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Evaluation of the function status of the ulnar nerve in carpal tunnel syndrome

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ABSTRACT. Many carpal tunnel syndrome (CTS) patients have symptoms in both the median and ulnar digits more frequently than in the median digits alone. This is possibly because of close anatomical contiguity of the carpal tunnel and Guyon's canal, and the high pressure may also affect the latter, causing indirect compression of ulnar nerve fibers. Thus, we evaluated the functional status of the ulnar nerve in patients with CTS in order to investigate the relationship between ulnar nerve impairment and sensory symptoms of the ulnar territory. Electrophysiological studies were conducted in CTS patients and healthy controls. CTS patients were divided into the mild/moderate group and severe group; they were further divided into the symptomatic and asymptomatic subgroups according to the sensory symptom of the fifth digit region. The findings suggest that CTS patients could have coexisting ulnar nerve wrist entrapments that might exacerbate the severity of CTS. Sensory impairment in the ulnar territory was observed more frequently in the mild/moderate stage of CTS, which is associated with ulnar nerve involvement. These findings also suggest that damage

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to the ulnar nerve fibers caused by compression forces in Guyon's canal may underlie the ulnar spread of symptoms in CTS.

Key words: Carpal tunnel syndrome; Ulnar nerve; Nerve conduction; Electromyography

INTRODUCTION

Carpal tunnel syndrome (CTS) is caused by increased pressure and the consequent compression of the median nerve within the carpal tunnel. Clinical symptoms include numbness, tingling, burning, and pain in the median nerve distribution (Werner, 2013). However, many CTS patients have symptoms in both the median and ulnar digits more frequently than in the median digits alone (Zanette et al., 2006, 2010). It is reported that 42% of sensory symptoms spread to the ulnar nerve territory (Ginanneschi et al., 2008a). The close anatomical contiguity between the carpal tunnel and Guyon's canal may suggest that the pathologic processes that cause CTS might also affect the ulnar nerve and may thus explain the extra-median spread of sensory symptoms (Ginanneschi et al., 2008a; Vargas et al., 2013). The aim of our study was to investigate involvement of ulnar nerve to obtain insights into the pathogenesis of the extra-median spread of sensory symptoms frequently reported in CTS patients. Accordingly, this study evaluated ulnar nerve function in CTS by using a neurophysiological approach as well as analyze the relationship between ulnar nerve impairment and sensory symptoms in the ulnar territory.

MATERIAL AND METHODS

Subjects and classification

The study subjects were 55 CTS patients from neurology or orthopedics outpatient clinic in our hospital from November 2011 to October 2012. All patients had CTS diagnosed according to the 2002 criteria of the American Academy of Neurology (American Association of Electrodiagnostic Medicine et al., 2002). CTS patients were divided into the mild/moderate group (N = 35) and severe group (N = 20) according to the Stevens standard (Stevens, 1997); their mean ages were 54.4 ± 5.3 and 53.1 ± 4.8 years, respectively. Each group was further subdivided into the symptomatic and asymptomatic subgroups according to the presence of sensory symptoms in the fifth digit region. Thus, there were 23 and 12 symptomatic and asymptomatic patients in the mild/moderate group, and 2 and 18 symptomatic and asymptomatic patients in the severe group, respectively. Patients had no clinical or electrophysiological signs of pathological conditions such as thoracic outlet syndrome, myelopathy, polyneuropathy, radiculopathy, myopathy, and other neurological diseases; disorders predisposing them to CTS (e.g., wrist fracture, pregnancy, diabetes, etc.); or Martin-Gruber connection. The control group included 20 age- and gender-matched healthy subjects (6 males and 14 females; mean age, 53.7 ± 5.4 years; range, 38-65 years) The study was approved by the Ethics Committee of the our hospital, and all subjects (controls and patients) gave their informed consent to participate in the study.

Neurophysiological evaluation

Motor conduction studies

During all testing, patients were in the supine position with their eyes closed. Tests were performed in a quiet room with a temperature of 22°-25°C. We investigated the conduction of the median, ulnar, and dorsal ulnar cutaneous nerves. Neurophysiological examination was performed by using the Keypoint.net (Medoc Ltd., Ramat Yishai, Israel) electromyogram device. The distribution of sensory symptoms of patients were recorded simultaneously.

Compound muscle action potentials (CMAPs) were recorded by surface electrodes placed over the motor points of the abductor digiti minimi and abductor pollicis brevis muscles for the ulnar and median nerves, respectively. Motor nerve conduction velocities, distal motor latencies (DMLs), and CMAP amplitudes were calculated. DML was measured from the negative take-off, and CMAP amplitude was defined as the height from the baseline to the first negative peak of the action potential. The median nerve was stimulated 7 cm from the active electrode to the tendons of the flexor carpi radialis and palmaris longus muscles at the wrist and elbow for the DML test.

Sensory conduction studies

Sensory nerve conduction velocity (SCV) and sensory nerve action potential (SNAP) amplitudes were recorded antrorsely by ring electrodes from the first and third digits for the median nerve and from the fifth digit for the ulnar nerve. Sensory nerve conduction of the dorsal ulnar cutaneous branch was also examined by using the standard antidromic technique: the surface active recording electrode was placed on the dorsum of the hand between the fourth and fifth metacarpal bones, and the reference electrode was placed on the proximal phalanx of the fifth digit. The nerve was stimulated at the wrist 8 cm from the active electrode between the ulnar bone and tendon of flexor carpi ulnaris muscle.

Electromyography

In CTS subjects, needle electromyography of the abductor digiti minimi and abductor pollicis brevis muscles was performed to exclude C8 and T1 radicular involvement. Nerve conduction studies of the lower extremities (i.e., tibial and peroneal nerve conduction studies included motor and sensory evaluation) were performed to exclude polyneuropathies. The normal values refer to the Cui standard (Cui, 2006).

Statistical analysis

Statistical analysis was performed by using SPSS version 17.0 (SPSS Inc., Chicago, IL, USA). Data are reported as mean ± SD. Comparisons between groups were made using independent sample *t*-tests. The level of significance was set at $P < 0.05$.

RESULTS

Conduction parameters of the median nerve

In the mild/moderate group, compared to the control group, the mean first digit-wrist and third digit-wrist SNAP and SCV were significantly lower and DML was more prolonged ($P < 0.01$). However, CMAP amplitude was not significantly different between groups ($P > 0.05$). Compared to the mild/moderate group, the severe group had slower SCV, prolonged DML of the ulnar nerve, and reduced SNAP and CMAP amplitudes ($P < 0.01$).

Conduction parameters of the ulnar nerve

Compared to the control group, the mild/moderate group had a significantly slower fifth digit-wrist SCV of the ulnar nerve ($P < 0.01$). However, the SNAP amplitude did not differ between groups. Compared to the mild/moderate group, the severe group had significantly slower fifth digit-wrist SCV of the ulnar nerve and reduced SNAP amplitude ($P < 0.01$).

However, there were no significant differences among the control, mild/moderate, and severe groups with respect to DML, CMAP amplitude, motor nerve conduction velocities above the elbow/wrist, SCV, or SNAP amplitude of the dorsal cutaneous ulnar nerve ($P > 0.05$; Table 1).

Table 1. Median and ulnar nerve conduction results of carpal tunnel syndrome patients and controls.

Group	Mild/moderate group (N = 35)	Severe group (N = 20)	Control group (N = 20)
Mean age (years)	54.4 ± 5.3	53.1 ± 4.8	53.7 ± 5.4
Median nerve			
DML (ms)	3.97 ± 0.24*	5.22 ± 0.86*	3.37 ± 0.26
CMAP (mV)	10.75 ± 1.78*	5.06 ± 1.17*	11.18 ± 1.93
MCV (wrist-elbow)	57.32 ± 3.16	57.00 ± 3.27	58.26 ± 3.40
M1 SNAP (μV)	9.57 ± 2.13*	5.31 ± 2.60*	18.13 ± 2.09
M1 SCV (m/s)	33.00 ± 5.96*	27.02 ± 5.53*	52.89 ± 3.65
M3 SNAP (μV)	7.81 ± 2.28*	3.90 ± 1.32*	16.44 ± 1.39
M3 SCV (m/s)	34.46 ± 7.19*	26.36 ± 4.98*	54.58 ± 3.22
Ulnar nerve			
DML (ms)	2.66 ± 0.23	2.67 ± 0.23	2.59 ± 0.23
CMAP (mV)	11.95 ± 1.51	12.22 ± 1.44	12.67 ± 1.06
MCV (wrist-elbow)	63.04 ± 3.07	62.24 ± 4.16	63.58 ± 3.79
U5 SNAP (μV)	14.09 ± 2.10*	12.61 ± 1.67*	14.35 ± 1.74
U5 SCV (m/s)	52.32 ± 2.81**	50.63 ± 2.90**	58.62 ± 3.21
Dorsal cutaneous ulnar nerve			
SNAP (μV)	17.51 ± 2.13	17.78 ± 2.31	18.39 ± 2.85
SCV (m/s)	54.71 ± 1.76	53.98 ± 2.56	55.11 ± 1.84

Data are reported as menas ± SD. DML, distal motor latency; CMAP, compound muscle action potential; MCV, motor conduction velocity; SNAP, sensory nerve action potential; SCV, sensory nerve conduction velocity., M1, median nerve, first digit-wrist; M3, median nerve, second digit-wrist; U5, ulnar nerve, fourth digit-wrist. *$P < 0.01$, **$P < 0.05$.

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Conduction parameters of the ulnar nerve in CTS patients with respect to symptoms in the fifth digit

Twenty-five CTS patients (45.4%) had paresthesia in the fifth digit in addition to symptoms in the median nerve innervation area. Among them, 23 (65.7%) and 2 (10%) were mild/moderate and severe CTS patients, respectively.

Compared to the asymptomatic subgroup, the symptomatic mild/moderate subgroup had significantly slower ulnar nerve wrist-fifth digit SCVs and lower SNAP amplitude ($P < 0.01$). However, there were no significant differences between these subgroups with respect to ulnar nerve wrist-abductor digiti minimi DMLs or CMAP amplitude (Table 2).

Table 2. Conduction parameters of the ulnar nerve in mild/moderate carpal tunnel syndrome patients with respect to symptoms in the little finger.

Group	N	DML (ms)	CMAP (mV)	U5 SNAP (μV)	U5 SCV (m/s)
Symptomatic	12	2.68 ± 0.24	11.66 ± 1.35	13.51 ± 1.84	51.59 ± 2.70
Asymptomatic	12	2.63 ± 0.20	12.50 ± 1.72	15.21 ± 2.16	53.72 ± 2.58
P		0.57	0.12	0.02	0.03

Data are reported as means ± SD. For abbreviations, see Table 1.

DISCUSSION

CTS is a neuropathy caused by the entrapment of the median nerve at the level of the carpal tunnel; its symptoms typically follow the median nerve distribution, but sensory symptoms involving the ulnar nerve territory and symptoms affecting the whole hand are frequently reported as well (Nora et al., 2004, 2005). Ulnar nerve territory symptoms can be attributed to concomitant ulnar nerve entrapment in CTS cases with neurologic signs of ulnar damage (Gozke et al., 2003). At present, CTS patients associated with ulnar nerve involvement do not receive sufficient attention. Nevertheless, a neurophysiological approach is the most sensitive method for evaluating peripheral nerve compression damage (Claes et al., 2013). In the present study, we classified the CTS severity of all patients including those with and without fifth digit symptoms by using neurophysiological methods. Thus, we evaluated the functional status of the ulnar nerve to elucidate the possible pathogenic mechanisms of ulnar nerve territory symptoms.

Compared to the control group, the mild/moderate group had a significantly slower fifth digit-wrist SCV of the ulnar nerve; however, SNAP amplitude did not differ significantly. Meanwhile, compared to the mild/moderate group, the severe group had further slowed fifth digit-wrist SCV of the ulnar nerve and reduced SNAP amplitude; however, there were no significant differences among the 3 groups with respect to DML, CMAP amplitude, or the motor nerve conduction velocities above the elbow/wrist, or the SCV, or SNAP amplitude of the dorsal cutaneous ulnar nerve. These results indicate that ulnar nerve sensory conduction impairment occurs during the early stages of CTS and increases with the increasing severity of median nerve involvement. Furthermore, involvement of the ulnar digital nerve branches without the involvement of the dorsal ulnar cutaneous branch, which does not pass through Guyon's canal, suggests the location of damage is Guyon's canal. As the floor of Guyon's canal is anatomically linked to the ulnar portion of the roof of the carpal tunnel, it is very likely that compressive forces, which are due to well-documented high carpal tunnel pressure in CTS, are transmitted to Guyon's canal and then to the ulnar nerve (Ginanneschi et al., 2008b). The volume and pressure of Guyon's canal are elevated in CTS patients, while pres-

sure decreases after carpal tunnel release (Ablove et al., 1996), supporting this hypothesis. In addition, Mondelli et al. (2009) demonstrate an improvement in conduction values in sensory ulnar fibers in some patients with CTS after carpal tunnel release. This further supports the hypothesis that in CTS, ulnar fibers may be subjected to compressive forces in Guyon's canal owing to the high pressure in the carpal tunnel.

Second, in order to study the relationship between ulnar nerve territory sensory symptoms and ulnar nerve involvement, each group was divided into symptomatic and asymptomatic subgroups according to the presence of sensory symptoms in the fifth digit region. Nearly half of the CTS patients (25, 45.4%) had paresthesia in the fifth digit in addition to symptoms in the median nerve innervation area; among them, 23 and 2 had mild/moderate and severe CTS, respectively. Previous reports indicate that an extra-median spread of symptoms is more frequent in the mild than in severe stage of CTS (Caliandro et al., 2006; Zanette et al., 2006). Moreover, severe CTS is more often associated with the typical median distribution.

In the mild/moderate group, ulnar nerve wrist-fifth digit SCVs were significantly slower and SNAP amplitudes were significantly lower in the symptomatic group than in the asymptomatic group. Therefore, compression of the ulnar nerve at the wrist may explain the ulnar spread of sensory symptoms in mild/moderate CTS patients. The present results are similar to those of Yemisci et al. (2011). Ablove et al. (1996) report that the volume and pressure in Guyon's canal are elevated in CTS patients and that spontaneous relief of ulnar nerve symptoms occurs after isolated carpal tunnel release. These findings suggest the ulnar nerve abnormalities observed may be attributable to functional (i.e., axonal membrane potential and ion channel changes) rather than morphological (i.e., focal nerve demyelination) factors (Ablove et al., 1996). Some studies (Tecchio et al., 2002) suggest the extra-median spread of symptoms is associated with higher levels of pain and paresthesia, and that central nervous system mechanisms of plasticity probably underlie the spread of symptoms in CTS patients. Tecchio et al. (2002) report evidence of enlargement of the hand representation in the sensory cortex in CTS patients with an all-digit distribution. The present results corroborate this finding.

In the severe group, only 2 patients had paresthesia in the fifth digit; however, ulnar nerve involvement was more serious in this group. The relief of ulnar nerve symptoms in severe CTS patients may be explained as follows. In early CTS, ectopic discharge from damaged ulnar sensory afferents plays a dominant role in conditioning extra-median spread of symptoms. In more severe CTS, central mechanisms may suppress or mask ulnar symptoms; for example, chronic or tonic stimulation of nociceptive afferents of the median nerve could exert some gating of sensory processing from low-threshold ulnar fibers, which are responsible for ectopic discharge. This may explain why the sensory symptoms in the ulnar territory improve after local anesthesia of the median nerve at the wrist in patients with severe CTS (Rossi et al., 2003).

To sum up, the present study documents a significant change in ulnar nerve conduction in the mild/moderate stage. The most plausible hypothesis seems to be damage to ulnar fibers by compressive forces in Guyon's canal as a consequence of high pressure in the carpal tunnel. However, some limits of the present study are worth mentioning. Skin thickness and temperature variaions in the hand may be potential confounding variables. In furthermore, the observed changes in ulnar nerve sensory conduction would presumably be reflected by ulnar DML. Possible reasons for this are that minimal impairment of ulnar nerve motor fibers may not be detectable by standard electrophysiology and that biophysical differences in the properties of sensory and motor axons could account for a lower susceptibility of motor fibers to compression compared to sensory fibers.

CONCLUSION

These results suggest that the pathological processes leading to median nerve entrapment in CTS patients may also affect ulnar nerve sensory fibers in Guyon's canal as well as ulnar nerve impairment increased as a result of the elevated severity of median nerve involvement. Furthermore, the ulnar spread of symptoms is more frequent in the mild/moderate stage. Therefore, compression of the ulnar nerve at the wrist may explain the ulnar spread of sensory symptoms in mild/moderate CTS patients.

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