10.5152/turkjnephrol.2020.3706

Case Report

Turkish Journal of Nephrology

Life-Threatening Hypokalemia Presenting with Paralysis after Percutaneous Nephrolithotomy in a Patient with Nephrolithiasis: A Case Report

Cenk Gökalp[1], Tuncer Bahçeci[2], Eda Otman[3], Fulya Doğan[3], Merve Güner[3], Soner Duman[3]

[1]Department of Nephrology, Ege University School of Medicine, İzmir, Turkey
[2]Department of Urology, Ege University School of Medicine, İzmir, Turkey
[3]Department of Internal Medicine, Ege University School of Medicine, İzmir, Turkey

Abstract

Distal renal tubular acidosis (Type 1 RTA) is an acid-base disorder characterized by impaired urinary acidification by the distal tubule. Osteomalacia, rickets, nephrocalcinosis, nephrolithiasis, and electrolyte imbalance can be seen in these cases. We report the case of a 24-year-old male patient who had low back pain with a history of nephrolithiasis. Routine laboratory results revealed mild hypokalemia and hyperchloremia. After percutaneous nephrolithotomy surgery, he developed muscle paralysis with a serum potassium level of 1.3 mEq/L. After intensive treatment of hypokalemia, the patient had normal anion gap metabolic acidosis, high urine pH, high transtubular potassium gradient, nephrocalcinosis, and hypercalciuria and was thus diagnosed as having distal RTA. After stabilization of the patient, he was discharged with oral potassium, bicarbonate, and spironolactone medications. Hypokalemia and hyperchloremia in a patient with a history of recurrent renal stones should be kept in mind for a possible distal RTA diagnosis. Prompt diagnosis and treatment of this situation not only reduces the formation of renal stones, but also may be lifesaving in rare cases.

Keywords: Distal renal tubular acidosis, hypokalemia, nephrolithiasis

Corresponding Author: Cenk Gökalp ✉ cenkshome@yahoo.com

Received: 19.12.2018 **Accepted:** 27.01.2019

Presented in: This study was presented at the 32th National Cogress of Nephrology, Hypertension, Dialysis and Transplantation, 2015, Antalya, Turkey

Cite this article as: Gökalp C, Bahçeci T, Otman E, Doğan F, Güner M, Duman S. Life-Threatening Hypokalemia Presenting with Paralysis after Percutaneous Nephrolithotomy in a Patient with Nephrolithiasis: A Case Report. Turk J Nephrol 2020; 29(2): 174-6.

INTRODUCTION

Distal renal tubular acidosis (Type 1 RTA) is a disease characterized by impaired urinary acidification by the distal tubule (1). This rare clinical condition may be congenital or acquired, as in proximal RTA. Hypokalemia, hyperchloremic metabolic acidosis, inability to acidify urine pH<5.5, nephrocalcinosis, and nephrolithiasis are among the most common findings of patients with distal RTA. Herein, we report a case of hypokalemic paralysis after percutaneous nephrolithotomy (PNL) in a patient with recurrent renal stone history.

CASE PRESENTATION

A 24-year-old male patient was admitted to our urology clinic with a complaint of low back pain. The patient's medical history revealed that he had undergone nephrolithotomy operations in the past and there was no medication and comorbidity history. When family history was questioned, we learned that his father had a history of nephrolithiasis and died because of lung cancer 14 years ago. Abdominal computed tomography (CT) was performed due to the suspicion that the present complaints of the patient may still be related to nephrolithiasis. The abdominal CT of the patient showed extensive stones in the right ureter and filling bilateral renal calyxes (Figure 1, 2), and PNL was planned for the treatment. The patient's blood pressure was 110/70 mm Hg, pulse pressure was 76 beats/min, and body temperature was 36.7 °C; he had hypokalemia (K^+: 3.4 mEq/L) and hyperchloremia (Table 1).

The patient underwent PNL in the urology clinic and had symptoms of weakness and palpitation. Inability to

Table 1. Blood biochemistry of the patient pre- and post-surgery and at discharge

	Pre-surgery	Post-surgery	Discharge
AST (U/L)	19	35	32
ALT (U/L)	13	22	37
ALP (U/L)		**203**	132
Protein (g/dL)		6.5	6.7
Albumin (g/dL)		3.8	3.6
BUN (mg/dL)	36	38	24
Creatinine (mg/dL)	0.98	1	0.79
Sodium (mEq/L)	139	135	141
Potassium (mEq/L)	**3.4**	**1.3**	4.9
Chloride (mEq/L)	**120**	105	107
Calcium (mg/dL)		8.7	9
Magnesium (mg/dL)		2.56	
Phosphorus (mg/dL)		**1.26**	3.19
Blood pH		**7.25**	7.39
Blood PCO_2 (mm Hg)		**23.9**	**34.8**
Blood HCO_3 (mmol/L)		**10.5**	**20.9**
Urine Na^+ (mEq/L)		43	
Urine K^+ (mEq/L)		7.2	
Urine Cl^- (mEq/L)		33	
Urine creatinine (mg/dL)		48.8	
Urine pH		6.5	8.0
Urine Ca^{++} (mg/24 h)		**731**	
Urine PO_4 (mg/24 h)		715	
Blood osmolarity (mOsm.L)		303	
Urine osmolarity (mOsm.L)		323	

Abnormal laboratory results are highlighted as bold.
BUN: blood urea nitrogen; AST: aspartate transaminase; ALT: alanine aminotransferase; ALP: alkaline phosphatase

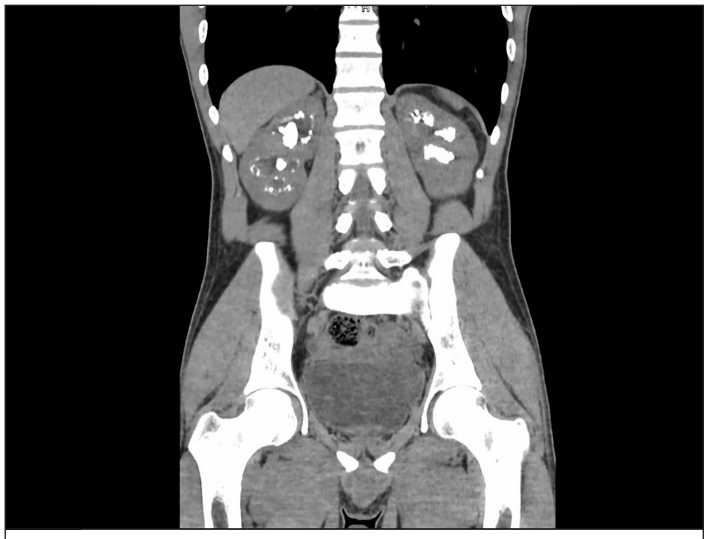

Figure 1. Computerized tomography showing bilateral renal calculi filling renal pelvises.

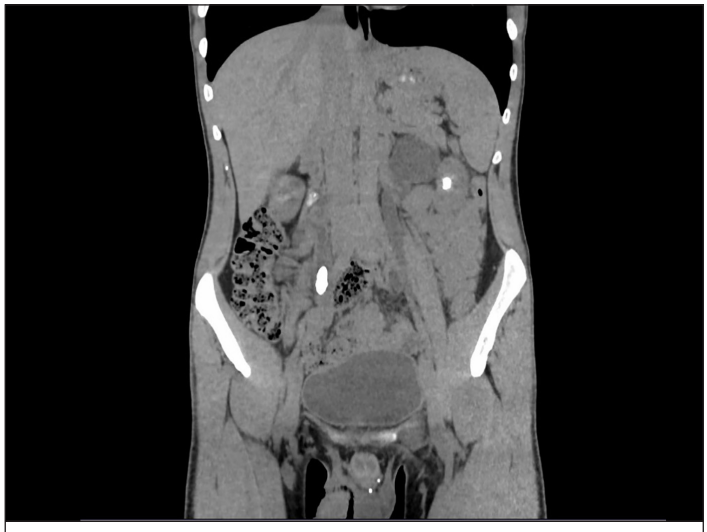

Figure 2. Computerized tomography showing urolithiasis in the right ureter.

move the lower and upper extremities was developed 6 h after the surgery. Serum potassium level was observed to be very low (K^+: 1.3 mEq/L) in the blood biochemistry of the patient, and intravenous potassium infusion was started at 20 mEq/h from the central vein and 80 mEq potassium was given orally. After 4 h of treatment, the patient had no significant elevation of serum potassium level, and he was given continuous intravenous potassium replacement at 15 mEq/h and 120 mEq potassium was administered orally. Blood gas analysis of the patient revealed normal anion gap metabolic acidosis and urine pH 6.5. The calculated transtubular potassium gradient (TTKG) of the patient was 5.2. The patient had normal anion gap metabolic acidosis, high urine pH, high TTKG, possible medullary nephrocalcinosis, and hypercalciuria and was thus diagnosed as having distal (Type 1) RTA and treated with intravenous potassium and bicarbonate infusions with oral potassium and spironolactone (200 mg/day). Genetic analysis of the patient revealed ATP6V0A4

Main Points

- Renal tubular acidosis may cause osteomalacia, nephrolithiasis and electrolyte disorders.
- Recurrent nephrolithiasis should be investigated for metabolic disorders.
- Prompt diagnosis of renal tubular acidosis is mandatory for appropriate management in severe cases.

gene mutation, which is associated with autosomal recessive distal RTA. After the rise of the patients serum potassium level above 2.5 mEq/L, complaints like palpitations and paralysis resolved. Additionally, hypophosphatemia secondary to hypokalemia and possible hydration at operation were observed in the blood biochemistry of the patient. The blood phosphorus level returned to normal after the correction of hypokalemia without any phosphorus replacement. After a total of 760 mEq intravenous and 960 mEq oral replacement of potassium, the patient was discharged with oral potassium, bicarbonate, and spironolactone medications.

DISCUSSION

RTA is a metabolic acidosis pattern that develops due to the impaired ability of renal tubules to maintain the acid-base homeostasis. Distal RTA is characterized by the impairment of hydrogen ion excretion from the collecting duct. The most common causes in adults are autoimmune diseases (Sjögren's syndrome, rheumatoid arthritis, systemic lupus erythematosus, cryoglobulinemia, etc.), hypercalciuria, drugs (amphotericin, lithium, ifosfamide, etc.), and some genetic mutations (2). Genetic forms may have autosomal dominant (luminal HCO_3^--Cl^- exchanger defect) or autosomal recessive (basolateral H^+-ATPase defect) inheritance.

Due to the defective H^+ secretion in distal RTA, less NH_4^+ is captured in the tubule lumen; this condition results in a higher urine anion gap. A patient with a high anion gap metabolic acidosis and hypokalemia with a urine pH>5.5 should be suspected to have distal RTA. The plasma bicarbonate level is usually below 15 mEq/L. Hypercalciuria and reduced citrate excretion can also be observed. Osteomalacia, rickets, nephrocalcinosis, nephrolithiasis, and electrolyte imbalance can be seen in untreated cases.

CONCLUSION

The goal of treatment is to correct metabolic acidosis and electrolyte abnormalities. Sodium bicarbonate treatment for the correction of metabolic acidosis can also reduce the loss of potassium and calcium.

Informed Consent: Written informed consent was obtained from the patient who participated in this case.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - C.G.; Design - C.G.; Supervision - S.D.; Data Collection and/or Processing - T.B., E.O., F.D., M.G.; Analysis and/or Interpretation - T.B., E.O.; Literature Search - F.D., M.G.; Writing - C.G., T.B., E.O., F.D., M.G.; Critical Reviews - S.D.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declare that they have received no financial support for this case.

REFERENCES

1. Rodríguez Soriano J. Renal tubular acidosis: The clinical entity. J Am Soc Nephrol 2002; 13: 2160-70. [Crossref]
2. Stedwell RE, Allen KM, Binder LS. Hypokalemic paralyses: A review of the etiologies, pathophysiology, presentation, and therapy. Am J Emerg Med 1992; 10: 143-8. [Crossref]