completion of dexamethasone and in what patient cohort and using what objective parameters. To further address this unmet need, we propose ILD physicians, respiratory and general physicians, intensivists, and interested others collaborate to generate clinically valid research questions that can be answered by a randomized control trial.

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LETTERS

Re: A Role for Steroids in COVID-19–associated Pneumonitis at Six-Week Follow-Up?

From the Authors:

We thank Denneny and colleagues for their insightful comments and would like to take this opportunity to clarify some of the points raised in their letter.

Early on in the first wave, we began to realize it would be important to understand the natural recovery and the incidence of pulmonary sequelae after coronavirus disease (COVID-19) infection (1). We carefully evaluated 837 recovering patients, first by telephone at 3–4 weeks after discharge, when 316 (38%) reported they were already back to their physical functional baseline. After face-to-face structured assessment and COVID-19 multidisciplinary team meeting discussion, only 59/837 (7%) had persistent post–COVID-19 interstitial changes with associated symptoms and physiological impairment and were therefore referred to the interstitial lung disease (ILD) service. At this stage, 24 patients had either minimal inflammatory infiltrates (<15%) on computed tomography (CT) or an absence of persistent symptoms or physiological impairment, similar to the self-resolving cohort Denneny and colleagues describe. However, this left 35 patients with persistent symptoms causing significant distress at a median of 61 days (9 wk) after discharge from the hospital. This represents only 4.2% of the surviving population, and although almost all our patients were recovering over time, this group of patients demonstrated a large ongoing symptom burden that was directly attributable to the ongoing inflammatory changes seen on CT, predominately organizing pneumonia. This left a clinical decision: to observe or to treat.

Given the risk factors for severe COVID-19 pneumonitis, we agree that steroid therapy is not without risk in this group. Individual patient discussion was key, and hence, five patients did not commence steroid treatment after review. It is therefore worth noting that the quoted proportions of patients with comorbidity refers to the whole cohort referred to the ILD service and not to the treatment group, who had lower rates of comorbidity. All patients had weekly telephone support and diabetes team input as appropriate. As a result, we saw no major complications of treatment.

This is observational work, and in keeping with Denneny and colleagues’ observations, we also saw an improvement in the small number of patients who did not receive treatment and completed 12-week follow-up. However, this improvement was of lower magnitude than the improvement we saw in our patients at 3 weeks after treatment. This study was commenced before the RECOVERY data were published, and only 19% of patients we treated had received any
steroids as inpatients. From the limited literature available, it appears that steroids have a mortality benefit in the acute illness, and this study suggests that there may well also be a morbidity benefit in patients with persistent disease. What will become clear in subsequent waves is whether acute administration is sufficient to obviate the small number of patients who have persistent ILD after COVID-19. We wholeheartedly agree that it behooves ILD physicians to collaborate in order to elucidate the optimal timing and dosing of steroids in COVID-19 pneumonitis and particularly post–COVID-19 ILD, ideally with a randomized controlled trial.

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