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CASE STUDY REPORT

A CASE OF HYPERKALEMIC PARALYSIS SECONDARY TO RENAL TUBULAR ACIDOSIS TYPE 4

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ABSTRACT

AIMS: To identify the rare presentation of hyperkalemic paralysis in type 4 RTA
OBJECTIVES OF THE STUDY: 1. to identify rare presentation. 2. Early identification and management
METHODS: A 60 year old female patient diabetic, hypertensive, IHD came with sudden onset of weakness of both upper and lower limbs, on examination vitals stable, ecg suggestive of hyperkalemic changes, cns examination power of both upper and lower limb was 3\5, deep tendon reflexes were sluggish and sensory system was intact.
RESULTS: after through clinical examination and investigation patient was diagnosed as TYPE 2 DM, HTN IHD WITH. HYPERKALEMIC PARALYSIS SECONDARY TO RTA TYPE4
CONCLUSION: hyperkalemic paralysis is a rare presentation in adults with type4 RTA. Till date there are very less data available in the literature.

INTRODUCTION

Renal tubular acidosis (RTA) is a constellation of syndromes arising from different derangements of tubular acid transport. It often presents as renal stone disease with nephrocalcinosis, ricket/osteomalacia and growth retardation in children with ultimate short stature in adulthood and hypokalemic paralysis, primary hypothyroidism. But distal RTA presenting as a hyperkalemic paralysis (as in our case) is a rare occurrence, so far other cases of distal renal tubular acidosis (dRTA) have been reported.

Type 4 results from aldosterone deficiency or unresponsiveness of the distal tubule to aldosterone. Because aldosterone triggers Na resorption in exchange for K and hydrogen, there is reduced K excretion, causing hyperkalemia, and reduced acid excretion. Hyperkalemia may decrease ammonia excretion, contributing to metabolic acidosis. Urine pH is usually appropriate for serum pH (usually < 5.5 when there is serum acidosis). Plasma HCO_3 is usually > 17 mEq/L. This disorder is the most common type of RTA. It typically occurs sporadically secondary to impairment in the renin-aldosterone-renal tubule axis.

METHODS

A 60-year-old female patient presented to the emergency department (ED), with history of sudden onset of paresis, progressively in nature, worsening, generalized muscle weakness, inability to walk without support form four days. She denied having any fever, chronic diarrhea, headache, abdominal Pain, paresthesias, sensorineural deafness or any other drug Intake. She was known to have type 2 diabetes mellitus, hypertension and ischemic heart diseases

On physical examination, she was afebrile. Her pulse was regular with a rate of 40/min. Her blood pressure was 130/80 mm Hg, and had a respiratory rate of 22 breaths/min. She appeared uncomfortable and generally fatigued, but was alert and oriented. Her lungs were clear on auscultation. The heart rhythm was regular, no murmur, rub or gallop. Her abdomen was nontender no organomegaly, no palpable mass felt, no edema and all peripheral pulse felt. Neurologic examination revealed muscle power of 3/5 in upper and 2/5 in lower extremities. Cranial nerves II-XII were normal and symmetric. Bilateral plantor flexor. Dtr reflexes are diminised An electrocardiogram (ECG), urine analysis and basic metabolic panel were completed soon after arrival in ED. Her ECG was suggestive of hyperkalemic

changes. Initial laboratory testing revealed metabolic acidosis in abg, rbs 279mg/dl urea – 65.8mg/dl creat – 1.6mg/dl electrolytes na – 126.5mmol/l ka – 8.60mmol/l urinary ph was 5 and she was treated with calcium gluconate, insulin dextrose infusion, salbutamol nebulisation and long term treatment with tab fludrocortisone 0.1 mg od

DISCUSSION

To begin, we need a definition and differential diagnosis for hyperkalemic (type IV) renal tubular acidosis (RTA).

Inability of the kidney either to excrete sufficient net acid or to retain sufficient bicarbonate results in a group of disorders known as RTAs. These all are normal anion gap hyperchloremic acidoses; in their traditional classification, type IV refers to the only variant associated with hyperkalemia. Unlike other distal RTAs, the collecting duct here fails to excrete both protons and potassium .Such a situation arises when aldosterone is insufficient in either quantity or activity and/or because of some intrinsic (genetic) or acquired molecular defect in relevant transporters . Sufficiency of aldosterone is both quantitatively and functionally necessary for adequate sodium reabsorption by the epithelial sodium channel (ENaC) located on the luminal surface of principal cells in the terminal portions of the nephron, which under normal conditions leads to the lumen-negative potential essential for potassium and proton secretion (Figure 1A). In addition, aldosterone has a direct, Na independent, non genomic effect on proton secretion through up regulation of apical proton pumps on intercalated cells, in rodents at least.

Low levels of aldosterone or tubular unresponsiveness to this hormone are present in the majority of patients with hyperkalemia and impaired renal function before end stage.4,5 The most common medical conditions associated with hypo renin hypoaldosteronism include diabetes and various forms of interstitial disease, including amyloid, monoclonal gammopathies, and particularly the interstitial nephritis associated with non steroidal anti-inflammatory agents. In the last case, renin levels may be normal, and some patients with diabetes fail to respond with aldosterone synthesis or release despite hyperkalemia. Other situations in which hypoaldosteronism is present but not matched by hyporeninism include adrenal destruction (whether surgical, malignant, or hemorrhagic), Addison disease, angiotensin-converting enzyme inhibitor therapy or angiotensin receptor blockade ,critical illness (because of direct adrenal suppression), and inhibition of aldosterone synthesis by heparin. Hyporeninemic hypoaldosteronism is also predictable with _ blockade. Hyperkalemia, which is a rare occurrence in normal subjects, is the commonest electrolyte manifestation in renal impairment, and symptoms and signs mainly limited to cardiac and neurological systems. However, neurological manifestations are not commonly encountered in clinical practice. According to the case review of 73 patients common presentations of secondary hyperkalemic paralysis were diminished reflexes, quadriparesis/paralysis, respiratory involvement and sensory loss. Complete recovery was achieved in 89% of cases and did not correlate either with the absolute potassium level or the degree to which it was corrected. We report a case of hyperkalemic paralysis with pure motor involvement in a patient with renal involvement Hyperkalemia is defined as serum potassium concentration of greater than 5.5mEq/L. Renal impairment is the major cause of

hyperkalemia, accounting for 75% of cases [2]. Hyperkalemia is often asymptomatic and diagnosed on routine laboratory test. When symptoms are present, they are non specific and predominantly cardiac and neurological. Nevertheless, neurological manifestations are sparsely reported in the literature. Although, ECG is a useful tool to diagnose hyperkalemia prior to laboratory evidence, it is not a very sensitive method. Various studies showed poor correlation between ECG changes and serum potassium level . In a study by Acker and colleague nearly half the patients with serum potassium more than 6 mEq/L did not have ECG changes. Furthermore, some patients showed gradual progression in changes, others showed progression from benign to more fatal arrhythmias without warning. However, hyperkalemia was clinically suspected in this case by ECG on admission. Chronicity of the process may also have an influence on symptom and signs. Pre treatment potassium level of this patient was 10.1 mEq/L, and he did not have lethal arrhythmias. Most common neurological manifestation in hyperkalemia is ascending quadriparesis or quadriplegia with diminished or absent reflexes as in this case. Respiratory failure, sensory loss, bulbar weakness and myalgias could also occur . The serum potassium concentration leading to hyperkalemic paralysis ranged from 7.0 to 11.2 mEq/L with a mean of 9.0 mEq/L . However some cases have been reported with potassium level between 5 mmol/l and 7.0 mmol/l . This highlights lack of consistent correlation

between the serum potassium level and the severity of the weakness. Chronic renal failure is the commonest underlying cause for hyperkalemia as in our patient. Other causes reported in the literature are exogenous potassium supplementation, medications, Addison disease and rhabdomyolysis . The mechanism of secondary hyperkalemic paralysis is unclear.The weakness has been attributed to abnormalities at various locations in the motor axis, including the muscle, nerve and neuromuscular junction. Nerve conduction studies showed decreased nerve conduction velocities and increased F wave latencies, but Electromyography (EMG) studies were normal. On the basis of nerve conduction studies, abnormal depolarization of the nerve membrane described as a possible cause of secondary hyperkalemic paralysis; summary, although hyperkalemic paralysis is not a common occurrence, clinicians should be aware of the potentially life threatening neurological complication of hyperkalemia.

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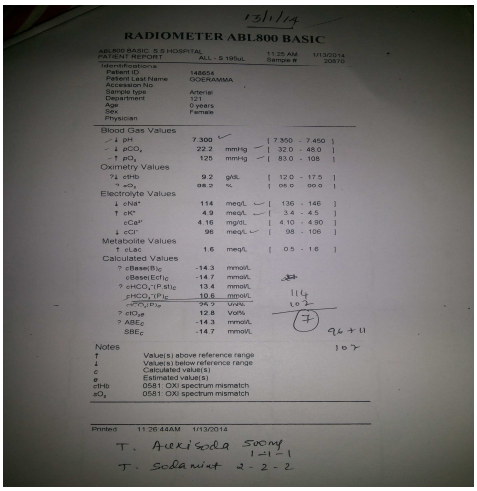
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