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MAJOR ARTICLE

COVID-19 in Hospitalized Adults With HIV

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Background. The spread of SARS-CoV-2 and the COVID-19 pandemic have caused significant morbidity and mortality worldwide. The clinical characteristics and outcomes of hospitalized patients with SARS-CoV-2 and HIV co-infection remain uncertain.

Methods. We conducted a matched retrospective cohort study of adults hospitalized with a COVID-19 illness in New York City between March 3, 2020, and May 15, 2020. We matched 30 people with HIV (PWH) with 90 control group patients without HIV based on age, sex, and race/ethnicity. Using electronic health record data, we compared demographic characteristics, clinical characteristics, and clinical outcomes between PWH and control patients.

Results. In our study, the median age (interquartile range) was 60.5 (56.6–70.0) years, 20% were female, 30% were black, 27% were white, and 24% were of Hispanic/Latino/ethnicity. There were no significant differences between PWH and control patients in presenting symptoms, duration of symptoms before hospitalization, laboratory markers, or radiographic findings on chest x-ray. More patients without HIV required a higher level of supplemental oxygen on presentation than PWH. There were no differences in the need for invasive mechanical ventilation during hospitalization, length of stay, or in-hospital mortality.

Conclusions. The clinical manifestations and outcomes of COVID-19 among patients with SARS-CoV-2 and HIV co-infection were not significantly different than patients without HIV co-infection. However, PWH were hospitalized with less severe hypoxemia, a finding that warrants further investigation.

Keywords. coronavirus disease 2019; HIV; severe acute respiratory syndrome coronavirus 2.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was identified as the causative agent of coronavirus disease 2019 (COVID-19) and has led to a significant burden of morbidity and mortality worldwide [1]. Infection with SARS-CoV-2 causes a broad range of symptoms, including fever, cough, dyspnea, sore throat, headache, nausea, vomiting, and diarrhea [2, 3]. Severe COVID-19 disease can progress to hypoxic respiratory failure, sepsis, and multiorgan system failure, which can be life-threatening [4]. Prior studies have revealed lymphopenia as a prominent laboratory feature of COVID-19 [2, 3]. Among hospitalized patients with COVID-19 in New York City, 90% had evidence of lymphopenia (<1500 per mm^3) [5]. Lymphopenia is also a prominent feature in HIV infection and has been identified as a predictor of HIV disease progression [6, 7]. The immune response and the role of the host immune system in the setting of infection with SARS-CoV-2 remain incompletely understood.

New York City was the early epicenter of the COVID-19 pandemic, with 192 840 confirmed cases reported to date, 16 232 confirmed COVID-19 deaths, and 4771 probable COVID-19 deaths as of May 22, 2020 [8]. The burden of COVID-19-related mortality by race and ethnicity in New York City showed that racial and ethnic minorities including Hispanics/Latinos and black individuals have higher rates of death compared with white and Asian populations [9]. The proportions of people with HIV (PWH) in New York City in 2018 were higher in racial and ethnic minorities: 46% of HIV cases were in black individuals, 36% in Hispanic/Latinos, and 11% in whites [10]. PWH with COVID-19 across racial and ethnic minorities warrant further study as a potential emerging vulnerable population.

Case reports and case series of SARS-CoV-2 and HIV co-infection among hospitalized patients with COVID-19 have been previously described; all reported recovery at the time of their reports [11–17]. A larger case series of 47 patients from Italy reported that 13 patients required hospitalization, 6 developed severe disease, 2 required mechanical ventilation, and 2 patients died; 45 (96%) patients fully recovered [18]. Two independent case reports of individuals with chronic HIV showed

Received 29 May 2020; editorial decision 23 July 2020; accepted 27 July 2020.

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DOI: 10.1093/ofid/ofaa327

delayed development SARS-CoV-2 antibody response, which may have implications for longer recovery periods and clinical outcomes [19, 20].

There is an absence of controlled data about the clinical characteristics and natural history of COVID-19 among individuals with HIV. Herein, we compare the clinical characteristics and outcomes of COVID-19 between hospitalized PWH and matched controls without HIV in a diverse population at the world's epicenter of the COVID-19 pandemic.

METHODS

Study Design

We conducted a matched retrospective cohort study of adults hospitalized with a COVID-19 illness at New York-Presbyterian/Weill Cornell Medical Center and its affiliate Lower Manhattan Hospital between March 3, 2020, and May 15, 2020. We included all PWH admitted to the hospital during this period and matched them in a 1:3 ratio to patients without HIV (control group) based on age, sex, and race/ethnicity.

Data Collection

We collected demographic data including age, sex, race/ethnicity, body mass index, medical comorbidities, and concomitant medications, as well as data on presenting symptoms. For all patients, we collected absolute lymphocyte counts upon presentation as well as peak values within 7 days of presentation of the following laboratory markers: C-reactive protein (CRP), procalcitonin, D-dimer, and lactate dehydrogenase. HIV-specific data included antiretroviral therapy regimens, CD4 nadir (if available in outpatient records), and most recent CD4 T-cell count, CD-8 T-cell count, CD4:CD8 ratio, and HIV-1 viral load obtained during hospitalization. We collected radiographic reports of initial chest x-ray studies and COVID-19 treatments received during hospitalization. All data were manually abstracted from the electronic health record by trained reviewers with a calibrated abstraction process that has been previously described and was demonstrated to have high inter-rater reliability [5].

Study Outcomes

We assessed respiratory status upon presentation and other hospital course events. Respiratory status upon presentation was determined by presence of hypoxemia and highest level of oxygen support within 3 hours of arrival to the emergency department. Levels of oxygen support included ambient air, nasal cannula, nonrebreather, noninvasive ventilation (bilevel or continuous positive airway pressure), and invasive mechanical ventilation. Other hospital course events included death, hospital discharge, hospital length of stay, admission to the intensive care unit, need for invasive mechanical ventilation during hospitalization, need for vasopressors, initiation of dialysis, and do not resuscitate/do not intubate (DNR/DNI) orders.

Statistical Analysis

Three-to-one matching was performed using the gmatch SAS macro, which is a greedy nearest neighbor matching algorithm [21, 22]. Matching was performed using the following characteristics: gender, age (+/-5 years), and race. Descriptive statistics were calculated, and means, standard deviations, medians, interquartile ranges, and percentages were reported. Bivariate analyses were performed using chi-square and Fisher exact tests for categorial variables, the Student *t* test for normally distributed continuous variables, and Wilcoxon tests for skewed continuous variables. Statistical significance was determined using an alpha of .05. All analyses were performed using Stata, version 14.

Patient Consent Statement

The design of this work was approved by the Weill Cornell Medicine Institutional Review Board. Written informed consent was waived, as it was an observational and retrospective analysis of our usual clinical practice. All patient data were anonymized for the purpose of analysis, and confidential data were protected in accordance with the ethical standards of the Helsinki Declaration.

RESULTS

Baseline Characteristics

Our study included PWH (n = 30) and a control group (n = 90) matched by age, sex, and race/ethnicity, as shown in Table 1. There were more active smokers (17% vs 4%) and former smokers (33% vs 22%; $P = .02$) among PWH compared with control patients. In addition, PWH more commonly had chronic obstructive lung disease as a comorbid diagnosis (13% vs 3%; $P = .07$). Chronic hepatitis B virus infection was significantly higher among PWH (20% vs 1%; $P < .001$). There were no significant differences between groups in body mass index, hypertension, diabetes mellitus, coronary artery disease, heart failure, stroke, chronic kidney disease, end-stage renal disease, asthma, cirrhosis, or chronic hepatitis C infection.

HIV Characteristics

In the PWH group, 29 of 30 patients were on antiretroviral therapy; 9 (31%) of the 29 were on regimens that contained a protease inhibitor (Table 2). Three patients did not have an HIV viral load or CD4 count recorded. Among the 27 PWH who had a recent HIV viral load and CD4 count available, all patients had undetectable viral loads (<20 copies/mL), and the median CD4 count (interquartile range [IQR]) was 332 (123–526)

Table 1. Baseline Characteristics of Hospitalized COVID-19 Patients With HIV Co-infection and Matched Controls

	PWH (n = 30), No. (%) or Median (IQR)	Control (n = 90), No. (%) or Median (IQR)	*P* Value
Age, y	60.5 (56.6–70.0)	60.5 (56.6–70.0)	.93
Sex			1.00
Male	24 (80)	72 (80)	
Female	6 (20)	18 (20)	
Race			.63
White	8 (27)	24 (27)	
Black	9 (30)	27 (30)	
Other	6 (20)	26 (29)	
Not specified	7 (23)	13 (14)	
Ethnicity			.92
Not Hispanic or Latino origin	10 (33)	35 (39)	
Hispanic or Latino origin	6 (20)	22 (24)	
Unknown or not specified	10 (33)	29 (32)	
Missing	4 (13)	4 (4)	
Body mass index, kg/m^2	27.2 (24.3–31.5)	28.1 (24.1–32)	.79
Smoking status			.02
Never	15 (50)	66 (73)	
Active smoker	5 (17)	4 (4)	
Former smoker	10 (33)	20 (22)	
Hypertension	12 (40)	48 (53)	.29
Diabetes mellitus	8 (27)	30 (33)	.65
Coronary artery disease	2 (7)	12 (13)	.51
Heart failure			1.00
HFpEF	0 (0)	1 (1)	
HFrEF	1 (3)	5 (6)	
Stroke	0 (0)	5 (6)	.33
Chronic kidney disease[a]	0 (0)	3 (3)	.33
End-stage renal disease	2 (7)	5 (6)	1.00
Chronic obstructive pulmonary disease	4 (13)	3 (3)	.07
Asthma	3 (10)	8 (9)	1.00
Cirrhosis	1 (3)	1 (1)	.44
Chronic hepatitis B	6 (20)	1 (1)	<.001
Chronic hepatitis C	1 (3)	1 (1)	.44

Abbreviations: COVID-19, coronavirus disease 2019; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; IQR, interquartile range; PWH, people with HIV.

[a]Defined by baseline serum creatinine >2 mg/dL.

cells/μL. Seven patients had a CD4 count <200 cells/μL during hospitalization.

Symptoms

Presenting symptoms did not differ significantly between the 2 groups (Table 3). Similar to prior studies of COVID-19 patients, the most common symptoms across both groups were fever (57% in PWH and 73% in control patients), cough (70% and 76%), and dyspnea (67% and 64%). Sputum production was uncommon (3% in PWH, 11% in control patients). Diarrhea occurred in approximately one-third of patients in both groups.

Laboratory and Radiographic Findings

Initial absolute lymphocyte counts and peak values of procalcitonin, D-dimer, and lactate dehydrogenase did not differ significantly between groups (Table 3). Both groups were found to have absolute lymphopenia, elevated D-dimer, and elevated lactate dehydrogenase. Although the difference in CRP between groups was not statistically significant, the median peak value in the control group was notably higher than in the PWH group (16.0 mg/dL vs 7.6 mg/dL; $P = .07$). Initial chest x-ray patterns were not significantly different between PWH and control patients. Approximately three-quarters of patients in both groups had either unilateral or bilateral infiltrates on initial chest x-ray.

COVID-19 Treatments

There were no significant differences in COVID-19 treatments received between PWH and control patients, including hydroxychloroquine (67% vs 50%; $P = .14$), systemic corticosteroids (13% vs 21%; $P = .43$), and remdesivir (0% vs 3%; $P = .57$).

Clinical Outcomes

Frequencies of clinical outcomes are reported in Table 4. Upon arrival at the hospital, 50% of all patients were hypoxemic, and there was no significant difference between the PWH and control groups. However, PWH were significantly less likely than the control group to require supplemental oxygen via nonrebreather (3% vs 14%) and invasive mechanical ventilation

Table 2. Antiretroviral Regimens of Hospitalized COVID-19 Patients With HIV Co-infection

	PWH (n = 29)
Protease inhibitor–containing regimens	6
Darunavir and cobicistat, dolutegravir	2
Lopinavir and ritonavir, lamivudine, and zidovudine	1
Ritonavir, darunavir, raltegravir	1
Ritonavir, darunavir, lamivudine, dolutegravir	1
Ritonavir, atazanavir, emtricitabine, and tenofovir	1
Non–protease inhibitor–containing regimens	23
Bictegravir, emtricitabine, and tenofovir	8
Elvitegravir, cobicistat, emtricitabine, and tenofovir	4
Dolutegravir, lamivudine, abacavir	2
Dolutegravir and rilpivirine	2
Dolutegravir, emtricitabine, and tenofovir	2
Emtricitabine and tenofovir, raltegravir, etravirine	1
Emtricitabine and tenofovir, darunavir, cobicistat	1
Tenofovir, emtricitabine, rilpivirine	1
Tenofovir, lamivudine, raltegravir	1
Dolutegravir, entecavir, lamivudine, abacavir	1

All regimens were continued during hospitalization except for 2 patients: (1) bictegravir, emtricitabine, and tenofovir to dolutegravir, lamivudine, and tenofovir; (2) atazanavir, ritonavir, emtricitabine, and tenofovir to dolutegravir, emtricitabine, and tenofovir.

Abbreviations: COVID-19, coronavirus disease 2019; PWH, people with HIV.

Table 3. Clinical Manifestations of Hospitalized COVID-19 Patients With HIV Co-infection and Matched Controls

	PWH (n = 30), No. (%) or Median (IQR)	Control (n = 90), No. (%) or Median (IQR)	*P* Value
Presenting symptoms			
Time of illness onset (days from symptom onset to hospital presentation), median (IQR)	7 (3–10)	7 (4–10)	.89
Fever	17 (57)	66 (73)	.11
Cough	21 (70)	68 (76)	.63
Sputum production	1 (3)	10 (11)	.29
Dyspnea	20 (67)	58 (64)	1.00
Sore throat	0 (0)	11 (12)	.63
Rhinorrhea or nasal congestion	1 (3)	8 (9)	.45
Headache	3 (10)	6 (7)	.69
Myalgias	4 (13)	23 (26)	.21
Nausea or vomiting	5 (17)	18 (20)	.79
Diarrhea	10 (33)	24 (27)	.49
Abdominal pain	3 (10)	6 (7)	.69
Chest pain	3 (10)	11 (12)	1.00
Anosmia	1 (3)	5 (6)	1.00
Ageusia	2 (7)	4 (4)	.64
Laboratory markers[a]			
Absolute lymphocyte count, median (IQR), $\times 10^3/\mu L$	0.9 (0.6–1.5)	0.9 (0.6–1.1)	.23
Missing	0	3	
C-reactive protein, median (IQR), mg/dL	7.6 (2.8–16.5)	16.0 (6.8–25.0)	.07
Missing	7	26	
Procalcitonin, median (IQR), ng/mL	0.16 (0.06–0.3)	0.24 (0.1–0.67)	.18
Missing	7	17	
D-dimer, median (IQR), ng/mL	1021 (427–3145)	954 (340–4455)	.96
Missing	11	32	
Lactate dehydrogenase, median (IQR), U/L	410 (283.5–535)	453 (366–587)	.15
Missing	6	16	
CD4 count, median (IQR), cells/μL	332 (123–526)	N/A	
CD4:CD8 ratio, median (IQR)	0.7 (0.3–1.0)	N/A	
HIV viral load, median (IQR), copies/mL	0 (0–0)	N/A	
Radiographic findings			
Initial chest x-ray			.63
No infiltrates	9 (30)	22 (24)	
Unilateral or bilateral infiltrates	21 (70)	68 (76)	

Abbreviations: COVID-19, coronavirus disease 2019; IQR, interquartile range; PWH, people with HIV.

[a]Absolute lymphocyte counts were collected by the initial value upon hospitalization. C-reactive protein, procalcitonin, D-dimer, and lactate dehydrogenase were collected as peak values of all available values within 7 days of hospitalization.

(0% vs 6%; $P = .04$) upon hospital arrival. There was no significant difference between intensive care unit admission, requirement of vasopressor support, initiation of dialysis, or death during hospitalization between the 2 groups. There were no differences in documented code status between groups. Most patients in both cohorts were full code (96% in PWH vs 78% in controls).

DISCUSSION

Our study is one of the largest reported observational cohort studies of hospitalized COVID-19 patients comparing the clinical manifestations and clinical outcomes of patients with HIV infection and matched controls. We found that there were significantly more active and former smokers, more patients with chronic obstructive pulmonary disease, and a significantly higher proportion of chronic hepatitis B viral infection in PWH compared with matched controls. These data are consistent with existing data on higher rates of smoking and HBV infection in HIV-positive individuals compared with the general population [23, 24]. We did not find significant differences between the PWH and control groups in presenting symptoms, laboratory parameters, radiographic findings, or clinical outcomes including death, need for invasive mechanical ventilation, length of stay, or frequency of hospital discharge.

Our study found that PWH co-infected with COVID-19 share similar laboratory derangements as the general COVID-19

Table 4. Clinical Outcomes of Hospitalized COVID-19 Patients With HIV Co-infection and Matched Controls

	PWH (n = 30), No. (%) or Median (IQR)	Control (n = 90), No. (%) or Median (IQR)	*P* Value
Hypoxemia upon hospital arrival	15 (50)	45 (50)	1.00
Highest level of supplemental oxygen required (within first 3 h of arrival)			.04
Room air	15 (50)	45 (50)	
Nasal cannula	13 (43)	27 (30)	
Nonrebreather	1 (3)	13 (14)	
Noninvasive ventilation[a]	1 (3)	0 (0)	
Invasive mechanical ventilation	0 (0)	5 (6)	
Need for invasive mechanical ventilation during hospitalization	4 (13)	18 (20)	.59
Need for vasopressors	4 (13)	18 (20)	.59
New initiation of dialysis	0 (0)	5 (6)	.33
Intensive care unit admission	4 (13)	21 (23)	.31
Hospital length of stay	6 (3–9)	5 (2–9.5)	.46
Hospital discharge[b]	24 (73)	64 (71)	.26
Death	2 (7)	14 (16)	.35

Abbreviations: COVID-19, coronavirus disease 2019; IQR, interquartile range; PWH, people with HIV.

[a]Bilevel or continuous positive airway pressure.

[b]Patients who neither were discharged nor died were either still hospitalized (n = 9) or transferred to another acute care hospital (n = 7) at the time of analysis.

population, including lymphopenia and elevated inflammatory markers [25, 26]. Peak CRP levels were lower in PWH compared with controls, which could suggest that PWH, given their relative state of immunodeficiency, do not mount as strong of an immune response to SARS-CoV-2.

Notably, we found that PWH were significantly less likely than control patients to require nonrebreather and invasive mechanical ventilation. This is despite no differences in initial chest x-ray findings, presenting symptoms, time of illness onset, prognostic markers such as lymphopenia or elevated D-dimer, or code status. Other case reports and studies have found similarly low morbidity in patients with HIV and SARS-CoV-2 co-infection [27, 28]. In our study, the PWH group had very well-controlled disease. One possible explanation could be that PWH are more likely to be hospitalized with less severe disease than patients without HIV due to concern from admitting physicians about underlying immunosuppression. Another possible contributing factor could be that PWH have an increased connection with our health care system given the need for primary care or infectious diseases specialists to be intimately involved in their care and disease management, and therefore earlier recognition and triage of symptoms.

It is also possible that PWH had lower rates of severe respiratory failure because many were already on antiretroviral therapy, which could have a protective effect against SARS-CoV-2 infection [29–31]. One-quarter of PWH were on protease inhibitors, including lopinavir/ritonavir, which has been shown to have activity against SARS-CoV-2 in vitro [32]. Although the pharmacodynamics of lopinavir/ritonavir are challenging and a randomized controlled trial of 2 protease inhibitors, lopinavir and ritonavir, did not show benefit beyond standard of care, 19 of 29 (66%) PWH in our study were taking tenofovir (either a disoproxil fumarate or alafenamide formulation), which has demonstrated in vitro activity against SARS-CoV-2 [33–36].

Lastly, it is possible that the relative immune-altered status in HIV infection may allow for less severe forms of COVID-19 and potentially favorable recovery outcomes. Persons with HIV who are well controlled on antiretroviral therapy have heightened levels of T-cell exhaustion, persistent immune activation with inversion of the CD4/CD8 ratio, and impaired immune responses that either collectively or independently may impact COVID-19 pathogenesis. There is emerging evidence that some patients with severe manifestations of COVID-19 develop a hyperactive immune response similar to cytokine release syndrome [37]. This phenotype is characterized by high fevers, elevated inflammatory markers, multi-organ failure, and high mortality rates. In particular, the hyperactivity of T cells may play a role in SARS-CoV-2-mediated lung injury. In peripheral blood flow cytometry analysis of postmortem tissue sampling of a patient who died from severe SARS-CoV-2 infection, there was an increased concentration of proinflammatory CCR6+ Th17 in CD4 T cells, as well as CD8 T cells with cytotoxic granules, suggesting that hyperactive and inflammatory T cells played a key role in lung injury [38].

Our study has limitations. First, our study has a relatively small sample size. Important differences between PWH and control patients might be detected with a larger study. Second,

the majority of PWH in our cohort were on antiretroviral therapy and were virally suppressed, which may limit our ability to comment on outcomes of COVID-19 in PWH who have uncontrolled HIV or advanced disease.

In conclusion, the clinical manifestations and outcomes of COVID-19 among patients with SARS-CoV-2 and HIV co-infection were not significantly different than patients without HIV co-infection. However, more PWH were current and former smokers and were admitted to the hospital with less severe hypoxemia. Levels of CRP among PWH compared with matched controls may indicate that relative immune dysfunction plays a protective role in COVID-19. Future studies should evaluate the role of the host immune system, antiretroviral agents, and immunomodulating agents in the setting of co-infection with SARS-CoV-2 and HIV.

Acknowledgments

This work was made possible through data provided by the Cornell COVID 19 Registry, led by Parag Goyal, MD, Justin Choi, MD, Laura Pinheiro, PhD, and Monika Safford, MD, of Weill Cornell Medicine. We would like to acknowledge Arthur Evans, MD, and Lishomwa Ndhlovu, MD, PhD, for their valuable feedback and review of this manuscript. We also acknowledge the commitment, resilience, and sacrifice of all frontline health care workers and our patients during this pandemic.

Financial support. This work was supported by the National Institutes of Health/National Center for Advancing Translational Sciences (grant numbers UL1TR00047, KL2-TR-002385 to J.J.C.) and the National Institute of Allergy and Infectious Diseases (grant number T32 AI007613 to C.D.J.). S.C.W. was supported by a Medical Scientist Training Program grant from the National Institute of General Medical Sciences of the National Institutes of Health (grant number T32GM007739 to the Weill Cornell/Rockefeller/Sloan Kettering Tri-Institutional MD-PhD Program).

Potential conflicts of interest. J.J.C. received research support from Roche Diagnostics and consulting fees from Allergan. M.J.G. received research support to Weill Cornell Medicine from Gilead Sciences and Regeneron and royalties from Springer and UpToDate. None of these activities relate to the current work. All other authors declare no competing interests. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

Author contributions. K.S. and C.D.J. contributed equally to drafting the manuscript. K.S., S.C.W., and J.J.C. collected the data. D.P.J. did the statistical analysis. T.M.E, M.A.V., and R.M.G. made substantial contributions to the interpretation of the data. C.D.J., M.J.G., and J.J.C. contributed equally to conceiving the study and supervising the data collection and analysis. All coauthors provided critical revisions to the intellectual content of the manuscript and final approval of the version to be published.

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