Response to: 'Comparative analysis of synovial inflammation after SARS-CoV-2 infection' by Alivernini *et al*

Few studies have described so far the occurrence of arthritis in patients infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[1] For this reason, we read with great interest the report by Alivernini *et al*[2] where they present two cases of polyarthritis in the context of a SARS-CoV-2 infection, with an exhaustive study of synovial tissue. In the immunohistochemical study of both biopsies, they observed inflammatory infiltrates mostly composed of CD3+, CD138+ and CD68+ cells. Furthermore, in the patient with rheumatoid arthritis, case 2, they also detected CD20+ cells. The study of synovial tissue in the context of SARS-CoV-2 arthritis is of scientific interest, and findings were similar to acute, reactive arthritis to other viral infections, in which an infiltrate of T lymphocytes, monocytes/macrophages and other inflammatory cells is also present.[3] In COVID-19, other tissues studied (pulmonary, myocardial and hepatic) also showed predominantly inflammatory, non-cytopathic changes. Local presence of SARS-CoV-2, demonstrated by reverse transcriptase PCR (RT-PCR), has been detected in several organs, including the lungs, heart, liver, kidneys, gastrointestinal tract, spleen, lymph nodes, skin and placenta.[4] Joints have been scantly assessed. In our series, all synovial fluid samples tested negative for SARS-CoV-2.[5] Here, the authors did not mention whether they had carried out RT-PCR for SARS-CoV-2 in the synovial tissue, what would have been of great interest, especially in case 1.

Regarding the complex issue of management of the infection in the context of patients under immunosuppressant agents, in case 2, methotrexate was stopped at admission. This practice in regard to immunosuppressants after COVID-19 development seems logical and matches the recent American College of Rheumatology recommendations.[6] Conversely, the European League Against Rheumatism[7] advocates discussing case by case between the rheumatologist and the patient due to possible protective effects of some disease-modifying antirheumatic drugs, especially those blocking interleukin-6. Severe COVID-19 poses features that resemble a cytokine release syndrome, and it can be hypothesised that timely intervention at milder stages may impede subsequent worsening.[8] Therefore, maintaining immunomodulatory treatment might help control or prevent the cytokine storm and the inflammatory reaction to SARS-CoV-2. This perspective will likely be clarified by the several ongoing trials testing biological and Janus kinase inhibitors for COVID-19.

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