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Case Report

Multifocal Neurologic Manifestation of Coronavirus Disease 2019 Infection: Report of 2 Cases

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Keywords
COVID-19; SARS-COV-2; Polyneuropathies; Neuropathy; Guillain-Barre syndrome; Myelitis

Abstract

Background: The most common neurological symptoms of coronavirus disease 2019 (COVID-19) were hypogeusia and hyposmia; neuropathy was recently reported in patients with COVID-19. It is not yet clear if the virus has the potential to affect the nervous system or it is just a co-incidental finding. In this study, we report 2 patients with a history of COVID-19 presenting neurological symptoms and weakness, who did not meet the Guillain-Barre syndrome (GBS) criteria.

Case Report: The first patient was a 56-year-old man who developed flu-like symptoms and was isolated for fourteen days after being diagnosed with COVID-19 through positive polymerase chain reaction (PCR) test and lung computed tomography (CT) scan. Three weeks later, the patient developed right-sided peripheral facial palsy, four-limb paresthesia, and progressive lower limb weakness. The second patient was a 62-year-old man with more or less the same presentation as patient number 1 except for lack of facial involvement and normal lower limbs' sensory nerve conduction study (NCS).

Conclusion: Here, we have reported 2 cases that developed neuropathic symptoms following the COVID-19 symptoms in the recovery period. The majority of the research regarding neuropathies in patients with COVID-19 studied the facial nerve involvement and GBS, and a sensory-predominant polyneuropathy is not yet reported, as far as we know. With the suspicion of an acute myelitis and sensory neuropathy, the patients were prescribed intravenous immunoglobulin (IVIg).

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Introduction

The coronavirus pandemic started in Wuhan, China, in late 2019. With the spread of the disease, the respiratory symptoms were developed in addition to those of the other systems, such as the nervous system.[1,2] Although the most common neurological symptoms were hypogeusia (5.6%) and

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hyposmia (5.1%), neuropathy has been recently reported in patients with coronavirus disease 2019 (COVID-19).[3-6] It is not yet clear if the virus has the potential to affect the nervous system or it is just a co-incidental finding.[2]

Case Report

In this study, we report two patients with a history of COVID-19 presenting neurological symptoms and weakness, who did not meet the Guillain-Barre syndrome (GBS) criteria.

The first patient was a 56-year-old man who developed flu-like symptoms on March 10, 2020 and was treated with hydroxychloroquine for a week and isolated for fourteen days after being diagnosed with COVID-19 through a positive polymerase chain reaction (PCR) test and lung computed tomography (CT) scan. Three weeks later, the patient developed right-sided peripheral facial palsy, four-limb paresthesia, numbness, impaired position, and vibration sensation in four limbs, and progressive lower limb weakness. The patient did not have diarrhea and the respiratory symptoms had considerably subsided; he was referred and admitted to a tertiary hospital four days later.

The vital signs were normal on examination. The patient was alert and afebrile; on cranial nerve examination, there was right-sided facial nerve palsy. The pupil reflex and the eye movements were normal. The gag reflex on both sides and the facial sensation were normal. The upper limb force was 4/5, and the lower limb force was 3/5 on distal and proximal muscles. The upper limb, patellar, and the Achilles tendon deep tendon reflexes (DTRs) were +2, +1, and absent, respectively. The proprioceptive and pain sensation on both sides were impaired. The patient did not have a sharp and definite sensory level and the Babinski reflex was negative on both sides; he had urinary retention and constipation.

The patients underwent electrodiagnostic (EDX) study including electromyography (EMG) and nerve conduction studies (NCS) for four limbs with the impression of GBS on the day after admission and 10 days later. The PCR was negative in the second EDX study. EDX findings revealed absent of upper limbs sensory response and decreased amplitude of lower limbs' sensory NCS with normal four limbs' motor NCS and decreased amplitude of right-side facial nerve compound muscle action potential (CMAP) in the first patient. Needle EMG showed decreased interference pattern in lower limbs with normal upper limbs study. There was no spontaneous activity in needle EMG. Late responses including F-wave and H-reflex were normal.

EDX findings were diagnosed as moderate to severe right-side peripheral facial nerve lesion with about 90% axonal loss in patient number 1 and sensory neuronopathy mainly involving upper limbs in both patients. Due to lack of criteria for GBS, findings were not conclusive for typical GBS. These findings (sensory neuronopathy, facial palsy, and paraparesis) could not be explained by known clinical syndrome but patchy and atypical presentation of infectious (viral) process such as COVID-19.

The second patient was a 62-year-old man with more or less the same presentation as patient number 1 except for lack of facial involvement and normal lower-limbs sensory NCS.

In the both patients, the magnetic resonance imaging (MRI) study of the thoracolumbar spinal cord segment showed cord edema without root involvement. The cerebrospinal fluid (CSF) parameters in lumbar puncture analysis were: white blood cell (WBC) count: 0 cells/µl, glucose: 62 mg/dl, and protein: 201 mg/dl for the first patient and similar data for the second patient.

With the suspicion of an acute myelitis and sensory neuronopathy, the patients were treated with 30 g intravenous immunoglobulin (IVIg) for five days. The lower limb force was 4/5 and the upper limb force was normal after the treatment. The right facial nerve palsy recovered in the first patient.

Discussion

Here, we have reported 2 cases who developed neuropathic symptoms following the COVID-19 symptoms in the recovery period. The majority of the research regarding neuropathies in patients with COVID-19 studied the facial nerve involvement and GBS, and a sensory-predominant polyneuropathy is not yet reported, as far as we know.[3,4,7]

In a study in China, a higher blood urea nitrogen (BUN) level and a lower lymphocyte and platelet number were reported in the central nervous system (CNS) involvement compared with the involvement of the peripheral nervous system (PNS) or absence of any involvements.[7]

The exact pathophysiology of nervous system involvement in COVID-19 is not yet clear. It seems that COVID-19 could lead to different manifestations of nervous system involvement.[8] Moreover, the patients with COVID-19 could develop symptoms of PNS involvement before or after the development of respiratory symptoms, which shows the need to follow patients with COVID-19 after the isolation period and to evaluate the patients with an acute polyneuropathy for a COVID-19 infection.[3,4] Due to the development of symptoms at different times, some theories are proposed for GBS occurrence in these patients, including the parainfectious profile (similar to some viruses such as Zika virus) or the classic postinfectious profile.[3,4]

Conclusion

The patients in our study did not have the typical EDX manifestations of GBS, and it seems that more research and evaluation of neurological sequelae is essential for a more accurate understanding of the mechanisms.

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None.

Conflict of Interest

Authors have no conflict of interest.

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