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UNRAVELING AN UNCOMMON ENCOUNTER: HYPOKALEMIC PERIODIC PARALYSIS WITH BRUGADA PHENOCOPY AMIDST HYPOKALEMIA

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ABSTRACT

Introduction: Hypokalemic periodic paralysis (HPP) presents a diagnostic challenge due to the painless muscle weakness it causes. This case discusses a patient with HPP along with electrocardiogram (ECG) findings of Brugada phenocopies (BrP) in the setting of hypokalemia. A review of the literature showed that it is the seventh documented example of BrP induced by hypokalemia alongside HPP.

Case description: A 43-year-old man presented to the emergency department with lower limb weakness. He attributed his symptoms to a substantial meal consumed after breaking his Ramadan fast, recalling a similar episode following heavy meals in the past. The patient was alert and oriented but demonstrated reduced strength in both upper and lower limbs. ECG revealed a Brugada type 1 pattern. Laboratory analysis revealed hypokalemia (2.5 mmol/l), elevated creatine kinase (326 U/l), and normal thyroid function. Following potassium supplementation, his symptoms resolved, and his ECG normalized.

Discussion: HPP occurs in the context of increased carbohydrate intake, potentially leading to rapid insulin release and activation of Na-K ATPase, enhancing cellular potassium absorption and lowering serum potassium levels. Symptoms range from weakness and fatigue to severe neuromuscular weakness and cardiac arrhythmias. Investigating hypokalemia requires excluding hypomagnesemia, thyroid function tests, and metabolic acidosis/alkalosis before considering HPP. Management involves gradual oral potassium repletion to avoid the risk of hyperkalemia associated with intravenous administration.

Conclusion: Clinicians should consider including HPP in differential diagnoses of patients presenting with weakness. In this case, electrophysiological evaluation suggested Brugada pattern induced by hypokalemia, which resolved with potassium supplementation.

KEYWORDS

Hypokalemia, hypokalemic periodic paralysis, Brugada phenocopy

LEARNING POINTS

- This case highlights the rarity and diagnostic challenges of hypokalemic periodic paralysis, offering critical insights into recognizing and managing such conditions in clinical practice.
- The case also demonstrates the importance of identifying reversible electrocardiogram changes like Brugada patterns, aiding in differentiation from persistent arrhythmias and avoiding unnecessary interventions.

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BACKGROUND

Hypokalemic periodic paralysis (HPP) is a rare genetic disorder, most commonly caused by autosomal dominant mutations in skeletal muscle ion channels[1,2]. These mutations impair normal muscle membrane excitability, leading to episodes of muscle weakness or paralysis. The condition is characterized by attacks of generalized or focal skeletal muscle weakness in the setting of low serum potassium levels, which typically resolve within hours[1,2]. In addition to causing neuromuscular symptoms, hypokalemia can also trigger cardiac electrophysiological disturbances, including Brugada phenocopies (BrP). BrPs are transient electrocardiogram (ECG) patterns that resemble Brugada syndrome but are caused by reversible, non-genetic clinical conditions that alter cardiac electrophysiology[3]. Unlike true Brugada syndrome, which is hereditary, BrPs are temporary and typically resolve once the underlying condition is addressed.

The paper discusses the rare case of a patient presenting with HPP alongside BrP in the setting hypokalemia that occurs following a heavy meal. The presentation signifies the complex interplay between metabolic disturbances, neuromuscular function, and cardiac electrophysiology, representing a rare, documented instance of the combination.

CASE DESCRIPTION

A 43-year-old male with medical history of hypertension and coronary artery disease presented to the emergency department with acute bilateral lower limb weakness. The patient reported normal muscle strength on the prior day, but he experienced sudden weakness upon waking up the following morning. He attributed the episode to a large meal consumed after breaking his Ramadan fast, noting similar but milder weakness following heavy meals in the past. He denied taking any new medications and denied use of alcohol or drugs. Further interrogation also revealed a family history of early onset cardiac-related deaths.

On examination, the patient was alert and oriented but demonstrated significantly reduced strength in both upper extremities (3/5) and complete weakness in both lower extremities (0/5 bilaterally). Sensation was normal in all extremities, and he had normal deep tendon reflexes. The remainder of the neurologic and physical exam was unremarkable. An ECG revealed normal sinus rhythm at a rate of 90 beats per minute, accompanied by first-degree atrioventricular (AV) block and coved ST elevations in leads V1-V3, consistent with a type I Brugada pattern *(Fig. 1)*. Laboratory analysis showed hypokalemia with a potassium level of 2.5 mmol/l, while the rest of the metabolic panel and urine electrolytes were normal. Creatine kinase was mildly elevated at 326 U/l, and thyroid and liver function tests were within normal limits.

Following potassium supplementation, the patient's weakness completely resolved, and a subsequent ECG showed normalization of the Brugada pattern *(Fig. 2)*. Despite the resolution of symptoms, the patient was admitted for

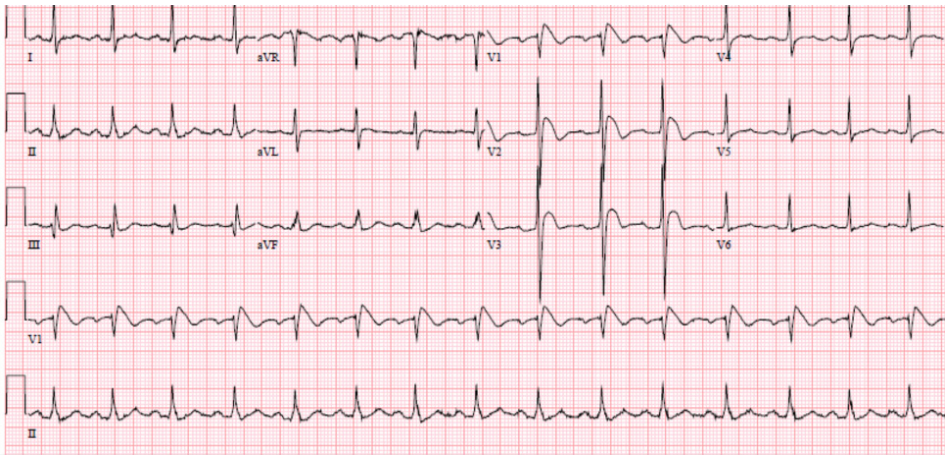

Figure 1. Electrocardiogram demonstrating Brugada syndrome.

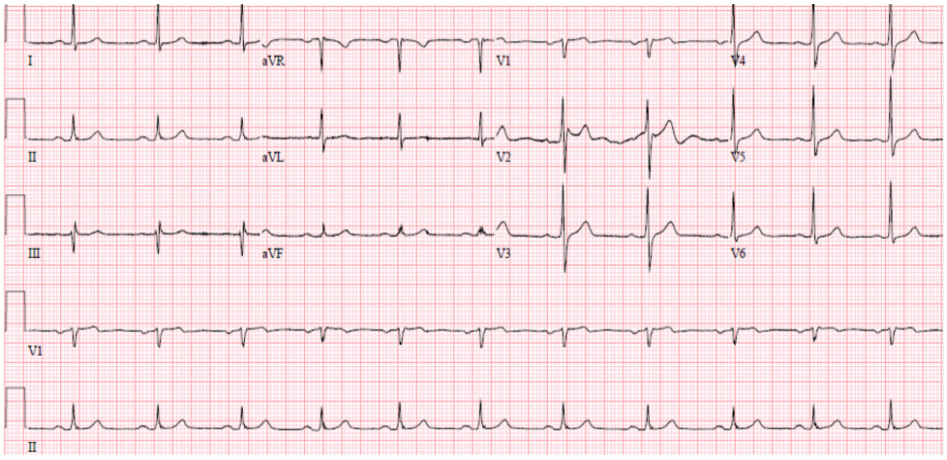

Figure 2. Electrocardiogram with normal sinus rhythm.

further evaluation of suspected Brugada syndrome and periodic paralysis. A computed tomography (CT) scan of the head and magnetic resonance imaging (MRI) of the spine were performed, both of which showed no significant pathological findings.

Given the absence of a history of syncope and the transient nature of the Brugada-like ECG changes, a diagnosis of true Brugada syndrome was deemed unlikely. Therefore, as per guidelines, an implantable cardioverter-defibrillator (ICD) was not recommended. An electrophysiology (EP) study was also conducted, and it revealed normal findings with no evidence of inducible sustained arrhythmias or bundle branch block.

Despite complete recovery, the patient was offered Brugada genetic testing due to concerns regarding his personal and family cardiac history risk. Genetic analysis was recommended, using a cardiomyopathy and arrhythmia panel covering 157 genes related to Brugada syndrome, which came back negative. Genetic testing for HPP confirmation was also recommended, but the patient opted out.

DISCUSSION

Periodic paralyses (PP) are rare autosomal dominant neuromuscular disorders caused by mutations in skeletal muscle sodium, calcium, and potassium channel genes[2]. These disorders are often associated with metabolic and electrolyte imbalances, which trigger episodes of muscle weakness or paralysis[1,2]. PP include hypokalemic paralysis, hyperkalemic paralysis, and Andersen-Tawil syndrome. Among these, HPP is the most prevalent, affecting almost 1 in 100,000 individuals. It is characterized by episodes of focal or generalized skeletal muscle paralysis in the setting of hypokalemia, with attacks lasting from hours to days[1,2]. HPP can be classified into familial and acquired types, each with distinct causes that influence treatment strategies. Familial HPP is a hereditary condition triggered by various factors such as high-carbohydrate meals, hypothermia, stress, infections, and corticosteroid use[1]. In contrast, acquired HPP arises in the setting of underlying conditions such as hyperthyroidism, known as thyrotoxic periodic paralysis (TPP), or other secondary metabolic disturbances. In our patient, a prior history of muscle weakness episodes supports the likelihood of familial HPP. The episode was likely triggered by a high-carbohydrate meal, leading to rapid insulin secretion, activation of the Na+/K+ ATPase pump, intracellular potassium shift, and subsequent hypokalemia-induced paralysis.

The diagnosis of HPP is typically suspected when an individual experiences sudden, flaccid muscle weakness commonly affecting the upper limbs, accompanied by decreased or normal deep tendon reflexes. A positive family or personal history of prior similar episodes strongly supports the diagnosis, which can be confirmed with genetic testing[2]. Genetic analysis in clinically suspected patients reveals heterozygous pathogenic mutations in up to 70% of cases[4]. Mutations usually cause gating pore cation leaks that disrupt muscle excitability. In the absence of a family history, the presence of hypokalemia during an episode is key for establishing the diagnosis. Nevertheless, further testing is necessary to rule out secondary or acquired HPP causes[2]. This includes ruling out hyperthyroidism which can cause TPP, hypomagnesemia, metabolic acidosis/alkalosis, and gastrointestinal or renal losses, all of which were negative in the case of our patient.

Electrocardiographic changes have been commonly reported in patients with HPP, and they are usually hypokalemia-induced[5]. These changes can include arrhythmias such as reentrant circuits and disruptions in activation-repolarization coupling. In the report by Gazzoni et al.[6], BrP ECG changes were documented in the setting of familial HPP, similar to our case. BrPs are reversible ECG changes that usually resolve with correction of the underlying electrolyte imbalance[3]. In cases of arrhythmias with HPP, it is helpful to evaluate patients comprehensively with a baseline ECG, ambulatory Holter monitoring, and cardiac genetic testing to determine the cause and guide management. This approach helps distinguish between transient conditions, like BrPs, and more serious, persistent arrhythmic disorders, ensuring appropriate care and avoiding unnecessary interventions such as ICD placement.

The treatment of hypokalemic paralysis episodes typically involves gradual oral potassium supplementation, carefully titrated to prevent the risk of rebound hyperkalemia[2,7]. This is typically done using oral potassium chloride, which is better absorbed, typically starting at a dose of 0.5 to 1 mEq/kg with a 30% repeat dosing in case of non-response to initial treatment. Monitoring is critical as the hypokalemia seen in HPP is due to a shift of potassium into cells rather than an absolute potassium deficit. In hereditary HPP, management extends beyond acute treatment to include preventive strategies aimed at avoiding triggers to reduce the frequency and severity of future episodes. In contrast, the treatment of acquired HPP focuses on addressing the underlying causes such as managing thyroid disorders or discontinuing offending medications.

CONCLUSION

In conclusion, this case uncovers the importance of considering HPP as a differential diagnosis in patients presenting with sudden-onset paralysis. Hypokalemia can act as a reversible trigger for both muscle weakness and Brugada-like ECG patterns. In our patient, prompt recognition and potassium supplementation effectively reversed both the paralysis and the BrP ECG changes. The case also highlights the need for a comprehensive diagnostic approach along with appropriate and timely management.

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