Predictors for Severe COVID-19 Infection

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Background. COVID-19 is a pandemic disease caused by a novel coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Predictors for severe COVID-19 infection have not been well defined. Determination of risk factors for severe infection would enable identifying patients who may benefit from aggressive supportive care and early intervention.

Methods. We conducted a retrospective observational study of 197 patients with confirmed COVID-19 admitted to a tertiary academic medical center.

Results. Of 197 hospitalized patients, the mean (SD) age of the cohort was 60.6 (16.2) years, 103 (52.3%) were male, and 156 (82.1%) were black. Severe COVID-19 infection was noted in 74 (37.6%) patients, requiring intubation. Patients aged above 60 were significantly more likely to have severe infection. Patients with severe infection were significantly more likely to have diabetes, renal disease, and chronic pulmonary disease and had significantly higher white blood cell counts, lower lymphocyte counts, and increased C-reactive protein (CRP) than patients with nonsevere infection. In multivariable logistic regression analysis, risk factors for severe infection included pre-existing renal disease (odds ratio [OR], 7.4; 95% CI, 2.5–22.0), oxygen requirement at hospitalization (OR, 2.9; 95% CI, 1.3–6.7), acute renal injury (OR, 2.7; 95% CI, 1.3–5.6), and CRP on admission (OR, 1.006; 95% CI, 1.001–1.01). Race, age, and socioeconomic status were not independent predictors.

Conclusions. Acute or pre-existing renal disease, supplemental oxygen upon hospitalization, and admission CRP were independent predictors for the development of severe COVID-19. Every 1-unit increase in CRP increased the risk of severe disease by 0.06%.

Keywords. predictors; risk factors; severe COVID-19.

In December 2019, the first pneumonia cases of unknown origin were identified in Wuhan city, Hubei province, China [1]. The pathogen was identified as a novel coronavirus (nCoV), now called severe acute respiratory syndrome coronavirus (SARS-CoV), with the disease termed coronavirus disease 2019 (COVID-19) [2]. Because of its rapid spread, the World Health Organization has declared COVID-19 as a pandemic [3]. As of 28 April 2020, a total of 3 090 844 confirmed cases had been reported in 184 countries [4]. COVID-19 infections have been described among asymptomatic (who never developed symptoms) as well as presymptomatic patients (who are not yet symptomatic) [5–10].

The clinical spectrum from the largest cohort of symptomatic patients with COVID-19 from China ranged from mild to critically ill cases [11]. Age was described as a strong risk factor for severe disease, with the highest case fatality occurring in those 80 years and older [11–13]. Preliminary data from the United States also suggested that adverse outcomes were most frequent among persons 85 years of age and older, but it was also recognized that severe infections could occur in adults of any age group [14, 15]. Comorbid conditions of hypertension, diabetes, and chronic lung and renal disease were also associated with severe infections and adverse outcomes [16–18]. Medications such as nonsteroidal anti-inflammatory drugs, angiotensin-converting enzyme (ACE) inhibitors, or angiotensin receptor blockers (ARBs) were suggested to increase the severity of infection [19], but currently there are no data to suggest a link between these medications and adverse outcomes. The determination of risk factors for severity in COVID-19 infection would enable the identification of high-risk patients who may benefit from close monitoring, aggressive supportive care, and early intervention.

To address this question, we collected clinical data from a cohort of hospitalized patients with the aim of identifying predictors for developing severe COVID-19 infections.

METHODS

Study Setting and Design

We conducted a single-center, retrospective observational study at a 776-bed tertiary care urban academic medical center. The study was approved by the Ascension St John Hospital Institutional Review Board. Patients with confirmed COVID-19 (positive real-time reverse transcriptase–polymerase chain reaction [RT-PCR] assay of a nasopharyngeal swab) from 8
March to 8 April 2020 were included. COVID-19 testing for all patients under investigation was done according to criteria defined per the US Centers for Disease Control and Prevention (CDC) [20]. Patients' nasopharyngeal swab specimens were sent to the Michigan Department of Health and Human Services (MDHHS) Bureau of Laboratories for RT-PCR testing after obtaining an nCoV identification number (ID) from the MDHHS Communicable Disease Division. These nCoV ID numbers were tracked by the institution's infection-control database. All studied subjects were identified using this database.

Data Collection
Data were collected from the electronic medical record (EMR) for all patients meeting the inclusion criteria. Clinical parameters included age, sex, race, residential zip code plus 4, presence of comorbid conditions (according to the Charlson Comorbidity Index, a validated, weighted index score that predicts mortality), patient's home medications, vital signs on presentation, presenting symptoms, laboratory and radiological findings on admission, status on intubation and intensive care unit (ICU) admission, and hospital discharge or inpatient death [23]. Clinical outcomes will be addressed in another report.

Definitions
A COVID-19 infection was defined as “severe” if the patient required mechanical ventilation. Obesity and severe obesity were defined according to CDC definitions [22]. Fever was defined as an axillary temperature of 37.5°C or higher. Lymphocytopenia was defined as a lymphocyte count of less than 1500 cells per cubic millimeter. Thrombocytopenia was defined as a platelet count of less than 150 000 per cubic millimeter. Acute renal injury or elevated creatinine on admission was defined as an increase in serum creatinine by 0.3 mg/dL or higher (≥26.5 μmol/L) within 48 hours or an increase in serum creatinine to 1.5 times baseline or higher, which is known or presumed to have occurred within the prior 7 days [24]. Patients with pre-existing renal disease were on dialysis, had a history of renal transplant, had uremic syndrome, or had a creatinine level greater than 3 mg/dL in prior admissions. Using the 9-digit zip code, the area deprivation rank for each individual patient was also obtained. The Area Deprivation Index (ADI) is a measurement of healthcare deprivation based upon where a person lives; it correlates with socioeconomic status [24]. A higher ADI means more deprived. On a national level the ADIs are ranked from 1 to 100 (1 = least deprived, 100 = most deprived) and on a state level the ADIs are ranked from 1 to 10 (1 = least deprived, 10 = most deprived).

Statistical Analysis
Statistical analysis was performed using SPSS version 26.0 (IBM Corporation, Armonk, NY). Descriptive statistics were generated to characterize the study population. Continuous variables were described as the mean with standard deviation (SD) or median with interquartile range (IQR). Univariable analysis was done using Student’s t test, analysis of variance followed by multiple pairwise comparisons using the Bonferroni correction of the P value, the Mann-Whitney U test, and chi-square analysis. Variables that were found to be significant (P < .05) predictors of severity were then entered a multivariable logistic regression model using a forward likelihood ratio algorithm. When 2 variables were measuring the same underlying factor, the variable with the highest univariable measure of association was used in the model. Results from the regression are reported as odds ratios with 95% confidence intervals. All reported P values are 2-sided.

RESULTS
A total of 197 hospitalized patients with confirmed COVID-19 were included in the analysis. The mean (SD) age of the cohort was 60.6 (16.2) years, 103 (52.3%) were male, and 156 (82.1%) were black. The mean body mass index of the cohort was 34.4 (9.0) kg/m². Of these patients, 161 (84.7%) had at least 1 comorbid condition listed. Hypertension was the most common underlying condition, which was present in 138 (70.1%) patients, followed by diabetes in 73 (37.1%) patients and chronic pulmonary diseases in 38 (19.3%) patients. History of contact with a sick person was present in 60 (31.6%) patients. The mean duration of symptoms prior to hospitalization was 5.4 (3.8) days.

Severe COVID-19 infection was noted in 74 (37.6%) patients, requiring intubation. The median time from hospitalization to intubation was 10.9 hours (IQR: 0.0, 58.2 hours). Time to intubation from the time of hospital admission was less than 2 hours in 22 (29.7%) patients and 2 hours or more in 52 (70.3%) patients. All 74 (100%) intubated patients with severe infection were admitted to the ICU compared with 1 (0.8%) patient among the nonsevere cases. The patients with severe infection tended to be older (mean [SD] age, 63.1 [15.9] vs 59.1 [17.3] years; P = .08) than nonsevere patients. Patients aged 60 years and older were significantly more likely to have severe infection (46 [62.2%] vs 58 [47.2%]; P = .04). Patients with severe infection were more likely to have at least 1 comorbid condition (64 [91.4%] vs 97 [80.8%]; P < .05) and were significantly more likely to have diabetes (36 [48.6%] vs 37 [30.1%]; P = .009), renal disease (22 [29.7%] vs 12 [9.8%]; P < .0001), and chronic pulmonary diseases (20 [27.0%] vs 18 [14.6%]; P = .03). Patients with severe infection were significantly more likely to be tachypneic (mean [SD] breaths/min, 27.6 [10] vs 22.8 [6.6]; P < .0001) and have low oxygen saturation (percentage [SD]) 91.4 [9.4] vs 94.7 [4.3]; P = .006) at the time of hospital admission.

There was no significant association found between race and severity of illness (P = .4). To examine this further, we also assessed the association between the median state ADI rank with both race and severity of disease. Although there was a
significant association between race and median state ADI rank (white median state rank, 6.0 [IQR, 2.8, 8] vs black median rank, 9.0 [IQR, 7, 9.75]) (P < .0001), there was no association between median state ADI rank and severity of disease.

The most common symptoms at the onset of illness in the studied cohort were cough (141 [73.1%]), shortness of breath (140 [71.1%]), and fever (116 [59.8%]). Among patients with severe disease, 28.4% were confused on presentation compared with 14.6% of those with nonsevere disease (P = .02). History of contact with a sick individual and presence of sore throat and cough were significantly less frequent in patients with severe disease compared with those with nonsevere disease. Patients with severe infection demonstrated an increased inflammatory response, including higher white blood cell counts, lower lymphocyte and platelet counts, and increased C-reactive protein (CRP) levels compared with those patients with nonsevere infection. Although patients with severe infection had significantly elevated procalcitonin levels, which raises concern for the presence of secondary bacterial infection, these patients also had acute renal injury on admission, which may have caused the elevated procalcitonin.

For all demographic data, clinical characteristics, and laboratory findings in univariate analysis shown in Table 1, we identified each variable that showed and/or reached statistical significance with P < .05 between severe and nonsevere infections. For multivariable logistic regression (Table 2), variables initially entered into the model included age, sex, chronic pulmonary disease, renal disease, altered mental status, fever within 24 hours of admission, respiratory rate on admission, oxygenation on admission, cough, shortness of breath, abnormal chest X-ray on admission, initial procalcitonin level, elevated creatinine from baseline, and initial CRP level. After 4 iterations, the model with the lowest −2-log likelihood value included 4 variables that were independent predictors for severity of COVID-19 infection, including presence of pre-existing renal disease, need for supplemental oxygen at the time of hospitalization, and elevated creatinine and CRP on admission laboratory findings.

DISCUSSION

Unlike SARS-CoV and Middle Eastern respiratory syndrome (MERS-CoV), COVID-19 has become a pandemic. While little has been reported regarding the predictors for severe COVID-19 infection, much is known regarding the risk factors and predictors for mortality [11, 25]. In our study we report pre-existing renal disease, supplemental oxygen requirement at admission, acute renal insufficiency, and CRP values on admission as independent predictors of severe COVID-19 infections.

The kidneys play a key role in immune hemostasis. Reduced renal function delays the clearance of circulating cytokines, leading to a persistent inflammatory state [26]. Renal dysfunction causes reduced lymphocyte numbers and function, creating an immunodeficient state, predisposing to severe infections [27]. Patients with renal disease often have the additional comorbidities of either diabetes or hypertension; yet interestingly, neither diabetes nor hypertension emerged as predictors of COVID-19 disease severity in the multivariable logistic regression analysis. Perhaps renal disease represents those patients with poorer control of either diabetes or hypertension (leading to the development of chronic kidney disease) and this explains why the latter 2 conditions fell out in multivariable analysis; well-controlled diabetes and hypertension may not portend the same risk as chronically uncontrolled disease. Some patients also had end-stage renal disease, which is a state of immune dysregulation in which buildup of uremic toxins and cytokines activates innate immune cells leading to a vicious cycle of further cytokine release and reactive oxygen species production [26]. Patients with kidney disease may also have received different antihypertensive or anti-hyperglycemic medications than those who did not have chronic kidney disease; we did not evaluate this specific potential in our study.

It is also interesting that acute renal insufficiency (whether in patients with chronic kidney disease or normal baseline renal function) was associated with adverse outcome. SARS-CoV-2 is strongly suspected to use ACE2 as its receptor, and ACE2 binding affinity has been shown to be one of the most important determinants of SARS-CoV infectivity [28]. Perhaps acute renal insufficiency reflects more efficient binding of SARS-CoV-2 to ACE2 given the location of ACE2 expression. Interestingly, ACE inhibitors and ARBs increase ACE2 expression, yet our analysis did not detect an association between ACE inhibitor or ARB use, or hypertension or diabetes, and disease severity. Persons taking ACE inhibitors or ARBs at home but presenting with renal insufficiency generally have those medications held upon admission. While SARS-CoV-2 enters cells by binding to ACE2, ACE2 also reduces inflammation [19]. If one hypothesizes that ACE2 expression decreases inflammation, and that withholding drugs that increase its expression was done mainly in patients with acute renal insufficiency, perhaps a pro-inflammatory reaction to drug withdrawal could contribute to an unfavorable outcome in such patients.

The need for supplemental oxygen for baseline hypoxia was an independent factor for severe disease in our study. A recently published study showed that oxygen saturation below 90% despite oxygen supplementation was a powerful predictor for a fatal outcome [29]. Given that ACE2 is expressed in lung epithelium, hypoxia may represent more avid binding to SARS-CoV-2 in those hosts. Interestingly, ACE2 is also expressed by endothelial cells, which represent one-third of lung cells [30]. The endothelium functions to promote vasodilation, fibrinolysis, and anti-aggregation; thus, endothelial damage may lead to a hypercoagulable state [31]. Accumulation of coagulation factors in lungs can drive ARDS through activation of
Table 1. Univariate Analysis of Predictors for Severe COVID-19 Infection

| Characteristics                      | Nonsevere (n = 123) (%) | Severe (n = 74) (%) | OR (95% CI)         | P     |
|--------------------------------------|-------------------------|---------------------|---------------------|-------|
| Age group                            |                         |                     |                     |       |
| <60 years                            | 65 (52.8)               | 28 (37.8)           | 1.8 (1.02, 3.3)     | .04   |
| >60 years                            | 58 (47.2)               | 46 (62.2)           |                     |       |
| Sex                                  |                         |                     |                     |       |
| Male                                 | 58 (47.2)               | 45 (60.8)           | 0.6 (0.3, 1.03)     | .06   |
| Female                               | 65 (52.8)               | 29 (39.2)           |                     |       |
| Race                                 |                         |                     |                     |       |
| White                                | 19 (15.8)               | 15 (21.4)           | 0.7 (0.3, 1.5)      | .38   |
| Black                                | 101 (84.2)              | 55 (78.6)           |                     |       |
| Comorbidities                        |                         |                     |                     |       |
| At least 1 comorbidity               | 97 (80.8)               | 64 (91.4)           | 2.32 (1.95, 5.69)   | .061  |
| Myocardial infarction                | 5 (4.1)                 | 4 (5.4)             | 1.34 (1.35, 5.19)   | .663  |
| Congestive heart failure             | 10 (8.1)                | 10 (13.5)           | 1.76 (1.70, 4.47)   | .226  |
| Peripheral vascular disease          | 0 (0.0)                 | 5 (6.8)             |                     |       |
| Cerebrovascular disease              | 9 (7.3)                 | 8 (10.8)            | 1.53 (1.57, 4.17)   | .398  |
| Dementia                             | 16 (13.0)               | 5 (6.8)             | .49 (1.17, 1.38)    | .169  |
| Chronic pulmonary disease            | 18 (14.6)               | 20 (27.0)           | 2.16 (1.06, 4.42)   | .03   |
| Connective tissue disease            | 0 (0.0)                 | 2 (2.7)             |                     |       |
| Peptic ulcer disease                 | 3 (2.4)                 | 2 (2.7)             | 1.11 (1.18, 6.81)   | .90   |
| Diabetes                             | 37 (30.1)               | 36 (48.6)           | 2.20 (1.21, 4.0)    | .009  |
| Hemiplegia                           | 5 (4.1)                 | 4 (5.4)             | 1.35 (1.35, 5.19)   | .66   |
| Renal disease                        | 12 (9.8)                | 22 (29.7)           | 3.91 (1.80, 8.51)   | <.0001 |
| Any malignancy                       | 6 (4.9)                 | 4 (5.4)             | 1.11 (1.30, 4.09)   | .87   |
| Metastatic solid tumor               | 3 (2.4)                 | 2 (2.7)             | 1.11 (1.18, 6.81)   | .91   |
| Mild liver disease                   | 2 (1.6)                 | 3 (4.1)             | 2.56 (4.2, 15.67)   | .29   |
| Moderate–severe liver disease        | 0 (0.0)                 | 1 (1.4)             |                     |       |
| AIDS                                 | 1 (0.8)                 | 0 (0.0)             |                     |       |
| Median CWIC (25th, 75th)             | 0 (0, 2)                | 1.5 (0, 3)          |                     | .001  |
| Hypertension                         | 82 (66.7)               | 56 (75.7)           | 1.56 (1.81, 2.98)   | .18   |
| Current tobacco smoker               | 5 (4.1)                 | 6 (8.3)             | 2.11 (1.21, 4.0)    | .22   |
| Obesity                              | 78 (64.5)               | 53 (71.6)           | 1.39 (1.74, 2.61)   | .30   |
| Morbid obesity                       | 29 (24.6)               | 19 (25.7)           | 1.06 (1.54, 2.07)   | .86   |
| Home medications                     |                         |                     |                     |       |
| Either ACEIs or ARBs                 | 40 (32.5)               | 27 (36.5)           | 1.19 (1.65, 2.18)   | .57   |
| Steroids                             | 6 (4.9)                 | 3 (4.1)             | .82 (0.20, 3.40)    | .79   |
| History of sick contact              | 44 (37.0)               | 16 (22.5)           | .51 (1.3, .9)       | .03   |
| Typical symptoms                     |                         |                     |                     |       |
| Fever                                | 72 (58.5)               | 44 (62.0)           | 1.15 (1.63, 2.10)   | .63   |
| Sore throat                          | 18 (14.6)               | 2 (2.9)             | .17 (0.04, .78)     | .01   |
| Headache                             | 13 (10.6)               | 4 (5.8)             | .52 (1.26, 1.67)    | .264  |
| Rhinorrhea                           | 7 (5.7)                 | 2 (2.9)             | .50 (0.10, 2.45)    | .38   |
| Shortness of breath                  | 83 (67.5)               | 57 (80.3)           | 1.96 (1.98, 3.94)   | .055  |
| Cough                                | 96 (78.0)               | 45 (64.3)           | .51 (0.27, 0.97)    | .038  |
| Hemoptysis                           | 1 (0.8)                 | 1 (1.4)             | 1.77 (1.11, 28.71)  | .69   |
| Fatigue                              | 41 (33.3)               | 21 (30.0)           | .86 (0.46, 1.62)    | .63   |
| Myalgia                              | 29 (23.8)               | 16 (22.9)           | .95 (0.47, 1.91)    | .89   |
| Nausea, vomiting                     | 23 (18.7)               | 9 (12.9)            | .64 (0.28, 1.48)    | .29   |
| Diarrhea                             | 32 (26.0)               | 13 (18.3)           | .64 (0.31, 1.32)    | .22   |
| Abdominal pain                       | 11 (8.9)                | 2 (2.9)             | .30 (0.06, 1.39)    | .11   |
| Altered mental status                | 18 (14.6)               | 21 (28.4)           | 2.31 (1.14, 4.71)   | .02   |
| Vital signs on admission              |                         |                     |                     |       |
| Supplemental oxygen required on hospitalization | 33 (26.8) | 44 (59.5)     | 4.0 (2.17, 7.38)    | <.0001 |
| Vital signs within first 24 hours    |                         |                     |                     |       |
| Temperature                          | 74 (60.2)               | 52 (70.3)           | 1.57 (1.85, 2.90)   | .15   |
| Abnormal chest X-ray on admission    | 84 (68.9)               | 68 (90.4)           | 4.27 (1.79, 10.16)  | .001  |
| Laboratory findings on admission     |                         |                     |                     |       |
| Leukopenia                           | 14 (11.4)               | 7 (9.5)             | .81 (0.31, 2.12)    | .67   |
protease-activated receptors. Microvascular permeability from endothelial injury can also facilitate viral invasion [31]. Thus, the direct effect of viral invasion and indirect effects through endothelial damage lead to severe hypoxia.

CRP level upon admission was also associated with severe infection. Every 1-unit increase in CRP increased the risk of severe disease by 0.06%. CRP is a homopentameric acute-phase inflammatory protein. Baseline CRP values are influenced by age, gender, smoking status, weight, lipid levels, and blood pressure, and by genetics [32]. Recent studies have reported that cases of severe COVID-19 exhibit increased plasma levels of interleukin (IL) 2, IL-6, IL-7, IL-10, granulocyte colony-stimulating factor (GCSF), tumor necrosis factor α (TNF-α), and others [33]. IL-6 is the main inducer of CRP gene expression, with IL-1 and TNF-α also playing a role [32]. Elevated CRP may reflect severe disease as an indirect marker of elevated IL-6 and TNF-α. CRP not only reflects inflammation, it also enhances the immune response. CRP can be irreversibly dissociated into monomeric subunits termed monomeric or modified CRP (mCRP) at either high concentrations of urea or elevated temperatures in the absence of calcium, and mCRP promotes monocyte chemotaxis and recruitment of circulating leukocytes to areas of inflammation. Modified CRP also binds immunoglobulin G (IgG) Fc receptors in an interaction leading to release of proinflammatory cytokines [32]. Thus, elevated CRP at admission may both reflect significant inflammation and itself drive further inflammation. And given that elevated levels of urea promote formation of mCRP, it fits that acute and chronic kidney disease may be associated with adverse outcomes.

In our data, having contact with a sick person was associated with a lower risk of severe disease. Perhaps some of those patients with contact with a sick person knew they were at risk of exposure and therefore were already attempting to minimize that risk through hand hygiene, masks, physical isolation, or using separate bathrooms, thus decreasing the potential amount of virus to which they were exposed. This would, however, only account for patients where someone else was symptomatic or exposed soon enough for the patient to be able to take precautions. Patients with known contact with a sick person may also have presented sooner due to a heightened suspicion of having contracted COVID-19, and thus received care more rapidly [34]. Recall bias may also have contributed. One other potential explanation is that the sickest patients were more confused or were intubated rapidly and therefore could not provide a history of contact with someone who was sick, which would have impacted the recording of a such contact in the EMR and thus our results.

Patients over 60 years old were significantly more likely to have severe infection. In multivariable analysis, however, age was not found to be an independent predicting factor for severe infection. This may reflect that the occurrence of kidney disease tends to be higher in older people and kidney disease was the stronger predictor in the model. Older age has been significantly associated with death in previous studies [35, 36]. This might be related to less robust immune responses, as older age has been linked with declined immune competence [37]. On the contrary, older macaques had a stronger host innate immune responses to viral infection after inoculation with SARS-CoV than the younger macaques due to differential expression of proinflammatory genes [38]. This proinflammatory state along with age-dependent functional deficits in T-cell and B-cell response could lead to poor outcomes [39]. This proinflammatory state is expressed by elevation in CRP. Elderly infected patients could be at risk of acute renal injury manifesting in elevation in serum creatinine on hospital admission. A study by Wang et al from China showed that patients admitted to the ICU were older and had a greater number of comorbid conditions,

### Table 1. Multivariate Logistic Regression Analysis of Predictors for Severe COVID-19 Infection

| Variables                        | OR (95% CI)        | P     |
|----------------------------------|--------------------|-------|
| Pre-existing renal disease       | 7.4 (2.5, 22.0)    | <.0001|
| Oxygen required on admission     | 2.9 (1.3, 6.7)     | .01   |
| Elevated creatinine from baseline| 2.7 (1.3, 5.6)     | .01   |
| Initial CRP value                | 1.008 (1.001, 1.01)| .02   |

Data are presented as n (%) unless otherwise indicated. Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; CI, confidence interval; COVID-19, coronavirus disease 2019; CWIC, Charlson Weighted Index of Comorbidity; OR, odds ratio.
including a significant elevation in creatinine on admission, than those not admitted to the ICU [13]. Thus, age in our studied cohort might have been a confounding factor with regard to elevated serum creatinine and CRP.

During the COVID-19 pandemic, racial and ethnic minorities, especially blacks, have been reported to be severely or disproportionately impacted. Our study did not show a disparity in severity by race, so we also investigated the relationship by ADI (as a proxy for socioeconomic status). No association was found between ADI and severity or between race and severity after controlling for ADI.

Our study has several limitations. This was a single-institution study among all the admitted patients, which makes generalizability of the findings difficult. Because of the retrospective nature of the study design, all variables in the studied patients were not available. Therefore, the role of some of these variables in predicting severity of the infection could have been underestimated. Last, the small sample size of our study and a predominantly black and overweight/obese cohort could have limited the generalizability of interpretation for some of the findings (for, eg, race). Nonetheless, our study did involve a population of black patients in the Detroit area and can provide valuable information on which factors are the most significant predictors of severe disease in that population.

CONCLUSIONS

Our study identifies the presence of renal disease and elevated creatinine and CRP on hospital admission and requirement for supplemental oxygen at the time of hospitalization as independent predictors for severe COVID-19 infection. Early identification of these risk factors can result in aggressive supportive care and prompt treatment intervention. Subsequent research involving multiple study sites and with a larger database can further validate the findings of our study and help in early clinical decision making.

Note

Potential conflicts of interest. The authors: No reported conflicts of interest. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest.

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