

A Case of HIV and SARS-CoV-2 Co-infection in Singapore

To the Editors:

As of April 10, 2020, there are close to 1.5 million cases of COVID-19 globally¹ and 37.9 million people living with HIV (PLHIV).² Most deaths in patients with COVID-19 disease have been in immunocompromised or elderly patients with little information on PLHIV. Concern arises from recent studies suggesting that the immune system function in HIV patients is not fully restored even after long-term chronic virologic suppression.^{3,4} So far, there is only 1 report on a patient from Wuhan who was newly diagnosed with HIV on screening before starting lopinavir/ritonavir for COVID-19 treatment.⁵ We report here a case of HIV and SARS-CoV-2 coinfection in a PLHIV on long-term antiretroviral therapy in Singapore.

A 37-year-old man presented to the emergency department of our public health institution with fever (38.6°C at maximum), sore throat, dry cough, and headache for the duration of 6 days. He returned from a 16-day trip to Paris and London 1 day before his symptom onset. In view of his travel history and presenting complaints of upper respiratory tract infection symptoms, he was immediately admitted to an isolation room.

His background medical history was significant for chronic HIV, diagnosed in late 2010. The CD4⁺ T-cell count was 201 cells/ μ L (12%) on diagnosis. He was initiated on tenofovir, lamivudine, and efavirenz and has been fully adherent to medications. His viral load has been undetectable since February 2011, and the CD4⁺ T-cell count increased to 900 cells/ μ L (36%) by 2015 (after which there were no further checks in view of the high-normal count). Efavirenz was switched to rilpivirine in September 2017 for financial considerations, but the patient has other-

wise never been on protease inhibitors in the course of his HIV treatment.

On presentation, the patient looked clinically well and was afebrile (37.2°C) with normal blood pressure and heart rate. His oxygen saturation was 100% on room air, and his respiratory rate after admission was 20 breaths per min. Lungs were clear on auscultation, and physical examination was otherwise normal. He had a normal complete blood count with no cytopenias, as well as normal renal and liver function tests on admission. Inflammatory markers were not raised: CRP < 5 mg/L [reference range 0–10 mg/L], LDH 404 U/L [reference range 250–580 U/L], procalcitonin < 0.06 ug/L [reference range <0.50 ug/L], and ferritin 77 ug/L [reference range 20–300 ug/L]. His chest radiograph was clear with no infiltrates or consolidation. Real-time reverse-transcriptase polymerase chain reaction assay for the detection of SARS-CoV-2⁶ was performed on a nasopharyngeal swab and returned positive the next day. The HIV viral load checked on admission remained undetectable, and the CD4⁺ count was 680 cells/ μ L (25%).

After admission, the patient remained clinically well with no further fevers or desaturation. His cough was severe but dry with minimal production of yellow sputum and reduced in intensity after day 11 of the onset of symptoms. No other symptoms developed. On day 9, day 11, and day 15 of symptom onset, repeat complete blood count, CRP, LDH, and ferritin remained normal. Serial chest radiographs also did not show any developing changes of pneumonia. The CD4⁺ count repeated on day 11 of symptom onset was 650 cells/ μ L (22%). He was not initiated on any off-label treatments specific for COVID-19 because his illness was mild. His routine antiretroviral therapy was continued. The patient remained well throughout the course of his admission and was transferred to a community isolation facility on day 22 of his illness. He was discharged home after another 14-day stay at the isolation facility, on having 2 negative SARS-CoV-2 polymerase chain reaction tests (performed on nasopharyngeal swabs) over 2 consecutive days.

Although our patient represents only 1 case, it is encouraging to report that he

recovered from a mild and uncomplicated clinical course of COVID-19 without treatment. He was not on any protease inhibitors in the course of his HIV treatment, which are reported to have activity against SARS-CoV-2. Although he has been virologically suppressed with a high-normal CD4⁺ T-cell count, it is not known whether a dampened immune response from having chronic HIV for more than 10 years may have contributed to the mild course of his illness. Larger case series analyses with a range of patients will certainly be needed before any firm conclusions can be drawn, but it can be seen from this case that not all PLHIV are at risk of severe COVID-19 disease.

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