Original Article

Guillain-Barré Syndrome (GBS) as a COVID-19 Complication: A Multicenter Study Conducted Throughout the 2-Year Pandemic

Ansari Behnaz[1,2], Kheradmand Mohsen[1,3], Mohammad Amin Najafi[1,3], Basiri Keivan[1,2], Khorvash Fariborz[1,3], Forouzandeh Negin[1], Ghasemi Majid[1,2]

[1]Isfahan Neurosciences Research Center, Alzahra Hospital, Isfahan University of Medical Sciences, Isfahan, Iran, [2]Department of Neuromuscular Disorders, Isfahan University of Medical Sciences, Isfahan, Iran, [3]Department of Neurology, Isfahan University of Medical Sciences, Isfahan, Iran

Abstract

Background: Several studies have been published on the coexistence of Guillain-Barré syndrome (GBS) and COVID-19, raising the question of whether SARS-CoV-2 plays a role in GBS pathogenesis or is just a coincidence. In this research, we discuss GBS characteristics in COVID-19 patients during the pandemic.

Material and Methods: This cross-sectional, multi-center study was performed on patients who were hospitalized in one of two GBS referral hospitals in Isfahan Providence between March 2020 to March 2022. All confirmed GBS and COVID-19 patients with no other known risk factor for GBS and no more than 28 days of COVID-19 treatment were entered into the trial.

Results: Totally, 45 patients were collected. The most prevalent EDX pattern was AIDP (57.8%). As compared to demyelinating injuries, axonal injuries were significantly more associated with lung involvement (*P* value: 0.022), higher GBS disability score (*P* value: 0.034), poorer response to treatment (*P* value: 0.021), higher serum inflammatory level, and older onset (*P* value: 0.013). The average period between the onset of COVID-19 and the onset of GBS symptoms was 18.07 ± 9.88 days. 46.3% of patients developed GBS within the first two weeks of COVID-19, a condition known as a para-infection. The axonal injury was significantly more prevalent in these individuals (*P* value: 0.045).

Conclusion: An earlier onset of neurological symptoms and para-infection conditions is associated with an increase in the frequency of axonal-type variations and, as a result, a poor prognosis. The majority of evidence in COVID-19-related GBS patients points to an autoimmune process.

Keywords: COVID-19, Guillain-Barré syndrome, pandemics, SARS-CoV-2

Address for correspondence: Dr. Kheradmand Mohsen, Department of Neurology, Alzahra Hospital, Isfahan University of Medical Sciences, Isfahan, Postal Code - 8174675731, Iran.
E-mail: kheradmand.moh3en@gmail.com
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Introduction

SARS-CoV-2 has some similarities with the SARS-CoV virus, including a strong proclivity to connect to the Angiotensin Converting Enzyme 2 (ACE2) receptor located on the surface of glial cells and neurons.[1,2] Guillain-Barré syndrome (GBS), along with ischemic stroke and encephalopathy, is one of the most common neurologic diseases among COVID-19 patients.[3,4] GBS is an acute polyradiculoneuropathy disease that, following the eradication of poliomyelitis, is considered one of the most common causes of flaccid paralysis in developed countries. The most serious consequence of GBS is its potential to lead to life-threatening Acute Respiratory Distress Syndrome (ADRS) if not treated

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right away.[5] This consequence emphasizes the need for early diagnosis and treatment of GBS throughout the COVID-19 course because early diagnosis and treatment of GBS can speed up the healing process and reduce morbidity.[6,7] Many infections (including HIV, Zika, SARS, MERS, EBV, CMV virus, and C. jejuni) have been identified in studies as potential GBS triggers.[8-15]

Several researches on the coexistence of these two diseases have been published, raising the issue of whether SARS-CoV-2 has a role in GBS pathogenesis or just occurred coincidentally. This research attempts to answer this issue by analyzing various factors.

Materials and Methods

Study design

This cross-sectional, multi-center study was conducted from March 2020 to March 2022. According to "Public Relations Of Isfahan University Of Medical Sciences," a total of 468,780 definite COVID-19 cases were identified in the Isfahan providence (the third most populated providence in Iran). Regardless of the patient's symptoms, all COVID-19 patients who required hospitalization were referred to the COVID-19 referral hospital (Al Zahra Hospital). Patients with a history of COVID-19 for more than two weeks were also referred to Kashani Hospital.

The criteria for inclusion of patients in this study were the presence of typical clinical symptoms and clinical course of GBS.[16] All patients underwent electrodiagnostic examination, and based on Rajabally's electrophysiological criteria,[17] they were confirmed and assigned to one of the subtypes. In patients where diagnostic doubts remained, Lumbar puncture (LP) was also performed to rule out GBS mimics. According to the GBS disability score,[18] patients' levels of disability at the time of hospitalization, after treatment, and their responses to treatment were assessed. Treatment responses were determined by comparing the GBS disability scores at the beginning and end of treatment.

Inclusion criteria

1) Definite COVID-19 diagnosis confirmed by positive SARS-CoV-2 virus reverse transcription polymerase chain reaction (RT-PCR) of nasopharyngeal samples or typical pulmonary involvement in high-resolution computed tomography (HRCT) for COVID-19; 2) a definite diagnosis of GBS, confirmed by clinical presentation and paraclinical evaluation (EDX and, if necessary, cerebrospinal fluid (CSF)).

Exclusion criteria

1) Existence of another possible trigger factor for GBS: All patients who had symptoms of other infections that were previously a known cause of GBS were excluded from the study. Patients who had gastrointestinal symptoms were screened for Cambylobacter infection. In addition, patients whose pulmonary imaging findings showed evidence of other pulmonary diseases, including mycoplasma infection, were evaluated to exclude this infection; *2)* a latency of more than 28 days (>4 weeks) between COVID-19 symptoms and GBS symptoms. 3) We excluded patients with critical illness neuropathy (CIN) from the study based on electrodiagnostic findings and performed LP and follow-up of the patients to see immunotherapy responses.

We defined two criteria for categorizing patients based on the coexistence of diseases: 1) para-infection: the onset of the diseases was less than 2 weeks apart, with objective active inflammation parameters in paraclinical evaluation (uptrend values of inflammatory factors including D-Dimer, WBC count ($\times 10^9$/L), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) on the first day of neurologic symptoms); 2) post-infection: has only one of the aforementioned items.

Potential biases

This includes the undervaluation of GBS patients because outpatient cases were not accessible.

Data analysis

Data analysis pooled descriptive analyses were conducted to assess differences among two main types of GBS variants for all patients. We assessed the differences in two groups for the above-mentioned variables using Chi-square test for categorical covariates and *t*-test for continuous covariates. Further, sub-analysis of the differences in frequencies and percentages was performed among three groups consisting of AIDP vs. AMSAN/AMAN vs. others, using Chi-square test. SPSS v16 was used to conduct the analysis.

Results

Totally, 45 patients (27 men and 18 women) were included. The average age was 54.66 ± 10.02 years (max: 84, min: 14, range: 70 years). The most common phenotype was the classic pattern (n = 41, 91%), which is defined as progressive sub-acute ascending paralysis from the lower limb, whereas the most common EDX pattern was AIDP (n = 26, 57.8%) [Table 1]. Fever was the most prevalent symptom of COVID-19 (89.7%), whereas anosmia was the least prevalent (12.8%). Another

Table 1: Frequency distribution of electrophysiological and phenotypes

	Subtype\Pattern	Frequency(number)
EDX Pattern	AIDP	57.8% (*n*=26)
	AMAN	17.8% (*n*=8)
	AMSAN	24.4% (*n*=11)
	Total axonal	42.2% (*n*=19)
GBS phenotypes	Classic pattern	91.1% (*n*=41)
	Pure Paraparesis	4.4% (*n*=2)
	Miller Fisher	4.4% (*n*=2)

EDX: Electrophysiological study; AIDP: *Acute Inflammatory Demyelinating Polyradiculopathy; AMAN:* Acute Motor Axonal Neuropathy; AMSAN: Acute Motor-Sensory Axonal Neuropathy; GBS: Guillain-Barré syndrome

noteworthy point is the existence of six individuals without any COVID-19 symptoms at the onset of neurological symptoms who developed COVID-19 symptoms later, either after the neurological symptoms; or COVID-19 was accidentally discovered in subsequent examinations.

The mean duration between the initiation of COVID-19 and the onset of neurological symptoms was lower in patients with axonal pattern. (12.00 ± 8.00 days; *P* value: 0.013) [Figure 1 and Table 2]. Positive RT-PCR nasopharyngeal samples were obtained in 42 of the patients (95.5%), and the rest were diagnosed with confirmatory highly suggestive HRCT features for COVID-19. According to HRCT, 13.5% of patients did not have any lung involvement at the time of neurologic symptoms. However, 100% of axonal cases had pulmonary involvement at the onset, which was significantly more than in AIDP types (*P* value: 0.022). Albuminocytological dissociation was found in three patients (2 AIDP, 1 AMAN). Among the patients who underwent LP, the average protein was high (79.25 ± 70.61 mg/dL), accompanied by a normal range of WBC (3.27 ± 1.27 per mm^3), which suggested albuminocytological disassociation. Additionally, the average CSFs' protein values were highest among the AIDP subtype (105.66 + 90.53 mg/dl) compared with axonal variants (52.83 + 33.13 mg/dl).

There was no significant difference between any of the GBS features in the para-infection and the post-infection group [Table 3], except for the electrodiagnostic pattern of the disease, which significantly had more frequent axonal damage versus demyelinating patterns in the para-infection group. Axonal-type injuries were more frequent in para-infection conditions (*P* value: 0.045). Patients with axonal patterns had higher ESR and CRP levels in comparison with those with demyelinating patterns (*P* value = 0.004 and 0.011, respectively).

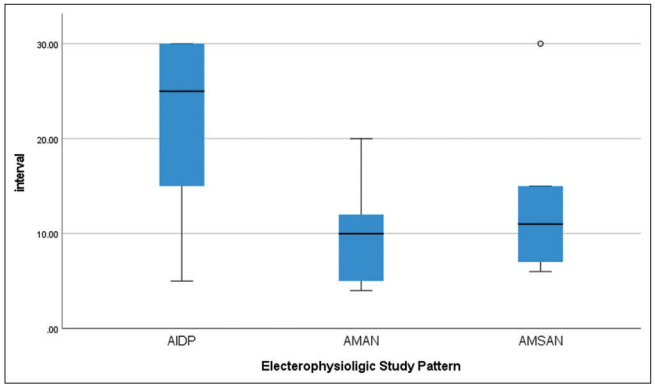


Figure 1: Simple box pilot of each electrophysiological subgroup of GBS compare with interval latency of neurological symptoms onset. *AIDP:* Acute Inflammatory Demyelinating Polyradiculopathy; AMAN: *Acute Motor Axonal Neuropathy; AMSAN: Acute Motor-Sensory Axonal Neuropathy; Interval: Average interval time between onset of COVID-19 and neurological symptoms*

Discussion

In the current study, we studied over 45 COVID-19-related GBS (CRG) individuals and compared their characteristics to those of non-CRG patients. We discuss each aspect of this accompanying in separate titles.

Demographic aspect

A previous study among non-CRG patients in Isfahan province concluded that the median age of patients was 42.78 ± 21.34 years.[19] Compared to the average age of our CRG patients (54.66 ± 10.021 years), GBS has developed at an older age in CRG patients. The age range is more similar to that of the Corona profile than to the age range of the non-CRG patients' profile, based on the average age of COVID-19 patients.[20] The majority of our patients were male (60%). Similar studies have demonstrated men's superiority[6,21] which corresponds to the profile of non-CRG patients.[19] Male predominance reflects the gender epidemiology of COVID-19; it is speculated that this is due to higher levels of ACE2 in males, providing more receptors for SARS-CoV-2 to infect.[22]

Interval time between symptoms

The average period from the initiation of COVID-19 to the onset of neurological symptoms is notable, since the inflammatory phase of COVID-19, which the symptoms of disease are aggravated, occurs around the same time (between 7 and 10 days of illness). Because several symptoms are shared by the two illnesses (particular tiredness, generalized weakness, and dyspnea), the diagnosis of GBS disease may be delayed or not performed at all.

Six of the patients were asymptomatic for COVID-19 at the onset of neurologic symptoms, or the diagnosis of COVID-19 was preceded by neurological symptoms. However, the SARS-CoV-2 virus was found in nasopharyngeal secretions of patients with lung involvement during hospitalization in all asymptomatic individuals. This conclusion is most likely attributable to COVID-19's long incubation time or to subtle symptoms in certain individuals. The remarkable thing point is that GBS may appear in mild COVID-19; not just in severe cases variant. Similar reports have been described in previous publications.[23,24]

EDX and paraclinical features

The most common EDX variation among CRG patients in our research was AIDP (55.1%). This is contrary to prior research on non-CRG patients in Isfahan, in which the majority of patients (24%) were found to have AMSAN.[19] We found significant differences in the frequency of AIDP and axonal variants at the onset of age. Axonal patterns evolved much later in life (mean: 50.11 ± 18.06 years in demyelinating versus 60.89 ± 13.56 years in axonal). Paradoxically, in the review article by Sriwastava S *et al*.,[25] among CRG patients, the mean age of the AIDP variants was significantly higher than that of the axonal type. In addition, the median time between disease onset was significantly (*P* value = 0.013) shorter in axonal types. As a result, a shorter interval of time between

Table 2: The patients' demographic, clinical, and prognostic characteristics

	Total ($n=45$)	Demyelinating ($n=26$)	Axonal ($n=19$)			*P**
		AIDP	Total	AMAN ($n=8$)	AMSAN ($n=11$)	AIDP versus Axonal
Age (year)	54.66±10.021	50.11±18.06	60.89±13.56	56.37±17.61	64.18±9.26	0.034
Sex						
Male	60% (27)	61.5% (16)	57.1% (12)	75.0% (6)	45.5% (5)	0.805
Female	40% (18)	38.5% (10)	42.9% (9)	25.0% (2)	54.5% (6)	
Asymptomatic COVID-19	13.6% (6)	16.0&(4)	10.5% (2)	0.0% (0)	18.2% (2)	0.600
Pulmonary involvement (based on HRCT)	86.4% (38)	73.1% (19)	100% (19)	100% (8)	100% (11)	0.022
Positive RT-PCR	95.5% (42)	96.2% (25)	94.7% (18)	87.5% (7)	100% (11)	0.820
Symptom's AIT**	18.07±9.88	21.73±9.49	12.00±8.00	10.20±6.41	13.80+9.73	0.013
Para/Post-infection state						
Para-infections	46.3% (19)	31.8% (7)	63.2% (12)	50% (4)	72.7% (8)	0.045
Post-infection	53.7% (22)	68.2% (15)	36.8% (7)	50% (4)	27.3% (3)	
CSF analysis (n=11)						
WBC counts (/mm)	3.27±1.27	3.00±1.58	3.50±1.04	3.33±1.52	3.66±0.57	0.545
Prot (mg/dL)	79.25±70.61	105.66±90.53	52.83±33.13	29.00±17.43	76.6+27.15	0.209
Glu (mmol/L)	76.87±18.49	76.40±5.68	77.16±25.64	63.33±5.85	91.00±32.18	0.949
Serum inflammatory markers						
WBC counts (/mL)	8751.16±2659.60	9092.00±2624.39	8277.77+2710.01	7575.00±2265.73	8840.00±3014.85	0.327
ESR (mm/hr)	32.12±25.42	23.50±18.42	49.36±29.41	33.00+25.97	58.71±28.70	0.004
CRP (mg/L)	24.11±30.288	14.18±20.19	39.71±37.18	34.60±41.39	42.55±36.93	0.011
Required respiratory equipment						
No equipment	33.3% (15)	46.2% (12)	15.8% (3)	25.0% (2)	9.1% (1)	0.089
Non-invasive	44.4% (20)	38.5% (10)	52.6% (10)	62.5% (5)	45.5% (5)	
Invasive	22.2% (10)	15.4% (4)	31.6% (6)	12.5% (1)	45.5% (5)	
Intubation time (n=10)	12.10±6.24	10.00±6.68	13.50±6.12	5.00±0.00	15.20±5.01	0.417
GBS disability score (after treatment)	2.66±1.59	2.23±1.47	3.26±1.59	2.75±1.75	3.63±1.43	0.030
Response to treatment	1.13±0.86	1.38±0.80	0.63±0.24	0.87±0.99	0.72±0.78	0.021
Mortality	8.9% (4)	7.6% (2)	10.5% (2)	12.5% (1)	9.1% (1)	0.741

AIDP: Acute Inflammatory Demyelinating Polyradiculopathy; AMAN: Acute Motor Axonal Neuropathy; AMSAN: Acute Motor-Sensory Axonal Neuropathy; EDX: Electrodiagnostic study test; DM: Diabetes mellitus; HTN: Hypertension; IHD: Ischemic heart disease; HLP: Hyperlipidemia; GBS: Guillain-Barré syndrome; CKD: Chronic kidney disease; HRCT: High-resolution computed tomography; CSF: Cerebrospinal fluid; WBC: White blood count; Prot: Protein; Glu: Glucose, ESR: Erythrocyte sedimentation rate, CRP : C-reactive protein. **The average interval time (AIT) between onset of COVID-19 and neurological symptoms. *Significant *P*-values considered lower than 0.05

COVID-19 manifestations and neurological symptoms predicts a higher risk of acquiring axonal damage and, as a result, a worse prognosis.

Although several infectious agents have been proposed as potential GBS triggers, it is now widely accepted that infection does not cause the disease *per se*.[26] GBS develops at least 2 weeks after the active phase of the illness as a result of autoimmune responses to these pathogens.

One of the key findings is that the existence of 19 individuals (46% of patients) happened as a para-infection profile rather than being caused by an antecedent infectious relationship. Other known pathogenic viruses, on the other hand, lead to GBS in at least 2–3 weeks. Another important finding was that para-infection was significantly (P value = 0.045) linked with axonal damage (63.2%) rather than demyelinating injury. (36.8%) Of course, the criteria chosen for para-infection individuals weren't exhaustive, and there were some drawbacks. One of the disadvantages is that the interval time is not always easy to assess because of the subtle COVID-19 symptoms in certain individuals. Additionally, it was impossible to accurately predict how long the infectious active phase would last because of the prolonged incubation period and differences in its duration among the patients.

Based on the results of our study, lung involvement in HRCT was more likely to cause axonal damage than demyelinating (P value = 0.022). Given that none of the patients' CSF had pleocytosis, we did not perform RT-PCR test on the CSF's samples. None of the previous studies reported positive RT-PCR results in CSF samples. The lack of evidence of virus presence in the CSF supports the most likely pathophysiology of the disease as an immune-mediated mechanism.

Table 3: Demographic, clinical, and prognostic features between para/post-infection groups

	Para-infection ($n=19$)	Post-infection ($n=22$)	*P*
Age	58.94±15.50	49.18±17.74	0.070
Sex			
Male	63.2% (12)	59.1% (13)	0.790
Female	36.8% (7)	40.9% (9)	
Treatment's response	1.00±0.94	1.27±0.70	0.296
Mortality	10.5% (2)	4.5% (1)	0.463
EDX pattern			
Demyelination	36.8% (7)	68.2% (15)	0.045
Axonal	63.2% (12)	31.8% (7)	
CSF finding			
WBC (/mm^3)	3.33±0.577	3.83±1.16	0.516
Prot (mg/dL)	90.50±19.36	84.33±100.20	0.908
Glu (mmol/L)	89.0±33.28	70.3±9.95	0.221

EDX: Electerodiagnostic study; AIDP: *Acute Inflammatory Demyelinating Polyradiculopathy; AMAN:* Acute Motor Axonal Neuropathy; AMSAN: Acute Motor-Sensory Axonal Neuropathy; CSF: Cerebrospinal fluid; Prot: Protein; Glu: Glucose

Conclusion

The patients were mostly imitating non-COVID-19-related GBS features. However, when the time interval between COVID-19 and the onset of GBS symptoms decreases, the similarities fade. An earlier onset of neurological symptoms and para-infection conditions is associated with an increase in the frequency of axonal-type variations and, as a result, a poor prognosis. The majority of evidence in CRG patients points to an autoimmune process throughout the COVID-19 course, including AIDP prevalence, responsiveness to immunomodulatory therapy, the lack of ganglioside antibodies in blood, and SARS-CoV-2 RNA in CSF samples. Further cohort and epidemiological studies are required to provide additional insight into the potential cause − effect relationship between COVID-19 and GBSs.

Ethics approval and consent to participate

This article was approved by the Research Committee of Isfahan University of Medical Sciences at 1400/05/31 and the code of ethics was also adopted (IR.MUI. MED. REC.1400.407).

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Nil.

Conflicts of interest

There are no conflicts of interest.

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