

SARS-COV-2 re-infection in a young male. Are we immune after an initial encounter?

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ABSTRACT

JMS: 2020; 23(4):38-40 DOI: <https://doi.org/10.33883/jms.v23i4.803>

We herewith report a case of severe SARS-COV-2 re-infection in an adult approximately thirteen weeks after he retested negative for initial infection. This case highlights re-infection with SARS-COV-2 is possible in an individual only a few weeks after recovery from the initial infection.

Patients who recover from COVID-19 are immune to re-infection with SARS-COV-2 and develop both cell and antibody-mediated immune response which depends upon the severity of the infection. The mild or asymptomatic disease leads to a weaker immune response that wanes off rapidly within few months compared to severe disease¹. However, uncertainty remains whether the immune response protects against subsequent re-infection and what level of neutralizing antibody is protective. We herewith describe a case of severe SARS-COV-2 re-infection in an

adult approximately 11 weeks after he tested negative following an asymptomatic detection of COVID 19 positivity upon contact tracing of COVID-19 cases.

Case

On August 22nd, 2020, a 28-years dental surgeon with a history of mild intermittent bronchial asthma presented with a 4-day history of high-grade fever, dry cough, and headache. Physical examination revealed a pulse of 75 beats/min, blood pressure of 120/80 mmHg, respiratory rate of 20 breaths/min, and bilateral crepitations upon chest

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Access this article online



Website:
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DOI:
<https://doi.org/10.33883/jms.v23i4.803>

How to cite this article: Khan U, Shah TH, Mantoo S, Mehfooz N, Dar AK, Shabir A, Siraj F, Bindroo M, Bhat F, Qadri S, Dangroo M, Koul A, Fomda B, Sofi F, Shah S, Jan R. SARS-COV-2 re-infection in a young male. Are we immune after an initial encounter?. JMS 2020;23(4):38-40

Received: 2020-12-03 Accepted: 2021-01-13



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auscultation. The rest of the examination was unremarkable. He was hypoxemic (PaO₂ 50mmHg) requiring supplemental oxygen @ 6 L/min to maintain his oxygen saturation.

Thirteen weeks ago, on 16th May 2020, the patient presented with a history of contact to a confirmed case of COVID-19. Real-time reverse transcription PCR on nasopharyngeal swab confirmed SARS-CoV-2 infection. He was managed as asymptomatic COVID19 (with isolation and monitoring). Repeat RT-PCR on day-12th of illness was again positive followed by a negative RT-PCR on day14th of illness. Six weeks following the detection of the infection, his serum tested negative for SARS-CoV-2 specific IgG antibodies.

The CBC was normal and serum ALT was high (218 IU/L, normal <40). D-dimer levels were 1250 mg/dL, IL-6 of 39.33 pg/mL (normal <7), ferritin of 327 ng/mL (Normal, 10-291) and a CRP level of 23 mg/L. Rests of the laboratory parameters were normal. High-resolution CT-chest revealed evidence of ground glassing in the posterior segment of the right upper lobe, basal segments of the right lower lobe, and left upper lobe (CORADS-6). Nasopharyngeal swab for RT-PCR was positive for SARS-COV-2.

The patient was managed as severe COVID-19 with oxygen, remdesivir, LMW-heparin, dexamethasone, antibiotics, and supportive care. The patient improved gradually and eventually was discharged after 10 days when RT-PCR for SARS CoV 2 was negative whereas Anti-SARS CoV 2 IgG antibodies were 4.15 (normal 0-1.4).

Discussion

Our case is the first documented case of reinfection in a patient from India. The first case of reinfection with SARS-COV-2 recently reported in Hong Kong 2 jolted the concept of a so-called "immunity passport" in the context of SARS-CoV-2 reinfection. However, the variability of the antibody response among the infected individuals argues against the potential protection of the recovered individuals for re-infection. Fan Wu et al reported that up to 30 % of recovered individuals had very low titers of neutralizing antibodies and a smaller proportion had undetectable levels.³ Undetectable IgG antibodies in our patient before the re-infection would place him in an 'unprotected' category.

The suboptimal antibody response in our patient may fortify

the concept that asymptomatic COVID-19 patients do not mount a robust antibody response.⁴ Patients with mild or asymptomatic COVID-19 have been reported to mount a weaker antibody response compared to those with severe disease.³ It is also possible that innate immune and T-cell response against the virus as happens with MERS-coronavirus may play a role in mild or asymptomatic disease.⁵ Recently ICU-hospitalized patients with COVID-19 have been found to generate a virus-specific reactive T-cell response, including among a subset of healthy, unexposed people, perhaps due to previous exposures to others.⁶ Another possibility for re-infection could be variation in the spike protein of the virus which induces neutralizing antibodies. This is because of differences in the spike protein of the virus as several mutations in the spike protein receptor-binding domain and N-terminal domain have been reported.⁷ This remains unexplored in our patient as the genomic analysis was not possible.

Important lacunae in our understanding of the dynamics of humoral immunity to SARS-CoV-2 is the longevity of the antibody response following the infection, as also the titers of the antibody response that could be potentially protective. The answers to these questions would also be important for the efficacy of the COVID vaccines in addition to the possible requirement of a revaccination/booster. The decay of antibody levels in other human coronavirus infections and the reports of subsequent reinfection in them also add to the possibility of a post-pandemic wave of COVID19.⁸ An interval of 98 days between two infections in our case suggests reinfection because viral RNA is undetectable one month after infection.¹ A severe infection following previous asymptomatic infection contests the previous claims of a milder disease in the event of a re-infection by the virus.⁸ A suboptimal antibody response following a confirmed molecular diagnosis emphasizes the adoption of continued infection control measures even when a person recovers from the asymptomatic infection.

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