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LETTER TO THE EDITOR

Long COVID from rheumatology perspective: a simple mimicker or promoter of autoimmunity?

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Dear editor,

We have read with great interest the review article by Sapkota et al. which has been recently published in the *Clinical Rheumatology* journal dealing with long COVID [1]. In this paper, the authors reported the symptoms and immunological findings of patients who were infected from severe acute respiratory syndrome coronovirus-2 (SARS-CoV-2). These symptoms and laboratory features share similarities with those of patients suffering from autoimmune rheumatic diseases (ARDs). They concluded that long COVID is a mimicker of ARDs and needs to be excluded to ensure a correct diagnosis [1].

Recently, we reported a patient who contracted SARS-CoV-2 infection and developed an erosive seronegative arthritis six months after infection [2]. Musculoskeletal, cutaneous, and other systemic manifestations, along with the presence of autoantibodies, are frequently observed in these patients. On the other hand, SARS-CoV-2 may trigger autoimmune responses and the development of de-novo manifestations of ARDs, as in our patient [3]. The pathogenesis of these phenomena is not well defined. One hypothesis implies the presence of autoantibodies against interferon (IFN) type-I, or inborn errors in the type-I IFN immunity [4, 5]. Another hypothesis is that SARS-CoV-2 might trigger autoimmune responses through molecular mimicry [3]. Several viruses have been implicated as possible etiological factors for the development of ARDs, mostly systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and others. Between viruses Epstein-Barr virus (EBV) is implicated in the pathogenesis of the above disorders [3, 6]. Indeed, EBV can trigger immune responses through molecular mimicry and is a polyclonal activator of B-cells and increases the production of rheumatoid factor (RF). Several studies suggested that molecular mimicry is a possible mechanism responsible for the development of ARDs in SARS-CoV-2 infection [7, 8]. Thus, SARS-CoV-2 may trigger autoimmunity and the possible development of the de novo manifestations of ARDs.

Declarations

Disclosures None.

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