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Case Report

A Case Report of in Two Times Psoriatic Arthritis and Autoimmune Hepatitis After Sars Cov2 Infection

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Abstract

Autoimmune diseases such as autoimmune hepatitis (AIH) and psoriatic arthritis (PsA) can occur in predisposed individuals in which an immune-mediated reaction against hepatocytes and joint cells is triggered by environmental factors. Lately, several cases are emerging that correlate SARS COV2 vaccination with the development of AIH [1]. We report a case of PsA and AIH triggered by two-stage SARS COV2 infection in a health worker (doctor) contracted in the workplace during the first pandemic wave.

Keywords: *Autoimmune Hepatitis; COVID-19; SARS-CoV-2; Psoriatic Arthritis*

Introduction

Autoimmune diseases such as autoimmune hepatitis (AIH) and psoriatic arthritis (PsA) can occur in predisposed individuals in which an immune-mediated reaction against hepatocytes and joint cells is triggered by environmental factors.

Case Presentation

Female patient, doctor, aged 42, affected from the age of 18 from mild inverse psoriasis, treated exclusively with steroid-based ointments locally and occasionally, she contracted a covid-19 infection in the workplace during the firstsevere covid pandemic.

The Cov2 Sars infection progressed with mild symptoms, predominantly lower back pain, headache, anosmia, and exertional dyspnea. It was not necessary to resort to hospitalization and home therapy was mainly with symptomatic pain medications. The positivity to the throat swab lasted about 3 months.

After 2 months the patient began to experience pain and edema of the left hand with functional impotence. An X-ray and an ultrasound of the hand and a rheumatological evaluation were performed which confirmed the change from psoriasis to psoriatic arthritis. Therefore, the patient received high dose steroid therapy and cyclosporine.

Blood checks were performed every 3 months to monitor hepatic and renal function, which were always normal. There was also a total remission of arthritic and cutaneous symptoms.

After 6 months from the onset of symptoms, the patient began to suffer from repeated episodes of vomiting and mild abdominal pain. Blood tests were performed which documented an increase in hepatic indices. In suspicion of cyclosporine intoxication, the drug was discontinued. Liver ultrasound was normal, except for a mild steatosis.

The symptoms worsened, however, as did the liver values. It was therefore decided to submit the patient to a liver blot and a liver biopsy. Rheumatological examinations have shown a positivity of ANA and ASMA (Figure 1).

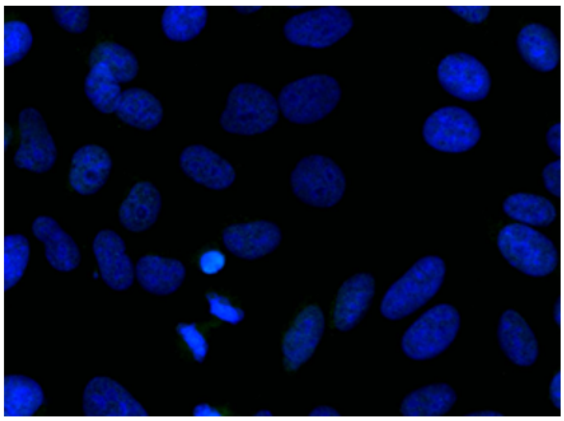

Figure 1

Liver biopsy showed an increase in the density of stromal collagen and inflammatory infiltrate consisting mainly of lymphocytes and some eosinophils. Areas of confluent necrosis and periportal fibrosis were also present in the hepatic lobules (Figure 2).

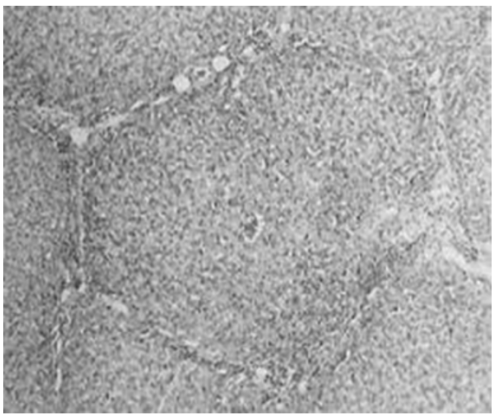

Figure 2

Confirmed the diagnosis of autoimmune hepatitis, the patient started therapy with azathioprine and prednisone.

One year later, the situation is under pharmacological control.

Discussion

Psoriatic arthritis (PsA) has been defined as inflammatory, usually seronegative, arthritis associated with psoriasis. Inflammatory arthritis occurs in 2 - 3% of the general population, but among patients with psoriasis the prevalence of inflammatory arthritis ranges from 6% to 42%.

Among possible trigger factors of autoimmune diseases, immunization and viral infections are mentioned in the literature, although the underlying immunological mechanism is still unclear. The most acceptable hypothesis involves the possible molecular mimicry existing between viral and epidermal proteins, and over activation of the immune system as a consequence of the viral attack [8]. Indeed, in autoimmune disorders, once the autoantibodies bind to their targets, namely self-structural proteins, several pathways are activated, including complement activation and deposition, and neutrophilic chemotaxis, with the release of proteases and elastases that lead to blister formation and of cytokines such as IL-6 and IL-8, which recruit additional immune cells [6,7]. The cytokine storm phenomenon in severely infected SARS-CoV-2 patients has been thoroughly explored and reported in critically ill COVID-19 patients [6,7]. Macrophages, considered one of the major immune populations residing in the lung parenchyma, have been suggested to play a critical role in the pathophysiology of SARS-CoV-2-induced acute respiratory distress syndrome (ARDS) and acute respiratory distress syndrome (ARDS) induced by SARS-CoV-2. Manifestations in critically ill patients. SARS-CoV-2 can trigger an overstimulation of the immune system [6] in genetically predisposed individuals which can lead to an excessive activation of local macrophages to produce a high level of inflammatory mediators such as: cytokines, chemokines and ferritin. The overproduction of cytokines by macrophages has been shown to improve the inflammatory process and trigger an unusually large amount of ferritin in the blood [7,8]. It has recently been shown that on admission to hospital, SARS-CoV-2 infected patients have a high level of ferritin [8] similar to what happens in other diseases triggered by other pathogenic viruses.

Conclusion

Coronavirus 2 (SARS-CoV-2) infection of severe acute respiratory syndrome has been associated with the development of autoimmune processes. Molecular mimicry has been suggested as a potential mechanism for these associations [2]. Growing evidence accumulated over the past year on the outbreak of the 2019 corona virus disease (COVID-19) pandemic, suggesting a strong association between severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and severe acute respiratory syndrome 2 (SARS-CoV-2) coronavirus infection. autoimmunity. Patient-reported inflammatory/autoimmune symptoms, the appearance of circulating autoantibodies and the diagnosis of several defined autoimmune diseases in a subset of patients infected with SARS-CoV-2, indicate the critical and fundamental effect of the SARS-CoV-2 virus. On human immunity, and its ability to trigger autoimmune diseases, in genetically predisposed individuals.

Overall, the link suggested above between SARS-CoV-2 infection and autoimmunity can be demonstrated by the concept of autoimmune/inflammatory syndrome induced by exposure to an external stimulus (such as: infections, adjuvant, vaccine and silicone) [6,9]. In this regard, a viral infection such as SARS-CoV-2 can trigger: i) a strong activation of the immune system; ii) the appearance of 'typical' clinical manifestations such as: myalgia, myositis, arthralgia, chronic fatigue, sleep disturbances, neurological manifestations, cognitive impairment, memory loss and pyrexia - all already reported in patients with SARS-CoV-2 infection; iii) the appearance of autoantibodies, which can lead to the development of autoimmune diseases in genetically predisposed subjects (e.g. HLA-DRB1 etc.) [8,9].

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