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Ventricular Bigeminy Presenting as Syncope and Seizure in the Setting of Hyponatremia: A Diagnostic Dilemma

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ABSTRACT

Syncope and altered sensorium in elderly patients often arise from overlapping cardiac, metabolic, and neurological etiologies, making accurate diagnosis challenging. A 71-year-old male presented with giddiness, excessive daytime sleepiness, and new-onset Generalized Tonic-Clonic Seizure (GTCS). Evaluation revealed symptomatic sinus bradycardia with ventricular bigeminy, significant hyponatremia, and type 1 respiratory failure in the background of chronic obstructive pulmonary disease. Continuous cardiac monitoring demonstrated intermittent severe bradycardia, while laboratory investigations confirmed electrolyte imbalance and elevated cardiac biomarkers. The coexistence of these conditions created a diagnostic dilemma, as each could independently explain the clinical presentation. Hyponatremia and hypoxia likely lowered the seizure threshold, while severe bradycardia may have contributed to convulsive syncope due to cerebral hypoperfusion. Additionally, seizure-related autonomic disturbances may have exacerbated cardiac arrhythmias, highlighting a complex bidirectional interaction.

Keywords: Syncope; Hyponatremia; Ventricular bigeminy; Bradycardia; Seizure

INTRODUCTION

Syncope is defined as a transient loss of consciousness due to global cerebral hypoperfusion, characterized by rapid onset, short duration, and spontaneous recovery [1]. Differentiating syncope from seizure activity is particularly challenging, as both may present with overlapping clinical features, including involuntary movements and post-event confusion. Hyponatremia is a well-recognized metabolic cause of altered sensorium

and seizures, particularly in the elderly, where impaired physiological reserve exacerbates neurological vulnerability [2]. Additionally, hypoxia and metabolic disturbances may lower the seizure threshold, further confounding clinical interpretation.

The clinical evaluation of syncope and altered sensorium in elderly patients represents a significant diagnostic challenge, particularly when multiple pathophysiological processes coexist. Cardiovascular, metabolic, and neurological etiologies frequently overlap, complicating the identification of a unifying diagnosis [3,4]. Cardiac arrhythmias such as ventricular bigeminy characterized by alternating sinus beats and premature ventricular contractions may reduce effective cardiac output, especially in the presence of bradycardia, leading to cerebral hypoperfusion and transient loss of consciousness [4]. Such hemodynamic instability may manifest as convulsive syncope, often mimicking primary seizure disorders.

In this context, we present the case of a 71-year-old male with giddiness, excessive daytime sleepiness, and new-onset generalized tonic-clonic seizures, wherein coexisting symptomatic bradycardia with ventricular bigeminy, hyponatremia, and respiratory compromise created a complex diagnostic dilemma. This case highlights the importance of a comprehensive, multidisciplinary approach in evaluating transient loss of consciousness in geriatric patients with multimorbidity.

CASE DESCRIPTION

A 71-year-old male presented with complaints of giddiness and excessive daytime sleepiness for 4–5 days. Further history from his wife revealed a recent episode of new-onset Generalized Tonic-Clonic Seizure (GTCS) associated with transient loss of consciousness. There was no prior history of seizures. He was a known hypertensive on regular treatment and had previously been diagnosed with narcolepsy, for which he was receiving modafinil. He also had Chronic Obstructive Pulmonary Disease (COPD) on dual inhaler therapy and was an ex-smoker who had quit 20 years earlier.

On admission, his blood pressure was 102/60 mmHg, pulse rate 48 beats per minute, and oxygen saturation 99% on supplemental oxygen at 4 L/min. General physical examination was unremarkable, with no pallor, cyanosis, clubbing, lymphadenopathy, or pedal edema. Systemic examination revealed no focal neurological deficits.

Arterial blood gas analysis demonstrated type 1 respiratory failure (pH 7.393, PaO_2 54.9 mmHg, $PaCO_2$ 41.6 mmHg, HCO_3^- 24.8 mmol/L). Electrocardiography revealed sinus bradycardia with heart rates below 40 beats per minute, along with ventricular bigeminy and conduction abnormalities (Figure 1). Continuous cardiac monitoring confirmed intermittent episodes of severe bradycardia. Two-dimensional echocardiography showed trivial aortic regurgitation, mild mitral regurgitation, left ventricular diastolic dysfunction, and preserved ejection fraction (60%). Cardiac biomarkers were elevated (Troponin-I 6.05, CK-MB 68 U/L, CPK-MB 7.4 ng/mL). Laboratory evaluation revealed significant hyponatremia. An incidental simple right renal cyst was also noted.

The patient was managed with continuous cardiac monitoring and supportive care, including oxygen therapy. Drug-induced bradycardia was considered but excluded due to the absence of an identifiable offending agent. The coexistence of sinus bradycardia with ventricular bigeminy, hyponatremia, and new-onset GTCS created a diagnostic dilemma, as each condition independently could account for syncope and altered sensorium. Post-ictal drowsiness, arrhythmia-induced cerebral hypoperfusion, and metabolic encephalopathy were all plausible

contributors. A working diagnosis of symptomatic sinus bradycardia with ventricular bigeminy in the setting of metabolic and possible neurological contributors was established, warranting multidisciplinary evaluation.

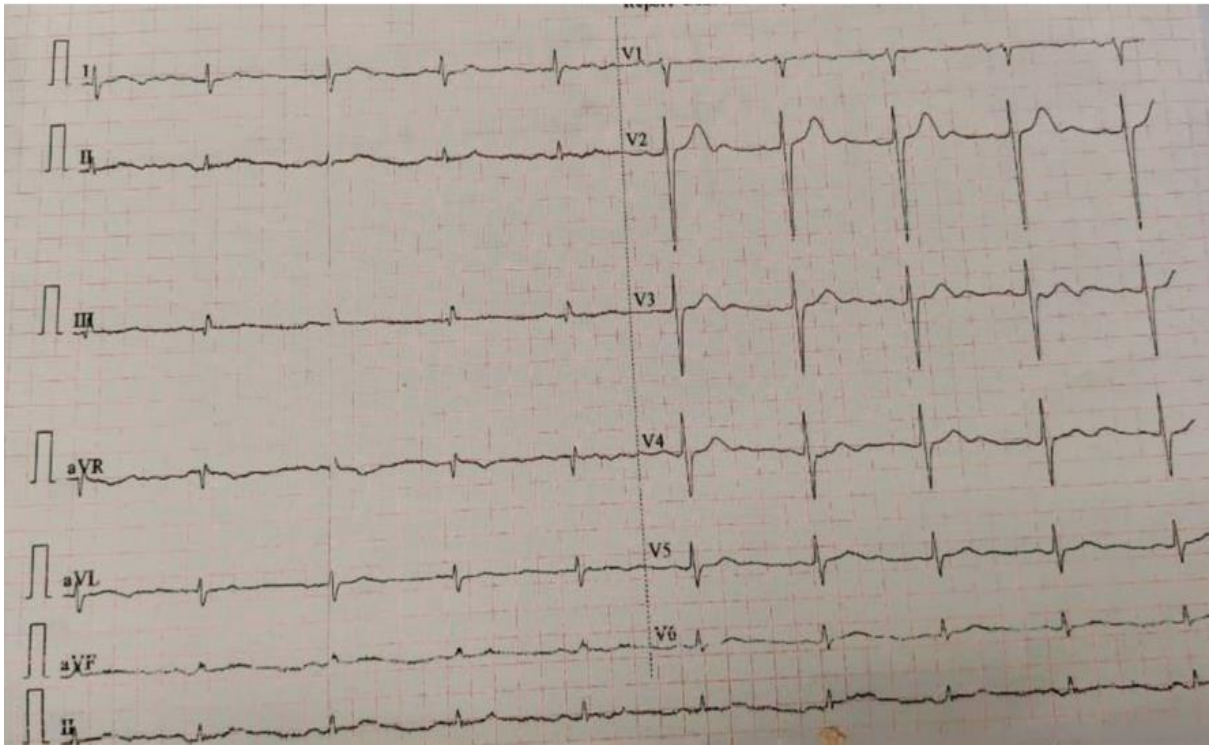

Figure 1: Twelve-lead electrocardiogram demonstrating diffuse tall, peaked T waves, most prominent in the precordial leads (V2–V6), with associated attenuation of P waves. These findings are characteristic of hyperkalemia. In the present case of partial hanging, hyperkalemia may be attributed to metabolic acidosis and rhabdomyolysis.

DISCUSSION

This case illustrates a complex interplay between respiratory, metabolic, and cardiovascular pathologies, each contributing to altered sensorium and transient loss of consciousness. Determining the primary etiology in such scenarios is essential yet inherently challenging. Hyponatremia is a well-established cause of seizures due to cerebral edema and neuronal dysfunction [4]. In this patient, chronic hypoxemia secondary to COPD may have contributed to inappropriate antidiuretic hormone secretion, resulting in dilutional hyponatremia [5]. Hypoxia further impairs cerebral adaptation to osmotic stress, thereby lowering the seizure threshold [6,7]. Thus, hyponatremia in conjunction with hypoxia likely played a significant role in precipitating the observed GTCS.

The presence of marked sinus bradycardia and ventricular bigeminy introduces an additional diagnostic dimension. Severe bradycardia can result in convulsive syncope, where transient cerebral hypoperfusion leads to loss of consciousness with seizure-like motor activity [8]. Conversely, seizures themselves can induce cardiac rhythm disturbances via autonomic dysregulation, including bradyarrhythmia and conduction abnormalities [8-12]. This bidirectional relationship complicates causal inference. Elevated cardiac biomarkers in this context likely reflect demand ischemia or transient myocardial injury secondary to systemic hypoxia and seizure activity, rather than primary coronary pathology [12]. Furthermore, hyponatremia has been reported as a

reversible cause of bradyarrhythmia, suggesting a possible metabolic contribution to the observed cardiac abnormalities [13].

CONCLUSION

This case highlights the diagnostic complexity arising from the interplay of cardiac, metabolic, and respiratory factors in elderly patients presenting with syncope and seizures. A comprehensive, multidisciplinary evaluation is essential to identify underlying mechanisms and prevent misdiagnosis in such overlapping clinical scenarios.

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