Case Report

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Guillain-Barré Syndrome Presenting with Facial Diplegia Due to SARS-CoV-2 Infection: A Case Series and Current Literature Review

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Abstract

The coronavirus disease-2019 (COVID-19) infection was first detected at the end of December 2019. Encephalitis, ischemic stroke, ataxia, and peripheral nerve diseases were reported in patients after the COVID-19 infection. Guillain-Barré syndrome (GBS) and cranial nerve involvement are also frequently observed. We report five patients with GBS after the COVID-19 infection. Four of them had facial diplegia. Electromyography of the three patients mentioned having acute motor and sensory axonal neuropathy, whereas two had demyelinating and mixed types. Mixed types were more frequent in studies, and a significant relationship was found with the demyelinating type. The high rate of facial diplegia in our cases suggests that more research should be conducted on the cranial nerve involvement in GBS patients with COVID-19 infection. Our aim was to focus on cranial nerve involvement in COVID-19 patients.

Keywords: Guillain-Barré syndrome, facial diplegia, SARS-CoV-2

Introduction

The coronavirus disease-2019 (COVID-19) infection was first detected at the end of December 2019. Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) is the agent responsible for this disease. It is spreading rapidly around the world as a cause of high mortality and morbidity. Cardiopulmonary effects of the disease are predominant, but neurological involvement is also seen. Headaches, a lost sense of taste and smell, and dizziness are the most common neurological symptoms. Encephalitis, ischemic stroke, ataxia, and peripheral nerve diseases were observed in patients after the COVID-19 infection (1). Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculopathy. In particular, it appears in the 1-4 week period after upper respiratory and gastrointestinal tract infections. Guillain-Barré syndrome generally presents with ascending weakness. However, variants of disease such as Miller Fisher syndrome, the pharyngocervicobrachial variant, facial diplegia, paresthesia, and pure sensory neuropathy are seen clinically (2). During the disease, the patient may develop respiratory distress and need an intensive care unit. Even death can occur. Therefore, it is important to keep GBS in mind as a differential diagnosis in patients with COVID-19 infection. We present five patients diagnosed with GBS after the COVID-19 infection. We contribute to the literature with a case series that presents GBS with cranial nerve involvement after COVID-19 infection.

Case Report

This was a single-center case series study, and the data on the patients were obtained retrospectively from their medical records. Five GBS patients after COVID-19 infection from March 2020 to October 2021 in the neurology clinic were described. Thorax computed tomography (CT) and COVID-19 reverse transcriptase-polymerase chain reaction (PCR) were used for the diagnosis of COVID-19 infection, and every patient was consulted with an infectious diseases specialist. The muscle strength of the patients was evaluated according to the Medical Research Council (MRC) scale, and lumbar puncture (LP) and electromyography (EMG)

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were performed at the diagnosis stage. The Brighton Collaboration GBS Working Group criteria were used for the diagnosis of GBS. Disability was calculated according to the Hughes Scale as: 0, healthy; 1, minor symptoms and can run; 2, can walk 10 m or more without help but can't run; 3, can walk 10 m with help; 4, bedridden or chair bound; 5, requiring ventilation for at least part of the day; 6, dead (3). Three of the patients were male, and two were female. Their mean age was 45.6 years [minimum (min) 32, maximum (max) 69]. The oropharyngeal COVID-19 swab sample of four patients was positive, and one of them was negative. Clinical features and thoracic CT were all compatible with COVID-19. The mean time between COVID-19 symptoms and the development of GBS was 11.4 days. (min 2, max 18 days). Facial diplegia was observed in addition to weakness in the extremities on the neurological examination of our four patients. Patients were investigated for metabolic disorders and other polyneuropathy causes, but no explanation for their clinical state was found. Lumbar puncture was performed, and albuminocytological dissociation was observed in all of them. COVID PCR of cerebrospinal fluid was negative. Ganglioside antibodies could not be studied. Electromyography was performed for all five patients. Acute motor and sensory axonal neuropathy (AMSAN) was reported in three patients' EMGs. The other two cases were evaluated as acute inflammatory demyelinating polyneuropathy (AIDP). They received 0.4 g/kg/day intravenous immunoglobulin for five days. The clinical and diagnostic findings of the patients are in Table 1.

Discussion

Guillain-Barré syndrome cases with the COVID-19 pandemic were seen after the SARS-CoV-2 infection. It was also mentioned that several neurological involvements existed. Symptoms and signs occur with damage to tissues in the peripheral and central nervous systems. The direct effect on cells, hypoxia, and immune effects on systems are the main mechanisms of this damage. It spreads to the nervous system with the involvement of the olfactory nerve and other cranial nerves. Secondary damage occurs because of the immune-mediated response, which has been supported by the finding of antibodies such as antiganglioside antibodies in some patients. In addition, mechanisms related to the angiotensin-converting enzyme 2 receptor have also been mentioned (4).

We presented five cases of diagnosed GBS after COVID-19 infection. Three of the patients were male, and two were female. The gender distribution was similar to the literature (5,6). The mean time between COVID-19 symptoms and the development of GBS was 11.4 days (min 2, max 18 days). The median range varies between 11 and 13 days (2). In a multicenter study in Italy, 20% of the patients were diagnosed with GBS after the COVID-19 infection, 80% of the patients were diagnosed with GBS while the current infection symptoms continued (3). Four of our patients' COVID PCR results were positive, but the patient whose symptoms persisted had a negative result. In the United Kingdom data, there was no increase in the number of GBS cases after the pandemic, and even a decrease was observed in March-May 2020. The use of masks and isolation was assumed to be the cause of the decrease, which was due to a decline in viral infections. In contrast, Filosto et al. (3) reported an increase in patients, according to their publication. Following COVID-19, GBS patients described a more serious course, increased autonomic dysfunction, and admission to an intensive care unit, with an apparent experience of the COVID-19 symptoms (3). According to several studies, there was not a significant increase in intensive care rates compared to the pre-covid period, and patients' clinical responses are good following GBS treatment (6,7). Clinical improvement was seen in our patients, but two of them had more severe COVID symptoms, which resulted in longer hospital stays and higher disability ratings. There are many reasons for bilateral facial paralysis. Trauma, infections like syphilis, metabolic diseases, acute leukemia, and autoimmune disorders like sarcoidosis can be expressed. Brainstem lesions such as pontine gliomas or hemorrhage may also be causes. Bilateral facial weakness is frequently observed in neuromuscular junction diseases. However, in these patients, there are other symptoms, such as bulbar symptoms and ophthalmoplegia, that are helpful in the diagnosis (8). GBS is a cause of bilateral facial paralysis. The rate of facial nerve involvement in GBS is 27-50%, and half of them are bilateral. The isolated facial paralysis variants are rare. Cranial nerve involvement is more common in patients with COVID-19 infection in the literature, similar to our cases (9). Three of the cases had AMSAN, and two had AIDP found in the EMG. In research, mixed and demyelinating types were more common, and a significant relationship with the demyelinating type was discovered (3,6). However, we observed axonal-type GBS dominance. Sedaghat and Karimi (10) also reported a patient with bilateral facial paralysis with AMSAN after COVID-19 infection, which had similar EMG and clinical findings to the patients in our study. The fact that the axonal type is more common in the normal GBS population in Asian countries may have led to this result (11). The assessment is also impacted by the size of our small group.

Table 1. Clinical and diagnostic findings of GBS patients after COVID-19 infection

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age (years)	**69**	**32**	**46**	**40**	**41**
Gender	Male	Male	Female	Male	Female
Medical history	Familial Mediterranean Fever	None	None	None	None
Symptoms of admission	Cough, hypoesthesia of lower extremities.	Weakness of the lower extremities.	Hypoesthesia of the fingers and toes and weakness of lower extremities.	Malaise and muscle aches.	Numbness in the left side of the face, difficulty in closing eyes and weakness in the lower extremities.
COVID-19 RT- PCR	Positive	Negative	Positive	Positive	Positive
CT chest findings	Pneumonic infiltrations with glass densities.	Patch-style glass densities.	Patch-style glass densities.	Consolidations ground-glass opacity.	Patch-style glass densities.
Neurological examination	Upper extremities 5/5 and lower extremities proximal muscles 4/5 and distal muscles 3/5 with MRC. DTR could not be obtained bilaterally in lower extremities, hypoactive in upper extremities. Plantar reflexes were bilaterally unresponsive.	He had facial diplegia. Upper extremities 4/5 and lower extremities 2/5 with MRC. DTR was hypoactive in all extremities. Plantar reflexes were bilaterally unresponsive.	She had facial diplegia. Upper extremities 4/5, proximal lower extremities 3/5 and distal lower extremities 5/5 with MRC. DTR was normoactive in the upper extremities and absent in the lower extremities. Plantar reflexes were bilaterally unresponsive. She had bilateral facial paralysis.	He had facial diplegia. Upper extremities 4/5, proximal lower extremities 5/5 and distal lower extremities 5/5 with MRC. DTR was hypoactive in the upper extremities and absent in the lower extremities. Plantar reflexes were bilateral flexors.	She had dysphagia, severe dysarthria, and facial diplegia. Upper extremities were 4/5, proximal lower extremities 3/5 and distal lower extremities 2/5 with MRC. DTR was normoactive in the upper extremities and absent in the lower extremities. Plantar reflexes were bilaterally unresponsive.
Duration between COVID-19 and GBS symptoms (days)	2	12	10	18	15
CSF findings	No cell, prt:182 mg/dL	No cell, prt: 180 mg/dL	No cell, prt: 243 mg/dL	No cell, prt: 148.7 mg/dL	No cell, prt: 125 mg/dL
EMG findings	AIDP prolonged DL and decrease in CV of median, tibial and peroneal nerves decreased F response frequency.	AMSAN low CMAP amplitude in peroneal nerve, absent sural sensory response.	AMSAN reduced CMAP amplitude in the ulnar and tibial nerves, reduced ulnar sensory amplitude.	AIDP prolonged DL and decrease in CV of tibial and peroneal nerves decreased F response frequency.	AMSAN low CMAP amplitude in tibial and peroneal nerves, absent sural sensory response.
Disability score at first month (Hughes scale)	4	1	2	1	3

DL: Distal latency, CV: Conduction velocity, CMAP: Compound muscle action potential, AMSAN: Acute motor and sensory axonal neuropathy, CSF: Cerebrospinal fluid, RT-PCR: Reverse transcriptase-polymerase chain reaction, DTR: Deep tendon reflex, COVID-19: Coronavirus disease-2019, EMG: Electromyography, GBS: Guillain-Barré syndrome, CT: Computed tomography

Conclusion

The COVID-19 infection has been in our lives for about two years, and its short- and long-term effects are not fully known yet. The impacts on the nervous system must be considered. We presented five GBS cases presenting with facial diplegia after the COVID-19 infection. In our cases, the axonal type was seen more frequently. But demyelinating is more common in the literature. It is thought that the predominance of this type in the normal GBS population in Asia may be a factor in the results. Also, four of the five patients (80%) had bilateral facial nerve involvement. This rate is higher than the normal GBS

population, but we had a small group for evaluation. More research on the cranial nerve involvement with COVID-19 infection is needed, as evidenced by the high occurrence of facial diplegia in our cases.

Ethics

Informed Consent: Consent information was obtained from the patient's family.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: I.K.A., Concept: K.G.K., Design: A.A., I.K.A., Data Collection or Processing: K.G.K., A.A., Analysis or Interpretation: M.F.P., E.G., Literature Search: K.G.K., Writing: K.G.K.

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