The Intercontinental Journal of
Internal Medicine

DOI: 10.51271/ICJIM-0025

Original Article

The relationship between albumin level and the development of acute kidney injury and mortality in critically COVID-19 patients

Sedrettin Koyun[1], İhsan Solmaz[2], Ramazan Danış[3], Yunus Tekin[4], Ömer Faruk Alakuş[5], Yusuf Yakut[6], Bilgin Bahadır Başgöz[2]

[1]Department of Internal Medicine, Kızıltepe State Hospital, Mardin, Turkiye
[2]Department of Internal Medicine, Gazi Yaşargil Training and Research Hospital, University of Health Sciences, Diyarbakır, Turkiye
[3]Department of Nephrology, Gazi Yaşargil Training and Research Hospital, University of Health Sciences, Diyarbakır, Turkiye
[4]Department of Internal Medicine, Siverek State Hospital, Şanlıurfa, Turkiye
[5]Department of Internal Medicine, Bismil State Hospital, Diyarbakır, Turkiye
[6]Department of Internal Medicine, Kocaköy State Hospital, Diyarbakır, Turkiye

Cite this article: Koyun S, Solmaz İ, Danış R, et al. The relationship between albumin level and the development of acute kidney injury and mortality in critically COVID-19 patients. *Intercont J Int Med*. 2024;2(1):7-11.

Corresponding Author: Sedrettin Koyun, mdsedriko@gmail.com

Received: 24/11/2023 ◆ **Accepted**: 27/02/2024 ◆ **Published**: 29/02/2024

ABSTRACT

Aims: TThe impact of the coronavirus pandemic, which began in 2019, has considerably diminished; however, its effects still persist. While respiratory symptoms have been prominent during the pandemic, acute kidney disease has emerged as a significant contributor to morbidity and mortality. This study aims to demonstrate that serum albumin levels can serve as a predictor for acute kidney injury (AKI) and mortality, owing to their cost- effectiveness and feasibility for use across various healthcare centers. The goal is to contribute to the reduction of AKI development and mortality rates.

Methods: The study was conducted by analyzing data from a total of 350 patients admitted to intensive care units of a training and research hospital due to COVID-19 between March 1, 2020, and April 30, 2021. Of these patients, 179 (51%) were male, and 171 (49%) were female. The data were examined retrospectively. Patients were categorized into two groups based on serum albumin levels: 2.5 mg/dl (severe hypoalbuminemia) and 2.5-3.5 mg/dl (mild hypoalbuminemia). Subsequently, the patients were further categorized into groups based on the presence or absence of AKI, and these groups were statistically compared. Demographic data, clinical information, and laboratory values of the patients were assessed. The diagnosis of acute kidney injury (AKI) was made according to KDIGO criteria. Data were compared using the SPSS version 24 software.

Results: Among the 350 patients included in the study, 115 had serum albumin levels ?2.5 mg/dl (severe hypoalbuminemia), and 235 had levels in the range of 2.5-3.5 mg/dl (mild hypoalbuminemia). Among those who developed AKI, the average albumin level was found to be 2.68 mg/dl, whereas in the group without AKI, the average albumin level was 2.76 mg/dl. Out of the total, 201 (57%) patients developed AKI, and 82% of them experienced mortality. In contrast, among the 149 (43%) patients who did not develop AKI, the mortality rate was 42%. Further subgroup analysis revealed the highest mortality rate of 88% among patients with severe hypoalbuminemia and AKI development. Conversely, the lowest mortality rate of 40% was observed in patients with mild hypoalbuminemia and no AKI.

Conclusion: This study aims to establish that low serum albumin levels should be regarded not only as a negative acute-phase reactant but also as a predictive factor for complications including acute kidney disease development and mortality. Lower albumin values are correlated with higher complication rates and increased mortality. Additionally, due to its widespread availability and low cost, serum albumin is a cost-effective diagnostic tool that can be utilized in almost any healthcare setting.

Keywords: COVID-19, hypoalbuminemia, acute kidney injury, intensive care unit, mortality

INTRODUCTION

According to the current data of the World Health Organization, approximately 769 million Coronavirus dissease–19 (COVID-19) cases have been seen worldwide and approximately 7 million of them have died.[1] Although the most common cause of COVID-19 infection-related mortality is acute respiratory failure, it is known that COVID-19 infection causes serious complaints by involving the cardiological, gastrointestinal, hepatological, nephrological, neurological, hematological, ocular and cutaneous systems in addition to respiratory system complaints.[2] Kidneys have a special importance among these organs affected by COVID-19 infection. The rate of developing acute kidney injury in those with COVID-19 infection is 28%. This rate increases up to 46% in those who are hospitalized and

followed up in the intensive care unit.[3] The mortality rate of patients who develop AKI due to COVID-19 infection is more than 5 times higher than that of those who do not develop AKI.[4]

Therefore, identifying high-risk patients for the development of AKI among COVID-19-related hospitalized patients, closely monitoring high-risk patients for the development of AKI, and taking measures to prevent the development of AKI, if possible, preventing the development of AKI damage is very important in order to increase the survival of patients and prevent the development of morbidity. Various tests have been developed for this purpose and are known to be useful in the early diagnosis of AKI. These can be listed as Cystatin-C, NGAL and KIM-1.[5] However, these tests are used only for clinical research purposes because they are expensive and not available in most centers. Therefore, they are not cost effective.

In this study, we aimed to evaluate the predictive ability of serum albumin level in predicting the development of AKI, morbidity and mortality in patients diagnosed with COVID-19 and treated in the intensive care clinic.

METHODS

Ethics

The study was carried out with the permission of Health Sciences University Gazi Yaşargil Training and Research Hospital Ethics Committee (Date: 28.04.2021, Decision No: 2021/840), and it was conducted in accordance with the Declaration of Helsinki.

Setting and Participants

This retrospective, single-center and observational study included patients aged 18 and over who were treated with a diagnosis of COVID-19 infection in the intensive care unit of a training and research hospital between 01.03.2020-30.04.2021. The participants were diagnosed with COVID-19 infection by PCR method. Patients with previously diagnosed glomerulonephritis or nephrotic syndrome, end-stage renal failure, end-stage heart failure, cirrhosis, malnutrition and protein-losing enteropathy, and patients under 18 years of age were not included in the study.

Patient Characteristics and Procedures

For all participants, demographic characteristics such as age and gender; clinical data such as diabetes hypertension, coronary heart disease, blood pressure value at the time of admission, initial oxygen support, length of hospital stay, need for dialysis, type of discharge; laboratory findings such as albumin amount, white blood cell count, neutrophil count, lymphocyte count, hemoglobin, platelets, glucose, urea, creatinine, aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, potassium, sodium, procalcitonin, ferritin, blood gas and C-reactive protein (CRP) were learned from the patient files retrospectively. Those with albumin values between 2.5 g/dl and 3.5 g/dl were defined as patients with mild hypoalbuminemia, and those with <2.5 g/dl were defined as patients with severe hypoalbuminemia (**Table 1**).

AKI development status in the patients was decided using the Kidney Dissease Improving Global Outcomes-Acute Kidney Injury (KDIGO-AKI) criteria and the patients were divided into 2 groups: those who developed AKI and those who did not develop AKI.

Table 1. Baseline characteristics of participants

Characteristic	Albumin≤ 2.5 n:115	2.5≤Albumin<3.5 n:235	p value
Age (year) mean±SD	69.5 (13.6)	65.4 (14.1)	**0.011**
Gender (%)	32.4±8.0	31.7±7.3	0.356
Male	17.4	33.4	
Female	15.4	33.7	
Mean blood pressure±SD (mm/Hg)			0.286
Systolic	124 (20.4)	126 (21)	
Diastolic	74.1 (13.5)	75.7 (13.8)	
Chronic diseases (%)			
Dm	8	24.6	0.122
HT	15.1	36.3	0.162
CAD	10	16.6	0.252
Initial oxygen support (%)			**0.002**
Mask+nasal	10	24	
Reservoir mask	4.2	12.8	
High flow	1.7	2	
CPAP	6.5	19.7	
Mechanical ventilation	9.4	8.2	
Hospitalization duration mean±SD	13.8 (min:2-max:90)	14.1 (min:2-max:129)	0.303
Need for dialysis (%)			0.422
Yes	4	6.3	
No	28.9	60.9	
Need for positive inotropes (%)			0.139
Yes	14	23.1	
No	18.9	44	
Albumin infusion (%)			**0.022**
Yes	21.4	35.1	
No	11.4	32	
Anti-inflammatory treatment (%)			**0.697**
Yes	14	27.1	
No	18.9	40	
Infective parameters (mean±SD)			
Crp	150 (84)	139 (80)	0.232
Procalcitonin	1.8 (4.5)	1.2 (3.9)	**0.009**
Ferritin	947 (776)	767 (654)	**0.016**
Hemogram parameters (mean±SD)	Albumin≤ 2.5 N:115	2.5≤Albumin<3.5 N:235	p value
Hgb	12.2 (2)	13.7 (8.7)	**<0.001**
Plt	269 (109)	266 (103)	0.86
Wbc	14.4 (6.7)	12.4 (6.7)	**0.003**
Neu	12.8 (6.6)	10.7 (6.3)	**0.001**
Lym	0.96 (0.62)	1.1 (1)	0.213
Neu/lym	21.5 (22)	14.2 (13.9)	**0.001**
Biochemical parameters (mean±SD)			
Urea	65 (47.5)	49.4 (27.3)	**0.002**
Sodium	138.7 (6.4)	135.7 (6)	**<0.001**
Potassium	4.3 (0.66)	4.23 (0.6)	0.496
Calcium	8.9 (0.5)	8.8 (0.5)	**0.034**
ALT	53.5 (75)	35 (27)	**0.01**
AST	58 (70)	45 (40)	0.104
Total bilurubin	0.9 (1.5)	0.6 (0.46)	**0.006**
D-dimer (mean±SD)	3089 (6718)	1513 (4007)	**<0.001**
Blood gas parameters (mean±SD)			
PH	7.35 (0.12)	7.38 (0.1)	**0.356**
HCO3	21.9 (5.5)	22.4 (4.2)	**0.585**
Lactate	2.9 (2.3)	2.8 (2.3)	0.785
AKI (n %)			**0.039**
Yes	75 (65)	126 (53)	
No	40 (34)	109 (46)	
Discharge type (n %)			**0.001**
Alive	25 (21.7)	92 (39.1)	
Death	90 (78.3)	143 (60.9)	

Dm: diabetes mellitus, HT: Hipertension, CAD: Coronary Artery Disease, Cpap:Continuous positive Airway Pressure, Crp: C-reaktive protein, Hgb: hemoglobine, Plt:platelet, Wbc:white blood cell, Neu: Neutrophile, Lym: Lymphocite, Ney/Lym: Neutrophile Lymphocite ratio, ALT: Alanine transaminase, AST: Aspartate transaminase, Hco3: Bicarbonate, AKI:acute Kidney Injury

The effect of the degree of hypoalbuminemia on the development of AKI and mortality and the mortality rates in patients who developed AKI were compared between the groups (Table 2).

Table 2. Albumin and acute kidney injury subgroups

Albumin(mg/dl)	n (%)	Acute kidney injury	n (%)
≤2.5 (severe hypoalbuminemia)	115 (67%)	Developed	201 (57%)
2.5-3.5 (mild hypoalbuminemia)	235 (33%)	Non-developed	149 (43%)
Total	**350 (100)**	**Total**	**350 (100)**

AKI:Acute kidney injury

Statistical Analysis

Statistical analyzes in our study were performed using IBM SPSS (version 24.0) package program. The suitability of the variables to normal distribution was examined using visual (histogram and probability graphs) and analytical methods (Kolmogorov-Smirnov test). Normally distributed numerical variables were analyzed using the "t test in independent groups" between two groups. Numerical variables that were not normally distributed were analyzed using the "Mann Whitney U test" between the two groups. "Chi-square analysis" and "Fisher's exact test" were used to compare nominal data. One Way Anova test was used to compare more than two groups. The power of all scoring models in the prediction of the mortality rate was tested with the receiver operator characteristics (ROC)-area under curve (AUC) analysis. ROC analysis was also used to state cut-off values and their sensitivity and specificity to estimate the risk of mortality in the ICU. Results are expressed with a 95% confidence interval. In statistical analysis, p values below 0.05 were considered statistically significant.

RESULTS

A total of 350 patients were included in our study. 179 (51%) of the participants were male. The total average age was 66.83 (min: 22-max: 102). The average age of men was 64.1±13.8 and the average age of women was 69.6±13.9. The initial albumin value was ≤2.5 mg/dl (severe hypoalbuminemia) in 115 of 350 patients and 2.5-3.5 mg/dl (mild hypoalbuminemia) in 235 patients. The mean initial creatinine in the severe hypoalbuminemia group was 1.15 (0.84) mg/dl, and in the mild hypoalbuminemia group, the initial creatinine average was 1.06 (0.49) mg/dl. The number of patients who developed AKI was 201 (57%) and AKI did not develop in 149 (43%) patients. The average albumin level was found to be 2.94 mg/dl in the mild hypoalbuminemia group that developed AKI, and 2.2 mg/dl in the severe hypoalbuminemia group that developed AKI.

When the follow-ups of the patients were examined, it was determined that hypoalbuminemia was correlated with AKI and mortality. When patients who developed AKI were evaluated, the rate of AKI development was found to be 53% in patients with mild hypoalbuminemia and 65% in patients with severe hypoalbuminemia ($p<0.05$). Death occurred in 166 (71.2%) of 201 patients who developed AKI. While death occurs in 69% of patients with mild hypoalbuminemia who develop AKI; this rate was found to be 73.3% in patients with severe hypoalbuminemia who developed AKI. AKI did not develop in the remaining 149 (43%) patients. The number of deaths in this group was 67 (44%). In our study, the total mortality rate was 66.6%. Death rate was 60.9% in the mild hypoalbuminemia group and this rate was 78.3% in the severe hypoalbuminemia group. This rate is statistically significant with the degree of low albumin level. (AUC:0.653; $p<0.05$). When the subgroups were compared among themselves, the highest mortality rate was seen in the patient group with severe hypoalbuminemia and AKI. The lowest mortality rate was seen in the group with mild hypoalbuminemia and without AKI (Table 3, 4).

Table 3. Acute kidney injury and mortality rates according to hypoalbuminemia groups

	AKI developed (n%)	Resulted in death (n%)
Mild hypoalbuminemia	126 (53.6)	143 (60.9)
Severe hypoalbuminemia	75 (65.2)	90 (78.3)
Total	**201 (57.4)**	**233 (66.5)**

AKI:Acute kidney injury

Table 4. Mortality rates according to hypoalbuminemia and acute kidney injury subgroups

	Mild Hypoalbuminemia (n%)	Severe Hypoalbuminemia (n%)
AKI non-developed	44 (40)	23 (59)
AKI developed	100 (79)	66 (88)
Total	**144 (61)**	**89 (77)**

AKI:Acute kidney injury

When the AKI groups were compared among themselves, statistical significance was detected in terms of age, length of stay, WBC count, neutrophil count, potassium level, D-dimer level and procalcitonin levels (Table 5).

Table 5. Comparison of acute kidney injury groups

Characteristic	AKI developed n:201	AKI non-developed n:149	p value
Age	68	65	**0.037**
Systolic blood pressure	124	127	0.303
Diastolic blood pressure	75	75	0.404
Hospitalization duration	12.3	15.8	**0.01**
Wbc	13.6	12.3	**0.045**
Neu	11.9	10.74	**0.047**
Lym	1.08	1.06	0.26
Neu/Lym	18	14.73	0.085
Hgb	13.5	12.91	0.934
Plt	271	262	0.487
ALT	44.2	38	0.183
AST	52	47	0.372
Total bilurubin	0.69	0.72	0.82
Calcium	8.8	8.9	0.469
Sodium	137	136	0.281
Potassium	4.3	4.1	**0.002**
CRP	147	136	0.085
D-dimer	2468	1440	**0.03**
Ferritin	891	740	0.142
Procalcitonin	1.8	1.05	**0.000**
Albumin	2.68	2.78	0.063

AKI: Acute kidney injury, Wbc: white blood cell, Neu: neutrophil, Lym: lymphocit, Hgb: hemoglobin, Plt: platelet, ALT: alanine aminotranferase, AST: aspartate aminotranferase

DISCUSSION

Our study was conducted with a total of 350 patients in a large center during the COVID-19 pandemic, by dividing patients with hypoalbuminemia into subgroups and

examining their effects on AKI development and mortality retrospectively. 179 (51%) of the participants were men and 171 (49%) were women. The death rate due to COVID-19 generally varies between 1.4-8%.[6] However, the development of acute kidney injury in addition to COVID-19 in patients followed in intensive care may cause the mortality rate to increase up to 80%, as some studies show.[7,8] Therefore, predicting AKI will play a key role in preventing mortality. In our study, in parallel with previous studies on albumin, it was determined that hypoalbuminemia caused both the development of AKI and an increase in mortality. However, unlike other studies, it is important that our study is conducted according to albumin levels and provides different perspectives.

In a meta-analysis by Silver et al.[3], which included 53 studies with COVID-19 patients and 30,657 patients, the rate of AKI development in non-intensive care patients was 12%, while this rate ranged between 35-57% in patients hospitalized in intensive care. In terms of AKI etiology, acute tubular damage stands out as the most common cause in patients followed in intensive care due to COVID-19.[9] Risk factors for the development of AKI are age, body mass index, acute circulatory and respiratory system failures, chronic liver disease, congestive heart failure, infections, peripheral vascular occlusive diseases, cancers, invasive procedures and high-risk surgeries.[10] Another important reason that precipitates the development of AKI is low albumin level.[8,11,12] In addition, markers that can predict AKI cases that occur due to intensive care hospitalization or various etiological reasons have been defined.[5] The most well-known of these are cystatin-C, NGAL (Neutrophil gelatinase-associated lipocalin) and KIM-1 (kidney injury molecule-1).

When the studies on this subject were reviewed, hypoalbuminemia was found to be a predictor for the development of AKI in both non-intensive care and intensive care patients, and it was also shown that the presence of hypoalbuminemia caused an increase in mortality.[11-13] Additionally, in a meta-analysis conducted by Hansrivijit et al.[13] in 2021, including 168,740 patients, it was shown that every 1 mg/dl decrease in albumin level caused a 1.68-fold increase in the development of AKI and a 1.18-fold increase in mortality. In the same study, it was determined that the risk for AKI development and mortality began to increase at albumin levels lower than 3.2 mg/dl. Studies on the effect of albumin level on the development of AKI and mortality are often designed to compare normal albumin levels with albumin levels ≤3.5 mg/dl.[7,14] Yang et al.[7] in a retrospective study with 740 patients were divided into 4 groups according to albumin levels. The highest mortality rate was found in the group with the lowest albumin level. Additionally, in a study conducted by Uyanık et al.[14] in Turkiye, the relationship between albumin values at the time of diagnosis and mortality was examined. The average albumin level was found to be 2.87±0.66 g/dl in deceased patients and 3.28±0.64 g/dl in living patients, and these values were statistically significant.

In our study, unlike previous studies, we aimed to retrospectively examine the relationship between serum albumin level and the development of AKI and mortality in COVID-19 patients and especially in intensive care patients. In our study, in parallel with the literature, a statistically high correlation was detected between the decrease in albumin level and the development of acute kidney injury and mortality (**Table 6**). However, in our opinion, it is important to conduct our study with patient groups with mild and severe hypoalbuminemia in terms of providing new perspectives.

Table 6. Albumin roc analysis in terms of Acute Kidney Injury development

Risk factor	AUC (95%)	Cut-off	P	Sensitivity (%)	Specificity(%)
	0.558 (0.497-0.619)				201 (57%)
Albumin	235 (33%)	2,65	0.064	45.3	37.6

AKI:Acute Kidney Injury

In the mild hypoalbuminemia group, the incidence of AKI was 53% and the mortality rate was 60%. In the group with severe hypoalbuminemia, the incidence of AKI was 65% and the mortality rate was 78.3%. When subgroup examinations were made, the highest mortality rate, reaching 90%, was seen in the group with severe hypoalbuminemia and AKI development; the lowest mortality rate, 40%, was found in the group with mild hypoalbuminemia and no AKI. As a result, as the albumin level decreased, more acute kidney disease and more death rates were detected. In our study, the total acute kidney injury incidence rate was found to be 57.4%, and the total death rate was 66.6% (**Table 7, 8**).

Table 7. Comparison of groups according to discharge method

Characteristic	Resulted in death n: 223	Not resulted in death n: 117	p value
Age	68.62	63.26	**<0.001**
Systolic blood pressure	124.52	128.9	**0.043**
Diastolic blood pressure	74.22	77.23	**0.013**
Hospitalization duration	13.3	14.89	**<0.001**
Wbc	13.78	11.64	**0.007**
Neu	12.23	9.81	**0.002**
Lym	0.97	1.28	**<0.001**
Neu/Lym	19.6	10.75	**<0.001**
Hgb	13.4	13	0.78
Plt	267	268	0.89
Alb	2.64	2.85	**<0.001**
Urea	60	43.4	**<0.001**
Cre	1.14	0.98	**0.003**
E-GFR	68	77	**0.003**
ALT	44.6	35.5	0.11
AST	53.2	43.7	0.13
Total bilurubin	0.71	0.69	0.69
Calcium	8.88	8.9	0.68
Sodium	137	135.8	0.12
Potassium	4.24	4.2	0.86
Crp	153	122	**0.001**
D-dimer	2284	1526	**0.03**
Ferritin	912	656	**0.001**
Procalcitonin	1.62	1.22	**<0.001**

Wbc: white blood cell, Neu: neotrophil, Lym: lymphocit, Hgb: hemoglobin, Plt: platelet, Alb: albumin, Cre: creatinine, E-GFR: estimated glomerular filtration rate, ALT: alanine aminotranferase, AST: aspartate aminotranferase

Table 8. Albumin Roc analysis for mortality

Risk factor	AUC (95%)	Cut-off	p	Sensitivity (%)	Specificity(%)
	0.653 (0.591-0.715)				201 (57%)
Albumin	235 (33%)	2,75	0.000	60.1	36.8

When the groups that developed and those that did not develop AKI were compared, there was a statistical significance between the groups in terms of age, length of stay, WBC, neutrophil count, potassium, d-dimer and procalcitonin. The average age of patients who developed

AKI was higher than those who did not develop AKI. All inflammation markers were detected higher in the group that developed AKI. There was a high correlation especially between the high initial procalcitonin value and the rate of AKI development (p<000). Similar risk factors were identified in the study conducted by Candido de Almeida et al.[15] Although there was no statistical significance between the mean albumin level in the groups that developed AKI and those that did not, there was a lower albumin level in the group that developed AKI (2.68 mg/dl versus 2.78 mg/dl).

When the effects of the parameters in our study on mortality were examined, age, hospitalization time, initial lymphocyte count, neutrophil/lymphocyte ratio, albumin level, initial urea, procalcitonin levels were found to be statistically highly significant (p<0.00). These findings were consistent with previous studies.[9,16] In studies conducted on this subject, male gender, diabetes, hypertension and coronary artery disease have also been identified as risk factors for mortality.[16] However, this could not be confirmed in our study (p>0.05). As mentioned before, this may be due to the difference in the patient group selected for our study.

Limitations

Since the patients included in our study were selected from patients hospitalized during the most intense period of the pandemic period, and as a natural result of retrospective studies, sufficient information could not be obtained about the patients' proteinuria, basal creatinine values and urine output amounts. Additionally, patients with normal albumin were requested to be included in the study as a control group, but only 4 of the patients examined in the same period were suitable for our study. Therefore, a control group could not be created. We continued our study by comparing subgroups of patients with hypoalbuminemia among themselves. In order to clarify this situation, prospective, large-scale, better designed studies are needed to predict the relationship between albumin values and AKI development, after eliminating the deficiencies that prevent us from giving clearer messages based on our study.

CONCLUSION

With this study, we aimed to show that low albumin is a predictive factor for the development of acute kidney disease and complications including mortality, rather than considering it only as an acute phase reactant. In our study, lower albumin levels were found to be associated with more complications and higher mortality rates. The availability of albumin in every center and the fact that it is a low-cost test is important in terms of cost-effectiveness. Other prospective, more comprehensive studies are needed to elucidate this issue.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of University of Health Sciences Gazi Yaşargil Training and Research Hospital Ethics Committee (Date: 28.04.2021, Decision No: 2021/840), and it was conducted in accordance with the Declaration of Helsinki.

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Author Contributions: All of the authors declared that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

Data Availability Statement: Data will be made available by the corresponding author on reasonable request.

REFERENCES

1. WHO Coronavirus (COVID-19) Dashboard | WHO Coronavirus (COVID-19) Dashboard With Vaccination Data [Internet]. [cited 2023 Aug 8]. Available from: https://covid19.who.int/
2. Lai CC, Ko WC, Lee PI, Jean SS, Hsueh PR. Extra-respiratory manifestations of COVID-19. *Int J Antimicrob Agents*. 2020;56(2):106024.
3. Silver SA, Beaubien-Souligny W, Shah PS, et al. The prevalence of acute kidney injury in patients hospitalized with COVID-19 infection: a systematic review and meta-analysis. *Kidney Med*. 2021;3(1):83-98.
4. Chávez-Valencia V, Orizaga-de-la-Cruz C, Lagunas-Rangel FA. Acute kidney injury in COVID-19 patients: pathogenesis, clinical characteristics, therapy, and mortality. *Diseases*. 2022;10(3):53.
5. Luft FC. Biomarkers and predicting acute kidney injury. *Acta Physiol*. 2021;231(1):e13479. doi: 10.1111/apha.13479
6. Grasselli G, Zangrillo A, Zanella A, et al. Baseline characteristics and outcomes of 1591 patients infected with SARS-CoV-2 admitted to ICUs of the Lombardy Region, Italy. *JAMA*. 2020;323(16):1574-1581.
7. Yang K, Yang N, Sun W, et al. The association between albumin and mortality in patients with acute kidney injury: a retrospective observational study. *BMC Nephrol*. 2023;24(1):332. doi: 10.1186/s12882-023-03323-x
8. Yazar S, Solmaz İ, Araç S, Sami Kalın B, Faruk Alakuş O. Are biochemical markers important in predicting intensive care unit admission and mortality in COVID-19 patients? *Van Med J*. 2021;28(3):428-433.
9. Mohamed MMB, Lukitsch I, Torres-Ortiz AE, et al. Acute kidney injury associated with coronavirus disease 2019 in urban New Orleans. *Kidney360*. 2020;1(7):614-622.
10. Joannidis M, Wiedermann CJ. Hypoalbuminemia as a Risk Factor for Acute Kidney Injury. In: Vincent JL, ed. Annual Update in Intensive Care and Emergency Medicine 2011. Annual Update in Intensive Care and Emergency Medicine 2011, vol 1. Springer: doi: 10.1007/978-3-642-18081-1_20
11. Francesco Violi et al. Is albumin predictor of mortality in COVID-19? *Antioxid Redox Signal*. 2021;35(2):139-142. doi: 10.1089/ars.2020.8142
12. Oster HS, Dolev Y, Kehat O, Weis-Meilik A, Mittelman M. Serum hypoalbuminemia is a long-term prognostic marker in medical hospitalized patients, irrespective of the underlying disease. *J Clin Med*. 2022;11(5):1207.
13. Hansrivijit P, Yarlagadda K, Cheungpasitporn W, Thongprayoon C, Ghahramani N. Hypoalbuminemia is associated with increased risk of acute kidney injury in hospitalized patients: a meta-analysis. *J Crit Care*. 2021;61:96-102.
14. Uyanik A. Factors affecting the prognosis of acute renal failure. Master Thesis. Ataturk University Medical Faculty. 2010. Available from: http://nek.istanbul.edu.tr:4444/ekos/TEZ/47475.pdf
15. de Almeida DC, do Franco MDCP, dos Santos DRP, et al. Acute kidney injury: Incidence, risk factors, and outcomes in severe COVID-19 patients. *PLoS One*. 2021;16(5):e0251048. doi: 10.1371/journal.pone.0251048
16. Grasselli G, Greco M, Zanella A, et al. Risk factors associated with mortality among patients with COVID-19 in intensive care units in Lombardy, Italy. *JAMA Intern Med*. 2020;180(10):1345-1355.