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Clinical and Electrophysiological Characteristics of Guillain–Barré Syndrome Across Pre-Pandemic, Pandemic, and Post-Pandemic Periods: A Ten-Year Retrospective Cohort Study

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ABSTRACT

Introduction: The COVID-19 pandemic markedly altered patterns of exposure to respiratory and gastrointestinal pathogens, healthcare-seeking behaviors, and environmental immune triggers. Guillain-Barré syndrome (GBS), an immune-mediated polyradiculoneuropathy frequently preceded by an infectious event, may therefore exhibit changes in clinical presentation or electrophysiologic subtype distribution. However, it remains unclear whether the clinical and electrophysiological characteristics of GBS have changed across pandemic periods.

Methods: This retrospective cohort included adults diagnosed with GBS between March 2015 and October 2025 at a tertiary referral center. Patients were grouped into pre-pandemic, pandemic, and post-pandemic periods. Clinical features, antecedent events, severity scores, and electrophysiologic subtypes (classified using Uncini criteria) were compared. Group comparisons were performed using chi-square, Fisher's exact test, and non-parametric tests where appropriate.

Results: Seventy patients were included: 31 pre-pandemic, 24 during the pandemic, and 15 post-pandemic. Age, sex, CSF findings, clinical and electrophysiological subtype distribution were similar across groups. Sensory and motor symptom subcategories and deep tendon reflexes did not differ. Antecedent event types differed significantly across periods (p=0.015). Admission Hughes score, MRC sum score, and mEGRIS score showed significant differences, indicating more severe presentation in the pre-pandemic group. Post-hoc comparisons suggested that these differences were primarily driven by comparisons between pre- and post-pandemic groups.

Conclusion: Despite pandemic-related epidemiological changes, the fundamental clinical and electrophysiological characteristics of GBS remained stable. However, differences in initial disease severity may reflect changes in environmental exposures and healthcare-seeking behavior rather than intrinsic alterations in disease pathophysiology

Keywords: COVID-19 pandemic, environmental influences, Guillain-Barré syndrome

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INTRODUCTION

The first case of COVID-19 in Türkiye was reported in March 2020, followed by the implementation of strict public health measures. In December 2020, vaccination with the inactivated Sinovac vaccine began, and was subsequently followed by the introduction of the mRNA-based BioNTech vaccine. In December 2021, another inactivated vaccine, Turkovac, was introduced. As the severity of the disease gradually decreased and its transmissibility became comparable to that of other circulating respiratory viruses, the Turkish Ministry of Health declared the end of the pandemic in May 2023 (1).

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive limb weakness, reduced or absent deep tendon reflexes, and variable involvement of sensory, cranial, autonomic, and respiratory systems. The global annual incidence is approximately 1–2 per 100,000 individuals.

Highlights

- **Clinical and electrophysiological characteristics of GBS remained stable across the pandemic periods.**
- **Distribution of antecedent events shifted significantly during the pandemic periods.**
- **In the post-pandemic group, milder level of disability were observed at the admission.**
- **Electrophysiological subtype distribution showed no significant temporal variation.**
- **Pandemic-related environmental factors did not alter the fundamental GBS phenotype.**

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Guillain-Barré syndrome is classified into two major subtypes: acute inflammatory demyelinating polyneuropathy (AIDP) and axonal variants, including acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN). These classifications are based on electrophysiological criteria, which have evolved over time (2–5). However, certain variants, such as Miller Fisher syndrome (MFS), often lack clear electrophysiological abnormalities in the early stages and are primarily diagnosed based on clinical features, with electrophysiological changes sometimes emerging during follow-up (6,7).

Infectious triggers, including Campylobacter jejuni, cytomegalovirus, Epstein–Barr virus, influenza-like illnesses, and gastrointestinal infections, play a central role in the pathogenesis of GBS through mechanisms of molecular mimicry (8–11).

The COVID-19 pandemic led to substantial changes in exposure to infectious agents, hygiene practices, reduced population mobility, and access to healthcare. Following the World Health Organization's declaration of the pandemic in early 2020, numerous case reports and series described GBS associated with SARS-CoV-2 infection. However, epidemiological studies have reported conflicting findings, with some demonstrating increased incidence or severity of GBS (12–14), while others reported a decline (15,16). These discrepancies may partly reflect regional differences in the prevalence of antecedent SARS-CoV-2 infection (12,15,17). For example, in 2020, the proportion of GBS cases associated with COVID-19 reached as high as 88% in Italy, whereas much lower rates (approximately 2%) were reported in countries such as Japan, where stricter public health measures were implemented (12,17).

Although previous studies have largely focused on the characteristics of COVID-19–associated GBS, there remains a paucity of data regarding the broader impact of the pandemic on the clinical and electrophysiological features of GBS. It therefore remains unclear whether pandemic-related changes have influenced disease severity or electrophysiological patterns of GBS. Given the substantial alterations in infection exposure and healthcare access during this period, understanding potential shifts in disease presentation is of considerable importance. Accordingly, the aim of this study was to evaluate differences in the clinical and electrophysiological characteristics of GBS across pre-pandemic, pandemic, and post-pandemic periods over a ten-year timeframe.

METHODS

Patients aged 18 years and older who presented to our tertiary referral center between March 2015 and October 2025 with acute or subacute onset progressive muscle weakness and/or sensory symptoms were included. Patients were classified according to the Brighton criteria. The pre-pandemic period included patients presenting between March 2015 and March 2020; the pandemic period included those presenting between March 2020 and May 2023; and the post-pandemic period included patients diagnosed from June 2023 onward, when SARS-CoV-2 began to demonstrate transmissibility and disease severity similar to other circulating upper respiratory tract viruses. Demographic data were analyzed separately for each period.

Exclusion criteria included acute polyneuropathies due to systemic, toxic, or other causes; transverse myelitis; acute myopathy; critical illness neuropathy/myopathy; and acute-onset chronic inflammatory demyelinating polyneuropathy mimicking GBS (Figure 1).

Clinical symptoms and neurological examination findings were extracted from the hospital database. Sensory, motor, autonomic,

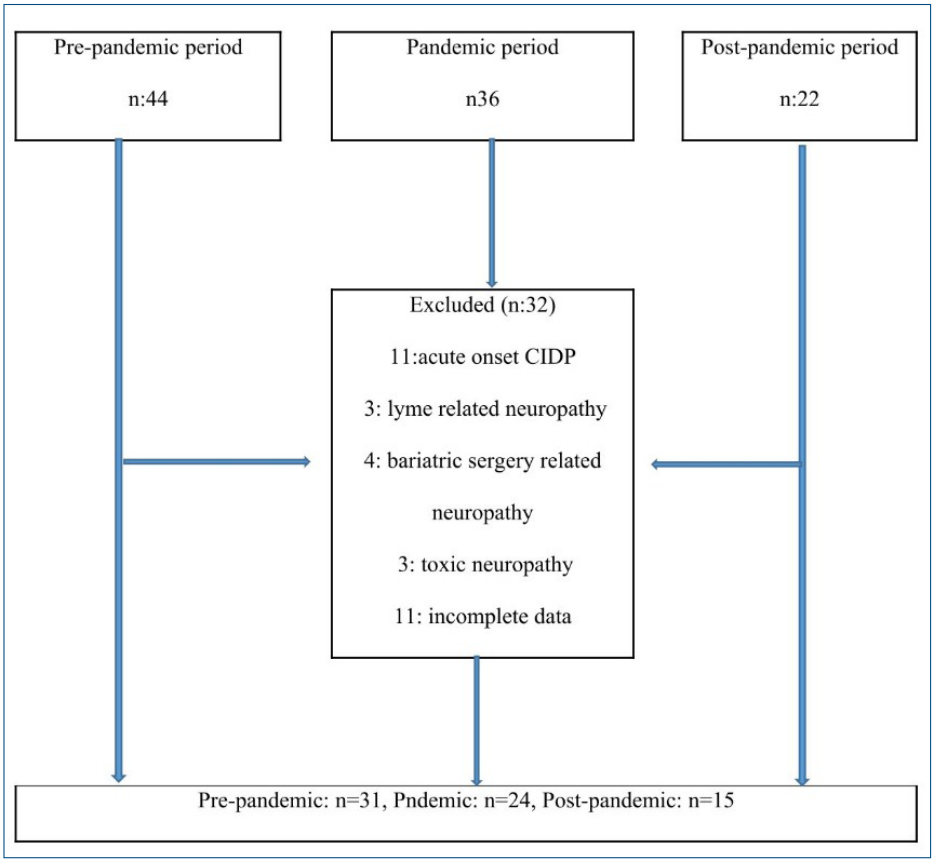

Figure 1. Flowchart of the study. CIDP, chronic inflammatory demyelinating polyneuropathy.

respiratory, cranial neuropathy, and ataxia symptom and sign categories were labeled positive if the patient had one or more findings in the respective category. Cerebrospinal fluid leukocyte counts and protein levels were recorded. Clinical severity was assessed using antecedent event presence and type, Medical Research Council (MRC) sum score and Hughes functional score at initial examination and at nadir, and Erasmus GBS Outcome Score and modified Erasmus GBS Respiratory Insufficiency Score at admission.

Electrodiagnostic evaluation was performed within the first 28 days from symptom onset. Patients whose studies clearly demonstrated features corresponding to GBS subtypes according to the criteria of Uncini et al. were included. Classification was based on all examinations performed for each patient (18).

Electrophysiological records were re-evaluated. Sensory nerve conduction studies, motor nerve conduction studies, and F-wave studies were performed in one upper and one lower extremity. Median and ulnar sensory and motor studies were evaluated in the upper extremities; tibial and peroneal motor and sural sensory studies were evaluated in the lower extremities. F-waves were recorded from the ulnar, median, and tibial nerves. All sensory studies were performed antidromically. All electrophysiological recordings were reviewed by the same investigator (N. P.).

All statistical analyses were performed using the IBM Statistical Package for Social Sciences (SPSS) program version 20.0 software package (IBM, Armonk, New York). Categorical variables were expressed as frequencies and percentages, and comparisons between the three study periods (pre-pandemic, pandemic, post-pandemic) were conducted using the Chi-square test of independence. Because many contingency tables contained cells with an expected frequency <5, which violates Chi-square assumptions, Fisher's Exact Test or Monte Carlo Exact Test was used as the primary inferential method when applicable. These exact tests were considered reliable indicators of statistical significance in the presence of sparse data. For ordinal variables with multiple ordered categories (e.g., MRC sum score, Hughes functional grading scale, mEGRIS score), the Linear-by-Linear Association test was additionally examined to evaluate potential monotonic trends across periods. However, final interpretation relied on Fisher's Exact Test outcomes when assumption violations were present.

Ethical approval was obtained from the local ethics committee (approval number: 96, date: 25 March 2026). The study was conducted in accordance with the Declaration of Helsinki.

RESULTS

After exclusions, 70 patients remained. Diagnostic certainty according to Brighton criteria was classified as level 1 in 38 patients, level 2 in 14 patients, and level 3 in 18 patients. No significant differences were observed in age (p=0.631) or sex distribution between groups (p=0.154). (Table 1)

No statistically significant difference was observed in the time from symptom onset to hospital admission across the study periods (p=0.278). However, a numerically shorter interval was noted in the post-pandemic period compared to the other groups.

Most patients presented with motor (57/70, 81.4%) and sensory (54/70, 77.2%) symptoms. No significant differences were observed between periods regarding sensory (Fisher p=0.168 and 0.233 for 3- and 2-category analyses, respectively) or motor symptom patterns (Fisher p=0.392). Deep tendon reflex (p=0.897), ophthalmoparesis (p=0.66), other cranial neuropathies (p=0.573), autonomic symptoms (p=0.624), ataxia (p=0.11), or respiratory symptoms/signs (p=0.554) showed no significant differences.

Although no difference was observed in the overall presence of antecedent events (p=0.782), categorization into upper respiratory tract infection (URTI), gastroenteritis (AGE), COVID-19 infection, and vaccination both Fisher's Exact Test and Monte Carlo estimates indicated a statistically significant association between pandemic period and antecedent event type (Fisher's Exact Test, p=0.015; Monte Carlo two-sided p=0.021, 99% CI=0.017–0.025). These results demonstrate that the distribution of antecedent events varied significantly across the three periods. Pre-pandemic period: URTI and gastroenteritis were the most common antecedent events. Pandemic period: COVID-19 infection emerged as a notable antecedent event, while traditional infectious triggers decreased in frequency. Post-pandemic period: COVID-19 remained present but diminished compared to the pandemic phase; vaccination-related antecedent events increased in proportion (Table 1).

Admission MRC sum scores differed significantly between groups (Kruskal-Wallis X^2=7.686, p=0.021), however, when the Bonferroni correction was applied, a value close to the threshold value of 0.0167 was found for this correction (p=0.0196). This difference indicated that patients had better muscle strength when they were admitted to the hospital in the post-pandemic period. No significant differences were observed in nadir MRC sum scores (p=0.188). Although no statistically significant difference was detected between GBS subtypes, the higher proportion of the Miller Fisher syndrome (MFS) subtype observed in the post-pandemic group may explain the lower frequency of motor weakness detected in these patients.

The mEGRIS with higher scores showed a statistically significant difference between the pre-pandemic and post-pandemic groups (Fisher's exact test, p=0.003), with higher scores observed in the post-pandemic period.

Although the overall distribution of Hughes functional scores between the periods showed only borderline statistical significance (Fisher's Exact Test, p=0.055), a significant linear trend toward better functional status over time was demonstrated (Linear-by-Linear Association, p=0.007). Patients in the post-pandemic period exhibited lower disability at the time of admission compared with the pre-pandemic group. In contrast, there was no difference in Hughes scores among patients at the peak of their illness.

Pairwise comparisons of clinical parameters across study periods are presented in Table 2. A significant difference in MRC sum score was observed between the pre-pandemic and post-pandemic groups (p=0.028), with higher scores in the post-pandemic period, indicating milder disease severity at hospital admission. Similarly, Hughes score differed between these two periods (p=0.055). In contrast, no significant differences were observed between the pre-pandemic and pandemic groups or between the pandemic and post-pandemic groups for these variables.

Regarding antecedent event type, a significant difference was found between the pre-pandemic and pandemic periods (p=0.015), while other pairwise comparisons were not statistically significant. For mEGRIS, a significant difference was observed between the pre-pandemic and post-pandemic groups (p=0.018), whereas other comparisons did not reach statistical significance.

Table 1. General clinical features GBS patients

	All GBS	Pre-pandemic	Pandemic	Post-pandemic	p
Age	52.9±17.5	52.5±3.1	54.3±3.6	51.9±4.8	0.631
Sex (F/M)	29/41	9/22	13/11	7/8	0.154
Symptom onset to admission, days, mean ± SD	6.5±6.4	6.8±1.2	6.9±1.3	5.13±1.5	0.278
Clinical features					
Sensory symptoms/signs, n (%)	54(77.2)	22(70.1)	18(75)	14(93.3)	0.227
Distal hypoesthesia	53(98.2)	22(100)	18(100)	13(92.9)	
Not recorded	16(21.8)	9(29.9)	6 (25)	1(6.7)	
Motor symptoms/signs, n (%)	57(81.4)	28(90.3)	20(85.3)	9(60)	0.33
Distal dominant weakness	13(18.6)	6(19.3)	5(20.8)	2(13.3)	
Distal/proximal weakness	41(58.6)	20(64.5)	14(58.3)	7(46.7)	
Proximal dominant weakness	3(4.3)	2(6.5)	1(4.2)	0	
Not recorded	13(18.6)	3(9.7)	4(16.7)	6(40)	
Cranial neuropathy (bulbar, facial diplegia, and paresis), n (%)	23(32.9)	11(35.5)	6(25)	6(40)	0.573
Ophthalmoparesis, n (%)	13(18.6)	5(16.1)	4(16.7)	4(26.7)	0.66
Autonomic symptoms/signs, n (%)	15(21.4)	5(16.1)	6(25)	4(26.7)	0.624
Ataxia, n (%)	15(21.4)	4(13)	5(20.8)	6(40)	0.11
Respiratory symptoms/signs, n (%)	9(12.9)	5(16.1)	2((8.3)	2(13.3)	0.554
MRC sum score, mean ± SD	47.85±17.65	43.77±2.31	49.87±2.17	53.06±1.93	**0.021**
Hughes score admission, mean ± SD	2.62±1.11	3±0.19	2.5±2.33	2.06±0.67	**0.023**
Hughes score nadir, mean ± SD	3.17±1.38	3.35±0.23	3.16±0.3	2.95±0.35	0.224
Symptom onset to nadir (days) mean ± SD	8.32±5.09	7.25±0.97	7.58±1.28	8.06±1.53	0.807
Deep tendon reflexes, n (%)					0.897
Hypo/areflexia	61(87.1)	28(90.3)	20(83.3)	13(86.7)	
Normal deep tendon reflexes	9(12.9)	3(9.7)	4(16.7)	2(13.3)	
Erasmus GBS outcome score, mean ± SD	2.87±2.48	3.61±0.51	2.50±0.45	1.93±0.44	0.134
Antecedent event n (%)	47(67.1)	21(67.7)	15(62.5)	11(73.3)	0.782
Antecedent event type n (%)					**0.015**
URTI	19(27.1)	9(29)	4(16.7)	6(40)	
SARS-Cov2	5(7.1)	0	5(20.8)	0	
Gastroenteritis history	20(28.6)	12(38.7)	4(16.7)	4(26.7)	
Others (vaccination, Surgery)	3(4.3)	0	2(8.3)	1(6.7)	
None	23(32.9)	10(32.3)	9(37.5)	4(26.7)	
Antecedent incident to symptom onset, mean ± SD	11.80±5.86	10.14±1.11	12±1.65	14.72±1.74	0.098
Symptom onset to LP (days) mean ± SD	7.55±5.77	7.25±0.97	7.58±1.28	8.06±1.23	0.931
CSF protein levels (mg/dL) mean ± SD	77.42±43.19	73.83±6.79	82.54±9.26	76.60±13.34	0.646
CSF leukocyte levels (cells/μL) mean ± SD	1.52±3.59	1.1±0.40	2.5±1.06	1.12±0.63	0.268
≤10, n (%)	67(95.7)	31(100)	22(91.7)	14(93.3)	
10–50, n (%)	3(4.3)	0	2(8.3)	1(6.7)	
>50, n (%)	0	0	0	0	
Treatment, n (%)					0.182
IVIg	63(98.2)	29(93.5)	20(83.4)	14(93.3)	
PE	1(1.4)	0	0	1(6.7)	
IVIg + PE	2(2.9)	0	2(8.3)	0	
No treatment	4(5.7)	2(6.5)	2(8.3)	0	
ICU admission, n (%)	11(15.7)	5(16.3)	4(16.7)	2(13.3)	0.959
Death, n (%)	3(4.3)	0	2(8.3)	1(6.7)	0.155
mEGRIS	1.32±0.87	1±0.78	1.70±0.24	2.33±2.32	**0.003**

CSF: cerebrospinal fluid; F: female; GBS: Guillain-Barré Syndrome; ICU: intensive care unit; IVIg: Intravenous immunoglobulin; LP: lumbar puncture; M: male; mEGRIS: modified Erasmus GBS respiratory insufficiency score; MRC: medical research council; PE: plasmapheresis; SD: standard deviation; URTI: upper respiratory tract infection.

Intensive care unit requirement did not differ between periods (Fisher p=1.000).

CSF protein and leukocyte distributions showed no significant differences (p=0.646 and p=0.268, respectively).

Almost all patients received intravenous immunoglobulin (IVIg) treatment, and treatment modality did not differ between periods (p=0.182).

Electrophysiological subtype distribution (p=0.661), and clinical subtype distribution (p=0.224) did not differ significantly (Table 3).

Table 2. Post-hoc pairwise comparisons of clinical characteristics across study periods

	MRC sum score	Hughes score	Antecedent incident type	mEGRIS
Pre-pandemic and Pandemic	0.185	0.111	**0.015**	0.056
Pre-pandemic and Post-pandemic	**0.028**	**0.055**	0.397	**0.018**
Pandemic and Post-pandemic	1.000	0.153	0.207	0.087
	Kruskal-Wallis test	Chi-square test	Chi-square test	Chi-square test

mEGRIS: modified Erasmus GBS respiratory insufficiency score; MRC: medical research council; data are presented as adjusted p-values for pairwise comparisons; continuous variables were analyzed using the Kruskal-Wallis test with Dunn-Bonferroni post-hoc correction, while categorical variables were compared using the Chi-square test; statistical significance was defined as p<0.05.

Table 3. GBS Subtype classifications and comparison of the electrodiagnostic subtypes in the pre-pandemic, pandemic and post-pandemic periods

	All GBS	Pre-pandemic	Pandemic	Post-pandemic	p
GBS subtypes n (%)					
AIDP	21(30)	9(29)	8(33.3)	4(26.7)	0.224
AMAN	11(15.7)	6(19.4)	5(20.8)	0	
AMSAN	25(35.7)	11(35.5)	8(33.3)	6(40)	
MFS	11(15.7)	4(12.9)	3(12.5)	4(26.7)	
Others	2(2.9)	1(3.2)	0	1(6.7)	
EDX subtype according to criteria of Uncini et al.[5] n (%)					0.661
Demyelinating	21(30)	9(29)	7(29.2)	5(33.3)	
Axonal	41(58.6)	19(61.3)	14(58.3)	8(55.3)	
Equivocal	1(1.4)	0	1(4.2)	0	
Inexcitable	1(1.4)	0	0	1(6.7)	
Normal	6(8.6)	3(9.7)	2(8.3)	1(6.7)	

AIDP: acute inflammatory demyelinating polyneuropathy; AMAN: acute motor axonal neuropathy; AMSAN: acute motor and sensory axonal neuropathy; EDX: electrodiagnostic study; GBS: Guillain-Barré syndrome; MFS: Miller Fisher syndrome.

DISCUSSION

This ten-year retrospective analysis revealed that although the COVID-19 pandemic dramatically altered infection exposure patterns and societal behaviors, the fundamental phenotype of Guillain-Barré syndrome remained stable. Core features such as cerebrospinal fluid findings, reflex abnormalities, and electrophysiological subtype distribution did not differ between periods.

A key finding of this study is the apparent dissociation between initial clinical presentation and disease severity at nadir. Although patients in the post-pandemic period presented with significantly milder clinical findings, the absence of differences in nadir severity suggests that the intrinsic disease course of GBS has remained stable over time. This indicates that the observed improvement at admission likely reflects external factors rather than changes in disease biology.

One possible explanation is earlier hospital presentation in the post-pandemic period, potentially driven by increased public awareness and heightened clinical vigilance following the COVID-19 pandemic. Improved access to healthcare services after the initial disruption may also have contributed to earlier diagnosis and hospitalization. However, the similar nadir severity across all periods implies that once the disease process is initiated, its progression and maximum severity are largely unaffected by these factors.

These findings highlight that while the pandemic may have influenced patient behavior and healthcare system dynamics, it did not fundamentally alter the natural history of GBS.

However, meaningful shifts were identified in antecedent event types and early clinical manifestations. The decline in respiratory antecedents is consistent with the global reduction in circulating respiratory viruses during periods of mask use and mobility restrictions.

SARS-CoV-2 has been proposed to trigger GBS through immune-mediated mechanisms, including molecular mimicry and cytokine-mediated inflammation (19–21). Structural similarity has been identified between human heat shock proteins and SARS-CoV-2 proteins, and the role of heat shock proteins in autoimmune processes underlying GBS has previously been demonstrated (22,23). However, our findings did not demonstrate any significant change in electrophysiological subtype distribution across periods. This suggests that while environmental triggers may have shifted, the core pathophysiological mechanisms underlying GBS remained stable.

Epidemiological studies including all patients registered in national health databases have reported that despite millions of individuals becoming infected over a short period, GBS remains a rare but serious complication of COVID-19. In a study by Sharma et al., the frequency of GBS among COVID-19 cases was 0.07%, compared with 0.08% among non–COVID-19 GBS cases (24). In a meta-analysis by Censi et al. evaluating the relationship between SARS-CoV-2 infection and GBS in Italy, the estimated incidence of GBS during the first pandemic wave was 0.013 per 1,000 SARS-CoV-2–infected individuals, which was lower than the incidence reported during Campylobacter jejuni and Zika virus outbreaks (25). A reduction in GBS risk was observed in some countries during the pandemic. Mandatory mask use, maintenance of social distancing, and restrictions on mobility implemented to control SARS-CoV-2 transmission may have reduced the circulation of infectious agents known to trigger GBS. Several studies have demonstrated a decline in Campylobacter jejuni and other gastrointestinal and respiratory infections during the pandemic (26–28).

In a study by Kim et al., which compared the incidence of eight legally notifiable viral respiratory infections in South Korea during the three years preceding the pandemic and a comparable ten-month pandemic period (March 9 to December 31), the incidence of these infections was found to be significantly reduced during the pandemic. The authors interpreted this finding as highly valuable in terms of infection control (29).

In the present study, a significant decrease in the incidence of upper respiratory tract infections (URTI) and acute gastroenteritis (AGE) was observed during the pandemic period, followed by a gradual return toward pre-pandemic levels in the post-pandemic period. In contrast, Arami et al. reported a significant increase in GBS frequency during the pandemic at a referral center in Iran (14). However, a three-year multicenter study including 177 cases from 13 referral centers in Istanbul, including our center, did not demonstrate an increase in GBS incidence (30), which is consistent with our findings.

The lack of increase in GBS incidence in our cohort may be attributed to strict adherence to public health measures, particularly in urban areas where the majority of the population resides. These measures likely reduced exposure to common infectious triggers such as URTI and AGE. While the pandemic introduced novel antecedent factors, including SARS-CoV-2 infection, our findings suggest that its long-term impact may involve a shift toward milder clinical presentations of GBS rather than an increase in overall disease incidence. Changes in health-related behaviors and hygiene practices during the pandemic may have played a role in altering the distribution of antecedent events.

Further large-scale, multicenter studies are warranted to confirm these observations and to better elucidate the mechanisms underlying these changes.

Although mEGRIS scores were higher in the post-pandemic period, this finding should be interpreted with caution. The increase appears to be influenced by three out of four patients with Miller Fisher syndrome presenting with bulbar involvement, which disproportionately elevates mEGRIS scores. Despite this, overall clinical severity at admission and nadir was not increased, and patients in the post-pandemic period demonstrated better functional status. Therefore, the higher mEGRIS scores in this group likely reflect a subgroup effect rather than a true increase in disease severity.

The distinction between parainfectious and postinfectious disorders in SARS-CoV-2–associated GBS is based solely on temporal criteria. However, given the very low rate of positive SARS-CoV-2 polymerase chain reaction results in cerebrospinal fluid reported in case reports and series, a postinfectious mechanism is more widely accepted. In our cohort, the mean interval between COVID-19 onset and GBS development was 12 days. All GBS cases temporally associated with SARS-CoV-2 infection exhibited mild disease severity for both COVID-19 and GBS. Infection-related symptoms had completely resolved before presentation. Hughes functional scores at admission ranged from 1 to 4, and the mean MRC sum score was 49. Although the literature reports more severe clinical courses, higher rates of cranial nerve involvement, and increased intensive care unit admissions in SARS-CoV-2–associated GBS (14,25), the cases followed at our center did not exhibit these characteristics. This discrepancy may be related to the single-center nature of the study and the inability to capture all cases from the relevant period.

The stability of electrophysiological subtypes despite environmental changes reinforces the notion that pandemic-related influences did not alter the underlying pathophysiology of GBS, but rather modified exposure to relevant immune triggers.

Limitations

Limitations of this study include its retrospective design, single-center setting, and variability in the timing of electrophysiological assessments. An additional limitation is the potential for selection bias related to changes in healthcare-seeking behavior during the pandemic. Mild cases may have been underrepresented, particularly during the early pandemic period, which could have influenced the observed differences in disease severity.

Ethics Committee Approval: Ethical approval has been obtained from the Ethics Committee of Ümraniye Training and Research Hospital. (approval number: 96, date: 25 March 2026).

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