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A case of SARS-CoV-2 reinfection in Ecuador

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See Online for appendix

Cases of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) reinfection have been reported in Hong Kong, Belgium, the Netherlands, and the USA.¹⁻⁴ Here we report the first confirmed case of SARS-CoV-2 reinfection in Ecuador and South America. For full methodological details, see the appendix (pp 1, 2).

On May 12, 2020, a 46-year-old man presented in Quito, Ecuador, with 3 days of intense headache and drowsiness. On May 16, he took a rapid antibody test that was positive for IgM and negative for IgG. On May 20, the patient tested positive on RT-PCR (*ORF3a* gene, Cq=36.85). Subsequently, the patient's symptoms improved, and he tested negative on RT-PCR on June 3 (appendix p 4).

On July 20, the patient presented once again with symptoms suggestive of COVID-19. This time the symptoms were more severe and included odynophagia, nasal congestion, fever of 38.5°C, back pain, productive cough, and dyspnoea. He received an RT-PCR test on July 22, with a positive result (*N* gene, Cq=30.82). Despite having moderate symptoms and dyspnoea, the patient did not require hospitalisation, and his clinical status improved after several days. A fourth RT-PCR test was done on Aug 4, which was negative (appendix p 4). Finally, an ELISA quantitative antibody test was done on Aug 18, showing anti-SARS-CoV-2 IgG (34.1 NovaTec Units [NTU]) and IgM (54.2 NTU; both interpreted as positive).

SARS-CoV-2 genome sequencing was done using Oxford Nanopore

Technologies' MinION, following the ARTIC Network protocols.⁵ Phylogenetic analysis revealed that the first infection variant belonged to clade 20A and lineage B1.p9, whereas the second infection variant belonged to clade 19B and lineage A.1.1 (appendix p 4). The mutation loci and amino acid changes are described in the appendix (p 5). No shared mutations were observed between the two sequences, further suggesting that both variants resulted from distinct evolutionary trajectories.

Although reinfections with coronaviruses that cause the common cold have been reported previously,⁶ the symptoms of reinfections are usually milder than those of the primary infections. It was therefore surprising that our patient showed more severe disease with the second infection than with the first, especially because the patient did not have any additional clinical conditions that could explain it. Increased severity of second SARS-CoV-2 infections has been reported previously: in a healthy man aged 25 years in Nevada, USA,² and in an immunocompromised woman aged 89 years in the Netherlands, which resulted in her death.⁴

The protective immune response to SARS-CoV-2 infection is not fully understood; however, protection against severe disease has been shown in animal models and inferred in humans infected with the virus.⁷ Interestingly, the antibody test performed during the first infection event showed the presence of specific anti-SARS-CoV-2 IgM and no IgG. However, it is not possible using conventional antibody tests

to determine whether a protective immune response developed.

BP-V and MB-W contributed equally. We declare no competing interests.

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