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COVID-19 and Inflammatory Diseases Triggered by the Immune System

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Perspective

Patients with immune-mediated inflammatory illnesses were thought to be at high risk for SARS-CoV-2 infection and severe COVID-19 at the start of the COVID-19 pandemic. Inflammatory arthritis, psoriasis, and inflammatory bowel illnesses, according to data collected over the past year, do not enhance the risk of SARS-CoV-2 infection or severe COVID-19 infection when compared to those who do not have these conditions. Furthermore, a growing body of evidence suggests that several drugs often used in patients with immune-mediated inflammatory illnesses, particularly cytokine inhibitors, may reduce the risk of severe COVID-19 [1]. Glucocorticoids and maybe B-cell-depleting therapies, on the other hand, appear to impair COVID-19 results. Furthermore, preliminary data on SARS-CoV-2 vaccination in patients with these diseases suggest that vaccination is well tolerated in patients with immune-mediated inflammatory diseases, though the immune response to vaccination may be reduced in this patient group, particularly those on methotrexate or CD20-targeted therapy.

SARS-CoV-2 was the third zoonotic coronavirus to transmit human-to-human and the first to spark a pandemic in 2019. Although the majority of COVID-19 patients have a self-limited upper respiratory tract infection, a small but significant number of patients suffer acute respiratory distress syndrome, which can quickly lead to multi organ failure and death [2].

An overproduction of proinflammatory cytokines such as tumour necrosis factor (TNF), interleukin (IL)-6, growth factors such as granulocyte-macrophage colony-stimulating factor, and chemokines such as IL-8 characterises severe COVID-19, a response known as cytokine storm. Some of the proinflammatory cytokines produced by COVID-19 can be used to treat patients with immune-mediated inflammatory disorders. Inflammatory arthritis, psoriasis, inflammatory bowel illness, and connective tissue disorders are all treated with cytokine inhibitors. As a result, the question of whether cytokine inhibition and immunomodulatory therapy could influence SARS-CoV-2 infection and outcomes has arisen [3]. We present data on the risk of SARS-CoV-2 infection and severe COVID-19 infection in patients with immune-mediated inflammatory illnesses of the joints, skin, and gut, as well as systemic diseases.

Infection with SARS-CoV-2 poses a risk

Patients with immune-mediated inflammatory illnesses are at a higher risk for infections, according to several datasets, and hence may be a vulnerable demographic in the COVID-19 pandemic. However, the situation is convoluted, and there is little direct evidence to support this theory. Patients with immune-mediated inflammatory disorders frequently receive immune-modulating medications and frequently have comorbidities such as cardiovascular, pulmonary, and metabolic disease, all of which increase infection susceptibility and are key drivers of infections in these patients. Although a larger incidence of comorbidities is a consistent predictor for increased infection risk, particularly respiratory tract infections, the effect of therapies cannot be generalised.

Glucocorticoids, for example, are linked to an increased risk of infection, showing that they have wide immunosuppressive effects that not only affect the innate immune system's function but also severely restrict adaptive immunity cells like T cells [4].

Most physicians considered individuals with immune-mediated inflammatory illnesses to be a potentially high-risk population for SARS-CoV-2 infection and a severe course of COVID-19 at the start of the epidemic. However, not just in rheumatology, but also in gastroenterology and dermatology, a large amount of evidence has now been gathered that provides a reassuring message.

COVID-19 and adverse outcomes

COVID-19 is a heterogeneous virus that can cause everything from asymptomatic infection to life-threatening disease and death. Although detecting asymptomatic and moderate infections is notoriously difficult and vulnerable to bias, serious sickness can be assessed using more strict methods such as hospitalisation, mechanical ventilation, and COVID-19-related death—endpoints that have been extensively studied. However, the data generated in this research must be examined for reliability and the possibility of selection bias or confounding. Local virus incidence, proportion of persons immunised, social behaviour, shielding practises, access to testing, and the quality of diagnostic methods, for example, all influence SARS-CoV-2 infection risk; this complexity could lead to measurement error and bias in the results [5].

In addition, for SARS-CoV-2 infection research, include adequate comparison groups is critical. The frequency of hospital admission is also affected by indirect factors such as the local SARS-CoV-2 incidence, general health state of the community, access to hospital care, and local health authority guidelines, so hospitalisation rates may not be interpreted unambiguously as an indicator of COVID-19 severity. Finally, COVID-19-related death may be the simplest way to assess the severity of the disease's progression. Normalization of results with regard to demographics, comorbidities, therapy, socioeconomic factors, and probable colliders is required even for COVID-19-related death to avoid misinterpretation, as demonstrated by the seemingly protective impact of current smoking on COVID-19 mortality.

References

1. Jin, Yuefei, Haiyan Yang, Wangquan Ji, and Weidong Wu, et al. "Virology, epidemiology, pathogenesis, and control of COVID-19." *Viruses* 12 (2020): 372.
2. Guan, Wei-jie, Zheng-yi Ni, and Yu Hu, et al. "Clinical characteristics of coronavirus disease 2019 in China." *N Engl J Med* 382 (2020): 1708-1720.
3. Toscano, Gianpaolo, Francesco Palmerini, Sabrina Ravaglia, and Luigi Ruiz, et al. "Guillain–Barré syndrome associated with SARS-CoV-2." *N Engl J Med* 382 (2020): 2574-2576.
4. Allez, Matthieu, Blandine Denis, Jean-David Bouaziz, and Maxime Battistella, et al. "COVID-19-Related IgA Vasculitis." *Arthritis Rheumatol* (Hoboken, NJ) 72 (2020): 1952-1953.
5. Galeotti, Caroline, and Jagadeesh Bayry. "Autoimmune and inflammatory diseases following COVID-19." *Nat Rev Rheumatol* 16 (2020): 413-414.

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