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Brief Communication

Transplantation of Kidneys from Deceased Adult Polycystic Donors

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Renal transplantation is the best treatment for end-stage renal disease. The discrepancy between donor organ supply and demand continues to widen. Maximum efforts should be made to make use of donor kidneys and we suggest that polycystic kidneys can be suitable marginal donor organs. Five polycystic cadaveric donor kidneys were transplanted in four recipients at our institution between year 2000 and 2004. The donor kidneys were either of normal size or moderately enlarged (less than 15 × 10 cm). Donor ages were 24, 46 and 55 years. All donors had normal serum creatinine at the time of organ retrieval. Recipients gave informed consent to be transplanted with the polycystic kidneys. Three of four recipients had primary graft function. The patient with primary nonfunction required graft nephrectomy 8 weeks post-transplantation. One patient died due to cardiovascular causes with a functioning graft 18 months after transplantation. Two patients remain well, 26 and 58 months after transplantation, with normal graft function. Our experience and the limited evidence from the literature suggest that, with careful selection of both donor and recipient, transplantation of cadaveric polycystic donor kidneys should be considered given the current organ shortage.

Key words: Deceased, donor, kidney, polycystic, transplant

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Introduction

Renal failure has serious adverse effects on the quality of life of affected individuals. Although dialysis provides an alternative to maintain survival, quality of life on dialysis is generally poor. Allograft renal transplantation is established as the best treatment for end-stage renal disease (ESRD). Transplant surgeons should aim to offer the best-quality organ to the recipient to ensure an excellent outcome. However, the widening gap between demand and supply of organs has necessitated exploring options including use of marginal donor organs. Example of marginal donors include non-heart-beating donors, donors with well-controlled hypertension, diabetes or mild proteinuria, donors older than 60 years and transplantation across the blood groups. We report our experience of adult cadaveric donor polycystic kidneys.

Patients and Methods

Three pairs of deceased adult polycystic kidneys were offered to our unit after surgeons at other centers declined to use these organs. The donor ages were 24, 46 and 55 years. All donors died of subarachnoid hemorrhage although none was hypertensive and all had normal serum creatinine at retrieval. The kidneys retrieved were polycystic but none was greater than 15 cm in size. Biopsy specimens were taken from each kidney of two pairs of donor kidneys; the other pair of kidneys was not sampled. In one pair of donor kidneys, the biopsies were examined by frozen section and in the other, the specimens were rapidly processed towards paraffin sections.

In the pair of kidneys, examined by rapidly processed paraffin sections, the biopsy needle cores showed the presence of cysts, but there was a good background kidney, with only 8% global glomerulosclerosis in the left donor kidney and only 18% global glomerulosclerosis in the right donor kidney. Both of these kidneys showed mild-to-moderate arteriosclerosis and arteriolosclerosis. The left kidney was implanted but the other was discarded because of prolonged cold ischemia time (>24 h).

In the pair of kidneys examined by frozen section, there was 30% global glomerulosclerosis in both kidneys and moderate chronic tubulo-interstitial fibrosis in the background but no cysts. It was not possible to clarify these chronic tubulo-interstitial changes further in these specimens, in the permanent/paraffin sections, due to marked fragmentation of the tissue following cryostat sectioning. Of these pre-implantation biopsy specimens, only one contained more than 25 glomeruli. After much deliberation this pair of organs was transplanted into one recipient to maximize the transplanted renal mass.

Four patients with established end stage renal failure were selected. Each patient was fully informed regarding the marginal donor status prior to giving consent for transplantation. One patient received a dual renal transplant. The donor and recipient details including cold ischemia times are shown in Table 1 and serum creatinine since transplantation is shown in Table 3.

Patients received an immune suppression regime consisting of cyclosporin and prednisolone together with either azathioprine or mycophenolate mofetil. In addition, two patients received the anti-CD25 monoclonal antibody basiliximab as part of their induction regime.

Table 1: Details of deceased donors and transplant recipients showing age, cause of end stage renal disease (ESRD), HLA mismatch, cold ischemia time (CIT) and immune suppression regime

Donor age (years)	Recipient age (years)	Cause of recipient's ESRD	Mismatch	CIT (h)	Immuno-suppression regime
46	63	Medullary cystic disease	1–1–0	21	PAC
46	72	Interstitial nephritis	2–2–0	19	PAC
24	65	Renal artery stenosis	1–1–0	18	BI/PMC
55	69	IgA nephropathy	1–2–0	21	BI/PMC

BI: Basiliximab induction; P: prednisolone; A: azathioprine; M: mycophenolate mofetil; C: cyclosporin

Table 2: Renal allograft function over time. Three out of four recipients had primary graft function that was sustained

Patient	Day 0	Day 1	2 months	8 months	18 months	26 months	58 months	Outcome
1	400	266	96	125	125	128	139	Alive and well
2	865	263	248	209	197			Died cardiac cause
3	722	400	163	178	113	110		Alive and well
4	765	752	866					Transplant nephrectomies

Table 3: Details of deceased polycystic donor kidneys and transplant recipients from the literature (ref = reference list; N/A = information not available)

Donor age (years)	Recipient age (years)	Cause of recipient's ESRD	Outcome	Ref
25	33	Familial mediterranean fever	8 year follow-up with normal creatinine	5
23	39	Focal sclerosing glomerulonephritis	Graft nephrectomy at 12.5 year for increased cyst size, pain and decreased RF	8
20	52	PCKD	3 year follow-up with normal creatinine	4
36	36	Diabetes	2.5 year follow-up with normal creatinine	3
19	48	Diabetes	1.5 year follow-up with normal creatinine	3
19	44	PCKD	10 year follow-up with normal creatinine	7
21	19	N/A	1 year follow-up with normal creatinine	6
21	20	N/A	1 year follow-up with normal creatinine	6

Results

Three of the four recipients had primary graft function. Two patients remain alive and well with functioning allografts, with follow up to 58 and 26 months (Table 2). Serial ultrasound scans in these two patients have shown no change in the cyst size or character. One recipient, with a functioning renal graft, died from cardiovascular disease 18 months after transplantation. The patient with dual renal transplantation had primary nonfunction. He developed pyelonephritis of the transplanted kidneys requiring graft nephrectomy 8 weeks post-transplantation.

Discussion

Autosomal dominant polycystic kidney disease (ADPKD) is an autosomal dominant inherited disorder characterized by presence of multiple renal cysts and slow progressive renal deterioration (1). Ten to fifteen percent of patients with ESRD have ADPKD as the etiology. However, there is approximately a 10-year lag period between the onset of symptoms and progression to end-stage renal failure. Churchill et al. predicted that the chance of developing ESRD in ADPKD patients is 23% by 50 years and 48% by 73 years of age (2). As 85% of people with ADPKD are asymptomatic until the fourth decade of life, some may die of unrelated causes, for example trauma, and their organs might be offered for organ donation.

To the best of our knowledge, eight cases using polycystic kidneys as donor organs for transplantation (Table 3) have been reported (3–8).

The obvious concern about transplanting a donor polycystic kidney is the possible compromised function at the outset and risk of rapid deterioration of function and ultimate failure. Other potential risks associated with donor polycystic kidneys include bleeding, infection and stone formation. In our series the patient receiving dual renal transplant developed pyelonephritis. This occurred in the setting of primary nonfunction warranting multiple percutaneous biopsies. None of the functioning grafts experienced pyelonephritis, bleeding into cysts or stone formation.

In ADPKD it is estimated that only 1% of nephrons become cystic per year. In a cohort of uni-nephrectomized ADPKD patients no significant acceleration in decline of renal function was seen (9). Hence there should be sufficient renal reserve to allow the transplanted polycystic

kidney to function. The natural history of the cysts in the transplanted kidneys is unclear. Whilst a decrease in size or even disappearance of renal cysts has occurred, others observed an increase in cyst size (3–8). Renal cell carcinoma occurring in ADPKD kidneys can be bilateral, multicentric and sarcomatoid but the incidence of renal cell carcinoma is no higher in ADPKD than in the general population (10) and should not be a concern for the recipient.

The number of cases in our report is small but in terms of the total number of polycystic donor cases equates to a further 50%. Follow-up data are limited to 12 years in the literature and, in our series, the two patients who remain alive with functioning grafts have good medium-term graft function (at 2 and 5 years).

Although the average age from the literature of cadaveric donor was 23 years, in our series the mean donor age was 45 years. It is of interest that the graft with primary nonfunction was from the oldest donor aged 55 years. In both the literature and our experience, successful functioning polycystic grafts have come from donors under 50 years. It may be advisable to limit the use of polycystic donor kidneys to donors aged less than 50 years, at which age less than 25% of patients with ADPKD demonstrate ESRD (2).

The published literature and our experience are now encouraging in the use of polycystic kidneys as marginal donors. Therefore, if polycystic kidneys are offered from suitable donors, they should not be turned down without full assessment by experienced members of the transplant team.

Such assessment should include confirmation that both of the donor kidneys were polycystic in appearance, as a unilateral cystic kidney would not be compatible with the diagnosis of ADPKD and would probably represent a nonfunctioning kidney.

Pre-implantation biopsies should be performed, as may be the case in other marginal donors. It has been shown that, while interstitial fibrosis/tubular atrophy was not a good predictor of graft outcome, >20% glomerulosclerosis was associated with poor graft outcome (11,12). However, samples containing fewer than 25 glomeruli might be unreliable in determining outcome based on glomerulosclerosis. The presence of significant vascular disease, in particular arteriolar hyalinosis, on pre-implantation biopsies may be a better pathological predictor of transplant failure (12,13). A scoring system for pre-implantation biopsies has been suggested that examined donor renal pathology in four areas: glomerulosclerosis, tubular atrophy, interstitial fibrosis and vascular disease. In a retrospective study, both a poor vascular disease score on pre-implantation biopsy and a low donor calculated creatinine clearance (<100 mL/min) were associated with delayed graft function (13). Prospective evaluation of these variables in assessment of future marginal donors is likely to be beneficial.

We propose the following criteria and precautionary measures before proceeding to transplantation of polycystic kidneys:

- Donors less than 50 years.
- Donor polycystic kidney size less than 15 cm in bipolar length.
- Donor should have normal creatinine at the time of retrieval.
- Pre-transplant renal biopsy should be performed. Discussion of these results should involve the pathologist, nephrologist and surgeon prior to considering organs for transplantation.
- Minimal cold ischemia time preferably less than 12 h and not greater than 24 h.
- Appropriately selected recipients, who may have a life expectancy of 10 years or less and who are fully informed regarding consent to receiving a polycystic graft.

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