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Research Article

CASE REPORT: HYPOKALEMIC PERIODIC PARALYSIS

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ABSTRACT

Hypokalemic Periodic Paralysis is one form of Periodic Paralysis, a rare group of disorders that can cause a sudden onset muscle weakness. Case of a 22 year old male is presented here. The patient was presented with weakness in lower limbs and difficulty in walking and standing. Laboratory evaluation revealed a markedly low potassium level. After normalizing patient's low potassium levels, his paralytic symptoms resolved and he was discharged with no neurologic deficits. Although rare, Periodic Paralysis must be differentiated from other causes of weakness and paralysis so that the proper treatment can be initiated quickly.

Key Words:

Familial, Hypokalemia, Periodic Paralysis,
Muscle Weakness, Potassium Infusion.

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INTRODUCTION

Periodic paralysis (PP) is a rare neuromuscular disorder related to a defect in muscle ion channels, characterized by episodes of painless muscle weakness, which may be precipitated by heavy exercise, fasting, or high-carbohydrate meals. It can occur in association with low serum potassium levels.¹ There are several types of Periodic Paralysis associated with metabolic and electrolyte abnormalities. Of these, Hypokalemic Periodic Paralysis (HPP) is the most common with a prevalence of 1 in 100,000.²

Hypokalemic periodic paralysis is the most predominant type of periodic paralysis, a rare channelopathy characterized by abnormal sarcolemmal excitability followed by episodic flaccid weakness. Hypokalemic paralysis may be caused by a short term shift of potassium into cells (caused by familial periodic paralysis or thyrotoxic periodic paralysis), or a larger deficit of potassium as a result of severe renal or gastrointestinal potassium loss.³

CASE REPORT

A 22 year old male, admitted to the casualty department presented with complaints of weakness in lower limbs for a few hours before admission. Patient complained of difficulty in standing and walking. The patient was slightly overweight with no history of similar illness in past or in family. There was no history of trauma, Diabetes mellitus or intake of any drugs prior to this illness.

On examination, the patient was conscious and oriented with BP 130/90 mm Hg, pulse rate 98 beats/minute, respiratory rate 20 breaths/minute and SPO₂ 99%. There was no sensory deficit. Pupils had normal size and reacted to light. Abdomen was soft and bowel sounds were present. Neurological examination was done. His muscle power was 2/5 in both lower limbs, his upper limbs power were normal and had no sensory deficit.

A complete blood count reveals declined levels of lymphocytes in differential counts as 13 % and absolute counts as 767 cells/cumm. Routine chemistry report shows elevated levels of glucose random {192 mg/dl} and declined level of potassium 2.4 mg/dl.

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Inj. KCl supplementation was given to the patient. With improvement in potassium levels, the symptoms started recovering. On the next day of admission the patient was transferred to the ward. He was treated with IV fluids, antacids, potassium infusion, and other supportive measures. Patient was followed up for one month. He had no episodes of muscle weakness and was advised to take potassium rich diet.

DISCUSSION

Differential diagnosis of acute onset of muscle weakness is extensive and it includes neurologic, metabolic and infectious causes. HPP may occur sporadically in the form of Familial Hypokalemic Paralysis (FHP), a poorly understood disorder which may occur spontaneously or as the result of autosomal dominant inheritance.⁴ This type of periodic paralysis is the result of disordered cellular potassium regulation perhaps due to sodium or calcium channel abnormalities. Mutations of the CACNA1S and SCN4A genes have been identified that cause abnormalities in sodium channels resulting in abnormal potassium ion flux.⁵

Though the serum potassium level is often alarmingly low, the other electrolytes remain normal. The most probable cause in the present case was metabolic i.e. hypokalemia. The patient was 22 years old and hypokalemic paralysis is more common among young adults. An acute paralytic episode was treated with potassium replacement and the patient recovered quickly. A diagnosis of hypokalemic paralysis should always be considered if a patient having hypokalemia presents with quick onset, are flexic, pure motor weakness in one or more limbs, without a change in consciousness or sphincter functions.⁶ Both, age of onset and response to potassium chloride therapy supports this diagnosis.

The patient was not identified with history of similar episodes. This paralytic attack can be prevented by controlling plasma potassium levels, avoidance of large glucose and salt loads (which promote intracellular shift), and maintenance of body temperature, acid-base balance, and cautious use of neuromuscular blocking agents.⁷ The specific treatment of hypokalemic paralysis is oral potassium supplementation, repeated at 15-30 minute interval, depending on the serum potassium level, and muscle strength.⁸

CONCLUSION

When a patient without a history of other underlying diseases presents with complaints of weakness or paralysis, periodic paralysis should be considered. It is associated with variations in serum potassium levels and if left untreated it can become more severe and fatal. Low potassium levels should be corrected rapidly with potassium infusion which in turn would resolve the symptoms. Preventive measures should be taken to reduce the recurrence of paralytic attacks.

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