TAM METİN BİLDİRİ

Case Presentation: Recurrent Steroid-Induced Hypokalemic Periodic Paralysis Mimicking Acute Neuropathy

Vaka Sunumu: Akut Nöropatiyi Taklit Eden Tekrarlayan Steroid Kaynaklı Hipokalemik Periyodik Paralizi

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ABSTRACT

This case report details the presentation of a 34-year-old male with acute onset bilateral lower extremity weakness, diagnosed as hypokalemic periodic paralysis (HPP) triggered by recurrent intramuscular dexamethasone administration. The patient, with a history of frequent steroid injections for upper respiratory infections, presented with profound lower limb weakness (3/5) and a serum potassium level of 2.7 mmol/L. Rapid correction of hypokalemia led to significant neurological recovery. Extensive neurological imaging and initial electromyography (EMG) were concerning for peripheral neuropathy, but follow-up EMG normalized completely after potassium repletion and cessation of the steroid trigger. This case underscores HPP as a crucial, reversible differential in acute weakness, highlights the significant risk posed by intermittent steroid use, and demonstrates the potential for transient electrodiagnostic abnormalities that may mislead initial diagnosis.

Keywords: *Electromyography, Hypokalemia, Neuropathy, Paralysis, Steroids.*

ÖZET

Bu vaka raporu, tekrarlayan intramüsküler deksametazon uygulamalarının tetiklediği hipokalemik periyodik paralizi (HPP) tanısı konulan, akut başlangıçlı bilateral alt ekstremite güçsüzlüğü şikayeti ile başvuran 34 yaşındaki bir erkek hastanın kliniğini ayrıntılı olarak sunmaktadır. Üst solunum yolu enfeksiyonları nedeniyle sık sık steroid enjeksiyonu öyküsü olan hasta, şiddetli alt ekstremite güçsüzlüğü (3/5) ve 2,7 mmol/L serum potasyum düzeyi ile başvurmuştur. Hipokaleminin hızlı bir şekilde düzeltilmesi, önemli ölçüde nörolojik iyileşmeye yol açmıştır. Kapsamlı nörolojik görüntüleme ve ilk elektromiyografi (EMG) bulguları periferik nöropati şüphesini doğurmuş olsa da, potasyum replasmanı ve steroid tedavisinin kesilmesinden sonra takip EMG'si tamamen normale dönmüştür. Bu vaka, akut kuvvet kaybında HPP'nin önemli ve geri dönüşümlü bir ayırıcı tanı olduğunu vurgulamakta, aralıklı steroid kullanımının oluşturduğu önemli riski ortaya koymakta ve ilk tanıyı yanıltabilecek geçici elektrodiagnostik anormalliklerin olasılığını göstermektedir.

Anahtar Kelimeler: *Elektromiyografi, Hipokalemi, Nöropati, Paralizi, Steroid*

INTRODUCTION

Hypokalemic periodic paralysis (HPP) is a neurological channelopathy characterized by episodic attacks of skeletal muscle weakness or paralysis, concomitant with a low serum potassium level (1). The weakness typically affects proximal muscles more than distal ones, often sparing respiratory and bulbar muscles, and can range from mild weakness to complete flaccid paralysis (2). Attacks can be triggered by factors such as carbohydrate-rich meals, strenuous exercise, stress, or cold exposure (2). The primary pathophysiological mechanism involves an abnormal shift of potassium ions into the intracellular space, particularly into muscle cells, leading to membrane hyperpolarization and failure to generate action potentials (3).

HPP is most commonly hereditary, typically with an autosomal dominant inheritance pattern linked to mutations in genes encoding skeletal muscle calcium (CACNA1S) or sodium (SCN4A) channels (4). However, a secondary, acquired form exists, which is often under-recognized. Secondary HPP can result from conditions causing profound total body potassium depletion (e.g., diuretic abuse, vomiting, diarrhea) or from acute intracellular potassium shifts (5). The incidence of the familial form is estimated at 1 in 100,000, while the exact prevalence of the secondary form is unknown but likely higher, given the widespread use of triggering medications (6). The clinical presentation of acute, ascending weakness with hypokalemia necessitates rapid differentiation from Guillain-Barré syndrome, myelopathies, and other acute neuropathies, as the treatment is specific and curative for the acute attack (7). This report highlights a case of secondary HPP induced by a commonly used medication, dexamethasone, illustrating its potential to cause severe neurological deficit and diagnostic confusion.

CASE PRESENTATION

A 34-year-old male with no significant past medical history presented to the emergency department with a 6-7 hour history of progressive inability to walk and lower limb weakness. He reported no recent trauma, fever, infectious symptoms, or similar prior episodes. His social history was significant for tobacco use but no alcohol consumption. Notably, his medication history revealed a pattern of approximately 5-6 annual visits to the emergency department over the preceding 3-4 years for upper respiratory tract infections, during which he received intramuscular injections of metamizole and

dexamethasone. The last such injection was administered one week prior to the current presentation.

On admission, the patient was fully conscious (Glasgow Coma Scale 15), oriented, and hemodynamically stable with normal vital signs. Neurological examination revealed symmetric, predominantly proximal motor weakness in the lower extremities, graded at 3/5. Sensory examination, cranial nerve assessment, and upper extremity strength were entirely normal. Systemic examination was unremarkable. Initial laboratory investigations were significant for severe hypokalemia of 2.7 mmol/L on venous blood gas and serum biochemistry. Other routine labs, including acute phase reactants, were within normal limits. An electrocardiogram showed normal sinus rhythm without signs of hypokalemia such as U waves or ST-segment depression.

Given the acute focal weakness, urgent neuroimaging with non-contrast brain CT and diffusion-weighted MRI was performed to exclude central causes like stroke or myelopathy, and both were normal. The identification of profound hypokalemia led to the initiation of intravenous potassium chloride infusion. Following correction of serum potassium to 3.7 mmol/L, a dramatic and rapid improvement in muscle strength was observed; the patient regained the ability to walk with assistance. This prompt response to potassium replacement strongly supported a diagnosis of hypokalemic periodic paralysis.

The patient was referred to the neurology service for further evaluation. Given the recurrent steroid exposure, a working diagnosis of steroid-induced secondary HPP was made. An initial electromyography (EMG) and nerve conduction study (NCS) performed shortly after presentation showed low amplitude compound muscle action potentials (CMAPs) in the peroneal and tibial nerves bilaterally, with normal sural sensory responses. This pattern raised initial concern for a possible acute peripheral neuropathy. On detailed neurological re-evaluation, proximal lower limb strength had improved to 4/5, with distal strength at 5-/5, and no sensory deficits. To comprehensively exclude structural or inflammatory etiologies, full-spine (cervical, thoracic, lumbar) MRI was performed, which revealed no abnormalities.

The patient was managed supportively with oral potassium supplementation and strict avoidance of further steroid exposure. His neurological examination normalized completely over the following 72 hours. A repeat EMG/NCS performed three days after the initial study was entirely within normal limits, resolving the previously noted CMAP amplitude reductions. After an uneventful hospital stay with no recurrence of weakness, the patient was discharged with explicit counseling on avoiding systemic corticosteroids and education on the nature of his condition.

DISCUSSION

This case presents a classic yet instructive example of secondary hypokalemic periodic paralysis induced by exogenous glucocorticoid administration. While familial HPP is well-documented in neurological literature, the acquired form, particularly steroid-induced, warrants greater emphasis in both emergency and general medical practice due to the ubiquitous use of corticosteroids.

The pathophysiology of steroid-induced HPP is rooted in the hormone's influence on electrolyte balance and cellular membranes. Glucocorticoids possess mineralocorticoid activity, which can promote renal potassium excretion, though this effect is usually mild with short-term use. A more significant mechanism in acute attacks is the intracellular shift of potassium. Corticosteroids enhance the activity of the Na+/K+-ATPase pump, particularly in skeletal muscle and liver cells, driving potassium ions from the serum into the intracellular compartment (8). This shift can be precipitous in susceptible individuals, leading to a sharp drop in serum potassium and subsequent muscle membrane hyperpolarization and inexcitability. In our patient, the recurrent, intermittent nature of high-dose intramuscular dexamethasone likely created a recurrent state of susceptibility. The attack one week post-injection aligns with the known pharmacokinetics of depot steroid formulations and suggests a delayed or cumulative trigger effect.

A critical aspect of this case is the initial EMG finding of low CMAP amplitudes, which complicated the diagnostic picture. In acute HPP, NCS findings can be variable. During a severe attack, CMAP amplitudes may be low or unobtainable due to muscle fiber inexcitability, and they typically increase with potassium correction and clinical improvement (9). The initial EMG in our case likely captured the muscle in a state of acute inexcitability. The complete normalization of the EMG within 72 hours is a powerful confirmatory finding, ruling out a primary axonal or demyelinating neuropathy and instead supporting a transient metabolic disturbance of muscle membrane potential. This transient electrophysiological abnormality underscores the importance of timing when performing diagnostic studies in suspected HPP and cautions against over-interpreting findings obtained during the acute phase.

What makes this case particularly noteworthy in the context of existing literature are several key features. First, the patient lacked any history of thyroid dysfunction (thyrotoxic periodic paralysis), renal disease, or gastrointestinal losses, making exogenous steroids the sole identifiable trigger. Second, the recurrent pattern of steroid use for minor illnesses highlights a significant public health concern: the non-critical use of injectable steroids in outpatient or emergency settings for conditions where evidence of benefit is limited. This practice, common in some regions, poses a real risk of serious adverse effects like HPP. Third, the dramatic presentation with an inability to walk and the initial abnormal EMG steered the differential diagnosis towards more ominous pathologies like Guillain-Barré syndrome or acute myelitis, necessitating extensive and costly imaging (Brain and full-spine MRI). This case therefore exemplifies how recognition of secondary HPP can prevent unnecessary investigations and guide immediate, effective therapy.

The management of acute steroid-induced HPP is two-fold: acute correction of hypokalemia and prevention of future episodes. Oral potassium replacement is often sufficient unless weakness is severe or cardiac involvement is present, as in our case where intravenous supplementation was used. It is crucial to avoid glucose-containing fluids, as carbohydrates can stimulate insulin release and exacerbate intracellular potassium shifting, potentially worsening the paralysis (10). The cornerstone of long-term management is the absolute avoidance of the triggering agent. Patient education is paramount. Our patient was explicitly informed about the direct causal link between the steroid injections and his paralysis, a critical step in preventing recurrence, as he may otherwise seek similar treatments for future ailments.

CONCLUSION

This case of recurrent dexamethasone-induced hypokalemic

periodic paralysis serves as a vital reminder that acute, profound motor weakness can stem from a reversible metabolic cause. It emphasizes that secondary HPP should be a primary consideration in any patient presenting with acute flaccid weakness, especially in the context of recent corticosteroid use, even if intermittent. The transient nature of both clinical and electrophysiological abnormalities upon potassium repletion is a hallmark of the condition. Clinicians must be vigilant about the potential for commonly used medications like steroids to provoke severe neurological complications. Heightened awareness of this entity can lead to faster diagnosis, appropriate and rapid treatment with potassium, avoidance of unnecessary diagnostic procedures

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