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ORIGINAL ARTICLE

A rare variant of Guillain-Barré syndrome: Isolated bilateral facial paralysis

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ABSTRACT

Objectives: This study aimed to investigate the frequency and characteristics of a rare variant of Guillain-Barré syndrome (GBS), isolated bilateral facial paralysis (iBFP), and the impact of the COVID-19 (coronavirus disease 2019) pandemic on the frequency of GBS and iBFP.

Patients and methods: This retrospective study reviewed 1,986 patients who were hospitalized, followed up, and treated between March 11, 2020, and August 11, 2023, and included 32 patients (22 males, 10 females; mean age: 51.6±19.3 years; range, 18 to 87 years) diagnosed with GBS in the analyses. Additionally, information was obtained from hospital records regarding admission dates, length of stay, age, sex, and diagnoses of patients admitted between January 1, 2019, and March 11, 2020, and five patients diagnosed with GBP were identified during this period.

Results: Eleven patients presented with facial paralysis, and iBFP was observed in five (15.6%) of them (4 males, 1 female). In iBFP patients, albuminocytological dissociation was detected in three cases, and the anti-GD1b IgG antibody was detected in one case. Electromyography findings of iBFP patients showed bilateral facial nerve involvement in only two cases, bilateral facial nerve involvement along with multiple A waves in other peripheral motor nerves in two cases, and facial nerve involvement along with AIDP findings in one case. The incidence of GBS was notably higher compared to the 15-month period before the COVID-19 pandemic (0.29% *vs.* 1.6%).

Conclusion: The frequency of iBFP was found to be higher than expected in our inpatient GBS patient group. It was noted that these patients were not associated with a specific anti-ganglioside antibody and had a good prognosis.

Keywords: COVID-19, facial diplegia with paresthesia, Guillain–Barré syndrome variants, peripheral facial paralysis.

Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculoneuropathy and is the most common cause of acute-onset flaccid paralysis.[1] Its incidence varies between 0.42 and 6.58 per 100,000 individuals and is more common in males.[2,3] Although it is classically defined as a triad of acute ascending symmetrical muscle weakness, areflexia, and albuminocytological dissociation in the cerebrospinal fluid (CSF), GBS is actually an umbrella diagnosis. It has many different subtypes and variants, such as Miller Fisher syndrome.[1,4] Miller Fisher syndrome, GBS, and their subtypes form a continuum of distinct and overlapping syndromes.[4]

Electromyography (EMG) findings classify GBS into two subtypes: demyelinating and axonal. Acute inflammatory demyelinating polyneuropathy (AIDP) is the most common type of GBS in Türkiye and Europe. It is characterized by symmetric demyelinating polyneuropathy, affecting both sensory and motor fibers bilaterally, with paralysis progressing from the lower to upper extremities.[5] Acute motor axonal neuropathy (AMAN) is one of the axonal variants in which only motor nerves are affected, presenting with muscle weakness.[5,6] Acute motor sensory axonal neuropathy (AMSAN) is the axonal type in which both motor and sensory fibers are affected.[5,6] However, there is much more clinical

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diversity than these three types, including the paraparetic variant, the pharyngeal-cervical-brachial variant, acute ataxic neuropathy, and others.[5,6]

Bilateral facial paralysis (BFP) with paresthesia is a rare variant of GBS, and facial diplegia may sometimes be accompanied by distal paresthesia.[7] The core features in the diagnostic criteria of this variant include facial paralysis (FP), which may be asymmetric, and areflexia/hyporeflexia in the extremities, which may also be normal. The absence of ophthalmoplegia, limb or neck weakness with ataxia, and peak of weakness occurring within 12 h to 28 days after onset followed by plateau are other features. Previous infection, presence of distal paresthesia at or before the onset of the disease, electrophysiological evidence of neuropathy, and albuminocytological dissociation in the CSF were defined as supportive features.[4,8]

Cranial nerve involvement is common in GBS, observed in 45 to 70% of cases.[9,10] Lower cranial nerves are most frequently affected, followed by the facial nerve.[10] Facial nerve involvement typically manifests bilaterally.[10] Cranial nerve palsies associated with SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) infection have also been frequently reported. The most commonly affected cranial nerves are the seventh, sixth, and third cranial nerves, with 57% of these cases being associated with GBS.[11] In another meta-analysis, it was found that 30 (42%) out of 72 patients diagnosed with COVID-19 (coronavirus disease 2019)-related FP had GBS; those with GBS predominantly exhibited bilateral FP (74%), while unilateral FP was more common in other cases (88%).[12]

About two-thirds of the patients diagnosed with GBS have a previous history of upper respiratory or gastrointestinal tract infection.[13,14] The most frequently detected agent is *Campylobacter jejuni*, and other associated pathogens are cytomegalovirus (CMV), Epstein-Barr virus (EBV), *Mycoplasma pneumoniae*, varicella zoster virus, HIV (human immunodeficiency virus), *Haemophilus influenzae (H. influenzae)*, and influenza A virus.[13,14] Many neurological complications have emerged during the pandemic, which started with the reporting of a COVID-19 case in Wuhan, China, in December 2019. The first GBS case was reported in January 2020.[15] In the following years, many cases diagnosed with post-COVID-19 GBS were reported, and it was interesting that cranial nerve involvement was more common in these cases.[16,17] In the review of Szewczyk et al.[18] published in September 2021, the number of patients with BFP in GBS cases associated with COVID-19 was 15, and only one of them was isolated BFP (iBFP).

This study aimed to investigate the frequency of peripheral FP and the characteristics of iBFP in patients with GBS. Since the study period included the COVID-19 pandemic, we also examined the impact of the pandemic on the frequency of GBS and the iBFP variant.

PATIENTS AND METHODS

The retrospective study reviewed 1,986 patients who were hospitalized, followed up, and treated in the inpatient clinic between March 11, 2020, and August 11, 2023. Thirty-two (1.6%) patients (22 males, 10 females; mean age: 51.6±19.3 years; range, 18 to 87 years) with GBS were identified and included in the study. The demographic data of these patients, along with their clinical presentations, CSF analyses, anti-ganglioside antibody panel (IgG- and IgM-type anti-GM1, -GM2, -GD1a, -GD1b, -GD3, -GT1a, -GT1b, -GQ1b, and -GB1b antibodies), and EMG findings, were analyzed. Additionally, information was obtained from hospital records regarding admission dates, length of stay, age, sex, and diagnoses of patients admitted between January 1, 2019, and March 11, 2020. It was determined that among 1,740 patients who were admitted during this period, only five (0.29%) were diagnosed with GBS and treated. However, the data for these five patients with GBS were not analyzed in detail. Only whether the clinical findings matched the iBFP variant was evaluated. The study protocol was approved by the Bezmialem Vakıf University Clinical Research Ethics Committee (date: 06.11.2023, no: E-54022451-050.05.04-128716). Written informed consent was obtained from all participants. The study was conducted in accordance with the principles of the Declaration of Helsinki.

The relationship of the distribution of cases with the months and seasons and the effect of the COVID-19 pandemic were investigated. Regarding the association with infection and vaccination, patients were evaluated for whether they had experienced COVID-19 or non-COVID-19 infections in the four weeks prior to the onset of symptoms and whether they had received a COVID-19 vaccine in the past month. Patients were classified based on their initial clinical presentation, whether it manifested as motor, sensory, cranial nerve involvement, or other neurological symptoms. Neurological examination findings were grouped into superficial and deep

TABLE 1
The main clinical, laboratory, and EMG characteristics of patients with GBS

Parameters	n	%	Mean±SD	Range
Age (year)			51.6±19.3	18-87
Sex				
Male	22	68.75		
Female	10	31.25		
Infection history (within the last 1 month) [5 of them COVID-19 (15.6%)]	22	69		
COVID-19 vaccination				
Male	2			
Clinical features				
5 bilateral facial (3 with deep sensory)				
5 motor + superficial sensory + ataxia				
3 motor + superficial sensory				
2 motor + superficial sensory + autonomic				
2 motor + superficial sensory + ataxia + autonomic				
2 motor + superficial sensory + ataxia + ophthalmoparesis + facial				
1 motor + superficial sensory + ataxia + bulbar + autonomic				
1 motor + superficial sensory + ataxia + ophthalmoparesis + autonomic				
1 motor + deep sensory + ataxia + ophthalmoparesis + bulbar + facial				
1 motor + superficial sensory + ataxia + facial				
1 motor + superficial sensory + ataxia + facial + autonomic				
1 motor + superficial sensory + bulbar				
1 motor + deep sensory + ataxia + bulbar				
1 motor + ataxia + ophthalmoparesis + bulbar				
1 motor + deep sensory				
1 sensory				
1 sensory + ophthalmoparesis				
1 deep sensory + ataxia + facial				
1 ataxia + ophthalmoparesis				
Cranial neuropathy involvement	19	59		
Facial nerve palsy	11	34		
Bilateral facial palsy				
Male	6			
Female	3			
Isolated bilateral facial palsy				
Male	4			
Female	1			
CSF protein				
Analyzed in 29, it was high (>45 mg/dL) in 25 of them				
Presence of anti-ganglioside antibody				
Examined in 25 patients, positive in two patients (anti-GM1 IgM + and anti-GM2 IgM +++ in one, anti-GD1b IgG + in one).				
Electromyography findings*				
AIDP	11			
AIDP + bilateral facial	3			
AMSAN	7			
AMSAN + bilateral facial	2			
Bilateral facial + multiple A	2			
Bilateral facial	2			
AMAN	1			
Normal (clinically two MFS, one ataxic GBS, one sensory GBS)	4			

EMG: Electromyography; GBS: Guillain-Barré syndrome; SD: Standard deviation; COVID-19: Coronavirus disease 2019; CSF: Cerebrospinal fluid; AIDP: Acute inflammatory demyelinating polyneuropathy; AMSAN: Acute motor sensory axonal polyneuropathy; AMAN: Acute motor axonal polyneuropathy; MFS: Miller-Fisher syndrome; * The electrophysiological classification was made according to the criteria of Uncini and colleagues.[19]

sensory impairment, facial nerve involvement, other cranial nerve involvement, motor, autonomic, or bulbar symptoms, and ataxia. Cases with FP were specifically addressed. It was noted whether FP occurred at the onset of symptoms or during follow-up.

In the CSF analysis, levels of protein and glucose, presence of cells, and albuminocytological dissosiation were recorded. Anti-ganglioside panel results were marked as positive, negative, or not examined. Possible associations between iBFP and the presence of particular anti-ganglioside antibodies were examined. Electromyography findings were classified based on the presence of demyelination, axonal (motor or sensorimotor) neuropathy, and facial nerve involvement.[19] Treatments (intravenous immunoglobulin [IVIG] and plasmapheresis) and response to treatments were evaluated.

RESULTS

The distribution of patients with GBS by period was as follows: five patients between January 1, 2019, and March 19, 2020; 32 patients between March 20, 2020, and May 1, 2023; and zero patients between May 1, 2023, and August 1, 2023. No GBS cases were admitted during the first two months of the pandemic when COVID-19 precautions were strict. No patients with GBS were admitted after the end of the COVID-19 pandemic in May 2023; the last patient was admitted on March 8, 2023. The incidence of GBS was notably higher compared to the 15-month period before the COVID-19 pandemic (0.29% *vs.* 1.6%). None of the five patients who were admitted before March 20, 2020, had the iBFP variant. Among the 32 patients who were admitted on or after March 20, 2020, five had the iBFP variant.

Demographic data of the patients who were admitted after March 20, 2020, are presented in Table 1. Twenty-three (72%) of the patients were followed during the spring and winter seasons (Figure 1). All cases of iBFP were observed between October and February.

Twenty-two (69%) patients had a history of recent infection within one month before the onset of GBS symptoms, and in five (15.6%) of them, COVID-19 infection was confirmed by polymerase chain reaction positivity (Table 1). Among these patients, three had peripheral FP, and one had bulbar involvement. There were two patients (both males) with a temporal relationship with the COVID-19 vaccine; one of them had bulbar involvement. Among the seven patients with a temporal relationship to COVID-19 infection or vaccination, five were males, with a mean age of 67.6±10.7 (range, 57 to 87) years.

The clinical, laboratory, and EMG characteristics of all patients are summarized in Table 1. Data of the five patients (4 males, 1 female; mean age: 46±20.9 years; range, 18 to 70 years) with iBFP are summarized in Table 2. The initial symptoms of 10 patients were motor, 14 were sensory, and eight were cranial symptoms. Twenty-one patients had both motor and sensory symptoms. One of the patients with iBFP also had sensory symptoms. On neurological examination, 29 patients had deep sensory impairment, 23 had muscle weakness, and 21 had superficial sensory deficit. Ataxia was observed in 18 patients, autonomic symptoms in seven, ophthalmoparesis in seven, and bulbar involvement in five (Table 1). The most common observed manifestation was deep sensory involvement

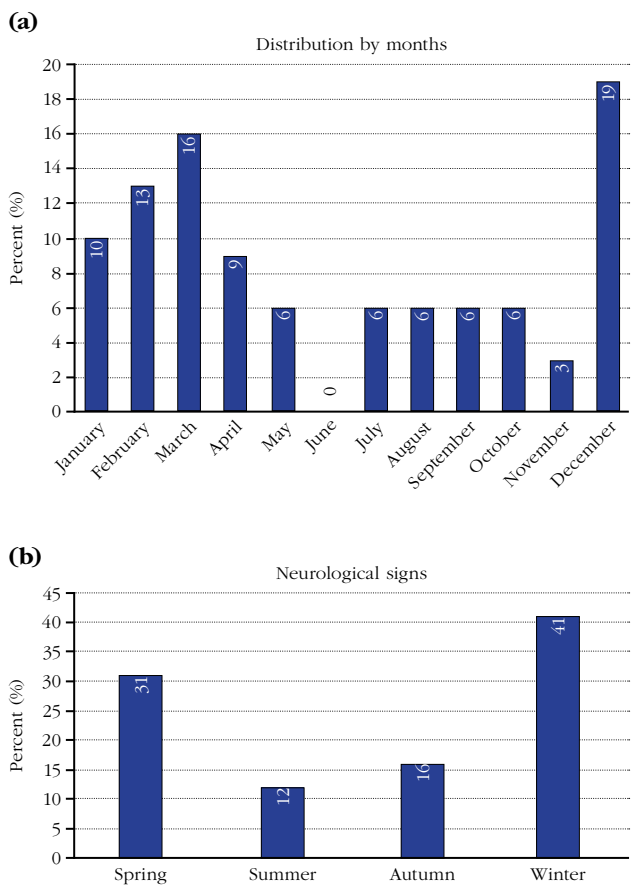

Figure 1. Distribution of Guillain-Barré syndrome cases according to **(a)** months and **(b)** seasons. It has been observed that the cases become more common in the December- March period and 72% of them were seen in the winter and spring seasons.

TABLE 2
The main clinical, laboratory, and EMG characteristics of patients with iBFP associated with GBS

Parameters	n	Mean±SD	Range
Age (year)		46±20.9	18-70
Sex			
Male	4		
Female	1		
CSF protein levels			
3 with high level (>45 mg/dL)	4		
CSF cells	4		
Anti-ganglioside antibodies			
1 with anti-GD1b IgG	5		
Electromiyography findings	5		
2 with bilateral PFP			
2 with bilateral PFP + multiple A waves			
1 with bilateral PFP + AIDP findings			
COVID-19 infection	3		
COVID-19 vaccination	0		

EMG: Electromyography; iBFP: Isolated bilateral facial paralysis; GBS: Guillain–Barré syndrome; SD: Standard deviation; CSF: Cerebrospinal fluid; PFP: Peripheral facial paralysis; AIDP: Acute inflammatory demyelinating polyneuropathy; IgG: Immunoglobulin G; COVID-19: Coronavirus disease 2019.

(24%), followed by motor and superficial sensory involvement (Figure 2). Eleven patients had FP; two cases were unilateral, and nine were bilateral. Facial paralysis initially occurred in seven patients and became bilateral in two during the course. Three of the five patients with iBFP also had deep sensory involvement revealed by neurological examination.

In the EMG examinations of the patients, findings consistent with AIDP were observed in 14 cases, with AMSAN in nine cases, and with AMAN in one case (Table 1). Among patients with iBFP, EMG findings showed bilateral facial nerve involvement only in two cases, bilateral facial nerve involvement along with multiple A waves in other peripheral motor nerves in two cases, and facial nerve involvement along with AIDP findings in one case (Table 2). Cerebrospinal fluid analysis was performed in 31 patients; increased CSF protein was observed in 25 patients (Tables 1, 2). Anti-ganglioside antibody panel was examined in 25 patients, and antibody positivity was detected in two patients (Tables 1, 2). One of these patients was an 18-year-old male who had AMSAN accompanied by BFP (anti-GM1 IgM+ and anti-GM2 IgM+++), while the other was a 38-year-old male with iBFP (anti-GD1b IgG+).

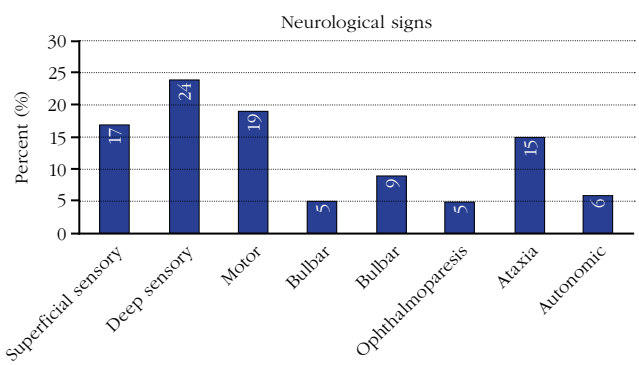

Figure 2. Distribution of neurological examination findings. 9% had facial nerve involvement.

All patients were treated with IVIG. Plasmapheresis (5 times) was administered to four patients who did not respond to IVIG. All patients with FP recovered with IVIG treatment.

DISCUSSION

In our study, it was found that the frequency of GBS was 1.6% in patients who were admitted to the inpatient clinic. The frequency of FP (34%) and involvement of other cranial nerves (59%) were similar to that reported in the literature. Interestingly, cases of iBFP (15.6%) were more common in our study group than expected (around 1%), and temporal relationship with COVID-19 was present in three of these patients.

In a multicenter study evaluating the frequency of pathogens monitored before GBS, *Campylobacter jejuni (C. jejuni)* infection was detected in 32% of patients, CMV infection in 13%, and EBV infection in 10%. Less frequently, *H. influenzae* was observed in 1%, parainfluenza in 1%, and influenza A virus in 1%.[13] Coronavirus disease 2019, which causes many autoimmune reactions, has emerged as a new triggering pathogen of GBS.[17,20] In a multicenter study by Fragiel et al.,[21] involving 61 emergency departments in Spain, a slight increase in relative GBS frequency was found between patient with COVID-19 (0.15‰) and without COVID-19 (0.02‰).

In our study, patients with GBS admitted before the COVID-19 pandemic were not thoroughly examined. However, the observation that only five patients with GBS were admitted in the 15 months prior to the COVID-19 pandemic and that no patients with GBS were admitted in the four months following the declaration of the end of the pandemic suggested a possible association with COVID-19 infection and vaccination. The increase in patients with GBS during the COVID-19 vaccination period further supported this claim. In our patient group, there were five (15.6%) patients with a clear temporal relationship to COVID-19 and two (6.3%) patients with a temporal relationship to COVID-19 vaccination. The retrospective nature and the single-center design of our study is insufficient for drawing general conclusions about the incidence of GBS and its variants. In the literature, the generally observed lack of a significant increase in GBS incidence was interpreted as potentially resulting from a reduction in the frequency of other infections that could trigger GBS during periods of strict lockdown measures.[20,22]

In a systematic review of reported cases of GBS associated with COVID-19 by Pimentel et al.[17] published in 2023, 436 post-COVID-19 patients were analyzed. The mean age of these patients was 61.38 years, with a predominance of males (67.2%). Similarly, 71.4% of the seven patients who had a temporal relationship with COVID-19 infection/vaccine in our study were males, with a mean age of 67.6±10.7 years. Three of these patients had the iBFP variant. According to Pimentel et al.,[17] the average onset of GBS symptoms occurred 19 days after infection, with hyporeflexia/areflexia, motor weakness, facial paresis/paralysis, and hypoesthesia being the main clinical features in most patients. The facial nerve was most commonly affected, followed by the lower cranial nerves and the third, fourth, and sixth cranial nerves.[17]

Traditionally, GBS is thought to be caused by anti-ganglioside antibodies produced against infectious agents, vaccines, and drugs that have molecular mimicry with gangliosides located in peripheral neurons.[23,24] However, there are also other causative mechanisms. It was reported that T cell-mediated cytokine storm may occur in AIDP, which is the most common form of GBS.[25] The presence of anti-ganglioside antibodies in our case series was 8% (2 out of 25 patients who underwent an anti-ganglioside antibody panel), which was less than generally reported. Interestingly, both of these patients had BFP, and one of them had the iBFP variant.

Acute unilateral peripheral FP is a relatively common neurological condition, with most cases being idiopathic Bell's palsy. In contrast, bilateral peripheral FP or facial diplegia is rare, and etiological investigations should be conducted carefully, considering infections affecting the peripheral facial nerve, autoimmune diseases, tumors, metabolic disorders, stroke, and toxic causes.[26] Facial nerve involvement is a common finding in GBS, observed in 24 to 60% of cases.[10] In some instances, the clinical presentation manifests as iBFP accompanied by paresthesias in the distal extremities.[7] This rare variant of GBS, which is observed in approximately 1%, was observed in 15.6% of the patients our study; three of these cases were associated with COVID-19. On the other hand, none of the five patients who were admitted before the COVID-19 pandemic had this variant.

In the cohort study conducted by Codeluppi et al.[27] in northern Italy in 2021, which included GBS patients associated with COVID-19 infection, a two-fold increase in facial nerve involvement in GBS was observed. The presence of cranial nerve damage, including oculomotor palsies and trigeminal nerve involvement, alongside disturbances in smell and taste during COVID-19 infection, suggested a higher incidence of cranial nerve involvement. In our study, FP was observed in 60% of GBS patients following COVID-19 infection.

Finsterer et al.[11] conducted a review in 2022 regarding the side effects of COVID-19 vaccines, which have become a part of our lives during the pandemic. The most commonly observed neurological side effects were headache, GBS, cerebral venous thrombosis, and myelitis. Similarly, in a review conducted by Finsterer et al.[28] in 2021, examining 19 cases of GBS following COVID-19 vaccination, the recombinant vaccine (ChAdOx1-S/nCoV-19), which combines

the COVID-19 vector with an adenovirus vector, was used in 14 patients. Cranial nerve involvement was detected in 12 patients, and all of them had BFP. In our patients, a temporal relationship with the vaccine was observed in only two patients, and neither of them had facial nerve involvement. The fact that the ChAdOx1-S/nCoV-19 vaccine was not used in Türkiye during the pandemic suggested that fewer GBS cases were observed after the vaccine.

Classically, in the CSF analysis of GBS patients, there is an increase in protein levels without a corresponding increase in cell count (albuminocytological dissociation).[4,5] Protein elevation due to disruption of the blood-brain barrier typically occurs after 48 h.[4] The cell count in the CSF generally does not exceed 10 mononuclear cells per cubic millimeter.[4,5] In our study, CSF analysis was performed in 31 patients, and albuminocytological dissociation was observed in 80% of them. No cells were observed in any of the patients.

In EMG findings of GBS, the AIDP form is most frequently encountered,[4,6] followed by the axonal forms AMAN and AMSAN.[6] In our study, AIDP was detected in 49% of the patients, while axonal involvement was observed in 30% of cases. Compared to the literature, axonal involvement was found to be higher in our study. In the EMG examination performed in GBS patients with iBFP, findings typically indicating demyelination of the facial nerves were observed.[5,29] Similarly, in our iBFP patient group, EMG examination revealed demyelination of the facial nerve as the most common finding, consistent with the literature.

In the immunological treatment of GBS, IVIG and plasmapheresis are used.[1,5] Although there are no significant differences between both treatment options, IVIG is preferred since it is not invasive and can be started quickly. In the comprehensive review of Pimentel et al.[17] during the COVID-19 period, 325 of 436 patients were treated with IVIG, and 45 were treated with plasmapheresis. The majority of the patients benefited from the treatment. In our study, all patients responded to IVIG treatment, and plasmapheresis treatment was not given in patients who had a recent COVID-19 infection.

The limitations of this study included the lack of a robust evaluation of patients with GBS before the COVID-19 pandemic. Moreover, some patients with COVID-19 may have been missed due to the lack of diagnostic studies at the beginning of the pandemic, false positivity for polymerase chain reaction, or asymptomatic presentation.

In conclusion, this study investigated iBFP, a rare variant of GBS, and its clinical course and noted that its frequency increased during the pandemic period. The clinical findings, EMG examinations, and treatments of the patients were discussed, and iBFP was found to be higher in our group of patients than expected. It was noted that these cases were not specifically associated with an anti-ganglioside antibody and had a good prognosis.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

1. Yuki N, Hartung HP. Guillain-Barré syndrome. N Engl J Med 2012;366:2294-304. doi: 10.1056/NEJMra1114525.
2. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: A systematic review and meta-analysis. Neuroepidemiology 2011;36:123-33. doi: 10.1159/000324710.
3. Wachira VK, Farinasso CM, Silva RB, Peixoto HM, de Oliveira MRF. Incidence of Guillain-Barré syndrome in the world between 1985 and 2020: A systematic review. Glob Epidemiol 2023;5:100098. doi: 10.1016/j.gloepi.2023.100098.
4. Wakerley BR, Yuki N. Mimics and chameleons in Guillain-Barré and Miller Fisher syndromes. Pract Neurol 2015;15:90-9. doi: 10.1136/practneurol-2014-000937.
5. Wakerley BR, Uncini A, Yuki N; GBS Classification Group; GBS Classification Group. Guillain-Barré and Miller Fisher syndromes--new diagnostic classification. Nat Rev Neurol 2014;10:537-44. doi: 10.1038/nrneurol.2014.138.
6. Ho TW, Mishu B, Li CY, Gao CY, Cornblath DR, Griffin JW, et al. Guillain-Barré syndrome in northern China. Relationship to Campylobacter jejuni infection and anti-glycolipid antibodies. Brain 1995;118:597-605. doi: 10.1093/brain/118.3.597.
7. Susuki K, Koga M, Hirata K, Isogai E, Yuki N. A Guillain-Barré syndrome variant with prominent facial diplegia. J Neurol 2009;256:1899-905. doi: 10.1007/s00415-009-5254-8.

8. Wakerley BR, Yuki N. Isolated facial diplegia in Guillain-Barré syndrome: Bifacial weakness with paresthesias. Muscle Nerve 2015;52:927-32. doi: 10.1002/mus.24887.
9. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 1990;27 Suppl:S21-4. doi: 10.1002/ana.410270707.
10. Bhargava A, Banakar BF, Pujar GS, Khichar S. A study of Guillain-Barré syndrome with reference to cranial neuropathy and its prognostic implication. J Neurosci Rural Pract 2014;5(Suppl 1):S43-7. doi: 10.4103/0976-3147.145200.
11. Finsterer J, Scorza FA, Scorza C, Fiorini A. COVID-19 associated cranial nerve neuropathy: A systematic review. Bosn J Basic Med Sci 2022;22:39-45. doi: 10.17305/bjbms.2021.6341.
12. Namavarian A, Eid A, Ziai H, Cheng EY, Enepekides D. Facial nerve paralysis and COVID-19: A systematic review. Laryngoscope 2023;133:1007-13. doi: 10.1002/lary.30333.
13. Jacobs BC, Rothbarth PH, van der Meché FG, Herbrink P, Schmitz PI, de Klerk MA, et al. The spectrum of antecedent infections in Guillain-Barré syndrome: A case-control study. Neurology 1998;51:1110-5. doi: 10.1212/wnl.51.4.1110.
14. Jasti AK, Selmi C, Sarmiento-Monroy JC, Vega DA, Anaya JM, Gershwin ME. Guillain-Barré syndrome: Causes, immunopathogenic mechanisms and treatment. Expert Rev Clin Immunol 2016;12:1175-89. doi: 10.1080/1744666X.2016.1193006.
15. Zhao H, Shen D, Zhou H, Liu J, Chen S. Guillain-Barré syndrome associated with SARS-CoV-2 infection: Causality or coincidence? Lancet Neurol 2020;19:383-4. doi: 10.1016/S1474-4422(20)30109-5.
16. Abu-Rumeileh S, Abdelhak A, Foschi M, Tumani H, Otto M. Guillain-Barré syndrome spectrum associated with COVID-19: An up-to-date systematic review of 73 cases. J Neurol 2021;268:1133-70. doi: 10.1007/s00415-020-10124-x.
17. Pimentel V, Luchsinger VW, Carvalho GL, Alcará AM, Esper NB, Marinowic D, et al. Guillain-Barré syndrome associated with COVID-19: A systematic review. Brain Behav Immun Health 2023;28:100578. doi: 10.1016/j.bbih.2022.100578.
18. Szewczyk AK, Skrobas U, Jamroz-Winiewska A, Mitosek-Szewczyk K, Rejdak K. Facial diplegia-complication or manifestation of SARS-CoV-2 infection? A case report and systemic literature review. Healthcare (Basel) 2021;9:1492. doi: 10.3390/healthcare9111492.
19. Uncini A, Ippoliti L, Shahrizaila N, Sekiguchi Y, Kuwabara S. Optimizing the electrodiagnostic accuracy in Guillain-Barré syndrome subtypes: Criteria sets and sparse linear discriminant analysis. Clin Neurophysiol 2017;128:1176-83. doi: 10.1016/j.clinph.2017.03.048.
20. Gomez F, Mehra A, Ensrud E, Diedrich D, Laudanski K. COVID-19: A modern trigger for Guillain-Barre syndrome, myasthenia gravis, and small fiber neuropathy. Front Neurosci 2023;17:1198327. doi: 10.3389/fnins.2023.1198327.
21. Fragiel M, Miró Ò, Llorens P, Jiménez S, Piñera P, Burillo G, et al. Incidence, clinical, risk factors and outcomes of Guillain-Barré in Covid-19. Ann Neurol 2021;89:598-603. doi: 10.1002/ana.25987.
22. Keddie S, Pakpoor J, Mousele C, Pipis M, Machado PM, Foster M, et al. Epidemiological and cohort study finds no association between COVID-19 and Guillain-Barré syndrome. Brain 2021;144:682-93. doi: 10.1093/brain/awaa433.
23. Guidon AC, Amato AA. COVID-19 and neuromuscular disorders. Neurology 2020;94:959-69. doi: 10.1212/WNL.0000000000009566.
24. Suh J, Mukerji SS, Collens SI, Padera RF Jr, Pinkus GS, Amato AA, et al. Skeletal muscle and peripheral nerve histopathology in COVID-19. Neurology 2021;97:e849-58. doi: 10.1212/WNL.0000000000012344.
25. Che Y, Qiu J, Jin T, Yin F, Li M, Jiang Y. Circulating memory T follicular helper subsets, Tfh2 and Tfh17, participate in the pathogenesis of Guillain-Barré syndrome. Sci Rep 2016;6:20963. doi: 10.1038/srep20963.
26. Keane JR. Bilateral seventh nerve palsy: Analysis of 43 cases and review of the literature. Neurology 1994;44:1198-202. doi: 10.1212/wnl.44.7.1198.
27. Codeluppi L, Venturelli F, Rossi J, Fasano A, Toschi G, Pacillo F, et al. Facial palsy during the COVID-19 pandemic. Brain Behav 2021;11:e01939. doi: 10.1002/brb3.1939.
28. Finsterer J, Scorza FA, Scorza CA. Post SARS-CoV-2 vaccination Guillain-Barre syndrome in 19 patients. Clinics (Sao Paulo) 2021;76:e3286. doi: 10.6061/clinics/2021/e3286.
29. Sardar S, Sasi S, Menik Arachchige S, Zahid M, Melikyan G. Isolated facial diplegia: A rare presentation of Guillain-Barre syndrome. Clin Case Rep 2021;9:e04473. doi: 10.1002/ccr3.4473.