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ORIGINAL ARTICLE

Incidence and Risk Factors for Acute Kidney Injury and Its Effect on Mortality in Patients Hospitalized From COVID-19

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Abstract

Objective: To determine the incidence of and risk factors for the development of acute kidney injury (AKI) and investigate the association between AKI and mortality in patients hospitalized with coronavirus disease 2019 (COVID-19) infection.

Patients and Methods: This retrospective case series includes the first 370 patients consecutively hospitalized with confirmed COVID-19 illness between March 10, 2020, and May 13, 2020, at a 242-bed teaching hospital. To determine independent associations between demographic factors, comorbid conditions, and AKI incidence, multivariable logistic regression models were used to estimate odds ratios adjusted for clinical covariates.

Results: Median age of patients was 71 (interquartile range, 59-82) years and 44.3% (145 of 327) were women. Patients with AKI were significantly older with a higher comorbid condition burden and mortality rate (58.1% [104 0f 179] vs 19.6% [29 of 148]; $P<.001$) when compared with those without AKI. Increasing age, chronic kidney disease, hyperlipidemia, and being of African American descent showed higher odds of AKI. Patients with AKI had significantly higher odds of mortality when compared with patients without AKI, and this effect was proportional to the stage of AKI. Increasing age and acute respiratory distress syndrome also revealed higher adjusted odds of mortality.

Conclusions: Acute kidney injury is a common complication among hospitalized patients with COVID-19 infection. We found significantly higher odds of AKI with increasing age and among patients with hyperlipidemia, those with chronic kidney disease, and among African Americans. We demonstrate an independent association between AKI and mortality with increasingly higher odds of mortality from progressively worsening renal failure in hospitalized patients with COVID-19 infection.

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In December 2019, a cluster of patients with pneumonia was reported in Wuhan, Hubei Province, China ,which was later identified to be caused by a novel coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[1] The illness caused by SARS-CoV-2, coronavirus disease 2019 (COVID-19), was declared as a pandemic by the World Health Organization on March 11, 2020. The illness mainly manifests as fever, cough, myalgia or fatigue, sputum production, headache, and diarrhea.[2] The severity of COVID-19 illness can run the spectrum from asymptomatic infection, self-limited flu-like illness, and acute pneumonia to sepsis leading to life-threatening complications, including acute respiratory distress syndrome (ARDS), acute cardiac injury, acute kidney injury (AKI), and septic shock.[3,4]

The reported incidence of AKI in COVID-19 has ranged from 0.5% to 27% among hospitalized patients.[2,3,5-7] Reports on the incidence of AKI in hospitalized patients from Western countries are lacking and much needed. For this study, we aimed to determine the incidence of AKI in patients hospitalized with COVID-19 infection and assess demographic factors and

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comorbid conditions that portend an increased risk for AKI in these patients. We also evaluated the association between AKI and mortality in these patients.

PATIENTS AND METHODS

Data Source

This retrospective case series includes the first 370 patients consecutively hospitalized with confirmed COVID-19 illness between March 10, 2020, and May 13, 2020, at a 242-bed teaching community hospital in the New York City metropolitan area. The hospital has a 12-bed intensive care unit and serves approximately 250,000 people in southern Westchester County, New York. Cases were confirmed through a positive result for SARS-CoV2 virus by reverse-transcriptase polymerase chain reaction testing of nasopharyngeal swab specimens. Data were manually abstracted from electronic health records by the authors and included demographic characteristics, comorbid conditions, and outcomes (AKI, ARDS, mortality, or discharge). Three authors (A. Nimkar, A. Naaraayan, and S.P.) independently reviewed the data for accuracy. Patient outcomes were followed up until June 10, 2020.

Definition of Patient Characteristics

Comorbid conditions derived from the patients or nursing home transfer forms were abstracted from physician documentation on the electronic health records. Patients with end-stage renal disease were all patients with renal disease receiving long-term dialysis treatment. Cardiac disease was defined as chronic heart conditions including but not limited to coronary artery disease, previous myocardial infarction, cardiac arrhythmias, congestive heart failure, presence of pacemaker or defibrillator device, and previous coronary artery bypass grafting or percutaneous coronary intervention. Body mass index (BMI; calculated as the weight in kilograms divided by the height in meters squared) was used to classify patients into normal weight (BMI, 18.5-24.9 kg/m^2), overweight (BMI, 25-29.9 kg/m^2), obese (BMI $\geq$30 kg/m^2), and malnourished (BMI $<$18.5 kg/m^2) categories, based on the classification by the World Health Organization.[8] Primary insurance coverage was classified as Medicare, Medicaid, private, or other (self-pay/no insurance). Race was categorized into 1 of the 4 groups: white, African American, Hispanic, and other.

Acute kidney injury was identified as defined by the criteria from Kidney Disease: Improving Global Outcomes and the International Society of Nephrology.[9] We did not have access to preadmission baseline creatinine values for most patients. Therefore, we considered the lowest creatinine level recorded during admission as the baseline and then retrospectively compared this "baseline" with the highest creatinine level recorded.[10] We did not include urine output in our determination of AKI given the heterogeneity of urine output recordings and the high degree of missing data. Using Kidney Disease: Improving Global Outcomes criteria, AKI was further characterized into stages on the basis of the maximal difference between baseline and peak creatinine levels during the hospital stay. Acute kidney injury was staged as follows: 1) stage 1, increase in serum creatinine level to 1.5 to 1.9 times baseline or increase in serum creatinine level by 0.3 mg/dL or greater ($\geq$26.5 μmol/L; to convert to μmol/L, multiply by 88.4); 2) stage 2, increase in serum creatinine level to 2.0 to 2.9 times baseline; 3) stage 3, increase in serum creatinine level to 3.0 times baseline or increase to 4.0 mg/dL or greater. Acute respiratory distress syndrome was defined as per the Berlin criteria.[11]

We could not determine AKI status for patients with only 1 creatinine value recorded during the hospital stay and thus excluded these patients from analysis (n=10). We also excluded patients with end-stage renal disease (n=28). Patients who did not yet have a definite outcome of mortality or discharge, that is, patients who were still being treated at the time of writing, were also excluded (n=5). After said exclusions, the final analysis included 327 patients.

Outcome Measures and Statistical Analyses

We computed median, interquartile range, frequency, and percentage as our descriptive variables. Differences in median and percentage were assessed using the Mann-Whitney and χ^2 test, respectively. We calculated odds ratios (ORs) for outcomes, AKI, and mortality by univariable- and age-sex–adjusted models. To determine independent associations between

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TABLE 1. Characteristics and Outcomes of Patients Admitted With Coronavirus Disease 2019[a]

Patient Characteristic	Overall (n=327)	Patients With AKI (n=179)	Patients Without AKI (n=148)	P[c]
Demographic characteristics				
Age (y), median (interquartile range)	71 (59-82)	75 (63-85)	67 (53.5-78)	<.001[b]
Male sex, no. (%)	182 (55.7)	101 (56.4)	81 (54.7)	.8
Race/ethnicity, no. (%)				
White	111 (33.9)	60 (33.5)	51 (34.5)	.9
African American	116 (35.5)	77 (43.0)	39 (26.4)	.002
Hispanic	65 (19.9)	26 (14.5)	39 (26.4)	.01[b]
Other	35 (10.7)	16 (8.9)	19 (12.8)	.3
Admission source home, no. (%)	212 (64.8)	108 (60.3)	104 (70.3)	.06
Insurance, no. (%)				
Medicare	125 (38.2)	70 (39.1)	55 (37.2)	.7
Medicaid	42 (12.8)	21 (11.7)	21 (14.2)	.5
Private insurance	147 (45.0)	84 (46.9)	63 (42.6)	.4
Self-pay or other	13 (4.0)	4 (2.2)	9 (6.1)	.08
Comorbid conditions, no. (%)				
Hypertension	209 (63.9)	126 (70.4)	83 (56.1)	.01[b]
Diabetes mellitus	139 (42.5)	87 (48.6)	52 (35.1)	.01[b]
Hyperlipidemia	114 (34.9)	76 (42.5)	38 (25.7)	.001[b]
Cardiac disease	98 (30.0)	59 (33.0)	39 (26.4)	.2
Chronic kidney disease	40 (12.2)	33 (18.4)	7 (4.7)	<.001[b]
Chronic obstructive pulmonary disease	44 (13.5)	24 (13.4)	20 (13.5)	.9
Stroke or dementia	91 (27.8)	59 (32.9)	32 (21.6)	.02
Smoking	56 (17.1)	29 (16.2)	27 (18.2)	.6
Malignancy	66 (20.2)	36 (20.1)	30 (20.3)	.9
Obesity	113 (34.6)	63 (35.2)	50 (33.8)	.8
Mortality, no. (%)	133 (40.7)	104 (58.1)	29 (19.6)	<.001[b]

[a]AKI = acute kidney injury.
[b]Difference in median and percentage were calculated using the Mann-Whitney and χ^2 test, respectively.
[c]Statistically significant.

demographic factors, comorbid conditions, and AKI incidence, multivariable logistic regression models were used to estimate ORs adjusted for clinical covariates. Demographic factors (age, sex, and race) and major comorbid conditions (hypertension, diabetes, and cardiac disease) were considered the 6 essential covariates and always included for adjustment in the multivariable model. In addition, covariates that showed significant odds in the age-sex–adjusted model were included in the multivariable models. As an additional covariate to calculate OR for mortality, ARDS was used. Two-sided $P<.05$ was considered statistically significant. Data were analyzed using Stata, version 13.0 (Stata Corp).

Statement of Ethics

The study was carried out in accordance with the Declaration of Helsinki and was approved by the departmental research review committee with a waiver of informed consent due to its retrospective design (approval number 20.5.01).

RESULTS

Median age of patients was 71 (interquartile range, 59-82) years and 44.3% (145 of 327) were women. The most commonly observed comorbid conditions in the 327 patients were hypertension (63.9% [n=209]), diabetes (42.5% [n=139]), hyperlipidemia (34.9% [n=114]), obesity (34.6% [n=113]), and cardiac diseases (29.9% [n=98]; Table 1). The overall mortality rate was 40.7% (133 of 327). In 179 (54.7%) patients, AKI was observed; 69 (21.1%) had stage 1, 42 (12.8%) had stage 2, and 68 (20.8%) had stage 3 AKI. In 137 of 179 (76.5%) patients, AKI was present on admission and another

TABLE 2. Odds of AKI in Patients Hospitalized With Coronavirus Disease 2019[a,b]

Demographic and Clinical Characteristics	Age-Sex–Adjusted Odds	*P*	Multivariable Analysis	*P*
Demographic characteristic				
Age	—	—	1.03 (1.01-1.05)	.007[c]
Male sex (vs female)	—	—	1.3 (0.8-2.2)	.3
Race (vs white)	—	—	—	—
African American	2.1 (1.2-3.7)	.01[c]	2.01 (1.1-3.6)	.02[c]
Hispanic	0.9 (0.4-1.7)	.7	0.9 (0.4-1.8)	.7
Other	0.8 (0.4-1.8)	.6	0.7 (0.3-1.6)	.4
Nursing home admit (vs home)	1.01 (0.6-1.7)	.9	—	—
Insurance (vs Medicare)			—	—
Medicaid	2.3 (0.9-5.4)	.07	—	—
Private	1.7 (0.9-2.9)	.07	—	—
Other	0.7 (0.2-2.4)	.5	—	—
Comorbid condition				
Hypertension	1.4 (0.9-2.3)	.2	0.9 (0.5-1.6)	.7
Diabetes mellitus	1.7 (1.1-2.7)	.03[c]	1.5 (0.9-2.4)	.1
Hyperlipidemia	1.8 (1.1-2.9)	.02[c]	1.8 (1.04-3.01)	.03[c]
Cardiac disease	0.9 (0.6-1.7)	.9	0.8 (0.5-1.4)	.4
Chronic kidney disease	3.7 (1.6-8.9)	.003[c]	3.3 (1.4-7.9)	.008[c]
Chronic obstructive pulmonary disease	0.8 (0.4-1.5)	.4	—	—
Stroke or dementia	1.2 (0.7–2.1)	.6	—	—
Smoking	0.7 (0.4–1.3)	.3	—	—
Malignancy	0.8 (0.4–1.4)	.4	—	—
BMI class (kg/m^2) (vs normal, 18.5-24.9)	—	—	—	—
Overweight, 25-29.9	0.6 (0.3-1.01)	.06	—	—
Obese, ≥30	1.05 (0.6-1.9)	.9	—	—
Underweight, <18.5	0.5 (0.1-1.9)	.3	—	—

[a]AKI = acute kidney injury; BMI = body mass index.
[b]Odds adjusted for race, hyperlipidemia, and chronic kidney disease in addition to 6 essential covariates (age, sex, race, hypertension, diabetes, and cardiac disease).
[c]Statistically significant.

26 (14.5%) patients developed AKI within 48 hours of admission. Twenty patients received urgent dialysis for stage 3 AKI. Patients with AKI were significantly older, less likely to be Hispanic, and had a higher prevalence of major comorbid conditions (hypertension, diabetes, hyperlipidemia, and chronic kidney disease [CKD]) when compared with patients without AKI. Mortality was significantly higher in patients with AKI when compared with patients without AKI (58.1% [104 of 179] vs 19.6% [29 of 148]; *P*<.001; Table 1).

On univariable analysis, age, hypertension, diabetes, hyperlipidemia, and CKD had higher odds of AKI in patients with COVID-19 infection (Supplemental Table 1, available online at https://mcpiqojournal.org). In the age-sex–adjusted model, race, diabetes, hyperlipidemia, and CKD showed higher odds of AKI. Covariates in the multivariable model for AKI thus included the 6 essential covariates identified in the methodology, along with race, hyperlipidemia, and CKD. In the multivariable model, increasing age (OR, 1.03 for every 1-year increase in age; 95% CI, 1.01 to 1.05; *P*=.007), African American race, presence of CKD, and hyperlipidemia showed higher odds of AKI (Table 2).

On univariable analysis, AKI, ARDS, increasing age, insurance, nursing home status, and several comorbid conditions showed higher odds of mortality (Supplemental Table 2, available online at https://mcpiqojournal.org). Age-sex–adjusted analysis demonstrated that AKI, ARDS, and BMI class had significant impacts on odds of mortality. Thus, these covariates were included in addition to the 6 preidentified essential covariates in the multivariable model

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TABLE 3. Odds of Mortality With AKI in Patients Hospitalized With Coronavirus Disease 2019[a,b]

Demographic and Clinical Characteristics	Age- and Sex-Adjusted Odds	*P*	Multivariable Analysis	*P*
AKI				
Stage 1	2.7 (1.4-5.2)	.003	2.8 (1.002-7.8)	.049[c]
Stage 2	5.1 (2.4-10.9)	<.001	3.3 (1.05-10.5)	.04[c]
Stage 3	9.2 (4.6-18.3)	<.001	4.8 (1.6-14.5)	.01[c]
ARDS	38.1 (17.4-83.4)	<.001	32.6 (12.8-83.6)	<.001[c]
Demographic characteristics				
Age	—	—	1.06 (1.03-1.1)	<.001[c]
Male sex (vs female)	—	—	1.5 (0.7-3.3)	.3
Race (vs white)	—	—	—	—
African American	0.8 (0.5-1.4)	.5	0.4 (0.2-1.1)	.08
Hispanic	1.5 (0.7-2.9)	.3	0.6 (0.2-2.1)	.4
Other	0.6 (0.2-1.3)	.2	0.5 (0.1-1.9)	.3
Nursing home admit (vs home)	1.4 (0.8-2.3)	.3	—	—
Insurance (vs Medicare)	—	—	—	—
Medicaid	1.4 (0.6-3.4)	.5	—	—
Private	0.7 (0.4-1.2)	.2	—	—
Other	0.8 (0.2-3.2)	.8	—	—
Comorbid condition				
Hypertension	1.1 (0.7-1.8)	.7	0.6 (0.2-1.3)	.2
Diabetes mellitus	1.3 (0.8-2.1)	.2	1.1 (0.5-2.3)	.9
Hyperlipidemia	1.4 (0.8-2.2)	.2	—	—
Cardiac disease	1.1 (0.7-1.9)	.7	1.7 (0.7-3.9)	.2
Chronic kidney disease	0.8 (0.4-1.6)	.5	—	—
Chronic obstructive pulmonary disease	0.7 (0.4-1.4)	.3	—	—
Stroke or dementia	1.1 (0.6-1.9)	.7	—	—
Smoking	0.8 (0.4-1.5)	.5	—	—
Malignancy	1.2 (0.7-2.1)	.5	—	—
BMI class (kg/m^2), (vs normal, 18.5-24.9)	—	—	—	—
Overweight, 25-29.9	1.8 (1.01-3.3)	.047	1.9 (0.7-5.5)	.2
Obese, ≥30	2.2 (1.1-4.1)	.02	1.6 (0.6-4.3)	.4
Underweight <18.5	1.3 (0.3-5.4)	.8	1.3 (0.1-14.9)	.8

[a]AKI = acute kidney injury; ARDS = acute respiratory distress syndrome, BMI = body mass index.
[b]Odds adjusted for AKI, ARDS, and BMI class in addition to 6 essential covariates (age, sex, race, hypertension, diabetes, and cardiac disease).
[c]Statistically significant.

for mortality. On multivariable analysis, patients with AKI had significantly higher odds of mortality when compared with patients without AKI, and this effect was proportional to the AKI stage (Table 3; Figure). In addition to AKI, advancing age and ARDS were the only other covariates to significantly affect in-hospital mortality (Table 3).

DISCUSSION

Although COVID-19 infection manifests primarily in the lungs, it is increasingly being recognized for involvement of the kidney, gastrointestinal-tract, heart, and coagulation system. Even as scientific data linking COVID-19 infection to kidney disease are expanding, there is a dearth of clinical data for the incidence of AKI in COVID-19 infection. We present one of the first reports assessing AKI among hospitalized patients with COVID-19 infection in the Western hemisphere. This is also one of the first studies proving the association of AKI with mortality in COVID-19. We saw increasing odds of mortality with progressively severe renal failure in these patients. This effect on mortality

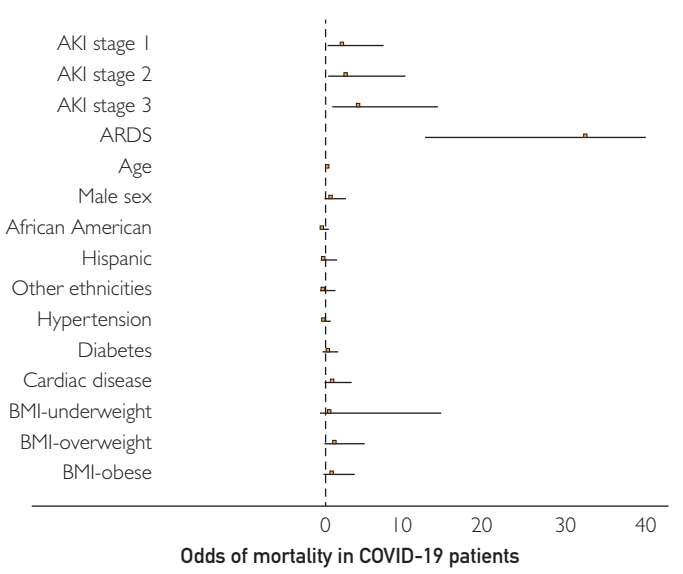

FIGURE. Adjusted odds of mortality in patients hospitalized with coronavirus disease 2019. AKI = acute kidney injury; ARDS = acute respiratory distress syndrome; BMI = body mass index.

persisted despite adjusting for demographics, comorbid conditions, and acute lung injury (ARDS), which has been shown to be the primary pathway for serious illness and mortality.[5,12] We also saw higher adjusted odds of mortality with increasing age and in patients with ARDS (Figure).

These findings are similar to the experience from China, although our data are based on multivariate analysis and are thus more robust.[5,13] Multivariate analysis by Chu et al[14] from the 2002 to 2003 SARS-coronavirus epidemic identified almost identical risk factors for mortality as our study. In their study, the authors described increasing odds of mortality with age, AKI, and ARDS, similar to our analysis, supporting the validity of our findings.

There are several mechanisms by which COVID-19 infection could affect the kidney. One of the mechanisms involves the cytokine release syndrome or the "cytokine storm" from sepsis in response to SARS-CoV-2.[2] Cytokine release syndrome has been reported in COVID-19 infection and could cause AKI by leading to intrarenal inflammation, increased vascular permeability, volume depletion, and cardiomyopathy, which can lead to cardiorenal syndrome. Studies have reported similar expression of angiotensin-converting enzyme 2 (ACE-2) among old and young individuals. However, there is a tendency for a stronger immune response, potentially leading to cytokine storm–related injury in the lungs of older individuals.[15] Increased mortality with increasing age could thus be a reflection of a more robust increase in cytokine levels in the elderly.

Organ cross-talk in COVID-19 infection could be another mechanism for AKI. Rhabdomyolysis leading to acute tubular necrosis, alveolar damage leading to renal medullary hypoxia, and acute tubular necrosis secondary to abdominal compartment syndrome from high peak airway pressure and intra-abdominal hypertension are some of the possible scenarios.[16] Systemic effects of severe sepsis such as endothelial damage leading to third-space fluid loss and hypotension, as well as endotoxin levels, could be another pathway for AKI in COVID-19 infection.

In addition to renal dysfunction as a result of immune dysregulation, emerging evidence suggests the possibility of a direct cytopathic effect of SARS-CoV-2 on the kidney. Although SARS-CoV-2 enters the human body mostly through lungs (and occasionally the gastrointestinal tract), RNA viremia has been reported during infection (15% of cases), thus allowing

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the virus to reach all organs in the body, including the kidneys.[2] In addition, ACE-2, which has been described as the most likely "receptor" for viral entry into human cells (along with serine proteases), is heavily expressed in tissues outside the lungs. Studies report higher expression of ACE-2 in intestine, testis, heart, and kidneys than in the lungs.[15,17] In a recent report of postmortem analysis of 26 patients with COVID-19 infection, 7 patients were found to have coronavirus particles with distinctive spikes in the renal tubular epithelium and podocytes.[18] In addition to viral particles, acute tubular necrosis, lymphocyte infiltration, and enhanced $CD68^{+}$ macrophages have been described in the interstitium with membrane attack complex (complement C5b-9) deposition on tubules.[6]

The incidence of AKI in our study was much higher than previously reported. This could partly be due to a difference in frequency of testing creatinine levels because detection of AKI is mainly based on acute changes in serum creatinine levels and the frequency of serum creatinine tests has a substantial impact on detection rate.[5] Pan et al demonstrated higher ACE-2 expression in kidneys of donors from Western countries compared with Asian populations, which could in part explain the higher rates of AKI in our study.[17] We used serum creatinine values during the entire hospitalization to calculate the difference between baseline and peak creatinine levels because kidney dysfunction in COVID-19 infection might not be readily evident at admission and progress during hospitalization.[13] This may be because the cytokine storm can occur a few days after illness onset, thus resulting in AKI days after hospitalization.[19] In our study, 35 of 179 (19.6%) AKI diagnoses were made past the traditionally used limit of the initial 48 hours of hospital stay. Using the entire hospital stay to calculate AKI instead of the first 48 to 72 hours may be another reason for a seemingly higher AKI incidence in our study.

We saw higher adjusted odds of AKI in older individuals, African Americans, and individuals with hyperlipidemia and history of CKD. Older age has consistently been reported as a risk factor for worse outcome, including mortality and AKI.[5,12] Our observation of higher odds of AKI in CKD and higher prevalence of CKD in patients with AKI is not surprising because CKD is a well-known risk factor for AKI.[20] We also found higher odds of AKI in patients with hyperlipidemia. Hypercholesterolemia and hypertriglyceridemia are known to be independent predictors of CKD.[21,22] In addition, increased total cholesterol and low-density lipoprotein cholesterol levels measured at baseline have been reported as independent risk factors for end-stage renal disease.[21,22] Despite the known associations of hyperlipidemia and kidney disease, the mechanisms behind increasing AKI with hyperlipidemia are not completely understood and should be investigated. Higher AKI among African Americans is not surprising because racial and ethnic minorities are increasingly being recognized as having a more severe clinical course and worse outcomes with COVID-19 infection.[23]

Overall, AKI had a significant impact on the outcome of death among patients hospitalized with COVID-19 infection. Patients who developed AKI had a much higher mortality rate (58.1% [104 0f 179] vs 19.6% [29 of 148]; $P<.001$) when compared with those without and our analysis revealed significantly higher odds of mortality in patients with COVID-19 infection with AKI.

This study has strengths and limitations. This is a single-center retrospective study. Previous creatinine values were available in only 172 of 327 (52.6%) patients and thus a decision to use the lowest creatinine level in the hospital stay as the patients' baseline creatinine level was made. When previous creatinine values were available, they were comparable to and not significantly different from the lowest creatinine values during the hospital stay ($P=.5$). We could not include urine output in our determination of AKI given the heterogeneity of urine output recordings and the high degree of missing data. The incidence of rhabdomyolysis was not recorded for most patients in the study. However, the authors believe that even if some of the AKI was secondary to rhabdomyolysis from COVID-19 infection, that these patients eventually had that complication is a finding that is novel and worth reporting.

Strengths of the study include the accuracy of data that were manually extracted from

patient charts, a relatively large cohort, and using a systematic approach for multivariable analysis.

CONCLUSION

In addition to lungs, kidneys are particularly prone to disruption by COVID-19 infection. We present one of the first reports describing the incidence of AKI and potential risk factors for the development of AKI in patients with COVID-19 infection. We found significantly higher odds of AKI with increasing age, among African Americans, patients with hyperlipidemia, and those with CKD. We demonstrate an independent association between AKI and mortality with increasingly higher odds of mortality from progressively worsening renal failure in hospitalized patients with COVID-19 infection .

SUPPLEMENTAL ONLINE MATERIAL

Supplemental material can be found online at https://mcpiqojournal.org. Supplemental material attached to journal articles has not been edited, and the authors take responsibility for the accuracy of all data.

Abbreviations and Acronyms: **ACE-2** = angiotensin converting enzyme 2; **AKI** = acute kidney injury; **ARDS** = acute respiratory distress syndrome; **BMI** = body mass index; **CKD** = chronic kidney disease; **COVID-19** = coronavirus disease 2019; **OR** = odds ratio; **SARS-CoV-2** = severe acute respiratory syndrome coronavirus 2

Potential Competing Interests: The authors declare no competing interests.

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