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ORIGINAL ARTICLE

Comparison of ulnar, median, and sural sensory nerve conduction studies between demyelinating and axonal forms of Guillain-Barré Syndrome

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Abstract

Background: Axonal GBS and acute inflammatory demyelinating polyradiculoneuropathy (AIDP) are two important subgroups of Guillain-Barré syndrome (GBS). It was aimed to compare sensory nerve conduction studies between AIDP and Axonal GBS patients.

Methods: Patients with clinical and electrodiagnostic features compatible with GBS were included in this retrospective study. The patients were divided into two groups using neurophysiological criteria such as Axonal GBS and AIDP. Medical Research Council (MRC) scores of the patients' muscles, median/ulnar/posterior tibial/peroneal/sural nerve conduction study findings were included in the analyses. Sural sparing pattern was considered as the abnormality of median/ulnar sensory nerve action potential (SNAP) and normal sural nerve SNAP (SS-M/SS-U).

Results: Twelve AIDP and 10 Axonal GBS patients were included in the study. MRC scores were not different between the two groups (p=0.895). SNAPs of the right median and ulnar nerves were smaller in AIDP patients than in Axonal GBS patients (p<0.001, p=0.004). SNAPs of the right and left sural nerves were not different between the two groups (p=0.140, p=0.099). SS-M / SS-U was observed in 1(10%)/1(10%) and 6(50%)/4(33%) of axonal GBS and AIDP patients, respectively (p=0.074 for SS-M, p=0.323 for SS-U). There was a positive correlation between right median / ulnar nerve SNAP amplitudes and sural nerve SNAP amplitudes (p=0.003 r=0.623 / p<0.001 r=0.850). A similar positive correlation was also found in AIDP and Axonal GBS subgroups.

Conclusions: This study indicated that sensory nerve conduction studies cannot be used to differentiate AIDP and Axonal GBS. There may be a relationship between SNAPs of median/ulnar nerves and SNAPs of sural nerves.

Keywords: Acute inflammatory demyelinating polyradiculoneuropathy, Guillain-Barré syndrome, Sensory nerve conduction study, Sural sparing.

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INTRODUCTION

Guillain-Barré syndrome (GBS) is a disease that progresses with weakness and sensory abnormalities that can improve or cause disability (1,2). The diagnosis of GBS can be made by clinical features, laboratory examinations, including cerebrospinal fluid examinations, and neurophysiological tests (1-5). Nerve conduction studies, which are important neurophysiological tests, not only provide the diagnosis but also allow the determination of the type of injury or the prediction of the prognosis (1-7). GBS can be divided into axonal and demyelinating forms using neurophysiological tests or pathology, allowing for a better understanding of the disease's pathophysiology. (1-3,5,8,9). Slowing of nerve conduction velocity, reduction in compound muscle action potential and compound nerve action potential amplitudes, conduction block, abnormal temporal dispersion, and F-wave abnormalities are some of the nerve conduction study findings in GBS (3-5,10). Both sensory and motor nerves may be affected, but the sural nerve may be spared in the early period, and this is known as the sural sparing pattern (10-13). The sural sparing pattern is also one of the important nerve conduction study findings of GBS (10-13). Therefore, sensory nerve conduction studies, including the sural sparing pattern, may play an important role in the differentiation of demyelinating GBS and axonal GBS. Moreover, it may provide important clues regarding the pathophysiology of axonal and demyelinating forms of GBS. In this study, it was aimed to compare the sensory nerve conduction study findings in axonal and demyelinating GBS forms.

MATERIALS AND METHODS

Subjects

GBS patients older than 18 years of age who applied to University of Health Sciences Adana City Training and Research Hospital (ACTRH) Clinical Neurophysiology Laboratory between September 2018 and March 2022 were included in this retrospective study. Patients were divided into two groups as acute inflammatory demyelinating polyradiculoneuropathy (AIDP) and Axonal GBS patients according to the neurophysiological criteria suggested by Rajabally et al (3). These criteria are shown in Table 1. In addition, Axonal GBS patients with sensory nerve conduction study abnormalities in at least two sensory nerves were defined as having acute motor sensory axonal neuropathy (AMSAN) and the others as having acute motor axonal neuropathy (AMAN) (1-4). Patients with the following characteristics were included in the study (1-5): 1) Clinical features compatible with GBS (sensory abnormalities beginning and spreading in the extremities and/or muscle weakness) 2) Elevated protein levels without cell increase in cerebrospinal fluid 3) Patients diagnosed with Axonal GBS or AIDP according to the neurophysiological criteria suggested by Rajabally et al (3). Patients with the following characteristics were excluded from the study: 1) A disease that can cause polyneuropathy, such as diabetes mellitus 2) Neurodegenerative diseases 3) Patients who do not meet the criteria for AIDP or Axonal GBS according to the neurophysiological criteria suggested by Rajabally et al (3). Neurological examination findings, medical research council (MRC) scores, and nerve conduction study findings of the patients were recorded (14). This study was approved by the clinical research Ethics Committee of the Adana City Training and Research Hospital (Date: 21.04.2022, Number: 1902).

Table 1. Neurophysiological criteria for Axonal GBS and AIDP

AIDP	Axonal GBS
Neurophysiological findings consistent with one of the following: 1) One of the following features is present in at least two nerves: *Motor NCV slower than the reference value by more than 30% *Distal motor latency delayed more than 50% compared to the reference value *F-wave latency delayed more than 20% compared to the reference value or more than 50% delay if the distal CMAP amplitude has decreased by more than 50% of the reference limit 2) Absence of F-wave in two nerves + additional parameter in another nerve 3) Proximal CMAP/distal CMAP amplitude ratio less than 0.7 in two nerves (other than tibial nerve) + additional parameter in another nerve	**If the CMAP amplitude is less than 10% of the reference value, there may be a nerve that meets the AIDP criteria. Apart from this, motor nerves should not have demyelinating features. In addition, it must have at least one of the following characteristics:** 1) Distal CMAP amplitude reduction of 80% relative to baseline in at least two nerves 2) Absence of F-waves in the two most nerves (Distal CMAP should be greater than 20% of the reference value) 3) Absence of F-wave in one nerve (Distal CMAP amplitude should be greater than 20% of reference value) or Proximal CMAP/distal CMAP amplitude ratio less than 0.7 in one nerve (other than tibial nerve) + distal CMAP amplitude in another nerve to reference value shrinkage by more than 80% 4) Proximal CMAP/distal CMAP amplitude ratio less than 0.7 in two nerves (other than tibial nerve) Inexcitable form: Absence of distal CMAP in all nerves

AIDP: acute inflammatory demyelinating polyradiculoneuropathy; CMAP: compound muscle action potential; GBS: Guillain-Barré syndrome; NCV: nerve conduction velocity. *: It is based on the criteria suggested by Rajabally et al (3).

Twenty-two GBS patients were included in the study. Prior to GBS complaints, eight of the patients had upper/lower respiratory tract infections, six had gastroenteritis, one had a history of vaccinations (tetanus and rabies vaccinations), and two had a history of surgery. In four of the eight patients with respiratory tract infections, the cause of infection was severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). No cells were found in the cerebrospinal fluid of the patients. Polymerase chain reaction (PCR) testing was performed for SARS-CoV-2 in patients who had coronavirus disease 2019 (COVID-19) prior to GBS. In these patients, fever, dyspnea, high white blood cell count, high c-reactive protein levels, which could indicate acute infection, were not present. Five patients had no event prior to GBS.

Nerve conduction studies

Nerve conduction studies were performed in Cadwell Sierra Summit EMG unit (Cadwell Laboratories, Kennewick, Washington, USA). Nerve conduction studies were performed on at least three extremities. Nerve conduction studies could not be performed on at least three extremities in each patient due to reasons such as edema in the extremities, vascular access, or the patient's inability to tolerate the procedure. Nerve conduction studies were performed if the temperature of the extremities was above 32^0C. Cold extremities were warmed. Previously suggested methods were used for nerve conduction studies (15-17). Surface electrodes were used for both stimulation and recording. Recommended reference values were used as reference values for nerve conduction studies (15-17). Low-high filters for sensory and motor nerve conduction studies were 20Hz-2kHz and 20Hz-10kHz, respectively. In sensory nerve conduction studies, the sensitivity and sweep rate was 10 μV/division and 1 ms/division, respectively. For motor nerve conduction studies, sensitivity and sweep rate were set as 2 mV/division and 5 ms/division, respectively. Both compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) amplitudes were calculated by measuring peak to peak. Sensory nerve conduction studies were performed antidromically. Sensory nerve conduction velocity was calculated using peak latency. CMAPs of median, ulnar, posterior tibial and peroneal nerves were obtained from abductor pollicis brevis, abductor digiti quinti, abductor hallucis, and extensor digitorum brevis muscles, respectively. In distal motor nerve conduction studies, the distance between the recording electrode and the stimulation point was 5 cm for the median and ulnar nerves, 10 cm for the posterior tibial nerve, and 8 cm for the peroneal nerve. Among the F-waves obtained after 10 stimulations, the minimum F-wave latency was included in the analyses. Normal sural nerve and abnormal median nerve SNAP were defined as sural sparing-median nerve abnormality (SS-M), while present sural nerve and abnormal ulnar nerve SNAP were defined as sural sparing-ulnar nerve abnormality (SS-U).

Statistical analysis

Statistical Package for the Social Sciences (SPSS IBM Corp; Armonk, NY, USA) 22.0 was used for statistical analysis. Numerical values were expressed as mean standard deviation (SD), median, and categorical variables as numbers and percentages. While the Mann-Whitney U test was used for the comparison between the groups given numerically, Pearson's Chi-squared test and Fisher's exact test were performed to compare the categorical variables between the groups. Spearman correlation test was applied for correlation analysis. A p value of <0.05 was considered statistically significant.

RESULTS

Twenty-two patients were included in the study. The mean age of the patients was 56.1±19.7 (min-max 18-88) years. Upper extremity, lower extremity and total MRC scores were 24.2±7.4 (min-max 4-30), 18.5±6.5 (min-max 4-26), 42.6±12.7 (12-56), respectively. The time interval between the onset of the complaints and the time of the nerve conduction study was 15.7±10.7 (min-max 5-45) days. There were 12 AIDP and 10 Axonal GBS patients. Clinical features of AIDP and Axonal patients are shown in Table 2. Three of the Axonal GBS patients met the diagnostic criteria of AMSAN and these patients had sensory abnormalities on neurological examination. Two AMAN patients had sensory complaints without any sensory abnormalities in the neurological examination. Two of the patients with a history of COVID-19 had AIDP and two had Axonal GBS.

Table 2. Clinical features among AIDP and Axonal GBS patients

Clinical feature	Axonal GBS (n=10)	AIDP (n=12)	p value
Age (years) mean ± SD (median)	56.3±24.4 (62.5)	55.9±15.9 (55.5)	0.668
Gender (male)	6	7	1.000
Duration of the symptoms (days)	14.7±9.3 (18.0)	16.6±12.0 (12.5)	0.842
MRC score of the upper extremities	24.0±8.3 (26.0)	24.3±6.9 (27.0)	0.866
MRC score of the lower extremities	18.4±5.2 (18.0)	18.5±7.7 (22.0)	0.688
Total MRC score	42.4±12.4	42.8±13.4	0.895

AIDP: acute inflammatory demyelinating polyradiculoneuropathy; GBS: Guillain-Barré syndrome; MRC: medical research council.

Motor and sensory nerve conduction studies were performed on 117 and 81 nerves, respectively. Motor nerve conduction study findings of 29 median nerves (21 right side, eight left side), 29 ulnar nerves (21 right side, eight left side), 36 posterior tibial nerves (22 right side, 14 left side) and 23 peroneal nerves (18 right side, 5 left side) were included in the analyses. The grouping of motor nerve conduction study findings according to axonal and demyelinating features is shown in Table 3. Sensory nerve conduction studies were performed on 24 median nerves (21 right side, three left side), 25 ulnar nerves (21 right side, four left side) and 32 sural nerves. Sensory nerve conduction study findings among AIDP and Axonal GBS patients are shown in Table 4. Right and sural nerve SNAP amplitude mean (min-max) values in all GBS patients were 12.0±11.9 (0-39.1) μV and 8.9±9.3 (0-23.8) μV, respectively. The sural nerve SNAP amplitude was either absent or above 5 μV. Figure 1 shows the comparison of number of patients with median, ulnar, sural SNAP amplitude abnormalities between AIDP and Axonal GBS patients. The number of patients with at least one abnormality in the ulnar, median, and sural nerve SNAPs was 12 (54.5%), 14 (63.6%), and 7 (31.8%), respectively. The number of patients with at least one ulnar, median, and sural nerve SNAP abnormality in the axonal GBS group was 3 (30%), 3 (30%), and 2 (20%), respectively, and the number of patients with at least one ulnar, median, and sural nerve abnormality in the AIDP group was 9 (75%), 11 (92%), and 5 (42%), respectively (p=0.084 for ulnar nerve SNAP, p=0.006 for median nerve SNAP, p=0.381 for sural nerve SNAP). SS-M and SS-U were found in 7 (31.8%) and 5 (22.7%) patients, respectively. SS-M was present in one (10%) Axonal GBS patient and six (50%) AIDP patients (p=0.074). SS-U was found in one (10%) Axonal GBS patient and four AIDP (33%) patients (p=0.323). The correlation between right sural nerve SNAP/NCV and clinical findings/median-ulnar nerve conduction study findings is shown in Table 5.

Table 3. Motor nerve conduction study findings according to axonal and demyelinating characteristics

	Number of nerves			
Motor nerve	Axonal	Demyelinating	Mild slowing or Mild reduction of CMAP amplitude	Normal
Right / Left Median nerve	6 / 1	9 / 3	4 / 3	2 / 1
Right / Left Ulnar nerve	11 / 3	7 / 3	2 / 0	1 / 2
Right / Left Posterior nerve	13 / 8	9 / 6	0 / 0	0 / 0
Right / Left Peroneal nerve	10 / 3	7 / 2	0 / 0	1 / 0

CMAP: compound action potential.

Table 4. Comparison of sensory nerve conduction studies between Axonal GBS and AIDP patients

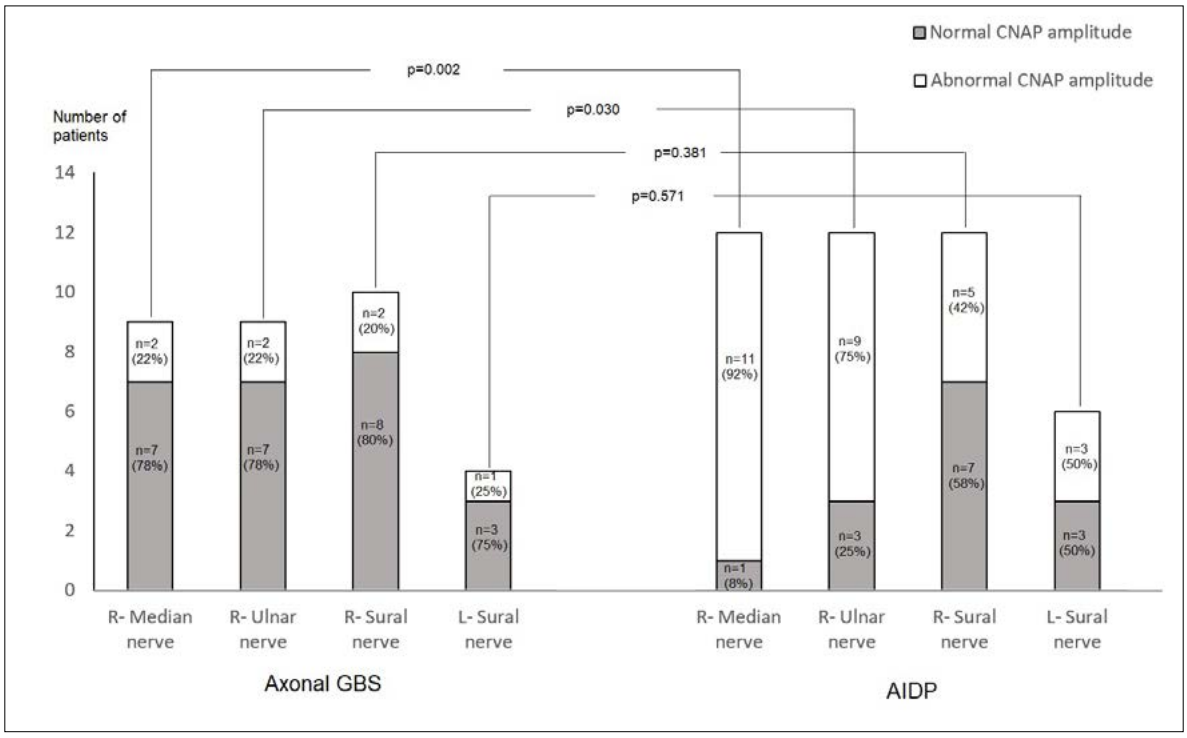
Sensory nerve conduction study	Axonal GBS Mean ± SD (median) (number)	AIDP Mean ± SD (median) (number)	p value
Median nerve			
R- SNAP amplitude (μV)	28.0±15.4 (36.8) (n=9)	2.6±5.8 (0) (n=12)	**<0.001**
R- NCV (m/s)	42.9±6.3 (45) (n=9)	36.7±12.7 (43) (n=3)	0.350
L- SNAP amplitude (μV)	7.7±6.7 (10.8) (n=3)	*	
L- NCV (m/s)	34.0±5.7 (34.0) (n=2)	*	
Ulnar nerve			
R- SNAP amplitude (μV)	27.6±17.7 (31.6) (n=9)	7.2±10.5 (0) (n=12)	**0.004**
R- NCV (m/s)	40.4±6.7 (39.0) (n=9)	37.2±8.3 (38.0) (n=5)	0.502
L- SNAP amplitude (μV)	9.9±6.9 (12.2) (n=4)	**	
L- NCV (m/s)	35.0±4.4 (37.0) (n=3)	**	
Sural nerve			
R- SNAP amplitude (μV)	16.6±13.2 (18.4) (n=10)	8.2±9.8 (6.9) (n=12)	0.140
R- NCV (m/s)	39.3±2.6 (39.5) (n=8)	40.1±4.6 (42.0) (n=7)	0.484
L- SNAP amplitude (μV)	14.9±10.3 (17.9) (n=4)	4.9±6.7 (3.1) (n=6)	0.099
L- NCV (m/s)	40.0±2.0 (40.0) (n=3)	37.0±1.7 (38.0) (n=3)	0.105

AIDP: acute inflammatory demyelinating polyradiculoneuropathy; SNAP: sensory nerve action potential; GBS: Guillain-Barré syndrome; NCV: nerve conduction potential. *: Nerve conduction studies were performed in five AIDP patients and SNAPs could not be achieved from these patients. **: Nerve conduction studies were performed in three AIDP patients and SNAPs could not be achieved from these patients.

Table 5. Correlation between clinical findings and sensory nerve conduction features, and right sural nerve NCV/SNAP

Clinical / Nerve conduction study feature	Right Sural SNAP amplitude (μV)	Right sural sensory NCV (m/s)
GBS patients		
MRC score of upper extremities	p=0.170, r=-0.303 (n=22)	p=0.093, r=0.449 (n=15)
MRC score of lower extremities	p=0.813, r=-0.054 (n=22)	p=0.258, r=0.312 (n=15)
Total MRC score	p=0.568, r=-0.129 (n=22)	p=0.090, r=0.453 (n=15)
Right median SNAP amplitude	p=**0.003**, r=0.613 (n=21)	p=0.677, r=-0.117 (n=15)
Right median sensory NCV	p=0.118, r=0.475 (n=12)	p=0.862, r=0.063 (n=10)
Right ulnar SNAP amplitude	p< **0.001**, r=0.850 (n=21)	p=0.954, r=-0.016 (n=15)
Right ulnar sensory NCV	p=0.065, r=0.506 (n=14)	p=**0.010**, r=0.682 (n=13)
AIDP patients		
MRC score of upper extremities	p=0.436, r=-0.227 (n=12)	p=0.376, r=0.364 (n=8)
MRC score of lower extremities	p=0.825, r=-0.065 (n=12)	p=0.583, r=0.230 (n=8)
Total MRC score	p=0.809, r=-0.071 (n=12)	p=0.513, r=-0.273 (n=8)
Right median SNAP amplitude	p=0.573, r=0.173 (n=13)	p=0.601, r=-0.220 (n=8)
Right median sensory NCV	p=0.684, r=0.316 (n=4)	p=1.000, r=0.000 (n=3)
Right ulnar SNAP amplitude	p=**0.005**, r=0.731 (n=13)	p=0.955, r=-0.024 (n=8)
Right ulnar sensory NCV	p=0.623, r=0.257 (n=6)	p=**0.024**, r=0.870 (n=8)
Axonal GBS patients		
MRC score of upper extremities	p=0.250, r=-0.402 (n=10)	p=0.129, r=0.584 (n=10)
MRC score of lower extremities	p=0.580, r=-0.200 (n=10)	p=0.444, r=0.317 (n=10)
Total MRC score	p=0.496, r=-0.245 (n=10)	p=0.130, r=0.582 (n=10)
Right median SNAP amplitude	p=**0.037**, r=0.697 (n=9)	p=0.342, r=0.388 (n=8)
Right median sensory NCV	p=0.262, r=0.419 (n=9)	p=0.861, r=0.075 (n=8)
Right ulnar SNAP amplitude	P=**0.004**, r=0.849 (n=9)	p=0.393, r=-0.352 (n=8)
Right ulnar sensory NCV	p=0.079, r=0.613 (n=9)	p=**0.384**, r=0.358 (n=8)

AIDP: acute inflammatory demyelinating polyradiculoneuropathy; SNAP: sensory nerve action potential; GBS: Guillain-Barré syndrome; MRC: medical research council; NCV: nerve conduction velocity.



AIDP: acute inflammatory demyelinating polyradiculoneuropathy; SNAP: sensory nerve action potential; GBS: Guillain-Barré syndrome.

Figure 1. Comparison of number of patients with median, ulnar, sural SNAP amplitude abnormalities between AIDP and Axonal GBS patients

DISCUSSION

GBS progresses with flaccid paralysis, which can lead to disability and even threaten life (1,2). Therefore, it is important to diagnose this important disease and treat it appropriately. Nerve conduction studies play a key role in the diagnosis of GBS. Nerve conduction studies are important not only for diagnosis but also for distinguishing between axonal and demyelinating forms of GBS (3-5). Although the separation of GBS into axonal and demyelinating forms may not be important for treatment, it will undoubtedly contribute to the understanding of the pathophysiology of GBS (3-5,8,9,18). In this current study, we divided GBS patients into axonal GBS and AIDP according to the proposed criteria of motor nerve conduction study. Therefore, we were able to compare the sensory nerve conduction study findings between these two forms. This was the reason why we did not use the proposed criteria, which included sensory nerve conduction studies.

Abnormalities of median and ulnar nerve SNAPs were more prominent in AIDP, which is the demyelinating form, and we concluded that the sural nerve conduction study findings were not different from each other in both forms. In addition, previous study showed that the absent median nerve and the present sural nerve pattern could not be used to differentiate AIDP from axonal GBS (13). The positive correlation between sural nerve SNAP amplitude / NCV and median / ulnar nerve SNAP amplitude / NCV in patients with AIDP and Axonal GBS may mean that other SNAPs are affected when sural SNAP is affected in some Axonal GBS, like AIDP. These findings suggest that there may be sensory abnormalities in both forms of GBS. On the contrary, there was a study suggesting that sural sparing would be useful in distinguishing between axonal and demyelinating forms of GBS (11). These findings indicate that more studies are needed on this subject. However, it should be noted that the time interval between the nerve conduction study and the onset of the complaints was different in our study,

and that nerve conduction study was not performed in every patient in the first days, and this is a weakness of our study. Similar to the median nerve, we found that the ulnar nerve SNAP was more significantly affected in AIDP than in axonal GBS. Considering all patients, the existence of an association between median/ulnar SNAP amplitude and sural nerve SNAP indicates a widespread involvement in GBS. However, sural sparing is a known condition in the acute phase of GBS (10-13,19).

In this study, sural sparing was present in approximately 30% of GBS patients and 50% of AIDP patients. These rates were similar to those in previous studies (10-13,19). Our findings showed that sural sparing or normal sural nerve SNAP could be in either form. In addition, median/ulnar nerve conduction studies were more prominently affected in AIDP compared to Axonal GBS. Although it is difficult to explain this situation, these findings can be explained by the reversible conduction failure seen in axonal GBS (4,5,18,20,21). Improvement of conduction failure in motor and sensory nerves may result in improvement of nerve conduction studies. Another explanation might be that many of the patients with Axonal GBS have acute motor axonal neuropathy. Similarly, the high rate of Axonal GBS patients in this current study can be explained by reversible conduction failure. In some patients, motor conduction block in AIDP and reversible conduction failure in Axonal GBS may have been misinterpreted (3-5,20), resulting in a high proportion of Axonal GBS patients. However, it should be noted that we used the recommended GBS diagnostic criteria.

For some, the axonal form of GBS may have a worse prognosis, but for others it may not be (6,7). According to some, recovery of axonal GBS is delayed or some AIDP patients may be misdiagnosed with Axonal GBS (4-7,20,21). For these reasons, the prognosis of axonal GBS may be misinterpreted. In this study, the patients were not followed up, but they had examination findings in the acute-subacute period and MRC scores were not different between axonal GBS and AIDP patients. Prospective studies involving the follow-up of axonal and demyelinating GBS patients are needed to elucidate this situation.

An important result found in our study was that four of the patients had COVID-19 prior to GBS. Cases of GBS associated with COVID-19 have been reported (22-24). Although the exact cause is unknown, GBS associated with COVID-19 may be associated with excessive cytokine release or immune reactions (22-24). Although cases of GBS associated with acute COVID-19 have also been reported (22), acute COVID-19 infection was not present in the cases in this study. PCR testing for SARS-CoV-2 was not performed in the cerebrospinal fluid but no cells were present in the cerebrospinal fluid. The findings in this current study may mean that GBS does not develop during acute infection and that GBS is not due to excessive cytokine release, and that previous COVID-19 triggers immune reactions. However, more studies are needed for the accuracy of these results.

This study had several limitations. Since it is a retrospective study, although the time interval between the time the nerve conduction study was performed and the onset of complaints did not differ between axonal GBS and AIDP patients, this interval was different between patients. Other limitations were the lack of follow-up of the patients and the low number of patients. Again, due to the small number of patients, AMAN and AMSAN patients could not be included in the analysis separately. The fact that this distinction was not made may have affected our results. We think that future studies involving these subgroups will be useful. One of the limitations was that anti-ganglioside antibodies were not available. Finally, not performing nerve conduction studies in all four extremities on every patient may be a limitation. However, it should be noted that some patients have a catheter or edema.

In conclusion, this study showed that sural nerve SNAP and sural sparing were not different between AIDP and Axonal GBS, but median/ulnar nerve SNAPs were different between AIDP and Axonal GBS. In addition, it was concluded that there is a positive correlation between median/ulnar nerve SNAP amplitudes and sural nerve SNAP amplitudes in GBS patients.

Declarations

The authors have no conflicts of interest to declare. The authors declared that this study has received no financial support.

This study was approved by the clinical research Ethics Committee of the Adana City Training and Research Hospital (Date: 21.04.2022, Number: 1902).

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