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Prednisolone/rituximab S

COVID-19 and SARS-CoV-2 pneumonia: case report

A 43-year-old woman developed COVID-19 and SARS-CoV-2 pneumonia during treatment with rituximab and prednisolone for rheumatoid arthritis (RA) [not all duration of treatment to reaction onset and routes stated]

The woman presented to the emergency department with a 24h history of fever, headache, myalgia, nonproductive cough and chest pain. She was diagnosed with COVID-19 infection on 29 November 2020. Her clinical examinations were normal. Hence, she was discharged. She had a medical history of seropositive rheumatoid factor and anticyclic citrullinated peptide RA for which she had been receiving rituximab for the last 6 years along with prednisolone 5–10 mg/day. She had completed 12 courses of rituximab, two 1g IV infusions 2 weeks apart. Her last dose was administered 4 months ago in August 2020. She had taken methotrexate, sulfasalazine and hydroxychloroquine in past, which she did not tolerate. Along with RA, she also had postsurgical hypothyroidism (thyroid neoplasm), dyslipidaemia and depressive syndrome and she had been receiving pantoprazole, levothyroxine sodium [levothyroxine], fluoxetine and fenofibrate, concomitantly. Fifteen days after the diagnosis of COVID-19 (14 December 2020), she again presented to the emergency department with persistent fever, headache, myalgia, cough, pleuritic chest pain, asthenia and nausea. On 15 December 2020, she was admitted to the COVID-19 ward. On admission, her laboratory findings were as follows: BP 125/80mm Hg, pulse 90 beats/min and oxygen saturation 96%, She had interstitial and subpleural infiltrates with ground-glass opacities with moderate involvement on both chest radiograph and CT. Arterial blood gas test revealed respiratory alkalemia with subtle hypoxemia and her analyses observed haemoglobin, 137 g/L, CRP 3.08 mg/dL, leucocytes 7400 × 109/L, lymphocytes 1060, platelets 384000 × 109/L, creatinine 0.72 mg/dL and ferritin 900 ng/mL. She was suspected to have SARS-CoV-2 pneumonia.

Thereafter, the woman was treated with oxygen therapy and her prednisolone dose was increased to 40 mg/day. Subsequently, flow cytometry revealed 1830 lymphocytes /µL with CD4 count 804 and no efficient B cells; quantitative serum immunoglobulin test showed low levels of IgG and IgM and COVID-19 serology test was negative. Despite her treatment, she remained highly febrile, inflammatory markers were elevated and radiological progression noted. While she was persistently symptomatic, no obvious clinical aggravation was observed and no additional oxygen therapy was required. On day 20 of her symptoms, she received piperacillin/tazobactam for SARS-CoV-2 pneumonia. However, no clinical, analytical or radiological responses were noted. Subsequently, she was scheduled to receive 5 day course of IV immune-globulin [human immunoglobulin] 0.4 mg/kg/day (total dose of 2 g/kg) and adjacent her dexamethasone dose to 9mg id. After 1 day of immune-globulin treatment, her fever subsided. On the next days, her clinical, analytical and radiographic findings also recovered and hence, oxygen supplementation was discontinued. She remained afebrile and asymptomatic and was discharged on Day 32, 48h after finishing the 5 days course of immune-globulin. After 2 and 6 weeks of discharge, she was evaluated in an outpatient clinic. She remained afebrile and asymptomatic and she began her normal activities. Laboratory results showed no abnormal findings and a significant radiologic improvement. She tolerated the tapering of prednisolone. No SARS-CoV-2 antibodies were detected 6 weeks following the discharge.

Vasconcelos J, et al. Intravenous immunoglobulin as a therapeutic option for patients with worsening COVID-19 under rituximab. BMJ Case Reports 14: No. 6, 28 Jun 2021.

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