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Eliciting History of Prior Severe Acute Respiratory Syndrome Coronavirus 2 Infection in Diagnosing Interstitial Lung Disease during the Coronavirus Disease 2019 Pandemic



To the Editor:

We read, with great interest, the article by Wong et al¹ in *CHEST* (September 2020) and agree that in the era of coronavirus disease 2019 (COVID-19), we must focus on a systematic approach to the diagnosis of interstitial lung disease (ILD). We highlight here an issue that is not included in the article by Wong and colleagues. Given the uncertainty surrounding long-term outcomes for survivors of COVID-19, including the possibility that these patients may go on to develop ILD, a systematic evaluation of the survivors is prudent,² both to gain a better understanding of long-term pulmonary consequences³ and to evaluate for potential clinical trial enrollment for treatment options.

Given the widespread pandemic, we anticipate two subsets of patients receiving a recent diagnosis of ILD and who need specific attention: first, those with a documented history of positive reverse transcription-polymerase chain reaction-confirmed infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and presenting with persisting dyspnea, abnormal chest imaging results, and pulmonary function test result derangements who will need additional testing to make a diagnosis of ILD. In the absence of other explanations for ILD, infection with SARS-CoV-2 could be considered an etiology of ILD in this group as a diagnosis of exclusion. Second, we anticipate a subset of patients without a documented history of COVID-19 but with a new diagnosis of ILD. In addition to the usual systematic approach to assessing the cause of ILD, obtaining a careful history of symptoms suggestive of COVID-19 is warranted in this group. Many symptoms of COVID-19 are not specific to the virus and can be noted with many other viral illnesses. However, asking for a history of anosmia and dysgeusia, as these symptoms are noted to be more frequently associated with COVID-19, will be valuable.⁴ The authors admit that this information will need to be interpreted in the context of each patient's risk factors for COVID-19, including asking about sick contacts and

the prevalence of local community spread at the time symptoms began.

Definitive research results will eventually become available regarding the long-term outcomes of COVID-19 and the role of antibody testing, but while we are in the midst of a pandemic, assessing patients with a new diagnosis of ILD for a history of prior COVID-19 may present more clinical usefulness in the short term.

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FINANCIAL/NONFINANCIAL DISCLOSURES: None declared.

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DOI: <https://doi.org/10.1016/j.chest.2020.10.028>

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Management Issues of Sarcoidosis in the Time of Coronavirus Disease 2019



To the Editor:

We read with interest the article by Sweiss et al¹ in a previous issue of *CHEST* (September 2020) that provides guidance on how to adjust immunosuppressive therapy in patients with sarcoidosis during the coronavirus disease 2019 (COVID-19) pandemic. The authors point out the importance of reducing the dose of immunosuppressive drugs to decrease the risk of infections and poor outcome. These recommendations are based on the results of some systematic reviews and meta-analyses that were conducted in other rheumatic diseases and that found an increased risk for serious infections, mostly with systemic glucocorticoids and biologic agents.

Although we agree on the need to consider attentively the dosage of immunosuppressive drugs in patients with sarcoidosis to prevent deterioration from COVID-19, we would like to highlight some points.

First, most studies that evaluated the risk of infection were conducted mostly in rheumatoid arthritis, not sarcoidosis.² Immunomodulation differs significantly in sarcoidosis vs the other rheumatic disorders, and the macrophage/T-cell system alteration and granulomas formation represent key steps to trigger and maintain persistent inflammation. Immunosuppressive drugs, therefore, could interfere differently with the immune system.

Second, the specific effect of immunosuppression in the management of COVID-19 is still under study.

Preliminary results from the RECOVERY trial have demonstrated that low dexamethasone doses significantly reduce the 28-day risk of death in patients with COVID-19 who are receiving invasive mechanical ventilation or oxygen.³

The interruption of the hyperinflammatory response has beneficial effects in the early phase of the disease; a rapid dose reduction of immunosuppressive agents such as glucocorticoids thus could worsen the COVID-19 progression. Considering also that an acute relapse of sarcoidosis could contribute to reduce the lung function, we suggest much caution in envisaging a drastic reduction of immunosuppressive therapy.

Third, specifically regarding tocilizumab, even if the compound has demonstrated some benefits in terms of oxygen status improvement in severe COVID-19 pneumonia, its effects on the global survival and in patients with sarcoidosis are unknown.⁴

Last, the recent observation that patients with autoimmune disorders who are treated with disease-modifying antirheumatic drugs are not at increased risk of severe COVID-19 does not exclude the fact that those patients who receive IL-6 or IL-12/IL-23 axis inhibitors might be protected against the severe forms of the disease.⁵

In this view, it could be useful to consider different treatment scenarios, based on the presence of COVID-19 coinfection, baseline treatment, and phase of both diseases. Ad hoc studies are needed urgently to investigate the interaction between the two disorders and how the immunosuppressive treatment could change their natural history.

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FINANCIAL/NONFINANCIAL DISCLOSURES: None declared.

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DOI: <https://doi.org/10.1016/j.chest.2020.09.267>

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Response



To the Editor:

We would like to thank Tana et al for their interest in our commentary.¹ Their comments highlight the ongoing controversy surrounding the issue of management of patients with sarcoidosis during the coronavirus disease 2019 (COVID-19) pandemic. Large cohort studies addressing the issue of COVID-19 in patients with sarcoidosis are lacking. Thus, any guidance is based on expert opinion and extrapolation from other diseases. Obviously, such extrapolation is subject to errors, especially if these diseases are significantly different.

Although we agree with the assertion of Tana et al that immunomodulation differs in sarcoidosis vs other rheumatic diseases, it is unclear how great these differences are. More importantly, it is unclear if these differences have any relevance in terms of the risk and outcomes of infection from immunosuppression.