Case Report

Recognizing hypokalemic periodic paralysis in the differentials of acute flaccid paralysis: A case-based approach

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ABSTRACT

Hypokalemic periodic paralysis (HypoPP) is a rare but important cause of acute flaccid paralysis that may be easily overlooked during initial evaluation. Timely recognition is essential, as management differs significantly from other neurological or metabolic causes of weakness. We report the case of a 51-year-old male who presented with an acute onset of flaccid quadriparesis. Despite initial treatment, the patient exhibited persistently low serum potassium levels, which did not respond adequately to standard potassium replacement. This unusual biochemical profile, combined with the clinical picture, led to a focused diagnostic approach that confirmed HypoPP. This case highlights the importance of including HypoPP in the differential diagnosis of acute flaccid paralysis, especially in patients with recurrent or persistent hypokalemia. Early identification and appropriate management can significantly alter patient outcomes and are essential to establish appropriate long-term management strategies to prevent recurrent episodes.

Key words: Acute flaccid paralysis, Case report, Channelopathy, Hypokalemia, Hypokalemic periodic paralysis

Hypokalemic periodic paralysis (HypoPP) is an autosomal dominant channelopathy resulting from mutations in genes encoding voltage-gated calcium (CACNA1S) or sodium (SCN4A) channels in skeletal muscle [1,2]. These genetic alterations lead to episodes of flaccid paralysis associated with hypokalemia. Other channelopathies that cause periodic paralysis include Andersen-Tawil syndrome, which results from mutations in the *KCNJ2* gene encoding inward rectifier potassium channels (Kir2.1). These defects impair potassium conductance across skeletal and cardiac myocytes, leading to episodic paralysis, cardiac arrhythmias, and dysmorphic features [3,4]. Mimics of HypoPP include gastrointestinal potassium losses, renal tubular disorders, barium poisoning, and certain endocrinopathies [5]. The cornerstone of acute management in HypoPP is prompt correction of serum potassium, while long-term therapy often involves carbonic anhydrase inhibitors [6].

We present this case to highlight the diagnostic challenges of HypoPP, which can mimic common neurological disorders such as stroke, leading to delays in treatment.

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CASE PRESENTATION

A 51-year-old male with a known history of type 2 diabetes mellitus and hypertension presented with neck pain of insidious onset and dull aching in nature for 2 weeks associated with progressive difficulty maintaining neck posture. These symptoms were accompanied by generalized weakness involving both upper and lower limbs, with greater impairment in the upper extremities, evidenced by an inability to grasp objects effectively.

General examination showed that the patient was afebrile, pulse was 82/min, blood pressure was 138/84 mmHg, respiratory rate was 18/min, and oxygen saturation was 97% on room air. No dysmorphic features or arrhythmias were noted. Neurological examination revealed grade IV power in both lower limbs and grade V in the upper limbs. Sensory and cranial nerve examinations were unremarkable.

Laboratory evaluation demonstrated severe hypokalemia, with an initial serum potassium concentration of 2.0 mmol/L. Despite intravenous potassium replacement, repeat levels remained critically low at 1.8 mmol/L. Magnetic resonance imaging (MRI) brain revealed no evidence of acute infarct or hemorrhage. Other possible causes for acute flaccid paralysis were also considered. Thyroid function tests were normal,

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excluding thyrotoxic periodic paralysis (TPP). Renal tubular acidosis (RTA) was also considered; however, arterial blood gas (ABG) analysis did not demonstrate a non-anion gap metabolic acidosis (NAGMA), and serum chloride levels were within the normal range (101 mmol/L), aiding in the exclusion of renal potassium-wasting disorders. In the absence of other identifiable etiology, a diagnosis of primary HypoPP was established.

The patient was subsequently initiated on oral potassium chloride supplementation and an aldosterone antagonist (spironolactone) to reduce renal potassium loss. Over the course of treatment, the patient demonstrated marked clinical improvement, with restoration of muscle strength and normalization of serum potassium levels.

DISCUSSION

The acute onset of limb weakness in our patient initially raised concerns for a possible cerebrovascular accident or a transient ischemic attack. Given the functional significance of the weakness and the patient's comorbidities, these were reasonable preliminary considerations.

Although hypokalemia was noted on initial laboratory workup, it was initially presumed to be an incidental finding, and prompt potassium replacement was initiated. However, brain MRI revealed no evidence of acute infarct or hemorrhage. The persistent hypokalemia despite adequate correction led to the reconsideration of potassium-wasting etiologies as the primary cause of weakness. This prompted a systematic evaluation of both renal and extrarenal causes of hypokalemia-induced paralysis. RTA, a common renal cause of hypokalemia, was considered; however, it was excluded based on normal ABG findings (no NAGMA), absence of hyperchloremia, and an unremarkable urine osmolar gap. TPP, another correctable cause of hypokalemic paralysis, especially in Asian men, was considered [3]. Notably, patients with TPP may typically not present with features of hyperthyroidism, and hypokalemia is a common presenting feature, further fuelled the probability of TPP [7]. The overactivity of Na^+-K^+ ATPase channel in a thyrotoxic patient leads to inward displacement of K^+ ions [8], thus causing low serum potassium levels. However, a normal thyroid ruled out the possibility of TPP.

Ultimately, the recurrent and refractory hypokalemia in the absence of other identifiable causes, combined with the acute flaccid motor weakness and exclusion of mimicking conditions, led to the diagnosis of HypoPP. This case highlights the importance of considering channelopathies like HypoPP in patients presenting with acute weakness and unexplained hypokalemia. It underscores the necessity of a methodical diagnostic approach to exclude secondary causes.

Given the patient's persistent hypokalemia despite repeated intravenous potassium supplementation, long-term management methods were instituted, which ranged from dietary modification to pharmacologic therapy. Diuretics such as carbonic anhydrase inhibitors have no role in acute attacks. However, they play an important role in reducing the frequency and intensity of the attacks [9]. Carbonic anhydrase inhibitors such as acetazolamide are commonly used as a first-line drug in the maintenance therapy for HyoPP. However, recent studies highlight the emerging role of dichlorphenamide in the management of HyoPP, especially in terms of potency as compared to acetazolamide [9]. Dichlorphenamide is emerging as a drug of choice in patients' refractory to acetazolamide [9-11]. Other potassium-sparing diuretics, such as mineralocorticoid receptor antagonist, are used as second-line drugs in the management of HyoPP. Our patient was initiated on a potassium-rich diet and tablet Aldactone as a part of the maintenance therapy. This approach not only supports serum potassium homeostasis but also mitigates further renal potassium loss.

HypoPP remains a diagnostic challenge due to its episodic presentation and broad differential diagnoses. This case underscores the importance of a systematic approach in evaluating acute flaccid paralysis, particularly in patients with unexplained hypokalemia. Through careful exclusion of more common etiologies such as cerebrovascular events, renal tubular disorders, and TPP, a diagnosis of HypoPP was established. Timely recognition and targeted management, including potassium supplementation, dietary optimization, and the use of potassium-sparing agents, led to a favorable clinical outcome. Increased awareness and early intervention are critical in preventing recurrence and potential complications, ultimately improving long-term patient prognosis.

CONCLUSION

This case emphasizes the importance of considering HypoPP in the differential diagnosis of acute flaccid paralysis. Early recognition and appropriate management can prevent recurrent episodes, improve prognosis, and reduce misdiagnosis with more common neurological conditions.

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