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Case Report

AN UNUSUAL CASE OF POLYURIA

Sunayana Lomte[1], Shrirang Bhurchandi[1], *Sourya Acharya[1] and Samarth Shukla[2]
[1]Department of Medicine, DMIMS University, J.N. Medical College, Sawangi (M), Wardha- 442004, Maharashtra
[2]Department of Pathology, DMIMS University, J.N. Medical College, Sawangi (M), Wardha- 442004, Maharashtra
**Author for Correspondence*

ABSTRACT
We present a case of a young adult with type 2 RTA who was presented with polyuria and polydipsia. A 23 years old male presented to us with complaints of polyuria, polydipsia and pain in abdomen for 2 months. There was no history of hematuria, polyphagia, nocturia, facial puffiness. On investigations, non-anion gap hyperchloremic metabolic acidosis was confirmed. USG KUB revealed bilateral nephrocalcinosis and right renal calculi. Diagnosis of distal tubular acidosis was confirmed by ammonium chloride challenge test.

Key Words: *Polyuria, Type 2 RTA.*

INTRODUCTION
Renal tubular acidosis is characterized by systemic acidosis due to inability of renal tubules to acidify the urine[1]. The acidosis is typically a hyperchloremic metabolic acidosis with normal anion gap (Yaqoob, 2002). There are four types of RTA depending upon which part of renal acid handling has been affected. Type 1 and type 2 may be inherited or acquired. Type 1or distal RTA characterized by hypokalemic hyperchloremic metabolic acidosis with distal nephrons failing to acidify urine leading to inability to fall the urinary PH below 5.5(Atkinson and Bourke, 1987; Masafumi *et al.*, 2004).

CASES
A 23 years old male presented to us with complaints of polyuria, polydipsia and pain in abdomen of 2 months' duration. There was no history of hematuria, polyphagia, nocturia, facial puffiness. The pain in abdomen was particularly in the lower right quadrant and also involved in right renal angle. There was no family history of diabetes, hypertension. On examination vitals were normal. There were no signs of dehydration. Abdominal examination revealed mild right renal angle tenderness.
On investigations a urine microscopy showed 3-4RBC's / HPF, urinary calcium was 400mg/day (normal less than 300mg/day), Urinary Ph- 6.0. Twenty-four hours' urine collection was more than 3.5 liters. Serum chloride 140meq/L, serum potassium 2.1mg/dl, serum sodium and serum magnesium were normal. Blood urea nitrogen 40mg%, serum creatinine 1.2mg%, serum calcium 8mg%. Fasting blood sugar levels and postprandial blood sugar levels were normal. A parathyroid hormone (PTH) profile was normal. Arterial blood gas reveled pH -7.28, PaO2- 102mmhg, PaCO2- 35mmhg, HCO3- 60mmol/L confirming non anion gap hyperchloremic metabolic acidosis.
USG KUB revealed bilateral nephrocalcinosis and right renal calculi. Diagnosis distal tubular acidosis was confirmed by ammonium chloride challenge test. Which was performed after overnight fasting, 0.1gm/kg bodyweight ammonium chloride was given orally and urine pH measured one hourly for 8 hours. Each urinary ph reading was more than 6.0.
He was treated with alkali replacement therapy 1gm NaHCO3 tablet 8 hourly, which corrected the metabolic acidosis and hypokalemia. The patients polyuira was also improved, a repeat 24 hours urine output came down to 1.8 liters.

DISCUSSION
Distal RTA (type 1) is more common than type 2 proximal RTA. Mechanism of distal renal tubular acidosis is excessive back diffusion of hydrogen ions from lumen into blood or inadequate transport of

hydrogen ions in the collecting duct (Atkinson and Bourke, 1987). Low ammonium excretion, rise in potassium excretions, decrease in tubular reabsorption of calcium causes renal hypercalciuria. The hypercalciuria, alkaline urine, low level of urinary citrate leads to development of nephrocalcinosis (Bihl and Myers, 2001).

The diagnosis of distal RTA is suggested by a hyperchloremic normal anion gap metabolic acidosis with urinary pH more than 5.5 (Masafumi *et al.*, 2004). Diagnosis of distal tubular acidosis was confirmed by ammonium chloride challenge (Bihl and Myers, 2001). Calcium phosphate stones, nephrocalcinosis can support diagnosis. Absence of bicarbonaturia distinguishes distal RTA from proximal RTA (Cogan, 1991). Polyuria in our case is explained by hypokalemia induced nephrogenic diabetes insipidus due to ADH resistance. Hypokalemia causes dysfunction of the K+/H + pump in the distal nephron leading to retention of Na+ and hence water and polyuria.

Alkali supplement should be given for metabolic acidosis, in the range of 0.5 to 2.0mmol/Kg body wt/day in 4-6 divided doses can be rises upto 4mmol/Kg body wt/day (Bihl and Myers, 2001).

Conclusion

To conclude, RTA should be suspected as a differential in all cases of polyura, non anion gap metabolic acidosis, hypokalemia and renal stones. A simple ammonium chloride challenge test confirms the diagnosis and therapy is usually by alkali suplimentation.

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