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# Association of Human Immunodeficiency Virus Infection with Outcomes Among Adults Hospitalized with COVID-19

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## Abstract

**Objective:** To evaluate the association of HIV infection with outcomes among people hospitalized with COVID-19.

**Design:** Prospectively-planned analysis of the American Heart Association's COVID-19 Cardiovascular Disease Registry.

**Setting:** 107 academic and community hospitals in the United States from March through December 2020

**Participants:** Consecutive sample of 21,528 adults hospitalized with COVID-19 at participating hospitals

**Main Outcome and Measure:** Primary outcome was pre-defined as in-hospital mortality. We used hierarchical mixed effects models to assess the association of HIV with in-hospital mortality accounting for patient demographics, comorbidities and clustering by hospital. Secondary

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**Data Access, Responsibility and Analysis:** Matthew Durstenfeld and Yifei Ma had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

**Data Sharing Statement:** Use of the data may be requested by submitting a research proposal to the AHA COVID-19 CVD Registry Research and Publications Committee at https://www.heart.org/-/media/files/professional/quality-improvement/covid-19-cvd-registry/gwtg-covid-rfp3_research-proposal-form_20201115.pdf?la=en.

outcomes included major adverse cardiac events (MACE), severity of illness, and length of stay (LOS).

**Results:** The registry included 220 people living with HIV (PLWH). PLWH were younger and more likely to be male, Non-Hispanic Black, on Medicaid, and active tobacco users. Of the study population, 36 PLWH (16.4%) died compared with 3,290 (15.4%) without HIV (Risk ratio 1.06; 95%CI 0.79–1.43; p=0.71). After adjustment for age, sex, race, and insurance, HIV was not associated with in-hospital mortality (aOR 1.13; 95%CI 0.77–1.6; p=0.54) with no change in effect after adding body mass index and comorbidities (aOR 1.15; 95%CI 0.78–1.70; p=0.48). HIV was not associated with MACE (aOR 0.99; 95%CI 0.69–1.44, p=0.91), COVID severity (aOR 0.96; 95%CI 0.62–1.50; p=0.86), or LOS (aOR 1.03; 95% CI 0.76–1.66; p=0.21).

**Conclusions:** In the largest study of PLWH hospitalized with COVID-19 in the United States to date, we did not find significant associations between HIV and adverse outcomes including in-hospital mortality, MACE, or severity of illness.

### Keywords

COVID-19; HIV; hospital; outcomes; mortality

## Introduction

There are limited data regarding risk of mortality among people living with human immunodeficiency virus (HIV; PLWH) hospitalized with coronavirus disease 2019 (COVID-19).[1] Excess cardiovascular risk due to traditional cardiovascular risk factors and heightened chronic inflammation/immune activation[2] may increase susceptibility among PLWH to cardiac injury or myocarditis from SARS-CoV-2 infection that results in higher mortality, cardiovascular complications, or post-acute sequelae of COVID-19 compared to non-HIV infected individuals. Thus, SARS-CoV-2 infection may present with different disease severity and result in different clinical outcomes among PLWH.[3] Although several studies have found higher risks of hospitalization[4, 5] and mortality[4, 6, 7] among PLWH with COVID-19, most case series of hospitalized patients suggest that the acute course of PLWH is similar to people without HIV.[8–13] With these conflicting data, whether coinfection with HIV portends worse outcomes among hospitalized patients with COVID-19 remains uncertain.[14] Therefore, we used the American Heart Association's COVID-19 CVD Registry powered by Get with the Guidelines® to study the association of HIV infection with COVID-19 outcomes among a large sample of hospitalized patients in the United States.

## Methods

### Data Source/Study Population

The Get With The Guidelines® programs are provided by the American Heart Association (AHA). As described previously, the AHA COVID-19 Cardiovascular Disease Registry is a quality improvement program that began in April 2020 and as of December 2020 included 21,528 people hospitalized at 107 participating hospitals, including a mix of academic and community hospitals from across the United States.[15] Approximately 2000 variables are

collected, including hospital characteristics, demographics, medical history, symptoms, vital signs, lab tests, and clinical outcomes. Records are uploaded by participating hospitals and a deidentified dataset is provided to investigators after review by a scientific review committee. The dataset included for this study includes records entered through December 2020.

Participating hospitals were instructed to upload data on all adult patients who had been hospitalized with confirmed COVID-19 and who had been discharged at the time of registry data upload regardless of preexisting cardiovascular disease. We included all patients uploaded to the registry in this analysis. Data were entered manually by each site and were not audited for accuracy.

## Explanatory Variables

The primary predictor was HIV status as defined by the medical record. Data regarding antiretroviral therapy, viral load, CD4 counts, and AIDS status were not included in the database. Other explanatory variables included age, sex, race, insurance, body mass index (BMI), other past medical history including hypertension, dyslipidemia, diabetes, smoking, chronic kidney disease, chronic lung disease, and time from symptom onset to admission. Insurance status was classified as Medicare, Medicaid, commercial, self/uninsured, and other/unable to determine. We also report values for initial laboratory tests upon admission to the hospital and treatment during hospitalization.

## Outcomes

Because the registry only included data through hospital discharge, the primary outcome was in-hospital mortality, defined by a discharge disposition of “expired.” We also conducted a sensitivity analysis in which we included those discharged to hospice as well. The first secondary outcome was a composite endpoint of major adverse cardiovascular outcomes (MACE) defined by in-hospital mortality, myocardial infarction, stroke, incident heart failure or myocarditis, and cardiogenic shock.[16] The next secondary outcome was the AHA COVID-19 Ordinal Scale defined as died, cardiac arrest, mechanical ventilation and shock requiring mechanical circulatory support, mechanical ventilation with shock requiring vasopressors/inotropes, mechanical ventilation only, and hospitalization only[16]. We also report components of MACE outcomes, mechanical ventilation, management in intensive care unit, and venous thromboembolism including deep venous thrombosis and pulmonary embolism. The next outcome was length of stay among survivors. Because PLWH were initially excluded from some COVID-19 clinical trials,[17] the final secondary outcome was participation in a COVID-19 clinical trial. Because our secondary outcomes were defined a priori as hypothesis generating to explain potential differences in in-hospital mortality, we did not correct for multiple testing.

## Missing Data

There were few missing data among our included covariates with exception of BMI, which was missing in 2,422 people (18 with HIV). We imputed missing values using multiple imputation with a random forest algorithm (MissRanger) based on the height, weight, age, sex, ZIP code, hospital, and history of diabetes. No other variables included in our models,

including outcome variables, had >1% missing values. We report laboratory findings only among those tested but did not include them in our models.

## Statistical Analysis

To characterize the study population stratified by HIV status, we report numbers and proportions for categorical variables and means with standard deviations for normally distributed continuous variables and median with interquartile range for non-normally distributed variables (ie time from symptom onset to admission). We used chi-squared test or Fisher's exact test for categorical variables, Student's t-test for normally distributed continuous variables, Mann-Whitney U test for non-normally distributed continuous variables, and Kruskal-Wallis test for ordinal variables.

We used hierarchical multivariable logistic regression mixed-effects models with hospital modeled as random effects and HIV and other covariates as fixed effects to examine conditional associations of HIV status with our primary and secondary outcomes adjusted for demographics and past medical history and account for clustering by hospital. For the primary outcome of in-hospital mortality, we report one model adjusted only for age, sex, race, and insurance and a second model adjusted for age, sex, race, body mass index, and comorbidities to estimate the conditional effect of HIV independent of those potential confounders. Included comorbidities were identified a priori as hypertension, dyslipidemia, diabetes, smoking, chronic kidney disease, and chronic lung disease based on prior reports of risk factors for severe disease. We also conducted a post-hoc sensitivity analysis including treatment with glucocorticoids or remdesivir as additional covariates, modeled odds of receiving treatment using hierarchical logistic regression, and tested for interactions with HIV status on mortality. For secondary outcomes we report only the fully adjusted models. For the ordinal severity scale we used a hierarchical multinomial logistic regression mixed-effects model for adjusted analysis. For length of stay we used a hierarchical Poisson regression mixed-effects model for adjusted analysis. P values for hierarchical models were generated by comparing the hierarchical model with HIV included with a similar model without HIV using ANOVA. Wald estimates of confidence intervals were used for hierarchical models. 2-sided P values of <0.05 were considered significant for the primary and secondary outcomes.

## Prespecified exploratory analysis

As an alternative approach to address confounding due to differences between those with and without HIV, we conducted a pre-specified exploratory propensity-matched analysis. We estimated propensity to have HIV using the same variables as our adjusted model, including facility and the imputed BMI value for those missing BMI. Then we used a 3:1 control to case nearest neighbor matching algorithm (MatchIt). The cases and controls were compared with a logistic regression model including the same variables as the adjusted hierarchical models.

## Data analysis and ethics approval

The American Heart Association Precision Medicine Platform (https://precision.heart.org/) was used for data analysis using R version 3.6.0 and SAS version 3.8. IQVIA (Parsippany,

New Jersey) serves as the data collection and coordination center. Dr. Durstenfeld & Mr. Ma had full access to the study data and take responsibility for the integrity of the data analyses. Institutional Review Board approval was granted by the University of California, San Francisco and the requirement for informed consent was waived.

## Results

Among 21,528 hospitalizations for COVID-19 at 107 hospitals, the cohort included 220 PLWH (1.0%), with baseline characteristics described in detail in Table 1. PLWH were younger (56.0+/−13.0 versus 61.3+/−17.9 years old, p<0.001), more likely to be male (72.3% versus 52.7%, p<0.001), and more likely to be Non-Hispanic Black (51.4% versus 25.4%, p<0.001). PLWH were more likely to be on Medicaid, government insurance for low-income individuals (44.5% versus 24.5%, p<0.001), and less likely to be on Medicare, government insurance for the elderly (20.9% versus 30.3%, p<0.001). PLWH had lower mean BMI (29.4 versus 30.8 $kg/m^2$, p=0.01) and were more likely to be active tobacco users (12.7% versus 6.5%, p<0.001). Despite having younger age, PLWH did not have significant differences in other comorbidities (Appendix: Supplementary Table A) consistent with a higher age-adjusted burden of traditional cardiovascular risk factors. Time from symptom onset to admission was not different by HIV status (median 5 days for both, p=0.75).

Admission laboratory findings and treatments are also shown in Table 1. PLWH had lower serum creatinine (1.00 vs 1.13. mg/dl, p<0.001) and higher d-dimer (850 vs 600 ng FEU/mL, p=0.01) compared to people without HIV, but no other significant differences. Although PLWH were less likely to be treated with glucocorticoids (26.4% vs 34.5%, p=0.01) and remdesivir (8.2% vs 15.1%, p=0.006), odds of treatment with those two therapies among PLWH were not statistically significantly lower compared those without HIV after accounting for patient characteristics, severity, and clustering by hospital (aOR 0.89, 95%CI 0.64–1.25, p=0.51 and aOR 0.81 95%CI 0.48–1.37, p=0.42, respectively).

Of the study population, 36 PLWH (16.4%) had the primary outcome of in-hospital mortality compared with 3,290 (15.4%) patients without HIV (Table 2). In-hospital mortality was not significantly different by HIV status in unadjusted analysis (Risk ratio 1.06, 95%CI 0.79–1.43; risk difference 0.9%, 95%CI −4.2 to 6.1%; p=0.71, Figure 1). After adjustment for age, sex, race, and insurance, we did not find a significant association of HIV with in-hospital mortality (Model 1: aOR 1.12 95%CI 0.76–1.64; p=0.58, Table 3). Results were similar after adding body mass index, hypertension, dyslipidemia, diabetes, smoking, chronic kidney disease, and chronic lung disease (Model 2: aOR 1.14, 95%CI 0.78–1.68; p=0.51; model coefficients listed in Supplemental Appendix B). Post-hoc sensitivity analysis including those discharged to hospice in the primary outcome yielded similar results (Model 2: aOR 1.12, 95%CI 0.77–1.64, p=0.55). Post-hoc sensitivity analysis adjusted for glucocorticoid and remdesivir use had similar results (aOR 1.13, 95%CI 0.76–1.69, p=0.54), and there were no significant interactions between HIV and treatments on mortality.

Among the entire population, 4,170 individuals (19.4%) had MACE. Among PLWH, there were 40 MACE (36 deaths, 4 with myocardial infarction, 2 with stroke, 0 with incident heart failure, 2 with myocarditis, and 1 with cardiogenic shock; Table 2). After

adjustment, HIV was not associated with increased risk of major adverse cardiac events (aOR 0.99, 95%CI 0.69–1.44; p=0.91). In an exploratory post-hoc analysis, we did not find significant associations between HIV and incidence of deep venous thrombosis and pulmonary embolism (Table 2).

Among those hospitalized with COVID-19, HIV was not associated with increased severity of illness as assessed by the American Heart Association's COVID Ordinal Scale (p=0.93). In adjusted models, HIV was not associated with increased severity of illness (aOR 0.96; 95%CI 0.62–1.50; p=0.86). Among those with HIV, 59 (26.8%) compared to 6,604 without HIV (30.7%) were managed in an intensive care unit (aOR 0.90, 95%CI 0.65–1.23; p=0.49). Similarly, 47 PLWH (21.4%) required endotracheal intubation and mechanical ventilation compared to 4,152 (19.3%) without HIV (aOR 1.00, 95%CI. 0.71–1.40; p=1.00).

HIV was also not associated with differences in length of stay by unadjusted analysis (median 6 days with or without HIV; p=0.73). After adjustment, HIV was not associated with an increased length of stay (aOR 1.02; 95% CI 0.98–1.07; p=1.00). Overall participation in a COVID-19 clinical trial was low and did not differ by HIV status (8.6% among PLWH and 9.2% among people without HIV, p=0.80). After adjustment, we did not find an association between HIV and clinical trial enrollment (aOR 1.07; 95% CI 0.63–1.83; p=0.79).

As a sensitivity analysis using propensity matching as an alternative approach to account for confounding, the estimated effect size was greater (aOR 1.52; 95% CI 0.94–2.44; p=0.09). The confidence interval largely overlaps with the primary analysis but could be compatible with clinically meaningfully increased risk at the population level. In the propensity-matched analysis, however, there was not a significant association between HIV and MACE (aOR 1.15; 95%CI 0.75–1.77; p=0.52), consistent with the primary analysis.

## Discussion

We found that HIV was not significantly associated with in-hospital mortality among people hospitalized with COVID-19 in both unadjusted and adjusted models among those hospitalized for COVID-19 at 107 hospitals participating in the AHA COVID-19 Cardiovascular Disease registry. We also did not find significant differences in major adverse cardiac events, severity of illness including intensive care unit admission and mechanical ventilation, or length of stay by HIV status. Lastly, enrollment in COVID-19 clinical trials was not lower among PLWH compared to people without HIV.

### Hypotheses regarding HIV as a risk factor

The biologic rationale underlying the impact of HIV infection on COVID-19 outcomes could include worsening of clinical sequelae or a paradoxical protective effect. First, PLWH have higher prevalence of risk factors for severe COVID-19 compared to those without HIV including hypertension, diabetes, cardiovascular disease, chronic kidney disease, smoking, and chronic lung disease.[4, 6] Although prevalence of most risk factors was similar except smoking in our study, given that PLWH were an average five years younger this represents a significantly increased age-adjusted risk factor burden. Nonetheless, in our study, the

severity of illness among HIV-infected individuals hospitalized with COVID-19 was similar to individuals without HIV, and inflammatory biomarkers were similar. Despite having higher d-dimer levels and higher rates of smoking, PLWH had similar rates of MACE and troponin levels compared to uninfected individuals. Given the limited data available in this registry, we were unable to investigate the role of antiretroviral therapy or differential serologic antibody response among PLWH, which may affect outcomes. [18][19]

### Distinction of our findings from population-based registry data

Our results among hospitalized PLWH contrast with findings of increased risk found in some population-based registry studies. In particular, a large population-based registry in the United Kingdom (OpenSAFELY)[20] and a South African cohort study[6] both found higher population-based mortality rates among PLWH with COVID-19 compared to those without PLWH. A population-based approach may yield different results if HIV status is associated with differential rates of infection or hospitalization.[4, 5] One study found lower rates of infection among PLWH but higher odds of severe disease.[19] In contrast to those studies, we estimated the association of HIV infection with in-hospital mortality conditional upon being admitted to a participating hospital as opposed to calculating the marginal risk across a population. Therefore, the results of our hospital-based analysis do not exclude a meaningfully increased population-level risk among PLWH because our analysis is restricted to those with severe disease requiring hospitalization.[20] Future studies may provide insights reconciling population-level differences in outcomes with our findings, which may be related to social determinants of health, differences in infection rates, and differences in severity.[14]

### Comparison with other studies of hospitalized PLWH

As the largest hospital-based analysis in the United States to date, our finding that HIV is not independently associated with in-hospital mortality is consistent with most prior reports among PLWH hospitalized with COVID-19.[8–13] The Veterans Aging Cohort Study, which included 253 HIV+/COVID+ veterans, most of whom were not hospitalized, found similar risk of hospital admission (aHR 1.09, 95% CI 0.85–1.41), intensive care unit admission (aHR 1.08, 95% CI 0.72–1.62), and death (aHR 1.08, 95% CI 0.66–1.75), consistent with our findings.[12] Similarly, Flannery et al found no differences in ventilator use, admission to the ICU, or in-hospital mortality among 99 PLWH (91 treated) admitted with COVID-19 to the Northwell Health System compared to people without HIV.[21] Our study has additional power to confirm and extend the generalizability of these findings to a broader population of hospitalized patients including non-veterans and those outside the New York metropolitan area.

Prior to our study, the largest studies of PLWH hospitalized for COVID-19 were the WHO Global COVID-19 Clinical Data Platform[22], the HIV-COVID-19 consortium study[23] and the United Kingdom-based ISARIC WHO CCP study[7] The largest international registry, the WHO Global COVID-19 Clinical Data Platform, included 15,522 PLWH, of whom 14,682 (94.6%) were from South Africa.[22] They found that PLWH were at increased risk of severe disease (aOR 1.06, 95%CI 1.02–1.11) and in hospital mortality (aHR 1.29, 95% CI 1.23–1.35). In a sensitivity analysis excluding South Africa, the risk of mortality

was no longer statistically significant among PLWH (aHR 1.16, 95% CI 0.90–1.51), which is similar to our effect estimate and confidence interval. The HIV-COVID-19 consortium study was a multicenter registry that included 286 PLWH at 36 institutions mostly in the United States, of whom 164 were hospitalized.[23] This descriptive study lacked a control group of people without HIV, but found a mortality rate of 16.5%, similar to our study. The ISARIC WHO CCP study, which included 122 PLWH hospitalized for COVID-19 and an HIV-negative control group, found a much higher overall mortality than our study or the consortium study (26.7% among PLWH, 32.1% among those without HIV).[7] They found no difference in crude mortality by HIV status, which is consistent with our findings. After adjustment for age, however, they found that HIV was associated with higher risk of mortality (HR 1.47, 95% CI 1.01–2.14), which we did not find in our study.

## Limitations

The strengths of our study are that this is a large study population with over 21,000 people hospitalized at 107 hospitals with rigorous clinical adjudication of outcomes and detailed clinical phenotyping. A major limitation is that the registry did not contain details regarding HIV severity and treatment such as treatment with antiretroviral therapy, CD4 counts, or viral loads. A second limitation is the lack of data on high-flow oxygen use and non-invasive ventilation. A third limitation is the lack of post-discharge outcomes including 30-day mortality. Despite diversity in hospital type and geographic location, it remains possible that the subset of hospitals participating in the registry may not be representative and that access to participating hospitals may vary by insurance status (which is different among those with HIV) and may result in selection bias. Therefore, we focused not a population-based analysis to estimate marginal effects of the average additional risk of HIV but rather the conditional effect: what additional risk might a person living with HIV face conditional upon being admitted to a participating hospital. While the absolute number of HIV-infected individuals with MACE and death was low, our study is the largest study of PLWH hospitalized with COVID-19 to date. As an observational study, a further threat to validity is unmeasured confounding; to address this concern, we performed a propensity score-matched analysis in which we achieved good matching. As data are entered by each individual site, measurement errors or differences data ascertainment may be present. We also did not account for calendar week or the local burden of the pandemic at a given hospital site as we hypothesized these should be non-differential with respect to HIV status.

## Conclusions

As the largest study to date of people with HIV hospitalized with COVID-19 in the United States, this registry-based study of over 21,000 people hospitalized for COVID-19 had 1% with a past medical history that included HIV. Our data provide evidence that despite increased traditional cardiovascular risk factors, PLWH hospitalized with COVID-19 in the United States are not at significantly elevated risk of complications from COVID-19 infection including in-hospital mortality, ICU admission, need for mechanical ventilation, major adverse cardiovascular events, or prolonged length of stay. Future studies evaluating the long-term sequelae of COVID-19 in the setting of HIV infection are needed.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgements:

The Get With The Guidelines® programs are provided by the American Heart Association (AHA). The AHA Precision Medicine Platform (https://precision.heart.org/) was used for data analysis. IQVIA (Parsippany, New Jersey) serves as the data collection and coordination center.

### Conflicts of Interest and Sources of Funding:

FR has received consulting fees from Novartis, Janssen, NovoNordisk, and HealthPals unrelated to this work. EAS reports unrelated research grants to BIDMC: AstraZeneca, BD, Boston Scientific, Cook, CSI, Laminate Medical, Medtronic and Philips and unrelated consulting/speaking fees from Abbott, Bayer, BD, Boston Scientific, Cook, CSI, Inari, Janssen, Medtronic, Philips, and VentureMed. RVP reports unrelated research support Janssen and Infraredx; consulting fees from Abbott Vascular; and scientific advisory board (minor equity interest) of Stallion Cardio, DocVocate, and HeartCloud. PYH has received honoraria from Gilead and Merck, research grant from Novartis, unrelated to this work. MSD, KS, and YM have no disclosures. AHA's suite of Registries is funded by multiple industry sponsors. AHA's COVID-19 CVD Registry is partially supported by The Gordon and Betty Moore Foundation (Palo Alto, California). Dr Durstenfeld was supported by NIH/NHLBI 5T32HL007731-28 and is now supported by NIH/NHLBI 5K12 HL143961. Dr. Rodriguez was funded by NIH/NHLBI K01HL144607 and AHA/Robert Wood Johnson Harold Amos Medical Faculty Development Program. Dr Secemsky is supported by NIH/NHLBI K23HL150290. Dr. Parikh is supported by AHA 18CDA34110335. Dr Hsue is supported by NIH/NIAID 2K24AI112393-06. The AHA COVID-19 CVD Registry Committee approved the research proposal and reviewed the manuscript prior to submission.

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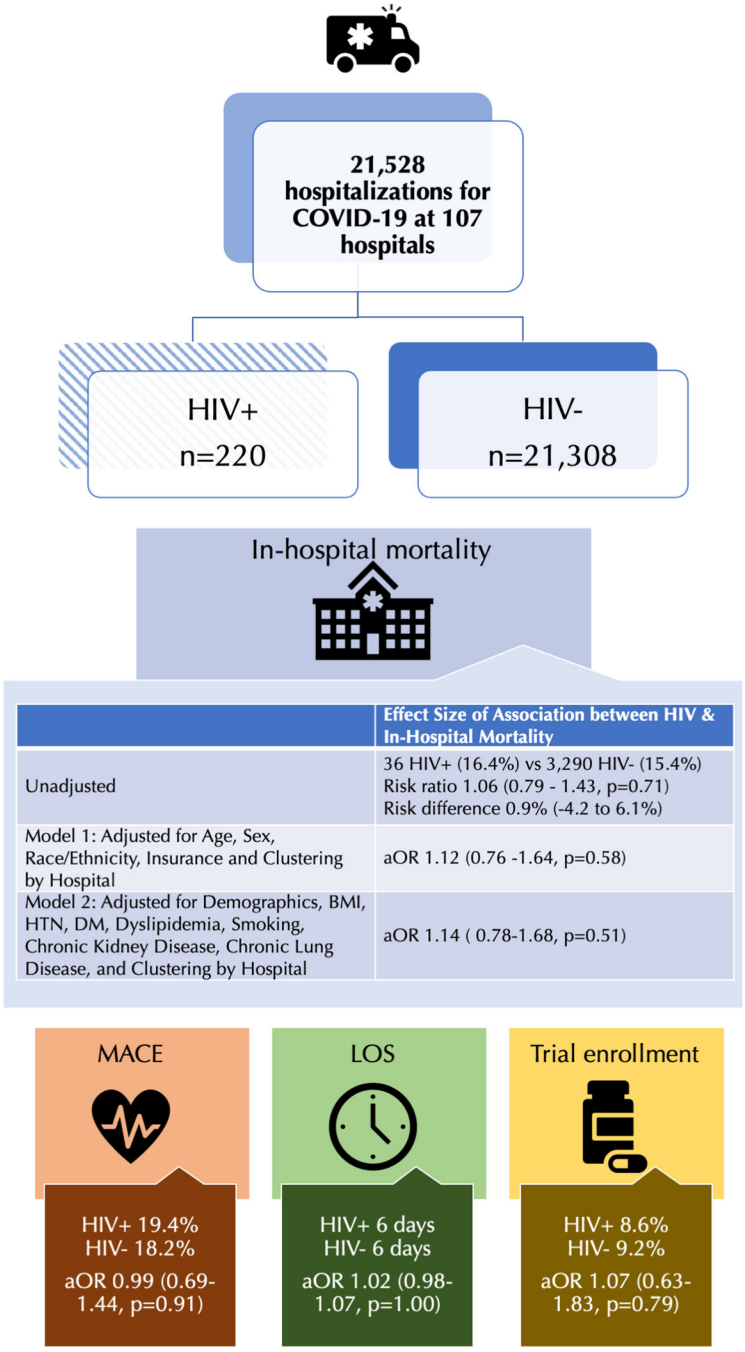


**Figure 1: Association of HIV with COVID-19 Outcomes among Hospitalized Patients in the American Heart Association COVID-19 CVD Registry (n=21,528)**

HIV was not found to have a significant association with in-hospital mortality among people hospitalized with COVID-19 in unadjusted analysis or after adjustment. HIV was also not found to have an association with major adverse cardiac events, length of stay, or clinical trial enrollment. aOR=adjusted odds ratio; MACE=major adverse cardiac events; LOS=Length of Stay.

**Table 1:**

Demographics, baseline characteristics, laboratories, and treatment by HIV status.

| | HIV positive (n=220) | HIV negative (n=21,308) | P value |
|---|---|---|---|
| Age | 56.0 ± 13.0 | 62.3 ± 17.9 | <0.001 |
| Female | 61 (27.7%) | 9,823 (46.1%) | <0.001 |
| Ethnicity/Race | | | <0.001 |
| Hispanic/Latinx | 60 (27.3%) | 5,418 (25.4%) | |
| Non-Hispanic Black | 113 (51.4%) | 5,418 (25.4%) | |
| Native American | 0 | 106 (0.5%) | |
| Asian | 1 (0.5%) | 856 (4.0%) | |
| Pacific Islander | 0 | 78 (0.4%) | |
| Non-Hispanic White | 26 (11.8%) | 8,176 (38.4%) | |
| Other/Unable to Determine | 20 (9.1%) | 1,256 (5.9%) | |
| Insurance | | | <0.001 |
| Medicare | 46 (20.9%) | 6,448 (30.3%) | |
| Medicaid | 98 (44.5%) | 5,228 (24.5%) | |
| Commercial | 56 (25.5%) | 6,638 (31.2%) | |
| Self/Uninsured | 13 (5.9%) | 2,077 (9.7%) | |
| Other/Unable to Determine | 7 (3.2%) | 917 (4.3%) | |
| Body Mass Index, kg/m$^2$ † | 29.4 ± 7.9 | 30.8 ± 8.5 | 0.01 |
| Past Medical History | | | |
| Hypertension | 125 (56.8%) | 12,548 (58.9%) | 0.58 |
| Dyslipidemia | 69 (31.4%) | 7,354 (34.5%) | 0.37 |
| Diabetes | 88 (40.0%) | 7,526 (35.3%) | 0.17 |
| Active Tobacco Use | 28 (12.7%) | 1,378 (6.5%) | <0.001 |
| Chronic Kidney Disease | 35 (15.9%) | 2,749 (12.9%) | 0.22 |
| Chronic Lung Disease | 48 (21.8%) | 3,967 (18.6%) | 0.26 |
| Symptom Onset to Admission (days) ‡ | 5.0 (IQR 2–9) | 5.0 (IQR 2–8) | 0.75 |
| Initial Laboratory Testing | | | |
| WBC Count, k/uL * | 6.70 (IQR 4.95–8.70) | 7.00 (IQR 5.10–9.70) | 0.07 |
| Platelet Count, k/uL * | 204 (IQR 143–260) | 204 (IQR 167–267) | 0.11 |
| Serum Creatinine, mg/dl * | 1.00 (IQR 0.94–1.64) | 1.13 (IQR 0.80–1.47) | <0.001 |
| C reactive protein, mg/L ‡ | 64 (IQR 24–100) | 57 (IQR 14–111) | 0.53 |
| Ferritin, ng/ml ‡ | 484 (IQR 273–1013) | 563 (IQR 239–1157) | 0.58 |
| Troponin, ng/L ‡ | 10 (IQR 0–50) | 11 (IQR 0–50) | 0.91 |
| BNP, pg/mL § | 34 (IQR 19–94) | 64 (IQR 23–223) | 0.08 |
| NT-Pro-BNP, pg/mL § | 299 (IQR 61–1753) | 217 (IQR 75–1607) | 0.94 |

| | HIV positive (n=220) | HIV negative (n=21,308) | P value |
|---|---|---|---|
| D-Dimer, ng FEU/mL [§] | 850 (IQR 300–1231) | 600 (IQR 410–1660) | 0.01 |
| Treatment During Hospitalization | | | |
| Azithromycin | 76 (34.5%) | 8,862 (41.6%) | 0.04 |
| Glucocorticoids | 58 (26.4%) | 7,346 (34.5%) | 0.01 |
| Remdesivir | 18 (8.2%) | 3,221 (15.1%) | 0.006 |
| Tocilizumab | 14 (6.4%) | 1,647 (7.7%) | 0.53 |
| Immunoglobulins | 2 (0.9%) | 143 (0.7%) | 0.66 |
| Convalescent Plasma | 13 (5.9%) | 1565 (7.3%) | 0.49 |

Baseline characteristics for people living with HIV (n=220) and people living without HIV (n=21,308) including demographics, past medical history, time from symptom onset to hospital admission, and admission laboratory results.

[*] Missing <5%.

[†] Missing 5–19%.

[‡] Missing 20–50%.

[§] Missing 60–85%.

**Table 2:**

Unadjusted outcomes by HIV status.

| | HIV positive (n=220) | HIV negative (n=21,308) | Unadjusted p value |
|---|---|---|---|
| **In-Hospital Mortality** | 36 (16.4%) | 3,290 (15.4%) | 0.78 |
| **Major Adverse Cardiac Event** | 40 (18.2%) | 4,130 (19.4%) | 0.72 |
| Died | 36 (16.4%) | 3,290 (15.4%) | |
| Myocardial Infarction | 4 (1.8%) | 651 (3.1%) | |
| Stroke | 2 (0.9%) | 310 (1.5%) | |
| Incident Heart Failure | 0 | 391 (1.8%) | |
| Myocarditis | 2 (0.9%) | 66 (0.3%) | |
| Cardiogenic Shock | 1 (0.5%) | 136 (0.6%) | |
| **Managed in ICU** | 59 (26.8%) | 6,545 (30.7%) | 0.24 |
| **Mechanical Ventilation** | 47 (21.4%) | 4,105 (19.3%) | 0.48 |
| **AHA COVID-19 Ordinal Scale** | | | 0.93 |
| Died | 36 (16.4%) | 3,290 (15.4%) | |
| Cardiac Arrest | 0 | 93 (0.4%) | |
| Mechanical Ventilation & Circulatory Support | 0 | 51 (0.2%) | |
| Mechanical Ventilation & Vasopressors/Inotropes | 12 (5.5%) | 775 (3.6%) | |
| Mechanical Ventilation Only | 6 (2.7%) | 1,188 (5.6%) | |
| Hospitalization Only | 166 (75.5%) | 15,911 (74.7%) | |
| **Venous Thromboembolism** * | | | |
| Deep Venous Thrombosis | 6 (2.7%) | 502 (2.4%) | 0.89 |
| Pulmonary Embolism | 3 (1.4%) | 382 (1.8%) | 1.00 |
| **Enrolled in COVID-19 Clinical Trial** | 19 (8.6%) | 1962 (9.2%) | 0.80 |
| **Length of Stay among Survivors, median** | 6 (IQR 4–11) | 6 (IQR 4–11) | 0.73 |

Unadjusted outcomes were similar among people with and without HIV. Major adverse cardiovascular outcomes (MACE) is defined as in-hospital mortality, myocardial infarction, stroke, incident heart failure or myocarditis, and cardiogenic shock. Totals of MACE do not add up to 100% as each patient was only counted once for MACE even with multiple MACE.

* Post-hoc exploratory analysis.

**Table 3.**

Adjusted effect size estimates for association between HIV

| | Adjusted Odds Ratio | 95% CI | P value |
|---|---|---|---|
| In-Hospital Mortality | 1.14 | 0.78–1.68 | 0.51 |
| Major Adverse Cardiac Event | 0.99 | 0.69–1.44 | 0.91 |
| AHA COVID-19 Ordinal Scale | 0.96 | 0.62–1.50 | 0.86 |
| Managed in ICU | 0.90 | 0.65–1.23 | 0.49 |
| Mechanical Ventilation | 1.00 | 0.71–1.40 | 1.00 |
| Deep Venous Thrombosis | 1.04 | 0.45–2.40 | 0.93 |
| Pulmonary Embolism | 0.80 | 0.25–2.55 | 0.69 |
| Enrolled in COVID-19 Clinical Trial | 1.07 | 0.63–1.83 | 0.79 |
| Length of Stay among Survivors | 1.02 | 0.98–1.07 | 1.00 |

Hierarchical logistic mixed effects models account for clustering by hospital as random effects and are adjusted for age, sex, race, insurance, body mass index, diabetes, hypertension, dyslipidemia, smoking, chronic kidney disease, and chronic lung disease as fixed effects. Length of stay is modeled using a hierarchical Poisson mixed effects model with the same covariates.