

Facial Nerve Palsy as a Neurological Manifestation of COVID-19

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Abstract

Facial nerve palsy is the most frequent acute mononeuropathy and it is often of viral etiology, although many other causes have been identified. It has recently been described as a potential manifestation of COVID-19. We report the case of a patient with recent history of diarrhea and malaise that was admitted to the hospital presenting right facial paresis with orbicular muscle involvement. Nasopharyngeal swab tested positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) on the real-time reverse transcription polymerase chain reaction and magnetic resonance imaging showed no structural changes. During the hospital stay, the patient showed clinical improvement, and no other symptoms were observed. This case presentation suggests a possible association between neuropathies and SARS-CoV-2 infection.

Keywords: Case, COVID-19, facial, mononeuropathy, report

INTRODUCTION

Facial nerve palsy (FNP) is the most frequent acute mononeuropathy and is typically characterized by a partial or complete inability to automatically move the affected side of the facial muscles. Although often assumed to be idiopathic (Bell's palsy), FNP etiology includes an extensive list of causes such as Ramsay–Hunt syndrome (varicella-zoster virus) or other viral infections, Lyme disease, aural infections and cholesteatomas, postsurgical insult, benign or malignant tumors, congenital malformations, systemic infections, autoimmune conditions, granulomatous diseases, and trauma.^[1] When a patient presents with isolated FNP, a careful neurological examination is required to rule out concomitant cranial nerve involvement. In some cases, complementary tests, such as cerebrospinal fluid (CSF) analysis or magnetic resonance imaging (MRI), may be useful to rule out associated conditions (e.g., ear disorders or neoplastic processes, central nervous system (CNS) infections, etc.).^[2] Recently, FNP has been described in case reports as a potential manifestation of COVID-19.^[3] Across the world, during the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic, there has been reported an increase in the incidence of FNP both in the adult and pediatric population.^[4,5]

CASE REPORT

In late May 2020, a 76-year-old woman with a medical history of high blood pressure, type 2 diabetes mellitus under insulin treatment, and recurrent episodes of urinary tract infection was admitted to the neurology department's stroke isolation ward with retroauricular and right frontal headache as well as right facial asymmetry for the past 24 hours. She did not mention other neurological symptoms and did not complain of aural symptoms, recent history of traumatic events, chills, cough, shortness of breath or any other symptoms on admission. She mentioned malaise and diarrhea the week before and that her husband had had fever and respiratory symptoms at the same time. She had remained asymptomatic ever since and had strictly complied with the lockdown measures implemented by the Spanish government. On admission, neurological examination revealed right facial

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paralysis with orbicular muscle involvement, but without Bell's palsy, consistent with right lower motor neuron type FNP. The corneal reflex was present. The rest of the neurological examination was unremarkable, without any focal deficits and with normal reflexes. Kernig's and Brudzinski's signs were negative. There were no external ear skin injuries or parotid swelling. The respiratory examination was normal, chest computed tomography (CT) scan showed a peripheral opacity in the left upper lobe without other relevant findings, and a nasopharyngeal swab tested positive for SARS-CoV-2 on real-time reverse transcription-polymerase chain reaction (RT-PCR). HIV and *Treponema pallidum* testing was negative. CSF was not analyzed, emergent CT scan showed no signs of intracranial bleeding, and brain MRI showed leukoencephalopathy secondary to chronic ischemic changes caused by microangiopathy without signs of acute or subacute ischemic events. The patient was started on a down-titration regimen of prednisone and was randomized to the local standard of care arm of WHO's "Solidarity" Clinical Trial. During hospital admission, she had no other symptoms and a slow clinical improvement of facial asymmetry was observed. She was discharged after 8 days of hospital stay. Ambulatory RT-PCR for SARS-CoV-2 remained positive until 4 weeks after symptoms onset.

DISCUSSION

SARS-CoV-2 infection has been associated with a wide range of symptoms, the most common being fever, cough, and fatigue according to a Chinese cohort study of 264 patients.^[6]

Nonetheless, coronaviruses are known to have neuroinvasive propensity and as with SARS and Middle East respiratory syndrome, current evidence suggests that COVID-19 pathophysiology may also involve the nervous system in up to 36.4% of cases.^[7]

Its clinical manifestations include nonspecific symptoms such as dizziness, headache, or impaired consciousness, but also acute cerebrovascular disease and skeletal muscle symptoms, suggesting the involvement of both the CNS and the peripheral nervous system.^[8] Anosmia and ageusia have also been described, although they are known to be present in other common respiratory tract infections.

In the past few months, a wide variety of neurological manifestations have been described in association with COVID-19. Among patients admitted to intensive care units, encephalopathy, corticospinal dysfunction, and delirium have been frequently reported while sporadic cases of Guillain-Barré syndrome, Miller Fisher syndrome, and even acute hemorrhagic necrotizing encephalopathy have also been described.^[9-12]

To date, the mechanism underlying the etiopathology of the neurological manifestations associated with COVID-19 remains unclear. Among the various hypotheses that have

been proposed, the most generally accepted ones are direct viral toxicity due to the neurotropism of the virus and immune-mediated injury triggered by a pro-inflammatory state characterized by a maladaptive cytokine profile, microglial activation, and adaptive autoimmunity.^[13,14] This inflammatory state, also known as cytokine release syndrome or cytokine storm, has been widely observed in severe cases and it is considered, at least partially, responsible for the high mortality associated with COVID-19.

This case report only suggests a possible association between cranial neuropathy and SARS-CoV-2 infection manifested as a parainfectious phenomenon. It should raise the awareness among physicians about atypical presentations of this infection. More cases and further research are needed to support causality.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Research quality and ethics statement

The authors followed applicable EQUATOR Network (["http://www.equator-network.org/](http://www.equator-network.org/)) guidelines, notably the CARE guideline, during the conduct of this report.

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Conflicts of interest

There are no conflicts of interest.

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