A rare case of cardiac arrest caused by hyponatremia

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SOUHRN

Hyponatremie je stanovena jako koncentrace sodíku nižší než 135 mmol/l. Jedná se o celosvětově nejčastější poruchu elektrolytů. Nejčastějšími projevy hyponatremie jsou gastrointestinální a neurologické obtíže. Příležitostně může vést hyponatremie k poruchám srdečního rytmu. V námi prezentované kazuistice vedla těžká hyponatremie k sinusové bradykardii s alternujícím stupněm atrioventrikulární blokády a srdeční zástavě. Obnovení sinusového rytmu bylo dosaženo až po úpravě koncentrace sodíku. Těžká hyponatremie a protrahovaná zástava oběhu však u pacienta vedly k malignímu otoku mozku. Tento jedinečný případ zdůrazňuje kritickou roli sodíku v srdeční elektrofyziologii a ukazuje důležitost monitorace koncentrace sodíku u pacientů se zástavou oběhu.

Keywords:
Beer potomania
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ABSTRACT

Hyponatremia, characterized by sodium levels below 135 mmol/l, is the most prevalent electrolyte disorder worldwide. It presents with a wide range of clinical symptoms, particularly in the neurological and gastrointestinal domains, occasionally leading to cardiac arrhythmias. In our specific case, severe hyponatremia resulting from potomania resulted in sinus bradycardia with alternating atrioventricular block and subsequent cardiac arrest. Restoration of sinus rhythm was achieved following correction of the sodium levels. However severe hyponatremia and long-lasting CPR resulted in brain oedema, which ultimately led to brain death. Per national regulations, the patient was enrolled in an organ donor program, resulting in successful organ transplants. This unique case underscores the critical role of sodium levels in cardiac electrophysiology and highlights the necessity of monitoring electrolyte levels in patients experiencing cardiac arrest.

Introduction

Hyponatremia represents the most prevalent electrolyte disturbance on a global scale. It is defined as a sodium level falling below 135 mmol/l.[1,2] Patients with hyponatremia can show various symptoms related to the nervous system and the gastrointestinal tract.[2,3] Neurological manifestations can range from somnolence to seizures, coma, and, in extreme cases, fatality. Additionally, hyponatremia has rarely been correlated with cardiac arrhythmias.[2,4] Arrhythmias linked with hyponatremia, as documented in the literature, include atrioventricular block and sinus bradycardia.[3,5] The specific cardiac arrhythmia associated with hyponatremia may exhibit variations dependent on the severity of the electrolyte imbalance.[2] This report details a unique case of acute hyponatremia, leading to sinus bradycardia and subsequent cardiac arrest.

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Case report

A 60-year-old male was admitted to our medical facility in January 2024 following a cardiac arrest, successfully addressed through cardio-pulmonary resuscitation (CPR). In the patient's medical history, notable factors include excessive beer consumption and chronic obstructive pulmonary disease (COPD). Preceding the incident, the patient was seated, engaging in leisure activities with his family when he suddenly lost consciousness and collapsed from his chair. Emergency services were promptly engaged, and Dispatcher Assisted CPR was initiated and continued by the emergency medical service team. The initial rhythm was asystole. The airway was secured with an orotracheal tube, and nearly 15 minutes into CPR, spontaneous circulation was successfully restored. The subsequent electrocardiogram (ECG) finding revealed atrial fibrillation, ST elevation in aVR and V_1 lead, and a new left anterior fascicular block, as depicted in **Figure 1A**.

Subsequently, the patient's heart rate declined from 143 beats per minute to 60 beats per minute, leading to another cardiac arrest with an entry rhythm of pulseless electrical activity. Timely CPR was initiated, resulting in the restoration of spontaneous circulation after 10 minutes. The observed rhythm was atrial flutter with a 2 : 1 atrioventricular block and left atrioventricular block, detailed in **Figure 1B**. Shortly thereafter, another cardiac arrest occurred, necessitating immediate CPR. Spontaneous circulation was restored in 4 minutes, with the rhythm identified as atrial flutter with a 2 : 1 atrioventricular block and left atrioventricular block with a new right bundle branch block, accompanied by ST elevations, as illustrated in **Figure 1C**.

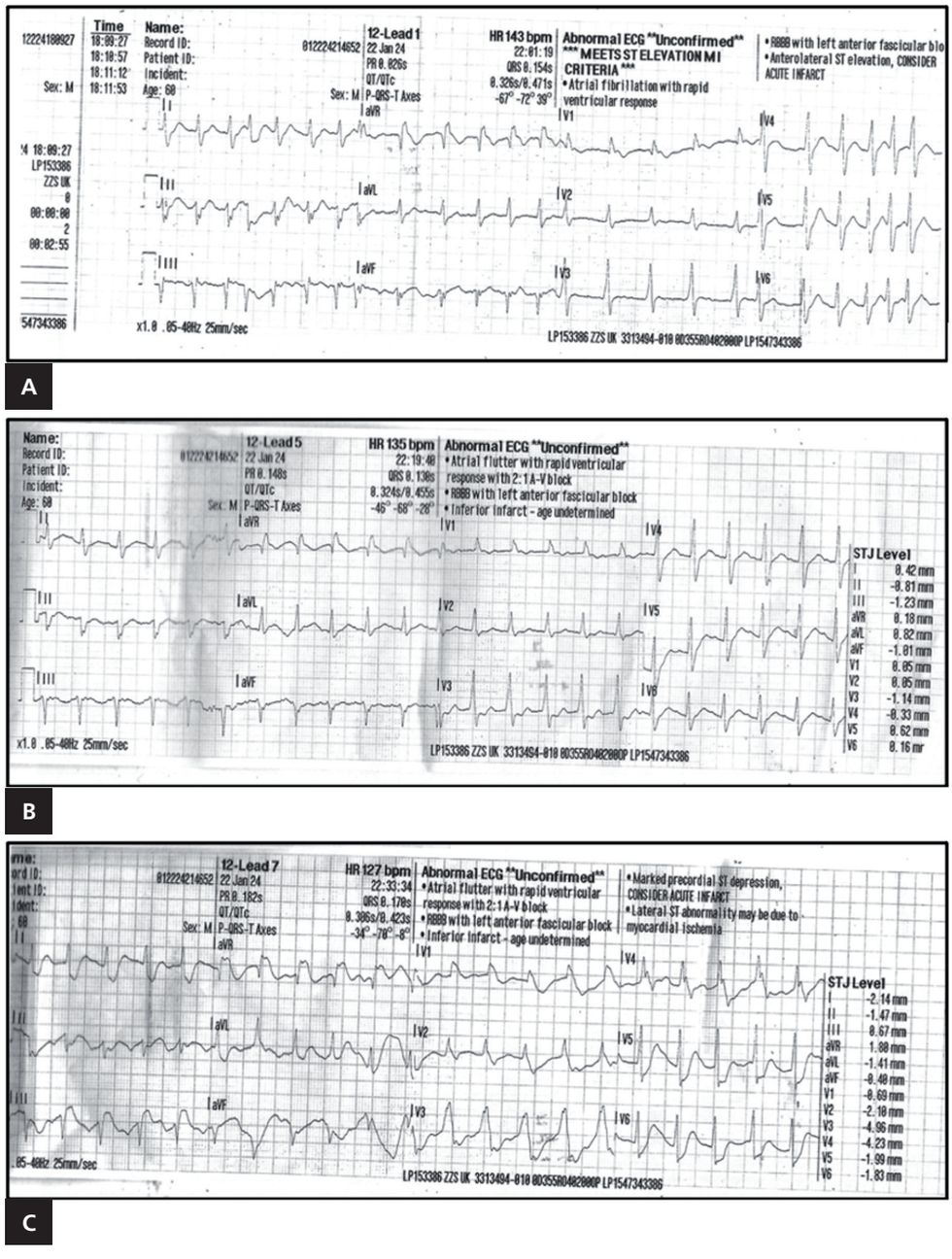

Fig. 1 – ECG (A–C) obtained by emergency service.

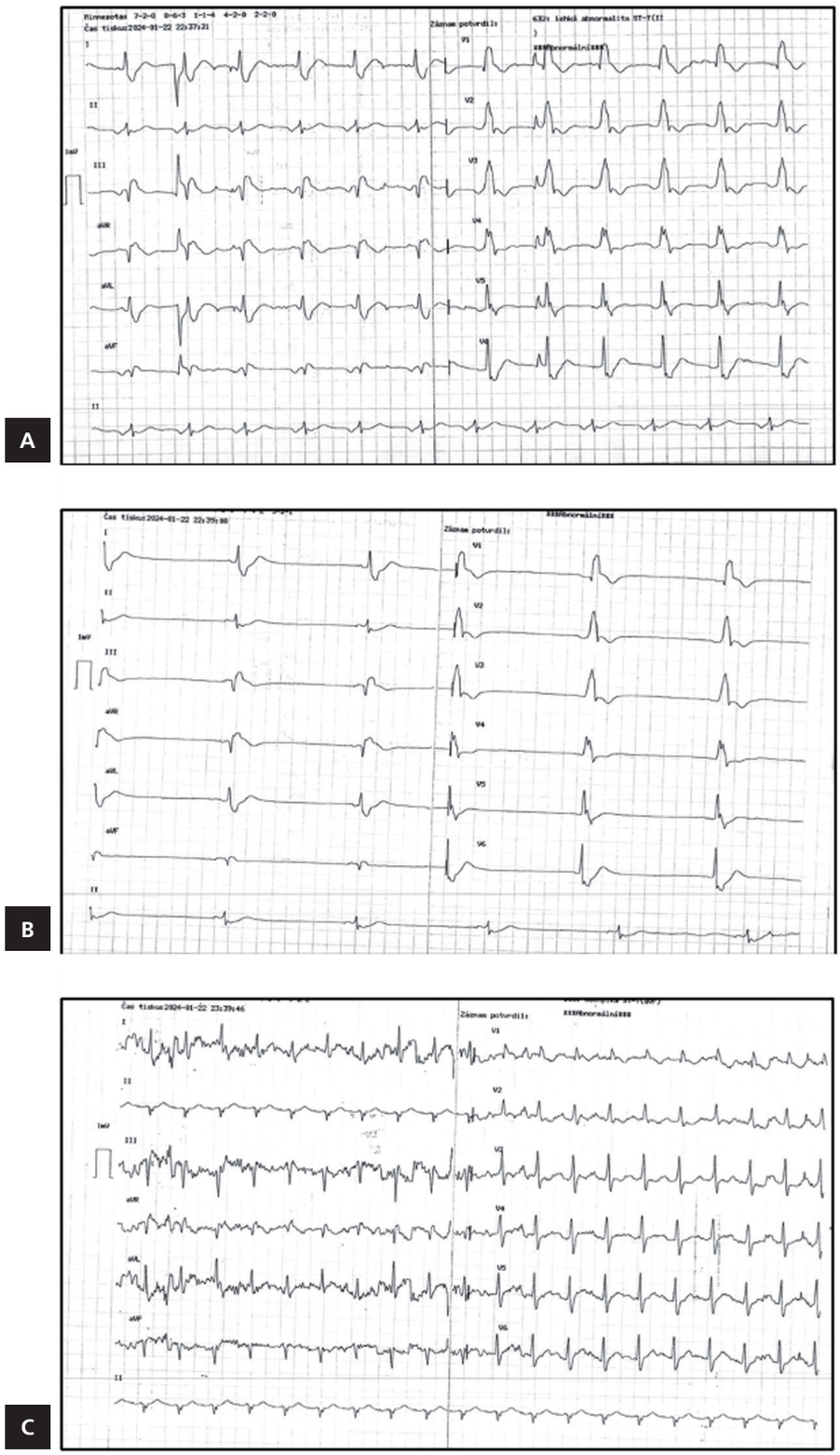

Fig. 2 – ECG (A–C) obtained at the Emergency Department.

Following these events, the patient was transferred to the Emergency Department, presenting with a sinus rhythm left anterior fascicular block, and right bundle branch block (**Fig. 2A**). Shortly after admission, another cardiac arrest ensued with a pulseless electrical activity as an entry rhythm (**Fig. 2B**). CPR was promptly initiated, and after 2 minutes, spontaneous circulation was restored. Due to a continuous decline in heart rate, dobutamine and noradrenaline infusions were initiated. A transthoracic echocardiogram revealed right ventricle dilatation without hypertrophy. A CT scan revealed brain oedema solely. There were no signs of pulmonary embolism, and subsequent

coronary angiography showed no evidence of coronary artery disease. Troponin levels were as low as 110 ng/l, with a peak value of 327 ng/l the following day. Laboratory tests on admission revealed acidosis, low osmolality, and severe hyponatremia falling to 108 mmol/l. A bolus of sodium chloride 5.85% was promptly administered. Post-administration, the QRS complex narrowed, and sinus rhythm with left anterior fascicular block was successfully restored, as depicted in **Figure 2A**. Dobutamine infusion was quickly discontinued. There were no other episodes of bradycardia or PEA during the hospital episode.

Upon admission to an ICU, natremia was 115 mmol/l. Hyponatremia correction was initiated, resulting in correction to 120 mmol/l within two hours and reaching 138 mmol/l within another two days. Following the hyponatremia correction sinus rhythm without any block was maintained. Two days after admission, diabetes insipidus manifested, and a follow-up brain CT scan revealed a significant diffuse brain oedema. The first neuron-specific enolase test recorded 237 µg/l (within 24 hours of admission), while subsequent tests were surpassing 370 µg/l at 48 and 72 hours from admission. Sedation was discontinued, and clinical signs of brain death emerged. Subsequently, in compliance with national regulations, angiography of brain vessels was conducted, confirming the diagnosis of brain death. The patient was enrolled in an organ donation program, with several organs successfully transplanted to other recipients. According to national law the autopsy was done after organ transplant. The autopsy revealed solely mild hypertrophy of right ventricle.

Discussion

Hyponatremia, although recognized as the most prevalent electrolyte disorder, remains relatively underexplored in its association with life-threatening arrhythmias.[1,2] Existing literature on this subject is sparse, with seminal contributions such as the work by Jeong, who first highlighted thiazide diuretic-induced hyponatremia as a causative factor in complete atrioventricular block.[6] Subsequent reviews by Liamis expounded on the severe consequences of thiazide diuretic-induced hyponatremia, particularly in elderly patients, with a focus on its impact on arrhythmias.[7] Notably, a recent case report by Zou in 2022 detailed severe arrhythmias attributed to hyponatremia, where the patient was also under diuretic therapy.[2]

In these documented cases, withdrawal of diuretics led to the normalization of sodium levels and the restoration of sinus rhythm, underscoring the causal relationship between hyponatremia and arrhythmias. Of particular interest in our presented case is the absence of thiazide diuretic use, no prior medical history of heart failure and importance of active correction of hyponatremia.

In our assessment, the most probable cause of hyponatremia was beer potomania. Our case aligns with the other reports where normalization of sodium levels resulted in the restoration of sinus rhythm, and subsequent episodes of arrhythmias were not observed.

Several theoretical frameworks attempt to elucidate the pathophysiological mechanisms underlying hyponatremia-induced arrhythmias. Empirical Trautwein's investigation demonstrated that markedly low sodium levels in the perfusing fluid of isolated heart muscle led to a reduction in contractions and excitability.[7] Additionally, studies by Nikolaidou on rabbit hearts highlighted the impact of low sodium levels on action potentials in the atrioventricular (AV) node.[8] Correspondingly, Yen Yu's physiological study in cell culture revealed that diminished sodium levels lowered the amplitude of action potentials.[9] Clinical observations by El-Sherif further supported these findings, emphasizing that hyponatremia, while infrequently electrophysiologically significant compared to other electrolyte disorders, did contribute to a shortened depolarization time and decreased action potential amplitude in the AV node.[10]

The comprehensive exploration of these physiological and clinical aspects provides valuable insights into the intricate interplay between hyponatremia and arrhythmias, shedding light on potential mechanisms and reinforcing the importance of considering this electrolyte imbalance in the context of cardiac electrophysiology.

Severe hyponatremia, prolonged CPR, and asphyxia are widely recognized as risk factors for malignant brain oedema.[11,12] It can be assumed that there was a period of inadequate ventilation prior to the intervention of the emergency medical service team. Consequently, all the aforementioned risk factors were evident in this particular case, contributing to the development of malignant brain oedema.

Despite hyponatremia presents most often as gastrointestinal and mild neurological symptoms, in our case, severe hyponatremia resulted in sinus arrest followed by cardiac arrest, without any prior neurological or gastrointestinal symptoms.

Conclusion

This case emphasizes the importance of recognizing hyponatremia as a reversible cause of cardiac arrest. It's crucial to carefully monitor and evaluate patients with hyponatremia and related changes in their electrocardiograms. Early detection and intervention can prevent negative cardiac outcomes.

Conflict of interest
None declared.

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None.

Ethical statement
The work was conducted in accordance with the Declaration of Helsinky and good clinical practice.

Informed consent
The informed consent was obtained from the patient or his family to use the data.

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