraemia gets worse and persists (3).

Severe hyponatremia patients who present with acute symptoms need urgent attention. The current recommendation is an immediate correction of 4-6 mmol/L for acute symptomatic hyponatraemia. If necessary, a 100-ml bolus of 3% saline may be given over a 10-minute period to treat severe symptoms. This procedure may be done up to three times. An infusion of hypertonic 3% saline can be administered at a rate of 0.5 to 2 ml/kg/hour for mild to moderate symptoms (4). The patient in this instance had an unstable bradycardia with a heart rate of 30 to 40 bpm. She denies having atrial fibrillation (AF) before; however, the ECG during the initial presentation was a very low heart rate of 30 to 40 bpm with atrial fibrillation. As she was confused and hypotensive, the maximal amount of IV atropine and IVI adrenalin were administered in accordance with the ACLS protocol. However, the patient only partially responded to the medication since the heart rate could not be maintained beyond 60 bpm and she was persistently hypotensive. As the sodium result was very low (Na <100 mmol/L), she was started on IVI hypertonic saline 3% with a weight-based approach of 1-2 ml/kg per hour. Some authors suggest increasing the rate to 4 to 5 mL/kg of body weight per hour for 1 to 2 hours for severe symptoms like coma and seizures. As Asians tend to have smaller frames than Americans or Europeans, weight-based approaches are preferred over fixed 100- to 150-mL infusion volumes of hypertonic saline 3%, and based on a recent guideline, it recommends discontinuing hypertonic saline infusion in patients with improved symptoms after a 5-mmol/L increase in sodium concentrations (5). After several hours of hypertonic saline correction, she has regained consciousness, and her heart rate has improved to 70 to 80 bpm.

CONCLUSION

The medical literature hardly ever mentions severe hyponatremia as a reversible cause of cardiac arrhythmia. A quick adjustment of sodium helped this patient's episode of unstable bradyarrhythmia, which was resistant to traditional ACLS protocols. This instance emphasises the need for close cardiac monitoring in individuals with low serum sodium levels and the significance of considering this electrolyte as one of the causes of bradyarrhythmia that doesn't respond to conventional therapy.

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