Abstract:
Coronavirus disease 2019 (COVID-19) patients have been increasingly reported to develop various neurological manifestations. We herein present a rare case of bilateral facial nerve palsy in a patient that occurred 5 weeks after the onset of COVID-19. The patient had no motor or sensory deficits in his extremities, and there were no other diseases that may have resulted in bilateral facial palsy. Based on these findings, we concluded that the facial palsy in this case may have been triggered by COVID-19.

Key words: COVID-19, bilateral facial nerve palsy

Introduction
A new coronavirus was identified as the cause of a cluster of pneumonia cases in Wuhan, a city in China, at the end of 2019. It has since spread rapidly and cases have increased worldwide (1). In February 2020, the World Health Organization (WHO) designated the viral disease as COVID-19 which stands for coronavirus disease 2019. Neurologic complications are known to occur in 36.4% of COVID-19 patients (2). Several cases of Guillain-Barré syndrome (GBS) post COVID-19 have already been reported (3-11), some of which are complicated by facial nerve palsy (3, 4, 6, 7, 9-11). However, only a few cases of bilateral facial nerve palsy without any other neurological symptoms, associated with COVID-19 have been reported in the past (9, 11-14). The objective of this clinical case report is to highlight a rare case of bilateral facial nerve palsy after severe COVID-19.

Case Report
A 43-year-old Japanese man presented to our department with bilateral facial paralysis that began on the left side of the face one week previously and thereafter spread bilaterally within a few days. The patient had been undergoing hemodialysis for end-stage renal disease since 17 years of age, and his medical history included hypertension and angina pectoris. Thirty-five days before the onset of facial paralysis, the patient developed a fever and was diagnosed with COVID-19 by a reverse transcription-polymerase chain reaction (RT-PCR) test, four days later. The patient was asymptomatic when admitted to our hospital for isolation; on the fifth day of hospitalization, his respiratory symptoms deteriorated rapidly and he required mechanical ventilation and intensive care management for 8 days. The patient did not experience any neurological complications during hospitalization. Respiratory rehabilitation resulted in a resolution of symptoms and the patient was discharged; however, he began to experience difficulty in moving the left side of his mouth, about 1 week after discharge.

On examination, the patient was afebrile, had left-dominant bilateral peripheral facial nerve palsy, and was unable to close his left eye; but, had no other neurological abnormalities such as weakness, sensory disturbance, diplopia, taste disorder, or auditory hyperesthesia. The patient’s deep tendon reflexes, ocular movements, and gait were normal. There were no remarkable laboratory findings, including hemoglobin A1c and angiotensin-converting enzyme levels. Serum IgG antibodies against Epstein-Barr virus viral capsid antigen (EBV-VCA) and cytomegalovirus were positive, but IgM was negative, suggesting a previous infection. Antiganglioside antibodies (GM1, GD1a, GD1b, GT1b, GQ1b, GA1, GM2, GM3, GD3, and GD2) were negative. Serum anti-nuclear antibody, anti-Ro/SS-A antibody, myeloperoxidase-anti-neutrophil cytoplasmic antibody, and proteinase 3-anti-neutrophil cytoplasmic antibody were also negative. Bilateral hilar lymphadenopathy was not seen in the chest CT. Nerve
conduction studies (NCS), performed 7 days after the onset of left facial paralysis, showed a decreased compound muscle action potential (CMAP) amplitude of bilateral facial nerves predominantly in the left (Figure), and no abnormality in 10 other nerves.

A clinical diagnosis of peripheral facial nerve palsy post COVID-19 was made and treatment was initiated with oral mecobalamin without immunotherapy, such as steroids, as requested. The patient’s symptoms had not worsened on a subsequent visit, 1 month later. However, NCS performed 41 days after the onset of facial nerve palsy, revealed a deterioration, the left side CMAP could not be recorded, and the amplitude on the right side decreased to 1.02 mV, thus indicating axonal damage.

A year later, the patient’s symptoms had improved, but he had not fully recovered; when he tried to close his eyes, the corners of his mouth lifted up, suggesting synkinesis.

The present report describes a patient with bilateral facial nerve palsy which developed 5 weeks after the onset of COVID-19. In a recent case series, 19 of 214 hospitalized patients (8.9%) with COVID-19 had peripheral nervous system manifestations, including taste impairment, smell impairment, vision impairment, and nerve pain (2). To the best of our knowledge, there are only five case reports on bilateral facial nerve palsy without other neurological findings: two of them are thought to be variants of GBS (9, 11), another two are thought to be bilateral Bell’s palsy after COVID-19 (12, 13), and the last one is a case of bilateral facial paralysis after a mixed infection with COVID-19 and EBV (14).

The patient had no limb weakness or sensory disorders, his deep tendon reflexes were normal, and NCS of the extremities were normal, thus we did not suspect GBS. In addition, since no other causes of bilateral facial nerve palsy were identified, we are of the opinion that the condition was idiopathic. This case meets the tentative definition of “probable association” between COVID-19 and neurological disease (15), and COVID-19 may have triggered the bilateral facial nerve palsy, but further research is needed to understand its pathogenesis.

In conclusion, clinicians should be aware that neurological complications, such as bilateral facial paralysis, may occur after COVID-19.

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

The authors state that they have no Conflict of Interest (COI).

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