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Recurrent and *de novo* glomerulonephritis following renal transplantation: higher rates of rejection and lower graft survival

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Abstract
Purpose In this retrospective study with case–control design, we aimed to determine the clinical and pathological characteristics of post-transplant glomerulonephritis (GN), and their effects on transplant recipients.
Methods One hundred and twenty renal transplant recipients with biopsy-proven recurrent or *de novo* primary GN were compared with two matched control groups including 120 transplant recipients with nonrecurrent primary GN (nonrecurrent GN group) and 120 transplant recipients with non-GN etiology (non-GN group). Primary outcome was allograft loss, and secondary outcomes were biopsy-confirmed cellular or antibody-mediated rejection.
Results In recurrent/*de novo* GN, nonrecurrent GN and non-GN groups, 54.2% ($n = 65$), 16.7% ($n = 20$) and 8.3% ($n = 10$) of patients reached primary outcome after a median follow-up of 96 (IQR: 56–149) months, respectively. Allograft loss was significantly higher in recurrent/*de novo* GN group compared to nonrecurrent GN and non-GN groups ($p < 0.001$). At 10 years, allograft loss rates in recurrent/*de novo* GN group were 54.2% for focal segmental glomerulosclerosis, 53.2% for membranoproliferative glomerulonephritis, and 33.4% for IgA nephropathy cases. Biopsy-confirmed rejection rate was significantly higher in the recurrent/*de novo* GN group ($n = 25$, 20.8%) compared to non-GN ($n = 8$, 6.7%) group ($p = 0.001$).
Conclusions Recurrent/*de novo* GN is associated with higher risk of rejection and worse allograft survival.

Keywords Renal transplantation · Graft survival · Graft rejection · Glomerulonephritis

Introduction

Renal transplant recipients, whose primary disease was glomerulonephritis (GN), suffer from a worse allograft survival as compared to patients with other primary renal diseases as the underlying etiology [1, 2]. One of the major factors playing a role in this unfavorable outcome is the risk of recurrence of primary GN in the allograft [1, 2]. The rate of recurrence has been reported to vary between 6 and 24.4% [1–6]. Reasons for varying rates of recurrence may be differences in study population, design and duration of follow-up, as well as different policies regarding allograft biopsy [1–6]. Therefore, it is very possible that this important complication may go underdiagnosed.

In recent years, with the implementation of various and improved immunosuppressive treatment regimens and prolongation of graft survival, effects of recurrence on allograft survival have become more important than ever [7]. Thus, numerous studies on the features of post-transplant recurrent/*de novo* GN have been conducted; however, very few have had an appropriate control group [5].

We, therefore, aimed to evaluate clinical and pathological characteristics of recurrent/*de novo* GN, effects of

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post-transplant GN on renal allografts and outcome of transplant recipients by using comparable control groups.

Materials and methods

Patients

A total of 120 patients (87 male, 33 female) who underwent a renal transplantation between 1980 and 2014 at hospitals of Istanbul Faculty of Medicine and Cerrahpasa Faculty of Medicine (both affiliated with Istanbul University) and developed biopsy-proven recurrent or *de novo* GN were analyzed in this retrospective case–control study, which was conducted between March 2015 and April 2016. Among these patients, 58 had focal segmental glomerulosclerosis (FSGS), 30 IgA nephropathy (IgAN), 15 membranoproliferative glomerulonephritis (MPGN), nine membranous nephropathy (MN), six atypical hemolytic uremic syndrome (aHUS), one C1q nephropathy and one lupus nephritis. Recurrent/*de novo* cases were stratified according to the timing of diagnosis: Early post-transplant recurrent/*de novo* disease was defined as a diagnosis within 12 months after transplantation, while patients were diagnosed to suffer from late post-transplant recurrent/*de* novo disease when this complication appeared after 1 year of transplantation. Each patient with recurrent/*de novo* GN was matched with two control groups.

The first control group (nonrecurrent GN group) included 120 patients (85 male, 35 female) who underwent a renal transplantation because of an end-stage renal disease (ESRD) caused by biopsy-proven primary GN and have no clinical and laboratory signs of recurrence (i.e., new onset proteinuria and/or increased serum creatinine level). Transplant recipients with macroscopic or microscopic hematuria were also excluded. In the nonrecurrent GN group, types of GN were as follows: FSGS ($n = 54$), IgAN ($n = 32$), MPGN ($n = 16$), MN ($n = 9$), lupus nephritis ($n = 3$), mesangioproliferative GN ($n = 3$), anti-glomerular basement membrane disease ($n = 1$), IgM nephropathy ($n = 1$) and fibrillary GN ($n = 1$).

The second control group consisted of 120 renal transplant recipients (86 male, 34 female) whose primary renal diseases leading to ESRD were other than GN (non-GN group), such as polycystic kidney disease, chronic pyelonephritis, vesicoureteral reflux (reflux nephropathy) or urolithiasis. Diabetic patients were not included in this group. The controls for each index case were chosen from the first consecutive patients who received renal grafts during the same period at the same centers and were matched with the index cases regarding age, gender, donor gender, donor type (living or deceased donor) and time of transplantation. All of the living donor transplantations were performed from relatives of the recipients. The features of study and control group patients are shown in Table 1.

Pre-transplant antihuman leukocyte antigen (HLA) antibodies were found negative in all transplant recipients. Initially, all patients were treated by triple maintenance immunosuppressive regimen including a calcineurin inhibitor (cyclosporine or tacrolimus), azathioprine or mycophenolate mofetil and prednisolone. Patients with a high risk of FSGS recurrence due to the history of rapid progression to ESRD received 5–8 courses of plasmapheresis (1 plasma volume/exchange) over the 2 weeks in the immediate perioperative period. Induction therapy (ATG Fresenius, 2 mg/kg/day for 3–7 days) was used in transplantations to high immunological risk recipients. All of the patients received intraoperative methylprednisolone bolus injection at the dosage of

Table 1 Demographics and baseline characteristics of the patients in three groups

	Recurrent/*de novo* GN ($n = 120$)	Nonrecurrent GN ($n = 120$)	Non-GN ($n = 120$)	p value
Clinical characteristics of recipients				
Male/female (*n*)	87/33	85/35	86/34	0.96
Age (years) (median–range)	44 (18–74)	45 (24–75)	44 (26–73)	0.81
BMI (kg/m^2) (mean ± SD)	23.7 ± 4	22.4 ± 3.9	22.4 ± 3.4	0.012
Time on dialysis (months) (median–IQR)	16 (6–36)	18 (6.75–36)	20 (10.5–50)	0.387
Duration of follow-up (months) (median–IQR)	85 (47–139)	97 (59.5–147.5)	110 (77.5–170)	< 0.001
Baseline characteristics of donors				
Living donor, *n* (%)	96 (80%)	96 (80%)	95 (79%)	0.983
Deceased donor, *n* (%)	24 (20%)	24 (20%)	25 (21%)	
Donor age (median–range)	46 (13–83)	47 (15–72)	48 (18–78)	0.682
Donor gender—male, *n* (%)	56 (47%)	57 (48%)	62 (52%)	0.658
HLA mismatches (mean ± SD)	3 ± 1	3 ± 0.9	2.9 ± 1	0.571

GN glomerulonephritis, *IQR* interquartile range, *BMI* body mass index, *SD* standard deviation, *HLA* human leukocyte antigen

500 mg. On postoperative day 1, patients received methylprednisolone beginning with a dose of 120 mg daily, with a rapid taper and reaching to maintenance dose of 10 mg daily within the first month and 5 mg daily within the first year. Target blood levels after the third month of transplantation were 50–150 and 5–10 ng/mL for cyclosporine (C0) and tacrolimus, respectively. If necessary, alterations were made in treatment strategies due to post-transplant complications (including transplant rejection), serious adverse events and drug intolerance during the follow-up. Patients who suffered from post-transplant FSGS recurrence were treated with an additional 5–8 courses of plasmapheresis. Prednisolone dose was increased and maintained at a daily dose of 7.5–10 mg in the patients who suffered from post-transplant recurrent/*de novo* GN.

Histopathological evaluation

Adequate renal biopsy specimens, which were defined as having seven or more glomeruli with at least two arteries, were evaluated. Three- to four-micrometer sections were used for all histochemical and immunohistochemical staining. 0.4–0.6-cm unfixed tissue was frozen with liquid nitrogen for immunofluorescence staining (IgG, IgM, IgA, C1q, C3 and fibrinogen). Remaining tissues were fixed in Hollande's fixative, embedded in paraffin and processed routinely for light microscopic evaluation (hematoxylin and eosin, periodic acid–Schiff, methenamine silver-periodic acid, Masson trichrome, Congo red). Banff 2013 diagnostic categories and related criteria were used for the final pathological diagnosis [8]. In order to standardize the definition of antibody-mediated rejection (AMR), renal allograft biopsies were reviewed and retrospectively rescored [8]. Immunofluorescence staining was graded by using a scale of 0–3. C4d staining was performed by immunohistochemistry on paraffin-embedded tissue blocks. Linear and circumferential staining in peritubular capillaries was regarded as positive according to the recent Banff scoring system (C4d > 0) [8]. A nephropathologist (YO) who was blinded to the previous pathology reports and clinical data confirmed the diagnoses by reviewing all the available biopsy samples.

Study outcomes

The primary outcome of the study was allograft loss, which was described as the loss of graft function leading to dialysis or retransplantation, or death with a functioning graft. Biopsy-confirmed cellular rejection or AMR was described as the secondary outcomes. Follow-up period was considered as the time interval between transplantation time and the last outpatient visit, allograft loss or death. The impact of recipient- and donor-related factors (transplant age, recipient gender, donor age and gender, donor type, HLA mismatches) on primary and secondary outcomes was analyzed.

Statistical analyses

Statistical analyses were performed using SPSS software for Windows (SPSS version 21.0, IBM Corp., Armonk, NY). Data are expressed as mean ± standard deviation (SD) when normally distributed or as the median [interquartile range (IQR)] otherwise. Parametric and nonparametric tests were used according to the distribution pattern of the data. Comparisons of continuous variables between two groups were assessed by using the unpaired *t* test or the Mann–Whitney *U* test, where appropriate. The differences in the proportions of different patient groups were compared by the Fisher's exact test. Allograft survival times were analyzed by the Kaplan–Meier method, and the allograft survival time for each patient was computed from baseline evaluation to the last follow-up or the primary outcome. Relationships were determined by Pearson's correlation coefficient, and Spearman rho was used for nonparametric correlations. Variables found to affect the outcomes in bivariate analyses were included in the multivariate Cox proportional hazards model. Variables were selected by backward elimination using likelihood ratio tests. All statistical tests were two sided, and a p value of 0.05 or less was considered to be statistically significant.

This study conformed to good medical and laboratory practices and to the recommendations of the *World Medical Association Declaration of Helsinki: Recommendations Guiding Physicians in Biomedical Research Involving Human Subjects* [9]. Our study was approved by the Istanbul Faculty of Medicine Ethical Committee and registered with ClinicalTrials.gov, number NCT02700516.

Results

Overall features

The baseline demographic, clinical and laboratory characteristics of the patients in the recurrent/*de novo* GN ($n = 120$), nonrecurrent GN ($n = 120$) and non-GN ($n = 120$) groups are shown in Table 1. In the recurrent/*de novo* GN group, the median time to diagnosis, defined as histopathological recurrent/*de novo* disease, was 39.5 (IQR: 15–88.75) months. Mean serum creatinine and median level of proteinuria were 1.93 ± 0.94 mg/dL and 3.04 (IQR: 0.5–4.4) g/24 h, respectively, at the time of histopathological diagnosis. There were 29 (24.2%) and 91 (75.8%) patients in early and late recurrent/*de novo* GN groups, respectively. The distribution of various GN according to these subgroups is shown in Table 2.

Table 2 The distribution of patients with early and late recurrent/*de novo* glomerulonephritis ($n = 120$)

	Early recurrent/*de novo* disease		Late recurrent/*de novo* disease		*p* value
	n	%	*n*	%	
Focal segmental glomerulosclerosis	18	31	40	68.9	
IgA nephropathy	2	6.6	28	93.3	
Membranoproliferative glomerulonephritis	2	13.3	13	86.6	
Membranous nephropathy	2	22.2	7	77.7	0.003
Atypical hemolytic uremic syndrome	5	83.3	1	16.6	
C1q nephropathy	0	0	1	100	
Lupus nephritis	0	0	1	100	

Study outcomes

In recurrent/*de novo* GN, nonrecurrent GN and non-GN groups, 54.2% ($n = 65$), 16.7% ($n = 20$) and 8.3% ($n = 10$) of patients reached primary outcome after a median of 96 (IQR: 56–149) months, respectively. The primary outcome, allograft loss was significantly more frequent in the recurrent/*de novo* GN group compared to nonrecurrent GN and non-GN control cases ($p < 0.001$). Allograft loss was also more frequent in the nonrecurrent GN group when compared with non-GN group ($p = 0.05$). Kaplan–Meier analysis revealed that 5-year and 10-year graft survival rates were 80.9% and 55, 96.3 and 85.4%, 97.2 and 95.2% for recurrent/*de novo* GN, nonrecurrent GN and non-GN groups, respectively (Fig. 1). Causes of allograft loss are explained in Table 3.

Sixty-five patients with recurrent/*de novo* GN experienced allograft loss at a median of 21 (IQR: 7–48) months after diagnosis. Among these patients, recurrent or *de novo* diseases were as follows: FSGS ($n = 35$), IgAN ($n = 16$), MPGN ($n = 12$), MN ($n = 1$), and aHUS ($n = 1$). Fifty one of these 65 patients experienced allograft loss due to recurrent disease. In 13 cases, chronic AMR was diagnosed as a result of a following kidney biopsy during the follow-up. One patient passed away with a functioning allograft with recurrent/*de novo* GN. At 10 years of follow-up, allograft loss rates were 54.2% for FSGS, 53.2% for MPGN and 33.4% for IgAN cases by Kaplan–Meier analysis (Fig. 2).

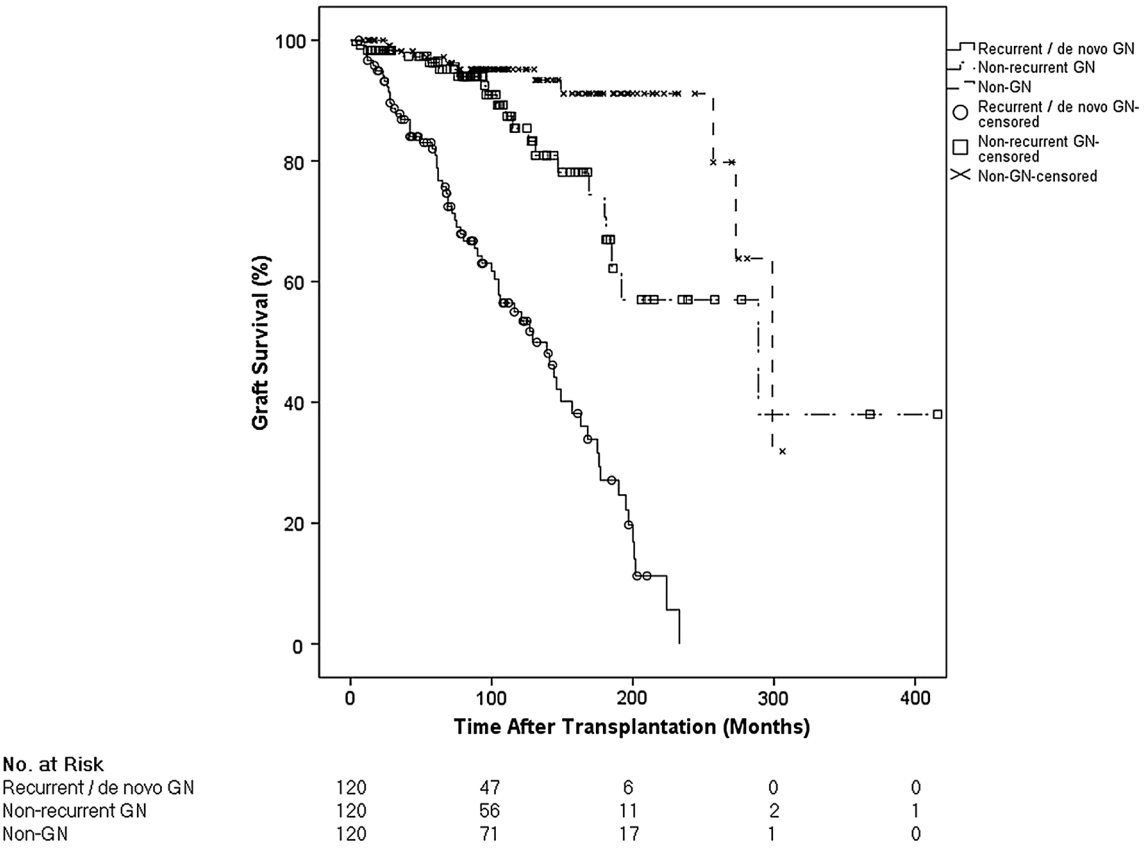

Fig. 1 Kaplan–Meier analysis of graft survival across study groups. Allograft loss was significantly more frequent in the recurrent/*de novo* GN group compared to nonrecurrent GN and non-GN control cases ($p < 0.001$) (GN, glomerulonephritis)

Table 3 Causes of allograft loss across study groups

	Recurrent/*de novo* GN ($n = 120$)	Nonrecurrent GN ($n = 120$)	Non-GN ($n = 120$)
Recurrent/*de novo* disease (%)	51 (78.4%)	0 (0%)	0 (0%)
Allograft rejection (%)	13 (20%)	8 (40%)	4 (40%)
Chronic allograft nephropathy (%)	0 (0%)	9 (45%)	2 (20%)
Death with a functioning allograft (%)	1 (1.5%)	2 (10%)	1 (10%)
Sepsis (%)	0 (0%)	1 (5%)	1 (10%)
BK virus nephropathy (%)	0 (0%)	0 (0%)	1 (10%)
Contrast nephropathy (%)	0 (0%)	0 (0%)	1 (10%)
Total	65	20	10

GN glomerulonephritis

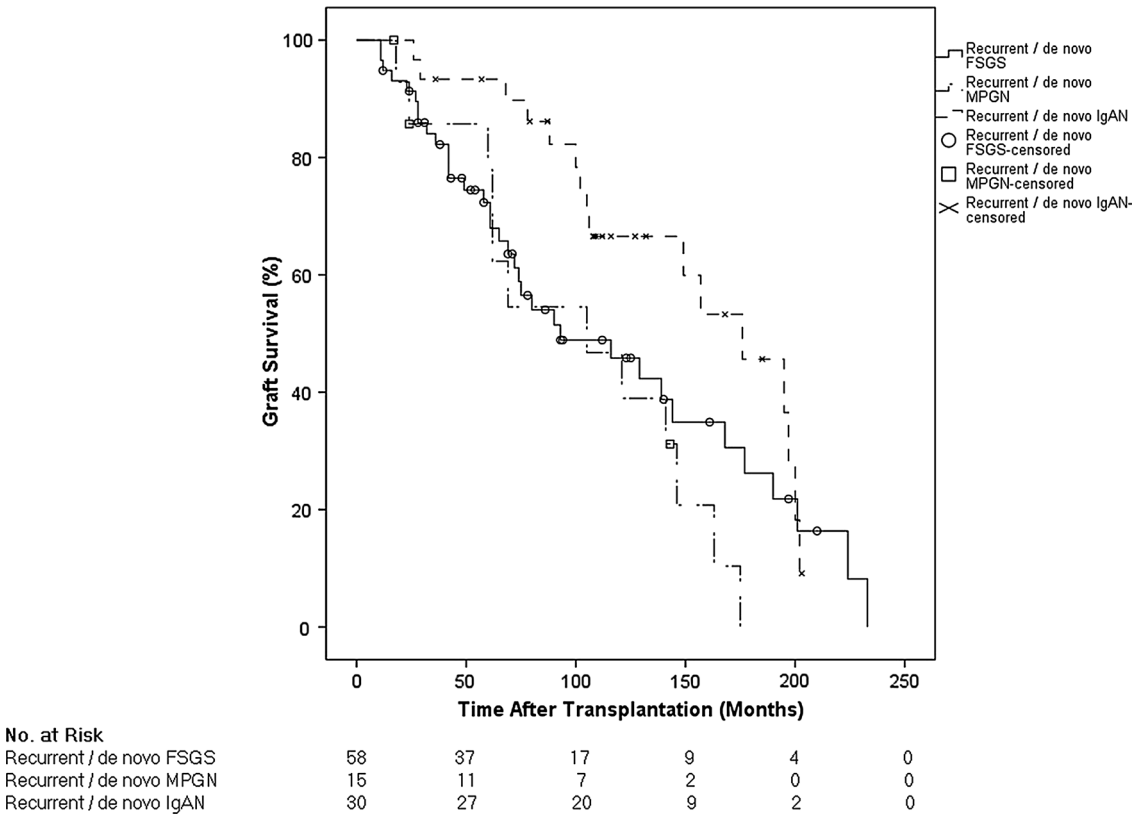

Fig. 2 Kaplan–Meier analysis of graft survival across recurrent/*de novo* GN subgroups. Patients with FSGS and MPGN had worse outcomes as compared to patients with IgAN ($p = 0.045$) (GN, glomerulonephritis; FSGS, focal segmental glomerulosclerosis; MPGN, membranoproliferative glomerulonephritis; IgAN, IgA nephropathy)

Overall 23 (35.4%) of 65 patients lost their grafts within one year after diagnosis of recurrent/*de novo* GN. Allograft loss rates were similar in patients with early (16/29, 55.2%) and late post-transplant GN (49/91, 53.8%) ($p = 0.901$).

Secondary outcome of the study, biopsy-confirmed graft rejection rate was noted in 48 patients. Among these, 21 developed acute cellular rejection, while 27 had AMR (2 acute and 25 chronic). Graft rejection rate was significantly higher in the recurrent/*de novo* GN group ($n = 25$, 20.8%) compared to non-GN ($n = 8$, 6.7%) group ($p = 0.001$). Thirteen of these 25 patients in the recurrent/*de novo* GN group suffered from graft loss, and remaining grafts continued to be functional during the follow-up. There was no statistically significant difference in rejection rates between nonrecurrent GN ($n = 15$, 12.5%) and non-GN groups ($p = 0.125$). Higher rejection rate in the recurrent/*de novo* GN group did not reach to statistical

significance when compared with the rejection rate in the nonrecurrent GN group ($p = 0.083$).

Immunosuppression

Although all patients were started on a triple maintenance immunosuppressive regimen, alterations during the follow-up period led to significant differences in treatment protocols among the study and control groups. The number of patients maintained with double therapies was significantly higher in the recurrent/*de novo* GN group ($n = 24$, 20%) compared to nonrecurrent GN ($n = 7$, 5.8%) and non-GN ($n = 11$, 9.2%) groups ($p = 0.002$); however, there was no significant difference between nonrecurrent GN and non-GN groups ($p = 0.327$). Detailed features with regard to immunosuppressive regimens are shown in Table 4. There were no differences regarding immunosuppressive regimens between patients suffered from graft rejection and patients without rejection ($p = 0.051$). The rates of patients using triple therapies in groups of patients suffered from rejection and patients without rejection were 93.8% (45/48) and 87.5% (273/312), respectively ($p = 0.209$).

Predictors of outcomes

Bivariate correlation analysis of all patients' characteristics revealed that graft loss was associated with age ($r = 0.122$, $p = 0.020$), history of biopsy-confirmed rejection ($r = 0.229$, $p < 0.001$) and post-transplant recurrent or *de novo* disease ($r = 0.446$, $p < 0.001$). In multivariate Cox regression analysis, diagnosis of recurrent/*de novo* GN (HR: 15.767, 95% CI 6.081–40.877, $p < 0.001$), nonrecurrent GN (HR: 2.942, 95% CI 1.055–8.207, $p = 0.039$) and biopsy-confirmed rejection (HR: 2.649, 95% CI 1.438–4.877, $p = 0.002$) predicted primary outcome, whereas age did not.

In recurrent/*de novo* GN group, bivariate correlation analysis revealed that post-transplant MPGN ($r = 0.196$, $p = 0.032$), serum creatinine ($r = 0.236$, $p = 0.015$) and albumin levels ($r = -0.389$, $p < 0.001$) at the time of diagnosis were significantly associated with allograft loss. In multivariate Cox regression analysis, only serum albumin levels at the time of diagnosis (HR: 0.526, 95% CI 0.322–0.861, $p < 0.001$) predicted primary outcome.

Bivariate correlation analysis of all patients' characteristics revealed that only recurrent/*de novo* GN ($r = 0.170$, $p = 0.001$) was significantly associated with secondary outcome (biopsy-confirmed rejection).

Discussion

One of the major findings of the present study is that recurrent/*de novo* GN significantly contributed to allograft dysfunction and subsequent graft loss, and, the worst allograft survival was found in this group. Moreover, allograft loss is markedly increased in patients with post-transplant recurrent/*de novo* MPGN and FSGS. Previous reports also suggested that recurrent/*de novo* GN was associated with a greater incidence of graft dysfunction and graft failure over the long term [1, 5].

Additionally, the present study revealed that 35.4% of the patients suffered from allograft loss within one year of diagnosis, thus demonstrating the profound impact of this condition on graft outcome. Another explanation for this adverse outcome may be related to the timing of allograft biopsy which could be performed late in the disease process.

Table 4 Immunosuppressive treatment regimens of three groups

	Recurrent/*de novo* GN ($n = 120$)	Nonrecurrent GN ($n = 120$)	Non-GN ($n = 120$)	p value
Triple therapies	96 (80%)	113 (94%)	109 (91%)	0.002
Tac + MMF + prednisolone	48	54	47	
CsA + MMF + prednisolone	22	31	31	
CsA + Aza + prednisolone	11	20	18	
mTORi + MMF + prednisolone	9	3	9	
Tac + Aza + prednisolone	5	5	4	
Tac + mTORi + prednisolone	1	0	0	
Double therapies	24 (20%)	7 (6%)	11 (9%)	0.002
MMF + prednisolone	19	7	7	
Aza + prednisolone	4	0	2	
CsA + prednisolone	0	0	2	
Tac + MMF	1	0	0	

GN glomerulonephritis, *Tac* tacrolimus, *MMF* mycophenolate mofetil, *CsA* cyclosporine, *Aza* azathioprine, *mTORi* mTOR inhibitors

The factors associated with the prognosis of patients within recurrent/*de novo* GN group were also studied in this study. Post-transplant MPGN and FSGS have the worst prognosis. This result confirms previous reports underlining that recurrent MPGN and FSGS are associated with a greater risk of graft dysfunction and graft failure in the long term [1, 10]. Particularly, MPGN was the most important risk factor for graft loss in recurrent/*de novo* GN group. Although proteinuria is a known risk factor for the progression of GN [11–13], after a multivariate Cox regression analysis, only serum albumin levels at the time of post-transplant GN diagnosis predicted allograft loss. The baseline proteinuria at the same time was not directly associated with allograft outcomes. Thus, as a negative acute phase reactant, serum albumin levels may additionally indicate the activity of disease process. There are several important questions regarding the role of proteinuria at the time of biopsy in the prognosis of glomerular diseases. Some studies have proven that proteinuria levels at diagnosis are often not a predictor of the outcome according to a Cox regression analysis; instead, these studies suggested that time-averaged proteinuria levels which represent the average level of proteinuria during the follow-up and proteinuria levels at 1 year or later may better indicate the prognosis [11–13]. Serum albumin levels may be a better marker of time-averaged proteinuria and/or inflammatory disease activation, thus significantly predicting allograft survival.

We were particularly interested in investigating the factors associated with post-transplant GN recurrence. In this study, a higher risk of post-transplant recurrent/*de novo* GN was found in recipients maintained on lower immunosuppression regimen with double therapies compared with triple immunosuppressive regimens. Lower level of immunosuppression, particularly steroid avoidance as a risk factor for recurrent GN, has also been described in previous studies [14, 15]. However, steroid avoidance more than six months following transplantation appeared to be associated with a similar low risk of IgAN recurrence as those on steroid maintenance treatment [15]. Potent immunosuppressive and/or antiproteinuric properties of triple immunosuppressive regimens may cause these superior results [16, 17].

Another factor which may affect post-transplant GN recurrence is the source of donor. Several previous reports did [18–21] or did not [22, 23] find living-related transplantation as a risk factor for GN recurrence. Further risk factors for FSGS recurrence are younger age, rapid progression to ESRD from the onset of proteinuria, collapsing variant of FSGS and previous transplant failure as a result of recurrent FSGS [24]. Our three study groups were matched regarding age, gender and donor source in order to investigate the effects of post-transplant recurrence on long-term graft outcomes. Thus, interpreting the study results and drawing conclusions about the effects of these matched factors on recurrence of primary GN is not feasible.

There have been numerous studies of post-transplant recurrent GN, but very few have had an appropriate control group. The strength of this parallel-group retrospective study is that recurrent/*de novo* GN, nonrecurrent GN and non-GN groups were matched and this led us to evaluate the influence of post-transplant GN on allograft functions. On the other hand, our study suffered from several limitations. The data were retrieved from a long period of retrospective observation. In addition, details for post-transplant anti-HLA antibodies were not available in all patients. As protocol biopsies were not performed in the study, we could not definitely exclude subclinical recurrent/*de novo* disease in nonrecurrent GN group.

In conclusion, post-transplant recurrent/*de novo* GN, particularly MPGN and FSGS, is an important cause of allograft loss. Serum albumin levels may be a better marker of predicting allograft survival and should be taken into consideration at any time if patients develop an early onset of post-transplant hematuria or proteinuria.

Compliance with ethical standards

Conflict of interest The authors do not have any conflicts of interest.

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