

The Relationship Between Body Mass Index and Mononeuropathies

Vücut Kitle İndeksi ve Mononöropatiler Arasındaki İlişki

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ABSTRACT

Aim: The study aimed to find out whether there is a relationship between the mononeuropathies of the median, ulnar, radial, peroneal, and sciatic nerves and body mass index (BMI).

Material and Methods: Patients whose clinical and electrodiagnostic findings were compatible with carpal tunnel syndrome (CTS), ulnar neuropathy at the elbow (UNE), radial neuropathy at the spiral groove (RNS), peroneal neuropathy at the fibular head (PNFH), and sciatic injury due to intramuscular injection (SNIII) were included in this retrospective cohort study. In addition, controls whose clinical and electrodiagnostic features were not compatible with mononeuropathy were included in the study. The BMI values of all participants were analyzed.

Results: One hundred thirty-one CTS patients, 53 UNE patients, 6 RNS patients, 25 PNFH patients, 72 SNIII patients, and 53 controls were included in the study. The BMI of CTS patients was higher than the BMI of controls ($p<0.001$), PNFH patients ($p<0.001$), and SNIII patients ($p<0.001$). The BMI of SNIII patients was lower than the BMI of controls ($p<0.001$), CTS patients ($p<0.001$), and UNE patients ($p<0.001$). The BMI of PNFH patients was lower than that of CTS patients ($p<0.001$) and UNE patients ($p=0.004$). No significant correlation was found between BMI values and electrodiagnostic classification of mononeuropathies in the groups.

Conclusion: This study showed that high BMI is a risk factor for CTS and low BMI is a risk factor for SNIII. There may also be a relationship between BMI and PNFH, but this should be confirmed by further studies.

Keywords: Body mass index; carpal tunnel syndrome; entrapment neuropathy; mononeuropathy; sciatic nerve injury.

ÖZ

Amaç: Bu çalışmada medyan, ulnar, radyal, peroneal ve siyatik sinirlerin mononöropatileri ile vücut kitle indeksi (VKİ) arasında bir ilişki olup olmadığının araştırılması amaçlandı.

Gereç ve Yöntemler: Klinik ve elektrodiagnostik bulguları karpal tünel sendromu (KTS), dirsekte ulnar nöropati (DUN), spiral olukta radyal nöropati (RNS), fibula başında peroneal nöropati (PNFB) ve intramüsküler enjeksiyona bağlı siyatik yaralanması (İMESY) ile uyumlu olan hastalar bu geriye dönük kohort çalışmasına dahil edildi. Ayrıca klinik ve elektrodiagnostik özellikleri mononöropati ile uyumlu olmayan kontroller çalışmaya dahil edildi. Tüm katılımcıların VKİ değerleri analiz edildi.

Bulgular: Yüz otuz bir KTS hastası, 53 DUN hastası, 6 RNS hastası, 25 PNFB hastası, 72 İMESY hastası ve 53 kontrol bu çalışmaya dahil edildi. KTS hastalarının VKİ değeri kontrollerin ($p<0,001$), PNFB hastalarının ($p<0,001$) ve İMESY hastalarının ($p<0,001$) VKİ değerinden daha yüksekti. İMESY hastalarının VKİ değeri kontrollerin ($p<0,001$), KTS hastalarının ($p<0,001$) ve DUN hastalarının ($p<0,001$) VKİ değerinden daha düşüktü. PNFB hastalarının VKİ değeri KTS hastalarından ($p<0,001$) ve DUN hastalarından ($p=0,004$) daha düşüktü. Gruplarda VKİ değerleri ile mononöropatilerin elektrodiagnostik sınıflandırması arasında anlamlı bir korelasyon bulunmadı.

Sonuç: Bu çalışma, yüksek VKİ değerinin KTS için bir risk faktörü olduğunu ve düşük VKİ değerinin ise İMESY için bir risk faktörü olduğunu göstermiştir. VKİ ve PNFB arasında da bir ilişki olabilir, ancak bu daha ileri çalışmalarla doğrulanmalıdır.

Anahtar kelimeler: Vücut kitle indeksi; karpal tünel sendromu; tuzak nöropati; mononöropati; siyatik sinir hasarı.

INTRODUCTION

Mononeuropathies are disorders that can lead to disability. They can occur as a result of trauma or compression of the nerve while passing through a narrow anatomical canal. Injury of the nerve as a result of increased pressure in the narrow canal is known as entrapment mononeuropathy (1-3). The most common entrapment mononeuropathy is carpal tunnel syndrome (CTS). Other common entrapment mononeuropathies include ulnar neuropathy at the elbow (UNE), radial neuropathy at the spiral groove (RNS), and peroneal neuropathy at the fibular head (PNFH) (1-3). Disorders such as diabetes mellitus or thyroid disease, activities during daily life or business life, and factors such as age or body mass index (BMI) may be risk factors for these entrapment mononeuropathies (4-9). The relationship between BMI and entrapment mononeuropathies has been analyzed in many studies. Most of the research was conducted on CTS and UNE (4-11). Although BMI is known to be a risk factor for CTS, there were conflicting results regarding the association between UNE and BMI (4,5,10-12). It has also been reported that sciatic nerve injury due to intramuscular injection (SNIII) is seen in thin patients due to a low amount of gluteal muscle mass (13-15).

In this study, it was aimed to find out whether SNIII and entrapment neuropathies are associated with BMI.

MATERIAL AND METHODS

Study Design

Individuals aged over sixteen years who applied to Adana City Training and Research Hospital (ACTRH) Clinical Neurophysiology Laboratory between September 2018 and October 2020, with both clinical and electrodiagnostic characteristics compatible with entrapment mononeuropathy or SNIII, and controls were included in this retrospective study. Individuals with the following conditions were excluded from the study: disease that could cause neuropathy, such as diabetes mellitus; polyneuropathy; clinical, electrodiagnostic, and imaging test findings consistent with cervical/lumbosacral radiculopathy or plexopathy. In addition, individuals with two or more different entrapment mononeuropathies were not included. The age, gender, and BMI of the patients were analyzed. It was also recorded whether the patients had weight loss or not. Individuals with a BMI of $<18.5 \text{ kg/m}^2$ were classified as underweight, individuals with a BMI of 18.5 to $<25 \text{ kg/m}^2$ as normal, and those with a BMI of $\geq 25 \text{ kg/m}^2$ as overweight (16). Ethics committee approval was received from the ACTRH Ethics Committee (date: 20.05.2020, and number: 57/869).

Electrodiagnostic Tests

The nerve conduction studies and the needle electromyography (EMG) were performed with a Cadwell Sierra Summit EMG unit (Cadwell Laboratories, Kennewick, Washington, USA). Electrodiagnostic tests were performed if the temperature of the patient's extremities was $\geq 32 \text{ }^\circ\text{C}$. Recording and stimulation were made with surface electrodes. Supramaximal nerve stimulation was applied. For sensory and motor nerve conduction studies, band filters are set to 20 Hz-2 kHz and 20 Hz-10 kHz, respectively. For the sensory nerve conduction study, the sensitivity and sweep speed were 10 $\mu\text{V/division}$ and 1 ms/division , respectively.

Sensitivity and sweep speed for motor nerve study were 2 mV/division and 5 ms/division , respectively. If the patient had mononeuropathy in the upper extremity, nerve conduction studies were performed in the bilateral upper extremities and one lower extremity. If the patient had PNFH or SNIII, nerve studies were performed in bilateral lower extremities and one upper extremity. A nerve conduction study was performed in one upper and one lower extremity of the controls. Protocols for the median, ulnar, peroneal, posterior tibial, superficial peroneal, and sural nerve conduction studies were made using conventional methods (17-19). Reference values of ACTRH Clinical Neurophysiology Laboratory were used for routine nerve conduction studies (18,19). Reference values for routine motor and sensory nerve conduction studies are shown in Table 1 and Table 2, respectively. Unlike other nerve conduction studies, radial nerve compound muscle action potential (CMAP) was recorded from the extensor indicis proprius muscle with a concentric needle electrode (20). The lower reference limit for the radial motor nerve conduction velocity (NCV) across the forearm-above spiral groove segment was 49.8 m/s (20). A superficial radial sensory nerve conduction study was made with surface electrodes using conventional methods (17). The lower reference limit for superficial radial nerve sensory nerve action potential (SNAP) amplitude and upper reference limit for superficial radial SNAP peak latency were 11 μV and 2.8 ms , respectively. The high pass and low pass band filters for needle EMG were 10 Hz and 10 kHz, respectively. Needle EMG was performed visually. Active denervation was carefully analyzed. Motor unit action potential (MUAP) analysis was performed during light muscle contraction. The sweep speed was 10 ms/division for both active denervation and MUAP analysis. Sensitivity for active denervation and MUAP analysis was 100 $\mu\text{V/division}$ and 200-500 $\mu\text{V/division}$, respectively. Ten to twenty MUAPs were analyzed. If the MUAP amplitude was $>4 \text{ mV}$ and the MUAP duration was $>15 \text{ ms}$, the MUAP was considered neurogenic.

Subjects

Carpal Tunnel Syndrome (CTS) Patients

Patients should have one of the following criteria clinically (21,22): *i*) paresthesia in the first three fingers or one of the first three fingers, *ii*) in neurological examination, sensory abnormality in the first three fingers or one of the first three fingers and/or weakness in hand muscles innervated by the median nerve. Patients should have one of the following characteristics in electrodiagnostic tests (21,22): *i*) slowing of median sensory NCV across second finger-wrist/first finger-wrist/third finger-wrist/palm-wrist segments (mild CTS), *ii*) in addition to the first electrodiagnostic criterion, the delay of the median nerve CMAP latency (moderate CTS), *iii*) delay of median nerve CMAP latency and absence of median nerve SNAP across first/second/third finger-to-wrist segments (severe CTS). Since the lesion localization could not be determined clearly, patients whose median nerve CMAP could not be obtained were not included in the study. Needle EMG was applied to abductor pollicis brevis, pronator teres, and first dorsal interosseous muscles of all CTS patients. Patients with

needle EMG abnormalities in the first dorsal interosseous and pronator teres muscles were not included in the study.

Ulnar Neuropathy at the Elbow (UNE) Patients

Patients with one of the following abnormalities in their neurological examination were included in the study (23,24): *i*) sensory abnormality in the fourth/fifth finger or in the medial palm or dorsomedial of the hand, *ii*) weakness of the muscles innervated by the ulnar nerve. If one of the following features was present in the nerve conduction study, the patient was considered to have UNE (18,23,24): *i*) slowing of ulnar motor NCV across below elbow-above elbow segment, *ii*) motor conduction block (decreasing the CMAP amplitude obtained by stimulation of the ulnar nerve above the elbow by more than 50% compared to that obtained by stimulation of the ulnar nerve below the elbow) across below elbow-above elbow segment, *iii*) abnormal latency difference in short segment (2 cm) ulnar motor nerve conduction study. UNE patients were divided into three groups according to electrodiagnostic classification (25): *i*) mild UNE: slowing of the ulnar motor NCV across the below elbow-above elbow segment or motor conduction block across this segment, *ii*) moderate UNE: in addition to the first criterion, reduction of SNAP amplitude across the 5th finger-wrist segment, *iii*) severe UNE: in addition to the first criterion, the absence of SNAP across the 5th finger-wrist segment. Since the location of the lesion could not be determined clearly in patients whose ulnar nerve CMAP could not be obtained, these patients were not included in the study. Needle EMG was applied to the

abductor digiti minimi, first dorsal interosseous, flexor carpi ulnaris, flexor digitorum profundus (ulnar), and abductor pollicis brevis muscles in patients with UNE. Patients with needle EMG abnormalities in the abductor pollicis brevis muscle were not included in the study (26). Patients whose electrodiagnostic findings were compatible with ulnar neuropathy in the wrist were excluded (27).

Radial Neuropathy at the Spiral Groove (RNS) Patients

Patients with one of the following neurological examination findings were included in the RNS group (28,29): Weakness in the dorsiflexion of the fingers or wrist. In addition to this criterion, there may be an abnormality in the sensory area innervated by the superficial radial nerve. Patients should have radial motor nerve conduction block (CMAP amplitude reduction >50%) across the below spiral groove-above spiral groove segment. The electrodiagnostic classification of RNS was as follows: *i*) mild RNS: radial motor conduction block, *ii*) moderate RNS: motor conduction block and reduced superficial radial nerve SNAP amplitude, *iii*) severe RNS: motor conduction block and absence of superficial radial nerve SNAP. Patients whose radial nerve CMAP could not be obtained were excluded from the study. The needle EMG findings of the triceps, abductor pollicis brevis, and first dorsal interosseous muscles of these patients should have been normal (28,29).

Peroneal Neuropathy at the Fibular Head (PNFH) Patients

PNFH was considered if one of the following features was present in the neurological examination of the patients (30,31): *i*) weakness of the muscles with peroneal

Table 1. Reference values for motor nerve conduction studies

Motor nerve conduction study parameter	Reference value
Median nerve terminal CMAP amplitude (mV) / latency (ms)	3.7 / 4.3
Median motor NCV across wrist - elbow segment (m/s)	49.1
Ulnar nerve terminal CMAP amplitude (mV) / latency (ms) ADM	8.0 / 2.9
Ulnar nerve terminal CMAP amplitude (mV) / latency (ms) FDI	6.4 / 4.9
Ulnar motor NCV across wrist - below elbow segment ADM / FDI	52.0 / 50.9
Ulnar motor NCV below elbow - above elbow segment ADM / FDI	43.0 / 45.7
Latency difference in short segment ulnar motor nerve conduction study (ms)	0.7
Posterior tibial nerve CMAP amplitude (mV) / latency (ms)	4.2 / 5.8
Posterior tibial motor NCV across ankle - popliteal fossa segment (m/s)	41.0
Peroneal nerve terminal CMAP amplitude (mV) / latency (ms) (EDB)	3.7 / 5.2
Peroneal nerve terminal CMAP amplitude (mV) (TA)	3.9
Peroneal motor NCV across ankle - below fibular head segment (m/s)	43.9
Peroneal motor NCV below fibular head - popliteal fossa segment EDB / TA (m/s)	40.1 / 41.0

CMAP: compound muscle action potential, NCV: nerve conduction velocity, ADM: abductor digiti minimi, FDI: first dorsal interosseous, EDB: extensor digitorum brevis, TA: tibialis anterior, CMAP amplitudes were measured from peak to peak

Table 2. Reference values for sensory nerve conduction studies

Sensory nerve conduction study parameter	Reference value
Median nerve	
2nd digit-wrist SNAP amplitude (uV) / sensory NCV (m/s)* / sensory NCV (m/s)**	10.3 / 40.9 / 44.6
1st digit-wrist SNAP amplitude / sensory NCV (m/s)*	4.3 / 34.0
3rd digit-wrist SNAP amplitude / sensory NCV (m/s)*	2.8 / 39.5
Palm-wrist SNAP amplitude / sensory NCV (m/s)*	14.7 / 37.5
Ulnar nerve 5th digit-wrist SNAP amplitude / sensory NCV (m/s)* / sensory NCV (m/s)**	7.1 / 38.8 / 42.0
Superficial peroneal nerve SNAP amplitude / sensory NCV (m/s)	5.3 / 37.0
Sural nerve SNAP amplitude / sensory NCV (m/s)	5.1 / 33.2

SNAP: sensory nerve action potential, NCV: nerve conduction velocity, *: Sensory NCV was calculated using peak latency, **: Sensory NCV was calculated using onset latency, SNAP amplitudes were measured from peak to peak

nerve innervation, *ii*) sensory abnormality in the skin area supplied by the peroneal nerve. Electrodiagnostic tests should have one of the following findings (17,32,33): *i*) slowing of the peroneal motor NCV across the below fibular head-popliteal fossa segment, *ii*) peroneal motor nerve conduction block (CMAP amplitude reduction >25%) across the below fibular head-the popliteal fossa segment (17). PNFH classification was as follows: *i*) mild: motor conduction block or slowing of motor NCV across the below fibular head-popliteal fossa segment, *ii*) moderate: reduced superficial peroneal nerve SNAP amplitude in addition to the first item, *iii*) severe: in addition to the first item, absence of the superficial peroneal SNAP. Patients whose peroneal nerve CMAP could not be obtained from both the tibialis anterior and extensor digitorum brevis muscles were not included in the study. The needle EMG of the medial gastrocnemius, biceps femoris (short head), and vastus lateralis muscles of the patients should have been normal (30,31,33).

Sciatic Nerve Injury due to Intramuscular Injection (SNIII) Patients

The complaints of the patients must have been related to intramuscular injection. If the patient had one of the following neurological examination findings, it was considered that the diagnosis was compatible with SNIII (15,19,34): *i*) weakness in the muscles innervated by the tibial, peroneal, or sciatic nerves, *ii*) sensory abnormality in the skin area supplied by the sciatic nerve branches. SNIII was considered if one of the following findings was present in the electrodiagnostic tests: *i*) abnormality in motor and sensory nerve conduction studies of the sciatic nerve branches, *ii*) needle EMG abnormality in the muscles innervated by the sciatic nerve or its branches. Electrodiagnostic classification of SNIII was as follows: *i*) mild: normal sural and superficial peroneal nerve SNAPs, *ii*) moderate: reduced sural or superficial peroneal nerve SNAP amplitude, *iii*) severe: absence of sural or superficial peroneal nerve SNAP. Needle EMG was performed on the tibialis anterior, medial gastrocnemius, peroneus longus, and biceps femoris (short head) muscles of the patients. In order to exclude lumbosacral radiculopathy/plexopathy, needle EMG was applied to the vastus lateralis, gluteus maximus, L3, L4, L5, and S1 paraspinal muscles (13-15).

Controls

The individuals who applied to the clinical neurophysiology laboratory and whose clinical and electrodiagnostic findings were not compatible with mononeuropathy or polyneuropathy or radiculopathy or plexopathy were included in the control group. Electrodiagnostic tests for controls were performed for joint, bone, or muscle pain. Neurological examinations of the controls were normal. In addition, individuals with diseases that could cause neuropathy and neurodegenerative diseases such as diabetes mellitus were excluded. Fifty-three healthy participants were analyzed.

Statistical Analysis

Categorical variables were summarized as percentage and frequency. The mean, standard deviation, and minimum and maximum of the numeric data were calculated for descriptive statistics. Pearson's chi-square and Fisher's exact tests were used to analyze categorical variables. The Shapiro-Wilk test was used to determine the distribution of the data. Kruskal-Wallis and Mann-Whitney U tests were used in the analysis of quantitative data. Bonferroni

correction was used for post hoc analysis and multiple comparisons. Spearman's rank correlation coefficient test was used for the correlation analysis. If p value was <0.05, it was considered statistically significant. The Statistical Package for the Social Sciences (SPSS IBM Corp; Armonk, NY, USA) 22.0 was used for statistical analysis.

RESULTS

One hundred thirty-one CTS patients, 53 UNE patients, 6 RNS patients, 25 PNFH patients, 72 SNIII patients, and 53 controls were included in the study. 19 (14.5%) of the CTS patients, 37 (69.8%) of the UNE patients, 5 (83.3%) of the RNS patients, 21 (84.0%) of the PNFH patients, 52 (72.2%) of the SNIII patients, and 30 (56.6%) of the controls were male. The rate of females in CTS patients was significantly higher than the rate of females in all other groups ($p < 0.001$). In addition, the rate of males in PNFH patients was higher than the control group ($p = 0.022$).

Age, height, weight, and BMI values among the groups were shown in Table 3. BMI values and BMI classification among groups were shown in Figure 1 and Figure 2, respectively. There was a history of weight loss in eleven PNFH patients and five SNIII patients. Six of the eleven PNFH patients with weight loss also had a history of prolonged repetitive leg posture, such as crossing the legs. The mean amount of weight loss in PNFH patients was 4.7 ± 2.3 (range, 2.5 to 8) kg/month. Except for PNFH and SNIII patients, none of the participants had a history of weight loss. Eleven PNFH patients had only a history of prolonged repetitive leg posture, four had only a history of prolonged sleep (two had a history of alcohol intake before sleep). In all RNS patients, RNS occurred after prolonged sleep (history of alcohol intake in two patients, history of heroin use in one patient). The side of mononeuropathies and the electrodiagnostic classification of mononeuropathies among the groups were shown in Table 4. There was no correlation between BMI values and electrodiagnostic classification of mononeuropathies in the groups (Table 5).

DISCUSSION

Different risk factors have been identified in different mononeuropathies (4-8,11,12). Disorders such as diabetes mellitus or thyroid disease, age, gender, activities during daily life or work, and BMI can be risk factors for some mononeuropathies (4-8). It is known that high BMI is a risk factor for CTS (4-7). This can be explained by the increased pressure in the carpal tunnel as a result of the increase in adipose tissue due to obesity and the subsequent injury to the median nerve (1,2,4,7,35). On the other hand, although there are studies showing that low BMI is a risk factor for UNE (10,11), there are also authors who report otherwise (12). It is thought that thin people have less amount of tissue protecting the ulnar nerve at the elbow, and consequently, the ulnar nerve in thin people may become more susceptible to compression (11). Although there was no difference between the BMIs of the control group and the UNE group in this study, the fact that most of the UNE patients had a BMI of $< 30 \text{ kg/m}^2$ may mean that UNE is less common in obese patients. However, it should be kept in mind that the distance can be measured more than it should be in nerve conduction studies in overweight patients (11). Future studies are needed to reveal whether there is a relationship between UNE and BMI.

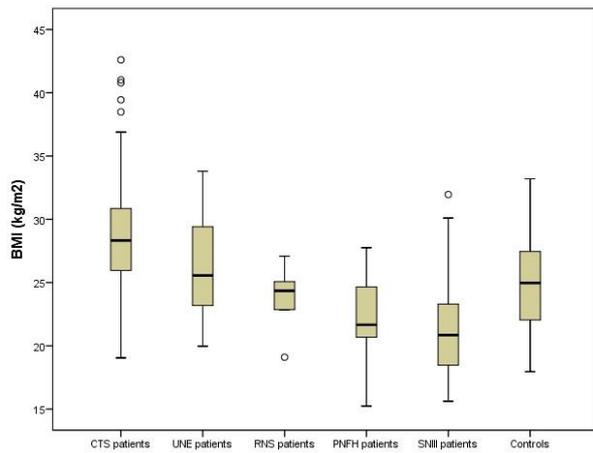


Figure 1. BMI among groups

BMI: body mass index, CTS: carpal tunnel syndrome, UNE: ulnar neuropathy at the elbow, RNS: radial neuropathy at the spiral groove, PNFH: peroneal neuropathy at the fibular head, SNIII: sciatic nerve injury due to intramuscular injection

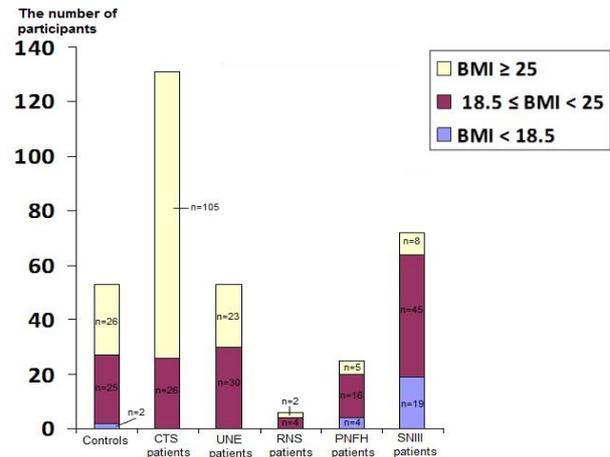


Figure 2. BMI classification among groups

BMI: body mass index, CTS: carpal tunnel syndrome, UNE: ulnar neuropathy at the elbow, RNS: radial neuropathy at the spiral groove, PNFH: peroneal neuropathy at the fibular head, SNIII: sciatic nerve injury due to intramuscular injection

Table 3. Age, weight, height, and BMI among groups, mean±SD, median (IQR₂₅₋₇₅) [min-max]

	CTS (n=131)	UNE (n=53)	RNS (n=6)	PNFH (n=25)	SNIII (n=72)	Controls (n=53)	p*
Age (years)	47.3±12.7 46 (38.0-56.0) [23-83]	42.0±14.9 42 (28.5-53.5) [18-77]	41.3±17.3 41 (22.8-59.8) [22-62]	32.8±16.0 30 (18.0-41.5) [18-82]	41.3±16.2 35 (30.0-54) [19-79]	38.8±12.8 38 (30.0-46.5) [18-69]	<0.001
Height (cm)	162.8±5.9 162 (159-165) [150-186]	172.1±7.9 173 (168-178) [150-191]	169.8±6.1 171 (164-175) [161-176]	176.2±7.6 176 (172-180) [160-191]	171.1±8.4 170 (165-177) [155-196]	170.1±7.9 172 (164-176) [150-185]	<0.001
Weight (kg)	76.5±12.3 76 (68.0-84.5) [54-110]	77.7±11.9 76 (68.0-86.0) [58-112]	68.8±10.0 70 (61.8-76.0) [52-82]	68.9±12.7 70 (59.0-80.0) [40-85]	62.0±8.9 60 (56.0-66.8) [47-87]	73.0±12.5 73 (63.5-84.0) [50-97]	<0.001
BMI (kg/m ²)	28.7±4.4 28.3 (25.7-30.9) [19.1-42.6]	26.3±4.1 25.6 (23.2-29.4) [19.9-33.8]	23.8±2.7 24.4 (21.9-25.6) [19.1-27.1]	22.1±3.4 21.7 (20.5-24.7) [15.2-27.8]	21.3±3.5 20.9 (20.5-24.7) [15.6-31.9]	25.1±3.9 24.9 (22.0-27.5) [17.9-33.2]	<0.001

BMI: body mass index, CTS: carpal tunnel syndrome, UNE: ulnar neuropathy at the elbow, RNS: radial neuropathy at the spiral groove, PNFH: peroneal neuropathy at the fibular head, SNIII: sciatic nerve injury due to intramuscular injection, SD: standard deviation, IQR₂₅₋₇₅: interquartile range (25th-75th percentile), *: Kruskal-Wallis test was used, Bonferroni correction was used for post hoc analysis and multiple comparisons: The ages of CTS patients were higher than that of PNFH patients (p<0.001), SNII patients (p=0.012), and controls (p=0.005); The heights of CTS patients were lower than that of controls (p<0.001), SNIII patients (p<0.001), UNE patients (p<0.001), and PNFH patients (p<0.001); The weights of SNIII patients were lower than that of controls (p<0.001), UNE patients (p<0.001), and CTS patients (p<0.001); The BMI of CTS patients was higher than that of controls (p<0.001), UNE patients (p<0.001), and SNIII patients (p<0.001); The BMI of SNIII patients was lower than that of controls (p<0.001), CTS patients (p<0.001), and UNE patients (p<0.001); The BMI of PNFH patients was lower than that of CTS patients (p<0.001), and UNE patients (p=0.004).

Table 4. Side and electrodiagnostic classification of mononeuropathies

Mononeuropathy	CTS (n=131)	UNE (n=53)	RNS (n=6)	PNFH (n=25)	SNIII (n=72)
Side of the mononeuropathy					
Right	25	38	3	18	22
Left	5	15	3	7	50
Bilateral	101	0	0	0	0
Electrodiagnostic classification					
Mild	51	36	6	20	7
Moderate	72	13	0	3	22
Severe	8	4	0	2	43

CTS: carpal tunnel syndrome, UNE: ulnar neuropathy at the elbow, RNS: radial neuropathy at the spiral groove, PNFH: peroneal neuropathy at the fibular head, SNIII: sciatic nerve injury due to intramuscular injection

Table 5. Correlation between BMI and electrodiagnostic classification among groups

Electrodiagnostic classification	BMI	
	r _s	p
CTS	0.098	0.413
UNE	0.219	0.186
PNFH	0.028	0.894
SNIII	-0.127	0.404

BMI: body mass index, r_s: Spearman correlation, CTS: carpal tunnel syndrome, UNE: ulnar neuropathy at the elbow, PNFH: peroneal neuropathy at the fibular head, SNIII: sciatic nerve injury due to intramuscular injection

In this study, the BMI of PNFH patients were less than the BMIs of CTS and UNE patients, although they were not different from the BMI of the controls. However, some of the patients had a history of weight loss. It is known that long-term repetitive leg postures and weight loss are associated with PNFH (30-32). This situation can be explained by the reduction of the tissues protecting the peroneal nerve due to weight loss and the peroneal nerve becoming more susceptible to injury due to compression, as we mentioned earlier (36). However, some patients with

PNFH associated with weight loss did not have a history of prolonged repetitive leg postures or prolonged sleep. For this reason, the reduction of nerve protective tissue may not be the only cause of PNFH. Metabolic changes due to weight loss may also have caused PNFH (37,38). These findings and the fact that the BMI of PNFH patients was lower than that of CTS and UNE patients found in this study may indicate that thin individuals are more likely to develop PNFH.

We found that the BMIs of SNIII patients were lower than the BMIs of controls, CTS patients, and UNE patients. This can be explained by the fact that the sciatic nerve becomes more sensitive to trauma as a result of the reduction of the tissue in the gluteal region that protects the sciatic nerve (13,15,39). Therefore, factors such as the length of the needle and the position of the patient should be taken into consideration when administering an intramuscular injection to individuals with low BMI (15). Intramuscular injection into the gluteus medius muscle instead of the gluteus maximus muscle can also inhibit SNIII (40).

Two RNS patients had a BMI of $>25 \text{ kg/m}^2$ but less than 30 kg/m^2 . The BMI of other RNS patients was normal. Although the findings in this study may indicate that BMI is not a risk factor for RNS, it should be noted that the number of patients is low. Further studies on BMI and RNS patients are needed.

Although one study found a relationship between CTS severity and BMI (41), there is also a study by Kouyoumdjian, which showed that there was no relationship between severely affected median nerve SNAP latency and high BMI (6). In our study, no correlation was found between the electrodiagnostic classification of CTS and BMI. In addition, a similar finding was found in patients with UNE, PNFH, and SNIII. These findings may suggest that factors other than BMI affect the severity of mononeuropathy after the development of mononeuropathy associated with BMI. We think that neurological examination and electrodiagnostic tests to be performed in patients with mononeuropathy after weight gain or weight loss will have interesting results.

This study had some limitations. First, the proportion of females in the CTS group was high. This was an expected result. CTS is known to be more common in women (9,42). In addition, there are studies reporting that UNE is more common in men (12,43). Given that gender is a risk factor for some mononeuropathies, this may have influenced our results. But it should be noted that we are making a comparison in a heterogeneous group. The male-gender ratio was higher in patients with PNFH. This finding may be important and needs to be confirmed by further studies. Second, the age of CTS patients was different from the participants in other groups. This finding supported the conclusion found in previous studies that age is a risk factor for CTS (9,44). Third, as we mentioned earlier, the numbers of RNS patients were low. Finally, the controls did not consist of completely healthy individuals. However, it should be noted that his neurological examinations were normal, and he had no neurological symptoms.

Despite these limitations, this study also had its strengths. We used both clinical and electrodiagnostic criteria for

mononeuropathy. Thus, it can be said that the diagnoses are more precise. In addition, neurological examinations and nerve conduction studies were performed in the upper and lower extremities of all participants, as a result, the possibility of more than one mononeuropathy was eliminated clinically or subclinically. We did not include patients with only the motor and sensory nerve conduction studies of the median and ulnar nerves, and all patients had nerve conduction studies of the upper and lower extremities.

CONCLUSION

This study showed that high BMI is a risk factor for CTS and low BMI is a risk factor for SNIII. There may also be a relationship between BMI and PNFH, but this should be confirmed by further studies.

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