Long COVID in people living with HIV

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Purpose of review
It is now recognized that SARS-CoV-2 infection can have a long-term impact on health. This review summarizes the current state of knowledge regarding Long COVID in people living with HIV (PLWH).

Recent findings
PLWH may be at elevated risk of experiencing Long COVID. Although the mechanisms contributing to Long COVID are incompletely understood, there are several demographic and clinical factors that might make PLWH vulnerable to developing Long COVID.

Summary
PLWH should be aware that new or worsening symptoms following SARS-CoV-2 infection might represent Long COVID. HIV providers should be aware of this clinical entity and be mindful that their patients recovering from SARS-CoV-2 infection may be at higher risk.

Keywords
coronavirus disease 2019, HIV, Long coronavirus disease, postacute sequelae of severe acute respiratory syndrome coronavirus-2 infection, severe acute respiratory syndrome coronavirus-2

INTRODUCTION

In the spring of 2020, shortly after the emergence of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), individuals began to report prolonged symptoms or unexpected changes in health after coronavirus disease 2019 (COVID-19) [1]. The number, diversity, and duration of symptoms was unprecedented, and the presence of this syndrome in people who had what was considered to be nonsevere acute illness (i.e. not requiring hospitalization) was unexpected. Patient anecdotes were reported widely in social and popular media, and a group of patient-researchers began conducting and publishing large internet-based surveys in the first attempts to characterize the syndrome. The medical research community followed their lead and the first peer-reviewed report of Long COVID was published in July 2020 [2]. After months of intense advocacy on the part of people experiencing this condition, the US Congress passed a more than $1 billion initiative to study Long COVID [3] and the Biden administration has also made commitments to these efforts [4]; similar programs have been developed in other settings [5,6]. Since that time, major efforts have been made to understand the epidemiology, natural history, biological mechanisms, and impact of Long COVID.

Long COVID, the term now favored by US research agencies [7], is one of several terms currently used to describe health problems that occur after COVID-19 [8]. Other names for the condition include post-COVID conditions, long-haul COVID, chronic COVID, post COVID-19 syndrome, and postacute sequelae of SARS CoV-2 infection (PASC). Ultimately, each of these serve as umbrella terms encompassing a range of conditions in the postacute period, ranging from established sequelae of severe COVID-19 (e.g. post-ICU and posthospitalization syndromes), incident medical diagnoses (e.g. pulmonary embolism, myocardial infarction, stroke, diabetes mellitus), mental health conditions (e.g. depression, anxiety), social conditions (e.g. isolation), as well as symptoms not attributable to another cause. The latter is most representative of Long COVID as described by individuals experiencing the condition.

In this review, we summarize the current state of knowledge regarding Long COVID in people living with HIV (PLWH). Although current data on the

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KEY POINTS

- Some people do not return to their baseline state of health and develop additional complications following SARS-CoV-2 infection.

- Although data are limited, several studies have reported an increased prevalence of Long COVID symptoms and other postacute sequelae of SARS-CoV-2 infection among PLWH compared with HIV-seronegative individuals.

- The risk factors for and pathophysiology of Long COVID remain incompletely understood.

- There are a number of reasons why PLWH might be vulnerable to developing Long COVID, including sociodemographic factors, medical comorbidities, and dysregulation of immunologic and/or physiologic systems that could be exacerbated by acute and postacute SARS-CoV-2 infection.

- HIV providers should assess patients for complications of SARS-CoV-2 infection when encountering them during the postacute period.

mechanisms of Long COVID are limited, we describe why PLWH might be vulnerable to developing this condition.

EPIDEMIOLOGY AND NATURAL HISTORY OF LONG COVID

Cohort studies, epidemiologic studies and systematic reviews suggest that up to 70% of individuals experience some form of Long COVID following SARS-CoV-2 infection, with smaller proportions (5–10%) experiencing severe, debilitating forms of this condition [9–13]. Prevalence estimates vary widely due to differences in case definition, which include differences in how Long COVID symptoms are ascertained and defined, particularly with regard to the inclusion of special populations (e.g. hospitalized individuals), the follow-up period (e.g. weeks or months), and the handling of symptoms that may have predated COVID-19 (e.g. exclusion of these symptoms or inadequate information to exclude them). In the United States, data from the Veterans Affairs (VA) Healthcare System has been instrumental in understanding the epidemiology of Long COVID in veterans [14–17], although a number of high-quality community-based studies have been undertaken [2,18–23]. Outside of the United States, there have been several notable efforts in the United Kingdom, Europe, Africa, and Asia [24–32]. Although the epidemiology has never been precisely understood and is potentially affected by the emergence of new variants [33], preexisting

vaccine-related and infection-related immunity [16,34], and the availability of therapeutics for acute infection [17,35], it is now accepted that even if the lower range of the prevalence estimate is accurate, the absolute number of people affected is large because of the scale of the pandemic. As a result, the economic, social, and healthcare impact is likely to be substantial [36].

Many initial studies of Long COVID focused on its natural history [13,14,37–40]. The most inclusive studies of Long COVID have identified dozens of symptoms associated with this condition [41,42], although it is unlikely that all symptoms experienced by a person following COVID-19 are directly attributable to SARS-CoV-2 infection or its sequelae. For this reason, separating related from unrelated symptoms is a major challenge in Long COVID research and clinical care [38]. In addition, Long COVID is characterized by substantial within-individual variability [38], with the waxing and waning of symptoms over time in some individuals complicating measurement. Clinical trajectories are highly variable, with some individuals improving over time, others plateauing, and some occasionally worsening (although substantial worsening is felt to be rare); many cases persist for months or even years. Finally, there is increasing acceptance that Long COVID is likely to constitute multiple syndromes or symptom clusters [39,40,43,44], including cardiopulmonary, neurocognitive, gastrointestinal, and musculoskeletal phenotypes. Some of these may in addition overlap with other postinfectious syndromes such as: myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS), postural orthostatic tachycardia syndrome (POTS), mast cell activation syndrome (MCAS), and postexertional malaise syndromes (PEM). Defining these phenotypes is of increasing importance as it may be the case that different pathophysiologic mechanisms drive different phenotypes. Although they continue to be identified, there are now several well established risk factors for Long COVID [14,18,26,45]. These include female sex, older and middle age, lower socioeconomic status and access to healthcare, and the presence of certain comorbid medical conditions including diabetes and obesity. Preexisting anxiety and depressive symptoms have also been associated with the development of Long COVID in some but not all studies [46,47] and mood symptoms may co-occur with physical symptoms in some individuals experiencing this condition.

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PLWH represent a specific group of interest and may be at increased risk for both post-COVID

complications as well as Long COVID. However, the data thus far are extremely limited. The presence of some PLWH were reported in early Long COVID cohorts but they were not characterized in detail [38]. Instead, the first in-depth descriptions of Long COVID in PLWH were case series. One series of 94 PLWH in Western India who were unvaccinated at the time of COVID-19 reported that 44% of individuals had at least one Long COVID symptom 30 or more days postinfection [48]. Cough and fatigue were the most commonly reported symptoms. This cohort was composed primarily of middle-aged men (median age 51, 73% men) and most (91%) were on suppressive antiretroviral therapy (ART). In a similar case series from an HIV clinic in Padua, Italy, 75 PLWH on suppressive ART who were unvaccinated at the time of symptomatic COVID-19 were followed for a median time of 6 months [49]. The cohort was predominantly constituted of middle-aged men (>80% men), with good immune reconstitution (median CD4+ T-cell count >500 cells/μl). One or more COVID-attributable symptoms or sequelae 4 or more weeks after symptom onset were reported in 27% of individuals in this study. The most common symptoms were fatigue, dyspnea on exertion, and recurrent headache. In both of these case series, those who had Long COVID symptoms were more likely to previously have had moderate-to-severe COVID-19.

Several attempts have also been made to determine whether PLWH have an increased likelihood of Long COVID compared with HIV-seronegative people. An analysis of 530 individuals who had presented to the emergency department or were hospitalized at NY Presbyterian Hospital-Cornell University in the spring of 2020 demonstrated that PLWH were more likely to report one or more Long COVID symptoms 12 months after COVID-19, after adjusting for age, sex, race, poverty, comorbidities, and severity of acute COVID-19 [adjusted risk ratio 1.75, 95% confidence interval (CI) 1.14–2.69] [50]. However, the cohort included only 10 PLWH (<2% of the total population studied).

Two more recent studies in the University of California, San Francisco-based Long-term Impact of Infection with Novel Coronavirus (LIINC) cohort built upon these findings. In the first [51■], 39 unvaccinated PLWH were compared with 43 unvaccinated HIV-seronegative individuals. Following adjustment for age, sex, hospitalization status, and time since infection, the authors found that PLWH had 4.01× the odds of reporting at least one COVID-attributed symptom and 2.72× the odds of reporting three or more COVID-attributed symptoms at a median of 4 months post-COVID. Certain symptoms, including fatigue, gastrointestinal symptoms, neurocognitive symptoms, and sleep problems were reported more commonly in PLWH. The authors identified lower levels of SARS-CoV-2-specific memory CD8+ T cells and higher levels of PD1+ SARS-CoV-2-specific CD4+ T cells among PLWH but did not find significant differences according to PASC status. They also found that interleukin-6 (IL-6) and IP-10 had a modest association with Long COVID overall, and that IP-10 levels in particular were associated with Long COVID symptoms in PLWH. A second study from this cohort [52■] evaluated 54 PLWH as part of an analysis of 280 individuals with prior SARS-CoV-2 infection to assess the contribution of chronic viral infections such as Epstein–Barr virus (EBV), cytomegalovirus (CMV), and HIV. It found using adjusted models that PLWH had increased odds of neurocognitive Long COVID symptoms at 4 months, when controlling for demographic factors, serologic evidence suggesting recent EBV reactivation, and CMV serostatus. This suggested that HIV infection itself was an independent risk factor for Long COVID in this cohort.

Finally, a recent study that has not yet been peer-reviewed has attempted to ascertain the likelihood of postacute sequelae of SARS-CoV-2 using data from 69 US healthcare organizations sourced from the TriNetX federated health research network [53■■]. The authors evaluated nearly 29 000 individuals between January 2020 and September 2022 who had ICD10 data indicating both SARS-CoV-2 infection and HIV infection. They compared PLWH with a similarly sized cohort of HIV-negative individuals, propensity matching for age, sex, race, ethnicity, BMI, and select comorbid conditions. They found a higher odds ratio in PLWH for several new diagnoses in an undefined post-COVID period: 'mental disorders', malignancy, heart disease, diabetes, and thrombosis. They also examined Long COVID symptoms and found body aches, headaches, gastrointestinal symptoms, respiratory symptoms, fatigue, and cognitive impairment to be more likely to be reported among PLWH. This study provides valuable information from a large US database and shows that certain symptoms and new onset health conditions were more likely to occur in PLWH in a mostly unvaccinated cohort after SARS-CoV-2 infection.

Based on these analyses, it remains possible and even likely, but not definitively proven, that HIV is a risk factor for Long COVID. Whether some of the excess risk for symptoms in PLWH can be attributed to factors such as smoking or comorbid conditions more frequent in this population requires studies with PLWH who do not have COVID-19 as a control group. Below, we outline the current research on the

pathobiology of Long COVID and delineate why PLWH may be more susceptible to develop postacute symptoms and sequelae [54] (Fig. 1).

RISK FACTORS FOR AND MECHANISMS OF LONG COVID: WHY PEOPLE LIVING WITH HIV MIGHT BE VULNERABLE

There is substantial overlap between the clinical risk factors for Long COVID and the demographics and clinical comorbidities of PLWH. First, severity of acute infection has been repeatedly identified as a predictor of Long COVID. Although initial small studies suggested that PLWH did not exhibit worse outcomes during acute SARS-CoV-2 infection [55–59], larger studies have shown that HIV status is a risk factor for severe outcomes even among the virologically suppressed [60–67]. This risk is most pronounced in those with comorbidities, viremia, and/or poor immune reconstitution [63,68–72]. Second, PLWH commonly exhibit metabolic comorbidities such as diabetes and obesity, as well as cardiovascular disease [73–81], which are also risk factors for Long COVID. Furthermore, other possible contributors to Long COVID or exacerbating factors such as mental health conditions, substance use, and lower socioeconomic status are common among PLWH [82–85]. People with well controlled HIV infection can also exhibit neurocognitive impairment despite ART [86,87]; this HIV-associated neurocognitive disorder (HAND) in some ways resembles the types of deficits present in neurocognitive Long COVID [88,89]. Because of these issues, PLWH may be at increased risk for developing Long COVID.

Acute COVID-19 is a highly inflammatory syndrome [90], and SARS-CoV-2 convalescent plasma donors have higher levels of inflammation than historical donors [91]. Multiple studies have now identified immune dysregulation and inflammation, specifically pathways involving IL-6, TNF-alpha, and in some cases IL-1B, among others, in those with Long COVID in comparison to individuals with full recovery [92–101]. Whether these
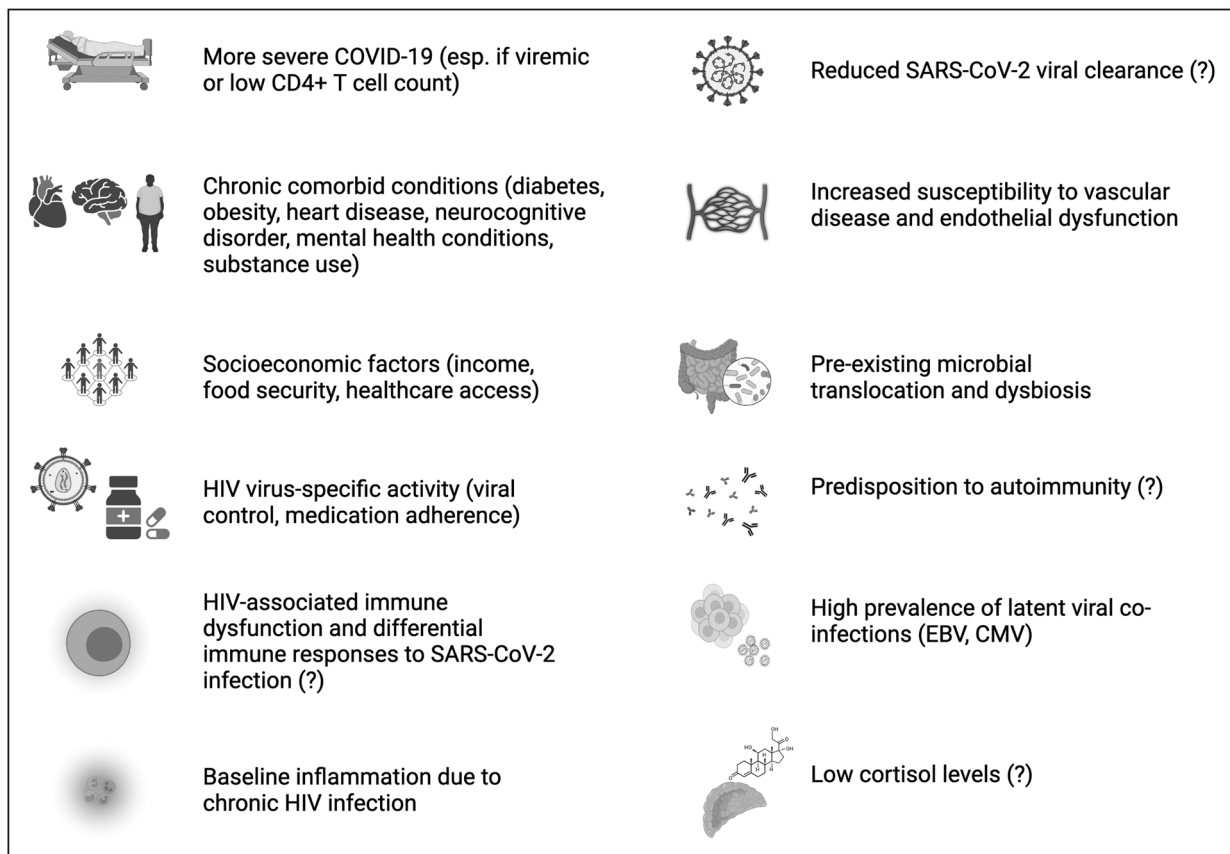

- More severe COVID-19 (esp. if viremic or low CD4+ T cell count)
- Chronic comorbid conditions (diabetes, obesity, heart disease, neurocognitive disorder, mental health conditions, substance use)
- Socioeconomic factors (income, food security, healthcare access)
- HIV virus-specific activity (viral control, medication adherence)
- HIV-associated immune dysfunction and differential immune responses to SARS-CoV-2 infection (?)
- Baseline inflammation due to chronic HIV infection
- Reduced SARS-CoV-2 viral clearance (?)
- Increased susceptibility to vascular disease and endothelial dysfunction
- Pre-existing microbial translocation and dysbiosis
- Predisposition to autoimmunity (?)
- High prevalence of latent viral co-infections (EBV, CMV)
- Low cortisol levels (?)

FIGURE 1. Potential contributors to Long COVID in people living with HIV. People living with HIV (PLWH) may be at elevated risk for Long COVID and other postacute sequelae following SARS-CoV-2 infection. While the risk factors for and mechanisms of such sequelae are incompletely understood, there are a variety of clinical, demographic, immunologic, and physiologic factors that could explain why PLWH might experience increased vulnerability to complications in the postacute phase of SARS-CoV-2 infection. '?' indicates potential contributors with limited strength of evidence. Created using Biorender.

perturbations drive Long COVID symptoms or are simply a result of other pathophysiologic processes as discussed below is unknown, as studies of immunomodulatory therapy in Long COVID have not yet been performed. However, it is well understood that PLWH – even those with viral suppression – have increased inflammation at baseline [102–106]. Therefore, they might be at higher risk for Long COVID following an additional SARS-CoV-2-initiated inflammatory insult. Although the cause of immune dysregulation in Long COVID is unknown, several mechanisms which may drive at least some cases of Long COVID have been proposed to contribute.

Recently, SARS-CoV-2 viral antigen persistence has been identified in a subset of individuals with Long COVID [107–111]. If persistence of viral genetic material or antigen drives Long COVID, it is possible that PLWH could exhibit reduced SARS-CoV-2 viral clearance from tissue reservoirs because of HIV-induced loss or dysfunction of tissue-specific immune cells. This could potentially be driven by dynamics of the humoral and cellular immune responses which in turn might contribute to Long COVID [94,96,98,99,112–118], although the role of the SARS-CoV-2 immune response in Long COVID and whether adaptive immune responses to SARS-CoV-2 differ by HIV status are unclear [51■,119–123].

Other proposed immunologic mechanisms of Long COVID could exacerbate baseline levels of immune dysregulation in PLWH. PLWH are known to have higher baseline levels of endothelial dysfunction [74]. Microvascular dysfunction, platelet activation, and clotting are currently under investigation as potential drivers of Long COVID [124–128]. It is believed that these processes could drive organ dysfunction, which manifests as inflammation and symptoms. Similarly, microbial translocation because of reduced integrity of the blood–gut barrier is another plausible mechanism of Long COVID supported by data from a recent study [129], as is dysbiosis [130–134]. Disruption of the gut epithelial barrier and microbiome dysbiosis are associated with HIV [135], and so PLWH might be at increased risk of Long COVID because of gut dysfunction. PLWH may also be primed to develop autoreactive immunity [136–138], which has been demonstrated during and just after acute SARS-CoV-2 infection [96,139–143], although the data in Long COVID are mixed [96,97,140,142,144,145].

People with HIV are also known to have high rates of human herpesvirus co-infection such as EBV and human CMV [146–148]. Several recent studies identified that direct viral measurement during acute COVID-19 or indirect serologic evidence suggesting recent EBV reactivation during the postacute phase was associated with increased odds of Long COVID [52■,96,149], and PLWH may be more susceptible to reactivation of this virus because of impairment of surveillance by CD8+ T cells [147]. Interestingly, CMV seropositivity was associated with lower odds of neurocognitive Long COVID symptoms in one Long COVID study in models controlling for HIV serostatus [52■]. In addition, HIV-specific factors including viral activity and immune responses could contribute. For example, an early study of PLWH after COVID-19 showed an increased frequency of viral blips of unclear clinical significance in the postacute period in comparison with historical controls [150]. This suggests that there may be unique mechanisms of Long COVID in PLWH, and therefore dedicated mechanistic studies of PASC and Long COVID in this subpopulation are warranted.

Finally, two independent studies identified an association of low cortisol levels with Long COVID [96,97]. The adrenal cortex, which is the site of cortisol production, is highly vascular and is a known site of SARS-CoV-2 infection [151,152]. Many symptoms of Long COVID overlap with those of adrenal insufficiency [153], although most adrenal insufficiency among PLWH in the modern ART era is subclinical [154,155].

MANAGEMENT OF LONG COVID

Although several clinical trials are now underway, there is no accepted treatment for Long COVID, and a detailed review of Long COVID management practices is beyond the scope of this article. The US CDC provides diagnostic guidelines [156], and other management guidelines for the primary care setting are available [157]. Generally, Long COVID management requires a holistic, patient-centered approach, focused on ruling out other medically treatable conditions that might mimic symptoms of Long COVID, detection of treatable complications of COVID-19, management of unexplained symptoms, and referral as appropriate for more intensive testing. For PLWH, evaluation of medication adherence and adverse effects, confirmation of virologic suppression and stable immune status, and attention to comorbid medical and psychosocial conditions are also important. Many nascent Long COVID clinics utilize a hub-and-spoke model to deliver coordinated care through a core team that partners with many medical subspecialties (e.g. pulmonology, neurology, cardiology, and psychiatry) and support services (e.g. pharmacy, physical and occupational therapy, social work, and behavioral health) [158]; this approach has for decades been used in many HIV and primary care clinics. Finally, we also note that PLWH continue to face substantial

stigmatization [159,160], and Long COVID and other poorly understood medical conditions can also result in stigma [161,162], although the scope of the problem has not been fully explored and few studies have addressed the qualitative experiences of people with HIV also experiencing Long COVID [163]. It is particularly important that HIV providers be aware of these issues and how they might complicate care for PLWH experiencing Long COVID.

CONCLUSION

Although Long COVID is now a well established clinical entity and public health challenge, many questions remain. More research examining the epidemiology of Long COVID in PLWH, in particular whether PLWH are at higher risk for developing Long COVID and whether the clinical manifestations or natural history differ between HIV-seropositive and HIV-seronegative people are needed. In order to distinguish the burden of HIV-specific morbidity among PLWH from that caused by COVID-19, studies of the impact of COVID-19 on the long-term health of PLWH may also need to make comparisons with PLWH who did not develop COVID-19 during the study interval. In addition, ongoing efforts will be needed to determine whether the development of Long COVID in PLWH is influenced by vaccination status, acute COVID-19 treatment, and the emergence of new variants to the same degree that it might be in HIV-seronegative individuals. As the mechanisms of Long COVID in the general population are further delineated, these need to be examined in PLWH, and attention needs to be paid to contributors that might be unique or more important in driving Long COVID among PLWH. And critically, efforts must be made to ensure that PLWH experiencing Long COVID are not systematically excluded from the nascent clinical trials that will soon be implemented to determine how best to manage Long COVID symptoms. Ultimately, regardless of whether it is more common, presents differently, or has different mechanisms, Long COVID is likely to remain an important new comorbid condition among a subset of PLWH recovering from SARS-CoV-2 infection for the foreseeable future.

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