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Risk factors for chronic kidney disease progression in patients with solitary kidney

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ABSTRACT

Introduction: The prevalence of chronic kidney disease (CKD) is rapidly increasing worldwide. Solitary kidney is also increasing in ranking among the CKD etiologies, because there has been a rapid increase in the number of radical nephrectomies due to an increased number of renal transplantations from live donors and an increased number of patients with renal cell carcinoma. The aim of the current study is to identify risk factors that affect the glomerular filtration rate (GFR) in individuals with solitary kidney.

Material and Method: The current study included 204 patients (75 with congenital, 129 with acquired solitary kidney). Laboratory data during the first and last admissions were recorded. Patients divided into two groups according to annual decline of eGFR. Group I and II consisted of patients whose annual decline eGFR was more than 1ml/min/1.73 m² and less than 1ml/min/1.73 m², respectively. In addition, patients were divided into two groups as patients with congenital and acquired solitary kidney. The first control is the first examination in the nephrology outpatient clinic for congenital solitary kidney patient and the post-operative examination on the fourteenth day after discharge from the hospital for the acquired solitary kidney patient. The final control is the examination within the last three months before reaching the primary endpoint of the study.

Results: Of the patients, 36.8% were male, and the average age was 57.16±15.04 years. The duration of the follow-up period was 6.48±3.69 years. Group I had higher rates of diabetes mellitus, cardiovascular disease, older age, higher mean blood pressure(MBP), glucose, CRP, total cholesterol (TC), LDL-cholesterol, non-HDL-cholesterol, triglyceride/non-HDL-cholesterol ratio and lower albumin. In the group with acquired solitary kidney, the patients were older, the incidence of cardiovascular diseases was higher, and the eGFR at the first and last admission was lower. There was no difference between acquired SK and congenital SK in terms of annual change in eGFR. In regression analysis CRP, LDL-cholesterol, non-HDL-cholesterol, TG/non-HDL-cholesterol ratio are independent risk factors on annual decline of eGFR. Having a congenital or acquired single kidney had no effect on the annual decline of eGFR. In addition, TC, TC/HDL-cholesterol, triglyceride/non-HDL-cholesterol, triglyceride/HDL-cholesterol ratios, non-HDL -cholesterol correlated with CRP positively.

Conclusıon:Patients with solitary kidney have higher risk of developing CKD. Inflammation and dyslipidemia must be paid attention to protect eGFR. Besides the atherosclerosis in the microcirculation, dyslipidemia affect eGFR through inflammation. Having a congenital or acquired single kidney has no effect on the annual decline of eGFR.

Keywords: Solitary kidney, inflammation, dyslipidemia, proteinuria, glomerular filtration rate

INTRODUCTION

Chronic kidney disease (CKD) is an important public health problem worldwide. In spite of the variability among different societies and locations, the prevalence of CKD increases on average 6% per year (1). With time, patients with CKD experience deterioration in their quality of life. Moreover, CKD patients have a 10 to 20-fold increase in mortality due to cardiovascular diseases (CVD) compared to the normal population. This rate can be up to 30-times in cases developing end-stage renal disease (ESRD) (2,3). Therefore, it is very important to closely monitor patients at risk for developing ESRD.

Although diabetes mellitus (DM), hypertension (HT), and glomerulonephritis are the most common causes in CKD etiologies, solitary kidney has recently taken its place among CKD etiologies, because there is an increasing number of patients developing ESRD worldwide, there has also been an increase in the number of renal transplantations from live donors. Additionally, due to an increase in the number of patients with renal cell CA, there have also been increases in the number of partial and radical nephrectomies. In the past, studies revealed that there was no difference between live donors and the general population in terms of developing ESRD and mortality rates(4,5). However, more recent studies in the USA and Norway reported that the risk of developing ESRD was increased by 8 and 11 times, respectively, in live donors (6,7). Likewise, a 2019 Korean study indicated that the risk of developing CKD increased by 3.26 times in live donors compared to the control group (8).

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The aim of the current study is to identify risk factors that affect the glomerular filtration rate (GFR) in individuals with solitary kidney.

MATERIAL AND METHOD

The study was carried out with the permission of Clinical Research Ethics Committee of Ankara Training and Research Hospital (Date: 21.04.2020, Decision No: E-20/201). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Inclusion and Exclusion Criteria

The study included 204 patients (75 with congenital solitary kidney and 129 with acquired solitary kidney) who were followed up with between 2005 and 2020. Patients between the ages of 18-80 and whose single kidney was shown radiologically were included in the study. Patients who needed intensive care due to complications in the postoperative period, had to use nephrotoxic agents, developed AKI, underwent dialysis, did not come for regular controls, and with partial nephrectomy or hypoplastic kidney, who had renal cell carcinoma with stage 2,3 or 4, other active malignancies, and chronic inflammatory disease were excluded from the study. The primary endpoint of current study was death or initiation of renal replacement therapy.

Laboratory Parameters

As well as demographic characteristics, serum urea, creatinine, and eGFR values, 24 h urine protein values at the first and last control, and complete blood count, albumin, CRP, total cholesterol (TC), HDL-cholesterol, LDL-cholesterol, non-HDL-cholesterol, and triglyceride levels at the first control were recorded. The first control is the first examination in the nephrology outpatient clinic for congenital solitary kidney patient and the post-operative examination on the fourteenth day after discharge from the hospital for the acquired solitary kidney patient. The final control is the examination within the last three months before reaching the primary endpoint of the study or the study end date.

GFR was calculated by the CKD-EPI (CKD Epidemiology Collaboration) equation. LDL-cholesterol was indirectly calculated using Friedewald formula. Non-HDL-cholesterol was calculated as HDL-cholesterol substracted from total cholesterol. Patients were classified according to anual change of eGFR as group I and II. Group I and group II consisted of patients whose annual decline of eGFR was more than 1 ml/min/1.73 m² and less than 1 ml/min/1.73 m², respectively. The annual decline of eGFR was calculated by the difference between baseline eGFR and the last eGFR divided by time interval in years. The annual decline in eGFR was grouped as above and below 1 ml/min/1.73 m² so that the groups were not affected by the decrease in EGFR after the age of fourty. Patients were also divided into two groups as patients with congenital and acquired solitary kidney.

Statistical Analyses

Analyses were conducted using SPSS (version 22.0). All data were fist checked for normality of distribution using the Kolmogrov-Smirnov and Shapirov-Wilk test. Normally distributed data are presented as the mean± standard deviation. Non-normally distributed data are represented as the median (inter-quartile range). Independent samples T test, was used to compare parametric continuous variables between groups. Mann Whitney U was employed for the comparison of non-parametric variables. Pearson's X^2 or Fisher's exact were used for categorical variables. Correlation analyses were performed using the Pearson /Spearman correlation coefficient. Univariate and multivariate cox regression analysis were used to identify independent risk factors on annual decline of eGFR. A significant difference was considered when $p<0.05$.

RESULTS

Of the patients, 36.8% were male, and the average age was 57.16±15.04 years. The duration of the follow-up period was 6.48±3.69 years, and 129 patients had nephrectomy. The causes of nephrectomy were nephrolithiasis (45.7%), renal mass (25.5%), unknown etiology (12.4%), donation (9.4%), infection (3.9%), and trauma (3.1%). Comparison of demographic characteristics between the two groups revealed that group I had higher rates of DM and cardiovascular disease, older age, and higher mean blood pressure values than group II. A difference between the groups according to the rates of congenital or acquired solitary kidney was not found. In addition, the incidence of proteinuria and eGFR values were similar at baseline. Compared to group II, group I had higher glucose, CRP, total cholesterol, LDL-cholesterol, non-HDL-cholesterol, triglyceride/non-HDL-cholesterol ratio and lower albumin (**Table 1**). In the group with acquired solitary kidney, the patients were older, the incidence of cardiovascular diseases was higher, and the eGFR at the first and last admission was lower. However, there was no difference in terms of annual change in eGFR (**Table 2**). In regression analysis CRP, LDL-cholesterol non-HDL-cholesterol, TG/non-HDL-cholesterol ratio are independent risk factors on annual decline of eGFR. Having a congenital or acquired single kidney had no effect on the annual decline of eGFR (**Table 3**). In addition, total cholesterol, total cholesterol/HDL-cholesterol, triglyceride/non-HDL-cholesterol, triglyceride/HDL-cholesterol ratios, and non-HDL-cholesterol correlated with CRP positively (**Table 4**).

Table 1. Comparison of basic characteristics and laboratory data between groups

Parameters	All patients (n:204)	Group I (n:123)	Group II (n:81)	P
Gender (Female) (%)	63.2	60.2	67.9	0.215
DM (%)	24.5	32.5	12.3	0.001
CVD (%)	12..7	17.1	6.2	0.002
HT (%)	67.2	70.7	61.7	0.325
Cause of SK Congenital (%)	36.8	34.1	40.7	0.456
Follow-up time (years)	6.48±3.69	7 (7) (min:0.5-max:21)	6 (5) (min:max:16)	0.521
Age (year)	57.16±15.04	63 (17) (min:22-max:80)	54 (23) (min:18-max:78)	<0.001
MBP (mmHg)	94.51±10.38	93 (19) (min:70-max:117)	93 (14) (min:73-max:120)	0.039
Urea at first admission (mg/dL)	39.47±17.9	36 (19) (min:17-max:112)	34 (18.5) (min:17-max:129)	0.321
Creatinine at first admission (mg/dL)	1.28±0.46	1.2 (0.56) (min:0.6-max:3.6)	1.18 (0.55) (min:0.7-max:2.45)	0.351
Proteinuria at first admission (mg/day)	324.75±766.71	100 (500) (min:100-max:4940)	100 (185) (min:100-max:1660)	0.444
EGFR atfirst admission (ml/min/1.73 m²)	63.42±53.74	57.1 (33.2) (min:12-max:76.1)	58 (32.3) (min:24-max:112)	0.412
Urea at last admission (mg/dL)	45.94±21.91	45 (26) (min:18-max:155)	34 (13.5) (min:17-max:66)	<0.001
Creatinine at last admission (mg/dL)	1.35±0.94	1.29 (0.64) (min:0.57-max:9.5)	0.96 (0.38) (min:0.58-max:1.77)	<0.001
Proteinuria at last admission (mg/day)	443.62±1087.95	100 (448) (min:100-max:6560)	100 (100) (min:100-max:3440)	<0.001
eGFR at last admission (ml/min/1.73 m²)	62.42±26.57	52 (29.2) (min:3-max:108)	80 (37.45) (min:34.9-max:127)	0.002
Annual change of eGFR (ml/min/1.73 m²)	0.49±9.74	-1.23 (0.482) (min:-44.2;max:+2.40)	4.07 (4.65) (min:+1.25;max:+43.6)	<0.001
Glucose (mg/dL)	109.26±31.65	99 (28) (min:72-max:250)	98 (17) (min:77-max:208)	0.023
Total Cholesterol (TC) (mg/dL)	193.06±48.57	195 (58) (min:95-max:409)	180 (58.5) (min:114-max:407)	0.011
HDL-cholesterol (mg/dL)	46.21±12.78	44 (13) (min:22-max:82)	45 (19.5) (min:19-max:89)	0.323
LDL-cholesterol (mg/dL)	113.11±34.21	116 (48) (min:50-max:224)	102 (42.5) (min:34-max:222)	0.014
Triglyceride (TG) (mg/dl)	176.64±110.54	156 (148) (min:48-max:640)	134 (80) (min:45-max:527)	0.546
TC/HDL-cholesterol ratio	4..42±1.5	4.46 (1.91) (min:2.2-max:9.74)	3..3 (1.76) (min:2.1-max:14)	0.016
LDL/HDL-cholesterol ratio	2.59±0.9	2.67±0.85 (min:0.95-max:5.96)	2.46±0.96 (min:0.33-max:5)	0.876
TG/HDL-cholesterol ratio	4.3±3.33	3..4 (4.12) (min:0.89-max:22.37)	3.13 (2.71) (min:0.79-max:21)	0.159
TG/non-HDL-cholesterol ratio	0.52±0.29	0.56±0.3 (min:0.1-max:1.35)	0.47±0.27 (min:0.19-max:1.32)	0.042
non-HDL-cholesterol (mg/dL)	146.85±47.31	151 (52) (min:65-max:367)	127 (59.5) (min:77-max:378)	0.002
Uric acid (mg/dL)	6.11±1.6	6.1 (2.5) (min:2.6-max:11.4)	5.9 (2.5) (min:2.6-max:9.8)	0.323
Albumin (g/dL)	4.32±0.42	4.3 (0.5) (min:3.1-max:5.29)	4.4 (0.42) (min:3.1-max:3.54)	0.036
CRP (mg/L)	1.93±2.12	1.1 (2.2) (min:0.2-max:15)	1 (1.5) (min:0.2-max:12)	0.006
WBC (10^6/L)	7722±2043	7500 (3200) (min:4100-12800)	7400 (2400) (min:3500-max:13300)	0.458
Neutrophil (10^6/L)	4832±1663	4600 (2300) (min:2000-max:9300)	4400 (1950) (min:1900-max:11400)	0.563
Lymphocyte (10^6/L)	2139±701	2100 (1100) (min:800-max:4310)	2000 (800) (min:800-max:4400)	0.741
Monocyte (10^6/L)	519±186	500 (200) (min:100-max:1070)	500 (200) (min:200-max:1400)	0.596
Platelet (10^9/L)	261019±77312	251 (97) (min:132-max:456)	242 (87) (min:116-max:717)	0.546
MPV (fL)	8.84±1.21	8.6 (1.6) (min:6.1-max:13.3)	8.7 (0.65) (min:6.6-max:11.9)	0.357
Hemoglobin (g/dL)	13.9±4.08	13.4 (1.9) (min:8.1-max:17.4)	13.7 (2.1) (min:9.7-max:13.7)	0.489
RDW (%)	14±1.51	13.8 (1.6) (min:11.3-max:21.7)	13.5 (1.5) (min:11.9-max:17.3)	0.583

Normally distributed data are presented as the mean± standard deviation. Non-normally distributed data are represented as the median (inter-quartile range) DM: Diabetes mellitus, HT: Hypertension, CVD: Cardiovascular diseases CRP:C-reactive protein, WBC: White blood cell, RDW: Red cell distrubition width, MPV: Mean Platelet Volume, GFR: Glomerular filtration rate, MBP: Mean blood pressure, SK:Solitary Kidney

Table 2. Comparison of basic characteristics and laboratory data between groups with congenital and acquired solitary kidney

Parameters	Group with congenital SK(n:75)	Group with acquired SK(n:129)	P
Gender (Female) (%)	39.5	60.5	0.296
DM (%)	30	70	0.312
CVD (%)	11.5	88.5	0.004
HT (%)	34.3	65.7	0.354
Follow-up time (years)	6 (6) (min:0.5-max:21)	7 (5) (min:1-max:19)	0.273
Age (year)	55 (27) (min:20-max:80)	60 (19) (min:25-max:80)	0.008
MBP (mmHg)	93 (17) (min:70-max:116)	93 (18) (min:77-max:120)	0.180
Urea at first admission (mg/dL)	34 (23) (min:17-max:113)	35 (17) (min:18-max:129)	0.662
Creatinine at first admission (mg/dL)	1.1 (0.52) (min:0.6-max:2.45)	1.2 (0.55) (min:0.7-max:3.6)	0.123
Proteinuria at first admission (mg/day)	100 (240) (min:100-max:4220)	100 (255) (min:100-max:4940)	0.832
eGFR at first admission (ml/min/1.73 m²)	64.5 (43.5) (min:19-max:120)	57 (27.7) (min:12-max:86)	0.041
Urea at last admission (mg/dL)	38 (27) (min:17-max:144)	41 (25) (min:18-max:155)	0.120
Creatinine at last admission (mg/dL)	1.04 (0.56) (min:0.5-max:5.61)	1.22 (0.59) (min:0.6-max:9.5)	0.043
Proteinuria at last admission (mg/day)	100 (340) (min:100-max:5360)	100 (290) (min:100-6560)	0.998
eGFR at last admission (ml/min/1.73 m²)	69±29 (min:7-max:127)	58±32.5 (min:3-max:118)	0.01
Annual change of eGFR (ml/min/1.73 m²)	0.78 (0.90) (min:-44.2;max:43.6)	0.77 (0.80) (min:-33.8. max:37.82)	0.321
Glucose (mg/dL)	97 (20) (min:80-max:250)	109 (23) (min:72-max:240)	0.387
Total Cholesterol (TC)(mg/dL)	189 (68) (min:95 max:409)	190 (59.5) (min:114-max:407)	0.551
HDL-cholesterol (mg/dL)	45 (17) (min:22-max:77)	43 (15) (min:19-max:89)	0.357
LDL-cholesterol (mg/dL)	112±32 (min:53-max:185)	113±35 (min:34-max:224)	0.957
Triglyceride (TG) (mg/dL)	134 (101) (min:45-max:537)	154 (127) (min:49-max:640)	0.066
TC/HDL-cholesterol ratio	4.13 (1.72) (min:2.2-max:9.74)	4.32 (1.95) (min:2.18-max:14)	0.193
LDL/HDL-cholesterol ratio	2.52±0.84 (min:0.9-max:4.96)	2.62±0.93 (min:0.3-max:5.96)	0.427
TG/HDL-cholesterol ratio	3 (3) (min:0.73-max:12.79)	3.3 (2.9) (min:0.79-max:22.37)	0.097
TG/non-HDL-cholesterol ratio	0.47±0.29 (min:0.14-max:1.11)	0.56±0.27 (min:0.1-max:1.35)	0.042
non-HDL-cholesterol (mg/dL)	141 (60) (min:65-max:367)	144 (52) (min:72-max:378)	0.389
Uric acid(mg/dL)	5.9±1.8 (min:2.6-max:11.4)	6.1±1.5 (min:2.6-max:9.5)	0.404
Albumin(g/dL)	4.3 (0.5) (min:3.2-max:5.3)	4.3 (0.5) (min:3.1-max:5.4)	0.835
CRP(mg/L)	1 (1.8) (min:0.28-max:2.2)	1 (1.5) (min:0.21-max:1.9)	0.819
WBC(10^6/L)	7700 (3300) (min:3500-max:12640)	7200 (2785) (min:4100-max:13300)	0.450
Neutrophil(10^6/L)	4600 (2100) (min:1900-max:9510)	4400 (2300) (min:2050-max:11400)	0.963
Lymphocyte(10^6/L)	2200 (1010) (min:800-max:4310)	200 (900) (min:800-max:4400)	0.131
Monocyte(10^6/L)	500 (300) (min:200-max:1400)	500 (200) (min:100-max:1100)	0.226
Platelet(10^9/L)	247 (193) (min:116-max:456)	250 (99) (min:132-max:717)	0.903
MPV(fL)	8.7 (1.3) (min:6.6-max:13.3)	8.6 (1.8) (min:6.1-max:11.9)	0.571
Hemoglobin(g/dL)	13.5 (1.8) (min:9.7-max:17)	13.6 (2.1) (min:8.1-max:13.2)	0.543
RDW(%)	13.9 (2) (min:11.3-max:18.3)	13.6 (1.5) (min:11.8-max:21.7)	0.438

Normally distributed data are presented as the mean± standard deviation. Non-normally distributed data are represented as the median (inter-quartile range) DM: Diabetes mellitus, HT: Hypertension, CVD: Cardiovascular diseases CRP:C-reactive protein, WBC: White blood cell, RDW: Red cell distrubition width, MPV: Mean Platelet Volume, GFR: Glomerular filtration rate, MBP: Mean blood pressure, SK: Solitary kidney

Table 3. Univariate and multivariate cox regression analysis for annual decline of eGFR (n:204)

Parameters	UNIVARIATE ANALYSIS			MULTIVARIATE ANALYSIS		
	HR	95%CI	P	HR	95%CI	P
Age	1.005	0.991-1.020	0.449			
DM	1.088	0.744-1.592	0.663			
CVD	1.055	0.659-1.691	0.823			
MBP	1.003	0.985-1.021	0.750			
Glucose	1	0.995-1.005	0.936			
CRP	0.919	0.839-1.008	0.043	0.902	0.819-0.993	0.036
Albumin	0.854	0.533-1.368	0.511			
TC	1	0.996-1.004	0.997			
LDL-cholesterol	0.999	0.994-1.003	0.050	0.985	0.973-0.997	0.014
Non-HDL- cholesterol	1	0.996-1.004	0.062	1..013	1.002-1.023	0.016
TG/non-HDL- cholesterol	0.727	0.392-1.350	0.313	0.391	0.165-0.926	0.033
Causes of SK	0.933	0.641-1.360	0.720			

DM: Diabetes mellitus, CVD: Cardiovascular diseases CRP:C-reactive protein, GFR: Glomerular filtration rate, MBP: Mean blood pressure, TC: Total Cholesterol, TG: Triglyceride SK: Solitary Kidney

Table 4. Correlation analysis between CRP levels and cholesterol parameters (n:204)

Parameters	r	P
TC	0.154	0.027
TC/HDL- cholesterol	0.168	0.016
TG/HDL- cholesterol	0.136	0.049
Non-HDL- cholesterol	0.175	0.012

CRP:C-reactive protein, MBP: Mean blood pressure, TC: Total Cholesterol, TG Triglyceride

DISCUSSION

Solitary kidney disease is either congenital (renal agenesis) or acquired. GFR is preserved through initial adaptation mechanisms and glomerular hyperfiltration. However, extreme loads (e.g., hypertension, dyslipidemia) on the kidney increase intra-glomerular pressure, which can lead to permanent renal damage. Hypertension is the best known of these extreme loads. Increased intra-glomerular pressure in patients with chronic uncontrolled hypertension can cause podocyte damage and impaired perm-selectivity of the cleft diaphragm, which can lead to proteinuria (9). Despite of similar levels of proteinuria at first control, proteinuria level at last control and MBP were higher in group-I and they were negatively correlated with eGFR. However in regression analysis the effects of MBP and proteinuria on CKD progression were not found.

Dyslipidemia is another extreme load experienced by the kidney. The relationship between CKD and dyslipidemia is often defined by atherosclerosis in the microcirculation. However dyslipidemia also plays a role in the development of CKD by directly causing inflammation. Damage of dyslipidemia, especially FFA, on the kidney occurs through two mechanisms. First mechanism: Tubule cells need energy for reabsorption. Maximum ATP production occurs during the breakdown of FFA in mitochondria. If this breakdown is more than enough, reactive oxygen radical production increases. This leads to renal damage (10). Second mechanism: Excessive accumulation of FFA in tubule cells causes structural changes which eventually trigger apoptosis. The SLC27A2 gene is responsible for this mechanism (11). The reason for the excessive accumulation of FFA in tubule cells are decreased HDL levels and diminished cholesterol efflux capacity (12). In current study total cholesterol, LDL-cholesterol non-HDL-cholesterol, triglyceride/non-HDL-cholesterol ratio were higher in group-I. In regression analysis CRP, LDL-cholesterol, non-HDL-cholesterol, TG/non-HDL-cholesterol ratio are independent risk factors on annual decline of eGFR. There was also a correlation between CRP and total cholesterol, total cholesterol/HDL-cholesterol, triglyceride/non-HDL-cholesterol, triglyceride/HDL-cholesterol ratios, and non-HDL-cholesterol. It was understood that cholesterol levels affect renal function through the inflammation. Similar to current study, clinical studies have reported a relationship between renal dysfunction and dyslipidemia (13,14). In a study conducted with 48054 participants, total cholesterol, triglyceride, non-HDL-cholesterol, triglyceride/HDL-cholesterol ratio, LDL-cholesterol /HDL-cholesterol ratio was higher in CKD group compared to control group. In regression analysis triglyceride/HDL-cholesterol ratio and non-HDL-cholesterol /HDL-cholesterol ratio are independent risk factors of CKD progression (OR:1.121; OR:1.14 respectively) (15). In a meta-analysis, high intensity statins were found to improve decline in eGFR in population with CKD not requiring dialysis compared control (16).

In the current study, both in the regression analysis and in the comparison of groups, it was found that having an acquired or congenital solitary kidney did not affect the annual change of eGFR. Whereas, eGFR was lower in the acquired solitary kidney group both in the first and last admission. The reason for this low level may be due to the fact that the patients are a little older and comorbidities such as cardiovascular diseases are more common. In addition, the factor causing nephrectomy in individuals with acquired solitary kidney may have indirectly affected the other healthy kidney. Studies have shown that the risk of developing CKD is slightly higher in patients with acquired solitary kidney (8,17). Jaoude et al. (17) reported an inverse relationship between GFR and follow-up time in patients with acquired solitary kidney but not in those with congenital solitary kidney. They suggested that the adaptive response following renal mass reduction may begin much earlier in the case of congenital solitary kidney than in that of acquired solitary kidney. Furthermore because mature glomeruli have low mitotic activity, acquired solitary kidney can have a worse functional adaption.

The present study had some limitations. It was a retrospective study and the patients' medical treatments, body mass indexes and habits such as smoking and alcohol could not be evaluated completely. A single measurement of cholesterol levels and CRP may not provide sufficient accuracy for predicting the renal outcome.

CONCLUSION

Individuals with solitary kidneys have a higher risk of developing CKD. Inflammation and dyslipidemia must be paid attention to protect GFR. Besides the atherosclerosis in the microcirculation, dyslipidemia affect GFR through inflammation. Having a congenital or acquired single kidney has no effect on the annual decline of eGFR

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Clinical Research Ethics Committee of Ankara Training and Research Hospital (Date: 21.04.2020, Decision No: E-20/201).

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The author has no conflicts of interest to declare.

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Author Contributions: The author declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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