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Clinical characteristics and outcomes of people living with HIV hospitalized with COVID-19: a nationwide experience

Maria Elena Ceballos[1], Patricio Ross[2], Martin Lasso[3], Isabel Dominguez[4], Marcela Puente[5], Pablo Valenzuela[6], Margarita Enberg[7], Michel Serri[8], Rodrigo Muñoz[9], Yazmin Pinos[10], Macarena Silva[11], Matías Noguera[12], Angelica Dominguez[13], Francisco Zamora[14] and on behalf of Chilean HIV/COVID-19 Study Group*

Abstract

In this prospective, multicentric, observational study, we describe the clinical characteristics and outcomes of people living with HIV (PLHIV) requiring hospitalization due to COVID-19 in Chile and compare them with Chilean general population admitted with SARS-CoV-2. Consecutive PLHIV admitted with COVID-19 in 23 hospitals, between 16 April and 23 June 2020, were included. Data of a temporally matched-hospitalized general population were used to compare demography, comorbidities, COVID-19 symptoms, and major outcomes. In total, 36 PLHIV subjects were enrolled; 92% were male and mean age was 44 years. Most patients (83%) were on antiretroviral therapy; mean CD4 count was 557 cells/mm^3. Suppressed HIV viremia was found in 68% and 56% had, at least, one comorbidity. Severe COVID-19 occurred in 44.4%, intensive care was required in 22.2%, and five patients died (13.9%). No differences were seen between recovered and deceased patients in CD4 count, HIV viral load, or time since HIV diagnosis. Hypertension and cardiovascular disease were associated with a higher risk of death (p = 0.02 and 0.006, respectively). Compared with general population, the HIV cohort had significantly more men (OR 0.15; IC 95% 0.07–0.31) and younger age (OR 8.68; IC 95% 2.66–28.31). In PLHIV, we found more intensive care unit admission (OR 2.31; IC 95% 1.05–5.07) but no differences in the need for mechanical ventilation or death. In this cohort of PLHIV hospitalized with COVID-19, hypertension and cardiovascular comorbidities, but not current HIV viro-immunologic status, were the most important risk factors for mortality. No differences were found between PLHIV and general population in the need for mechanical ventilation and death.

Keywords

HIV, COVID-19, SARS-CoV-2, coronavirus, pandemic

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Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the cause of coronavirus disease 2019 (COVID-19), emerged in China in late 2019 from a zoonotic source.[1] The virus has raised global concern due to its high transmission capability as well as its high morbidity and mortality.[2] Currently, over 29 million cases and 900,000 deaths have been reported worldwide.[3]

While most people with COVID-19 develop mild or uncomplicated illness, approximately 15% develop severe disease requiring hospitalization and oxygen support, and 5% develop a critical disease.[4]

Older age, obesity, hypertension, diabetes, and cardiovascular disease are associated with an increased risk of death in COVID-19.[5] An immunocompromised condition has been described as a risk factor for aggravation.[6]

[1]Department of Infectious Diseases, School of Medicine, Pontificia Universidad Católica de Chile, Santiago, Chile
[2]Department of Internal Medicine, School of Medicine, Pontificia Universidad Católica de Chile, Santiago, Chile
[3]Hospital Sótero del Río, Puente Alto, Chile
[4]Clínica Santa María, Santiago, Chile
[5]Hospital Del Salvador, Providencia, Chile
[6]Hospital DIPRECA, Las Condes, Chile
[7]Hospital Regional de Antofagasta Dr Leonardo Guzmán, Antofagasta, Chile
[8]Hospital FACH and Clínica Tabancura, Las Condes, Chile
[9]Hospital Clínico Magallanes, Punta Arenas, Chile
[10]Hospital Base San José Osorno, Osorno, Chile
[11]Hospital San Borja Arriarán, Santiago, Chile
[12]Pontificia Universidad Católica de Chile, Santiago, Chile
[13]Faculty of Medicine, Department of Public Health, Pontificia Universidad Católica de Chile, Santiago, Chile
[14]Hospital Barros Luco Trudeau, San Miguel, Chile
* A list of other author contributors is listed in the Acknowledgment section.

Corresponding author:
María Elena Ceballos, Department of Infectious Disease, Pontificia Universidad Católica de Chile, Diagonal Paraguay 362, 6th Floor, Santiago 8320000, Chile.
Email: mceballos@med.puc.cl

Notwithstanding, we are still learning about the impact of HIV infection on clinical outcomes and prognosis of patients infected with SARS-CoV-2.[7] Case series and cohort studies have not detected an increased risk of severe outcomes among people living with HIV (PLHIV).[8–10] However, data from large electronic data centers have shown an increase risk of COVID-19 mortality.[11–13] Despite Latin America has been one of the most affected areas in this COVID-19 pandemic, we are not aware of studies reporting outcomes for PLHIV with COVID-19 in this region. Chilean incidence of COVID-19 cases is currently of 2,493/100,000 population, one of the highest from the region, with a case fatality rate of 3.3%.[14]

In this nationwide prospective study, we describe the clinical, epidemiological, laboratory, and radiological characteristics, as well as the clinical outcomes of a cohort of hospitalized PLHIV with COVID-19 in 23 hospitals in Chile. Additionally, we make comparisons between these cohorts with data publicly available of Chilean general population admitted with SARS-CoV-2.

Methods

Study design

This is a prospective, observational cohort study. People living with HIV with confirmed SARS-CoV-2 infection and hospitalized in 23 hospitals of eight regions all over the country were recruited. These hospitals, distributed along the whole country (Figure 1), are currently responsible for the care of 27097 PLHIV, which represent two thirds of total PLHIV that are under medical care in the country.[15] Patients were enrolled between 16 April and 23 June 2020.

Age, gender, comorbidities (diabetes, hypertension, obesity, chronic obstructive pulmonary disease (COPD), asthma, chronic kidney disease, chronic liver disease, cardiovascular disease, cancer, and pharmacological immunosuppression), and HIV-specific variables were registered as baseline data.

Signs and symptoms of COVID-19 on admission, laboratory findings, chest X-rays or computed tomography (CT), oxygen and ventilatory support requirement, antivirals or other therapies used during hospitalization, and clinical outcomes were daily reviewed on clinical charts and nursing records and registered in a standardized study record file, until death or discharge.

In addition, we collected information about Chilean general population hospitalized due to COVID-19 on the same period, from data publicly available extracted from epidemiological reports of Chilean health authorities. Data from the general population consisted of age, sex, comorbidities (diabetes, hypertension, obesity, COPD, asthma, chronic kidney disease, chronic liver disease, and cardiovascular disease), COVID-19 signs and symptoms, and clinical outcomes such as admission to intensive care unit (ICU), need for mechanical ventilation, and death.[16–18]

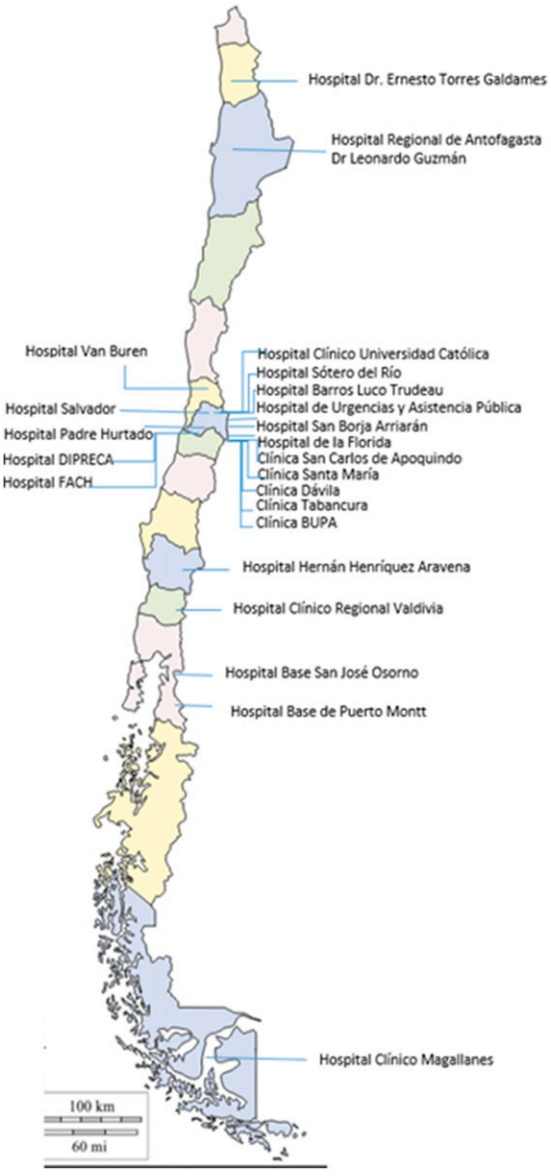

Figure 1. Distribution of the 23 Chilean hospitals included in the study.

We made comparisons between this general population and our HIV cohort.

The Ethics Committee of the School of Medicine of Pontificia Universidad Católica de Chile approved the study protocol. Informed consent was waived by the Committee. Data were encoded to maintain the anonymity of the patients.

We defined *confirmed SARS-CoV-2 infection* in patients with compatible clinical findings and a positive real-time polymerase chain reaction for SARS-CoV-2.[16] These tests were performed in the local designated hospital by nasopharyngeal swab. *Severe COVID-19 disease* was defined as dyspnea, respiratory rate greater than 30 breaths per min, oxygen saturation of 93% or less at room air, a ratio of

partial pressure of arterial oxygen to fractional concentration of oxygen in inspired air (PaO_2/FiO_2) of ≤300, or radiological progression of more than 50% in 48 h.[19] Definition of *multiorgan failure* has been described elsewhere.[20] The definition of *confirmed lung coinfection* was used when a patient had clinical symptoms or signs of bacterial or fungal pneumonia with a positive culture for a new pathogen from a lower respiratory tract specimen (including sputum, tracheal aspirates, or bronchoalveolar lavage fluid) or a positive urinary antigen for *Streptococcus pneumoniae* or *Legionella pneumophila*. The definition *suspect lung coinfection* was used when a patient had clinical symptoms or signs of bacterial or fungal pneumonia without microbiological confirmation. *Supressed HIV viremia* was considered with an HIV viral load less than 50 copies RNA/mL. *NLR* was the neutrophil-to-lymphocyte ratio. A ratio equal or greater than 3.04 was considered elevated.[21]

Statistical analysis

Continuous variables were expressed as mean and standard deviation if they were normally distributed or as median with quantiles 25 and 75 if they were not. Comparisons among groups—severe versus non-severe COVID-19 and deceased versus recovered patients—were done using the nonparametric Mann–Whitney test for numerical variables. Categorical variables were expressed as frequency and percentages. Comparisons among groups were performed using Fisher's exact test. When comparing our results with general population figures, 95% confidence intervals (95% CI) for prevalence and odds ratios were computed. Significance level was set at 0.05. Data entry and descriptive statistical analysis was done using Microsoft Office's Excel software. Statistical analyses were performed using SPSS 17.0.

Results

During the study period, 36 PLHIV with confirmed SARS-CoV-2 infection were hospitalized. In the same period, as of 22 June, 18,285 individuals from general population were hospitalized with COVID-19 in Chile.

Regarding the HIV cohort, a previous close contact with a person with SARS-CoV-2 infection was documented in 13 patients (36%). Three patients were foreigners. Most of the patients were on antiretroviral therapy (ART) at the time of COVID-19 diagnosis. Twenty-four out of 36 were receiving a backbone with two nucleoside reverse transcriptase inhibitors and a third drug. In these cases, the third drug was integrase strand transfer inhibitors in 19 patients, protease inhibitors in one, and non-nucleoside reverse transcriptase inhibitor in four patients. Five patients abandoned ART at the moment of admission and one was diagnosed with HIV at this hospitalization. A suppressed HIV viremia was found in most of the patients and 89% had a CD4 cell count >350 cells/mm^3. Patients over 50 years old had higher prevalence of hypertension and chronic kidney disease than patients younger than 50 ($p < 0.01$). One patient had hepatitis B coinfection, and no coinfection with hepatitis C was documented. Baseline characteristics of PLHIV and the comparison with Chilean general population hospitalized due to COVID-19 are shown in Table 1. People living with HIV had significantly lower age, higher proportion of men, and higher cardiovascular disease than general population.

Mean time between COVID-19 symptoms onset and hospital admission was 6.5 days. People living with HIV had significantly higher proportion of fever, dyspnea, chest pain, diarrhea, anosmia, and ageusia than general population. Main symptoms, laboratory, and radiological findings are shown in Table 2. For the 28 PLHIV that had a chest CT available during hospitalization, the prevailing pattern was ground-glass opacities, with bilateral and peripheral involvement.

Regarding potential anti-SARS-CoV-2 treatments, 13 patients (36.1%) received hydroxychloroquine, 4 (11%) lopinavir/ritonavir, and 10 (27.8%) corticosteroids. There were no patients treated with IL-6 antagonists.

Twenty-three patients (64%) received antibiotics for suspected lung coinfections, but only one of them had culture-confirmed bacterial pneumonia by *S. pneumoniae*. One patient had suspected *Pneumocystis jirovecii* pneumonia and another patient had a confirmed coinfection with human metapneumovirus.

Severe COVID-19 was developed by 16 patients (44.4%), with a mean of 10 days between symptoms onset and aggravation. COVID-19 complications were thromboembolic events in two patients, myocarditis in one patient, and arrhythmias in two patients (non-hydroxychloroquine associated). Median time staying in ICU was 7 days. Comparisons of need of ICU, mechanical ventilation, and death (case fatality rate) between the HIV cohort and general population can be seen in Table 3. People living with HIV had significantly higher ICU admission than general population, but no differences were seen in needing for mechanical ventilation and death.

Comparing PLHIV who did and did not develop severe COVID-19 ($n = 16$ and $n = 20$, respectively), no differences were seen in age, gender, been on ART, baseline HIV Centers for Disease Control and Prevention (CDC) stage, undetectable HIV viral load before admission, CD4 cell count before admission or during hospitalization, and comorbidities. A significant decrease in $CD4^+$ T-lymphocyte count was observed comparing known preadmission and admission values in patients that had severe COVID-19 ($p = 0.03$). Patients with severe illness had higher NLR ($p = 0.001$) and higher C-reactive protein ($p = 0.03$) during hospitalization.

From the 36 patients, 31 were discharged (86.1%) and five patients died (13.9%), three of them attributed to COVID-19. Two of these deceased patients had advanced

Table 1. Baseline characteristics of patients with HIV and general population admitted with COVID-19.

Variable	HIV cohort *N* = 36	General population *N* = 18,285	OR	(IC 95%)
Mean age, years (range)	44 (26–85)	NA	—	—
Age				
<50 years	26 (72%)	6027 (33%)		
>50 years	10 (28%)	12258 (67%)	**0.15**	**(0.07-0.31)**
Male gender	33 (91.7%)	10267 (56%)	**8.68**	**(2.66-28.31)**
Current tobacco use	10 (27.8%)	NA	—	—
Comorbidities				
Any	20 (55.6%)	NA	—	—
Diabetes	4 (11.1%)	3602 (19.7%)	0.50	(0.18–1.44)
Hypertension	6 (16.7%)	5723 (31.3%)	0.44	(0.18–1.06)
Obesity	5 (13.9%)	1243 (6.8%)	2.21	(0.86–5.70)
Chronic obstructive pulmonary disease	1 (2.8%)	805 (4.4%)	0.63	(0.09–4.57)
Asthma	0 (0%)	585 (3.2%)	0.43	(0.03–6.95)
Chronic kidney disease	4 (11.1%)	786 (4.3%)	2.78	(0.98–7.88)
Chronic liver disease	1 (2.8%)	201 (1.1%)	2.59	(0.35–19.00)
Cardiovascular disease[a]	4 (11.1%)	750 (4.1%)	**2.92**	**(1.03-8.28)**
Cancer	3 (8.3%)	NA	—	—
Pharmacological immunosuppression	2 (5.6%)	NA	—	—

Baseline HIV variables N = 36	
Mean time since HIV infection diagnosis, years (range)	7 (3–14)
Nadir CD4 count (at HIV diagnosis), cells/mm^3	332 (220–600)
CD4 count before admission[b], cells/mm^3[c]	557 (405–694)
Undetectable viral load before admission[b,d]	19 (68%)
Centers for Disease Control and Prevention stage at HIV diagnosis[e]	
A	13 (36.1%)
B	4 (11.1%)
C	10 (27.8%)
On ART[f]	30 (83.3%)
On second-line ART regimen	6 (16.6%)
CD4+ T-cell count during hospitalization[g], cells/mm^3	202 (168–446)
CD4+ T-cell difference between admission and previous six months (RIQ), cells/mm^3	202 (331)

ART: antiretroviral therapy; NA: not available. Data are presented as *N* (%) and median (quantile 25–quantile 75) for CD4 count. For CD4$^+$ T-cell difference, data are shown as median (interquartile range). Numbers in bold: significant values

[a]Cardiovascular disease includes congestive heart failure, coronary heart disease, arrhythmias, and cardiac devices.

[b]CD4+ T-cell count and HIV viral load before admission include previous 6 months before admission.

[c]Within 27 patients with available CD4 count.

[d]Within 28 patients with available viral load.

[e]Within 27 patients with data available.

[f]Antiretroviral therapy includes adherence during the last 3 months.

[g]Within 10 patients with available CD4 count during hospitalization.

age and chronic renal failure that deteriorated by COVID-19. Due to the ominous prognosis of these patients, treating physicians decided to withdraw life-sustaining interventions and provided terminal care. The third patient was younger but developed myocarditis and arrhythmias and died owing to a massive pulmonary thromboembolism. Two deaths were not directly attributed to COVID-19 (Kaposi sarcoma and abdominal sepsis). Mean time between symptom onset and death was 29 days.

People living with HIV who died had a higher prevalence of some comorbidities than patients who recovered (Table 4). Laboratory parameters, including HIV-related immune status, were not different between patients dying and those who did not, except for the NLR ratio > 3.04

Table 2. Clinical characteristics of patients with HIV and general population admitted with COVID-19.

Clinical characteristic	HIV cohort *N* = 36	General population *N* = 18285	OR	IC 95%
Signs and symptoms				
Cough	25 (69.4%)	11209 (61.3%)	1.43	0.70–2.91
Dyspnea	24 (66.7%)	8192 (44.8%)	**2.47**	**1.23–4.94**
Headache	15 (41.7%)	5990 (32.8%)	1.47	0.75–2.84
Fatigue or myalgia	15 (41.7%)	7607 (41.6%)	1.00	0.52–1.95
Chest pain	8 (22.2%)	2011 (11%)	**2.31**	**1.05–5.07**
Sore throat	10 (27.8%)	3218 (17.6%)	1.80	0.87–3.74
Anosmia	8 (22.2%)	878 (4.8%)	**5.66**	**2.57–12.45**
Ageusia	6 (16.7%)	750 (4.1%)	**4.69**	**1.95–11.30**
Abdominal pain	1 (2.8%)	1152 (6.3%)	0.43	0.06–3.13
Diarrhea	13 (36.1%)	1518 (8.3%)	**6.24**	**3.16–12.35**
Hypotension[a]	18 (50%)	NA	—	—
Fever ≥ 37.2°	32 (88%)	9088 (49.7%)	**7.42**	**(2.62–20.99)**
Lower SAP during hospitalization	97.6 (90–107)	NA	—	—
Lower DAP during hospitalization	58.9 (52–67.8)	NA	—	—
Laboratory and radiological characteristics of HIV-infected patients admitted with COVID-19				
Laboratory variable (unit; normal range)				
White blood cell count < 4 ($\times 10^9$ per L; NV 4–11)				16 (44.4%)
Lymphocyte count ($\times 10^9$ per L; NV 1–4)				950 (690–1227)
<1				21 (58.7%)
NLR				2.99 (1.82–7.72)
NLR ≥ 3.04				17 (48.6%)
Higher LDH during hospitalization (U/L; NV 135–214)				318 (237–471)
Higher C-reactive protein during hospitalization (mg/dL; NV < 0.5)				10.1 (2.95–19.65)
Higher serum ferritin during hospitalization (ng/mL; NV < 300)				790 (291–1606)
Higher D-dimer during hospitalization (ng/mL; NV < 500)				615 (340–1574)
Computed tomography scan (N = 28)				
Normal				4 (14.3%)
Consolidation				6 (21.4%)
Ground-glass opacities				20 (71.4%)
Crazy-paving pattern				2 (7.1%)
Bilateral findings				23 (82.1%)
Peripheral predominance of findings 21				(75%)

NV: Normal value; SAP: systolic arterial blood pressure; DAP: diastolic arterial blood pressure: LDH: Lactate dehydrogenase; NLR: Neutrophil-to-lymphocyte ratio (ratio with the lower lymphocyte during hospitalization and its concomitant neutrophils); NA: data not available. Data are represented as *N* (%) and median (quantile 25–quantile 75). *p* value < 0.05 is considered statistically significant. Numbers in bold: significant values.
[a]Hypotension was defined as systolic arterial pressure lower than 90 mmHg or diastolic arterial pressure lower than 60 mmHg.

which was significantly higher ($p = 0.044$) in patients who died.

Discussion

In the present study, we report the clinical course and outcomes for a cohort of 36 PLHIV that were hospitalized with COVID-19 during 2 months of the pandemic in Chile and compared them with data from Chilean general population hospitalized with SARS-CoV-2 infection. The HIV cohort had a higher proportion of men, which reflects the predominance of men over women in Chilean HIV epidemic.[22] Also PLHIV had a considerably lower age than those from the general population, with 72% of patients being less than 50 years, compared with only 33% for the general population. These results are similar to those reported from Geretti et al.,[12] which described significantly younger ages in PLHIV than HIV-negative people hospitalized with COVID-19.

We did not find significant differences with general population on comorbidities (except for cardiovascular disease that was higher in PLHIV); however, these results were not adjusted by age because disaggregated data for general population are not publicly available. Over a half of our patients from the PLHIV cohort had at least one comorbidity, which is high considering the low average age of our patients. This finding has been described previously[8,9] and may be related to the known premature aging and

Table 3. Major clinical outcomes of HIV-infected patients comparing with general population admitted with COVID-19.

	HIV cohort	General population		
	N = 36	*N* = 18285	OR	IC 95%
ICU admission	8 (22.2%)	2011 (11%)	**2.31**	**1.05–5.07**
Invasive MV	3 (8.3%)	1718 (9.4%)	0.87	0.27–2.85
Death (case fatality rate)	5 (13.9%)	4360 (23.8%)	0.52	0.20–1.33
Clinical outcomes of HIV-infected patients admitted with COVID-19				
Outcome				
Severe illness				16 (44.4%)
Oxygen requirements				21 (58%)
Thromboembolic events				2 (5.6%)
Myocarditis				1 (2.8%)
Myocardial ischemia				0 (0%)
Arrhythmias				2 (5.6%)
Acute kidney injury				4 (11%)
MOF				7 (19%)
ECMO				0 (0%)
Length of hospital stay, days				9.5 (7.75–17.5)

ICU: Intensive care unit; MV: Mechanical ventilation; MOF: Multiorgan failure; ECMO: Extracorporeal membrane oxygenation. Data are presented as *n* (%) or median (quantile 25–quantile 75). Numbers in bold: significant values.

frailty[23] that HIV infection produces, leading to the development of comorbidities at younger ages.

Regarding clinical symptoms, the group of Geretti and colleagues reported that some symptoms were higher in the group of PLHIV comparing with a group of HIV-negative patients.[12] A similar finding was found in our cohort of PLHIV, where a significantly higher proportion of fever, dyspnea, chest pain, anosmia, ageusia and diarrhea was found, compared to the general population.[16] We cannot assure that HIV status per se explains this higher presence of COVID-19 symptoms, but there are reports that show chronic lung inflammation even after effective ART,[24,25] which could explain some of these findings. Another explanation could be a bias regarding information collection since our patients were directly consulted about symptoms, while data from general population were extracted from epidemiological reports, so this issue could be underreported.

Lymphocytes expressing CD4 are significantly reduced in patients with more severe viral infection and in SARS-CoV-2 infection.[26] These findings were corroborated in our study, where we observed a significantly greater decrease in CD4 lymphocytes count between preadmission and hospitalization in patients who met the definition of severe COVID-19 compared with patients that had a more benign course of the disease. Considering that the NLR was identified as a powerful predictive and prognostic factor for severe COVID-19, we looked for NLR, using the cutoff of 3.04.[21] We found that this ratio was significantly higher in our patients with severe COVID-19 and deceased patients. Therefore, this ratio seems to be a useful prognostic tool in patients infected with SARS-CoV-2.

Severe illness in our cohort was 44%. This is similar than another report of hospitalized PLHIV with COVID-19[9,27] but higher than other multi-centre report.[28] Our cohort required ICU admission in 22% of cases, similar to the cohort of Shalev from New York.[29] However, we saw lower rates of need for mechanical ventilation (8.3%) and in-hospital mortality (13.9%) than other cohorts of PLHIV.[10,29] These cohorts report higher age of HIV patients, which in part, could explain this difference.[8,29]

Even if we found a significantly higher ICU admission rate in the HIV group than the general population, we found no overall differences in the need for mechanical ventilation and mortality. This finding must be observed with caution since our results are not adjusted by age, sex, or comorbidities because disaggregated data are not available in Chilean epidemiological reports and because the number of cases is small.

Interestingly, PLHIV who developed severe COVID-19, did not differ in age, gender, ART, baseline HIV CDC stage, undetectable HIV viral load before admission, CD4 cell count before admission or during hospitalization, and comorbidities to those PLHIV who had a more benign course of the disease. People living with HIV who died had a higher prevalence of some comorbidities such as hypertension and cardiovascular disease than patients who survived; however, HIV-related immune status or other HIV variables such as being on ART or having an undetectable

Table 4. Comparison of clinical characteristics and HIV parameters between recovered and deceased HIV-infected patients admitted with COVID-19.

Variable	Recovered (*N* = 31)	Deceased (*N* = 5)	*p*
Mean age, years (range)	41 (32–48)	57 (39–71)	0.090
Male gender	29 (93.5%)	4 (80%)	0.370
Baseline CD4 count before admission[a], cells/mm^3	564 (389–691)	543	0.417
Inhospital CD4 count, cells/mm^3	199 (148–442)	344	0.482
CD4 count decreased between preadmission[a] and at hospitalization, cells/mm^3	103 (316–329)	320	0.229
HIV viral load < 50 copies RNA/mL before admission[a]	15 (65.2%)	4 (80%)	0.999
On ART	26 (83.9%)	4 (80.8%)	0.99
Median time since HIV diagnosis, years	7 (4–12)	16 (3–17)	0.436
At least one comorbidity	13 (41.9%)	4 (80%)	0.167
Diabetes	2 (6.5%)	2 (40%)	0.084
Hypertension	3 (9.7%)	3 (60%)	**0.024**
Obesity	4 (12.9%)	1 (20%)	0.549
Cancer	2 (6.5%)	1 (20%)	0.370
Smoking	10 (32.3%)	0 (0%)	0.293
Cardiovascular disease[b]	1 (3.2%)	3 (60%)	**0.006**
Chronic obstructive pulmonary disease	1 (3.2%)	0 (0%)	0.99
Pharmacological immunosuppression	1 (3.2%)	1 (20%)	0.262
Chronic liver disease	1 (3.2%)	0 (0%)	0.99
Chronic kidney disease	2 (6.5%)	2 (40%)	0.084
Time from first symptoms to hospital admission	6 (4–8)	10	**0.043**
NLR ≥ 3.04	12 (43%)	5 (100%)	**0.044**
Length of hospital stay, days	9 (7–16)	19	0.017
Oxygen requirement or PaO2/FiO2 <300	16 (51.6%)	5 (100%)	0.062
Severe illness	11 (35.5%)	5 (100%)	**0.012**

ART: antiretroviral therapy; NLR: Neutrophil-to-lymphocyte ratio (ratio with the lower lymphocyte during hospitalization and its concomitant neutrophils). Data are presented as *n* (%) or median (quantile 25–quantile 75). For $CD4^+$ T-cell difference, data are shown as median (interquartile range). Quantiles are not shown in deceased patients because of the small sample size. Numbers in bold: significant values.
[a]CD4+ T-cell count and viral load before admission includes last 6 months. *p* value < 0.05 is considered statistically significant.
[b]Cardiovascular disease includes congestive heart failure, coronary heart disease, arrhythmias, and cardiac devices.

viral load were not different between patients dying and those who did not.

Our study has limitations. First, as all of the patients in this study were hospitalized, the reported COVID-19 severity may not be representative of the risk for the entire HIV population. Furthermore, it is notable that despite including a large number of hospitals along the country, with a number of PLHIV under follow-up of approximately 27000, the group of patients admitted is small. Third, data of general population available in epidemiological reports may have included patients with HIV, although HIV prevalence in the general population in Chile is low (0.5%). Last, data from epidemiological reports are not disaggregated, so findings reported here could not be adjusted by age, sex, or comorbidities. Nevertheless, we have provided a comprehensive report of the reality of PLHIV hospitalized with SARS-CoV-2 coinfection in our country. Strengths include a broad comparison with general population, laboratory-confirmed SARS-CoV-2 diagnosis in all PLHIV/COVID-19, and inclusion of all important HIV variables such as CDC stage at baseline, nadir CD4 count, CD4 count before and at admission, been on ART, and HIV viral load at admission. To the best of our knowledge, this is the first report of PLHIV coinfected with SARS-CoV-2 in Latin America.

Conclusions

The findings of this study do not support that PLHIV have a higher risk for aggravation or death from COVID-19 than the general population. We are still learning and understanding about the interactions between HIV and SARS-CoV-2. Further studies should clarify the effect of HIV on the overall risk of COVID-19.

Chilean HIV/Covid-19 Study Group

María Elena Ceballos (Department of Infectious Diseases. School of Medicine. Pontificia Universidad Católica de Chile), Patricio Ross (Department of Internal Medicine. School of Medicine. Pontificia Universidad Católica de Chile), Martín Lasso (Infectious Disease Specialist,

Hospital Sótero del Río), Isabel Dominguez (Infectious Disease Specialist, Clínica Santa María), Marcela Puente (Infectious Disease Specialist, Hospital Del Salvador), Pablo Valenzuela (Infectious Disease Specialist, Hospital DIPRECA), Margarita Enberg (Infectious Disease Specialist, Hospital Regional de Antofagasta Dr Leonardo Guzmán), Michel Serri (Infectious Disease Specialist, Hospital FACH and Clínica Tabancura), Rodrigo Muñoz (Infectious Disease Specialist, Hospital Clínico Magallanes), Yazmín Pinos (Infectious Disease Specialist, Hospital Base San José Osorno), Macarena Silva (Infectious Disease Specialist, Hospital San Borja Arriarán), Matías Noguera (Medical student, Pontificia Universidad Católica de Chile), Angélica Domínguez (Department of Public Health, Faculty of Medicine, Pontificia Universidad Católica de Chile), Francisco Zamora (Infectious Disease Specialist, Hospital Barros Luco Trudeau), Mabel Aylwin (Infectious Disease Specialist, Hospital Padre Hurtado), Carolina Chaín (Infectious Disease Specialist, Hospital Hernán Henriquez Aravena, Temuco), Olga López (Infectious Disease Specialist, Dr. Ernesto Torres Galdames), Loreto Rojas (Infectious Disease Specialist, Hospital Base de Puerto Montt), Ignacio Delama (Infectious Disease Specialist, Hospital Clínico Regional Valdivia), Rosana Benitez (Infectious Disease Specialist, Clínica Dávila y Clínica Bupa), Leonardo Siri (Infectious Disease Specialist, Hospital La Florida), Alvaro Rojas (Infectious Disease Specialist, Clínica San Carlos de Apoquindo), Gonzalo Wilson (Infectious Disease Specialist, Hospital Carlos Van Buren).

Authors' note

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Authors' contributions

MEC conceived, designed this investigation, and wrote the paper. PR helped to design the scheme of the investigation. PR, MN, FZ, ML, ID, MP, PV, MSerri, MSilva, ME, YP, and RM collected the original data. MEC and AD analyzed the data. PR contributed writing the paper.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethics committee approval

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ORCID iDs

Maria Elena Ceballos https://orcid.org/0000-0002-5569-6485
Matías Noguera https://orcid.org/0000-0002-6438-9735

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