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ORIGINAL ARTICLE

Guillain-Barre syndrome: A single-center experience

Gülsüm Aslı Tatlı, Ekrem Sarıkaya, Özlem Kayım Yıldız

Department of Neurology, Cumhuriyet University Faculty of Medicine, Sivas, Türkiye

ABSTRACT

Objectives: This study aimed to describe the clinical and electrophysiological characteristics and outcomes of Guillain-Barre syndrome (GBS) in the Sivas province.

Patients and methods: In this retrospective study, the medical files and the electrophysiological raw data of 69 patients (43 males, 26 females; mean age: 55.8±17 years; range, 20 to 88 years) with GBS who were admitted between January 1, 2011, and January 1, 2023, were reviewed. The outcomes were in-hospital mortality and the modified Rankin Scale (mRS) score at hospital discharge.

Results: The subtypes of GBS were as follows: acute inflammatory demyelinating polyradiculoneuropathy (n=33, 47.8%), acute motor axonal neuropathy (n=10, 14.5%), acute motor and sensory axonal neuropathy (n=11, 15.9%), Miller Fisher syndrome (n=2, 2.9%), and unclassified (n=13, 18.8%). Sixteen (23.2%) patients were admitted to the intensive care unit due to respiratory insufficiency or dysautonomia. Fourteen (20.3%) of the patients needed mechanical ventilation. The median mRS score at hospital discharge was 2 (IQR, 1-5). The in-hospital mortality rate was 8.7%. Age, blood glucose level on admission, bulbar symptoms, dysautonomia, respiratory compromise necessitating mechanical ventilation, and intensive care unit admission were found to be independently associated with in-hospital mortality. Age, cerebrospinal fluid glucose levels, and the length of hospital stay were positively correlated, and symptom duration on admission and serum albumin levels were negatively correlated with mRS scores at discharge.

Conclusion: Axonal variants were common and associated with unfavorable outcomes in the Sivas province.

Keywords: Guillain-Barre syndrome, mechanical ventilation, mortality, prognosis.

Guillain-Barre syndrome (GBS) is the most common cause of acquired acute polyneuropathy worldwide, with an annual incidence of 1 to 2 in 100,000 individuals.[1] Males are affected more than females, and the incidence of the disease increases with age.[1] The typical manifestation of the disease is ascending sensorimotor dysfunction with diminished or abolished deep tendon reflexes; however, there is considerable clinical heterogeneity, and subtypes and variants of GBS exist.[2] The diagnosis is based on patient history, neurological and electrophysiological examination findings, and the results of the cerebrospinal fluid (CSF) analysis.[2] Electrophysiological examinations help in differentiating the subtypes of GBS, including acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor and sensory axonal neuropathy (AMSAN).[3] There are also variants of the disease with distinctive clinical features, such as ophthalmoplegia, ataxia, and areflexia in Miller Fisher syndrome (MFS).[4]

A substantial geographical variability in the clinical manifestations, severity, subtypes and prognosis of GBS was reported.[5] This study aimed to determine the prognostic factors in GBS patients who were admitted to a tertiary hospital in the Sivas province.

PATIENTS AND METHODS

In this retrospective study, medical files of 69 consecutive patients (43 males, 26 females;

Correspondence: Özlem Kayım Yıldız, MD. Cumhuriyet Üniversitesi Tıp Fakültesi Nöroloji Anabilim Dalı, 58140 Sivas, Türkiye.
E-mail: ozlemkayim@yahoo.com
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mean age: 55.8±17 years; range, 20 to 88 years) with GBS who were admitted to the Cumhuriyet University Faculty of Medicine Department of Neurology between January 1, 2011, and January 1, 2023, were reviewed. The diagnostic criteria proposed by Leonhard et al.[2] were used to diagnose the classical forms and variants (e.g., MFS) of GBS. Progressive (up to four weeks) bilateral weakness of extremities and absence or decrease of deep tendon reflexes were required for the diagnosis of the classical forms of GBS.[2] To diagnose MFS, the presence of ophthalmoplegia, ataxia, and areflexia was required.[2] The patients aged 18 years or over who were admitted within two weeks of symptom onset and who underwent a standardized electrophysiological examination performed in the neurophysiology laboratory were included. The electrophysiological raw data were reviewed by one of the authors according to the electrophysiological classification proposed by Uncini et al.[3] The findings were considered abnormal when they were outside the normal range of our laboratory (the supplementary table shows the normal values of our laboratory). Patients with equivocal electrophysiological findings due to performance in very early stages were included into the study if they had clinical (flask weakness with diminished or abolished deep tendon reflexes progressing over less than four weeks) and laboratory (albuminocytological dissociation in CSF) evidence of GBS. These patients were labeled as the unclassified subtype. The exclusion criteria were as follows: disease progression of more than four weeks, fever, sensory level suggesting a myelopathy, sphincter dysfunction, findings indicating upper motor neuron involvement, including spasticity, hyperreflexia, clonus, and extensor plantar reflexes, CSF pleocytosis (>50×10^6/L), and any other clinical, imaging, or electrophysiological evidence indicating an alternative diagnosis. This study protocol was approved by the Cumhuriyet University Non-Interventional Clinical Research Ethics Committee (date: 21.09.2023, no: 2023-09/40). Due to the retrospective design of the study, the requirement for informed consent was waived. The study was conducted in accordance with the principles of the Declaration of Helsinki.

All patients underwent lumbar puncture. Albuminocytological dissociation (increased protein level in the absence of pleocytosis) was considered a supportive feature; however, the presence of normal protein level was not considered an exclusion criterion in a patient with otherwise typical GBS. Patients with hypercapnia, hypoxemia, rapidly progressive muscle weakness, or autonomic disturbances were admitted to the intensive care unit (ICU), and mechanical ventilation was applied to patients with severe respiratory compromise.

The demographic characteristics of the patients, comorbidity status, symptom duration on admission, antecedent events, clinical features (ophthalmoplegia, facial paralysis, bulbar symptoms, neuropathic pain and dysautonomia), treatments received, mechanical ventilation, ICU admission, the length of hospital and ICU stay, in-hospital mortality, and the modified Rankin Scale (mRS) score at hospital discharge, as well as the laboratory parameters on admission and the CSF findings, were analyzed. Clinical outcomes were in-hospital mortality and mRS scores at hospital discharge.

Statistical analysis

Data were analyzed using IBM SPSS version 23.0 software (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to determine if the variables were normally distributed. Data were presented as frequency (%), mean ± standard deviation (SD) or median (interquartile range, IQR), as appropriate. The chi-square test was used for comparisons of the categorical variables. Student's t-test, the independent sample t-test, one-way analysis of variance, and the Kruskal-Wallis test with post hoc analyses were used for comparisons of continuous variables between groups. Multivariate analysis was used to determine the variables independently associated with clinical outcomes (in-hospital mortality and mRS scores at discharge). A p-value <0.05 was considered statistically significant.

RESULTS

Table 1 shows the patients' demographics, comorbidity status, antecedent events, symptom duration on admission, clinical characteristics, GBS subtypes, laboratory parameters, results of CSF analysis, treatments received, mechanical ventilation requirement, ICU admission rates, duration of hospital and ICU stay, in-hospital mortality rate, and mRS scores at hospital discharge.

The most common comorbid diseases were hypertension, diabetes, and coronary heart disease. More than half of the patients had antecedent events within four weeks of symptom onset, upper respiratory tract infections being the most common, followed by gastroenteritis, trauma, surgery, and

TABLE 1
The demographic and the clinical characteristics of patients with GBS

	n	%	Mean±SD	Median	IQR
Age (year)			55.8±17		
Sex					
Female	26	37.7			
Male	3	62.6			
Comorbid diseases					
Diabetes mellitus	14	20.3			
Hypertension	23	33.3			
Coronary heart disease	13	18.8			
Preceding event					
Upper respiratory tract infection	14	20.3			
Gastroenteritis	13	18.8			
Other (trauma, surgery, vaccination)	10	14.4			
Unknown	32	46.4			
Symptom duration on admission (day)				7	3.3-10
GBS subtypes					
AIDP	33	47.8			
AMAN	10	14.5			
AMSAN	11	15.9			
MFS	2	2.9			
Unclassified	13	18.8			
Inability to walk	51	73.9			
Ophthalmoplegia	6	8.7			
Facial paralysis	9	13			
Bulbar symptoms	26	37.7			
Neuropathic pain	30	43.5			
Dysautonomia	9	13			
ICU admission	16	23.2			
Mechanical ventilation	14	20.3			
Stay in hospital (day)				15	11-23
Stay in ICU (day)			8.3±7		
Treatment					
IVIG	21	30.4			
PLEX	26	37.7			
IVIG + PLEX	11	15.9			
None	11	15.9			
mRS score at hospital discharge				2	1-5
In hospital mortality	6	8.7			

GBS: Guillain-Barre syndrome; SD: Standard deviation; IQR: Interquartile range; AIDP: Acute inflammatory demyelinating polyradiculoneuropathy; AMAN: Acute motor axonal neuropathy; AMSAN: Acute motor and sensory axonal neuropathy; MFS: Miller Fisher syndrome; ICU: Intensive care unit; IVIG: Intravenous immunoglobulin; PLEX: Plasma exchange; mRS: Modified Rankin Scale.

TABLE 2
The serum and CSF analysis results of patients with GBS

	Mean±SD	Median	IQR
Hemoglobin (g/dL)	14.3±2		
White blood cells ($\times 10^9$/L)		8.2	6.8-10.9
Platelet (×/μL)		262.5	228.3-337
Serum albumin (g/dL)	40.1±4.6		
Blood glucose (g/dL)		113.5	97.3-129.8
C-reactive protein (g/dL)		4.6	2.8-13.3
Erythrocyte sedimentation rate (mm/h)		15	4-47
CSF protein (g/dL)		69.6	48.3-125
CSF glucose (g/dL)		70	65.5-84.5

CSF: Cerebrospinal fluid; GBS: Guillain-Barre syndrome; SD: Standard deviation; IQR: Interquartile range.

TABLE 3
The differences among GBS subtypes

	AIDP			AMAN			AMSAN			MFS			Unclassified			
	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (year)			57.3±16.8			54.3±13.9			65.4±12.4			29.5±4.9			48.8±18.7	<0.05*
Mechanical ventilation	4	12.1		3	30		6	54.5		0	0		1	17.7		<0.05**
Bulbar symptoms	7	21.2		8	72.7		0	0		0	0		0	0		<0.05†
mRS score at hospital discharge, median (IQR)			2 (1-3.5)			4.5 (1.8-5.3)			5 (2-5)			2 (1-2)			1 (1-2)	<0.05‡

GBS: Guillain Barre syndrome; SD: Standard deviation; mRS: Modified Rankin scale; IQR: Interquartile range; * MFS *vs.* AIDP, p=0.039, MFS *vs.* AMSAN, p=0.031; ** AMSAN *vs.* AIDP, p=0.004; AMSAN *vs.* unclassified, p=0.012; † AIDP *vs.* AMAN, p=0.003; ‡ AMSAN *vs.* AIDP, p=0.024, AMSAN *vs.* unclassified p=0.004; AMAN *vs.* unclassified, p=0.023. AIDP: Acute inflammatory demyelinating polyradiculoneuropathy; AMAN: Acute motor axonal neuropathy; AMSAN: Acute motor and sensory axonal neuropathy; MFS: Miller-Fisher syndrome.

vaccination. The median symptom duration on admission was 7 (range, 3.3-10) days. The most common GBS subtype was AIDP, followed by AMSAN and AMAN; there were two (2.9%) patients with MFS. The subtype could not be determined in 13 (18.8%) patients due to early electrophysiological evaluation or equivocal findings. A control electrophysiological evaluation in this group either could not be performed or yielded equivocal findings. The frequency of the clinical features was as follows: inability to walk in 51 (73.9%), bulbar symptoms in 26 (37.7%), neuropathic pain in 30 (43.5%), facial paralysis in nine (13%), dysautonomia in nine (13%), and ophthalmoplegia in six (8.7%), two of whom had MFS. The most common therapy received was plasma exchange (37.7%), followed by intravenous immunoglobulin (IVIG, 30.4%). Eleven (15.9%) patients were treated with plasma exchange followed by IVIG therapy due to nonsatisfactory response to plasma exchange and severe respiratory insufficiency or dysautonomia. These patients did not have a disease course that suggested chronic inflammatory demyelinating polyradiculoneuropathy. Eleven (15.9%) patients did not receive IVIG or plasma exchange since they had mild, nondisabling symptoms that did not interfere with mobilization and nonprogression. Sixteen (23.2%) patients were admitted to the ICU, mostly due to respiratory insufficiency and autonomic disturbances. Mechanical ventilation was needed by 20.3% of the patients. The median mRS score at hospital discharge was 2 (IQR, 1-5). The median length of hospital stay was 15 (IQR, 11-23) days, and the mean duration of ICU stay was 8.3±7 days.

Table 2 shows the patients' laboratory data on admission. The median CSF protein level was 69.6 (IQR, 48.3-125; range, 25 to 422; normal range: 15 to 45) g/dL.

Significant differences among GBS subtypes regarding age, bulbar symptoms, mechanical ventilation need, and mRS scores at hospital discharge were found. Patients with MFS were younger than other patients. None of the patients with MFS had bulbar symptoms or needed mechanical ventilation. Bulbar symptoms were not present in patients with AMSAN, but they were common in patients with AMAN. Patients with AMSAN had the highest rate of mechanical ventilation and mRS scores at hospital discharge (Table 3).

In-hospital mortality

The in-hospital mortality rate was 8.7% (n=6; Table 1). A multivariate analysis revealed

TABLE 4
Factors associated with in-hospital mortality

	Expired					Survived					
	n	%	Mean±SD	Median	IQR	n	%	Mean±SD	Median	IQR	*p*
Age (year)			75.2±14.2					53.9±16.2			0.002-0.010*
Hemoglobin (g/dL)			12.6±1.5					14.5±2			0.034
Blood glucose (mg/dL)				186.5	111.3-245				108	96.5-125.5	0.037-0.001*
Bulbar symptoms	5	83.3				21	33.3				0.004-0.004*
Mechanical ventilation	6	100				8	12.7				0.000-0.000*
Dysautonomia	3	50				6	9.5				0.006-0.001*
ICU admission	6	100				10	15.9				0.000-0.000*

SD: Standard deviation; IQR: Interquartile range; ICU: Intensive care unit. * Multivariate analysis revealed significant association.

TABLE 5
The correlation of the mRS scores

	r	*p*
Age	0.319	0.022
Symptom duration on admission	–0.367	0.028
Serum albumin level	–0.420	0.004
CSF glucose level	0.348	0.024
Hospital stay duration	0.451	0.001

mRS: Modified Rankin Scale; CSF: Cerebrospinal fluid.

that age was independently associated with mortality during hospitalization. The mean age was 75.2±14.2 years in the mortality group and 53.9±16.2 years in the surviving patients (p=0.002 in univariate analysis and p=0.010 in multivariate analysis). Although the hemoglobin level on admission was found to be lower in the mortality group than that in the surviving patients (12.6±1.5 *vs.* 14.5±2; p=0.034), a multivariate analysis did not show an independent association (p>0.05). The median blood glucose level on admission was 186.5 (111.3-245) mg/dL in the mortality group and 108 (96.5-125.5) mg/dL in the surviving patients, and it was independently associated with in-hospital mortality (p=0.001). However, a diagnosis of diabetes did not influence mortality.

As expected, the presence of bulbar symptoms and dysautonomia were independently associated with death during hospitalization. The mechanical ventilation requirement and ICU admission rates were 100% in the mortality group and 12.7% and 15.9% in the surviving patients (p=0.000 for both comparisons; Table 4). The mortality rate was 43% in mechanically ventilated patients and 37.5% in patients who were admitted to the ICU.

The mRS scores at hospital discharge were positively correlated with age, CSF glucose levels, and the length of hospital stay and negatively correlated with symptom duration on admission and serum albumin levels (Table 5). Moreover, the median mRS score was higher in patients who were admitted to the ICU than in patients without ICU admission (5 [IQR, 5-6] *vs.* 1 [IQR, 1-3]; p=0.000). In other words, unfavorable short-term outcome was associated with older age, shorter symptom duration and lower serum albumin levels on admission, admission to the ICU, and longer hospitalization duration (Table 5).

DISCUSSION

In this study, we found that GBS affected males more than females, and the most common subtype was AIDP in our geographical region; these findings are in accordance with previous findings.[1] Approximately 30% of our patients had axonal subtypes of GBS, including AMAN and AMSAN. The frequency of axonal subtypes of GBS was reported to be between 6% and 17% in European countries.[6] Kiraz et al.[7] evaluated the subtypes of GBS in Van, Türkiye, and reported that 51% had AIDP, 25% had AMAN, and 24% had AMSAN subtypes. Çetiner et al.[8] reported that 30% of patients had axonal variants in Kütahya. Konuşkan et al.[9] reported that 37% of children with GBS had AMAN and 12% had AMSAN in Türkiye. These results suggest that axonal variants may be common in Türkiye.

Although it was stated that approximately 70% of GBS patients reported symptoms of preceding infections,[10] we found that approximately 40% of patients had any antecedent event, including upper respiratory tract infections, gastroenteritis,

trauma, surgery, and vaccinations. However, the possibility of under recording of preceding events in the patients' files cannot be excluded due to the retrospective design of our study. Despite the relationship between axonal subtypes of GBS and Campylobacter jejuni infection is well-known, we did not find any association between the type of the preceding event and GBS subtypes.[11] Again, this finding may be due to a limitation of our study due to its retrospective design.

Up to 30% of patients with GBS develop respiratory insufficiency necessitating mechanical ventilation.[12] Almost one-fifth of our patients needed respiratory support. Respiratory compromise was more common in patients with axonal subtypes, particularly AMSAN. Additionally, patients with AMAN and AMSAN had higher mRS scores at discharge. Axonal forms of GBS were associated with more severe course of the disease, including respiratory insufficiency and worse outcomes.[13] One striking finding of our study was the common occurrence of bulbar symptoms in AMAN patients. The involvement of cranial nerves was reported to be uncommon in patients with AMAN.[13]

The in-hospital mortality rate in our study was 8.7%. Despite advancements in medical care, 3 to 10% of patients with GBS die.[2,14] We found that age, blood glucose level on admission, the presence of bulbar symptoms and dysautonomia, mechanical support requirement, and ICU admission were independently associated with short-term mortality. The deceased patients were more than 20 years older than the surviving patients (75.2±14.2 *vs.* 53.9±16.2 years) in our study. The association of older age with mortality in GBS is well-documented.[14-17] Furthermore, as expected, endotracheal intubation and mechanical ventilation were associated with increased mortality, as in our study.[17,18] In our study, all deceased patients needed mechanical ventilation and were admitted to the ICU. Forty-three percent of the mechanically ventilated patients and 37.5% of the patients who were admitted to the ICU died. The most common causes of death were ventilator-associated pneumonia, sepsis, and autonomic disturbances. The mortality rate in mechanically ventilated GBS patients was reported to be 20 to 38%.[19,20] The high frequency of axonal variants may have contributed to the higher mortality rate in our study.

Thirteen percent of our patients had autonomic disturbances. Most of the symptoms suggesting dysautonomia were blood pressure instability and cardiac arrhythmias. Two-thirds of GBS patients were reported to have autonomic symptoms, including gastrointestinal motility dysregulation and vasomotor dysfunction.[21] Due to the retrospective design of our study, mild and moderate autonomic symptoms may not have been recorded in patient files. We found that autonomic dysfunction and bulbar symptoms were independently associated with in-hospital mortality, as previously reported.[2,21]

Both blood and CSF glucose levels were reported to be associated with short-term prognosis in GBS.[22,23] Blood glucose level on admission was independently associated with mortality, and CSF glucose levels were positively correlated with mRS scores at discharge. Although higher CSF protein levels were associated with a more severe disease course, we did not find any association between CSF protein levels and mortality or mRS scores.[24] However, we found that the serum albumin level was inversely correlated with mRS scores, as Jahan et al.[25] reported.

In our study, the median interval between symptom onset and hospital admission was 7 days. We found that patients who were admitted later had lower mRS scores. This finding indicates that patients with more severe clinical manifestations reached their nadir in a shorter time period and were admitted earlier. A shorter interval between symptom onset and hospital admission was associated with a more severe disease course.[18]

There were several limitations to our study. First, due to its retrospective design, data regarding the antecedent events and autonomic disturbances may have been incomplete. A repeated electrophysiological examination was not performed in all cases; therefore, subtype classification was not possible in some cases who underwent electrophysiological study in the very early days of symptom onset.

In conclusion, our results suggested that GBS axonal subtypes, which may be associated with worse outcomes, could be common in the Sivas province. The short-term unfavorable prognosis was associated with advanced age, axonal subtypes, bulbar symptoms, autonomic disturbances, respiratory compromise necessitating mechanical ventilation, ICU admission, higher blood and CSF glucose levels, and lower serum albumin levels.

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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SUPPLEMENTARY TABLE
Normal values of our electrophysiology laboratory

Nerve	Motor				Sensory		
	DML (msec)	Amplitude (mV)	CV (m/sec)	F latency (msec)	SL (msec)	Amplitude (μV)	CV (m/s)
Median	4.4	4	49	31	3.5	20	50
Ulnar	3.3	6	49	32	3.1	17	50
Radial	3.3	2	49		2.8	20	50
Peroneal	6.5	2	44	56			
Tibial	5.8	4	41	56			
Sural					4.4	6	40
Facial	3.1	1					

DML: Distal motor latency; CV: Conduction velocity; SL: Sensory latency.