Case Report

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Hypokalemic Periodic Paralysis: A Case Report

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Abstract

Introduction: Hypokalemic Periodic Paralysis is one form of Periodic Paralysis, a rare group of disorders that can cause sudden onset weakness. Although rare, Periodic paralysis must be differentiated from other causes of weakness and paralysis so that the proper treatment can be initiated quickly.

Case Report: A 72 year-old elderly male presented to the emergency room with sudden onset of paralysis. He had no respiratory or swallowing difficulty and was able to move his neck and facial muscles.

Neurologic exam revealed flaccid paralysis bilateral lower extremities which involved the proximal and distal muscles. Sensation was intact but deep tendon reflexes were slightly diminished to 3 out of 4 throughout. RFT deranged with serum creatinine level of 1.7 (0.6-1.2mg/dl), potassium level of 1.6 (3.5–5 mmol/L), magnesium level of 0.9 (1.3-2.1meq/l).

Electrocardiogram revealed bradycardia and left axis deviation. Two hours after initiation of intravenous potassium replacement, the patient's neurologic symptoms started resolving.

The patient was diagnosed with Hypokalemic Periodic Paralysis and was started on calcium channel blocker for control of blood pressure. He was discharged home with an appointment to follow up.

Conclusion: Periodic Paralysis should be kept in mind when a patient comes with sudden onset weakness or paralysis, especially when other diseases have been ruled out. It can be life threatening if the treatment is improper, but intervention and subsequent correction of potassium abnormalities can clear the symptoms completely. The underlying etiology should be searched properly to avoid recurrence or persistence of the paralysis.

Keywords: Familial Hyperkalemic Paralysis; Hypokalemic Periodic Paralysis; Thyrotoxic Periodic Paralysis.

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Key Messages: The most frequent form of Periodic paralysis is hypokalemic periodic paralysis (HPP).

The abrupt onset of weakness, which can range in severity from mild, transient weakness to severe impairment leading to respiratory failure, is what makes the condition most distinctive.

Stress factors like viral fever or medications like beta-agonists, insulin, or

steroids can worsen it. Low potassium levels and muscular dysfunction are brought on by marked reduction in sodium and calcium ion channels.

In addition to potassium replacement, the heart rhythm and serum potassium are closely monitored during treatment. Prophylaxis can involve the use of spironolactone and acetazolamide.

INTRODUCTION

Hypokalemic Periodic Paralysis is one form of Periodic Paralysis, a rare group of disorders that can cause sudden onset weakness. A case of a 72 year old male is presented here. The patient presented with sudden onset paralysis of his extremities. Laboratory evaluation revealed a markedly low potassium level. The patient's paralysis resolved upon repletion of his low potassium and he was discharged with no neurologic deficits. Further workup revealed there was Hypomagnesaemia in this patient.

CASE REPORT

A 72 year-old elderly male presented to the emergency room with sudden onset paralysis. The patient had gone to bed at 10 pm with no weakness and awoke at midnight unable to move his upper or lower extremities. The weakness was bilateral involving both the lower extremities. He was unable to walk and get up from sitting and squatting position. He had no respiratory or swallowing difficulty and was able to move his neck and facial muscles. He denied any pain or paresthesia. Prior to this episode, the patient had been healthy and denied any recent diarrheal, chest pain, shortness of breath, or weight change. He reported several episodes of dizziness and tiredness while working in his farm field. He did not take any other medications than his regular antihypertensives and denied use of alcohol or drugs, or significant changes in diet or activity levels. He is a known case of old CVA and hypertension on Tab Atenolol 50 mg and Amlodipine 5 mg. His parents and brother had no history of similar episodes and no other significant illnesses.

On physical exam, the patient's heart rate was 42 bpm and blood pressure was 110/80 mmHg, respiratory rate was 16com and Spo2 was 98% at room air. He was moderately built and nourished and normal in overall appearance His skin was cool and dry, and the oral mucosa was moist. No jugular venous distension, goiter or lymphadenopathy were appreciated. Cardiac evaluation revealed bradycardia with a regular rhythm and no murmurs. Examination of the lungs and abdomen were unremarkable. There were no deformities or enema of the extremities and distal pulses were present and equal bilaterally. Neurologic exam revealed flaccid paralysis bilateral lower extremities which involved the proximal and distal muscles. Sensation was intact but deep tendon reflexes were slightly diminished to 3 out of 4 throughout. Cranial nerve function was grossly intact. Fundoscopic examination revealed bilateral normal fundus study. 2D echo revealed grade 1 LV diastolic dysfunction, with LV EF=55%.

Routine chemistry, complete blood count were normal, RFT deranged with serum creatinine level of 1.7 (0.6-1.2mg/dl), potassium level of 1.6 (3.5–5 mmol/L), magnesium level of 0.9 (1.3-2.1meq/l)

Electrocardiogram revealed revealed bradycardia and left axis deviation.

CT brain plain study revealed a well circumscribed round soft tissue density lesion measuring ~ 2.5x2.4 cm with few specs of calcifications noted in the subcutaneous plane in high parietal region ~ likely trichelemmal cyst.

Inj. Potassium chloride 40 mEq in 500ml RL over 6 hours, Inj Magnesium 2 gm in 100 ml NS over 30 minutes was given. Two hours after initiation of intravenous potassium replacement, the patient's neurologic symptoms started resolving. His heart rate was normal, however repeat electrocardiogram revealed a normal sinus rhythm with bradycardia. Follow up studies were performed to determine the etiology of the patient's hypokalemia. Urine sodium and potassium levels were measured to rule out adrenal involvement and were found to be normal. The patient was diagnosed with Hypokalemic Periodic Paralysis and was started on calcium channel blocker for control of blood pressure. He was discharged home with an appointment to follow up.

DISCUSSION

Weakness is the most common and general complaint both in the inpatient and outpatient

units. While there are several possible differential diagnoses for it (Table 1), the emphasis is significantly narrowed when a patient exhibits a clear decline in muscle strength on examination.[1]

Table 1: Causes of acute weakness

Neurologic
Stroke
Post-seizure Paralysis
Myasthenia Gravis
Cataplexy
Multiple Sclerosis
Inflammatory
Polymyositis
Dermatomyositis
Infectious
Polio
Diphtheria
Botulism
Metabolic
Porphyria
Alcohol/Opiates
Electrolyte disorder

Table 2: Causes of Hypokalemia

Potassium Depletion – Renal
Increased aldosterone
Diuretics
Hypomagnesemia
Renal Tubular Acidosis (Type I and II)
Metabolic alkalosis
Liddle's syndrome
Potassium Depletion – Extra renal
Decreased intake
Vomiting/Diarrhea
Zollinger-Ellison Syndrome
Fistulas
Potassium Shift into Cells
Increased insulin
Alkalosis
Thyrotoxic Periodic Paralysis
Familial Hypokalemic Paralysis

The possibility of death from nerve compression brought on by tumours and stroke should be ruled out first. Postictal paralysis or disorders of the motor neuron are some of the more frequent causes. For a diagnosis, a thorough history is required, paying close attention to the dates, durations, and different body areas affected.[1,2]

Periodic paralysis is a differential that is typically overlooked during the patient's initial work-up. Periodic paralysis comes in a variety of forms that are connected to problems in electrolytes and metabolism. The most frequent of these is hypokalemic periodic paralysis (HPP). Prevalence is 1 in 100,000 people. The underlying aetiology may affect the signs and symptoms. The abrupt onset of weakness, which can range in severity from mild, transient weakness to severe impairment leading to respiratory failure, is what makes the condition most distinctive. Stress factors like viral fever or medications like beta-agonists, insulin, or steroids can worsen it. Low potassium levels and muscular dysfunction are brought on by marked reduction in sodium and calcium ion channels.[1] Tendon reflexes may be absent, yet sensation is typically unaffected, because there is a defect with muscle contraction rather than nerve conduction. Although serum potassium levels are extremely low, total body potassium is within normal limits, and a change in serum level indicates a potassium shift inside cells.[2] Despite being present, electrocardiographic alterations do not match serum levels.[3] Because the patient may be clinically normal and the serum levels and electromyography may be within the normal range, it is challenging to diagnose it between two episodes. Since HPP can arise in a variety of circumstances, a thorough investigation is necessary to make a diagnosis. Familial Hypokalemic Paralysis (FHP), a form of HPP, can occur sporadically. It can be inherited either as autosomal dominant or spontaneously.[4] Because of defective sodium or calcium channel function, there is disrupted cellular potassium control in this form.[2,5] Defects in sodium channels caused by mutations in the CACNA1S and SCN4A genes result in aberrant potassium ion flow. In addition to potassium replacement, the heart rhythm and serum potassium are closely monitored during treatment. Prophylaxis can involve the use of spironolactone and acetazolamide.[2]

Conflict of Interest: Nil

REFERENCES

1. Jurkat-Rott K, Lerche H, Lehmann-Horn F: Skeletal muscle channelopathies.J Neurol 2002, 249(11):1493-1502.
2. Lin SH, Lin YF, Chen DT, Chu P, Hsu CW, Halperin ML: Laboratory tests to determine the cause of hypokalemia and paralysis. Arch Intern Med 2004, 164(14):1561-1566.
3. Kelley DE, Gharib H, Kennedy FP, Duda RJ Jr, McManis PG: Thyrotoxic periodic paralysis. Report

of 10 cases and review of electromyographic findings. Arch Intern Med 1989, 149(11):2597-2600.

4. Fontaine B, Vale-Santos J, Jur ka t- Rott K, Reboul J, Pl assa rt E, R im e CS, Elbaz A, Heine R, Guimaraes J, Weissenbach J, *et al*.: Mapping of the hypokalaemic periodic paralysis (HypoPP) locus to chromosome 1q31-32 in three European families. Nat Genet 1994,6(3):267-272.
5. W ang W, Jia ng L, Ye L, Zhu N, Su T, Guan L, Li X, Ning G: Mutation screening in Chinese hypokalemic periodic paralysis patients. Mol Genet Metab 2006, 87(4):359-363.