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Rates of Acute Kidney Injury in Coronavirus Disease-19

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ABSTRACT:

BACKGROUND:

Kidney involvement is common in COVID-19, with up to 75% of patients experiencing renal abnormalities. Acute kidney injury (AKI) has been identified as a frequent complication, affecting approximately 17% to 37% of hospitalized individuals and it's associated with increased in-hospital mortality.

OBJECTIVE:

To evaluate the rate of acute kidney injury in patient diagnosed with COVID-19 through serial evaluation of urine output and serum creatinine in order to elicit the diagnosis of acute kidney injury.

PATIENTS AND METHODS:

One hundred patients with Covid -19 infection were selected from isolation centers and consultation clinic and were followed in a period of 2 weeks and evaluated for the occurrence of acute kidney injury (AKI) using the KIDGO criteria, and was correlated with severity, inflammatory markers, comorbidities and patients' demographics.

RESULTS:

The incidence of AKI in this study was 22%. The analysis revealed significant differences across various parameters. The AKI group had a notably higher mean age of 66.7 ± 14.6 years compared to 45.0 ± 18.1 years in the No AKI group. Co-morbidities, particularly the presence of two or more, diabetes, hypertension, and heart disease, were more prevalent in the AKI group. The severity of COVID-19 was strongly linked to AKI, with a majority of severe cases observed in the AKI group. The distribution of KIDGO stages revealed that the majority of cases were categorized as Stage 1, constituting 59.1% (n=13). Stage 2 and Stage 3 were observed in 22.7% (n=5) and 18.2% (n=4) of the cohort, respectively. Assessing kidney outcomes, 36.4% (n=8) of the patients experienced recovery, while 31.8% (n=7) died, and an equal percentage persisted with renal complications.

CONCLUSION:

Acute kidney injury in COVID 19 patients was common and it's related to severity, old age, hypertension, diabetes and elevated inflammatory markers. The occurrence of AKI can predict higher mortality among hospitalized patients.

KEYWORDS: Acute kidney injury,COVID-19, Glomerular filtration rate,SARS-COV-2,Prognosis.

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INTRODUCTION:

The emergence of COVID-19 in Wuhan, China, in 2019 marked the third instance of a coronavirus transitioning from animals to humans, following the patterns of Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV) and Middle East Respiratory Syndrome Coronavirus (MERS-CoV) [1,2]. The global impact of COVID-19 is substantial, with 444,875,049 reported cases and 6,013,568 deaths worldwide as of 5/3/2022, and 2,307,555 cases with 25,041 deaths in Iraq . The virus exhibits a wide age group susceptibility, with severe cases more prevalent in older individuals, nursing home residents, and those with chronic medical conditions. Human-to-human transmission, primarily through respiratory droplets and aerosols, plays a pivotal role in the virus's spread [3].

Transmission primarily occurs through respiratory particles released during coughing, sneezing, or talking, with infections possible through inhalation or direct contact with mucous membranes [4]. COVID-19 manifests with a spectrum of symptoms, including fever, cough, shortness of breath, and fatigue, making clinical diagnosis challenging [5]. Notably, COVID-19 can cause acute kidney injury (AKI), particularly in severe cases, emphasizing the need for

understanding the virus's impact on various organ systems [6]. The virus's affinity for ACE2 receptors is implicated in multi-organ involvement, affecting the heart, liver, gastrointestinal tract, bone marrow, and kidneys [7–9]. The pathophysiology of COVID-19 AKI involves inflammatory responses, endothelial injury, coagulation activation, and renin–angiotensin system modulation, with factors like hypoxia, low cardiac output, nephrotoxic drugs, mechanical ventilation, and hypotension contributing to AKI development in critically ill patients [10,11].

The high prevalence of acute kidney injury (AKI) in COVID-19 patients, ranging from 17% to 37% [12], and its strong association with increased mortality underscore the need for region-specific data [13]. AKI was reported in 8.02% of patients in Thi Qar, Iraq, and was linked to higher mortality, increased dialysis needs, and more in-hospital complications [14].

In Iraq, where healthcare resources and patient demographics may differ from global trends, evaluating AKI rates in COVID-19 patients is crucial for early identification of renal complications, optimizing management strategies, and improving patient outcomes. Thus, this study aims to evaluate the rate of acute kidney injury in covid 19 patients and its correlation to the severity of disease, inflammatory markers and patients' comorbidities.

PATIENTS AND METHOD:

Research Design and Settings

This cross-sectional analytical study took place in various isolation centers in Baghdad, Iraq, spanning from May 2021 to January 2022. It encompassed patients admitted to the ward and those under home isolation follow-up. Ethical and scientific approval for the research was obtained from the Scientific Committee at the Department of Medicine, Iraqi board of medical specializations. Verbal consent was obtained from all patients before starting data collection and after explaining the aims of the study and assuring confidentiality. Samples were collected by convenience method.

Data Collection

Patient data were gathered on the first day of diagnosis, with a two-week follow-up conducted to assess the incidence of acute kidney injury based on sequential changes in serum creatinine, and urine output. Demographic information (age, gender), comorbidities (diabetes mellitus, hypertension), inflammatory markers (LDH, CRP, serum ferritin, D-dimer), and lymphocyte count were also collected. Severity classification was assigned as mild, moderate, or severe.

Inclusion Criteria

The study included 100 patients aged 16 and above diagnosed with COVID-19 based on clinical symptoms, PCR positivity, and radiological findings.

Exclusion Criteria

1. Patients with a history of chronic kidney disease undergoing renal replacement therapy or recipients of kidney transplants.
2. Vaccinated individuals and those with a documented history of previous COVID-19 infection confirmed by PCR.
3. Asymptomatic patients were excluded.

Definitions

The KDIGO criteria were employed for acute kidney injury diagnosis, encompassing an increment in serum creatinine by 0.3 mg/dl within 48 hours, an increase in serum creatinine to 1.5 times baseline within the prior 7 days, or urine volume less than 0.5 ml/kg/h for 6 hours. Patients meeting the AKI criteria were further staged into three categories according to KDIGO guidelines [15], considering age, gender, serum creatinine, and self-reported race. Recovery was defined by the Acute Disease Quality Initiative 16 consensus groups as the absence of AKI based on both serum creatinine and urine output criteria within 7 days of AKI onset. COVID-19 was diagnosed and classified according to the National Institute of Health [16], which divided the infection into three types: mild, moderate, and severe illness.

Statistical analysis

Continuous parameters were checked for normality using graphical (bar chart) and mathematical methods (Shapiro test) and thus the mean ± SD were used for parametric variables and median (range) were used for non-parametric variables. Categorical variables were expressed as percentages. Welch Two Sample T-test and Mann-Whitney U test was used for assessing the statistical significance between two means. One-way ANOVA test and T test were used to check if the means of dependent variables are significantly different from each other. R Statistical Packages were used for statistical analysis. and P values less than 0.05 will be considered to be statistically significant.

RESULTS:

The incidence of AKI in this study was reported to be 22%. Patients with AKI were significantly older (66.7 ± 14.6 years) compared to those without AKI (45.0 ± 18.1 years, $p < 0.001$). The gender distribution was similar between groups

(p = 0.7). A significantly higher proportion of patients with AKI had multiple comorbidities

(50.0% vs. 10.3%, p < 0.001), particularly diabetes (40.9% vs. 15.4%, p = 0.016), hypertension (59.1% vs. 23.1%, p = 0.001), and heart disease (18.2% vs. 2.6%, p = 0.021). In terms of treatment, a significantly higher percentage of AKI patients received remdesivir (86.4% vs. 30.8%, p < 0.001), whereas supportive care was more common in non-AKI patients (59.0% vs. 9.1%). Oxygen therapy requirements were notably higher in AKI patients, with only 4.5% maintaining room air compared to 65.4% in the non-AKI group (p < 0.001). Additionally, AKI patients had significantly lower oxygen saturation levels at admission (87.9% ± 7.1 vs. 95.2% ± 4.6, p < 0.001) (table 1).

Table 1: Description of study demographics, past-medical history and management options stratified by the presence and absence of AKI.

Characteristic	Overall, N = 100[1]	No AKI, N = 78[1]	With AKI, N = 22[1]	P-value[2]
Age (years)	49.8 ± 19.5	45.0 ± 18.1	66.7 ± 14.6	**<0.001**
Gender				0.7
Male	53 (53.0%)	42 (53.8%)	11 (50.0%)	
Female	47 (47.0%)	36 (46.2%)	11 (50.0%)	
Co-morbidities				
Diabetes	21 (21.0%)	12 (15.4%)	9 (40.9%)	**0.016**
Hypertension	31 (31.0%)	18 (23.1%)	13 (59.1%)	**0.001**
Heart disease	6 (6.1%)	2 (2.6%)	4 (18.2%)	**0.021**
Others	12 (12.0%)	9 (11.5%)	3 (13.6%)	0.7
PCR (+)	93 (93.0%)	73 (93.6%)	20 (90.9%)	0.6
Treatment				**<0.001**
Supportive	48 (48.0%)	46 (59.0%)	2 (9.1%)	
Remdesivir	43 (43.0%)	24 (30.8%)	19 (86.4%)	
Favipiravir	9 (9.0%)	8 (10.3%)	1 (4.5%)	
Oxygen supply				**<0.001**
Room Air	52 (52.0%)	51 (65.4%)	1 (4.5%)	
Continuous positive airway pressure (CPAP)	23 (23.0%)	12 (15.4%)	11 (50.0%)	
Nonbreathable face mask (NRM)	21 (21.0%)	12 (15.4%)	9 (40.9%)	
O_2 Mask	4 (4.0%)	3 (3.8%)	1 (4.5%)	
SpO_2 (%)	93.6 ± 6.0	95.2 ± 4.6	87.9 ± 7.1	**<0.001**

[1]Mean ± SD; n (%)
[2]Welch Two Sample t-test; Pearson's Chi-squared test; Fisher's exact test

AKI patients had significantly more severe COVID-19 (95.5% vs. 34.6%, p < 0.001) and higher inflammatory biomarkers, including CRP, LDH, and ferritin (p < 0.001). They also had elevated WBC counts and lower lymphocyte levels as was documented in table 2.

Table 2: Description of COVID-19 severity and biomarkers grouped by the presence and absence of AKI.

Characteristic	Overall, N = 100[1]	No AKI, N = 78[1]	With AKI, N = 22[1]	P-value[2]
COVID severity				**<0.001**
Mild	50 (50.0%)	49 (62.8%)	1 (4.5%)	
Severe	48 (48.0%)	27 (34.6%)	21 (95.5%)	
Moderate	2 (2.0%)	2 (2.6%)	0 (0.0%)	
Biomarkers				
CRP	68.3 ± 73.3	50.5 ± 63.7	131.1 ± 71.5	**<0.001**
LDH	417.6 ± 368.7	319.5 ± 209.6	765.2 ± 563.4	**0.001**
D-dimer	1.6 ± 3.1	1.4 ± 3.3	2.3 ± 2.2	0.2
Ferritin	587.6 ± 655.5	437.8 ± 543.0	1,118.7 ± 752.0	**<0.001**
WBC	9.8 ± 5.3	9.0 ± 4.6	12.8 ± 6.4	**0.014**
Lymphocyte count	1.1 ± 0.8	1.2 ± 0.8	0.7 ± 0.3	**<0.001**

[1]Mean ± SD; n (%)
[2]Welch Two Sample t-test; Pearson's Chi-squared test; Fisher's exact test
Normal reference lab values: C-reactive protein (CRP) <10 mg/L, Lactate Dehydrogenase (LDH) 140-280 U/L, D-dimer <0.5 µg/mL, Serum Ferritin 24-336 µg/L, WBC 4.5-11.0 × 109/L, lymphocyte count 1 – 4.8 × 109/L.

Serial renal function assessments (table 3) showed a slight increase in serum creatinine and blood urea levels after 7 days, which partially improved by day 14. Urine output remained normal in most patients, though oliguria was observed in 13.0% at day 7 and 12.0% at day 14, indicating transient renal impairment in some cases.

Table 3: Serial measurement of renal function tests and urine output on follow-up.

Parameters	At admission[1]	After 7 days[1]	After 14 days[1]
Serum creatinine	0.9 ± 0.2	1.1 ± 0.7	1.0 ± 0.6
Blood urea	40.2 ± 23.8	44.2 ± 34.4	40.8 ± 32.2
Urine output			
Normal	100 (100.0%)	87 (87.0%)	88 (88.0%)
Oliguria[2]	0 (0.0%)	13 (13.0%)	12 (12.0%)

[1]Mean ± SD; n (%)
[2]Oliguria is defined as < 0.5 ml/ kg/ 6 hours

Among the 22 individuals with AKI included in the study, the distribution of KIDGO stages revealed that the majority of cases were categorized as Stage 1, constituting 59.1% (n=13). Stage 2 and Stage 3 were observed in 22.7% (n=5) and 18.2% (n=4) of the cohort, respectively. Assessing kidney outcomes, 36.4% (n=8) of the patients experienced recovery, while 31.8% (n=7) died, and an equal percentage persisted with renal complications as was illustrated in figure 1.

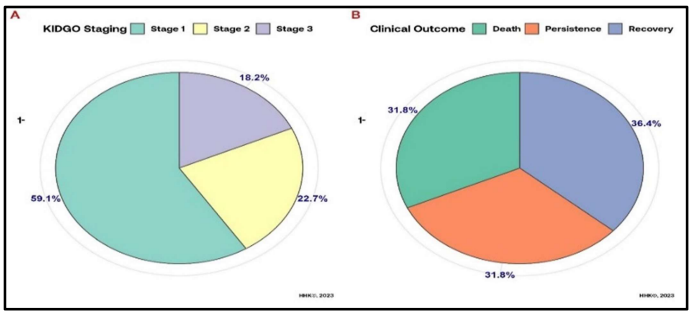

Figure 1: In patients with AKI (N=22); (A): Proportion of KIDGO staging for the severity of AKI; (B) incidence of mortality, recovery and persistence of AKI within the study follow-up period.

DISCUSSION:

The present study revealed a mean age of 49.79 ± 19.52, with 53% being male and 47% female. Regarding comorbidities, 21% had diabetes, and 31% were hypertensive. Among the cases, 48 were classified as severe requiring hospitalization, 50 were mild to moderate and treated at home, and 2 cases were lost to follow-up. Out of the 100 cases, 22 developed acute kidney injury (AKI), with 1 being mild and 21 requiring hospitalizations for severe COVID-19 infection, indicating a robust association between COVID-19 severity and AKI development. The global variation in AKI rates in COVID-19 was highlighted, with a retrospective study in Baghdad showing a 23.2% incidence, an Egyptian study reporting 14%, and a large cohort study in China finding that 39% of inpatients experienced AKI, distributed across different stages [17–19]. The high incidence of AKI in COVID-19 patients can be attributed to various factors, including hemodynamic disturbances, heightened inflammation, coagulation dysfunction, organ interactions, or direct SARS-CoV-2 involvement in the kidneys [20].

Pooling data from 13 hospitals in New York (total sample size of 9657), the study revealed a 39.9% incidence of Acute Kidney Injury (AKI). Among the AKI cases, 17% were categorized as stage 1, 8.7% as stage 2, and 14.2% as stage 3 [21]. The observed variability in AKI rates is attributed to differences in patient inclusion criteria, treatment strategies, follow-up procedures, and the varying proportion of severe cases across the hospitals.

AKI was more prevalent in the older age group, consistent with findings from studies such as those conducted in Egypt and Baghdad teaching hospital [17,18]. This association can be attributed to the higher occurrence of comorbidities, increased use of multiple medications, and the tendency for more severe disease in older individuals. In our sample, AKI patients exhibited elevated levels of LDH, CRP, d-dimer, lymphopenia, and serum ferritin compared to non-AKI patients, aligning with a study from China that established a connection between lymphopenia and AKI development. The link between age, inflammatory markers, and AKI development was also observed in local studies in Baghdad teaching hospital and reports from Egypt, suggesting a potential correlation with disease severity and activation of the coagulation cascade in patients with reduced renal function.

Furthermore, our study revealed an association between hypertension and the development of AKI, in line with reports from China where hypertension was significantly linked to AKI development [19]. This association may be indicative of the heightened severity of COVID-19 in individuals with hypertension, as corroborated by various global studies showing a higher prevalence of severe COVID-19 in patients with a history of hypertension. Notably, medications like ACE inhibitors and ARBs, known for their RAAS inhibition, were identified as potential contributors to AKI development due to their upregulation of ACE2 expression in kidney tubules [22,23]. Additionally, diuretics were identified as a risk factor for AKI development. Lastly, our study found an association between diabetes and AKI, supported by research in Iran, where diabetic patients admitted for COVID-19 exhibited a higher incidence of AKI during hospitalization compared to non-diabetic individuals [24].

Our findings indicate a significant correlation between the development of COVID-19-associated Acute Kidney Injury (AKI) and elevated blood urea levels on the initial day of presentation. In a retrospective study conducted at a single center in the University of Science and Technology (Wuhan, China) with 305 enrolled patients, it was observed that higher levels of initial blood urea nitrogen (BUN) and D-dimer were linked to increased mortality rates [25]. Another retrospective investigation involving a cohort of 12,413 COVID-19 patients revealed that elevated baseline levels of BUN and Serum creatinine were associated with a heightened risk of COVID-19 mortality and severity [26]. The elevated levels of blood urea observed in these cases may be attributed to hypovolemia resulting from decreased oral intake and fluid consumption associated with severe COVID-19 disease [26]. Furthermore, such elevations in blood urea could potentially serve as an early indicator for the development of acute AKI in hospitalized patients.

The present investigation did not reveal a significant correlation between elevated initial serum creatinine and disease severity. This lack of association might be attributed to the exclusion of patients with chronic kidney disease (CKD) from the study. Our study observed a full recovery rate of 36%, which is notably lower than the findings in a study from Egypt [17], where an 84.6% recovery rate was reported. Similarly, reports from Korea indicated a 67% recovery rate. In contrast, an observational retrospective cohort study within the Mass General Brigham health care system demonstrated that among 251 patients with

COVID-19-associated acute kidney injury (AKI), 56% recovered to within 20% of baseline creatinine [27]. The comparatively lower recovery rate in our study could be linked to the shorter duration of follow-up, delayed presentation of our patients, varying criteria for admission and disease severity. Notably, our isolation centers often admit more severe cases with lower oxygen levels, which may contribute to the differences observed in recovery rates compared to other studies.

Our investigation revealed an increased mortality rate among patients with Acute Kidney Injury (AKI), constituting 10% of the total sample compared to 7% ($p < 0.001$). This finding aligns with numerous global reports, including a study from Portugal where mortality was elevated in AKI patients (28.3% vs. 5.9%, $p < 0.001$), accompanied by an extended hospital stay (26.5±26.2 days vs. 17.1±19.6 days, $p=0.007$) [24].

The present investigation has certain limitations. The follow-up duration was restricted to two weeks; extending this period could provide a more comprehensive understanding of recovery rates, the emergence of chronic kidney disease (CKD), and the prognosis for patients who developed Acute Kidney Injury (AKI). Additionally, for mild cases managed with home isolation, the assessment of urine output relied on self-reported data. Notably, our study did not incorporate treatment plans and medications, which could offer insights into the causes of AKI, given that many drugs used for management possess potential nephrotoxicity. A more inclusive consideration of these factors could enhance the clarity and depth of our findings.

CONCLUSION:

The incidence of AKI in this study was reported to be 22%. The study highlights a significant association between COVID-19 and acute kidney injury (AKI), with AKI patients exhibiting greater disease severity, elevated inflammatory biomarkers, and worse clinical outcomes. Serial renal function assessments revealed transient impairment in some patients, emphasizing the need for close monitoring. Patient admitted for covid 19 should be carefully evaluated for the development of AKI especially cases with elevated inflammatory markers and with diabetes and hypertension and elevated initial blood urea level and take rapid measures for its correction , further studies should evaluate patients with history of CKD, and recipient of kidney transplantation.

Source of funding

This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors.

Ethical approval

Ethical and scientific approval for the research was obtained from the Scientific Committee at the Department of Internal Medicine, Iraqi board for medical specialization. All procedures performed in the present study involving human participants were in accordance with the ethical standards of the institutional and or national research committee and with the 1964 Declaration of Helsinki and its later amendments. Verbal consent was obtained from all patients before starting data collection and after explaining the aims of the study and assuring confidentiality.

Consent for publication

All authors have read and approved the final version of the manuscript and have agreed to its submission for publication. No patients' identifiable information was included in this manuscript. This manuscript has not been published elsewhere and is not under consideration for publication elsewhere.

Availability of data and material

The dataset used in this study will be made available upon request. Interested readers may contact the corresponding author to request access to the data. The data are available for non-commercial purposes only and are subject to certain limitations, including restrictions on redistribution and confidentiality concerns.

Conflict of Interest

The authors declare no financial or non-financial competing interests that could be perceived as having influenced the study design, data collection, interpretation of the results, or any other aspect of the research.

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