

# Nephrocalcinosis: A Case Report

## Nefrokalsiniz: Olgu Sunumu

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### Abstract

Nephrocalcinosis is characterized by increased calcium deposition within the renal parenchyma, most commonly affecting the renal medulla and less frequently the cortex, and may result in progressive renal dysfunction. It is often detected incidentally through imaging modalities such as abdominal radiography, ultrasonography, or computed tomography (CT). We report the case of an 18-year-old female with a history of recurrent nephrolithiasis and growth retardation who presented with acute left flank pain, gross hematuria, and mild dehydration. Laboratory evaluation revealed hypercalciuria, hyperchloremic metabolic acidosis with a normal anion gap, and inappropriately alkaline urine. Imaging studies demonstrated bilateral medullary nephrocalcinosis and a 17-mm calculus at the left ureteropelvic junction, resulting in grade 3 hydronephrosis. Non-contrast CT confirmed the diagnosis and excluded infectious or obstructive complications. The ureteral stone was successfully removed via ureteroscopy without complications. Based on clinical and laboratory findings, a diagnosis of distal renal tubular acidosis (dRTA) was established, and alkali therapy with sodium bicarbonate and potassium citrate was initiated. Nephrocalcinosis is frequently associated with dRTA, a disorder characterized by impaired urinary acidification leading to alkaline urine in the presence of hyperchloremic metabolic acidosis. Early recognition is crucial to prevent recurrent nephrolithiasis and long-term renal impairment.

**Keywords:** Distal renal tubular acidosis, hematuria, nephrocalcinosis, nephrolithiasis

### Öz

Nefrokalsinoz, böbrek parankiminde, en sık böbrek medullasını ve daha az sıklıkla korteksi etkileyen ve ilerleyici böbrek fonksiyon bozukluğuna yol açabilen artmış kalsiyum birikimi ile karakterizedir. Genellikle karın radyografisi, ultrasonografi veya bilgisayar tomografisi (BT) gibi görüntüleme yöntemleriyle tesadüfen tespit edilir. Tekrarlayan nefrolitiazis ve büyüme geriliği öyküsü olan 18 yaşında bir kadın hastanın akut sol yan ağrısı, makroskopik hematüri ve hafif dehidratasyon ile başvurduğu bir olgu sunulmuştur. Laboratuvar değerlendirmesinde hiperkalsiüri, normal anyon açıklığı ile hiperklorik metabolik asidoz ve uygunsuz derecede alkali idrar saptandı. Görüntüleme çalışmaları, bilateral medüller nefrokalsinoz ve sol üreteropelvik kavşakta 17 mm'lik bir taş olduğunu ve bunun sonucunda 3. derece hidronefroz oluştuğunu gösterdi. Kontrastsız BT, tanıyı doğruladı ve enfeksiyon veya obstrüktif komplikasyonları ekarte etti. Üreter taşı, üreteroskopi yoluyla komplikasyonsuz bir şekilde başarıyla çıkarıldı. Klinik ve laboratuvar bulgularına dayanarak distal renal tübüler asidoz (dRTA) tanısı konuldu ve sodyum bikarbonat ve potasyum sitrat ile alkali tedaviye başlandı. Nefrokalsinoz, sıklıkla hiperklorik metabolik asidoz varlığında idrar asitliğinin bozulması ve alkali idrar oluşumu ile karakterize bir bozukluk olan dRTA ile ilişkilidir. Tekrarlayan nefrolitiazis ve uzun süreli böbrek yetmezliğini önlemek için erken teşhis çok önemlidir.

**Anahtar kelimeler:** Böbrek, hematüri, nefrokalsinoz, nefrolitiazis

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## Introduction

Nephrocalcinosis is characterized by an intense increase in calcium content in the kidneys. It can be effective at the molecular level or vary from microscopic to macroscopic levels and ultimately leads to progressive renal impairment (1). It predominantly affects the renal medulla, although the cortex may be involved less frequently. In the context of hypercalciuria, the increased concentration and supersaturation of urine lead to the accumulation of calcium crystals within the renal parenchyma (2).

Incidental diagnoses are frequently encountered in patients. Key imaging modalities for diagnosis include abdominal scanning, kidney ultrasonography, and computed tomography (3). Nephrocalcinosis is commonly regarded as a manifestation of a systemic disorder, thus requiring a thorough evaluation to ascertain its underlying causes (3). The differential diagnosis encompasses conditions such as primary hyperparathyroidism, medullary sponge kidney, sarcoidosis, vitamin D intoxication, distal renal tubular acidosis (dRTA), and various inherited tubulopathies. In alignment with clinical suspicion, the initial laboratory assessment should encompass serum levels of calcium and phosphate, parathyroid hormone, vitamin D, as well as urinalysis and urine electrolytes (4).

This case report details an 18-year-old female patient diagnosed with nephrocalcinosis, for whom written informed consent was procured prior to the preparation of the case presentation.

## Case Report

The family of the patient presented an 18-year-old female to the pediatric emergency department of the hospital. She had a documented history of growth retardation, with a height of 148 cm (<3<sup>rd</sup> percentile) and a weight of 38 kg (<3<sup>rd</sup> percentile) according to age- and sex-matched reference values. She had a history of recurrent kidney stones. Patient's complaints were sudden, severe pain in the left flank and gross hematuria that developed over the preceding 12 hours.

Upon admission, vital signs were appropriate for late adolescence. Body temperature was 36.3 °C, heart rate was 104 bpm, blood pressure was 110/60 mmHg, and respiratory rate was 30 breaths/min. Physical examination revealed a mildly distressed patient who appeared slightly dehydrated but otherwise non-toxic. Palpation of the left flank produced tenderness, while the midline spine was non-tender. Abdominal assessment demonstrated

localized tenderness, with rebound and guarding noted in the left lower quadrant of the abdomen. No abnormalities were observed in the remaining systems.

The patient's blood tests, peripheral blood smear and urinalysis results were summarized in Table 1.

Arterial blood gas analysis demonstrated metabolic acidosis with a normal anion gap (pH: 7.21, HCO<sub>3</sub><sup>-</sup>: 15.7 mmol/L, pCO<sub>2</sub>: 41.3 mmHg). The calculated serum anion gap was 9 mEq/L. Urinary pH was persistently alkaline (pH: 8.0), and the urine anion gap was positive, supporting impaired renal acid excretion. Urinary calcium excretion was elevated at 4.9 mg/kg/day. Urine culture was negative, and inflammatory markers normalized during follow-up, excluding urinary tract infection.

An anteroposterior abdominal radiograph was obtained as part of the initial evaluation to assess for radiographic signs suggestive of an acute abdomen and to exclude gross abdominal pathology (e.g, appendicitis, complicated acute cholecystitis, ectopic pregnancy, diabetic ketoacidosis, sickle cell crisis, noncomplicated hepatobiliary disease and non-complicated acute cholecystitis, gallstones, viral gastroenteritis/viral syndrome, Henoch-Schönlein purpura, constipation, mononucleosis) (Figure 1).

Ultrasonography revealed the presence of bilateral medullary nephrocalcinosis, characterized by calcification in the medullary regions of both kidneys, in addition to calyceal stones with an average diameter of 1 centimeter.

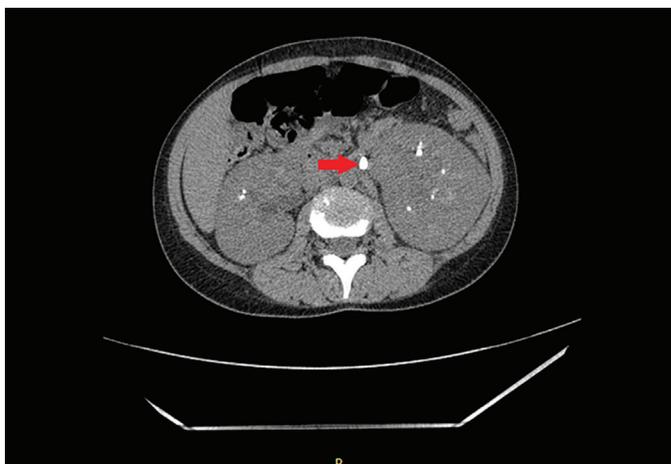


**Figure 1.** Anteroposterior abdominal radiograph taken at the patient's initial presentation (medullary nephrocalcinification in the right and left renal fossae)

**Table 1. The patient's blood tests, peripheral blood smear and urinalysis results**

Test category	Test name	Result	Unit	Pediatric reference range	Observation
Blood tests	Hemoglobin	11.8	g/dL	11.5-15.5	Within range
	WBC	7.66	$\times 10^9/L$	4.0-10.5	Within range
	Platelets	355	$\times 10^9/L$	150-450	Within range
	Urea	8.3	mmol/L	2.5-7.1	Slightly high
	Creatinine	58	$\mu\text{mol/L}$	27-88	Within range
	Uric acid	155	$\mu\text{mol/L}$	120-330	Within range
	CRP	105	mg/L	<10	Significantly high
	Sodium	138	mmol/L	135-145	Within range
	Chloride	113	mmol/L	98-107	High
	Potassium	3.9	mmol/L	3.5-5.0	Within range
	Phosphorus	1.26	mmol/L	1.29-2.26	Slightly low
	Calcium	2.10	mmol/L	2.12-2.62	Slightly low
	Magnesium	0.95	mmol/L	0.7-0.9	Slightly high
	ALP	80	U/L	130-560 (during growth)	Low
Peripheral blood smear	PNL	62.5	%	25-60	Slightly high
	Lymphocytes	27.1	%	30-65	Slightly low
Urinalysis	Appearance	Turbid	-	Clear	Abnormal
	Color	Red	-	Yellow	Abnormal
	pH	8.0	-	5.0-8.0	Alkaline
	Nitrite	Negative	-	Negative	Normal
	Density	1010	-	1003-1030	Normal
	Protein	+++	Dipstick	Negative	Positive
	Glucose	Negative	-	Negative	Normal
	Ketone	Negative	-	Negative	Normal

WBC: White blood cell, CRP: C-reactive protein, PNL: Percutaneous nephrolithotomy, ALP: Alkaline phosphatase



**Figure 2.** Calcification in the renal medulla together with mixed nephrocalcinosis and proximal left ureteral stone (red arrow)

At the left uteropelvic junction, a stone sitting proximally to the ureter with a diameter of 17 mm is observed, grade 3 hydronephrosis is observed in the left kidney.

Non-contrast CT excluded pyelonephritis, abscess formation, or other infectious complications and confirmed mild left-sided hydronephrosis. The findings indicated the presence of medullary nephrocalcinosis, a 17 mm calculus located in the proximal left ureter, and mild hydronephrosis on the left side (Figure 2). The patient underwent ureteroscopic stone extraction without perioperative complications. Post-procedural follow-up demonstrated resolution of pain and hematuria.

Following initiation of alkali therapy, the patient showed improvement in metabolic acidosis, normalization of serum bicarbonate levels, and remained clinically stable without recurrence of hematuria or flank pain during follow-up.

As a result, the case was characterized by inappropriately alkaline urine in the setting of hyperchloremic normal anion gap metabolic acidosis and growth retardation. Based on these findings, a diagnosis of dRTA was established, and

oral sodium bicarbonate (3 mEq/kg/day) and potassium citrate therapy were initiated.

## Discussion

Nephrocalcinosis has a multifactorial etiology. It can arise secondary to dRTA but may also result from hypercalcemia, hyperoxaluria, or exposure to certain medications such as furosemide, vitamin D analogues, amphotericin B, ifosfamide, and lithium. In some instances, however, nephrocalcinosis remains idiopathic (5).

dRTA is defined by the presence of persistently alkaline urine, even in the context of hypokalemic, hyperchloremic metabolic acidosis (6). This pathology arises from a primary defect in the urinary acidification process, specifically the impaired excretion of protons in the collecting ducts (7). The type A intercalated cells within the connecting tubules and collecting ducts exhibit insufficient proton excretion as ammonium, which is essential for the elimination of ammonia, the acidification of urine, and the production of new bicarbonate (8,9).

The net acid excretion is quantified by the total of urinary ammonium and titratable acidity, with bicarbonate being subtracted from this sum (10). In clinical practice, urinary phosphate often serves as an indicator of titratable acidity, while urinary bicarbonate is typically disregarded if the urine pH is below 6.5. The function of type A intercalated cells, along with overall acid excretion, generally increases in response to systemic acidosis (11).

Patients diagnosed with dRTA frequently exhibit signs of hyperchloremic metabolic acidosis with a normal anion gap, often accompanied by hypokalemia. In pediatric and adolescent populations, dRTA may present as growth retardation, failure to thrive, kidney stones, rickets, or nephrocalcinosis, which are often the initial manifestations of the condition. Furthermore, polyuria may arise due to impaired urine concentration resulting from hypercalciuria, hypokalemia, or nephrocalcinosis (12-14). Based on the presence of hyperchloremic normal anion gap metabolic acidosis, persistently alkaline urine, positive urine anion gap, hypercalciuria, nephrocalcinosis, and growth retardation, dRTA was diagnosed.

During childhood, nephrocalcinosis usually presents in the medullary rather than cortical region (15). dRTA is often associated with significant hypercalciuria, predisposing to kidney stones, bone demineralization, nephrocalcinosis,

and potentially progressing to chronic kidney disease requiring dialysis in adolescence or early adulthood (16). The patient exhibited nephrocalcinosis, ureteral calculi, hyperchloremic metabolic acidosis characterized by a normal anion gap, recurrent episodes of dehydration suggestive of polyuria, and growth retardation.

## Conclusion

dRTA is a rare metabolic disorder that can lead to serious clinical complications. It should be included in the pediatric differential diagnosis showing marked growth retardation, tubular dysfunction, polyuria, refractory rickets, and hypokalemic metabolic acidosis.

A continuum appears to exist linking recurrent nephrolithiasis, nephrocalcinosis, and distal renal tubular acidosis. Diagnosis is established through clinical suspicion supported by characteristic laboratory findings. Clinicians are advised to pursue further evaluation in children with recurrent urinary tract infections, renal stone formation, or growth retardation due to diagnostic challenges at early stages.

dRTA should be considered in adolescents presenting with recurrent nephrolithiasis, nephrocalcinosis, growth retardation, and hyperchloremic metabolic acidosis. Early diagnosis and appropriate alkali therapy are essential to prevent long-term renal damage.

## Ethics

**Informed Consent:** This case report details an 18-year-old female patient diagnosed with nephrocalcinosis, for whom written informed consent was procured prior to the preparation of the case presentation.

## Footnotes

### Authorship Contributions

Surgical and Medical Practices: D.A., E.N.A.Ö., P.E., Concept: D.A., E.N.A.Ö., P.E., K.Ö., Design: D.A., E.N.A.Ö., P.E., K.Ö., Data Collection or Processing: D.A., E.N.A.Ö., P.E., Analysis or Interpretation: D.A., K.Ö., Literature Search: D.A., E.N.A.Ö., P.E., K.Ö., Writing: D.A., K.Ö.

**Conflict of Interest:** No conflict of interest was declared by the authors.

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