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Emergency Medicine

WEAK IN THE KNEES: A CASE OF HYPOKALEMIC PERIODIC PALSY PRESENTING TO THE EMERGENCY DEPARTMENT

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ABSTRACT Hypokalemic Periodic Paralysis (HPP) is a rare but clinically significant neuromuscular disorder that is primarily characterized by recurrent, episodic muscle weakness or paralysis associated with abnormally low serum potassium levels. The severity of symptoms is not always proportional to the degree of potassium deficiency, with rapid declines in potassium levels often leading to pronounced clinical manifestations, even if the serum concentration remains above 2.5 mEq/L. The pathophysiology of HPP involves transcellular potassium shifts from the extracellular to the intracellular space, disrupting muscle contraction and impairing neuromuscular function. HPP may be familial, with genetic mutations in ion channels being implicated, or secondary, triggered by factors such as high-carbohydrate meals, physical exertion, stress, or underlying endocrine conditions like hyperthyroidism. The diagnosis relies heavily on clinical evaluation, electrolyte analysis, and occasionally genetic testing. Acute management includes prompt potassium replacement therapy, magnesium supplementation, and close cardiac monitoring to avert life-threatening complications, including arrhythmias. Long-term management involves lifestyle modifications aimed at preventing recurrences, including avoiding known triggers and maintaining a potassium-sparing diet, along with the use of potassium-sparing medications. With appropriate therapy and continuous monitoring, most patients experience complete recovery between episodes and can maintain normal, active lifestyles. However, frequent or poorly managed attacks can result in chronic muscle weakness and significant morbidity. This case highlights the importance of early recognition, intervention, and follow-up in improving outcomes for individuals with HPP.

KEYWORDS : Hypokalemic periodic palsy, periodic palsy

INTRODUCTION

Hypokalemic periodic paralysis (HPP) is a rare neuromuscular disorder characterized by recurrent episodes of muscle weakness or paralysis, typically associated with abnormally low serum potassium levels [1]. Notably, the severity of symptoms does not always correlate with the absolute degree of hypokalemia [2]. For instance, while serum potassium levels above 2.5 mEq/L are generally considered less likely to produce symptoms, a rapid decline in potassium can still precipitate significant muscular dysfunction [3].

Hypokalemia may result from several mechanisms, including genetic mutations affecting ion channels (as seen in familial forms), excessive renal or gastrointestinal potassium loss, or transcellular shifts of potassium from the extracellular to the intracellular compartment [4]. Prompt recognition and correction of hypokalemia are essential, as both hypokalemia and hyperkalemia carry a risk of serious complications, including cardiac arrhythmias and neuromuscular impairment[5].

A thorough medical history and physical examination are key to identifying the underlying cause [6]. Understanding the physiology of potassium homeostasis is fundamental to diagnosing and managing disorders of potassium balance. As the most abundant intracellular cation, potassium is vital for numerous physiological processes, especially for maintaining resting membrane potential-largely governed by the ratio of intracellular to extracellular potassium concentrations, as described by the Nernst equation [7].

CASE REPORT

A 20-year-old previously healthy male presented to the Emergency Department with complaints of bilateral lower limb weakness and giddiness. He was apparently asymptomatic until four days prior to presentation, when he developed intermittent high-grade fever associated with myalgia, not accompanied by chills or rigors, and partially relieved with over-the-counter medications. The last fever spike occurred 12 hours before arrival. About eight hours prior to presentation, he developed sudden-onset, symmetrical weakness of both lower limbs, which was flaccid and progressive, without associated sensory loss, trauma, or bladder/bowel involvement. Six hours before arrival, he also developed giddiness and difficulty walking. There were no upper limb symptoms or cranial nerve involvement. He denied any past history of similar episodes, recent medication use, or family history of neuromuscular disorders.

On initial assessment in the Emergency Department, the patient was conscious and oriented, with a Glasgow Coma Scale (GCS) of E4V5M6, and bilaterally reactive pupils. The airway was patent, with the patient vocalizing spontaneously. Breathing was spontaneous, respiratory rate was 18/min, with SpO_2 of 99% on room air, and bilateral vesicular breath sounds without added sounds. Circulation was stable: pulse rate 78/min, blood pressure 128/80 mmHg, all peripheral pulses were palpable, S1S2 was audible, and IV access was secured using an 18G cannula. On exposure, the patient was afebrile, with no signs of trauma, rashes, or bleeding sources. General examination revealed no pallor, icterus, cyanosis, clubbing, lymphadenopathy, or edema.

The patient had complaints of fever for four days, followed by giddiness for six hours, and progressive symmetrical lower limb weakness for the past eight hours. On neurological examination, motor power was preserved in the upper limbs (5/5) and decreased in the lower limbs (2/5 bilaterally). Reflexes were 2+ in biceps, triceps, and supinator bilaterally, while knee and ankle reflexes were absent, and plantar responses were mute bilaterally. Sensation was intact.

Laboratory investigations revealed: hemoglobin 14.6 g/dL, serum sodium 140.7 mmol/L, potassium 1.85 mmol/L, and chloride 105.4 mmol/L. Arterial blood gas showed a pH of 7.349, pCO_2 34.5 mmHg, pO_2 46.2 mmHg, HCO_3 18.6 mmol/L, lactate 1.57 mmol/L, and blood glucose 133.6 mg/dL. Upper limb motor examination remained normal, while lower limb motor strength was decreased bilaterally.

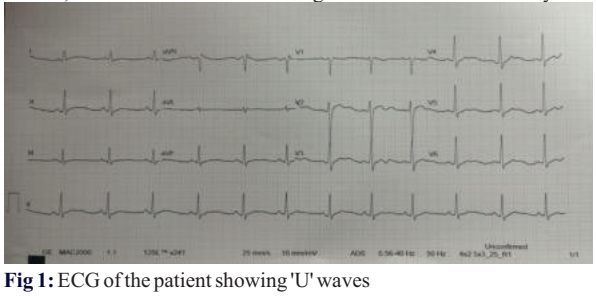

Fig 1: ECG of the patient showing 'U' waves

The patient was managed with IV potassium chloride 60 mEq over 4–5 hours in 500 mL normal saline, IV hydration, magnesium sulphate 2 g in 100 mL NS over 20 minutes, and oral syrup potassium chloride. He was placed on continuous cardiac monitoring, and urine output and serum potassium were monitored closely. Potassium levels were repeated every 2 hours, and after initial correction, potassium

improved to 3.7 mmol/L, with corresponding improvement in lower limb strength and disappearance of U waves on ECG. Urine output remained adequate at 70 mL/hr. By the time of discharge, serum potassium had normalized to 4.8 mmol/L, and the patient's lower limb power had returned to baseline.

DISCUSSION

Hypokalemic Periodic Paralysis (HPP) is a rare neuromuscular disorder characterized by transient episodes of muscle weakness or flaccid paralysis that occur in association with low serum potassium levels. The severity of these episodes can vary, ranging from mild weakness to complete paralysis, often triggered by identifiable factors. The condition may be familial, typically inherited in an autosomal dominant pattern, or secondary to acquired causes, particularly involving electrolyte imbalances. In familial HPP, the disorder is most commonly associated with mutations in genes encoding for voltage-gated ion channels, such as CACNA1S (which encodes for calcium channels) and SCN4A (which encodes for sodium channels). In contrast, secondary HPP arises from conditions that lead to excessive potassium loss (either renal or gastrointestinal) or shifts of potassium from the extracellular to the intracellular compartment. Such causes include hyperthyroidism (especially thyrotoxic periodic paralysis), renal tubular acidosis, diuretic abuse, infections like dengue fever, and excessive insulin or carbohydrate intake.

The pathophysiology of HPP centers around the abnormal movement of potassium, which disrupts the resting membrane potential of muscle cells. Potassium, being the primary intracellular cation, plays a crucial role in muscle cell function. When potassium shifts into the cells, the serum potassium levels drop, and the muscle fibers become hyperpolarized, impairing their ability to depolarize and contract, which results in weakness or paralysis. Familial cases of HPP are typically triggered by factors like stress, rest, high-carbohydrate meals, or vigorous exercise, while secondary HPP is often precipitated by an underlying condition such as viral infections (e.g., dengue) or thyroid dysfunction.

Clinically, patients present with sudden-onset, symmetrical flaccid paralysis, which often affects the lower limbs but may also involve the upper limbs in more severe cases. The episodes are typically brief (lasting hours to days) and may occur spontaneously or after certain triggers like a high-carbohydrate meal or exercise. During an episode, the patient typically experiences weakness that may be accompanied by giddiness, and the reflexes are diminished or absent, particularly in the affected limbs. Sensory function is usually preserved, and cranial nerve involvement is rare. In secondary HPP, additional features of the underlying condition, such as fever, palpitations, or tremors, may also be present.

The diagnosis of HPP is primarily clinical and relies on a history of episodic weakness associated with hypokalemia (serum potassium < 3.5 mmol/L) during attacks. Electrocardiographic changes such as U waves, flattened T waves, and prolonged PR intervals are commonly observed in the setting of hypokalemia. In some cases, urine potassium levels and magnesium levels may be measured to determine the cause of the hypokalemia (renal versus non-renal loss). In familial HPP, genetic testing may reveal mutations in ion channel genes. In secondary HPP, further investigations are needed to identify the underlying cause, such as thyroid function tests, imaging, or viral serology.

Several conditions must be considered in the differential diagnosis, including Guillain-Barré syndrome, myasthenia gravis, botulism, and spinal cord lesions, all of which can present with acute flaccid paralysis. However, HPP is distinguished by the absence of sensory deficits, the presence of characteristic ECG changes during episodes of weakness, and a history of triggering events.

The management of HPP is based on the prompt correction of potassium deficiency, which is essential for reversing the paralysis and preventing complications. In cases with severe or symptomatic hypokalemia, intravenous potassium chloride is administered, typically over several hours while closely monitoring the ECG for any changes in cardiac rhythm. In less severe cases, oral potassium supplementation can be used. In addition to potassium, magnesium supplementation may be necessary in the presence of hypomagnesemia, which can also contribute to muscle weakness. Cardiac monitoring is essential, as rapid potassium shifts can lead to dangerous arrhythmias. In cases of familial HPP, medications like carbonic anhydrase inhibitors (e.g., acetazolamide) can help prevent episodes by mildly acidifying the blood and promoting potassium movement out of cells.

In secondary HPP, treatment revolves around managing the underlying condition, whether it's correcting hyperthyroidism, treating diarrhea-induced potassium loss, or addressing insulin or drug-induced hypokalemia. Additionally, patients should be advised to avoid triggers such as high-carbohydrate meals and excessive physical exertion. Long-term management involves regular potassium monitoring and the potential use of potassium-sparing agents in cases with recurrent attacks. Preventive strategies also include adequate hydration, a potassium-rich diet, and lifestyle modifications such as avoiding strenuous exercise.

The prognosis for most patients with HPP is generally good, with complete recovery between episodes. However, repeated or severe episodes can lead to persistent weakness or myopathy. In the case of familial HPP, some patients may experience progressive muscle weakness if the episodes are not well managed. Therefore, prevention of recurrent attacks is crucial for maintaining muscle function and preventing permanent disability.

Hypokalemic Periodic Paralysis is a potentially reversible condition with important neurological and electrolyte management implications. Early recognition and prompt correction of hypokalemia are key to preventing severe complications and ensuring recovery. With appropriate treatment and lifestyle modifications, most patients can lead normal lives without long-term disability

CONCLUSIONS

In conclusion, hypokalemic Periodic Paralysis (HPP) is a rare but important clinical condition that requires early recognition and prompt intervention. The condition is often characterized by sudden-onset, reversible muscle weakness or paralysis due to low serum potassium levels, which can be triggered by factors such as high-carbohydrate meals, exercise, stress, or underlying conditions like hyperthyroidism [8,9,10]. The pathophysiology revolves around shifts of potassium from the extracellular to intracellular space, disrupting muscle function and leading to impaired muscle contraction [11].

While familial HPP is typically genetically inherited, secondary HPP can result from a variety of factors including viral infections, endocrine disorders, or electrolyte imbalances [12,13,14]. Diagnosis relies heavily on clinical signs, electrolyte findings, and sometimes genetic testing [15,16]. Effective management revolves around the correction of hypokalemia and addressing any underlying causes or precipitating factors. Potassium replacement therapy, magnesium supplementation if required, and cardiac monitoring are essential steps to manage acute episodes and prevent life-threatening complications like arrhythmias [17,18].

Long-term management involves preventing recurrences through lifestyle modifications, such as avoiding triggers, maintaining a potassium-rich diet, and using potassium-sparing medications in recurrent cases [19]. With appropriate treatment and monitoring, the majority of patients recover fully between episodes, and the prognosis is generally favorable. However, persistent or frequent attacks without proper management can lead to chronic muscle weakness and complications, emphasizing the importance of early intervention and careful follow-up [20].

In summary, while HPP is a rare disorder, it is crucial to recognize the condition promptly, treat episodes aggressively, and take preventive measures to reduce recurrence. With careful management, most patients can lead normal, active lives with minimal long-term effects [21].

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