Response to: ‘Correspondence on ‘Second COVID-19 infection in a patient with granulomatosis with polyangiitis on rituximab” by Tampe et al

We read with great interest the report by Tampe et al regarding their patient with granulomatosis with polyangiitis on rituximab who developed a serological response to SARS-CoV-2 and attenuated viral spread only after B cell reconstitution.1 This is an important observation, which is logical and further supports the recommendations to time the SARS-CoV-2 vaccine towards the end of the rituximab cycle and, if possible, to delay rituximab until 2–4 weeks after the second SARS-CoV-2 vaccination.2 3 While we agree with the authors that waiting for B cell reconstitution might improve response to the SARS-CoV-2 vaccine, patients on rituximab can have prolonged B cell depletion (as seen in this case) lasting months or years4—making this strategy impractical for many patients. In addition, it appears that the SARS-CoV-2 vaccines induces both B and T cell responses,5 although this remains incompletely understood. Given the urgency of the pandemic, timing the vaccine with rituximab dosing intervals rather than B cell reconstitution is likely a better strategy for most patients. Booster vaccinations will also likely need to be evaluated in rituximab-treated and other immunocompromised patients, as such strategies may be helpful in overcoming inadequate vaccine responses.

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