Bendamustine/rituximab

Prolonged SARS-CoV-2 viral shedding : case report

A 71-year-old man developed prolonged SARS-COV-2 viral shedding following treatment with bendamustine and rituximab for grade 3A, stage IIIB follicular lymphoma [route and dosage not stated]

The man who, had dyspnea was admitted to the hospital. Of note, he had a history of grade 3A, stage IIIB follicular lymphoma which was in complete remission. Nine months prior to the current admission, he had received six cycles of bendamustine and rituximab therapy. Notably, at that time, he was not vaccinated for SARSCoV-2. Five weeks prior to the current admission, he experienced fatigue and subsequently, he was found to be positive for SARS-COV-2, N501Y variant, with a cycle threshold (Ct) value of 29.3. Hence, he had COVID-19 infection. At the time of diagnosis, his COVID-19 symptoms were not severe; therefore, he was home isolated and received unspecified symptomatic treatment. However, after, two weeks from the diagnosis, the PCR testing of his nasopharyngeal specimen (NPS) revealed persistently positive results for the N501Y variant; the Ct value was 32.6. Subsequently, he was discharged. At the time of discharged, he still had a few COVID-19 symptoms, including lethargy and dizziness. Therefore, he continued his self-isolation. It was reported that, on the morning of current admission, he had mild confusion and difficulty in breathing; therefore, he was referred to the emergency department of the hospital. On admission, he reported a temperature of 36.4°C, pulse rate of 77 beats/minute, blood pressure of 110/62mm Hg, oxygen saturation of 87% on room air and respiratory rate of 22 breaths/minute. Subsequent physical examination showed decreased breath sounds at the lung base. Blood examination was indicative of lymphopenia and elevated C-reactive protein levels. Moreover, his CD4+ T lymphocyte count was severely low. Subsequent chest radiography showed bilateral diffuse infiltrates and chest computed tomography revealed diffuse ground-glass opacities in the peripheral and middle lobes with consolidation in the lower lobes. Thereafter, PCR results of NPS revealed positive results for SARSCoV-2, N501Y variant with a Ct value of 21.3. Electrochemiluminescent immunoassay test revealed negative results for for both SARS-CoV-2 total antibody and SARS-CoV-2 spike protein [duration of treatment to reaction onset not stated].

Thereafter, the man received off label dexamethasone and remdesivir and oxygen therapy. On day 5, his oxygen therapy was discontinued. Subsequently, his general condition improved. On day 5, the CRP level decreased, however, on day 15, it was again increased. It was reported that, he had mild fever but no respiratory symptoms. However, as compared to his chest radiograph on admission, his current chest radiograph did not show any clear improvement compared to that obtained on admission. Subsequently, his treatment with dexamethasone was stopped. It was speculated that the mild fever and persistent CRP positivity were due to prolonged and relapsed COVID-19 infection due to cessation of off label dexamethasone. However, a gradual improvement in CRP levels and high body temperature were noted without any medication. On day 28, he was afebrile and was not in cardiorespiratory distress. Subsequently, he was discharged and based on a report of persistent viral shedding, for following month, he was instructed to observe home isolation, it was reported that, his viral shedding had lasted for approximately two months after bendamustine and rituximab therapy. After 10 days from discharge, the PCR test was reported as negative, his CRP level declined to the normal range. Moreover, his chest radiography did not reveal any abnormal opacity. After 2 months, from discharge, his SARS-COV-2 antibody levels results remained negative. Eventually, the development of prolonged SARS-COV-2 viral shedding was attributed to bendamustine and rituximab.

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