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Guillain-Barré syndrome after Covid-19 infection☆

Síndrome de Guillain-Barré tras infección por COVID-19

To the Editor

COVID-19 *is an infectious disease caused by Severe Acute Respiratory Syndrome Coronavirus* (SARS-CoV-2) which has several symptoms and whose severity lies mainly in pulmonary involvement. However, in addition to pulmonary involvement, other levels of involvement have been described, including various forms of neurological compromise.[1,2]

Next, we report a case of Guillain-Barré Syndrome (GBS) after COVID-19 infection. A 77-year-old male with a history of hypertension, dyslipidaemia, trigeminal neuralgia, and chronic obstructive pulmonary disease who was admitted during two weeks for bilateral interstitial pneumonia secondary to COVID-19 infection. During admission, he received treatment with lopinavir/ritonavir, hydroxychloroquine, and piperacillin/tazobactam, and was discharged in good general condition and without the need for supplemental oxygen therapy. After a week at home, he returned to the emergency department due to an insidious onset and progressive course condition, consisting of left facial asymmetry with difficulty in bilateral eyelid closure and dysarthria, as well as inability to open the mouth and dysphagia for both fluids and for solids. The initial neurological examination revealed severe facial diplegia, dysarthria with dysphagia, and a loss of gag reflex. Strength was preserved, except for a slight weakness in the cervical flexor muscles, without weakness in the lower limbs or sphincter abnormalities, with the rest of the examination being inane. Suddenly, the patient experienced 2 self-limiting episodes of acute respiratory failure with facial cyanosis and desaturation of up to 75% lasting 1–2 min, reason why he was admitted to the Intensive Care Unit for airway isolation.

In the initial ancillary tests, laboratory results and cranial tomography did not show relevant abnormalities; however, the lumbar puncture revealed an albumin-cytological dissociation with a spinal fluid protein concentration of 77.7 mg/dl.

Given the clinical suspicion of GBS with bulbar involvement, treatment with immunoglobulins (Ig) was started at a dose of 0.4 mg/Kg for 5 days. During this period, an MRI was performed that ruled out abnormalities compatible with Bickerstaff encephalitis. Without clear improvement after a first cycle of IGs, an electromyogram (EMG) was performed, which showed axonal and sensory-motor demyelinating polyneuropathy with significant involvement of bilateral VII CP and compatible with the clinical suspicion of GBS, deciding a 2nd° Ig cycle after which progressive clinical improvement was observed, leading to discharge.

In summary, GBS is an acute immune-mediated inflammatory polyradiculoneuropathy, the response of which causes the destruction of myelin in the peripheral nerves, it can occur at any age and in most cases is triggered by an infection. The typical clinical characteristics are progressive muscle weakness, usually symmetrical and starting in the lower limbs, with absent or diminished tendon reflexes, however in a small percentage, it can start in the upper limbs or facial muscles, with different variants of this disease having been described.[3]

The case that we have reported is a GBS with predominantly axonal bulbar involvement in a patient with previous COVID-19 infection, a complication that has been scarcely described in the literature. Specifically, our patient showed bilateral weakness in facial muscles, as well as the oropharyngeal and cervical muscles, with preservation of strength and reflexes in the lower limbs, which is compatible with a variant of GBS known as pharyngeal-cervical-brachial weakness.

In relation to COVID-19, multiple nonspecific central nervous system manifestations have been described, such as headache, dizziness/vertigo, impaired level of consciousness, ataxia, epilepsy, acute cerebrovascular accidents, and encephalopathy. On the other hand, peripheral nervous system involvement includes hyposmia, hypogeusia, neuralgia, and skeletal muscle injury with rhabdomyolysis.[4,5] Some of the neurological manifestations can appear even before pneumonia or respiratory symptoms and, therefore, it is important to take this fact into account.

In conclusion, although the physiopathological mechanism by which neurological involvement occurs in COVID-19 infection is not yet well known, it seems that, like other viruses, it could be related as an etiological agent of GBS. In our case, albuminocytologic dissociation in lumbar puncture and EMG support the diagnosis. If we add the epidemiological history to this, we can support the causality of both.

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Conflicts of interest

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