COVID-19 in Patients with Chronic Kidney Disease in New York City

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ABSTRACT

Background Coronavirus disease 2019 (COVID-19) has affected millions of people, and several chronic medical conditions appear to increase the risk of severe COVID-19. However, our understanding of COVID-19 outcomes in patients with chronic kidney disease (CKD) remains limited.

Methods This was a retrospective cohort study of patients with and without CKD consecutively admitted with COVID-19 to three affiliated hospitals in New York City. Pre-COVID-19 CKD diagnoses were identified by billing codes and verified by manual chart review. In-hospital mortality was compared between patients with and without underlying CKD. Logistic regression was used to adjust this analysis for confounders and to identify patient characteristics associated with mortality.

Results We identified 280 patients with CKD, and 4098 patients without CKD hospitalized with COVID-19. The median age of CKD group was 75 (65-84) years, and age of non-CKD group 62 (48-75) years. Baseline (pre-COVID-19) serum creatinine in patients with CKD was 1.5 (1.2-2.2) mg/dL. In-hospital mortality was 30% in patients with CKD vs. 19.9% in patients without CKD (p<0.001). The risk of in-hospital death in patients with CKD remained significantly higher after adjustment for comorbidities (hypertension, diabetes mellitus, asthma, and chronic obstructive pulmonary disease), adjusted OR 1.4 [1.1-1.9]. When stratified by age, elderly patients with CKD (above age 70) had higher mortality than their age-matched control patients without CKD. In patients with CKD, factors associated with in-hospital mortality were age (adjusted OR 1.09 [1.06-1.12]), baseline and admission serum phosphorus (adjusted ORs 1.5 [1.03-2.1] and 1.4 [1.1-1.7]), serum creatinine on admission >0.3 mg/dL above the baseline (adjusted OR 2.6 [1.2-
Conclusions CKD is an independent risk factor for COVID-19 associated in-hospital mortality in elderly patients. Acute on chronic kidney injury increases odds of in-hospital mortality in CKD patients hospitalized with COVID-19.

INTRODUCTION

The coronavirus disease 2019 (COVID-19) global pandemic has affected millions of people. In spring 2020, New York City (NYC) was the epicenter of the COVID-19 pandemic in the U.S.. Several chronic medical conditions, such as hypertension, diabetes, and obesity, have been reported as risk factors for severe COVID-19. Chronic kidney disease (CKD) affects approximately 15% of the adult US population. CKD is associated with both immune activation, marked by systemic inflammation, and immune deficiency, manifesting as increased susceptibility to infections. Patients with CKD have higher mortality from non-COVID-19 associated pneumonia compared to patients with preserved renal function. The United States Centers for Disease Control and Prevention (CDC) warns that having chronic kidney disease of any stage increases the risk for severe illness from COVID-19. However, studies of COVID-19 outcomes in patients with pre-dialysis CKD remain scarce. Herein we characterize a cohort of patients with CKD and a control cohort of patients without CKD who were hospitalized with COVID-19 in spring and summer 2020 in NYC.

METHODS

This was a retrospective cohort study which was conducted at a major academic center and three affiliated hospitals in NYC. The study was approved by the Institutional Review Board of Weill Cornell Medicine. Patients with COVID-19 diagnosis (by reverse-transcriptase
polymerase chain reaction of nasopharyngeal swab specimens) that required in-patient admission were included. Patients who were diagnosed with COVID-19 in the emergency department but did not require admission were not included. We included all COVID-19 positive hospitalized patients who were discharged by August 31st, 2020. Data were abstracted electronically using institutional reporting database. The cohort was screened for patients with CKD using the ICD-10 diagnostic code of CKD (N18) in electronic medical records at any point since 2015 pre-COVID-19 diagnosis. CKD status was validated by manual chart review. Health care provider-recorded diagnosis of CKD either in prior medical records or in the emergency department was used for validation. Co-morbid conditions were identified by their ICD-10 codes.

Statistics
Continuous variables are presented as median (interquartile range). T-test was used to determine significance of differences between normally distributed continuous variables and chi-square test was used for the proportions. Covariates included in the multivariate analysis of the effect of CKD on mortality included sex, race and ethnicity, and the presence of comorbidities – hypertension, diabetes, asthma, and chronic obstructive pulmonary disease (COPD). To determine risk factors for in-hospital death in COVID-19 patients with underlying CKD, we used logistic regression. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for variables significant in univariate analysis; adjustment covariates included age, sex, race, and ethnicity. We used STATA for statistical analyses.

RESULTS
Our two cohorts included 280 patients with CKD and 4098 patients without a pre-existing diagnosis of CKD (Table 1). CKD group was older than the non-CKD group (median age 75 vs 62 years) years and had higher percentage of males (62.8% vs. 55.1%). Percentage of Black patients was higher in the CKD group than in non-CKD group (18.9% vs. 10.1%), indicating
disproportionate vulnerability of Black patients to COVID-19, consistent with previous reports. Comorbid conditions included hypertension, diabetes, asthma, COPD, and congestive heart failure; all of these conditions were more common in patients with pre-existing CKD than in patients without CKD.

Patients with CKD required mechanical ventilation more frequently than patients without CKD (Table 1). Patients with CKD were also more frequently diagnosed with septic shock. Acute kidney injury (AKI) was diagnosed in 53.6% of patients with CKD vs. in 21.8% of those without underlying CKD. In-hospital mortality was 30.0% in patients with CKD and 19.9% in patients without CKD (unadjusted p<0.001) (Table 1 and Figure 1a). After adjustment for demographic characteristics and comorbid conditions (Supplemental table S1), CKD remained a significant risk factor for in-hospital mortality (adjusted OR, 1.4 [1.1-1.9]). Importantly, when both CKD and non-CKD cohorts were stratified by age, patients aged over 70 years had higher mortality than their age-matched counterparts without CKD (Figure 1b).

Within CKD cohort, the subgroup of patients who died in the hospital was older than the subgroup of those who survived (Table 2 and Figure 2). As expected, higher in-hospital mortality was observed with higher CKD stages (Supplemental figure S1). In univariate analysis, other factors associated with increased odds of mortality were: having primary language other than English (OR 1.9 [1.1-3.1]), BMI<25 kg/m2 (OR 1.8 [1.1-3.0]), pre-COVID-19 BUN (1.03 [1.01-1.05]), serum phosphorus on admission (OR 1.3 [1.1-1.5]), CRP (OR 1.1 [1.05-1.2]), elevated serum creatinine on admission (OR 2.9 [1.6-5.6]), and diagnosis of AKI during hospitalization (OR 3.9 [2.2-7.1]). Baseline use of angiotensin receptor blockers and higher lymphocyte count on admission were associated with increased odds of survival (OR 0.5 [0.3-0.9] and 0.5 [0.2-0.9] respectively). After adjustment for demographic characteristics (Figure 2), variables that remained significantly associated with mortality were age (OR 1.09 [1.06-1.12]), admission serum phosphorus (1.1 [1.4-1.7]), admission CRP (1.05 [1.02-1.11]), admission serum creatinine above baseline (2.6 [1.2-5.4]), and diagnosis of AKI during hospitalization (OR 3.9 [2.2-7.1]).
hospitalization (4.6 [2.3-8.9]). In addition, in multivariate analysis baseline serum phosphorus was significantly associated with mortality (adjusted OR 1.5 [1.03-2.1]).

**DISCUSSION**

Identification and stratification of risk factors for severe COVID-19 disease are important for the development of effective preventative strategies and interventions. While emerging data support the role of hypertension, diabetes, and coronary artery disease as risk factors for severe COVID-19\(^1\), the significance of CKD as an underlying condition for severe COVID-19 remains less well understood. In a meta-analysis of early reports from China, no study individually found CKD as significant predictor of severe COVID-19. However, when data of individual studies were pooled, a significant association between CKD and severe COVID-19 was observed [OR 3.03 (1.09–8.47)].\(^7\) In a cohort of 5700 patients from NYC area hospitalized with COVID-19, the reported prevalence of CKD was 5% and ESRD 3.5% based on the available ICD-10 diagnostic codes in medical history\(^12\). COVID-19 in dialysis patients received close attention mainly from the standpoint of developing preventative guidelines, given frequent aggregation of these patients in hemodialysis units.\(^13\) However, data on the outcomes of COVID-19 in pre-dialysis CKD patients remain scarce.\(^8\) In a recent multicenter study of patients admitted to the intensive care units across the United States, the presence of pre-existing kidney failure treated by maintenance dialysis was strongly associated with in-hospital death, whereas pre-existing non-dialysis CKD had an intermediate association, compared to no pre-existing CKD\(^9\).

In the present study, we reported the role of CKD in COVID-19-associated in-hospital mortality based on the cohort of 4378 patients hospitalized with COVID-19, 280 of whom carried the diagnosis of CKD prior to COVID-19 pandemic. Patients with CKD had approximately 50% higher in-hospital mortality than patients without CKD had. In a cohort of 1603 patients admitted with COVID-19 in Spain, underlying CKD was a risk factor for in-hospital death with hazard ratio
1.59 [1.06-2.37]. In a multicenter study of 4264 COVID-19 patients admitted to the intensive care units in the United States, CKD had a slightly lower risk of in-hospital death, hazard ratio 1.25 [1.08-1.44]. While in both studies associations were adjusted for age, a stratification of mortality by age was not reported. It has been well established that COVID-19 disproportionately affects elderly within the general population, while the role of age in patients with CKD and COVID-19 remained less well understood. In our cohort, the overall difference in mortality between CKD and non-CKD cohorts was most pronounced in the elderly patients. In the subgroup of patients younger than 70, we did not observe differences in mortality between patients with and without underlying CKD. Our results highlight that CKD may be particularly significant risk factor for mortality in elderly patients with COVID-19 and warrant further analysis of the role of CKD as risk factor for adverse outcomes in COVID-19 separately in younger, middle age, and elderly population.

CKD-specific patient characteristics that may be responsible for adverse outcomes of COVID-19 have received little attention to date. In addition to advanced age, several CKD complications were associated with in-hospital death in patients with CKD in our cohort. It has been well established that obesity is a risk factor for adverse outcomes of COVID-19. In our univariate analysis, BMI appeared to follow the reverse epidemiology in patients with CKD. Indeed, patients with BMI<25kg/m² were at higher risk for mortality. This phenomenon has been described for all-cause mortality in patients with CKD. Our findings warrant further investigation of nutritional parameters in patients with CKD and COVID-19 and their role in outcomes.

While most patients in our CKD cohort did not have severe hyperphosphatemia, pre-COVID-19 and admission serum phosphorus were independently associated with in-hospital death in patients with CKD. Distorted phosphorus homeostasis, a hallmark of CKD-mineral and bone disorder (MBD), has profound vascular effects in patients with CKD. Severe COVID-19 frequently leads to endothelial injury. In future studies, it would be of interest to clarify if
vascular component of CKD-MBD predisposes patients with CKD to more severe vascular injury from COVID-19.

AKI is a common complication of COVID-19 in hospitalized patients\textsuperscript{20}. It was suggested that early evaluation of renal reserve in the course of COVID-19 may inform therapeutic interventions\textsuperscript{21}. In patients with CKD, episodes of acute on chronic kidney injury have been characterized as novel risk factors for disease progression\textsuperscript{22}. At the same time, the role of acute on chronic kidney injury in outcomes of COVID-19 in patients with CKD has not been fully elucidated. In our cohort of patients hospitalized with COVID-19, patients with CKD were diagnosed with AKI 2.5 times more frequently than patients without CKD. Patients with CKD who died in the hospital had AKI more frequently than those patients with CKD who survived. The diagnosis of AKI during hospitalization was the strongest predictor of in-hospital death in CKD patients with COVID-19 among the potential risk factors that we analyzed (adjusted OR 4.6 [2.3-8.9]). In future studies, it would be important to investigate the mechanisms of acute on chronic kidney injury in patients with COVID-19 and underlying CKD, and to test the effectiveness of preventative measures (e.g., early and aggressive fluid resuscitation in patients without oliguria) in improving COVID-19 outcomes in patients with CKD.

The strengths of our study included analysis of a large single-center cohort of consecutively hospitalized patients with COVID-19 who had underlying CKD, and comparing the outcomes with control group of patients without CKD. Our study had limitations. Baseline laboratory data were not available for all patients. Our analysis was focused on in-hospital mortality and we did not follow patients post discharge. Electronic data abstraction may not have provided the degree of granularity that can be achieved in a fully manual chart review. While we presented the largest to date single-center cohort of COVID-19-positive patients with CKD, an even larger sample size would be required to render more precise estimates of the effects that baseline CKD-specific patient characteristics may have on outcomes.
In conclusion, our findings indicate that pre-dialysis CKD is an independent risk factor for in-hospital death in elderly patients with COVID-19. Ongoing control of CKD complications, as well as early and aggressive measures to prevent the development of acute on chronic kidney injury may serve as opportunities to improve outcomes of COVID-19 in elderly patients with CKD. Further evaluation of the role that CKD may play in COVID-19 outcomes in different age groups is warranted.

DISCLOSURES
J.J.C. reported research support from NIH/NCATS grant KL2-TR-002385, Roche Diagnostics, and Allergan outside the submitted work. The spouse of M.E.C. is a cofounder and shareholder, and serves on the Scientific Advisory Board of Proterris, Inc. All remaining authors have nothing to disclose.

FUNDING
O.A. reported receiving research support from the National Institutes of Health (NIH) National Institute of Diabetes and Digestive and Kidney Diseases K08 DK114558, and is a recipient of the Rohr Family Clinical Scholar Award (Weill Cornell Medicine). M.E.C. reported research support from NIH grants R01 HL133801, R01 HL055330, and R01 HL132198.

ACKNOWLEDGEMENTS
We thank the staff at Weill Cornell Medicine and affiliated New York Presbyterian hospitals for their care of patients with COVID-19. Weill Cornell Medicine Institutional COVID-19 research data repository received support from NewYork-Presbyterian Hospital and Weill Cornell Medical College, including the Clinical and Translational Science Center (UL1 TR000457) and Joint Clinical Trials Office. We greatly appreciate the assistance of Sajjad Abedian, Weill Cornell
Medicine research informatics analyst, and the help of Weill Cornell medical students, Keba Shaaban and Rachel Abramson with reviewing medical records.

AUTHOR CONTRIBUTIONS

O Akchurin: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Supervision; Validation; Visualization; Writing - original draft; Writing - review and editing
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All authors approved the final version of the manuscript.

SUPPLEMENTAL MATERIAL

Supplemental Table S1. Role of CKD in COVID-19-associated in-hospital mortality: multivariate analysis

Supplemental Figure S1. In hospital mortality in COVID-19 patients with pre-existing CKD by
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### Table 1. Demographic and clinic characteristics of hospitalized COVID-19 patients, with and without underlying CKD.

| Patient characteristics                  | No CKD (n=4098) | CKD (n=280) | p     |
|------------------------------------------|-----------------|-------------|-------|
| Age, years                               | 62 (48-75)      | 75 (65-84)  | <0.001|
| Male sex                                 | 2244 (55.1%)    | 176 (62.8%) | 0.009 |
| Borough of residence:                     |                 |             |       |
| Manhattan                                | 742 (18.1%)     | 100 (35.8%) | <0.001|
| Queens                                   | 2657 (64.9%)    | 111 (39.8%) | <0.001|
| Brooklyn                                 | 454 (11.1%)     | 41 (14.7%)  | 0.06  |
| The Bronx                                | 154 (3.8%)      | 19 (6.8%)   | 0.012 |
| Race and ethnicity:                       |                 |             |       |
| White race                               | 1099 (26.8%)    | 87 (31.2%)  | 0.11  |
| Black race                               | 415 (10.1%)     | 52 (18.9%)  | <0.001|
| Asian race                               | 635 (15.5%)     | 42 (15.1%)  | 0.83  |
| Hispanic ethnicity                        | 1292 (31.7%)    | 58 (20.8%)  | <0.001|
| Comorbidities:                            |                 |             |       |
| Hypertension                             | 1684 (41.1%)    | 232 (82.8%) | <0.001|
| Diabetes mellitus                        | 1052 (25.7%)    | 155 (55.3%) | <0.001|
| Congestive heart failure                 | 339 (8.3%)      | 109 (38.9%) | <0.001|
| Asthma                                   | 815 (8.3%)      | 38 (13.6%)  | 0.003 |
| COPD                                     | 233 (2.6%)      | 44 (15.7%)  | <0.001|
| Baseline laboratory characteristics      |                 |             |       |
| Serum creatinine, mg/dL                  | 0.8 (0.7-1.1)   | 1.5 (1.2-2.2) | <0.001|
| BUN, mg/dL                               | 16 (12.5-21.2)  | 31 (20.2-44) | <0.001|
| Hemoglobin, g/dL                         | 12.1 (10.7-13.5)| 11.6 (9.8-13.1)| <0.001|
| Outcomes                                 |                 |             |       |
| Mechanical ventilation                   | 329 (8.02%)     | 37 (13.2%)  | 0.002 |
| Septic shock                             | 169 (4.1%)      | 27 (9.6%)   | <0.001|
| Acute kidney injury                      | 892 (21.8%)     | 142 (53.6%) | <0.001|
| In-hospital mortality                    | 815 (19.9%)     | 84 (30%)    | <0.001|

Data are shown as median (interquartile range) or n (%). COPD, chronic obstructive pulmonary disease. BUN, blood urea nitrogen.
Table 2. Demographic and clinical characteristics of hospitalized COVID-19 patients with underlying CKD.

| Patient characteristics | All patients (n=280) | Survivors (n=196) | Non-survivors (n=84) | p     |
|-------------------------|----------------------|-------------------|----------------------|-------|
| Age, years              | 75 (65-84)           | 70 (59-80)        | 86 (76-90)           | <0.001|
| Male sex                | 176 (63.1%)          | 125 (64.1%)       | 51 (60.7%)           | 0.59  |
| Borough of residence:   |                      |                   |                      |       |
| Manhattan               | 100 (35.8%)          | 73 (37.2%)        | 27 (32.1%)           | 0.39  |
| Queens                  | 111 (39.8%)          | 69 (35.2%)        | 42 (50.0%)           | 0.02  |
| Brooklyn                | 41 (14.7%)           | 31 (15.8%)        | 10 (11.9%)           | 0.39  |
| The Bronx               | 19 (6.8%)            | 15 (7.7%)         | 4 (4.8%)             | 0.37  |
| Race and ethnicity:     |                      |                   |                      |       |
| White race              | 87 (31.2%)           | 53 (27.0%)        | 34 (40.4%)           | 0.02  |
| Black race              | 52 (18.6%)           | 40 (20.4%)        | 12 (14.3%)           | 0.22  |
| Asian race              | 42 (15.1%)           | 25 (12.8%)        | 17 (20.2%)           | 0.11  |
| Hispanic ethnicity      | 58 (20.8%)           | 45 (22.9%)        | 13 (15.5%)           | 0.15  |
| Maintenance medications (pre-COVID-19): | | | | |
| ACE-inhibitors          | 62 (22.1%)           | 46 (23.5%)        | 16 (19.1%)           | 0.41  |
| Angiotensin receptor blockers | 93 (33.2%)       | 73 (37.2%)        | 20 (23.8%)           | 0.02  |
| Diuretics               | 118 (42.1%)          | 82 (41.8%)        | 36 (42.9%)           | 0.87  |
| Sevelamer               | 23 (8.2%)            | 15 (7.6%)         | 8 (9.5%)             | 0.61  |
| Calcitriol              | 28 (10%)             | 21 (10.7%)        | 7 (8.3%)             | 0.53  |
| Antihyperlipidemic agents | 186 (66.4%)        | 133 (67.8%)       | 53 (63.1%)           | 0.43  |
| Comorbidities:          |                      |                   |                      |       |
| Hypertension            | 232 (82.9%)          | 162 (82.6%)       | 70 (83.3%)           | 0.89  |
| Diabetes mellitus       | 155 (55.4%)          | 112 (57.1%)       | 43 (51.2%)           | 0.36  |
| Congestive heart failure| 109 (38.9%)          | 72 (36.7%)        | 37 (44.1%)           | 0.25  |
| Asthma                  | 38 (13.6%)           | 29 (14.8%)        | 9 (10.7%)            | 0.36  |
| COPD                    | 44 (15.7%)           | 31 (15.8%)        | 13 (15.4%)           | 0.94  |
| BMI, kg/m²              | 25.7 (21.7-29.2)     | 26 (22.4-30.5)    | 24.2 (21.3-29.2)     | 0.40  |
| EGFRL (mL/min/1.73 m²)  |                      |                   |                      |       |
| N=192                   | 1.5 (1.2-2.2)        | 1.4 (1.1-2.0)     | 1.6 (1.3-2.7)        | 0.21  |
| BUN, mg/dL              | 31 (21-44)           | 29 (19-42)        | 35 (25-53)           | 0.01  |
| Hemoglobin, g/dL        | 11.6 (9.8-13.1)      | 11.8 (10-13.2)    | 11.3 (9.4-12.5)      | 0.23  |
| Ferritin, ng/mL         | 160.5 (60-357.5)     | 104.1 (52-349)    | 262.9 (139-473)      | 0.02  |
| Phosphorus, mg/dL       | 3.6 (3.2-4.2)        | 3.6 (3.2-4.1)     | 3.7 (3.2-4.3)        | 0.91  |
| PTH, pg/mL              | 81.9 (57.8-120.5)    | 80.8 (60.1-96.7)  | 92.1 (46.2-197.9)    | 0.46  |
| CRP, mg/dL              | 3.6 (0.8-4.8)        | 2.2 (0.6-4.6)     | 4.2 (2.4-5.6)        | 0.02  |
| Lymphocyte count, x 10⁷ mm⁻³ | 1.2 (0.9-1.7) | 1.2 (0.9-1.7) | 1.2 (0.8-1.8) | 0.72  |

| Laboratory characteristics on admission: | |
|-----------------------------------------|-----------------------------------------|
| Creatinine, mg/dL                       | n=268 1.98 (1.4-3.2)                  |
| BUN, mg/dL                              | n=228 43 (27-69)                      |
| Hemoglobin, g/dL                        | n=270 11.8 (9.8-13.6)                 |
| Ferritin, ng/mL                         | n=185 688.9 (351-1318.0)              |
| Phosphorus, mg/dL                       | n=188 3.85 (3.3-5)                    |
| CRP, mg/dL                              | n=181 10.4 (4.9-19.3)                 |
| Lymphocyte count, x 10⁷ mm⁻³            | n=248 0.8 (0.5-1.2)                   |

| Laboratory characteristics at discharge: | |
|-----------------------------------------|-----------------------------------------|
| Creatinine, mg/dL                       | n=153 1.8 (1.2-3.0)                  |
| Phosphorus, mg/dL                       | n=85 4.1 (3.1-5.2)                    |

Data are shown as median (interquartile range) or n (%). ACE, angiotensin-converting enzyme. ARB, angiotensin receptor blocker. BMI, body mass index. PTH, parathyroid hormone.
FIGURE LEGENDS

Figure 1. In-hospital mortality in COVID-19 patients with and without CKD, overall (a) and stratified by age (b).

Figure 2. Analysis of factors associated with in-hospital mortality in COVID-19 patients with CKD.
ACE, angiotensin-converting enzyme. ARB, angiotensinreceptor blocker. BMI, body mass index. GFR, glomerular filtration rate. CRP, C-reactive protein. BUN, blood urea nitrogen. AKI, acute kidney injury.
* Adjusted by age, sex, race and ethnicity.
** Creatinine rise was defined as admission serum creatinine >0.3 mg/dL above the baseline (pre-COVID-19) serum creatinine.
Figure 1. In-hospital mortality in COVID-19 patients with and without CKD, overall (a) and stratified by age (b).
Figure 2. Analysis of factors associated with in-hospital mortality in COVID-19 patients with CKD.