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LETTER

Psoriasis and psoriatic arthritis: How to manage immunosuppressants in COVID-19 days

Dear Editor,

We read with interest the article "COVID-19 and psoriasis: it is time to limit treatment with immunosuppressants? A call for action" by Conforti et al[1] and, given its actual relevance, we would like to give our comments and contribution from a rheumatologic point of view.

The authors underline the need of reassessing psoriatic patients under immunosuppressive treatment, hence more susceptible to infections, and consider limiting or reducing its administration in endemic areas for Coronavirus Disease 2019 (COVID-19), suggesting eventual switch to topical drugs or medication with lower impact on the immune system.

In the setting of rheumatic disease, in particular psoriatic arthritis (PsA), rheumatologists and dermatologists share indeed many treatments, such as methotrexate, anticytokine biologic therapy (anti-tumor necrosis factor (TNF)-alpha, anti-interleukin (IL)-17, anti-IL-12/23), small molecules, and less frequently cyclosporine. Thus, we are facing similar issues in a region where COVID 19 is definitely endemic, with tens of thousands of cases described to date in Italy.

Even if it is well documented a slight augmented risk of infections with these therapies,[2] we believe that, in accordance with European League Against Rheumatism, American College of Rheumatology, and Italian Society of Rheumatology, unjustified discontinuation of immunosuppressants in PsA, as in other rheumatic disorders, may lead to disease flares, sometimes more harmful than the therapy itself.

Disease flare implies systemic inflammation and immunological disruption, two recognized factors responsible for increasing susceptibility to infection in systemic polyarthritis.[3]

Furthermore, an active disease entails the need of a medical reassessment, which is best to be avoided at this time, given the higher risk of contagion due to moving around and being in the hospital.

Diabetes and metabolic syndrome are acknowledged as associated with PsA and psoriasis[4]; these comorbidities in the setting of a poorly controlled disease may worsen due to inflammation itself.

As already mentioned by the authors, during SARS-CoV-2 (causative agent of COVID-19) infection, pre-existing disorders (such as diabetes, cardiovascular disease, obesity) are detrimental augmenting the risk of severe respiratory syndrome and consequent higher mortality rate.

Hence, when no signs of infection are present, it is even more crucial not to interfere, without a proper indication, with the basal balance of a complex multifaceted disease such as PsA.

It goes without saying that if any signs or symptoms suggestive for infection occur, caused by SARS-CoV-2 or any other infectious etiology, patients should follow their current practice of interrupting immunosuppressive therapy.

Emerging evidence describes a subgroup of patients with severe COVID-19 characterized by a cytokine storm, with increased level of multiple mediators—IL-2, IL-6, IL-7, granulocyte-colony stimulating factor, TNF-alpha, ferritin—reflecting hyperinflammation.[5]

Given these observations, in China it has recently been approved a multicenter randomized controlled (ChiCTR2000029765) trial using tocilizumab (IL-6 receptor blockade indicated in many rheumatic diseases) in patient with elevated IL-6 and SARS-CoV-2 pneumonia.[6] Moreover, a phase 2 study has been approved by the Italian Regulatory Drug Agency and will enroll 330 patients with pneumonia and early respiratory failure, with mortality reduction at 1 month as primary outcome (Multicenter study on the efficacy and tolerability of tocilizumab in the treatment of patients with COVID-19 pneumonia).

When lung and systemic injury results from a hyperactivation of the immune system, immunosuppression might be considered as a therapeutic option, supporting the intricacy of the interaction among the virus and the immunological response depending on every single individual.

In conclusion, before interrupting a chronic therapy, even if patients with PsA have an increased risk of comorbidities and serious infections compared with patients with psoriasis,[7] we suggest evaluating not only the infectious profile of immunosuppressants but also the underlying inflammatory nature of psoriatic disease itself, especially if severe and/or associated with articular involvement.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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