Mild COVID-19 in ANCA-associated vasculitis treated with rituximab

Treatment for antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis usually raises questions about the risk of infections. Particular attention has been given to the impact of drugs such as cyclophosphamide and B-cell depleter therapies on the severity of COVID-19. Monti et al suggest that receiving biological disease-modifying antirheumatic drugs may not increase risk of COVID-19. Furthermore, Guiplain et al reported a woman treated with rituximab and low-dose prednisone due to granulomatosis with polyangiitis proteinase 3-antineutrophil cytoplasmic antibody (PR3-ANCA) vasculitis, who developed pneumonia associated with COVID-19 with a milder evolution than expected in other series.

Here, we present a 64-year-old woman diagnosed of myeloperoxidase-ANCA microscopic polyangiitis in 2014, with secondary hypertrophic pachymeningitis, sinusiitis and constitutional syndrome. Her main comorbidities were hypercholesterolaemia and areata alopecia. On 25 November 2019, vasculitis relapsed and was treated with two infusions of 1000 mg rituximab given 2 weeks apart, in addition to three bolus of 125 mg methilprednisolone. The patient improved and received maintenance treatment with 7.5 mg/day prednisone and a double-strength tablet of trimeprprop-sulfamethoxazole 3 days a week.

While on holidays at her daughter’s house in The Netherlands, on 7 March 2020 her husband developed fever >38°C, odynophagia, discomort and dyspnnea. He was finally admitted to a hospital and diagnosed with COVID-19 infection by reverse transcription PCR (RT-PCR) on a nasopharyngeal swab. At the same time, she presented feverish, asthenia, myalgia, lack of appetite, headache, anosmia and dysgeusa, and was treated with paracetamol at home, improving after 2 weeks. One month later, after returning home in Spain, she came to our clinic for evaluation due to her exposure to COVID-19.

Blood test results showed positive immunochromatographic-specific IgG antibodies assay for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). IgM antibodies and nasopharyngeal swab (RT-PCR) were both negative. Cytometric studies revealed 10 B lineage cells. Serum immunoglobulin sequences. The impact of rituximab treatment on SARS-CoV-2 infection remains to be clarified. As hyperinflammation underlies the mechanism of severe COVID-19, the immunocompromised situation may prevent them from virus-induced cytokine storm syndrome. Transcriptome data including RNA-seq and GeneChip human genome arrays show that glucocorticoids and some disease modifying anti-rheumatic drugs (DMARDs) (foliculzumab, methotrextate, hydroxylchloroquine, tofacitnihm, azathiprine and so on) could suppress the cytokine profile represented in severe COVID-19, but there are scarce data about rituximab. Rituximab largely depletes peripheral B cells, including memory B cells, from blood. It also modulates the immune system through different mechanisms including antibody-dependent cellular cytotoxicity, complement-dependent cytotoxicity and apoptosis induction. In addition, early stem cells and B-precursors do not express CD20 on their surface, which gives them resistance to the effect of rituximab. However, studies have shown that patients treated with anti-CD20 agents had a severely impaired immunogenicity to vaccines and therefore, against emerging infections such as COVID-19.

Our patient’s presentation of COVID-19 with mild symptoms did not fit the expected clinical course in an immunosuppressed patient. This suggests that B-cell depleter therapy may not be a risk factor for severe forms of COVID-19 but favour a milder course of the disease and the generation of specific antibodies, whose future protective role remains unclear.


1Department of Internal Medicine, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain
2Systemic Autoimmune Disease Unit, Department of Internal Medicine, Instituto de Investigación Sanitaria del Principado de Asturias (ISPA), Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain
3Autoimmunity Section, Immunology Department, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain
Correspondence to Silvia Suárez-Díaz, Department of Internal Medicine, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain; silvia_porto@hotmail.es

Contributors SS-D: writing the paper, CM-C: writing the paper, RC-H: clinical support; CS-C: language supervision; LM-A: Immunological diagnosis; LC-M: patient’s primary physician and head of the autoimmune diseases unit.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; internally peer reviewed.

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Received 11 June 2020
Accepted 12 June 2020
Published Online First 7 August 2020

ORCID iD Silvia Suárez-Díaz http://orcid.org/0000-0002-8558-5452

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