Journal of Cytokine Biology
ISSN: 2576-3881
Short Communication Open Access

Auto Immune Response to COVID-19

George Kassiotis*

Department of Infectious Disease, St Mary's Hospital, Imperial College London, London W2 1PG, UK

Coronavirus is rapidly spreading over the world and is continuously doing havoc. According to studies, cytokine storms and T-helper abnormalities play a key role in the majority of acute cases of the condition. A variety of drugs have been recommended to treat or control the condition, but they have all been withdrawn due to negative effects. Melatonin, as an intrinsic molecule, has pharmacological anti-inflammatory and antioxidant capabilities, but its concentration diminishes with age, making older people more susceptible to numerous ailments. The COVID-19 outbreak raises serious concerns about vaccine efficacy for those taking the anti-CD20 drug ocrelizumab. The humoral response to SARS-CoV-2 infection and vaccination has been demonstrated to be reduced by ocrelizumab, although the T-cell response to vaccination has yet to be fully defined [1]. We wanted to see what B and T-cell responses to SARS-CoV-2 vaccination looked like in ocrelizumab-treated patients, and what characteristics correlated with vaccine immunogenicity. PwMS receiving glatiramer acetate, Interferon-ß, Dimethyl fumarate, Cladribine or Natalizumab had intact humoral and cellular immune responses following vaccination against SARS CoV-2. B-cell depleting therapies reduced B-cell responses but did not affect T cell responses. Sphingosin-1-Phospate (S1P) inhibitors strongly reduced humoral and cellular immune responses.

We reasoned that patients who did not have a humoral response to the SARS-CoV-2 vaccine would nevertheless have healthy T-cell responses. Selenium, an essential trace element, is critical for human health and, in particular, for the proper functioning of the immune system. Se deficit in COVID-19 patients was linked to disease severity and death risk during the current epidemic. Selenium has been linked to a positive immunological response after vaccination, although it's unclear whether this holds true for SARS-CoV-2 vaccinations. The immunological factors involved in protection against SARS-CoV-2 infection are not well defined or understood [2]. Previous knowledge of the related SARS virus and other human coronaviruses, on the other hand, may be valuable. Anti-SARS-CoV-2 antibodies measured in population-based serosurveys could give a pattern for assessing infection levels and tracking the epidemic's progression. To combat the COVID-19 pandemic brought on by SARS-CoV-2 infection, significant progress has been achieved in identifying infected individuals and detecting those who have a positive immunological response to the virus. Attempts to develop a vaccine against the coronavirus are now underway.

The protocol followed the manufacturer's instructions. Plasma not immediately processed was stored cell-free under −17 °C for a maximum of 2 weeks. The concentration of released interferon-gamma in the plasma is then determined. The interferon-gamma concentration in the plasma of the BLANK represents the individual interferon-gamma background and was subtracted from the interferon-gamma concentration of the plasma, in tubes TUBE and STIM. After BLANK subtraction, the interferon-gamma concentration in the STIM condition must still be higher than the BLANK value itself in order to ensure a sufficient number and stimulability of the immune cells. Concentrations greater than 2500IU/µl were not titrated in the clinical routine analysis. The manufacturer defines values greater than 120 IU/µl to be stimulable by SARS-CoV-2 antigens values below 100IU/µl are considered negative, values in between are borderline results [3-4].

To have a better understanding of SARSCoV-2 immunoreactivity COVID-19 individuals with humoral immune deficiencies, such as those with solid and hematologic tumours, patients with primary and acquired immune deficiencies, and recipients of solid organ and hematopoietic stem cell transplants, may benefit more from passive immunotherapy. SARS-CoV-2 is rapidly changing from its wild-type form to a variety of variations and spreading around the world. Because so many patients have been vaccinated with diverse vaccines, developing a high-throughput technology for evaluating antibody responses and surrogate neutralising capabilities against several SARS-CoV-2 variants is critical. Clinical prognosis has been linked to the volume and quality of humoral responses to SARS-CoV-2 [5]. Although the elicitation of humoral responses against various viral proteins is quick and occurs in the majority of infected individuals, the degree of these responses varies greatly among them and is positively related to the severity of COVID-19 disease. Coronaviruses are RNA viruses that have evolved to infect a variety of avian, amphibian, and mammalian hosts. Despite their widespread distribution and potential impact, knowledge of coronavirus host immunity is limited, owing in part to the absence of overt pathogenicity of endemic human coronaviruses, which generally cause common colds. This study demonstrates that S1P inhibitors impair the cellular and humoral immune response in SARS CoV-2 vaccination, whereas patients receiving B-cell depleting therapies mount an intact cellular immune response. These data can support clinicians in counselling their PwMS and NMOSD patients during the COVID 19 pandemic.

Conflict of interest

The author declare no conflict of interest.

References

1. Wang L, Wang Li, Bai M (2014) Blastic plasmacytoid dentritic cell neoplasm:two cases report and literature review. 35: 1124-1126.
2. Thind SK, Taborda CP, Nosanchuk JD (2015) Dendritic cell interactions with Histoplasma and Paracoccidioides. Virulence 6: 424-432.
3. Han J, Li Y, Wang P J (2017) Blastic plasmacytoid dentritic cell neoplasm with myeloproliferative neoplasms:a case report . 38: 256.
4. Brok M, Nierkens S, Figdor CG (2005) Dendritic cells: tools and targets for antitumor vaccination. 4:699-710.
5. Collin M, Ginhoux F (2019) Human dendritic cells. Semin Cell Dev Biol 86: 1-2.

***Corresponding author:** George Kassiotis, Department of Infectious Disease, St Mary's Hospital, Imperial College London, London W2 1PG, UK, E- mail : gaosge.kasiotis@crick.ac.uk

Received: 10-Feb-2022, Manuscript No. jcb-22-53938; **Editor assigned:** 12- Feb -2022, PreQC No. jcb-22-53938 (PQ); **Reviewed:** 19-May-2022, QC No. jcb-22-53938; **Revised:** 24-May-2022, Manuscript No. jcb-22-53938 (R); **Published:** 30-May-2022, DOI: 10.4172/2161-0681.1000412

Citation: Kassiotis G (2022) Auto Immune Response to COVID-19. J Cytokine Biol 7: 412.