

Acute flaccid paralysis associated with distal renal tubular acidosis - a rare case report

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Abstract

Renal tubular acidosis (RTA) is a constellation of syndromes arising from different derangements of tubular acid transport. Distal (Type 1) renal tubular acidosis (dRTA) is characterized by inability to secrete hydrogen ions from the distal tubule. In recent years, molecular biology techniques have identified the genetic factors involved in inadequate urinary excretion of H⁺ and ammonium in patients with distal RTA.

Keywords: renal tubular acidosis, Hypokalemia, flaccid paralysis, Proximal myopathy

Introduction

Renal tubular acidosis (RTA) is a constellation of syndromes arising from different derangements of tubular acid transport.^[1] Distal (Type 1) renal tubular acidosis (dRTA) is characterized by inability to secrete hydrogen ions from the distal tubule. It was first described in 1946.^[2] In recent years, molecular biology techniques have identified the genetic factors involved in inadequate urinary excretion of H⁺ and ammonium in patients with distal RTA^[3]. The aetiology of dRTA is diverse and can be either inherited or acquired. Common clinical presentations of dRTA in the paediatric age group include polyuria, nocturia, failure to thrive, constipation, abnormal breathing and nephrolithiasis. Though persistent hypokalemia is frequently seen in dRTA, hypokalemic muscular paralysis is uncommon and rarely described in children.

Case report:

Nine year old boy, born out of second degree consanguineous marriage presented with acute flaccid paralysis. He had significant past history of pathological fractures 2 episodes after trivial fall. Developmental domains were age appropriate. There was no history of polyuria, polydipsia, nocturia and abnormal breathing. Physical examination revealed short stature with height age of 4 years with waddling gait and hypotonia, diminished lower limb reflexes and muscle tone, previously diagnosed as a acute flaccid paralysis. On investigations found to have low potassium (1.7meq/l) and he was subsequently

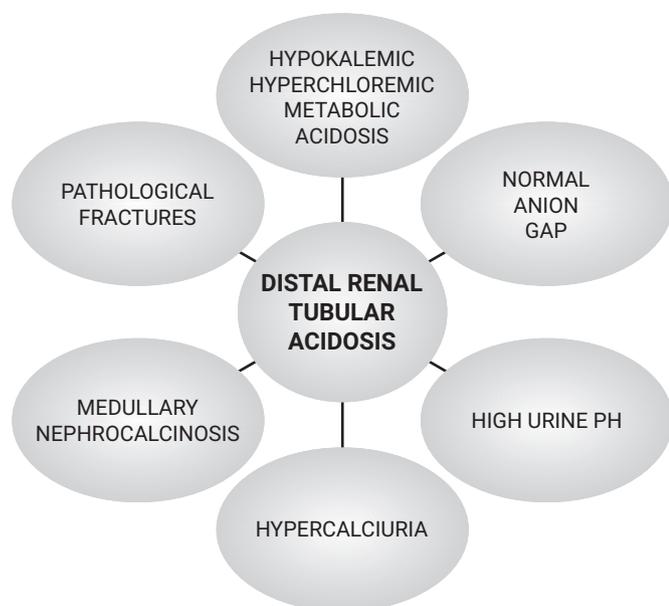
investigated for hypokalemic paralysis. Diagnosis of distal renal tubular acidosis was made, based on hypokalemic hyperchloremic metabolic acidosis with normal anion gap, high urine pH, hypercalciuria, medullary nephrocalcinosis, with past history of pathological fracture and exclusion of other differential diagnosis. The child showed complete symptomatic recovery upon commencement of standard treatment for distal renal tubular acidosis. On follow up height is increasing, no acidosis and potassium within normal limits.



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Discussion

Hypokalemic periodic paralysis (HPP) is a disorder that characterized by attacks of skeletal muscle paralysis depending on the changes in serum potassium levels, and can occur due to primary and secondary causes. One of the secondary causes of HPP is distal renal tubular acidosis (dRTA). Renal tubular acidosis (RTA) is diagnosed by measuring pH from the first urine in the morning in addition to simultaneous measurement of serum electrolytes, urea, creatinine, and ABG analysis. A positive urinary anion gap in addition to a pH > 5.5 and hypokalemia suggests DRTA (type 1). Symptoms secondary to hypokalemia become prominent with a serum potassium level below 2.5 - 3 mEq/L. The main finding in HPP is symmetric loss of muscle strength, especially in shoulder and pelvic muscles. Rarely, an asymmetrical involvement may also be observed, in which unilateral arm or leg is involved.⁴ Our patient had nausea and vomiting in addition to equal and symmetrical loss of muscle strength in both upper and lower extremities. Our patient had low potassium in the blood sample, and normal anion gap metabolic acidosis in ABG analysis. Among the differential diagnoses, Type 4 RTA was excluded since our patient had hyperchloremic metabolic acidosis in addition to a urine pH greater than 5.5, a low serum potassium levels.

Conclusion: This case report highlights the importance of considering hypokalemia and renal tubular acidosis in the differential diagnosis of acute flaccid paralysis. Early diagnosis will prevent costly investigations and enable rapid clinical recovery in the affected child.

References:

1. Ramen C. Basak et al. Distal Renal Tubular Acidosis, Hypokalemic Paralysis, Nephrocalcinosis, Primary Hypothyroidism, Growth Retardation, Osteomalacia and Osteoporosis Leading to Pathological Fracture. *Oman Medical Journal* 2011;26(4): 271-274.
2. Randula Ranawaka et al. A child with distal (type 1) renal tubular acidosis presenting with progressive gross motor developmental regression and acute paralysis. *BMC Res Notes* 2017; 10:618
3. Norma E. Guerra-Hernández et al. Distal renal tubular acidosis in two children with acquired Hypothyroidism. *Nefrologia* 2018;3 8(6):655-659
4. Herrin JT. Renal Tubular Acidosis. In: Barratt TM, Avner ED, Harmon WE (eds). *Pediatric Nephrology*. Lippincott Williams & Wilkins, Baltimore; 1999. p.565-77.

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