

## LETTER TO THE EDITOR

## COVID-19 crossing paths with AIDS in the homeless

To the Editor,

The coronavirus disease of 2019 (COVID-19) pandemic has created new challenges and magnified existing ones for immunocompromised individuals who may be at risk for worse clinical outcomes. Severe COVID-19 has been associated with a hyperimmune response characterized by a surge in cytokine release described as a cytokine release syndrome (CRS).<sup>1</sup> Among immunocompromised patients, the inability to mount an immune response may be protective against a poor outcome.

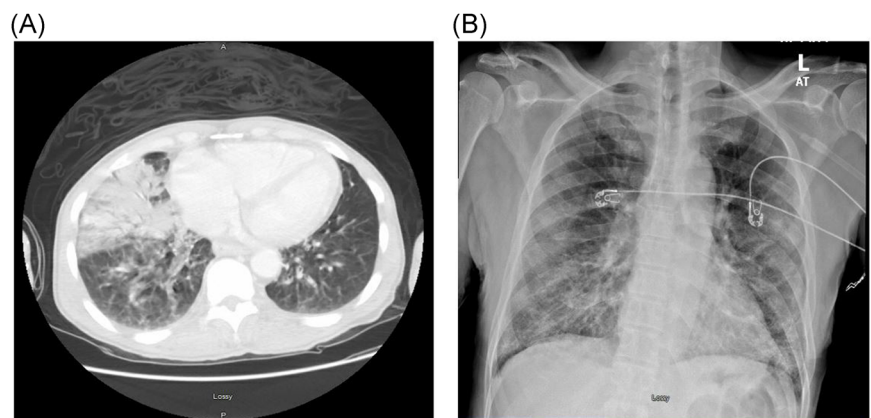
For individuals living in homeless shelters, the lack of testing and assistance can contribute to a rapid spread of new outbreaks, which may limit a region's ability to control the pandemic.<sup>2</sup> In this report, we describe the clinical course of two homeless patients with a history of acquired immune deficiency syndrome (AIDS) who were admitted for COVID-19. The details of their immunological profile and in-hospital outcomes are described and the pathophysiological consequences of immune dysfunction in the setting of COVID-19 discussed.

Patient 1 is a 51-year-old homeless male with a previous diagnosis of AIDS, not on antiretroviral therapy (ART), polysubstance abuse who had presented to the emergency department due to alcohol intoxication. He had denied any fevers, cough, or dyspnea before his presentation after he was initially stabilized. His vital signs were stable upon presentation except for an oxygen saturation of 86% on ambient air for which he was transitioned to nasal canula for oxygen therapy but escalated to 15 L of non-rebreather to maintain an oxygen saturation >92%. Chest imaging showed bilateral infiltrates on chest x-ray and ground glass opacities on computed tomography. A nasopharyngeal swab was positive for the novel severe acute respiratory coronavirus (SARS-CoV-2) (Figure 1). Sputum cultures showed concomitant infection with *Streptococcus pneumoniae*. His detailed clinical

course is given in Table 1. The patient reported a nadir CD4 cell count of 10.2 cells per uL and a corresponding HIV RNA PCR of 26 900 copies per mL. Over the course of his stay, he mounted a maximum temperature of 100.8°F on hospital day 3. He was managed conservatively with supportive oxygen therapy, azithromycin, and piperacillin tazobactam for the superimposed infection. He was not started on either chloroquine or hydroxychloroquine due to an increased risk of arrhythmia with combination therapy with azithromycin. He was discharged to a shelter uneventfully on hospital day 10.

Patient 2 is another homeless male, aged 63 years, with a previous history of AIDS, not on ART, hypertension and hyperlipidemia who presented with a 3-day duration of worsening nonproductive cough, subjective fevers, myalgias, and dyspnea. He self-reported exposure to individuals who had been previously diagnosed with SARS-CoV-2 infection. A nasopharyngeal swab specimen was consistent with the SARS-CoV-2 infection. Upon presentation to the emergency room, he was afebrile and hemodynamically stable saturating at 93% ambient air. Further details on his clinical presentation including baseline and peak laboratory findings, and imaging characteristics are given in Table 1 and Figure 1, respectively. His immunological profile showed a CD4 cell count of 116/uL, and a HIV RNA PCR viral load of 2 540 000 copies per mL. He received supportive treatment with nasal canula oxygen therapy, acetaminophen for headaches and antibiotic treatment for the pneumonia. Sputum cultures were negative for other infectious etiology of pneumonia. The patient was discharged to a homeless shelter on hospital day 7 with no COVID-19-associated complications.

Although limited by few studies and case series, the current evidence regarding the association between HIV and COVID-19 shows that people with HIV are not at an increased risk for worse



**FIGURE 1** Computed tomography chest imaging showing a right lung middle lobe consolidation with bilateral ground glass opacity (A) in patient 1 and an anterior posterior chest X-ray (B) showing bilateral lower lobe infiltrates

**TABLE 1** Baseline demographic, clinical, and treatment characteristics of patients

Variable	Patient 1	Patient 2
Age, y	51	63
Sex	M	M
BMI, kg/m <sup>2</sup>	22	18
Race	AA	AA
Symptoms at presentation		
Fever	No	No
Dyspnea	Yes	Yes
Cough	No	Yes
Nausea/emesis	Yes	No
Myalgias	Yes	No
Vital signs baseline (peak)		
Temp	98.8 (100.8)	98.3 (98.7)
RR, per min	17 (25)	17 (20)
HR, per min	66 (99)	94 (110)
Oxygen Saturation	86 (85)	90 (90)
SBP, mm Hg	153 (180)	130 (143)
Baseline (peak) lab. values		
WBC, ×10 <sup>9</sup> /L	5.1 (11)	2.4 (5.7)
Lymphocytes, ×10 <sup>9</sup> /L	18	34 (49)
Hemoglobin, g/dL	12.7 (12.9)	12.8 (13)
Platelets, ×10 <sup>9</sup> /L	88 (155)	454 (459)
AST, units/L	108 (131)	53
ALT, units/L	78 (84)	43
ALP, IU/L	135 (135)	92
BUN, mg/dL	11 (24)	33
Creatinine, mg/dL	0.8 (0.9)	1.6
CRP, mg/L	...	4.95 (4.95)
ESR, h	...	131 (131)
Ferritin, ng/mL	742 (1264)	2292 (2292)
Superimposed infection	Streptococcus pneumoniae	None
HAART regimen	None	None
Immunological profile		
CD4 cell count	10.2	116.3
HIV RNA PCR copies	26 900	2 540 000
Management		
Hydroxychloroquine	NA	NA
Corticosteroids	NA	NA
Tocilizumab	NA	NA
Antibiotics	Azithromycin, bactrim, zosyn	Bactrim
Other	...	Zinc sulfate, vitamin C
Outcomes		
Length of hospitalization, d	10	7
ICU admission, n (%)	0 (0)	0 (0)


**TABLE 1** (Continued)

Variable	Patient 1	Patient 2
Mechanical ventilation, n (%)	0 (0)	0 (0)
In-hospital death, n (%)	0 (0)	0 (0)
Readmission	0 (0)	0 (0)

Abbreviations: ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase; BMI, Body mass index; BUN, blood urea nitrogen; CD4, cluster of differentiation 4; CRP, C reactive protein; HAART, highly active antiretroviral therapy; HIV RNA PCR, human immunodeficiency virus ribonucleic acid polymerase chain reaction; HR, heart rate; ICU, intensive care unit; RR, respiratory rate; SBP, systolic blood pressure; SR, erythrocyte sedimentation rate; WBC, white blood cell count.

outcomes.<sup>3-7</sup> The poor clinical outcomes in COVID-19 have been associated with laboratory features of CRS. These include abnormal levels of inflammatory cytokines (IL-6, IL-10, IL-2, and IFN- $\gamma$ ) and a decrease in CD4 and CD8 cells.<sup>1</sup> In chronic untreated HIV infection, progression to AIDS is inevitable and plasma levels of inflammatory mediators (IL-6, IFN- $\gamma$ , and TNF) are typically elevated.<sup>8</sup> The reason for this sustained elevation in cytokines is unclear; however, it is likely that a chronic hyperinflammatory state may serve a protective role against COVID-19-related complications. A better understanding of ways in which these mediators may drive immune homeostasis is key to the management of these individuals. In both patients, the initiation of ART was withheld during their acute state of infection with SARS-CoV-2. Instead, ART was initiated weeks after their successful discharge to a homeless shelter.

In conclusion, we describe our experience with AIDS and COVID-19 in two homeless patients who were expected to be at an increased risk for worse outcomes. Despite their poor immunological profile, their clinical course was uncomplicated, and both were successfully discharged alive with scheduled followup and monitoring.

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