RESEARCH PAPER

Hypokalemic Paralysis: A Case Series Highlighting Diverse Etiologies

Dr Pooja Rajesh Naik[1], Dr Ganesh Pujar[2,*], Dr. Tejashree P[3]

[1]Assistant Professor, Department of General Medicine, KLE Jagadguru Gangadhar Mahaswamigalu Moorusavirmath medical college and Hospital, Hubli, KLE academy of Higher Education and Research, Deemed to be University, Belagavi, Karnataka, India 590010

[2]Senior Resident, Department of General Medicine, Karnataka Medical College and Research Institute (KMCRI), Vidya nagar, Hubballi, RGUHS University

[3]Consultant Physician, Department of General Medicine, United hospital, 30, Madhavan Park Circle, Bairasandra Extension, Jaya Nagar 1st Block, Jayanagar 3rd Block East, Jayanagar, Bengaluru, Karnataka 560011

***Corresponding author: Dr Ganesh Pujar, Senior Resident, Department of General Medicine, Karnataka Medical College and Research Institute (KMCRI), Vidya nagar, Hubballi, RGUHS University.**

Email: ganesh374147@gmail.com

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ABSTRACT

Background: Hypokalemic paralysis is a rare but potentially life-threatening neurological emergency characterized by acute flaccid muscle weakness associated with low serum potassium levels. It may be primary (periodic paralysis) or secondary to renal, metabolic, drug-induced, endocrine, or autoimmune causes. **Case Series:** We describe a series of six patients presenting with acute hypokalemic paralysis due to diverse etiologies. The first case involved tenofovir-induced proximal renal tubular acidosis (type 2 RTA). The second case was hypokalemic quadriparesis associated with uncontrolled type 2 diabetes mellitus and systemic stress. The third case represented hypokalemic periodic paralysis with coexisting alcohol-related peripheral neuropathy. The fourth case involved primary Sjögren's syndrome presenting with distal renal tubular acidosis and hypokalemic paralysis. The fifth case demonstrated overlap connective tissue disease (Sjögren's syndrome with systemic sclerosis features) complicated by hypokalemia. The sixth case was thyrotoxic hypokalemic periodic paralysis, a classic endocrine cause due to intracellular potassium shift. **Conclusion:** This case series highlights the heterogeneous etiologies of hypokalemic paralysis and underscores the importance of identifying reversible secondary causes, including drug-induced renal tubular dysfunction, autoimmune disorders, and endocrine abnormalities, to prevent recurrence and life-threatening complications.

Keywords: Hypokalemic paralysis; Quadriparesis; Periodic paralysis; Renal tubular acidosis; Tenofovir; Sjögren's syndrome; Connective tissue disease; Thyrotoxicosis.

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INTRODUCTION

Hypokalemic paralysis is characterized by episodic or acute onset muscle weakness resulting from reduced serum potassium levels. It is broadly classified into primary hypokalemic periodic paralysis and secondary hypokalemic paralysis, the latter caused by renal tubular disorders, gastrointestinal losses, drugs, insulin excess, alcohol use, endocrine abnormalities, and autoimmune diseases [1]. Severe hypokalemia may result in respiratory failure, cardiac arrhythmias, rhabdomyolysis, and acute kidney injury if not promptly treated [2]. This case series illustrates six distinct etiologies of hypokalemic paralysis encountered in clinical practice.

CASE SERIES

Case 1: Tenofovir-Induced Hypokalemic Quadriparesis

A 52-year-old female with HIV infection on tenofovir, lamivudine, and dolutegravir for 12 years presented with acute onset symmetrical quadriparesis without sensory, bowel, or bladder involvement. Neurological examination revealed hypotonia, power of 2/5 in all limbs, and absent deep tendon reflexes.

Laboratory investigations showed severe hypokalemia (1.81 mmol/L), hyperchloremia (120 mmol/L), normal anion gap metabolic acidosis (pH 7.277, HCO_3^- 11.8 mmol/L), elevated urinary potassium, albuminuria, and reduced eGFR (45 ml/min). A diagnosis of proximal renal tubular acidosis (type 2) with hypokalemic paralysis secondary to tenofovir toxicity was made.

The patient was treated with intravenous and oral potassium supplementation. Tenofovir was discontinued and replaced with abacavir (600 mg). She made a complete neurological recovery with no recurrence on follow-up.

Case 2: Hypokalemic Quadriparesis with Uncontrolled Diabetes Mellitus

A 38-year-old male presented with sudden onset progressive quadriparesis, similar to a prior episode. There was no bladder or bowel involvement. Examination showed reduced tone and muscle power of 2/5 in all limbs with bilaterally flexor plantar responses.

Investigations revealed serum potassium of 2.3 mEq/L, hyperglycemia (GRBS 240–248 mg/dL), leukocytosis with neutrophilia, and sinus tachycardia on ECG. ABG values were near normal. A diagnosis of hypokalemic paralysis secondary to metabolic stress and uncontrolled diabetes mellitus was considered.

The patient was managed with potassium supplementation, intravenous fluids, insulin therapy, and antibiotics. Muscle strength improved following normalization of serum potassium.

Case 3: Hypokalemic Periodic Paralysis with Alcohol-Related Neuropathy

A 40-year-old male with type 2 diabetes mellitus and chronic alcohol consumption presented with acute onset bilateral lower limb weakness and difficulty standing from a squatting position. Upper limb strength was preserved, while lower limb power was 2/5 with reduced reflexes.

A diagnosis of hypokalemic periodic paralysis with coexisting alcohol-related peripheral neuropathy was made. The patient was treated with intravenous and oral potassium, methylcobalamin, and supportive care, resulting in gradual neurological improvement.

Case 4: Primary Sjögren's Syndrome with Hypokalemic Paralysis

A middle-aged female, presented with acute onset symmetrical flaccid quadriparesis without sensory, bowel, or bladder involvement. Neurological examination revealed hypotonia with reduced deep tendon reflexes and preserved cranial nerve function. She reported a history of dry eyes and dry mouth for several months.

Laboratory evaluation showed severe hypokalemia with normal anion gap metabolic acidosis, suggestive of distal renal tubular acidosis. Renal function was preserved. Autoimmune evaluation revealed positive anti-SSA (Ro) antibodies, and Schirmer's test was abnormal, confirming the diagnosis of primary Sjögren's syndrome.

The patient was treated with intravenous potassium supplementation followed by oral potassium citrate, resulting in rapid and complete recovery of muscle strength. She was initiated on disease-specific therapy under rheumatology care and advised long-term electrolyte monitoring.

Case 5: Overlap Connective Tissue Disease with Hypokalemia

Adult female, presented with episodic muscle weakness, fatigue, and myalgias. She had a long-standing history of Raynaud's phenomenon, sicca symptoms, and progressive skin tightening over the fingers. Examination revealed sclerodactyly, reduced oral aperture, and dry oral mucosa.

Laboratory investigations demonstrated hypokalemia with features of distal renal tubular acidosis. Autoimmune workup showed positivity for anti-SSA antibodies, along with clinical features suggestive of systemic sclerosis. A diagnosis of overlap connective tissue disease (Sjögren's syndrome with systemic sclerosis features) was made.

She was managed with potassium and alkali supplementation along with immunomodulatory therapy. Her muscle strength improved following correction of electrolyte imbalance, and she remains under rheumatologic follow-up.

Case 6: Thyrotoxic Hypokalemic Periodic Paralysis

A 29-year-old male with no known comorbidities presented with sudden onset painless weakness of all four limbs on awakening in the early morning hours. The weakness predominantly involved proximal muscle groups and progressed over a few hours. There was no sensory loss, cranial nerve involvement, or bowel or bladder dysfunction. The patient reported recent weight loss, palpitations, heat intolerance, and increased appetite.

On examination, he was tachycardic with fine tremors of the hands. Neurological examination revealed hypotonia with muscle power of 2/5 in all four limbs and diminished deep tendon reflexes. Plantar responses were flexor. Laboratory evaluation revealed severe hypokalemia (serum potassium 1.9 mEq/L) with normal renal function and no acid–base disturbance. Electrocardiography showed flattened T waves and prominent U waves. Thyroid function tests demonstrated suppressed thyroid-stimulating hormone levels with elevated free T3 and T4, confirming thyrotoxicosis.

A diagnosis of thyrotoxic hypokalemic periodic paralysis was made. The patient was treated with intravenous potassium supplementation and oral propranolol. Antithyroid therapy was initiated following endocrine consultation. Muscle strength

improved dramatically within 24 hours, and serum potassium normalized. The patient was advised definitive management of hyperthyroidism to prevent recurrence.

DISCUSSION

Hypokalemic paralysis represents a spectrum of disorders with varied etiologies. While primary periodic paralysis is genetic, secondary causes are more frequently encountered in clinical practice [1]. This case series highlights renal, metabolic, autoimmune, drug-induced, and endocrine mechanisms contributing to hypokalemia.

Tenofovir-associated proximal renal tubular dysfunction results in Fanconi syndrome and type 2 RTA with bicarbonate and potassium loss, predisposing to paralysis [3–5]. In uncontrolled diabetes mellitus, hypokalemia may result from insulin-mediated intracellular potassium shift and stress-induced catecholamine excess [6]. Chronic alcohol consumption further contributes through poor intake, renal losses, and peripheral neuropathy.

Autoimmune disorders such as Sjögren's syndrome are well-recognized causes of distal renal tubular acidosis, leading to hypokalemia and neuromuscular weakness [7]. Overlap connective tissue disorders further increase the risk of renal involvement and electrolyte disturbances [8].

Thyrotoxic hypokalemic periodic paralysis, as illustrated in Case 6, is caused by enhanced Na^+/K^+-ATPase activity driven by thyroid hormone excess, leading to intracellular potassium shift without total body potassium depletion [9]. Early recognition is crucial, as treatment with beta-blockers and control of thyrotoxicosis prevents recurrence.

CONCLUSION

Hypokalemic paralysis is a reversible but potentially fatal neurological emergency with diverse etiologies. In addition to metabolic and drug-induced causes, autoimmune and endocrine disorders such as Sjögren's syndrome and thyrotoxicosis must be actively considered. Prompt potassium correction combined with targeted etiological treatment ensures favorable outcomes and prevents recurrence.

ETHICAL STATEMENT

Written informed consent was obtained from all patients for publication of their clinical details. Patient anonymity has been strictly maintained, and no identifiable personal information has been disclosed. As this is a retrospective descriptive case series without any interventional procedures, approval from the institutional ethics committee was not required, in accordance with local ethical guidelines.

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