Letter

Case of Giant Cell Arteritis After SARS-CoV-2 Vaccination: A Particular Phenotype?

To the Editor:

We read with great interest the article by Mehta et al about the similarities and discriminators between giant cell arteritis (GCA) and the coronavirus disease 2019 (COVID-19).¹ Viruses have been suspected to be implicated in the pathogenesis of GCA, especially the varicella zoster virus,² but a clear association has not been confirmed.³ No connection between SARS-CoV-2 and GCA has been established as yet, but an increased number of GCA diagnoses was reported during the COVID-19 pandemic.⁴ Cases of GCA or polymyalgia rheumatica after influenza vaccination were also reported.⁵ Here we report a case of GCA development after receipt of a SARS-CoV-2 vaccine. Written informed consent was provided by the patient.

A 70-year-old man was admitted for headache and hyperesthesia of the scalp. These symptoms started a few days after his first dose of the ChAdOx1 nCoV-19 SARS-CoV-2 vaccine (AZD1222). The clinical examination found an induration of the left temporal artery, no vascular murmur, and no jaw claudication. The fundus examination and the fluorescein angiography were normal.

The blood tests showed an anicteric cholestasis (gamma-glutamyltransferase 112 U/L, alkaline phosphatase 40 U/L), C-reactive protein (CRP) at 5.0 mg/L, and a prolonged activated partial thromboplastin time at 1.6. SARS-CoV-2 serology was positive for IgG (1336 U/mL) and negative for IgM.

The cerebral computed tomography (CT) scan was normal. The contrast-enhanced CT did not show any sign of vasculitis. An 18F-fluorodeoxyglucose positron emission tomography did not detect large vessel vasculitis. The biopsy of the temporal artery showed intima thickening, fragmentation of internal elastic lamina, and moderate infiltration of the media with giant cells, confirming the diagnosis of GCA. The upper CRP level observed during the hospitalization was 13.5 mg/L. The patient was given 0.5 mg/kg/day of prednisone and the symptoms regressed almost completely the following day. The CRP normalized and the anicteric cholestasis decreased rapidly. HLA test showed HLA-DR4.

To the best of our knowledge, this the first case of GCA after a SARS-CoV-2 vaccine. We found similarities with a case reported after SARS-CoV-2 infection.⁶ Both patients had low levels of CRP for GCA and presented HLA-DR4. The increased prevalence of HLA-DR4 in GCA has been studied for a long

time.⁷ This allele could be correlated with the development of GCA after a viral infection. The low level of CRP could be a confounding variable in the diagnosis of GCA.

In conclusion, we suggest an interaction between GCA and not only SARS-CoV-2 infection but also the SARS-CoV-2 vaccine, perhaps with a particular phenotype with low levels of CRP and HLA-DR4. In this situation, we reiterate that a low level of CRP does not rule out the diagnosis of GCA.

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The authors declare no conflicts of interest relevant to this article.

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