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COVID-19 as a trigger of Guillain-Barré syndrome: A review of the molecular mechanism

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Abstract

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) caused a pandemic with serious complications. After the coronavirus disease 2019 (COVID-19), several post-acute COVID-19 syndromes (PACSs) and long-COVID sequels were reported. PACSs involve many organs, including the nervous, gustatory, and immune systems. One of the PACSs after SARS-CoV-2 infection and vaccination is Guillain-Barré syndrome (GBS). There is a lower chance of getting GBS after a SARS-CoV-2 infection compared to other infections. However, the high prevalence of COVID-19 and severe complications of GBS, e.g., autonomic dysfunction and respiratory failure, highlight the importance of post-COVID-19 GBS. It is while patients with simultaneous COVID-19 and GBS seem to have higher admission rates to the intensive care unit, and demyelination is more aggressive in post-COVID-19 GBS patients. SARS-CoV-2 can trigger GBS via several pathways like direct neurotropism and neurovirulence, microvascular dysfunction and oxidative stress, immune system disruption, molecular mimicry, and autoantibody production. This review discusses the most recent evidence regarding the molecular and cellular mechanisms of GBS after SARS-CoV-2 infection and vaccination.

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