Comment on: Palindromic rheumatism following COVID-19 infection evolved to rheumatoid arthritis after COVID-19 reinfection

Sirs,

We have read with interest the article by E. Ben-Chetrit and E. Ben-Chetrit, which was published recently in *Clinical and Experimental Rheumatology*, dealing with rheumatoid arthritis (RA) development following COVID-19 (1).

In this paper, the authors concluded that a temporal relationship exists between the RA development following severe acute respiratory syndrome Coronavirus-2 (SARS-CoV-2) infection. Recently, we reported a patient with COVID-19 who developed an erosive seronegative RA six months after SARS-CoV-2 infection (2).

Autoimmune rheumatic diseases (ARDs) are frequently described in patients with COVID-19. These are expressed as autoimmune phenomena such as the presence of autoantibodies or/and organ-specific ARDs. Indeed, the clinical features range from the discovery of an autoantibody, mainly antinuclear antibodies (ANA), or antiphospholipid antibodies (APL), to organ-specific and systemic diseases like thyroiditis, myocarditis, myositis, peripheral neuropathy, to antiphospholipid syndrome (APS), RA-like disease and many others (3). Some investigators concluded that COVID-19 is a mimicker of ARDs and should be excluded to ensure a correct diagnosis (4). On the other hand, COVID-19 may trigger autoimmune responses and the development of ARDs, as in the case reported above (5). This hypothesis is supported by the fact that SARS-CoV-2 might trigger autoimmune responses through molecular mimicry. Still, several other viruses have been implicated as possible aetiologic factors for the development of RA. Among them is the Epstein-Barr virus (EBV), which can induce immune responses through molecular mimicry and is a polyclonal activator of B-cells with the generation of autoantibodies, primarily rheumatoid factor (RF) (6).

Recent studies support the hypothesis of molecular mimicry as a possible mechanism responsible for the development of ARDs in patients who contracted SARS-CoV-2 infection (7, 8). Thus, SARS-CoV-2 may trigger autoimmunity and lead to the potential progression of ARDs, among which is RA.

A.A. Drosos, *MD, FACR*
E. Pelechas, *MD*
A.I. Venetsanopoulou, *MD, PhD*
P.V. Voulgari, *MD*

ORCID iD:
A.A. Drosos: 0000-0002-2232-0326
E. Pelechas: 0000-0002-9383-5722
A.I. Venetsanopoulou: 0000-0003-4280-9193
P.V. Voulgari: 0000-0002-5193-2284

Rheumatology Clinic, Department of Internal Medicine, Medical School, University of Ioannina, Ioannina, Greece.

Please a address correspondence to:
Alexandros A. Drosos,
Rheumatology Clinic,
Department of Internal Medicine,
Medical School, University of Ioannina,
Ioannina 45110, Greece.
E-mail: adrosos@uoi.gr

Competing interests: none declared.

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