



Covid-19 and People with HIV Infection: Outcomes for Hospitalized Patients in New York City

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Summary: There are limited data on COVID-19 clinical outcomes for people with HIV (PWH). We report outcomes for a cohort of hospitalized PWH with COVID-19 compared to demographically matched comparators. Major adverse outcomes were similar for PWH and matched comparators.

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Abstract

Background: There have been limited data regarding the clinical impact of COVID-19 disease on people with HIV (PWH). In this study we compared outcomes for PWH with COVID-19 disease to a matched comparison group.

Design: We identified 88 PWH hospitalized with laboratory confirmed COVID-19 in our hospital system in New York between March 12 and April 23, 2020. We collected data on baseline clinical characteristics, laboratory values, HIV infection status, COVID-19 treatment, and outcomes from this group and matched comparators (one PWH to up to five patients by age, sex, race/ethnicity and calendar week of infection). We compared baseline clinical characteristics and outcomes (death, mechanical ventilation, hospital discharge) for these two 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. PWH hospitalized with COVID-19 had high proportions of HIV virologic control on antiretroviral therapy. PWH had greater proportions of smoking ($p < 0.001$) and comorbid illness than demographically similar uninfected comparators. There was no difference in COVID-19 severity on admission by HIV status ($p = 0.15$). Poor outcomes for hospitalized PWH were frequent but similar to proportions in comparators; 18% required mechanical ventilation and ultimately 21% died during follow-up (compared with 23% and 20% respectively). There was similar cumulative incidence of death over time by HIV status ($p = 0.94$).

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

Keywords: Human Immunodeficiency Virus (HIV), Coronavirus-19 (COVID-19), SARS-CoV-2

Background

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 >400,000 deaths to-date.[1] Patients with immunocompromise have overall poorer outcomes from most serious infections, but this may vary based on the type of immune deficiency and the specific pathogens. HIV infection is one of the most common causes of immunocompromise globally, with over 1 million people with HIV (PWH) in the United States alone.[2]

Despite the substantial reach of the COVID-19 pandemic in areas with high HIV prevalence, there have been very limited data regarding the clinical impact of SARS-CoV-2 infection on people with HIV (PWH). Although large proportions of PWH 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 causing respiratory failure and multiorgan dysfunction.[4] Lack of knowledge regarding the interaction of HIV- and COVID-19-related immune dysfunction and have led to uncertainty regarding appropriate clinical and policy guidance for the management and counseling of PWH during the SARS-CoV-2 pandemic.

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

Methods

Study Cohort

We identified all hospitalized patients with laboratory confirmed SARS-CoV-2 infection at five hospitals in the Mount Sinai Health System admitted between March 12 and April 23, 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 then selected an HIV uninfected comparator group (from the larger laboratory confirmed SARS-CoV-2 cohort) using a similar methodology to that used by the Veterans Aging Cohort Study for comparisons by HIV status.[5] We identified 405 HIV uninfected patients, matching one PWH to up to five 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 were then collected including demographics, all diagnostic codes and procedures, as well as clinical laboratory measurements and outcomes (death, mechanical ventilation, discharge). 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 IL-6) reflected the admission value, collected within 48 hours of hospital presentation. Comorbidities were defined using relevant diagnostic codes (International Classification of Diseases 10th Edition). The most recent value prior to hospital admission during the preceding 12 months were determined for prior CD4, CD4 percentage of lymphocytes, and HIV viral load measurements and each of these biomarkers were also ascertained, when available, after the diagnosis of COVID-19 disease. HIV status, HIV clinical variables and antiretroviral medications were confirmed by chart review by two infectious diseases physicians (KS and TS). Adverse outcomes included death and mechanical ventilation, which were identified from electronic records and confirmed by chart review.

Data Analysis

We then compared baseline characteristics, treatments and outcomes, testing to assess significant differences between PWH and comparators using the Wilcoxon test for continuous variables and the chi-square test for categorical variables. We also stratified baseline laboratory measures by COVID-19 severity for PWH, testing for differences in measures by severity category using ordinal logistic regression. We then 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 then 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 PWH 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 PWH from COVID-19 by fitting separate multivariable competing risk models using demographic factors and significant univariate predictors (one 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 PWH compared to uninfected persons. All analyses were conducted using STATA Version 15. This study was approved by our Institutional Review Board.

Results

Of the 4,402 patients hospitalized during the study period for COVID-19, 88 (2%) were PWH. The median age of PWH hospitalized with COVID-19 disease was 61 (Table 1; intraquartile range [IQR] 54-67) and most PWH were black (40%) or Hispanic/Latino (30%). Patients did not differ significantly by HIV status when comparing age, sex, or race/ethnicity due to the matching algorithm. Consistent with trends noted in HIV cohort studies,[9] PWH also had greater proportions of smoking (55% versus 23%; $p<0.001$) and comorbid illnesses than demographically-similar uninfected comparators, most notably chronic obstructive pulmonary disease (COPD; 10% vs. 3%; $p<0.001$), cirrhosis (6% vs. 2%; $p=0.02$) and a history of cancer diagnosis (17% vs. 6%; $p=0.001$). PWH and uninfected persons had similar COVID-19 severity on admission as measured by oxygen supplementation requirements ($p=0.15$).

All PWH 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/mm³ although among the 26 patients from this group with available prior CD4 percentage measurements (within 12 months), 73% had lower CD4 percentage levels on admission (median decline 4% [IQR 0-9%]). Among PWH 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 PWH. Laboratory measures on admission were similar for PWH and uninfected patients except for lower ferritin ($p=0.002$) and white blood cell counts ($p=0.02$) for PWH. Higher CRP values were present in PWH with more severe COVID-19 on admission (Table S2; $p=0.005$). The majority of PWH were treated with hydroxychloroquine and azithromycin; experimental or expanded-access agents were used less frequently.

Most hospitalized PWH 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 PWH 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 ($p=0.94$; 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 (Table S3).

Comparisons of PWH who died to those who were still alive or discharged at the end of follow-up revealed few differences in patients who died compared to those who did not. We did find, however, differences in the proportions who were organ transplant recipients who died versus those who had not died (Table 2; 17% versus 1%; $p=0.006$). In univariate comparisons PWH who died were less likely to have been treated with nucleoside reverse transcriptase inhibitors (NRTIs) than those who did not die during hospitalization (89% versus 99%; $p=0.04$). PWH with higher median CRP and procalcitonin levels on admission were also more likely to die during admission (both $p<0.008$). In separate models adjusting for age, sex and race/ethnicity significance persisted for organ transplant recipient status (sub-hazard ratio for death [SHR]: 3.85; 95% confidence interval [CI]: 1.87-7.94) and NRTI use (SHR 0.31; 05% CI: 0.12-0.80) as predictors of death for PWH.

Discussion

Among patients from five hospitals in a large health system during the peak of the Spring 2020 NYC SARS-CoV-2 epidemic, we found that PWH 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 PWH 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 PWH with COVID-19 that has been described to date and adds to existing limited evidence that HIV might not be associated with more severe COVID-19 disease course.

Concern that COVID-19 disease 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] Outcomes analyses for PWH during the COVID-19 pandemic have been limited however, consisting of small case reports or series.[12] A single center in Spain reported outcomes for five male PWH hospitalized with COVID-19. Four of the five 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 PWH similar to those described for the general population.[14-16] This is broadly consistent with our finding that respiratory failure requiring mechanical ventilation and death were not more frequent in PWH when compared to demographically similar persons with COVID-19. The lack of outcome differences is even more striking when noting that COPD, cancer and smoking, risk factors linked to worse COVID-19 outcomes in several previous studies, were far more prevalent in PWH 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 disease in PWH 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 from mostly HIV uninfected persons.[23, 24] Neither biomarker differed on presentation for patients by HIV status nor was either associated with COVID-19 severity at presentation for PWH. Among PWH, 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 PWH. Organ transplantation was associated with death for PWH in our study, however, 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 infection.[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 five hospitals within a NYC large health system that is one of the largest HIV care providers in the United States. Our sample size of PWH was limited but we were nonetheless able to identify a large, well-matched comparison group for comparison of outcomes. To maximize our comparison group size, we limited our matching strategy to demographic and temporal factors using a similar method to the largest American HIV cohort study although this yielded differences in comorbidity profiles for the two groups.[5, 29] This difference in comorbidity profiles may have also been influenced by more frequent medical care for PWH, leading to an imbalance in the ascertainment of comorbid diagnoses. However, the marked difference in smoking history for PWH versus HIV uninfected persons 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 to a similar comparison group. Verification of this finding in other large cohorts is warranted to improve understanding of the impact of COVID-19 on PWH. If confirmed, investigation of specific factors contributing to similar outcomes in this large group of patients with immune disturbance may provide greater insight into the pathogenesis of SARS-CoV-2.

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Funding, Conflicts of Interest, and Acknowledgements

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Table 1. Clinical characteristics, laboratory measures and treatments for people with HIV (PWH) and matched comparators hospitalized in New York City with COVID-19 infection			
	Patients with HIV (N=88)	HIV Uninfected (N=405)	<i>p</i>
Demographics			
Age – median (IQR) ^a	61 (54-67)	60 (55-67)	0.71
Female – no. (%)	22 (25)	97 (24)	0.84
Race/Ethnicity – no. (%)			0.99
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)	0.76
Hypertension	33 (38)	135 (33)	0.46
Obesity	9 (10)	34 (8)	0.58
Chronic Obstructive Pulmonary Disease	8 (9)	7 (2)	<0.001
Cirrhosis	5 (6)	6 (2)	0.02
Coronary Artery Disease	6 (7)	46 (11)	0.21
Chronic Kidney Disease	19 (22)	56 (14)	0.06
Organ Transplant	4 (5)	7 (2)	0.11
Cancer	15 (17)	26 (6)	0.001
Current or Former Smoker	48 (55)	93 (23)	<0.001
HIV Clinical Characteristics – no. (%)			
Anti-retroviral therapy (ART)	88 (100)	--	
ART Class			
Integrase	69 (78)	--	

Protease Inhibitor	15 (17)	--	
Non-Nucleoside Reverse Transcriptase Inhibitors	8 (9)	--	
Nucleoside Reverse Transcriptase Inhibitors	85 (97)	--	
CD4 cell count, admission (N=57)			
<50 cells/mm ³	5 (9)	--	
50-200 cells/mm ³	19 (33)	--	
201-500 cells/mm ³	26 (46)	--	
>500 cells/mm ³	7 (12)	--	
CD4 cell count, prior to admission (N=46)			
<50 cells/mm ³	1 (2)	--	
50-200 cells/mm ³	6 (13)	--	
200-500 cells/mm ³	19 (41)	--	
>500 cells/mm ³	20 (44)	--	
HIV RNA level, admission or within previous 12 months (N=82) ^l			
<50 copies/ul	66 (81)	--	
>50 copies/ul	16 (19)	--	
COVID-19 Severity on Admission Based on Supplemental Oxygen Requirement			0.15
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 (Non-invasive Ventilation or Mechanical Ventilation)	18 (21)	88 (22)	
Laboratory Values – median (IQR)			
White blood cell count – k/ul ^b	7.2 (4.9-8.8)	7.9 (5.6-10.7)	0.02
Creatinine – mg/dl ^b	1.2 (0.83-2.1)	1.1 (0.85-1.98)	0.66
Ferritin – ng/ml ^c	692 (285-1,334)	984 (469-2,432)	0.002

D-dimer – ug/ml ^d	1.98 (0.89-4.91)	1.86 (0.92-4.11)	0.80
C-reactive protein – mg/dl ^e	119 (81-192)	135 (72-234)	0.30
Procalcitonin – ng/ml ^f	0.21 (0.09-0.77)	0.28 (0.10-0.75)	0.54
Lactate Dehydrogenase – U/L ^g	428 (312-595)	448 (345-615)	0.21
Interleukin-6 – pg/ml ^h	64.1 (31.9-117)	68.1 (35.5-149)	0.57
Alanine aminotransferase -- U/L ⁱ	32 (21-49)	33 (21-58)	0.70
Treatments – no. (%)			
Hydroxychloroquine	67 (76)	328 (81)	0.30
Azithromycin	66 (75)	301 (74)	0.90
Tocilizumab	3 (3)	29 (7)	0.20
Experimental and Expanded-Access Agents ^k	4 (5)	13 (3)	0.53

^a IQR denotes interquartile range

^b No missing values in PWH

^c Values available for PWH: 77/88, uninfected: 347/405

^d Values available for PWH: 43/88, uninfected; 207/405

^e Values available for PWH: 77/88, uninfected: 354/405

^f Values available for PWH: 79/88, uninfected: 335/405

^g Values available for PWH: 79/88, uninfected: 343/405

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Table 2. Characteristics of PWH with Covid-19 who died during follow-up versus patients alive or discharged			
	Patients with HIV who Died (N=18)	Patients with HIV Alive (N=70)	<i>p</i>
Demographics			
Age – median (IQR) ^a	62 (57-67)	58.5 (53-67)	0.30
Female – no. (%)	5 (28)	17 (24)	0.76
Race/Ethnicity – no. (%)			0.13
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)	0.60
Hypertension	6 (33)	27 (39)	0.70
Obesity	1 (6)	8 (11)	0.46
Chronic Obstructive Pulmonary Disease	2 (11)	6 (9)	0.74
Cirrhosis	1 (6)	4 (6)	0.98
Coronary Artery Disease	0 (0)	6 (9)	0.20
Chronic Kidney Disease	6 (33)	13 (19)	0.18
Organ Transplant	3 (17)	1 (1)	0.006
Cancer	1 (6)	14 (20)	0.15
Current or Former Smoker	11 (61)	37 (53)	0.53
HIV Clinical Characteristics – no. (%)			
Anti-retroviral therapy	18 (100)	70 (100)	-
ART Class			

Integrase	13 (72)	56 (80)	0.47
Protease Inhibitor	5 (28)	10 (14)	0.18
Non-Nucleoside Reverse Transcriptase Inhibitors	0 (0)	8 (11)	0.13
Nucleoside Reverse Transcriptase Inhibitors	16 (89)	69 (99)	0.04
CD4 cell count, admission (N=57)			0.40
<50 cells/mm ³	1 (8)	4 (9)	
50-200 cells/mm ³	6 (46)	13 (30)	
201-500 cells/mm ³	6 (46)	20 (45)	
>500 cells/mm ³	0 (0)	7 (16)	
CD4 cell count, prior to admission (N=46)			0.15
<50 cells/mm ³	0 (0)	1 (3)	
50-200 cells/mm ³	3 (38)	3 (8)	
201-500 cells/mm ³	2 (24)	17 (45)	
>500 cells/mm ³	3 (38)	17 (44)	
HIV RNA level, admission (N=55)			0.67
<50 copies/ul	12 (92)	37 (88)	
>50 copies/ul	1 (8)	55 (12)	
HIV RNA level, prior to admission (N=63)			0.36
<50 copies/ul	10 (91)	41 (79)	
>50 copies/ul	1 (9)	11 (21)	
Laboratory Values – median (IQR)			
White-cell count – k/ul ^b	6.7 (5.3-11.5)	7.3 (4.9-8.5)	0.35
Ferritin – ng/ml ^c	911 (312-2,651)	682 (267-1,288)	0.23
D-dimer – ug/ml ^d	1.9 (0.91-5.7)	2.1 (0.89-4.9)	0.95
C-reactive protein – mg/dl ^e	209 (140-249)	101 (73-173)	0.002
Procalcitonin – ng/ml ^f	0.74 (0.21-3.2)	0.19 (0.10-0.62)	0.008
Lactate Dehydrogenase – U/L ^g	442 (319-634)	426 (297-560)	0.50

Interleukin-6 – pg/ml ^h	131 (64-305)	57 (27-98)	0.004
Treatments – no. (%)			
Hydroxychloroquine	14 (78)	53 (76)	0.86
Azithromycin	12 (67)	54 (77)	0.36
Tocilizumab	1 (6)	2 (3)	0.57
Experimental and Expanded-Access Agents	2 (11)	2 (3)	0.13

^a IQR denotes interquartile range

^b No missing values in PWH

^c Values available for PWH: 77/88

^d Values available for PWH: 43/88

^e Values available for PWH: 77/88

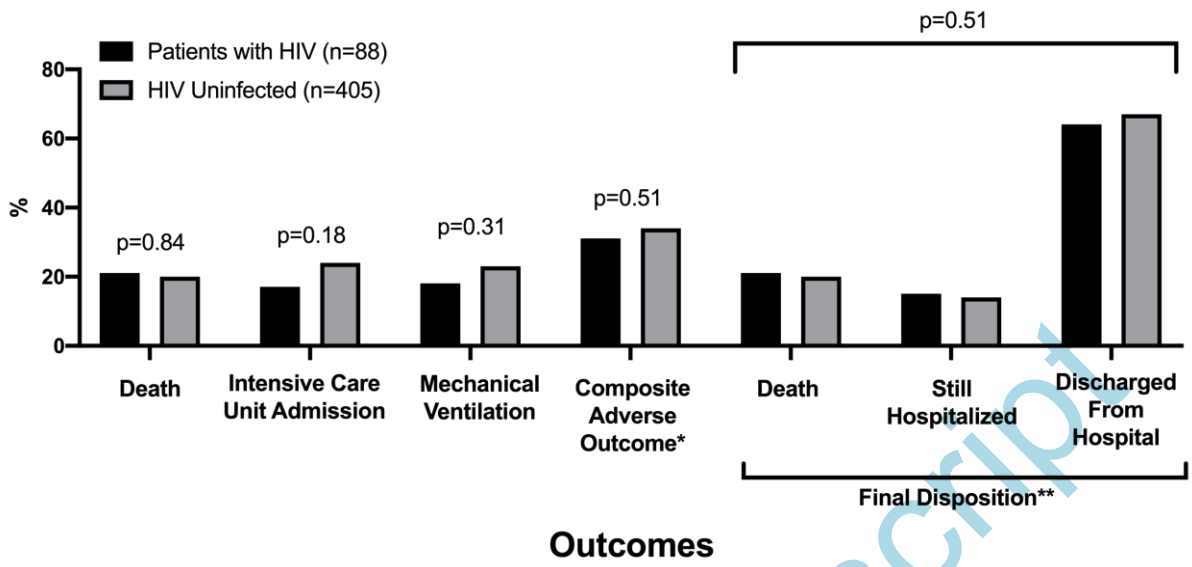
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Figure 1. Major outcomes for people with HIV (PWH) and matched comparators hospitalized in New York City with COVID-19. (*Composite outcome including death, intensive care unit admission or mechanical ventilation; **Mutually exclusive categorical outcomes)

Figure 2. Cumulative incidence of death comparing PWH to matched uninfected comparators during COVID-19 pandemic, March-April 2020 in New York City

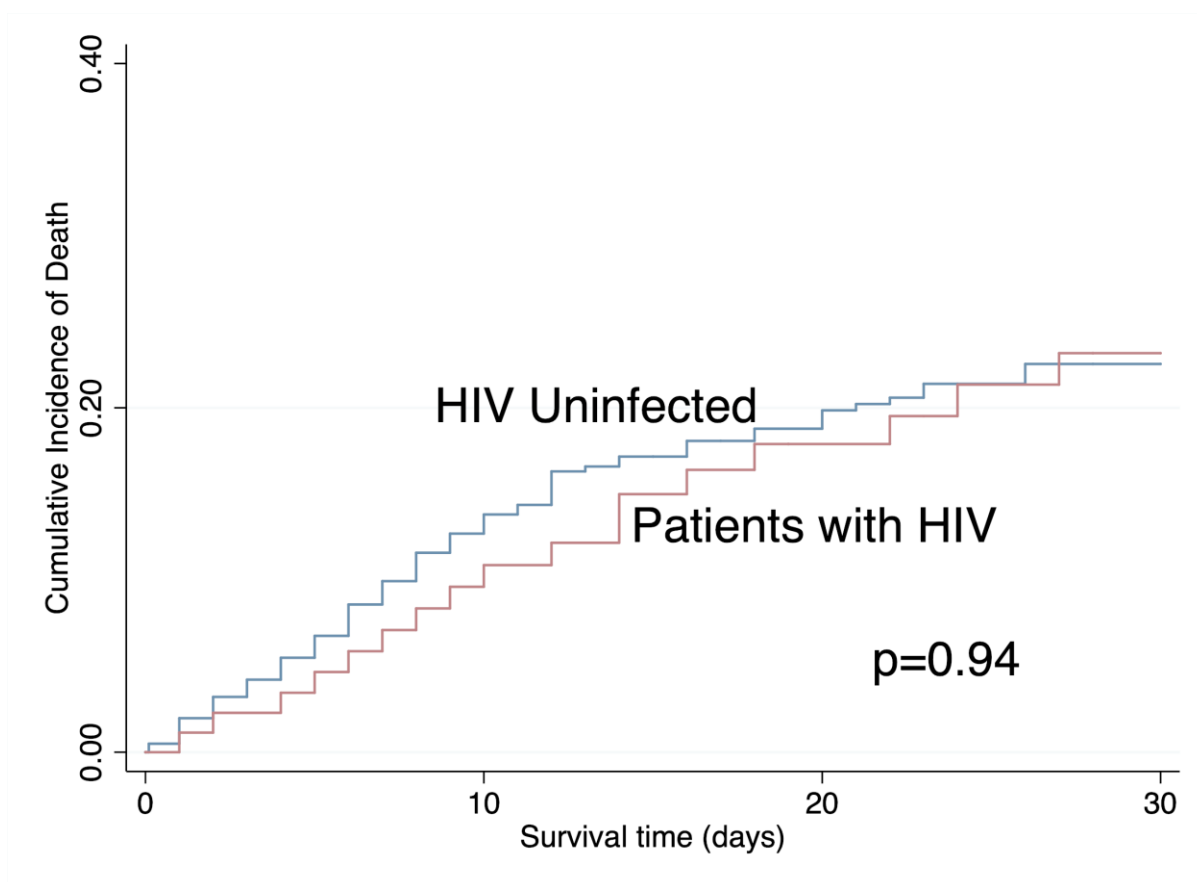
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Figure 1



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Figure 2



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