NÖRO PSİKİYATRİ ARŞİVİ
ARCHIVES OF NEUROPSYCHIATRY

RESEARCH ARTICLE

Arch Neuropsychiatry 2022;59:255–259
https://doi.org/10.29399/npa.27855

Association Between Guillain-Barré Syndrome and COVID-19 Infection: Experience of a Turkish Neurophysiology Laboratory

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ABSTRACT

Introduction: Coronavirus disease 2019 (COVID-19) has affected the practice of neurology and other medical fields. The neurological complications associated with SARS-CoV-2 infection are remarkable. In this study, we investigated the clinical and electrophysiological characteristics of patients with Guillain-Barré syndrome (GBS) caused by COVID-19 diagnosed between 1 September 2020 and 30 November 2020.

Methods: This study included patients diagnosed with GBS clinically and electrophysiologically between September-November 2020 (pandemic period) and September-November 2019 (prepandemic period). Patients with GBS during the pandemic period, who were diagnosed with COVID-19 within 6 weeks before neuropathic symptoms developed, were included in the study. Pandemic period GBS patients were grouped as GBS associated with COVID-19 (n=13), and prepandemic period patients were grouped as GBS non-associated with COVID-19 (n=7). Demographic, clinical, electrophysiological and laboratory data of these two patient groups were compared.

Results: The most common symptoms were fever and cough (46.2%) in GBS associated with COVID-19 group and diarrhoea (71.4%) in GBS non-associated with COVID-19 group during active infection period. In the GBS associated with COVID-19 patients, lung involvement was apparent in 12 (92.3%) during active viral infection. A positive and significant correlation was observed in GBS associated with COVID-19 patients between comorbid factors and a need for ventilation support.

Conclusion: GBS cases associated with COVID-19 may have a more severe course, especially if they have comorbidities. It is important to define the unique clinical, electrophysiological, and laboratory findings of such patients to optimise follow-up, treatment and management.

Keywords: Coronavirus disease 2019 (COVID-19), Guillain-Barré syndrome, SARS-CoV-2

Cite this article as: Eroğlu Durmaz Ş, Uluca Kaya B, Gümüşyayla Ş. Association Between Guillain-Barré Syndrome and COVID-19 Infection: Experience of a Turkish Neurophysiology Laboratory. Arch Neuropsychiatry 2022;59:255–259.

INTRODUCTION

The world is experiencing an unprecedented pandemic of coronavirus disease 2019 (COVID-19), affecting millions. Compared to seasonal influenza, COVID-19 is more contagious, has a longer incubation period, and is associated with longer hospital stays and more deaths (1–5). In common with most medical disciplines, the practice of neurology has been affected by the pandemic. COVID-19 symptoms range from those of a simple cold to severe acute respiratory syndrome with fever, cough, and shortness of breath. In terms of neurological symptoms, anosmia is the most common. However, cerebrovascular disease, headache, encephalopathy, Guillain-Barré syndrome (GBS), and myalgia have also been reported; our experience with treatment has increased on a case-by-case basis during the pandemic (3,5–10).

GBS is a rare immune system-mediated inflammatory polyradiculoneuropathy that is associated with a precursor infection. Examples include *Campylobacter jejuni*, influenza viruses, Epstein-Barr Virus (EBV), cytomegalovirus (CMV), and more recently Zika virus (11–15). Its incidence increases worldwide after epidemics (15,16). Although the underlying mechanism of post-infection GBS remains unknown, it is possible that humoral molecular mimicry is in play; 40–70% of GBS cases develop after infections and autoantibodies against glycolipids are detected in more than 50% of patients (17).

Highlights

- **Neurological complications of COVID-19 are high among those with severe and critical illnesses.**
- **We found that most of the cases with GBS due to COVID-19 had lung involvement.**
- **Comorbid factors and ventilation need in pandemic period GBS patients significantly correlate.**

We explored whether COVID-19 causes GBS by examining patients diagnosed during the COVID-19 pandemic, and we compared their clinical characteristics to those of pre-pandemic GBS patients.

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Received: 20.04.2021, **Accepted:** 05.12.2021, **Available Online Date:** 07.11.2022

METHODS

The medical records of patients who visited the Ankara City Hospital Clinical Neurophysiology Laboratory between 1 September and 30 November 2020 were retrospectively reviewed. We have included patients diagnosed with GBS clinically and electrophysiologically between September-November 2020 (pandemic period) and September-November 2019 (prepandemic period) to this study. This study was approved by the Ministry of Health and the ethics committee of Ankara City Hospital. GBS was diagnosed according to Ausbury and Ornblath criteria (18). Demographics and clinical histories (age, sex, neuropathic complaints and the times of onset, any antecedent infection and initial symptoms, the time between infection onset and that of the neuropathic complaint, and any comorbidity) were retrieved from patient files. We recorded muscle strength, any sensory impairment, status of reflexes, any autonomic dysfunction, any cranial nerve disorder, and any need for mechanical ventilation. Hughes scoring and Medical Research Council (MRC) scoring of muscle strength were performed at admission, discharge, and 3 months after admission (19, 20). Treatments (intravenous immunoglobulin [IVIG], plasmapheresis, or both) were noted.

Electromyography (EMG) records were obtained from the Clinical Neurophysiology Laboratory. The Hadden criteria were used to distinguish acute inflammatory demyelinating polyneuropathy (AIDP) from acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN). The serum-specific SARS-CoV-2 IgG/IgM antibody test results, and the results of the SARS-CoV-2 reverse transcriptase polymerase chain reaction (RT-PCR) tests on nasopharyngeal and oropharyngeal swabs were retrieved from the patient files.

The GBS patients were divided into two groups: those with GBS associated with COVID-19 and those with GBS non-associated with COVID-19. GBS associated with COVID-19 group included the patients who were diagnosed between 1 September and 30 November 2020 whose neuropathic symptoms commenced within 6 weeks from the onset of acute COVID-19 infection and whose nasopharyngeal and/or oropharyngeal swabs were positive for SARS-CoV-2 in RT-PCR, or whose sera were positive for SARS-CoV-2 IgG antibody. GBS non-associated with COVID-19 group was the patients with GBS who were diagnosed between September 1 and November 30, 2019. We compared the data of these two groups with each other.

Statistical Analyses

Patients' sex, initial symptoms of COVID-19 infection and GBS, lung involvement status, treatment type and response, any need for ventilation support, and the numbers and proportions of comorbid diseases were recorded. The Kruskal-Wallis non-parametric test was used to compare the ages of the two groups. Sex was compared using the chi-square and Pearson chi-square tests. The non-parametric Mann-Whitney U-test was employed to compare continuous variables. Spearman correlations were calculated to determine the directions and extents of relationships among variables. IBM SPSS Statistics for Windows ver. 21.0 (released 2012; IBM Corp., Armonk, NY, USA) was used for statistical analyses and some calculations; MS Excel 2007 was employed for other calculations. Statistical significance was accepted at a $p<0.05$.

Table 1. Demographic, clinical, and laboratory characteristics of patients with GBS associated with COVID-19 and GBS non-associated with COVID-19

	GBS associated with COVID-19 group (n=13)	**GBS non-associated with COVID-19 group (n=7)**	
	% (n)	**% (n)**	**p-value**
COVID-19 initial symptoms	Flu-like 30.7 (4)	-	
	Cough 46.2 (6)	-	
	Dyspnoea 7.7 (1)	Dyspnoea 14.3 (1)	
	Diarrhoea 7.7 (1)	Diarrhoea 71.4 (5)	
	URTI 7.7 (1)	URTI 14.3 (1)	
GBS initial symptoms	Assendan Weakness 61.5 (8)	Assendan Weakness 100 (7)	0.067
	Facial diplegia and Assendan Weakness 30.8 (4)		
	Facial Diplegia 7.7 (1)		
SARS-CoV-2 RT-PCR test results	Positive 69.2 (9)		
	Negative 30.8 (4)		
Pulmonary involvement	Present 92.3 (12)		
	Absent 7.7 (1)		
GBS subtype	AIDP 61.5 (8)	AIDP 71.4 (5)	0.602
	AMSAN 30.8 (4)	AMSAN 28.6 (2)	
	AMAN 7.7 (1)	-	
Treatment	IVIG 69.2 (9)	IVIG 85.7 (6)	0.347
	PE+IVIG 7.7 (1)	PE+IVIG 14.3 (1)	
	PE 23.1 (3)		
Response to treatment	None 23.1 (3)	_	0.094
	Partial 61.5 (8)	Partial 57.1 (4)	
	Complete 15.4 (2)	Complete 42.9 (3)	
A need for mechanical ventilation	Absent 76.9 (10)	Absent 85.7 (6)	0.648
	Present 23.1 (3)	Present 14.3 (1)	
Comorbidities	No 38.5 (5)	No 71.4 (5)	0.158
	DM 30.8 (4)	DM 14.3 (1)	
	DM+HT 23.1 (3)	_	
	HT 7.7 (1)	HT 14.3 (1)	

AIDP: Acute Inflammatory Demyelinating Polyneuropathy; AMAN: Acute Motor Axonal Neuropathy; AMSAN: Acute Motor Sensory Axonal Neuropathy; COVID-19: Coronavirus Disease-19; DM: Diabetes Mellitus; GBS: Guillain-Barré Syndrome; HT: Hypertension; IVIG: Intravenous Immunglobulin; PE: Plasma Exchange; SARS-CoV-2 RT-PCR: Severe Acute Respiratory Syndrome Coronavirus-2 Reverse Transcriptase Polymerase Chain Reaction; URTI: Upper Respiratory Tract Infection.

Table 2. The admission, discharge, and 3-month Hughes and MRC scores of patients with GBS associated with COVID-19 group and GBS non-associated with COVID-19 group

	GBS associated with COVID-19 group (13)	GBS non-associated with COVID-19 group (7)	
	Median (IQR)	Median (IQR)	p-value
Admission Hughes score	4 (3–4)	3 (2–4)	0.285
Discharge Hughes score	4 (2–4.5)	2 (1–4)	0.182
Three-month Hughes score	3 (1.5–4)	1 (0–1)	0.07
Admission MRC score	26 (19–34)	32 (24–38)	0.403
Discharge MRC score	32 (16–36)	36 (28–40)	0.186
Three-month MRC score	36 (22–39)	40 (38–40)	0.032

COVID-19: Coronavirus Disease-19; GBS: Guillain-Barré Syndrome; MRC: Medical Research Council.

RESULTS

Thirteen patients who visited our EMG laboratory between 1 September and 30 November 2020 were clinically and electrophysiologically diagnosed with GBS. All had been infected with SARS-CoV-2 within the prior 6 weeks, and all were seropositive for SARS-CoV-2 IgM/IgG antibody. There were 13 patients in the GBS associated with COVID-19 group and 7 in GBS non-associated with COVID-19. The median age was 59 years (interquartile range [IQR] 51.5–61.3 years) in the GBS associated with COVID-19 group and 57 years (IQR 43.5–73.5 years) in the GBS non-associated with COVID-19 group. There were seven (53.8%) males in the GBS associated with COVID-19 group and four (57.1%) in GBS non-associated with COVID-19 group. The clinical characteristics of all patients are shown in Table 1. The initial symptoms of the antecedent infections in GBS associated with COVID-19 patients were fever and cough in six (46.2%), flu-like symptoms in four (30.8%), dyspnoea in one (7.7%), and diarrhoea in one (7.7%). Those in GBS non-associated with COVID-19 patients were throatache in one (14.3%), diarrhoea in five (71.4%), and dyspnoea in one (14.3%) (Table 1). The initial GBS symptom in GBS associated with COVID-19 patients was ascending weakness in 12 (92.3%), Facial diplegia and Ascending Weakness 30.8 (4), Facial Diplegia 7.7 (1) (Table 1). All patients with GBS non-associated with COVID-19 presented with ascending weakness; none had facial diplegia (Table 1). The median time between infective and neurological symptoms was 25 days (IQR 7–37 days) in GBS associated with COVID-19 group and 21 days (IQR 14–27 days) in GBS non-associated with COVID-19 group. The median hospitalisation times was 8 days (IQR 7–25.5 days) and 7 days (IQR 6–22 days) in the former and latter groups, respectively.

The nasopharyngeal and oropharyngeal swab RT-PCR tests in GBS associated with COVID-19 patients were positive in nine (69.2%) and negative in four (30.8%) (Table 1). The COVID-19 antibody seropositivity rate was 100%. Pulmonary involvement was evident in 12/13 patients (92.3%) during active infection (Table 1). The groups did not differ in terms of comorbid diseases (Table 1). Using the Hadden classification, eight (61.5%) GBS associated with COVID-19 patients were considered to exhibit AIDP, four (30.8%) had AMSAN, and one (7.7%) had AMAN. Of the seven patients with GBS non-associated with COVID-19 (71.4%) five had AIDP and two were diagnosed with AMSAN, respectively; the GBS type did not differ between the two groups (Table 1). Nine (69.2%) GBS associated with COVID-19 patients were given IVIG, three (23.1%) underwent plasmapheresis, and one (7.7%) received both treatments. Six (85.7%) GBS non-associated with COVID-19 patients received IVIG and one (14.3%) received both IVIG and plasmapheresis (Table 1). Two GBS associated with COVID-19 patients showed complete responses, eight had partial responses, and three had no response (Table 1). Among GBS non-associated with COVID-19 patients, three exhibited complete responses and four had partial responses; no patient was unresponsive (Table 1). During hospital follow-up, ventilation support was required by three patients with GBS associated with COVID-19 and one with GBS non-associated with COVID-19; the two groups did not differ in this respect (Table 1).

The median Hughes and MRC scores are given in Table 2. The two groups did not differ significantly. We found a significant positive correlation between comorbid factors and a need for ventilation support in only the GBS associated with COVID-19 group (p=0.03).

DISCUSSION

Throughout the pandemic, the neurological complications associated with SARS-CoV-2 infection have been remarkable. The potential involvement of the peripheral nervous system during COVID-19 infection is of great interest (21). As para– and post-infectious GBS cases associated with COVID-19 are increasingly being reported, it is important to investigate the link between SARS-CoV-2 infection and GBS (21–32). Given the similarity between recent GBS cases triggered by SARS-CoV-2 infection and those caused by MERS-CoV infection, we speculated that SARS-CoV-2 infection might also precede GBS (21). Thus, we explored the relationship between SARS-CoV-2 infection and GBS by comparing the characteristics of GBS cases encountered during certain months of the pandemic and in the same months of the year prior to the pandemic. Our first finding was the difference in the initial symptoms of the antecedent infections, namely, diarrhoea in 71.4% of cases with GBS non-associated with COVID-19 but only 7.7% of cases with GBS associated with COVID-19; the initial symptoms in the remaining COVID-19-related cases were respiratory tract infections. In many cases of GBS associated with COVID-19 reported in the literature, GBS developed after respiratory symptoms (21–33).

Another interesting finding was that 5 of 13 patients with GBS associated with COVID-19 had facial diplegia, while no cases with GBS non-associated with COVID-19 had facial diplegia. Facial diplegia is common in GBS associated with COVID-19 reported to date (21,34–37). Thus, cranial neuropathies are common in GBS associated with COVID-19 cases.

The median time between the initial symptoms of active COVID-19 infection and the neuropathic symptoms was 25 days in cases with GBS associated with COVID-19, and 21 days (from non-COVID-19 infection) in cases with GBS non-associated with COVID-19. Although there are some studies to date suggesting that GBS due to COVID-19 may be in parainfectious form, based on the data dealing with chronological evolution, the response to IVIG, and the absence of SARS-CoV-2 RNA in CSF may suggest a prominent post-infectious immune mediated mechanism rather than a para-infectious one (22,23,28,32,34,38–40). We have found that the median time between the initial symptoms of active COVID-19 infection and the neuropathic symptoms of GBS associated with COVID-19 cases was 25 days. Our finding has also supported the post-infectious mechanism.

In a meta-analysis of patients with GBS associated with COVID-19 treated between January and August 2020, most evidenced the classic sensorimotor presentation of the demyelinating GBS subtype. Most were treated with IVIG or plasmapheresis, and two-thirds responded (16).

We found that most patients with GBS associated with COVID-19 had the demyelinating type of GBS and 76.9% exhibited complete or partial responses to treatment.

Although pulmonary involvement was apparent in 12 (92.3%) of 13 GBS associated with COVID-19 cases, 1 patient lacked such involvement (7.7%). Pulmonary involvement refers to changes in lung parenchyma consistent with COVID-19 which is shown in thorax computed tomography in the COVID-19 associated with GBS group. Toscano *et al.* found that three of five GBS associated with COVID-19 cases reported pulmonary involvement (23). However, SARS-CoV-2 causes acute/lethal pneumonia in 15% of cases (41–44). We found that the vast majority of GBS cases had histories of pneumonia. In one study, patients with severe COVID-19 symptoms exhibiting rapid clinical deterioration were at greater risks of serious neurological complications (45).

We found a significant, positive correlation between comorbid factors and a need for ventilation support in patients with GBS associated with COVID-19 but not in the other group. Thus, comorbid disease worsens both COVID-19 infection and GBS. It is likely that comorbid factors may contribute not only to the infective phase of the COVID-19 disease, but also to the severity of the whole clinical picture. However, large patient numbers are needed to take into account the comorbid factors related to this issue.

Our two groups did not differ in terms of hospitalisation duration, ventilation requirements, or the admission, discharge, or 3-month Hughes and MRC scores. However, our patient numbers were small. In Filosto et al.'s study, in which pandemic and pre-pandemic period GBS patients were compared, in GBS group related to COVID-19it differs from that in our study because the demyelinating subtype was more common and the MRC score was lower than non-COVID-19 GBS (11). However, showing that GBS related to COVID-19 progresses more severely, and as we have shown in our study that GBS may progress worse in patients with co-morbidity in the group of COVID-19 associated with GBS; it is our consensus that it is probable that the systemic disorder due to COVID-19 may have contributed to the severity of the overall clinical picture.

As is true of other medical disciplines, neurology has changed during the pandemic. The virus causes neurological diseases; neurologists have thus focussed on patient care. The neurological complications of COVID-19 remain mostly unknown. More experience is required. It appears that COVID-19 can trigger GBS. Viral mimicry (particularly during postinfectious COVID-19-associated GBS) should be investigated. It should not be forgotten that the SARS-CoV-2 virus is neurotropic. Encounters with both parainfectious and postinfectious GBS will continue. A limitation of our study is that our case numbers were low (compromised by the pandemic). Another limitation was the retrospective design of the study. Prospective studies are needed on this subject. In addition, due to the pandemic situation, the cerebrospinal fluid examination of the patients could not be performed completely and information on the pathogenesis could not be obtained. Besides, since EMG was performed very early in some of the patients, the distinction between axonal and demyelinating may not have been made correctly in some patients. Prompt reporting of disease symptoms and potential associations with COVID-19 are important in terms of public health. Careful consideration of causation and associations is required to advance our understanding of this new viral pathogen and the sequelae of infection.

Ethics Committee Approval: This study was approved by the Ministry of Health and the ethics committee of Ankara City Hospital. (Date: 03.02.2021, Number:E1-21-1481).

Informed Consent: Informed consent was obtained from the patients.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept- ŞED, BUK, ŞG; Design- ŞED, BUK, ŞG; Supervision- ŞED, BUK, ŞG; Resource- ŞED, BUK, ŞG; Materials- ŞED, BUK, ŞG; Data Collection and/or Processing- ŞED, BUK, ŞG; Analysis and/or Interpretation- ŞED, BUK, ŞG; Literature Search- ŞED, BUK, ŞG; Writing- ŞED, BUK, ŞG; Critical Reviews- ŞED, BUK, ŞG.

Conflict of Interest: The authors declared that there is no conflict of interest.

Financial Disclosure: None.

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