Original Research

Clinical Characteristics and Laboratory Biomarkers for Patients with Suspected COVID-19 Infection Within HCA Healthcare

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Abstract

Background
The coronavirus infection (COVID-19), also known as the Severe Acute Respiratory Syndrome Virus 2 (SARS-CoV-2), caused significant illness and a worldwide pandemic beginning in 2020. Early case reports showed common patient characteristics, clinical variables and laboratory values in these patients. We compared a large population of American COVID-19 patients to see if they had similar findings to these smaller reports. In addition, we examined our population to identify any differences between mild or severe COVID-19 infections.

Methods
We retrospectively accessed a de-identified, multi-hospital database managed by HCA Healthcare to identify all adult emergency department (ED) patients that were tested for COVID-19 from January 1st, 2020–April 30th, 2020. We collected clinical variables, comorbidities and laboratory values to identify any differences in those with or without a SARS-CoV-2 infection.

Results
We identified 44,807 patients who were tested for SARS-CoV-2. Of those patients, 6,158 were positive for COVID-19. Male patients were more likely to test positive than female ones (15.0% vs. 12.6%, $p < 0.001$). The most frequently positive tests occurred in age groups 40–49, 50–59 and 60–69 (16.9%, 15.3% and 14.1% respectively). Both African Americans (20.2%) and Hispanics (20.8%) were more likely to test positive than Caucasians (8.3%, $p < 0.001$). Hypertension and diabetes were more common in those with positive tests, and multiple laboratory biomarkers showed significant differences in severe infections.

Conclusions
This broad cohort of American COVID-19 patients showed similar trends in gender, age groups and race/ethnicity as previously reported. Severe COVID-19 disease was also associated with many positive laboratory biomarkers.

Keywords
COVID-19; SARS-CoV-2; coronavirus infections/diagnosis; coronavirus infections/complications; biomarkers/blood; clinical characteristics; pandemics; retrospective studies

Introduction
In late December 2019, a novel coronavirus named the Severe Acute Respiratory Virus 2 (SARS-CoV-2) began circulating within humans in the Wuhan province of China.[1] Over the next couple of months, this coronavirus (COVID-19) spread through China and then began to spread internationally. The disease initially spread to Iran and Italy, and then it made its way to the United States. The first hotspots in the United States occurred in February and March of 2020 on the West Coast followed rapidly by the New York City tri-state area.[1–4] In April 2020, SARS-CoV-2 began to spread uncontrollably around the country as well as the rest of the world.

Rapid diagnosis of COVID-19 patients has also been problematic. The virus was novel, and no tests existed before 2020 for this specific pathogen. The World Health Organization was able to produce the first diagnostic test for COVID-19[5] and the Centers for Disease Control (CDC) developed its own diagnostic test for the United States. However, the turnaround time for testing was slow, often taking many days to get a result. This delay left a void in health care, and caused emergency department (ED) providers to be unable to identify patients with COVID-19 early in the disease process. In order to identify as many cases as possible, anyone who suspected they were ill from traveling or were knowingly exposed to the disease were required to quarantine at home, or they were admitted to the hospital for treatment until their test results came back.

Clinical findings of the novel coronavirus infection were first reported by Huang et al. in January 2020, a case series of 41 infected patients from the Wuhan province in China.[1] More cases were subsequently reported from China[6,7] and Italy[8] identifying clinical characteristics of patients with COVID-19. Then a case series from New York reported presenting characteristics, comorbidities and outcomes of hospitalized patients.[3] Common clinical findings included fever, hypoxia and dyspnea. Many patients had abnormal chest x-rays with bilateral ground glass infiltrates, and their white blood cell count showed lymphopenia. Other laboratory findings in China that were abnormal included an elevated D-dimer, lactate dehydrogenase, troponin-I and procalcitonin.[1] Therefore, many clinicians in America began testing patients suspected of having COVID-19 for signs of inflammation or other biomarkers for infectious diseases (i.e., lactic acid or C-reactive protein) while waiting for official COVID-19 test results.

HCA Healthcare owns 184 hospitals in the United States and the United Kingdom. They maintain a central registry of all their patients, which gives us a unique opportunity to investigate data on a large cohort of SARS-CoV-2 patients. We accessed this database to confirm previously reported trends in SARS-CoV-2 patients, clinical variables and laboratory biomarkers so as to better assist health care providers with early identification of COVID-19 patients. Populations across the world are different, and it would be interesting to see if Americans with COVID-19 follow the same clinical characteristics and biomarker patterns that have been seen in other parts of the world as well as if any new biomarker(s) could reliably predict severe COVID-19 disease or complications.

Methods

We retrospectively accessed the central HCA Healthcare database containing billing and medical record data from 162 EDs within their multihospital system in the United States. HCA Healthcare owns facilities in 18 states and all of them were included. The HCA Healthcare institutional review board deemed this study exempt from oversight. Data was abstracted out of the database from January 1st, 2020 through April 30th, 2020 for all adult patients (18+) who were tested for SARS-CoV-2. Safe Harbor de-identification techniques were utilized so that no protected health information was taken out of the central database for our analysis. However, we were able to collect demographic data, including the subjects' age (years; if age > 89 we had to list them as 89 according to Safe Harbor de-identification techniques), gender and race or ethnicity. Clinical variables collected included first day vital signs, ED status (admission or discharge home), type of inpatient ward (floor, step down unit or intensive care unit [ICU]), final diagnoses (via ICD-10 codes) and final status (discharged from ED, discharged from hospital, deceased or still admitted at time of the data pull). Laboratory biomarkers collected included routine tests (complete blood count with differential counts, chemistry analysis, lactic acid, troponin-I and pregnancy test), coagulation tests (activated partial thromboplastin time [PTT], prothrombin time [PT], international normalized ratio [INR], D-dimer and fibrinogen) and markers of inflammation (erythrocyte sedimentation rate [ESR], C-reactive protein [CRP], procalcitonin, interleukin-6, ferritin and lactic acid dehydrogenase [LDH]). Results from the SARS-CoV-2 test were also collected. Normal values for all labs were compared to ensure equivalence between locations. For the D-dimer test, if results were reported in D-dimer units (DDUs), they were multiplied by two to get equivalent fibrinogen equivalent units (FEUs) for purposes of combined analysis.

The subjects were divided into groups for

analysis. First we compared subjects that tested negative for SARS-CoV-2 (COVID-) versus ones that tested positive (COVID+) by looking at clinical characteristics (vital signs) and past medical history. Then we subdivided the COVID+ group into those with non-severe infections (either discharged home from the ED or admitted to the hospital) and those admitted with severe infections. Infections were considered severe if the subject at any time during their hospitalization was in the ICU or had final diagnosis codes for severe sepsis, septic shock, systemic inflammatory response syndrome (SIRS) with organ dysfunction, adult respiratory distress syndrome or acute respiratory failure. SIRS without organ dysfunction was considered a non-severe infection.

Statistical analysis was performed using Sas 9.4, and tables were created in Excel. Descriptive statistics were used to evaluate patient demographics and comorbidities, including chi-squared analysis. Variance analysis and student t-test were used to evaluate significance in clinical characteristics and laboratory values. An alpha level of 0.05 was accepted as the level of statistical significance for all comparisons.

Results

Table 1 identifies the baseline characteristics of all patients who were tested for SARS-CoV-2 between January 1st, 2020 and April 30th,

Table 1. Baseline Characteristics of Patients Tested for SARS-CoV-2, No. (%)

	Total (n=44,807)	COVID - (n=38,649)	COVID + (n=6158)	P value
Gender				
Female	23680 (52.8)	20691 (53.5)	2989 (48.5)	
Male	21127 (47.2)	17958 (46.5)	3169 (51.5)	p<0.001
Ages				
18–29		4168 (89.7)	480 (10.3)	
30–39		4728 (87.2)	695 (12.8)	
40–49		4627 (83.1)	944 (16.9)	*
50–59		6324 (84.7)	1145 (15.3)	†
60–69		7325 (85.9)	1205 (14.1)	
70–79		6719 (87.2)	986 (12.8)	
≥80		4758 (87.1)	703 (12.9)	
Race				
White	29317 (65.4)	26527 (68.6)	2790 (45.3)	‡
African American	8417 (18.8)	6732 (17.4)	1685 (27.4)	‡
Other	6142 (13.7)	4666 (12.1)	1476 (24.0)	‡
Asian	932 (2.1)	724 (1.9)	207 (3.4)	
Ethnicity				
Hispanic	8000 (17.9)	6338 (16.4)	1662 (27.0)	p<0.001
Non-Hispanic	36807 (82.1)	32311 (83.6)	4496 (73.0)	
Comorbidities				
Hypertension	17900 (39.9)	15205 (39.3)	2695 (43.8)	p<0.001
Cardiovascular Disease	17164 (38.3)	15375 (39.8)	1789 (29.1)	p<0.001
Hypercholesterolemia	14938 (33.3)	13024 (33.7)	1914 (31.1)	p<0.001
Diabetes	13150 (29.3)	11152 (28.9)	1998 (32.4)	p<0.001
COPD	13750 (30.7)	12579 (32.5)	1171 (19.0)	p<0.001
Malignancy	5282 (11.8)	4826 (12.5)	456 (7.4)	p<0.001
Chronic Kidney Disease	7675 (17.1)	6747 (17.4)	928 (15.1)	p<0.001
Chronic Liver Disease	2194 (4.9)	2010 (5.2)	184 (3.0)	p<0.001

* p<0.001 between this age group and all others except 50–59

† p<0.001 between this age group and all others except 40–49 or 60–69

‡ p<0.001 between White, African American, and other races

2020 in the HCA Healthcare hospital system. We found 44,807 tests for SARS-CoV-2 were performed, with 6,158 (13.7%) tests returning positive results (COVID+). Overall, fewer male patients were tested than females (21,127 vs. 23,680), yet the prevalence of COVID+ was higher in males than females (15.0% vs. 12.6%, p < 0.001). In the COVID+ cohort, the most prevalent age groups were the 40–49, 50–59, and 60–69 age groups. Comparing individual age groups, the 40–49 age group was statistically more likely to test positive than all other age groups except for the 50–59 age group (all with p < 0.001). The 50–59 age group was statistically more likely to test positive than all other age groups except for the 40–49 and 60–69 age groups (all with p < 0.001). Lastly, the 60–69 age group was statistically more likely to test positive than the 18–29 age group (p < 0.001). Comparing races, the majority of tests were performed on Caucasian patients (65.4%), yet the positivity of COVID+ was higher in African American, Asian and other racial groups (p < 0.001). Similarly comparing ethnicities, the majority of tests were performed on non-Hispanic patients (82.1%), yet the prevalence of COVID+ was higher in the Hispanic ethnicity group (p < 0.001).

When we studied the presence of comorbidities in patients tested for SARS-CoV-2, we noticed some differences as well. Patients that tested positive for COVID-19 were more likely to have hypertension (43.8% vs. 39.3%, p < 0.001) and diabetes mellitus (32.4% vs. 28.9%, p < 0.001) versus those who tested negative. However, patients that tested positive for COVID-19 were less likely to have cardiovascular disease (29.1% vs. 39.8%, p < 0.001), hypercholesterolemia (31.1% vs. 33.7%, p < 0.001), chronic obstructive pulmonary disease (COPD) (19.0% vs. 32.5%, p < 0.001), malignancy (7.4% vs. 12.5%, p < 0.001), chronic kidney disease (15.1% vs. 17.4%, p < 0.001) or chronic liver disease (3.0% vs. 5.2%, p < 0.001) versus those who tested negative.

Clinical variables for patients with COVID-19 are shown in **Table 2**. Many patients with SARS-CoV-2 had specific abnormalities in their

Table 2. Vital signs and clinical characteristics of ED patients tested for SARS-CoV-2

	Total Tested (n=44,807)	COVID -	COVID +	t-test P value	Chi-squared P value
Heart Rate (n=44,358), median beats per min (IQR)		97 (83–112)	98 (86–110)	p=0.64	
Systolic Blood Pressure (n=44,287), median mmHg (IQR)		145 (130–165)	142 (129–157)	p<0.001	
Diastolic Blood Pressure (n=44,287), median mmHg (IQR)		84 (75–92)	82 (75–90)	p<0.001	
Oxygen saturation (n=43,975), median % (IQR)		95 (92–97)	94 (90–96)	p<0.001	
Respiratory Rate (n=44,146), median breaths per min (IQR)		19 (18–23)	20 (18–24)	p<0.001	
Temperature (n=44,113), median oC (IQR)		37.0 (36.8–37.4)	37.5 (37.0–38.4)	p<0.001	
Admitted to ICU, No. (%)	9805 (21.9)	8144 (21.1)	1661 (27.0)		p<0.001
SIRS without organ dysfunction, No. (%)	11239 (25.1)	9153 (23.7)	2086 (33.9)		p<0.001
SIRS with organ dysfunction, severe sepsis or septic shock, No. (%)	5207 (11.6)	4226 (10.9)	981 (15.9)		p<0.001
ARF, No. (%)	12274 (27.4)	9712 (25.1)	2562 (41.6)		p<0.001
ARDS, No. (%)	417 (0.9)	128 (0.3)	289 (4.7)		p<0.001

n = number of subjects with that recorded vital sign (some data was missing)

Abbreviations: Interquartile Range (IQR), Celsius (C), Intensive Care Unit (ICU), Systemic Inflammatory Response Syndrome (SIRS), Acute Renal Failure (ARF), Acute Respiratory Distress Syndrome (ARDS)

ED vital signs. Those who tested positive for SARS-CoV-2 were statistically more likely to have hypoxia (SpO_2 < 93% on room air) than those who tested negative (42.1% vs. 28.2%, p < 0.001), a temperature greater than 38° C (34.4% vs. 13.0%, p < 0.001) or a respiratory rate greater than 16 breaths/min (92.2% vs. 90.4%, p < 0.001). Our analysis did not find any difference for heart rate greater than 100 bpm or systolic blood pressure greater than 120 mmHg between groups.

We found that 9,805 (21.9%) of all patients tested for COVID-19 were admitted to an ICU setting. Those who tested positive for SARS-CoV-2 were more likely to be admitted to the ICU than those who tested negative (27.0% vs. 21.1%, p < 0.001). COVID+ patients were also more likely than COVID- patients to have SIRS without organ dysfunction or any type of severe infection, which we defined as SIRS with organ dysfunction, severe sepsis, septic shock, acute renal failure or acute respiratory distress syndrome (all with p < 0.001).

Lastly, we compared laboratory biomarkers in COVID+ patients with severe versus non-severe infections. These results can be seen in **Table 3**. Many laboratory tests showed significant differences, including statistically higher values of total white blood cell count (WBC), neutrophil count, blood urea nitrogen (BUN), glucose, lactic acid, aspartate aminotransferase (AST), LDH and CRP for those with severe COVID-19 infections (all with p < 0.005). D-dimer was also significantly higher in those with severe COVID-19 (p = 0.023).

Discussion

HCA Healthcare maintains a large electronic database that is prime for researching large groups of patients. To our knowledge, this study represents the first multi-state or multi-regional cohort of sequentially identified ED patients with COVID-19 in the United States. The database confirmed many baseline trends and clinical characteristics of COVID-19 patients previously reported in isolated series in China,[1,6,7] Italy,[8] New York City[3] and Washington state.[2] The geographic area of our cohort consisted of 18 states. However, more than 50% of the patients resided in Florida or Texas. In addition, most of the prior publications evaluated populations of COVID+ patients without comparing them to concurrent COVID- patients who also presented to the ED. The database allowed us to compare all ED patients that were tested for SARS-CoV-2. We also had the ability to compare severe and non-severe COVID-19 disease to find some significant differences.

The COVID+ patients in our cohort were more likely to be older (40–69 yrs) and male. These factors may be attributable to the severity of disease in older patients, as younger patients with COVID have milder disease and may not presents as often to the ED for treatment. Similar trends were found in prior studies from China, the United States and Italy.[3,8–11] These prior studies also identified hypertension and diabetes mellitus as common co-existing medical conditions present in patients with COVID-19. Our cohort confirmed this association with hypertension and diabetes mellitus to testing positive for SARS-CoV-2. However, our cohort also showed that patients with a history of cardiovascular disease, hypercholesterolemia, COPD, malignancy, chronic kidney disease or chronic liver disease were more likely to test negative for SARS-CoV-2. Since COVID-19 is primarily a disease of the respiratory tract, it is interesting that patients with COPD were more likely to test negative for SARS-CoV-2. One hypothesis to explain this outcome is that COPD patients are frequently on inhaled steroids.

The database also confirmed the previous reports that COVID-19 illness is more likely to occur in minority ethnic demographic groups versus Caucasians.[11–13] This outcome occurred despite the majority of tests being performed in our cohort on Caucasian patients (65.4%). We cannot make any assumptions as to why minority ethnic groups are at higher risk of infection as there are too many unaccounted cofactors needed to better evaluate disadvantaged socioeconomic groups.

Our study was able to compare clinical characteristics of all ED patients tested for SARS-CoV-2. We found that COVID+ patients were more likely to have a fever (temperature > 38° C), hypoxia (SpO_2 < 93% on room air) and tachypnea (respiratory rate > 16) than COVID- patients. This characteristics presented in

Table 3. Laboratory biomarkers of COVID+ patients on admission to hospital, median (IQR)

	Normal Range	Non-Severe Covid Infections	Severe Covid Infections	P value
White blood cell count (n=826), x109/L	3.6–11.0	5.7 (4.5–7.7)	7.53 (5.5–9.7)	p<0.001
Neutrophil count (n=625), x109/L	1.6–8.2	4.2 (3.2–5.7)	4.9 (3.5–7.5)	p<0.001
Lymphocyte count (n=694), x109/L	1.1–4.7	1.13 (0.85–1.6)	0.94 (0.6–1.3)	p=0.259
Platelet count (n=885), x109/L	150–400	204 (161.5–255)	203 (160–266)	p=0.106
Hemoglobin (n=919), g/dL	12.0–16.0	13.2 (12.0–14.5)	13.4 (11.9–14.7)	p=0.784
Activated partial thromboplastin time (n=284), s	25.1–36.5	29.8 (27.7–32.9)	30.2 (27.5–34.0)	p=0.055
Prothrombin time (n=383), s	9.4–12.5	12.2 (10.9–13.8)	12.4 (11.0–13.8)	p=0.875
International Normalized Ratio (n=182), U	1–1.4	1.1 (1.0–1.2)	1.1 (1.0–1.3)	p=0.507
D-dimer (n=163), mg/L FEU	<500	820 (504–1,400)	1,248 (620–2,640)	p=0.023
Sodium (n=900), mmol/L	136–145	137 (134–139)	136 (133–139)	p=0.478
Potassium (n=927), mmol/L	3.5–5.1	3.8 (3.6–4.1)	3.9 (3.5–4.3)	p=0.151
Chloride (n=846), mmol/L	98–107	103 (100–106)	103 (99–107)	p=0.334
Bicarbonate (n=1057), mmol/L	21–32	25 (23–27)	24 (22–27)	p=0.008
Blood urea nitrogen (n=880), mg/dL	7–18	13 (9–19)	18 (12–29)	p<0.001
Creatinine (n=901), mg/dL	0.6–1.3	0.98 (0.77–1.21)	1.10 (0.81–1.59)	p=0.088
Blood Urea Nitrogen/Creatinine (n=176), ratio	9.3–24.4	15 (10.8–20.8)	16 (12.4–20)	p=0.981
Glomerular filtration rate (n=672), mL/min	>60	60 (60–60)	60 (50–60)	p<0.001
Glucose (n=1027), mg/dL	70–110	117 (101–161)	125 (104–182)	p=0.002
Lactic acid (n=705), mmol/L	0.4–2.0	1.2 (0.9–1.6)	1.6 (1.2–2.2)	p<0.001
Troponin-I (n=668), ng/dL	<0.034	0.015 (0.012–0.020)	0.02 (0.015–0.065)	p=0.163
Aspartate aminotransferase (n=689), U/L	15–37	35 (26–50)	45 (28–69.5)	p<0.001
Alanine aminotransferase (n=730), U/L	10–60	35 (24–54)	36 (23–61)	p=0.312
Lactic acid dehydrogenase (n=237), U/L	84–246	260 (204–310)	334 (251–473)	p<0.001
C-reactive protein (n=261), mg/L	<1.0	5.10 (2.23–9.16)	9.28 (5.00–16.05)	p=0.004
Erythrocyte Sedimentation Rate (n=43), mm/hr	<20	33 (4–59)	49 (33–72)	p=0.166
Procalcitonin (n=189), ng/mL	<0.50	0.08 (0.05–0.24)	0.14 (0.05–0.42)	p=0.257
Ferritin (n=203), ng/mL	8–388	373 (195–718)	623 (208–1283)	p=0.173
Fibrinogen (n=21), mg/dL	200–393	370 (358–424)	523 (439–604)	p=0.269
Interleukin-6 (n=14), pg/mL	<15.5	91 (20–163)	79 (41-261)	p=0.604

n = number of COVID+ subjects with that laboratory test, first value if multiple

Abbreviations: Interquartile Range (IQR), liters (L), seconds (s), grams (g), milligrams (mg), units (U), fibrinogen equivalent units (FEU), millimoles (mmol), deciliters (dL), minutes (min), nanograms (ng), millimeters (mm), hours (hr), picograms (pg)

similar research from the United Kingdom that showed COVID+ patients had tachypnea and required increasing amounts of supplemental oxygen.[14] Our analysis did not find any difference for heart rate greater than 100 bmp or systolic blood pressure greater than 120 mmHg between groups. In our cohort, COVID+ patients were also more likely to have SIRS without organ dysfunction or any type of severe infection.

Lastly, our results add to the growing literature that inflammatory markers are elevated in those with severe illness and indicated a risk

of mortality.[15–21] In our study, higher levels of total WBC, neutrophil count, BUN, glucose, lactic acid, AST, LDH, D-dimer and CRP were associated with severe illness. Of these, LDH and CRP showed the strongest correlation to severe illness ($p < 0.001$), and D-dimer showed the third strongest correlation to severe illness ($p = 0.023$). However, our research suggests that other inflammatory markers may be less associated with severe COVID-19 infections as their differences were not statistically significant. This finding is in contrast to other studies that showed elevated levels of ESR, procalcitonin, ferritin, fibrinogen and interleukin-6 may be associated with severe infections.[7,9,15–22]

Limitations

Our study has several limitations. First, it is retrospective by design, which limits its strength, and we cannot make any causative conclusions. Second, the time period from which we obtained our data was early in the COVID-19 pandemic. Since then the infective pandemic grew in size and went through two more peaks in the United States and other countries. Even though the database from which we abstracted our results has a large collection of hospitals and EDs (162) in 18 states, most are located in Southern or Southeastern states. The two states with the most hospitals represented in our cohort were Florida and Texas, which together account for 56% of the American inpatient hospital beds in the HCA Healthcare system. Even though we accessed a large database, it is not reflective of a nationwide sampling. Yet, our results were similar to other international reports and the New York City area. Lastly, the database was limited by many specific patient details. For instance, we did not have access to historical details in provider notes to ascertain how many days of illness occurred prior to ED presentation, nor do we know the reason for SARS-CoV-2 testing. We can, however, assume the majority were done for diagnostic purposes. We also do not know the method of SARS-CoV-2 testing at each site, but, based on the dates of our study, we can be fairly certain they were all done with reverse transcriptase polymerase chain reaction methods as other methods (i.e., rapid antigen testing) were not developed yet. We may have also encountered some variability in laboratory measurements between hospitals that use different analyzers for the same test. For instance, D-dimer can be measured in multiple ways. Combining the data to give a single value may have introduced some error into our results. Finally, it is possible that a single patient may have been included in our database more than once if they had separate hospital encounters/admission and had a SARS-CoV-2 test performed during each encounter.

Conclusion

This large multi-state database of EDs in the United States confirmed common baseline characteristics, clinical variables and laboratory biomarkers of patients with SARS-CoV-2 found internationally. Males, non-Caucasian minority ethnic groups, patients aged 40–69 and those with a history of hypertension or diabetes were most associated with testing positive for SARS-CoV-2. Confirmed cases were more likely to be febrile, tachypneic and/or hypoxic. Lastly, those with severe COVID-19 disease were more likely to have elevated levels of many biomarkers with LDH and CRP showing the strongest correlation.

Conflicts of Interest

The authors declare they have no conflicts of interest.

Drs. Gutovitz, Hanson and Jehle are employees of Grand Strand Regional Medical Center, a hospital affiliated with the journal's publisher.

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