Chapter 7

Ultrasonography in severe carpal tunnel syndrome

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Abstract

Introduction – In up to 30% of patients with carpal tunnel syndrome (CTS), the cross-sectional area (CSA) of the median nerve may not be enlarged. We hypothesize that this could be the result of secondary atrophy of the nerve in severe CTS. The aim of this study is to measure the ultrasonographic CSA of the median nerve at the wrist in patients with severe CTS.

Methods – In 14 consecutive patients with clinically and electrophysiologically defined severe CTS, the CSA of the median nerve was measured and compared with controls.

Results – CSA of the median nerve exceeds the upper limit of normal in the majority of patients with severe CTS.

Discussion – Atrophy of the median nerve in severe CTS does not explain negative ultrasonographic test results. Instead, the CSA of the median nerve is enlarged in most patients with severe CTS.
Introduction

Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy. Usually, the clinical symptoms are very characteristic, which permits a reliable clinical diagnosis. However, many surgeons require electrodiagnostic testing in order to confirm the diagnosis; this has a sensitivity of 85% and specificity of 95%. Electrodiagnostic testing may, however, be perceived as very unpleasant by many patients. With respect to this, ultrasonography of the median nerve, which is emerging as a diagnostic test in CTS, has advantages over electrodiagnostic studies: it is painless, and it also gives anatomical and morphological information about the carpal tunnel and the nerve itself. An abnormally enlarged cross-sectional area of the median nerve is a characteristic ultrasonographic finding in CTS and has been reported to have a sensitivity and specificity of 70 to 82% and 57 to 96%, respectively. Besides enlarged cross-sectional area, other findings can include flattening at the level of the hook of the hamate and an increase in cross-sectional area at the level of the pisiform bone compared to the cross-sectional area in the forearm.

In up to 30% of patients with clinically defined CTS and electrophysiologically confirmed CTS, however, the cross-sectional area at the level of the pisiform bone is not enlarged. In an old autopsy study of a patient with CTS accompanied by weakness and muscle atrophy, a reduction in fiber size under the flexor retinaculum was found. We hypothesize that in patients with severe CTS, the cross-sectional area of the median nerve is not enlarged; it may, in fact, be reduced because of secondary atrophy of the median nerve at the carpal tunnel after severe axonal damage.

The aim of this study is to measure the ultrasonographic cross-sectional area of the median nerve at the carpal tunnel in patients with severe carpal tunnel syndrome in order to test this hypothesis.

Materials and Methods

Subjects

In this observational cohort study, 14 consecutive patients with clinically and electrophysiologically severe carpal tunnel syndrome were included.

Clinically severe carpal tunnel syndrome was considered to be present in patients who had pain and/or paresthesia in and restricted to the sensory distribution of the median nerve, with 2 or more of the following criteria: (1) nocturnal paresthesias, (2) reproduction or aggravation of paresthesias or pain...
by provocative tests (Tinel or Phalen signs), (3) aggravation of paresthesias
by activities such as driving a car, riding a bike, holding a book, or holding a
telephone, and (4) relief of symptoms by shaking the hand. These clinical criteria
have previously been used in other studies. Also, atrophy of the abductor
pollicis brevis muscle had to be present on visual inspection and palpation of
the muscle.

Patients with previous carpal tunnel release or carpal tunnel syndrome
possibly caused by a traumatic injury were excluded from this study.

For inclusion in the study as severe CTS, patients had to have reasonably
strong arguments for the presence of severe axonal damage of the median
nerve. These included visible atrophy of the abductor pollicis brevis muscle
exclusively, as well as the presence of fibrillation potentials and positive sharp
waves and severe reduction or absence of motor unit action potentials after
maximal voluntary effort. Patients with either normal or minimally reduced
compound muscle action potentials (CMAPs) or sensory nerve action potentials
(SNAPs) were excluded.

**Electrodiagnostic evaluation**

All patients underwent standardized motor and sensory nerve conduction
studies (NCS), as well as electromyography (EMG) of the abductor pollicis brevis
muscle in the symptomatic hand, in accordance with our laboratory’s standard
procedure in CTS. EMG and NCS were performed using a Viking Myograph type
IV (Nicolet Biomedical Inc, Madison, WI, USA). Skin temperature was maintained
at a minimum of 31.0°C.

In all sensory nerve conduction studies, the proximal electrode was placed
at the first interphalangeal joint, and the distal recording electrode was placed at
a distance of at least 3 cm, if feasible. Stimuli with a duration of 0.3 msec were
applied.

Sensory nerve conduction studies compare the onset latencies of the
median nerve and the ipsilateral ulnar nerve over the same distance, recorded
from the ring finger. Conduction velocity of the ulnar nerve should be at least
50 m/s. A difference in onset latency more than 0.4 ms or the absence of the
median SNAP is considered to be consistent with CTS. In segmental sensory
conduction studies across the wrist SNAPs were recorded from digit 2 and 3
after stimulation of the median nerve at the palm and at the wrist, where the
distance palm-to-digit is half of the distance between the stimulation sites of the
wrist and digit.
Absence of SNAPs or a difference in conduction velocity between the palm-to-digit and palm-to-wrist greater than 10 m/s, is considered to be consistent with CTS.

Median nerve motor nerve conduction studies were performed by stimulating the median nerve at the wrist and at the cubital fossa. CMAPs were recorded from the thenar eminence by means of surface electrodes at a distance of 6 cm from the stimulation site at the wrist. A distal motor latency of > 4.0 ms is considered to be consistent with CTS.

In addition to our standard protocol, needle electromyography of the abductor pollicis brevis muscle was conducted in every patient with a concentric needle electrode.

**Ultrasonographic evaluation**

Ultrasonographic examination was performed by an experienced electrodiagnostic technician, using a Philips Diagnostic Ultrasound System (model iU22) with a 5-17 MHz linear array transducer. All wrists were examined in neutral position with palm up and fingers semi-extended. The median nerve was visualized about 7 cm proximal to the carpal tunnel in longitudinal and transverse planes in order to confirm identification of the nerve. At the inlet of the carpal tunnel, which is defined as the proximal margin of the flexor retinaculum between the scaphoid tubercle and the pisiform bone, the cross-sectional area (CSA) was measured by means of the direct tracing method. The distal wrist crease was used as an external landmark. The inner margin of the hyperechoic sheath was considered to be the margin of the nerve. In case of a bifid median nerve, the cross-sectional area of the extra branch was added to the cross-sectional area of the main branch, since the size criterion for bifid median nerves is higher than for non-bifid median nerves.\(^7\)

In all patients, the wrist circumference at the distal wrist crease was measured by a marking gauge and a measuring tape with a precision of 1 mm. In case of bilateral symptoms, only the most symptomatic side was examined by ultrasonography. Ultrasonographic examination was performed on the same day as the electrodiagnostic studies. The technician was blinded to the results of the nerve conduction studies and electromyographic examination.

As the population of patients in this study appeared to be rather old, we tested an additional group of 12 healthy persons with comparable age characteristics. This group fitted well within in the reference values for CSA of the original reference group.
Statistical Analysis

Data concerning clinical variables, nerve conduction studies, electromyography and ultrasonography were processed using Microsoft Office Excel and Access 2003, and all statistical analyses were performed using SPSS Statistics 17.0.

Our laboratory reference values were collected previously for nerve conduction studies as well as for quantitative ultrasonography of the median and ulnar nerves. By using the regression equations based on L/R side and circumference of the wrist, we calculated how the observed CSA differed from the mean of normal in order to express this as a Z-score. So, by definition, a Z-score < 2 would be a normal result. The following regression equations were used:

- right side, \( Z_{\text{right}} = \frac{[\text{measured CSA} - 0.86 \times \text{wrist circumference (cm)}]}{1.45} \)
- left side, \( Z_{\text{left}} = \frac{[\text{measured CSA} - 1.06 \times \text{wrist circumference (cm)}]}{1.55} \)

The Mann-Whitney U was used for testing differences between patients and controls.

Results

Study population

In the 14 included patients, gender was equally distributed, and the mean age was 71.8 years (range 52 – 86). The median duration of symptoms was 6.0 months (range 2 – 24). For more detailed demographic and clinical features of the patients see Table 1.

Since the mean age of the patient group was relatively old in comparison to the controls, CSA of the median nerve of 12 asymptomatic elderly people was determined and used as an extra, age-matched, control group.

Electrophysiology

In all patients, fibrillation potentials and/or positive sharp waves were found in the abductor pollicis brevis muscle on needle electromyographic examination. CMAPs were absent in 12 patients and were severely reduced in amplitude (i.e., < 0.6 mV) in 2 patients. Median SNAPs were completely absent in 11 patients.
The overall mean CSA in patients was 17.7 mm$^2$ (SD, 5.22); 17.6 mm$^2$ (SD, 7.23) and 17.8 mm$^2$ (SD, 3.64) for the left and right median nerve respectively. Detailed data on mean CSA and mean $Z$-scores of the CSA are shown in Table 2.

The boxplots in Figure 1 show the spread of the $Z$-scores as well as 95% confidence intervals of the mean of the median nerve CSA in patients with severe CTS as well as in elderly controls. The boxplots in Figure 2 show the spread of the mean CSA as well as 95% confidence intervals, not corrected for wrist circumference.

**Ultrasonography**

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### Table 2. Mean CSA and Z-score of the CSA of the median nerve and 95% confidence intervals in patients and elderly controls

<table>
<thead>
<tr>
<th></th>
<th>Patients (n = 14)</th>
<th>Elderly (n = 12)</th>
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<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>95% CI</td>
</tr>
<tr>
<td>Mean CSA (mm²)</td>
<td>17.7 ± 5.22*</td>
<td>14.7 – 20.7</td>
</tr>
<tr>
<td>Mean CSA left (mm²)</td>
<td>17.6 ± 7.23</td>
<td>9.97 – 25.2</td>
</tr>
<tr>
<td>Mean CSA right (mm²)</td>
<td>17.8 ± 3.64</td>
<td>14.7 – 20.8</td>
</tr>
<tr>
<td>Z-score of the CSA</td>
<td>4.93 ± 3.40*</td>
<td>2.96 – 6.89</td>
</tr>
</tbody>
</table>

* P < 0.001; CI = Confidence Interval
Figure 1. Boxplots: $Z$-scores of CSA of the median nerve. CSA, cross-sectional area

Figure 2. Boxplots: mean CSA of the median nerve. CSA, cross-sectional area
Discussion

Previous studies demonstrated that, in CTS patients, the degree of electrophysiological abnormalities is associated with CSA of the median nerve at the wrist. To our knowledge, no such data have been published with regard to severe CTS with axonal damage.

The main finding of this study is that in severe CTS the CSA of the median nerve clearly exceeds the upper limit of normal in the majority of cases (11 out of 14). This rejects our hypothesis that, in patients with severe CTS, atrophy of the median nerve is not an explanation for the normal ultrasonographic findings. In three cases, the cross-sectional area of the nerve was within normal limits, but not one patient had a cross-sectional area below the lower limit of normal. We do not have a clear explanation for the normal ultrasonographic findings in these cases, but since the values are within normal ranges, one can assume there is still no clear indication for atrophy of the nerve.

In this study, the diagnosis of CTS was based on carefully defined clinical criteria, complemented by extensive electrodiagnostic studies. Reference values are derived from previous research in the same laboratory using the same equipment and performed by the same technician.

In most of the patients, motor and sensory compound action potentials were absent or very low. This may of course be the result of axonal damage. However, conduction block may contribute to this if stimulation is applied proximal to the zone of compression. Only in the case of second and third digit involvement, palmar stimulation can reliably distinguish this cause of low SNAPs. In motor conduction studies, palmar stimulation may be applied, yet this may cause unintentional stimulation of ulnar branches in the palm and may lead to recording CMAPs from ulnar-innervated muscles in the thenar eminence. We did not perform this test.

The rather small sample size is a relative limitation of the study. Nevertheless, differences between groups are statistically significant and numerically relevant.

The finding of the relatively old age of these patients is remarkable. As these ages lie outside our set of reference values, we measured the CSA in 12 healthy controls whose ages matched those of the patient group. The CSA values appeared to be within the range of our reference values. Therefore, the observed large CSA in this group of patients cannot be attributed to old age. From previous reports it is known that severe CTS, accompanied by severe electrophysiological abnormalities, is more frequent in the elderly, and that older patients more often present with severe CTS accompanied by thenar
atrophy.\textsuperscript{13} However, ultrasonographic findings have not been reported.

Another issue worth considering is the occurrence of diabetes among these patients with severe CTS. In one previous study among diabetics, mean calculated CSA was larger in patients with clinical evidence of diabetic polyneuropathy as compared with patients without polyneuropathy.\textsuperscript{14} In our study, however, patients with clinical evidence of polyneuropathy were not included. Moreover, SNAPs of the ulnar nerve were normal in all patients. After exclusion of the 4 diabetics, our results remained essentially the same. Future studies will be necessary to collect reference values in diabetics, in order to use ultrasonography for diagnostic purposes.

In this study, the duration of symptoms ranged from 2 to 24 months (median = 6 months); severe CTS with axonal damage occurred after both short and long-standing complaints.

In conclusion, atrophy of the median nerve in clinically and electrophysiologically severe CTS does not explain negative ultrasonographic test results. Instead, based on these results, we conclude that the CSA of the median nerve is enlarged in most patients with severe CTS.
References


