3 Not All Parenchymal Changes on Computed Tomography Are Interstitial Lung Disease

To the Editor:

We thank the authors for their report outlining the impact of steroid therapy on symptoms, radiology, and lung function parameters (1). We would, however, like to challenge some of the findings.

One concern is that a significant proportion (45.7%) of the “interstitial lung disease” cohort had required mechanical ventilation and 54.5% intensive care admission and is therefore not fully representative of those recovering from coronavirus disease (COVID-19). It is of course possible that persistent computed tomography (CT) changes are associated with the severity of the acute illness, hence the overrepresentation. However, the criteria used by the authors to determine the need for CT imaging (desaturation, abnormal lung function, or abnormal chest X-ray [CXR]) may have influenced this and resulted in missed radiological abnormalities; in the absence of baseline data, it is not possible to conclude that a normal lung function test result is not relatively normal for the patient.

The authors refer to the post–severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection changes on CT as “post–COVID-19 ILD,” but this is an unsubstantiated claim at the present time. The significant improvement in gas transfer and forced vital capacity after steroid therapy is of course staggering, but without a control arm, it is difficult to draw firm conclusions that active therapy is required. As the authors suggest, further study, such as differential cell count analysis of bronchoalveolar fluid, would be useful to ascertain if a persistent inflammatory state is truly the driver for the CT changes. The 15% parenchymal involvement used in this study might not be the optimal cutoff in someone with a significantly inflammatory phenotype on bronchoalveolar lavage.

Overall, 17.1% of patients in the post–COVID-19 interstitial lung disease (ILD) cohort had received inpatient steroid therapy. Inpatient steroid therapy might influence post–COVID-19 radiology changes, especially if the pathology being treated is organizing pneumonia (OP), a highly steroid-responsive condition. With this in mind, the prevalence of CT abnormalities might be lower in the second wave when dexamethasone is part of standard medical care (2). Ground glass changes are a common finding at follow up after OP (3); therefore, the radiology changes noted might simply reflect parenchymal recovery after OP rather than persistent inflammation.

In the SARS-CoV-2 pandemic, improvement in radiology findings was seen with time (4, 5). How to best define “persistent” change with regard to timing of imaging is unknown, but 6 weeks might be too soon and overestimate the prevalence of abnormalities.

It is important to carefully balance the risks of initiating steroid therapy in this cohort. Weight gain during lockdown is a notable issue, and the impact of steroid therapy on body mass index in this cohort would have been interesting to see.

Although the improvement in lung function is remarkable, we should approach the improvement in symptoms with caution as breathlessness post–COVID-19 is multifactorial, including recovery after acute illness, weight gain, and deconditioning. From our single center experience, anxiety regarding further infection often deters patients from exercising outdoors, adding to the problem.

Although this study shows interesting results, there remain many uncertainties, and the etiology of radiological changes and optimal timing of imaging remain speculative at this stage. Longitudinal and mechanistic studies are required to fully understand the pulmonary sequelae post–COVID-19.

Regardless, we thank the authors for their timely and coordinated effort in describing their cohort and response to steroids.

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