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ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Domenico De Berardis, MD, PhD, Adjunct Professor, Chief Doctor, NHS, Department of Mental Health, Teramo 64100, Italy. domenico.deberardis@aslteramo.it

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ORIGINAL ARTICLE

Retrospective Study

COVID-19 among African Americans and Hispanics: Does gastrointestinal symptoms impact the outcome?

Hassan Ashktorab, Adeleye Folake, Antonio Pizuorno, Gholamreza Oskrochi, Philip Oppong-Twene, Nuri Tamanna, Maryam Mehdipour Dalivand, Lisa N Umeh, Esther S Moon, Abdoul Madjid Kone, Abigail Banson, Cassandra Federman, Edward Ramos, Eyitope Ola Awoyemi, Boubini Jones Wonni, Eric Otto, Guttu Maskalo, Alexandra Ogando Velez, Sheldon Rankine, Camelita Thrift, Chiamaka Ekwunazu, Derek Scholes, Lakshmi Gayathri Chirumamilla, Mohd Elmugtaba Ibrahim, Brianna Mitchell, Jillian Ross, Julencia Curtis, Rachel Kim, Chandler Gilliard, Joseph Mathew, Adeyinka Laiyemo, Angesum Kibreab, Edward Lee, Zaki Sherif, Babak Shokrani, Farshad Aduli, Hassan Brim

ORCID number: Hassan Ashktorab 0000-0002-4048-4666; Adeleye Folake 0000-0003-3047-0436; Antonio Pizuorno 0000-0002-4118-6310; Gholamreza Oskrochi 0000-0003-1905-4490; Philip Oppong-Twene 0000-0001-5517-0937; Nuri Tamanna 0000-0001-5347-1250; Maryam Mehdipour Dalivand 0000-0003-2671-6325; Lisa N Umeh 0000-0002-8323-3541; Esther S Moon 0000-0002-2896-6099; Abdoul Madjid Kone 0000-0002-3160-3192; Abigail Banson 0000-0002-5448-4632; Cassandra Federman 0000-0002-3545-7719; Edward Ramos 0000-0002-7420-9638; Eyitope Ola Awoyemi 0000-0001-5958-3261; Boubini Jones Wonni 0000-0002-0755-4538; Eric Otto 0000-0003-4397-3470; Guttu Maskalo 0000-0002-3287-1543; Alexandra Ogando Velez 0000-0002-0321-7323; Sheldon Rankine 0000-0001-5634-2956; Camelita Thrift 0000-0003-4308-565X; Chiamaka Ekwunazu 0000-0001-8677-3001; Derek Scholes 0000-0002-9176-684X; Lakshmi Gayathri Chirumamilla 0000-0003-3414-7804; Mohd Elmugtaba Ibrahim 0000-0003-0024-8543; Brianna Mitchell 0000-0001-8305-4212; Jillian Ross 0000-0002-2880-732X; Julencia Curtis

Hassan Ashktorab, Adeleye Folake, Philip Oppong-Twene, Nuri Tamanna, Maryam Mehdipour Dalivand, Lisa N Umeh, Esther S Moon, Abdoul Madjid Kone, Abigail Banson, Cassandra Federman, Edward Ramos, Eyitope Ola Awoyemi, Boubini Jones Wonni, Eric Otto, Guttu Maskalo, Alexandra Ogando Velez, Sheldon Rankine, Camelita Thrift, Chiamaka Ekwunazu, Derek Scholes, Lakshmi Gayathri Chirumamilla, Mohd Elmugtaba Ibrahim, Brianna Mitchell, Jillian Ross, Julencia Curtis, Rachel Kim, Chandler Gilliard, Joseph Mathew, Adeyinka Laiyemo, Angesum Kibreab, Edward Lee, Zaki Sherif, Babak Shokrani, Farshad Aduli, Department of Medicine, Gastroenterology Division and Cancer Center, Howard University College of Medicine, Washington, DC 20060, United States

Antonio Pizuorno, Faculty of Medicine, La Universidad del Zulia, Maracaibo 4002, Zulia, Venezuela

Gholamreza Oskrochi, College of Engineering and Technology, American University of Middle East Kuwait, Egaila 54200, Kuwait

Zaki Sherif, Department of Biochemistry and Molecular Biology, Howard University College of Medicine, Washington, DC 20060, United States

Hassan Brim, Pathology and Cancer Center, Department of Biochemistry and Molecular Biology, Howard University College of Medicine, Washington, DC 20060, United States

Corresponding author: Hassan Ashktorab, PhD, Professor, Department of Medicine, Gastroenterology Division and Cancer Center, Howard University College of Medicine, 2041 Georgia Avenue, N.W., Washington, DC 20060, United States. hashktorab@howard.edu

Abstract

BACKGROUND

The coronavirus disease 2019 (COVID-19) disproportionately affected African Americans (AA) and Hispanics (HSP).

0000-0002-9159-3942; Rachel Kim 0000-0002-8994-968X; Chandler Gilliard 0000-0002-0438-8837; Joseph Mathew 0000-0002-8646-9998; Adeyinka Laiyemo 0000-0001-9699-4879; Angesum Kibreab 0000-0002-4850-9071; Edward Lee 0000-0002-6717-3112; Zaki Sherif 0000-0002-1772-7041; Babak Shokrani 0000-0003-2201-6188; Farshad Aduli 0000-0002-9145-6358; Hassan Brim 0000-0001-7055-2298.

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AIM

To analyze the significant effectors of outcome in African American patient population and make special emphasis on gastrointestinal (GI) symptoms, laboratory values and comorbidities

METHODS

We retrospectively evaluated the medical records of 386 COVID-19 positive patients admitted at Howard University Hospital between March and May 2020. We assessed the symptoms, including the GI manifestations, comorbidities, and mortality, using logistic regression analysis.

RESULTS

Of these 386 COVID-19 positive patients, 257 (63.7%) were AAs, 102 (25.3%) HSP, and 26 (6.45%) Whites. There were 257 (63.7%) AA, 102 (25.3%) HSP, 26 (6.45%) Whites. The mean age was 55.6 years (SD = 18.5). However, the mean age of HSP was the lowest (43.7 years *vs* 61.2 for Whites *vs* 60 for AAs). The mortality rate was highest among the AAs (20.6%) and lowest among HSP (6.9%). Patients with shortness of breath (SOB) (OR2 = 3.64, CI = 1.73-7.65) and elevated AST (OR2 = 8.01, CI = 3.79-16.9) elevated Procalcitonin (OR2 = 8.27, CI = 3.95-17.3), AST (OR2 = 8.01, CI = 3.79-16.9), ferritin (OR2 = 2.69, CI = 1.24-5.82), and Lymphopenia (OR2 = 2.77, CI = 1.41-5.45) had a high mortality rate. Cough and fever were common but unrelated to the outcome. Hypertension and diabetes mellitus were the most common comorbidities. Glucocorticoid treatment was associated with higher mortality (OR2 = 5.40, CI = 2.72-10.7). Diarrhea was prevalent (18.8%), and GI symptoms did not affect the outcome.

CONCLUSION

African Americans in our study had the highest mortality as they consisted of an older population and comorbidities. Age is the most important factor along with SOB in determining the mortality rate. Overall, elevated liver enzymes, ferritin, procalcitonin and C-reactive protein were associated with poor prognosis. GI symptoms did not affect the outcome. Glucocorticoids should be used judiciously, considering the poor outcomes associated with it. Attention should also be paid to monitor liver function during COVID-19, especially in AA and HSP patients with higher disease severity

Key Words: COVID-19; Pandemic, Gastrointestinal manifestation; Liver; African Americans; Hispanics

Core Tip: The Coronavirus disease 2019 (COVID-19) disproportionately affected African Americans and Hispanics. Understanding the transmission dynamics, a different array of symptoms and the impact of the presence of other chronic diseases in minority patients can provide important hints about the progression of the pandemic and treatment options, especially in areas where access to equal health services is limited, unequal and challenging for underserved populations is broad. This study presents the findings of a comprehensive analysis of COVID-19 patients in a Washington DC tertiary hospital that caters primarily to minority populations. The main objective of this study was to define major effectors of outcome in this patient population. We sought to determine clinical and gastrointestinal (GI) factors associated with differences in outcomes. Special emphasis was made on GI symptoms, laboratory values and comorbidities.

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INTRODUCTION

A new cluster of pneumonia cases in China surfaced in December of 2019. On January 7, 2020, the agent causing this pneumonia was identified and named severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) for SARS-CoV-2 instead of SARS-CoV-1 that prevailed in 2003[1]. The disease caused by this new virus, coronavirus disease (COVID-19), was recognized by the World Health Organization as a pandemic on March 11, 2020. Since its appearance, the virus has infected more than 70 million people and caused more than 1.5 million deaths worldwide, as of December 20, 2020 [2]. The pandemic has affected over 150 countries and significantly impacted all racial and socioeconomic segments of the population. COVID-19 affected more than 22 million people within the United States and caused the death of more than 373 thousand deaths as of January 10, 2021, by far one of the most affected areas in the world. The most affected states are California, Texas, Florida, Georgia, and New York [3].

The United States has a population of 328239523 and is one of the most diverse countries in the world, with 13.4% African Americans (AA) and 18.5% Hispanics (HSP). Health disparity is a significant issue in the United States, reflecting differences in access to healthcare, education, lifestyle, and socioeconomic status[4]. This minority population has been disproportionately affected by the pandemic[5]. For example, in Chicago, IL[6], the rates of COVID-19 positive cases as of September 21 are more excellent among Latinos and African Americans [30232 (47.8% of the total cases) cases and 13273 (27.3% of the total cases) cases respectively] in comparison to Whites [11092 cases (17.5% of the total cases)]. The same applies to mortality rates, with deaths from Latinos and African Americans are 33.1% and 42.7%, respectively, while Whites represent just 19.1% of the total deaths. This same scenario was reproduced in many other states, with reported cases and deaths in minorities much higher than their percentage within the general population.

The most common explanation for such disproportionate impact of COVID-19 in these minority populations might be the higher prevalence of chronic conditions or comorbidities when compared to Whites (European Americans)[7]. These populations are probably also living in conditions that promote breeding and incubation for infection and transmission of the virus. Apart from the clinical and environmental causes, there may be genetic/biological factors that may also play a role in predisposing them to more severe disease and more unsatisfactory outcomes. Understanding the transmission dynamics, a different array of symptoms and the impact of the presence of another chronic disease in minority patients can provide important hints about the progression of the pandemic and treatment options, especially in areas where access to equal health services is limited, unequal and challenging for underserved populations is broad[8,9].

This study presents the findings of a comprehensive analysis of COVID-19 patients in a Washington DC tertiary hospital that caters primarily to minority populations. The main objective of this study was to define significant outcome effectors in this patient population. Special emphasis was made on gastrointestinal (GI) symptoms, laboratory values and comorbidities.

MATERIALS AND METHODS

Patients' selection

This retrospective study used data from 386 adult patients hospitalized for COVID-19 at Howard University Hospital. Demographic, clinical, and pathological features of these patients were collected. Using the medical record number for each patient, we searched all reports in the hospital system and analyzed all available charts and doctors' notes to collect all relevant data for our study. Howard University Institutional Review Board approved this study. The main features, prognosis assessment and potential effectors relate to the outcome of patients were collected. This is a retrospective study covering patients from the first wave and for a period 6 mo (March

to August 2020) when protocols and management of patients were evolving with increasing clinical parameters being captured and ordered by treating physicians. Also, because our institution mainly caters to minorities, Caucasians were not well represented in our cohort.

Collected data

The following information was collected for all COVID-19 patients: Demographics (date of the report, age, gender, height, weight, body mass index), symptoms (fever, cough, shortness of breath, abdominal pain, anorexia, diarrhea, nausea, vomiting, fatigue), comorbidities (cardiac disease, diabetes mellitus, hypertension, immunocompromised status, alcohol consumption) and laboratory values: lymphocytes count (reference value: 0.9-3.2 × 10^9), C-reactive protein (CRP, reference value: < 10 mg/dL), D-dimers (reference value: < 10 mg/dL), Ferritin (reference value: 20-400 ng/mL), Creatinine (reference value: 0.6-1.2 mg/dL), Alanine transaminase (ALT, reference value: 0-55 IU/L), Alkaline Phosphatase (ALP reference value: 0-50 IU/L), Aspartate Transaminase (AST, reference value: 0-50 IU/L), Albumin (reference value: 3.2-5.5 g/dL), Procalcitonin (reference value: < 0.50 ng/mL), treatment (Hydroxychloroquine, Glucocorticoid, Intubation and Mechanical ventilation) and outcome (alive or dead). For laboratory clinical values, reference lab values were used to determine normal from out-of-range values.

Data synthesis and statistical analysis

Patient demographics, symptoms, underlying comorbidities, treatment, and outcomes were compared among AA, EA, LAT, and other ethnic groups. Predictors of hospital mortality evaluated by using logistic and/or multiple logistic regression using four models to assess the effect of each risk factor with: (1) No adjustment (OR1); (2) Adjusted for gender, age, ethnicity, and center (OR2); (3) The previous (OR2) model, further adjusted for comorbidities (OR3); and (4) The previous (OR3) model, further adjusted for disease severity (OR4). Each OR and the associated 95% confidence interval for OR were calculated. The 95% confidence interval was investigated to see if it contains unity. Present of unity (*i.e.*, OR=1) in any confidence interval means that the concerned risk factor is not statistically and significantly affects mortality. SPSS version 26 (SPSS Inc., Chicago, IL, United States) was used for this analysis.

RESULTS

Adults over 50 years were predominantly affected by COVID-19, and age was considered as a significant effector of outcome

Among the 386 patients, the mean age was 55.6 years. There were 257 AAs, 102 HSP (Latin Americans), and 26 Caucasians (CAU, European Americans), while 14 were other races. With respect to mortality, 20.6% (53 patients) AAs, 11.5% (3 patients) CAU and 6.9% (7 patients) HSP died (Table 1). The mean age for those who survived was 53.4 years old and 67.2 years old for the deceased. The mean age of the deceased among the AA was 68.4 years, HSP 51.1 years and CAU 84.6 years. Among deceased, the average length of hospital stay in days was highest in EA (25.5 d) followed by AA (11.1 d) and HSP (6.1 d). Using less than 35 years old as a reference, those within the groups of over 75 years old had 5.80 more risks to die in OR2 analysis (CI = 1.52-22.1), and those between 65 years old to 74 years old had 9.88 times more risk to die (OR2, CI = 2.62-37.17) (Table 2).

Male mortality was higher despite equal infection rates in both genders

Our cohort was homogeneously distributed for gender with 50.1% females and 49.9% males (Table 1). The male death rate was 19.1% (37 patients), while the females' rate was 13.6% (26 patients, Table 2). For AA (140 males and 117 females), 21.4% of males and 19.7% of females died in the hospital. In contrast with Caucasians CAU (15 males and 11 females), none of the females died because of the infection and three males (20%) were deceased. For HSP (39 males and 63 females), 10.3% (4 patients) male patients and 4.8% (3 patients) females patients died (Table 1).

Ethnicity did not influence in determining outcomes in our cohort

The race was not associated with outcome. Using AA as a reference, overall CAU had less risk of dying (OR3 = 0.38, CI = 0.08-1.83) and HSP with 0.55 Less risk than comparing with AA (OR3, CI = 0.21-1.47) (Table 2). The African Americans in our

Table 1 Overall baseline characteristics of the study population

	African Americans, *n* (%)	European- Americans, *n* (%)	Latin-Americans, *n* (%)
Indices			
Discharge status (*n* = 385)			
Alive (*n* = 322)	204 (79.4)	23 (88.5)	95 (93.1)
Dead (*n* = 63)	53 (20.6)	3 (11.5)	7 (6.9)
Sex (*n* = 386)			
Female (*n* = 191)	117 (45.5)	11 (42.3)	63 (61.2)
Male (*n* = 195)	140 (54.5)	15 (57.7)	40 (38.8)
BMI (*n* = 332)			
Normal (*n* = 127)	75 (31.9)	12 (52.2)	40 (54.1)
Overweight (*n* = 94)	69 (29.4)	6 (26.1)	19 (25.7)
Obese (*n* = 111)	91 (38.7)	5 (21.7)	15 (20.3)
Baseline comorbidities			
Cardiac disease (*n* = 377)			
No (*n* = 236)	141 (55.3)	18 (72)	77 (79.4)
Yes (*n* = 141)	114 (44.7)	7 (28)	20 (20.6)
Hypertension (*n* = 373)			
No (*n* = 183)	105 (41.7)	10 (40)	68 (70.8)
Yes (*n* = 190)	147 (58.3)	15 (60)	28 (29.2)
Immunosuppression (*n* = 369)			
No (*n* = 335)	225 (88.9)	21 (87.5)	89 (96.7)
Yes (*n* = 34)	28 (11.1)	3 (12.5)	3 (3.3)
Presenting symptoms			
Fever (*n* = 383)			
No (*n* = 174)	107 (41.8)	12 (46.2)	55 (54.5)
Yes (*n* = 209)	149 (58.2)	14 (53.8)	46 (45.5)
Cough (*n* = 380)			
No (*n* = 151)	93 (36.8)	6 (23.1)	52 (51.5)
Yes (*n* = 229)	160 (63.2)	20 (76.9)	49 (48.5)
Shortness of breath (*n* = 381)			
No (*n* = 144)	85 (33.2)	9 (36)	50 (50)
Yes (*n* = 237)	171 (66.8)	16 (64)	50 (50)
Fatigue (*n* = 236)			
No (*n* = 143)	78 (53.8)	11 (55)	54 (76.1)
Yes (*n* = 93)	67 (46.2)	9 (45)	17 (23.9)
Nausea (*n* = 362)			
No (*n* = 315)	208 (86.7)	19 (82.6)	88 (88.9)
Yes (*n* = 47)	32 (13.3)	4 (17.4)	11 (11.1)
Vomiting (*n* = 375)			
No (*n* = 332)	216 (87.1)	22 (88)	94 (92.2)
Yes (*n* = 43)	32 (12.9)	3 (12)	8 (7.8)
Abdominal pain (*n* = 343)			

No (*n* = 296)	205 (87.6)	17 (81)	74 (84.1)
Yes (*n* = 47)	29 (12.4)	4 (19)	14 (15.9)
Diarrhea (*n* = 377)			
No (*n* = 317)	203 (81.2)	22 (88)	92 (90.2)
Yes (*n* = 60)	47 (18.8)	3 (12)	10 (9.8)
Anorexia (*n* = 325)			
No (*n* = 238)	147 (68.7)	16 (80)	75 (82.4)
Yes (*n* = 87)	67 (31.3)	4 (20)	16 (17.6)
Laboratory results			
Lymphocyte count (*n* = 380)			
Normal (*n* = 193)	120 (47,1)	14 (53.8)	59 (59.6)
Low (*n* = 163)	120 (47.1)	10 (38.5)	33 (33.3)
Elevated (*n* = 24)	15 (5.9)	2 (7.7)	7 (7.1)
CPK (*n* = 293)			
Normal (*n* = 99)	69 (32.5)	7 (38.9)	23 (36.5)
Elevated (*n* = 194)	143 (67.5)	11 (61.1)	40 (63.5)
D-Dimer (*n* = 309)			
Normal (*n* = 39)	23 (10)	6 (30)	10 (17.2)
Elevated (*n* = 270)	208 (90)	14 (70)	48 (82.8)
Procalcitonin (*n* = 276)			
Normal (*n* = 163)	114 (55.9)	13 (72.2)	36 (66.7)
Elevated (*n* = 113)	90 (44.1)	5 (27.8)	18 (33.3)
Ferritin (*n* = 273)			
Normal (*n* = 98)	73 (35.4)	5 (41.7)	20 (36.4)
Elevated (*n* = 175)	133 (64.6)	7 (583)	35 (63.6)
AST (*n* = 332)			
Normal (*n* = 185)	129 (54.7)	13 (54.2)	43 (59.7)
Elevated (*n* = 147)	107 (45.3)	11 (45.8)	29 (40.3)
ALT (*n* = 329)			
Normal (*n* = 231)	168 (72.1)	16 (66.7)	47 (65.3)
Elevated (*n* = 98)	65 (27.9)	8 (33.3)	25 (34.7)
ALP (*n* = 317)			
Normal (*n* = 260)	191 (86)	17 (81)	52 (70.3)
Elevated (*n* = 57)	31 (14)	4 (19)	22 (29.7)
Hydroxychloroquine (*n* = 371)			
No (*n* = 291)	185 (92.4)	24 (96)	82 (88.2)
Yes (*n* = 80)	68 (26.9)	1 (04)	11 (11.8)
Interventions			
Glucocorticoids (*n* = 371)			
No (*n* = 303)	202 (73.1)	20 (80)	81 (87.1)
Yes (*n* = 68)	51 (20.2)	5 (20)	23 (12.9)
Mechanical ventilation (*n* = 376)			
No (*n* = 320)	204 (81.3)	23 (92)	93 (93)

Yes (*n* = 56)	47 (18.7)	2 (8)	7 (7)

BMI: Body mass index.

study had the highest mortality as they consisted of an older population compared to the Hispanic and other ethnic groups. In other words, if controlling for age, sex, ethnicity, center and other potential confounding variables, age is the most crucial factor in determining the mortality rate.

Body mass index was a minor effector of outcome

In our cohort, 127 patients had a normal body mass index (BMI) (< 25), 111 were obese (BMI > 30), and 94 were overweight (25 < BMI < 30) (Tables 1 and 2). In the obese group, 19.8% died, followed by 19.1% in the overweight group and 15% in the normal BMI group. Among the AA patients, the death rate was high for normal weight (22.7%) compared to the obese (20.9%) and overweight (21.7%). There was no significant association between high BMI (overweight and/or obesity) and poor outcome (OR were not significant) for BMI.

Fever and shortness of breath were more common in AA

Shortness of breath, cough and fever were the most common symptoms in our cohort. Overall, 62% (237 patients) reported shortness of breath, followed by 59.7% (229 patients) cough, and 54.6% (209 patients) had a fever as their symptom (Table 1). There were race-based differences concerning the presentation of symptoms. Fever (58.2%) and shortness of breath (66.8%) were more commonly seen in AA when compared to CAU and HSP. Whereas cough was the most common presentation of the EA (76.9) when compared to the AA (63.2%) and HSP (48.5%). While cough and fever were the most common symptoms, neither cough (OR 2 = 0.87 with a CI = 0.46-1.63) nor fever (OR2 = 1.07, CI = 0.58-1.96) were significant for poor outcome. However, shortness of breath was highly associated with mortality (OR2 = 3.64, CI = 1.73-7.65), even after adjusting for age, sex, and ethnicity (Table 2). In AAs, CAU and HSP, the above symptoms affected the outcome similarly as in the overall cohort.

Diarrhea was the most prevalent GI symptom, and GI symptoms did not affect outcome

Diarrhea (15.9%) was the most common GI symptom among the overall cohort, followed by nausea (13%), vomiting (11.3%), anorexia (26.4%), and abdominal pain (13.8%) (Table 1). This distribution of GI symptoms was the same within racial groups. Diarrhea was reported in 18.8%, 12%, and 9.8% in AAs, CAU and HSP, respectively.

Hypertension and diabetes mellitus were the most prevalent chronic conditions

Hypertension and diabetes mellitus was present in 51% and 37.4% of patients, respectively (Table 2). They were followed by cardiac disease in 19% of the cohort and immunocompromised status in 9.1%. All the ethnic groups have hypertension followed by diabetes mellitus as the most common pre-existing comorbidities. Only cardiac disease was significantly associated with poor outcomes in the crude calculation (OR = 1.86, 95%CI = 1.00-3.47). However, after we adjust for age, gender, race, this becomes not significant (OR = 0.96, 95%CI = 0.47-1.05) (Table 2).

AST and ALT were the most altered in the liver test panel and are associated with poor outcomes

AST was elevated in 147 patients (43.8%) (Table 2). ALT values were high in 98 patients (29.6%) and ALP in 60 patients (18.2%). The same trend was encountered in the race groups, with AST elevated in 45.3% and 40.2%. AST with an OR ratio of 7.90 (3.93-15.9) has the highest association with poor outcomes (Table 2). In OR2 analysis adjusted for age, gender, and ethnicity, elevated AST was associated with poor outcomes in a statistically significant manner. Elevated AST added 8 times more risk to the patients. Worth noting that AST is not specific to the liver. For the ALT enzyme, which is more liver-specific, the OR associating with death was 2.64 (1.36-5.01) after adjustment for age, race, and gender.

Glucocorticoids treatment did not improve patients outcome

Overall, 18% (68 patients) received glucocorticoids. Concerning outcome, 39% (27

Table 2 Association between clinical laboratory markers and death as an outcome

	Alive, *n* (%)	Dead, *n* (%)	OR1 (95%CI)	OR2 (95%CI)	OR3 (95%CI)	OR4 (95%CI)
Age (*n* = 384), yr						
< 35 (*n* = 66)	63 (95.5)	3 (4.5)	-	-	-	-
35-44 (*n* = 50)	48 (96)	2 (4)	0.87 (0.14-5.44)	0.65 (0.10-4.19)	0.71 (0.094-5.50)	0.27 (0.39-1.90)
45-54 (*n* = 45)	41 (91.1)	4 (8.9)	2.04 (0.43-9.63)	1.64 (0.33-7.96)	1.85 (0.31-11.07)	0.69 (0.13-3.64)
55-64 (*n* = 95)	85 (89.5)	10 (10.5)	2.47 (0.65-9.34)	1.63 (0.39-6.66)	1.54 (0.30-7.93)	0.70 (0.15-3.20)
65-74 (*n* = 63)	37 (58.7)	26 (41.3)	14.7 (4.17-52.13)	9.88 (2.62-37.17)	9.91 (2.09-46.81)	3.08 (0.73-13.01)
≥ 75 (*n* = 65)	47 (72.3)	18 (27.7)	8.04 (2.23-28.90)	5.80 (1.52-22.1)	5.95 (1.23-28.80)	2.10 (0.49-8.98)
Sex (*n* = 385)						
Female (*n* = 191)	165 (86.4)	26 (13.6)	-	-	-	-
Male (*n* = 194)	157 (80.9)	37 (19.1)	1.49 (0.86-2.58)	1.44 (0.78-2.65)	1.43 (0.75-2.70)	1.17 (0.58-2.36)
Ethnicity (*n* = 385)						
African American (*n* = 257)	204 (79.4)	53 (20.6)	-	-	-	-
European American (*n* = 26)	23 (88.5)	3 (11.5)	0.50 (0.14-1.73)	0.54 (0.14-1.96)	0.38 (0.08-1.83)	1.00 (0.22-4.50)
LatinX (*n* = 102)	95 (93.1)	7 (6.9))	0.28 (0.12-0.64)	0.52 (0.21-1.30)	0.55 (0.21-1.47)	0.64 (0.23-1.76)
BMI (*n* = 332)						
Normal (*n* = 127)	108 (85)	19 (15)	-	-	-	
Overweight (*n* = 94)	76 (80.9)	18 (19.1)	1.34 (0.66-2.73)	1.02 (0.47-2.23)	1.00 (0.46-2.19)	
Obese (*n* = 111)	89 (80.2)	22 (19.8)	1.40 (0.71-2.75)	1.22 (0.58-2.56)	1.18 (0.56-2.49)	
Baseline comorbidities (no, always as reference)						
Cardiac disease (*n* = 375)						
No (*n* = 302)	257 (85.1)	45 (14.9)	-	-		
Yes (*n* = 73)	55 (75.3)	18 (24.7)	1.86 (1.00-3.47)	0.96 (0.47-1.95)		
Diabetes mellitus (*n* = 377)						
No (*n* = 236)	202 (85.6)	34 (14.4)	-	-		
Yes (*n* = 141)	112 (79.4)	29 (20.6)	1.53 (0.89-2.65)	1.15 (0.63-2.11)		
Hypertension (*n* = 373)						
No (*n* = 183)	156 (85.2)	27 (14.8)	-	-		
Yes (*n* = 190)	155 (81.6)	35 (18.4)	1.30 (0.75-2.25)	0.79 (0.42-1.51)		
Immune suppression (*n* =369)						
No (*n* = 355)	282 (84.2)	53 (15.8)	-	-		
Yes (*n* = 34)	25 (73.5)	9 (26.5)	1.91 (0.84-4.33)	2.19 (0.87-5.53)		
Presenting symptoms						
Fever (*n* = 383)						
No (*n* = 174)	149 (85.6)	25 (14.4)	-	-		
Yes (*n* = 209)	171 (81.8)	38 (18.2)	1.32 (0.76-2.29)	1.07 (0.58-1.96)		
Cough (*n* = 380)						
No (*n* = 151)	125 (82.8)	26 (17.2)	-	-		
Yes (*n* = 229)	193 (84.3)	36 (15.7)	0.89 (0.51-1.55)	0.87 (0.46-1.63)		
Shortness of breath (*n* = 381)						
No (*n* = 144)	133 (92.4)	11 (7.6)	-	-	-	-
Yes (*n* = 237)	185 (78.1)	52 (21.9)	3.39 (1.70-6.75)	3.64 (1.73-7.65)	3.60 (1.71-7.56)	2.38 (1.07-4.09)

Fatigue (*n* = 236)						
No (*n* = 143)	125 (87.4)	18 (12.6)	-	-		
Yes (*n* = 93)	80 (86)	13 (14)	1.12 (0.52-2.42)	0.71 (0.30-1.67)		
Nausea (*n* = 362)						
No (*n* = 315)	264 (83.8)	51 (16.2)	-	-		
Yes (*n* = 47)	42 (89.4)	5 (10.6)	0.61 (0.23-1.63)	0.86 (0.30-2.50)		
Vomiting (*n* = 375)						
No (*n* = 332)	277 (83.4)	55 (16.6)	-	-		
Yes (*n* = 43)	39 (90.7)	4 (9.3)	0.51 (0.17-1.50)	0.631 (0.20-1.99)		
Abdominal pain (*n* = 343)						
No (*n* = 296)	248 (83.8)	48 (16.2)	-	-		
Yes (*n* = 47)	44 (93.6)	3 (6.4)	0.35 (0.10-1.18)	0.50 (0.14-1.82)		
Diarrhea (*n* =377)						
No (*n* = 317)	264 (83.3)	53 (16.7)	-	-		
Yes (*n* = 60)	52 (86.7)	8 (13.3)	0.76 (0.34-1.70)	0.74 (0.31-1.78)		
Anorexia (n = 325)						
No (*n* = 238)	208 (87.4)	30 (12.6)	-	-		
Yes (*n* = 87)	69 (79.3)	18 (20.7)	1.80 (0.94-3.44)	1.30 (0.63-2.68)		
Laboratory results (quartiles determined based on the entire study population; q1 always as reference)						
Lymphocyte count (*n* = 380)						
Normal (*n* = 193)	177 (91.7)	16 (8.3)	-	-		
Low (*n* = 163)	121 (74.2)	42 (25.8)	3.84 (2.06-7.14)	2.77 (1.41-5.45)		
Elevated (*n* = 24)	19 (79.2)	5 (20.8)	2.91 (0.95-8.83)	3.27 (0.97-11.04)		
CRP (*n* = 293)						
Normal (*n* = 99)	91 (919)	8 (8.1)	-	-	-	-
Elevated (*n* = 194)	149 (76.8)	45 (23.3)	3.43 (1.55-7.61)	3.19 (1.39-7.29)	3.11 (1.38-7.04)	2.77 (1.21-6.34)
D-Dimer (*n* = 309)						
Normal (*n* = 39)	39 (100)	0 (0)	L. Reg. N/A: No death for normal D-dimer; Chi-square *P* = 0.001 highly significant			
Elevated (*n* = 270)	212 (78.5)	58 (21.5)				
Procalcitonin (*n* = 276)						
Normal (*n* = 163)	151 (92.6)	12 (7.4)	-	-		
Elevated (*n* = 113)	67 (59.3)	46 (40.7)	8.63 (4.30-17.3)	8.27 (3.95-17.3)		
Ferritin (*n* = 273)						
Normal (*n* = 98)	88 (89.8)	10 (10.2)	-	-		
Elevated (*n* = 175)	131 (74.9)	44 (25.1)	2.95 (1.41-6.18)	2.69 (1.24-5.82)		
Hydroxychloroquine (*n* = 371)						
No (*n* = 291)	249 (80.8)	42 (66.7)	-	-		
Yes (*n* = 80)	59 (19.2)	21 (33.3)	2.11 (1.16, 3.83)	1.93 (0.98 - 3.79)		
ALP (*n* = 317)						
Normal (*n* = 260)	221 (85)	39 (68.4)	-	-		
Elevated (*n* = 57)	39 (15)	18 (31.6)	2.61 (1.36 – 5.03)	3.52 (1.65-7.51)		
AST (*n* = 332)						
Normal (*n* = 185)	174 (94.1)	11 (5.9)	-	-		

Elevated (*n* = 147)	98 (66.7)	49 (33.3)	7.90 (3.93-15.9)	8.01 (3.79-16.9)
ALT (*n* = 329)				
Normal (*n* = 231)	197 (85.3)	34 (14.7)	-	-
Elevated (*n* = 98)	72 (73.5)	26 (26.5)	2.09 (1.17-3.73)	2.64 (1.36-5.01)
Interventions (no as reference)				
Glucocorticoids (*n* = 371)				
No (*n* = 303)	267 (88.1)	36 (11.9)	-	-
Yes (*n* = 68)	41 (60.3)	27 (39.7)	4.88 (2.68-8.87)	5.40 (2.72-10.7)
Mechanical ventilation (*n* = 376)				
No (*n* = 320)	303 (94.7)	17 (5.3)	-	-
Yes (*n* = 56)	17 (30.4)	39 (69.6)	40.8 (19.3-86.5)	35.2 (15.3-81.1)

OR1 = Crude, without any adjustment; OR2 = Adjusted for age, sex, ethnicity; OR3 = Multivariable, adjusted for age, sex, ethnicity, and other potential confounding variables (to be decided later). The final model suggest that Age, shortness of breath (SOB) and C-reactive protein (CRP) is significantly increase the risk of mortality. Baseline comorbidity, ethnicity and gender are not significant after controlling for SOB and CRP. BMI: Body mass index; CRP: C-reactive protein; ALP: Alkaline phosphatase; AST: Aspartate transaminase; ALT: Alanine transaminase.

patients) of those that received glucocorticoid died. Among AA, 20.2% received glucocorticoids, of these 45% of them died. Among CAU, 20% were on glucocorticoids (5 patients), and only one died. In HSP, 12.9% received glucocorticoids, and 3 of them died. In HSP, 12.9% received glucocorticoids, and 3 of them died. Among glucocorticoid-treated patients, there was an overall risk of 5.40 times more than those that did not receive them to die (OR2, CI = 2.72-10.7) (Table 2).

Mechanical ventilation associated with poor outcome

Overall, 14.9% (56 patients) received mechanical ventilation. The proportion of patients receiving mechanical ventilation was highest among the AA (18.7%) compared to CAU (8%) and HSP (7%) (Table 1). Patients on mechanical ventilation were 40 times more susceptible to poor outcomes. Even after controlling the analysis for age, sex and ethnicity, patients on mechanical ventilation were still 35 times (OR = 35.2, CI= 15.3-81.1) more prone to poor outcomes (Table 2).

DISCUSSION

The United States has one of the most diverse populations[10], exhibiting discrepancies in healthcare access, socioeconomic status, and wealth distribution, all of which lead to disparities in health outcomes across different groups, especially in minorities [11]. African Americans and Hispanics, who are known to carry a heavy burden of comorbidities compared to the general population, are likely to have these comorbidities exacerbated by the current COVID-19 pandemic and likely to exacerbate outcomes from the virus infection[12].

This study comprehensively analyzed features and effectors of COVID-19 patients hospitalized in a Tertiary Care University Hospital in Washington DC, which caters primarily to minorities. We found that elevated liver enzymes, procalcitonin, ferritin, CRP and D-dimers are robust markers of poor prognosis in minority patients receiving medical care for COVID-19. GI symptoms, although not indicative of outcome in COVID-19 patients, were prevalent in our cohort. Anorexia (26.4%), diarrhea (15.8%), abdominal pain (13.8%), nausea (13%) and vomiting (11.3%) were the main symptoms of the GI tract-associated symptoms. GI symptoms are likely important latent markers to manifest as the virus persists within the GI system, even after clearing the respiratory system.

In this cohort, the patients that died were more likely to be older than 50 years; this agrees with Center for Disease Control (CDC) reports for hospitalized patients in which 74.5% were older than 50 years[13]. Interestingly, Hispanics had the youngest patients' group; the mean age was 43.2 for survivors, while the deceased was 51.1 years. Rodriguez-Diaz *et al*[14] reported similar findings with their Hispanics being less than 35 years. The high rate of young people within the HSP in this study is likely

a reflection of the occupation these patients are involved in, making them highly exposed to the virus and unlikely to benefit from the protections that remote working offers to others.

Both genders were equally represented in our cohort. Many studies, including a CDC report found in a study of 1482 patients (54.4% males)[13], report more males than female infections, generally explained by men's likely higher exposures in many societies. Males had higher mortality in our cohort. This was explained by potential protective immune functions on the X chromosome[15] or protective effects hypothetically attributed to female hormones. This trend was reproduced in the 3 races included in this study (AA, CAU, and HSP). Worth noting that AA had the highest mortality rate in our cohort when compared to CAU and HSP. While this finding may be affected by sample sizes of the 3 groups, Doumas *et al*[16] have also reported that AA displays the worst outcome in the United States across different racial and ethnic groups.

Obesity has been described as a negative marker in COVID-19 patients' outcomes, primarily because of obesity-associated pro-inflammation, excessive oxidative stress, impaired immunity, and a creator/trigger of metabolic syndrome[17,18]. Our findings showed that 20.2% were obese, 18.6% overweight and 14.8% regular BMI patients died. However, this trend of high BMI with death was not statistically significant. This contrasts with what was found by Malik *et al*[19]'s meta-analysis of 10233 COVID-19 confirmed cases where they showed a significant association between obesity and COVID-19 severity and poor outcomes.

Shortness of breath was seen in 62%, cough in 59.7%, fever in 54.6% and fatigue (39.5%). Overall, in our cohort, although very common, these symptoms were not all related to outcome/severity or mortality.

Shortness of breath was significantly associated with higher mortality in the HSP group only. This contrasts with Li *et al*[20] who reported that in the Henan province in China, fatigue and expectoration were signs of severe COVID-19 infection and prognostic markers for outcome in their patients, and shortness of breath was prognostic only for males. Can *et al*[21] also reported similar presentations distribution in their COVID-19 patients that had fatigue (46.5%), cough (69.4%) and fever (41.4%).

The presence of GI symptoms points to the likelihood of the virus affecting different systems at once, even though others hypothesize that such non-respiratory symptoms are associated with inflammations triggered by infections of other systems besides the lungs. Angiotensin-converting enzyme 2 (ACE2) receptor, the target of the virus SPIKE protein and transmembrane protease serine 2 (TMPRSS2) that is needed for its cleavage and entry into the cells, was reported to be expressed in the GI system, and as such, we cannot rule out that GI symptoms may be the result of a direct virus effect on these systems. It is noteworthy that many patients who tested negative for the virus in the respiratory specimens continued to test positive for the virus RNA in their stools after they recovered from the disease[22]. It is possible that in the foreseeable future, many new disorders in the GI system, as well as neurological and cardiovascular systems, of which SARS-CoV-2 is an etiological element may occur among patients who recovered from COVID-19 infection.

Diabetes mellitus and hypertension, two of the most common chronic conditions in the United States were not significantly associated with higher mortality in our cohort. Cardiac disease in heart failure or coronary artery disease was present in 19% of our cohort and was significantly associated with higher mortality (P = 0.028). This contrasts with the Almeida-Petitto *et al*[23]*'s* report, where cardiovascular disease is related to outcome and diabetes mellitus and hypertension. When the analysis was done within the race groups in this study, none of the comorbidities were significant for AA, CAU or HSP.

Laboratory test values are essential markers for the general assessment of underlying determinants of observed symptoms and prognosis. In OR2 analysis, low lymphocyte elevated CRP, elevated procalcitonin, elevated ferritin and elevated AST and ALT were significantly associated with severe outcome and death in our cohort. These findings corroborate with Bastug *et al*[24]'s cohort, where they reported that D-dimer and CRP are significant predictors of mortality, but not AST, LDH, ferritin, or lymphocytes. COVID-19 induces systemic inflammation and a prothrombic state which translates to elevated CRP, procalcitonin, and D-dimer levels which are the worst prognostic factors[25].

Liver function test has been well documented in COVID-19 patients. In our cohort, elevated AST was highly significant for higher mortality. AST was found to be elevated in 147 patients (43.8%); ALT was found to be elevated in 98 patients (29.6%). None of our findings for the overall cohort were translated to the race group analyses. While elevated AST is associated with the highest OR for poor outcomes, not all AST

can be assigned to the liver. However, the more liver-specific ALT enzyme did associate with a higher OR of poor outcome, although not to the same magnitude as AST associated OR. These findings confirm that liver function alterations that might stem from viral-induced liver damage of triggered inflammation are related to severe outcomes. Wang *et al*[27] described histological damage to the liver in a cohort of 156 patients. Their findings stated that infected hepatocytes displayed conspicuous mitochondrial swelling, endoplasmic reticulum dilatation, glycogen granule decrease, massive hepatic apoptosis, and some binuclear hepatocytes. These are hallmarks of typical lesions of viral infection origin. As stated above, for the GI system of recovered patients, liver health and status need to be monitored in millions of recovering patients to avoid a resurgence of new liver diseases linked to the sequels of the pandemic.

Treatments implemented in our hospital (21.1% with hydroxychloroquine and 18% with glucocorticoids) did not seem to have benefited our patients. Indeed, there was even higher mortality in those receiving them (hydroxychloroquine with $P = 0.019$, and glucocorticoids with $P < 0.0001$). Similar findings were reported by Budhathoki *et al*[28] who stated that using corticosteroids made the viral clearance duration and hospital stay longer in the treated group, real-time polymerase chain reaction took 3 d more to come negative when compared to the non-treated group. Patel *et al*[29] also reported an adverse outcome risk using hydroxychloroquine and even worse when adding azithromycin. It is worth mentioning that the present cohort of our study corresponds to the first wave of COVID-19 patients where treatment protocols were still being developed and tried.

CONCLUSION

In conclusion, elevated liver enzymes, ferritin, CRP, and D-dimers are robust markers of poor prognosis. The African Americans in our study displayed the highest mortality as they consisted of an older population compared to the Hispanic group. GI symptoms did not correlate with the outcome; however, they may manifest essential features as the virus persists within the GI system, even after clearing from the respiratory system. Attention should also be paid to monitor liver function during COVID-19, especially in African Americans and Hispanic patients with higher disease severity. Our study showed that digestive symptoms and liver injury are not uncommon in patients with COVID-19. Increased attention should be paid to the care of African Americans and Hispanics' unique group of patients.

ARTICLE HIGHLIGHTS

Research background

The coronavirus disease 2019 (COVID-19) disproportionately affected African Americans and Hispanics. The United States has one of the most diverse populations, exhibiting discrepancies in healthcare access, socioeconomic status, and wealth distribution, all of which lead to disparities in health outcomes across different groups, especially in minorities.

Research motivation

To evaluate the clinical manifestations, comorbidities, and laboratory parameters in COVID-19 patients and identify risk factors that may be related to poor prognosis.

Research objectives

To clarity whether clinical manifestations and laboratory parameters are related to the prognosis of COVID-19 positive African American (AA) patients.

Research methods

This study is a retrospective analysis. Patient demographics, symptoms, underlying comorbidities, treatment, and outcomes were compared among AA, Caucasians, Hispanics, and other ethnic groups. Predictors of hospital mortality evaluated by using logistic and/or multiple logistic regression. SPSS version 26 (SPSS Inc., Chicago, IL, United States) was used for this analysis.

Research results

A total of 386 COVID-19 positive patients, 257 (63.7%) were AAs, 102 (25.3%) Hispanics, and 26 (6.45%) Whites. The mortality rate was highest among the AAs (20.6%) and lowest among Hispanics (6.9%). Patients with shortness of breath (OR2 = 3.64, CI = 1.73-7.65) and elevated AST (OR2 = 8.01, CI = 3.79-16.9) elevated Procalcitonin (OR2 = 8.27, CI = 3.95-17.3), AST (OR2 = 8.01, CI = 3.79-16.9), ferritin (OR2 = 2.69, CI = 1.24-5.82), and Lymphopenia (OR2 = 2.77, CI = 1.41-5.45) had a high mortality rate. Glucocorticoid treatment was associated with higher mortality (OR2 = 5.40, CI = 2.72-10.7)

Research conclusions

The African Americans were the most affected population due to severe acute respiratory syndrome coronavirus 2 in our study with high mortality. Predictors of poor outcomes in our study are Age > 50, shortness of breath, increased liver enzymes, CRP, Ferritin, Procalcitonin. Injudicious use of glucocorticoids resulted in poor outcomes. The presence of gastrointestinal symptoms did not increase disease severity.

Research perspectives

In the future, we will continue to follow COVID-19 positive patients to analyze the causes of death and the risk factors that may lead to death. And we will devote ourselves to finding predictors related to the prognosis of minority patients with COVID-19.

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