A YOUNG GIRL WITH EPIGASTRIC PAIN AND EPISODIC GENERALIZED MOTOR WEAKNESS: A CASE REPORT

Munnam Sohail Jafar, Ahmad Malik, Choudhary Naim Muhammad

Jinnah Hospital Lahore Pakistan

ABSTRACT

A young female presented with epigastric pain, scant vomiting and generalized weakness. Ultrasound abdomen was normal. Serum lipase and amylase were raised. Arterial blood gases (ABGs) revealed metabolic acidosis, electrocardiogram (ECG) showed U-waves, serum potassium was 2.1, urine pH and urinary sodium were 6 and 82 mEq/L respectively and urine anion gap was raised. A diagnosis of distal renal tubular acidosis and acute pancreatitis was made. She was made pain free and nil per os (NPO) was ordered. For her renal tubular acidosis she was given intravenous potassium citrate followed by sodium bicarbonate. The patient got clinically improved and was then discharged home on oral potassium citrate.

Key words: Acute pancreatitis, Hypokalemia, Renal tubular acidosis.

INTRODUCTION

The term "renal tubular acidosis" (RTA) refers to a group of disorders in which hyperchloremic metabolic acidosis develop because of inability of the renal tubules to perform net renal acid excretion, despite the absence or out of proportion decrease in the glomerular filtration rate. Renal acid excretion is carried out by either bicarbonate conservation or hydrogen excretion[1]. RTA has four types, namely distal (type 1 RTA), proximal (type 2 RTA), type 3 and type 4 RTA, which can be distinguished based on clinical, pathophysiological, and molecular criteria[2].

CASE REPORT

A 25 years old female presented to the emergency with a history of epigastric pain radiating to back for 1 day. It was slow onset, progressive, did not change with position, aggravated with food intake. It was associated with multiple episodes of non-bloody vomiting, which was scant in amount and contained food particles.

The patient also complained about off and on generalized weakness of all limbs for the past 15 months which spontaneously got better. She could not bear her weight on her limbs during these episodes. They were not associated with weakness of any part of the body, seizures, altered state of consciousness, fever, sensations of insects crawling under the skin, balance problems, bowel/bladder incontinence, diarrhea or vomiting.

Her past medical record showed a hospital admission for acute pancreatitis 6 months back. There was no history of medicine intake except antacids and omeprazole for her pain. Her personal history was not significant for alcohol, smoking or drugs of abuse.

On admission, she was vitally stable except her heart rate was 100/min. There was mild tenderness in the epigastric region without any guarding or rebound tenderness. Bowel sounds were audible. Motor and sensory system were unremarkable with normal plantars, deep tendon reflexes and sensations.

A stat ECG revealed U waves. ABGs showed non anion gap metabolic acidosis with bicarbonate 5.4 mEq/L and plasma anion gap of 10. Ultrasound abdomen showed no gallstones. The patient was made pain free and NPO was ordered. Lipase and amylase levels were raised and serum potassium level turned out to be

Correspondence: Dr Munnam Sohail Jafar, 32-B, Allama Iqbal Medical College Jinnah Hospital Lahore Pakistan
Email: munnamjafar@gmail.com
Received: 03 May 2016; revised received: 17 Feb 2017; accepted: 02 Mar 2017

2.1 mEq/L. Urine pH was found to be 6 and the urine anion gap was 21. A diagnosis of distal renal tubular acidosis and acute pancreatitis was made.

Patient was treated with intravenous potassium citrate and afterwards with intravenous sodium bicarbonate. Sodium bicarbonate replacement resulted in painful carpal spasms due to accompanying hypocalcemia, which only improved temporarily with intravenous calcium carbonate administration. So a concurrent magnesium (Mg) deficiency was suspected and investigated. Mg levels were found to be low and replaced. After the treatment, patient got clinically improved and was discharged home on oral potassium citrate.

DISCUSSION

Typical findings of type 1 distal RTA (dRTA) include low serum bicarbonate, normal serum anion gap, hyperchloremia and hypokalemia. Hypokalemia in dRTA is sometimes severe and produces muscle weakness. However, the mechanisms leading to hypokalemia are likely multifactorial and not well understood[2]. Chronic metabolic acidosis leads to sequestration of protons in bone which stimulate osteoclasts and make the patient prone to osteoporosis. Metabolic acidosis also leads to nephrocalcinosis because it causes proximal tubular reabsorption of citrate which results in hypocitraturia that promotes calcium phosphate precipitation. It is also suggested that dRTA is more prevalent in autoimmune diseases like Sjögren syndrome, SLE, primary biliary cirrhosis, autoimmune hepatitis and autoimmune thyroiditis. However, the pathophysiological mechanism of dRTA in relation to autoimmunity remains unclear[3].

Treatment is not only targeted to correct hypokalemia and acidosis but is also aimed to improve growth, prevent nephrolithiasis and skeletal abnormalities. A mixture of sodium and potassium citrate salts is recommended. Citrate salts correct the metabolic acidosis and prevent nephrolithiasis secondary to hypocitraturia[4]. Hypokalemia should be corrected before correcting acidosis, as alkali replacement can worsen the hypokalemia which can lead to dangerous consequences. Correcting hypokalemia also improves the musculoskeletal symptoms.

Hypokalemia should always be considered as a cause of muscle weakness. Nonspecific symptoms of muscle weakness can easily be overlooked and attributed to somatization secondary to anxiety, especially in females during busy emergency hours. A high degree of suspicion along with ECG and ABGs can help in establishing a quick bedside provisional diagnosis. We were unable to determine the etiology of RTA in our case, however, owing to the association of distal RTA with autoimmune diseases, a possible autoimmune etiology must always be sought.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

REFERENCES

1. Basu G, Sudhakar G, Mohapatra A. Renal tubular acidosis. Clinical queries: nephrology 2013; 2(4): 166-78.
2. Gil-Peña H, Mejía N, Santos F. Renal tubular acidosis. The Journal of pediatrics 2014; 164(4): 691-8.
3. Both T, Zietse R, Hoorn EJ, van Hagen PM, Dalm VA, van Laar JA, et al. Everything you need to know about distal renal tubular acidosis in autoimmune disease. Rheumatology international 2014; 34(8): 1037-45.
4. Batlle D, Haque SK. Genetic causes and mechanisms of distal renal tubular acidosis. Nephrology Dialysis Transplantation 2012; 27(10): 3691-704.