SARS-CoV-2 infection in a patient with psoriasis treated with risankizumab

Christina Huang MD, Tiffany Chen MD FRCPC DABD

Affiliations: Division of Dermatology, University of Toronto, Toronto, Ontario, Canada

Corresponding Author: Tiffany Chen, Women's College Hospital, 76 Grenville Street, Toronto, ON, M5S 1B2, Tel: 416-323-6400, Email: tiffany.chen@medportal.ca

Keywords: biologics, IL23, psoriasis, SARS-CoV-2, severe acute respiratory syndrome coronavirus 2, risankizumab

To the Editor:
Since the emergence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in December 2019, the novel virus has spread worldwide, resulting in a global pandemic. The contagious nature and severe consequences of the infection raise concern for immunosuppressed patients, including those treated with biologic therapy.

We report the case of a 40-year-old man suffering from plaque psoriasis of the face, scalp, and body treated with risankizumab, an IL23 p19 inhibitor. The patient had failed both psoralen plus ultraviolet light A (PUVA) and topical therapy. He was otherwise healthy.

The patient was started on risankizumab on August 12, 2020 owing to worsening psoriasis. He received his second injection on September 9, 2020. Ten days later (September 19, 2020), he developed a fever of 101.8 degrees Fahrenheit. On September 20, 2020, he tested positive for SARS-CoV-2. Aside from intermittent fatigue, there were no additional symptoms. His fever resolved on September 20, 2020 and he reported feeling "really well" on September 21, 2020. The patient had no known contact with individuals infected by SARS-CoV-2. Subsequently the patient went on to receive his next injection in December as per the dosing regimen.

Although the mechanisms underlying SARS-CoV-2 infection are unclear, the cytokines associated with severe cases requiring intensive care unit admissions include IL1, IL2, IL7, IL10, granulocyte colony-stimulating factor, inducible protein 10, monocyte chemoattractant protein 1, macrophage inflammatory protein 1a, interferon-γ, and TNF [1]. Prior reports have linked Th17 to severe immune injury in SARS-CoV-2 infection [2,3]. This data has led some authors to propose that inhibition of IL23, which is essential to Th17 phenotype, may play a protective role in the setting of SARS-CoV-2 infection by attenuating key cytokines [4-6].

Our patient contracted SARS-CoV-2 after the initiation of risankizumab. His symptoms were mild and resolved within three days. This is a shortened disease course—symptoms and fever persist an average of 8 and 6.5 days, respectively [7]. Other studies have reported SARS-CoV-2 infection in patients treated with risankizumab [5] and guselkumab [8]. Like our patient, these patients developed minor symptoms and did not experience any life-threatening manifestations of SARS-CoV-2. In one study, symptoms improved drastically one day after guselkumab injection, despite failing to respond to paracetamol [8].

These findings raise questions about the potential role of IL23 inhibitors in counteracting the "cytokine storm" seen in severe cases of SARS-CoV-2 infection. This exaggerated immune response is both ineffective towards the virus and deleterious to the body, leading in some cases, to acute respiratory failure, end organ damage, and death. In our case, the effect of risankizumab on the patient's disease course is unknown since patients with SARS-CoV-2 infection experience variable disease manifestations and may be asymptomatic. However, despite contracting SARS-CoV-2 infection, our patient treated with risankizumab achieved a full recovery.

Real world cases such as ours are needed to understand the implications of SARS-CoV-2 infection in patients receiving biologic therapies for psoriasis. Future reports may help to determine if there is a potential role for biologic therapies in the development and outcomes of SARS-CoV-2 infection.

Potential conflicts of interest

Dr. Chen has served as Consultant, speaker, or advisory boards for Amgen, Aralez, AbbVie, Bausch Health, Eli Lilly, Galderma, Janssen, LEO Pharma, Pfizer, Sanofi Genzyme, and Sun Pharma. Dr. Huang has no interests to declare.

References

1. Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet*. 2020;395:497-506. [PMID: 31986264].
2. Wu D, Yang XO. TH17 responses in cytokine storm of COVID-19: An emerging target of JAK2 inhibitor Fedratinib. *J Microbiol Immunol*. 2020;53:368-70 [PMID: 32205092].
3. Xu Z, Shi L, Wang Y, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. *Lancet Repir Med*. 2020;8:420-2. [PMID: 32085846].
4. Kiss N, Lőrincz K, Medvecz M, et al. Coronavirus disease 2019 in a psoriatic patient with concomitant chronic obstructive pulmonary disease under treatment with risankizumab. *Dermatol Ther*. 2020;33:14186. [PMID: 32794375].
5. Wang CJ, Truong AK. COVID-19 infection on IL-23 inhibition. *Dermatol Ther*. 2020. [PMID: 32584451].
6. Ward M, Gooderham M. Asymptomatic SARS-CoV2 infection in a patient receiving risankizumab, an inhibitor of IL23. *JAAD case reports*. 2020;7:60-1. [PMID: 33173806].
7. Chang D, Mo G, Yuan X, et al. Time kinetics of viral clearance and resolution of symptoms in novel coronavirus infection. *AM J Respir Crit Care Med*. 2020;201:1150-2. [PMID: 32200654].
8. Benhadou F, Del Marmol V. Improvement of SARS-CoV2 symptoms following Guselkumab injection in a psoriatic patient. *J Eur Acad Dermatol Venereol.* 2020;34:363-4 [PMID: 32379925].