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Diagnostic Value of Sensory Nerve Conduction Studies in Acute Inflammatory Demyelinating Polyradiculoneuropathy

Akut Enflamatuvar Demiyelinizan Poliradikülonöropatide Duyusal Sinir İletim Çalışmalarının Tanısal Değeri

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Abstract

Objective: The diagnosis of acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is based mainly on motor nerve conduction studies (NCSs), which may lead to inconclusive results, especially early in the course of the disease. The present study aimed to evaluate sensory NCSs as an additional tool to aid the identification of this illness.

Materials and Methods: We retrospectively evaluated the sensory and motor nerve conduction findings of patients with AIDP and compared them with laboratory controls. The sensitivity and specificity of the NCS parameters and sural sparing pattern (SSP) were also assessed. The NCS patterns were categorized as normal, abnormal, and no response. The association of sensory nerve action potential (SNAP) amplitude patterns and effect of the timing of the electrodiagnostic examination on sensory and motor NCS patterns were analyzed.

Results: The most sensitive sensory nerve conduction findings were reduced ulnar (79.3%) and median (75.9%) SNAP amplitudes, which were more sensitive than the compound muscle action potential amplitudes and forearm motor nerve conduction velocities of these nerves. Employing ulnar SNAP for SSP identification was more useful than using the median SNAP. The timing of the electrodiagnostic studies did not affect the patterns of the sensory nerve conduction parameters.

Conclusion: The reduction in the median and ulnar SNAP amplitudes along with SSP, with the ulnar SNAP amplitude used for comparison, is beneficial for diagnosing AIDP regardless of the timing of the electrodiagnostic examination.

Keywords: Acute inflammatory demyelinating polyradiculoneuropathy, electrodiagnosis, Guillain–Barré syndrome, nerve conduction study

Öz

Amaç: Akut enflamatuvar demiyelinizan poliradikülonöropatinin (AİDP) elektrofizyolojik tanısı esas olarak motor sinir iletim çalışmalarına (SİÇ) dayanmaktadır ve özellikle hastalığın erken dönemlerinde kesin sonuç için yetersiz kalabilmektedir. Bu çalışmanın amacı duyusal SİÇ'lerinin AİDP tanısına katkısını değerlendirmektir.

Gereç ve Yöntem: AİDP hastalarının duyusal ve motor sinir iletim bulgularını retrospektif olarak değerlendirildi ve laboratuvar kontrolleri ile karşılaştırıldı. SİÇ parametrelerinin ve sural korunma paterninin (SSP) duyarlılığı ve özgüllüğü de değerlendirildi. SİÇ paternleri normal, anormal ve yanıtsız olarak kategorize edildi. Duyusal sinir aksiyon potansiyeli (DSAP) amplitüd paternlerinin birbirleri ile ilişkisi ve elektrodiagnostik inceleme zamanlamasının duyusal ve motor SİÇ paternleri üzerindeki etkisi analiz edildi.

Bulgular: En duyarlı duyusal sinir iletim bulguları, azalmış ulnar (%79,3) ve median (%75,9) DSAP amplitüdleri olup, bu sinirlerin birleşik kas aksiyon potansiyeli amplitüdleri ve ön kol motor sinir iletim hızlarından daha hassastır. SSP tanımlaması için ulnar DSAP kullanımı, median DSAP kullanımından daha anlamlıdır. Elektrodiagnostik çalışmaların zamanlaması, duyusal sinir iletim parametrelerinin paternlerini etkilememektedir.

Sonuç: Median ve ulnar DSAP amplitüdlerinde azalma ile birlikte karşılaştırma için ulnar DSAP amplitüdünün kullanıldığı SSP, AİDP tanısına elektrodiagnostik inceleme zamanından etkilenmeksizin katkı sağlayabilir.

Anahtar Kelimeler: Akut enflamatuvar demyelinizan poliradikülopati, elektrodiyagnoz, Guillain–Barré sendromu, sinir iletim çalışması

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Introduction

Guillain–Barré syndrome (GBS) is one of the leading causes of acute flaccid paralysis and is the most common cause of acute acquired inflammatory polyneuropathy (1). Varying in its geographical distribution, the most common subtype of GBS is acute inflammatory demyelinating polyradiculoneuropathy (AIDP), which displays demyelinating features in electrodiagnostic studies (2). Therefore, electrophysiological evaluation is crucial for classifying subtypes and prognosis estimation. Subtype classification criteria, which have been used until recently, are based solely on motor nerve conduction findings (3,4,5,6). However, several studies have reported that sensory nerve conduction abnormalities not included in the traditional electrodiagnostic criteria are frequently observed in AIDP and other subtypes (7,8). The sural sparing pattern (SSP), which indicates relatively preserved sural sensory nerve action potential (SNAP) amplitude compared with the ulnar nerve, has recently been introduced to the diagnostic criteria of AIDP (9). Further sensory nerve conduction studies (NCSs) have the potential to be of use in AIDP diagnosis. We therefore undertook this retrospective study to characterize sensory abnormalities that can contribute to the traditional criteria sets.

Materials and Methods

Patients

Files of patients with a monophasic, acute bilateral flaccid paralysis evaluated in the electromyography (EMG) laboratory between 2006 and 2013 and with diagnostic certainty consistent with the Brighton criteria for GBS (10) were reviewed. Patients had to fulfill the Ho et al. (4) and Hadden et al. (5) criteria for AIDP to be included in the study. Other subtypes, such as acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy, and Miller Fisher syndrome, were excluded. Neurological examination findings were recorded at the time of the EMG examination. Mean values of the deltoid, biceps, wrist dorsiflexors, iliopsoas, and tibialis anterior muscle strength were graded using the Medical Research Council Scale. Deep tendon reflexes were categorized as absent, hypoactive, and normoactive. Cerebrospinal fluid examination findings were obtained. The study was approved by the Ethics Committee of Gazi University (protocol number: 2013-163, date: 2023.10.21). As the study was conducted retrospectively, consent forms were not obtained from the patients.

Nerve Conduction Studies

Motor and sensory NCSs were performed using Neuropack Σ MEB-5504K (Nihon Kohden, Tokyo, Japan), Counterpoint (Dantec, Skovlunde, Denmark), and Keypoint (Medtronic, Skovlunde, Denmark) machines in accordance with previously described methods (11). Stimulation was delivered through bipolar surface electrodes with a pulse duration of 100 μs. Recordings were made using silver/silver chloride surface electrodes. Motor NCSs included distal and F-wave latencies in addition to nerve conduction velocities (NCVs) of the forearm segments of the median and ulnar nerves, as well as knee–ankle segments of the peroneal and tibial nerves. Compound muscle action potential (CMAP) amplitudes were measured from peak to peak. Temporal dispersion was defined as a prolongation of the negative peak duration of the proximal CMAP by more than 30% compared with the distal CMAP (9). Sensory NCSs of the finger–wrist segments of the median and ulnar nerves were performed orthodromically, and the sural nerve was evaluated antidromically. Latencies were measured to the negative peak of the SNAP, and SNAP amplitudes were measured from peak to peak. The results obtained from only one upper and lower limb on the same side were considered. When both sides were examined, the side with more prominent nerve conduction abnormalities was chosen. The NCS results of the patients were compared with laboratory controls. The SSP was assessed by employing the two different methods described by Uncini et al. (9) and Umapathi et al. (12). The NCS patterns were categorized as normal, abnormal, or no response.

Statistical Analysis

The sensitivity and specificity of the NCS parameters were calculated as follows: sensitivity = true positivity/true positivity + false negativity; specificity = true negativity/true negativity + false positivity. The normality of distribution was verified using the Shapiro–Wilk test. Associations between SNAP patterns were evaluated using the Kappa coefficient to assess their compatibility. A Mann–Whitney U test analyzed the involvement patterns of sensory NCSs, considering the timing of the electrodiagnostic examination. The impact of the timing of the electrodiagnostic examination on the motor and sensory NCS patterns was analyzed through univariate binary logistic regression analysis. The statistical analysis was performed using IBM SPSS V25 (Chicago, IL, USA) software.

Results

Participants

Forty-eight patients with GBS were identified. Seven patients had undergone a control electrodiagnostic examination, and after excluding other subtypes, a final group consisting of 29 patients with AIDP was established (Figure 1). Diagnostic certainty was level 1 in 19 patients (65.52%), level 2 in 8 (27.59%), and level 3 in 2 (6.90%), according to the Brighton criteria. There were 17 men and 12 women in the AIDP group, with a mean age (standard deviation) of 53.80 (17.03). Presentation findings included ascending paralysis, speech and swallowing difficulties, sensory symptoms, facial weakness, and shortness of breath in 18 patients (62.07%), 3 patients (10.30%), 4 patients (13.80%), 3 patients (10.30%), and 1 patient (3.40%), respectively. Antecedent events were notable for upper respiratory system infection in 9 patients (31%) and gastroenteritis in 2 patients (6.90%), including a patient with both conditions. One patient (3.40%) had prior immunization and 1 had otitis. The remaining 17 patients (58.60%) reported no antecedent events.

The muscle strength evaluation revealed mean values of 3.50 for the deltoid, 3.80 for the biceps brachii, 3.30 for the wrist dorsiflexors, 3 for the iliopsoas, and 2.50 for the tibialis anterior. Deep tendon reflexes were absent in 26 patients (89.70%) and globally hypoactive in 3 (10.30%).

Cerebrospinal fluid protein elevation (>40 mg/dl) was determined in 21 patients (72.40%). The protein level was normal in 1 (3.50%). Seven patients refused a spinal tap.

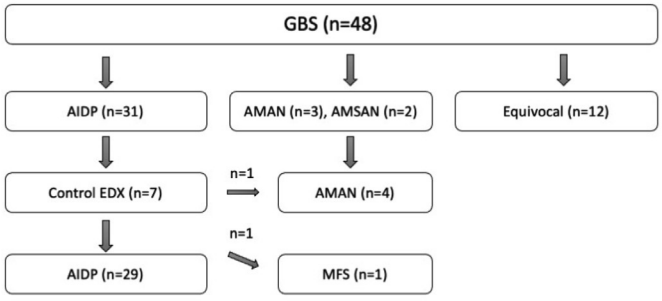

Figure 1. Selection of patients with acute inflammatory demyelinating polyradiculoneuropathy

AMAN: Acute motor axonal neuropathy, AMSAN: Acute motor and sensory axonal neuropathy, EDX: Electrodiagnostic examination, GBS: Guillain–Barré syndrome, MFS: Miller Fisher syndrome, n: Number

Nerve Conduction Studies

The abnormal findings from the motor and sensory NCSs are presented in Table 1. The variables with the highest sensitivity were tibial F-wave latency, peroneal F-wave latency, median distal latency, and peroneal CMAP amplitude, in descending order. The ulnar SNAP amplitude had the same sensitivity as the peroneal distal latency. Similarly, the median SNAP amplitude showed the same sensitivity as the ulnar F-wave latency. Conduction block was identified in at least one nerve segment in 11 patients (37.90%). Likewise, temporal dispersion was observed in the same number of patients. Six patients (20.69%) displayed both phenomena.

The analysis of the compatibility of the median and ulnar SNAP patterns with the sural SNAP pattern revealed consistent median and sural and, to a lesser degree, ulnar and sural SNAP patterns (Table 2).

Table 1. Frequencies of patterns (%) and sensitivity and specificity of nerve conduction study parameters in patients with acute inflammatory demyelinating polyradiculoneuropathy

	Normal	Abnormal	NR	Sensitivity	Specificity
Median					
DL	4 (14.3)	24 (85.7)	0 (0)	85.7	92
MNCV	9 (32.1)	16 (57.1)	3 (10.7)	67.9	96
CMAP Amp	13 (46.4)	15 (53.6)	0 (0)	53.6	96
mFL	6 (21.4)	16 (57.1)	6 (21.4)	78.6	92
SNCV	8 (27.6)	1 (3.4)	20 (68.9)	72.4	92
SNAP Amp	7 (24.1)	2 (6.9)	20 (68.9)	75.9	100
Ulnar					
DL	15 (51.7)	13 (44.8)	1 (3.4)	48.3	100
MNCV	17 (58.6)	10 (34.5)	2 (6.9)	41.4	100
CMAP Amp	14 (48.3)	14 (48.3)	1 (3.4)	51.7	100
mFL	7 (24.1)	12 (41.4)	10 (34.5)	75.9	96
SNCV	9 (32.1)	2 (7.1)	17 (60.7)	65.5	100
SNAP Amp	5 (17.9)	6 (21.4)	17 (60.7)	79.3	100
Peroneal					
DL	6 (20.7)	19 (65.5)	4 (13.8)	79.3	100
MNCV	8 (27.6)	17 (58.6)	4 (13.8)	72.4	100
CMAP Amp	5 (17.2)	20 (68.9)	4 (13.8)	82.8	100
mFL	2 (6.9)	7 (24.1)	20 (68.9)	93.1	96
Tibial					
DL	10 (34.5)	16 (55.2)	3 (10.3)	65.5	92
MNCV	9 (31.0)	17 (58.6)	3 (10.3)	69.0	96
CMAP Amp	9 (31.0)	17 (58.6)	3 (10.3)	69.0	100
mFL	1 (3.4)	16 (55.2)	12 (41.4)	96.6	96
Sural					
SNCV	13 (44.8)	0 (0)	16 (55.2)	55.2	100
SNAP Amp	12 (41.4)	1 (3.4)	16 (55.2)	58.6	100

CMAP Amp: Compound muscle action potential amplitude, DL: Distal latency; mFL: Minimal F-wave latency, MNCV: Motor nerve conduction velocity of the forearm segments of the median and ulnar nerves, knee-ankle segments of the peroneal and tibial nerves, NR: No response, SNAP Amp: Sensory nerve action potential amplitude, SNCV: Sensory nerve conduction velocity of the finger-wrist segments of the median and ulnar nerves, sura-ankle segment of the sural nerve

The SSP was calculated using median and ulnar SNAP amplitudes separately. Although median SNAP calculations revealed an SSP in 6 patients (20.70%), the ulnar SNAP calculations resulted in an SSP in 8 patients (27.60%). No patients exhibited an SSP with median SNAP calculations alone without an SSP with ulnar SNAP, which made the Umapathi formula no more valuable than using the ulnar SNAP alone for SSP calculations. Although the sural SNAP was unobtainable in 16 patients (55.20%), an SSP was present in 8 (61.54%) of the 13 patients with an obtainable sural SNAP. Only 1 patient (7.69%) with an obtainable sural SNAP had amplitude reduction.

Electrodiagnostic studies were performed 12 ± 9.51 (2–40) days after the onset of symptoms. The SNAP patterns of the median, ulnar, and sural nerves were evaluated, taking the timing of the examination into account. No significant difference existed between the timing of the electrodiagnostic studies and SNAP involvement patterns (Table 3). Although timing of the electrodiagnostic studies was not a risk factor in the sensory NCSs, an independent risk factor was identified in terms of the peroneal knee–ankle NCV (Table 4).

Discussion

This study revealed that the sensory nerve conduction parameters had a high sensitivity in diagnosing AIDP. The sensory NCVs of the median and ulnar nerves were more sensitive than the motor NCV of the forearm segments. Moreover, the median and ulnar SNAP amplitudes were more sensitive than the CMAP amplitudes of the same nerves. They also had comparable sensitivities with the peroneal and tibial motor NCVs and CMAP amplitudes. More specific electrophysiological markers diagnostic of AIDP have been reported previously. One study indicated that an ulnar ratio ≥0.78 obtained by dividing the palmar cutaneous by the dorsal branch SNAP amplitude of the ulnar nerve could exclude AIDP with high sensitivity and specificity (13) because the dorsal branch is relatively spared in this disease. Another study by the same group suggested that a low-amplitude medial plantar response could be diagnostically useful, especially early in the course of the disease, when the motor nerve conduction findings may be inconclusive (14). Sensory ratio (sural plus radial SNAPs/median plus ulnar SNAPs) is reportedly another marker in the diagnosis of AIDP (15). However, sensory nerve conduction abnormalities of the routinely studied median and ulnar nerves can still provide further evidence for AIDP in the appropriate clinical context.

Comparison of the sural SNAP with the median, ulnar, or both SNAPs has been recommended to identify SSP (9,12).

Table 2. Association of sensory nerve action potential patterns

	Sural		Kappa value	*P*
	Normal	NR + abnormal		
Median				
Normal	7	0	0.621	<0.001
NR + abnormal	5	17		
Ulnar				
Normal	5	1	0.386	0.019
NR + abnormal	7	16		

NR: No response

Table 3. Timing of the electrodiagnostic examination in relation to the involvement patterns of the sensory nerve action potential amplitudes (days, median, range in parentheses)

SNAP Amp	Normal	NR + abnormal	*P*
Median	7 (7-15)	9 (2-40)	0.980
Ulnar	7 (7-15)	10 (2-40)	0.854
Sural	8.5 (5-26)	8 (2-40)	0. 913

NR: No response, SNAP Amp: Sensory nerve action potential amplitude

Table 4. Effect of the timing of the electrodiagnostic study on abnormalities in nerve conduction variables

	OR (95% CI)	*P*	Accuracy
Median			
DL	1.180 (0.892-1.562)	0.246	85.7
MNCV	1.077 (0.951-1.219)	0.242	67.9
CMAP Amp	0.939 (0.854-1.032)	0.193	64.3
mFL	1.085 (0.928-1.268)	0.308	78.6
SNCV	1.074 (0.942-1.224)	0.288	72.4
SNAP Amp	1.048 (0.929-1.183)	0.447	75.9
Ulnar			
DL	0.995 (0.916-1.080)	0.900	51.7
MNCV	1.066 (0.972-1.169)	0.174	65.5
CMAP Amp	1.009 (0.929-1.095)	0.835	62.1
mFL	1.048 (0.929-1.183)	0.447	75.9
SNCV	1.068 (0.952-1.198)	0.260	65.5
SNAP Amp	1.052 (0.921-1.202)	0.453	79.3
Peroneal			
DL	0.992 (0.900-1.095)	0.877	79.3
MNCV	0.881 (0.785-0.988)	**0.030**	79.3
CMAP Amp	1.099 (0.911-1.325)	0.324	82.8
mFL	1.077 (0.829-1.398)	0.578	93.1
Tibial			
DL	1.192 (0.981-1.450)	0.078	69.0
MNCV	0.981 (0.900-1.068)	0.654	69.0
CMAP Amp	1.102 (0.955-1.272)	0.184	65.5
mFL	1.158 (0.686-1.955)	0.583	96.6
Sural			
SNCV	1.024 (0.940-1.116)	0.589	51.7
SNAP Amp	1.031 (0.942-1.128)	0.504	58.6

CMAP Amp: Compound muscle action potential amplitude, DL: Distal latency, mFL: Minimal F-wave latency, MNCV: Motor nerve conduction velocity of the forearm segments of the median and ulnar nerves, knee-ankle segments of the peroneal and tibial nerves, NR: No response; SNAP Amp: Sensory nerve action potential amplitude, SNCV: Sensory nerve conduction velocity of the finger-wrist segments of the median and ulnar nerves, sura-ankle segment of the sural nerve, OR: Odds ratio, CI: Confidence interval

The present study demonstrated that 61.54% of patients with an obtainable sural response exhibit the SSP pattern. It is also important to note that using the ulnar SNAP amplitude to identify SSP is a more sensitive method than employing the median SNAP. There is no need to include the median nerve in the calculations because the ulnar SNAP is invariably affected in that type of situation, which is consistent with the literature (9,16). The compatibility of the pattern of involvement of the sural nerve with the median nerve (Table 2) is more prominent, supporting this finding. Derksen et al. (16) demonstrated that although the specificity of the median and ulnar nerves was similar in the identification of SSP (0.91 vs. 0.93), the sensitivity of the ulnar nerve was higher compared with the median nerve (0.35 vs. 0.26). We observed that the sural nerve involvement pattern in AIDP is either an absent SNAP or, when recordable, completely normal, indicating an "all or nothing" phenomenon.

Electrodiagnostic studies interpreted using the classic criteria may be equivocal regarding subtype classification in the early stages of GBS (17,18,19), as the subtype may change in repeated studies. Therefore, a serial study approach is recommended for subtype discrimination (9,20,21,22,23,24,25). Motor NCSs are normal early in the course of the disease and may remain unaffected even longer in variants such as Miller Fisher syndrome (26,27,28). However, SNAP abnormalities are common in GBS (18,29,30). A recent study reported that in a group composed of 36 patients with GBS, in which 11 were diagnosed with AMAN, 40% demonstrated a reduced SNAP amplitude, whereas SSP was reported only in 25% of patients in the first week of the disease (31). Additionally, employing a binary logistic regression analysis, we revealed that time had no effect on SNAP patterns, which may contribute to diagnosing AIDP in the early stages. The limitations of our study are its retrospective design and the small number of patients suitable for inclusion.

Conclusion

In conclusion, the reduction in median and ulnar SNAP amplitudes along with SSP, with the ulnar SNAP amplitude used for comparison, is beneficial for diagnosing AIDP regardless of the timing of the electrodiagnostic examination, which may obviate serial studies.

Ethics

Ethics Committee Approval: The study was approved by the Ethics Committee of Gazi University (protocol number: 2013-163, date: 2023.10.21).

Informed Consent: Retrospective study.

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Concept: S.G.K., B.C., Design: S.G.K., B.C., Data Collection or Processing: S.G.K., B.C., R.K., Analysis or Interpretation: S.G.K., B.C., R.K., Literature Search: S.G.K., R.K., Writing: S.G.K., B.C., R.K.

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