© Aktualn Neurol 2022, 22 (2), p. 67–74
DOI: 10.15557/AN.2022.0007

Received: 20.11.2021
Accepted: 06.09.2022
Published: 07.12.2022

Şencan Buturak[1], Halit Fidancı[2], İlker Öztürk[1], Zülfikar Arlıer[1]

Conduction block and positive sharp waves/fibrillation potentials in entrapment neuropathies of the ulnar, radial, and peroneal nerves

Blok przewodzenia i dodatnie fale ostre/fibrylacje w przebiegu neuropatii z uwięźnięcia w obrębie nerwów łokciowych, promieniowych i strzałkowych

[1] Department of Neurology, Adana City Training and Research Hospital, Adana, Turkey
[2] Department of Neurology, Division of Clinical Neurophysiology, Adana City Training and Research Hospital, Adana, Turkey
Correspondence: Dr. Halit Fidancı, Department of Neurology, Division of Clinical Neurophysiology, Adana City Training and Research Hospital 01060, Yüregir, Adana, Turkey, tel.: +90322455900-1147, fax: +903223440305, e-mail: dr.halitfidanci@gmail.com

ORCID iDs
1. Şencan Buturak https://orcid.org/0000-0002-7496-5628
2. Halit Fidancı https://orcid.org/0000-0001-6573-9090
3. İlker Öztürk https://orcid.org/0000-0002-2333-9360
4. Zülfikar Arlıer https://orcid.org/0000-0003-2645-648X

Abstract

Introduction: Entrapment mononeuropathies can cause motor conduction block, positive sharp waves, and fibrillation potentials. **Aim:** The study aims to find whether there is a relationship between positive sharp waves/fibrillation potentials and conduction block observed in entrapment mononeuropathies. **Materials and methods:** Patients with ulnar neuropathy at the elbow, radial neuropathy at the spiral groove, and peroneal neuropathy at the fibular head were included in this retrospective study. Nerve conduction study and needle electromyography results of the patients were analysed. **Results:** The study included a total of patients with 67 ulnar neuropathy, 8 radial neuropathy, and 27 peroneal neuropathy. All radial and peroneal neuropathy patients and 30 ulnar neuropathy patients had positive sharp waves/fibrillation potentials in at least one muscle. Twenty-three ulnar neuropathy patients with these potentials, 6 radial neuropathy patients, and 18 peroneal neuropathy patients had conduction block ($p < 0.001$). The reduction of compound muscle action potential amplitude in percentage recorded from the abductor digiti quinti/first dorsal interosseous across the elbow segment in ulnar neuropathy patients with and without positive sharp waves/fibrillation potentials was 41.9 ± 35.9/46.6 ± 36.1% and 7.6 ± 16.5/10.4 ± 16.5%, respectively ($p < 0.001/p < 0.001$). The distal compound muscle action potential amplitudes of ulnar neuropathy patients with these potentials were lower than those of ulnar neuropathy patients without these potentials ($p = 0.029$ – abductor digiti quinti, $p = 0.017$ – first dorsal interosseous). No correlation was found between the severity of positive sharp waves/fibrillation potentials and muscle strength in patients with these potentials ($p > 0.05$). **Conclusions:** Positive sharp waves/fibrillation potentials and motor conduction block can be seen together in patients with entrapment mononeuropathies. We concluded that there may be no relationship between the severity of these potentials and muscle strength.

Keywords: conduction block, electrodiagnostic testing, entrapment mononeuropathy, fibrillation potential, positive sharp wave

Streszczenie

Wstęp: Mononeuropatie z uwięźnięcia mogą powodować blok przewodzenia ruchowego, dodatnie fale ostre i fibrylacje. **Cel:** Celem badania było ustalenie, czy istnieje związek pomiędzy dodatnimi falami ostrymi/fibrylacjami a blokiem przewodzenia obserwowanym w przebiegu mononeuropatii z uwięźnięcia. **Materiał i metody:** Do retrospektywnego badania włączono pacjentów z neuropatią nerwu łokciowego, neuropatią nerwu promieniowego w bruździe spiralnej i neuropatią nerwu strzałkowego w okolicy główki kości strzałkowej. Analizie poddano wyniki przeprowadzonego u pacjentów badania przewodnictwa nerwowego i elektromiografii igłowej. **Wyniki:** Do badania włączono 67 pacjentów z neuropatią nerwu łokciowego, 8 pacjentów z neuropatią nerwu promieniowego i 27 pacjentów z neuropatią nerwu strzałkowego. U wszystkich uczestników z neuropatią nerwu promieniowego i strzałkowego oraz 30 pacjentów z neuropatią nerwu łokciowego wykazano dodatnie fale ostre/fibrylacje w co najmniej jednym mięśniu. U 23 pacjentów z neuropatią nerwu łokciowego, u których stwierdzono te potencjały, a także u 6 uczestników badania z neuropatią nerwu promieniowego i 18 pacjentów z neuropatią nerwu strzałkowego odnotowano blok przewodzenia ($p < 0{,}001$). Spadek amplitudy mięśniowych potencjałów czynnościowych

(w ujęciu procentowym) w mięśniu odwodzicielu palca małego/mięśniu pierwszym grzbietowym międzykostnym w odcinku łokciowym u pacjentów z neuropatią nerwu łokciowego z dodatnimi falami ostrymi/fibrylacjami i bez nich wyniósł odpowiednio 41,9 ± 35,9/46,6 ± 36,1% i 7,6 ± 16,5/10,4 ± 16,5% ($p < 0{,}001/p < 0{,}001$). Amplitudy potencjałów czynnościowych mięśni dystalnych u pacjentów z neuropatią nerwu łokciowego z fibrylacjami były niższe niż u pacjentów z neuropatią nerwu łokciowego bez tych potencjałów ($p = 0{,}029$ – mięsień odwodziciel palca małego, $p = 0{,}017$ – mięsień pierwszy grzbietowy międzykostny). Nie stwierdzono zależności pomiędzy nasileniem dodatnich fal ostrych/fibrylacji a siłą mięśniową u pacjentów z tymi potencjałami ($p > 0{,}05$). **Wnioski:** U pacjentów z mononeuropatią z uwięźnięcia mogą współwystępować dodatnie fale ostre/fibrylacje i blok przewodzenia. Uzyskane wyniki mogą wskazywać na brak zależności między nasileniem tych potencjałów a siłą mięśniową.

Słowa kluczowe: blok przewodzenia, badanie elektrodiagnostyczne, mononeuropatia z uwięźnięcia, fibrylacje, dodatnie fale ostre

INTRODUCTION

Entrapment mononeuropathy occurs when a peripheral nerve is injured as a result of compression passing through a narrow anatomical space (Lundborg and Dahlin, 1996; Wahab et al., 2017). Carpal tunnel syndrome, ulnar neuropathy at the elbow (UNE), radial neuropathy at the spiral groove (RN), and peroneal neuropathy at the fibular head (PNFH) are among the most common entrapment mononeuropathies (Lundborg and Dahlin, 1996; Wahab et al., 2017). Entrapment mononeuropathies can be diagnosed by clinical and electrodiagnostic tests, and by imaging methods (Dong et al., 2012; Hobson-Webb and Juel, 2017; Wahab et al., 2017). Nerve conduction studies and needle electromyography (EMG) are not only useful for the diagnosis but also provide information about the severity of mononeuropathy and the location of the lesion. They are also important for the differential diagnosis (Bowley and Doughty, 2019; Doughty and Bowley, 2019; Katirji, 1999; Landau and Campbell, 2013; Omejec and Podnar, 2015; Wahab et al., 2017; Wang and Weiss, 2013). Slowing of nerve conduction velocity (NCV), and conduction block (CB) found in nerve conduction studies are important findings for entrapment mononeuropathies (Bowley and Doughty, 2019; Doughty and Bowley, 2019; Katirji, 1999; Wang and Weiss, 2013). Positive sharp waves (PSWs) and fibrillation potentials (FPs) that can be detected by needle EMG may indicate axonal degeneration; these active denervation findings may also be associated with CB (Katirji, 1999). This study aims to obtain information about the presence of PSWs and FPs in entrapment mononeuropathies in which conduction block is frequently observed in nerve conduction studies.

MATERIALS AND METHODS

Individuals who were admitted to the Clinical Neurophysiology Laboratory of Adana City Training and Research Hospital (ACTRH) between September 2018 and November 2020, and whose clinical and electrophysiological findings were compatible with UNE, RN, and PNFH were included in this retrospective cohort study. Patients with the following characteristics or diseases were excluded from participation: polyneuropathy, a disease that may cause polyneuropathy such as diabetes mellitus, neurodegenerative disease, clinical and electrodiagnostic findings compatible with cervical/lumbosacral radiculopathy or brachial/lumbosacral plexopathy, mononeuropathy associated with major trauma, more than one entrapment mononeuropathy. The clinical and electrodiagnostic findings of the enrolled patients were analysed. The Medical Research Council (MRC) scale was used for determining muscle strength (Kleyweg et al., 1991). Ethics committee approval was received from the ACTRH Ethics Committee (Decision No.: 72/1182).

Electrodiagnostic tests were performed with a Cadwell Sierra Summit EMG unit (Cadwell Laboratories, Kennewick, Washington, USA). Nerve conduction studies were performed when the temperature of the patient's extremities was >32°C. Cold extremities were heated. Surface electrodes were used for stimulation and recording in nerve conduction studies. The nerves were stimulated supramaximally. Sweep speed and sensitivity for sensory nerve conduction studies were 1 ms/division and 10 μV/division, respectively. Sweep speed and sensitivity for the motor nerve conduction studies were set as 5 ms/division and 2 mV/division, respectively. Nerve conduction studies were performed in at least three extremities in all patients. Data from previous studies were used for the methods and reference values of nerve conduction studies of the median, ulnar, peroneal, and posterior tibial nerves (Fidancı et al., 2020a, 2020b). The lower reference limits for the ulnar NCV across the 5th finger-wrist segment and sensory nerve action potential (SNAP) amplitude were 38.8 m/s and 7.1 μV, respectively. The lower reference limits for the ulnar motor NCV across wrist-below elbow and below elbow-above elbow segments were 52 m/s (abductor digiti quinti, ADQ)/50.9 m/s (first dorsal interosseous, FDI) and 43 m/s (ADQ)/45.7 m/s (FDI), respectively. The lower reference limit for the ulnar nerve compound muscle action potential (CMAP) amplitude was 8.0 mV (ADQ)/6.4 mV (FDI). The upper reference limit for distal ulnar CMAP was 2.9 ms (ADQ)/4.9 ms (FDI), respectively (Fidancı et al., 2020b). In the short segment ulnar motor nerve conduction study, the stimulation points were the medial epicondyle (ME) and at 2 cm intervals towards the distal (D) and proximal (P) directions.

DOI: 10.15557/AN.2022.0007

In the short segment of ulnar nerve conduction study recorded from the ADQ/FDI muscles, the upper reference values of latency differences in D4–D2, D2–ME, ME–P2, P2–P4, P4–P6 segments were 0.6/0.5, 0.6/0.7, 0.7/0.8, 0.5/0.7, and 0.6/0.5 ms, respectively (Fidancı et al., 2020a). The stimulation and recording for radial motor nerve conduction were performed with surface electrodes and concentric needle electrodes (length = 50 mm, diameter = 0.46 mm, Bionen Medical Devices, Florence, Italy), respectively (Oh, 2003). For the superficial radial sensory nerve conduction study, the recommended methods were used (Chen et al., 2016). In the forearm-arm segment, the lower reference limit for the radial motor NCV was 49.8 m/s (Oh, 2003). The lower reference limit for the amplitude of the superficial radial SNAP was 11 μV. The superficial radial SNAP peak latency was set at 2.8 ms as the upper reference limit (Chen et al., 2016). The upper/lower reference limits for the peroneal nerve CMAP were as follows: 1) distal CMAP amplitude: 3.7 mV (extensor digitorum brevis, EDB), 3.9 mV (tibialis anterior, TA); 2) distal CMAP latency: 5.2 ms; 3) NCV across ankle-below fibular head segment: 43.9 m/s; 4) NCV across below fibular head segment: 40.1 m/s (EDB), 41 m/s (TA) (Fidancı et al.; 2020a). The lower reference limits for the amplitude of the superficial peroneal nerve NCV and SNAP were 37.0 m/s and 5.3 μV, respectively (Fidancı et al.; 2020a). Needle EMG was performed visually. Concentric needle electrode electrodes (length = 50 mm, diameter = 0.46 mm, Bionen Medical Devices, Florence, Italy) were used for the recording. The PSWs and FPs were carefully analysed. The classification of the PSWs/FPs was made as follows (Daube and Rubin, 2009): 0) absence of PSWs and FPs; 1) single PSW or FP in at least two areas; 2) a moderate number of PSWs or FPs in three or four areas; 3) PSWs or FPs in all areas; 4) PSWs or FPs filling the screen in all areas. Motor unit action potential (MUAP) was analysed during mild muscle contraction. If the MUAP amplitude was >4 mV, and the duration was >15 ms, this was considered neurogenic. If any muscle of the patient had PSWs/FPs, the patient was considered to have PSWs/FPs. Entrapment mononeuropathy patients were divided into two groups: with and without PSWs/FPs.

Conduction block: If the CMAP amplitude reduction in percentage obtained with proximal nerve stimulation from the ADQ or FDI muscles in UNE and the EDB or TA muscles in PNFH, and the extensor indicis proprius (EIP) muscle in RN was higher than 50% compared to the CMAP amplitude obtained with distal nerve stimulation (American Association of Electrodiagnostic Medicine; Olney, 1999; Oh et al., 1994). The patients were divided into groups according to the presence or absence of CB.

UNE patients: One of the following had to be present during the neurological examination: 1) sensory abnormality in the area of the skin supplied by the ulnar nerve; 2) weakness of the muscles with ulnar nerve innervation. Electrodiagnostic tests should reveal one of the following across the below elbow – above elbow segment (CMAP recorded from ADQ and/or FDI): 1) slowing of ulnar motor NCV; 2) motor CB or CMAP amplitude reduction in percentage CMAP amplitude reduction in percentage >14.9% (ADQ) and >22.7% (FDI) (Fidancı et al., 2020b); 3) abnormal latency delay in short segment (2 cm) ulnar motor nerve conduction study. Depending on the tolerance of the patients, needle EMG was applied to the ADQ, FDI, flexor carpi ulnaris, flexor digitorum profundus (ulnar), and abductor pollicis brevis muscles in patients with UNE. There should be no abnormality other than the ulnar innervated muscles in the needle EMG.

RN patients: Except for the triceps muscle, the neurological examination must reveal a weakness in the muscles innervated by the radial nerve. Furthermore, patients may have a sensory abnormality in the neurological examination in the area of the skin innervated by the superficial radial nerve. There must be a motor CB of the radial nerve across the forearm – above the spiral groove segment or needle EMG abnormalities in the EDC, EIP, and brachioradialis muscles. Needle EMG in the abductor pollicis brevis, FDI, deltoid, and triceps muscles of the RN patients must be normal (Wang and Weiss, 2013).

PNFH patients: The neurogenic examination of the patients must reveal a weakness in the peroneal nerve innervation muscles and/or a sensory abnormality in the skin area innervated by the peroneal nerve. One of the following should be present across the below fibular head-popliteal fossa in the peroneal nerve conduction study performed by recording from the EDB and/or TA muscles: 1) slowing of the peroneal motor NCV; 2) motor CB or abnormal CMAP amplitude reduction in percentage >25% (Chen et al., 2016). Needle EMG was performed on the patients' TA, peroneus longus, vastus lateralis, medial gastrocnemius, the short head of the biceps femoris, gluteus medius, gluteus maximus, L3, L4, L5, and S1 paraspinal muscles of the patients. There should be no needle EMG abnormality in the muscles other than peroneal innervated muscles (Masakado et al., 2008).

Statistical analysis: Categorical variables were summarized as percentages and frequencies. Mean ± standard deviation was calculated for descriptive statistics. The Pearson's Chi-square and Fisher's exact tests were used to analyse categorical variables. The Spearman's test was used for correlation. The $p < 0.05$ value was considered to be statistically significant. The Statistical Package for the Social Sciences (SPSS IBM Corp; Armonk, NY, USA) 22.0 was used to perform the statistical analysis.

RESULTS

Sixty-seven UNE patients (49 males, 18 females), eight RN patients (seven males, one female), and 27 PNFH patients (21 males, six females) were included in the study. The mean age of patients with UNE, RN, and PNFH was 41.4 ± 14.4 (range 18–77), 39.3 ± 15.4 (range 22–62), and 31.9 ± 15.9

 DOI: 10.15557/AN.2022.0007

Neurological examination and nerve conduction studies	Number of patients (%)
UNE patients (*n* = 67)	
Side of the UNE – right/left/bilateral	22 (33)/45 (67)/0 (0)
Sensory abnormality in 4th–5th digits/medial palm	64 (96)/47 (70)
Weakness in ADQ/FDI muscles	29 (43)/31 (46)
Reduced ulnar nerve CMAP amplitude – ADQ/FDI*	15 (22)/7 (13)
Reduced ulnar nerve SNAP amplitude across 5th finger-wrist segment	22 (33)
Slowing of ulnar motor NCV across below elbow-above elbow segment – ADQ/FDI*	32 (48)/35 (64)
Abnormal ulnar nerve CMAP latency in short segment motor nerve conduction study – ADQ/FDI*	63 (94)/47 (86)
Abnormal ulnar nerve CMAP latency across E–D2/P2–E/P4–P2 segments	13 (19)/52 (78)/2 (3)
Abnormal ulnar nerve CMAP amplitude reduction in percentage across below elbow-above elbow segment – ADQ/FDI*	26 (39)/15 (27)
PSWs/FPs in ADQ/FDI/FCU†/FDP‡	24 (36)/27 (40)/9 (14)/8 (13)
Neurogenic MUAP ADQ/FDI/FCU†/FDP‡	28 (42)/28 (42)/20 (31)/15 (23)
RN patients (*n* = 8)	
Side of the RN – right/left/bilateral	5 (62.5)/3 (37.5)/0 (0)
Sensory abnormality in the skin area innervated by superficial radial nerve	6 (75)
Weakness in dorsiflexion of fingers/wrist	8 (100)/8 (100)
Reduced superficial radial nerve SNAP amplitude	0 (0)
Slowing of radial motor NCV across forearm – above spiral groove segment	2 (25)
Abnormal radial nerve CMAP amplitude reduction in percentage across below spiral groove – above spiral groove segment	6 (75)
PSWs/FPs in EIP/EDC/BR	5 (62.5)/8 (100)/8 (100)
Neurogenic MUAP EIP/EDC/BR	0 (0)/0 (0)/0 (0)
PNFH patients (*n* = 27)	
Side of the PNFH – right/left/bilateral	18 (67)/9 (33)/0 (0)
Sensory abnormality in dorsum of foot/lateral of leg/dorsum of foot + lateral of leg	10 (37)/1 (4)/11 (41)
Sensory abnormality in dorsum of foot or lateral of leg	22 (82)
Weakness in dorsiflexion of foot/eversion of foot/dorsiflexion of foot or eversion of foot	26 (96)/23 (85)/27 (100)
Reduced peroneal nerve CMAP amplitude – EDB/TA	11 (41)/7 (26)
Reduced superficial peroneal nerve SNAP amplitude	6 (22)
Neurological examination and nerve conduction studies	**Number of patients (%)**
Slowing of peroneal motor NCV across below fibular head-popliteal fossa segment – EDB§/TA¶	17 (63)/17 (63)
Abnormal peroneal nerve CMAP amplitude reduction in percentage across below fibular head-popliteal fossa segment – EDB§/TA	20 (77)/24 (89)
PSWs/FPs in TA/PL	27 (100)/18 (67)
Neurogenic MUAP – TA/PL	1 (4)/0 (0)

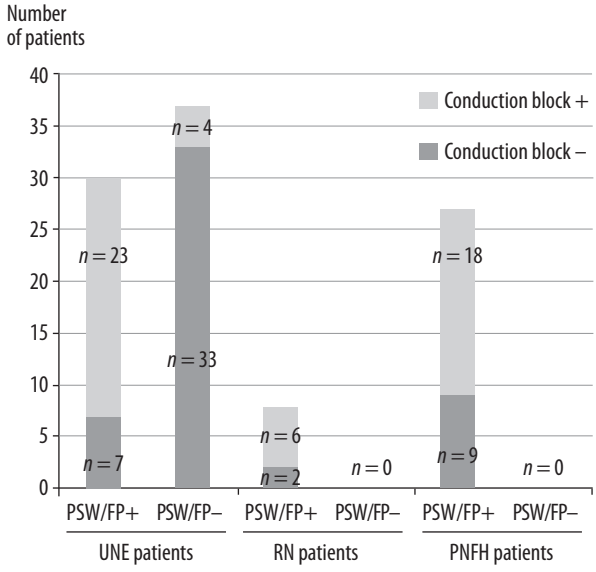
* Fifty-five patients were examined. † Sixty-five patients were examined. ‡ Sixty-four patients were examined. § Peroneal nerve CMAP could not be obtained from the EDB muscle in one patient. ¶ NCV could not be calculated in one patient since CMAP was not obtained from the TA muscle by stimulating the peroneal nerve at the popliteal fossa. **ADQ** – abductor digiti quinti; **BR** – brachioradialis; **CMAP** – compound muscle action potential; **EDB** – extensor digitorum brevis; **E–D2** – elbow – 2 cm distal to the medial epicondyle; **EDC** – extensor digitorum communis; **EIP** – extensor indicis proprius; **FCU** – flexor carpi ulnaris; **FDI** – first dorsal interosseous; **FDP** – flexor digitorum profundus (ulnar); **MUAP** – motor unit action potential; **NCV** – nerve conduction velocity; **P2–E** – 2 cm proximal to the medial epicondyle – elbow; **P4–P2** – 4 cm – 2 cm proximal to the medial epicondyle; **PL** – peroneus longus; **PNFH** – peroneal neuropathy at the fibular head; **RN** – radial neuropathy at the spiral groove; **SNAP** – sensory nerve action potential; **TA** – tibialis anterior; **UNE** – ulnar neuropathy at the elbow.

Tab. 1. Abnormalities in neurological examination and electrodiagnostic tests of the patients

(range 16–82) years, respectively. For patients with UNE, RN, and PNFH, the time between the onset of complaints and the time of electrodiagnostic tests was 93.6 ± 56.8 (21–180), 29.0 ± 8.1 (range 20–40), and 34.9 ± 13.4 (range 21–60) days, respectively. The interval was ≤60 days in patients with RN and PNFH, and 41 patients with UNE.

Following a prolonged sleep, six RN patients and four PNFH patients developed entrapment mononeuropathy. One patient with PNFH that developed entrapment mononeuropathy also had a history of weight loss. Among the PNFH patients, 13 had a history of prolonged repetitive leg posture, five had a history of weight loss, and five had a history of both weight loss and repetitive leg posture. There was no history of weight loss or prolonged sleep in the patients with UNE. Forty-two UNE patients and one RN patient had a history of performing activities with excessive use of the upper extremities. One RN patient complained about a burn on a body area other than the extremity with a drop hand after anaesthesia for surgery.

The abnormalities in neurological examination and nerve conduction studies of the patients are shown in Tab. 1. The motor CB and PSWs/FPs found in patients with UNE, RN, and PNFH are shown in Fig. 1. Motor conduction block was present in 23 of 30 UNE patients with PSW/FB



FP – fibrillation potential; **PNFH** – peroneal neuropathy at the fibular head; **PSW** – positive sharp wave; **RN** – radial neuropathy at the spiral groove; **UNE** – ulnar neuropathy at the elbow.
The rate of conduction block was higher in UNE patients with PSWs/FPs than in those without PSWs/FPs ($p < 0.001$, Fisher's exact test).

Fig. 1. Motor conduction block in patients with UNE, RN, and PNFH with and without PSWs/FPs

in at least one muscle ($p < 0.001$). Comparisons of findings from the nerve conduction study and MRC scores of the ADQ/FDI muscles in UNE patients with and without PSWs/FBs are shown in Tab. 2. Dorsiflexion and eversion MRC scores in PNFH patients were 2.9 ± 1.3 (range 0–5) and 3.2 ± 1.5 (range 0–5), respectively. Finger extension, wrist extension, and brachioradialis muscle MRC scores of RN patients were 2.0 ± 1.5 (range 0–4), 1.9 ± 2.0 (range 0–4), and 2.9 ± 1.6 (range 1–4), respectively. A total of 24 PNFH patients and six RN patients recovered completely after repeated neurological examinations over a 5-month period. Follow-up neurological examinations of UNE patients were not available in our laboratory archive. Tab. 3 shows the correlations between MRC scores, the severity of PSWs/FPs, and CMAP amplitude reduction.

DISCUSSION

CB is characterised by impaired impulse conduction along the nerve axon. CB is observed in nerve conduction studies as the CMAP amplitude and area obtained by stimulation of the nerve at the proximal region decreases in comparison with the CMAP amplitude obtained by the stimulation of the nerve at the distal region (American Association of Electrodiagnostic Medicine; Olney, 1999; Oh et al., 1994). PSWs and FPs can be seen in entrapment mononeuropathies in addition to CB. This is caused by peripheral nerve denervation. PSWs and FPs found in a mononeuropathy process without demyelination may be associated with a poor prognosis (Gilchrist and Sachs, 2004). In such cases, aggressive treatments may be an option. However, PSWs and FPs found in entrapment mononeuropathy may also be associated with CB (Katirji, 1999). The high rate of CB in UNE, RN, and PNFH patients with PSWs/FPs found in

Electrodiagnostic or clinical parameter	**UNE patients with PSWs/FPs** Mean ± *SD* (min–max) *n* (number)	**UNE patients without PSWs/FPs** Mean ± *SD* (min–max) *n* (number)	***p* value**
Ulnar nerve CMAP amplitude reduction in percentage across elbow (ADQ) [%]	41.9 ± 35.0 (0–97.7) *n* = 30	7.6 ± 16.5 (0–85.8) *n* = 37	<0.001*
Ulnar nerve CMAP amplitude reduction in percentage across elbow (FDI) [%]	46.6 ± 36.1 (1.1–98.0) *n* = 23	10.4 ± 16.5 (0–89.3) *n* = 32	<0.001*
Ulnar nerve motor NCV across elbow (ADQ) [m/s]	37.0 ± 11.2 (14–59) *n* = 30	48.0 ± 7.5 (33–65) *n* = 37	<0.001*
Ulnar nerve motor NCV across elbow (FDI) [m/s]	37.0 ± 9.9 (18–58) *n* = 23	47.8 ± 10.0 (33–69) *n* = 32	<0.001*
Distal ulnar nerve CMAP amplitude (ADQ) [mV]	9.8 ± 3.7 (0.7–15.4) *n* = 30	12.3 ± 3.3 (6.4–18) *n* = 37	0.029*
Distal ulnar nerve CMAP amplitude (FDI) [mV]	11.5 ± 6.0 (2.2–25.4) *n* = 23	16.4 ± 6.8 (6.4–25.5) *n* = 32	0.017*
Ulnar nerve SNAP amplitude across the 5th digit-wrist segment [μV]	7.1 ± 5.3 (0–15) *n* = 30	7.9 ± 5.3 (0–18) *n* = 37	0.904*
Symptom duration day	82.7 ± 57.1 (21–170) *n* = 30	102.5 ± 55.8 (21–180) *n* = 37	0.072*
MRC score of ADQ muscle	4.1 ± 1.0 (2–5) *n* = 30	4.9 ± 0.4 (4–5) *n* = 37	<0.001*
MRC score of FDI muscle	4.0 ± 1.0 (2–5) *n* = 30	4.8 ± 0.5 (3–5) *n* = 37	<0.001*

* Mann–Whitney *U* test.
ADQ – abductor digiti quinti; **CMAP** – compound muscle action potential; **FP** – fibrillation potentials; **MRC** – Medical Research Council scale; ***n*** – number; **NCV** – nerve conduction velocity; **PSW** – positive sharp waves; ***SD*** – standard deviation; **SNAP** – sensory nerve action potential; **UNE** – ulnar neuropathy at the elbow.

Tab. 2. Comparison of electrodiagnostic/clinical findings between UNE patients with and without PSWs/FPs

 DOI: 10.15557/AN.2022.0007

MRC score		Severity of PSWs/FPs		CMAP reduction in percentage [%]	
UNE patients		**ADQ**	**FDI**	**ADQ**	**FDI**
ADQ*	*p* value/R	0.956/0.012	0.063/−0.362	<0.001/−0.490	0.001/−0.587
	n	24	27	67	55
FDI*	*p* value/R	0.796/−0.056	0.025/−0.429	<0.001/−0.499	0.001/−0.572
	n	24	27	67	55
ADQ†	*p* value/R	<0.001/−0.662	<0.001/−0.499		
	n	67	67		
FDI†	*p* value/R	<0.001/−0.672	<0.001/−0.512		
	n	67	67		
RN patients		**EIP**	**EDC**	**EIP**	
Finger dorsiflexion	*p* value/R	0.680/−0.174	0.359/−0.376	0.352/−0.381	
	n	8	8	8	
Wrist dorsiflexion	*p* value/R	0.771/−0.123	0.553/−0.248	0.080/−0.652	
	n	8	8	8	
PNFH patients		**EDB**	**TA**	**EDB**	**TA**
Dorsiflexion of foot	*p* value/R	0.888/0.028	0.963/−0.009	0.001/−0.609	0.007/−0.507
	n	27	27	26	27
Eversion of foot	*p* value/R	0.479/0.142	0.286/−0.213	0.002/−0.569	0.002/−0.574
	n	27	27	26	27

Spearman correlation test was used. * When the severity of PSWs/FPs and MRC scores were correlated, patients with PSWs/FPs were considered. † When the severity of PSWs/FPs and MRC scores were correlated, UNE patients with and without PSWs/FPs were considered together.
ADQ – abductor digiti quinti; **CMAP** – compound muscle action potential; **EDB** – extensor digitorum brevis; **EDC** – extensor digitorum communis; **EIP** – extensor indicis proprius; **FDI** – first dorsal interosseous; **FP** – fibrillation potential; **MRC** – Medical Research Council scale; **NCV** – nerve conduction velocity; **PL** – peroneus longus; **PNFH** – peroneal neuropathy at the fibular head; **PSW** – positive sharp wave; **RN** – radial neuropathy at the spiral groove; **SNAP** – sensory nerve action potential; **TA** – tibialis anterior; **UNE** – ulnar neuropathy at the elbow.

Tab. 3. Correlation between MRC scores and severity of PSWs-FPs/CMAP amplitude reduction in patients

this study may support this conclusion. This may be due to the loss of a few axons in the area of severe demyelination (Katirji, 1999). If the PSWs and FPs found in entrapment mononeuropathy are due to CB, the degeneration process of the axons and these active denervation findings will resolve with the disappearance of the CB. Thus, the muscle weakness of the patient will improve. The finding in our study that there was a relationship between motor CB and muscle strength in UNE and PNFH patients supports this situation. This may imply that CB is one of the parameters that can be used in the follow-up of patients. Such a relationship in RN patients was not found in our study, which can be explained by the low number of RN patients.

Some argue that if PSWs/FPs continue after the motor CB is eliminated, this suggests that these active denervation findings are not dependent on CB; however, it is known that PSWs and FPs can persist for months (Kim et al., 2011). For these reasons, using active denervation findings in electrodiagnostic classifications or when making treatment decisions may be inconvenient. It seems more appropriate to use the findings obtained from clinical symptoms, nerve conduction studies, and imaging methods. Imaging methods may be useful in determining the aetiology and treatment. It is known that entrapment mononeuropathies may develop due to masses that can be detected by imaging methods (Dong et al., 2012; Hobson-Webb and Juel, 2017). UNE in the retroepicondylar groove often exhibits features of demyelination, while UNE in the humeroulnar aponeurotic arcade often shows features of axonal degeneration (Omejec and Podnar, 2015, 2016). The reduction in SNAP and CMAP amplitudes observed in nerve conduction studies may also be evidence of axonal degeneration (Katirji, 1999). For all these reasons, when making a treatment decision for entrapment mononeuropathies, not only PSWs/FPs, but also clinical findings, electrodiagnostic tests, and imaging results of the patient should be evaluated together.

The number of patients without PSWs/FPs was available in UNE relative to other patient groups. This may be related to the time when electrodiagnostic tests were performed in UNE patients (Gooch and Weimer, 2007). However, the interval between the onset of the symptoms and the time when the electrodiagnostic tests were performed was not different between UNE patients with and without PSWs/FPs. When UNE patients were divided into two groups, i.e. those with and those without PSWs/FPs, it was found that UNE with PSWs/FPs had slower ulnar motor NCV and CMAP amplitude reduction was more pronounced across the elbow segment than among the patients without PSWs/FPs. This observation may suggest that the development of PSWs/FPs is associated with CB. Electrodiagnostic findings supporting demyelination were statistically more abundant in the UNE group with PSWs/FPs; however, CMAP amplitudes were lower in the UNE group with PSWs/FPs, albeit it was statistically less pronounced. The findings suggest

DOI: 10.15557/AN.2022.0007 AKTUALN NEUROL 2022, 22 (2), p. 67–74

that PSWs/FPs found in UNE are due to both axonal degeneration and secondary axonal degeneration due to CB. This supports the idea that some axons are damaged in areas of severe demyelination (Katirji, 1999). In the presence of severe demyelination, the degree of conduction block will increase, and the NCV will slow. This is consistent with the finding in this study that motor CB is more common and more severe across the elbow segment in UNE patients with PSWs/FPs. Unfortunately, there were no patients without PSWs/PPs among RN and PNFH patients. On the other hand, the complete recovery of the majority of RN and PNFH patients, similar to UNE, may suggest that demyelination is more prominent than axonal degeneration in RN, and PNFH (Katirji, 1999; Wang and Weiss, 2013). Some may argue that these findings show that PSWs/FPs indicate a good prognosis in entrapment mononeuropathies. Interestingly, it was found in some studies that EMG abnormalities observed in radiculopathy may be associated with a good prognosis (Rigler and Podnar, 2007; Savage et al., 2015). We do not know whether a similar situation will occur in entrapment mononeuropathies, but it should always be remembered that PSWs/FPs may be due to axonal degeneration with a poor prognosis.

In this study, we also performed a correlation analysis of MRC and the PSWs/FPs severity scores. We conducted the correlation assessment in all UNE patients with and without PSWs/FPs separately, due to the absence of PSWs/FPs in some UNE patients. When all UNE patients were included in the correlation analysis, an inverse correlation was found between MRC and PSWs/FPs severity scores. This situation can be explained by the treatment of symptoms as a result of the disappearance of denervation in patients without PSWs/FPs (PSWs/FPs severity in these patients was included in the analysis as "0"). However, when UNE patients with PSWs/FPs were included in the correlation analysis, we found no correlation between the PSWs/FPs severity and MRC scores. All PNFH and RN patients had PSWs/FPs in at least one muscle, and we found similar correlation results in these entrapment neuropathies. The findings may indicate that there is no correlation between the severity of these spontaneous discharges and muscle strength in entrapment mononeuropathy patients with PSWs/FPs (Masakado et al., 2008).

Another finding of our study was that different muscles were variously affected in needle EMG. There was a higher proportion of PSWs/FPs in the distal muscles of UNE patients, and the TA muscle of PNFH patients compared to other muscles on which EMG was performed, though a limitation was that a wide variety of muscles were left out of the analysis. This finding in UNE, PNFH, and RN can be explained by the topography of the nerve fascicles (Eliaspour et al., 2012). However, considering the low number of RN patients and not applying needle EMG to various muscles such as EDB in PNFH patients, it would be beneficial to confirm this finding in RN and PNFH patients with further studies.

However, the study also has some limitations. The CMAP area and duration, and the temporal dispersion of the nerves are not used for the diagnosis of CB. Amplitude reduction is not a parameter that clearly distinguishes CB from temporal dispersion (Olney and Miller, 1984). We believe that further studies including these parameters would be beneficial. Secondly, the interval between the time when the electrodiagnostic test was performed and the onset of the patients' symptoms ranged from 21 to 180 days. Although this interval was <60 days in most patients, this is also a limitation. PSWs/FPs are strongly related to the duration of mononeuropathy, as we mentioned before (Gooch and Weimer, 2007). Using the reference values of the short segment ulnar motor nerve conduction study across the elbow segment may lead to misdiagnosis, which may be another limitation. However, it should be noted that the clinical findings of the patients were compatible with UNE. Furthermore, this was a retrospective study. Further prospective studies, including patient follow-up, would contribute to a greater understanding of the pathophysiology of PSWs/FPs and CB seen in entrapment mononeuropathies.

CONCLUSIONS

This study showed that PSWs/FPs found in UNE, RN, and PNFH may coexist with CB. Secondary axonal degeneration due to CB and/or axonal degeneration may be the cause of PSWs/FPs, and further studies on this subject may be interesting. Subsequent studies including the follow-up of patients would be beneficial to confirm the findings obtained in this study. In entrapment mononeuropathies with PSWs and FPs, aggressive treatments such as surgical procedures may not be required. While selecting a treatment, the findings of clinical, electrodiagnostic tests and imaging tests should be evaluated together. In contrast to the strong correlation between muscle strength and CB severity in entrapment mononeuropathies, the findings in this study may indicate that there is no correlation between the severity of these spontaneous discharges and muscle strength in entrapment mononeuropathy patients with PSWs/FPs.

Conflict of interest

The authors have no conflicts of interest to declare.

Acknowledgements

None.

Ethical approval

Ethics committee approval was received from the Adana City Training and Research Hospital (ACTRH) Ethics Committee (Decision No.: 72/1182). The procedures applied to all human participants were in accordance with the ethical standards of the ACTRH ethics committee and the 1964 Helsinki declaration and its later amendments.

References

American Association of Electrodiagnostic Medicine; Olney RK: Guidelines in electrodiagnostic medicine. Consensus criteria for the diagnosis of partial conduction block. Muscle Nerve Suppl 1999; 8: S225–S229.

Bowley MP, Doughty CT: Entrapment neuropathies of the lower extremity. Med Clin North Am 2019; 103: 371–382.

Chen S, Andary M, Buschbacher R et al.: Electrodiagnostic reference values for upper and lower limb nerve conduction studies in adult populations. Muscle Nerve 2016; 54: 371–377.

Daube JR, Rubin DI: Needle electromyography. Muscle Nerve 2009; 39: 244–270.

Dong Q, Jacobson JA, Jamadar DA et al.: Entrapment neuropathies in the upper and lower limbs: anatomy and MRI features. Radiol Res Pract 2012; 2012: 230679.

Doughty CT, Bowley MP: Entrapment neuropathies of the upper extremity. Med Clin North Am 2019; 103: 357–370.

Eliaspour D, Sedighipour L, Hedayati-Moghaddam MR et al.: The pattern of muscle involvement in ulnar neuropathy at the elbow. Neurol India 2012; 60: 36–39.

Fidancı H, Öztürk İ, Köylüoğlu AC et al.: Bilateral nerve conduction studies must be considered in the diagnosis of sciatic nerve injury due to intramuscular injection. Neurol Sci Neurophysiol 2020a; 37: 94–99.

Fidancı H, Öztürk İ, Köylüoğlu AC et al.: The needle electromyography findings in the neurophysiological classification of ulnar neuropathy at the elbow. Turk J Med Sci 2020b; 50: 804–810.

Gilchrist JM, Sachs GM: Electrodiagnostic studies in the management and prognosis of neuromuscular disorders. Muscle Nerve 2004; 29: 165–190.

Gooch CL, Weimer LH: The electrodiagnosis of neuropathy: basic principles and common pitfalls. Neurol Clin 2007; 25: 1–28.

Hobson-Webb LD, Juel VC: Common entrapment neuropathies. Continuum (Minneap Minn) 2017; 23: 487–511.

Katirji B: Peroneal neuropathy. Neurol Clin 1999; 17: 567–591.

Kim SJ, Kim WR, Kim HS et al.: Abnormal spontaneous activities on needle electromyography and their relation with pain behavior and nerve fiber pathology in a rat model of lumbar disc herniation. Spine (Phila Pa 1976) 2011; 36: E1562–E1567.

Kleyweg RP, van der Meché FG, Schmitz PI: Interobserver agreement in the assessment of muscle strength and functional abilities in Guillain-Barré syndrome. Muscle Nerve 1991; 14: 1103–1109.

Landau ME, Campbell WW: Clinical features and electrodiagnosis of ulnar neuropathies. Phys Med Rehabil Clin N Am 2013; 24: 49–66.

Lundborg G, Dahlin LB: Anatomy, function, and pathophysiology of peripheral nerves and nerve compression. Hand Clin 1996; 12: 185–193.

Masakado Y, Kawakami M, Suzuki K et al.: Clinical neurophysiology in the diagnosis of peroneal nerve palsy. Keio J Med 2008; 57: 84–89.

Oh SJ: Nerve conduction in focal neuropathies. In: Oh SJ: Clinical Electromyography: Nerve Conduction Studies. 3rd ed., Lippincott Williams & Wilkins, Philadelphia 2003: 231–234.

Oh SJ, Kim DE, Kuruoglu HR: What is the best diagnostic index of conduction block and temporal dispersion? Muscle Nerve 1994; 17: 489–493.

Olney RK, Miller RG: Conduction block in compression neuropathy: recognition and quantification. Muscle Nerve 1984; 7: 662–667.

Omejec G, Podnar S: Precise localization of ulnar neuropathy at the elbow. Clin Neurophysiol 2015; 126: 2390–2396.

Omejec G, Podnar S: Proposal for electrodiagnostic evaluation of patients with suspected ulnar neuropathy at the elbow. Clin Neurophysiol 2016; 127: 1961–1967.

Rigler I, Podnar S: Impact of electromyographic findings on choice of treatment and outcome. Eur J Neurol 2007; 14: 783–787.

Savage NJ, Fritz JM, Kircher JC et al.: The prognostic value of electrodiagnostic testing in patients with sciatica receiving physical therapy. Eur Spine J 2015; 24: 434–443.

Wahab KW, Sanya EO, Adebayo PB et al.: Carpal tunnel syndrome and other entrapment neuropathies. Oman Med J 2017; 32: 449–454.

Wang LH, Weiss MD: Anatomical, clinical, and electrodiagnostic features of radial neuropathies. Phys Med Rehabil Clin N Am 2013; 24: 33–47.

DOI: 10.15557/AN.2022.0007