Multiple demyelinating sensory and motor mononeuropathy associated with COVID-19: a case report

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Abstract

The involvement of the nervous system may occur in 36.4% of patients with COVID-19. Cases have been described of cerebrovascular diseases, encephalitis, encephalopathies, and changes in smell and taste. Two months after being discharged from hospital with COVID-19, a 63-year-old male patient presented with a predominantly demyelinating sensory and motor mononeuropathy. A diagnostic possibility of multiple sensory and motor demyelinating mononeuropathy (Lewis-Sumner syndrome) was made. Treatment with human immunoglobulin was initiated. COVID-19 may be associated with multiple demyelinating sensory and motor mononeuropathy.

Keywords COVID-19 · SARS-CoV-2 · Neuromuscular disorders · Peripheral nervous system · Multiple sensory and motor demyelinating mononeuropathy

The involvement of the nervous system may occur in 36.4% of patients with COVID-19 (Mao et al. 2019). Cases have been described of cerebrovascular diseases, encephalitis, encephalopathies, and changes in smell and taste (Munhoz et al. 2020). With regard to the impairment of the peripheral nervous system and muscles, cases have been described of Guillain-Barré syndrome, isolated cranial neuropathies, Miller-Fischer syndrome, myositis, and rhabdomyolysis (Mao et al. 2019; Munhoz et al. 2020; Paliwal et al. 2020; Katyal et al. 2020). However, there have been no reports in the literature of multiplex mononeuritis associated with COVID-19.

Herein we describe a case of multiplex mononeuritis possibly associated with COVID-19.

Ten months ago, a 63-year-old male patient presented with fever and respiratory symptoms. He presented no neurological symptoms at the onset of the disease. The RT-PCR test confirmed SARS-CoV-2 by nasal swab. It was necessary to admit the patient to ICU due to respiratory failure, where he required orotracheal intubation with invasive mechanical ventilation for 20 days. After being discharged from ICU, the patient presented with generalized muscle weakness, and a diagnosis of critical illness myopathy was considered. Electroneuromyography revealed a myopathic pattern. The patient was discharged 2 months after onset of the condition. He continued to suffer from an overall reduction in strength and with physiotherapy he presented indications of rehabilitation. There was a gradual and total recovery of motor strength after discharge and there were no sensory complaints.

Two months after discharge, he began to complain of a loss of tactile sensitivity in the first and second fingers of his right hand and on the back of both feet, and also noticed atrophy just in his right biceps. This condition developed over approximately 30 days. The evolution of sensory and motor symptoms occurred at the same time and stability occurred during the last 6 months.

He had a personal history of systemic arterial hypertension, hypothyroidism, vitamin D deficiency, vitiligo, and one kidney.

The neurological examination demonstrated that his segmental strength was preserved, although with atrophy of the right biceps. The deep tendon reflexes were normal, except for areflexia on investigation of the right bicipital reflex. There was tactile hypoaesthesia around the area of the right ulnar nerve and both fibular nerves.
Electroneuromyography, performed 5 months after the onset of the sensory complaints, was compatible with a predominantly demyelinating multiple sensory and motor mononeuropathy. This demonstrated a lack of response in the right ulnar nerve motor conduction; increased latency, a reduction in speed and blocked left ulnar nerve motor conduction above and below the elbow; a right median nerve motor conduction block at the wrist; a lack of bilateral sensitive response in the ulnar nerve; increased latency and decreased left fibular nerve motor conduction; a lack of motor response in the right fibular nerve; increased latency and decreased motor amplitude in both tibial nerves; and a lack of sensory response in both fibular nerves. Electromyography revealed an asymmetric denervation pattern.

The study of cerebrospinal fluid (CSF), conducted 6 months after the onset of the sensory condition, revealed 2 cells/mm³ (90% lymphocytes and 10% monocytes), protein of 143 mg/dl and glucose of 74 mg/dl. The IgG index was 0.88, which demonstrated intrathecal IgG production. A PCR was not performed for SARS-CoV-2 on the CSF.

Serum tests demonstrated no significant changes, including markers for autoimmune diseases (antinuclear factor), vasculitis (neutrophil cytoplasmic antibody and cryoglobulin testing), and serology (serology for HIV, syphilis, hepatitis B, and hepatitis C).

Our conclusion rested on the diagnostic possibility of multiple sensory and motor demyelinating mononeuropathy (Lewis-Sumner syndrome) and treatment with human immunoglobulin was initiated. Although just one treatment cycle has been performed, there has been no clinical response.

As far as we are aware, this is the first reported case of a possible association between multiple demyelinating mononeuropathy and COVID-19. There was a close temporal relationship (2 months) between the end of the clinical manifestation of COVID-19 and the onset of symptoms compatible with the manifestation of multiple mononeuropathy, corroborating with a causal relationship. The association between multiple demyelinating mononeuropathy and other chronic viral infections, such as HIV and hepatitis C, has been previously described (Kaku and Simpson 2014; Mariotto et al. 2014).

Other inflammatory neuropathies, like Guillain-Barré Syndrome, may be caused by viral infections such as cytomegalovirus, herpes simplex virus, varicella-zoster virus, Epstein-Barr virus, hepatitis (A, B, C, and E), human immune deficiency virus, dengue virus, chikungunya virus, and Zika virus. The physiopathology appears to be through molecular mimicry (Rodríguez et al. 2018).

However, the possibility of a temporal coincidence between multiple demyelinating mononeuropathy and COVID-19 should not be ruled out. Nevertheless, there have been reports of post-infectious immune-mediated neurological manifestations associated with COVID-19, such as acute disseminated encephalomyelitis and Guillain-Barré syndrome (Koralnik and Tyler 2020; de Miranda Henriques-Souza et al. 2021) Acute disseminated encephalomyelitis in a COVID-19 pediatric patient. This case draws attention to the possibility of this new association.

Declarations

Conflict of interest The authors declare that there is no conflict of interest. There was no financial support. This case report was approved by the institution’s ethics committee and the patient gave his written consent.

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