Fernando S., Mendonza A.

DELAYED ONSET GUILLAIN-BARRÉ SYNDROME FOLLOWING COVID-19: EVIDENCE OF PROLONGED IMMUNE DYSREGULATION IN A PATIENT WITH PRE-EXISTING AUTOIMMUNITY

Tutors: Senior Lecturer Zakharova A.G., Senior Lecturer Primak S.V.
Department of Propaedeutics of Internal Diseases
Belarusian State Medical University, Minsk

Relevance. Patients with pre-existing autoimmune diseases have a significantly increased risk of developing new-onset autoimmune conditions, with reported incidence rates of approximately 3.8% (38.10 per 1,000 individuals) following COVID-19 infection. While Guillain-Barré syndrome (GBS) typically manifests within 6 weeks after a COVID-19 infection, deviations from this timeline warrant further investigation.

Aim: to elucidate the pathophysiological mechanisms underlying the delayed onset of GBS two years following COVID-19 infection, with particular focus on how pre-existing autoimmunity and prolonged corticosteroid therapy may alter immune signaling. Specifically, this study explores how these factors may predispose to GBS while modulating and delaying its neuroinflammatory clinical presentation.

Materials and methods. A retrospective analysis of a single patient case from the 6th Clinical Hospital, Minsk, Belarus, was conducted, supplemented by a focused review of current literature from scientific databases.

Results and their discussion. This is a case of a 68-year-old female with a history of Weber-Christian panniculitis with secondary vasculitis (M35.6), first diagnosed during hospitalization from 25 April to 8 May 2018. She was subsequently admitted from 23 June to 30 June 2022 with severe SARS-CoV-2 infection and bilateral interstitial pneumonia. Her final hospitalization (29 February-9 March 2024) resulted in a diagnosis of Guillain-Barré syndrome (GBS) with tetraparesis, and she died on 9 March 2024.

Weber-Christian panniculitis is a rare autoimmune disorder. Emerging data indicate that patients with pre-existing autoimmune diseases are at an increased risk of developing new-onset autoimmune conditions following a COVID-19 infection. This patient was diagnosed with Guillain-Barré syndrome (GBS) two years after contracting COVID-19, whereas GBS typically occurs within six weeks of an acute COVID-19 infection. Emerging evidence suggests that persistent viral reservoirs may remain detectable for up to two years. In this context, COVID-19 may act as an immunological stressor superimposed on an already dysregulated immune system, potentially triggering a prolonged subclinical phase. This mechanism could explain the delayed manifestation of GBS, requiring a cumulative period of immune instability of approximately two years.

Further, the patient had been on long-term methylprednisolone therapy for nearly seven years prior to the diagnosis of GBS. Chronic corticosteroid use may suppress inflammation and attenuate pain, which may obscure early neuropathic symptoms such as paresthesia and mild weakness. Additionally, steroid-induced myopathy may have contributed to the misattribution of proximal muscle weakness to medication side effects rather than an evolving neuropathic process. Collectively, these factors, along with the anti-inflammatory effects of long-term steroid therapy, may have increased the threshold of inflammatory activity required to produce clinically detectable nerve damage, thereby delaying the diagnosis of GBS.

Conclusions. This case highlights the diagnosis of Guillain-Barré syndrome two years after the onset of severe COVID-19 in a patient with pre-existing autoimmune disease and prolonged corticosteroid use, suggesting that altered immune signaling may both predispose to and delay neuroinflammatory complications.