Correspondence on ‘Factors associated with COVID-19-related death in people with rheumatic diseases: results from the COVID-19 Global Rheumatology Alliance physician-reported registry’

I read with great interest the recent work from the COVID-19 European League Against Rheumatism registry authored by Strangfeld et al and the COVID-19 Global Rheumatology Alliance describing the risk factors for COVID-19 mortality in patients with rheumatic diseases.

It should perhaps not be surprising that high disease activity in rheumatic diseases is associated with mortality from COVID-19. In rheumatoid arthritis, high disease activity has been shown to be an independent risk factor for severe infection, with a 30% increase in relative risk for every 0.5-unit increase in the 28-joint Disease Activity Score (DAS28). The same authors found no association between either biologic or glucocorticoid use and severe infections. The linear relationship between serious infections and disease activity has also previously been described by Emery et al. Reported serious infections are, however, largely bacterial in nature; susceptibility to severe viral infection appears to be less well understood. Infections are also a leading cause of morbidity and mortality in lupus. Flares in lupus disease activity have been shown to be an independent risk factor for susceptibility to infection, including viral infections. All being said, it is clear that having a disordered immune system due to uncontrolled autoimmune disease predisposes to infection in general and the same appears to be true for susceptibility to severe COVID-19 infection.

The study by Strangfeld et al clearly showed that there is an association between high-dose glucocorticoid use, as defined by a prednisolone dose of 10 mg or above, and COVID-19 mortality. Glucocorticoids are highly effective immunosuppressants and have even been shown to reduce mortality in severe COVID-19 infection by reducing inflammatory burden. It is therefore reasonable to assume that they predispose to infection. In rheumatic diseases, it is well known that glucocorticoids increase the risk of bacterial infection, with one study finding a 10-fold increase in infections with doses of prednisolone 10 mg and above. However, what is less clear is whether glucocorticoids are an independent risk factor for COVID-19 mortality or if they are in covariance with disease activity. Current rheumatological practice aims to minimise glucocorticoid exposure due to their systemic side effects. High-dose glucocorticoids are therefore reserved for flaring patients with a rheumatic diagnosis who have, by definition, high disease activity. There will, however, be a number of patients taking a maintenance dose of prednisolone greater than 10 mg, but these patients will also likely have more severe or difficult-to-treat autoimmune disease and therefore have more disordered immune systems.

There are very few, if any, medical reasons to receive high-dose glucocorticoids in the absence of an inflammatory process. This makes the process of untangling disease activity, high-dose glucocorticoid use and COVID-19 mortality a tricky one.

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