Transpl Int (1998) 11: 22–27

ORIGINAL ARTICLE

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Lesions in donor kidneys: nature, incidence, and influence on graft function

Received: 5 March 1997
Received after revision: 8 July 1997
Accepted: 21 August 1997

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Abstract The aim of this study was to assess the influence of kidney-donor transmitted pathology on graft function. Light and immunofluorescent microscopic findings from a surgical biopsy taken prior to transplantation from 114 cadaveric kidney donors were analyzed. Moderate to severe mesangial IgA deposits were considered consistent with IgA nephropathy. Pathological abnormalities were correlated together with donor age, number of mismatches, and type of immunosuppression by multivariate statistical analysis with the serum creatinine values from patients who experienced no acute rejection at 1 year. Serum creatinine values ($n = 52$) were not correlated with either nonspecific light microscopic lesions or immunofluorescent deposits found in the majority of kidney donors or with changes consistent with IgA nephropathy observed in 9 % of the cases. There was, however, a correlation with donor age, which was also correlated with the extent of chronic lesions ($P < 0.001$).

Key words IgA nephropathy, kidney transplantation, biopsy

Introduction

There is little data available on kidney donor pathology transmitted to the recipient at the time of renal transplantation and even less on their functional consequences for the graft [3]. The aim of the present work was to report the light and immunofluorescent microscopic abnormalities found in a series of cadaveric kidney donor biopsies taken at the time of renal transplantation, with special emphasis on their influence on graft function.

Materials and methods

From September 1992 to January 1995, a "zero-hour" biopsy was taken from 114 consecutive cadaveric kidney donors dispatched by Eurotransplant. Before implantation and revascularization, all kidneys were reperfused and a wedge biopsy measuring ± 5 × 5 × 10 mm^3 was performed at the upper pole of the kidney. Hemostasis was completed before unclamping using a 0 chromic catgut suture. Postoperative immunosuppression consisted of a quadruple drug induction regimen. A 10-day course of 4 mg/kg per day i.v. R-ATG (Fresenius, Germany) was administered, along with cyclosporin A at a starting dose of 5–8 mg/kg per day, slowly tapered according to 12-h trough blood levels (between 200 and 400 ng/ml, whole blood; nonspecific Abbott TDX assay) and to serum creatinine level. Azathioprine was given at a constant dose of 1 mg/kg per day, and prednisolone was started at 0.5 mg/kg per day and slowly tapered down to 0.1 mg/kg per day by month 9. Variants of the immunosuppressive regimen included: antithymoglobulins, cyclosporin, azathioprine, and steroids; FK506, azathioprine, and steroids; and cyclosporin, azathioprine, and steroids.

Material for light microscopy was fixed in Duboscq-Brazil, embedded in paraffin, and stained with hematoxylin and eosin, periodic acid-Schiff, periodic acid silver methenamine, Masson's trichrome, and phosphotungstic acid-hematoxylin. Tissue for immunofluorescence examination was snap-frozen and sections were incubated with fluorescein-conjugated antisera monospecific for IgG, IgA, IgM, fibrinogen, C3, and C1 q components of complement.

Glomerular, tubular, interstitial, and arterial acute and chronic lesions were semiquantitatively scored on a 1–3 scale according to their extension in the glomerular, tubular, interstitial, or arterial structures (0 %–29 % = 1; 30 %–59 % = 2; ≥ 60 % = 3). Acute lesions included intracapillary thrombi, tubular epithelial cell degeneration, vacuolization, and interstitial edema. Chronic lesions included glomerular global sclerosis, tubular atrophy, interstitial inflammatory cells, arterial hyalin deposits, interstitial and intimal sclerosis. Both brightness and extension of linear, granular, glomerular, tubular, and arterial immune deposits were semiquantitatively scored on a 0.5, 1, 2, 3 scale as traces, discrete or +, moderate or + + and severe or + + + amounts, respectively.

The sum of the scores of acute lesions, chronic lesions, and immune deposits in each biopsy represented the "acute lesion", "chronic lesion", and "immunofluorescent deposit" indexes, respectively. Broad transcortical, inflammatory, tubulointerstitial sclerosis that was sharply delineated from the adjacent normal cortex was considered consistent with the histological diagnosis of chronic pyelonephritis. Diffuse mesangial IgA deposits (+ + to + + +), in isolation or in association with various combinations of mesangial IgG, IgM, fibrinogen, C3, and C1 q (scored at the same value or lower than IgA) were considered specific immunofluorescent deposits consistent with IgA nephropathy. The following were considered nonspecific findings: linear IgG, fibrinogen; mesangial traces or + amounts of IgA in isolation; mesangial IgG, IgM, fibrinogen, C3, C1 q, in isolation or in various combinations, with or without traces or + amounts of mesangial IgA; granular C3 on glomerular vascular poles, on arterial walls and on tubular basement membranes.

Indication for renal graft biopsy was based on a 20 % increase in the serum creatinine level when all other causes of graft dysfunction had been ruled out and/or on appearance of proteinuria and/or hematuria. All biopsies were performed with an 18-gauge needle and a biopsy gun device under ultrasonic guidance in order to exclude any obstructive nephropathy.

Acute graft failure due to acute tubular necrosis was defined as the need to resort to hemodialysis at least once during the 1st week post-transplantation in the absence of any sign of rejection.

Results were expressed as means ± standard deviation. A univariate regression equation was calculated when indicated using linear or exponential regression where appropriate. Stepwise multivariate regression was used to study the influence of a set of variables on the creatinine level at 1 year. Whenever possible, appropriate transformation was applied to whole variables. The odd ratios and the corresponding confidence intervals were calculated. Actuarial survival rates were calculated using the Kaplan-Meier method; *P* values comparing survival rates were calculated using the log-rank test. We analyzed graft survival considering all deaths as graft loss. Other statistical methods used were the chi-square analysis and the Mann-Whitney U-test, when required.

Results

The median time of first warm ischemia was 0 min (range 0–20 min) and that of cold ischemia was 24.02 h (range 14.03–46.07 h).

Of the 114 grafts, 12 (11 %), 22 (19 %), 25 (22 %), 30 (26 %), 19 (17 %), and 6 (5 %) were distributed over the 12–19, 20–29, 30–39, 40–49, 50–59, and 60–74 years donor age groups with a mean age of 38 ± 14 years. The female/male ratio was 0.60 (43/71). In addition to pathological changes consistent with specific nephropathies in

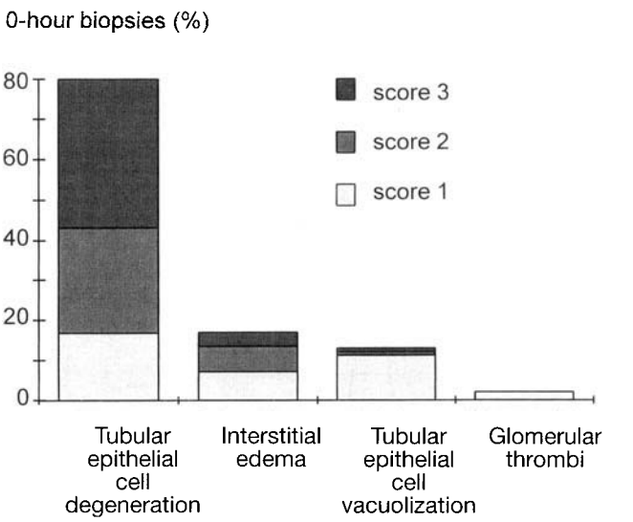

Fig. 1 Type, incidence, and score distribution of light microscopic acute lesions in "zero-hour" biopsies (n = 114)

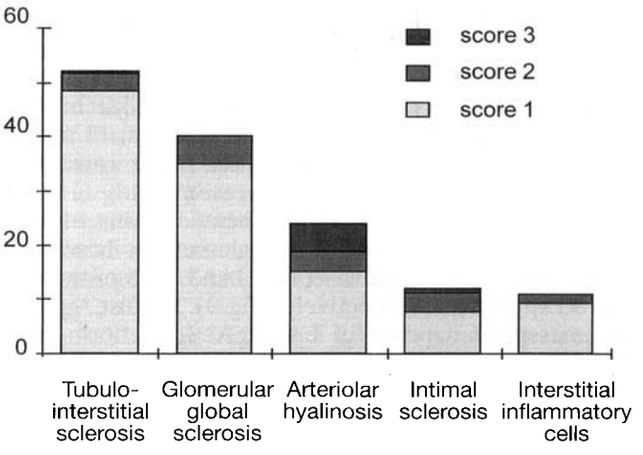

Fig. 2 Type, incidence, and score distribution of light microscopic chronic lesions in "zero-hour" biopsies (n = 114)

11 biopsies, all specimens revealed nonspecific lesions (n = 112) and/or immune deposits (n = 99) by light and immunofluorescent microscopy, respectively. Tubular epithelial cell degeneration, characterized by brush border loss, luminal exfoliation, and/or luminal epithelial cellular deliquescence, extended to more than 60 % of the tubules (score 3) in half of the cases and was the most frequent acute lesion found in 91 of the 114 (80 %) biopsies (Fig. 1). Interstitial edema, sometimes extensive, and tubular epithelial cell vacuolization, mostly focal, were found in 19 of the 114 (17 %) and 15 of the 114 (13 %) biopsies, respectively. In two cases, focal intracapillary glomerular thrombi were noted. Tubulointerstitial and glomerular global sclerosis, extending to less than 30 % of the tubulointerstitial tissue and glomeruli, re-

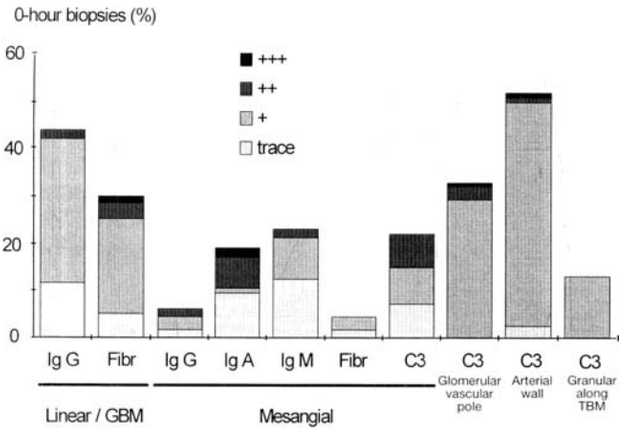

Fig. 3 Type, incidence, and score distribution of nonspecific and specific immunofluorescent deposits in "zero-hour" biopsies ($n = 114$). (*Fibr* fibrinogen, *GBM* glomerular basement membranes, *TBM* tubular basement membranes)

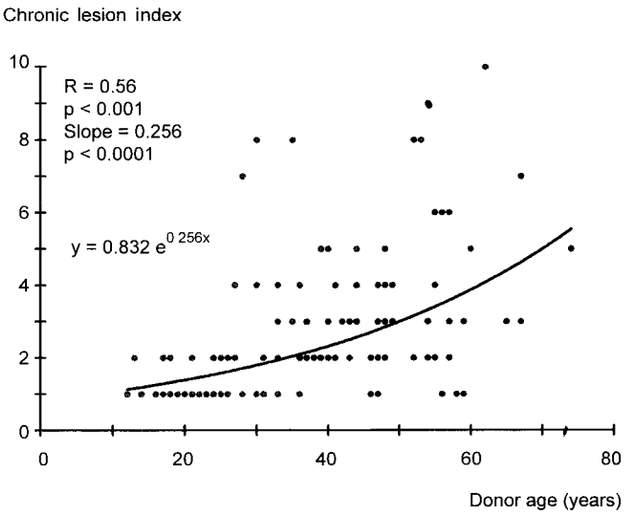

Fig. 4 Relationship between donor age and "chronic lesion" index of donor kidney ($n = 89$)

spectively (score 1) in most of the cases, were the most frequent chronic lesions found in 59 of the 114 (52 %) and in 46 of the 114 (40 %) biopsies, respectively (Fig. 2). Usually, discrete arterial hyaline deposits and intimal sclerosis were observed in 27 of the 114 (24 %) and 14 of the 114 (12 %) biopsies, respectively. Mild, chronic, interstitial, inflammatory cells were noted in 11 of the 114 (10 %) biopsies. Chronic lesions were consistent with chronic pyelonephritis in one case (1 %).

Weak to moderate, diffuse, linear deposits of IgG and of fibrin were noted along glomerular basement membranes in 50 of the 114 (44 %) and in 35 of the 114 (30 %) specimens, respectively (Fig. 3). Diffuse, granular, mesangial deposits of IgG, IgA, IgM, fibrinogen, and C3 were found, in various combinations, in 7 of the 114 (6 %), 22 of the 114 (19 %), 26 of the 114 (23 %), 5 of the 114 (4 %), and 25 of the 114 (22 %) biopsies, respectively. In ten of them (9 %), mesangial IgA deposits were observed (+ + or + + + , in association with various combinations of equal or lower amounts of mesangial IgG, IgM, fibrinogen, or C3), findings considered consistent with IgA nephropathy. Granular C3 deposits were observed on glomerular vascular poles in 38 of the 114 (33 %), arteriolar walls in 59 of the 114 (52 %) and, occasionally, along tubular basement membranes in 13 of the 114 (11 %) biopsies, respectively. Severe tubular epithelial cell degeneration (score 3) was noted in 3 out of 12 patients with post-transplantation acute graft failure due to acute tubular necrosis.

Univariate regression analysis showed that, unlike the "acute lesion" or "immunofluorescent deposit" indexes, the "chronic lesion" index could be correlated with donor age. An exponential regression line with a significant correlation coefficient ($R = 0.56$; $P < 0.001$) and slope ($R = 0.256$; $P < 0.0001$) showed a positive correlation between these two latter parameters (Fig. 4).

Multivariate regression analysis was carried out using the serum creatinine values at 1 year following transplantation as the dependent variable and several clinico-pathological parameters as independent variables. These included donor age, number of mismatches, type of immunosuppressive regimen, presence or absence of specific or nonspecific immune deposits, and acute or chronic lesions, as well as "acute lesion", "chronic lesion", and "immunofluorescent deposit" indexes. Recipients who experienced acute rejection during the 1st year post-transplantation were excluded from this analysis. Donor age was the only variable that had a statistically significant impact on the serum creatinine values available in 52 recipients at 1 year post-transplantation ($P = 0.015$; Table 1). A linear regression line with a significant correlation coefficient ($R = 0.44$; $P = 0.001$) and slope ($R = 0.0083$; $P = 0.015$) showed a mild correlation between donor age and serum creatinine values in these patients (Fig. 5).

The fate of immune deposits was evaluated in 42 graft biopsies taken an average of 4 months after transplantation (range 0.5–24 months) from 42 patients. Deposits considered as nonspecific (linear IgG, fibrinogen; mesangial traces of IgA in isolation; mesangial IgM, C3, in various combinations, with or without traces or + amounts of mesangial IgA; granular C3 on glomerular vascular poles, on arterial walls, and tubular basement membranes) had been observed in isolation in 35 of the 42, and in association with deposits considered as specific [diffuse mesangial deposits of IgA (+ + or + + +), associated with various combinations of equal or lower amounts of mesangial IgG, IgM, fibrinogen, C3, consistent with IgA nephropathy] in 7 of the 42 0-h biopsies. The following changes in nonspecific im-

Table 1 Factors likely to influence serum creatinine of recipients without rejection 1 year after transplantation ($n = 52$) (*IF* immunofluorescent)

	P	OR 1[a]	OR	OR 2
Donor age	0.015	1.0017	1.0083	1.0150
Number of mismatches	0.057	0.9922	1.2940	1.6874
Specific IF deposits	0.096	0.6146	0.8003	1.0420
Nonspecific IF deposits	0.150	0.9274	1.2157	1.5936
Chronic lesions	0.554	0.6581	0.9087	1.2550
Acute lesions	0.230	0.8998	1.1747	1.5336
Immunosuppression	0.980	0.7671	1.0028	1.3110
IF deposit index	0.110	0.9501	1.2368	1.6099
Chronic lesion index	0.400	0.8440	1.1320	1.5183
Acute lesion index	0.600	0.6988	0.9277	1.2317

[a] OR 1 and OR 2 are, respectively, the inferior and the superior limits of the odds ratios (OR) confidence intervals calculated for $\alpha = 0.05$

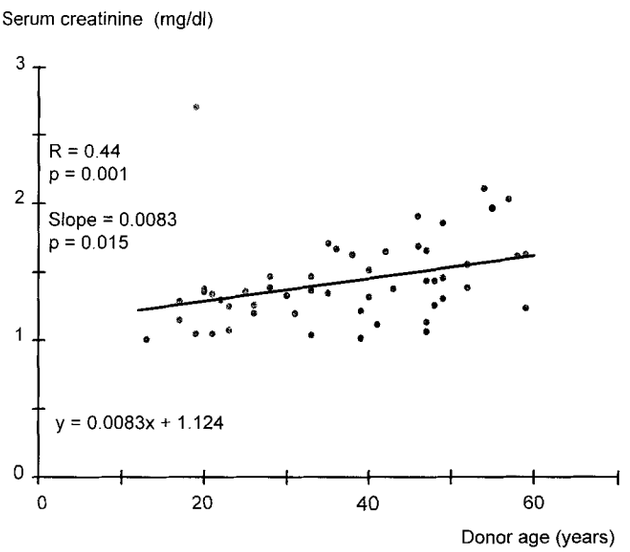

Fig. 5 Relationship between donor age and serum creatinine values 1 year after kidney transplantation in recipients ($n = 52$) without rejection episodes during the 1st post-transplant year

mune deposits in these 42 rebiopsied patients were observed. Out of 22 donors with traces ($n = 5$), + ($n = 16$), and + + ($n = 1$) amounts of linear IgG, clearance was achieved by 18 (82 %) and maintenance by 4 of the receivers (score +). Out of 15 donors with traces ($n = 4$), + ($n = 9$), + + ($n = 1$), and + + + ($n = 1$) amounts of linear fibrinogen, clearance was achieved by 11 (73 %) and maintenance by 4 of the receivers as traces ($n = 1$), + ($n = 2$), and + + + ($n = 1$) amounts. Donor traces of mesangial IgA deposits were cleared by all receivers ($n = 3$). Out of 5 donors with traces ($n = 3$) and + ($n = 2$) amounts of mesangial IgM deposits, clearance was achieved by all of the receivers. Out of 10 donors with traces ($n = 3$), + ($n = 5$), and + + ($n = 2$) amounts of mesangial C3 deposits, clearance was achieved by 9 (90 %) and maintenance by 1 of the receivers (score + +). Out of 15 donors with granular vascular polar C3 deposits (score +), clearance was achieved by all of the receivers. Out of 29 donors with granular C3 arterial deposits (score +), clearance was achieved by 14 (48 %) and maintenance by 15 of the receivers (score +). Out of 2 donors with granular C3 deposits along tubular basement membranes (score +), clearance was achieved by 1 (50 %) and maintenance by 1 of the receivers (score +).

The fate of mesangial deposits considered as specific in 7 of the 42 rebiopsied patients was as follows. Four of these receivers (4/42) had native kidney diseases, i.e., chronic glomerulonephritis of unknown etiology, IgA nephropathy, polycystic kidney disease, and membranoproliferative glomerulonephritis type I. The 0-h biopsy of their grafted kidney contained + + IgA, + + IgM; + + IgA, + IgG, + + C3; + + + + IgA, + IgG, + + C3; and + + IgA, traces of IgM, + + C3 mesangial deposits, respectively; yet, they achieved clearance of all deposits within 14 days, 1, 2, and 6 months post-transplantation, respectively. In 3 of the 42 receivers whose native kidney disease was chronic glomerulonephritis of unknown etiology and whose 0-h biopsies respectively contained mesangial + + IgA associated with mesangial traces of IgM, fibrinogen; + + IgG, trace amounts of IgM, + + C3; and + IgM, + fibrinogen, + + C3, we observed the following changes: in the first case, a decrease to + mesangial IgA deposits (associated with mesangial + IgM, + fibrinogen, + + C3 and + C1 q) at 9 months post-transplantation; in the second case, an increase to + + + mesangial IgA (associated with mesangial + + IgG, + + IgM, + + C3, + + C1 q) at 5 months postoperatively; and, in the third case, maintenance of + + mesangial IgA deposits (associated with mesangial + IgM and traces of fibrinogen) at 6 months post-transplantation.

Actuarial graft and patient survival rates, as well as the number of functioning grafts in patients with no immunofluorescent deposits ($n = 15$), nonspecific deposits only ($n = 89$), and Ig A deposits (+ + / + + +) consistent with IgA nephropathy ($n = 10$) in the 0-h biopsy, were

Table 2 Follow-up of grafted patients according to immunofluorescent deposits in the 0-hour biopsy (*NA* not applicable)

	0-hour biopsy						
	No immunofluorescent deposits ($n = 15$)		Nonspecific immunofluorescent deposits ($n = 89$)		IgA deposits (++/+++) consistent with IgA nephropathy ($n = 10$)		Total patients
	Rebiopsy	No rebiopsy	Rebiopsy	No rebiopsy	Rebiopsy	No rebiopsy	
At 1 year Number	7	8	35	54	7	3	114
Actuarial graft survival rate	85.7 %	100.0 %	91.4 %	96.3 %	85.7 %	100.0 %	93.9 %
Actuarial patient survival rate	100.0 %	100.0 %	100.0 %	98.1 %	100.0 %	100.0 %	99.1 %
Acute rejection	4/7 (57.1 %)	0/8 (0.0 %)	27/35 (77.1 %)	7/54 (13.0 %)[a]	5/7 (71.4 %)	0/3 (0.0 %)	43/114 (37.7 %)
Serum creatinine level (mg/dl)	2.60 ± 1.73	1.37 ± 0.24	1.99 ± 0.99	1.44 ± 0.32	1.40 ± 0.32	1.28 ± 0.26	1.66 ± 0.78
Functioning graft	6	8	32	52	6	3	107
At 3 years Number	6	6	32	45	6	2	97
Actuarial graft survival rate	71.4 %	100.0 %	71.4 %	94.4 %	85.7 %	NA	84.2 %
Actuarial patient survival rate	100.0 %	100.0 %	96.9 %	98.1 %	100.0 %	NA	96.2 %
Serum creatinine level (mg/dl)	2.04 ± 0.76	1.42 ± 0.30	1.67 ± 0.47	1.48 ± 0.32	1.51 ± 0.35	NA	1.56 ± 0.41
Functioning graft	4	6	22	42	5	0	79
Graft loss	2	0	10	3	1	2	18
Acute rejection	0	0	3	2	1	0	6
Chronic rejection	1	0	4	0	0	0	5
Death[b]	0	0	1	0	0	2	3
Infection	0	0	1	0	0	0	1
Nephropathy recurrence	1	0	1	1	0	0	3

[a] Rejection based on clinical criteria
[b] Death with functioning graft

not statistically different at 1 year. By contrast, the actuarial graft survival rate at 3 years of the subgroup of rebiopsied patients with nonspecific immunofluorescent deposits only ($n = 32$) and a high incidence of acute rejection was significantly lower than that of the subgroup of nonrebiopsied patients with nonspecific immunofluorescent deposits only ($n = 45$) and not statistically significantly different in all the other subgroups including that of changes consistent with IgA nephropathy. The serum creatinine level at 1 year was significantly higher in the rebiopsied patients than in those who were not when subgroups of patients with no immunofluorescent deposits (2.60 mg/dl vs 1.37 mg/dl; $P < 0.022$) and nonspecific immunofluorescent deposits only (1.99 mg/dl vs 1.44 mg/dl; $P < 0.003$) were compared. There was, however, no statistically significant difference in serum creatinine values at 1 year between the two subgroups of patients (with or without rebiopsy) with changes consistent with IgA nephropathy. At 3 years, no statistically significant difference in serum creatinine values was found between the different subgroups (Table 2).

Discussion

Nonspecific light microscopic lesions and/or immunofluorescent deposits were observed in all kidney donor biopsies. They were associated with lesions consistent with chronic pyelonephritis in one case and with immune deposits consistent with IgA nephropathy in ten cases.

The tubular epithelial cell degeneration observed in our series was similar to the necrotic and lytic appearances noted in pre- and postperfusion kidney donor biopsies [6]. As with the latter, it may have been attributed to hypoxia and/or procurement. What is interesting in our series is that, even when extensive, this epithelial cell degeneration did not correlate with the occurrence of acute renal failure due to acute tubular necrosis in the early post-transplantation period.

We observed vascular, glomerular, or tubulointerstitial sclerosis in half of the kidney donors, which is in accordance with findings of other studies [3]. Their extension to less than 30 % of the biopsy was consistent with the unremarkable past medical history of the donors, according to Eurotransplant. They most likely resulted from aging since they were correlated with donor age in our series, as in others [3]. Despite a rather equal distribution of donor age groups in our and other series [3], arteriosclerotic lesions were less prominent in our cases. Interestingly, tubulointerstitial and intimal sclerosis (involving less than 30 % of the parenchyma in more than half of the biopsies) were transmitted by the donor in 48 % (55/114) and 8 % (9/114) of the cases, respectively, and thus should not necessarily be attributed to chronic rejection. Unlike donor age, sclerosis of renal structures

in donor kidneys did not correlate with serum creatinine values at 1 year post-transplantation. This would seem to corroborate the interesting observation already stressed by others, that a negative impact of donor age on graft function has only been noted in cadaveric series (as here) but not in those including living related donors [5].

The linear IgG and/or fibrinogen deposits along glomerular basement membranes were weak and never associated with C3 or necrotic inflammatory lesions. Since they were not observed in autopsy kidneys or in early graft biopsies, they were probably related to ischemia. It could be that like the weak, nonimmune linear deposition of IgG along biochemically altered glomerular basement membranes in diabetes [1], reversible alterations of the glomerular basement membranes, resulting in a transient, weak, probably nonimmune adherence of proteins, might have been induced. The sparse granular C3 deposits at glomerular vascular poles and on arteriolar walls may have resulted from complement activation in extracellular matrices by cell remnants issued from normal cell turnover [4]. Nonspecific immune deposits were not correlated with donor age or with graft function at 1 year after transplantation. The poorer graft survival in patients needing a rebiopsy than in those who did not, regardless of the presence of nonspecific immunofluorescent deposits in the 0-h biopsies, seems to indicate that such deposits play no role in graft outcome, whereas acute rejection episodes do [7].

Mesangial IgA deposits consistent with IgA nephropathy were incidentally found in 9% of our biopsies, which is close to the reported incidence of 5%–12% in healthy kidney donors [2, 3]. These deposits were cleared in more than half of the cases (4/7) less than 1 year after transplantation, in accordance with findings from the studies mentioned above [2, 3]. Interestingly, their clearance was among the fastest in the native kidney of our single patient with Berger's disease. These results suggest that the mesangial substrate plays an important role in the pathogenesis of deposition and/or removal of IgA. Finally, in our series, as in others [2, 3], persisting and even increasing mesangial IgA deposits had no impact on short-term graft outcome. In this group of patients with inherited changes consistent with IgA nephropathy, the good graft survival rate, irrespective of acute rejection episodes, may have been due to the small number of patients.

In conclusion, though nonspecific kidney donor pathology seems almost invariably to have been transmitted to the recipient in our series, short-term graft function was unaffected. This is a welcome finding in view of the present shortage of kidney grafts. Whilst, as already stated, assessment of chronic lesions in the donor kidney before transplantation [3], we showed that acute tubular lesions, the lesions most frequently and extensively transmitted, did not account for either early or short-term graft dysfunction. The influence of donor-transmitted changes consistent with IgA nephropathy on long-term graft function has yet to be determined with a larger number of cases and a longer follow-up period. Finally, whether acute light microscopic lesions and nonspecific immunofluorescent deposits should be attributed to hypoxia or reperfusion techniques is a question that can only be answered in future studies by resorting to "zero-hour" biopsies from the same kidney, not only before revascularization but also before procurement and cold ischemia.

Acknowledgement We are grateful to Dr. C. Craddock-de Burbure for revising the manuscript.

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