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LETTER

The course of COVID-19 infection and prolonged sneezing and nasal congestion in a patient using ixekizumab

Dear Editor,

With the onset of the Coronavirus disease 2019 (COVID-19) pandemic, we had concerns about managing the treatments of the patients who received biological therapy. To share our experience, we described the clinical course of a patient with COVID-19 who used ixekizumab.

A 46-year-old female patient with hypertension, bronchiectasis, allergic rhinitis, house dust allergy, chronic plaque-type psoriasis, and psoriatic arthritis with a positive QuantiFERON blood test used ixekizumab 160 mg, perindopril/indapamide combination, and received isoniazid for tuberculosis prophylaxis with a dose of 300 mg/day. She continued ixekizumab treatment for 6 months. After the last dose of the ixekizumab, the patient had complaints of mild fever (37.5°C) and coughing for 3 days. She contacted a person who had a positive polymerase chain reaction (PCR) 6 days ago for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The patient's nasopharyngeal swab PCR test was found to be positive for SARS-CoV-2. At admission, the patient's oxygen saturation was 98%, and her lung tomography had a bilateral ground-glass appearance. She was hospitalized in the infectious diseases service, and she began to receive favipiravir (1600 mg orally twice a day on the first day, and followed by 600 mg orally twice a day), enoxaparin sodium solution s.c. 60 mg/0.6 mL/day, and paracetamol 500 mg/day p.o. On the third day of the treatment, her fever decreased, and she had a complaint of mild shortness of breath. Favipiravir treatment was completed and stopped on the fifth day. At the end of the first week, the dyspnea was completely recovered, and the fever did not occur after discharge. After a week, symptoms of sneezing and nasal congestion started. Two SARS-CoV-2 nasopharyngeal swab samples repeated 30 days after the initial diagnosis yielded negative results. One ixekizumab dose was skipped, and the treatment continued 1 month later. The patient had not new psoriatic lesions, and joint complaints during and after the COVID 19 infection. The psoriasis area severity index (PASI) value of the patient, which was 11.8 when she started the ixekizumab treatment, was measured as 1.2 in the sixth week of the treatment. The PASI value did not change during and after the COVID-19 infection.

Interleukin-17 (IL-17) was considered to promote viral persistence by inhibiting the apoptosis of virus-infected cells and to cause Th17-related cytokine storm syndrome.[1] In a recent study of the Italian psoriasis-biologics-COVID working group, the researchers thought that biological therapies could reduce the cytokine storm which may lead to multiorgan failure, ARDS, and death.[2] Therefore, inhibition of IL-17 may have contributed to the mild course of the disease in the present case.

Although the assumption that IL-17 causes asthma pathology is not clear, it was revealed that IL-17A contributed to airway hyperresponsiveness in mouse models, and increased levels of airway IL-17A were observed in humans with severe asthma.[3] The patient developed sneezing and nasal congestion symptoms in the second week after the last administration of ixekizumab. This may be due to the re-increase of IL17A levels in the respiratory tract mucosa which causes airway sensitivity.

It was thought that COVID-19 infection was less severe in patients with airway allergy.[4] The patient also had a house-dust allergy. This may have a role in the mild course of the disease in the patient.

With regard to the tuberculosis co-infection, Th1 immunity developed in association with exposure to tuberculosis antigen might be responsible for the low mortality rate of COVID-19.[5] Quantiferon, an indicator of having previously encountered tuberculosis bacilli, was positive in our patient. This may be one of the reasons for the mild course of the disease.

Cohort studies and case reports report that biological treatments in patients with psoriasis do not increase the risk of COVID-19 and do not worsen the course of COVID-19.[1,2] Based on these finding, we continued the treatment of the patient.

In conclusion, IL-17 inhibition did not worsen the course of the COVID-19 disease in our patient. The patient had significant risk factors for the severe course of COVID-19 infection such as hypertension and obesity. However, several other factors such as exposure to tuberculosis antigen, accompanying history of respiratory allergy, and using IL-17A inhibitor may have contributed to the good prognosis of the COVID-19 disease in the patient.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

AUTHORS CONTRIBUTION

Semih Güder and Hüsna Güder: Conceptualization; Semih Güder: Data collection; Semih Güder and Hüsna Güder: Writing of the manuscript; Semih Güder and Hüsna Güder: Manuscript editing and revision.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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