COVID-19 in hospitalized patients with and without acute kidney injury

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Abstract

***Introduction*: Acute kidney injury (AKI) has emerged as a significant complication in coronavirus disease 2019 (COVID-19) patients, contributing to adverse clinical outcomes. This study aimed to investigate the association between AKI and COVID-19 infection, identifying risk factors and evaluating renal dysfunction severity according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines.**

***Methods*: A retrospective cohort analysis was conducted at a single-center hospital in Houston, Texas, involving 919 hospitalized COVID-19 patients from March 2020 to February 2022. Patient data, demographics, clinical profiles, severity scores, and outcomes were collected from electronic medical records.**

***Results*: Of the 919 patients, 214 (23.28%) developed AKI, with older age, male sex, black race, obesity, hypertension, diabetes mellitus, congestive heart failure, chronic kidney disease (CKD), acute respiratory distress syndrome (ARDS), invasive ventilation, and use of diuretics and vasopressors being significant risk factors. AKI severity, categorized by KDIGO guidelines, showed a higher mortality rate in patients with more severe stages, emphasizing the importance of early recognition and intervention.**

***Conclusions*: This study highlights the high incidence of AKI in COVID-19 patients and its association with adverse clinical outcomes, particularly in those with identified risk factors and severe disease. Comprehensive management strategies targeting identified risk factors and understanding the complex pathophysiological mechanisms contributing to AKI are essential in improving patient outcomes.**

Key words: COVID-19, AKI, acute kidney injury, creatinine phosphokinase, biomarkers, COVID-19 prognostic factors, predictors of outcome.

Introduction

Coronavirus disease 2019 (COVID-19) is caused by the severe acute respiratory syndrome coronavirus 2, primarily impacting the respiratory system. Most infected individuals have mild or no symptoms, but about 15% develop a severe form of the disease requiring intensive care unit (ICU) admission and oxygen therapy. (1,2) Social determinants of health, such as socioeconomic status, access to healthcare, and structural inequalities, contribute to different risks and impacts on vulnerable populations. (3) Additionally, pre-existing health conditions and comorbidities, commonly found in certain populations, such as cardiovascular disease, diabetes, obesity, and respiratory conditions, can increase the risk of severe illness and complications from COVID-19. (2)

One of the most common complications in hospitalized and post-COVID patients is acute kidney injury (AKI), which can be explained due to the affinity of the spike protein in angiotensin-converting enzyme (ACE) receptors. These receptors are found in the lungs, gastrointestinal tract, heart, kidneys, and brain, and they are targeted by the spike protein of

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the virus. (4)
AKI in COVID-19 can occur as a new condition or worsen in patients with pre-existing renal diseases. Various pathophysiological causes contribute to this renal compromise, including direct mechanisms such as spike proteins binding to ACE receptors in the kidneys, as well as indirect mechanisms like inflammation, multiple organ dysfunction, sepsis, hemodynamic disturbances, cytokine storm, disseminated intravascular coagulation, dehydration, and hypoxemia. (5) (**Figure 1**)
The most relevant factors in developing AKI include older age, male sex, black ethnicity, obesity, hypertension, diabetes mellitus, congestive heart failure, chronic kidney disease (CKD), acute respiratory distress syndrome (ARDS), invasive ventilation, and the use of diuretics and vasopressors. (1) However, the severity of the disease also plays a significant role in the development of AKI. (2)
The purpose of this study was to investigate the association between AKI and COVID-19 infection, identify risk factors, and evaluate renal dysfunction severity according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines.

Methods

Study design and participants

The present study was a retrospective cohort analysis conducted at a single-center hospital in Houston, Texas from March 2020 to February 2022**.** It involved a total of nine hundred ninety-four patients who were diagnosed with COVID-19 that required hospital admission.
Our inclusion criteria were adult patients aged 18 years and above who were hospitalized for COVID-19 infection. Our exclusion criteria were patients under the age of 18 and patients with pre-existing CKD.

Data collection

We utilized the hospital's electronic medical record system, Meditech^{TM}, to collect patient data for the study. This included demographic information, clinical data, severity scores, imaging results, laboratory tests, treatment details, length of hospital stay, and patient outcomes.
To confirm infection, we employed various methods such as reverse transcription polymerase chain reaction (RT-PCR), SARS Antigen Fluorescent Immunoassay (Sofia), or IgG/IgM rapid tests, in addition to clinical diagnosis and imaging. The demographic and clinical data encompassed factors like age, gender, race, comorbidities, body mass index (BMI), home medications, symptoms, vital signs upon admission, and hospital treatment. Severity scores, including the Sequential Organ Failure Assessment (SOFA), Acute Physiology and Chronic Health Evaluation (APACHE) II scores, and Ichikado computed tomography (CT) scores, were also recorded. Laboratory data consisted of complete blood count, electrolyte levels, blood biochemistry, and infection-related biomarkers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), D-dimer, ferritin, procalcitonin (PCT), creatine phosphokinase (CPK), troponin, prothrombin time (PT), partial thromboplastin time (PTT), and fibrinogen.
Continuous variables were categorized using relevant cutoff points based on clinical perspectives. The study divided patients into three age groups: young (18-59), middle-aged (60-74), and old adults (75 years old). In this study, patients exhibiting renal impairment were classified into three distinct groups based on the KDIGO Guidelines for acute kidney injury (AKI). (6) The classification was determined by comparing their current creatinine levels to their respective baseline creatinine values. Patients falling within Stage 1 (S1) presented with creatinine levels ranging from 1.5 to 1.9 times higher than their baseline. Those categorized under Stage 2 (S2) showed creatinine levels elevated between 2.0 to 2.9 times compared to baseline, while Stage 3 (S3) encompassed patients whose creatinine levels surpassed 3.0 times the baseline measurement. The present study was conducted following ethical guidelines and was approved by the Institutional Review Board of the hospital before data collection. Patient information was anonymized and handled with strict confidentiality to protect individual privacy.

Statistical analysis

The statistical analysis was conducted using IBM SPSS Statistics version 24 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation (SD), while categorical data were presented as frequencies and percentages. Descriptive statistics were used to summarize demographic and clinical characteristics of both patient groups as well as chi-square, t-test, U Mann-Whitney test, Kruskal-Wallis test, and linear regressions. The data was collected and analyzed by all authors.

Results

During the period of March 2020 to February 2022 there were 946 patients who were admitted for COVID-19. This study included 919 patients, 214 (23.3%) developed AKI, 80 (8.7%) were on S1, 46 (5%) were on S2, 88 (9.6%) were on S3, and the

remaining did not develop AKI. Among those patients, 496 (44.1%) were female and 513 (55.8%) were male. Five hundred and twenty patients (56.5%) were classified as young adults, 300 (32.6%) were middle-aged adults, and 99 (10.8%) were old adults. As for ethnicity 189 (20.5%) were Caucasian, 496 (53.9%) were Hispanic, 184 (20%) were Black ethnicity, 21 (2.3%) were Asian, and 18 (2%) were from other races. Out of the 919 patients, only 742 (80.7%) survived. Two hundred and twenty-four (24.3%) patients had a past medical history of diabetes mellitus, 333 (36.2%) had hypertension, 25 (2.7%) had chronic obstructive pulmonary disease (COPD), 17 (1.8%) had myocardial infarction, 29 (3.2%) had congestive heart failure, 20 (2.2%) had peripheral vascular disease, and 21 (2.3%) had a history of stroke/transient ischemic attack (TIA). Forty-two patients (4.6 %) used tobacco, 1 (0.1%) used electronic cigarettes, 77 (8.4 %) used alcohol, and 11 (1.2%) used illicit drugs. Only 4 (.4%) patients required renal replacement therapy, 52 patients (5.7%) were on vasopressin, and 114 (12.4%) were on remdesivir (**Table 1**). One hundred and fifty-one patients (16.6%) were intubated, of which 96 (10.55%) patients had AKI ($p<0.001$).

SOFA score during hospitalization was 2 (1-3), the median highest APACHE score during hospitalization was 8 (6-13), and the median Ichikado score on admission was 150 (120-200).

The median highest CRP level was 76.80 (37.95-139), the median highest CPK level was 150 (64-388), the median highest procalcitonin level was 0.12 (-49 to -0.60), and the median highest ESR was 78 (50-109). For prothrombin time (PT) and partial thromboplastin time (PTT), the medians were 12.2 (11.49-13.50) and 33 (29-38), respectively. The median D-dimer level was 0.95 (0.44-3.23).

Chi-square tests showed an association between race and the development of acute kidney injury with $p=0.002$ (**Table 2**). Regarding age, the group with no AKI had 427 (60.6%) young adults, 214 (30.4%) middle-aged adults, and 64 (9.1%) old adults; in S1 AKI patients there were 36 (45%) young adults, 24 (30%) middle-aged adults, and 20 (25%) old adults; in S2 AKI patients there were 16 (34.8%) young adults, 23 (50%) middle-aged adults, and 7 (15.2%) old adults; in S3 AKI patients there were 41 (46.6%) young adults, 39 (44.3%) middle-aged adults, and 8 (9.1%) old adults. The group with no AKI had 72 (10.2%) deaths and 633 (89.8%) survivals; in S1 AKI patients there were 34 (42.5%) deaths and 46 (57.5%) survivals; in S2 AKI patients there were 23 (50 %) deaths and 23 (50%) survivals; in S3 AKI patients there were 48 (54.5%) deaths and 40 (45.5%) survivals. Two hundred and thirty-three (33%) patients had hypertension in the group with no AKI, 45 (56.3%) patients in the S1 group, 21 (45.7 %) patients in the S2 group, and 34 (38.6%) patients in the S3 group; sixteen (2.3%) patients had congestive heart failure in the group with no AKI, 6 (7.5%) patients in S1 group, 5 (10.9 %) patients in S2 group, and 2 (2.3 %) patients in the S3 group. Sixteen (2.3%) patients were on vasopressin in the group with no AKI, 16 (20%) patients in the S1 group, 5 (10.9%) patients in the S2 group, and 15 (17%) patients in the S3 group.

A chi-square analysis showed that there was no statistically significant difference in outcomes observed between patients who were administered remdesivir and those who were not ($p=0.338$). In the SI group, there were 3 recorded deaths among those who received remdesivir and 6 patients who survived. In the S2 group, 2 deaths were reported among those who received remdesivir, with none surviving. Lastly, in the S3 group, there were 5 deaths and 5 survivors in the remdesivir group.

A t-test analysis showed a significant correlation existed between age and AKI ($p<0.001$). However, there was no substantial link found between BMI and AKI ($p=0.943$).

A Kruskal-Wallis test with post-hoc analysis revealed a statistically significant difference between AKI S1 showing that the S1 group had a significantly higher D-dimer than the no AKI group, and the no AKI group had a significantly higher D-dimer than the S2 group ($x^2[3]=77.915$, $p<0.001$). No AKI group had a higher fibrinogen level than the S3 group ($x^2[3]=18.034$, $p<0.001$). The S1 group had a higher PTT than the no AKI group, and the no AKI group had a higher PTT than the S2 and S3 groups ($x^2[3]=53.819$, $p<0.001$). Regarding ESR, the no AKI group had a higher level than the S2 and S3 groups ($x^2[3]=27.398$, $p<0.001$). S1 patients had a significantly higher ferritin level than the no AKI group, but the no AKI group had a significantly higher ferritin level than the S2 and S3 group ($x^2[3]=84.641$, $p<0.001$). The no AKI group had a significantly higher aspartate transferase (AST) level than the S1, S2, and S3 groups ($x^2[3]=63.220$, $p<0.001$). S2 and S3 patients had a higher alanine transaminase (ALT) level than the no AKI group ($x^2[3]=52.877$, $p<0.001$). The S1 group had a significantly higher troponin level than the no AKI group, but this group had a higher troponin level than S2 patients ($x^2[3]=35.565$, $p<0.001$). The S1 group had a higher CPK level than the no AKI group, but the no AKI group had a higher CPK level than the S2 and S3 groups ($x^2[3]=75.673$, $p<0.001$). The no AKI group had a higher CRP level than the S3 patients ($x^2[3]=23.375$, $p<0.001$).

Discussion

The results from this study provide crucial insights into the relationship between demographic factors, comorbidities, and clinical outcomes in COVID-19 patients with AKI. The high prevalence of AKI among COVID-19 patients, as observed in this study, aligns with findings from previous research highlighting the kidney's susceptibility to injury during viral infections. (7,8)

Interestingly, our study revealed age as a significant risk factor for AKI, with young adults being more vulnerable. This finding contradicts some earlier studies suggesting that older adults were at higher risk. (9) Such discrepancies might be attributed to differences in sample demographics and regional variations in disease severity.

In contrast to the study conducted by Chan L. et al., which stated that there was no connection between hypertension and diabetes with severe AKI. (10) A study conducted among hospitalized COVID-19 patients with AKI in New York, USA, unveiled that factors contributing to the risk of AKI encompassed advanced age, diabetes mellitus, heart-related ailments, Black ethnicity, high blood pressure, as well as the requirement for ventilator support and vasopressor medications. (11,12)

Black ethnicity is more susceptible to the virus and experiences worse outcomes. (13,14) They also have a higher risk of developing AKI due to COVID-19 infection. Black ethnicity patients exhibit higher levels of inflammatory markers and are more predisposed to thrombosis, coagulopathy, and elevated levels of D-dimer. (15) Additionally, the presence of the apolipoprotein L1 (APOL1) allele in Black ethnicity individuals has been suggested to increase the risk of AKI by 1.2 times compared to white patients. (16-18)

Individuals who experienced AKI likely underwent proximal tubular damage, peritubular red blood cell clumping, and the formation of blood clots in the kidney's glomerular capillaries due to ischemic collapse. (19)

The impact of comorbidities on AKI development is evident from the higher prevalence of hypertension and other medical conditions among AKI patients. (20) However, the study does not delve into the potential interplay between these comorbidities and the severity of COVID-19, which could be crucial in understanding their role in AKI pathogenesis.

In a study led by Pei G. et al., it was observed that upon admission, 65% of patients exhibited proteinuria and 41% had hematuria; however, only a minor proportion, 4.7%, eventually experienced the onset of AKI. In our study, we encountered instances of proteinuria with a mean of 25.59 (75.825). It could prove insightful to delve into a discourse concerning the potential link between hematuria and the emergence of AKI in hospitalized patients. (21)

The varying mortality rates across different stages of AKI underscore the importance of early detection and intervention to improve patient outcomes (**Figure 2**). The significant association between race and AKI development raises important questions regarding potential genetic or socio-economic factors contributing to differential risks. (22)

In some comparative studies, remdesivir was mentioned to be a very effective treatment against COVID-19, and it is supposed to have a minimal effect on the development of AKI. In comparison, our study revealed a significance on patients that were treated with remdesivir, had AKI, and additionally, there were some that died. (23)

To strengthen the validity of these findings, future studies could consider multi-center collaborations to increase sample size and diversity. Additionally, exploring the use of other treatments or medications not examined in this study may provide further insights into AKI prevention and management. Overall, these results contribute significantly to the existing body of knowledge on AKI in the context of COVID-19 and highlight the importance of targeted interventions to mitigate the impact of this complication on patient outcomes.

Conclusions

Our study sheds light on the high incidence of AKI in COVID-19 patients and its association with worse clinical outcomes, particularly in patients with identified risk factors and severe disease. Applying the KDIGO guidelines, we were able to categorize AKI severity and observe its impact on patient mortality. Early recognition, close monitoring, and appropriate management of AKI are crucial in mitigating the impact of this complication on COVID-19 patient outcomes. Future research should focus on further elucidating the specific mechanisms behind AKI in COVID-19, as this knowledge can inform targeted therapies and interventions to improve patient outcomes. Additionally, efforts to address social determinants of health and healthcare disparities may help reduce the burden of AKI and COVID-19 on vulnerable populations.

Conflicts of interest

The authors have no conflict of interest in the preparation of this manuscript. This research received no specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

This study was presented as an oral presentation at the Society of Critical Care Medicine Congress in San Francisco, in January 2023.

Table 1. Demographics, clinical characteristics, and comorbidities of patients admitted with COVID-19 infection

	AKI	%	No AKI	%	p-value
Number of patients	214	22.85%	705	77%	
Mean age	60.28		54.23		≤0.001
Gender					
- Female	79	8.60%	327	35.60%	≤0.015
- Male	135	14.70%	378	41.10%	≤0.015
Mean BMI	31		31.04		≤0.943
Ethnicity					
- Caucasian	56	6.09%	133	14.47%	≤0.004
- Hispanic	114	12.40%	382	41.56%	
- Black ethnicity	31	3.37%	153	16.64%	
- Other	8	0.87%	10	1.08%	
- Asian	2	0.21%	19	2.06%	
Comorbidities					
- Diabetes mellitus	56	6.1%	168	18.3%	≤0.485
- Hypertension	100	10.90%	233	25.4%	≤0.001
- Peripheral vascular disease	7	0.80%	13	1.40%	≤0.210
- Congestive heart failure	13	1.40%	16	1.70%	≤0.005
- COPD	10	1.10%	15	1.60%	≤0.045
- Dementia	6	0.70%	9	1.00%	≤0.123
- Myocardial infarction	1	0.10%	16	1.70%	≤0.087
- Connective tissue disorder	4	0.40%	6	0.70%	≤0.209
- Peptic ulcer	5	0.50%	11	1.20%	≤0.447
- Liver disease	2	0.20%	9	1.00%	≤0.687
- Hemiplegia	3	0.30%	1	0.10%	≤0.014
- Solid tumor	4	0.40%	5	0.50%	≤0.131
- Lymphoma	1	0.10%	1	0.10%	≤0.371
- Leukemia	0	0.00%	3	0.30%	≤0.339
- Other	86	9.40%	251	27.30%	≤0.223
- Stroke/TIA	2	0.20%	19	2.10%	≤0.131
Tobacco	17	7.00%	25	10.30%	≤0.183
Alcohol	23	9.50%	54	22.30%	≤0.901
Illicit drugs	1	0.40%	10	4.01%	≤0.129

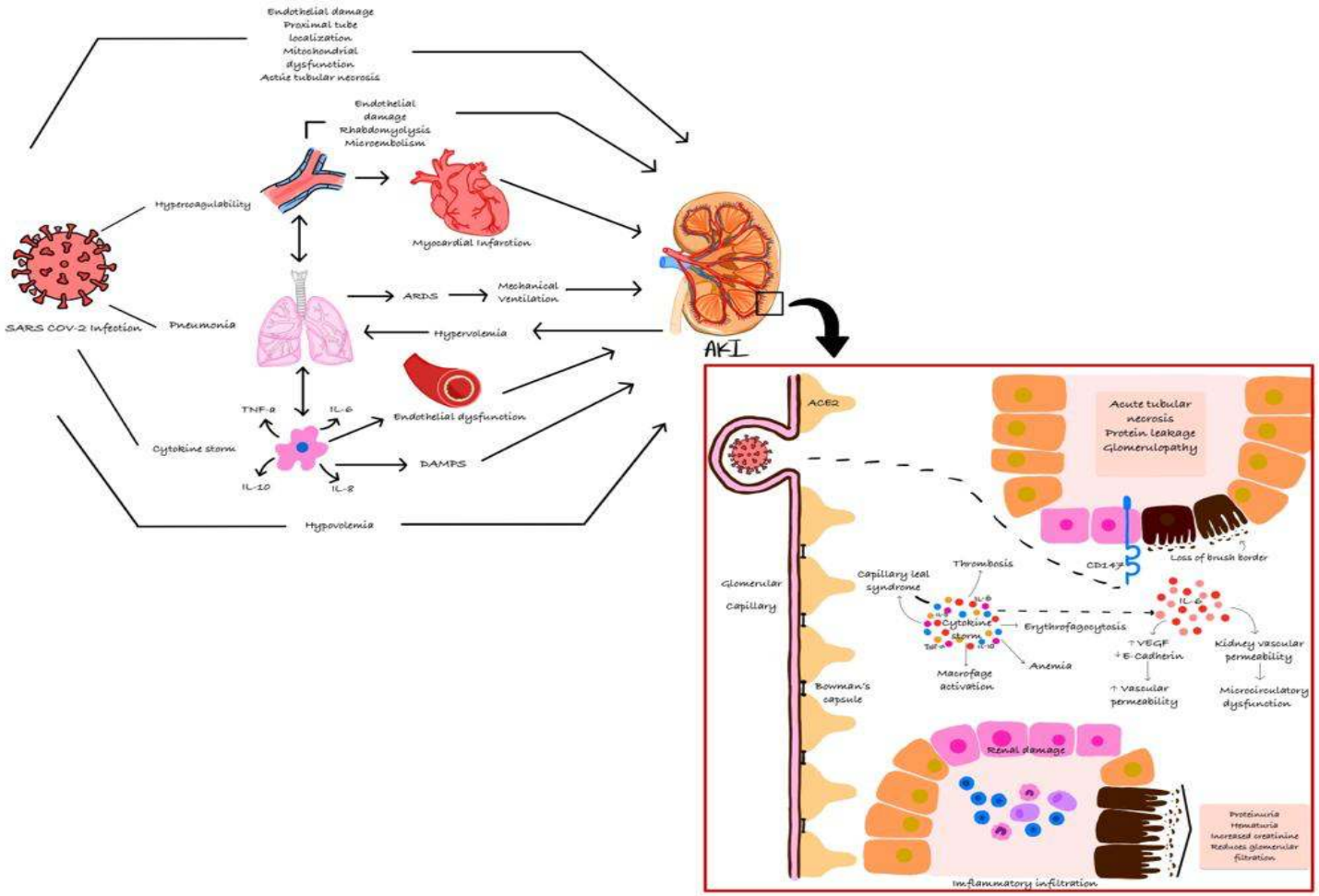
Legend: COVID-19=coronavirus disease 2019; AKI=acute kidney injury; BMI=body mass index; COPD=chronic obstructive pulmonary disease; TIA=transient ischemic attack.

Table 2. Association between race and stages of AKI: Analysis according to the KDIGO guidelines

Race	No AKI		Stage 1		Stage 2		Stage 3	
	n	%	n	%	n	%	n	%
Caucasians	133	19.1%	20	25.6%	13	28.3%	23	26.4%
Hispanics	382	54.8%	38	48.7%	26	56.5%	50	57.5%
Black ethnicity	153	22%	18	23.1%	5	10.9%	8	9.2%
Asian	19	2.7%	2	2.6%	0	0%	0	0%
Other	10	1.4%	0	0%	2	4.3%	6	6.9%
Total	697	100%	78	100%	46	100%	87	100%

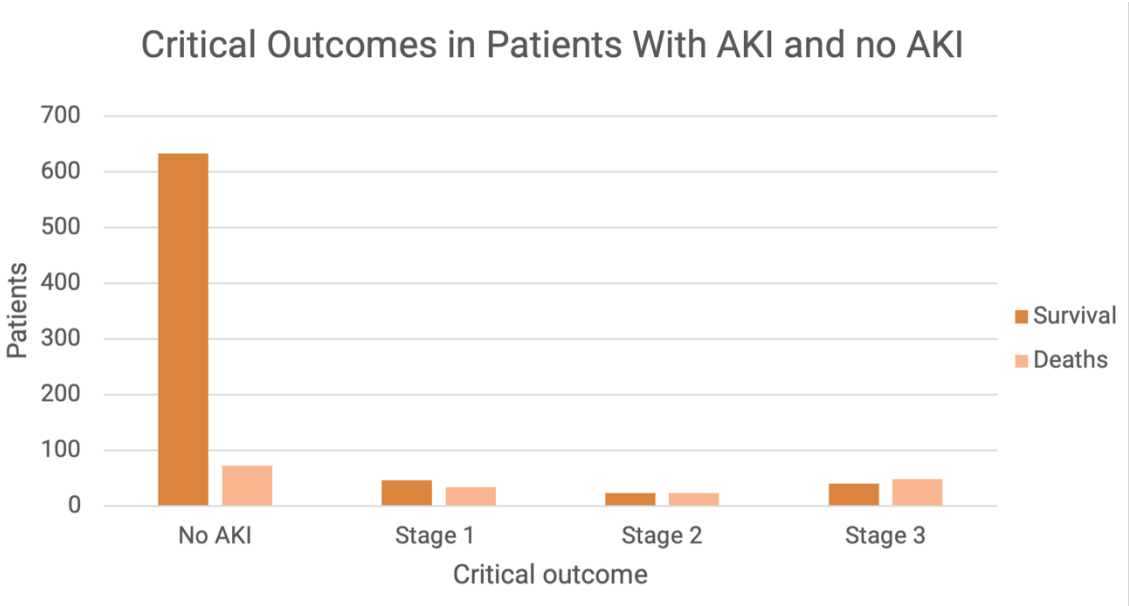
Legend: AKI=acute kidney injury; KDIGO=Kidney Disease: Improving Global Outcomes.

Figure 1. Direct and indirect physiopathology pathways of acute kidney injury due to COVID-19



Legend: COVID-19=coronavirus disease 2019; ARDS=acute respiratory distress syndrome; SARS CoV-2=severe acute respiratory syndrome coronavirus 2; TNF-α=tumor necrosis factor-alpha; IL=interleukin; DAMPs=damage-associated molecular patterns; AKI=acute kidney injury; VEGF=vascular endothelial growth factor.

Figure 2. Distribution of critical outcomes in relation to stages of acute kidney injury according to the KDIGO guidelines



Legend: KDIGO=Kidney Disease: Improving Global Outcomes; AKI=acute kidney injury.

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