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Case Report

Hypokalemic paralysis due to distal renal tubular acidosis type 1 in patient of mucormycosis on amphotericin B: a case report

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ABSTRACT

A 42-year-old male patient who is a known case of DM and mucormycosis on treatment presented with sudden onset difficulty in moving all 4 limbs followed by decreased depth of respiration for 4 hours. The patient was known case of DM for 10 years and was on OHA for the same, he had history of biopsy diagnosed rhino mucormycosis 4 months ago and was on treatment for the same. On initial examination the tone was hypotonic in all4 limbs along with power of 3+, respiration was shallow and patient was bedridden unable to stand on his own, he was ambulatory 6 days before presenting to hospital. Potassium-1.7 mEq/l, ABGA pH-7.18, HCO_3-10 Meq/l, urine osmolality 220 mOsm/l, urine pH-7.0, potassium-to-creatinine rstio (K/Cr)-3.9 mEq/ml, urine K-22 mEq/ml. Distal RTA (dRTA) is the classical form of RTA, being the first described. Distal RTA is characterized by a failure of H+ secretion into lumen of nephron by the alpha intercalated cells of the medullary collecting duct of the distal nephron. This failure of acid secretion may be due to a number of causes, and it leads to an inability to acidify the urine to a pH of less than 5.3. This case study enumerates the potentially dangerous side effects of amphotericin B in patients which can precipitate RTA type 1 leading to severe hypokalaemia and acidosis, thus all patients receiving amphotericin B should be cautiously warned regarding side effect of hypokalaemia and prophylactic potassium syrup supplementation may be given in predisposed patients.

Keywords: Distal renal tubular acidosis, Hypokalaemia paralysis, Amphotericin B

INTRODUCTION

Renal tubular acidosis (RTA) is a medical condition that involves an accumulation of acid in the body due to a failure of the kidneys to appropriately acidify the urine. In renal physiology, when blood is filtered by the kidney, the filtrate passes through the tubules of the nephron, allowing for exchange of salts, before it drains into the bladder as urine. The metabolic acidosis that results from RTA may be caused either by insufficient secretion of hydrogen ions (which are acidic) into the latter portions of the nephron (the distal tubule) or by failure to reabsorb sufficient bicarbonate ions (which are alkaline) from the filtrate in the early portion of the nephron (the proximal tubule).[1] Clinically, patients may present with vague symptoms such as dehydration, mental status changes, or delayed growth in adolescents. The metabolic acidosis caused by RTA is a normal anion gap acidosis.[2]

This study presents a curious presentation of RTA in the form of hypokalaemia paralysis and shallow respiration in the patient.

CASE REPORT

A 42-year-old male patient who is a known case of DM and mucormycosis on treatment presented with sudden onset difficulty in moving all 4 limbs followed by decreased depth of respiration for 4 hours. The patient was known case of DM for 10 years and was on OHA for

the same, he had history of biopsy diagnosed rhino mucormycosis 4 months ago and was on treatment for the same.

On initial examination the tone was hypotonic in all 4 limbs along with power of 3+, respiration was shallow and patient was bedridden unable to stand on his own, he was ambulatory 6 days before presenting to hospital.

The following positive lab reports were obtained:

Table 1: Lab reports.

Investigations	As seen in current case report	Normal reference range[3]
S. potassium (mmol/L)	1.7	3.5-5
S. Chloride (mmol/L)	122	96-106
ABGA pH	7.08	7.35-7.45
ABGA HCO_3 (mmol/L)	10	22-26
Urine osmolality (mOsm/kg)	220	500-850
Urine pH	7.2	4.5-7
Potassium-to-creatinine ratio (urine)	3.9	0.6-2.3
Urine potassium (mEq/ml)	62	<20

Diagnosis

Based on the clinical examination showing weakness of all 4 limbs hyporeflexia, decreased depth of respiration and the lab values of potassium less than 2 mEq/ml suggesting severe hypokalaemia, a diagnosis of transient hypokalaemia paralysis was made. There was also drastic improvement in symptoms after potassium supplementation.

Based on the urine examination showing increased fractional excretion of potassium along with ABGA findings suggestive of acidosis, with alkaline urine pH led to the diagnosis of RTA 1 (due to the severity of acidosis and hypokalaemia RTA 2 proximal counterpart was excluded).

On elaborating patients' history, we were currently on Amphotericin B injections for treatment of mucormycosis.

Thus, a diagnosis of Amphotericin B induced RTA 1 with transient hypokalemic paralysis was made.

DISCUSSION

Distal RTA (dRTA) is the classical form of RTA, being the first described. Distal RTA is characterized by a failure of H+ secretion into lumen of nephron by the alpha intercalated cells of the medullary collecting duct of the distal nephron.

This failure of acid secretion may be due to a number of causes, and it leads to an inability to acidify the urine to a pH of less than 5.3. Because renal excretion is the primary means of eliminating H+ from the body, there is consequently a tendency towards acidemia. There is an inability to excrete H^+ while K^+ cannot be reclaimed by the cell, leading to acidemia (as H^+ builds up in the body) and hypokalemia (as K^+ cannot be reabsorbed by the alpha cell).

This leads to the clinical features of dRTA in other words, the intercalated cells' apical H+/K+ antiporter is non-functional, resulting in proton retention and potassium excretion.[4]

Since calcium phosphate stones demonstrate a proclivity for deposition at higher pHs (alkaline), the substance of the kidney develops stones bilaterally; this does not occur in the other RTA types.

Distal RTA has also been linked to specific genetic mutations in ATP6V1B1 and ATP6V0A4 will present with symptoms within the first year of life, while those with mutation of the SLC4A1 have delayed onset around 10 years of age as seen in Rodríguez study.[5]

In a large number of patients acquired causes for it are seen such as Sjogren's syndrome, primary biliary cirrhosis, SLE and various drugs most commonly amphotericin B and lithium.[6]

The treatment is usually symptomatic to counteract acidosis sodium bicarbonate is given and to overcome symptoms of hypokalaemia, iv supplementation of potassium is given keeping in mind the rate of infusion of potassium infusion should never exceed 20 mEq/hr.[7]

The Burgess study also iterates the possibility of D RTA in patients receiving amphotericin B.[8]

The Usami et al study, the aim of this retrospective study was to examine the appropriate potassium supplementation conditions to treat hypokalemia induced by L-AMB.[9] The subjects were 100 hematological patients who received L-AMB for the first time between April 2012 and March 2013. A total of seven patients were excluded. Of the remaining 93 patients, 48 (51.6%) were assigned to the group receiving supplemental potassium (supplementation group), and 45 (48.4%) were assigned to the group without potassium supplementation (non-supplementation group). Hypokalemia greater than grade 3 was exhibited by 50 of the 93 (53.8%) patients.

The McCurdy et al study showed findings in 25 normal controls, six patients with other forms of renal disease and six with RTA.[10] Five of the patients on amphotericin

B had defects in acid excretion characteristic of distal RTA. These defects could not be attributed to decreased glomerular filtration. A tubular defect in acid excretion, superimposed on an ischemic kidney, appears to underlie both the hypokalemia and the nephrocalcinosis characteristic of amphotericin nephropathy. Concomitant alkali therapy during prolonged amphotericin B administration might prove prophylactically useful.

CONCLUSION

After potassium iv supplementation patient regained movement and power in all 4 limbs and was discharged on syrup potchlor and sodium bicarb tablets and was advised to continue amphotericin B cautiously.

This case study enumerates the potentially dangerous side effects of amphotericin B in patients which can precipitate RTA type 1 leading to severe hypokalaemia and acidosis, thus all patients receiving ampho B should be cautiously warned regarding side effect of hypokalemia and prophylactic potassium syrup supplementation may be given in predisposed patients.

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