Correspondence

A concomitant diagnosis of COVID-19 infection and systemic lupus erythematosus complicated by a macrophage activation syndrome: A new case report

Dear Editor,

Systemic lupus erythematosus (SLE) is an autoimmune, chronic, and multifactorial disease with multi-systemic involvement, characterized by the breaking of tolerance toward nuclear self-antigens inducing the production of pathogenic autoantibodies.

Viral antigens are already known to be potential triggers of SLE. Few reports theorized that SARS-CoV-2 could also be implicated in the SLE pathogenesis.[1]

A 25-year-old female patient, with no particular medical history, presented to our dermatology department with a 15-day history of a rash with diffuse myalgias, asthenia, and fever.

Upon physical examination, the patient was conscious, afebrile with stable underlying respiratory status. Skin examination revealed a diffuse maculopapular exanthema with palmar-plantar involvement, periorbital edema (Fig. 1a), infiltrated purpuric lesions of the lower limbs, with multiple labial and palatal erosions. (Fig. 1b). Anamnesis did not objectify photosensitivity, hair loss, or joint paint, nor any drug intake in the previous year. HIV, syphilis, and rickettsiosis were suspected and serologically discarded in our patient.

Chest computed tomography (CT) scan was performed and showed bilateral pulmonary infiltrates of probable viral origin, the polymerase chain reaction PCR test on a nasopharyngeal swab specimen was positive for SARS-CoV-2 RNA, and the patient was admitted to a COVID-19 intensive care unit (ICU).

Figure 1 Systemic lupus erythematosus in a female patient: (a) Periorbital edema, (b) Multiple labial and palatal erosions

During her stay in the ICU, the biological analysis revealed an aregenerative normocytic normochromic anemia, leukopenia, neutropenia, and lymphopenia. The immunological assessment revealed positive antinuclear antibodies, positive DNA antibodies, low complement levels, negative antiphospholipid antibodies, and positive proteinuria. The cardiac ultrasound objectified a stage 3 mitral insufficiency without pericardial effusion. Skin biopsy with direct immunofluorescence was in keeping with SLE with leukocytoclastic vasculitis.

The patient was diagnosed with SLE combined with COVID-19.

After 4 days, she also developed a high level of ferritinemia at 1010 and hypofibrinemia. The LDH level was 510, high liver enzymes and hypertriglyceridemia were noted, which led to the diagnosis of macrophage activation syndrome (MAS). The patient was started on steroid therapy with methylprednisolone along with the usual treatment for COVID-19. The follow-up was marked by clinical and biological improvement.

The literature data concerning the association of lupus and COVID-19 is sparse. To our knowledge, few cases reported the onset of SLE following COVID-19,[2,3] but only one case report prior to this one documented the concomitant occurrence of clinical manifestations of SLE and COVID-19 infection in an 18-year-old female.[4]

Classically, it is accepted that genetic, epigenetic, environmental, hormonal, and immunoregulatory mechanisms triggers the loss of autoimmunity tolerance and consequently the multivisceral dysfunction observed in various autoimmune diseases. In particular, during SLE, it is well established that viruses induce the activation of an aberrant innate and acquired immune response in genetically predisposed individuals (especially those with IL-6 genetic polymorphisms), resulting in high cytokines release, mainly TNF-α, IL-6 and IL-1β, IL-17, IL-18, and elevated chemokines CCL3, CCL5, CCL2, and CXCL10. On the same note, higher serum levels of these same proinflammatory cytokines were found in patients with severe COVID-19, which could explain the onset of SLE in COVID-19 patients.[1]

Another interesting aspect of our case is the development of MAS syndrome following the SLE diagnosis. The MAS is probably due to a cytokine "storm" and immune dysregulation caused by the SARS-CoV-2 leading to severe tissue damage.[5]

This being said, clinicians should remain careful while diagnosing SLE in the setting of COVID-19 as clinical symptoms of COVID-19 can easily mimic SLE symptoms, and

temporary autoantibodies can be detected as a response to infections, therefore a clinical and immunological follow-up of these patients is recommended to finally retain the SLE/COVID-19 diagnosis.

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