Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

trial (NCT02915159[9]) with a similar design also failed to show efficacy of abatacept on any primary or secondary endpoint (including lack of a clinically relevant improvement in symptoms), and this resulted in the discontinuation of clinical development of abatacept for Sjögren's syndrome.[9] These two negative RCTs[4,9] also show, along with the experience with anti-tumour necrosis factor and rituximab, that repurposing biologics used in rheumatoid arthritis for Sjögren's syndrome might not be successful.

The three trials in *The Lancet Rheumatology* also illustrate the remaining challenges in trial design in Sjögren's syndrome. Using ESSDAI as primary outcome might result in a large placebo effect (up to 55% of patients in the placebo group showed a clinically relevant decrease of at least 3 points in the iscalimab trial[3]). Whether this finding is explained by a time-varying decrease in ESSDAI related to a natural history of the disease (eg, spontaneous alleviation of arthralgias or synovitis, purpura, or parotid swelling), heterogeneous clinical assessment in multicentre trials, difficulties in discriminating disease activity from damage, or the scoring system itself, remains to be determined.

Given the large number of ongoing or future trials that use ESSDAI as the primary outcome, re-evaluating the use of this score as a primary outcome might be important. NECESSITY, a European initiative, will combine data from previous randomised trials, including these three,[3-5] to identify new clinical outcomes in primary Sjögren's syndrome. An initiative from OMERACT on clinical outcomes in primary Sjögren's syndrome is also progressing on this crucial topic.

Despite the small sample sizes and inconsistent results for patient-reported symptoms, and the remaining challenges regarding trial design in Sjögren's syndrome, the positivity of two randomised trials allows us to consider the glass half-full rather than half-empty regarding the future for therapeutic management of Sjögren's syndrome.

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Symmetric peripheral polyarthritis developed during SARS-CoV-2 infection

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For the **COVID-19 Global Rheumatology Alliance** see https://rheum-covid.org/

For the **EULAR Rheumatological Database** see https://www.eular.org/eular_covid19_database.cfm

During the COVID-19 pandemic a number of considerations regarding the interaction of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) with rheumatic diseases have arisen.[1-4] International initiatives were launched (eg, the COVID-19 Global Rheumatology Alliance and the European League Against Rheumatism COVID-19 Rheumatological Database) aimed at examining the health outcomes of patients with rheumatic diseases and COVID-19 on the basis of sociodemographic factors, comorbidities, clinical presentations of COVID-19, and ongoing immunosuppressive drugs before the virus infection.

Aggressive inflammatory responses observed in the severe cases of COVID-19 seem to be linked to a dysregulation of host innate immunity.[5,6] Although many aspects of the hyperinflammatory response in severe

COVID-19 remain to be fully elucidated, it seems that certain patients develop a cytokine storm; this is one of the major reasons why some immunomodulating agents, which inhibit the activity of specific cytokines, are under investigation for the treatment of severe COVID-19 manifestations. Recently, particular attention has been given to reported cases of multisystemic inflammatory disease and atypical Kawasaki disease with concomitant SARS-CoV-2 infection.[7] In view of these considerations and looking at the problem from the rheumatologists' perspective, the suspicion that COVID-19 might represent a trigger for systemic autoimmune diseases is materialising.

We here describe a case of a man aged 45 years with a familial history of ankylosing spondylitis; he was in good health until mid-March, 2020, when he presented with acute symmetric polyarthritis of the metacarpophalangeal and proximal interphalangeal joints of the hands, associated with diffuse myalgia. 1 week after the onset of articular signs and symptoms, he also presented with anosmia and dysgeusia without any respiratory symptoms. On the same day, his wife presented with fever and respiratory symptoms and for these reasons, at the end of March, they underwent nasopharyngeal and oropharyngeal swabs, which were positive for SARS-CoV-2 in both patients. No specific treatments were prescribed after the positive nasopharyngeal and oropharyngeal swab results, and the patient was continuously monitored. During the third week after the onset of arthritis, he had complete remission of anosmia and dysgeusia and partial remission of articular symptoms of the hands and muscular symptoms. Nasopharyngeal and oropharyngeal swabs were repeated twice, once in April and once in mid-May, and both were negative for SARS-CoV-2. However, in the second half of May, he had a worsening of the inflammatory articular symptoms and myalgia, for which he underwent a rheumatological evaluation in the Rheumatology Unit at Azienda Ospedaliero Universitaria Pisana (Pisa, Italy). During the visit, he presented with swollen and tender joints of the hands and ultrasound examinations showed a slight effusion of the right wrist and bilateral effusion of the fifth proximal interphalangeal joint, in the absence of synovial hyperplasia or power Doppler signal. Laboratory tests showed increased concentrations of creatine phosphokinase (279 U/L; normal range <170 U/L), a moderate increase in the erythrocyte sedimentation rate (13 mm/h; normal range 2–10 mm/h), and normal concentrations of C-reactive protein (1·6 mg/L; normal range <8 mg/L); moreover, further laboratory tests were negative for rheumatoid factor and positive for anticyclic citrullinated peptide. Medium doses of methylprednisolone were prescribed (starting from 16 mg with progressive tapering). A few days after the rheumatological evaluation a routine echocardiograph, in absence of any symptoms, showed a moderate pericardial effusion. During short term follow-up in the first week of June, the patient reported complete remission of articular symptoms and clinically significant improvement of overall myalgia; at the end of June, the patient confirmed complete remission, and the corticosteroids were suspended after gradual tapering. However, after the suspension of corticosteroids, the patient reported a slight exacerbation of arthralgia during the first week of July. Further follow-ups in the coming months will tell us whether the case can be considered as an arthritis reactive to the SARS-CoV-2 virus or whether this condition represents the onset of a chronic inflammatory process in which the virus might have acted as a trigger.

The main question raised by this case is whether SARS-CoV-2 infection can accelerate the onset of an autoimmune disease. Currently, no specific answer can be formulated; however, what seems to be crucial is the need for further investigations on the specific mechanisms underlying the virus interaction with the immune system, especially regarding the potential association between the SARS-CoV-2 infection and systemic autoimmune diseases.

We declare no competing interests. The patient has provided informed consent for the publication of this paper.

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