Clinical Infectious Diseases

MAJOR ARTICLE

Coronavirus 2019 and People Living With Human Immunodeficiency Virus: Outcomes for Hospitalized Patients in New York City

Keith Sigel,[1] Talia Swartz,[2] Eddye Golden,[3] Ishan Paranjpe,[3,4] Sulaiman Somani,[3,4] Felix Richter,[3,4] Jessica K. De Freitas,[3,4] Riccardo Miotto,[3,4] Shan Zhao,[3,5] Paz Polak,[6] Tinaye Mutetwa,[1] Stephanie Factor,[2] Saurabh Mehandru,[7] Michael Mullen,[2] Francesca Cossarini,[2] Erwin Bottinger,[3,8] Zahi Fayad,[9,10] Miriam Merad,[6,11,12] Sacha Gnjatic,[6,11,12] Judith Aberg,[2] Alexander Charney,[4,13,14] Girish Nadkarni,[3,11,15,a] and Benjamin S. Glicksberg[3,4,a]

[1]Division of General Internal Medicine, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [2]Division of Infectious Diseases, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [3]Hasso Plattner Institute for Digital Health at Mount Sinai, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [4]Department of Genetics and Genomic Sciences, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [5]Department of Anaesthesia, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [6]Department of Oncologic Sciences, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [7]Division of Gastroenterology, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [8]Digital Health Center, Hasso Plattner Institute, University of Potsdam, Professor-Dr.-Helmert-Strasse 2–3, Potsdam, Germany, [9]BioMedical Engineering and Imaging Institute, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [10]Department of Radiology, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [11]Department of Medicine, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [12]Precision Immunology Institute, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [13]Pamela Sklar Division of Psychiatric Genomics, Icahn School of Medicine at Mount Sinai, New York, New York, USA, [14]Department of Psychiatry, Icahn School of Medicine at Mount Sinai, New York, New York, USA, and [15]Charles Bronfman Institute for Personalized Medicine, Icahn School of Medicine at Mount Sinai, New York, New York, USA

Background. There are limited data regarding the clinical impact of coronavirus disease 2019 (COVID-19) on people living with human immunodeficiency virus (PLWH). In this study, we compared outcomes for PLWH with COVID-19 to a matched comparison group.

Methods. We identified 88 PLWH hospitalized with laboratory-confirmed COVID-19 in our hospital system in New York City between 12 March and 23 April 2020. We collected data on baseline clinical characteristics, laboratory values, HIV status, treatment, and outcomes from this group and matched comparators (1 PLWH to up to 5 patients by age, sex, race/ethnicity, and calendar week of infection). We compared clinical characteristics and outcomes (death, mechanical ventilation, hospital discharge) for these groups, as well as cumulative incidence of death by HIV status.

Results. Patients did not differ significantly by HIV status by age, sex, or race/ethnicity due to the matching algorithm. PLWH hospitalized with COVID-19 had high proportions of HIV virologic control on antiretroviral therapy. PLWH had greater proportions of smoking ($P < .001$) and comorbid illness than uninfected comparators. There was no difference in COVID-19 severity on admission by HIV status ($P = .15$). Poor outcomes for hospitalized PLWH were frequent but similar to proportions in comparators; 18% required mechanical ventilation and 21% died during follow-up (compared with 23% and 20%, respectively). There was similar cumulative incidence of death over time by HIV status ($P = .94$).

Conclusions. We found no differences in adverse outcomes associated with HIV infection for hospitalized COVID-19 patients compared with a demographically similar patient group.

Keywords. human immunodeficiency virus; coronavirus 2019; severe acute respiratory syndrome coronavirus 2.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the causative agent of coronavirus disease-19 (COVID-19), has infected millions of people worldwide since its identification in December 2019, resulting in more than 400 000 deaths to date. [1] Patients who are immunocompromised have overall poorer outcomes from most serious infections, but this may vary based on the type of immune deficiency and the specific pathogens. Living with human immunodeficiency virus (HIV) is one of the most common causes of immunocompromise globally, with more than 1 million people living with HIV (PLWH) in the United States alone [2].

Despite the substantial reach of the COVID-19 pandemic in areas with high HIV prevalence, there are very limited data regarding the clinical impact of SARS-CoV-2 infection on PLWH. Although large proportions of PLWH have achieved HIV virologic suppression in the current era of widespread antiretroviral use, mild immunodeficiency and chronic immune activation have been widely recognized as drivers of HIV complications [3]. COVID-19 also leads to marked immune dysregulation, with severe cases manifesting a dramatic hyperinflammatory syndrome that causes respiratory failure and multiorgan dysfunction [4]. Lack of knowledge regarding the interaction of HIV- and COVID-19–related immune

Received 15 May 2020; editorial decision 18 June 2020; accepted 22 June 2020; published online June 28, 2020.

[a]G. N. and B. S. G. contributed equally to this work.

Correspondence: K. Sigel, Mount Sinai School of Medicine, One Gustave Levy Place, Box 1087, New York, NY 10029 (Keith.Sigel@mssm.edu).

Clinical Infectious Diseases® 2020;71(11):2933–8

DOI: 10.1093/cid/ciaa880

dysfunction have led to uncertainty regarding appropriate clinical and policy guidance for the management and counseling of PLWH during the SARS-CoV-2 pandemic.

In this study, we assessed the outcomes of COVID-19 in PLWH, comparing a hospitalized cohort of PLWH from our New York City health system and a demographically matched group of comparator patients. We evaluated factors associated with mortality for PLWH hospitalized with COVID-19.

METHODS

Study Cohort

We identified all hospitalized patients with laboratory-confirmed SARS-CoV-2 infection at 5 hospitals in the Mount Sinai Health System admitted between 12 March 2020 and 23 April 2020. From this cohort, we identified 88 patients with HIV-related diagnostic codes or those being treated with antiretroviral medications identified from inpatient or outpatient medication orders. We selected an HIV-uninfected comparator group (from the larger laboratory-confirmed SARS-CoV-2 cohort) using a methodology similar to that used for the Veterans Aging Cohort Study for comparisons by HIV status [5].We identified 405 patients not living with HIV, matching 1 PLWH to up to 5 patients by age (±2.5 years), sex, race/ethnicity, and calendar week of infection (to account for temporally associated changes in COVID-19 management and differences in follow-up time).

Variables

Electronic health record (EHR) data, including demographics, all diagnostic codes and procedures, as well as clinical laboratory measurements and outcomes (death, mechanical ventilation, discharge), were collected. Smoking status was ascertained from a structured EHR element. Oxygen supplementation requirements on admission were collected, verified via chart review, and used to categorize COVID-19 severity using published criteria [6]. Laboratory values (white blood cell count, creatinine, alanine aminotransferase [ALT], ferritin, D-dimer, C-reactive protein [CRP], procalcitonin, lactate dehydrogenase, and interleukin [IL]-6) reflected the admission value, collected within 48 hours of hospital presentation. Comorbidities were defined using relevant diagnostic codes (*International Classification of Diseases, Tenth Edition*). The most recent value prior to hospital admission during the preceding 12 months was determined for prior CD4, CD4 percentage of lymphocytes, and HIV viral load measurements. Each of these biomarkers was also ascertained, when available, after the diagnosis of COVID-19. HIV status, HIV clinical variables, and antiretroviral medications were confirmed by chart review by 2 infectious diseases physicians (K. S. and T. S.). Adverse outcomes included death and mechanical ventilation, which were identified from electronic records and confirmed by chart review.

Data Analyses

We compared baseline characteristics, treatments, and outcomes, testing to assess significant differences between PLWH and comparators using the Wilcoxon test for continuous variables and the χ^2 test for categorical variables. We also stratified baseline laboratory measures by COVID-19 severity for PLWH, testing for differences in measures by severity category using ordinal logistic regression. We compared differences in time to death to assess disease trajectory for hospitalized patients by HIV status by fitting unadjusted cumulative incidence function curves with hospital discharge as a competing risk. Curves were compared using the test of Pepe and Mori [7]. To compare cumulative incidence of death by HIV status accounting for potential confounding factors, we fit a multivariable survival model using Fine-Grey competing risk methods, including demographics, COVID-19 severity, comorbid conditions, and laboratory values that differed by HIV status [8].

Finally, we compared characteristics of PLWH who died during hospitalization to those who were discharged or still alive at the end of follow-up. We explored the independent association of significant factors associated with death for PLWH who died from COVID-19 by fitting separate multivariable competing risk models using demographic factors and significant univariate predictors (1 model for each predictor). Based on our sample size, our primary analysis of proportion of hospital deaths by HIV status had 80% power to detect a 15% increase in the absolute risk of death for PLWH compared with uninfected persons. All analyses were conducted using Stata version 15. Our institutional review board approved the study .

RESULTS

Of the 4402 patients hospitalized for COVID-19 during the study period, 88 (2%) were PLWH. The median age of PLWH hospitalized with COVID-19 was 61 years (Table 1; interquartile range [IQR], 54–67), and most PLWH were black (40%) or Hispanic/Latino (30%). Patients did not differ significantly by HIV status when age, sex, or race/ethnicity were compared due to the matching algorithm. Consistent with trends noted in HIV cohort studies [9], PLWH also had greater proportions of smoking (55% vs 23%; $P < .001$) and comorbid illnesses than demographically similar uninfected comparators, most notably chronic obstructive pulmonary disease (COPD; 10% vs 3%; $P < .001$), cirrhosis (6% vs 2%; $P = .02$), and a history of cancer diagnosis (17% vs 6%; $P = .001$). PLWH and uninfected persons had similar COVID-19 severity on admission as measured by oxygen supplementation requirements ($P = .15$).

All PLWH admitted for COVID-19 were prescribed antiretroviral therapy, and most (78%) were receiving integrase inhibitor–based regimens. The majority (58%) of patients with CD4 measurements on hospital admission had counts >200 cells/mm^3. However, among the 26 patients from this group

Table 1. Clinical Characteristics, Laboratory Measures, and Treatments for People Living With Human Immunodeficiency Virus and Matched Comparators Hospitalized in New York City With Coronavirus 2019

Characteristic	Patients Living With HIV (n = 88)	People Not Living With HIV (n = 405)	*P* Value
Demographics			
Age, median (IQR), y	61 (54–67)	60 (55–67)	.71
Female, no. (%)	22 (25)	97 (24)	.84
Race/Ethnicity, no. (%)			.99
White	17 (19)	76 (19)	
Black	35 (40)	170 (42)	
Hispanic	26 (30)	115 (28)	
Other	10 (11)	44 (11)	
Comorbidities, no. (%)			
Diabetes	24 (27)	117 (29)	.76
Hypertension	33 (38)	135 (33)	.46
Obesity	9 (10)	34 (8)	.58
Chronic obstructive pulmonary disease	8 (9)	7 (2)	<.001
Cirrhosis	5 (6)	6 (2)	.02
Coronary artery disease	6 (7)	46 (11)	.21
Chronic kidney disease	19 (22)	56 (14)	.06
Organ transplant	4 (5)	7 (2)	.11
Cancer	15 (17)	26 (6)	.001
Current or former smoker	48 (55)	93 (23)	<.001
HIV clinical characteristics, no. (%)			
ART	88 (100)		
ART class			
Integrase	69 (78)		
Protease inhibitor	15 (17)		
Nonnucleoside reverse transcriptase inhibitors	8 (9)		
Nucleoside reverse transcriptase inhibitors	85 (97)		
CD4 cell count, admission (n = 57), cells/mm^3			
<50	5 (9)		
50–200	19 (33)		
201–500	26 (46)		
>500	7 (12)		
CD4 cell count, prior to admission (n = 46), cells/mm^3			
<50	1 (2)		
50–200	6 (13)		
200–500	19 (41)		
>500	20 (44)		
HIV RNA level, admission or within previous 12 months (n = 82), copies/uL[a]			
<50	66 (81)		
>50	16 (19)		
Coronavirus 2019 severity on admission based on supplemental oxygen requirement			.15
Mild (no supplemental oxygen)	16 (18)	88 (22)	
Moderate (nasal cannula or venturi mask)	39 (44)	129 (32)	
Moderate/Severe (non-rebreather mask, high-flow nasal cannula)	15 (17)	98 (24)	
Severe (noninvasive ventilation or mechanical ventilation)	18 (21)	88 (22)	
Laboratory values, median (IQR)			
White blood cell count, k/μL[b]	7.2 (4.9–8.8)	7.9 (5.6–10.7)	.02
Creatinine, mg/dL[b]	1.2 (0.83–2.1)	1.1 (0.85–1.98)	.66
Ferritin, ng/mL[c]	692 (285–1334)	984 (469–2432)	.002
D-dimer, μg/mL[d]	1.98 (0.89–4.91)	1.86 (0.92–4.11)	.80
C-reactive protein, mg/dL[e]	119 (81–192)	135 (72–234)	.30
Procalcitonin, ng/mL[f]	0.21 (0.09–0.77)	0.28 (0.10–0.75)	.54
Lactate dehydrogenase, U/L[g]	428 (312–595)	448 (345–615)	.21
Interleukin-6, pg/mL[h]	64.1 (31.9–117)	68.1 (35.5–149)	.57
Alanine aminotransferase, U/L[i]	32 (21–49)	33 (21–58)	.70
Treatments, no. (%)			
Hydroxychloroquine	67 (76)	328 (81)	.30
Azithromycin	66 (75)	301 (74)	.90
Tocilizumab	3 (3))	29 (7)	.20
Experimental and expanded-access agents[j]	4 (5)	13 (3)	.53

Abbreviations: ART, antiretroviral therapy; HIV, human immunodeficiency virus; IQR, interquartile range.

[a] Admission HIV RNA values available for 55 patients; values from previous 12 months used for 27 patients.

[b] No missing values in people living with HIV (PLWH).

[c] Values available for PLWH: 77/88, people not living with HIV: 347/405.

[d] Values available for PLWH: 43/88, people not living with HIV: 207/405.

[e] Values available for PLWH: 77/88, people not living with HIV: 354/405.

[f] Values available for PLWH: 79/88, people not living with HIV: 335/405.

[g] Values available for PLWH: 79/88, people not living with HIV: 343/405.

[h] Values available for PLWH: 54/88, people not living with HIV: 246/405.

[i] Values available for PLWH: 85/88, people not living with HIV: 400/405.

[j] Experimental and expanded-access agents included trial or off-label use of remdesivir, sarilumab, and/or anakinra.

with available prior CD4 percentage measurements (within 12 months), 73% had lower CD4 percentage levels on admission (median decline, 4%; IQR, 0%–9%). Among PLWH with HIV viral RNA testing on admission or in the 12 months prior, proportions of viral suppression were high (81%).

Inflammatory markers, including ferritin, CRP, and D-dimer, were measured for most hospitalized PLWH. Laboratory measures on admission were similar for PLWH and uninfected patients except for lower ferritin (P = .002) and white blood cell counts (P = .02) for PLWH. Higher CRP values were present in PLWH with more severe COVID-19 on admission (Supplementary Table 2; P = .005). The majority of PLWH were treated with hydroxychloroquine and azithromycin; experimental or expanded-access agents were used less frequently.

Most hospitalized PLWH were discharged from the hospital during the follow-up period (Figure 1). There was no significant difference in intensive care use by HIV status in our cohort. Poor outcomes for hospitalized PLWH were still frequent but similar to proportions in comparators; 18% required mechanical ventilation and 21% died during follow-up compared with 23% and 20%, respectively. Competing risks analysis showed similar cumulative incidence of death over time by HIV status with no significant difference in the curves (P = .94; Figure 2).

HIV was not significantly associated with risk of death in multivariable competing risks analysis after adjusting for demographics, COPD, smoking, baseline ferritin level, and baseline white blood cell count (Supplementary Table 3).

Comparisons of PLWH who died with those who were still alive or discharged at the end of follow-up revealed few differences in patients who died compared with those who did not. However, we did find differences in the proportions who were organ transplant recipients who died vs those who did not die (Table 2; 17% vs 1%; $P = .006$). In univariate comparisons, PLWH who died were less likely to have been treated with nucleoside reverse transcriptase inhibitors (NRTIs) than those who did not die during hospitalization (89% vs 99%; $P = .04$). PLWH with higher median CRP and procalcitonin levels on admission were also more likely to die during admission (both $P < .008$). In separate models adjusting for age, sex, and race/ethnicity, significance persisted for organ transplant recipient status (subhazard ratio for death [SHR], 3.85; 95% confidence interval [CI], 1.87–7.94) and NRTI use (SHR, 0.31; 05% CI, .12–.80) as predictors of death for PLWH.

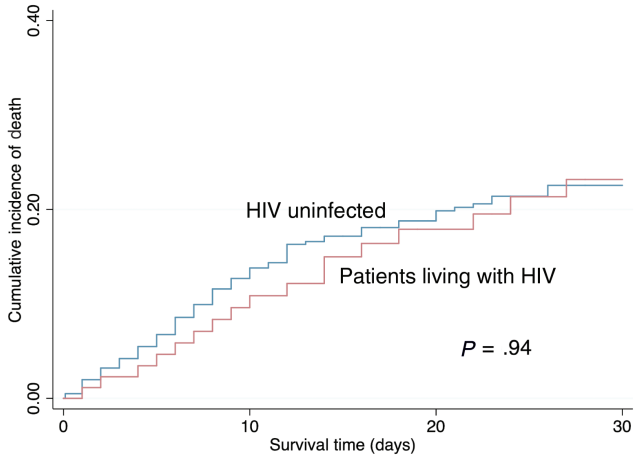

Figure 2. Cumulative incidence of death comparing people living with HIV to matched uninfected comparators during the coronavirus 2019 pandemic, March 2020–April 2020, in New York City. Abbreviation: HIV, human immunodeficiency virus.

DISCUSSION

Among patients from 5 hospitals in a large health system during the peak of the spring 2020 New York City SARS-CoV-2 epidemic, we found that PLWH hospitalized with COVID-19 had a substantial burden of comorbid illness and smoking but had good HIV disease control. Although mortality and other adverse outcomes were high among PLWH in our cohort, these were not worse than a demographically and temporally matched group with similar COVID-19 severity at presentation and fewer comorbidities. Our study represents the largest and most diverse cohort of PLWH with COVID-19 that has been described to date and adds to existing limited evidence that HIV might not be associated with a more severe COVID-19 course.

Concern that COVID-19 might be more severe in persons with immunodeficiency or immune dysregulation has been raised since the emergence of the earliest cases [10]. Uncontrolled series of immunosuppressed persons, such as kidney transplant recipients and cancer patients, have shown high mortality rates [11]. However, outcomes analyses for PLWH during the COVID-19 pandemic have been limited, consisting of small case reports or series [12]. A single center in Spain reported outcomes for 5 males living with HIV hospitalized with COVID-19. Four of the 5 men had been discharged, and the fifth was in intensive care at the time of their report [13]. Larger uncontrolled series from New Jersey (13 hospitalized patients), New York City (31 hospitalized patients), and Madrid, Spain (28 hospitalized patients) also subsequently demonstrated COVID-19 outcomes for PLWH similar to those described for the general population [14–16]. This is broadly

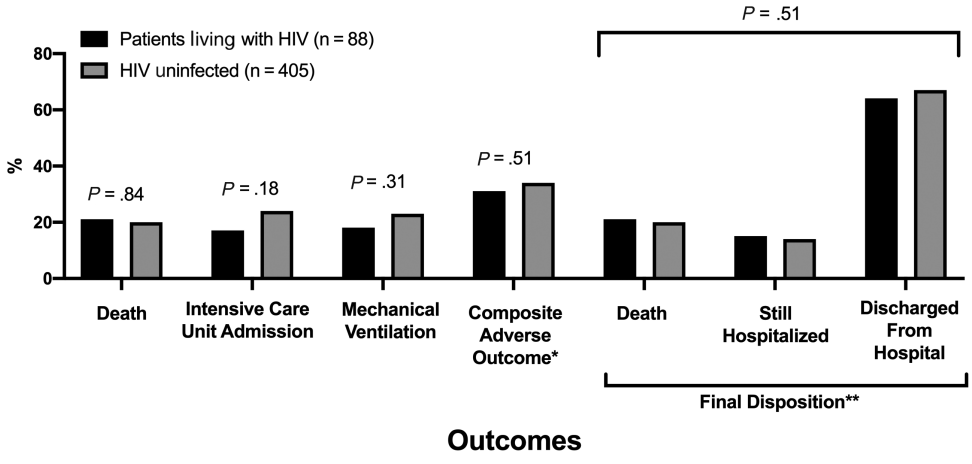

Figure 1. Major outcomes for people living with HIV and matched comparators hospitalized in New York City with coronavirus 2019. (*Composite outcome including death, intensive care unit admission, or mechanical ventilation. **Mutually exclusive categorical outcomes). Abbreviation: HIV, human immunodeficiency virus.

Table 2. Characteristics of People Living With Human Immunodeficiency Virus With Coronavirus 2019 Who Died During Follow-up vs Patients Alive or Discharged

Characteristic	Patients Living With HIV Who Died (n = 18)	Patients Living With HIV Alive (n = 70)	*P* Value
Demographics			
Age, median (IQR), y	62 (57–67)	58.5 (53–67)	.30
Female, no. (%)	5 (28)	17 (24)	.76
Race/Ethnicity, no. (%)			.13
White	5 (28)	12 (17)	
Black	3 (17)	32 (46)	
Hispanic	8 (44)	18 (26)	
Other	2 (11)	8 (11)	
Comorbidities, no. (%)			
Diabetes	4 (22)	20 (29)	.60
Hypertension	6 (33)	27 (39)	.70
Obesity	1 (6)	8 (11)	.46
Chronic obstructive pulmonary disease	2 (11)	6 (9)	.74
Cirrhosis	1 (6)	4 (6)	.98
Coronary artery disease	0 (0)	6 (9)	.20
Chronic kidney disease	6 (33)	13 (19)	.18
Organ transplant	3 (17)	1 (1)	.006
Cancer	1 (6)	14 (20)	.15
Current or former smoker	11 (61)	37 (53)	.53
HIV clinical characteristics, no. (%)			
ART	18 (100)	70 (100)	-
ART class			
Integrase	13 (72)	56 (80)	.47
Protease inhibitor	5 (28)	10 (14)	.18
Nonnucleoside reverse transcriptase inhibitors	0 (0)	8 (11)	.13
Nucleoside reverse transcriptase inhibitors	16 (89)	69 (99)	.04
CD4 cell count, admission (n = 57), cells/mm^3			.40
<50	1 (8)	4 (9)	
50–200	6 (46)	13 (30)	
201–500	6 (46)	20 (45)	
>500	0 (0)	7 (16)	
CD4 cell count, prior to admission (n = 46), cells/mm^3			.15
<50	0 (0)	1 (3)	
50–200	3 (38)	3 (8)	
201–500	2 (24)	17 (45)	
>500	3 (38)	17 (44)	
HIV RNA level, admission (n = 55), copies/μL			.67
<50	12 (92)	37 (88)	
>50	1 (8)	55 (12)	
HIV RNA level, prior to admission (n = 63), copies/μL			.36
<50	10 (91)	41 (79)	
>50	1 (9)	11 (21)	
Laboratory values, median (IQR)			
White-cell count, cells/L[a]	6.7 (5.3–11.5)	7.3 (4.9–8.5)	.35
Ferritin, ng/mL[b]	911 (312–2651)	682 (267–1288)	.23
D-dimer, μg/mL[c]	1.9 (0.91–5.7)	2.1 (0.89–4.9)	.95
C-reactive protein, mg/dL[d]	209 (140–249)	101 (73–173)	.002
Procalcitonin, ng/mL[e]	0.74 (0.21–3.2)	0.19 (0.10–0.62)	.008
Lactate dehydrogenase, U/L[f]	442 (319–634)	426 (297–560)	.50
Interleukin-6, pg/mL[g]	131 (64–305)	57 (27–98)	.004
Treatments, no. (%)			
Hydroxychloroquine	14 (78)	53 (76)	.86
Azithromycin	12 (67)	54 (77)	.36
Tocilizumab	1 (6)	2 (3)	.57
Experimental and expanded-access agents	2 (11)	2 (3)	.13

Abbreviations: ART, antiretroviral therapy; HIV, human immunodeficiency virus; IQR, interquartile range.

[a] No missing values in people living with HIV (PLWH).

[b] Values available for PLWH: 77/88.

[c] Values available for PLWH: 43/88.

[d] Values available for PLWH: 77/88.

[e] Values available for PLWH: 70/88.

[f] Values available for PLWH: 79/88.

[g] Values available for PLWH: 54/88.

consistent with our finding that respiratory failure that requires mechanical ventilation and death were not more frequent in PLWH when compared with demographically similar persons with COVID-19. The lack of outcome differences is even more striking when it is noted that COPD, cancer, and smoking, which were identified as risk factors linked to worse COVID-19 outcomes in several previous studies, were far more prevalent in PLWH in our cohort than in comparator patients [17, 18].

Immunomodulatory effects of SARS-CoV-2 have been associated with severe sequelae including a cytokine release syndrome [4]. Several factors have supported theoretical risks of worse COVID-19 in PLWH, including incomplete immune reconstitution and evidence of persistent immune activation in many patients prior to the pandemic [19, 20]. Furthermore, increased IL-6 and D-dimer measures have been independently associated with chronic HIV infection [21, 22] and have also been closely linked with COVID-19 severity in data mostly from persons not living with HIV [23, 24]. Neither biomarker differed on presentation for patients by HIV status nor was either associated with COVID-19 severity at presentation for PLWH. Among PLWH, CD4 decline was noted in the majority of patients with available data, consistent with existing immunologic data on COVID-19 natural history, but the decrease in CD4 percentage was not large [25]. We did not find evidence of associations between immunologic measures (either decreases from pre–COVID-19 values or low values at the time of presentation) and adverse COVID-19 outcomes for PLWH. However, organ transplantation was associated with death for PLWH in our study, suggesting that non-HIV causes of immunodeficiency may be more prominent risks for severe outcomes.

Antiretroviral medications have been evaluated for treatment of COVID-19 [26]. Several agents, including lopinavir and ritonavir, have demonstrated possible in vitro activity against SARS-CoV-2 [27, 28]. We found an adjusted association

between NRTI use and lower mortality, although our analyses were not subject to correction for multiple statistical tests and could have been confounded by other factors.

Our study benefited from data collected from diverse patient groups from 5 hospitals within a New York City large health system that is one of the largest HIV care providers in the United States. Our sample size of PLWH was limited, but we were nonetheless able to identify a large, well-matched comparison group to compare outcomes. To maximize our comparison group size, we limited our matching strategy to demographic and temporal factors using a method similar to that of the largest American HIV cohort study, although this yielded differences in comorbidity profiles for the 2 groups [5, 29]. This difference in comorbidity profiles may have also been influenced by more frequent medical care for PLWH, leading to an imbalance in the ascertainment of comorbid diagnoses. However, the marked difference in smoking history for PLWH vs persons not living with HIV supports likely differences in the true prevalence of these comorbid diseases by HIV status in our cohort. In addition, some measures, such as laboratory data, were missing for substantial portions of the sample, in proportions too large for imputation. Nonetheless, our key exposures and outcomes were manually verified and provide important information for this vulnerable population.

In conclusion, we found no differences in adverse outcomes associated with HIV infection for hospitalized COVID-19 patients compared with a similar comparison group. Verification of this finding in other large cohorts is warranted to improve our understanding of the impact of COVID-19 on PLWH. If confirmed, investigation of specific factors that contribute to similar outcomes in this large group of patients with immune disturbance may provide greater insight into the pathogenesis of SARS-CoV-2.

Supplementary Data

Supplementary materials are available at *Clinical Infectious Diseases* online. Consisting of data provided by the authors to benefit the reader, the posted materials are not copyedited and are the sole responsibility of the authors, so questions or comments should be addressed to the corresponding author.

Notes

Acknowledgments. The authors acknowledge Courtney Chan and Marina Makram for their assistance in this project.

Financial support. This work was supported by the National Center for Advancing Translational Sciences, National Institutes of Health (U54 TR001433-05).

Potential conflicts of interest. G. N. reports grants and personal fees from Renalytix AI; other payments from Pensieve Health; and personal fees from AstraZeneca, Reata, BioVie, and GLG Consulting. Z. F. reports grants from Daiichi Sankyo, Amgen, and Bristol Myers Squibb; personal fees from Alexion, GlaxoSmithKline, and Trained Therapeutix Discovery; and payments from Siemens Healthineers. Z. F. has a patent with Trained Therapeutix Discovery, licensed to Trained Therapeutix Discovery. S. J. reports grants and personal fees from Merck, Neon Therapeutics, OncoMed, Bristol-Myers Squibb, Genentech, Immune Design, Agenus, Janssen R&D, Pfizer, Takeda, and Regeneron. J. A. reports grants and personal fees from Gilead, Merck, ViiV, and Janssen; personal fees from Theratech and Medicure; and grants from Regeneron and Frontier Technology. M. M. reports grants from Regeneron, personal fees from Takeda, personal fees from Genentech, personal fees from Compugen, personal fees from Pionyr, and personal fees from Myeloid Therapeutics. All other authors report no potential conflicts. 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.

References

1. COVID-19 Projections. Available at: https://covid19.healthdata.org/united-states-of-america/new-york. Accessed 7 May 2020.
2. Giroir BP. The time is now to end the HIV epidemic. Am J Public Health **2020**; 110:22–4.
3. So-Armah KA, Tate JP, Chang CH, et al; VACS Project Team. Do biomarkers of inflammation, monocyte activation, and altered coagulation explain excess mortality between HIV infected and uninfected people? J Acquir Immune Defic Syndr **2016**; 72:206–13.
4. Tay MZ, Poh CM, Rénia L, MacAry PA, Ng LFP. The trinity of COVID-19: immunity, inflammation and intervention. Nat Rev Immunol **2020**; 20:363–74.
5. Justice AC, Dombrowski E, Conigliaro J, et al. Veterans Aging Cohort Study (VACS): overview and description. Med Care **2006**; 44:S13–24.
6. Messner CB, Demichev V, Wendisch D, et al. Ultra-high-throughput clinical proteomics reveals classifiers of COVID-19 infection. Cell Syst **2020**. In press.
7. Pepe MS, Mori M. Kaplan-Meier, marginal or conditional probability curves in summarizing competing risks failure time data? Stat Med **1993**; 12:737–51.
8. Bakoyannis G, Touloumi G. Practical methods for competing risks data: a review. Stat Methods Med Res **2012**; 21:257–72.
9. Mayer KH, Loo S, Crawford PM, et al. Excess clinical comorbidity among HIV-infected patients accessing primary care in US community health centers. Public Health Rep **2018**; 133:109–18.
10. Ballester-Arnal R, Gil-Llario MD. The virus that changed Spain: impact of COVID-19 on people with HIV. AIDS Behav **2020**. In press.
11. Akalin E, Azzi Y, Bartash R, et al. Covid-19 and kidney transplantation. N Engl J Med **2020**; 382:2475–7.
12. Zhu F, Cao Y, Xu S, Zhou M. Co-infection of SARS-CoV-2 and HIV in a patient in Wuhan City, China. J Med Virol **2020**.
13. Blanco JL, Ambrosioni J, Garcia F, et al; COVID-19 in HIV Investigators. COVID-19 in patients with HIV: clinical case series. Lancet HIV **2020**; 7:e314–6.
14. Okoh AK, Bishburg E, Grinberg S, Nagarakanti S. COVID-19 pneumonia in patients with HIV—a case series. J Acquir Immune Defic Syndr **2020**.
15. Shalev N, Scherer M, LaSota ED, et al. Clinical characteristics and outcomes in people living with HIV hospitalized for COVID-19. Clin Infect Dis **2020**. In press. ciaa635: 1–4.
16. Vizcarra P, Perez-Elias MJ, Quereda C, et al. Description of COVID-19 in HIV-infected individuals: a single-centre, prospective cohort. Lancet HIV **2020**; doi:10.1016/S2352-3018(20)30164-8
17. Alqahtani JS, Oyelade T, Aldhahir AM, et al. Prevalence, severity and mortality associated with COPD and smoking in patients with COVID-19: a rapid systematic review and meta-analysis. PLoS One **2020**; 15:e0233147.
18. Liang W, Guan W, Chen R, et al. Cancer patients in SARS-CoV-2 infection: a nationwide analysis in China. Lancet Oncol **2020**; 21:335–7.
19. Mehandru S, Poles MA, Tenner-Racz K, et al. Lack of mucosal immune reconstitution during prolonged treatment of acute and early HIV-1 infection. PLoS Med **2006**; 3:e484.
20. Paiardini M, Müller-Trutwin M. HIV-associated chronic immune activation. Immunol Rev **2013**; 254:78–101.
21. Armah KA, McGinnis K, Baker J, et al. HIV status, burden of comorbid disease, and biomarkers of inflammation, altered coagulation, and monocyte activation. Clin Infect Dis **2012**; 55:126–36.
22. Neuhaus J, Jacobs DR Jr, Baker JV, et al. Markers of inflammation, coagulation, and renal function are elevated in adults with HIV infection. J Infect Dis **2010**; 201:1788–95.
23. Li X, Xu S, Yu M, et al. Risk factors for severity and mortality in adult COVID-19 inpatients in Wuhan. J Allergy Clin Immunol **2020**; 146:110–8.
24. Zhou F, Yu T, Du R, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet **2020**; 395:1054–62.
25. Qin C, Zhou L, Hu Z, et al. Dysregulation of immune response in patients with COVID-19 in Wuhan, China. Clin Infect Dis **2020**. In press.
26. Cao B, Wang Y, Wen D, et al. A trial of lopinavir-ritonavir in adults hospitalized with severe covid-19. N Engl J Med **2020**; 382:1787–99.
27. Martinez MA. Compounds with therapeutic potential against novel respiratory 2019 coronavirus. Antimicrob Agents Chemother **2020**; 64:e00399–20.
28. Elfiky AA. Ribavirin, remdesivir, sofosbuvir, galidesivir, and tenofovir against SARS-CoV-2 RNA dependent RNA polymerase (RdRp): a molecular docking study. Life Sci **2020**; 253:117592.
29. Goulet JL, Fultz SL, Rimland D, et al. Aging and infectious diseases: do patterns of comorbidity vary by HIV status, age, and HIV severity? Clin Infect Dis **2007**; 45:1593–601.