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ACUTE FLACCID PARALYSIS PRESENTING AS RENAL TUBULAR ACIDOSIS IN CHID: A CASE REPORT

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ABSTRACT

Background: Acute Flaccid Paralysis with symptoms of hypokalaemia and paralysis which was initially mistaken for the Guillain-Barre syndrome, however it was later diagnosed as Distal Renal Tubular Acidosis (dRTA). Renal tubular acidosis is a constellation of syndromes arising from different derangements of tubular acid transport. Common clinical presentations of dRTA in the paediatric age group include polyuria, nocturia, failure to thrive, constipation, abnormal breathing and nephrolithiasis. Though persistent hypokalemic is frequently seen in dRTA, hypokalemic muscular paralysis is uncommon and rarely described in children. **Case presentation:** A case of 4 years6 months old male presented with chief complaints of unable to stand and walk, unable to sit, unable to hold his neck since morning. Motor examination revealed weakness in the lower limbs with decrease in tone and power and absent deep tendon reflexes of the lower limbs. Blood investigations revealed hypokalaemia. An ABG was done which showed hyperchloremic normal anion gap metabolic acidosis, which raised the suspicion of renal tubular acidosis. Urine pH, urine anion gap and ultrasound abdomen pointed towards Distal Renal Tubular Acidosis. The child was treated accordingly and improved clinically. **Conclusion:** Whenever a child presents with weakness with neck drop, should raise a suspicion of hypokalaemia, subsequent ABG should be performed to rule out Renal Tubular Acidosis. This case highlights the importance of considering hypokalaemia 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.

Keywords: Acute Flaccid Paralysis (AFP), Distal Renal Tubular Acidosis (dRTA), Hypokalemia, Hypokalemic Periodic Paralysis

INTRODUCTION

Renal tubular acidosis is a constellation of syndromes arising from different derangements of tubular acid transport [1]. Common clinical presentations of dRTA in the paediatric age group include polyuria, nocturia, failure to thrive, constipation, abnormal breathing and nephrolithiasis. Though persistent hypokalemic is frequently seen in dRTA, hypokalemic muscular paralysis is uncommon and rarely described in children. Acute Flaccid Paralysis with symptoms of hypokalaemia and paralysis which was initially mistaken for the Guillain-Barre syndrome, however it was later diagnosed as Distal Renal Tubular Acidosis (dRTA).

CASE REPORT

A 4 year 6 months old male child, 2nd in birth order born out of 2nd degree consanguineous marriage, resident of Secunderabad was brought by mother with chief complaints of unable to stand and walk since morning, unable to sit and hold his neck since morning. The child was apparently asymptomatic a night before, later developed sudden onset weakness of bilateral lower limb progressing to neck soon after waking up in the morning.

No history of fever, rash, vomiting, loose stools, head trauma, seizure, altered sensorium, sore throat, recent history of vaccination, bowel and bladder incontinence, difficulty in lifting hand above head, difficulty in buttoning t-shirt or slipping of footwear. On detailed history mother complained of not gaining weight since one year. History of excessive thirst and passing excessive urine since last 1 month.

Child was thin built, malnourished, enamel chip off at multiple areas of teeth with widening of wrist joint and short stature. Anthropometry revealed microcephaly with severe stunting. Motor examination revealed weakness in bilateral lower limbs with decrease in tone and power of both lower limbs with absent deep tendon reflexes and plantar reflex with no sensory loss and autonomic disturbances. Investigations revealed normocytic anemia with hypokalemia (1.9meq/l) resistant to potassium correction. The child was investigated for hypokalaemia paralysis. An ABG was done which revealed hyperchloremic normal anion gap metabolic acidosis (pH –7.24, HCO_3 –13.67, Anion gap – 8, Chloride - 120) with urine pH –7, with positive urinary anion gap (U. Na+ = 13.8, U.K+ =119, U.Cl- =130). Urinary electrolytes revealed elevated Calcium and potassium in urine. Ultrasound abdomen showed B/L medullary

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Nephro-calcinosis with insufficient Vit-D3 levels (16.56 ng/ml).

Based on clinical and laboratory evidence child was diagnosed with dRTA as he presented with hypokalemia, hyperchloremic normal anion gap metabolic acidosis, alkaline urinary pH , positive urinary anion gap , medullary nephron-calcinosis with symptoms of sudden onset paralysis, polyuria, polydipsia, anaemia, stunting and vit D deficiency.

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.

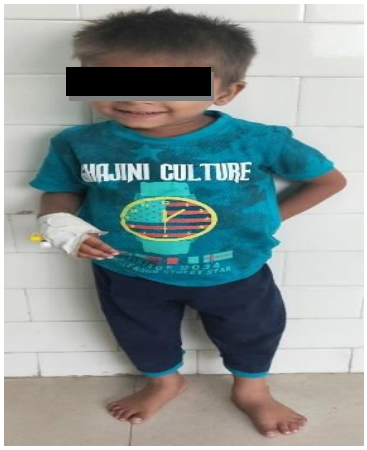

Fig 1: Hypokalaemia periodic paralysis (HPP)

DISCUSSION

Hypokalaemia periodic paralysis (HPP) (Fig:1) is a disorder that is 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) [2]. 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 [3,4]. The main finding in HPP is symmetric loss of muscle strength, especially in shoulder and pelvic muscles [5]. Rarely, an asymmetrical involvement may also be observed, in which unilateral arm or leg involved [6]. Our patient had symmetrical loss of muscle strength in both upper and lower extremities with low potassium in the blood sample and hyperchloremic normal anion gap metabolic acidosis on ABG analysis. It is important to differentiate from other causes of hypokalemia like familial periodic paralysis (FPP), thyrotoxicosis, hyperaldosteronism and gastrointestinal loss; because the treatment is different and bicarbonate therapy can deteriorate familial hypokalemic periodic paralysis; on the other hand it is the mainstay of therapy in RTA. This case highlights the importance of considering hypokalaemia and renal tubular acidosis in the differential diagnosis of acute flaccid paralysis.

CONCLUSION

Whenever a child presents with weakness with neck drop, should raise a suspicion of hypokalaemia, subsequent ABG should be performed to rule out Renal Tubular Acidosis. This case highlights the importance of considering hypokalaemia 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.

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