Bladder Rupture in Immediate Postrenal Transplant Period of Uncertain Cause

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Abstract

Bladder rupture in patients undergoing renal transplant is rare. A 26-year-old man underwent a deceased-donor renal transplant. Postoperatively, he showed a good clinical course, but after removal of the urethral catheter, he complained of pain in the lower aspect of the abdomen and in the left flank. Findings of abdominal computed tomography and retrograde cystography revealed an extraperitoneal bladder rupture. We did not identify specific causes for this condition and believe that the bladder rupture was spontaneous. However, we could not rule out the possibility of traumatic rupture by the double-J ureteral stent. We decided to manage this case conservatively, with an indwelling urethral catheter and antibiotics, instead of by surgical repair. Results of repeated serial cystography during the treatment showed decreased contrast extravasation, and cystographic findings at 6 weeks showed no leakage of contrast medium. We confirmed complete healing of the ruptured bladder and removed the urethral catheter. Since then, the patient has maintained good renal function without any complications.

Key words: Computed tomography, Retrograde cystography, Ureteral stent, Urethral catheter, Urologic complication

Introduction

Although the incidence of urologic complications in renal transplant has decreased over the past decades, they remain an important cause of posttransplant morbidity and mortality, with the incidence rate recently reported to be between 10% and 12.5%.1, 2 Various types of urologic complications have been described in many studies, but bladder rupture is rare. Here we report an unusual case of bladder rupture after renal transplant, which was treated successfully through conservative management.

Case Report

A 26-year-old man with end-stage renal disease secondary to IgA nephropathy underwent a renal transplant on July 27, 2010. He had been dependent on hemodialysis for 4 years and in an anuric state for 3 years. Preoperative examination revealed normal chest radiographic and electrocardiographic results. Laboratory studies found the following values: hemoglobin, 118 g/L (11.8 g/dL); white blood cells, 6.43 × 10^9/L (6430/mm^3); platelets, 206 000 × 10^9/L (206 000/mm^3); sodium, 134 mmol/L (134 mEq/L); potassium, 5.0 mmol/L (5.0 mEq/L); serum urea nitrogen, 25.8 mmol/L (72.4 mg/dL); and creatinine, 1242 μmol/L (14.05 mg/dL).

During the operation, the left kidney from a deceased donor was transplanted into the right iliac fossa of the recipient. The renal artery and vein of the donor kidney was anastomosed to the external iliac artery and vein of the recipient in an end-to-side fashion. The ureter of the donor kidney was anastomosed to the right side of the recipient’s bladder using an antireflux technique. A double-J ureteral stent (7F, 24 cm, Urosoft, Angiomed, Bard, Karlsruhe, Germany) was inserted by ureteroneocystostomy. There were no immediate surgical complications.
Postoperatively, the patient had good urine output, and his creatinine level gradually fell to 139.7 μmol/L (1.58 mg/dL) on the seventh postoperative day. He received an immunosuppressive regimen of basiliximab, mycophenolate mofetil, tacrolimus, and prednisone. On postoperative day 7, the urethral catheter was removed. The next day, the patient complained of pain in the left lower quadrant of the abdomen and the left flank region. The pain was aggravated during voiding, and his creatinine level rose to 219.2 μmol/L (2.48 mg/dL).

To evaluate the cause of pain and decreased renal function, we performed imaging studies. Color Doppler ultrasonography findings revealed no substantial abnormality except fluid collection around the transplanted kidney located in the right iliac fossa. However, results of nonenhanced abdominal computed tomography (CT) revealed a defect at the left lateral wall of the bladder and fluid collection in the left pelvic extraperitoneal space. The CT also showed that a part of the ureteral stent had pulled out from inside the bladder into the pelvic space through the left lateral wall defect (Figure 1A).

We believed that the CT finding was indicative of bladder rupture and urine leakage, and retrograde cystography was performed via the urethral catheter. The cystography showed contrast medium leaking into the extraperitoneal space from the left lateral bladder wall (Figure 1B). Through the result of this imaging study, we concluded that the cause of pain was bladder rupture. For treatment of the bladder rupture, the ureteral stent was removed.

We decided to manage the patient conservatively by inserting an indwelling urethral catheter for diversion of the urine hoping for spontaneous healing, and antibiotics were administered intravenously to the patient. One week after insertion of the urethral catheter, the patient’s symptoms improved, and the serum creatinine level fell to 106.1 μmol/L (1.20 mg/dL). The patient was discharged with the indwelling urethral catheter in place and receiving a regimen of oral antibiotics.

Follow-up cystography was performed 2 and 4 weeks later, and the results showed decreased but persistent leakage of contrast medium. However, cystography performed after 6 weeks showed no leakage of contrast medium and complete healing of the bladder rupture (Figure 2). The urethral catheter was removed, and the patient has remained asymptomatic ever since.

**Discussion**

Several types of urologic complications are possible after renal transplant. The major urologic complications are usually associated with an implanted ureter, such as ureteral leakage and stricture. Other minor complications include bladder outlet obstruction, recurrent urinary tract infection, and hydroceles. Among these complications, bladder rupture is rare, and only 1 case has been reported, which described spontaneous bladder rupture in the recipient of a transplanted kidney that had cytomegalovirus infection.

In our case, there was no definite causative factor for the bladder rupture, but 2 possible causes were considered. First, there was the possibility that the bladder rupture was spontaneous. Spontaneous bladder rupture is a relatively rare condition, which occurs in the absence of bladder trauma and is known to be associated with bladder tumors, radiation injury, ureterovesical stones, urinary catheterization, postpartum patients, atonic bladder, normal saline irrigation, and binge alcohol consumption.
consumption. In our case, the patient’s bladder had contracted because he had been in an anuric state for 3 years at the time of renal transplant, and the bladder rupture was found after removing the indwelling urethral catheter. We believed that sudden increased pressure inside the bladder after removing the urethral catheter might be associated with spontaneous rupture of the contracted bladder.

Second, trauma on the bladder wall caused by the ureteral stent might lead to bladder rupture. This possibility is based on the CT finding showing that a part of the ureteral stent had pulled away from the bladder and entered the pelvic cavity. However, such a possibility was considered to be unlikely based on our clinical experience, and we thought that the ureteral stent migrated into the pelvic cavity through a bladder wall defect after spontaneous bladder rupture. Nevertheless, we could not completely rule out the possibility of traumatic bladder rupture by the ureteral stent.

Many transplant centers (including ours) use ureteral stents routinely to prevent urologic complications. Although routine placement of ureteral stents in a renal transplant is controversial, many reports support the cost-effectiveness of routinely using ureteral stents. If a bladder rupture in our case truly resulted from trauma caused by the ureteral stent, this emphasizes that close attention should be given when placing a ureteral stent. This is underscored by the fact that many transplant centers favor the routine use of ureteral stents.

Diagnosing bladder rupture is often difficult, especially during the postrenal transplant period, because the symptoms are vague and nonspecific. Renal transplant recipients with lower abdominal pain, dysuria, and decreased renal function should be carefully examined. There is no criterion standard imaging modality for diagnosing bladder rupture. Abdominal CT, coupled with retrograde cystography, has been reported to be the method of choice. In our case, we initially performed abdominal CT to differentially diagnose the patient’s abdominal pain. Bladder rupture was suggested by the CT findings and was confirmed by results of retrograde cystography.

Generally, bladder rupture is categorized into intraperitoneal and extraperitoneal ruptures. Intraperitoneal rupture is associated with high mortality and morbidity, and usually requires surgical repair. On the other hand, most cases of extraperitoneal ruptures can be managed conservatively with transurethral catheter drainage and prophylactic antibiotics. In our case, we used conservative management. There were several reasons for administering conservative treatment. First, bladder rupture was confined to the extraperitoneal space. Second, the patient’s clinical condition was relatively good. Third, the risk of needing reoperation was considered relatively high during the early transplant period.

In conclusion, bladder rupture after renal transplant is rare. We experienced a case of unexpected bladder rupture in a renal transplant recipient. We could not identify a specific cause of bladder rupture, but the patient was successfully treated through conservative management. This case highlights 2 points. First, bladder rupture should be considered in the differential diagnosis in the renal transplant recipient with abdominal pain and decreased renal function. Second, conservative treatment can be successfully applied to a renal transplant recipient with bladder rupture according to the type of bladder rupture and the patient’s condition.

References