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Letters to the Editor

SARS-CoV-2 infection triggering a giant cell arteritis

Infección por SARS-CoV-2 como desencadenante de una arteritis de células gigantes

Dear Editor:

Introduction

Different infectious agents have been suggested to be involved in the pathogenesis of both classical and self-limited Giant Cell Arteritis (GCA).

Case report

On 14th March 2020, a 50-year-old-man without past medical history was assessed through teleconsultation with a dermatologist during the state of alarm due to Covid-19 in Spain. He reported high fever, cough and severe headache with bilateral temporal arteries thickening. No diagnostic tests could be performed at that time. As a non-severe SARS-CoV-2 infection was suspected, and visual or osteomuscular alterations were not reported, we opted for a late referral to specialized care and remote monitoring of the symptoms.

One month later, the patient presented not any more Covid-19 symptoms but reported persistent headache and temporomandibular joint pain. Clinical examination revealed swelling and inflammation of his right temple, where a filiform pulse was noted. A notable improvement from the previous temporal thickening was observed. At that time, several diagnostic tests were performed. Blood tests yielded normal or negative results, including erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and autoimmunity profile; Covid-19 IgM/IgG Rapid Test (VivaCheck Biotech (Hangzhou) Co., Ltd.) was positive for both IgG and IgM; and a Doppler ultrasound of the right temporal artery showed a dark halo around lumen with a marked flow impairment, suggesting arterial wall inflammation, while left temporal artery Doppler echography was normal.

Two weeks later, an FDG PET-CT scan was performed, showing a slight increase of metabolic activity in the abdominal aorta, with a maximum standardized uptake value of 2.3 g/ml compared to 2.2 g/ml in the liver, without current active vasculitis signs.

Follow-up at three weeks revealed spontaneous clinical improvement with no corticosteroid treatment needed and a new temporal artery Doppler ultrasound was performed showing a resolution of arterial wall inflammation and blood flow.

Taking into account the complementary tests and the clinical evolution, we conclude that the most likely diagnosis was a Giant Cell Arteritis (GCA).[1] Given the coincidence in time with the surrounding SARS-CoV-2 infection we hypothesize that the virus could have acted as a trigger, because of its affinity for vascular endothelia. Varicella Zoster Virus (VZV),[2] *Chlamydia pneumoniae*, Parvovirus B19 and Epstein Barr Virus, have been suggested to trigger GCA. Our patient presented atypical clinical features of CGA with spontaneous resolution, which supports a virus-related pathogenesis. In addition, other vasculitis, such as Kawasaki disease in children or neurological complication with CNS vasculitis-like pattern, have been recently linked to Covid-19,[3,4] which supports our hypothesis.

Our main limitation is the lack of histological confirmation. However, the absence of any biologic abnormality in blood tests could be explained by the fact that biologic tests were performed after the patient presented with symptoms.[5] At the same time, we should take into account that general systemic symptoms, evaluated by telephone triage or similar, might be wrongly attributed to Covid-19, leading to the delayed diagnosis of this rheumatologic condition, which in turn could prompt to an irreversible visual loss, highlighting the severity of indirect morbidity related to Covid-19.

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Contributors

All authors have made substantial contributions in each of the following aspects: study conception and design, analysis and interpretation of data, draft manuscript, critical review of its intellectual content and definitive approval of the final version.

Patient and public involvement

This research was done without patient involvement. Patients were not invited to comment on the study design and were not consulted to develop patient relevant outcomes or interpret the results. Patients were not invited to contribute to the writing or editing of this document for readability or accuracy.

Competing interests

No, there are no competing interests for any author.

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Influenza vaccine in patients on biologic therapy; also with belimumab☆

Vacuna de la gripe en pacientes tratados con fármacos biológicos; también con belimumab

To the Editor:

We have read with interest the article recently published in your journal by Richi et al. on influenza vaccine response in patients receiving biologic therapies[1]. Multiple biologics were included in the study, but we have missed belimumab, a biologic agent approved for the treatment of systemic lupus erythematosus (SLE)[2].

The pivotal study of belimumab in SLE, BLISS-76, which included patients treated with placebo or monthly intravenous belimumab for 76 weeks, assessed a group of patients who had received different vaccinations, including the influenza vaccine. Antibodies were determined at baseline and at 52 weeks, and the percentage of change in the levels and the proportion of patients who maintained the levels were assessed. No significant changes were observed in the antigens of the vaccine received in 2007–8, nor in the percentage that maintained titers. In patients who received influenza vaccination, overall, the titers increased significantly, although it was higher in patients with placebo than those treated, although in some strains, namely Brisbane 10 and 59, the percentage of patients with titres >1:10 was lower in treated patients[3]. The authors conclude that belimumab treatment does not affect pre-existing antibodies in response to influenza vaccination in SLE patients, and that there does not appear to be an increased risk of inadequate response to vaccination during belimumab treatment.

The current recommendations with the available results are in favour of influenza vaccination of patients with SLE under treatment with belimumab[4]. We vaccinate all our treated patients in our routine practice.

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Reply☆

Respuesta

We appreciate the opportunity to respond to the interesting letter that Callejas JL et al. have sent to your journal commenting on our work. Although we recruited patients with connective tissue diseases in our study, none of the patients included suffered from systemic lupus erythematosus (SLE). As belimumab is a drug approved exclusively for the treatment of SLE and there were no participants with SLE, we were not able to study the behaviour of the vaccine in patients receiving belimumab treatment. The European League Against Rheumatism (EULAR) vaccine recommendations, updated in 2019, include annual influenza vaccination for patients with inflammatory autoimmune diseases receiving immunosuppressive treatments.[1] These treatments include beli-

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