

Case of acute arthritis following SARS-CoV-2 infection

In a recent issue of *ARD*, López-González and colleagues reported case series of acute arthritis during COVID-19 admission; the authors concluded that all four cases of acute arthritis in their report were due to crystal-proven gout flares or calcium pyrophosphate deposition disease.¹ Here we would like to share another case of COVID-19 presenting delayed arthritis without crystal deposition.

A 57-year-old, male, Japanese patient with a history of hypertension and hyperlipidaemia was admitted to our hospital with cough, fever and malaise of 3 days' duration. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) RNA was positive at admission on real-time (RT) PCR using nasopharyngeal swabs (SRL Inc). Chest X-rays and CT showed multiple, peripheral, ground-glass opacities in the bilateral lungs. The oxygen saturation of the peripheral arteries ranged from 91% to 94% without oxygen therapy.

The patient's symptoms, oxygenation and general condition as well as radiological findings spontaneously improved without specific treatment for SARS-CoV-2 during the 12-day hospitalisation. RT-PCR using the nasopharyngeal swabs for SARS-CoV-2 was negative on day 12. However, his fever recurred on day 15, and multiple joint pain appeared in the left wrist, the right shoulder and the bilateral knees on day 17. His viral pneumonia resolved and did not recur. His serum C reactive protein level increased from 0.4 mg/dL to 4.8 mg/dL between days 12 and 19. RT-PCR for SARS-CoV-2 using a nasopharyngeal swab returned positive on day 14. Antinuclear antibodies, rheumatoid factor, anticyclic citrullinated peptide antibodies, hepatitis B virus surface antigen, antihepatitis C virus antibodies and anti-human immunodeficiency virus antibodies were negative. A joint examination revealed slight swelling of the right knee. Synovial fluid aspirated from the right knee joint on day 23 was negative for SARS-CoV-2 RNA on RT-PCR and free from crystals on polarised microscopic examination. SARS-CoV-2 RNA was not detected in the serum or whole blood. The arthritis resolved spontaneously on day 27, and RT-PCR for SARS-CoV-2 using nasopharyngeal swabs was negative on days 19 and 21.

Viral arthritis is caused by several mechanisms induced by direct invasion and immune complex formation. Viral particles may act as antigenic components in immune complexes formed by the humoral response to viral infection. These immune complexes may be preferentially deposited in the joints, leading to arthralgia and arthritis.

SARS-CoV-2 viraemia reportedly occurs in 15% of COVID-19 cases² but was absent in the present case, which shared clinical

features with arthritis due to infection by hepatitis B virus, hepatitis C virus, parvovirus or alphavirus, such as chikungunya. Since the aetiology of acute arthritis in COVID-19 is still unknown,³ we wished to share the present case of acute arthritis following SARS-CoV-2 infection.

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