

Relapsing Urinary Tract Infection Due to Rectourethral Fistula in a Renal Transplant Recipient

Böbrek Nakli Alıcısında Rektoüretral Fistüle Bağlı Tekrarlayan İdrar Yolu Enfeksiyonu

ABSTRACT

Objectives: Urinary tract infection (UTI) is the most common cause of bacterial infection in renal transplant recipients. It occurs frequently in the early period because of the high-dose immunosuppressive agents and urethral catheterizations. Relapsing UTI may lead to graft dysfunction and further evaluations have to be performed for predisposing factors. We report the case of a renal transplant recipient who presented with relapsing bacterial UTI due to a rectourethral fistula.

Case: A 24-year-old male patient underwent a successful renal transplantation from a living donor on May 2008. He had a history of surgical intervention for anal atresia and rectourethral fistula. He was hospitalized five times because of relapsing bacterial UTI after transplantation. We investigated the presence of an anatomical abnormality and found a rectourethral fistula. After the surgical repair of the fistula the UTI did not relapse.

Conclusion: Relapsing infections are not uncommon and anatomical abnormalities can lead to relapsing UTI in transplant recipients. Further investigations must be performed regarding the factors that might contribute to the development of UTIs in the presence of relapsing UTI.

KEY WORDS: Renal transplantation, Rectourethral fistula, Urinary tract infection

ÖZ

Giriş: Böbrek nakli alıcılarında bakteriyel enfeksiyonların en sık nedeni idrar yolu enfeksiyonudur (İYE). Yüksek doz immün baskılayıcı ilaç kullanımı ve üreter kateterizasyonu nedeni ile sıklıkla nakil sonrası erken dönemde görülür. Tekrarlayan İYE graft işlev bozukluğuna neden olabilir ve bu durumda kolaylaştırıcı nedenler açısından ileri inceleme yapılması gerekmektedir. Yazımızda, rektoüretral fistüle bağlı tekrarlayan bakteriyel İYE olan bir böbrek nakli alıcısı olgu sunulmuştur.

Olgu: Anal atrezi ve rektoüretral fistül nedeni ile cerrahi operasyon öyküsü olan 24 yaşında erkek hastaya Mayıs 2008’de başarılı canlı böbrek nakli uygulandı. Nakil sonrası tekrarlayan İYE nedeni ile 5 kez hastaneye yatırışı oldu. Anatomik anormallik açısından yapılan incelemelerde rektoüretral fistül tespit edildi. Fistülün cerrahi onarımından sonra hastanın takiplerinde İYE tekrarlamadı.

Sonuç: Anatomik anormallikler tekrarlayan İYE’na neden olabilir ve sıklıkla karşımıza çıkabilir. Tekrarlayan İYE varlığında altta yatan kolaylaştırıcı faktörlerin belirlenmesi için ileri incelemeler yapılmalıdır.

ANAHTAR SÖZCÜKLER: Böbrek nakli, Rektoüretral fistül, İdrar yolu enfeksiyonu

INTRODUCTION

Urinary tract infections (UTIs) are the most frequent post-transplantation infections. They are associated with significant graft dysfunction and relapse (1). Despite a high

recurring infection rate, uncomplicated UTI has a good prognosis (2). Some factors might contribute to the development of relapsing UTIs. We report a renal transplant recipient who presented with relapsing bacterial UTI due to a rectourethral fistula.

Ayşegül ORUÇ KOÇ¹
Alparslan ERSOY²

- 1 Yozgat State Hospital, Department of Nephrology, Yozgat, Turkey
- 2 Uludağ University Faculty of Medicine, Department of Nephrology, Bursa, Turkey



Received : 15.01.2014

Accepted : 22.04.2014

Correspondence Address:

Ayşegül ORUÇ KOÇ
Yozgat Devlet Hastanesi,
Nefroloji Bölümü, Yozgat, Turkey
Phone : +90 506 204 93 50
E-mail : aysegul13072@yahoo.com

CASE

A 24-year-old male patient underwent a successful renal transplantation from a living donor on May 2008. He had been on the hemodialysis program for 3 years because of end-stage renal disease with a lower urinary tract anomaly. He had undergone a surgical intervention for anal atresia on his 2nd day of life. Retrograde urethrography revealed a rectourethral fistula when he complained of anal incontinence and aberrant drainage of urine from the anus when five years old. The fistula was repaired with a three-stage procedure (sigmoid loop colostomy, fistula repair, colostomy closure) 17 years ago. During his follow-ups, end-stage renal disease was diagnosed when he was 20 years old and a hemodialysis program was initiated. He had 1000-1500 cc diuresis during hemodialysis and no history of repeated UTI. Pre-transplant investigations did not reveal a fistula. He underwent a successful renal transplantation from a living donor on May 2008. Posttransplant immunosuppressive treatment consisted of basiliximab, prednisolone, mycophenolate mofetil and cyclosporine. He was discharged with a serum creatinine of 1.6 mg/dL on the 6th day. There was no surgical complication or a polyuric period during hospitalization. He had no complaint at the first month but his laboratory tests revealed a leukocyte count of 14,020 per mm³. Serum creatinine was 1.7 mg/dL. Urine analysis showed 30 leukocytes per HPF. Culture of the urine sample indicated *Escherichia coli* that was sensitive to first line antibiotics. He was treated with 10 days of oral ciprofloxacin. The ureteric stent was then removed. Asymptomatic sterile pyuria continued during his follow-up. On the 4th month of transplantation he presented at the emergency service with fever, diarrhea and pain over the graft region. On physical examination his temperature was 38.8 °C, and he had tenderness over the graft region. He had leukocytosis with a total white cell count of 18,600 per mm³ and the differential count showed 86% polymorphs, 10% lymphocytes and 4% band forms. Urine analysis revealed 32 leukocytes per HPF. Serum creatinine was 4.4 mg/dL. Cyclosporine 0 hour trough level was 173 ng/dL. Graft Doppler ultrasonography showed good anastomotic flow with no evidence of urinary tract obstruction. Renal scintigraphy revealed only delayed excretion and cyclosporine was switched

to tacrolimus. Blood and urine cultures grew *E. coli*. After parenteral hydration and ceftriaxone treatment for 14 days because of the pyelonephritis diagnosis, the urine culture became negative. Serum creatinine level decreased to 2.1 g/dl. He was then hospitalized 5 times because of pyelonephritis in a 5 month period and extended-spectrum beta-lactamase positive (ESBL +) *E. coli* grew in urinary cultures each time. All the episodes were treated with meropenem for 14 days and he was discharged from hospital with negative urine cultures. Serum creatinine levels showed fluctuations between 2.2 to 3.1 mg/dl during these attack periods (Table I). Because of the relapsing UTIs, further investigations were performed to look for any predisposing factors and especially anatomic abnormalities as soon as possible in the infection-free period. Renal ultrasonography and scintigraphy were performed during all the episodes and revealed no anatomic abnormality. Uroflowmetry performed at the 7th month of transplantation was normal and showed 50 cc residual urine. Retrograde urethrography revealed a rectourethral fistula at the 8th month of the kidney transplantation. The patient underwent a rectourethral fistula repair. The pyuria disappeared after the surgery and the UTI did not relapse during the follow-up (Figure 1A,B).

DISCUSSION

Post-transplant UTI occurs frequently in the early postoperative period. The incidence of UTI after renal transplantation varies from 6% to 86%. Factors that might contribute to the development of UTIs are related to the graft recipient, prolonged period of hemodialysis before grafting, duration of catheterization, history of UTI before transplantation, renal allograft, anatomical features of the recipient (VUR, PCKD), female sex, diabetes mellitus and the infection-causing organism (3, 4). Post-transplant UTI is also frequent in patients with preexisting bladder or ureterovesical junction dysfunction managed by cutaneous ureterostomy or bladder augmentation. *E. coli* is the most common microorganism and is involved in 29% to 60% of cases (5). Our patient had multiple episodes of UTI in the first year after transplantation. All the episodes were caused by *E. coli*. These episodes were associated with significant graft dysfunction, which recovered once the infection

Table: The outcomes of patient during hospitalizations.

Hospitalization day	Creatinine BT (mg/dl)	Creatinine AT (mg/dl)	Urine culture	Treatment
1.1.	4.4	2.1	<i>E. coli</i>	Ceftriaxone/14d
1.2.	2.4	2.0	ESBL(+) <i>E. coli</i>	Meropenem/14d
1.3.	2.5	2.0	ESBL(+) <i>E. coli</i>	Meropenem/14d
1.4.	3.1	2.2	ESBL(+) <i>E. coli</i>	Meropenem/10d
1.5.	2.6	2.1	ESBL(+) <i>E. coli</i>	Meropenem/14d

BT: Before treatment, **AT:** After treatment.

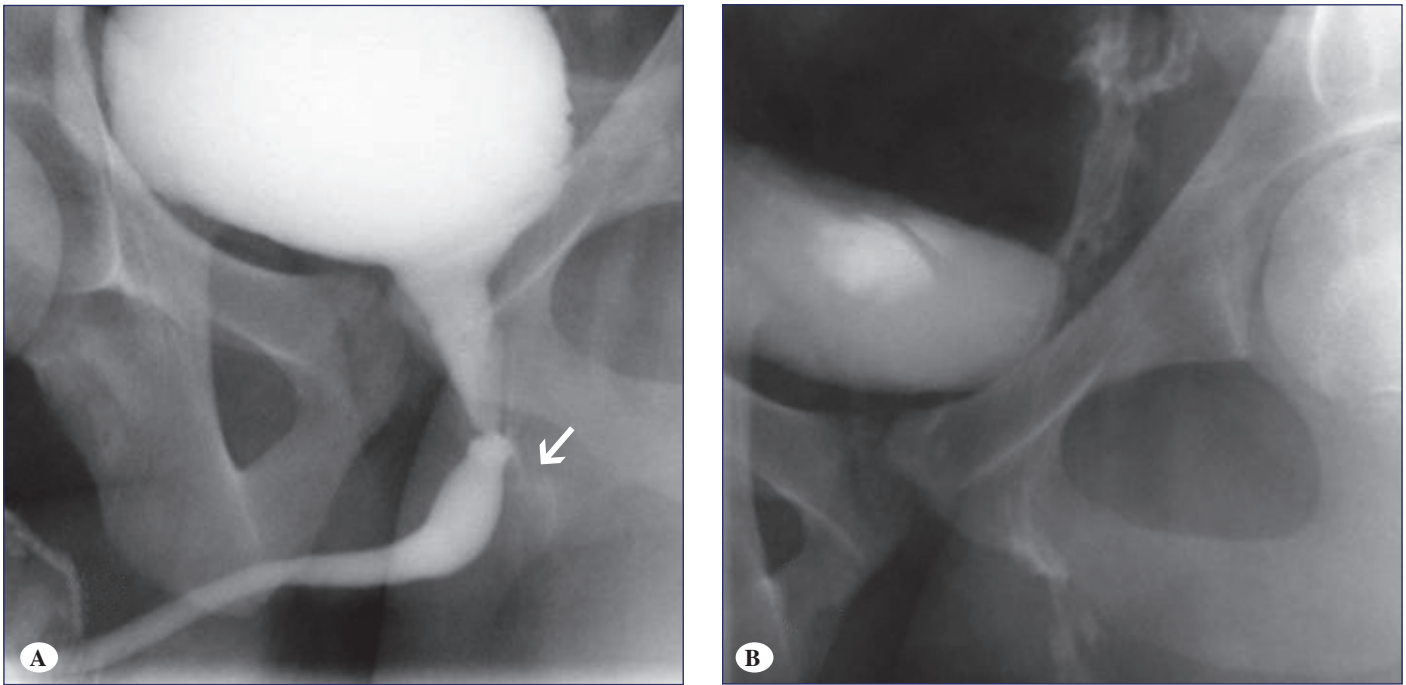


Figure 1: Retrograde urethrography images of the fistula. **A)** Fistula tract that begins from the membranous urethra, **B)** Contrast artifacts in the bowel loop(s).

was controlled. We excluded acute rejection, drug toxicity, obstructive or vascular abnormalities with the clinical course and imaging investigations. Although we removed the ureteric stenting, which might be associated with complications such as UTI and reflux (5), the UTIs attacks continued.

Relapsing UTI is defined as UTI that results from a failure to eradicate the original infection. Functional or anatomical abnormalities such as a stone, obstructive uropathy, and ureterovesical junction complications must be excluded in the presence of relapsing UTI. The most common findings are ureteral reflux, strictures at the ureterovesical junction, and neurogenic bladder. Further investigation is recommended to find out possible risk factors such as anatomical abnormalities in patients who have relapsing UTI (3). We performed further investigations after the 3rd attack because of the lower urinary tract (LUT) abnormality and surgery history. Renal ultrasonography and scintigraphy were performed for all the episodes and revealed no anatomic abnormality. Retrograde urethrography done due to the previous history of rectourethral fistula proved the recurrence of the same anatomic abnormality in the urinary tract. Our patient had no history of significant UTI before transplantation. He also had a predisposing anatomical abnormality and the immunosuppressive treatment might have facilitated UTI episodes.

Anomalies of the LUT can be associated with upper urinary tract damage and is responsible for renal failure in 20-30% of children with end-stage renal disease. These anatomical

abnormalities increase the risk of UTIs after transplantation. However, several studies have reported that the graft survival for patients undergoing reconstruction of a LUT anomaly is comparable to those with a normal urinary tract (6). Imperforate anus is a relatively common congenital anomaly in children. Fistulous communications between the blind rectum and urinary tract are common in boys with high anomalies (7). Boys with an anorectal malformation therefore must be investigated for fistulas. Rectourethral fistulas may be congenital or acquired. Acquired etiologies include inflammatory, neoplastic or traumatic factors (8). Our patient had a LUT anomaly that was responsible for renal failure. He was born with an imperforate anus and later a fistula was recognized and repaired. Colostomy, fistula repair and colostomy closure, called a three-stage procedure, is the conventional procedure for rectourethral fistulas. Our patient had also undergone the three-stage procedure when the fistula was first recognized. Experiences in surgery, standardized bowel preparations and effective antibiotics have diminished the enthusiasm for colostomy. Effective single procedures have recently been reported in the literature. Colostomy is recommended when antibiotics alone cannot control the inflammation and infection associated with the fistula (8, 9). After transplantation, the recurrent rectourethral fistula was repaired with a successful single-stage procedure in our patient. The single stage procedure was used for the rectourethral fistula repair as there were no signs of inflammation. We chose this minimal invasive procedure because our patient was an immunocompromised.

In conclusion, UTI is an important and preventable cause of morbidity, mortality and graft failure in recipients. Anatomic urinary tract abnormalities can be a predisposing factor for relapsing UTI. Additional investigations must be performed to find the predisposing anatomical abnormality that might lead to relapsing UTI.

REFERENCES

1. Chuang P, Parikh CR, Langone A: Urinary tract infections after renal transplantation: A retrospective review at two US transplant centers. *Clin Transplant* 2005;19:230-235
2. Sqalli TH, Laboudi A, Arrayhani M, Benamar L, Amar Y, Ouzeddoun N, Bayahia R, Rhou H: Urinary tract infections in renal allograft recipients from living related donors. *Saudi J Kidney Dis Transpl* 2008;19:551-553
3. de Souza RM, Olsburgh J: Urinary tract infection in the renal transplant patient. *Nat Clin Pract Nephrol* 2008;4:252-264
4. Schmaldienst S, Dittrich E, Hörl WH: Urinary tract infections after renal transplantation. *Curr Opin Urol* 2002;12:125-130
5. Ranganathan M, Akbar M, Ilham M, Chavez R, Kumar N, Asderakis A: Infective complications associated with ureteral stents in renal transplant recipients. *Transplant Proc* 2009;41:162-164
6. Djakovic N, Wagener N, Adams J, Gilfrich C, Haferkamp A, Pfitzenmaier J, Toenshoff B, Schmidt J, Hohenfellner M: Intestinal reconstruction of the lower urinary tract as a prerequisite for renal transplantation. *BJU Int* 2008;103:1555-1560
7. Daradka I, Hazza I: The effect of rectourogenital fistula in high imperforate anus. *Saudi J Kidney Dis Transpl* 2007;18:186-190
8. Hanus T: Rectourethral fistulas. *Int Braz J Urol* 2002;28:338-345
9. Zheng S, Xiao X, Huang Y: Single-stage correction of imperforate anus with a rectourethral or a rectovestibular fistula by semi-posterior sagittal anorectoplasty. *Pediatr Surg Int* 2008;24:671-676