

Case Report

Ureteroscopy combined with percutaneous nephroscopic balloon expansion alleviates ureteral obstruction caused by xanthogranulomatous pyelonephritis in ureteropelvic junction of transplant kidney

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Abstract: Post renal transplant ureteral obstruction is uncommonly found at the ureteropelvic junction of the renal graft. Xanthogranulomatous pyelonephritis rarely occurs in renal graft recipients, leading to worsening of renal graft function. Here, we report a case of xanthogranulomatous pyelonephritis associated with ureteral obstruction in the ureteropelvic junction of the transplant kidney. *E. coli* grew in a urine culture. Color Doppler ultrasonography showed a renal graft 13.5 × 7.2 × 7.0 cm in size, an abnormal suprarenal echo 7.0 × 8.0 cm in size and pelvic dilation (1.9 cm). The renal allograft function was improved by appropriate antibiotics therapy and urinary diversion by percutaneous nephrostomy insertion. Percutaneous balloon dilatation followed by temporary antegrade ureteric catheter placement was performed as definite treatment. The function of the patient's renal graft was recovered. In conclusion, xanthogranulomatous pyelonephritis is a rare cause of renal allograft dysfunction and can be associated with ureteral obstruction. Early diagnosis, appropriate antibiotic therapy and urinary diversion by percutaneous nephrostomy insertion can improve the function of the renal allograft, allowing balloon dilatation and ureteric stent insertion.

Keywords: Renal transplant, obstructive nephropathy, xanthogranulomatous pyelonephritis, nephoscopy balloon expansion

Introduction

Ureteral obstruction is a common urological complication following renal transplantation and threatens the survival of the renal graft [1]. Therefore, early diagnosis and prompt alleviation of ureteral obstruction are critical to an uneventful post transplant outcome [2]. Obstruction at the ureteropelvic junction of the renal graft is uncommon as the proximal ureter is supplied by small branches from the main renal artery.

Xanthogranulomatous pyelonephritis is a severe, chronic bacterial infection of the kidneys and is characterized by destruction of the renal parenchyma and the presence of granulomas, abscesses and lipid-laden macrophages [3]. Xanthogranulomatous pyelonephritis

can be easily misdiagnosed for renal malignancy or renal tuberculosis and surgically resected. Xanthogranulomatous pyelonephritis has also been reported in renal graft recipients [4-6]. However, there has been no report on xanthogranulomatous pyelonephritis associated with obstruction of the ureteropelvic junction in graft recipients. Here, we report one case of xanthogranulomatous pyelonephritis associated with ureteral obstruction of the renal graft that was successfully managed by appropriate antibiotics therapy and percutaneous balloon dilatation followed by temporary antegrade ureteric stent placement.

Case report

A 51-year old male underwent cadaveric renal allograft transplantation at our hospital in

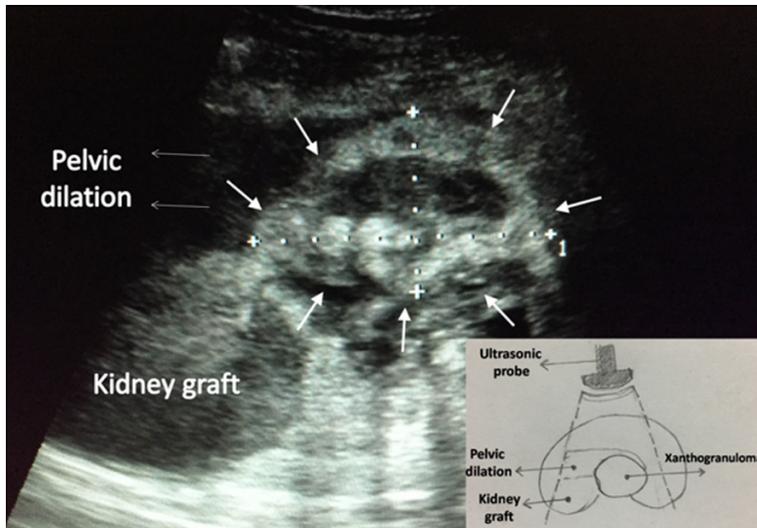


Figure 1. Preoperative color Doppler ultrasonography in a 51-year-old male who developed acute renal failure 6 months after receiving cadaveric renal allograft transplantation. Ultrasonography reveals a renal graft $13.5 \times 7.2 \times 7.0$ cm in size, an abnormal suprarenal echo 7.0×8.0 cm in size and pelvic dilation (1.9 cm). Arrows indicate granuloma in the renal allograft before treatment.

August 2014. His postoperative immunosuppressive therapy included cyclosporine, mycophenolate mofetil and prednisolone. The graft recipient had an uneventful postoperative recovery with a daily urine output of 2000 to 3000 mL. Six months post transplant, the recipient felt a dwindling urine output (1000 mL/day), and acute renal failure developed (serum creatinine, $260 \mu\text{mol/L}$). *E. coli* grew in a urine culture. Color Doppler ultrasonography showed a renal graft $13.5 \times 7.2 \times 7.0$ cm in size, an abnormal suprarenal echo 7.0×8.0 cm in size and pelvic dilation (1.9 cm) (**Figure 1**).

To protect renal graft function, we performed percutaneous nephrostomy insertion and drained a diurnal urine volume of 3000 mL. Serum creatinine returned to normal four days later. Percutaneous antegrade pyelography was carried out to further understand the cause of hydronephrosis of the renal graft and disclosed narrowing of the ureteropelvic junction while the lower part of the ureter was well visualized and had no apparent dilatation. Abdominal multislice CT scan by triple phase enhancement showed patchy inhomogeneous enhancement, which was approximately 7.0×7.8 cm in size, in the upper course of the pelvis and ureter (**Figure 2**). The lesions surrounded blood vessels in the renal sinus, the pelvis and

the ureter. Renal graft biopsy showed that the renal parenchyma was largely destroyed and replaced with numerous lipid laden macrophages and a moderate amount of lymphocytes and scant erythrocytes and necrotic tissues. The capillary loops of the renal corpuscles were well perfused, and there was mild proliferation of mesangial cells and the mesangial matrix, but no marked thickening of the basement membrane was observed (**Figure 3A** and **3B**). No infiltration of inflammatory cells was seen in the glomeruli. Though no apparent atrophy of the renal tubules was observed, vacuolation of renal tubule epithelial cells was observed and numerous lymphocytes and lipid laden macro-

phages were dispersed in the interstitium. No fibrosis was present in the interstitium. Immunohistochemistry revealed CD10 (-), vimentin (+), CK (-), CD68 (+), p63 (-), CK-7 (-), and SV40 (-) (**Figure 3C** and **3D**). The patient was diagnosed with xanthogranulomatous pyelonephritis and ureteral obstruction of the renal graft.

The patient was given meropenem (0.5 g, three times daily). Meanwhile, a stent was placed in the ureter of the renal graft to alleviate obstruction. Percutaneous balloon dilatation was carried out using a F4.0 STORZ rigid ureteroscope and a 15 cm hydrophilic guidewire. The guidewire was passed through the working channel of the ureteroscope, which was then introduced into the urinary bladder via the urethra. After the anterior wall of the bladder was observed and the opening of the renal graft ureter was located, the hydrophilic guidewire was advanced into the ureter of the renal graft in a retrograde manner. Intraoperative roentgenography confirmed entry of the guidewire into the renal graft pelvis; however, we failed to advance the ureteroscope into the ureter because of the unique position of the ureter opening. Instead, the ureteroscope was advanced into the renal pelvis via the percutaneous nephrostomy insertion. A foreign body pincer was then introduced via the ureteroscope to retract out of the body

Xanthogranulomatous pyelonephritis in transplant kidney

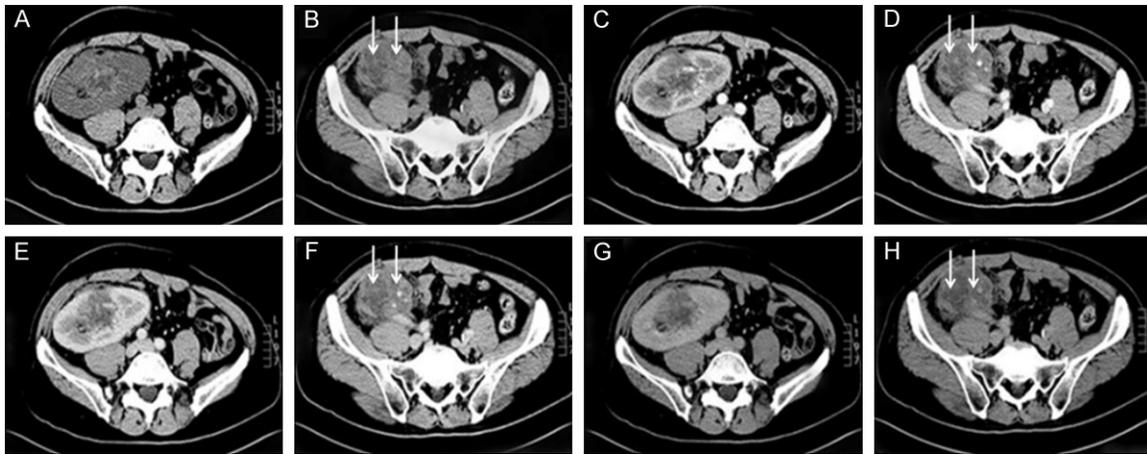


Figure 2. Abdominal multislice CT scan by triple phase enhancement reveals patchy inhomogeneous enhancement, 7.0 × 7.8 cm in size, in the upper course of the pelvis and ureter. The lesions surround the blood vessels in the renal sinus, the pelvis and the ureter. A and B: Plain CT scan; C and D: The cortex phase; E and F: The parenchyma phase; G and H: The excretion phase.

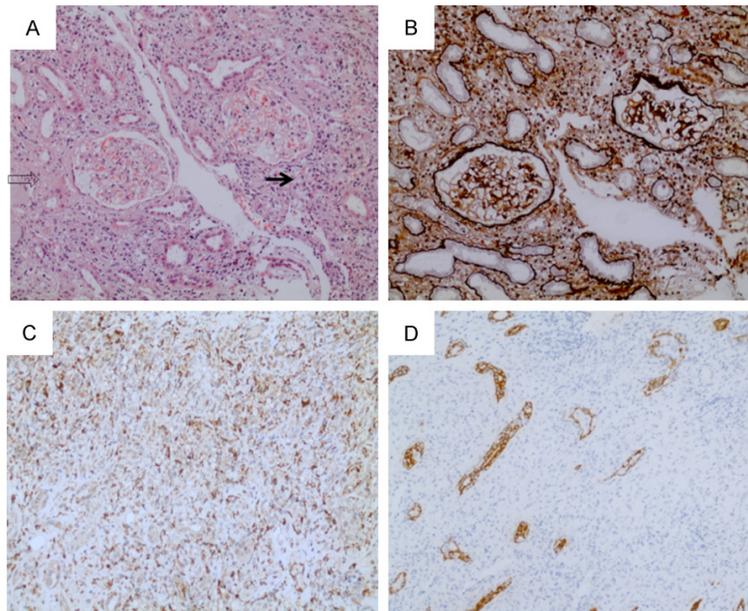


Figure 3. Pathological examination of the renal graft biopsy specimen shows that the renal parenchyma is largely destroyed and replaced with numerous lipid laden macrophages and a moderate amount of lymphocytes and scant erythrocytes and necrotic tissues (A: H&E staining, ×200, lymphocytes and scant erythrocytes are indicated by arrows; hollow arrows indicate the necrotic tissues). The capillary loops of the renal corpuscles are well perfused, and there is mild proliferation of mesangial cells and the mesangial matrix, but no marked thickening of the basement membrane is observed. No infiltration of inflammatory cells is seen in the glomeruli (B: PASM staining, ×200). IHC staining of CD68 shows that most of interstitial infiltrating cells are positive, suggesting that they are macrophages (C: Immunostaining for CD68, ×200). At the same time, IHC staining of CK is negative in interstitial infiltrating cells (D: Immunostaining for CK, ×100).

the proximal end of the guidewire while the distal end of the guidewire remained outside of

the urethra. After the guidewire was tightened from both the proximal and distal end and kept in place, an X-Force U30 ureter balloon dilation stent (length of balloon, 6 cm) was cannulated into the hydrophilic guidewire and advanced superiorly. Under roentgenography guidance, the balloon was positioned in the ureteropelvic junction of the renal graft and the balloon was inflated and maintained at 2.8×10^3 kPa for 15 min. Thereafter, the balloon stent was withdrawn and a double-J stent was advanced from the proximal end of the hydrophilic guidewire and under roentgenography guidance the upper end of the double-J stent was positioned within the renal graft pelvis and the lower end within the urinary bladder. Meropenem (0.5 g, three times daily) was continued for 10 days postoperatively.

The percutaneous nephrostomy insertion stent was removed three days after surgery.

The ureteric catheter was removed five months after placement. The patient was followed up

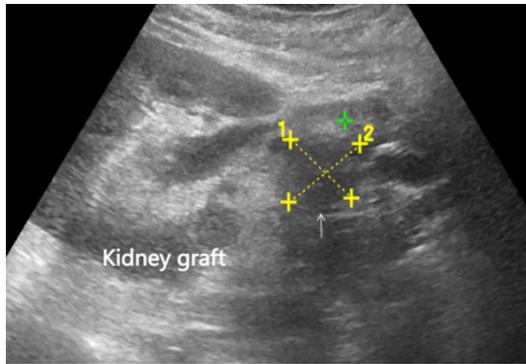


Figure 4. Renal ultrasonography at the 24 month follow up visit shows no pelvic dilation and the suprarenal echo is reduced to 3.0 × 2.7 cm in size. Arrow indicates granuloma in the renal allograft post treatment.

for 36 months and the renal graft functioned normally at the time of writing up this report, with serum creatine maintained at 20-130 μmol/L and renal ultrasonography showed no pelvic dilation and the suprarenal echo was reduced to 3.0 × 3.0 cm in size (**Figure 4**).

Discussion

Ureteral obstruction is a common complication of renal transplantation that may threaten the survival of the renal graft. Prompt and correct identification of the cause of ureteral obstruction and rapid alleviation of obstruction are important to a successful outcome. In this paper, we report a rare case of obstruction of the ureteropelvic junction in a renal graft recipient. The unique feature of this reported case is the association of renal allograft xanthogranulomatous pyelonephritis with ureteral obstruction. Post renal transplant ureteral obstruction may ensue as a result of errors in surgical technique such as retaining an unnecessary long ureter [7], periureteral fibrosis, viral infections, renal ischemia or graft rejection [8, 9] and may occur in any part of the ureter, with the distal ureter close to the ureterovesical junction being the most frequent site of obstruction. Rarely does ureteral obstruction occur in the ureteropelvic junction as in the current case. Though there are occasional reports of xanthogranulomatous pyelonephritis in renal allograft recipients [4, 10], our case is the first reported case of obstruction of the ureteropelvic junction associated with xanthogranulomatous pyelonephritis in a renal allograft.

Xanthogranulomatous pyelonephritis is a chronic bacterial infection of the kidneys and uri-

nary tract obstruction and urinary calculi are considered to be predisposing factors [11-13]. Xanthogranulomatous pyelonephritis rarely occurs in renal allografts and fewer than ten cases have been reported [6, 10, 14]. Our patient underwent renal allograft transplantation six months prior to the onset of xanthogranulomatous pyelonephritis. We speculated that obstruction of the ureteropelvic junction, if present prior to the onset of xanthogranulomatous pyelonephritis, may be a predisposing factor for xanthogranulomatous pyelonephritis. Another likely predisposing factor was altered immunologic competence in the patient because of immunosuppressive therapy. The clinical manifestations of xanthogranulomatous pyelonephritis were nonspecific in our case: a febrile patient with a failing renal graft. The diagnosis was suggested by a positive urine culture for *E. coli* and established by histological findings consistent with xanthogranulomatous pyelonephritis [15].

Though treatment with antibiotics may be successful for xanthogranulomatous pyelonephritis [10], because of the presence of ureteric obstruction in our case, we opted for urinary diversion by percutaneous nephrostomy insertion to minimize renal damages. Furthermore, appropriate antibiotics based on sensitivity data were prescribed. These measures led to improved renal function in the patient. We then performed percutaneous balloon dilatation and ureteric stenting for definite treatment, which, together with antibiotics therapy, led to the recovery of renal function in the patient. Options for post renal transplant ureteral obstruction include ureteroneocystostomy with excision of the stenotic segment and re-implantation, ureteroureterostomy using the recipient ipsilateral ureter, or balloon dilatation. In the current case, because obstruction occurred at the ureteropelvic junction, ureteroneocystostomy or ureteroureterostomy would be invasive and surgically challenging and ureteral obstruction would also highly likely recur. In addition, because the ureter of the renal graft is typically implanted in the superior portion of the urinary bladder, it is extremely difficult to locate the ureter opening under a ureteroscope and to carry out catheterization in a retrograde manner.

In conclusion, xanthogranulomatous pyelonephritis is a rare cause of renal allograft dysfunction and can be associated with ureteral

obstruction. Early diagnosis, appropriate antibiotic therapy and urinary diversion by percutaneous nephrostomy insertion can improve the function of the renal allograft, allowing balloon dilatation and ureteric catheter insertion for definite treatment. The case reported herein is the first case of xanthogranulomatous pyelonephritis associated with ureteral obstruction at the ureteropelvic junction in the renal allograft that was successfully treated with antibiotic therapy and balloon dilatation and ureteric stenting.

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Disclosure of conflict of interest

None.

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