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Outcomes of Percutaneous Management of Anastomotic Ureteral Strictures in Renal Transplantation: Chronic Nephroureteral Stent Placement with and without Balloon Dilatation

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Abstract
Purpose This study was designed o evaluate outcomes of percutaneous management of anastomotic ureteral strictures in renal transplants using nephroureteral stents with or without balloon dilatation.
Methods A retrospective audit of 1,029 consecutive renal transplants was performed. Anastomotic ureteral strictures were divided into two groups: nephroureteral stent only (NUS) and NUS+PTA (nephroureteral stent plus percutaneous transluminal angioplasty), with each cohort subdivided into early versus late presentation (obstructive uropathy occurring <90 day or >90 days from transplant, respectively). Overall and 6-month technical success were defined as removal of NUS any time with <30 % residual stenosis (any time lapse less or more than 6 months) and at >6 months, respectively. Patency was evaluated from NUS removal to last follow-up for both groups and compared.
Results Sixty-seven transplant patients with 70 ureteric anastomotic strictures (6.8 %, $n = 70/1{,}029$) underwent 72 percutaneous treatments. 34 % were late (>90 days, $n = 24/70$), and 66 % were early (<90 days, $n = 46/70$). Overall technical success was 82 % ($n = 59/72$) and 6-month success was 58 % ($n = 42/72$). Major and minor complications were 2.8 % ($n = 2/72$), and 12.5 % ($n = 9/72$). NUS+PTA did not improve graft survival ($p = 0.354$) or patency ($p = 0.9$) compared with NUS alone. There was no difference in graft survival between treated and non-treated groups ($p = 0.74$).
Conclusions There is no advantage to PTA in addition to placement of NUS, although PTA did not negatively impact graft survival or long-term patency and both interventions were safe and effective. Neither the late or early groups benefited from PTA in addition to NUS. Earlier obstructions showed greater improvement in serum creatinine than later obstructions.

Keywords Nephrostomy/Nephrostogram · Urogenital interventions · Urogenital · End stage renal disease · Urinary tract · Recanalization/Recanalisation · Nephroureteral stent

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Introduction

Renal transplantation is the treatment of choice for patients with end-stage renal disease (ESRD), having nearly doubled from 8,878 transplants in 1,988 to 16,893 transplants in 2013 [1, 2]. With the advances in surgical technique and immunomodulation over the past three decades, the rate of

urologic complications has decreased from 10–25 % to 2–14.1 % [3–5]. Unfortunately, ureteral stricture, obstruction, and leak remain the most common urologic complications, especially in the early period, and remain a significant cause of morbidity and mortality for renal transplant recipients, especially if prompt interventional therapy is not instituted [6–8].

While ureteral complications have not shown to threaten graft or patient survival when treated promptly and adequately, it is the advent of percutaneous and endourologic techniques that has allowed patients to undergo less invasive treatments than reexploration and open repair of the ureteral anastomosis [9]. Interventional radiology plays a key role in the management of complications in kidney transplantation, most of which can be diagnosed and treated by minimally invasive interventional radiologic techniques [10, 11]. Placement of percutaneous nephrostomy (PCN) followed by nephroureteral stenting (NUS) has become routine management in the obstructed transplant ureter [5, 12–15]. More recently, treatment with high-pressure and cutting balloons, also known as percutaneous balloon dilation, has been added to the management of transplant ureteral strictures, albeit with mixed results [7, 16–19]. To date, it is not clear whether chronic (over months), indwelling, ureteric stent placement (NUS or endoscopically placed) is the ideal treatment or whether balloon dilation has a role in the management of post-transplant ureteric anastomotic strictures. The purpose of this study was to provide additional clinical data in a larger series of transplant ureteral strictures and to determine the utility of percutaneous balloon dilatation when combined with NUS versus NUS alone.

Materials and Methods

After approval by the Institutional Review Board, a retrospective audit of 1,029 patients receiving renal transplants between January of 1998 and December 2010 was performed. Anastomotic ureteral strictures were divided into two cohorts: those receiving only NUS, and those receiving a combination of NUS and percutaneous balloon dilatation. The treatment method (to augment with balloon dilation) was operator-dependent and the general intent of chronic NUS ureteric stenting was stenting the ureteric stricture for months (usually 3–6 months). Additionally, early strictures were defined as strictures occurring within 3 months (within 90 days) of transplantation, and late strictures were defined as occurring more than 3 months following transplantation. The early and late groups were based on prior work by Fontaine et al., [17] who also defined early and late strictures as occurring before and after a 3-month interval between transplantation and initial percutaneous intervention.

Ureteric patency was determined by antegrade pyelograms during the initial PCN and during subsequent NUS changes, which were routinely performed every 6–8 weeks. Overall technical success was defined as ability to place the NUS and to remove it leaving a patent ureter (<30 % residual stenosis) regardless of how much time lapsed from the initial stenting (NUS placement) to the NUS removal. Technical failures were defined as presence of the NUS at graft nephrectomy, presence of the NUS at surgical revision of the anastomosis, or death with the NUS still in place (NUS dependence). Technical success at 6 months was defined as ability to remove the NUS at 6 months or earlier (intubation period of 6 months or less) leaving a patent ureter (<30 % residual stenosis). Technical failure at 6 months was defined as presence of the tube at last available follow up after 6 months from placement (NUS dependence for 6 months or more).

Primary unassisted patency (PUP) was defined as the time between tube removal and last available follow-up or graft loss date, not requiring additional intervention or de novo NUS placement. Patients who required de novo NUS placement due to stricture were considered restenoses. Obstructions requiring de novo NUS or PCN placement due to clot or stones were not considered restenoses. Graft loss was determined if the patient underwent graft nephrectomy or suffered renal failure requiring dialysis. Graft survival was measured from the date of PCN placement until last follow-up or graft loss date.

Peak serum creatinine before decompression was recorded, with the highest value available 1–7 days before the procedure. The trough in serum creatinine after decompression within 1–14 days was recorded. Serum creatinine values were then compared for early and late groups using the Mann–Whitney *U* test. Minor complications were defined as blood clots in the collecting system, partial tube dislodgement not requiring de novo access, and local infection requiring antibiotics. Major complications included hemorrhage requiring blood transfusion, sepsis, perforation of the renal pelvis, tube dislodgement requiring de novo access, and death.

A p value <0.05 was considered statistically significant. Demographic comparisons for age, time from transplant, baseline creatinine, NUS size, and intubation period were compared using the Mann–Whitney *U* test. Comparisons between cohorts for gender, concomitant transplant (solitary renal vs. additional solid organ transplant), transplant location (RLQ vs. LLQ), donor type (living vs. deceased) were performed using the Fisher exact test. Etiology of kidney disease was compared between the cohorts using the Chi square test. Graft survival and patency was estimated using a nonparametric Kaplan–Meier survival curve. Graft survival was measured from both the transplant date and from the date from which the NUS tube was pulled.

Table 1 Patient demographics for the NUS-only and the NUS+PTA groups, and the early and late groups

Demographics Criteria		NUS only group ($n = 27$)	NUS+PTA group ($n = 40$)	p value	Early group ($n = 46$)	Late group ($n = 24$)	p value
Age (years)	Mean (SD)	52 (14)	46 (15.4)	0.120*	50 (15.2)	44 (16.5)	0.139*
	Range	21–70	13–71		13–71	16–69	
Gender	Male	19 (70 %)	27 (68 %)	0.804**	32 (70 %)	13 (54 %)	0.202**
	Female	8 (30 %)	13 (32 %)		14 (30 %)	11 (46 %)	
Concomitant transplant	Solitary	26 (96 %)	36 (90 %)	0.336**	44 (96 %)	22 (91 %)	0.378***
	Kidney pancreas	0 (0 %)	2 (5 %)		1 (2 %)	0 (0 %)	
	Kidney liver	1 (4 %)	2 (5 %)		1 (2 %)	2 (8 %)	
Transplant position	RLQ	16 (62 %)	24 (60 %)	0.952**	27 (59 %)	14 (58 %)	0.977**
	LLQ	11 (38 %)	16 (40 %)		19 (41 %)	10 (42 %)	
Days from transplant	Mean (SD)	204 (378)	515 (1,121)	0.147*	35.4 (27.7)	1090 (1,274)	<0.0000001*
	Range	4–1704			2–86	92–5795	
Etiology of kidney disease	DM	10 (37 %)	15 (38 %)	0.477***	18 (39 %)	7 (29 %)	0.897***
	HTN	5 (19 %)	9 (23 %)		10 (22 %)	5 (21 %)	
	Other	14 (52 %)	15 (38 %)		22 (48 %)	11 (46 %)	
Donor type	Living	9 (33 %)	10 (25 %)	0.458**	14 (30 %)	9 (38 %)	0.550**
	Deceased	18 (67 %)	30 (75 %)		32 (70 %)	15 (62 %)	
Baseline creatinine	Mean (SD)	4 (1.8)	3.3 (2.1)	0.090*	3.79 (2.22)	3.23 (1.66)	0.423*
	Range	1.2–7.4	1.2–10.8		1.2–10.8	1.2–6.9	
Largest NUS size (French)	Mean (SD)	9.7 (2.01)	11.7 (1.76)	0.00015*	10.8 (2.05)	10.9 (2.41)	0.951*
	Range	8–16	8–14		8–14.5	8–16	
Intubation period (mo)	Mean (SD)	6.6 (17.3)	7.8 (8.6)	0.003*	7.01 (13.67)	7.8 (10.53)	0.824*
	Range	0.3–93	0.3–52		0.3–93	1–52	
Management technique	NUS only				20 (43 %)	10 (42 %)	0.884**
	NUS+PTA				26 (57 %)	14 (58 %)	

The only significant differences were found in the largest tube size and the total intubation period for the NUS and NUS+PTA groups

SD standard deviation, *RLQ* right lower quadrant, *LLQ* left lower quadrant, *DM* diabetes mellitus, *HTN* hypertensive nephropathy

* Mann–Whitney *U* test; ** Fischer exact test, *** Chi square

Censored group graft survival analysis with intervals also was performed. Comparison of the different patency rates and graft survival rates was made by the log-rank test. Values that were not estimable were denoted with the abbreviation "*n/e*."

Results

Seventy ureteral anastomotic strictures were identified in 67 transplant patients, representing 6.8 % of all evaluated transplants ($n = 70/1{,}029$), which underwent 72 percutaneous interventions. Average time from transplant was 528.6 days (range 2–5,903 days; standard deviation 1,191). Patients presenting early were 66 % of the total strictures ($n = 46/70$) and late presenters were 34 % of the total strictures ($n = 24/70$). One patient crossed over from the NUS group to the NUS+PTA group after undergoing a second placement of NUS for a recurrent stricture. One patient from the NUS+PTA group received a total of three transplants, all of which underwent NUS+PTA. Detailed results of this demographic analysis can be found in Table 1 (for NUS-only vs. NUS+PTA) and Table 2 (for early vs. late presentation).

One patient with a ureteric stricture died of sepsis after placement of the PCN and did not receive either balloon dilation or NUS. Another patient underwent surgical revision of the ureteral anastomosis immediately after placement of the PCN. These patients were included in the analysis on the basis of intent to treat (both were considered technical failures). The overall complication rate was 15 % ($n = 11/72$). The minor complication rate was 12.5 % ($n = 9/72$), including four partial tube migrations not requiring de novo access, three nonobstructing clots in the collecting system, one case of fungal pyelonephritis, and one access site abscess resolving with antibiotic therapy. The major complication rate was 2.8 % ($n = 2/72$), including one death due to sepsis and one tube dislodgement requiring de novo PCN access. There were three restenoses in the NUS only group (10.7 %, 3/28) and four in the NUS+PTA group (9.5 %, 4/42).

Table 2 Kaplan–Meier mean and median graft survival times in months for graft survival

Graft survival subgroup analysis and serum creatinine	Group	Number of observations	NUS Only	NUS+PTA	Number of events (graft loss)	Mean (SD) survival times (mo)	Log-rank test NUS or NUS+PTA versus early or late presentation
Survival from transplant	Early	44	19	25	12	90.6 (8.7)	$p = 0.944$
	Late	23	8	15	9	113.6 (19.1)	$p = 0.094$
Survival from tube removal	Early	44	19	25	12	90.6 (8.7)	$p = 0.999$
	Late	23	8	15	9	52.2 (9.9)	$p = 0.182$

	Pre-procedure Cr	Post-procedure Cr	Mann–Whitney U test Pre versus post	NUS versus NUS+PTA
Serum creatinine (Cr)				
NUS	4.0	2.67	$p = 0.011$	$p = 0.064$
NUS+ PTA	3.3	2.0	$p = 0.0011$	
Early Presentation	3.79	1.93	$p < 0.0001$	
Late presentation	3.23	2.7	$p = 0.28$	

Transplants presenting with strictures before or after 90 days (early and late groups) did not benefit from PTA in addition to NUS placement ($p = 0.944$ and $p = 0.094$, respectively). When measuring from time of tube removal, neither early or late presentation grafts benefitted from PTA in addition to NUS placement ($p = 0.999$ and $p = 0.182$, respectively). Mean creatinine pre-procedure measured 4.0 in the NUS group and 3.3 in the balloon dilation group. The post-procedure creatinine averaged 2.67 and 2.0 in the NUS and balloon dilation groups, showing a statistically significant decrease of $p = 0.011$ and $p = 0.0011$, respectively. There was no difference in posttreatment serum creatinine between the NUS and balloon dilation groups ($p = 0.064$). Early stricture mean creatinine was 3.79, and late strictures averaged 3.23. There was a significant decrease in the serum creatinine for patients presenting with early strictures to a mean of 1.93 ($p < 0.0001$). Late strictures showed a decrease in serum creatinine of a mean 3.2–2.7 ($p = 0.28$)

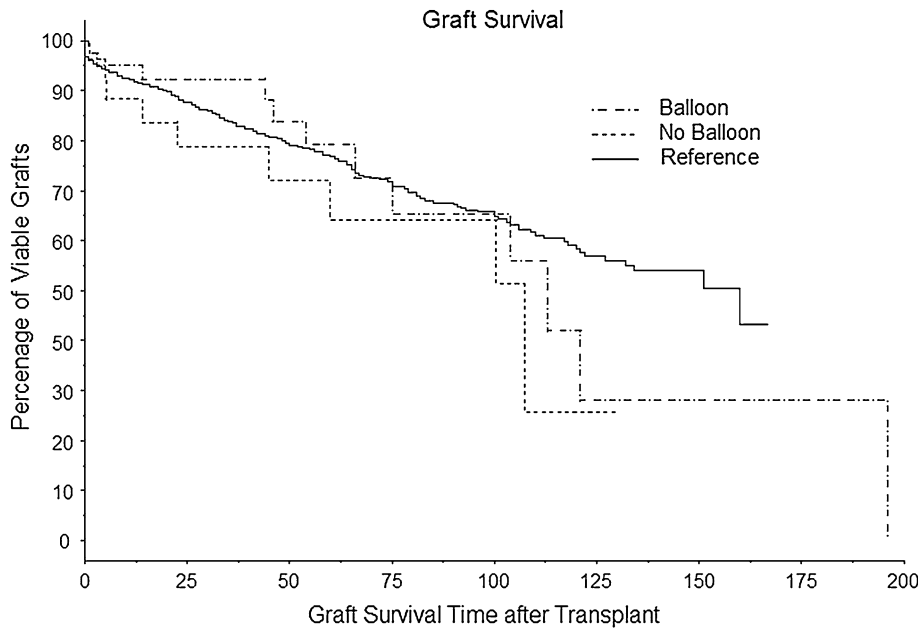

Fig. 1 Kaplan–Meier estimates for graft survival comparing treatment versus nontreatment groups. The *solid line* denotes the graft survival curve for the reference group (transplants not receiving NUS or NUS+ balloon dilation); the *thick hatched line* denotes the graft survival curve for the graft procedures in which a balloon was used, and the *thin hatched line* denotes the graft survival curve for the graft procedures in which no balloon was used. There was no difference in graft survival when comparing the NUS only group versus the nontreated population ($p = 0.17$) and when comparing the NUS+PTA group versus the nontreated population ($p = 0.74$)

Improvement in renal function measured in serum creatinine before and after the procedures was statistically significant in all groups except strictures, which presented late; results are summarized in Table 2. Overall technical success was 82 % ($n = 59/72$). Technical success at 6 months was 58 % ($n = 42/72$).

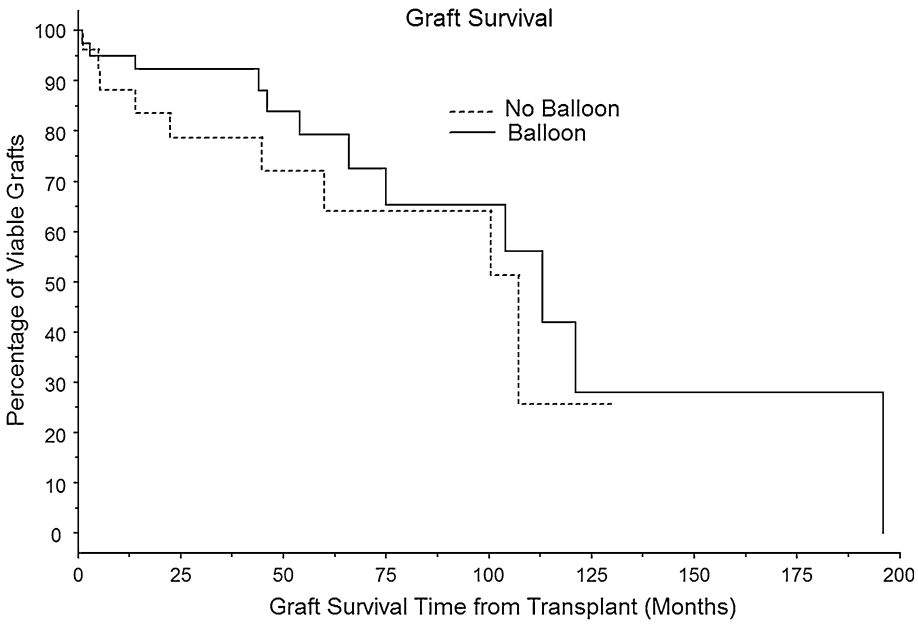

Fig. 2 Kaplan–Meier estimates for graft survival. The *solid line* denotes the graft survival curve for the graft procedures in which balloon dilation was used, and the *hatched line* denote the graft survival curve for the graft procedures in which a balloon dilation was not used. Addition of balloon dilation to NUS treatment did not show increased graft survival ($p = 0.354$)

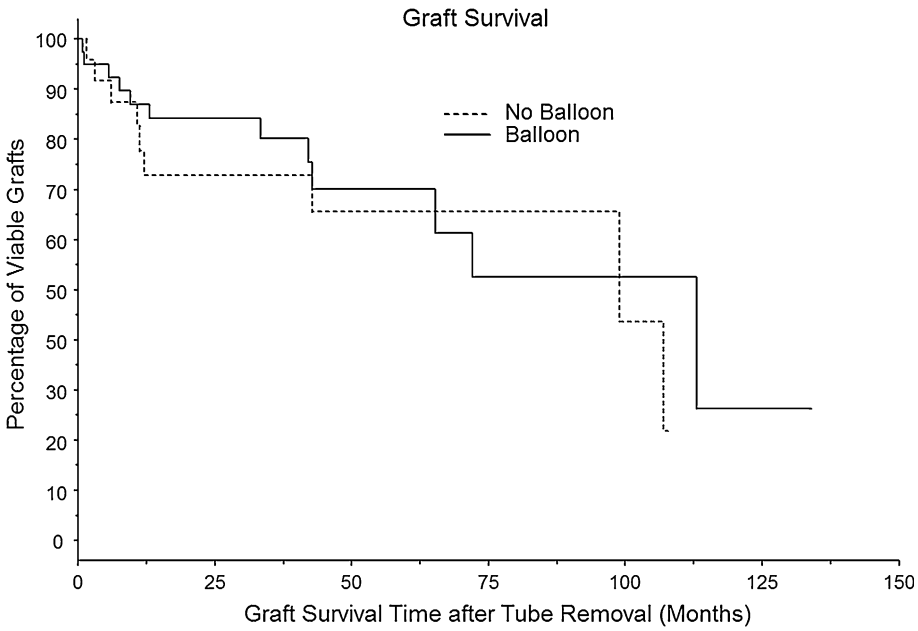

Fig. 3 Kaplan–Meier estimates for graft survival from removal of the NUS tube. The *solid line* denotes the graft survival curve for the graft procedures in which a balloon was used and the *hatched line* denotes the graft survival curve for the graft procedure in which a balloon was not used. From the time of tube removal, no improvement in graft survival was observed between the groups ($p = 0.567$)

Of the 1,029 consecutive patients who underwent renal transplantation, 244 renal grafts were lost (23.7 %). Mean time for graft loss was 119 ± 2.7 months, with a median time to graft loss of 156 months (95 % confidence interval [CI] 131 months, *n/e*). The use of NUS or NUS and balloon dilation had no impact in the overall graft survival of the treated patients compared with the overall data set ($p = 0.17$, $p = 0.74$, respectively; Fig. 1). Overall graft survival at 1, 3, and 6 years was 92 % ± 3, 79 % ± 6, and 59 % ± 9 for the 67 transplants requiring intervention. The graft survival curves for the NUS and the NUS+PTA groups are shown in Fig. 2. A detailed subgroup analysis of graft survival measured from transplant and from tube removal in the NUS and NUS+PTA groups in the early and late strictures is summarized in Table 2. There was no statistically significant difference in graft survival from time of transplant between the NUS and the NUS+PTA groups ($p = 0.354$) and no statistically significant difference when measuring survival from the time of tube removal between the NUS and NUS+PTA groups (0.567) (Fig. 3).

Censored group graft survival analysis with intervals showed that graft survival from transplant at intervals of 1, 6, and 10 years was 86 % ± 7, 78 % ± 12, 29 % ± 17 for the early group and 96 % ± 2, 73 % ± 8, 61 % ± 13 for the late group, which was statistically significant ($p < 0.05$). However, the Kaplan–Meier analysis (Fig. 4) did not show a

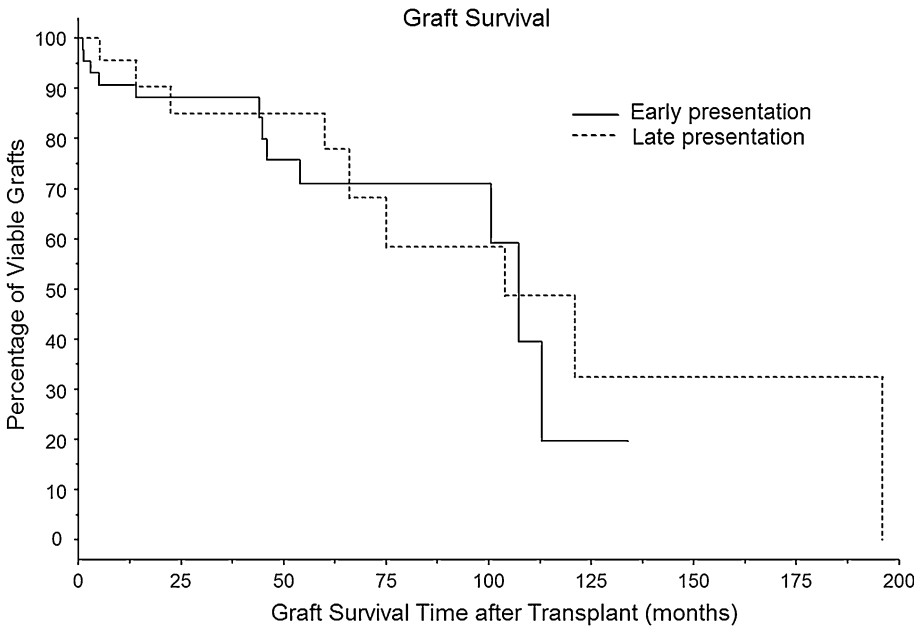

Fig. 4 Kaplan–Meier estimates for graft survival. The *solid line* denotes the graft survival curve for the early presentation patients, and the *hatched line* denotes the graft survival curve late presentation patients. There was no difference in graft survival between late presenters and early presenters from transplant date ($p = 0.692$)

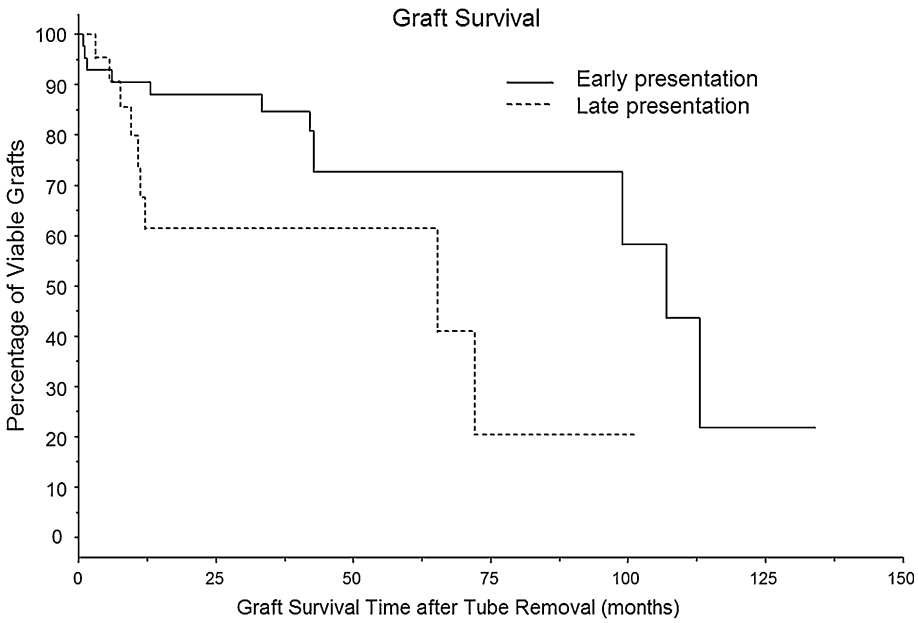

Fig. 5 Kaplan–Meier estimates for graft survival. The *solid line* denotes the graft survival curve for the early presentation patients, and the *hatched line* denotes the graft survival curve late presentation patients. There was a statistically significant difference between the late and early groups ($p = 0.032$) when measuring from tube removal, with a median graft survival for the early group of 107 months (95 % CI 99.0, *n/e*) versus 65.3 months for the late group (95 % CI 10.8, *n/e*; Table 2)

significant difference between the early and late groups when measuring from transplant date ($p = 0.692$). When measuring from tube removal, there was a statistically significant difference between the late and early groups ($p = 0.032$), with a median graft survival for the early group of 107 months (95 % CI 99.0, *n/e*) versus 65.3 months for the late group (95 % CI 10.8, *n/e*; Fig. 5).

Overall PUP at intervals of 1, 3, and 5 years was 80 % ± 6, 74 % ± 8, and 73 % ± 10, respectively. NUS-only and NUS+PTA PUP at 1, 2, and 3 years was 85 % ± 7, 79 % ± 10, 79 % ± 11, and 76 % ± 8, 76 % ± 9, 70 % ± 10, respectively ($p = 0.25$). Early and late PUP at 1, 2, and 3 years was 78 % ± 6, 75 % ± 8, 71 % ± 8 and 80 % ± 11, 80 % ± 17, 80 % ± 17, respectively ($p > 0.05$). In the NUS group, two patients had restenoses requiring replacement of the NUS, and one of these went for balloon dilation. In the NUS+PTA group, a single patient required two de novo PCN procedures secondary to restenosis of the ureter following balloon dilatation.

Discussion

Despite widespread adoption of the ureteroneocystostomy technique of implantation, nonvascular complications of renal transplantation are still encountered with significant

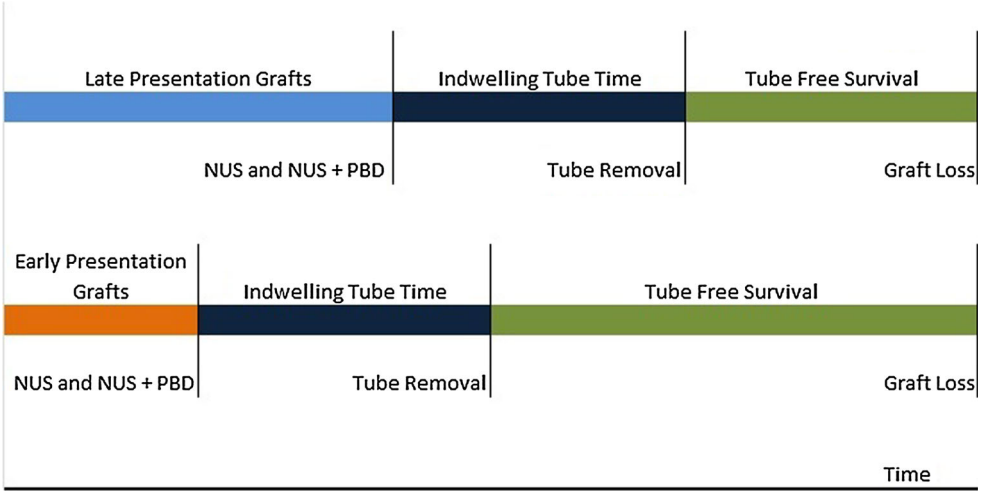

Fig. 6 Graphic demonstrating a possible bias in the increased tube-free graft survival for early presentation grafts, which may not be meaningful, because overall tube graft survival was not significantly different between the two groups. Early presentation grafts had the tubes placed and removed earlier, but the total indwelling time was similar (Table 1, $p = 0.824$)

frequency [13]. These include perinephric collections, lymphocele, abscess, hematoma, urinoma, leak, stricture, and obstruction [3, 12]. It is estimated that 2–10 % of recipients experience ureteral stricture, with up to 90 % of these resulting from ischemia of the distal ureteroneocystostomy [4, 13]. Causes of obstruction other than ischemia also include high anastomosis at the mobile anterior dome of the bladder, extrinsic compression, and intrinsic obstruction, which can be further divided into extrinsic compression from lymphocele, abscess, and hematoma, and into intrinsic obstruction from edema, clot, tumor, or calculus. Other causes quoted in the literature include ureteropelvic fibrosis, mycetoma, and crossing vessels, and in our experience, a single patient with a gravid uterus [11]. This series shows an overall urologic complication rate of renal transplantation of 6.8 %, which is consistent with the range of 2–10 % quoted in the literature [4, 13]. Our data show that PCN placement, followed by NUS placement is safe and effective, with overall patency >70 % at 5 years. Balloon dilatation of the transplanted ureteral strictures did not improve patency or graft survival, despite having no adverse effect on the same. There was a significant drop in serum creatinine after the procedure, representing an improvement in renal function following decompression of the transplanted collecting system. This benefit was most noticeable in grafts presenting with early strictures, in both grafts treated with NUS alone or NUS and balloon dilation. The improvement in function was not superior in grafts treated with NUS and balloon dilation versus NUS alone.

Several series have reported overall success rates for percutaneous intervention in posttransplant ureteric strictures ranging from 30 to 92 % [5–7, 16–23]. Technical success (as defined by ability to remove the PCN) has been reported between 43 and 100 % in early presenters and 16–66 % in late presenters. Using the stricter definition of technical success (intubation period under 6 months), we showed a 58 % ($n = 42/72$) success rate, which is in line with the prior series. We can confirm that technical success at 6 months is lower in patients presenting with late strictures (>3 months from transplant).

Intervention with NUS or NUS and balloon dilation has no negative effect on overall graft survival when compared with the entire data set ($p = 0.198$, $p = 0.8$). It is likely that transplant kidneys requiring placement of a nephrostomy tube and NUS with or without balloon are not adversely affected by the procedure. The most important conclusion that can be drawn from this study is that no improvement in function, patency, or graft survival was shown in the group treated with NUS and balloon dilation versus the group treated with NUS alone. While there is no defined treatment algorithm for stenosis of renal transplant ureters, these results suggest that balloon dilatation should not be a routine part of therapy, being reserved for recalcitrant strictures which do not respond to NUS alone.

When observing the interval censored data, there was a difference in graft survival from patency from the transplant date, favoring longer graft survival for patients presenting after 90 days from transplant. However, the Kaplan–Meier curves were not significantly different and did not support the results seen in the censored interval analysis. The significant difference ($p = 0.032$) between graft survival measured from tube removal favoring the early group is most likely secondary to bias related to tube placement time, because graft loss is a multifactorial problem, as demonstrated in Fig. 6. We thus conclude that while grafts that presented early may have longer tube-free graft survival, the lack of difference in overall graft survival from both groups indicates that both early and late grafts were lost due to other factors and the decreased tube-free graft survival in late grafts may not be meaningful.

The restenosis rate in the NUS group was 10.7 % (3/28) and in the NUS+PTA group the restenosis rate was 9.5 %

($n = 4/42$). The range of restenosis rates in the literature is 0–40 %. Aytekin et al. [7, 18, 21, 22, 24, 25] observed a recurrent stricture rate of 44 % in the early group and 40 % in the late group [7]. Pappas et al. and Voegeli et al. [23, 24] had two recurrent strictures, with a rate of 17 % ($n = 2/12$). Both of the restenoses in the study by Pappas et al. [23] occurred in the late group and all patients underwent NUS placement without PTA. Bachar et al. observed restenosis rates of 38 % following PDB ($n = 8/21$), with five patients undergoing surgery and three with chronic indwelling internal double J stents [21]. Yong et al. [18] had a restenosis rate of 33 % ($n = 3/9$), all of them in patients who underwent balloon dilation.

An alternative treatment for ureteral strictures is surgical revision of ureteroneocystostomy. Lehmann et al. found a 14 % restenosis rate in patients with repeat ureteroneocystostomy, considerably lower than the restenosis rates described in the above studies [25]. In a large series of 1,157 renal transplants, Alberts et al. found 142 urologic complications, 82 of which were treated with PCN, and 60 underwent surgery. This study quoted a surgical success rate of 92 %, with an 8 % restenosis rate requiring further surgical revision ($n = 5/60$). The overall complication rate for ureteroneocystostomy in this study was 22 %, with 6 of 60 nonurological complications and 7 of 60 urological complications [26]. A smaller study by Sandhu et al. [13] showed a urological complication rate of 40 %, with one leak and three obstructions, two of which required nephrectomy for pyelonephritis, in ten patients undergoing repeat ureteropyelostomy. Further comparison between restenosis rates of NUS placement with and without PDB versus ureteral reconstruction may be warranted, especially because the series presented here demonstrates a similar restenosis rate in the treated population.

Although relatively benign, PCN with NUS placement is not without risks, and the procedure possesses a distinct side-effect profile. Complications of NUS placement include bladder discomfort/irritation, bacteriuria, urosepsis, hematuria, flank/loin pain, dislocation, renal pelvic perforation, and tube fragmentation [28]. Minnee et al. [27] have quoted a relative risk of UTI in transplant patients with NUS of 1.49. The treatment of urologic complications in renal transplants is not limited to NUS and balloon dilation, however, and there is a small but growing interest in other devices and methods related to the prevention of leaks, strictures, and obstructions. Cantasdenir et al. reported successful use of a self-expanding metallic stent, which also was shown to be successful by Burgos et al. [28, 29]. The series by Burgos et al. included 11 patients and demonstrated 73 % patency at 48 months with self-expanding metallic stents [28]. In addition, use of an antireflux device has been reported, albeit without any apparent benefit to the prevention of renal transplant vesicoureteral reflux or UTI [30]. Another option that may merit further investigation is the use of Holmium:Yag laser therapy in transplant ureteric strictures, as shown by Kristo et al., who demonstrated 100 % success rate with a 24 month follow-up in strictures <2 cm in length [22].

We believe our series to be the largest one to date in the available literature evaluating the efficacy of interventional radiological procedures in the treatment of renal transplant ureteric strictures. A few of the conclusions made by previous authors were confirmed by our study, including increased failure rates in patients presenting with strictures after 3 months. Our recommendation based on our results is that PCN with NUS placement after 24–48 h should be first line therapy, reserving balloon dilation to patients with recalcitrant strictures and high operative risk.

Conclusions

PCN followed by NUS and balloon dilation are safe and effective techniques for managing ureteral strictures following renal transplantation in the early and late periods. While placement of NUS with or without a PTA improves renal function, there is no significant advantage in terms of graft survival to balloon dilatation in addition to placement of NUS, although balloon dilation did not negatively impact graft survival or long-term patency. Neither the late or early groups benefited from NUS and balloon dilation versus NUS alone. Patients presenting with earlier obstructions were more likely to show improvement in serum creatinine than patients presenting later.

Conflict of interest Andre Uflacker , Daniel Sheeran , Minhajuddin Khaja , James Patrie , Gustavo Elias , Wael Saad have no conflict of interest.

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