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Median Nerve Changes Following Steroid Injection for Carpal Tunnel Syndrome

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Abstract

Introduction—Neuromuscular ultrasound is a painless, radiation-free, and high-resolution imaging modality for assessment of the peripheral nervous system. The purpose of this study was to use neuromuscular ultrasound to assess the changes that occur in the median nerve following steroid injection for carpal tunnel syndrome (CTS).

Methods—Ultrasound and nerve conduction studies were performed at baseline and 1 week, 1 month, and 6 months after steroid injection in 19 individuals (29 wrists) with CTS.

Results—Significant changes were noted in median nerve cross-sectional area ($p < 0.001$), mobility ($p = 0.001$), and vascularity ($p = 0.042$) at the distal wrist crease following steroid injection, and the nerve cross-sectional area correlated with symptom score and electrodiagnostic parameters. Changes in the ultrasonographic parameters were seen within one week of injection.

Discussion—These findings suggest neuromuscular ultrasound is potentially helpful for the assessment of individuals undergoing treatment for CTS, as typical changes can be expected following successful treatment.

Keywords

Carpal tunnel syndrome; Ultrasound; Median nerve; Steroid; Injection

Introduction

Carpal tunnel syndrome (CTS) is a common condition in the United States with 300,000 new cases and \$500 million in related costs per year.¹ The exact pathophysiology of CTS is unknown, but it is thought to result from chronic compression of the median nerve as it passes through the rigid carpal tunnel.² Diagnosis is typically made through a combination

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of patient history, clinical examination, and electrodiagnostic studies.³ However, over the past 10 years neuromuscular ultrasound, which demonstrates enlargement of the median nerve proximal to the entrance of the carpal tunnel, has emerged as another method for the diagnosis of CTS.^{4, 5} There are several treatment options for CTS, and local steroid injection into the carpal tunnel has proven to be an effective treatment, providing symptomatic relief for up to 6 months.⁶

This study was designed to assess the ultrasonographic changes (including nerve area, echogenicity, mobility, and vascularity) that occur within the median nerve of those undergoing steroid injection for CTS, with the goal of evaluating two major issues. First, it was thought that the type and timing of ultrasonographic changes observed following steroid injection could improve our understanding of the pathophysiology of CTS. Second, it was thought that knowledge of typical neuromuscular ultrasound changes following successful steroid injection would provide clinicians with key information to assess the median nerves of individuals with failed CTS intervention.

Materials and Methods

Participants

Prior to the collection of data, this study was approved by the Institutional Review Boards at Wake Forest University School of Medicine and Korea University College of Medicine. At their initial visit, all participants provided signed informed consent. A total of 19 individuals (29 affected wrists) with clinical, electrodiagnostic, and ultrasonographic evidence of CTS were recruited at Wake Forest University School of Medicine (9 participants) and Korea University College of Medicine (10 participants) during 2008 and 2009. Study inclusion criteria included numbness or pain in a median nerve distribution, nerve conduction studies consistent with CTS per AANEM guidelines,⁷ and a median nerve cross-sectional area at the wrist greater than 12mm². In addition, either the participants' symptoms were not severe enough to warrant surgery or they had expressed a preference for medical treatment. Exclusion criteria included previous surgical or injection CTS treatment. Each participant completed the Levine-Katz CTS self-assessment questionnaire⁸ and underwent median motor and sensory nerve conduction studies (NCS) and median nerve ultrasonography at baseline (prior to steroid injection) and at 1 week, 1 month, and 6 months after injection. The Levine-Katz CTS self-assessment questionnaire has both a symptom score (a mean score for each hand, which ranges from 1 to 5 with 5 being the most severe symptoms) and a function score (a single mean score, which ranges from 1 to 5 with 5 being the most severe functional limitations).⁸

Injection

At the initial visit, a steroid injection near the median nerve in the affected wrist was administered after obtaining baseline electrical and ultrasonographic measurements. The affected wrists were first cleaned with isopropyl alcohol and superficially injected with a small amount of lidocaine. Then, under ultrasound guidance, a 23-gauge needle was inserted at the proximal wrist crease, just ulnar to the palmaris longus tendon, at a 30-degree angle to the skin and aiming towards the index finger. A combination of 1 cc of lidocaine and 1 cc of triamcinolone acetonide (40mg) was injected into the area surrounding the median nerve.

Nerve Conduction Studies

Nerve conduction studies (NCS) across the affected wrists were performed at baseline and at 1 week, 1 month, and 6 months after injection. The studies were performed with the participant in a supine position using standard surface and ring electrodes and established protocols, and hand temperatures were maintained above 32 degrees Celsius.⁷ During the

initial examination, both median and ulnar motor and antidromic sensory studies were performed. In addition, when necessary, mixed median-ulnar comparison studies and electromyography were performed to clearly establish the diagnosis of CTS. At the follow-up visits, only median motor and antidromic sensory studies to digit 2 were performed.

Ultrasound Studies

The affected wrist was assessed with ultrasound at baseline and at 1 week, 1 month, and 6 months after injection. The ultrasound examination included measurement of the median nerve cross-sectional area at the distal wrist crease (DWC) and mid-forearm, as well as assessment of median nerve echogenicity, mobility, and vascularity at the DWC. Ultrasound imaging of the median nerve was performed using an Esaote Biosound MyLab 25 (Esaote Group, Genoa, Italy) ultrasound device with an 18 MHz linear-array transducer at Wake Forest University and a Philips ATL HDI 3500 (Philips Medical Systems, Bothell, WA, USA) ultrasound device with a 12 MHz linear-array transducer at Korea University. Only one ultrasonographer at each center performed all the studies (MSC at Wake Forest University and JSY at Korea University).

To perform the ultrasound studies, the transducer was first used to obtain a transverse view of the median nerve at the DWC. The transducer was moved 2–3 centimeters distal and proximal to the DWC to find the site of maximal nerve enlargement, and the cross-sectional area of the median nerve was measured at this site using the “continuous trace” function. At this same site of maximal enlargement, the median nerve echogenicity, mobility, and vascularity were assessed. Echogenicity was assessed subjectively, and rated as either “normal (2), slightly decreased (1), or decreased (0)” based on visual inspection of the image, with normal nerve echogenicity showing a honeycomb pattern with a mixture of dark fascicles interspersed amongst a brighter background. To assess median nerve mobility, the participant was asked to repeatedly flex and extend the fingers and wrist, while the transducer was kept over the distal wrist crease. Mobility was rated as “normal (2), slightly decreased (1), or decreased (0).” Normal mobility is seen when the median nerve dives deep to the flexor tendons during finger and wrist flexion. Vascularity was assessed by placing the power Doppler box over the median nerve, and slowly increasing the gain. If color flow was seen in the nerve prior to other structures (in particular, the flexor tendons) then vascularity was rated as either “increased (2) or slightly increased (1)” based on the degree of color flow, and “normal” (0) when there was no early color Doppler signal in the nerve. The transducer was then moved to the mid-forearm to measure the cross-sectional area of the median nerve at this location, again at the site of maximal nerve enlargement. This mid-forearm measurement served as an internal control.

Statistical Analysis

Changes in the following variables were assessed over time: Levine-Katz symptom and function scores; median nerve motor and sensory latencies (converted to velocity for sensory studies) and amplitudes; and median nerve cross-sectional area (at DWC and mid-forearm), echogenicity, mobility, and vascularity assessed with ultrasound. Variables with normal distribution were analyzed using one-way repeated measures ANOVA, and variables with non-normal distribution were analyzed with Friedman repeated measures ANOVA of ranks. For each variable, p values for the trends were calculated. Correlation was assessed using Pearson product-moment correlation coefficients.

Results

Seven participants were female (36.8%), 2 with bilateral symptoms and injections, and twelve participants were male (63.2%), 8 with bilateral symptoms and injections. The mean

age of the participants was 56.5 years (range, 27–82 years), and the mean BMI was 27.4 (range, 19.5–44.7). Nine of the participants were seen at Wake Forest University and 10 at Korea University (Table 1).

Three participants (4 wrists) were lost to follow-up prior to their last visit. Additionally, two participants (one wrist each) underwent carpal tunnel release surgery between the 1 and 6 month visits. Thus, the 6-month data for these 2 wrists were not included in the statistical analyses. We performed statistical analyses in which the missing variables from the 6 wrists were assigned values to indicate they did not change over time, and this did not affect the statistical significance of any of the results.

The following mean baseline values were recorded: motor latency 5.32ms, motor amplitude 7.59mv, sensory velocity 31.98m/s, sensory amplitude 15.29uv, DWC area of 15.86mm², forearm area of 6.56mm², nerve echogenicity of 0.9 (“2” is normal), nerve mobility of 0.4 (“2” is normal), and nerve vascularity of 0.6 (“0” is normal) (Table 2). The following variables showed significant changes over time: symptom score ($p<0.001$), function score ($p<0.001$), motor latency ($p=0.005$), motor amplitude ($p=0.029$), sensory velocity ($p<0.001$), sensory amplitude ($p=0.007$), DWC area ($p<0.001$), nerve mobility ($p=0.001$), and nerve vascularity ($p=0.042$) (Table 3). The following variables correlated significantly with the DWC nerve cross-sectional area at the 0.05 significance level (reported as r , p -value): symptom score (0.53, 0.044), motor latency (0.3, 0.002), and motor amplitude (−0.25, 0.010) (Table 4). The following variables correlated significantly with symptom score at the 0.05 significance level (reported as r , p -value): DWC area (0.53, 0.044), motor latency (0.33, $p<0.001$) and sensory amplitude (−0.24, 0.013) (Table 4). All of the above analyses were also performed using only the data from Wake Forest University, and no significant differences were noted. This indicates that statistically similar results were obtained from Wake Forest University and Korea University.

Discussion

Nineteen individuals (29 total wrists) with CTS underwent successful ultrasound-guided steroid injection near the median nerve within the carpal tunnel. Based on the Levine-Katz questionnaire, hand symptoms and function both significantly improved following steroid injection ($p<0.001$), and the improvement was noted one week after the injection. At the six month visit, the mean symptom score increased from the nadir seen 1 month after injection, but it still did not approach the mean baseline score. This persistent effect of steroid injection for CTS is consistent with other reports, which suggest a mean benefit of 4.5 months, and some individuals have symptomatic improvement lasting well over 1 year.^{9, 10}

The electrodiagnostic parameters (median nerve motor latency, motor amplitude, sensory velocity, and sensory amplitude) also showed significant improvement following steroid injection. Interestingly, only the sensory velocity began to decline at the 6 month follow-up, in the same manner as the symptom score, whereas the other electrodiagnostic parameters continued to improve even at 6 months. Similar electrodiagnostic findings have been reported previously following steroid injection for CTS, with the median nerve sensory velocity beginning to decline at 6 months. This is consistent with it being a sensitive parameter for the assessment of CTS.¹¹

Three ultrasonographic parameters (median nerve DWC cross-sectional area, mobility, and vascularity) showed significant improvement following steroid injection, but nerve echogenicity did not ($p=0.064$). As expected, the cross-sectional area of the median nerve at the forearm did not change following injection. This is the first study to assess the ultrasonographic findings after steroid injection for CTS, but there are a few studies that

have used ultrasound to assess the median nerve following surgical release for CTS. Two of these studies demonstrated decreases in median nerve cross-sectional area at the wrist following surgical release, similar to the results we recorded after injection,^{12, 13} and the other noted normalization of median nerve morphology, with resolution of the abnormal median nerve flattening ratio seen in CTS.¹⁴ Ultrasonographic assessment of median nerve mobility and vascularity has not previously been reported following any intervention for CTS, so it is noteworthy that these parameters also improved following steroid injection. In addition, at 6 months, mobility and vascularity values stopped improving and began to return toward the baseline, similar to the symptom score and sensory velocity, which suggests these parameters may be sensitive for the assessment of CTS. However, it should be noted that subjective scales that have not been validated were used to assess median nerve mobility and vascularity. Therefore, further research into these parameters is needed.

Previous studies have used ultrasound to quantify nerve enlargement in conditions such as Charcot-Marie-Tooth disease and multifocal motor neuropathy,^{15, 16} leading to speculation that the ultrasonographic nerve enlargement seen in some conditions, including CTS, could be related to demyelination and re-myelination. Our results suggest this is an unlikely solitary explanation for the nerve enlargement seen with CTS, because the nerve size decreased within 1 week of a steroid injection near the median nerve. Therefore, we postulate that at least some of the ultrasonographic median nerve enlargement seen with CTS is related to rapidly reversible causes, such as inflammation and edema, which have been demonstrated in animal models of nerve compression.¹⁷ The decrease in median nerve vascularity, as measured by Doppler flow, also implicates vascular congestion as a component of the nerve enlargement seen in CTS.

This study had some limitations. First, the sample size was not large, since only 19 individuals (29 wrists) were studied. This is because the study was designed as an exploratory pilot study. While this sample size was large enough to detect differences in many nerve conduction and ultrasound parameters over time, a larger sample size may have provided even more power to detect significant differences in other parameters, such as median nerve echogenicity. Second, all participants received steroid injections and there was no control group, so those performing the nerve conduction studies and ultrasound could have been biased. To prevent this as much as possible, given the constraints of the study design, previous nerve conduction study and ultrasound values were not reviewed or referenced at any point during, or prior to, the collection of data. Finally, some of the measurements, such as median nerve echogenicity, mobility, and vascularity have not been extensively studied with regard to reliability. To minimize potential measurement variability, only two ultrasonographers performed all the studies, but inter and intra-rater reliability still could have affected the results. It is reassuring that no difference was found in the data from Wake Forest and Korea Universities, but further investigation into the reliability of these measures is needed.

In conclusion, ultrasound study of the median nerve at the DWC demonstrated significant changes in nerve cross-sectional area, mobility, and vascularity following steroid injection for CTS. Knowledge of these changes will help clinicians, particularly in the evaluation of individuals with failed CTS interventions. Median nerve cross-sectional area and vascularity changed within one week of steroid injection, indicating that at least some of the pathophysiologic changes seen in CTS are rapidly reversible. Finally, median nerve mobility and vascularity are two ultrasonographic parameters that should be investigated further as potentially sensitive measures of median nerve dysfunction in CTS. Neuromuscular ultrasound in general deserves further investigation for all aspects of focal neuropathies, as it is painless, readily accessible, involves no radiation, and provides detailed anatomic information.

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Abbreviations

CTS	carpal tunnel syndrome
DWC	distal wrist crease

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Table 1

Patient Population

Variable	Value (Standard Deviation)
Total Number of Participants	19
Number from Wake Forest University	9
Number of Subjects with Bilateral CTS	10
Total Number of Wrists Initially Studied	29
Total Number of Wrists Followed Entire 6 Months	23
Mean Age (years)	56.5 (11.5)
% Female	36.8
Mean Body Mass Index	27.4 (5.5)

Table 2

Baseline Characteristics

Variable	Value (Standard Deviation)
<i>Subjective Findings:</i>	
Mean Symptom Score	2.7 (0.7)
Mean Function Score	2.1 (0.8)
<i>Electrodiagnostic Parameters:</i>	
Mean Motor Latency	5.32ms (1.1)
Mean Motor Amplitude	7.59mV (3.3)
Mean Sensory Velocity	31.98m/s (6.7)
Mean Sensory Amplitude	15.29uV (10.8)
<i>Ultrasound Parameters:</i>	
Mean Distal Wrist Crease Nerve Area	15.86mm ² (3.5)
Mean Forearm Nerve Area	6.56mm ² (1.6)
Mean Echogenicity Score	0.9 (0.5)
Mean Mobility Score	0.4 (0.6)
Mean Vascularity Score	0.6 (0.8)

Table 3

Measurements Over Time

Variable	Baseline	7 Days	30 Days	180 Days	p-value
<i>Subjective Findings</i>					
Mean Symptom Score (Standard Deviation)	2.7 (0.7)	1.8 (0.4)	1.6 (0.5)	1.8 (0.7)	<0.001
Mean Function Score (Standard Deviation)	2.1 (0.8)	1.6 (0.5)	1.4 (0.4)	1.6 (0.7)	<0.001
<i>Electrodiagnostic Parameters</i>					
Mean Motor Latency (Standard Deviation)	5.32 (1.1)	5.11 (1.1)	5.02 (1.0)	4.71 (0.9)	0.005
Mean Motor Amplitude (Standard Deviation)	7.59 (3.3)	8.49 (4.0)	8.83 (4.1)	9.12 (4.0)	0.029
Mean Sensory Velocity (Standard Deviation)	31.98 (6.7)	35.47 (7.7)	36.30 (7.9)	36.25 (7.4)	<0.001
Mean Sensory Amplitude (Standard Deviation)	15.29 (10.8)	16.16 (9.6)	18.60 (10.7)	20.59 (13.0)	0.007
<i>Ultrasound Parameters</i>					
Mean DWC Nerve Area (Standard Deviation)	15.86 (3.5)	13.15 (3.5)	12.83 (3.8)	12.50 (3.3)	<0.001
Mean Forearm Nerve Area (Standard Deviation)	6.56 (1.6)	6.52 (1.5)	6.40 (1.5)	6.10 (1.3)	0.335
Mean Echogenicity Score (Standard Deviation)	0.9 (0.5)	1.1 (0.7)	1.1 (0.6)	0.9 (0.6)	0.064
Mean Mobility Score (Standard Deviation)	0.4 (0.6)	0.7 (0.7)	1.0 (0.6)	0.9 (0.6)	0.001
Mean Vascularity Score (Standard Deviation)	0.6 (0.8)	0.1 (0.4)	0.1 (0.4)	0.2 (0.5)	0.042

Table 4

Significant Correlations

Variable Pair	Correlation Coefficient (r)	p-value
DWC Nerve Area-Motor Latency	0.30	0.002
DWC Nerve Area-Motor Amplitude	-0.25	0.010
DWC Nerve Area-Symptom Score	0.53	0.044
Symptom Score-Motor Latency	0.33	<0.001
Symptom Score-Sensory Amplitude	-0.24	0.013