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KIDNEY HEALTH CONSEQUENCES OF PATIENTS WITH GLOMERULONEPHRITIS; BEFORE AND AFTER SARS-COV2 INFECTION

SARS-COV2 ENFEKSİYONUNDAN ÖNCE VE SONRASI GLOMERÜLONEFRİTLİ HASTALARIN BÖBREK SAĞLIĞI

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ABSTRACT

Objective: The virus that causes severe acute respiratory syndrome (SARS-CoV-2) was first identified in Wuhan, China, in December 2019. Recent studies have proven that SARS-CoV-2 is also a nephrotrophic virus.

Methods: Our study aimed to evaluate kidney function and general kidney health of patients with previously diagnosed glomerular diseases and follow-up after SARS-CoV-2 infection. For this purpose, the data of 36 patients who were diagnosed with and routinely followed up for glomerulonephritis and had SARS-CoV-2 infection at the Kocaeli University Faculty of Medicine Hospital nephrology outpatient clinics between January 2020 and January 2022 were examined before and after the infection.

Results: No significant differences were observed in serum creatinine, estimated glomerular filtration rate, and 24-hour urine protein values after infection. There was an increase in platelet and albumin levels following the SARS-CoV-2 infection. A significant decrease was detected in 24-hour urine creatinine values.

Conclusion: The results of the study showed that kidney function and general kidney health of patients with SARS-CoV-2 infection diagnosed with glomerulonephritis were not different when compared to their condition before SARS-CoV-2 infection.

Keywords: COVID-19 infection, glomerulonephritis, chronic kidney disease, vaccine

ÖZ

Amaç: Şiddetli akut solunum yolu sendromuna neden olan virüs (SARS-CoV-2) ilk olarak Aralık 2019'da Çin'in Wuhan kentinde tanımlandı. Son çalışmalar SARS-CoV-2'nin de nefrotrofik bir virüs olduğunu kanıtlamıştır.

Yöntem: Çalışmamızın amacı daha önce glomerüler hastalık tanısı konmuş hastaların böbrek fonksiyonlarını ve genel böbrek sağlıklarını değerlendirmek ve SARS-CoV-2 enfeksiyonundan sonra böbrek sağlığı kontrolunü yapmaktır. Bu amaçla Ocak 2020 ile Ocak 2022 arasında Kocaeli Üniversitesi Tıp Fakültesi Hastanesi nefroloji polikliniklerinde glomerülonefrit tanısı konulan ve rutin olarak takibi yapılan, SARS-CoV-2 enfeksiyonu olan 36 hastanın verileri enfeksiyondan öncesi ve sonrası olarak incelendi.

Bulgular: Enfeksiyondan sonra serum kreatinin, tahmini glomerüler filtrasyon hızı ve 24 saatlik idrar protein değerlerinde anlamlı bir fark gözlenmedi. SARS-CoV-2 enfeksiyonundan sonra trombosit ve albümin seviyelerinde artış görüldü. 24 saatlik idrar kreatinin değerlerinde anlamlı bir düşüş tespit edildi.

Sonuç: Çalışmanın sonuçları, glomerülonefrit tanısı konulan SARS-CoV-2 enfeksiyonlu hastaların böbrek fonksiyonlarının ve genel böbrek sağlıklarının, SARS-CoV-2 enfeksiyonundan önceki durumlarıyla karşılaştırıldığında farklı olmadığını gösterdi.

Anahtar Kelimeler: COVID-19 enfeksiyonu, glomerülonefrit, kronik böbrek hastalığı, aşı

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Introduction

The virus that causes severe acute respiratory syndrome (SARS-CoV-2) was first identified in December 2019. Within months, the virus spread throughout the world and was declared a pandemic at the beginning of March 2020. SARS-CoV-2, viral pneumonia, and multi-organ involvement.[1] Chronic kidney disease has also been identified as a risk factor because it causes immune deficiency.[2]

Possible targets and facilitating factors for the virus to cause infection in human cells are cell membrane-bound receptor Angiotensin Converting Enzyme 2 (ACE2), Transmembrane Serine Protease 2 (TMPRSS2), Furin and Basigin (CD147). The expression of ACE2, TMPRSS2, Furin, and CD147 is not specific to the lung, and their expression in various tissues, including the brain, intestine, and kidney, may expose these organs to SARS-CoV-2 infection.[3] Renal parenchymal cells, specifically proximal tubular cells, secrete high levels of ACE2. The expression of genes related to injury, inflammation, and fibrosis is increased in kidney tubule cells and podocytes in which SARS-CoV-2 RNA was found.[4] Many studies have reported that SARS-CoV-2 RNA and protein were detected using electron microscopy in the kidneys of COVID-19 patients.[5]

In a post-mortem renal histopathologic study of SARS-CoV-2 patients; It was shown that 85% of patients had AKI and 74% had acute glomerular damage.[6] In another study conducted in living patients, biopsies were performed. The pathological conditions identified included acute tubular damage, minimal change disease, membranous glomerulonephritis, anti-GBM disease, and lupus nephritis. SARS-CoV-2 infection has been reported to have the potential to affect innate or adaptive immune responses, triggering new glomerular diseases or exacerbating pre-existing autoimmune conditions.

Thus, further attention should be focused to the long-term impact of SARS-CoV-2 infection and kidney function monitoring. This study aimed to evaluate the kidney function and general kidney health of patients with previously diagnosed glomerular diseases and follow-up after SARS-CoV-2 infection.

Methods

This study included 36 patients diagnosed with glomerulonephritis who had SARS-CoV-2 infection and were followed up at Kocaeli University Faculty of Medicine Hospital Nephrology Outpatient clinics between January 2020 and January 2022.

During the data collection process, all patients who were diagnosed with glomerulonephritis after renal biopsy using the hospital automation system retrospectively and who had COVID-19 were included in the study. In light of the fact that the antiviral medication molnupiravir was effective against SARS-CoV-2 and was available in our country from 2022 onwards, none of the patients received such treatment. However, all inpatients were administered low molecular weight heparin and oral or intravenous prednol treatment.

Patient age, sex, comorbidities, laboratory values (hemoglobin, hematocrit, platelet, urea, BUN, creatinine, eGFR, aspartate aminotransferase, alanine aminotransferase, total protein, albumin, electrolyte values, 24-hour urine protein, creatinine, albumin, C-reactive protein, low-density lipoprotein, and uric acid), and pathology results were collected from the hospital electronic data system. Patients with a history of malignancy and pregnant women were excluded from this study. Patients with an average follow-up of two months (1-3mos) after SARS-CoV-2 infection were included in the study. None of the patients received any antiviral treatment for SARS-CoV-2 as there was no effective treatment available at that time.

This study complied with the principles of the Declaration of Helsinki. Ethical approval for the study was obtained from the ethics committee of Kocaeli University Hospital (GOKAEK-2021/170).

Statistical Analysis

Data analysis was performed using IBM SPSS 20.0 program. The Shapiro-Wilk test was used to assess conformity with a normal distribution. Normally distributed variables were expressed as mean standard deviation and non-normally distributed variables as medians. Frequencies were used to represent categorical variables. Differences between the dependent samples were examined using the Wilcoxon signed-rank test. The Mann-Whitney U test was used to determine the differences between the groups. The chi-squared test was used to analyse the relationships between categorical variables. To evaluate the two-way hypotheses, we considered the differences in biochemical parameters to be statistically significant at $p<0.05$.

Results

We retrospectively scanned the data of 36 patients who had SARS-CoV-2 infection with a previous diagnosis of glomerulonephritis and were followed up between January 2020 and January 2022, using the hospital information registry data system and patient files. Seventeen patients were female (47.2%) and 19 were male (52.8%). The mean age of the patients was 43 years (range: 19–68 years). 44% of the patients had been diagnosed with glomerulonephritis for more than five years.

As for the comorbid conditions, 20 (55.5%) patients had comorbidities namely: diabetes mellitus (9/25%), hypertension (16/44.4%), hypothyroidism (4/11.1%), and hyperlipidaemia (8/22.2%).

Regarding the subtypes of glomerulonephritis according to the kidney biopsy results, nine (25%) patients were diagnosed with IgA nephropathy, seven (19.4%) with FSGS, six (16.7%) with membranous nephropathy, four (11.1%) with membranoproliferative glomerulonephritis,

three (8.3%) with minimal change disease, two (5.6%) with chronic glomerulonephritis, two (5.6%) with lupus nephritis, two (5.6%) with mesangioproliferative glomerulonephritis, and one (1%) with tubulointerstitial nephritis 2.8).

The cases were grouped according to the COVID-19 dates: 18 people (50%) had contracted SARS-CoV-2 in 2020 and 18 (50%) in 2021. Twenty-eight of 36 cases (77.8%) recovered simultaneously, while 8 (22.2%) required hospitalization. Thoracic tomography was not necessary in 24 patients (66.6%). Among the 12 patients who underwent the procedure, seven (58.3%) exhibited pneumonic infiltrations, while five (41.6%) did not.

Four people did not receive the COVID-19 vaccine throughout the pandemic and 32 were vaccinated. Of the 32 patients, 19 (59.3%) had received the vaccine prior to being infected with SARS-CoV-2, while 13 (40.6%) had received the vaccine afterwards. Of the eight hospitalized patients, six (75%) did not receive the vaccine. The 24-hour urine albumin and protein levels of people who were hospitalized due to SARS-CoV-2 infection were found to be significantly higher than those of people who were not hospitalized (Table 3).

There was a substantial increase in platelet and albumin levels following SARS-CoV-2 infection (Table 1, Figure 1 and 2). A significant decrease was detected in 24-hour urine creatinine values (Figure 3) and spot urine erythrocyte values ($p < 0.05$). No significant changes were observed in other laboratory values (Tables 1 and 2).

The disease activity spectrum of the patients was as follows: 25 (69.4%) were in remission without taking immunosuppressives, 9 (25%) were in remission with ongoing immunosuppressive therapy, and 2 (5.6%) were not in remission despite immunosuppressive agents. Immunosuppressant agents included corticosteroids (3 patients), cyclosporine (1 patient), corticosteroids+CSI (2 patients), corticosteroids+ tacrolimus (3 patients), corticosteroids+cyclophosphamide (1 patient), and corticosteroids+MMF (1 patient).

Table 1. The change in laboratory values before and after SARS CoV-2 infection

Parameter	Before SARS-CoV-2 infection Median (IQR)	After SARS-CoV-2 infection Median (IQR)	p value
Hemoglobin, g/dL	13.7 (13.1-14.9)	13.8 (12.9-14.9)	0.414
Hematocrit, %	41.2 (38.6-43.8)	40.3 (37.8-43.2)	0.385
Platelet, μ/L	255,000 (216,625-314,500)	272,800 (215,500-314,500)	**0.044**
Urea, mg/dl	30 (24.3-43.7)	30.4 (24.9-45.3)	0.437
BUN, mg/dl	14.7 (11.8-23.6)	14 (11.9-21)	0.120
Creatine, mg/dl	0.82 (0.71-1.16)	0.82 (0.74-1.18)	0.566
GFR, ml/dk	101.5 (69.1-111.2)	95.7 (66.9-114.1)	0.623
AST, U/L	20.3 (16-25)	19.5 (14.2-25.7)	0.329
ALT, U/L	22 (14.2-33.2)	21.4 (13.2-27.7)	0.966
Total protein, g/dl	7.1 (6.7-7.4)	7 (6.7-7.5)	0.622
Albumin, g/dl	4.3 (3.9-4.4)	4.4 (4-4.5)	**0.048**
Sodium, mEq/L	139.9 (138.1-141)	139 (138-140.4)	0.214
CRP, mg/dl	2.4 (1.1-5)	2.1 (1.2-4.5)	0.981
LDL, mg/dl	126 (105-173)	114 (95-144)	0.128
24-hour urine protein mg/day	683 (284.6-2.105.8)	921.3 (320.2-1.737.4)	0.937
24-hour urine albumin	375.9 (56.9-1492.3)	360.7 (70.2-1107.4)	0.789
Spot urine erythrocyte	1.5 (0-7.7)	1 (0-3)	**0.026**

BUN: Blood Urea Nitrogen AST: Aspartate Aminotransaminase ALT: Alanine Transaminase CRP: C reactive protein
IQR: Interquartile range

Table 2. The change in laboratory values before and after SARS CoV-2 infection

Parameter	Before SARS-CoV-2 infection Mean ± SD	After SARS-CoV-2 infection Mean ± SD	p value
Chloride, mEq/L	102.5 ± 3.5	101.8 ± 2.5	0.215
Potassium, mg /dL	4.4 ± 0.4	4.6 ± 0.42	0.086
Phosphorus, mg/dL	3.5 ± 0.6	3.6 ± 0.7	0.262
Uric acid, mg/dL	6.1 ± 1.53	5.8 ± 1.54	0.212

SD: Standard deviation

Table 3. Values before and after SARS-CoV-2 infection of hospitalized patients and out-patients

Parameter 1/2	Hospitalization Median (IQR)	No hospitalization Median (IQR)	p* value
Urea 1, mg/dl	36.5 (26.8-59.9)	29 (21.6-42.7)	0.168
Urea 2, mg/dl	43.9 (23.8-41.6)	28.4 (29.2-70.7)	**0.044**
Urea difference	3.2 (-4.5-5)	-2.5 (-5.5-3.6)	0.358
p value**	0.674	0.269	-
BUN 1, mg/dl	20.4 (14.5-28.9)	14 (11.1-20)	0.107
BUN 2, mg/dl	20.3 (13.5-32.9)	13.2 (11-19.8)	**0.044**
BUN difference	0.0 (-9.4-2.3)	-1.6 (-3.7-1.5)	0.780
p value**	0.833	0.113	-
K 1, mmol/L	4.6 (3.9-4.9)	4.5 (4.2-4.7)	0.955
K 2, mmol/L	4.8 (4.7-5.2)	4.5 (4.2-4.7)	**0.012**
K difference	0.19 (0.09-0.76)	0.11 (-0.35-0.41)	0.193
p value**	**0.018**	0.487	-
P 1, mg/dl	3.2 (3-3.8)	3.5 (3.2-3.9)	0.339
P 2, mg/dl	3.9 (3.3-4.5)	3.4 (3-4.1)	0.221
P difference	-0.7 (-0.8 – (-0.5))	-0.005 (-0.53-0.5)	**0.015**
p value**	0.123	0.973	-
24-hour urine albumin 1, mg/dl	1703.2 (586.5-3097.6)	199.1 (40.9- 857.8)	**0.006**
24-hour urine albumin 2, mg/dl	1611.4 (685.2-2248.1)	212.8 (56.2-1037.1)	**0.012**
24-hour urine albumin difference	-183.9 (-1109.4-550.2)	16.1 (-38.9-157.3)	0.466
p value**	0.575	0.327	-
24-hour urine protein 1, mg/dl	2398.4 (893.1-4197)	476.8 (213-1399.2)	**0.009**
24-hour urine protein 2, mg/dl	2000.4 (1099.7-2697.7)	485.8 (310.2-1605.4)	**0.044**
24-hour urine protein difference	-215.6 (-2369.4-466.5)	20.1 (-101.9-221.7)	0.489
p value**	0.401	0.569	-

BUN: Blood Urea Nitrogen, K: Potassium, Parameter 1: Before SARS-CoV-2 infection, Parameter 2: After SARS-CoV-2 infection, IQR: Interquartile range

*p value between group analysis, **p value within group analysis

Table 4. Values before and after SARS-CoV-2 infection of hospitalized patients and outpatients

Parameter 1/2	Hospitalization Median (IQR)	No hospitalization Median (IQR)	p* value
P 1, mg/dl	3.2 (3-3.8)	3.5 (3.2-3.9)	0.339
P 2, mg/dl	3.9 (3.3-4.5)	3.4 (3-4.1)	0.221
P difference	-0.7 (-0.8 – (-0.5))	-0.005 (-0.53-0.5)	**0.015**
p value**	0.123	0.973	-
24-hour urine albumin 1, mg/dl	1703.2 (586.5-3097.6)	199.1 (40.9- 857.8)	**0.006**
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24-hour urine protein 2, mg/dl	2000.4 (1099.7-2697.7)	485.8 (310.2-1605.4)	**0.044**
24-hour urine protein difference	-215.6 (-2369.4-466.5)	20.1 (-101.9-221.7)	0.489
p value**	0.401	0.569	-

P: Phosphorus, Parameter 1: Before SARS-CoV-2 infection, Parameter 2: After SARS-CoV-2 infection, IQR: Interquartile range

*p value between group analysis, **p value within group analysis

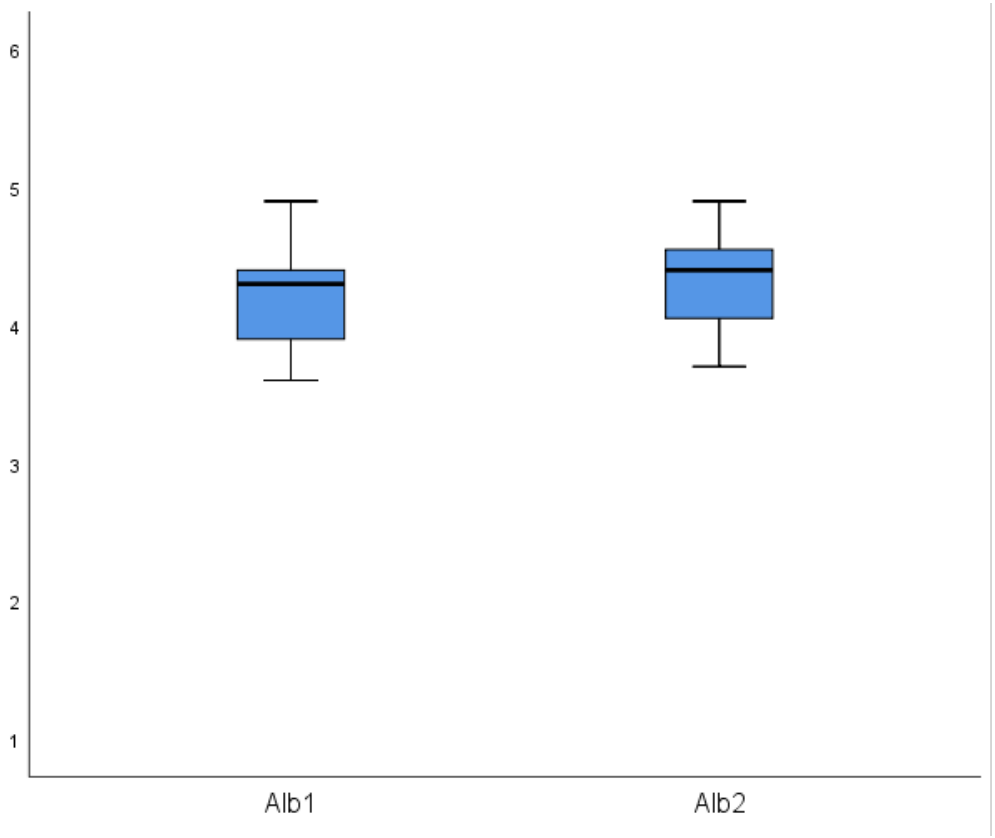

Figure 1. Change in serum albumin levels after COVID-19 infection

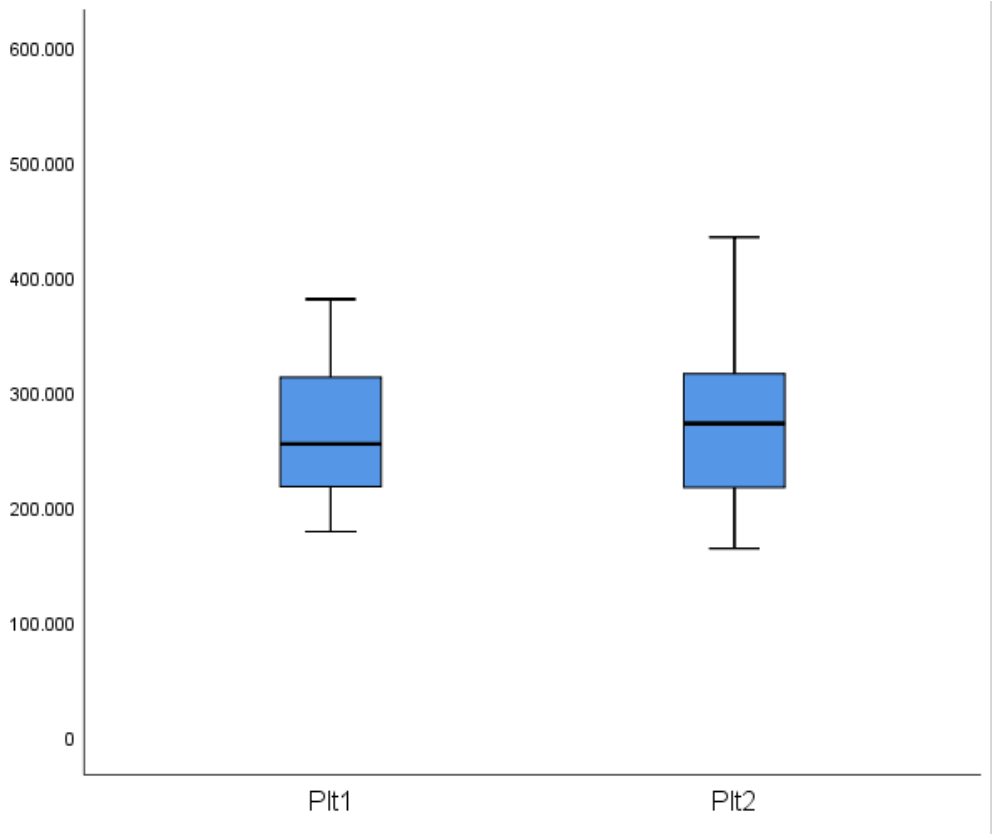

Figure 2. Changes in thrombocyte number after COVID- 19 infection

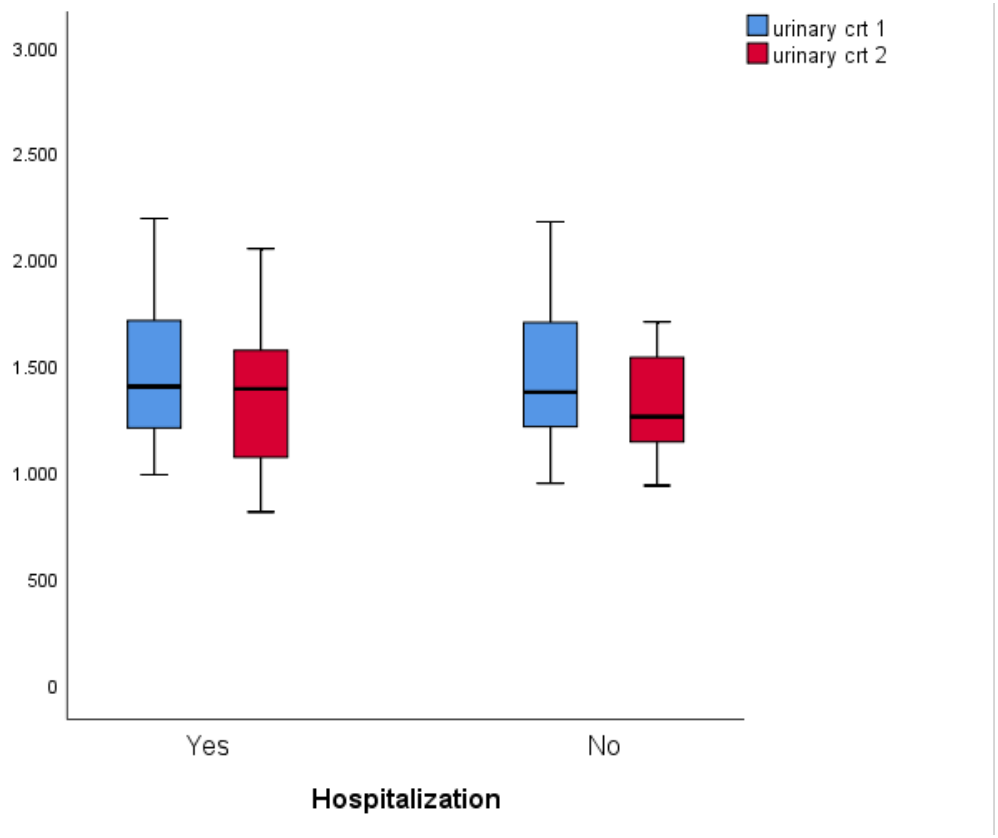

Figure 3. Decrease in 24-hour urine creatinine secretion after COVID-19 infection in hospitalized and non-hospitalized patients

Discussion

Unlike other studies on COVID-19 and glomerular diseases, our study focused on a younger population.[7] Studies have shown that kidney cells secrete proteins that can induce SARS-CoV-2 infection. However, how often kidney cells are infected and whether this infection contributes to kidney disease are not clearly delineated.[8] A study on kidney biopsy results in patients infected with SARS-CoV-2 showed that although acute tubular damage was more common, both glomerular and tubular damage developed.[7] Another study examining postmortem biopsy pathologies showed that glomeruli were preserved in most kidneys and that AKI occurring in most of the deceased was associated with acute tubular damage.[9]

In our study, no significant differences were observed in the proteinuria values of the patients diagnosed with glomerulonephritis. A study has shown that ACE 2, the functional receptor of SARS-CoV-2 infection, is highly expressed in tubules rather than glomeruli in the kidney. Accordingly, the main effect of SARS-CoV-2 infection on the kidney is thought to be tubular damage. The observation that the tubulointerstitial infiltration by inflammatory cells like macrophages during acute tubular damage while the glomeruli are usually preserved confirms this "***primarily tubular***" hypothesis.[10] This might also explain why a significant change in the magnitude of proteinuria in our patients did not occur following SARS-CoV-2 infection. Additionally, the patients included in the study had SARS-CoV-2 infection without hospitalization or serious organ failure. Epidemiologically, they were infected with SARS-CoV-2 in the second wave of the pandemic, during which milder cases were more prevalent than in the initial days of the pandemic. This may have contributed to the more favourable clinical presentation of the patients.

Philipp et al. demonstrated that out of 59 individuals with a history of immune-mediated glomerular illness and SARS-CoV-2 infection, 16.9% experienced acute kidney injury.[11] In our study, we compared pre- and post-COVID serum creatinine levels in our patients and observed no significant differences. Likewise, the average pre/post-COVID eGFR values were not significantly different, despite a slight increase following SARS-CoV-2 infection. It is important to emphasize that retrospectively assessing the values in our study, without considering them individually, may lead to a misleading understanding of AKI.

Although many studies have mainly reported tubular involvement with SARS-CoV-2 infection, it has also been shown that AKI may develop secondary to glomerular involvement with heavy proteinuria and podocyte damage.[12] It has been shown that advanced age, obesity, hypertension and diabetes are among the risk factors for AKI to develop. Most studies in the literature included hospitalized COVID-19 patients with a relatively higher mean age.[13] The reason for the insignificant correlation between creatinine level and eGFR changes in our study was attributed to the younger age of the patients and the mild nature of the infection without hospitalization.

In a study by Wang et al., the risk of worsening glomerulonephritis after COVID-19 was found to be 35%. It has also been shown that patients diagnosed with glomerulonephritis and vaccinated with the COVID-19 vaccine had lower rates of worsening. In our study, we discovered that the vaccination rate among hospitalized patients was relatively low at 75%, and there was no noticeable protective effect associated with vaccines. The reason behind this variation is believed to be due to the fact that our study's cases primarily experienced the infection during the second wave. Studies in the literature have shown that the development of AKI is less common in patients treated in the intensive care unit in the second wave of COVID-19 compared with first-wave patients.[14]

In our study, the mean platelet count following SARS CoV2 infection significantly increased compared with that before infection. However, studies reporting the opposite do exist in hospitalized COVID-19 patients with thrombocytopenia and an associated suboptimal prognosis.[14] We believe that the reason for the increase in the platelet count in our patients might be the homeostatic response to platelet consumption. There is still uncertainty and no consensus regarding the normal platelet count in hospitalized COVID-19 patients.[15] The hemoglobin levels in our patients before and after COVID-19 infection were not different.

In our study, no significant differences were detected in AST, ALT, and total protein levels after SARS-CoV-2 infection compared to pre-infection levels. A previous study has shown increased AST and ALT levels, indicating liver damage to the level of 1-2 times the upper limit of normal in the acute phase of SARS-CoV-2 infection. A COVID-19 study found that older people with liver disease, alcohol addiction, and obesity were more likely to suffer from liver damage. The prognosis for COVID-19 patients with liver damage was generally good, and in 95.6% of patients, liver function tests returned to normal within two months of discharge,[16] which might be the reason why no statistically significant difference was observed in transaminase levels after the infection period in our study. Furthermore, none of the patients in the present study developed serious organ failure. Therefore, serum albumin levels were higher after the COVID-19 infection. This finding could be incidental and does not correlate with the findings of previous studies. Previous studies have demonstrated that low serum albumin levels are associated with disease severity, despite the discrepancies in albumin levels among study populations based on factors such as age, inflammation status, sex, and geographical location.[17] This finding could be attributed to the fact that our patients remained relatively healthy when compared with hospitalized and more severely ill patients.

Studies have shown that non-COVID-19 viral infections play a role in the recurrence of nephrotic syndrome and IgA nephropathy in pediatric patients.[17] Although the mechanism underlying disease exacerbation is not fully understood, a dysregulated immune response to infection has been reported. It has been shown that none of the patients in these reports developed serious disease, and all responded to glucocorticoid therapy without serious complications. In contrast, some studies have suggested that lymphopenia caused by SARS-CoV-2 may have a protective effect against the progression of glomerular diseases such as drugs that suppress the immune system. Studies conducted with glucocorticoids have shown that dexamethasone reduces mortality in ARDS due to SARS-CoV-2, but has no positive or negative effect on moderately severe infection.[18] In a single-center study, cyclosporine use reduced mortality. The use of immunosuppressive agents such as rituximab, cyclophosphamide, and mycophenolate has been shown to result in worse outcomes in patients with COVID-19.[19]

No significant difference was observed in LDL levels before and after SARS-CoV-2 infection in patients with glomerulonephritis. Researchers have demonstrated that dyslipidemia, especially when accompanied by obesity, exacerbates the severity and progression of COVID-19.[19] However, there are no studies in the literature on the effects of SARS-CoV-2 infection on LDL levels.

The limitations of this study were the short follow-up period for consequences, limited number and younger age range of patients, and exclusion of more serious forms of COVID-19 infection.

Many patients developed SARS-CoV-2 infections during the second wave of the COVID-19 pandemic. Most patients recover from SARS-CoV-2 infection without hospitalization or ARDS development. Most hospitalized patients were from a group of patients who had not been vaccinated with the COVID-19 vaccine. Considering proteinuria and eGFR values after SARS-CoV-2 infection, there was no change in the disease activity of primary glomerulonephritis.

Ethical Approval

This study complied with the principles of the Declaration of Helsinki. Ethical approval for the study was obtained from the ethics committee of Kocaeli University Hospital (GOKAEK-2021/170).

Conflict of Interest

The authors declare that they have no conflict of interest.

Author Contributions

BD, OG, ME, SGB: Concept-Design; SGB, OG: Supervision; BD, ME: Data Collection and/or Processing; BD, ME, SGB, OG: Analysis and/or Interpretation; SGB, BD, OG: Literature Review; BD, OG, SGB, ME: Writer; SGB, OG: Critical Review

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