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*Case Report*

# SARS-CoV-2 reinfection: Two cases from Ethiopia

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## ABSTRACT

Reinfection with SARS-CoV-2 has infrequently been reported in the literature and never from Ethiopia or Africa. We describe two individuals with documented recurrent COVID-19 disease admitted to Eka Kotebe Hospital, Addis Ababa, Ethiopia.

**Keywords:** SARS-CoV-2, Reinfection, COVID-19

## INTRODUCTION

Reinfection with severe acute respiratory syndrome virus 2 (SARS-CoV-2) is a rare phenomenon. The efficiency and duration of the immune responses in protecting the host from reinfection remain to be elucidated.[1,2] Mild to serious disease following reinfection has been reported from several countries.[3-6] To the best of our knowledge, however, there has been no report of reinfection from Africa in general and from Ethiopia in particular. Here, we report two individuals with SARS-CoV-2 reinfection from Addis Ababa, Ethiopia, the first cases in the nation and in the continent.

## CASE REPORT

### Case 1

As part of an orphanage workplace required screening, a 42-year-old previously healthy man from Addis Ababa tested positive for SARS-CoV-2 by nasal swab RT-PCR in late July 2020. He was asymptomatic at that time and isolated at home for 2 weeks. His repeat nasal swab RT-PCR test was negative 16 days after the initial test. Seven months later, the patient developed fever, myalgia, arthralgia, headache, and dry cough followed by shortness of breath. He tested positive for SARS-CoV-2 by nasal swab RT-PCR 5 days after symptom onset. He was admitted to Eka Kotebe Hospital, the first COVID-19 treatment center in Addis Ababa, Ethiopia.

Physical examination revealed an acutely ill, obese (BMI = 33.8 kg/m$^2$) man, in respiratory distress. His vital signs were remarkable for a respiratory rate 28 breaths/min and $SpO_2$ 81% on room air. He had diffused coarse crackles bilaterally on the lower 2/3$^{rd}$ lung fields. His hemoglobin (Hb) A1C was 8.7%. His other laboratory tests included the following: White blood cell (WBC) 7700/mm$^3$ (reference range 4400–10,000/m$^3$), Hb 15.3 g/dl (13.5–17.5 g/dl), platelet count 292,000/mm$^3$ (152,000–450,000/mm$^3$), creatinine (Cr) 0.7 mg/dl (0.5–1.3 mg/dl), blood

urea nitrogen (BUN) 13 mg/dl (12–24 mg/dl), aspartate transaminase (AST) 12 u/l (0–50.0 u/l), alanine transaminase (ALT) 13 u/l (0–50.0), alkaline phosphatase (ALP) 109 u/l (30–120 u/l), sodium (Na) 139 mmol/l (135–145 mmol/l), potassium (K) 3.9 mmol/l (3.5–5.1 mmol/l), and chloride (Cl) 101 mmol/l (98–111 mmol/l). Chest X-ray showed bilateral lower zone and peripheral opacities.

He was treated with supplemental oxygen (10 L/min), awake prone ventilation, antibiotics (ceftriaxone 1 gram [g] intravenous [IV] twice daily [bid] and then cefepime 1 g IV bid in addition to vancomycin 1 g IV bid), dexamethasone (6 mg IV bid), therapeutic anticoagulation (unfractionated heparin 17,500 international units subcutaneous [subq bid]), and adjusted regular insulin. He clinically improved and was discharged home, off supplemental oxygen, 3 weeks after admission. At discharge, he had a negative nasopharyngeal RT PCR for SARS-CoV-2.

### Case 2

On monthly COVID-19 screening as per protocol of his workplace, a 54-year-old previously healthy male nurse tested positive for SARS-CoV-2 by nasopharyngeal RT-PCR in late October 2020. He had only mild low back pain. He was admitted to an isolation center and discharged, symptom free, 12 days later with a negative nasal swab RT-PCR. In late January 2021, he developed watery diarrhea, profound fatigue, arthralgia, myalgia, and a high-grade fever. Over the next 24 h, his condition worsened with sore throat, dry cough, and shortness of breath. He tested positive for SARS-CoV-2 by nasopharyngeal RT-PCR and was admitted to Eka Kotebe Hospital.

On admission, his blood pressure was 120/80 mmHg, respiratory rate 22 breaths/min, temperature 36.5°C, and $SpO_2$ 83% on room air. He had bilateral fine crackles on chest examination. His laboratory tests included the following: WBC 8170/$mm^3$ (4000–10,000/$mm^3$), Hb 15.6 g/dl (13.5–16.5 g/dl), platelet count 115,000/$mm^3$ (150,000–450,000/$mm^3$), Cr 1.07 mg/dl (0.81–1.44 mg/dl), BUN 20.4 mg/dl (17–43 mg/dl), ALT 36 u/L (0–50 u/l), AST 32 u/l (0–50 u/l), ALP 73 u/l (30–120 u/l), Na 138 mmol/l (136–145 mmol/l), K 3.8 mmol/l (3.5–5.1 mmol/l), Cl 103 mmol/l (98–107 mmol/l), and Hb A1C 5.7%. Chest X-ray showed bilateral lower lung zone haziness consistent with pneumonia.

He was treated with supplemental oxygen (up to 5 L/min), awake prone ventilation, antibiotics (cefepime 1 g IV 3 times daily and vancomycin 1 g IV bid), dexamethasone (6 mg IV once daily), and enoxaparin (40 mg subq daily). He was discharged 2 weeks later, clinically improved, with a negative nasopharyngeal RT-PCR.

## DISCUSSION

We report two previously healthy patients with documented reinfection with SARS-CoV-2.

Most SARS-CoV-2 infections result in detectable levels of antibodies 10–14 days after symptom onset. The extent and duration of immune protection remain largely unknown. Those with mild presentations may have less immune response, which may explain reinfection in our patients. Further, Zhao *et al*. from China reported that 40% of asymptomatic individuals and 12.9% of symptomatic ones were seronegative 8 weeks after infection.[2] Serologic testing for post-infection antibody response was not performed in our patients.

Our patients did not have any immunologic disorder nor were they on any immunosuppressive treatment that could put them at risk for reinfection. Whole-genome sequencing was also not done to assess if they were infected with phylogenetically different strains. Nonetheless, given that the time span between the first and second episodes of SARS-CoV-2 infection in our cases was several months (>3 and >6 months), we think that the cases are indeed true reinfection cases. This notion is supported by a recent review indicating that reinfection cases with persistent SARS-CoV-2 shedding in a significant proportion of patients occurs up until 38 days.[7]

Even though reinfection cases with persistent SARS-CoV-2 shedding occur in a significant proportion of patients up until 38 days[7] and a meta-analysis by Arafkas *et al*. reported no clinical reinfection after a 70-day period following first infection,[8] our cases and other reports have clearly shown that reinfection is a possibility after 3 months.[4,9-11]

Some patients with reinfection were reported to have severe disease,[3-6] and this was also the case in our patients. Those with mild or asymptomatic disease are likely to get reinfected possibly due to a higher dose of the virus, greater virulence, or antibody-dependent enhancement.[6,12] The reason for the occurrence of severe disease after initial asymptomatic or mild infection is not well known but it could be from a genetically mutated variant with increased tendency to cause severe infection. Reinfection severe disease could also be due to the presence of a high genetic burden as reported by Horowitz *et al*.[13] However, thus far, there is no evidence of this finding in Ethiopia.

## CONCLUSION

Our cases and prior reports indicate that infection may not confer long lasting immunity. This also leaves unanswered the question of how long vaccine induced antibodies will protect against subsequent infection. For now, known personal and public health measures should remain primary prevention strategies.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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**Conflicts of interest**

There are no conflicts of interest.

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