Case Report

Hyperkalemic paralysis in primary adrenal insufficiency

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Abstract

Hyperkalemic paralysis due to Addison's disease is rare, and potentially life-threatening entity presenting with flaccid motor weakness. This case under discussion highlights Hyperkalemic paralysis as initial symptomatic manifestation of primary adrenal insufficiency.

Keywords: Hyperkalemic paralysis, primary, adrenal insufficiency

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Introduction

Periodic paralyses are a heterogeneous group of muscle disorders characterized by episodic weakness. Besides the hereditary hyperkalemic paralysis, a secondary form exists and it occurs most frequently in renal insufficiency, Addison's disease and with certain medications such as angiotensin converting enzyme inhibitors, potassium sparing diuretics, nonsteroidal anti-inflammatory drugs, etc., We report a case of secondary hyperkalemic paralysis (SHPP) in a patient with primary adrenal insufficiency.

Case Report

A 60-year-old female with 2 years history of hypothyroidism presented to our emergency department with the complaints of dry cough and fever for 3 days and severe weakness of all limbs for 3 h before presenting to the hospital. The history of presenting complaints revealed that for last 7-8 months she had experienced occasional brief episodes of limb weakness, which tends to occur at rest following exertion. For the last 3 days, the patient experienced increasing episodes of weakness and along with her meals she had also consumed approximately 200 ml coconut water per day. There was no history of recent animal bite, illicit drug or alcohol abuse. She had no past medical history of hypertension, diabetes mellitus, tuberculosis, and chronic kidney disease. Her current medications included tablet thyroxine 100 mcg before breakfast.

On presentation, she was afebrile. Her heart rate was 80/min, respiratory rate was 20/min, systolic blood pressure was 86 mmHg, and oxygen saturation of 100% on room air. Physical examination revealed hyperpigmentation of the palmar creases and the knuckles and patchy hyperpigmentation of the oral mucosa [Figures 1 and 2]. Neurological examination revealed fully intact mental status, 1/5 power in both upper and lower extremities on the medical Research Council scale and diminished deep tendon reflexes in all extremities. Superficial, deep, and cortical sensations were intact and cranial nerves; fundoscopy was normal. Examination of abdomen, cardiovascular, and respiratory systems were unremarkable.

The initial laboratory tests showed serum sodium of 123 mmol/L, serum potassium 10.4 mmol/L, serum chloride 103 mmol/L, serum creatinine 0.89 mg/dL,

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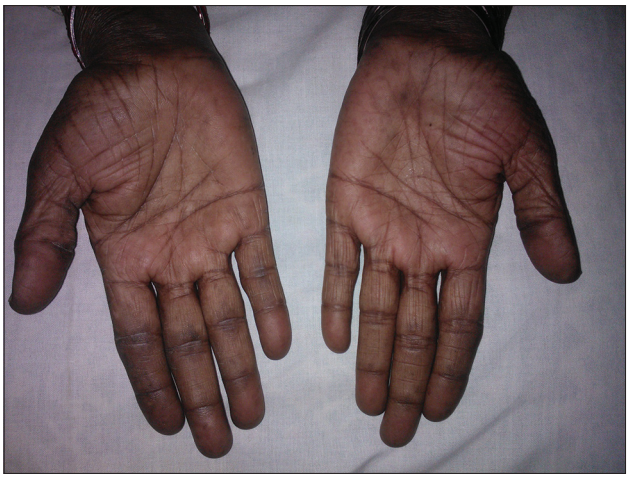

Figure 1: Hyperpigmentation of the palmar creases

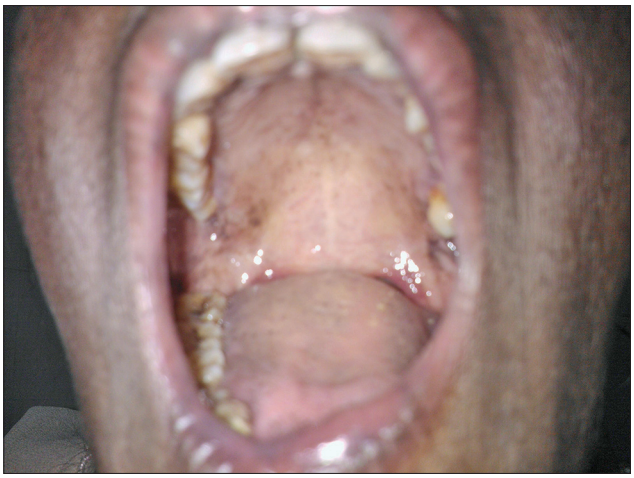

Figure 2: Patchy hyperpigmentation of the oral mucosa

and random blood sugar 99 mg/dL. Arterial blood gas analysis showed metabolic acidosis with high anion gap: pH 7.29, PCO_2 24, PO_2 82, HCO_3 12, anion gap 20. Electrocardiogram (ECG) showed tall and peaked T-waves. Transtubular potassium gradient value was 5.1. Other hematological and biochemical tests, including complete blood counts, erythrocyte sedimentation rate, urine analysis, serum calcium, serum magnesium, creatine phosphokinase, thyroid and liver function tests were all within the normal limits. Hepatitis B surface antigen, hepatitis C virus, and human immunodeficiency virus serology was negative. Chest X-ray and abdominal ultrasound examination did not reveal any abnormality.

Hypotension, hyperpigmentation, hyponatremia, and hyperkalemia suggested adrenal crisis due to acute stress in long standing primary adrenal insufficiency. She was treated with normal saline, antibiotics, 10 ml of 10% calcium gluconate intravenously, 10 mg of nebulized salbutamol, slow intravenous injection of 10 units of regular insulin added to 50 ml glucose 50% and 100 mg intravenous bolus of hydrocortisone every 6 hourly. Her limb power improved to 4/5 in few hours. After 6 h serum sodium was 126 mmol/L, potassium 7.1 mmol/L, bicarbonate 17 mmol/L, and ECG abnormalities resolved. Intravenous hydrocortisone was tapered over 3 days and replaced with long acting glucocorticoid prednisolone in replacement dosage. Her serial serum potassium and sodium levels after steroids replacement returned to normal level. Adrenal function test results showed: Baseline cortisol 2.87 mcg/dL (normal level 6-26 mcg/dL), increasing to 3.01 mcg/dL (normal > 20 mcg/dL) 60 min after short synacthen test. Autoantibody screening showed positive thyroid autoantibodies. Adrenal autoantibodies against adrenal cytoplasm, 21-hydroxylase, 17-alpha hydroxylase and side chain cleavage enzyme could not be done.

Discussion

Hyperkalemic paralysis is divided into primary and secondary forms. Primary hyperkalemic periodic paralysis (PHPP) has an autosomal dominant inheritance pattern, and occurs due to mutations in the sodium channel gene in chromosome 17q23-25. Episodes of PHPP usually begin in the first decade of life, often precipitated by fasting or rest after exercise, unlike in hypokalemic periodic paralysis. Affected individuals have multiple brief episodes of paralysis due to episodes of hyperkalemia.[1,2] SHPP occurs in acute and chronic renal dysfunction, Addison's disease, rhabdomyolysis, excessive ingestion of potassium and drugs such as angiotensin converting enzyme inhibitors, potassium sparing diuretics, nonsteroidal anti-inflammatory drugs, cotrimoxazole, etc., Renal impairment is the most common cause, followed by Addison's disease and medications.[3]

Review of the literature revealed several case reports of SHPP associated with adrenal insufficiency. In their study Sowden and Borsey have reported hyperkalemic periodic paralysis as initial symptomatic manifestation of Addison's disease in a patient with diabetes mellitus.[4] Hyperkalemic paralysis in established cases were attributed to inadequate steroid replacement or excess administration of potassium supplements.[5-9]

Increased intake of potassium, from either exogenous or endogenous sources, can result in hyperkalemia, but the rise in plasma potassium concentration is usually transient unless an underlying defect in renal potassium excretion is present as well.[10] The persistence of

hyperkalemia in the face of an increased potassium load should suggest an underlying defect in the potassium homeostatic system. One must consider the possibility of either a defect in the renin-angiotensin-aldosterone axis or a primary tubular potassium secretory defect. Impaired potassium secretion in the presence of an intact renin-aldosterone axis is evidence of a renal tubular defect in potassium secretion. In the patient under discussion, hyperkalemia, hyponatremia, hyperpigmentation, and abnormal short synacthen test suggested long standing primary adrenal insufficiency and hyperkalemia periodic paralysis as initial symptomatic manifestation.

Although renin and aldosterone levels were not measured, the possibility of hyporeninaemic hypoaldosteronism is unlikely because this patient had normal chloride levels, abnormal adrenal stimulation tests, good neurological recovery and normalization of potassium level following treatment with prednisolone 5 mg and fludrocortisone 0.1 mg daily.

Conclusion

Clinicians should be aware of this rare, but potential serious initial manifestation of Addison's disease.

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