CASE REPORT

Hypokalemic Periodic Paralysis

Abidullah Khan[1], Muhammad Farooq[2]

ABSTRACT

Hypokalemic Periodic Paralysis is a group of rare inherited disorders that can cause temporary and often recurrent episodes of acute flaccid paralysis. Several conditions e.g. thyrotoxicosis, increased carbohydrate and salt dietare known to cause or precipitate it. A case of a 26 years age male is presented here who reported with sudden onset of muscle weakness with concomitant hypokalemia after a high carbohydrate diet. The patient's paralysis resolved upon replacement of potassium. Periodic Paralysis must be differentiated from other causes of sudden onset paralysis, so that the proper treatment can be initiated in time to prevent complications due to this rare disease.

Key Words: *Hypokalemia, Hypokalemic Periodic Paralysis, Flaccid Paralysis.*

Case Report

Male soldier 26 years of age presented to the emergency room with sudden onset weakness of all four limbs. The patient had gone to sleep after taking a heavy dinner andwoke upnext morning with inability to move all his four limbs. The weakness involved both proximal and distal muscles. There was no respiratory or swallowing difficulty. He denied any muscle pain and there were no sensory symptoms. Prior to this episode, the patient had been healthy and denied any recent diarrhea, sore throat, vaccination, shortness of breath, palpitation, weight loss or heat intolerance. There was no history of psychomotor illness. Two years ago he had experienced a similar episode of muscle weakness for which he was admitted in hospital and recovered completely after potassium replacement. Duration of attack both times was about 4-6 hours and both times it occurred after a carbohydrate-rich meal. He did not take any medications and denied use of alcohol. There was no family history of neuromuscular disease.

On examination, BPwas 130/80 mmHg, pulse74/ minute, RR14/minute, temperature 99F. Neurological examination revealed flaccid paralysis of all limbs with power 2/5. Deep tendon reflexes were overall diminished and plantars were normal. There was no sensory loss, cranial nerve involvement, bladder/bowel or higher function disturbance. There were no signs of hyperthyroidism and rest of the systemic examination was un remarkable.

Baseline investigations such as blood complete picture, urinalysis, random blood sugar, serum urea, serum creatinine and liver enzymes were all normal. Serum electrolytes revealed hypokalemia with a potassium level of 3.2 mmol/l; however serum sodium, calcium, magnesiumand phosphate levels were normal. Creatinine phosphokinase(CK) levels, thyroid profile, serum Aldosterone / Renin and 24 hour urinary sodium and potassium levels were also sent and were subsequently found to be normal. The patient was meanwhile managed in ICU with potassium replacement and cardiac monitoring. Within few hours his symptoms resolved completely and muscle power was 5/5. A repeat electrocardiogram revealed a normal sinus rhythm and rate with normal T waves.

Based on the typical presentation and the dramatic response to potassium replacement, a clinical diagnosis of hypokalemic periodic paralysis (HypoKPP) was made. Considering the nature of his job, he was counseled in detail regarding trigger identification, such as avoiding carbohydrate-rich meals, long distance travel and strong physical exercise, in order to prevent future attacks.

Discussion

Hypokalemic paralysis is characterized by episodes of acute muscle weakness associated with hypokalemia.[1] Possible etiological factors include

[1]*Department of Medicine*
63 Medical Battalion, Multan Cantt
[2]*Department of Medicine*
Islamic International Medical College
Riphah International University, Islamabad

Correspondence:
Maj. Dr. Abidullah Khan
63 Medical Battalion
Multan Cantt
E-mail: drabid2424@yahoo.com

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decreased potassium intake, urinary losses (e.g. primary/secondary aldosteronism, cushing's syndrome, diuretics, hypomagnesemia, renal tubular acidosis etc), GI losses (e.g. diarrhea, vomiting etc.), and potassium shift into the cells (e.g. by insulin, alkalosis or periodic paralysis). Hereditary defects causing hypokalemia are Bartter's syndrome and Gitelman syndrome. In this case, secondary causes of hypokalemia and thyrotoxicosis were sequentially ruled out on the basis of clinical evaluation and battery of laboratory investigations. Although desirable, NCS/EMG studies were not available at our facility. Hencea clinical diagnosis of HypoKPP was made on the basis of typical presentation and recurrent episodes triggered by high carbohydrate diet.

HypoKPP is a genetic disorder/channelopathy in which affected individuals (majority of them young males) may experience episodes of acute flaccid paralysis with concomitant hypokalemia. The paralytic attacks are reversible, usually leading to paraparesis or quadriparesis. They can last several hours and sometimes days. Some individuals have only one episode in a lifetime; more commonly, crises occur repeatedly. The major triggering factors are carbohydrate-rich meals and rest after strenuous exercise; hence symptoms frequently occur early morning on awakening. Other triggers include stress, viral illness, fatigue, and medications such as beta-agonists, insulin or steroids. HypoKPP is caused by mutations in the genes that control the development and function of certain ion channels in the muscle membrane.[2] Although the serum potassium level is often low, other electrolytes usually remain normal. Infact, total body potassium is actually normal with the change in the serum level merely reflecting an acute shift of potassium into cells.[3] Many variants are recognized including thyrotoxic periodic paralysis (TPP) which is mainly observed in Asians and familial periodic paralysis.[1-5] They are of sudden onset and sometimes even with life-threatening respiratory failure. As this is primarily a problem with muscle contraction rather than nerve conduction, tendon reflexes are diminished but sensation is intact. Electrocardiographic changes are common, but changes do not correlate well with the measured serum level. Diagnosis in between the paralytic episodes is difficult as the patient will have normal strength and potassium levels. Electromyography may reveal abnormalities in some patients but is often normal especially in between the episodes.[2-5]

Management of HypKPP includes potassium replacement and identifying and avoiding triggers of attack. Potassium may be replaced orally or intravenously depending on potassium levels and clinical condition. When given intravenously the rate of potassium administration should not exceed 20 mmol/hour. To calculate the amount of potassium supplementation one should have an estimate of potassium deficit. On average a reduction of serum potassium by 0.3 mmol/L suggests a total body deficit of 100mmol/L.[2,4-6] Biochemical parameters including electrolytes such as potassium, sodium, magnesium, calcium and phosphate, ECG and cardiac rhythm monitoring are essential components of management of HypoKPP. Although completely reversible on potassium replacement in the majority of the affected individuals, some of the patients with HypoKPP may develop myopathy leading to exercise intolerance in later life; others may be at increased risk of post anaesthetic weakness.

Conclusion

A high index of suspicion is required for the timely diagnosis of HypoKPP in any patient presenting with acute flaccid paralysis. Identifying and avoiding triggers of attack are integral component of the management of HypoKPP.

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