HHS Public Access

Author manuscript

Muscle Nerve. Author manuscript; available in PMC 2014 October 01.

Published in final edited form as:

Muscle Nerve. 2014 October ; 50(4): 517–522. doi:10.1002/mus.24183.

Muscle Intrusion as a Potential Cause of Carpal Tunnel Syndrome

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Abstract

Introduction—To determine if there is an association between flexor digitorum and lumbrical muscle intrusion into the carpal tunnel and carpal tunnel syndrome (CTS).

Methods—513 manual laborers (1,026 wrists) were evaluated with ultrasound to determine if those with CTS had more muscle intrusion into the carpal tunnel than those without CTS. 190 of the participants without CTS at baseline (363 wrists) were followed over 1 year to determine if muscle intrusion at baseline predicted the development of CTS.

Results—Participants with CTS had more muscle within the carpal tunnel with the wrist in the neutral ($P = 0.026$) and flexed positions ($P = 0.018$) than those without CTS. Baseline muscle intrusion did not predict the development of CTS at 1 year.

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Disclosure: Drs. Cartwright, Walker, Arcury, Chen, and Quandt; and Ms. Newman and Mora have nothing to disclose.

Dr Cartwright participated in study concept and design, acquisition of data, analysis and interpretation of data, and critical revision of the manuscript for important intellectual content

Dr. Walker participated in study concept and design, acquisition of data, analysis and interpretation of data, and critical revision of the manuscript for important intellectual content

Ms. Newman participated in study concept and design, analysis and interpretation of data, and critical revision of the manuscript for important intellectual content

Dr. Arcury participated in study concept and design, analysis and interpretation of data, and critical revision of the manuscript for important intellectual content

Ms. Mora participated in study concept and design, acquisition of data, analysis and interpretation of data, and critical revision of the manuscript for important intellectual content

Dr. Chen participated in study concept and design, analysis and interpretation of data, and critical revision of the manuscript for important intellectual content

Dr. Quandt participated in study concept and design, acquisition of data, analysis and interpretation of data, critical revision of the manuscript for important intellectual content, and study supervision

Conclusions—Muscle intrusion into the carpal tunnel is associated with CTS, but muscle intrusion alone does not predict the development of CTS over the course of a year.

Keywords

Carpal tunnel syndrome; incidence; prevalence; ultrasound; EMG

Introduction

Carpal tunnel syndrome (CTS) is a common condition; it affects 2.7% of people and costs over $500 million annually in United States healthcare spending.[1, 2] CTS is thought to be caused by chronic irritation of the median nerve at the wrist as it passes through the fibroosseous carpal tunnel along with 9 finger flexor tendons. Risk factors for CTS such as female gender, obesity, pregnancy, hypothyroidism, rheumatoid arthritis, diabetes, and family history have been identified, but it remains unclear why some with risk factors develop CTS and others do not.[1] One theory is that increased pressure within the carpal tunnel leads to chronic compression of the median nerve, which causes microvascular trauma and results in a median mononeuropathy at the wrist.[3] While several aspects of this theory are plausible, the initial cause of increased pressure within the tunnel in those with idiopathic CTS is not understood.

Over the past 20 years, neuromuscular ultrasound has been used to examine median mononeuropathy at the wrist, and it is now an accepted modality for the diagnosis of CTS.[4] Ultrasound provides anatomic information about the median nerve and contents of the carpal tunnel that cannot be obtained with nerve conduction studies. Our Diagnostic Neurology laboratory has been using ultrasound to evaluate suspected cases of CTS for more than 10 years. During that time we have noted many individuals who have muscles, either flexor digitorum or lumbricals, entering the carpal tunnel with certain finger and wrist movements.[5] In addition, there are scattered case reports in the surgical literature over the past 4 decades of CTS suspected to be secondary to anomalous muscle intrusion into the carpal tunnel.[6, 7] Given these findings, we postulated that idiopathic CTS may be secondary to increased pressure within the carpal tunnel caused by repetitive muscle intrusion into the tunnel. This study was conducted to explore that hypothesis.

Methods

Participants

This study is part of a larger investigation into health disparities in Latino poultry processing workers, including dermatologic, pulmonary, and musculoskeletal conditions. Participant recruitment and study design have been discussed in detail previously.[8] Prior to initiation, this study was approved by the Institutional Review Board at Wake Forest School of Medicine, and all participants provided signed informed consent. Participants were paid $40 for attending the initial data clinic and $60 for attending the one-year follow-up clinic.

Participants were recruited from 4 counties in western North Carolina. All participants self-identified as Latino and worked full-time in manual labor positions. Each attended a data

collection clinic that occurred on 1 of 12 Sundays evenly distributed from June 2009 to November 2011. Those without CTS at baseline (defined by symptoms and nerve conduction studies) were asked to return for repeat evaluations a year later.

1,526 individuals underwent baseline screening; 957 were eligible for enrollment, 742 underwent interviews, 518 attended baseline data collection clinic, and 513 had ultrasound, nerve conduction studies, and filled out hand diagrams at the baseline data collection clinic (1,026 wrists). Two-hundred sixty-four participants were identified as not having CTS at baseline in either hand and were invited to return to a second data collection clinic a year later. Of those, 173 (65.5%) returned for 1-year follow-up. In addition, 17 workers who were invited back for the dermatologic portion of the study and did not have CTS in 1 of wrist at baseline were included in the follow-up data analyses to increase the number of individuals studied. This resulted in 363 total wrists without CTS at baseline that underwent follow-up at 1 year.

Data Collection

During the data collection clinics the participants were asked about hand symptoms, filled out a Katz hand diagram,[9] underwent bilateral nerve conduction studies, and were evaluated with neuromuscular ultrasound of both wrists. The interview and hand diagram were conducted in Spanish. Nerve conduction studies were performed with a Teca TD10 Electromyograph (Teca Corporation, Pleasantville, NY) by a technician with at least 5 years of experience who was blinded to the clinical and ultrasonographic information. Wrist temperature was maintained greater than 32°C, and median and ulnar antidromic sensory studies were performed by stimulating at the wrist and recording with ring electrodes 14 cm distally on the 2nd and 5th fingers, respectively. Onset and peak sensory latencies were recorded. If a median nerve sensory nerve action potential was not obtainable, then an orthodromic median motor study was conducted.

Ultrasound of the wrist was performed using a Biosound MyLab25 ultrasound device (Esaote Group, Genoa, Italy) with an 18 MHz linear array transducer. Participants were seated with their forearms supinated and resting on a table, and ultrasonography was done by a neurologist with at least 5 years of neuromuscular ultrasound experience. The ultrasonographer was blinded to the clinical and nerve conduction study data. The site of maximum median nerve enlargement at the wrist was identified, and the cross-sectional area of the median nerve at this site was measured. The trace function was used to outline the outer portion of the nerve, erring just to the inside of the bright epineurium. Next, the cross-sectional area of the muscle entering the carpal tunnel was measured at the level of the distal wrist crease. This was done with the wrist in the neutral position, and with the wrist fully extended and fully flexed. When the fingers and wrist were fully extended, the amount of flexor digitorum muscle entering the tunnel was measured, and with the fingers and wrist fully flexed the amount of lumbrical muscle entering the tunnel was measured. To position the fingers and wrist in full extension and flexion, participants were instructed to actively move their fingers and wrist, and none of the positioning was obtained with passive movement. The muscle measurements were performed using the trace function and same

method as for the nerve, and in some cases the results of >1 tracing were added together to get the total cross-sectional area of multiple muscles entering the carpal tunnel (Figure 1).

Defining CTS

A combination of symptoms (based on the hand diagram) and nerve conduction study results was used to define the presence of CTS. First, if the hand diagram scored a “1”, “2”, or “3” then the diagram was assigned a score of “1”, and if not it was assigned a “0.” Next, median and ulnar peak latencies were compared. If the median was less than 0.49 ms longer than the ulnar, it was scored as “0”; if it was 0.50 to 0.79 ms longer, it was scored as “1”; and if it was greater than 0.80 ms longer, it was scored as “2.” The hand diagram and nerve conduction score were then summed, and a total score of 0 was defined as “no CTS,” 1–2 as “possible CTS,” and 3 as “CTS.” Similar CTS case definitions, with 0.50 ms and 0.80 ms cut-offs for peak latency difference, have been used previously in large-scale CTS screening studies.[10] This scoring system was applied to each wrist, and those that scored a 0 bilaterally during the initial visit were invited to return 1 year later. In addition, 17 wrists from the dermatologic portion of the study also scored a 0 at baseline in 1 wrist and were evaluated 1 year later.

Statistical Analyses

In general, continuous variables were calculated as means and standard deviations, and discrete variables were calculated as percentages and frequencies. The initial group of 513 participants (1,026 wrists) was evaluated to determine the amount of muscle intrusion into the carpal tunnel with the wrist in the neutral, extended, and flexed positions; and multivariate nominal logistic regression, controlling for data collection site, participant dwelling (to account for related individuals), wrist side, age, gender, and body mass index (BMI) was used to examine the association between the muscle area in wrists and the prevalence of CTS at baseline. Hard cut-offs of >15 mm^2 of muscle in any wrist position, >30mm^2 of muscle in any position, and any muscle intrusion in any position were also evaluated in this model in those with and without CTS. Next, those without CTS at baseline and with 1 year follow-up data (190 participants, 363 wrists) were evaluated to determine if muscle intrusion predicted the development of CTS. Two models were created, one in which CTS was strictly defined as only those participants who developed “CTS” (total score = 3) and one in which the definition of CTS was more flexible and included both “possible CTS” and “CTS” (total score = 1–3). In each model, the following variables were evaluated: age, gender, BMI, muscle area (in neutral, extended, and flexed wrist positions), presence of muscle area in any position > 15 mm^2, presence of muscle area in any position > 30 mm^2, and any muscle intrusion into the tunnel. No differences were detected between the strict and less strict models. Because only 2 participants developed CTS over 1 year by the strict definition, only data from the less strict model are presented. The models were also run excluding the 17 individuals with only unilateral data, and this did not change any of the results significantly. All models were checked using the score test for the proportional odds assumption, and all models were non-significant, indicating they were appropriate models. Finally, it should be noted that all models were adjusted for clustering within a participant (referred to as “wrist side”), to control for the lack of independence between 2 wrists in the same individual.

Results

Of the 1,026 wrists that were evaluated, 632 (61.6%) met criteria for “no CTS,” 346 (33.7%) met criteria for “possible CTS,” and 48 (4.7%) met criteria for “CTS.” After controlling for age, gender, and BMI, we found that 4 wrist variables were significantly different among the 3 CTS classifications: nerve area ($P < 0.001$), muscle area with the wrist in neutral position ($P = 0.017$), muscle area with the wrist in flexed position ($P = 0.020$), and any muscle within the tunnel ($P = 0.003$) (Table 1). This shows that those with CTS had more muscle within the tunnel with the wrist in the neutral and flexed positions, and they were more likely to have any muscle entering the tunnel. All of the participants who met criteria for “CTS” had some muscle entering the tunnel, whereas 9.1% of those defined as “no CTS” had no muscle intrusion at all.

Of the 363 wrists with no CTS at baseline, at 1 year follow-up 312 (86.0%) continued to meet the criteria for “no CTS,” 49 (13.5%) changed to “possible CTS,” and 2 (0.6%) changed to “CTS” (Table 2). After controlling for participant characteristics, none of the baseline ultrasonographic measurements of muscle predicted the development of CTS at 1 year in the 363 wrists evaluated, in either the strict or less strict CTS definition models (Table 3).

Discussion

In this study, the amount of muscle entering the carpal tunnel was evaluated in 1,026 wrists of 513 manual laborers. Those who met criteria for “CTS” had more muscle intrusion into the carpal tunnel with the wrist in the neutral and flexed positions than those with “possible CTS,” and those with “possible CTS” had more muscle intrusion than those with “no CTS.” An additional finding of significance was that 100% of those with “CTS” had some degree of muscle intrusion into the tunnel, 96.5% of those with “possible CTS” had some degree of muscle intrusion, and 90.9% of those with “no CTS” had some degree of muscle intrusion. These findings suggest that muscle intrusion into the carpal tunnel, with the wrist in the neutral or flexed position (resulting in lumbrical intrusion into the tunnel), is associated with CTS. Case reports dating back several decades have suggested this possibility, and there are a few other studies that have demonstrated lumbrical muscle intrusion into the tunnel and a possible association with CTS. Cobb and colleagues in 1994 examined 5 cadaver wrists using radiopaque markers on the transverse carpal ligament and lumbrical muscles and showed that with complete finger flexion the lumbricals moved an average of 30 mm into the carpal tunnel.[11] This same group, in 1995, measured the pressure within the carpal tunnel in cadavers with the fingers flexed before and after removal of the lumbrical muscles, and they demonstrated a significant decrease in carpal tunnel pressure after removal of the muscles when the fingers were fully flexed compared to when the lumbricals were in place.[12]

While this study showed an association between lumbrical muscle intrusion and CTS, it did not show the same association between flexor digitorum muscle intrusion and CTS. Those who met criteria for possible CTS had more flexor digitorum muscle intrusion than those with “no CTS,” but those with “CTS” had less flexor digitorum intrusion than both the “no

CTS" and "possible CTS" groups. This is an unexpected finding, because, similar to the literature on lumbrical muscle intrusion, there are case reports and other studies that suggest flexor digitorum intrusion into the tunnel as a cause of CTS.[6, 13] There are several possible explanations why an association between flexor digitorum intrusion and CTS was not detected in this study. First, it is possible that there is not a strong association between CTS and the intrusion of anatomically normal flexor digitorum muscle, and only anomalous flexor digitorum intrusion causes CTS, as described in case reports. Second, it is possible that flexor digitorum intrusion is associated with CTS, but, once the median nerve starts to enlarge, there is no room for the muscle to enter. This might be suggested by the increased amount of muscle within the tunnel in those with "possible CTS" compared to those with "no CTS." However, this is not consistent with the lumbrical findings. Third, perhaps the participants in this study, Latino manual laborers (half being poultry processing workers), perform specific repetitive tasks that cause more damaging lumbrical intrusion than flexor digitorum intrusion. Finally, it is important to note the site of muscle measurement in this study, which was at the distal wrist crease. Lumbrical muscles measured at this site, by definition, traversed the entire length of the carpal tunnel, whereas flexor digitorum muscle at this site may have intruded only slightly into the proximal portion of the tunnel. Further investigation in other populations and at multiple sites within the tunnel will be needed to determine if flexor digitorum intrusion has any association with CTS.

It is worth noting that most participants in this study had some degree of muscle intrusion. We previously studied healthy volunteers with no symptoms of CTS and found that muscle intrusion occurred in only 80%, whereas in this study 90.9% of those with "no CTS" had muscle intrusion.[5] The most likely explanation is that this study evaluated manual laborers, so they likely had more muscle hypertrophy and intrusion than our previous group of volunteers from our medical center. To some degree, this matches the hypothesis that manual labor leads to muscle intrusion, which leads to increased carpal tunnel pressure and results in median mononeuropathy at the wrist. It should also be noted that specific cut-offs were investigated to determine if > 15 mm^2 or > 30 mm^2 of any muscle type was associated with CTS, and they were not. This may be because these cut-offs included both lumbrical and flexor digitorum measurements, and in this study only lumbrical intrusion was associated with CTS. Alternatively, it is possible that it is an issue of methodology, and these pre-determined cut-offs were not important statistically.

The second portion of the study was prospective and followed 363 wrists (190 manual laborers) over a year to determine if muscle intrusion predicted the development of CTS. These data were examined multiple ways, and no association was detected between muscle intrusion at baseline and the development of CTS. One limitation of this portion of the study was that only 2 wrists (0.6%) developed "CTS" at 1 year. A larger number (49 wrists, 13.5%) developed "possible CTS" at 1 year, but the low number of those that converted to "CTS" likely limited the ability to create robust predictive models. A study with longer follow-up would likely result in more incident cases and improve the ability to assess the importance of muscle intrusion for development of CTS.

Other limitations were present in this study. First, more sensitive electrodiagnostic studies are available for detection of median mononeuropathy at the wrist, such as mixed palmar

comparison studies.[14] We chose to use a peak median-to-ulnar sensory comparison, because it has previously been described in large-scale screening studies of CTS, it is not technically challenging, and it is efficient, especially in a setting outside of our electrodiagnostic laboratory. However, this may have systematically decreased our ability to detect mild cases of CTS. Second, 34.5% of those without CTS at baseline did not follow up at 1 year for repeat studies. This could have introduced bias, as perhaps those who developed CTS were more likely to stop working in manual labor and therefore not complete follow-up. Third, this study included only Latino manual laborers, so the results may not be generalizable to all populations. Finally, it is possible that the ratio of muscle intrusion to the size of the carpal tunnel itself is even more important than the absolute amount of muscle intrusion. Measurement of the area of the entire carpal tunnel can be obtained with ultrasound, but it is often challenging technically. Given the large number of ultrasonographic evaluations performed in this study, we did not think it would be feasible to also measure carpal tunnel size.

Overall, we found the results of this exploration to be quite interesting. Further studies, perhaps of longer duration, with measurement at more sites within the tunnel, and with measurement of the area of the tunnel itself, should be considered to more extensively explore the relationship between idiopathic CTS and muscle intrusion into the carpal tunnel. Since CTS is a common and costly condition,[1, 2] a thorough understanding of this relationship is important and could lead to novel preventive and therapeutic approaches for CTS.

Acknowledgments

Financial Support: Dr. Quandt has funding from the CDC/NIOSH (R01OH009251) to study occupational injuries in Latino poultry workers and Dr. Cartwright has funding from the NIH/NINDS (1K23NS062892) to study neuromuscular ultrasound.

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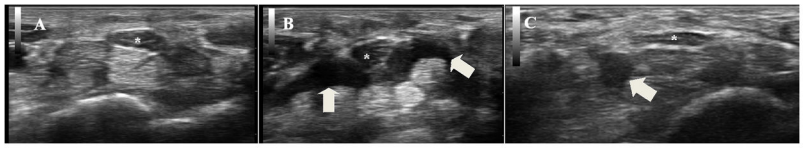

Figure 1.

Images A and B are cross-sectional views obtained with ultrasound at the left distal wrist crease in an individual with lumbrical muscle intrusion into the carpal tunnel during finger and wrist flexion. Image A was obtained with the wrist in the neutral position. The nerve is marked with an asterisk (*), and deep to the nerve are the flexor tendons. Image B was obtained with the fingers and wrist flexed. Deep and lateral to the nerve, on either side, are hypoechoic and anechoic regions (arrows), which are the intruding lumbrical muscles. The tendons are the hyperechoic, round structures that are now displaced deep to the lumbrical muscles. Image C is from the left wrist of an individual with flexor digitorum muscle intrusion during finger and wrist extension, and the nerve (*) and muscle (arrow) are again marked.

Table 1

Muscle Intrusion into the Carpal Tunnel in 1,026 Wrists of Latino Manual Workers

Parameter	No CTS N = 632 (61.6%) Mean (SD)	Possible CTS N = 346 (33.7%) Mean (SD)	Definite CTS N = 48 (4.7%) Mean (SD)	*P*-value
Age	32.7 (9.8)	37.6 (10.2)	41.4 (12.3)	< 0.001[c]
Gender (% women)	44.8 %	46.0 %	58.3%	0.390[c]
Height (cm)	158.5 (8.6)	156.8 (7.9)	155.0 (9.3)	0.003[c]
Weight (kg)	70.7 (13.0)	73.3 (13.7)	77.5 (18.4)	0.003[c]
BMI	28.1 (4.5)	29.8 (4.8)	32.2 (7.1)	< 0.001[c]
Median Nerve Area (mm^2)	10.2 (2.3)	12.4 (3.5)	14.8 (5.6)	< 0.001[d]
Muscle Area Neutral (mm^2)	6.1 (8.7)	8.0 (10.4)	8.2 (10.6)	0.017[d]
Muscle Area Extended (mm^2) [a]	12.4 (13.2)	13.1 (14.1)	11.2 (9.4)	0.733[d]
Muscle Area Flexed (mm^2)[b]	16.6 (18.5)	20.3 (19.5)	21.0 (17.5)	0.020[d]
Muscle Area in Any Position > 15 mm^2	61.9 %	67.4 %	64.6 %	0.265[d]
Muscle Area in Any Position > 30 mm^2	28.4 %	30.8 %	29.2 %	0.537[d]
Any Muscle Intrusion	90.9 %	96.5 %	100 %	0.003[d]

[a] Flexor digitorum superficialis muscle

[b] Lumbrical muscle

[c] Multivariate logistic regression controlling for site, dwelling, and wrist side

[d] Multivariate logistic regression controlling for site, dwelling, wrist side, age, gender, and BMI

Table 2

Baseline Parameters in the 363 Wrists that Underwent One-Year Follow-up

Parameters	No CTS (312 wrists)	Possible CTS (49 wrists)	CTS (2 wrists)
Age	30.5 (7.9)	31.5 (11.1)	28.0 (1.4)
Gender (% women)	47.8 %	42.9 %	50.0 %
Height (cm)	158.0 (8.1)	158.5 (9.4)	159.5 (18.8)
Weight (kg)	69.6 (12.6)	71.1 (13.4)	75.8 (9.5)
BMI	27.9 (4.7)	28.2 (4.1)	30.0 (3.3)
Muscle Area Neutral (mm2)	5.9 (8.2)	7.2 (9.5)	3.0 (4.2)
Muscle Area Extended (mm2)	12.8 (13.1)	14.8 (15.6)	0 (0)
Muscle Area Flexed (mm2)	16.0 (17.8)	17.2 (17.8)	16.0 (9.9)
Muscle Area in Any Position > 15 mm2 (%)	60.77 %	61.22 %	50.00 %
Muscle Area in Any Position > 30 mm2 (%)	27.01 %	30.61 %	0 %
Any Muscle Intrusion (%)	91.59 %	93.75 %	100 %

Table 3

Muscle Intrusion (Less Strict Model, 51 Incident Cases of Possible CTS or CTS)

Parameters	Adjusted Odds Ratio	*P*-Value[a]
Age	1.010	0.724
Gender		0.755
Men	1.136	
Women	---	
Height	0.913	0.416
Weight	1.106	0.356
BMI	0.795	0.395
Muscle area in neutral	1.013	0.609
Muscle area in extended	1.010	0.586
Muscle area in flexed	1.008	0.677
Muscle Area in Any Position > 15 mm^2	0.663	0.380
Muscle Area in Any Position > 30 mm^2	0.848	0.808
Any Muscle Intrusion	1.305	0.700

[a] Multivariate logistic regression controlling for site, dwelling, wrist side, age, sex, and BMI