De novo urethral stricture disease in renal transplant recipients

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ABSTRACT

Introduction: With routine catheterization and low urine output pre-transplant, renal transplant recipients (RTRs) may be at risk of urethral stricture disease post-transplant. The objective of this study was to characterize new urethral stricture disease in males following renal transplant.
Methods: Retrospective chart review was carried out on all male RTRs at Vancouver General Hospital who developed urethral strictures from October 2009–2019. Descriptive analyses were conducted on patient characteristics. Comparative analyses against non-stricture RTRs were carried out.
Results: Of 636 RTRs, 18 (2.8%) developed a postoperative urethral stricture. Median time from transplant to stricture discovery was 56 days (range 8–618 days). One-third of stricture patients had prior risk factors for stricture formation. Post-transplant, 77.8% presented symptomatically, with 61.1% requiring intervention. Overall graft survival rate was 88.9% among the RTR stricture group; 16.7% experienced acute rejection and 22.2% had delayed graft function (DGF). There was no significant association between developing postoperative urethral stricture and urinary tract infection (Chi-squared [X^2]=0.04,

KEY MESSAGES

- Urethral stricture disease has a higher prevalence among postoperative renal transplant patients compared to the general population.
- Stricture disease should be considered post-transplantation in patients with voiding dysfunction, even without prior risk factors.
- From our study, urethral strictures do not appear to affect graft survival, although multicenter studies are needed to elucidate any relationship between urethral stricture and graft survival.

p=0.84; odds ratio [OR] 0.81, 95% confidence interval [CI] 0.1–6.21), DGF (X^2=0.14, p=0.70; OR 0.8, CI 0.26–2.48), or acute rejection (X^2=2.02, p=0.14; OR 2.55, CI 0.71–9.12).
Conclusions: De novo post-transplant urethral stricture rates appear to occur at a higher rate than the general population and contribute to patient morbidity. Stricture disease should be considered post-transplantation in patients with voiding dysfunction, even if they don't have prior risk factors. Multicenter studies should be considered to elucidate any relationship between urethral stricture and graft survival.

INTRODUCTION

Urethral stricture disease is the pathologic narrowing of the urethra causing lower urinary tract obstruction. A stricture is caused by fibrosis to the urethral epithelium and corpus spongiosum, which results in luminal constriction. Urethral stricture disease is relatively common in males with an estimated prevalence of 0.6–0.9% in developed countries, though rates vary geographically.[1,2] Stricture etiology is most commonly idiopathic, followed by iatrogenic causes like repeated previous instrumentation or catheterization. A history of urinary tract infection (UTI) or prior urethral reconstructive surgery can also lead to strictures.[3,4] Patients often present with obstructive voiding symptoms and lower urinary tract symptoms (LUTS), leading to decreased quality of life. Significant complications may result such as UTI, acute urinary retention, and, at the extreme, renal failure.[5] Treatment for urethral strictures can be challenging, with formal reconstructive procedures from subspecialized urologists often being required.

Incidence and management of postoperative urethral strictures in renal transplant recipients (RTRs) is not well characterized. In British Columbia, over 300 renal transplants are performed each year.[6] Many patients transition from significantly decreased urine output pre-transplant to producing multiple litres of urine per day post-transplant. This can unmask an underlying lower urinary tract obstruction such as urethral stricture, which can prove problematic once a postoperative Foley catheter is removed. Additionally, routine Foley catheterization in the transplantation process itself may predispose patients to stricture formation.[7]

Currently, little is reported in the literature regarding urethral stricture formation following renal transplant. Reported incidence rate of urethral strictures in RTRs ranges from 1.1–6.6%, but generally is thought to be higher than population risk.[8,9] Urethral stricture has been shown to be a relatively common cause of postoperative voiding dysfunction in male RTRs aged 40–59 years (23%).[10] Patients with postoperative strictures generally present with dysuria, frequency, or hydronephrosis.[11] To the best of our knowledge, no research has been carried out to described postoperative urethral strictures in RTRs using a Canadian population. The objective of this study was to characterize urethral stricture disease in male RTRs from a single

high-volume Canadian transplant center. The secondary objective was to identify risk factors associated with the formation of urethral strictures in RTR patients.

METHODS

A single-center retrospective chart review was conducted on adult male RTRs (aged 19 years and older) who received a renal transplant at Vancouver General Hospital (VGH) between October 1, 2009 and October 1, 2019, and who developed a postoperative urethral stricture. This study was approved by the University of British Columbia Clinical Research Ethics Board (CREB) (#H19-03271).

VGH institutional electronic medical records and the British Columbia (BC) provincial renal database (Patient Records and Outcome Management Information System, PROMIS) were used to identify all patients who received a renal transplant between October 1, 2009 and October 1, 2019. Inclusion criteria included male sex and age 19 years or older. To identify patients who developed strictures post-transplant, patient records were searched for "urethral stricture", "dilation", "urethroplasty", and "urethrotomy". Patients were excluded if they only had a history of pre-transplant stricture without subsequent post-transplant stricture, or had a stricture discovered incidentally intraoperatively during Foley catheter insertion. For the purpose of this study, a patient was considered to have a postoperative urethral stricture if there was either a cystoscopic confirmation of the diagnosis or a urethral dilation/reconstruction procedure after the date of the renal transplant surgery. A patient was considered to have a preoperative urethral stricture if there was either a known history of urethral stricture as stated in the chart preoperatively or a urethral dilation/reconstruction procedure before the date of the renal transplant surgery. Data was abstracted from patient charts including demographics; past medical and urologic history; and preoperative, operative, postoperative, and follow-up data.

For comparison, baseline data was collected from all adult male RTRs (aged 19 years and older) who received a renal transplant at VGH between October 1, 2009 and October 1, 2019, and who did not develop urethral stricture post-transplant. Data regarding donor kidney type, UTI, delayed graft function (DGF), and acute rejection (AR) was gathered from the BC provincial renal database (PROMIS).

In our centre, patients were given antibiotic prophylaxis before renal transplantation. Induction regimen included either Basiliximab or anti-thymocyte globulin (ATG). Patients were catheterized with an indwelling urethral catheter postoperatively for 4–7 days following renal transplant. A double-J ureteric stent was kept *in situ* for 6–8 weeks. The ureteric stent was routinely removed by transplant surgery using office cystoscopy during the first follow-up appointment. Renal transplant recipients were closely followed by transplant nephrology, with appointments 3 times per week for the first month. Immunosuppressants included tacrolimus and mycophenolate mofetil with or without steroids or prednisone taper.

Collected data were de-identified and stored on an encrypted Microsoft Excel© (version 16.57) document in a password-protected computer. For RTRs who developed urethral strictures

post-transplant, descriptive statistics were used to analyze patient characteristics and demographics. Contingency tables were created to determine if there was an association between post-transplant stricture occurrence and donor kidney type, UTI, DGF, and AR. The urethral stricture population was compared against the non-stricture population. Chi-square statistics (X^2) were calculated with p-value <.05 considered statistically significant. Odds ratios (OR) were calculated with 95% confidence intervals (CI). All analyses were performed using Microsoft Excel©. Figure 1 was created with BioRender.com.

RESULTS

During the 10-year period between October 2009–2019, 636 adult male patients underwent renal transplantation at our centre. Of these patients, 18 RTRs (2.8%) were identified as having urethral strictures post-transplant and thus were included in this study (Figure 1). The 618 patients who did not develop strictures post-transplant were used for comparison.

Baseline characteristics of the study population at time of renal transplant are described in Table 1. The median age at time of transplantation was 54.5 years. When evaluating established risk factors for urethral stricture, 1 patient (5.6%) had a known history of UTI, 3 (16.7%) had a known history of bladder outlet obstruction (BOO), 2 (11.1%) had a known history of urethral instrumentation, and 2 (11.1%) had a history of stricture prior to kidney transplantation. No information was available regarding how these strictures were treated. No patients had a known history of prior STI, urethritis, prostatitis, brachytherapy, self-intermittent catheterization, or indwelling catheterization. For roughly half of patients (55.6%), the cause of their end-stage renal disease (ESRD) was diabetic nephropathy. Other causes of ESRD included hypertension (11.1%), IgA nephropathy (11.1%), and glomerulonephritis (5.6%). Only 1 patient had their renal transplant pre-dialysis; the remainder of patients were on hemodialysis at the time of transplant. Before renal transplant, 16.7% of patients were anuric and 88.9% had a urine output of <1 L. Type of kidney donor included living (33.3%), neurological determination of death (NDD; 27.8%), and donation after circulatory death (DCD; 38.9%). Of the 18 total, 3 patients (16.7%) experienced acute rejection (AR) and 4 patients (22.2%) experienced delayed graft function (DGF). Overall, the 1-year and 3-year graft survival rates were both 100%; the 5-year graft survival rate was 88.9% (Table 1).

Renal transplant operative and postoperative details for the 18 RTRs who developed urethral strictures are described in Table 2. The majority of patients underwent Basiliximab induction regimen (77.8%) versus ATG (22.2%). Two patients suffered postoperative urinary retention and one developed a UTI during their initial postoperative hospitalization. Intraoperative and postoperative Foley size varied between patients (16–18 French). Estimated blood loss for the transplant procedure was minor (<250cc) for the majority of patients (94.4%) (Table 2).

Characteristics of the urethral strictures that developed postoperatively for the 18 RTRs are described in Table 3. Following renal transplantation, the median time to stricture discovery

was 56 days (range, 8–618 days) (Table 3). For most patients, strictures occurred in multiple locations, most commonly in the fossa navicularis (83.3%), pendulous urethra (77.8%), and bulbous urethra (50.0%). Of the 18 patients, 14 (77.8%) presented in an outpatient setting with symptoms of their postoperative strictures: lower urinary tract symptoms (LUTS; 61.1%), UTI (27.8%), acute urinary retention (5.6%), and/or stent retention (5.6%). Stricture was confirmed by cystoscopic examination. The remaining 4 strictures were incidentally found upon routine cystoscopy during transplant stent removal. For management, of the 4 strictures found incidentally, 3 required no treatment, while 1 was treated with dilation. Of the 14 who presented symptomatically, 10 were treated with either dilation (71.4%), urethroplasty (50%), and/or urethrotomy (35.7%). Overall, 4 patients (22.2%) required clean intermittent catheterization (CIC). All patients with incidentally found strictures had only one occurrence. Almost two-thirds of patients with symptomatic strictures had at least one recurrence (Table 3).

To determine if there was an association between post-transplant urethral stricture occurrence and donor kidney type, UTI, DGF, and AR, contingency tables were created (Table 4, Table 5). Patients who developed strictures post-transplant (n = 18) were compared against those who did not develop strictures (n = 618) (Figure 1). There was no significant association between stricture post-transplant and donor kidney type (X^2=2.53, p =.28). There were no increased odds of developing a postoperative stricture with differing donor kidney type: NDD (OR 2.29, CI 0.80–6.57), DCD (OR 0.67, 0.26–1.75), and living (OR 0.86, CI 0.32–2.31) (Table 4). There was no significant association between stricture post-transplant and UTI (X^2=0.04, p=.84; OR 0.81, CI 0.1–6.21), DGF (X^2=0.14, p=.70; OR 0.8, CI 0.26–2.48), or AR (X^2=2.02, p=.14; OR 2.55, CI 0.71–9.12) (Table 5).

DISCUSSION

In our centre, the incidence rate of urethral stricture following renal transplant was 2.8% over the period studied. This falls within the reported range of 1.1–6.6% for RTR patients and is comparable to the published rate of 2.5% from Tsaur *et al*.[8,9,10] This rate is also higher than that published for the general population[1,2], reaffirming that RTR patients have a higher likelihood of developing urethral strictures.

One-third of patients who developed urethral stricture disease post-transplant had prior risk factors for stricture formation. Risk factors included a history of UTI, LUTS, or instrumentation, and these would be important to question during a pretransplant assessment. Almost all (88.9%) of the patients with a postoperative urethral stricture had no previous noted history of urethral stricture. Thus, the absence of previous stricture history does not preclude patients from later developing postoperative urethral strictures. Of note, two-thirds of stricture patients produced less than 250cc of urine per day pre-transplant, which could mask pre-existing strictures. Given the relatively higher prevalence of *de novo* strictures found in our study, urethral stricture may be considered in RTRs with newfound urinary complaints (e.g., LUTS and UTI) after transplant, regardless of previous risk factors.

Foley catheterization has been identified as an important predisposing factor for urethral stricture formation.[7] End-stage renal disease is a chronic condition often requiring prolonged catheterization, which likely contributes to the higher incidence of strictures seen in this population. In this study, patients were catheterized with an indwelling urethral catheter postoperatively for 4–7 days following renal transplant. A literature review showed no studies regarding urethral stricture rates from comparable hospitalized populations with routine catheterization for other indications, so it is hard to comment on how this catheterization may have impacted stricture rates. Intraoperative and postoperative Foley catheter size varied by patient. Stricture formation has been associated with larger urethral catheter size, which has led to recommendations to use smaller catheter sizes for shorter durations.[12,13] Removal of the catheter may have also unmasked strictures, as patients were generally producing minimal urine before receipt of the donor kidney. More work is needed to identify the optimal catheterization protocol during and after renal transplantation.

The median time from transplant to stricture presentation was 8 weeks. This differs from Xie *et al.*, which reported a mean time from transplant to urethral stricture of 4.4 months (range 2–7 months), and Gökçe *et al.*, which reported a time from transplant to urethrography and cystoscopy of 17.5 months (range 1–85 months).[9,11] At our centre, all RTRs had their ureteric stent removed via office cystoscopy 6–8 weeks after transplant surgery. Stent removal is typically the first in-person follow-up appointment most transplant patients have with transplant urology, which is likely when concerns regarding stricture-related symptoms would be first discussed, or else found incidentally on cystoscopy. Therefore, most of the urethral strictures were likely identified during a stent removal appointment. Most post-transplant urethral strictures were seen in the fossa navicularis or pendulous urethra, followed by the bulbar urethra; other studies have found the most common location to be the membranous urethra.[9,11] Given that not all of patients who developed urethral strictures postoperatively presented symptomatically, there may be an element of urethral stricture overdetection that is not clinically significant. Nonetheless, the majority of RTRs (77.8%) with postoperative strictures did have clinical symptoms including LUTS and UTI, which has been found in other studies.[11] Importantly, nearly half of patients with postoperative urethral strictures had more than one occurrence of stricture, supporting surveillance for recurrence.

Not all patients with urethral strictures required treatment. Of the 7 managed conservatively, 3 had presented asymptomatically, reaffirming that there may be a degree of stricture overdetection on cystoscopy that may not be clinically significant. However, similar to the literature, the majority required operative management for their strictures such as urethrotomy and urethroplasty.[8,11] Furthermore, 22.2% of patients required CIC to help manage their urethral stricture, which is an added morbidity as well as an increased risk of infection.[14,15]

Finally, comparative analysis revealed that there was no statistically significant association between developing postoperative urethral stricture and an increased risk of UTI,

DGF, or AR. Moreover, the 1-, 3-, and 5-year graft survival rates in patients who develop post-transplant strictures (100%, 100%, 88.9%) were comparable to overall graft survival rates (our institution: 94% at 1 year; 85% at 3 years) (national data: 64.6-82.1% at 5 years)[16]. Thus, urethral strictures do not appear to affect overall graft survival, although this is a limited observation given the study design and the lack of power to directly compare populations. Urethral stricture has been associated with UTI, which can increase morbidity in a population that is already immunosuppressed and taking prophylactic antibiotics.[4] Further research should be done to investigate this potential increased morbidity.

This study has several limitations given its nature as a single-centre retrospective review. Results may have lower external validity given that data was abstracted from one transplant center. Urethral stricture disease has a low incidence in renal transplant recipients, resulting in a smaller sample size. As a retrospective study, some data was missing; further, it is possible that some data was under-reported, including the incidence of BOO, instrumentation, and UTI. Routinely at our centre, Transplant Urology does not follow all RTRs after stent removal unless referred by their Transplant Nephrologists or a primary care physician, so there could be some patients with strictures who developed later who were lost to follow-up, thus underestimating the true prevalence of the disease.

Further research may be carried out to identify risk and protective factors that influence the prevalence of urethral strictures, as well as the impact of renal transplantation. Since the rates of urethral strictures post-renal transplantation are low, larger cohorts of patients through multicentre studies may be needed to describe population trends, increase statistical power, and identify clinical significance. Moreover, the long-term effects on graft function for RTR patients with urethral strictures should be described.

CONCLUSIONS

Urethral stricture disease is a less common but important complication affecting renal transplant patients. This study is the first to characterize urethral stricture disease in male RTRs from a high-volume Canadian transplant center. Our data show that there is a slight increased incidence of urethral stricture disease in RTRs with a calculated prevalence of 2.8%, though a history of risk factors, time to presentation, and risk of recurrence vary greatly. As the majority of strictures present symptomatically, urethral stricture is a potential etiology of newfound postoperative voiding dysfunction to consider. Overall, outcomes are reassuring. There is no significant association between developing postoperative urethral stricture and donor kidney type, UTI, DGF, or AR. Urethral strictures do not appear to affect overall graft survival. Further research using larger patient cohorts to better describe risk and protective factors, as well as long-term effects on graft function, is needed.

REFERENCES

1. Santucci RA, Joyce GF, Wise M. Male urethral stricture disease. *J Urol* 2007;177:1667-74.
2. Anger JT, Buckley JC, Santucci RA, et al. Trends in stricture management among male Medicare beneficiaries: Underuse of urethroplasty? *Urology* 2011;77:481-5.
3. King C, Rourke KF. Urethral stricture is frequently a morbid condition: Incidence and factors associated with complications related to urethral stricture. *Urology* 2019;132:189-94.
4. Stein DM, Thum DJ, Barbagli G, et al. A geographic analysis of male urethral stricture aetiology and location. *BJU Int* 2013;112:830-4.
5. Rourke KF, Welk B, Kodama R, et al. Canadian Urological Association guideline on male urethral stricture. *Can Urol Assoc J* 2020;14:305-16.
6. BC Transplant, Provincial Health Services Authority. 2021 Organ donation and transplantation summary [cited 2022 Feb 28]. http://www.transplant.bc.ca/Documents/Statistics/2021-BC-Organ-Donation-Transplant-Summary.pdf. Accessed February 12, 2022.
7. Lumen N, Hoebeke P, Willemsen P, et al. Etiology of urethral stricture disease in the 21st century. *J Urol* 2009;182:983-7.
8. Krajewski W, Dembowski J, Kołodziej A, et al. Urological complications after renal transplantation – a single centre experience. *Cent European J Urol* 2016;69:306-11.
9. Gökçe AM, Kaya C, Kara VM, et al. Effects of urethral stricture on allografts in kidney transplantation. *Transplant Proc* 2015;47:1336-9.
10. Tsaur I, Jones J, Melamed RJ, et al. Postoperative voiding dysfunction in older male renal transplant recipients. *Transplant Proc* 2009;41:1615-8.
11. Xie L, Lin T, Wazir R, et al. The management of urethral stricture after kidney transplantation. *Int Urol Nephrol* 2014;46(11):2143-5.
12. Hammarsten J, Lindqvist K, Sunzel H. Urethral strictures following transurethral resection of the prostate. The role of the catheter. *Br J Urol* 1989;63:397-400.
13. Fenton AS, Morey AF, Aviles R, et al. Anterior urethral strictures: Etiology and characteristics. *Urology* 2005;65:1055-8.
14. Jhanwar A, Sokhal AK, Singh K, et al. Assessment of quality of life in patients of urethral stricture on clean intermittent catheterization following direct vision internal urethrotomy. *Urol Ann* 2018;10:395-9.
15. Wyndaele JJ. Complications of intermittent catheterization: Their prevention and treatment. *Spinal Cord* 2002;40:536-41.
16. Hart A, Lentine KL, Smith JM, et al. OPTN/SRTR 2019 Annual data report: Kidney. *Am J Transplant* 2021;21:S2:21-137.

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FIGURES AND TABLES

Figure 1. Inclusion and exclusion criteria to identify RTRs who did and did not develop postoperative urethral strictures.

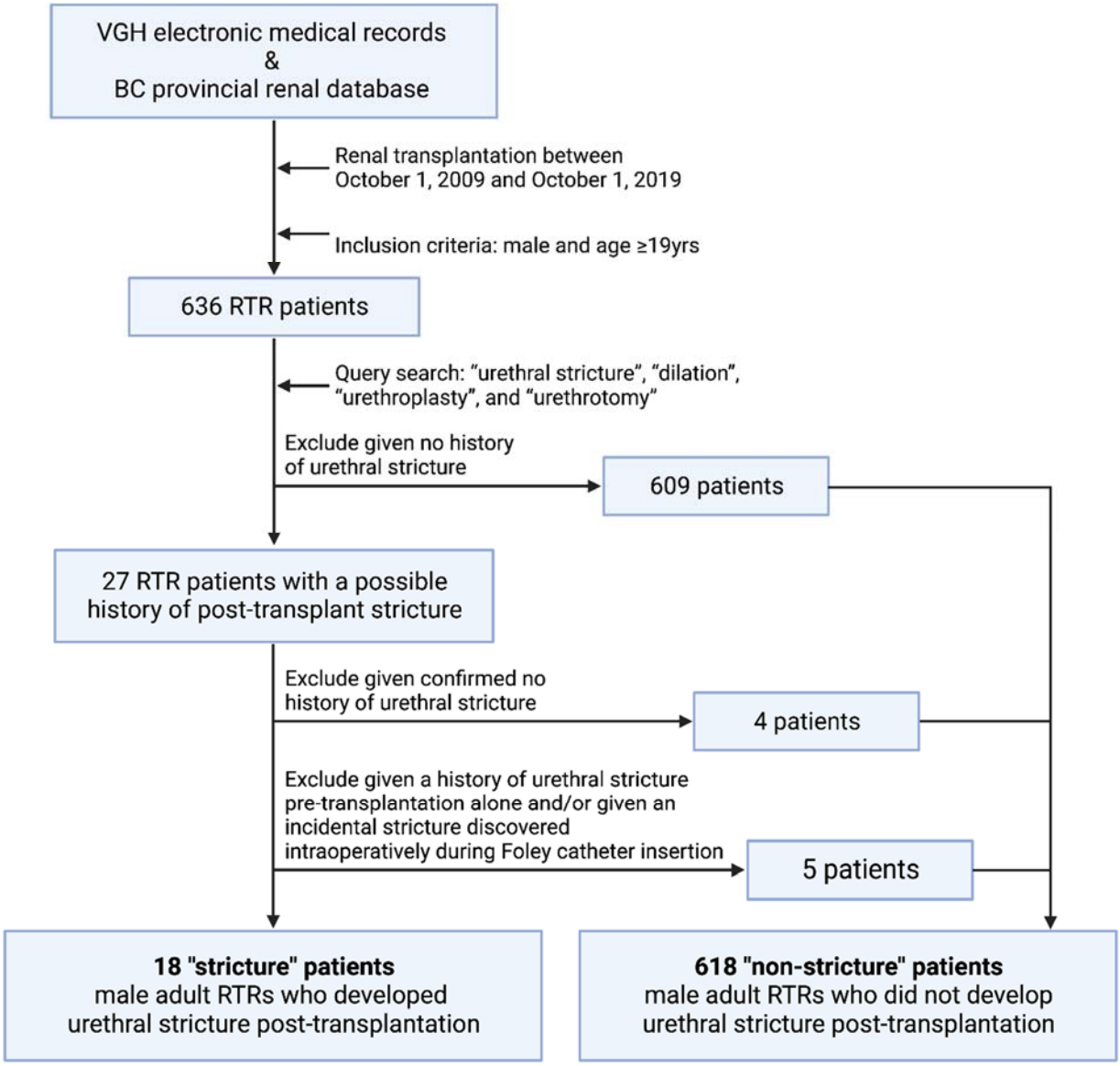

Table 1. Baseline characteristics of the RTR urethral stricture group at time of renal transplant			
Characteristics		**Number of patients (n = 18)**	**Percent of total (%)**
Demographics			
	Age (mean, IQR) [yrs]	54.5, 26	–
	BMI (mean) [kg/m^2]	27.8, 5.6	–
	BMI ≥30	5	27.8%
Risk factor for stricture		6	
	History of UTI	1	5.6%
	History of BOO	3	16.7%
	History of instrumentation	2	11.1%
	History of previous urethral stricture	2	11.1%
Reason for ESRD			
	Diabetes	10	55.6%
	Hypertension	2	11.1%
	IgA nephropathy	2	11.1%
	Glomerulonephritis	1	5.6%
	Other[1]	1	5.6%
	Unknown	2	11.1%
Time on dialysis (median, IQR) [days]		1089.5, 979	
Dialysis type			
	HD	17	94.4%
	PD	0	0%
	Pre-dialysis	1	5.6%
Urine output pre-transplant			
	0cc	3	16.7%
	1–250 cc	9	50.0%
	251–1000 cc	4	22.2%
	>1000 cc	2	11.1%
Type of donor			
	Living	6	33.3%
	NDD	5	27.8%
	DCD	7	38.9%
Graft outcome			
	Acute rejection	3	16.7%
	Delayed graft function	4	22.2%
	1-year graft survival rate	18	100.0%
	3-year graft survival rate	18	100.0%
	5-year graft survival rate	16	88.9%

[1]Other: congenital renal dysplasia, focal segmental glomerulosclerosis, Crohn's disease.

BMI: body mass index; BOO: bladder outlet obstruction; DCD: donation after circulatory death; ESRD: end-stage renal disease; HD: hemodialysis; IQR: interquartile range; NDD: neurological determination of death; PD: peritoneal dialysis; UTI: urinary tract infection.

Table 2. Operative and postoperative characteristics of the RTR urethral stricture group at time of renal transplant

Operative characteristics		**Number of patients (n = 18)**	**Percent of total (%)**
Induction regimen			
	Basiliximab	14	77.8%
	ATG	4	22.2%
Complications			
	Postoperative retention	2	11.1%
	Postoperative UTI	1	5.6%
Intraoperative Foley size			
	18 French	5	27.8%
	20 French	5	27.8%
	Unknown	8	44.4%
Postoperative Foley size			
	16 French	1	5.6%
	18 French	5	27.8%
	20 French	4	22.2%
	Unknown	8	44.4%
Estimated blood loss			
	≤250 cc	17	94.4%
	>250 cc	1	5.6%

ATG: anti-thymocyte globulin; UTI: urinary tract infection.

Table 3. Characteristics of the RTR postoperative urethral strictures		
Postoperative urethral strictures	**Number of patients (n = 18)**	**Percent of total (%)**
Median time from transplant to stricture discovery (range) [days]	56 (8–618)	
Post-transplant stricture locations		
Fossa navicularis	15	
Pendulous urethra	14	
Bulbar urethra	9	
Membranous urethra	2	
Prostatic urethra	1	
Asymptomatic presentation	4	22.2%
No treatment / surveillance	3	75.0%
Required treatment	1	25.0%
Dilation	1	
Symptomatic presentation	14	77.8%
Presenting complaint		
LUTS	11	78.6%
UTI	5	35.7%
AUR / AKI	1	7.1%
Stent retention	1	7.1%
No treatment / surveillance	4	28.6%
Required treatment	10	71.4%
Dilation	10	
Urethrotomy	5	
Urethroplasty	7	
Required CIC	4	28.6%
Recurrent strictures		
1 recurrence	5	
2 recurrences	4	

AKI: acute kidney injury; AUR: acute urinary retention; CIC: clean intermittent catheterization; LUTS: lower urinary tract symptoms; UTI: urinary tract infection.

Table 4. Contingency table comparing donor kidney type between RTRs who did and did not develop postoperative urethral strictures				
	Stricture	**No stricture**	**X^2 (2, n=636) (p)**	**OR (95% CI)**
NDD	5	89	2.53 (p=0.28)	2.29 (0.80, 6.57)
DCD	7	301		0.67 (0.26, 1.75)
Living	6	228		0.86 (0.32, 2.31)

CI: confidence interval; DCD: donation after circulatory death; NDD: neurological determination of death; OR: odds ratio.

Table 5. Contingency table comparing UTI, DGF, and AR between RTRs who did and did not develop postoperative urethral strictures				
	Stricture	**No stricture**	**X^2 (1, n=636) (p)**	**OR (95% CI)**
UTI	1	42	0.04 (p=0.84)	0.81 (0.10, 6.21)
No UTI	17	576		
DGF	4	162	0.14 (p=0.70)	0.8 (0.26, 2.48)
No DGF	14	456		
AR	3	45	2.21 (p=0.14)	2.55 (0.71, 9.12)
No AR	15	573		

AR: acute rejection; CI: confidence interval; DGF: delayed graft function; OR: odds ratio; UTI: urinary tract infection.