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Case Report

Internal Medicine Section

# Nephrolithiasis in a Second Renal Allograft Recipient: A Rare Cause of Graft Dysfunction

AALAYA HARIDAS[1], MANIKANTAN SHEKAR[2], RAMA VENKATESHPRASAD[3], AMEYA CHOGLE[4], SANKETH NETHULA[5]



## ABSTRACT

Renal allograft nephrolithiasis is an uncommon but clinically significant complication which may compromise graft function. While structural and infective factors are well recognised, metabolic abnormalities such as hyperoxaluria and hypocitraturia are increasingly being recognised as contributors to post renal transplant nephrolithiasis. This case describes a 49-year-old male, a second renal transplant recipient who developed de novo nephrolithiasis with ureteric obstruction in the transplanted kidney, complicated by a urinary tract infection and graft dysfunction. His first transplanted renal graft failure was due to chronic active Antibody mediated rejection and recurrent chronic pyelonephritis, which required a graft nephrectomy. He subsequently underwent a deceased donor renal transplantation. He had episodes of urinary tract infection and was treated with antibiotics. One month later he was presented with graft dysfunction, imaging revealed multiple renal calculi with proximal ureteric calculi with mild hydroureteronephrosis. He underwent ureteroscopy lithotripsy with double-J stenting, after which there was stabilisation of graft function. Metabolic evaluation showed hyperoxaluria, 24-hour urine oxalate level was 77.5 mg/day (normal range 7-44), along with significant hypocitraturia with urinary citrate excretion of 50 mg/day (normal range 116-924). Serum calcium, phosphorous and uric acid were within normal range, while parathyroid hormone was elevated, with vitamin D deficiency. This suggested an oxalate driven lithogenic mechanism. Associated hypocitraturia may have further promoted calcium oxalate crystal stone formation. This case highlights hyperoxaluria and hypocitraturia as an important metabolic contributor to post renal transplant nephrolithiasis and emphasises the need for metabolic evaluation. Early imaging, timely urology intervention, prevention of urinary tract infections, correcting metabolic and mineral bone abnormalities and optimisation of immunosuppressive therapy are essential to maintain graft function.

**Keywords:** Hyperoxaluria, Hypocitraturia, Ureteroscopy, Urinary tract infections

## CASE REPORT

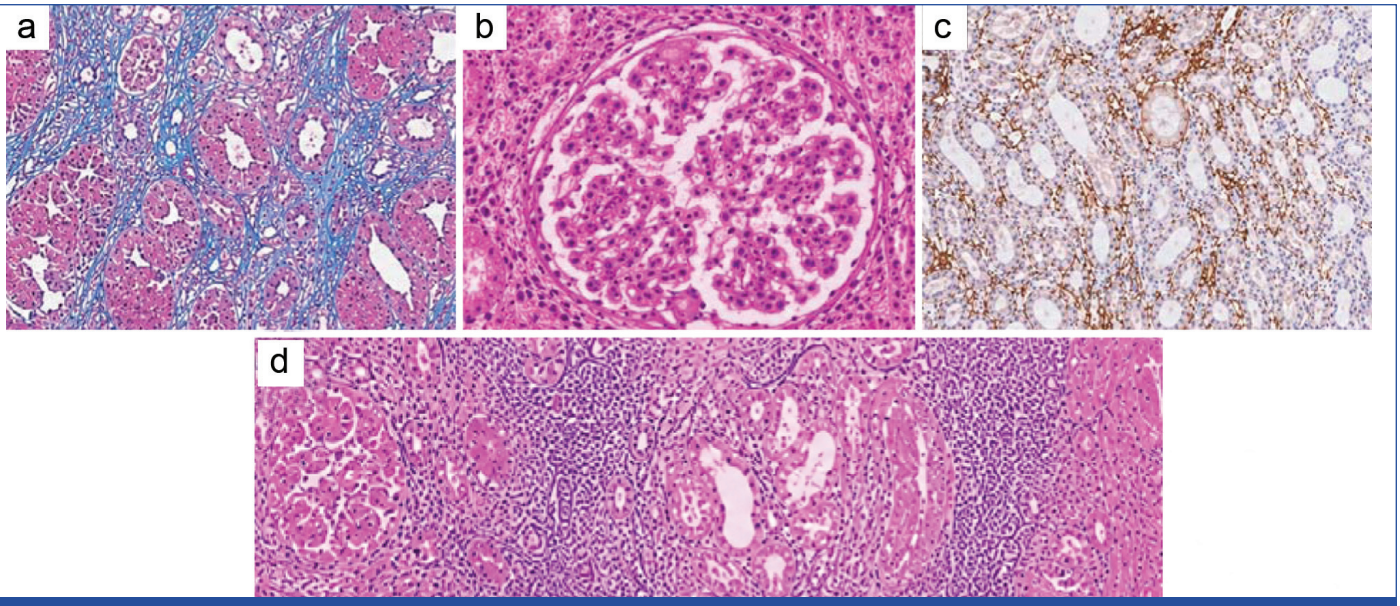
A 49-year-old male patient who initially presented in 2017 with complaints of shortness of breath, oliguria and pedal oedema. On further evaluation, he was diagnosed as end-stage kidney disease with ultrasound imaging showing bilaterally small kidneys with loss of corticomedullary difference. There was no prior history of urinary tract infection, history suggestive of renal stones. There was no family history of chronic kidney disease or nephrolithiasis. He was initiated on haemodialysis and was on twice weekly schedule with minimal residual urine output. During this period, he had no hospital admissions for any acute events. In 2018, he underwent a living related, ABO compatible renal transplantation, with spouse being the donor. He received induction with anti-thymocyte globulin. The post-transplant course was uneventful and he achieved a nadir creatinine of 1.6 mg/dL and was continued with adequate dose of immunosuppression with tacrolimus, mycophenolate mofetil and oral corticosteroids.

In 2021, he developed recurrent episodes of urinary tract infection, with acute pyelonephritis for which he received antibiotics. Radio-imaging showed acute pyelonephritis of graft kidney with no evidence of renal stones or hydroureteronephrosis. He subsequently developed graft dysfunction with increase in creatinine to 2.1 mg/dL. Allograft biopsy was done which showed 30% interstitial fibrosis and tubular atrophy, without significant evidence of acute cellular rejection, antibody mediated rejection, otherwise unremarkable. His tacrolimus trough levels were within normal range and immunosuppression was continued.

Subsequently, his graft function deteriorated with recurrent urinary tract infection. In April 2024, his creatinine increased to 6 mg/dL, he developed uremic features and following which he was initiated on haemodialysis and was put on twice weekly schedule. In August 2024 he underwent graft nephrectomy in view of recurrent chronic pyelonephritis. Histopathological examination of the explanted renal graft showed chronic pyelonephritis and chronic active antibody mediated rejection, Banff score: g2, ptc2, cg2, ci3, ct3, i2, t1, i-IFTA >50% with 30% parenchymal infarction [Table/Fig-1] [1]. He was continued on haemodialysis for one year on a thrice weekly schedule. There was no history of nephrolithiasis during this period. Mineral bone disease parameters were regularly monitored and medically managed.

In September 2025, he underwent a deceased donor, ABO compatible renal transplantation with anti-thymocyte globulin induction. Complement-dependent cytotoxicity crossmatch done prior to transplant was negative. The donor was a 40-year-old male deceased, following a road traffic accident, with no prior history of renal dysfunction, diabetes mellitus or systemic hypertension. The early post-transplant period was complicated by delayed graft function, needing haemodialysis. Suspecting antibody mediated rejection, he received plasmaphereses and intravenous immunoglobulin and there was improvement in renal function. He was discharged after 12 days, with a nadir creatinine of 1.4 mg/dL, with optimised doses of tacrolimus, oral steroids and mycophenolate mofetil as immunosuppressive medication along with prophylaxis using Valganciclovir and trimethoprim-sulfamethoxazole.

One-month post-transplant, he developed complaints of dysuria. Ultrasound imaging did not show any collection or features of pyelonephritis. Urine culture showed growth of *Escherichia coli* and he was treated with levofloxacin on Outpatient Department (OPD) basis. In December 2025, three months post-transplantation, he presented with left iliac fossa discomfort. His creatinine was 2.6 mg/dL [Table/Fig-2]. Non-contrast computed tomography of the abdomen revealed intrarenal calculi with a proximal ureteric stone of 5.5 mm causing mild hydroureteronephrosis [Table/Fig-3]. He underwent ureteroscopic lithotripsy with double-J stenting. Urine culture grew

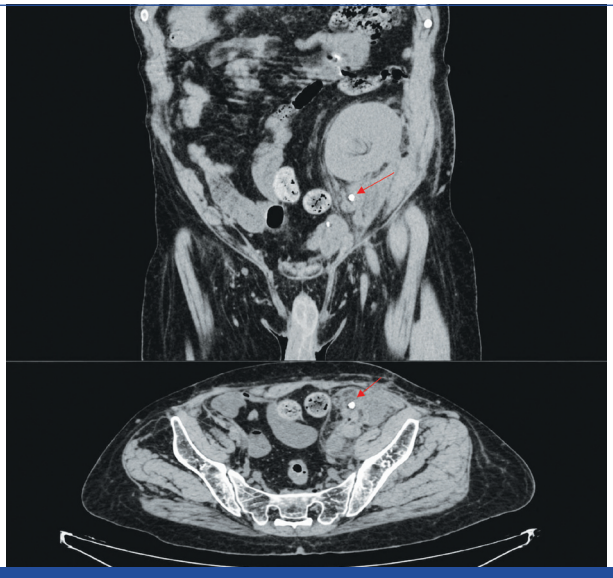



**[Table/Fig-1]:** Histopathological findings in the explanted first renal allograft; a) Severe interstitial fibrosis and tubular atrophy involving >50% of cortical area (ci3, ct3, i-IFTA >50%) (Masson trichrome, 100x); b) Transplant glomerulopathy with duplication of glomerular basement membranes (cg2) (PAS, 400x); c) Positive C4d staining in peritubular capillaries, supportive of antibody-mediated rejection (C4d IHC, 200x); d) Chronic pyelonephritis with dense chronic inflammatory infiltrates and interstitial scarring (H&E, 200x). Banff scores: g2, ptc2, cg2, ci3, ct3, i2, t1, i-IFTA >50%

*Enterococcus faecalis*, and was treated with tablet linezolid based on culture sensitivity reports. Post-procedure, his renal function improved (creatinine 1.6 mg/dL). Stone analysis was not done as stone fragments were unavailable for analysis. Metabolic evaluation including urine analysis was done four weeks after treatment of urinary tract infection are presented in [Table/Fig-4].

| Parameters | Result | Reference range |
|---|---|---|
| Creatinine (mg/dL) | 2.6 | 0.7-1.2 |
| Blood urea nitrogen (mg/dL) | 19 | 5-18 |
| Sodium (mmol/L) | 137 | 136-145 |
| Potassium (mmol/L) | 4.1 | 3.5-5.1 |
| Bicarbonate (mmol/L) | 22 | 22-29 |
| Chloride (mmol/L) | 103 | 98-107 |
| Calcium (mg/dL) | 8.8 | 8.5-10.5 |
| Phosphorus (mg/dL) | 3.3 | 2.5-4.5 |
| Uric acid (mg/dL) | 6.2 | 3.4-7 |
| Intact parathyroid hormone (pg/mL) | 181.5 | 15-65 |
| Vitamin D (ng/mL) | 20.04 | 30-100 |

**[Table/Fig-2]:** Serum biochemical parameters.



**[Table/Fig-3]:** Coronal and axial non-contrast computed tomography images showing intrarenal calculi with proximal ureteric calculus in the transplanted kidney causing mild hydroureteronephrosis.

| Parameter | Result | Reference range |
|---|---|---|
| Total urine volume (mL) | 5000 | - |
| Urine creatinine excretion (g/day) | 1.07 | 1.04-2.4 |
| Oxalate (mg/day) | 77.5 | 7-44 |
| Calcium (mg/day) | 32 | 100-300 |
| Citrate (mg/day) | 50 | 116-924 |
| Sodium (mmol/day) | 189 | 40-220 |
| Uric acid (mg/day) | 413 | 250-750 |
| Phosphorus (mg/day) | 382 | 400-1300 |

**[Table/Fig-4]:** 24-hour urine analysis.

These findings were consistent with hyperoxaluria and hypocitraturia, low urinary calcium excretion and normal uric acid levels along with vitamin D deficiency, supporting an oxalate predominant lithogenic mechanism rather than hypercalciuric or uric acid related stone disease. The patient was advised high fluid intake, dietary oxalate restriction, calcium supplementation with meals, Potassium citrate supplementation and vitamin D repletion. The elevated urine volume likely reflects preserved renal allograft function and high oral fluid intake, which is commonly encouraged in transplant recipient's with nephrolithiasis. At 6-month follow-up, serum creatinine remained stable at 1.5 mg/dL with no further episodes of urinary tract infection.

## DISCUSSION

Renal allograft nephrolithiasis is a rare but an important complication noted after kidney transplantation, with a reported incidence of 0.2-1.7% [2]. Sandberg M et al., in one of the largest recent series involving transplant recipients reported an incidence of nephrolithiasis post renal transplant as 1.4% [3]. Often, due to denervation of the transplanted kidney, patients present atypically with graft dysfunction or urinary tract infection rather than renal colic pain [4]. The most common clinical sign in such cases is haematuria. Several structural factors such as ureteric stents, altered urinary anatomy are well associated with renal stones in post-transplanted patients [5]. Additionally, urinary tract infections are common after kidney transplantation and may contribute to infection-related calculi in susceptible patients [6]. Metabolic abnormalities such as hyperoxaluria and hypocitraturia, are an under-recognised significant risk factor in such cases [7]. Careful donor evaluation is important in such cases and any history of renal dysfunction, stone disease prior to transplantation is important.

In this case, the first renal graft dysfunction was attributed to chronic pyelonephritis and chronic antibody-mediated rejection, needing

nephrectomy. The patient had no history of renal stones. During the second transplantation, which was from a deceased donor, there was no history of renal dysfunction, urinary tract infection or renal stones. The patient underwent a second transplantation and had a history of delayed graft function, in suspicion of active antibody mediated rejection, plasmapheresis and intravenous immunoglobulin was given, after which he achieved a stable graft function. He developed episodes of urinary tract infection which were managed with antibiotics and conservative treatment. Three months post transplantation he developed renal stones with mild hydroureteronephrosis and following urological intervention, his graft function had stabilised. In this case, the presence of hyperoxaluria associated with hypocitraturia and low urinary calcium excretion supports an oxalate-predominant lithogenic mechanism rather than hypercalciuria or uric acid related stone disease. Associated hypocitraturia may further promote calcium oxalate crystal aggregation and stone formation. Since there was no history of renal stones prior to the second transplantation, hyperoxaluria was thought to be due to a secondary cause rather than primary hyperoxaluria.

Several transplant specific factors contribute to nephrolithiasis in renal allografts. Hyperoxaluria, hypocitraturia, recurrent urinary tract infection, hyperparathyroidism, hypercalciuria, and reduced graft function are recognised contributors to stone formation in allograft kidney. Prolonged use of antibiotics like fluoroquinolones and cotrimoxazole can cause loss of *Oxalobacter formigenes* in the gut, which decreases oxalate degradation in gut. Antibiotic-induced gut dysbiosis, due to loss of *Oxalobacter formigenes* is a recognised risk factor for hyperoxaluria [8]. The patient had history of urinary tract infection treated with antibiotics. Reduced graft function may further impair oxalate handling and contribute to crystal deposition. Reduced glomerular filtration rate may increase systemic oxalate retention and urinary supersaturation. This promotes calcium oxalate crystal deposition and stone formation. Sandberg M et al., also demonstrated lower estimated glomerular filtration rates among stone formers in transplanted kidneys [3].

Hypocitraturia further contributes to calcium oxalate stone formation by reducing the inhibition of urinary calcium crystal aggregation [9]. Vitamin D deficiency reduces intestinal calcium absorption, thereby decreasing luminal calcium available to bind dietary oxalate and potentially increasing intestinal oxalate absorption [10]. Vitamin D levels insufficiency and high parathyroid hormone levels were seen in this patient.

The patient had mildly reduced 24-hour urinary phosphorus excretion (382 mg/day), this finding was interpreted cautiously. Urinary phosphate excretion is influenced by dietary phosphate intake, renal function, vitamin D status, and tubular handling, and therefore was not considered the primary determinant of the patient's lithogenic profile. The overall metabolic evaluation was more consistent with hyperoxaluria and hypocitraturia. A diet rich in oxalate containing food like spinach, nuts, chocolate can cause hyperoxaluria [9]. Enteric hyperoxaluria is more commonly seen with fat malabsorption due to conditions like chronic diarrhoea, pancreatic insufficiency, inflammatory bowel disease or post-bariatric surgery and ileal resection. Excess vitamin C, pyridoxine deficiency is also known to increase endogenous oxalate production [11]. There was no such history in this case.

Secondary hyperoxaluria in this patient was likely multifactorial, including history of urinary tract infections, possible antibiotic induced gut dysbiosis, vitamin D deficiency resulting in reduced intestinal calcium availability and transplant related metabolic alterations. Although stone composition analysis was not available, the presence of significant hyperoxaluria with hypocitraturia supports an oxalate-predominant lithogenic mechanism. This case underscores the importance of evaluation of metabolic parameters in renal transplant recipients who are found to have nephrolithiasis. Comprehensive metabolic evaluation including 24-hour urinary oxalate and citrate assessment is therefore important in patients presenting with nephrolithiasis post renal transplant.

## CONCLUSION(S)

This case highlights hyperoxaluria and hypocitraturia as an important metabolic risk factor to post-transplant nephrolithiasis. Comprehensive metabolic evaluation, early imaging with timely urological intervention and prevention of recurrent urinary tract infections are essential to preserve the graft function of the transplanted kidney.

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**PARTICULARS OF CONTRIBUTORS:**

1. Senior Resident, Department of Nephrology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
2. Professor and Head, Department of Nephrology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
3. Assistant Professor, Department of Nephrology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
4. Senior Resident, Department of Nephrology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.
5. Senior Resident, Department of Nephrology, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India.

**NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:**
Dr. Aalaya Haridas,
112, First Leaf Villas, Hyderabad, Telangana, India.
E-mail: haridasaalaya@gmail.com



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