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Ureteric complications in recipients of kidneys from donation after circulatory death donors

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Abstract

A large increase in the use of kidneys from donation after circulatory death (DCD) donors prompted us to examine the impact of donor type on the incidence of ureteric complications (UCs; ureteric stenosis, urinary leak) after kidney transplantation. We studied 1072 consecutive kidney transplants (DCD n=494, live donor [LD] n=273, donation after brain death [DBD] n=305) performed during 2008-2014. Overall, there was a low incidence of UCs after kidney transplantation (3.5%). Despite a trend toward higher incidence of UCs in DCD (n=22, 4.5%) compared to LD (n=10, 3.7%) and DBD (n=5, 1.6%) kidney transplants, donor type was not a significant risk factor for UCs in multivariate analysis (DCD vs DBD HR: 2.33, 95% CI: 0.77-7.03, *P*=.13). There was no association between the incidence of UCs and donor, recipient, or transplant-related characteristics. Management involved surgical reconstruction in the majority of cases, with restenosis in 2.7% requiring re-operation. No grafts were lost secondary to UCs. Despite a significant increase in the number of kidney transplants from DCD donors, the incidence of UCs remains low. When ureteric complications do occur, they can be treated successfully with surgical reconstruction with no adverse effect on graft or patient survival.

KEYWORDS
DCD kidney transplantation, ureteric complications

1 | INTRODUCTION

Ureteric complications (UC) after kidney transplantation are relatively uncommon but represent a significant cause of early and late morbidity. UCs comprise urinary leaks and stenosis, and their incidence in recently reported series ranges between 2.7% and 9.2%.[1-4] The majority of UCs occur during the first year after transplantation; risk factors for early complications may include increased donor age, delayed graft function, and multiple renal arteries,[5] whereas later complications may be associated with acute rejection, BK virus nephropathy, or recurrent urinary infections.[6,7] The stented extravesical anastomosis has now become the standard technique for ureteric implantation as it is associated with a relatively low complication rate.[8-10] When performing the ureteric implantation, preservation of the ureteric blood supply and avoidance of an unnecessarily long ureter are both thought to be important factors in minimizing UCs.[1]

Increased demand for kidney transplantation has prompted an expansion of the deceased donor pool by greater use of "marginal" donor kidneys, including those from elderly donors and those with significant cardiovascular morbidity. There has also been a large increase in the use of kidneys from donation after circulatory death (DCD) donors. It is widely thought that ischemic damage of the donor ureter due to compromised arterial blood supply may be a contributory factor to UCs and the warm ischemic injury integral to DCD may increase the risk of UCs following kidney transplantation. In an analysis of kidney transplants performed in our center from 1998 to 2008, we have previously reported that the incidence of ureteric stenosis was similar in kidneys transplanted from live donors (LD), donation after brain death

(DBD) donors, and DCD donors.[7] Since then, DCD transplant activity at our center has increased substantially, and we now perform twice as many DCD as DBD kidney transplants and routinely use DCD kidneys from elderly donors (>60 years old).[11] We therefore thought it would be timely to re-examine the impact of this change in clinical practice on the incidence and nature of UCs after kidney transplantation.

2 | METHODS

2.1 | Study population

A single-center, retrospective, observational, cohort study was performed to examine the impact of donor type (DCD, DBD, LD) on the incidence of UC after kidney transplantation. Data on all kidney transplants performed at the Cambridge Transplant Centre were prospectively collected, and analyses were performed as part of a service evaluation. The study population comprised 1072 recipients of a single kidney transplant, including simultaneous kidney and pancreas transplants, performed between September 2008 and December 2014 (census date December 31, 2014). Dual kidney transplants and recipients of combined and multivisceral transplants were excluded from the analysis. All DCD kidneys were procured from controlled, Maastricht category 3 and 4 donors,[12] who incurred irrecoverable brain injury, but did not meet the criteria for diagnosis of brain stem death. Kidney procurement was performed as previously described,[13] and donation was pursued for a minimum of 4 hours after withdrawal of life-supporting treatment. Data were retrieved from a prospective, cross-audited, computerized database and by detailed case note review. Operating theater logs were also examined (in addition to the database search and case note review) to ensure identification of all recipients with UCs. The study was approved by the local institutional review board as a service evaluation.

2.2 | Clinical information and variable definitions

Live donor kidneys were retrieved laparoscopically. Kidney transplants were routinely placed extraperitoneally in the left or right iliac fossa, and ureteroneocystostomy was performed using interrupted absorbable sutures (5/0 polydioxanone) over a double JJ (pigtail) ureteric stent. Ureteric stents were removed cystoscopically after approximately 6 weeks or earlier if indicated clinically by the presence of a urinary tract infection (UTI). Immunosuppression was administered according to standard protocols, as described previously.[7] Screening for BK viremia by polymerase chain reaction was performed routinely during the study period.

Ureteric complications were defined as ureteric stenosis or urinary leak after ureteroneocystostomy. A diagnosis of ureteric stenosis was suspected by the presence of hydronephrosis of the transplant kidney on ultrasound examination and confirmed in all cases by a nephrostogram after radiologically guided insertion of a percutaneous nephrostomy tube. A urine leak from the vesicoureteric anastomosis was defined as the leak of urine from the abdominal wound, the presence of a perinephric urine collection (identified on radiological imaging and confirmed by biochemical analysis of the aspirate), or the presence of a leak identified by an antegrade nephrostogram. Where surgical intervention was deemed necessary, it comprised re-implantation of the donor ureter onto the bladder or creation of a donor ureter to native ureter ureteroureterostomy or creation of a donor pelvis to native ureter pyeloureterostomy. All ureteric reconstructions were performed over a double pigtail ureteric stent, which was removed after approximately 6 weeks.

Extended criteria donors (ECD) were defined as those ≥60 years or those aged 50-59 years with two of the following three features: hypertension; terminal serum creatinine >115 mmol/L; or death from cerebrovascular accident.[14] Delayed graft function was defined as the provision of dialysis in the first week after transplantation, and primary nonfunction was defined as a graft that never achieved sufficient function to allow discontinuation of dialysis, excluding acute vascular thrombosis.[15] UTIs were defined as urine samples from which bacteria were identified at microscopy and after culture. Recipient sensitization was defined as HLA-specific antibody reactivity (calculated reaction frequency [cRF]) against a panel of 10 000 consecutive UK organ donors.[16]

2.3 | Statistical methods

Data are summarized as mean (SD) or median (interquartile range) as appropriate. For comparison of fixed covariates, Fisher's exact, Student's *t*, and Mann-Whitney *U* tests were used. The time origin for survival and time to UC analysis was the date of transplantation. For the analysis of UCs incidence, follow-up was diagnosis of the UC, and censoring took place at the end of the study period if UC had not occurred. For covariates that were fixed at transplantation (ischemic time and donor and recipient characteristics), time to UC was summarized using the Kaplan-Meier method, and curves were compared using the standard log-rank test. Covariates that occurred at varying times post-transplant (UTI) were treated as time dependent in a Cox proportional hazards regression analysis. Fixed covariates that were significant at the 10% level in Kaplan-Meier tests were included in multivariate Cox regression models. Hazard ratios and 95% CIs from these analyses are presented. When analyzing the incidence of UTIs, a nested case-control study was constructed in which the comparison group consisted of one kidney transplant recipient immediately before and one kidney transplant recipient immediately after every case of UC (n=74); UTIs occurring within 1-year post-transplantation in the comparison group were analyzed. The relationship between incidence of UC and graft and patient survival used Cox regression with UC as a time-dependent marker and graft loss or patient death as the outcome.

3 | RESULTS

During the 75-months study period, a total of 1072 kidney transplants (DCD n=494, LD n=273, DBD n=305) were performed at the Cambridge Transplant Centre. Of these, nine (0.8%) patients developed a urinary leak at a median (SD) of 28 (10) days after transplantation. A further 28 (2.6%) patients developed a ureteric stenosis, at a median (SD) of 68 (85) days following transplantation. The 37 patients with UCs had similar clinical characteristics to the 1035 patients who did not develop

TABLE 1 Clinical characteristics of transplants performed at the Cambridge Transplant Centre during the study period (September 2008 and December 2014)

	UCs (n=37)	No UCs (n=1035)	Log-rank test on time to UCs
Transplant type, n (%)			
Kidney	34 (92)	911 (88)	0.43
SPK	3 (8)	124 (12)	
Male donors, n (%)	18 (49)	548 (53)	0.60
Male recipients, n (%)	29 (78)	643 (62)	0.05
Donor age (y), mean (SD; range)	51 (16, 14-78)	49 (16, 5-82)	0.43[a]
Recipient age (y), mean (SD; range)	50 (15, 22-72)	49 (13, 17-75)	0.42[a]
Donor extended criteria status, n (%)			
Yes	16 (43)	346 (33)	0.20
No	20 (54)	664 (64)	
Not known	1 (3)	25 (2)	
Donor cause of death, n (%)			
Neurological	24 (65)	676 (65)	0.89
Respiratory	1 (3)	36 (3)	
Organ failure	0 (0)	14 (1)	
Cardiovascular	0 (0)	11 (1)	
Drug overdose	0 (0)	5 (0)	
Unknown	2 (5)	32 (3)	
Living donor	10 (27)	261 (25)	
Renal artery multiplicity, n (%)	9 (24)	289 (28)	0.61
HLA mismatch level[b]			
1	1 (3)	82 (8)	0.30
2	6 (16)	266 (26)	
3	21 (57)	484 (47)	
4	9 (24)	202 (20)	
Unknown	0 (0)	1 (0)	
Sensitization, n (%)			
0-15	32 (86)	750 (72)	0.26
15-50	3 (8)	109 (11)	
50-85	1 (3)	91 (9)	
85-100	1 (3)	85 (8)	
Re-transplant, n (%)	5 (14)	110 (11)	0.57
Cold ischemic time, mean (SD; range, in h)[c]	10.6 (5.9, 1.2-22.2)	11.7 (6.2, 1-34.9)	0.49

(Continues)

UCs (Table 1). The median duration of follow-up for patients with and without UCs was 813 days (range: 0-2216 days) and 1037 days (range 0-2528 days), respectively. Four of the nine patients with a ureteric leak were treated successfully by conservative management, and five were treated by surgical intervention with re-implantation of the donor ureter onto the recipient urinary bladder. The 28 cases of ureteric stenosis were all confirmed by an antegrade percutaneous nephrostogram; 16 cases (57.2%) involved the vesicoureteric junction or distal ureter, three cases (10.7%) the mid-ureter, and three cases (10.7%) the proximal ureter, and two cases (7.1%) had a long stricture (involving over half of the ureteric length). In four cases (14.3%), the involved ureteric segment was not specified. Twenty-six of the 28 cases of ureteric stenosis were treated by surgical reconstruction. One patient was treated for acute rejection following which their graft function improved and no further intervention was deemed necessary, and a further patient was treated with antegrade ureteric stent insertion. Only one patient presented with a recurrence of ureteric stenosis (overall recurrence rate was 2.7%) following ureteric reconstruction and was treated by creation of a donor pelvis to native ureter pyeloureterostomy.

The principal aim of this study was to determine whether there was any association between kidney donor type and incidence of UCs. As shown in Table 2, the incidence of UCs was numerically higher in

TABLE 2 (Continued)

	UCs (n=37)	No UCs (n=1035)	Log-rank test on time to UCs
Prolonged cold ischemic time[d], n (%)	5 (14)	137 (14)	0.98
Warm ischemic time, mean (SD; range in min)[e]	7.9 (5.1, 1-20)	9.2 (5.4, 1-50)	0.29
Delayed graft function[f], n (%)	19 (51)	393 (38)	0.10
Primary nonfunction[f], n (%)	1 (3)	26 (4)	0.94
Urinary tract infection[g], n (%)	16 (43)	27 (36)	0.55
BK virus infection[h], n (%)	4 (11)	188 (20)	0.20

SPK, simultaneous pancreas and kidney transplant; UCs, ureteric complications.

[a]Cox regression was used for donor and recipient age analyzed as continuous variables. Classifying donor age ≥60 y vs <60 y had no association with incidence of UCs (*P*=.61, log-rank). Classifying recipient age ≥60 y vs <60 y had no association with incidence of UCs (*P*=.17, log-rank).

[b]HLA mismatch level was defined according to UK allocation policy for kidneys from brain death donors and was based on the mismatch between donor and recipient at the HLA-A, HLA-B, and HLA-DR loci: level 1 was a 000 HLA-A, HLA-B, and HLA-DR mismatch; level 2 was a 0 HLA-DR plus 0/1 HLA-B mismatch; level 3 was a 0 HLA-DR plus 2 HLA-B mismatch or a 1 HLA-DR plus 0/1 HLA-B mismatch; and level 4 was a 2 HLA-DR or a 1 HLA-DR plus 2 HLA-B mismatch.

[c]Cold ischemic time was not available for 25 kidneys in the "No UCs" group. Cox regression was used for cold ischemic time analyzed as continuous variable.

[d]Cold ischemic time >18 h compared to ≤18 h (log-rank test). Cold ischemic time was not available for 25 kidneys in the "No UCs" group.

[e]Warm ischemia time was not available for 15 kidneys in the "No UCs" group.

[f]Delayed graft function and primary nonfunction status were not available for eight patients in the "No UCs" group.

[g]When analyzing the incidence of urinary tract infections, a nested case-control study was constructed in which the comparison group consisted of one kidney transplant recipient immediately before and one kidney transplant recipient immediately after every case of UC (n=74).

[h]For patients with UCs, BK viremia before the diagnosis of a UC was considered. BK viremia status was not available for 105 patients (two patients in the "UCs" group and 103 patients in the "No UCs" group).

TABLE 2 Incidence of ureteric complications according to kidney donor type

		Ureteric complications		
Donor type	N	Total (%)	Stenosis (%)	Leak (%)
LD	273	10 (3.7)	8 (2.9)	2 (0.7)
DCD	494	22 (4.5)	16 (3.2)	6 (1.2)
DBD	305	5 (1.6)	4 (1.3)	1 (0.3)
Total	1072	37 (3.5)	28 (2.6)	9 (0.8)

LD, live donors; DCD, donors after circulatory death; DBD, donors after brain death.

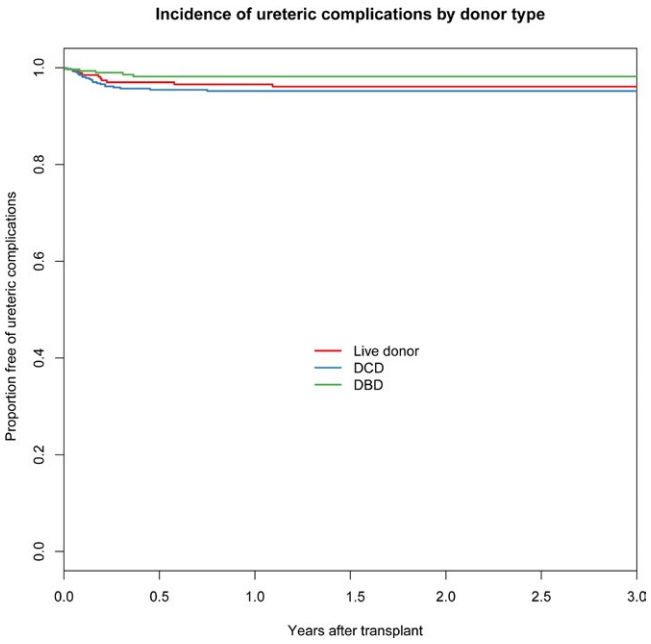

FIGURE 1 Incidence of ureteric complications according to kidney donor type. Incidence of ureteric complications (ureteric stenosis and urinary leak) in recipients from live donors (LD), donors after circulatory death (DCD), and donors after brain death (DBD)

recipient of DCD kidneys (4.5%) than for those who received LD kidneys (3.7%) and DBD kidneys (1.6%). In univariate analysis (Figure 1 and Table 3), recipients of DCD kidneys had a significantly greater risk of developing UCs compared to recipients of DBD kidneys (HR: 2.77, 95% CI: 1.05-7.31, *P*=.04), whereas the risk of UCs was similar for recipients of LD and DBD kidneys (HR: 0.46, 95% CI: 0.16-1.35, *P*=.16). Cold ischemic time was similar in patients with (mean: 10.6 hours, SD: 5.9, range: 1.2-22.2 hours) and without UCs (mean: 11.7 hours, SD: 6.2, range: 1.0-34.9 hours). Delayed graft function occurred in 51% of patients with UCs and 38% of patients without UCs and was not significantly different between the two groups (*P*=.10). There was no association between incidence of UCs and kidney donor characteristics (age, gender, cause of death, and kidneys from ECD). Similarly, there was no association between UCs and transplant recipient characteristics (age, gender, and sensitization to HLA) or transplant-related factors (re-transplantation, HLA mismatch level, and renal artery multiplicity). Four (11%) of the recipients with a UC developed BK viremia before the diagnosis of UC was made, and this was not significantly different to the incidence of BK viremia within 1 year after transplantation in patients without UCs (20%; HR: 0.51,

TABLE 3 Risk factors for ureteric complications using single covariate and multiple covariate Cox proportional hazards regression analysis

Variable	Hazard ratio (95% CI)	Significance
Single covariate models		
Donor type		
DCD vs LD	1.27 (0.60-2.69)	.53
LD vs DBD	0.46 (0.16-1.35)	.16
DCD vs DBD	2.77 (1.05-7.31)	.04
Recipient sex (male)	2.19 (1.00-4.79)	.05
Delayed graft function (yes)	1.75 (0.91-3.36)	.10
Urinary tract infection (yes)	1.16 (0.61-2.23)	.65
Multiple covariate model		
Donor type		
DBD	1.00	Reference
LD	2.63 (0.88-7.85)	.08
DCD	2.42 (0.91-6.44)	.08
Recipient sex (male)	2.17 (0.98-4.79)	.055
Delayed graft function (yes)	1.70 (0.84-3.41)	.14

LD, live donors; DCD, donors after circulatory death; DBD, donors after brain death.

95% CI: 0.18-1.44, *P*=.20). We have also examined whether the warm ischemic insult integral to DCD organ procurement might contribute to the risk of developing UCs in recipients of DCD kidneys. There was no association between UCs and warm ischemia time (defined as the time between donor circulatory arrest and cold in situ perfusion) which was similar in patients with (mean: 7.9 minutes, SD: 5.1, range: 1-20 minutes) and without UCs (mean: 9.2 minutes, SD: 5.4, range: 1-50 minutes).

The potential association between UTI and the risk of developing UCs was examined in a nested case-control study. The incidence of UTI, before the diagnosis of UC, in recipients with UCs was 43% (occurring at a mean [SD] of 20 [17] days after transplantation), and the incidence of UTI (diagnosed within 1 year of transplantation) mean [SD] of 64 [88] days) in the control group, chosen as the recipients before and after each patient with UC, was 38% (HR: 1.16, 95% CI: 0.61-2.23, *P*=.65).

We next performed multivariable Cox proportional hazards regression analysis to determine risk factors for the development of UCs. As shown in Table 3, DCD kidney transplantation was not associated with a significant increase in the risk of UCs; this was also the case when the analysis was repeated, excluding cases of urinary leak (DCD vs DBD HR: 2.33, 95% CI: 0.77-7.03, *P*=.13). There was a trend toward increased risk of UCs in male kidney transplant recipients but that did not reach statistical significance in multivariable analysis (HR: 2.17, 95% CI: 0.98-4.79, *P*=.055). Graft and patient survival were not affected by occurrence of a UC (HR: 1.70, 95% CI: 0.45-8.70, *P*=.37 for graft survival and HR: 0.58, 95% CI: 0.14-2.99, *P*=.58 for patient survival). There were no cases of graft loss or patient death as a direct consequence of UCs and their treatment.

4 | DISCUSSION

This large single-center retrospective cohort analysis showed that UCs remain an uncommon complication (3.5% overall) following renal transplantation. This is broadly similar to that observed in our previous analysis of an earlier transplant cohort where the overall rate of UCs was 2.7%.[7] Although UCs are uncommon, they give rise to significant morbidity and the vast majority of UCs in the present series required surgical intervention. No grafts were lost as a direct result of UCs. It was notable that the incidence of UCs was highest in recipients of DCD kidneys and lowest in recipients of DBD kidneys, although this difference was no longer statistically significant on multivariate analysis. Again, this is consistent with our earlier findings where the incidence of UCs was similar in recipients of DCD and DBD kidneys.

Urinary leaks were relatively rare (0.8%) in the present series, and this is likely attributable to the routine use of ureteric stents.[10] Urinary leaks typically occurred in the early postoperative period, and in four of the nine patients, the ureteric leak resolved with conservative management alone. Ureteric stenosis was three times more common than urinary leakage in this study and most commonly occurred in the first few months after kidney transplantation and often in the days following cystoscopic removal of the ureteric stent. The etiology of ureteric stenosis is not known, and it has been suggested that factors such as poor ureteric blood supply, UTI, and BK virus infection may all contribute. No obvious associations between these factors and the presence of ureteric stenosis were evident in our analysis. Our study showed a trend toward a increased risk of UCs in male kidney transplant recipients; a pathophysiological basis in support of this observation has not been described, but it is tempting to speculate that the presence of prostatic hypertrophy might be a contributing factor. In the present series, as in our earlier series,[7] the majority of UCs were treated by surgical intervention. Surgery was successful in all but one patient who required an additional surgical procedure to be performed. Others have reported on the effective treatment of ureteric stenosis by endourological approaches, most notably ureteric dilatation, although these approaches are associated with lower success rates and more complications than open surgical intervention.[17] It was notable that no recipient deaths or graft failures occurred in the present series as a direct result of UCs.

Kidneys from DCD donors are increasingly used for transplantation. It is notable that, in our previous analysis of an earlier transplant cohort,[7] 22% of kidneys were from DCD donors, whereas in the present study, DCD kidneys accounted for 46% of the entire cohort. While kidneys from such donors inevitably incur a period of significant ischemia-reperfusion injury, as evidenced by a higher rate of delayed graft function, they have comparable graft survival to that seen in recipients of kidneys from DBD donors.[18,19] Moreover, as highlighted in the present study, kidneys from DCD donors have a numerically higher incidence of UCs compared to DBD kidneys, but the incidence of UCs is <5% and is not statistically significant when other variables are taken into account. Notably, we found no association between the warm ischemia time integral to DCD donation and risk of UCs, although the warm ischemia time was relatively short in this cohort.

It is important to acknowledge some limitations of the present study. This was a single-center, retrospective, observational, cohort study that investigated a relatively uncommon complication after kidney transplantation raising the possibility of a type II error. A randomized controlled trial would have been required to definitively address the association between donor type and risk of UCs, but this would have to include a large sample size to ensure adequate power and is, therefore, likely to be impractical. Despite the limitations of the study design, the present study is one of the largest of its kind to date, in which DCD transplants comprised almost half of the entire cohort. It is also important to acknowledge that, despite performing a nested case-control study to investigate the potential association of UTI and the risk of developing UCs, inclusion of the entire patient cohort might have been more informative.

In conclusion, the present series shows that, despite a significant increase in the number of kidney transplants from DCD donors, the incidence of UCs remains low. No obvious predisposing factors to UCs were identified in our analysis. When UCs do occur, they can be treated successfully with surgical reconstruction with no adverse effect on graft or patient survival.

AUTHORS' CONTRIBUTIONS

TJM and DHM: Performed acquisition of data, analysis and interpretation of data, and statistical analysis. OB: Performed acquisition of data. KSP and AJB: Carried out research design and drafting of manuscript. JAB: Carried out research design, analysis and interpretation of data, and drafting of manuscript. VK: Carried out research design, acquisition of data, analysis and interpretation of data, drafting of manuscript, and study supervision.

CONFLICT OF INTEREST

The authors report no conflict of interests.

REFERENCES

1. Davari HR, Yarmohammadi H, Malekhosseini SA, Salahi H, Bahador A, Salehipour M. Urological complications in 980 consecutive patients with renal transplantation. *Int J Urol*. 2006;13:1271–1275.
2. Nie ZL, Zhang KQ, Li QS, Jin FS, Zhu FQ, Huo WQ. Urological complications in 1,223 kidney transplantations. *Urol Int*. 2009;83:337–341.
3. Eufrasio P, Parada B, Moreira P, et al. Surgical complications in 2000 renal transplants. *Transplant Proc*. 2011;43:142–144.
4. Zavos G, Pappas P, Karatzas T, et al. Urological complications: analysis and management of 1525 consecutive renal transplantations. *Transplant Proc*. 2008;40:1386–1390.
5. Karam G, Hétet JF, Maillet F, et al. Late ureteral stenosis following renal transplantation: risk factors and impact on patient and graft survival. *Am J Transplant*. 2006;6:352–356.
6. Rajpoot DK, Gomez A, Tsang W, Shanberg A. Ureteric and urethral stenosis: a complication of BK virus infection in a pediatric renal transplant patient. *Pediatr Transplant*. 2007;11:433–435.
7. Saeb-Parsy K, Kosmoliaptsis V, Sharples LD, et al. Donor type does not influence the incidence of major urologic complications after kidney transplantation. *Transplantation*. 2010;90:1085–1090.
8. Mangus RS, Haag BW. Stented versus nonstented extravesical ureteroneocystostomy in renal transplantation: a metaanalysis. *Am J Transplant*. 2004;4:1889–1896.
9. Alberts VP, Idu MM, Legemate DA, Laguna Pes MP, Minnee RC. Ureterovesical anastomotic techniques for kidney transplantation: a systematic review and meta-analysis. *Transpl Int*. 2014;27:593–605.
10. Wilson CH, Rix DA, Manas DM. Routine intraoperative ureteric stenting for kidney transplant recipients. *Cochrane Database Syst Rev*. 2013;6:CD004925.
11. NHSBT. Annual Activity Report. 2015. http://nhsbtmediaservices.blob.core.windows.net/organ-donation-assets/pdfs/kidney_activity.pdf. Accessed May, 2016.
12. Kootstra G, Daemen JHC, Oomen APA. Categories of non-heart-beating donors. *Transplant Proc*. 1995;27:2893–2894.
13. Kosmoliaptsis V, Salji M, Bardsley V, et al. Baseline donor chronic renal injury confers the same transplant survival disadvantage for DCD and DBD kidneys. *Am J Transplant*. 2015;15:754–763.
14. Port FK, Bragg-Gresham JL, Metzger RA, et al. Donor characteristics associated with reduced graft survival: an approach to expanding the pool of kidney donors. *Transplantation*. 2002;74:1281–1286.
15. Mallon DH, Summers DM, Bradley JA, Pettigrew GJ. Defining delayed graft function after renal transplantation: simplest is best. *Transplantation*. 2013;96:885–889.
16. Howell WM, Harmer A, Briggs D, et al. British Society for Histocompatibility & Immunogenetics and British Transplantation Society guidelines for the detection and characterisation of clinically relevant antibodies in allotransplantation. *Int J Immunogenet*. 2010;37:435–437.
17. Kwong J, Schiefer D, Aboalsamh G, Archambault J, Luke PP, Sener A. Optimal management of distal ureteric strictures following renal transplantation: a systematic review. *Transpl Int*. 2016;29:579–588.
18. Summers DM, Johnson RJ, Hudson A, Collett D, Watson CJ, Bradley JA. Effect of donor age and cold storage time on outcome in recipients of kidneys donated after circulatory death in the UK: a cohort study. *Lancet*. 2013;381:727–734.
19. Summers DM, Johnson RJ, Allen J, et al. Analysis of factors that affect outcome after transplantation of kidneys donated after cardiac death in the UK: a cohort study. *Lancet*. 2010;376:1303–1311.

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