

Intraperitoneal Urine Leak Following Renal Transplant: The Role of Radionuclide Imaging

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CASE PRESENTATION

A 26-yr-old woman with a history of chronic renal failure and a cadaver renal transplant presented with complaints of weakness and dizziness. One year prior to the present admission the patient was started on hemodialysis for biopsy-proven membrano-proliferative glomerulonephritis. One month later she was converted to peritoneal dialysis. Except for several episodes of peritonitis, she had done well. Three weeks prior to the current admission a cadaver renal transplant was performed without complication. Postoperatively she remained anuric. A renal scan was performed the next day (Fig. 1). The scan demonstrated mildly decreased perfusion and poor function suggestive of acute tubular necrosis (ATN). Following the renal scan, she was treated with OKT-3, Imuran, prednisone, and peritoneal dialysis. Over the next four days, the patient had minimal urine output, but her creatinine decreased from 18.6 on admission to 15.1. A follow-up renal scan was performed (Fig. 2). The perfusion at this time had markedly improved, but no definite excretion of radiotracer into the collecting system was seen. The findings were again felt to be consistent with ATN.

Peritoneal dialysis and immunosuppressive therapy were continued. The patient continued to have no urine output, and, two days following the second renal scan, she began to complain of abdominal cramping. A renal scan was performed two days later (Fig. 3). Perfusion was again felt to be normal. Although there was no definite visualization of the collecting system, activity within the pelvis was interpreted as representing bladder. It was felt that there was a slight improvement in

function but that ATN persisted. One day later, the patient had an episode of hypotension with a blood pressure of 80/40. Intravenous hydration was initiated. The blood pressure improved but the patient continued to complain of abdominal cramping.

Over the next several days, she had no urine output and her creatinine remained between 11 and 15. It was thought that she had severe ischemic damage to the transplanted kidney. OKT-3 therapy was discontinued and cyclosporine was started. The patient was sent home on peritoneal dialysis. Three days later she was readmitted with persistent abdominal cramping, diarrhea, and anuria. Her past medical history is notable for toxemia of pregnancy in 1982 and asthma. Her medications at the time of admission included prednisone, Imuran, cyclosporine, and atenolol. On physical examination, she was an obese black female with a systolic blood pressure of 74. Her pulse was 90 and her respiratory rate was 18. Her lungs were clear. Heart rate was regular and she had no murmurs or rubs. Her abdomen was soft but diffusely tender without rebound or guarding. Her chronic abdominal peritoneal dialysis catheter was in place. The incision site from the transplant was clean and dry. Her skin turgor was poor.

Laboratory tests revealed a hemoglobin of 10.4 and a hematocrit of 33.0. Her white blood cell (WBC) count was 12,700. BUN was 63 and creatinine was 11.4. Calcium was 11.2 and phosphate was 8.3. She was thought to be volume depleted secondary to diarrhea. Fluids were administered intravenously and the dialysis fluid was cultured. Following rehydration, the patient's blood pressure rose to 120/70. A renal biopsy performed two days after admission revealed moderate acute rejection. It was elected to treat her with six daily doses of 500 mg of intravenous methylprednisolone. Over the next five days, the patient felt well but continued to be anuric. It was felt that ATN might mask the response to steroids. Meanwhile, her serum creatinine began to fall steadily at a rate of ~ 1 mg/dl/day to 8 mg/dl. Analysis of the dialysate revealed a creatinine of 20, indicating that urine was present in the fluid.

An antegrade pyelogram under ultrasound guid-

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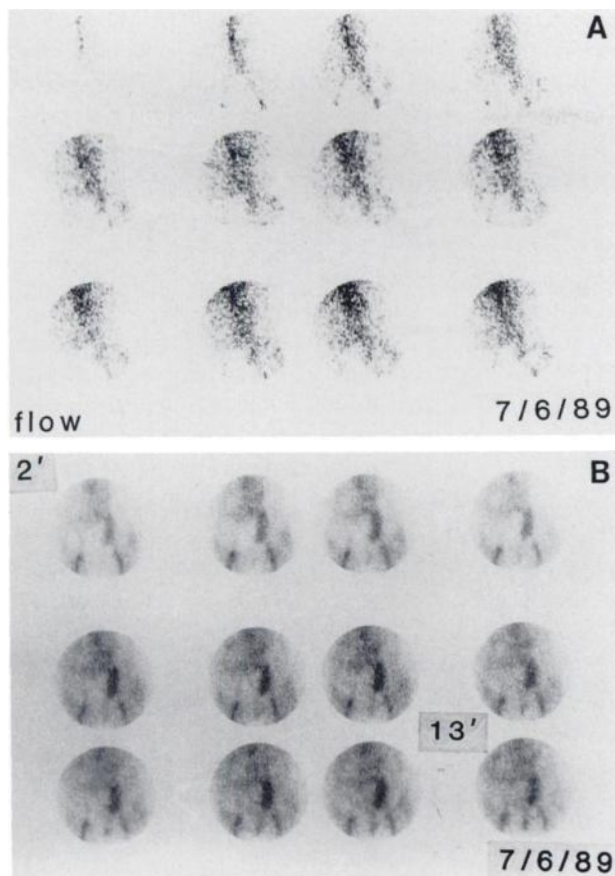


FIGURE 1

Renal scan: ^{99m}Tc -DTPA. This renal scan, performed one day post-transplant, demonstrates slightly delayed perfusion and nonvisualization of the collecting system. These findings are suggestive of acute tubular necrosis. The area of photon deficiency adjacent to the kidney was felt to represent peritoneal dialysis fluid.

ance was unsuccessful. There was no hydronephrosis present but a fluid collection surrounding the transplanted kidney was noted. This was felt to most likely represent intraperitoneal dialysis fluid (Fig. 4). One day later, indigo carmen dye was administered intravenously. The dye was noted to be present in the fluid coming out of the peritoneal dialysis catheter, indicative of an intraperitoneal leak. A follow-up renal scan was performed that day, one week after admission (Fig. 5). This scan demonstrated activity spreading diffusely across the lower pelvis and up the sides of the abdomen on decubitus views indicating a urine leak.

DISCUSSION

This case is instructive in the use of renal scans and the complications of renal transplantation. In our institution, renal scanning is performed routinely on the first postoperative day. Occasionally, if there are concerns, the scan is performed as soon as the patient leaves the recovery room to determine the perfusion to the graft. Absence of perfusion usually signifies arterial

or venous occlusion or, rarely, hyperacute rejection (1). In general, if there is no perfusion to the graft, it has been irretrievably lost. Unlike normal kidneys, the allograft has no collateral circulation that could maintain viability of the organ if the main artery or vein is occluded. If there is no perfusion on the radionuclide scan, the patient is typically returned to the operating room as soon as possible with the usual outcome being allograft nephrectomy.

The most common cause of primary dysfunction of the graft with the maintenance of good perfusion is acute tubular necrosis. It occurs in ~40% of all cadaveric grafts but in less than 5% of grafts from living donors. This discrepancy is probably due to the shorter cold storage time for the graft from a living donor and the guaranteed excellent health of the donor. In ATN, blood flow to the graft is well preserved but function is decreased or absent (2). The pathophysiology of ATN is not well understood despite 40 yr of study. Four theories have been put forth to explain it: (1) blood is shunted away from filtering nephrons, (2) there is a decrease in hydraulic permeability of the glomerular filtering membrane due to injury, (3) sloughing of tubular epithelial cells allows the glomerular filtrate to

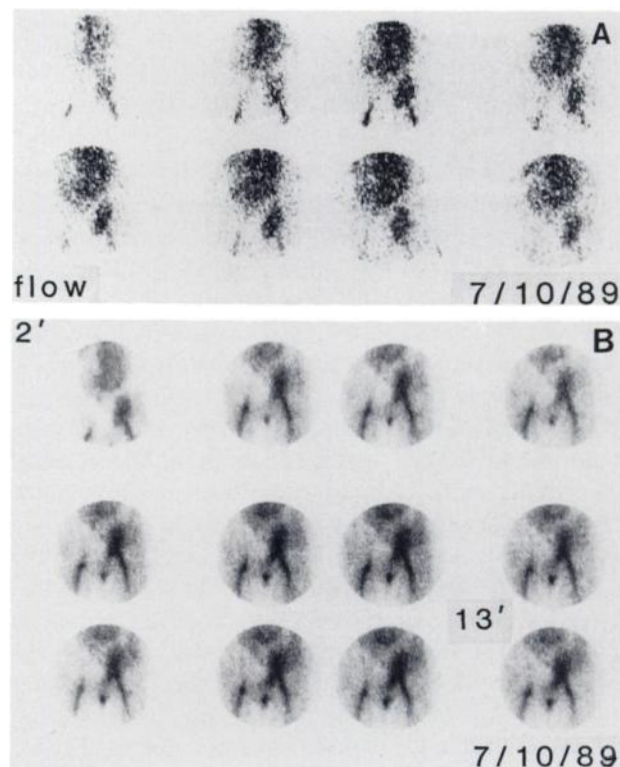


FIGURE 2

Renal scan: ^{99m}Tc -DTPA. Four days post-transplant, the renal scan shows improved perfusion. Again, there is no definite excretion of the radiotracer into the collecting system. These findings are indicative of continuing ATN. The activity in the midline of the lower pelvis represents normal vascularity of the perineum.

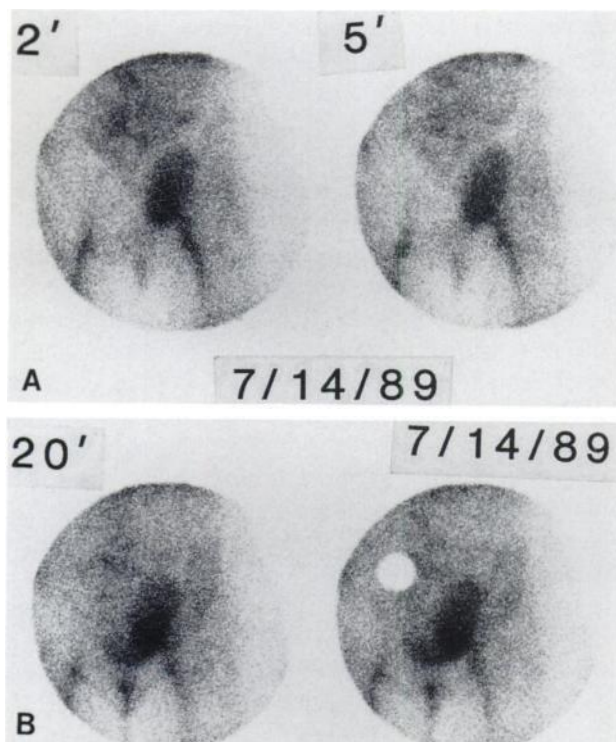


FIGURE 3

Renal scan: ^{99m}Tc -DTPA. There is now prompt perfusion to the transplanted kidney but no distinct visualization of the collecting system. Radioactivity is clearly seen in the pelvic region and was thought to represent bladder. This finding was felt to indicate that there was a slight improvement in renal function but that ATN was still present.

diffuse back into the blood stream, and (4) sloughed epithelial cells mix with tubular proteins normally present forming casts which obstruct the tubules. None of these theories fit all the clinical and experimental observations.

Other complications that can be discovered by radionuclide scanning are ureteral obstruction and extravasation of urine (2). These disorders occur in less than 5% of renal transplants and are usually manifest more than one week after surgery. The most common cause of ureteral obstruction or a urinary leak is ischemia of the ureter or renal pelvis. Native ureters have a triple blood supply with vessels from the renal pedicle, the adjacent lumbar arteries, and the urinary bladder. The last two vascular supplies are lost when the kidney is removed from the donor. The vascular supply to the ureter from the renal pedicle is tenuous, at best, and easily damaged. With ureteral ischemia, the ureter becomes fibrotic and obstructed or breaks down and leaks. Ureteral ischemia usually becomes evident one week or more after surgery. However, in the immediate postoperative period, there may be obstruction because the uretero-neocystostomy is too "tight." Alternatively, the ureter may pull loose from the bladder when the kidney is positioned and the wound is closed.

There are a number of renal diseases, including focal segmental glomerulosclerosis, IgA nephropathy (Berger's disease), anti-glomerular basement membrane disease (Goodpasture's syndrome), and membrano-proliferative glomerulonephritis which can recur in renal allografts and cause decreased function. This patient had membrano-proliferative glomerulonephritis. However, these illnesses are not known to recur in the immediate postoperative period such that they might cause dysfunction of the graft.

Even though this patient's transplanted kidney had ATN with poor function, it had a relative good blood supply and was, therefore, subject to rejection. The cornerstone of immunosuppression today is cyclosporine; however, it is a nephrotoxic drug which may prolong the course of ATN or even totally prevent eventual recovery of function (3). For that reason, many programs do not start using cyclosporine until the kidney is functioning. Instead, early on, prednisone and azathioprine are used, often with the addition of a monoclonal or polyclonal heterologous serum directed against T-lymphocytes. In this case, OKT-3, a monoclonal antibody directed against the CD-3 marker found on all T-lymphocytes, was used. This drug has been shown to be effective in the prevention of rejection; acute rejection is very unusual while OKT-3 is being administered (4). However, since OKT-3 is a potent immunosuppressive drug, it cannot be used indefinitely due to the risk of infection. After 14 days of therapy with OKT-3, the patient was switched to cyclosporine.

Since the repeat scans appeared similar to the first, it was concluded that the patient had prolonged ATN from which she might recover, given time. Studies have shown that ~ 10% of cases of ATN may take 4 wk or longer to heal. Some may never recover.



FIGURE 4

Ultrasound coronal pelvis. The ultrasound demonstrates a normal appearing kidney in the left iliac fossa. No hydronephrosis is present. A fluid collection is seen inferior and medial to graft.

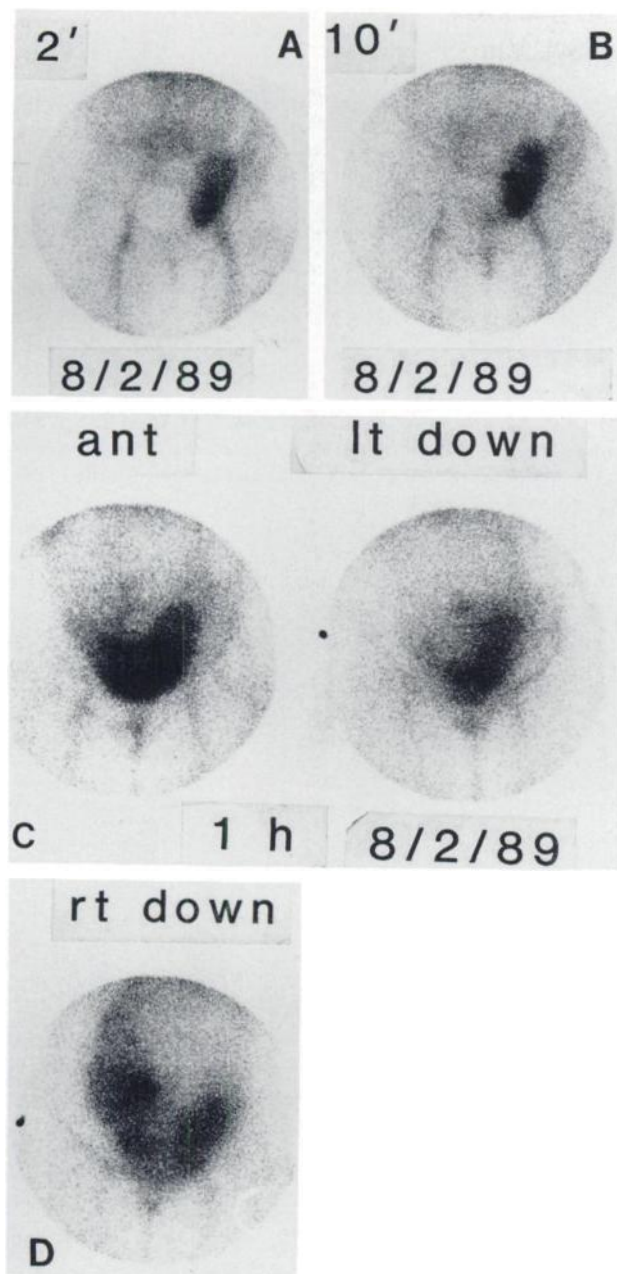


FIGURE 5
Renal scan: ^{99m}Tc -DTPA. There is mildly delayed blood flow to the renal graft with good concentration of the radiotracer. Intrarenal transit is delayed until 7 min postinjection. The activity spreads diffusely across the lower pelvis and, on decubitus views, tracks laterally up into the abdomen. This finding is strongly suspicious for a urine leak.

A biopsy performed during the patient's subsequent hospital stay showed acute cellular rejection. Since the course of OKT-3 had been completed only a few days earlier, it was elected to treat her with high-dose intravenous methylprednisolone. Though she remained anuric during this time, her serum creatinine began to fall about 1 mg/dl/day. Therefore, one must suspect that there had been improvement in renal function. But where was the urine? The kidney, as with most renal

transplants, is located in the retroperitoneum. The ultrasound showed no hydronephrosis and intraperitoneal fluid is not unexpected in a patient on peritoneal dialysis.

The creatinine level of the dialysate was higher than that of the serum. This can only mean that urine is present in the dialysate. This, in addition to the indigo carmine test and the final renal scan, indicates that there is communication between the kidney and the peritoneal cavity.

There are three mechanisms which can allow urine to enter the peritoneal cavity. If the kidney was placed in the peritoneum, as is done occasionally when the kidney is large and the recipient is small, any leak of urine would collect in the peritoneum. This is not the case here. Second, a urine leak occurring in a patient with a tear in the peritoneum may allow this to occur. However, if this were the case, dialysis fluid would have leaked through the peritoneal tear and out of the patient's incision. This did not occur. The third and best explanation is that the ureter had been attached to the peritoneum rather than the bladder. This is a rare error, and has happened one other time in our experience with almost 1,500 transplants.

PATHOLOGY

The patient was taken back to the operating room immediately following the final renal scan. It was discovered that the ureter of the transplanted kidney had been implanted into a "tongue" of peritoneum folded over the bladder. A ureterocystostomy was performed. The patient tolerated the procedure well.

CONCLUSION

The traditional surgical method for connecting the transplanted ureter to the recipient's bladder is called the "Politano-Leadbetter" technique (5). In that procedure, the bladder is opened, the ureter is pulled through a tunnel created obliquely through the bladder wall, the ureter is sewn into place from inside the bladder, and the cystostomy incision is closed. This procedure has a couple of disadvantages. First, a substantial length of ureter is needed. Second, the bladder is an extremely well vascularized organ and bleeds profusely. In the past decade, the extravesical uretero-neocystostomy or "Lich procedure" has become popular and is used by many transplant surgeons (6). In this procedure, the bladder is filled with saline through a Foley catheter. The bladder muscularis is dissected until the mucosa appears in the wound or a gush of saline appears. The ureter is sewn into place from outside the bladder and the muscularis is closed over the distal ureter for ~ 2 cm creating a non-refluxing tunnel. The

Lich procedure has the advantages of causing far less bleeding than the Politano-Leadbetter procedure, is faster, and a shorter length of ureter can be used.

There are several reasons why the ureter may have been sewn into the peritoneum rather than the bladder. First, the patient was quite obese; the surgeon was working in a deep hole with poor exposure. Second, the peritoneum overlying the bladder had been markedly thickened by prior episodes of peritonitis. Even though the peritoneal cavity had been drained of dialysis solution, residual fluid remained in dependent areas, e.g., around the bladder. The bladder was filled with saline causing the adherent fold of peritoneum to rise into the field. The thickened peritoneum was dissected until a gush of saline (in this case dialysis fluid) appeared and the ureter was sewn into place. Following the correct placement of the ureter, the patient had an immediate diuresis, a falling serum creatinine level, and was discharged five days later. When seen in clinic, 4 mo after

the final initial surgery, she was doing well with a serum creatinine level of less than two.

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