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Ex vivo reconstruction of the donor renal artery in renal transplantation: a case–control study

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Summary

Transplantation of renal allografts with anatomic variability or injured vasculature poses a challenge to the transplanting surgeon but can be salvaged for transplantation with *ex vivo* bench reconstruction of the vasculature. We investigated whether renal allograft function is impaired in these reconstructed allografts; compared to the donor-matched, un-reconstructed allograft. Reconstructed allografts were transplanted into 60 patients at our institution between 1986 and 2012. A control group was selected from the matched pair of the recipient in deceased donor transplantation. We found no significant difference in the overall graft and patient survival rates ($P = 1.0$, $P = 0.178$). Serum creatinine levels were not significantly higher in the study group at 1, 3 and 12 months postoperatively. There were two cases of vascular thrombosis in the study group that were not related to the *ex vivo* reconstruction. A significantly greater proportion of reconstructed patients were investigated with a colour duplex ultrasound postoperatively (0.007). Although we have demonstrated a higher index of suspicion of transplant failure in patients with a reconstructed allograft, this practice has proven to be a safe and useful technique with equivocal outcome when compared to normal grafts; increasing the organ pool available for transplantation.

Introduction

Renal transplantation is the treatment of choice for end-stage kidney failure as it improves both the life expectancy and quality of life of the recipient. This surgical technique was originally described in 1951 and has only evolved slightly in 60 years [1]. Our institution is the only kidney transplantation centre in the Republic of Ireland, performing approximately 160 transplants per year [2]. There has been significant progress in transplantation over the last decade with improved graft and patient survival secondary to better immunosuppressive drugs, standardization in surgical technique and improvements in organ preservation and postoperative care. Currently, the main issues in transplantation are the shortage of available organs and growing numbers of patients awaiting transplantation, so it is imperative to attempt to increase the organ pool available for transplantation [3]. Renal allografts with anatomic variability or injured vasculature pose a technical challenge to the transplanting surgeon [4]. The presence of vascular anomalies, disease of the vessels or iatrogenic injury to the vasculature during organ procurement may necessitate reconstruction or repair of these vessels *ex vivo* prior to transplantation to salvage these allografts for transplant [5,6].

Few studies have addressed outcome following arterial reconstruction. There are two case–control studies in the literature comparing renal allografts with injured or diseased vasculature with standard renal allografts. In each of these studies, the groups are matched in terms of recipient demographic factors such as age, sex and premorbid conditions, and show favourable outcomes [7,8]. A case series of 104 allografts requiring reconstruction of anatomic anomalies or injured vasculature demonstrated that by using these techniques, they salvaged 30 suboptimal or previously deemed unusable grafts [9].

This is the first case–control study in the English literature comparing graft outcome in deceased donor renal transplantation in reconstructed donor allografts with standard donor allografts. It is the largest case–control study of this nature in the literature and is novel in study design as it is the first study comparing the outcome of matched pairs of renal allografts from the same donor, with the reconstructed allograft in the study group (SG) and it matched allograft from the same donor in the control group (CG).

The aim of our study was to investigate whether graft function and survival are impaired in renal allografts with a reconstructed renal artery compared to the matched, unreconstructed allograft from the same donor.

Materials and methods

Patients

Of a total of 3510 kidney transplants performed at our institution between January 1986 and December 2012, we identified 84 patients (2.4%) who received an allograft with a reconstructed renal artery. We excluded 15 recipients of a live donor allograft and those who received a cadaveric unpaired single kidney, nephron-dosing (dual) kidney transplants and en-bloc kidneys; leaving a total of 60 cases and 60 controls. The organ procurement and transplantation techniques were performed according to standard techniques. First-time transplants were generally transplanted into the right side of the recipient. As described by Chopin *et al.* [10], vena caval extension was routinely used for all right-sided kidneys to obtain additional length for the right renal vein. The ureteral anastomosis was routinely performed over a ureteric stent. All transplants were performed in a single centre by a senior surgeon on a rolling on-call rota.

Study design

The study design was an observational, retrospective, case–control study. The CG was formed from the patients who received the donor-matched allograft with a standard renal artery not requiring any form of reconstruction or repair. Each case was therefore matched to one control. The STROBE guidelines for the reporting of observational case–control studies were utilized for the reporting of this study [11]. The cases were identified from the operative records of each patient and from the National Renal Transplant Registry in Beaumont Hospital, Ireland. Prior to data collection, institutional review board approval was gained for this study and only patients on the renal transplant database were included in this study for which ethical approval has previously been awarded.

The eligibility criterion for this study was all patients on record in our institution who received a renal allograft with a reconstructed renal artery and with a donor-matched allograft that did not require reconstruction transplanted into another patient. 'Reconstruction of the renal artery' was defined as any surgical incision and/or repair of the main renal artery or polar artery for any reason that took place ex vivo prior to transplantation.

Statistical analysis

Stata® Release 12 software (StataCorp LP, College Station, TX, USA) was used for all analysis. Mean and minimum–maximum ranges are quoted unless otherwise stated. Statistical analyses were performed by paired t-test for normally distributed data, Wilcoxon's signed-rank test for non-normally distributed data and McNemar's test for comparing matched data with binary outcomes. Kaplan–Meier survival curves were plotted to estimate graft and patient survival.

Results

Demographics

There were 42 males and 18 females in the SG and 36 males and 24 females in the CG ($P = 0.362$). The mean age at transplantation was 44 (17–71) years in the SG and 49 (42–71) years in the CG ($P = 0.5732$). The main subtypes of end-stage renal failure in the study group were IgA nephropathy (23.3%), reflux nephropathy (16.7%), diabetes mellitus (10%), adult polycystic kidney disease (APKD) (10%) and glomerulonephritis (10%). In the control group, the main subtypes were glomerulonephritis (16.7%), APKD (13.3%), IgA nephropathy (11.7%), diabetes mellitus (11.7%) and hypertension (11.7%). 34 (57%) patients in the SG and 26 (43%) patients in the CG received a right donor kidney ($P = 0.366$), and the left kidney was therefore transplanted into 26 (43%) patients in the SG and 34 (57%) patients in the CG. Matching donor characteristics was found because of the use of paired kidneys with a mean donor age of 41 years ranging from 15 to 65 years. Patient demographics are outlined in Table 1 and the anatomic description of the vascular reconstruction performed is outlined in Table 2.

Forty-two per cent ($n = 25$) of patients in the study group and 45% ($n = 27$) of patients in the study group required quadruple immunosuppressive induction therapy with a calcineurin inhibitor [cyclosporine (CsA) prior to 2001 and tacrolimus (FK) from 2001 onwards]; an antimetabolite (azathioprine (A) prior to 2002 and mycophenolate mofetil (MMF) from 2002 onwards; prednisolone(DC) and anti-thymoglobulin (ATG-Fresenius®, Munich, Germany) or Basiliximab (Simulect®) because of increased immuno-

Table 1. Patient demographics.

Variable		Case	Control	*P*-value
Male (%)		70	60	0.362
Female (%)		30	40	0.362
Age (years)		44	49	0.57
Diabetes mellitus (%)		10	11.7	1.0
Previous transplants (%)	1st	90	87	0.79
	2nd	6	13	0.387
Preoperative dialysis (%)	Haemodialysis	65	66.7	1.0
	CAPD	26.7	25	1.0
	Pre-emptive	8.3	8.3	1.0
PRA	Mean	12 (0–99)	10 (0–96)	
	<80%	51 (85%)	49 (98%)	0.01
	>80%	9 (15%)	1(2%)	0.01
HLA miss-matches (total HLA-A, HLA-B, HLA-DR))		3 (0–6)	4 (0–6)	1.0
Immunosuppression (%)	Triple Therapy (CSA/FK + A/MMF+ Steroids)	58	55	0.625
	Quadruple Therapy (Triple + ATG/Simulect)	42	45	0.625
Kidney (%)	Right	57	43	0.366
	Left	43	57	0.366

CAPD, continuous ambulatory peritoneal dialysis; PRA, panel reactive antibodies (determined by the use of the complement-dependant cytotoxicity assay, NIH Basic technique); HLA, human leucocyte antigen; CSA, cyclosporine; FK, tacrolimus; A, azathioprine; MMF, mycophenolate mofetil; ATG, antithymocyteglobulin.

Table 2. Type of vascular reconstruction.

Repair of Injury/diseased vessel (%)	Branch vessel	8.4
	Main vessel	16.7
	Arterial patch	1.7
	Excision and repair of aneurysm	10
	Total	36.7
Reconstruction (%)	Branch vessel E-S to main vessel	18.3
	Two arteries anastomosed together	6.7
	Branch vessel anastomosed to patch	10
	Main vessel E-E to donor graft	13.3
	S-S anastomoses of 2 patches	13.3
	Main vessel E-E to synthetic graft	1.7
	Total	63.3

E-S, end-to-side; E-E, end-to-end; S-S, side to side.

Table 3. Operative outcomes and postoperative complications.

Variable		Case	Control	*P*-value
WIT (mins)		37.5 (22–69)	36 (17–77)	0.4956
CIT (hrs)		19 (8–35)	18 (11–27)	0.132
Transplant biopsy post operatively (%)		40	28	0.2649
CDUS post operatively (%)		28	8	0.007
Vascular complications (%)	Thrombosis	3	0	0.5
	Haemorrhage	2	2	1.0
Urine leaks (%)		5	3	1.0

WIT- warm ischaemia time; CIT- cold ischaemia time; CDUS- colour duplex ultrasound.

logical risk ($P = 0.625$). The remainder of patients received standard CsA- or FK-based triple therapy.

The mean panel reactive antibody was 12% (range 0–99%) in the SG and 10% (range 0–96%) in the CG ($P = 0.699$). 51 (85%) patients in the SG and 49 (98%) patients in the CG had a documented PRA of <80% ($P = 0.015$), while 9 patients in the SG and 1 patient in the CG had a documented PRA >80% ($P = 0.015$). Unfortunately, the PRA was not documented in 10 patients in the CG so these were omitted. There was a mean of 3 (0–6) HLA mis-matches in the study group and 4 (0–6) in the control group ($P = 1.0$).

As part of our departmental protocol, transplant patients also received an antipneumocystis prophylactic antibiotic in the form of trimethoprim-sulphamethoxazole during first 6 months of transplant; 3 doses of a broad spectrum penicillin-based antibiotic for peri-operative prophylaxis and valgancyclovir was also administered for cytomegalovirus (CMV) prophylaxis if either the donor or recipient was CMV positive.

Operative outcomes

The mean number of arterial anastomoses at transplantation was 1 [1,2] in both the SG and CG. The mean warm ischaemia time was 37.5 (22–69) min in the SG, comparable to 36 (17–77) min in the CG ($P = 0.4956$). The

Table 4. Graft and patient outcomes.

Variable		Case	Control	*P*-value
Overall graft survival rate (%)		81.7	80	1.0
Mean length of follow-up (months)		73 (1–288)	66 (1–192)	0.5786
1 yr graft survival rate (%)		93	96.7	1.0
5 yr graft survival rate (%)		84	86	1.0
Mean length of graft survival (months)		83	82	0.835
Postoperative creatinine (μmol/L)	1 month	165 (71–430)	143 (78–313)	0.08
	3 months	145 (66–332)	138 (68–330)	0.4385
	12 months	134 (66–278)	136 (70–314)	0.6961
eGFR (ml/min/1.73 m^2)		50.8 (18.8–89.9)	56.71 (42.5–69.2)	0.991
DGF (%)		20	12	0.26
Days spent on dialysis		7 (1–27)	5 (1–14)	1.0
Overall patient survival rate (%)		90%	82%	0.178

eGFR, estimated glomerular filtration rate; DGF, delayed graft function.

mean cold ischaemia time was 19 (8–35) h in the SG and 18 [11–27] h in the CG. There was one graft anomaly in the SG and 2 in the CG; all 3 cases were that of a duplex ureter.

17 (28%) patients in the SG and 5(8%) patients in the CG were investigated in the immediate postoperative period with a colour duplex ultrasound (CDUS) of their transplant kidney ($P = 0.007$), while 24(40%) patients in the SG and 17(28%) patients in the CG went on to have a biopsy of their transplant kidney in the postoperative period ($P = 0.2649$). Biopsies were only performed if there was clinical suspicion of rejection or deterioration of allograft function. Operative outcomes are outlined in Table 3.

Graft survival

The overall graft survival rate was 81.7% in the SG and 80% in the CG ($P = 1.0$). This was after a mean follow-up period of 73 months (range 1–288) in the SG and 66 months (range 1–192) in the CG ($P = 0.5786$). There were four early failures (i.e. failure within one year post-transplantation) in the SG giving a 1-year graft survival rate of 93%. 2 early failures in the CG gave a 1 year graft survival rate of 96.7% ($P = 1.0$). The causes of early failure in the SG were primary nonfunction in a patient that sustained a renal vein thrombosis and underwent a transplant nephrectomy on day 3 post-transplant; rejection at 1 month post-transplant; discontinuation of immunosuppressive therapy due to sepsis leading to graft loss one month post-transplant and the final early failure was secondary to primary non function leading to the patient undergoing a transplant nephrectomy on day 3 post operatively. The other grafts in the SG were lost to chronic allograft nephropathy ($n = 3$) at 183, 120 and 30 months post-transplant; and chronic rejection ($n = 3$) at 38, 66 and 90 months post-transplant. The final patient lost their graft at 92 months post-transplant with failure of the immunosuppressive therapy leading to severe acute rejection.

Both early failures in the CG were caused by primary nonfunction leading to graft failure at 1 and 5 days postoperatively. The other 10 grafts were lost to chronic rejection ($n = 3$) at 33, 159 and 48 months post-transplant; chronic allograft nephropathy ($n = 3$) at 113, 21 and 74 months; noncompliance with immunosuppressive therapy ($n = 2$) leading to graft loss at 83 and 85 months; and recurrence of the primary disease ($n = 2$) at 45 and 59 months post-transplant.

The mean length of transplant survival was 83 months in the SG and 82 months in the CG ($P = 0.835$). These graft survival rates are illustrated in Kaplan–Meier survival curves in Fig. 1a and b. Graft and patient outcomes are outlined in Table 4.

Graft outcomes

The mean creatinine at one month postoperatively in the study group was 165 (71–430) μmol/L compared to 143 (78–313) μmol/L in the control group ($P = 0.08$). At 3 months postoperatively, it was 145 (66–332) μmol/L in the SG and 138 (68–330) μmol/L in the CG ($P = 0.4385$), and at 12 months postoperatively, it was comparable at 134 (66–278) μmol/L in the SG and 136 (70–314) μmol/L in the CG ($P = 0.6961$).

The mean eGFR in the SG of all functioning grafts at latest follow-up was 50.82 (18.8–89.9) ml/min/1.73 m^2 according to the MDRD equation while that of the CG was 56.71 (42.5–69.2) ml/mil/1.73 m^2 ($P = 0.991$).

Delayed graft function (DGF) was defined as the requirement of dialysis in the first postoperative week and occurred in 12(20%) patients in the SG and 7(12%) patients in the CG ($P = 0.26$). SG patients with DGF spent a mean of 7 [1–27] days on dialysis postoperatively while

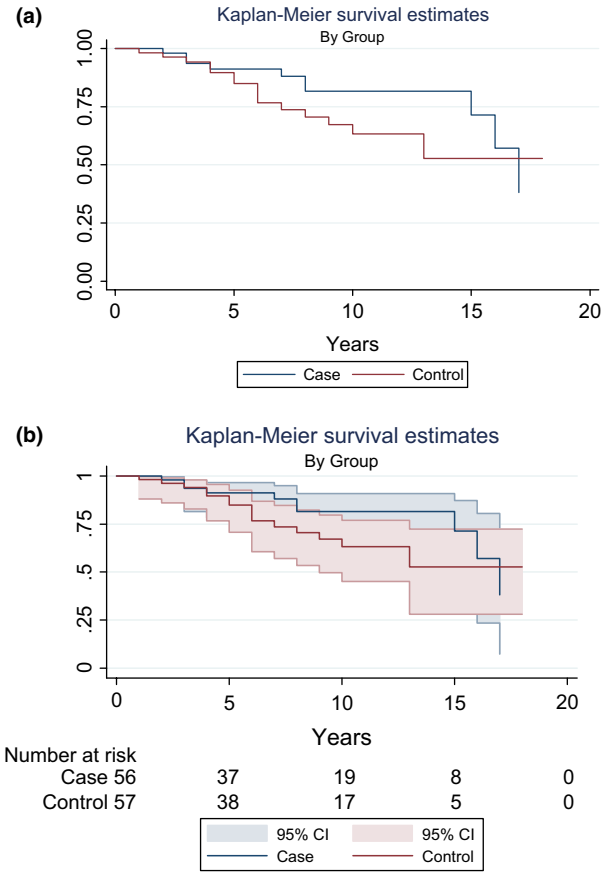

Figure 1 (a) Kaplan–Meier estimate of graft survival. (b) Kaplan–Meier estimate of graft survival with 95% confidence interval.

CG patients with DGF spent a mean of 5 [1–14] days ($P = 1.0$). 2 SG patients and 3 CG patients had primary nonfunction of their renal allograft.

Patient survival

The overall patient survival rate was 90% in the SG and 82% in the CG ($P = 0.178$). Three patients in the SG died with a functioning graft from malignancy at 197 months post-transplant, unknown causes at 45 months post-transplant and a cerebrovascular accident at 33 months post-transplant. The other 3 patients in the SG died 27, 135 and 36 months after transplant failure from unknown causes ($n = 2$) and sepsis ($n = 1$). Six patients in the CG died with a functioning graft; from sepsis ($n = 1$) at 6 months post-transplant; unknown causes ($n = 1$) at 85 months post-transplant; malignancy ($n = 3$) at 67, 113 and 19 months post-transplant; and cardiac failure ($n = 1$) at 19 months post-transplant. The other 5 patients in the CG died 22, 12, 42 and 22 months and 3 days after transplant failure. The causes of death were sepsis ($n = 1$), unknown causes ($n = 2$), cardiac failure ($n = 1$) and liver failure ($n = 1$).

Postoperative complications

Two patients in the SG sustained a vascular thrombosis of their allograft; one patient had a renal vein thrombosis leading to primary nonfunction of their graft; the *ex vivo reconstruction* in this case was a repair of a middle pole vessel injured at procurement. This patient is alive and dialysis-dependent at most recent follow-up. The other patient sustained an ischaemic thrombosis of an upper pole branch vessel and the reconstruction in this case was to the bifurcation of the main renal artery at the hilum of the kidney. This patient is alive with a functioning graft and a serum creatinine of 222 μmol/L at 3 months post-transplant. There were no documented cases of vascular thrombosis in the control group and there were no documented cases of post-operative renal artery stenosis in either group.

One patient in the SG had a surgical re-exploration for haemorrhage. This patient had haemorrhagic cystitis and a urine leak; the *ex vivo* reconstruction was S-S anastomosis of 2 aortic patches and after 72 months follow-up this patient is alive with a functioning graft and serum creatinine of 162 μmol/L. Haemorrhage was from the bladder only. 1 patient in the CG also had surgical re-exploration for postoperative haemorrhage; this patient had haemorrhage from the arterial anastomosis and subsequently had primary nonfunction of their graft. This patient subsequently died 3 days after graft failure from cardiac failure. 10(16.7%) patients in the study group were transfused a mean of 3 [1–8] units of red cells postoperatively, while 11 (18.3%) of controls were transfused a mean of 3 [1–6] units.

Five cases and four controls had documented urological or surgical complications post-transplantation. There were three urine leaks in the SG; the vascular reconstruction in each of these cases was an E-S anastomosis of a lower pole branch vessel to the main renal artery; E-S anastomosis of a lower pole vessel to the aortic patch and a S-S anastomosis of two aortic patches. An obstructed graft in the SG required a percutaneous nephrostomy and ureteroneopyelostomy; in this case, there had been an injury to the donor ureter at procurement and the vascular reconstruction was a patch repair of a partially severed branch renal artery. There was one case of a superficial wound dehiscence in the study group; the reconstruction in this case was an excision of an intimal dissection of the donor renal artery and E-E anastomoses. There were 2 urine leaks, one superficial wound dehiscence and one deep wound dehiscence in the CG. Postoperative complications are also outlined in Table 3.

Discussion

Ex vivo bench reconstruction of the vasculature prior to transplantation is a well-recognized and widely used

technique [7,12–16] with potential early and late complications associated such as bleeding from the anastomosis, vascular thrombosis and transplant renal artery stenosis [7,17]. Several techniques exist for *ex vivo* repair or reconstruction of donor renal arteries [6,14,18–20]. A simple repair of an injured part of the vessel may be carried out on a branch artery, main vessel or the aortic patch. A diseased area of the vessel, such as an aneurysm can be excised and repaired. Techniques for reconstructing multiple vessels include end-to side(E-S) anastomosis of a branch vessel to the main artery, anastomosis of a branch vessel to the aortic patch, side-to-side(S-S) anastomosis of two aortic patches, anastomosing two renal arteries together to form a single stem, end-to-end (E-E) anastomosis of the main renal artery to a donor arterial graft, E-E anastomosis of the main renal artery to a synthetic graft and anastomosis of a polar artery to the inferior epigastric artery of the recipient. A series with 25% of multiple arteries concluded that the type of anastomosis (E-E versus E-S) does not affect the long-term outcome [21].

Higher risks of graft loss or vascular complications have been previously associated in the literature with multiple renal graft arteries, along with a short renal vein without donor vena caval extension [22]. The side of procurement determines the length of the vessels; with a longer artery and shorter vein, right kidneys run a higher risk of arterial kinking [23,24]. This can be avoided by using Chopin's method [10] of caval extension of the right renal vein to facilitate the arterial and venous anastomosis and reduce the rate of arterial thrombosis and stenosis without increasing the rate of venous complications. Cava extension of the right renal vein is routinely employed in our centre for these reasons. Although a slightly higher number of right deceased donor (DD) kidneys were transplanted in the SG, the difference was not significant. Recent evidence has also suggested that right DD kidneys may have a worse outcome when compared to left DD kidneys [16,25]. However, this has not been shown when comparing paired kidney transplants where caval extension is employed for right DD kidneys. Phelan *et al.* showed that the side of the DD kidney (left or right) appears to have no impact on early or late allograft outcome [24]. Bordei *et al.* reported an increased incidence of arterial anomalies in the left kidney compared to the right, based on anatomic and radiologic investigations [26], we have not shown this in our study as the majority of kidneys requiring an *ex vivo* reconstruction were that of right kidneys.

In the event of other vascular problems such as moderate atheroma of the donor renal artery; the atheromatous cuff was excised to enable an anastomosis to a nonatheromatous plaque. The majority of *ex vivo* interventions of the allograft renal artery were reconstructions of either multiple renal arteries or repair of a vessel damaged at procurement. *Ex vivo* reconstruction of the donor renal artery is associated with higher rates of arterial complications postoperatively. *Bessede et al.* found *ex vivo* reconstruction of the artery to be a significant risk factor for the development of arterial thrombosis and arterial stenosis postoperatively [16]. We did find an increased incidence of vascular complications in the SG compared with the CG, but these differences were not statistically significant. Two of the three patients in the SG with a urine leak post op had a reconstruction of a lower pole vessel, so this may have been the causative factor; but 2 patients in the CG also had a urine leak and, again, this difference was not significant ($P = 1.0$).

The vascular complications that occurred in the SG may not have been secondary to the *ex vivo* reconstruction as one patient sustained a renal vein thrombosis and the *ex vivo* reconstruction in this case was a repair of a middle pole vessel injured at procurement. The second patient had ischaemic infarction of an upper pole branch when the reconstruction was on the bifurcation of the main renal artery at the hilum of the kidney.

There was a significantly greater number of patients with an *ex vivo* intervention who were investigated with a postoperative CDUS of their transplanted kidney indicating that there was a higher index of clinical suspicion in this group of patients. More patients in the SG also had a transplant biopsy postoperatively, but this was not statistically significant. CDUS has been routinely applied in the assessment of post operative transplant kidneys; both in the immediate and long term [27,28]. The indication for CDUS in the immediate postoperative period is dysfunction of the transplant kidney, it will identify thrombosis of the transplant renal artery or vein if present [29]. It is also useful after any intervention on the transplant kidney; such as a transplant biopsy or placement of a nephrostomy. CDUS will identify complications of these procedures such as the development of an intrarenal arteriovenous fistula or an intrarenal pseudoaneurysm. Transplant renal artery stenosis (TRAS) is the most common vascular complication in renal transplants during the first 3 postoperative years, the risk of developing TRAS is increased in recipients of a reconstructed allograft [4]. CDUS is especially useful for the identification of TRAS during the long-term follow-up of reconstructed allografts [28]. Magnetic resonance angiography (MRA) can then be used to confirm the results of CDUS but has technical limitations for patients with metallic implants or artefact created by surgical clips.

We found an increased incidence of DGF in the SG but the incidence of rejection and failure was higher in the CG; however, these results were not statistically significant. There was no difference in the mean days spent on dialysis in the patients with DGF in the study and control groups. The 1-year graft survival rate was marginally higher in the

CG. Kaplan–Meier survival curves show similar allograft survival patterns with slightly better estimated survival rates in the SG compared to the CG. The postoperative creatinine was higher in the SG at 1 and 3 months postoperatively but fell to a level below that of the CG at 12 months post-transplantation. There was an increased incidence of vascular thrombosis in the SG compared to the CG. The incidence of renal artery stenosis, bleeding requiring surgical re-exploration and urological or surgical complications were equivocal among the groups. A greater number of patients in the CG received a postoperative blood transfusion, but there was no difference in the mean number of units transfused (3 in each group).

Our results are similar to those previously quoted in the literature. Zhang *et al.* compared 32 renal allografts with injured graft blood vessels to 60 renal transplant patients with noninjury during the same term. They found no difference in the 1-year graft survival (96.9% and 98.3% in the study and control group, respectively), postoperative 1 year acute rejection and the incidence of stenosis of vascular anastomosis. Their rates of delayed graft function were also similar to ours with a rate of 21.9% in the study group and 18.3% in the control group [7]. Minana *et al.* reported similar results for rejection episodes and creatinine values, but a significant proportion of cases developed renal artery stenosis (25%, $P = 0.052$) which was diagnosed with the onset of arterial hypertension. These patients were treated with percutaneous angioplasty and did not lead to graft failure in any of the cases. They concluded that allografts with arteries damaged during procurement or as a result of pathological changes such as arteriosclerosis or aneurysm are salvageable after bench reconstruction before transplantation [8]. Neither of these deceased donor studies reported on predictors of failure.

The use of the paired allograft approach allowed us direct comparison of reconstructed and unreconstructed kidneys and we consider this to be a more accurate method than matching recipient demographic factors where donor variables cannot be accounted for. However, our groups were also similar in terms of the recipient demographic factors and immunosuppressive protocols. There was no significant difference between the groups in terms of the gender and age of the recipient, subtype of end-stage renal failure, incidence of diabetes mellitus, antigen mis-match, postoperative immunosuppressive regime, warm and cold ischaemia time and length of follow-up. However, there was a statistically significant difference in the proportion of low risk (PRA <80%) and highly sensitized (PRA>80%) individuals in each group with a greater proportion of patients with a PRA>80% in the SG.

This study has several limitations. As a retrospective, observational case–control single-centre study it has inherent weaknesses; namely selection and recall bias. We were unable to report on donor variables but matching donor characteristics are found with the use of paired transplants in this study. This is a single-centre study but by limiting this study to one centre, we have reduced the confounding effects of multiple surgeons and multiple peri-operative protocols. We were unable to ascertain the incidence of postoperative hypertension owing to the retrospective nature of this study. The presence of postoperative hypertension may be an indication of the presence of renal artery stenosis and has been reported in other studies of this nature, but we were unable to evaluate this fully. We were also unable to portray detailed anatomic follow-up of the recipient vessels as 77% of our reconstructed grafts were functioning at latest follow-up there was no indication to perform transplant angiography to assess the vessel calibre. The endpoints chosen instead to assess graft outcome were DGF, patient and graft survival, serum creatinine and eGFR (ml/min/1.73 m^2). These markers are used in a large proportion of studies assessing renal allograft outcome. We would, however, recommend that a prospective case–control study of the same nature in a large volume centre be undertaken to assess the true incidence of renal artery stenosis and other vascular complications in this group of patients.

Conclusion

These results demonstrate comparable outcome in renal allografts with *ex vivo* reconstruction of the renal artery. We have also shown a statistically significantly increase in the number of patients with a reconstructed allograft investigated with a Doppler US of their transplanted kidney postoperatively. This may indicate a higher index of suspicion of transplant rejection in this cohort of patients. However, this marker of clinical suspicion did not affect overall graft outcome as shown by the 1-year survival rates and overall incidence of failure among the groups. We believe that our results confer increased validity as we have excluded a major source of confounding by matching the groups in terms of the allograft donor, and having further similarities among the recipient demographics. This study conclusively shows that *ex vivo* reconstruction of the donor renal artery in renal transplantation has equivocal graft and patient outcomes when compared to their donor-matched, unreconstructed allograft. We would advocate the judicious use of this technique where appropriate, to salvage suboptimal renal allografts in order to increase the organ pool available for transplantation.

Authorship

LM: involved in the study design, data analysis and drafting of manuscript. ND and GS: are involved in data collection.

CD: also involved in data analysis. RP, PM and DH: supervision was conducted. ME: supervision and drafting of manuscript were carried out. DL: supervision, study design and drafting of manuscript are carried out.

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