PRIMARY GLOMERULONEPHRITIS: A SINGLE-CENTER RETROSPECTIVE EXPERIENCE

YAVUZ AYAR[*], ALPARSLAN ERSOY[*], FATMA EZGI CAN[**], MUSTAFA GÜLLÜLÜ[*], ISMAIL BAYRAKCI[***], DILAY DEMIRAYAK[***], HAKAN DÜGER[***], TUGBA OCAK[***], AYSEGÜL ORUC[*], ABDÜLMECIT YILDIZ[*], BAYRAM KORKUT[***], AHMET BILGEHAN SAHIN[***], NIHAL YÜCEL CAMCI[***], BERNA AYTAC VURUSKAN[****]

*Uludag University Medical Faculty, Internal Medicine, Department of Nephrology, Bursa, Turkey - **Uludag University Medical Faculty, Department of Biostatistics, Bursa, Turkey - ***Uludag University Medical Faculty, Internal Medicine, Bursa, Turkey - ****Uludag University Department of Pathology

ABSTRACT

***Aim**: Primary glomerular diseases (PGD) are seen frequently. Age, gender, geographical characteristics and genetic affect the distribution of the disease. Despite the treatment, a part of the disease may progress to end-stage renal disease. We evaluated the PGD patients and retrospectively regarding to their clinical and histopathological characteristics.*

***Materials and methods**: In this study PGD patients, who have had renal biopsy between 1st January 2009 and 31st December 2014 were evaluated. Clinical and laboratory characteristics, response of the treatment, biopsy evidences, and the risk factors associated with mortality were analyzed. We also compared the cases of primary glomerulonephritis retrospectively. The mean value of the follow-up periods was 22 months (range: 8 - 76 months).*

***Results**: The median age was 42 years (18 - 80) in 264 patients. When five groups were compared, there was statistically important difference between the groups (p <0.001). The distributions of PGD subgroups were 40.5% for membranous glomerulonephritis (MGN), 20.4% for IgA nephropathy (IgAN), 25.7% for focal segmental glomerulosclerosis (FSGS), 7.9% for minimal change disease (MCD) and 5.3% for membranoproliferative glomerulonephritis (MPGN). The distrubition of gender was not significantly in the five PGD groups (p=0.269). Nephrotic syndrome was the most biopsy indication in MGN patients (76.6%). Nephritic syndrome was more seen in immunoglobulin A nephropathy (IgAN) patients (63%). Chronic kidney disease was more detected in focal segmental glomerulosclerosis (FSGS) patients as biopsy indication (p<0.001). Minimal change disease (MCD) and IgAN patients were more received with steroid treatment (85.7% and 55.5% respectively). Cyclophosphamide was more received in MGN patients. Serum IgG levels were more higher in FSGS and IgAN patients (p<0.001). Partial remission was more seen in MCD patients. Complete remission was more detected in MGN and FSGS patients. 29.1% of IgAN patients were not received treatment response. Six year renal survival rates were 84.1% (MGN), 87.1% (IgAN), 95.3% (MCD), 64.3% (MPGN) and 76.5% (FSGS) respectively.*

***Conclusion**: Biopsy maintains its diagnostic importance in glomerular diseases. Renal functions at diagnosis are important in response to treatment. Early diagnosis, follow-up and appropriate immunosuppressive medications affect mortality and clinical progress in PGD.*

***Keywords**: Primary glomerular disease, proteinuria, renal biopsy.*

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Introduction

Glomerulonephritis are the most of third common reasons worldwide those cause end stage renal disease (ESRD). Of all glomerular diseases, primary glomerulopathies were the most frequent (54.2%). In Turkey, at end of 2014, renal replacement treatment was performed in 4238 patients who developed ESRD in the course of glomerulonephritis (İncident of patients were 7.22%)[1]. Primary glomerular diseases (PGD) were the most frequent glomerular diseases in all type of glomerulopathies (54.2%). Factors such as gender, age and geographical location affect the distribution of glomerulonephritis. Data from different countries indicate that primary glomerulonephritis are accounted for 54.8-70.8% of all glomerulonephritis cases. According to data obtained from the Brazilian reg-

istry, the most common primary glomerular disease in the adult population is focal and segmental glomerulosclerosis (29.7%), followed by membranous nephropathy (20.7%), IgA nephropathy (IgAN) (17.8%), minimal change disease (9.1%), membranoproliferative glomerulonephritis (7%). In another review was included 40 studies of incidence of PGD from Europe, North and South America, Canada, Australasia and the Middle East. Rates for the individual types of disease were found to be in adults, 0.2/100 000/year for membrano-proliferative glomerulonephritis (MPGN), 0.6/100 000/year for minimal change disease (MCD), 0.8/100 000/year for focal segmental glomerulosclerosis (FSGS), 1.2/100 000/year for membranous nephropathy (MGN) and 2.5/100 000/year for IgAN[2, 3].

In our study, we retrospectively compared PGD patients who have been diagnosed following renal biopsy and analyzed their clinical and laboratory characteristics, responses to treatment. Our clinic serves a population of about 3 million. This is the first reported PGD study from the northwest region of Turkey.

Materials and methods

The medical records of patients who have had renal biopsy in Nephrology Clinics between 1st January 2009 and 31st December 2014 were evaluated retrospectively. Age, sex, serum immunoglobulin, complement, creatinine levels before and after treatment, albumin, estimated glomerular filtration rate (eGFR), lipid levels, daily urinary protein excretion (UPE) were evaluated in our patients. In addition, responses to treatment of the cases of primary glomerulonephritis, and applied treatments were investigated. The cases that had UPE<300mg/day were considered as complete remission. Cases were considered as partial remission if: had UPE>300mg/day and <3.5 g/day or 50% decreasing according to the initial value (4). First response to treatment was investigated 3 months after the treatment initial earliest. Patients who had diabetic or hypertensive glomerular findings were excluded. eGFR was calculated according to the Modification of Diet in Renal Disease (MDRD) formula [eGFR = 186 × Pcr (Plasma creatinine)-1.154 × age-0.203 × 1.212 (if black) × 0.742 (if female)].

Statistical analysis

Continuous variables were expressed by mean ± standard deviation or median (minimum-maximum) values; categorical variables were expressed by frequency and corresponding percentage values. Wilcoxon signed rank test and paired samples t test was used for within comparisons where between groups comparisons were performed by using Pearson chi-square test, Fisher Freeman Halton test, Kruskal Wallis test if appropriate. In order to compare before and after treatment values percent changes were computed and Kruskal Wallis test was applied to compare these values between the groups. The data was statistically processed by IBM SPSS version 22 software (IBM Acquires SPSS Inc., Somers, NY, USA). In all statistical analysis $p < 0.05$ was accepted as statistically significant for the results.

Results

The median age was 42 years (18 - 80) in 264 patients. When five groups were compared, there was statistically important difference between the groups ($p < 0.001$). Male sex was predominant in all PGD subgroups ($p=0.269$). MGN patients were older than other groups ($p<0.001$). Serum urea levels were higher in MPGN group. During the biopsy, nephrotic syndrome was more seen in MGN patients (76.6%). Nephritic syndrome was more detected in IgAN group (63%). Chronic kidney disease was more seen in FSGS patients (22.1%) ($p<0.001$). Serum IgG and C3c levels were more higher in IgAN group($p<0.001$ and $p=0.005$, respectively). Minimal change disease (MCD) and IgAN patients were more received with steroid treatment (85.7% and 55.5% respectively). Cyclophosphamide was more received in MGN patients. Cyclosporine A (CsA) was more received in FSGS patients (41.1%). The distribution of immunosuppressive therapy was different in all groups ($p=0.266$). Responses of treatment were different in all PGD groups ($p=0.338$). Partial remission was more seen in MCD group (47.6%). Complete remission was more detected in MGN patients (55.1%). Resistance to treatment was more seen in FSGS group (27.9%). Considering of clinical results, 66.7% of MCD patients were lived with treatment, 28.6% of MPGN with hemodialysis, 8.8% of FSGS with renal transplantation were followed. Death rate of MCD patients was higher (4.8%). Rejection rate was higher in FSGS group (2.9%). Laboratory and clinical datas are summarized in Table 1.

	MGN (n=107)	**IgAN (n=54)**	**MCD (n=21)**	**MPGN (n=14)**	**FSGS (n=68)**	**p value**
Age (n)	50 (23:79)	41 (21:80)	40 (18:70)	34 (20:59)	36.50 (20:76)	**p<0.001**
Gender (Female/Male) (%)	43/64 (40.20%/59.80%)	13/41 (24.07%/75.93%)	7/14 (33.30%/66.70%)	5/9 (35.70%/64.30%)	30/38 (44.10%/55.90%)	0.269
Urea (mg/dL)	38 (9:136)	48 (12:180)	30 (13:110)	57 (25:118)	46 (18:181)	**0.002**
Biopsy indication						
Nephrotic Syndrome	82 (76.60%)	11 (20.40%)	14 (66.70%)	6 (42.90%)	28 (41.20%)	**p<0.001**
Nephritic Syndrome	18 (16.80%)	34 (63%)	7 (33.30%)	6 (42.90%)	25 (36.80%)	
CKD	7 (6.50%)	9 (16.70%)	0 (0%)	2 (14.30%)	15 (22.10%)	
Serum IgG (mg/dL)	764 (179:2320)	1195 (253:1950)	489 (189:1160)	793.50 (324:1032)	1030.50 (148:1570)	**p<0.001**
Serum IgM (mg/dL)	114 (16:838)	108.20 (24.90:254)	123 (24.10:142)	143 (40.10:203)	106 (17.80:294)	0.670
Serum IgA (mg/dL)	196 (25.50:797)	389 (169:634)	169 (25.50:431)	175.50 (111:194)	186 (44.90:733)	**p<0.001**
Serum C3c (mg/dL)	126 (17.80:202)	135 (88.60:176)	128 (78.20:233)	115 (36.30:163)	127 (17:168)	**0.005**
Serum C4 (mg/dL)	31 (8.19:72.90)	31.40 (11.70:52.60)	27.70 (16.50:50.30)	28.80 (15.10:36.10)	29.90 (11.80:46)	0.104
Treatment						
Steroid	26 (24.3%)	30 (55.5%)	18 (85.7%)	4 (28.6%)	19 (27.9%)	0.266
Cyc	52 (48.6%)	4 (7.4%)	2 (9.5%)	4 (28.6%)	7 (10.3%)	
CsA	13 (12.1%)	7 (13%)	1 (4.8%)	2 (14.3%)	28 (41.1%)	
Aza	6 (5.6%)	5 (9.2%)	0	2 (14.3%)	6 (8.8%)	
MMF	0	1 (1.8%)	0	2 (14.3%)	1 (1.5%)	
ACE or ARB	5 (4.7%)	7 (13%)	0	0	7 (10.3%)	
Response of treatment						0.338
Partial	23 (21.50%)	14 (25.50%)	10 (47.60%)	4 (28.60%)	12 (17.60%)	
Complete	59 (55.10%)	25 (45.50%)	7 (33.30%)	7 (50%)	37 (54.40%)	
None	25 (23.40%)	16 (29.10%)	4 (19%)	3 (21.40%)	19 (27.90%)	
Clinical result						
LWT	82 (76.60%)	40 (24.10%)	14 (66.70%)	9 (64.30%)	43 (63.20%)	
Hemodialysis	5 (4.70%)	4 (7.40%)	0 (0%)	4 (28.60%)	9 (13.20%)	
LWOT	4 (3.70%)	2 (3.70%)	2 (9.50%)	0 (0%)	3 (4.40%)	
Uncontrolled	12 (11.20%)	4 (7.40%)	4 (19%)	0 (0%)	3 (4.40%)	
Death	3 (2.80%)	1 (1.90%)	1 (4.80)	0 (0%)	1 (1.50%)	
RT	1 (0.90%)	2 (3.70%)	0 (0%)	1 (7.10%)	6 (8.80%)	
PD	0 (0%)	1 (1.90%)	0 (0%)	0 (0%)	1 (1.50%)	
RTHD	0 (0%)	0 (0%)	0 (0%)	0 (0%)	2 (2.90%)	

Table 1: Laboratuary and clinical findings and distribution of primary and secondary glomerulonephritis.
MGN: Membranous glomerulonephritis. IgAN: IgA nephropathy. MCD: Minimal change disease. MPGN: Membranoproliferative glomerulonephritis. FSGS: Focal segmental glomerulosclerosis. ACE: Angiotensin converting enzyme. ARB: Angiotensin reseptör blockers. Cyc: Cyclophosphamide. CsA: Cyclosporine. Aza: Azathioprine. MMF: Mycophenolate mofetil. LWT: Life with treatment. LWOT: Life without treatment. RT: Renal transplantation. PD: Peritoneal dialysis. RTHD: Renal transplantation, graft loss, hemodialysis

When renal biopsy findings were evaluated, global and segmental sclerosis were higher in FSGS cases (p<0.001). Interstitial inflammation and fibrosis rates were more common higher in MGN, FSGS and IgAN cases (75.9%) (p<0.001). Number of crescent was dominant in IgAN patients and tubular atrophy in FSGS group (p<0.001). Mesengial proliferation was more seen in IgAN cases (p<0.001).

Vascular involvement was more common in FSGS patients (p<0.001) (Table 2).

	MGN (n=107)	IgAN (n=54)	MCD (n=21)	MPGN (n=14)	FSGS (n=68)	p value
Total number of glomeruli (n)	12 (5:44)	13 (7:41)	12 (7:36)	13.50 (8:32)	12 (7:37)	0.351
Global sclerosis (n)	1 (0:10)	2 (0:7)	0 (0:3)	2 (0:3)	2 (0:12)	**<0.001**
Segmental sclerosis (n)	0 (0:4)	0 (0:3)	0 (0:2)	0 (0:2)	1 (0:6)	**<0.001**
Crescent presence	0 (0:0)	0 (0:7)	0 (0:0)	0 (0:0)	0 (0:0)	**0.003**
Mesangial proliferation (Present/absent) (n,%)	14/93 (13.10%/86.90%)	52/3 (94.50%/5.50%)	0/21 (0%/100%)	7/7 (50%/50%)	45/23 (66.20%/33.80 %)	**<0.001**
Thickening of the basement membrane (Present/absent) (n,%)	107/0 (100%/0%)	15/40 (27.30%/72.70%)	0/21 (0%/100%)	13/1 (92.90%/7.10%)	9/59 (13.20%/86.80 %)	**<0.001**
Interstitial inflammation (Present/absent) (n,%)	103/4 (96.30%/3.70%)	55/0 (100%/0%)	16/5 (76.20%/23.80%)	14/0 (100%/0%)	68 (100%/0%)	**<0.001**
Interstitial fibrosis (Present/absent) (n,%)	75/32 (70.10%/29.90%)	42/13 (76.40%/23.60%)	1/20 (4.80%/95.20%)	10/4 (71.40%/28.60%)	67/1 (98.50%/1.50%)	**<0.001**
Vascular involvement (Present/absent) (n,%)	45/62 (42.10%/57.90%)	25/30 (45.50%/54.50%)	2/19 (9.50%/9.50%)	6/8 (42.90%/57.10)	51/17 (75%/25%)	**<0.001**
Tubular atrophy (Present/absent) (n,%)	79/28 (73.80%/26.20%)	39/16 (70.90%/29.10%)	4/17 (19%/81)	12/2 (85.70%/14.30%)	63/5 (92.60%/7.40%)	**<0.001**
IgG (n)	3 (0:3)	0 (0:1)	0 (0:0)	0 (0:3)	0 (0:2)	**<0.001**
IgM (n)	0 (0:3)	0 (0:2)	0 (0:0)	0 (0:0)	0 (0:3)	**0.043**
IgA (n)	0 (0:3)	3 (2:3)	0 (0:0)	0 (0:0)	0 (0:3)	**<0.001**
C3c (n)	0 (0:3)	0 (0:3)	0 (0:0)	2 (0:3)	0 (0:3)	**<0.001**
C1q (n)	0 (0:3)	0 (0:1)	0 (0:0)	0 (0:0)	0 (0:0)	0.750
Fibrin (n)	0 (0:2)	0 (0:2)	0 (0:0)	0 (0:2)	0 (0:2)	0.507

Table 2: Renal biopsy staining and findings.
MGN: Membranous glomerulonephritis. IgAN: IgA nephropathy. MCD: Minimal change disease. MPGN: Membranoproliferative glomerulonephritis. FSGS: Focal segmental glomerulosclerosis.

	Pre:treatment	Post:treatment	p value
MGN (n=107)			
Creatinine (mg/dL)	0.90 (0.30:3.60)	0.90 (0.40:5.79)	0.730
Serum albumin (g/dL)	2.80 (1.10:5)	3.80 (1.70:4.80)	**<0.001**
UPE (g/day)	5.62 (0.60:38)	1.33 (0.02:28)	**<0.001**
eGFR (mil/min)	81.94 (17.90:267.54)	82.46 (10.35:212)	0.960
Total cholesterol (mg/dL)	298 (104:678)	239 (130:1986)	**<0.001**
LDL cholesterol (mg/dL)	195 (45:515)	152 (67:330)	**<0.001**
Triglyceride (mg/dL)	241 (70:919)	171 (52:421)	**<0.001**
HDL cholesterol (mg/dL)	44 (5:123)	46 (19:135)	**0.009**
IgAN (n=54)			
Creatinine (mg/dL)	1.20 (0.60:5.40)	1.20 (0.50:9.90)	0.220
Serum albumin (g/dL)	3.70 (1.80:4.50)	4 (2.30:4.60)	**<0.001**
UPE (g/day)	1.95 (0.10:11)	0.63 (0.01:5.60)	**<0.001**
eGFR (mil/min)	74.73±38.56	74.65±43.54	0.976
Total cholesterol (mg/dL)	228 (123:420)	203 (100:420)	**<0.001**
LDL cholesterol (mg/dL)	142 (67:279)	126 (36:348)	**<0.001**
Triglyceride (mg/dL)	174 (77:405)	135 (63:254)	**<0.001**
HDL cholesterol (mg/dL)	42 (18:86)	42 (19:92)	0.718
MCD (n=21)			
Creatinine (mg/dL)	0.70 (0.40:1.40)	0.80 (0.50:1.50)	0.692
Serum albumin (g/dL)	2.50 (1.40:4.80)	4 (1.80:4.50)	**0.001**
UPE (g/day)	3.62 (0.78:46)	1.18 (0.01:10)	**<0.001**
eGFR (mil/min)	65.91 (15.05:217.13)	75.61 (5:217.13)	0.398
Total cholesterol (mg/dL)	223 (123:705)	214.50 (68:602)	**0.044**
LDL cholesterol (mg/dL)	149 (17:412)	134.50 (34:487)	**0.020**
Triglyceride (mg/dL)	172 (65:882)	133.50 (36:411)	**<0.001**
HDL cholesterol (mg/dL)	43 (27:84)	45 (12:100)	**0.018**

Table 3: Pre and posttreatment laboratuary findings intragroup comparisons.
MGN: Membranous glomerulonephritis. IgAN: IgA nephropathy. MCD: Minimal change disease. MPGN: Membranoproliferative glomerulonephritis. FSGS: Focal segmental glomerulosclerosis. UPE: Urinary protein excretion. eGFR: Estimated glomerular filtration rate. LDL: Low dansity lipoprotein. HDL: High dansity lipoprotein.

When pre-treatment and post-treatment laboratory results of PGD patients were compared, it was seen that while serum levels of albumin increased, total cholesterol and UPE levels decreased in all groups (p<0.001) (Table 3). Percentage change of laboratory results before and after treatment were investigated between all of PGD groups, and while increasing in serum levels of albumin (p=0.010), decreasing in total cholesterol (p=0.005), LDL cholesterol (p=0.011) and triglyceride (p=0.036) levels were significantly more seen in all groups (Table 4). Six year renal survival rates were 84.1% (MGN), 87.1% (IgAN), 95.3% (MCD), 64.3% (MPGN) and 76.5% (FSGS) respectively. We did not find any factors who effected renal survival in multivariate analysis.

	MGN (n=107)	IgAN (n=54)	MCD (n=21)	MPGN (n=14)	FSGS (n=68)	p value
Creatinine (mg/dL)						
Pretreatment	0.90 (0.30:3.60)	1.20 (0.60:5.40)	0.70 (0.40:1.40)	1.05 (0.60:3.04)	1.10 (0.50:4.60)	**0.027**
Posttreatment*	0 (-0.65:6.29)	0 (-0.54:2.96)	0.14 (-0.38:0.58)	-0.04 (-0.33:5.31)	0.11 (-0.64:2.70)	0.101
Serum albumin (g/dL)						
Pretreatment	2.80 (1.10:5)	3.70 (1.80:4.50)	2.50 (1.40:4.80)	3.35 (1.60:5.20)	3.40 (0.80:4.80)	**<0.001**
Posttreatment*	0.24 (-0.39:1.64)	0.08 (-0.26:0.87)	0.38 (-0.40:1.29)	0.19 (-0.04:1.06)	0.15 (-0.49:2.58)	**0.010**
UPE (g/day)						
Pretreatment	5.62 (0.60:38)	1.95 (0.10:11)	3.68 (0.70:22)	4.20 (1:17)	3.62 (0.78:46)	**<0.001**
Posttreatment*	-0.73 (-1:1.54)	-0.69 (-0.97:2.67)	-0.91 (-1:2.86)	-0.69 (-1:0.09)	-0.67 (-1:4.56)	0.072
eGFR (mil/min)						
Pretreatment	81.94 (17.90:267.54)	69 (10:157.04)	110.13 (42.66:186.51)	70.11 (20:146.05)	65.91 (15.05:217.13)	**0.025**
Posttreatment*	0 (-0.90:1.23)	0 (-0.80:0.90)	-0.14 (-0.45:0.54)	0.05 (-0.88:0.61)	-0.12 (-0.77:2.26)	0.102
Total cholesterol (mg/dL)						
Pretreatment	298 (104:678)	228 (123:420)	318 (203:722)	285 (154:394)	223 (123:705)	**<0.001**
Posttreatment*	-0.15 (-0.66:9.18)	-0.12 (-0.60:1.16)	-0.24 (-0.68:0.45)	-0.19 (-0.50:0.05)	-0.06 (-0.71:1.34)	**0.005**
LDL cholesterol (mg/dL)						
Pretreatment	195 (45:515)	142 (67:279)	189 (99:619)	171.50 (3.50:215)	149 (17:412)	**0.001**
Posttreatment*	-0.18 (-0.74:1.26)	-0.16 (-0.76:2.59)	-0.3 (-0.74:1.59)	-0.09 (-0.45:47)	-0.12 (-0.79:7.18)	**0.011**
Triglyceride (mg/dL)						
Pretreatment	241 (70:919)	174 (77:405)	228 (78:425)	143 (98:429)	172 (65:882)	**0.006**
Posttreatment*	-0.23 (-0.86:1.27)	-0.21 (-0.64:0.48)	-0.22 (-0.64:0.43)	-0.14 (-0.68:0.65)	-0.17 (-0.87:2.06)	**0.036**
HDL cholesterol (mg/dL)						
Pretreatment	44 (5:123)	42 (18:86)	50 (27:145)	50 (35:90)	43 (27:84)	0.995
Posttreatment*	0.07 (-0.66:5)	0.02 (-0.48:1.19)	0.04 (-0.64:1.11)	-0.05 (-0.61:0.14)	0.07 (-0.71:0.73)	0.952

Table 4: Pre and posttreatment percentage changes in groups.
*MGN: Membranous glomerulonephritis. IgAN: IgA nephropathy. MCD: Minimal change disease. MPGN: Membranoproliferative glomerulonephritis. FSGS: Focal segmental glomerulosclerosis. UPE: Urinary protein excretion. eGFR: Estimated glomerular filtration rate. LDL: Low dansity lipoprotein. HDL: High dansity lipoprotein. *Percentage change values was compared.*

Discussion

This study was the first study from our province that analyze PGD patients according to biopsy findings, treatment responses, clinical

results and laboratuary findings. PGDs are from the most three causes advance to ESRD. The subgroups of the disease vary by many factors such as genetic, geographical location, gender, age. Renal biopsy is the gold standard for diagnosis and beginning of treatment[5, 6]. The commonest type of PGD alterable in many countries and regions. We found MGN was the most frequent type of PGD (40.5%). FSGS was the commonest type of PGD encountered in Saudi Arabia and IgAN is the most detected type of PGD in Australia and west of France. MGN was more seen in China[5-8]. The mean age of PGD patients were higher in MGN cases and male gender was predominantly in our study such as other studies[5, 9].

Nephrotic syndrome was the most clinical evidence in our study by the time of biopsy (53.4%). the frequency of nephrotic syndrome among patients submitted to renal biopsy has ranged from 17 to 49.5%[10, 11]. Angiotensin converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARB) were more use in PGD as primary treatment. Steroid treatment was more received in MCD and MGN, cytotoxic agents were more used in FSGS and MGN[5, 9]. Response of treatment was different in PGD. Complete remission was seen between 37.4% and 47.9%. Partial remission alter between 13.9% and 25.4%. Patients with no response were seen between 12.8% and 19.9%[11]. We found similar results in treatment and response of treatment.

The clinical course was different in type of PGD. FSGS had poor prognosis and renal survival among PGD patients. Although the treatment 50% of FSGS patients progress to ESRD and the recurrence rates were higher after transplantation[5, 12]. ESRD and recurrence were higher in our FSGS patients. Generally renal survival was lower in FSGS and MPGN[5, 9, 13]. We demonstrated similar results in our study.

The absence of this study was its retrospective nature and limit comparisons with datas from other regions of Turkey.

Consequently, our study gives epidemiologic, clinical and laboratuary informations on the evaluation of PGD. Renal biopsy is gold standard in diagnosis for PGD patients. Early diagnosis, follow-up and appropriate immunosuppressive medications affect mortality and clinical progress in PGD. The multiplicity of biopsy findings and proteinuria are significant for the choice of treatment. Renal survival is poor in some PGD subtypes such as FSGS and MPGN. The glomerulopathies can repeat although renal transplantation.

Monitoring and treatment of patients must be closely followed. Genetic studies, biomarkers can support the diagnosis such as kidney biopsy.

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Corresponding author
YAVUZ AYAR
Uludag University Faculty of Medicine, Department of Nephrology, 16059 Gorukle-Bursa
(Turkey)