

CASE REPORT

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Acquired non-thyrotoxic hypokalemic periodic paralysis: A case report

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ABSTRACT

Introduction: Periodic paralysis is a group of rare neuromuscular disorders presenting with episodes of painless paralysis often incited by stress such as exercise. Differentiating hypokalemic periodic paralysis (HPP) from secondary hypokalemia can present a challenge. **Case Report:** A 26-year-old Hispanic male with no significant past medical history presented with periodic muscular weakness, areflexia, and paralysis. The patient's potassium level on admission was 1.6 mEq/l with no other electrolyte abnormalities or electrocardiogram changes. There was a complete resolution of symptoms with IV and oral potassium. The diagnosis of acquired non-thyrotoxic HPP was given based on the patient's history, his presenting symptoms, ruling out secondary causes of hypokalemia, and resolution with correction of potassium levels. **Conclusion:** Hypokalemic periodic paralysis most commonly affects Asians, and is tightly associated with thyrotoxicosis. Treatment of HPP relies on rapid identification and understanding of underlying cause. In an acute disabling attack, as seen in this patient, oral potassium and 24 hour cardiac monitoring for rebound hyperkalemia are recommended. We report an uncommon case

of acquired non-thyrotoxic HPP, as indicated by the patient's history, presentation, initial laboratory values, and resolution after potassium correction.

Keywords: Muscular weakness, Non-thyrotoxic hypokalemic, Periodic paralysis, Potassium level

How to cite this article

Kumar KK, Kadhim Z, Mohan JK. Acquired non-thyrotoxic hypokalemic periodic paralysis: A case report. *Edorium J Neurol* 2016;3:1–3.

Article ID: 100007N06KK2016

doi:10.5348/N06-2016-7-CR-1

INTRODUCTION

Periodic paralysis is a diverse group of rare neuromuscular disorders caused by channelopathies. Clinically, patients present with episodes of painless paralysis often elicited by muscular stress such as exercise or viral illness. Periodic paralysis is most commonly characterized by hypokalemia due to mutations in calcium channels [1–3]. Estimated prevalence of hypokalemic periodic paralysis (HPP) is 1:100,000 with up to one third of cases representing novel mutations [3, 4]. The majority of HPP cases are hereditary; of the minority of acquired cases most are associated with thyrotoxicosis or permanently lowered potassium levels. Differentiating HPP from secondary hypokalemia can present a diagnostic challenge. In one case series of 97 patients initially diagnosed with periodic paralysis, 24 were ultimately found to have secondary hypokalemia

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Received: 25 September 2015
Accepted: 19 November 2015
Published: 29 February 2016

[5]. We report a young Hispanic male presenting with periodic muscle weakness, areflexia and hypokalemia diagnosed with acquired non-thyrotoxic HPP.

CASE REPORT

A 26-year-old Hispanic male, with no significant past medical or family history, presented with a two-week history of progressively worsening extremity weakness and areflexia. The patient experienced one week of copious, watery diarrhea five days prior to admission. The patient denied any current diarrheal bowel movements, headache, neck stiffness, fever, chills, nausea, vomiting, cold/heat intolerance, skin/hair/nail changes, back pain, saddle anesthesia, loss of bladder or bowel control, dysuria, hematuria, recent surgery, any past medical history, any precipitating factors outside of his diarrheal illness (including vigorous exercise and a heavy or a carbohydrate-rich meal), and any family history of similar paralytic events. Physical examination revealed muscular weakness in the upper and lower extremities bilaterally, 0+ areflexia in the patella and ankles bilaterally, and 2+ reflexes in the biceps and triceps. The patient needed assistance for mobility with an otherwise negative neurological examination. Thyromegaly was not appreciated. Initial laboratory tests demonstrated potassium of 1.6 mEq/l and magnesium of 1.3 mg/dl. A lumbar puncture, urine toxicology, and urinalysis were negative. The patient's electrocardiogram on presentation showed no signs of hypokalemia and his chest X-ray was normal. The patient received 1000 mL intravenous normal saline, 50 mL magnesium sulfate, and 250 mL intravenous potassium. The patient was given 40 mEq oral potassium every 30 minutes until his hypokalemia resolved. The patient's laboratory values the following morning showed potassium of 4.4 mEq/l, and magnesium of 2.0 mg/dl. The patient's physical examination was subsequently normal the following morning, approximately 23 hours after presenting, revealing 2+ reflexes and 5/5 motor strength in his upper and lower extremities bilaterally.

DISCUSSION

Differentiating HPP from secondary causes of hypokalemia can present a diagnostic challenge. HPP most commonly affects Asians, and is tightly associated with thyrotoxicosis [6]. We report a Hispanic patient with acquired non-thyrotoxic periodic paralysis, which demographically represents an uncommon case that presented atypically. Paralytic attacks typically last several hours [7], as seen in the patient. Though most attacks are preceded by a carbohydrate rich meal or vigorous exercise, stress such as a viral illness is also a common inciting event [8]. Our patient's paralytic attack was preceded by

diarrheal illness that was presumably gastroenteritis. The mean potassium level for cases of HPP is 2.4 mEq/l with potassium values less than 2.0 mEq/l often suggesting secondary causes of hypokalemia [4]. Hypokalemia most often occurs secondary to dehydration (from chronic diarrhea, chronic laxative abuse, vomiting, or sweating), chronic kidney disease, diabetic ketoacidosis, or renal tubular acidosis. Though the patient did report a seven-day history of diarrhea, he denied any symptoms of hypokalemia until five days after the diarrhea had resolved, which is more suggestive of a viral illness as a stress inciting factor to HPP. Additionally, absent signs of dehydration, distal RTA, and other secondary causes of hypokalemia, as well as the accompanying symptoms which presented in this patient suggest HPP to be the most likely diagnosis. Current guidelines recommend against provocative testing, such as glucose loading, insulin administration, and ACTH administration, and were not performed. In an acute disabling attack, as seen in this patient, oral potassium and 24 hour cardiac monitoring for rebound hyperkalemia are recommended and were performed in this patient. When a patient has persistent paralytic attacks, preventive lifestyle modifications have been suggested including avoidance of vigorous exercise and reduction in dietary carbohydrates. If lifestyle modifications are not effective, the clinician should consider adding medications; oral potassium supplementation, dichlorphenamide, acetazolamide, and spironolactone have been shown to reduce frequency of attacks [8–11]. The patient's initial presentation with absent signs of dehydration, distal renal tubular acidosis, and other secondary causes of hypokalemia, as well as the accompanying symptoms which presented in this patient suggest hypokalemic periodic paralysis (HPP) to be the most likely diagnosis.

CONCLUSION

The case presented here is an uncommon example of acquired non-thyrotoxic hypokalemic periodic paralysis (HPP), as this is a Hispanic patient with no known family history as evidenced by the patient's history, presentation, initial laboratory values, and resolution after potassium correction. This case highlights the importance of having a high index of suspicion in a patient presenting with hypokalemia and flaccid paralysis.

Author Contributions

Keerthana K. Kumar – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Zuhail Kadhim – Analysis and interpretation of data,
Revising it critically for important intellectual content,
Final approval of the version to be published

Jagan K. Mohan – Analysis and interpretation of data,
Revising it critically for important intellectual content,
Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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