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Unraveling the Diverse Etiologies of Hypokalemia- A Case Series of Hypokalemic Periodic Paralysis

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ABSTRACT:

On a regular basis, a physician would come across a number of patients presenting with muscular weakness of different severities. Among the causes of the muscular weakness, hypokalemia remains as one of the important causes. This condition is called Hypokalemic Periodic Paralysis. It is a neurological manifestation of hypokalemia and it is completely reversible with appropriate management. Hence, identifying the cause of hypokalemia in each individual at the time of presentation is very important to make a diagnosis, treat accordingly and prevent the further episodes. In this article, three cases of hypokalemic periodic paralysis are presented with typical muscular weakness caused by hypokalemia which in turn is due to different causes and are recovered by giving potassium correction.

Keywords: Hypokalemia, Paralysis, Hypokalemic Periodic Paralysis, Potassium.

1. Introduction

Hypokalemia is a condition when potassium level in bloodstream is lower than the normal (<3.5mEq/l). This manifests with different signs and symptoms such as muscle twitches, muscle paralysis, hypotension, arrhythmias, excessive urination, and excessive thirst. Hypokalemic Periodic Paralysis (HPP) is a neurological manifestation of hypokalemia. The tendency to rapidly shift between the intracellular and extracellular compartments makes potassium ions vulnerable to changes. It is a rare disorder of channelopathy with a prevalence of 1 in 100,000. It is characterized by the sudden onset of muscle paralysis. Males are most commonly affected than females. Heavy carbohydrate meals, stress, infection, glucose infusion, steroids, alcohol use, hypothermia, and strenuous exercise can trigger this condition. Periodic paralysis refers to short-lived, episodic hyporeflexia with or without myotonia. The episodes can last from a few minutes to several days, with spontaneous recovery. It can be primary or secondary to other causes. The calcium or sodium ion channel mutation such as CACNA1S, SCN4A, and KCNJ2 can cause primary HPP. Secondary HPP is associated with hyperthyroidism, renal tubular acidosis, Gitelman syndrome, Barters syndrome, primary hyperaldosteronism and hypokalemia secondary to gastrointestinal losses. In this article a series of three cases of hypokalemic periodic paralysis caused by autoimmune disorder (Sjogren's syndrome), endocrine disorder (thyrotoxic periodic paralysis), and renal system involvement (Gitelman syndrome).

Case 1:
A 32-year-old female presented to the emergency with c/o weakness of both upper and lower limbs for the past 4 days. It was insidious in onset and progressive in nature. There was no history of diarrhoea, vomiting. Patient gave a history of dryness of eyes from the past 6 months, for which she is on topical lubricant. Patient also gave a history of joint pain for the past 4 months. She had a history of similar episode one year ago and She gave a family history of her mother having similar complaints. She had no h/o DM, HTN, COPD, CKD. Clinical examination revealed stable vitals, CNS examination revealed LMN type of quadriparesis with power of 2/5 in all four limbs with normal higher mental funtion. Laboratory investigation – normal blood sugar, normal HbA1c, serum calcium – 9.8 mg/dL. ECG showed prominent U wave with flattening of T wave in all leads. On ABG analysis, pH was 7.3 with normal anion gap, PCO2 -35.1, PO2 -72, serum Na+ -138 mmol/l, serum k+ - 2.6 mmol/L, serum bicarbonate 14.7 mmol/L, and serum chloride 111 mmol/L. Urinary pH was 7.5 with positive anion gap. Serum magnesium was 1.8 mg/dL suggestive of RTA. Tear break up time test revealed severe dry eye of right eye(<5s) and moderate dry eye of left eye(7s), Schirmer test was positive. Thyroid profile was normal and Sickling test was negative. Ultra sonogram of abdomen and pelvis showed no abnormality. Serological test-ANA-positive, RA factor +, SSA + + +, SSB + +, anti Ro + +, with negative anti CCP. Patient was given potassium, bicarbonate supplementation, a provisional diagnosis of Sjogren's syndrome was made and the patient was started on treatment after which he showed recovery.

Case 2:
A 24-year-old male came to the hospital with complaints of weakness in all four limbs for the past five days. It was an acute presentation involving the lower limbs first and later rapidly progressed involving the upper limbs within 24 hours. There was no sensory or bowel and bladder involvement. The patient had a history of significant weight loss of about 10kgs over the past three months and also had a history of palpitations. There was no history suggestive of similar complaints in the past. On examination, the patient was conscious and oriented.Pulse rate showed tachycardia of 124 beats per minute while other vitals were normal. Neurological

examination revealed power of 3/5 in lower limbs and 4/5 in upper limbs. Deep tendon reflexes were depressed. ECG showed sinus tachycardia, ST depression and "u" waves. Blood investigations revealed hypokalemia(serum K+ - 2.9) while renal and liver parameters were normal. As the patient had history of weight loss, palpitations and ECG showed sinus tachycardia TFT was done which showed low TSH(0.32 μIU/mL) and elevated T3(1.85 ng/ml) and T4(22.31μg/dL) values suggestive of hyperthyroidism. USG neck was done which revealed diffuse enlargement of the gland with increased vascularity. Patient was given potassium correction after which he showed improvement. He was also started on beta blockers and carbimazole to treat thyrotoxicosis.

Case 3:
A 47-year-old male presented to the casualty with c/o weakness of bilateral lower limbs for the past four days, which was insidious in onset and gradually progressive which later involved the bilateral upper limb. He had a history of working under the sun for the past five days. Patient had similar episode 6 months ago. He had no h/o DM, HTN, COPD, CKD. On physical examination, the patient was conscious, oriented, afebrile and his vitals were stable. Systemic examination revealed quadriparesis with a power of 3/5 in all four limbs, diminished deep tendon reflexes, and sensory examination was normal. ABG was done, which showed metabolic alkalosis, pH was 7.47, PCO2-43.4, PO2-85.4, serum Na+ -138 mmol/l, serum k+ - 2.6 mmol/L, serum bicarbonate - 33 mmol/L, and serum chloride - 96 mmol/L. The patient was started on potassium correction, after which his weakness improved. Pseudohypokalemia was ruled out, and evaluation for hypokalemia was initiated. He had urinary potassium loss of 22meq/l, and TTKG (trans tubular K+ gradient) was elevated. The urinary calcium and creatinine ratio was <0.15 and as the patient was not on diuretics, a possible diagnosis of Gitelman syndrome was made.

2. Discussion and Conclusion

Muscle weakness is usually thought to be due to neuronal damage, but it can also be caused by a few systemic abnormalities and hypokalemia is one of them. The causes of hypokalemia can be divided into three main groups-potassium depletion by renal causes, potassium depletion by extra renal causes, and potassium shift into cells. Renal causes include increased aldosterone, hypomagnesemia, renal tubular acidosis (Type I and II), metabolic alkalosis, and Liddle's syndrome. Extra renal causes include decreased intake, vomiting, diarrhea, Zollinger-Ellison Syndrome and fistulas. Intracellular shift of potassium is caused by increased insulin, alkalosis, hyperthyroidism, thyrotoxic periodic paralysis, and familial hypokalemic paralysis. There are various manifestations of hypokalemia such as muscle twitches, muscle cramps, muscle paralysis, hypotension, arrhythmias, excessive urination, and excessive thirst.
The evaluation for the cause of Hypokalemic periodic paralysis should begin from the time of presentation of a patient to the clinic. A proper history taking helps in making a clinical suspicion. Periodic paralysis is suspected when patient presents with acute onset flaccid weakness involving proximal muscles with decreased or normal deep tendon reflexes. Thorough neurological system examination and ABG, serum electrolytes, urine analysis forms an integral part in establishing a diagnosis. This case series signifies the importance of evaluating the various causes of hypokalemia to promptly start on appropriate treatment and prevent recurrence.

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