Humoral and T-cell immune response following three doses of SARS-CoV-2 mRNA vaccine in giant cell arteritis: analysis of a prospective cohort

Dear Sir,

The present study describes SARS-CoV-2-specific humoral and cellular response in patients with giant cell arteritis (GCA) receiving three mRNA vaccine doses.

GCA patients followed up at the Rheumatology clinic of Policlinico San Matteo, Pavia, Italy, were assessed for their immune status before (T2) and after (T3) the third anti-SARS-CoV-2 mRNA vaccine (BNT162b2 Pfizer/BioNTech or mRNA-1273 Moderna). Anti-SARS-CoV-2 trimeric spike (S-IgG) and neutralising (NtAbs) antibodies were measured (cut-off 33.8 Binding Antibody Units (BAU)/mL and 1:10 dilutions, respectively). T cell response was estimated via enzyme-linked immunospot assay (cut-off 10 IFN-γ spot-forming units [SFU]/10 PBMC). Patients contracting COVID-19 between the onset of the pandemic and T3 were excluded. Healthy controls were used as a comparison.

Forty-two patients were enrolled (Table I), 31 (73.8%) were females, median age 73 years (IQR 69-82). Thirty-three (78.6%) were on glucocorticoids (GCs) at a median dose of 3.8 mg/day prednisone-equivalents (IQR 1.3-5.0). Methotrexate (MTX) was the additional immunosuppressant in 17 (40.5%) patients, while six (14.3%) were treated with subcutaneous tocilizumab 162 mg/week (TCZ). At T2, 95.2% patients retained S-IgGs, 69.0% NtAbs, and 76.2% neutralising GC+MTX group (57.1%) had a detectable seroconversion at T3, while TCZ did not (2, 3). Patients who got COVID-19 had a poorer T-cell response: only eight patients taking GC+MTX (57.1%) had a detectable cellular response vs. 25 (92.6%) of the non-GC+MTX group (p=0.04).

Three patients (7.1%) experienced a GCA flare 24±5 days after the third dose. Nine (21.4%) had COVID-19 at a median of 3.1 months (IQR 2.0-6.3) after the vaccine, none of them requiring hospitalisation.

To conclude, three vaccine doses conferred excellent seroconversion rates in a cohort of elderly patients receiving immunosuppressants. Nevertheless, immunosuppressive treatment reduced S-IgGs and NtAbs titres and influenced the cellular response. As experienced with the first immunization round, GC partially contributed in decreasing the cellular immune response to the third dose (1). MTX significantly impaired humoral immune response and it might contribute to decreasing the cellular one, especially when combined with GCs, while TCZ did not (2, 3). Patients who got COVID-19 had a favourable outcome, in accordance with the previous literature evidence (4).

Table I. SARS-CoV-2-specific immune response 6 months after the second vaccine dose (T2), and three weeks after the third (T3) compared to healthy controls. Humoral and cellular response in patients treated with MTX was weaker to those who were not.

<table>
<thead>
<tr>
<th>Table I</th>
<th>SARS-CoV-2-specific immune response 6 months after the second vaccine dose (T2), and three weeks after the third (T3) compared to healthy controls. Humoral and cellular response in patients treated with MTX was weaker to those who were not.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall T2 overall T3</td>
<td>value (n=42)</td>
</tr>
<tr>
<td>S-IgGs, n (%)</td>
<td>90 (95.2)</td>
</tr>
<tr>
<td>S-IgG titre, BAU/mL, median (IQR)</td>
<td>138 (85-320)</td>
</tr>
<tr>
<td>NtAbs, n (%)</td>
<td>90 (95.2)</td>
</tr>
<tr>
<td>NtAbs titre, t:10 dilutions, median (IQR)</td>
<td>10 (5-40)</td>
</tr>
<tr>
<td>T cell responders, n (%)</td>
<td>32 (76.2)</td>
</tr>
<tr>
<td>T cell response, IFN-γ SFU/10^5 PBMC, median (IQR)</td>
<td>15 (10-39)</td>
</tr>
</tbody>
</table>

References
1. MONTI S, FORNARA C, DELVINO P et al: Immunosuppressive treatments selectively affect the humoral and cellular response to SARS-CoV-2 in vaccinated patients with vasculitis. Rheumatology

Letters to the Editors
Clinical and Experimental Rheumatology 2023

A. BARTOLETTI1,2, MD
C. FORNARA1, MD
P. DELVINO1,2, MD
G. FRANCHI1, MD
A. FERRAREI1, MS
F. BALDANTI3, Prof
C. MONTUCCIO2, Prof
S. MONTI1,2, MD, PhD
1Department of Internal Medicine and Therapeutics, Università di Pavia;
2Division of Rheumatology, Fondazione IRCCS Policlinico San Matteo, Pavia;
3Department of Microbiology and Virology, Fondazione IRCCS Policlinico San Matteo, Pavia;
4PhD in Experimental Medicine, University of Pavia, Italy.

Please address correspondence to: Alice Bartoletti, Division of Rheumatology, Fondazione IRCCS Policlinico San Matteo, Department of Internal Medicine and Therapeutics, Università di Pavia, Piazzale Golgi 2, 27100 Pavia, Italy. E-mail: alice.bartoletti01@universitadipavia.it

Competing interests: none declared.
Letters to the Editors

