Obstructive Uropathy due to Bilateral Sliding Hernia in a Renal Transplant Patient with Incidental RCC in Native Kidney

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ABSTRACT

BACKGROUND: Ureteral obstruction is a common complication after kidney transplantation. Ureteral obstruction caused by inguinal hernia, however, is a rare complication of transplantation and requires urgent surgical repair to prevent allograft loss.

CASE PRESENTATION: A 58-year-old man presented with allograft dysfunction 18-years after renal transplant. He was compliant with medications and given the long duration of allograft survival, a primary renal etiology was suspected. Thus, the initial work-up included allograft biopsy that was unremarkable. Three months later, worsening allograft function prompted further evaluation. At this time, allograft ultrasound and computed tomography led to the diagnosis of ureteral obstruction due to uretero-inguinal herniation of left kidney transplant secondary to bilateral sliding inguinal hernias. The patient was also found to have incidental renal cell carcinoma of the left native kidney. A percutaneous nephrostomy tube was placed and then followed by surgical repair with ureteral reimplantation, herniorrhaphy with mesh, and left native nephrectomy.

CONCLUSIONS: Mechanical obstruction can occur years after kidney transplantation. Even though it is uncommon, ureteral obstruction due to inguinal herniation is critical. Early detection of this complication and surgery can salvage the allograft and prolong function.

ABBREVIATIONS: RCC: renal cell carcinoma; PCN: Percutaneous Nephrostomy; ACKD: Acquired Cystic Kidney Disease

KEYWORDS: kidney transplantation, ureteral obstruction, inguinal hernia, uretero-inguinal hernia, renal cell carcinoma

BACKGROUND

Ureteral obstruction is a common complication of renal transplantation with ureteral stones, vesico-uretero reflux, infection, and rejection being the most common causes. Most cases occur early after transplantation and are related to surgical causes or ischemic strictures. However, uretero-inguinal hernia causing ureteral obstruction is a rare complication following kidney transplant with the most common causes being redundancy of transplant ureter and anterior positioning of the ureter in relation to the spermatic cord. ^{1,2,3} The orientation of the kidney allograft may play a significant part in location of the ureter, with upward orientation of the renal hilum resulting in superficial location of the ureter, close to the inguinal canal and susceptible to herniation.⁴ Other possible risk factors include male sex and obesity; herniation can present a decade or more delay between kidney transplantation and presentation.

CASE PRESENTATION

A 58-year-old Caucasian man with history of hypertension, end stage kidney disease secondary to IgA nephropathy, now 18 years post second live donor kidney transplant to the left iliac fossa, presented with decreased urine output for two days, poor oral intake and nausea.

He was maintained on a triple immunosuppression regimen of prednisone, tacrolimus and azathioprine. He had enjoyed stable allograft function with baseline serum creatinine of 1.5 mg/dl until a few months before his presentation, when creatinine on routine labs rose to 2.6 mg/dL. A diagnostic allograft biopsy showed frequent peritubular capillaritis, severe arteriosclerosis, diffuse arterial hyalinosis, mild to moderate interstitial fibrosis, and tubular atrophy with negative C4d staining. Donor specific antibodies were negative and donor derived cell-free DNA was < 0.12% (normal). No radiological imaging was done at that time. With the biopsy findings, there were no changes made to his immunosuppressive medications.

Upon subsequent presentation, blood work at an outside hospital showed serum creatinine of 5.2 mg/dl, and allograft ultrasound showed hydronephrosis. He denied fever, urinary frequency, dysuria, pain at the transplant site or hematuria. Upon his transfer to our institution, vital signs revealed blood pressure of 130/80 mm/Hg, heart rate 74 bpm, saturating 99% on room air with body mass index of 24.5. Physical exam was unremarkable with no abdominal allograft tenderness on palpation and no evidence of hernia. Laboratory studies were remarkable for sodium 126 mEq/L, potassium 5 mEq/L, bicarbonate 13 mEq/L, anion gap 19, BUN 100 mg/dL with serum creatinine of 7.4 mg/dl. Urinalysis was



negative for blood and protein, negative for leukocyte esterase, negative for nitrite and had no evidence of red blood cells. Urine culture was negative for infection. BK virus PCR was undetectable.

We repeated allograft ultrasound at our institution, confirming severe hydroureteronephrosis of the transplant kidney extending to the uretero-vesicular junction (Figures 1,2).

Allograft function continued to worsen over the next day with creatinine peaking at 10.5 mg/dl and oliguria with urine output dropping to 50 ml per day. Computed tomography of the patient's abdomen and pelvis without IV contrast revealed severe hydronephrosis of the left lower quadrant transplant kidney, secondary entrapment of the ureter following herniation into the left inguinal canal, herniation of the urinary bladder into the right inguinal canal with circumferential urinary bladder wall thickening (**Figure 3**), and two suspicious native left renal masses concerning for renal cell carcinoma (**Figure 4**).

Antegrade transplant nephrostogram demonstrated severe left transplant hydronephrosis with abrupt ureter obstruction in the inguinal canal. Glidewire was able to pass the obstruction into the bladder, however a 4 french catheter could not pass the obstructed ureter. Percutaneous nephrostomy (PCN) was placed with brisk urine output of 3.7 L and improved allograft function down to 2.3 mg/dl over the next few days (**Figures 5,6**).

Figure 1,2. Ultrasound image of the transplanted kidney showing severe hydronephrosis with hydroureter.



Two weeks later, he underwent mobilization and reimplantation of the left transplant ureter, left herniorrhaphy with mesh and left native radical nephrectomy. The left PCN tube was removed. Given the extent of the surgery and the lack of symptoms, the small sliding right inguinal hernia was not intervened upon. One week after surgery, a complete recovery of his allograft function was achieved and creatinine recovered to its baseline of 1.5 mg/dl.

Figure 3. Abdominal computed tomography with sagittal view showing severe hydroureteronephrosis with uretero-inguinal herniation (arrow) and bladder herniation (star).



Figure 4. Abdominal computed tomography with axial view showing the left two renal masses in the left native kidney (arrow).





Figures 5,6. Anterograde nephrostogram demonstrating obstruction in the distal ureter at the level of the inguinal hernia (arrow); Figure 6. Percutaneous nephrostomy placement (arrow).



Pathologic examination of the left native renal masses demonstrated a 3.1 cm mass in the upper pole, a 2.1 cm mass in the middle pole with 2 additional microscopic foci (< 5mm). histology revealed papillary renal cell carcinoma (RCC) type 1 with free ureteral, vascular, and perinephric margins and without lymph nodes invasion (T1aN0Mx).

After 2 years of follow-up, the allograft function remains stable at 1.5 mg/dl while maintained on the same immunosuppressive regimen using prednisone, tacrolimus and azathioprine.

DISCUSSION

Our case represents an unusual and underdiagnosed cause of obstructive uropathy due to ureteral and bladder entrapment in bilateral sliding hernias in a nonobese man 18 years after kidney transplantation. After decompression of the hydroureteronephrosis with percutaneous nephrostomy, reimplantation of the left ureter was performed in addition to left herniorrhaphy. Subsequently, he had complete recovery of the allograft function. Imaging should have been obtained with the initial presentation of renal transplant dysfunction. It is likely that this represented the early signs of partial ureteral obstruction. Case reports have described this complication over the past few decades, but this is the first to demonstrate bilateral sliding inguinal hernias, complete ureteral obstruction and concomitant management of renal cell cancer as we have described.

Ureteral entrapment in a hernia is rare. It was first described in 1880 with less than 140 cases reported in the

literature.6 Uretero-inguinal hernias are more common in men, typically in the fifth and sixth decades of life. Many cases occur in patients with a history of kidney transplant given the anterior location of the transplanted ureter within the space of Retzius. It occurs more commonly on the right than the left side.^{5,7} Pre-operative management with nephrostomy tube insertion with or without antegrade ureteric stent was most frequently employed first for immediate decompression of the collecting system to prevent irreversible graft dysfunction.8-12 Hernia repair and herniorrhaphy are usually performed.¹³⁻¹⁶ This complication should be considered in the differential diagnosis as one considers transplant ureteral obstruction as a cause of acute kidney injury in a renal allograft.¹⁷ There is a possible role for elective repair of inguinal or incisional hernias in renal transplant patients.¹⁸

Our case illustrates the additional incidental findings of two small, low-grade papillary renal cell carcinomas of the left native kidney that were surgically removed with left radical nephrectomy. Asymptomatic RCC in this case could have been easily missed if radiological imaging of the abdomen was not done.

The risk of the development of *de novo* RCC in renal transplant recipients is 15–100 times higher than the general population. Due to the long-term use of immunosuppressive medications, transplant recipients are at increased risk of malignancies, with skin cancers and non-Hodgkin lymphoma the most common cancers after kidney transplantation.¹⁴ Carcinoma of the native kidney accounts for less than 5% of all malignancies found in transplant recipients with the incidence ranging between 0.3–4.8%. The median time of the kidney transplant to the development of RCC is reported to be 4 to 5 years after transplant.²⁰⁻²²

Multiple risk factors for *de novo* RCC after kidney transplant have been identified, including acquired cystic kidney disease (ACKD), male sex, African-American races, older age (65 years and older), those with a longer pretransplant dialysis interval, a donor aged at least 50 years, immunosuppressive medications, and microscopic hematuria.^{20,21} Early detection has better prognosis with the consideration of screening all transplant patients for ACKD and RCC.²³ As in our case, RCCs of native kidneys are more frequently incidental findings, low-grade and with overall favorable prognosis.¹⁰ Nephrectomy is the main treatment as it is curative for most cases without metastases and without compromising the graft function by stopping immunosuppressive medications.^{21,24,25,26}

There are no guidelines for RCC screening in kidney transplant recipients. While the American Society of Transplantation and the Kidney Disease Improving Global Outcomes Clinical Practice Guidelines for the Care of the Kidney Transplant Recipient do not recommend routine screening for renal cancer after kidney transplant, the European Association of Urology recommend annual screening of native



and transplant kidney for the detection of renal tumors.²⁷⁻²⁹ These additional screenings may help with incremental findings of malignancies that, in transplant recipients, might be advantageous with early diagnosis and treatment.

CONCLUSION

Mechanical obstruction of a renal allograft may develop years after transplantation and should be considered in the differential diagnosis of renal transplant dysfunction. This case emphasizes the need for imaging in patients with renal transplant dysfunction as part of the complete workup for prerenal, intrinsic renal and post renal causes of acute allograft injury. Awareness of transplant uretero-inguinal herniation as a cause of acute allograft dysfunction is important as early surgical intervention is critical to avoid allograft loss. Despite bilateral sliding inguinal hernias and complete ureteral obstruction, allograft salvage was accomplished by ureteral reimplantation and unilateral inguinal herniorrhaphy.

Furthermore, RCC in kidney transplant recipient is usually detected incidentally with the possibility of being present in the recipient's own kidneys before transplant. The possible role of native renal radiological screening for patients undergoing kidney transplantation is unclear, but nevertheless the imaging might be helpful when the studies are done for other clinical scenarios. Although more robust guidelines are needed for routine nonspecific screening and detection of RCC in renal transplant recipient, the relative cost to benefit ratios must be weighed clinically as well as economically.

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