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A patient with acute quadriparesis in the emergency department: Unmasking the diagnosis through electrocardiography

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Abstract:

Electrolyte abnormalities may masquerade as acute neurological or cardiac emergencies in the emergency department (ED). In such atypical presentations, a timely bedside electrocardiography (ECG) often provides the earliest and most critical clue, facilitating an accurate diagnosis, guiding appropriate management, and improving patient outcomes. A 32-year-old male presented with rapidly evolving flaccid quadriparesis without sensory or autonomic involvement. A bedside ECG provided a critical clue, revealing a Mobitz type I block with ischemia-mimicking ST–T changes and prominent U waves. This prompted urgent testing, which confirmed profound hypokalemia and hypomagnesemia. Swift intravenous repletion reversed both paralysis and conduction abnormalities within 72 h. Although electrolyte abnormalities are known to produce typical ECG changes, their presentation with conduction disturbances such as Wenckebach block is rare and easily misinterpreted as ischemia or structural heart disease. Prompt recognition of these atypical ECG findings at the bedside is critical to avoid unnecessary invasive workups and to initiate lifesaving electrolyte correction.

Keywords:

Electrocardiography, hypokalemia, quadriparesis

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Introduction

Acute flaccid quadriparesis requires careful consideration of various causes, including autoimmune, toxic, metabolic, and infectious disorders. Although autoimmune and demyelinating etiologies often dominate the differential diagnosis, severe metabolic disturbances, particularly hypokalemia and hypomagnesemia, remain under recognized yet fully reversible culprits. These electrolyte imbalances can also produce dramatic electrocardiographic changes that may closely mimic acute coronary syndromes (ACS). Although hypokalemia-related atrioventricular conduction disturbances, including Mobitz type I block, have been described in isolated case reports, these reports predominantly focus on rhythm abnormalities in nonemergency settings. In contrast, ischemia-mimicking ST–T changes accompanied by Wenckebach conduction, particularly in association with acute neuromuscular weakness and concomitant hypomagnesemia, represent a diagnostic challenge that remains critically underreported in the literature. This distinction is clinically important, as such electrocardiogram (ECG) patterns

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may closely simulate ACS and lead to inappropriate management if the metabolic etiology is not promptly recognized. We report a case of severe electrolyte imbalance presenting as quadriparesis with a Wenckebach phenomenon on ECG, which resolved entirely following electrolyte correction.

Case Report

A 32-year-old male presented to the emergency department (ED) with acute-onset bilateral lower-limb weakness, which progressed over several hours to involve the upper limbs and trunk, resulting in flaccid quadriparesis at presentation. The neurological symptoms were preceded by intermittent nonprojectile vomiting of undigested food for 5 days, followed by multiple episodes of diarrhea (approximately 5/day) over the preceding 3 days. He denied sensory symptoms, cranial nerve involvement, bowel or bladder incontinence, respiratory difficulty, chest pain, or palpitations. There was no history of toxin ingestion, alcohol excess, drug use, or similar prior episodes, and he had no known comorbidities.

On arrival (time 0), the patient appeared emaciated and clinically dehydrated. He was alert and oriented, with a pulse rate of 88/min, blood pressure of 110/70 mmHg, respiratory rate of 20/min, and oxygen saturation of 98% on room air. He was afebrile and hemodynamically stable. Neurological examination revealed symmetrical flaccid quadriparesis, predominantly affecting proximal muscle groups, with markedly diminished deep tendon reflexes. Sensory examination, cranial nerve evaluation, and autonomic assessment were normal.

A 12-lead ECG obtained within 15 min of ED presentation demonstrated second-degree atrioventricular block with Wenckebach (Mobitz type I) phenomenon, PR interval prolongation, apparent QT prolongation, diffuse ST-segment depression in leads II, III, augmented vector foot, and V3–V6, ST-segment elevation in augmented vector right, low-amplitude T waves, and prominent U waves fused with T waves forming TU complexes [Figure 1]. In view of these ischemia-mimicking changes, ACS was considered; however, the absence of chest pain, normal cardiac biomarkers, and unremarkable bedside echocardiography made this unlikely. Neurological differentials, including Guillain–Barré syndrome and central causes, were considered but deemed less likely due to the absence of sensory, cranial nerve, or autonomic involvement.

Within 60 min of ECG acquisition, urgent laboratory evaluation revealed profound hypokalemia (1.03 mEq/L) (normal range 3.5–5.1 mEq/L) and hypomagnesemia (1.4 mg/dL) (normal range 1.9–2.5 mg/dl). Thyroid function tests were normal, excluding thyrotoxic periodic paralysis, and imaging revealed no renal or adrenal pathology. Immediate electrolyte correction was initiated with continuous cardiac monitoring. Intravenous potassium chloride was started at 20 mEq/h and titrated to 40 mEq/h based on serial serum potassium measurements, along with oral potassium supplementation (40 mEq every 4 h). Magnesium deficiency was corrected with an initial 2 g intravenous magnesium sulfate bolus, followed by additional supplementation as required.

Over the subsequent 72 h, the patient demonstrated progressive neurological improvement, with complete

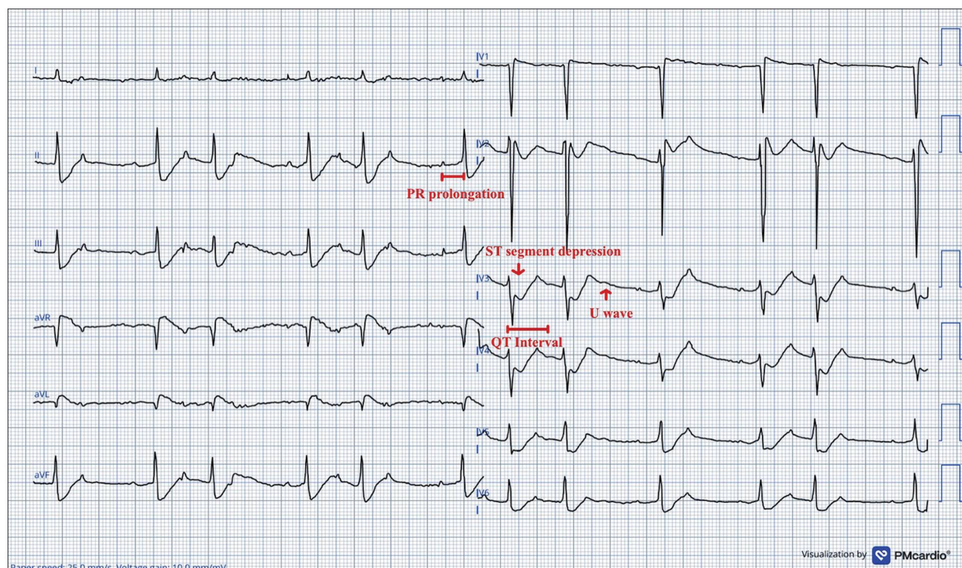

Figure 1: Electrocardiogram on arrival demonstrating second-degree heart block with Wenckebach phenomenon with diffuse ST-segment depression, ST elevation in augmented vector right, PR prolongation, apparent QT prolongation, and prominent U waves forming TU complexes

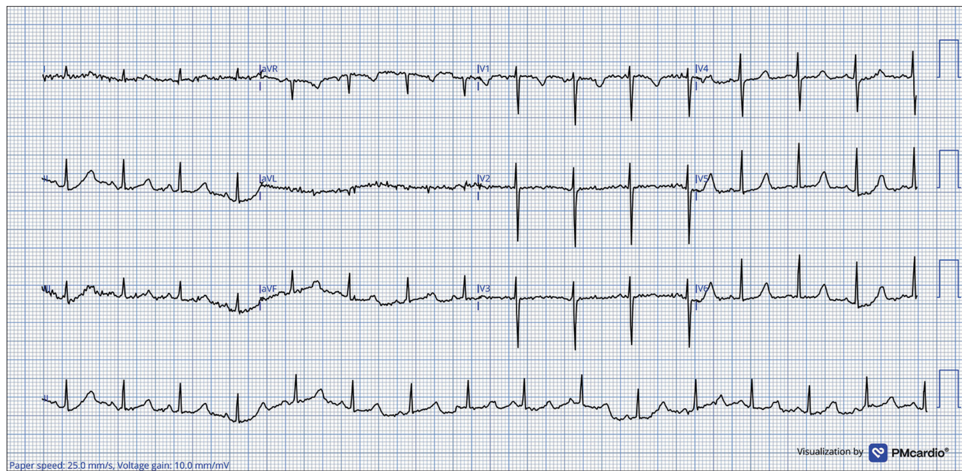

Figure 2: Electrocardiogram after electrolyte correction demonstrating normal sinus rhythm

recovery of motor strength and reflexes. Repeat ECG showed resolution of atrioventricular block and normalization of ST–T changes, and serum electrolytes improved to potassium 3.12 mEq/L and magnesium 1.6 mg/dL [Figure 2]. The complete and parallel reversal of neuromuscular and electrocardiographic abnormalities confirmed severe electrolyte imbalance as the primary etiology. Written informed consent was obtained from the patient for the publication of this case report.

Discussion

Acute flaccid quadriparesis presenting to the ED often necessitates rapid differentiation between broad etiologies, including neurological, neuromuscular, metabolic, and electrolyte-related disorders. Severe electrolyte imbalance is a reversible but frequently overlooked cause, in which the ECG plays a decisive role, serving as an early, noninvasive indicator that often precedes laboratory confirmation. Severe hypokalemia predisposes patients to neuromuscular paralysis and malignant cardiac arrhythmias. In the general population, hypokalemia (serum K^+ <3.5 mEq/L) has a prevalence of approximately 1.9%, rising to 5.5% in the ED settings. Severe hypokalemia (≤2.5 mEq/L), though relatively uncommon, with a reported ED prevalence of 0.4%, carries significant clinical risk.[1,2]

Approximately 40% of hypokalemic patients presenting to the ED exhibit at least one ECG abnormality attributable to potassium imbalance.[3] The earliest manifestation is often a reduction in T wave amplitude, but the severity and pattern of ECG findings are more reflective of the rate of potassium shift from the patient's baseline than of the absolute serum level.[4,5] The hypokalemic ECG index, which sums the ST-segment depressions and U wave amplitudes in leads II and V3, has been proposed to estimate serum potassium levels.[6,7] While these changes evolve with progressive potassium decline, there are no universally accepted serum thresholds for specific ECG abnormalities.

In the ED, diffuse electrocardiographic abnormalities should prompt consideration of a systemic or metabolic process rather than regional myocardial ischemia, which typically follows a defined coronary territory. QTc prolongation, diffuse ST-segment depression, and prominent U waves resulting from potassium deficiency appear atypical when multiple electrolyte disturbances coexist, mimicking ACS. Recognition of these diagnostic pitfalls is essential to prevent unnecessary activation of the invasive ACS pathways.

Careful consideration of the global distribution of changes, absence of reciprocal ST elevation, and presence of U waves can help distinguish metabolic derangements from true ischemia. Early recognition is critical, as severe hypokalemia, particularly when accompanied by hypomagnesemia, is associated with a high risk of malignant arrhythmias and conduction disturbances. Therefore, prompt correction of electrolytes is an emergency priority, as timely potassium and magnesium replacement can rapidly reverse ECG abnormalities, prevent unnecessary ACS-directed interventions, and significantly reduce morbidity and mortality.

In our patient, the presence of Mobitz type I (Wenckebach) atrioventricular block further complicated the ECG interpretation. Although hypokalemia is known to cause characteristic repolarization abnormalities, clinically significant atrioventricular conduction disturbances are rarely reported and may be mistaken for intrinsic conduction system disease or ischemia. A focused review of the literature reveals only isolated case reports describing Wenckebach block associated with hypokalemia, most of which were identified outside the ED or lacked ischemia-mimicking ECG changes or concomitant hypomagnesemia.[8,9]

While hypokalemia often takes clinical precedence, hypomagnesemia is an equally critical contributor to both neuromuscular weakness and cardiac instability. Its deficiency exacerbates potassium loss, prolongs repolarization, and increases the risk of Torsades de pointes and ventricular arrhythmias, especially when coexisting with hypokalemia. A U-shaped association has been demonstrated between serum potassium levels and all-cause mortality, both in the general population and in patients with comorbidities such as heart failure, chronic kidney disease, and diabetes mellitus, with more severe hypokalemia conferring progressively higher mortality risk.[10]

In this case, early bedside ECG interpretation in the ED redirected the diagnostic approach toward a metabolic etiology, enabling prompt electrolyte correction and preventing unnecessary activation of ACS or stroke pathways, with complete neurological and electrocardiographic recovery within 72 h.

Conclusion

This case highlights the importance of early electrocardiographic evaluation in the ED, where the ECG may serve as the first and most accessible clue to a severe electrolyte imbalance. Acute metabolic disturbances, including hypokalemia, should always be considered in the differential diagnosis of patients presenting with sudden-onset neurological deficits that may mimic acute stroke. However, clinicians must remain vigilant, as distinguishing these metabolic mimics from true ACS or stroke can still present diagnostic challenges, even with early ECG evaluation. Concomitant hypomagnesemia can amplify electrocardiographic abnormalities, delay recovery, and increase the risk of malignant arrhythmias, highlighting the need for prompt recognition and simultaneous correction of both abnormalities to ensure rapid and complete clinical resolution.

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Author contribution statement

S. Anukarthika: Writing – original draft, writing-review and editing, visualization, data curation, conceptualization. Mari Selva Ganesh: Writing – review and editing, visualization, project administration, data curation. V. T. Amrithanand: Writing – review and editing, supervision, conceptualization.

Conflicts of interest

None Declared.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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