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Effect of donor age on the outcome of living-related kidney transplantation

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Summary

The study compared the results of kidney transplantation from living-related donors older and younger than 60 years. The 273 kidney graft recipients were divided into group 1 (115 recipients of older grafts) and group 2 (158 recipients of younger grafts). The frequency of acute rejection (AR) episodes was similar in both groups but slow graft function occurred more frequently in group 1. The frequency of chronic renal allograft dysfunction in the first post-transplant year was significantly higher in group 1 than in group 2. Patient and graft survival was significantly worse in group 1. Risk factors for graft loss were the difference between donor and recipient age and AR. Donor age and graft function were risk factors for patient death. Although kidneys from older donors provide a statistically poorer transplant outcome, they are clinically acceptable, especially when waiting time is prolonged and access to dialysis limited.

Introduction

In the last two decades, there has been remarkable discrepancy between the growing number of dialysis patients and the rate at which kidney transplantations are being performed. The gap between the supply of available kidneys and the demand for them has been progressively increasing [1,2]. As a consequence, donor selection criteria have expanded to include nonheart beating donors and donors of advanced age [1]. Thus, donor nonimmunological factors have become important determinants of renal allograft survival [3,4]. In addition, the lack of cadaveric organs for transplantation has resulted in an increased number of kidney transplants from living donors [5].

Our previous analysis showed that more frequent acute complications and more progressive chronic kidney-graft failure occurred during the first post-transplant year in the recipients of grafts from older donors [6]. That was why we suggested that kidney grafts from donors older than 60 should be used for living-related kidney transplantation with precautions, especially from those older than 70 years. Nevertheless, insufficient cadaveric renal transplantation in our country has led to a continually increasing number of living-related kidney transplantations reaching 70% of all kidney transplantations in our institution. As the vast majority of our patients were adults, almost 40% of their donors were older than 60, and among them 30% were more than 70 years old. The constant lack of cadaveric kidneys and the insistence by patients and their families for older donors to be accepted has contributed to the increase in the number of transplants from older living-related donors, in spite of the results and suggestions of our previous analysis.

Recent studies have reported significantly poorer graft function and survival associated with the use of older cadaver donors, but suggested that a poorer outcome cannot be *a priori* attributed to older living donors [7]. Moreover, fewer studies have evaluated the effect of living donor age on patients and graft outcome, especially in long-term studies, and the results are inconsistent [8,9]. The present study was undertaken 10 years after our first

analysis on the use of older donors in living-related kidney transplantation, with the aim of contributing to the solution of the question about the effect of donor age on graft outcome after living kidney transplantation.

Patients and methods

This retrospective study included medical records of 273 renal transplant patients who received their first graft from living-related donors at our institute between January 1987 and December 1999. They were regularly followed-up in our out-patient department until their death, return to dialysis or until December 2003. The end of the period analyzed was chosen to provide a minimum of 48 months for patient follow-up. According to donor age, the recipients were divided into group 1 consisting of 115 patients receiving a graft from donors older than 60 years (donor age 60–85 years), and group 2 composed of the remaining 158 patients, who received a graft from donors <60 years old (donor age 34–59 years). Data on the donors and recipients are summarized in Table 1.

The kidney donors and recipients were subjected to an extensive immunological, medical, physical and radiological examination in accordance with the European Best Practice Guidelines for Kidney Transplantation [10]. Serum creatinine level (Jaffe's method), 24 h creatinine clearance (CCr) and ^{99m}TcDTPA glomerular filtration rate (GFR) were used to assess global kidney function before donation. Only donors with normal age-adjusted GFR were accepted for further evaluation. Clearance of ^{99m}TcDTPA was used for assessment of both total GFR and the relative contribution of each kidney to overall GFR (single kidney GFR- SKGFR). This was considered as the baseline function of the kidney graft [11].

Graft function was assessed by serum creatinine levels (Jaffe's method). Acute rejection (AR) was defined as an increase in serum creatinine by 25% or more, characteristic clinical and ultrasound features in the presence of low

Table 1. Data on donors and recipients.

	Group 1	Group 2	*P*
Number of patients	115	158	
Donors			
Age (years)	66.31 ± 4.68	49.78 ± 6.38	0.0001†
Sex (f/m)	58/57	111/47	0.0009*
SKGFR, ml/min	49.9 ± 15.2	53.1 ± 12.4	0.05
Recipients			
Age (years)	38.32 ± 7.51	28.27 ± 7.50	0.0001†
Sex (f/m)	27/88	54/104	0.05*
Underlying kidney disease			
GN	68	106	NS
PN	15	35	NS
ADPKD	8	0	0.0008*
DN	7	1	0.008*
Others	10	16	NS
HD (months)	32.20 ± 25.7	25.85 ± 24.5	0.001‡
Difference			
Age(years)	28.05 ± 7.43	21.75 ± 7.07	0.0001†
Sex	50	85	NS*
F donor—M recipient	72	40	NS
ABO mismatches	11	12	NS*
HLA mismatches			
0	7	25	0.01*
1/2	32/68	46/85	NS*
3	8	2	0.01*
PRA (No. of patients)			
<50%	110	153	NS*
>50%	5	5	NS

*χ^2 test.

†*t*-test for independent samples.

‡Mann–Whitney *U*-test.

f, female; m, male; SKGFR, single kidney glomerular filtration rate; GN, glomerulonephritis; PN, pyelonephritis; ADPKD, adult dominant polycystic kidney disease; DN, diabetic nephropathy; HD, hemodialysis; PRA, panel reactive antibodies.

or normal cyclosporine levels, and good response to treatment with pulse methyl-prednisolone (1 g) for three consecutive days. Only in 10 cases, AR was histologically confirmed. Slow graft function (SGF) was defined as a lack of serum creatinine decrease below 300 μmol until the seventh postoperative day in the absence of AR and urinary tract or renal graft vessel obstruction. This included both patients that needed hemodialysis in the first 7 days post-transplant and patients not needing dialysis. A progressive and irreversible decline in graft function, irrespective of the cause, was designated as chronic renal allograft dysfunction (CRAD) [12]. Chronic graft nephropathy (CAN) denotes a chronic graft dysfunction associated with the following histological changes: patchy fibrosis of the interstitium with or without inflammation, tubular atrophy, glomerular sclerosis and vascular endarteritis.

Immunosuppression

Triple drug immunosuppression consisting of azathioprine, cyclosporine A and prednisolone was applied in the majority of patients both in group 1 and 2 (109 vs. 148). The remaining recipients were treated with cyclosporine (5 vs. 8) or azathioprine (1 vs. 2 patients) both in combination with steroid. Our immunosuppressive protocol was changed during the study as previously described in detail [13]. Induction immunosuppression based on ALG or ATG was applied in immunologically high-risk patients (different but compatible ABO blood groups, more than three HLA mismatches, a high index in mixed lymphocyte culture between donor and recipient or panel reactive antibody titers >50%). Until 1996, sequential therapy was used. Thus, cyclosporine A was introduced in a dose of 10 mg/kg BW only when graft function had become established (serum creatinine <300 μmol/l). Since 1996, cyclosporine was started 2 days preoperatively in all patients in a dose of 6–8 mg/kg BW due to a lack of medication for induction therapy. The dose was adjusted to achieve 12-h trough levels of 150–200 ng/ml during the first 6 months, then 150 ng/ml to the end of the first year and 100–150 ng/ml after the first post-transplant year. Azathioprine was given in a dose of 2 mg/kg/day unless leukopenia developed. Methyl-prednisolone was given i.v. in a dose of 500 mg during the operation, 250 mg in the next 2 days and 125 mg on the third post-transplant day, and after that prednisolone dose was tapered during the next 3 months to 0.15 mg/kg BW.

The database included donor (age, gender), recipient (age at transplantation, gender, peak PRA levels, underlying kidney disease, time spent on hemodialysis), transplant variables (HLA-A, B, and DR mismatches, SGF, AR) and graft function.

Statistical methods

The results were expressed as mean values with standard deviations (mean ± SD). The significance of differences between the mean values for the groups was calculated using the Mann–Whitney *U*-test and Student's *t*-test. Chi-square was used to compare frequency. Patient and graft survival was calculated using the Kaplan–Meier survival analysis, while the difference in survival between the examined groups of patients was calculated by the Cox–Mantle test. Risk factors for development of AR and SGF were analyzed using multivariate logistic regression, and for patients and graft outcome using the multivariable Cox proportional hazards model. The impact of covariates on the outcome for patients and grafts and/or serum creatinine concentration was tested first by a univariate analysis. Only those covariates significant by univariate analysis were used in multivariate analysis. Multivariate regression analysis was performed using step-wise selection. All analyses were performed using the SPSS statistical software package (Version 10; SPSS, Inc., Chicago, IL, USA). *P*-values <0.05 were considered statistically significant.

Results

Figure 1 presents the number of recipients of kidney grafts from living-related donors younger and older than 60 years transplanted in our institution from 1980 to the end of 2003. The frequency of older donors varied from 16% in 1989 to 56% in 1995. Only kidney graft recipients of living-related donors transplanted between 1987 and 1999 and regularly followed-up in our outpatient department were included in the present analysis.

Patient characteristics pretransplant

Data on the patients analyzed are presented in Table 1. The majority of donors were parents of recipients (96.3%), rarely siblings (2.5%) or grandparents (1.1%). Both donors and recipients from group 2 were significantly younger than donors and recipients from group 1 ($P = 0.0001$). Table 1 showed that the two recipient groups examined differed also in hemodialysis duration and HLA matches with their donors. Although there were more female donors and recipients in group 2, the gender difference between the groups was statistically insignificant. Global kidney function of all donors, expressed as CCr, was normal (95.04 ± 2.5 ml/min) (data not presented). Warm and cold ischemia times were similar in both groups. No surgical complications were noted during the donor nephrectomy and kidney transplantation. No severe anatomic abnormality in the grafts was found either.

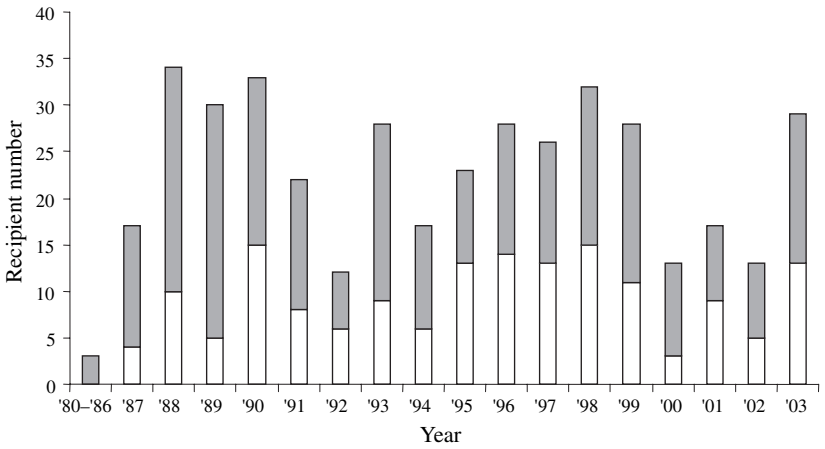

Figure 1 Number of patients receiving kidney grafts from living-related donors younger and older than 60 years in our institution. White column-donor older than 60, grey column-donor younger than 60 years.

Graft function follow-up and frequency of acute and chronic graft dysfunction

Table 2 presents the number of patients with acute kidney graft dysfunction as well as chronic graft dysfunction. The number of patients with one or more AR episodes was similar in both studied groups. However, SGF occurred more frequently in group 1 than in group 2 ($P = 0.001$). Hemodialysis was not necessary in all patients with SGF but in seven patients from group 1 and 5 patients from group 2. By the end of the first 3 months 27 patients with SGF from group 1 and 14 patients with SGF from group 2 did not reach normal graft function and they were designated as CRAD. In addition, CRAD developed due to other reasons but not SGF in eight recipients from group 1 and three recipients from group 2 in the first 3 months as well as in 18 and 14 patients before the end of the first year (Table 2). Thus, in the first post-transplant year CRAD developed more frequently in group 1 than in group 2. A graft biopsy was taken from 14 patients from group 1 and 23 patients from group 2 who developed CRAD. Histological analysis showed chronic allograft nephropathy in 12 patients from group 1 and 21 patients from group 2 and recurrent glomerulonephritis in two patients from each group.

Table 2. Evolution of renal graft function in both studied groups.

	Group 1 ($n = 115$)	Group 2 ($n = 158$)	P*
AR	34	50	n.s.
SGF	35 (7)	23 (5)	0.001
CRAD			
0–3rd month	27 ± 8	14 ± 3	0.01
4–12th month	18	14	0.04

*χ^2 test. N.s., not significant; CRAD, chronic renal allograft dysfunction; Data in parentheses indicate patients hemodialyzed during the first 7 days after transplantation. Data for CRAD from 0 to 3 months indicate those with slow recovery of graft function (27 from group 1 and 14 from group 2) and those with newly developed chronic graft failure (eight and three patients from groups 1 and 2, respectively).

According to univariate analysis, the following variables were found to be significant risk factors for development of acute kidney graft dysfunction: recipient gender, polycystic kidney disease as an underlying kidney disease, time spent on hemodialysis, peak PRA levels, donor age and age difference between donor and recipient, HLA B mismatches, established graft function, period after transplantation. Only these variables were used in the multivariate analysis. The multivariate analysis indicated that donor age was a risk factor for SGF ($P = 0.002$), while HLA B-mismatches significantly increased the risk factor for AR ($P = 0.03$) (Table 3).

Mean serum creatinine profiles during the 5-year follow-up period are presented in Fig. 2. Significant differences in mean serum creatinine levels between the groups were maintained almost throughout the entire period. The higher SGF rate was probably one of the main causes of slower graft function improvement in recipients from group 1. In the same group 22 (64.7%) recipients did not fully recover graft function after AR, while 23 (46%) recipients of younger kidney grafts did not fully recover graft function after an AR episode. Although the difference was not significant, it could influence late graft function. Multivariate analysis indicated the risk factors that affected serum creatinine levels at different points of the follow-up period (Table 4). It can be seen that donor age and immunological factors (HLA mismatches, PRA titer, AR) had the highest influence on older graft function. On the contrary, serum creatinine of younger graft recipients was higher in males, in those who experienced SGF, and in recipients with a greater difference between donor and recipient age, indicating that their graft function mainly depended on nonimmunological factors (Table 4).

Table 3. Risk factors for AR and SGF.

	B	SE	*P*	Odds ratio (95% CI)
AR				
HLA B MM	0.63	0.29	0.03	1.88 (1.0610–3.3511)
Period after transplantation	–0.007	0.003	0.04	0.99 (0.9865–0.9999)
SGF				
Donor age	0.9424	0.3111	0.002	2.5662 (1.3948–4.7214)

B, coefficient; SE, standard error of B; CI, confidence interval for odds ratio HLA B; MM = mismatches in HLA B.

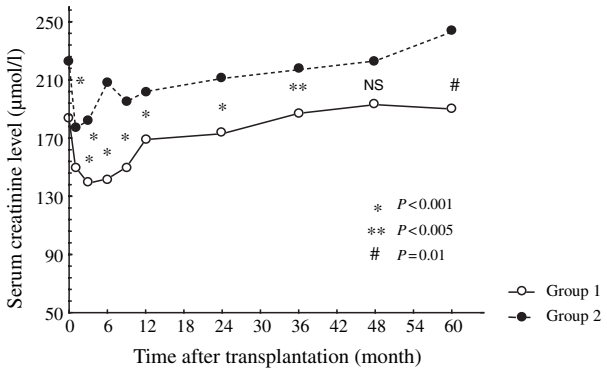

Figure 2 Serum creatinine level monitored 5 years after transplantation in patients receiving a kidney from a donor older (group 1) and younger than 60 years (group 2).

Patients and graft survival

Patient and graft survival over 5 years is shown in Fig. 3. Patient survival for the first five post-transplant years was 98%, 94%, 89%, 82% and 82% for group 1 and 99%, 98%, 98%, 97% and 93% for group 2. A significant difference was observed from the second post-transplant year until the end of the studied period ($P = 0.002$). Graft survival was 88.6%, 79.5%, 71%, 63.3% and 55.8% for group 1 (half-life 84 months) and 97%, 89%, 82.6%, 78.3% and 71.3% for group 2 (half-life 120 months) for every year, when a patient's death was counted as graft loss. Older grafts from group 1 had significantly poorer survival than grafts from group 2, during the entire studied period ($P = 0.001$). Graft survival censored for patient death was 91.2%, 82.7%, 75.8%, 70.5% and 63.4% for group 1 (half-life 96 months) and 97.4%, 90.7%, 84.4%, 81.4% and 75.7% for group 2 (half-life 168 months) for every year, and the difference was statistically significant from the second post-transplant year ($P = 0.02$–0.004). In addition, graft survival in recipients receiving a graft from donors aged 60–70 years and those receiving a graft from donors aged 70–80 years was compared. No significant difference in graft survival between

Table 4. Risk factors for high serum creatinine level at different points of the follow-up period in group 1 (receiving graft from older donor) and group 2 (receiving graft from younger donor).

	Group 1			Group 2		
Month after Tx	Risk factors	β	*P*	Risk factors	β	*P*
6	Donor age	0.213	0.033	SGF	0.415	0.000
				Recipient gender	–0.193	0.008
				Donor age	0.157	0.029
9	AR	0.309	0.001	SGF	0.365	0.000
	HLA A MM	–0.211	0.029	Recipient gender	–0.233	0.003
	HLA B MM	–0.210	0.034	D-R age difference	0.198	0.011
	Donor age	0.301	0.002			
	HD, month	–0.220	0.020			
12	D-R age difference	0.349	0.000	SGF	0.313	0.000
	AR	0.294	0.002	D-R age difference	0.202	0.010
	PRA	–0.197	0.035			
24	AR	0.274	0.01	SGF	0.297	0.001
	HLA A MM	0.230	0.032	D-R age difference	0.223	0.009
	Recipient gender	–0.269	0.04	D-R gender difference	–0.276	0.03
	D-R gender difference	0.320	0.039	PRA	0.275	0.01

D, donor; R, recipient; SGF, slow graft function; HLA A MM-mismatches in HLA A and B. For explanation of other abbreviations, see previous tables.

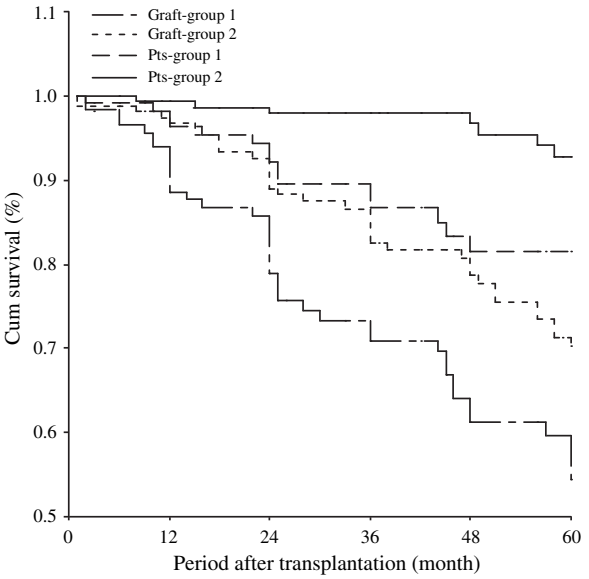

Figure 3 Patient and graft survival rate over 5 years.

Table 5. The causes of patient deaths and graft losses.

	Group 1	Group 2
Patient death	16	7*
Cardiovascular disorders	6 (2)	0
Infection	3	2
CNS insult	2 (2)	3
Pancreatitis	0	1
Cancer	1	0
ARDS	0	1 (1)
Intestinal bleeding	1	0
Unknown	2	0
Graft loss	47	41
Patient death	16	7
Chronic allograft nephropathy	12 (4)	21 (1)
Recurrent glomerulonephritis	2	2
Irreversible AR	3 (3)	2 (1)
Arterial thrombosis	0	2 (2)
CRAD – biopsy unproven	14 (5)	7

*$P = 0.009$.

Data in parentheses indicate patient death or graft loss occurring during the first post-transplant year CNS-central nervous system; ARDS-adult respiratory distress syndrome.

these two subgroups of older graft recipients was found (data not presented).

The causes of patient death and graft loss are shown in Table 5. Graft function was preserved in all patients who died. Although similar number of patients from two groups lost the grafts during entire follow-up period, graft loss in the first post-transplant year occurred more frequently in group 1 than in group 2 (16 vs. 5, $P = 0.001$).

The Cox proportional hazard model revealed that the high-risk factor for patient death was independently associated with increase of donor age ($P = 0.01$), polycystic kidney disease as an underlying kidney disease ($P = 0.004$) and graft function ($P = 0.0000$) (Table 6). The latter implied that a high serum creatinine level was followed by a poor patient outcome. The difference between donor and recipient age was found to be a significant risk factor for graft survival. In addition, patients with AR episodes had a 1.5 times higher risk for graft loss than patients without AR.

Graft survival was also calculated considering the presence of AR (Fig. 4). Inside both studied groups, grafts which experienced AR survived less than grafts without AR and the difference was significant ($P = 0.016$ for group 1 and $P = 0.025$ for group 2). Thus, the half-life for grafts from group 1 with AR was 60 months, but 108 months for grafts with no AR. For grafts from group 2 the half-life with and without AR was 80 and 129 months, respectively. Furthermore, the older grafts from group 1 with AR had a significantly shorter survival time than grafts from group 2 with AR ($P = 0.019$). In the absence of AR, the outcome for older and younger grafts was similar ($P = 0.07$).

Discussion

The present study shows that donor age has a detrimental effect on short- and long-term renal allograft function and survival. These data confirm our previous results [6] and are in accordance with the majority of published studies. Some of these were single-center reports analyzing small numbers of patients [8,14,15], while others included many patients [16] or presented data from well-known registries of transplant patients [17–22].

Table 6. Risk factors for lower patient and graft survival.

	B	SE	*P*	Odds ratio (95% CI)
Patient				
Donor age	0.053	0.02	0.015	1.054 (1.0118–1.0977)
Graft function	4.28	1.016	0.000	72.764 (9.9326–533.065)
ADPKD	1.53	0.63	0.015	4.606 (1.3456–15.7709)
Graft				
AR	0.45	0.20	0.02	1.5627 (1.0512–2.3229)
D-R age difference	0.05	0.01	0.000	1.0518 (1.0270–1.0772)

For explanation of abbreviations, see previous tables.

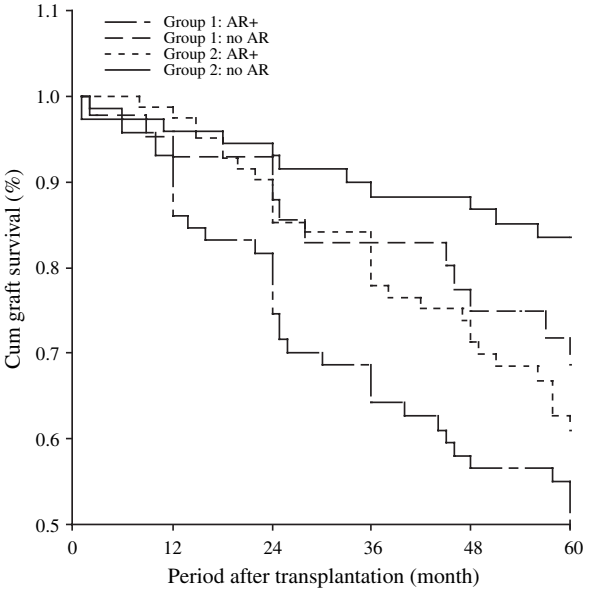

Figure 4 Five-year graft survival in the two examined groups considering AR.

However, several teams pointed out that donor age is not necessarily associated with an inferior allograft outcome. Donor age did not have the same influence in cadaveric and living donor kidney recipients or in recipients of different race [23–25]. Recently, Pessione *et al.* reported that the detrimental effect of donor age on graft survival was not an independent factor. Cardiovascular diseases in the donor were found to be associated with poorer graft survival, independent of donor age, but both risk factors had a cumulative effect [21]. The significant impact of cardiovascular diseases in donors on graft outcome had already been described [26–29]. These data may explain the different results obtained in kidney transplantation from older cadavers and living donors. Nevertheless, fewer studies referred to older living donors and reported controversial results [8,30–33]. These controversies partly originated from the differences in the population examined, in the age defined as the border for older donors and in the methods of analysis.

The present study confirmed the data on the significant influence of AR on graft survival obtained by other authors [34,35]. When we compared the graft survival in patients with one or more AR episodes and those without AR, we obtained results comparable with those of Kerr *et al.* [24], whose series was considerably more numerous. Namely, AR episodes markedly diminished graft survival of both older and younger kidney grafts, but in the absence of AR, survival of older and younger grafts was similar. Although the low number of histologically confirmed AR diminished the significance of our results, characteristic clinical, laboratory and ultrasound features of AR in the presence of low or normal cyclosporine levels and good response to the treatment with pulse methyl-prednisolone in all patients indicated that AR was really involved [10,36].

Donor age significantly predicted the long-term recipient survival i.e. the older the donor kidney, the worse the recipient survival. This could be partly due to recipient age, because our recipients, being mostly children of older kidney donors, were significantly older than the recipients of younger kidneys. In addition, they were longer on hemodialysis before transplantation. Both conditions have already been reported to be associated with higher comorbidity in patients on hemodialysis [37]. The older age and comorbidity contributed to the high mortality rate of older kidney recipients. The main cause of patient death for older graft recipients was cardiovascular disease, which together with cerebrovascular insult, accounted for 50% of all causes of death. A close correlation between donor age and cardiovascular mortality has already been reported [16,38], as well as an increasing frequency of graft loss due to patient death [39].

Data presented here indicated that donor age affected not only graft survival, but also graft function and both had a significant influence on recipient survival. A similar finding was reported by Keith *et al.* [40] for cadaveric kidney transplantation. Different methods were used for estimation of graft function during follow-up, most frequently CCr calculated by the Cockcroft–Gault formula [38,41], but also serum creatinine [16,37] as in the present study.

The analysis of factors influencing graft function revealed that donor age and the age difference between donors and recipients were the most significant risk factors modifying graft function in the first post-transplant year in both groups. Later on, i.e. after the second post-transplant year, donor age disappeared from the risk factors affecting graft function, but the other risk factors differed between the two groups. In recipients of older grafts, immunological factors (HLA mismatches, PRA titer, AR) had the greatest influence on graft function. In contrast to this, graft function in younger graft recipients was predominantly affected by nonimmunological factors (male gender, SGF). Although SGF was the main risk factor for poorer younger graft function, its influence on older graft function should not be disregarded, particularly due to the significantly higher incidence of SGF after transplantation of old grafts. A high incidence of SGF including DGF in older grafts, as well as its negative effect on graft function and outcome, has been reported elsewhere in other series [16,41]. There is also evidence that SGF strongly predisposed to AR which was found to be a significant risk factor for both graft function and graft survival, especially for older kidney grafts [42,43].

Nevertheless, AR occurred at a similar rate in both our groups but this is not a solitary result. Thus, some authors reported a similar incidence of AR in recipients of older and younger kidneys [16] while others found AR more frequently in older donor kidney recipients [41]. Regardless of its frequency, AR was shown to be one of the main risk factors for graft function and survival [34,35]. Moreover, Matas *et al.* [23] identified AR as the only significant risk factor for late graft failure in a group of living graft transplantations. In our study, CRAD, irrespective of its cause, started earlier in recipients of older kidneys and in the first post-transplant year its frequency was significantly higher in this group than in the recipients of younger kidneys. This might be related to a higher proportion of SGF as well as AR which did not resolve with fully functional recovery in older grafts. A similar problem was stressed by Meier–Kriesche *et al.* [38].

Our study also indicated the risk factors for older kidney graft function and graft and patient survival. These data are important because they direct toward strategies that could reduce or even avoid the influence of these risk factors and diminish the difference in transplant outcome between older and younger graft recipients. Special attention should be paid to the early events due to their great influence on patient and graft outcome. This involves careful selection of donors with high HLA compatibility, improving surgery and preservation techniques, minimization of ischemia times, use of drugs with high immunosuppressive potency and low nephrotoxicity, etc. The significance of these measures was clearly illustrated by disappearance of the difference in graft survival between older and younger donors in the absence of AR. Also, prevention of cardiovascular diseases and other co-morbidities, as important causes of patient death and graft loss, must not be neglected. Improvement of economic conditions in our country and the health service should enable the use and improvement of these measures in our practice allowing better transplantation results (incidence of SGF, AR recovery, patient and graft survival) to be expected.

Finally, considering all the results obtained, it is obvious that kidneys from older donors provide a poorer transplant outcome. However, the universal shortage of organs led other authors with similar results to suggest that they were acceptable for many patients and better than hemodialysis treatment [21,24,40,44]. In our country with a low number of cadaveric kidney transplantations and limited resources for hemodialysis treatment, older donors remain an unavoidable source of organs. This statement is supported by the fact that the mortality rate in our country was four times higher for hemodialysis than for transplant patients [45], and also, although glomerular hyperfiltration is frequent, unchanged kidney function was maintained during the long-term follow-up period of donors after donation of the kidney [46].

In conclusion, despite worse graft function and poorer patient and graft survival, kidney transplantation from living-related older donors may be an acceptable practice especially when wait times are prolonged or access to dialysis limited.

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