Case Report

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Hyperkalemic Periodic Paralysis and Prompt Recovery in an Elderly Patient with Comorbidities: A Case from Addis Ababa, Ethiopia

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ABSTRACT

Hyperkalemic periodic paralysis (HPP) is a rare disorder typically of genetic origin, and its occurrence as a secondary condition in the elderly is exceptional. We report the case of an 80-year-old Ethiopian man with severe pulmonary hypertension and a suspected myeloproliferative disorder who presented with acute lower limb paralysis. Initial investigations revealed an extreme serum potassium level of 9.08 mmol/L with corresponding electrocardiographic changes, including widened QRS complexes and peaked T waves. Emergent treatment with intravenous calcium gluconate, insulin-dextrose, and fluids rapidly corrected the hyperkalemia, leading to the complete reversal of paralysis and cardiac abnormalities. This case is notable for the patient's advanced age and the severity of the hyperkalemia, highlighting the multi-factorial pathogenesis of secondary HPP where renal impairment, polypharmacy, and underlying hematologic disease may interact. It further demonstrates that prompt adherence to fundamental electrolyte management protocols is critical in preventing fatal outcomes in complex geriatric patients.

Keywords: Hyperkalemic periodic paralysis, Electrolyte imbalance, Muscle weakness, geriatric, secondary hyperkalemia, Ethiopia

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1. Introduction

Hyperkalemic periodic paralysis (HPP) is a rare neuromuscular disorder characterized by recurrent episodes of skeletal muscle weakness or paralysis, precipitated by elevated serum potassium levels. Classically linked to autosomal dominant mutations in the SCN4A gene, which encodes voltage-gated sodium channels in skeletal muscle, HPP disrupts membrane excitability, leading to transient depolarization and impaired muscle contraction. [(1)] While hereditary forms typically manifest in childhood or early adulthood, secondary or acquired hyperkalemic paralysis triggered by renal dysfunction, metabolic derangements, or pharmacological agents may present in older people, often complicating diagnosis and management.[(2)]

In geriatric populations, HPP poses unique challenges due to the convergence of age-related physiological decline, poly-pharmacy, and comorbidities, such as chronic kidney disease (CKD), cardiovascular disorders, and endocrine abnormalities. These factors amplify susceptibility to hyperkalemia and obscure the recognition of neuromuscular manifestations, delaying critical interventions.[(3)] Furthermore, life-threatening hyperkalemia (serum $K^+ > 7.0$ mmol/L) is rare in HPP, as most genetic variants induce milder potassium fluctuations. However, in individuals with superimposed acute kidney injury (AKI), the use of potassium-wasting diuretics, or myeloproliferative disorders, conditions that perturb potassium homeostasis, severe hyperkalemia may develop, escalating risks of fatal cardiac arrhythmias or respiratory muscle paralysis.

This case report details an 80-year-old male with acute flaccid paralysis and extreme hyperkalemia (9.08 mmol/L), a presentation exceptional in both magnitude and clinical complexity. The patient's comorbidities, including a suspected myeloproliferative disorder (MPD), severe pulmonary hypertension (PHTN), and AKI, highlight the multi-factorial etiology of hyperkalemia in HPP-like presentations. Specifically, diuretic therapy for PHTN, potential tumor lysis from MPD, and renal hypoperfusion likely synergized to precipitate this crisis. The case underscores the diagnostic pitfalls in distinguishing primary HPP from secondary paralysis in elderly patients, where overlapping pathologies demand a nuanced approach. Additionally, it demonstrates the critical role of rapid, protocol-driven management, even in resource-constrained settings, in reversing metabolic and neuromuscular threats.

This case is clinically significant for three reasons. First, it demonstrates that extreme, non-familial HPP can manifest in the geriatric population, expanding the typical demographic profile of the disease. Second, the severity of hyperkalemia (9.08 mmol/L) approaches the upper limits of survivable potassium levels, offering a rare window into the acute management of profound electrolyte-induced toxicity. Third, the patient's comorbidities, including a suspected myeloproliferative disorder, renal dysfunction, and poly-pharmacy, illustrate the multi-factorial nature of secondary HPP, where multiple acquired defects in potassium excretion and distribution converge. This report underscores the importance of prioritizing physiological principles over age-related diagnostic assumptions when managing acute paralysis with electrolyte derangement.

2. Method

Study Design: This is a single, descriptive case report.

Study Area and Setting: The patient was evaluated and managed at Care Land General Hospital, a private institution in Addis Ababa, Ethiopia, that

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serves a growing and significant number of communities in the region.

Study Period: The patient presented to the emergency department on December 22, 2023, and was immediately admitted to the Intensive Care Unit (ICU), where all acute management and stabilization were performed.

Clinical Protocol and Materials: The patient was managed according to the hospital's emergency protocol for severe hyperkalemia with cardiac toxicity.

3. Case report

An 80-year-old male presented to the hospital with a three-day history of acute-onset bilateral lower extremity weakness, urinary incontinence, epigastric pain, and nausea. He reported easy fatigability and a burning sensation in his legs for the past year but denied recent trauma, potassium-rich dietary intake, vomiting, diarrhea, or cardio-respiratory symptoms. His medical history was significant for severe pulmonary hypertension (PHTN) with grade III left ventricular diastolic dysfunction and severe tricuspid regurgitation on recent echocardiography, as well as an ongoing investigation for suspected myeloproliferative disorder (MPD) pending bone marrow biopsy results. He also reported a fall from a horse three years prior without any sequelae. Chronic medications included spironolactone (25 mg daily), furosemide (40 mg daily), gabapentin (300 mg daily), and allopurinol (100 mg daily).

On admission, vital signs were stable (blood pressure: 131/60 mmHg; pulse: 50–60 bpm; respiratory rate: 20/min; SpO_2: 95% on room air). Physical examination revealed that the patient appeared acutely ill but was alert and oriented. Neurological assessment demonstrated bilateral lower extremity motor power of 2/5 and upper extremity power of 3/5, with preserved cranial nerve function and no focal deficits. Cardiovascular, respiratory, and abdominal examinations were unremarkable.

Initial laboratory investigations revealed life-threatening hyperkalemia (9.08 mmol/L), hyponatremia (129.4 mmol/L), acute kidney injury (creatinine: 1.45 mg/dL; urea: 154 mg/dL), moderate normocytic anemia (hemoglobin: 7.2 g/dL), leukocytosis (WBC: 46.57 × 10^3/µL with neutrophilia), and thrombocytosis (platelets: 563 × 10^3/µL). Electrocardiography showed widened QRS complexes and peaked T waves (Fig. 1), consistent with hyperkalemia-induced cardiotoxicity. Serial electrolytes demonstrated a gradual decline in potassium (from 9.08 to 5.76 mmol/L over four days) and resolution of AKI (creatinine: 0.83 mg/dL by day 5). During the hospitalization, hemoglobin levels fluctuated (range: 7.2–9.4 g/dL), and leukocytosis improved (WBC: 19.72 × 10^3/µL).

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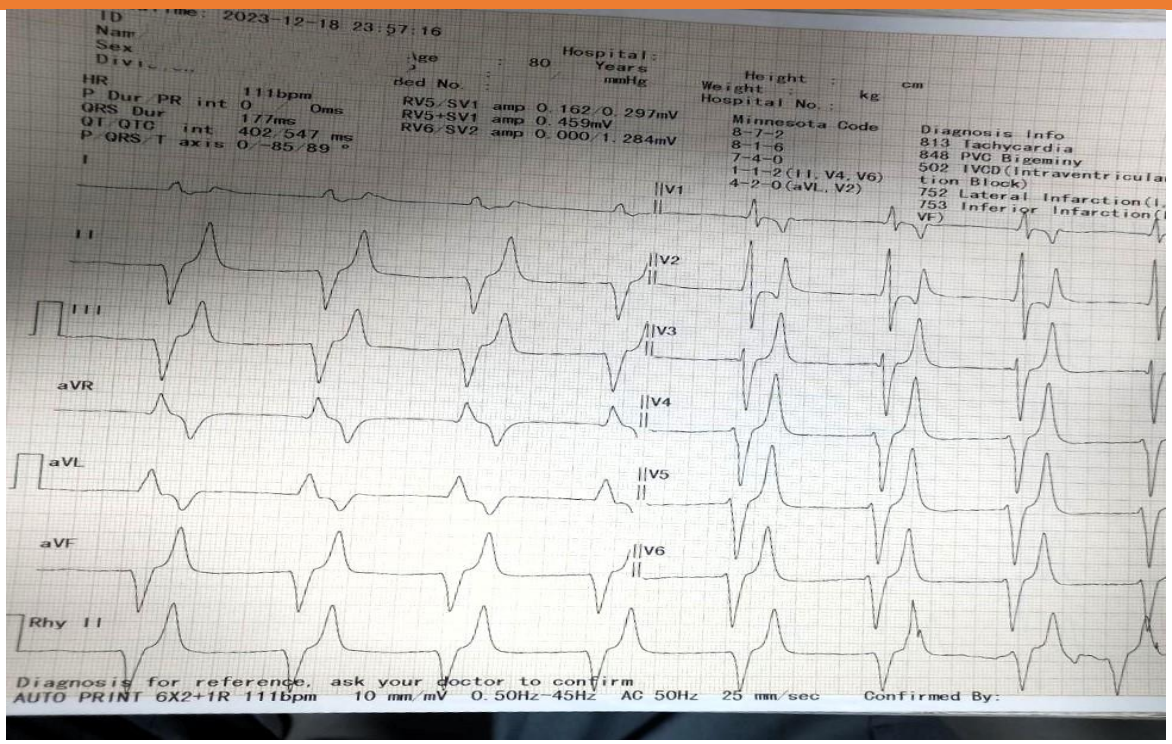

Fig 1: Initial ECG at admission showing wide QRS and peaked T waves, courtesy of Besufekad Taye. MD, ECCM Addis Ababa University

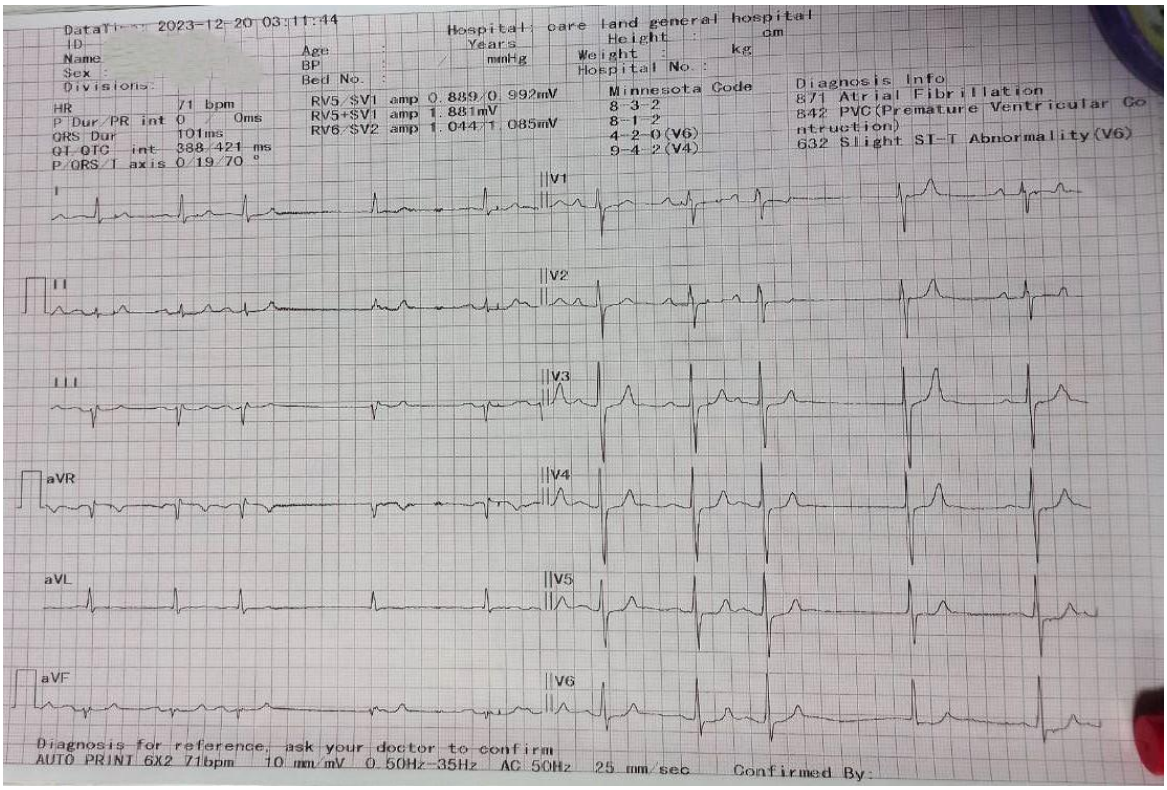

Fig 2: Last ECG of the patient after stabilization showing corrected QRS and T waves, courtesy of Besufekad Taye. MD, ECCM Addis Ababa University

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Treatment and outcome

The patient was admitted to the intensive care unit (ICU) with working diagnoses that included severe hyperkalemia (secondary to tumor lysis syndrome vs. MPD vs. acute-on-chronic kidney injury), hyperkalemic periodic paralysis, severe PHTN, and moderate anemia of undetermined etiology. He was managed emergently with intravenous calcium gluconate (2 g), insulin-dextrose (40 g dextrose + 10 IU regular insulin), nebulized salbutamol (10 puffs), and furosemide (60 mg IV bid). Normal saline and sodium bicarbonate were administered for volume expansion and correction of acidosis. A hematology consultation was sought for suspected tumor lysis syndrome (TLS) or MPD-related complications.

4. Discussion

This case of an 80-year-old male with acute hyperkalemic paralysis and life-threatening hyperkalemia (9.08 mmol/L) underscores the complex interplay of aging, comorbidities, and polypharmacy in precipitating electrolyte emergencies. While hyperkalemic periodic paralysis (HPP) is classically linked to SCN4A gene mutations causing skeletal muscle sodium channel dysfunction, secondary triggers renal impairment, myeloproliferative disorders (MPD), and potassium-altering medications, likely dominated in this geriatric patient. The rapid resolution of paralysis and hyperkalemia with standard therapies highlights the importance of distinguishing primary HPP from acquired hyperkalemic paralysis, particularly in older adults with multi-factorial risk factors.

Pathophysiology and Contributing Factors

The patient's severe hyperkalemia likely arose from a confluence of factors:

A. Renal Dysfunction: Acute kidney injury (creatinine: 1.45 mg/dL) impaired potassium excretion, exacerbated by diuretic therapy (spironolactone and furosemide). [2] Spironolactone, a potassium-sparing agent, poses a significant risk of hyperkalemia in patients with renal insufficiency, particularly in the elderly with reduced glomerular filtration rates. [2]

B. Myeloproliferative Disorder (MPD): The suspected MPD raises the possibility of tumor lysis syndrome (TLS), though the absence of elevated uric acid or phosphate makes this less likely. Alternatively, marked leukocytosis (WBC: 46.57 × 10^3/μL) may reflect cellular potassium release or cytokine-driven membrane instability.

C. Pulmonary Hypertension (PHTN) Management: Diuretic therapy for PHTN likely contributed to volume depletion, worsening renal perfusion, and promoting potassium retention.

D. Aging Physiology: Age-related declines in renal function, muscle mass, and β-adrenergic responsiveness impair potassium regulation and exacerbate neuromuscular susceptibility to hyperkalemia.

Clinical and Therapeutic Considerations

The patient's presentation, flaccid paralysis, peaked T waves, and widened QRS complexes, aligns with hyperkalemia-induced depolarization block in skeletal and cardiac muscle. The absence of a family history or childhood-onset symptoms argues against hereditary HPP, favoring secondary paralysis from acute-on-chronic hyperkalemia. Prompt intervention with calcium gluconate (membrane stabilization), insulin-dextrose (intracellular potassium shift), salbutamol (β-agonist-mediated cellular uptake), and furosemide (enhanced excretion) successfully averted fatal arrhythmias or respiratory compromise. Notably, despite severe initial hyperkalemia, renal recovery (creatinine: 0.83 mg/dL by day 5) and potassium normalization were achieved without dialysis, underscoring the efficacy of conservative management in resource-constrained settings. [4]

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Differential Diagnoses and Diagnostic Challenges

Alternative causes of acute paralysis, such as Guillain-Barré syndrome or spinal cord pathology, were excluded by the temporal association with hyperkalemia, the absence of sensory deficits, and the rapid response to electrolyte correction. Similarly, rhabdomyolysis and adrenal insufficiency were deemed unlikely given normal creatine kinase levels and the clinical context. [2] This case emphasizes the need to prioritize reversible metabolic etiologies in elderly patients presenting with acute weakness, particularly those on nephrotoxic or potassium-affecting medications.

Implications for Geriatric Care

This case illustrates critical lessons for managing hyperkalemia in older adults:

Poly-pharmacy Vigilance: Potassium-sparing diuretics require rigorous monitoring in patients with renal impairment or hematologic disorders.

Multidisciplinary Approach: Collaboration among nephrology, hematology, and critical care teams is essential to address overlapping contributors (e.g., MPD, AKI, and diuretic effects).

Resourcefulness in Resource-Limited Settings: Basic interventions, calcium, insulin, and β-agonists, remain cornerstone therapies even when advanced options like dialysis are unavailable.

Limitations and Unanswered Questions

This case has several limitations. The pending bone marrow biopsy leaves the role of MPD-associated mechanisms speculative. Additionally, genetic testing for SCN4A mutations was unavailable, although the clinical picture strongly favored secondary triggers. Long-term management will hinge on optimizing diuretic regimens, definitively addressing the suspected MPD, and preventing recurrent AKI.

5. Conclusion

This case reinforces hyperkalemia as a reversible yet life-threatening cause of paralysis in elderly patients. It highlights the need for heightened suspicion of multi-factorial hyperkalemia in aging populations, particularly those with renal dysfunction, hematologic disorders, or high-risk medications. Timely, protocol-driven intervention can mitigate catastrophic outcomes, even in complex clinical scenarios.

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Competing interests

The authors declare that they have no competing interests regarding the publication of this case report. There are no financial, personal, or professional conflicts of interest that could potentially bias the reporting or interpretation of the findings. The authors have no affiliations or financial involvement with any organization or entity that has a direct or indirect interest in the subject matter discussed in this case report. This competing interest statement is provided in the interest of transparency and to ensure the integrity and impartiality of the research presented.

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