



Diagnosis of Renal Tubular Acidosis in Patients With Acute Pyelonephritis

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Case Presentation

A 35-year-old woman with no relevant medical history presented to the emergency department (ED) complaining of right flank pain and fever for one day. A physical examination revealed costovertebral angle knocking pain. A biochemical examination revealed hypokalemia, pyuria, and leukocytosis with left shift. A kidneys, ureter, and bladder X-ray (KUB) revealed multiple bilateral kidney stones (Fig. 1). Acute pyelonephritis was suspected. The biochemical analysis of serum and urine is presented in Table 1. The results revealed hyperchloremia, normal anion gap metabolic acidosis, positive urine anion gap, a urine pH of 6.956,

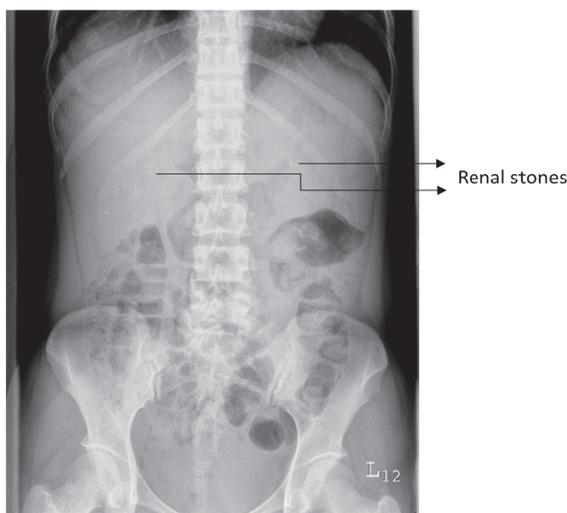


Fig. 1. Multiple bilateral renal stones in a kidneys, ureter, and bladder (KUB) X-ray.

Table 1. Biochemistry, immune, and urine profiles

	Serum	Urine
pH	7.402	6.956
pCO ₂	27.8	21.4
HCO ₃ ⁻	16.9	4.7
Na ⁺	130 (at ER)	17
K ⁺	2.6 (at ER)	4.3
Cl ⁻	112	20
Cr	1.01 (at ER)	96.4
ANA	1:80 (—)	
Anti-dsDNA	(—)	
RA	(—)	
Anti-SSA (Ro)	(—)	
Anti-SSB (La)	(—)	

a low urine minus blood pCO₂ (U-B pCO₂) level, and a low fractional excretion of bicarbonate (FEHCO₃⁻). Immune profiles were within the normal range. Type 1 (distal) renal tubular acidosis (RTA) was diagnosed. An inherited or medullary-sponge kidney-related disease was suspected. Six hundred milligrams of potassium citrate three times a day was prescribed to treat the hypokalemia and prevent urolithiasis. The patient was asymptomatic and in good health three months after treatment.

Discussion

Although renal-stone—inducing acute pyelonephritis and hypokalemia—is a common disease

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encountered by ED physicians, other underlying diseases should also be considered. The clinical features of RTA are nephrocalcinosis, hypokalemia, hyperchloremia, and normal anion-gap metabolic acidosis. Distal tubules play a role in generating bicarbonates. Damage to the distal tubules may cause elevation of urine pH as a result of the inability to excrete acid in the distal tubules; such damage is associated with hypokalemia caused by the failure of H/K ATPase. Autoimmune diseases, such as systemic lupus erythematosus and Sjogren syndrome, are the most common cause of distal RTA in adults.¹ Medullary-sponge kidney-related nephrocalcinosis, chronic pyelonephritis, chronic interstitial nephritis and obstructive uropathy have also been discussed in association with distal RTA.

Laboratory findings of distal RTA include a normal plasma anion gap, low urinary ammonium, hypokalemia, positive urine anion gap (Urine $\text{Na}^+ + \text{K}^+ - \text{Cl}^-$), minimal urine pH higher than 5.5, FEHCO_3^- less than 5%, and U-B pCO_2 in alkaline urine equal to or less than zero. Conversely, in proximal RTA, the FEHCO_3^- value is usually more than 20%, and the U-B pCO_2 gradient should be greater than 20 mmHg in normal individuals.

Distal RTA is the most common form of primary RTA in Western countries. The incidence of distal RTA is 2.3% in the patients with calcium-containing stones, and tends to predominantly affect women.² Confirming the diagnosis of RTA is difficult and often

delayed. In this case report, diagnosis of distal RTA is through the typical symptoms of acute pyelonephritis combined with renal stones and hypokalemia. The possibility of distal RTA should always be considered, especially in the patients with renal stones and hypokalemia. Acid-base status, chloride, anion gap, and urine parameters should also be examined for a differential diagnosis. Treatment includes correction of hypokalemia and alkali replacement.³ Hypokalemia should be corrected first because alkali replacement can exacerbate hypokalemia and therefore lead to dangerous consequences. Early treatment also reduces the incidence of nephrocalcinosis, recurrence of renal stones, and progression to chronic kidney disease.⁴

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