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The relative importance of donor age in deceased and living donor kidney transplantation

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Summary

In deceased donor kidney transplantation donor age is known to influence graft survival. The influence of living donor age on graft survival is questioned. We compared the influence of living and deceased donor age on the outcome of renal transplantation. All 1821 transplants performed in our center between 1990 and 2009 were included in the analysis. Observation was until April 2012. A total of 941 patients received a deceased donor kidney and 880 a living donor kidney. In multivariate Cox analysis, recipient age, maximum and current panel reactive antibodies, transplant year, HLA-mismatches, donor age, donor gender, donor type, delayed graft function, and calcineurin inhibitor (CNI) and prednisone as initial immunosuppression were found to have a significant influence on death-censored graft failure. The influence of both living and deceased donor age followed a J-shaped curve, above 30 years the risk increased with increasing age. Donor type and donor age had an independent influence. The graft failure risk of deceased donor transplantation is almost twice that of living donor transplantation so that a 60-year-old living donor kidney has the same graft failure risk as a 20-year-old deceased donor kidney.

Introduction

To keep pace with the waiting list, more kidney donations are accepted from living extended criteria donors (ECD). Although donor hypertension and obesity play a role, the most prominent characteristic of both living and deceased ECDs is that they are older than standard criteria donors (SCD) [1–5].

In deceased donor kidney transplantation donor age is known to influence graft survival, in living donor kidney transplantation this influence is less clear. The composition of living and deceased donor recipient populations is different in many respects; this probably explains part of the difference in graft survival in these populations.

Until now the influence of donor age has been studied in deceased or living donor kidney transplantation populations separately, ruling out comparison because of heterogeneity of the populations [2,6–13]. Besides, in many studies age was categorized resulting in small elderly populations [3,9–11,14]. In most living donor populations donor age range is narrow because of donor selection, so the influence of age cannot be studied properly.

Deceased donor kidney transplantations have been performed in our center since 1971 and living donor kidney transplantations since 1981. Only in the very beginning were high recipient and deceased donor age exclusion criteria. The wide distributions in recipient and donor age in our population allowed us to study the influence of age as a continuous variable on the risk of graft failure, both in deceased and living donor kidney transplantation. How important is living donor age and how does it compare to deceased donor age?

Methods

All 1821 transplants performed in our center between January 1990 and December 2009 were included in the analysis. Observation was until April 2012 or until graft

failure, death, or lost to follow-up. 27 patients were lost to follow-up with a median time after transplantation of 31 months (0–160). Standard immunosuppression was cyclosporine, prednisone in 1990, but was changed to prednisone, cyclosporine, and mycofenolate mofetil (MMF) in 1996, whereas tacrolimus was introduced in 1998 as substitute for cyclosporine. In patients that started on triple therapy, prednisone was tapered and discontinued at 4 months after transplantation.

Screening of our potential living kidney donors has been described thoroughly [15]. Absolute contra-indications for donation are body mass index >35 kg/m^2, GFR <80 ml/min, hypertension with end-organ damage, history of invasive malignancies, diabetes mellitus, pregnancy, intravenous drug abuse, major cardio respiratory disease, human immunodeficiency virus positivity, hepatitis B or C infection, psychiatric disorders, and systemic disease. Living donor age itself has never been a contraindication for donation.

In our center deceased donor kidneys are accepted from heart beating donors and donation after cardiac death (DCD) donors. We primarily accept donors after controlled cardiac death (Maastricht category III). Uncontrolled Maastricht category II donors are accepted under strict conditions only.

We studied graft failure censored for death, uncensored graft failure, and patient death. ANOVA and Chi-square tests were performed to test the difference between living and deceased donor populations and between donor age categories. Kaplan–Meier analysis was performed, including donor age and type (living vs. deceased). For Kaplan–Meier analysis, donor age was subdivided into the categories 0–39, 40–59, and 60 years and older. Univariate and multivariate Cox proportional hazard analyses were performed, including all variables mentioned in Table 1 and donor type, which was included as a categorical variable (heart beating, DCD, living). Backward elimination was chosen as the method of variable selection. Transplantation year was included to correct for time related changes in diagnostics, treatment options, and experience. Donor and recipient age were included as continuous variables. Initial immunosuppression was included as six binary variables consisting of any combination of immunosuppressants with or without: (i) CNI (tacrolimus, cyclosporine), (ii) induction therapy (rATG, IL2-blocker, OKT3), (iii) mTOR inhibitor (rapamycin, everolimus), (iv) MMF, (v) prednisone, and (vi) other (azathioprine, trial medication). The proportional hazards assumption was tested for donor type with a log-minus-log plot. The analyses were performed using Statistical Package for the Social

Table 1. Characteristics for deceased donor (DD) and living donor (LD) kidney recipients.

	All *N* = 1821	DD *N* = 941	LD *N* = 880	*P*-value
Recipient age (mean ± SD)	47.8 ± 14.2	49.4 ± 13.5	46.1 ± 14.8	<0.001*
Male recipients (%)	62	61	63	ns†
Maximum PRA (median; % >5%)	5; 44	9; 58	4; 28	<0.001*
Current PRA (median; % >5%)	0; 17	0; 24	0; 10	<0.001*
Transplant year (median)	2002	1999	2005	<0.001*
Previous transplants (%)				
0	81	76	86	<0.001†
1	15	18	11	
2+	5	6	3	
Pre-treatment (%)				
Hemodialysis	55	70	39	<0.001†
Peritoneal dialysis	29	27	31	
Pre/Trans	16	3	30	
HLA-mismatches (mean ± SD)	2.8 ± 1.6	2.6 ± 1.5	3.0 ± 1.7	<0.001*
DR mismatches (mean ± SD)	0.8 ± 0.7	0.7 ± 0.7	1.0 ± 0.7	<0.001*
Donor age (mean ± SD)	47.6 ± 14.7	45.7 ± 16.1	49.6 ± 12.7	<0.001*
Male donors (%)	50	55	44	<0.001†
Delayed graft function (%)	24	42	5	<0.001†
CNI as initial immunosuppression (%)	95	94	95	ns†
Induction therapy (%)	13	14	11	ns†
mTOR inhibitor (%)	6	3	10	<0.001†
MMF (%)	66	57	75	<0.001†
Prednisone (%)	94	93	96	0.005†
Other immunosuppression (%)	8	5	10	<0.001†

*ANOVA and †Chi-square to test significance between DD and LD. CNI, calcineurin inhibitor; MMF, mycofenolate mofetil; PRA, panel reactive antibodies.

Sciences (SPSS) PASW 17.0.2 for Windows (IBM Corporation, Armonk, NY, USA). *P*-values ≤ 0.05 were considered significant.

Results

A total of 941 patients received a deceased donor kidney and 880 a living donor kidney. There were 94 donors after cardiac death (Maastricht category III $n = 91$, Maastricht category II $n = 3$). In Table 1 transplantation characteristics are shown. There were missing values in 15 cases (0.8%). Recipients of a living donor kidney were significantly younger than recipients of a deceased donor kidney, whereas living donors were significantly older than deceased donors. The distribution of donor and recipient age was also different between the living and deceased donor populations (Fig. 1a and b). Very young donors were present in the deceased donor population but absent in the living donor population. Age in the living donor population was shifted to the right (older donors) in comparison to the deceased donor population. In addition to recipient and donor age there were significant differences between the living and deceased donor populations (Table 1).

There were 507 graft failures; 341 in recipients of deceased donor kidneys and 166 in recipients of living donor kidneys. In Table 2 numbers and causes of graft failure are shown for age categories and donor types. There was no significant difference between the age groups regarding numbers of graft failures (Table 2a). However, in the eldest donor age group never functioning grafts occurred significantly more often and least in the youngest donor age group. When comparing recipients of kidneys from heart beating, DCD, and living donors, there was a significant difference in the number of graft failures (Table 2b). Living donor kidneys failed less often than heart beating donor kidneys. The incidences of chronic rejection and recurrence of original disease was also different between the populations.

In *Kaplan–Meier analysis*, graft survival censored for death was significantly different in the three donor age categories in the deceased donor population ($P < 0.001$), but not in the living donor population ($P = 0.08$) (Fig. 2). Graft survival censored for death after living donor transplantation was better than after deceased donor transplantation for all donor age categories, $P = 0.008$ for 0–39 years, $P < 0.001$ for 40–59 years, and $P < 0.001$ for 60 years and older, respectively.

The influence of all variables shown in Table 1 and the influence of donor type on graft failure risk were studied in the *Cox proportional hazards analysis*. In univariate Cox analysis, recipient age, maximum panel reactive antibodies (PRA), current PRA, transplant year, previous transplants, pre-treatment, total number of HLA-mismatches, donor age, donor type, delayed graft function, and CNI treatment, induction therapy, MMF treatment, and prednisone as initial immunosuppression had a significant influence on the risk of graft failure, censored for death. The influence of donor age was not linear, but exponential (data not shown). The other variables described in Table 1 did not significantly influence this

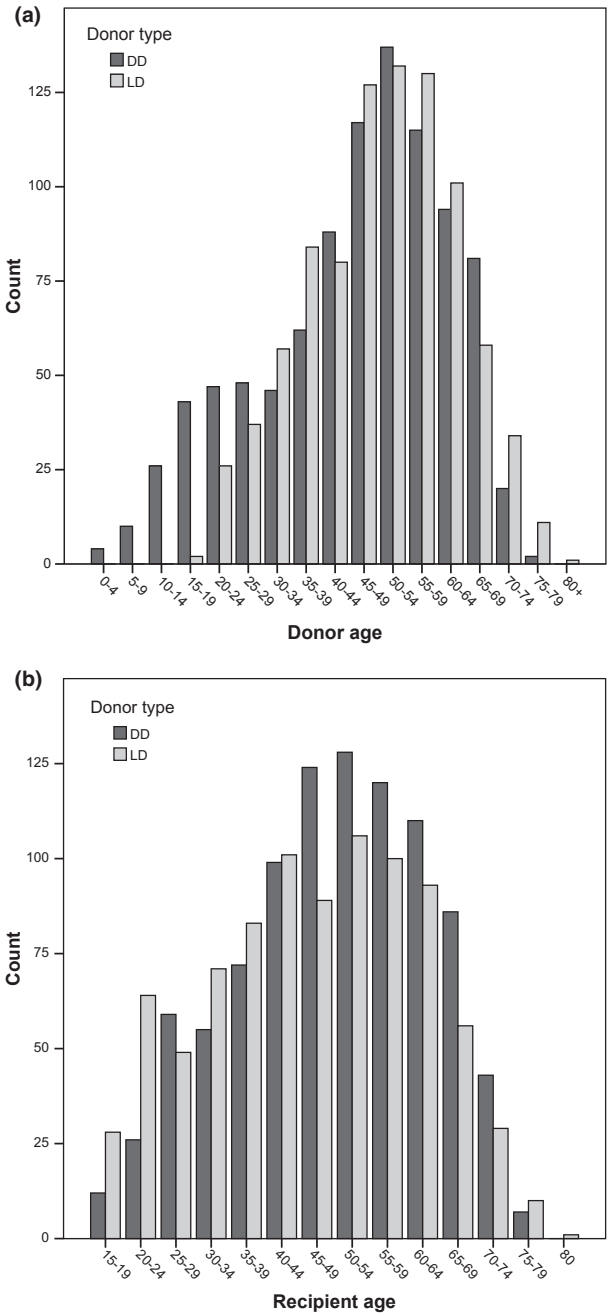

Figure 1 (a) Donor and (b) recipient age distributions in deceased (DD) versus living donor (LD) kidney transplantation.

Table 2. Numbers and causes of graft failure per (a) donor age category and (b) donor type.

	Donor age (years)			
(a)	≤39	40–59	≥60	*P**
N	492	926	402	
Numbers of failures	134	242	130	0.064
Failure causes (*n*)				
Chronic rejection	68	121	58	0.534
Acute rejection	18	27	14	0.753
Technical problems	17	33	10	0.226
Recurrence original disease	11	15	7	0.624
Never functioning graft	3	23	27	<0.001
Other	17	23	14	0.632

*Chi-square to test significance among all three groups.

	Donor type			
(b)	HB	DCD	Living	*P**
N	847	94	880	
Numbers of failures	317	24	166	<0.001
Failure causes (*n*)				
Chronic rejection	149	5	93	0.003
Acute rejection	41	1	17	0.344
Technical problems	46	3	11	0.039
Recurrence original disease	11	2	20	0.001
Never functioning graft	40	11	3	<0.001
Other	30	2	22	0.409

*Chi-square to test significance among all three groups.
DCD, donation after cardiac death; HB, heart beating.

risk. In the final multivariate Cox model, a number of factors were found to have a significant influence on the relative risk [RR or Exp(B)] of graft failure, censored for death (Table 3a). All variables not present in Table 3a had been excluded via backward elimination in previous runs. The influence of DCD was not significantly different from heart beating donation, whereas the risk of living donation was significantly lower than that of heart beating donation. Donor age had a quadratic influence on the risk of graft failure (Fig. 3a). Between the ages of 20 and 40 years graft failure risk hardly changed (relative risk, respectively, 0.60 and 0.63 in comparison to 20-year-old deceased donor). However, between living donor ages of 40 and 60 years the relative risk of graft failure increased from 0.63 to 1.01 in comparison to 20-year-old deceased donor. The interaction terms between donor type and either HLA-mismatches, current PRA, maximum PRA, recipient age, and donor age were not significant. There was neither interaction between donor and recipient age nor between donor age and transplant year.

Table 3b shows the results of the multivariate Cox analysis with death and/or graft failure as the event studied (univariate results not shown). As the square of donor age was also significant, the influence of donor age followed a J-shaped curve (Fig. 3b).

Table 3c shows the results of the multivariate Cox analysis with patient death as the event studied (univariate results not shown). The proportional hazards assumption was not violated.

Discussion

The present study shows that in Kaplan–Meier analysis living donor age appears not to have a significant influence on graft survival censored for death. However, this lack of influence of donor age in Kaplan–Meier analysis could be caused by the fact that this analysis does not take the influence of other variables into account. Moreover, the continuous variable age had to be distributed into arbitrary categories to be suitable for Kaplan–Meier analysis. As shown in Fig. 1 age distribution in the deceased and living donor populations is not comparable which means that the results of these separate Kaplan–Meier analyses cannot be compared.

In Cox analysis donor age turns out to have a significant influence on the risk of graft failure censored for

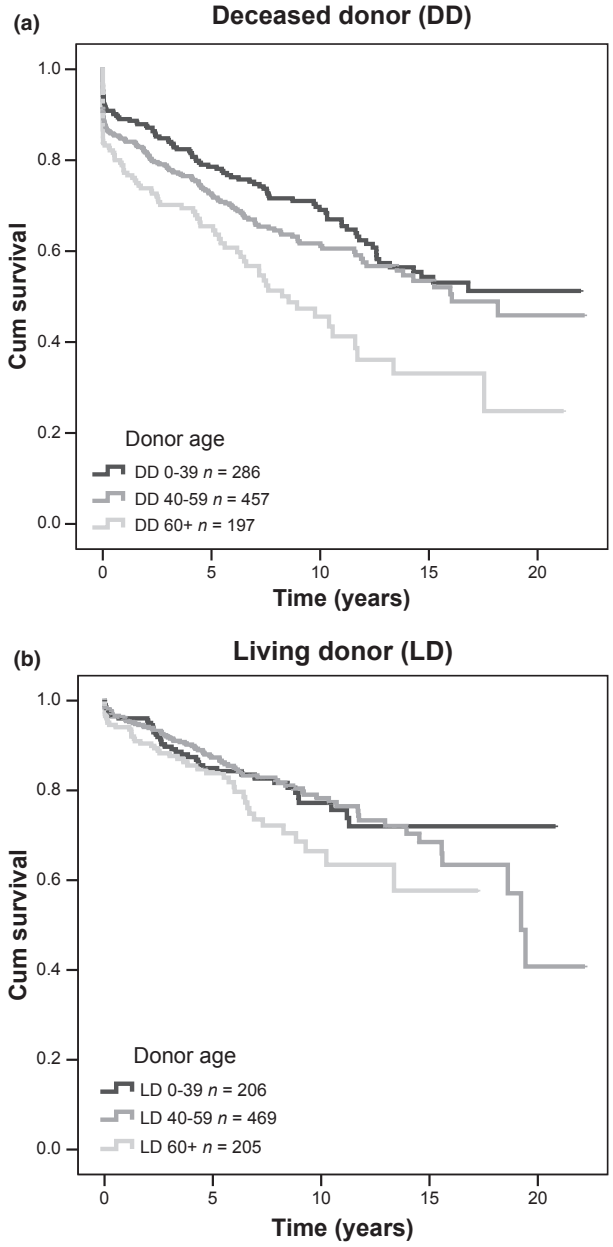

Figure 2 Kaplan–Meier curve comparing death-censored graft survival after (a) deceased donor transplantation ($P < 0.001$) and (b) living donor transplantation (ns) for three donor age categories.

death and the risk of uncensored graft failure independent of donor type. This means that donor age influences graft survival in both living and deceased donor transplantation. The risk of graft failure in recipients of a kidney transplantation increases with increasing donor age according to a quadratic equation. However, the risk in deceased donor transplantation is almost twice that of living donor transplantation so that the graft failure risk for a recipient of a 60-year-old living donor kidney is the same as that of a recipient of a 20-year-old deceased donor kidney. As there is no interaction between donor and recipient age regarding graft failure risk it is not necessary to take age difference between donor and recipient into consideration.

In the literature the influence of increasing donor age on the risk of graft failure has been studied in different ways. In some studies the influence of age was studied in Kaplan–Meier analysis where age had to be categorized [16,17]. In other studies age was studied in a Cox analysis, either as a categorical [3,6,9–11,13,14,18] or as a continuous covariate [7,8,11]. Subdivision in categories is arbitrary and not all studies use the same definitions for 'elderly' and 'old'. On top of that, most studies included donor age as a dichotomous variable: old versus young [3,9–11,13,16–18]. As aging is a continuous process, its effect most probably follows a continuous line. Categorization probably does not reflect the natural aging process and thus the influence on graft failure risk.

In *deceased donor transplantation*, donor age is known to have a negative effect on overall graft survival [6] and death-censored graft survival [7]. In 1999, we described the influence of deceased donor age as a continuous variable on overall and death-censored graft survival in multivariate Cox analysis as a J-shaped curve [8]. The risk of graft failure was highest for recipients of older and extremely young donor kidneys. The risk was lowest for the age categories between 20 and 40 years.

There are few studies that describe the influence of donor age on graft survival in populations that received either *living or deceased donor kidney transplantation*. In all these studies, donor age was included as a categorical variable. In Cox proportional hazards analysis with age as a categorical variable, Matas and colleagues [9] found an unfavorable effect of donor age (≥50) on overall and death-censored graft survival in the population with deceased donor transplantation, but no effect in living donor transplantation population. Kerr and colleagues [10] reported the same results with donors aged 55 years or older. However, both groups performed separate analyses for deceased and living donor transplantation populations. In both studies, the cut-off age for elderly donors was relatively low as was the number of elderly donors included. As we showed, probably as a result of selection, living and deceased donor recipient populations are not comparable (Table 1). This means that the results of separate analyses in two different populations cannot be compared. Although the results of both analyses are different it does not mean that the results of both programs are different.

In UNOS database Gill studied the influence of donor age on graft survival of recipients of living or deceased donor kidneys [14]. Age was defined as a categorical

Table 3. Results of the multivariate Cox proportional hazards analysis. Failure event is (a) censored for death, (b) uncensored, and (c) censored for graft failure.

(a)	*N* = 1821, 502 events		
Variable (reference category)	Exp(B)	95% CI	*P*
Recipient age (per year)	0.984	0.977–0.990	<0.001
Maximum PRA (per %)	0.995	0.990–1.000	0.045
Current PRA (per %)	1.015	1.008–1.021	<0.001
Transplant year (per year)	0.974	0.954–0.993	0.008
HLA-mismatches (per HLA-mismatch)	1.107	1.040–1.178	0.001
Donor age (per year)	0.970	0.943–0.998	0.033
Donor age^2 (per year^2)	1.001	1.000–1.001	0.001
Donor gender (female)	0.835	0.699–0.998	0.047
Donor type (heart beating)			<0.001
DCD	1.056	0.669–1.667	0.816
Living	0.603	0.478–0.760	<0.001
Delayed graft function (no)	2.006	1.629–2.471	<0.001
CNI as initial immunosuppression (no)	0.236	0.174–0.321	<0.001
Prednisone as initial immunosuppression (no)	0.710	0.514–0.980	0.037
(b)	*N* = 1821, 832 events		
Variable (reference category)	Exp(B)	95% CI	*P*
Recipient age (per year)	1.013	1.007–1.019	<0.001
Maximum PRA (per %)	0.996	0.992–1.000	0.028
Current PRA (per %)	1.011	1.006–1.016	<0.001
Transplant year (per year)	0.978	0.958–0.999	0.037
HLA-mismatches (per HLA-mismatch)	1.057	1.009–1.108	0.021
Donor age (per year)	0.977	0.957–0.998	0.033
Donor age^2 (per year^2)	1.000	1.000–1.001	0.003
Donor type (heart beating)			<0.001
DCD	1.200	0.844–1.706	0.309
Living	0.651	0.543–0.781	<0.001
Delayed graft function (no)	1.770	1.499–2.091	<0.001
CNI as initial immunosuppression (no)	0.282	0.214–0.371	<0.001
MMF as initial immunosuppression (no)	0.812	0.662–0.997	0.047
Prednisone as initial immunosuppression (no)	0.630	0.486–0.817	<0.001
(c)	*N* = 1821, 330 events		
Variable (reference category)	Exp(B)	95% CI	*P*
Recipient age (per year)	1.071	1.060–1.082	<0.001
Transplant year (per year)	0.941	0.918–0.965	<0.001
Donor type (heart beating)			0.012
DCD	1.777	1.027–3.075	0.040
Living	0.783	0.591–1.036	0.087
Delayed graft function (no)	1.341	1.022–1.759	0.034
CNI as initial immunosuppression (no)	0.366	0.208–0.645	<0.001
Prednisone as initial immunosuppression (no)	0.609	0.406–0.915	0.017

CNI, calcineurin inhibitor; DCD, donation after cardiac death; MMF, mycofenolate mofetil; PRA, panel reactive antibodies.

variable with four elderly groups above 55 years of age (9.7% of the population) compared with one young population below 55 years (90.3%). An increasing risk of graft failure was found with increasing age independent of donor type. Although the influence of younger donor age categories was not separately analyzed in this study, results for the elderly population showed the same trend we found in our study. In another study, Gill performed a multivariate analysis restricted to elderly recipients aged 60 years or older. They found superior graft survival results with older (>55) living donor kidneys compared to extended criteria deceased donor kidneys, but results

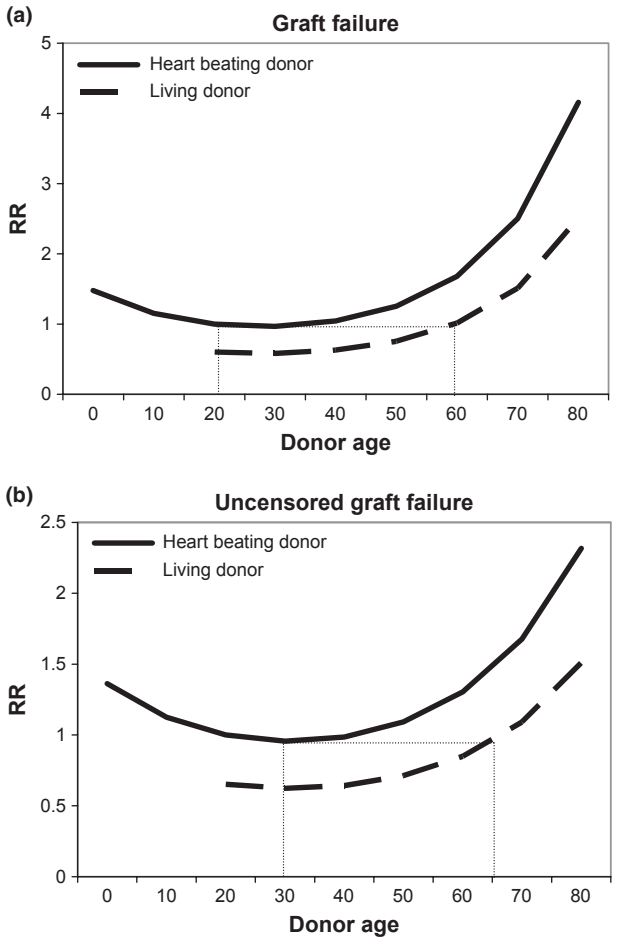

Figure 3 Calculated relative risk (RR) of (a) graft failure censored for death and (b) uncensored graft failure with increasing donor age for heart beating and living donor transplantation. The reference value is a 20 year old heart beating donor. The dotted lines demonstrate the comparison of the risk between recipients of a living donor kidney and a heart beating donor kidney.

were inferior to results from young living donor kidneys [3]. Young found no difference for (death-censored) graft loss between older living donor transplantation and deceased SCD in adult recipient transplantation [11].

In *living donor transplantation*, donor age analyzed in multivariate Cox proportional hazards analysis as a continuous variable did not show a significant influence on graft loss [11]. However, in this study only 73 (5.8%) elderly donors aged 60 years or older were included. Dols [12] studied donor age as a dichotomous (<60 years vs. ≥60 years) variable. In multivariate analysis they found no difference in death-censored graft survival between recipient populations transplanted with an older living donor kidney and a young living donor kidney. In a population of living donor kidney recipients Toma and colleagues [13] studied the influence of living donor age as a time-dependent variable. They found that living donor age (high ≥60 years vs. low <60 years) was the most important risk factor for long-term overall graft failure. A meta-analysis on the impact of transplantation of kidneys from extended criteria living donors on transplantation outcome revealed that recipients of kidneys from younger living donors had better outcomes than kidney recipients from older living donors [2]. Elderly donor age was defined as above 60 years of age.

The meta-analysis also showed that the negative influence of increasing donor age appeared to diminish in time [2]. This is in line with our findings during the period 1983–1997 where transplant results improved over time [8]. The current study confirms this effect of transplant year on the graft failure risk. A probable explanation is growing experience, improved medical care for concomitant disease, and improvements in diagnostics.

Our study also shows that initial use of CNI and of prednisone is associated with a decreased graft failure and patient death risk, whereas other immunosuppressants have no significant influence.

In the present study we showed that in our population, a kidney from any living donor below age 60 has better graft survival than a 20-year-old deceased donor kidney. Between the ages of 20 and 40 years living donor graft failure risk hardly changes whereas over the age of 40 the relative risk of graft failure increases. This means that awaiting a deceased donor kidney is not an option when a living donor is available. Older living donor kidney transplantation certainly is better than remaining on the waiting list [19].

In conclusion, elderly living donors should not be rejected on the basis of their age only. Although there is an advantage for patients receiving a young living donor kidney (below age 40), even transplantation with an older living donor kidney provides comparable or better graft survival outcomes than with a deceased donor kidney.

Authorship

ML and JIR: participated in research design and performance, data analysis and statistical analysis and in the writing of the manuscript. WW: participated in research design and performance and in the writing of the manuscript. JAKG: collected data for analysis and participated in the writing of the manuscript. JW and JNMI: participated in the writing of the manuscript.

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