Prevalence and Prognosis of Post-transplant Glomerulonephritis in Kidney Transplant Biopsies, A Single-Center Report

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Original Paper

Introduction. Recurrence of glomerulonephritis (GN) after kidney transplant (Tx) may be associated with allograft loss. This study aimed to evaluate the frequency and prognosis of de novo or recurrent post-Tx GN.

Methods. We reviewed 1305 kidney Tx biopsy samples obtained between 2006 and 2020. The biopsy specimens were divided into post-Tx GN (recurrent or de novo) and control groups (i.e., no detectable GN in biopsy). Demographic and baseline characteristics of the patients and kidney survival rates were analyzed.

Results. From 1305 kidney transplanted biopsies, 350 repeated biopsies for transplant rejection were excluded. Among 955 analyzed biopsies, (mean age: 40.4 ± 13.48 years, mean transplantation duration: 4.54 ± 3.98 years, 74.6% males), the frequency of GN was 10.78%. The most common recurrent post-Tx GN was IgA nephropathy (22.3%), followed by secondary focal segmental glomerulonephritis (FSGS, 19.4%), primary FSGS (19.4%), and membranous glomerulonephritis (17.5%). In the post-Tx GN group, the mean serum creatinine and proteinuria were 3.28 ± 1.97 mg/dL and 2730 ± 1244 mg/d at the biopsy time and 4.14 ± 1.86 mg/dL and 2020 ± 1048 mg/d, at the end of the study. There was a significant relationship between baseline serum creatinine and graft loss ($P < .001$). One-, five-, and ten-year graft survival rates were 97%, 81%, and 63% in the post-Tx GN, and 100%, 92%, and 59% in the control group. The median time to graft loss after biopsy, (graft survival after biopsy), was significantly lower in the post-Tx GN group ($P < .000$). The other accompanying factors had no significant impact on graft survival.

Conclusion. The median time to graft loss after biopsy was significantly lower in post-Tx GN. Baseline serum creatinine had a significant association with graft loss. Optimal management of recurrent or de novo GN should be a main focus of post-transplant care.

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INTRODUCTION

Glomerulonephritis (GN) is one of the most common causes of end-stage kidney disease worldwide.[1] In addition, GN may involve the kidney allograft and recurrent and de novo GN are detected in 3 to 18.5% of the patients receiving a kidney transplant, which may lead to a 30% decrease in the 5-year graft survival rate.[2,3] The

probability of recurrent GN has been reported between 2.5% to 50% in different studies.[4–6] This disparity may be linked to several factors such as variable follow-up periods, inconsistent follow-up contact, incomplete biopsy data, different diagnostic criteria and demographic characteristics, and discordant reporting practices.[7]

To date, the prevalence, etiology, pathogenesis, prognosis, and mean interval to post-transplant (post-Tx) GN are not fully elucidated. The treatment and outcome of post-Tx GN are also somewhat unknown. Timely diagnosis and treatment of post-Tx GN may play an important role in increasing allograft and patient survival.

Multiple factors including male gender, younger recipient age, living-related donors, and compatibility HLA matching have been indicated to be associated with disease recurrence in the transplant recipient, but the risk factors are not well identified.[8–10] Recurrence of GN is associated with adverse allograft outcomes including allograft loss.[8,11,12] Management and diagnosis of recurrent post-Tx GN are important to improve long-term kidney allograft outcome and also provide a unique opportunity to explore the pathogenesis of native kidney disease.[13] Understanding the pathogenesis of recurrent GN and implementing protocols for the prediction, prevention, and treatment of recurrent GN can strongly affect the kidney transplant outcome.

In this study, we investigated the prevalence, risk factors, and prognosis of post-Tx GN in biopsy specimens performed on transplanted kidneys in Hasheminejad Kidney Center, Tehran, the main referral kidney center of the country, during a 16 years period.

MATERIALS AND METHODS

Population Study

In this retrospective cohort study, the target population included all kidney Tx biopsies in Hasheminejad Kidney Center, performed between 2006 and 2020. The biopsy specimens demonstrating recurrent or de novo GN were classified as post-Tx GN group. The remainder of the biopsy specimens were labeled as the control group.

Demographic data, the underlying disease leading to kidney transplantation, duration of dialysis before Tx, type of Tx donor (living vs. cadaveric), serum creatinine level and the amount of daily proteinuria at the time of biopsy (baseline) and at the end of the study, the immunosuppressive protocols, the last patient status (survived vs. deceased) and graft status (functioning vs. on kidney replacement therapy) and the results of kidney pathology were collected in questionnaires.

Data Analysis

Qualitative data were presented as mean ± SD or median (interquartile range) (IQR), where appropriate. Kolmogronov-Smirnov, independent sample t-test, ANOVA, Chi-square, and ROC-curve were used for analysis. The frequency of GN and patient and allograft outcomes were evaluated by using IBM SPSS Statistics (Version 16).

Ethics

This study was carried out under the supervision of the research ethics committee of the Iran University of Medical (IR.IUMS.FMD.REC.1400.126). Patients' consent was taken by phone call.

RESULTS

A total of 1305 biopsy specimens were obtained in the determined time frame. The re-biopsy specimens with no new findings other than their preceding counterparts (n = 350) were excluded.

The frequency of post-Tx GN in the examined 955 biopsy specimens was 10.78% (103 cases).

In 103 post-Tx GN, the mean age of patients was 43.08 ± 12.59 years, and 79% were male. The mean duration of Tx at the time of biopsy was 7.48 ± 70.71 years. The mean post-Tx creatinine was 1.3 ± 0.3 mg/dL. The mean baseline serum creatinine and proteinuria level (at the time of biopsy) were 3.28 ± 1.41mg/dL and 2730 ± 1244 mg/d, respectively. At the last follow-up, the mean serum creatinine level and proteinuria were 4.14 ± 2.68 mg/dL, 2020 ± 1048 mg/d; respectively.

In the control group composed of 79.9% males and 20.10% females, the mean age was 40.16 ± 13.56 years. The mean creatinine of these patients at the biopsy time was 3.17 ± 1.78 mg/dL and the mean creatinine at the end of the study was 2.91 ± 2.35 mg /dL.

There was no significant difference between sex distribution of control and post-Tx GN group (P = .27), and no significant difference in mean age (P = .38) and serum creatinine level at time of biopsy (P = .52). There was significant difference

in serum creatinine level between the two groups in last follow- up ($P < .01$) (Table 1).

Based on the ROC curve analysis, the mean level of baseline serum creatinine of 2.8 mg/dL was defined as the cutoff which could predict graft loss with a sensitivity of 43% and specificity of 40% in patients with post-Tx GN ($P = .06$) (AUC = 0.38, CI 95%: 0.25 to 0.50).

In the study population of 955 subjects, 65% had living and 35% had cadaveric donors, compared to 72,8% and 27.2% in the post Tx GN patients, respectively ($P > 0.05$).

Among patients diagnosed with post-Tx GN, the underlying disease before kidney transplantation was unknown in 51 patients (55.49%) and kidney biopsy had been performed in only 25 patients (24.27%), however, in 91% of patients nephrotic syndrome and proteinuria were reported in past medical history. In the patients who had a previous kidney biopsy, 82.7% showed recurrence of primary GN and 17.3% had de novo GN.

Table 2. Frequency of Various Underlying Diseases in Patients with Post-TX GN

Primary Disease	Frequency (Percent)
Unknown	51 (49.5)
Hypertension	8 (7.8)
Membranous Glomerulonephritis	7 (6.8)
IgA Nephropathy	7 (6.8)
Hereditary	5 (4.9)
Stone-Hypertension	4 (3.9)
FSGS	4 (3.9)
ADPKD	4 (3.9)
Lupus Nephritis	3 (2.9)
Diabetes Mellitus	3 (2.9)
Congenital	2 (1.9)
Proteinuria	1 (1)
Amyloidosis	1 (1)
RCC	1 (1)
Interstitial Nephritis	1 (1)
MPGN	1 (1)
Total	103 (100)

Abbreviations: ADPKD, autosomal dominant polycystic kidney disease; MPGN, membranoproliferative glomerulonephritis; RCC, renal cell carcinoma.

Table 1. Demographic Characteristics and Mean Serum Creatinine in the Case and Control Groups

	Sex (male)	Age (mean ± SD)	Serum Creatinine at Biopsy (mg/dL) (mean ± SD)	Serum Creatinine at Last Follow-up (mg/dL) (mean ± SD)	Proteinuria at Biopsy (mg/d) (mean ± SD)	Proteinuria at Biopsy Last Follow-up (mg/d) (mean ± SD)
Control (852)	79.9%	40.16 ± 13.56	3.17 ± 1.78	2.91 ± 2.35[d]	– [a]	– [a]
Case (103)	79%	43.08 ± 12.62	3.28 ± 1.41[b]	4.14 ± 2.68[b,d]	2730 ± 1244[c]	2020 ± 1048[c]

[a]not applicable
[b]$P < .001$
[c]$P < .001$
[d]$P < .001$

Table 3. Frequency of Post-Tx GN by the Underlying Disease

Recurrent GN	Primary Disease													
	IgA Nephropathy	Membranous GN	Unknown	FSGS	HTN	DM	MGN	Congenital	RCC	ADPKD	MPGN	Interstital Nephratis	Lupus Nephritis	Amyloidosis
IgA Nephropathy	7	1	13	1	1	0	0	0	0	0	0	0	0	0
Secondary FSGS	0	0	9	4	4	1	1	1	0	0	0	0	0	0
Primary FSGS	1	0	13	3	1	0	0	0	1	1	0	0	0	0
Membranous GN	1	5	6	0	1	0	0	1	0	2	1	1	0	0
Crescentic GN	0	0	1	0	5	0	0	0	0	0	0	0	1	0
Non-specific GN	0	0	2	0	2	0	0	1	0	1	0	0	0	0
Glomerulosclerosis	0	0	1	0	0	2	0	0	0	0	0	0	0	0
Lupus Nephritis	0	0	0	0	0	0	0	0	0	0	0	0	2	0
Amyloidosis	0	0	0	0	1	0	0	0	0	0	0	0	0	1
MPGN	0	0	0	0	0	1	0	0	0	0	0	0	0	0
ICGN	0	0	1	0	0	0	0	0	0	0	0	0	0	0

Abbreviations: ADPKD, autosomal dominant polycystic kidney disease; DM, diabetes mellitus; FSGS, focal segmental glomerulosclerosis, GN, glomerulonephritis; HTN, hypertension; ICGN, immune complex mediated glomerulonephritis; MPGN, membranoproliferative glomerulonephritis; RCC, renal cell carcinoma.

Table 4. Patient Characteristics of the Post-Tx GN Group

Diagnosis (no.)	Mean Age (years) (± SD)	Mean Duration of Transplantation (± SD) (months)	Mean Duration of Dialysis (± SD) (months)	Mean Creatinine After Transplantation (± SD) (mg/dL)	Mean Creatinine Before Biopsy (± SD) (mg/dL)	Mean Proteinuria Before Biopsy (± SD) (mg/d)	Mean Proteinuria at the End of Study (± SD) (mg/d)	Mean Creatinine at the End of the Study (± SD) (mg/dL)
IgA Nephropathy (23)	46.61 (± 12.33)	120.09 (± 72.27)	10.115 (± 10.12)	1.357 (± 0.24)	3.33 (± 3.02)	2358.91 (± 1751.72)	1173.758 (± 804.35)	2.74 (± 1.82)
Primary FSGS (20)	40.85 (± 12.70)	35.40 (± 42.26)	17.30 (± 9.20)	1.520 (± 0.47)	4.51 (± 4.11)	4609.30 (± 7257.98)	1800.00 (± 2287.03)	4.82 (± 3.07)
Secondary FSGS (20)	45.45 (± 14.40)	103.95 (± 88.66)	15.45 (± 8.85)	1.300 (± 0.21)	3.865 (± 1.56)	1845.60 (± 1854.76)	1145.50 (± 1431.91)	5.44 (± 2.63)
MGN (18)	40.22 (± 11.57)	76.33 (± 50.28)	14.56 (± 11.39)	1.333 (± 0.30)	2.26 (± 0.75)	2804.33 (± 1728.07)	1200.00 (± 1230.50)	2.98 (± 2.75)
Crescentic GN (7)	33.86 (± 12.56)	70.29 (± 54.74)	12.14 (± 7.13)	1.300 (± . 0.27)	5.53 (± 5.89)	2430.14 (± 2367.66)	1714.29 (± 1564.64)	3.90 (± 2.08)
Non-specific GN (6)	45.00 (± 8.27)	118.00 (± 70.96)	24.00 (± 7.59)	1.233 (± 0.24)	3.63 (± 0.76)	3928.33 (± 2739.76)	933.33 (± 1053.88)	4.05 (± 1.79)
Glomerulosclerosis (3)	48.00 (± 14.73)	92.00 (± 55.43)	26.00 (± 9.17)	1.400 (± 0.35)	2.70 (± 1.73)	2794.97 (± 2570.84)	1400.00 (± 1276.72)	5.93 (± 3.10)
Lupus Nephritis (2)	42.00 (± 5.66)	150.00 (± 8.49)	12*	1.300 (± 0.36)	2.80 (± 0.14)	5314.50 (± 430.63)	1550.00 (± 2192.03)	1.4*
Amyloidosis (2)	44.50 (± 12.02)	108.00 (± 67.88)	39.00 (± 46.67)	1.350 (± 0.07)	3.85 (± 0.36)	600.00 (± 848.53)	600.00 (± 848.53)	6.50 (± 2.12)
Immune Complex Mediated GN (1)	32.00*	168.00*	24.00*	1.200*	5.00*	3700.00*	4000.00*	9.00*
MPGN (1)	57.00*	12.00*	18.00*	2371*	2.100*	_	2100.00*	4.00*
Total Post-Tx GN (103)	43.08 (± 12.56)	88.3 (± 70.71)	17.51 (± 11.06)	1.3 (± 0.3)	3.28 (± 1.41)	2730 (± 1244)	2020 (± 1048)	4.14 (± 2.68)
P	.254	.015	.001	.627	.050	.524	.499	.044

*Standard deviation not calculated because of low number of cases.

In the total study population, 103 kidney biopsies were diagnosed as post-Tx GN including 23 IgA Nephropathy (22.3%), 20 secondary FSGS (19.4%), 20 primary FSGS (19.4%), 18 membranous glomerulonephritis (17.5%), 7 crescentic GN (6.8%), 6 non-specific GN (5.8%), 3 glomerulosclerosis (2.9%), 2 lupus nephritis (1.9%), 2 amyloidosis (1.9%), 1 membranoproliferative glomerulonephritis (1%) and 1 immune complex-mediated GN (1%). The most common treatment regimen recorded during the biopsy period was cyclosporine, mycophenolate, and prednisolone. As shown in Table 2, if there was a difference between the primary and the recurrent disease, it was classified as de novo GN (5 patients (4.8%) (Tables 3, 4).

Table 4 shows the demographic and laboratory characteristics of the patients at the time of biopsy and end of follow-up.

At the end of the study, 65% of all studied (995) patients had functional grafts, 19.1% lost their allografts and underwent hemodialysis and 15.8% died.

Among the 103 patients with the diagnosis of post-Tx GN, 48 patients (46.6%) had functional grafts with GFR above 15 ml/min, 36 patients (35%) had graft loss, who underwent dialysis, and 19 patients (18.4%) died due to infection, heart disease, cancer, and other causes. In 852 control patients, 588 (65.5%) had functional graft with GFR above 15 ml/min, 159 patients (18.7%) had graft loss, who underwent dialysis, and 135 patients (15.8%) died. So the rate of graft loss was significantly higher in post- Tx GN group ($P < .0003$) (Table 5).

Mean graft survival after biopsy was 5.84 ± 0.12 years, with 3.13 ± 0.33 years in post-Tx GN and 6.11 ± 0.12 years in control groups ($P < .001$). Mean patient survival after biopsy was 7.37 ± 0.11 years, with 5.24 ± 0.47 years in post-Tx GN and 7.48 ± 0.11 years in control group ($P < .001$).

Overall mean patient survival was 18.67 ± 0.99 years, with 21.70 ± 1.58 years in post-Tx GN and

Table 5. Patient Outcome

Outcome	Dead no (%)	Graft Loss no (%)	Live with Functioning Graft no (%)
Control (852)	135 (15.8)	159 (18.7)	588 (65.5)
Case (103)	19 (18.4)	36 (35)	48 (46.6)

Note: Graft loss was significantly higher in post-Tx GN group ($P < .001$).

15.06 ± 0.38 years in control groups (P = .093) Mean graft survival was 12.89 ± 0.41 years, with 13.70 ± 1.51 months in post-Tx GN and 11.98 ± 0.27 years in control groups (P = .412)

In the post-Tx GN group one-, five- and eight-year patient survival rates after biopsy were 91, 53 and 26%; respectively. In the control group, the corresponding rates were 95, 83, and 66%, respectively.

In the post-Tx GN group one-, five- and eight-year graft survival rates after biopsy were 72, 19, and 4%, respectively. In the control group, the corresponding rates were 90, 67, and 40%, respectively.

Regarding total patient survival rates, in the post-Tx GN group one, five-, and ten-year patient survival rates were 98, 94, and 83%, respectively. In the control group, the corresponding rates were 100, 95, and 79%, respectively.

In the post-Tx GN group one, five-, and ten-year total graft survival rates were 97, 81, and 63%, respectively. In the control group, the corresponding rates were 100, 92, and 59%, respectively.

Median time to graft loss (total graft survival) was not different between the post-Tx GN and control groups (P = .412), however median time to graft loss after biopsy, (graft survival after biopsy), was significantly lower in the post-Tx GN group (P < .001) (Figures 1, 2).

Cox regression model analysis of the accompanying factors, including diabetes mellitus (P = .949, 95% CI: 0.793 to 1.282), hypertension (P = .829, 95% CI: 0.836 to 1.250), smoking (P = .686, 95% CI: 0.686 to 1.281), family history of kidney disease (P = .916, 95% CI: 0.747 to 1.123), and gender (P = .799, 95% CI: 0.806 to 1.323) showed none of them as a risk factor for graft survival.

DISCUSSION

In this retrospective cohort study, a total of 1305 kidney transplanted biopsies were examined, of which, 955 biopsies were analyzed after exclusion of 350 repeated biopsies specimens due to recurrent rejection

We found 103 (10.78%) cases of post-Tx GN in a duration of 103 months after transplantation, among all biopsied patients. Similarly, An *et al.* in a cohort of 996 kidney transplant patients, reported 9.7% and 17% of post-Tx GN recurrence after 5 and 10 years, respectively.[14] A retrospective study of ESKD patients also reported a similar percentage of 10.3% GN recurrence rate in kidney transplant recipients.[15]

In our study among 103 recipients with biopsy-

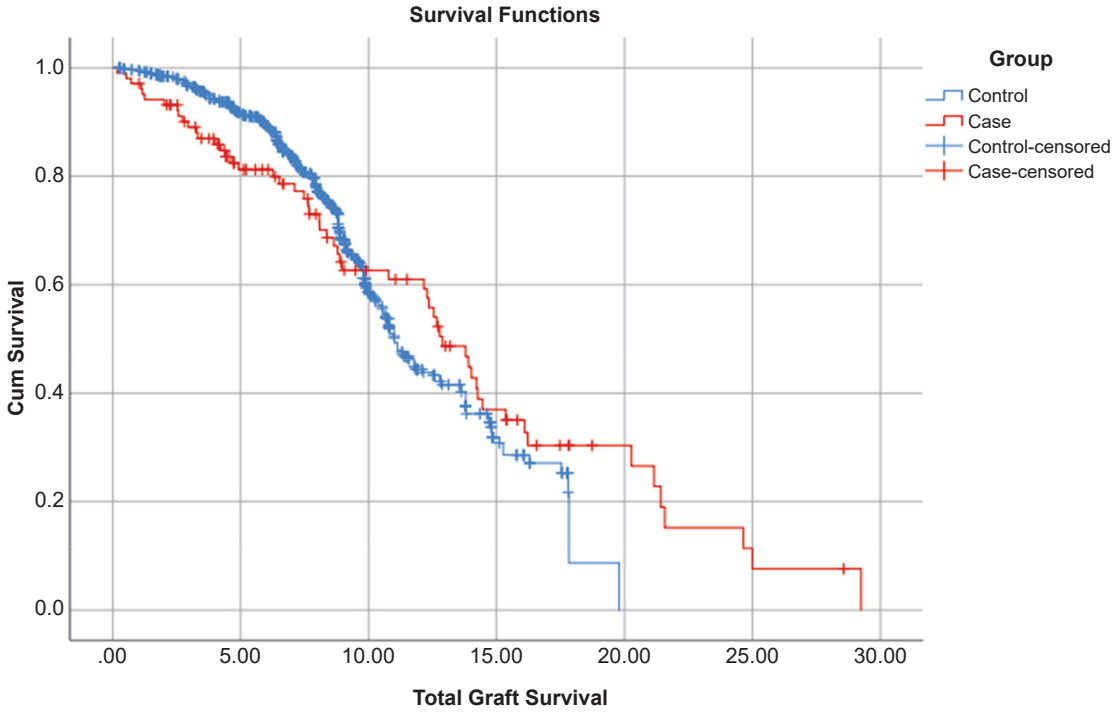

Figure 1. Kaplan-Meier Curve Demonstrating Median Time to Graft Loss (Total Graft Survival) Between Post-transplant Glomerulonephritis (Case) and Control Groups (P = .412).

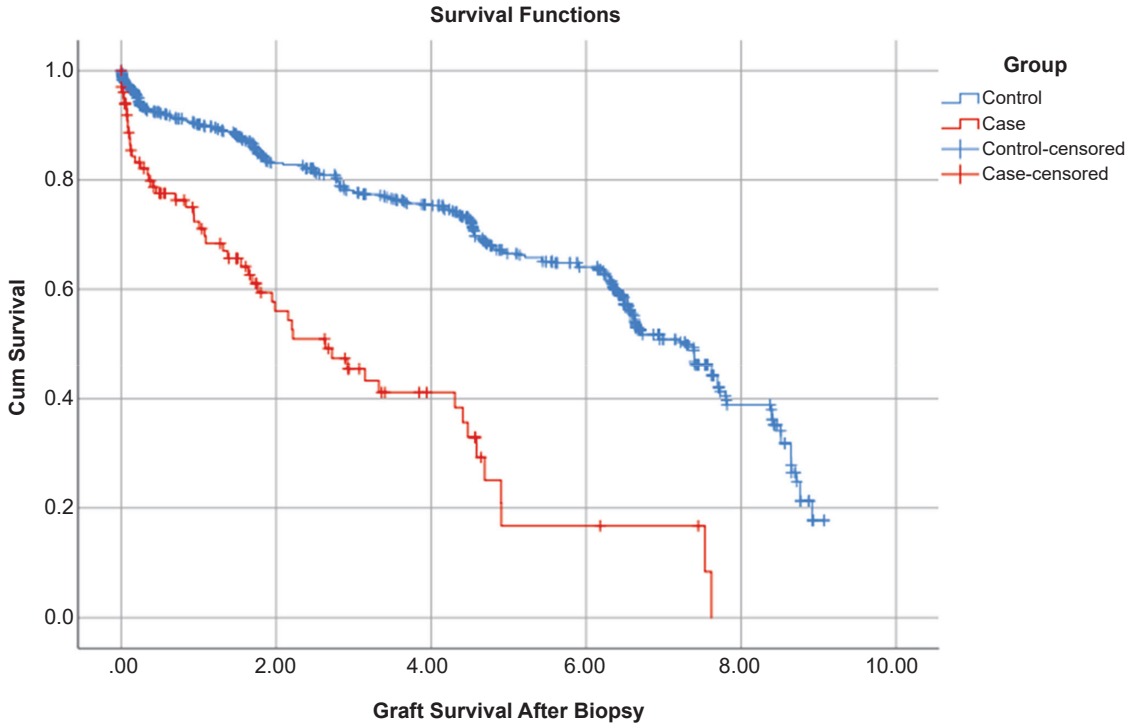

Figure 2. Kaplan-Meier Curve Demonstrating Median Time to Graft Loss After Biopsy (Graft Survival After Biopsy) Between Post-transplant Glomerulonephritis (Case) and Control Groups ($P < .001$).

proven GN, IgA nephropathy was the leading cause of GN, followed by primary and secondary FSGS each in 19.4% of patients, while in the study by Takahiro Shinzato *et al.* secondary FSGS was found in 14.5% of cases.[16] Secondary FSGS after kidney transplantation can be induced by progressive obesity, as manifested by glomerular hypertrophy, as well as kidney hypertrophy, younger age of the recipients and older donor.[17,18] Allen *et al.*, in a retrospective study of 4637 kidney transplant recipients, reported a similar distribution of GN subtypes.[15] Cheigh *et al.* reported a comparable incidence of post-Tx FSGS in 154 renal allografts.[19] However, different rates of MPGN are reported among patients with post-Tx GN. While Lorenz *et al.* reported a similar rate of MPGN with our study (1.6 and 0.9%, respectively), MPGN was found more frequently in the study by Allen *et al.* (5.7%).[15,20] Recurrence of MPGN is seen in 27 to 65% of cases after renal transplant resulting in graft loss in up to 50% of cases. The recurrence rate seems to be even higher in the second transplant.[18]

Graft failure occurred in 35% of transplant recipients with post-Tx GN in our study. However, Allen *et al.* reported graft failure in 44.2% of post-Tx GN cases.[15] Nonetheless, studies agree that post-Tx GN may lead to a two-fold increase in all-cause mortality.[15,18]

Among recipients with post-Tx GN examined in our study, the mean serum creatinine and proteinuria before biopsy were 3.27 ± 1.97 mg/dL and 2730 ± 1244 mg/d, respectively. In a study of 327 kidney transplant recipients, Hohage *et al.* reported proteinuria of 0.25 to 1.0 g/d in 25.5% of their patients at 6 months after transplant.[21] In this study proteinuria was associated with a marked reduction in 5-year graft survival (58.9 vs. 85.6% in proteinuric compared to non-proteinuric recipients). On the other hand, the authors showed that age, gender, hemodialysis duration and cold ischemia time were not correlated with proteinuria. Given the evidence provided by Hohage *et al,* it is safe to conclude that even non-nephrotic proteinuria may exert a deleterious effect on long-term graft survival.[21]

Briganti *et al.* studied the impact of recurrent post-Tx GN on graft failure. Among 1505 patients, recurrent post-Tx GN led to graft failure in 52 patients, with a 10-year incidence of 8.4%. However, post-Tx GN and other causes of graft failure had a comparable rate of graft failure after 10 years. The authors concluded that although

post-Tx significantly affects graft survival, it may not necessitate alterations in kidney transplant prioritization.[8] Similarly, Kessler *et al.* studied 84 patients receiving kidney transplants due to IgA nephropathy or Henoch-Schönlein purpura nephropathy, and concluded that recurrent post-Tx GN may not impact 8-year graft survival.[22]

In addition to the above studies, long- term survival rates reported in the published literature have only demonstrated negligible differences between the post-Tx GN and control groups. Survival analyzes conducted regarding different time frames suggest that the findings of our study are in alignment with previous reports of survival rates. Nevertheless, graft survival rates of GN patients seems to be following an improving trend in the past few decades. Hariharan *et al.* reported one- and five-year graft survival rates of 88 and 74%, respectively in the post-Tx GN patients; the more recent studies, including ours, do not report improved survival rates. In this regard, better management of the recurrent disease and more efficacious utilization of renin-angiotensin-aldosterone axis inhibitors seem to have contributed to favorable results.[11,12,23] Likewise, Requião-Moura *et al.* have stated that renin-angiotensin-aldosterone axis inhibitors are able to improve graft outcomes independent of other variables.[24]

An *et al.* reported tacrolimus and bazilximab to be associated with post-Tx GN more frequently.[14]

Despite advancements in the epidemiology, pathogenesis, and classification of post-Tx GN, the proper approach to achieve improved survival outcomes has yet to be established. Such consensus requires a global effort to archive the clinical manifestations, pathological findings, modes of treatment, and outcomes of post-Tx GN. As the Kidney Disease Improving Global Outcomes (KDIGO) protocol for post-Tx GN recommends, screening is the basis of post-Tx GN management.[25] Documentation of routine screening data in a global database may provide a fertile ground for future research which are necessary to reach a consensus on an efficient approach to improve post-Tx GN outcomes.

Electron microscopy (EM) is a helpful complementary study in a number of post Tx GN cases such as differentiation of C3 glomerulopathy subtypes.[26] In our study EM was not available for evaluation of kidney biopsies, that would be a limitation in cases without definite diagnosis.

Our study showed that a wide range of glomerulonephritides, either relapsing or de novo may recur after kidney transplantation which reduces the lifespan of the graft.

CONCLUSION

In conclusion our study showed that a wide range of glomerulonephritides, either relapsing or de novo, may recur after kidney transplantation, which is associated with a decrease in graft survival. Close monitoring of the kidney function and development of proteinuria and timely biopsy may help to implement therapeutic measures to control post- Tx GN and prevent graft loss.

ABBREVIATIONS

GN: Glomerulonephritis
ESKD: End-stage kidney disease
MPGN: Membranoproliferative Glomerulonephritis
HLA: Human leukocyte antigen
GFR: Glomerular filtration rate
FSGS: Focal segmental glomerulosclerosis
IgA: Immunoglobulin A
MGN: Membranous glomerulonephritis
ICGN: Immune complex GN

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