

Introduction

Carpal tunnel syndrome (CTS) is the most common peripheral compressive neuropathy. The prevalence of CTS in the general population is approximately 4% and its symptoms result in roughly 500,000 decompressions performed annually. Diagnosis of carpal tunnel syndrome is made primarily by history and physical examination. Electrodiagnostic studies are a helpful adjunct for confirming the diagnosis and understanding disease severity. More recently, ultrasonography has emerged as a useful diagnostic for CTS. Previous studies have demonstrated patients with CTS exhibit increased median nerve cross-sectional area (CSA) as compared to well patients. However, it is unclear what other risk factors may result in an increased median nerve size as measured by ultrasound and whether these risk factors worsen disease severity in patients with CTS

Diabetes mellitus is increasingly prevalent in the population and is a known risk factor for CTS. Diabetic neuropathy is the most common microvascular complication in diabetes with an overall prevalence of 30% and approximately 1/3rd of patients with diabetes develop CTS. Given the increasing use of ultrasonography in the diagnosis of CTS, our goal was to evaluate the influence of diabetes on CTS severity as well as the CSA of the median nerve in patients with CTS.

Methods

Patients with clinically diagnosed CTS were seen in the outpatient setting from October 2014 through February 2021 and enrolled. Outcome measures were collected including from the Boston carpal tunnel questionnaire (BCTQ) – both functional status scale (FSS) and symptom severity scale (SSS) – as well as CTS-6. Median Nerve CSA was measured at the carpal tunnel inlet, just proximal to the level of the pisiform, as demonstrated below:

Ultrasound Level for Median Nerve CSA

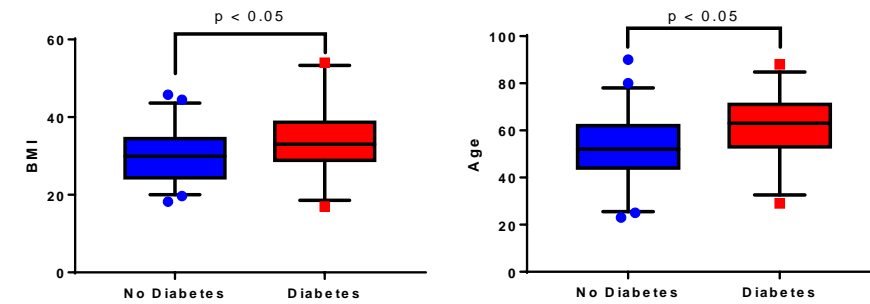
Median Nerve on Ultrasound (A), CSA 22mm²



A chart review was performed to identify patients with a known diagnosis of diabetes. Parameters were collected including most recent A1c, polypharmacy (>2 diabetes medications), and insulin pharmacotherapy. Statistics were performed using GraphPad Prism 7.00 using Mann-Whitney testing for numerical variables and Chi-squared testing for categorical variables. A p-value < 0.05 was determined to be statistically significant. Data are plotted with boxes indicating the interquartile range, horizontal line signifying the mean, and whiskers signifying 2.5-97.5% ile.

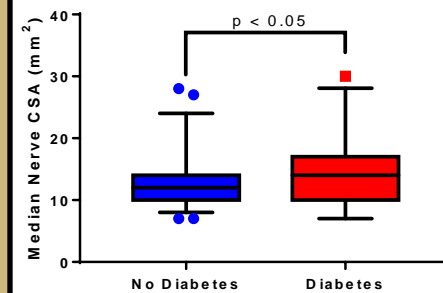
Patient Demographics

Demographic	No Diabetes	Diabetes	p-value
Patients	99	55	
Wrists	122	82	
% Bilateral	23.2	47.3	0.002
BMI Mean (95% CI)	29.9 (28.6 – 31.2)	33.8 (31.8-35.8)	0.002
Age Mean (95% CI)	52 (50 – 55)	62 (59 – 65)	0.014
% Female	72.7%	65.5%	0.345



Patients with CTS and diabetes are: **Older** (mean age 52 vs 62), **more obese** (mean BMI 29.9 vs 33.8), and have **more bilateral disease** (23.2% vs 47.3%)

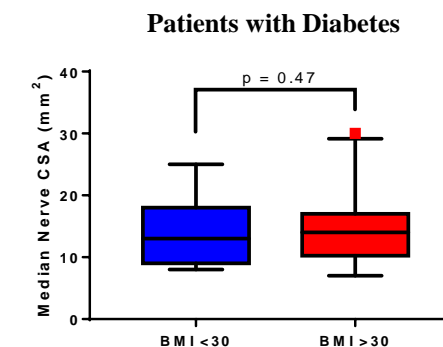
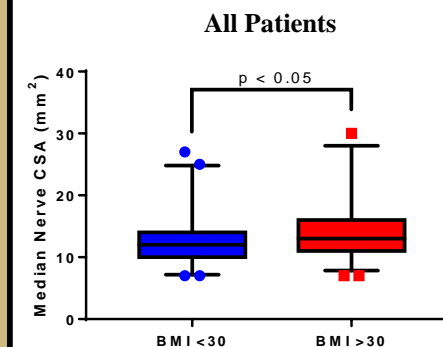
Influence of Diabetes and Obesity on Median Nerve CSA



In patients with CTS, **diabetic** patients have a **larger median nerve CSA** (mean 12.8mm² vs 14.5mm²)

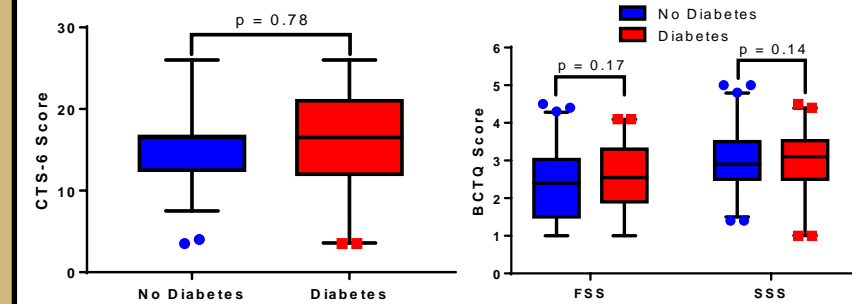
Obese patients have a larger median nerve CSA (mean 12.7mm² vs 14.0mm²)

Obesity does not increase median nerve CSA in patients with diabetes



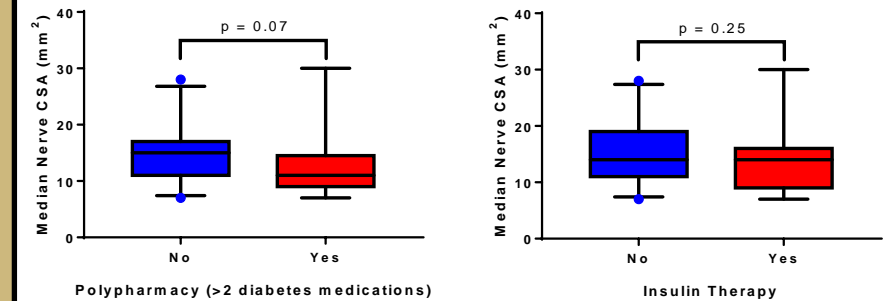
Patient Reported Symptom Severity

Diabetes **does not** increase disease severity in patients with CTS

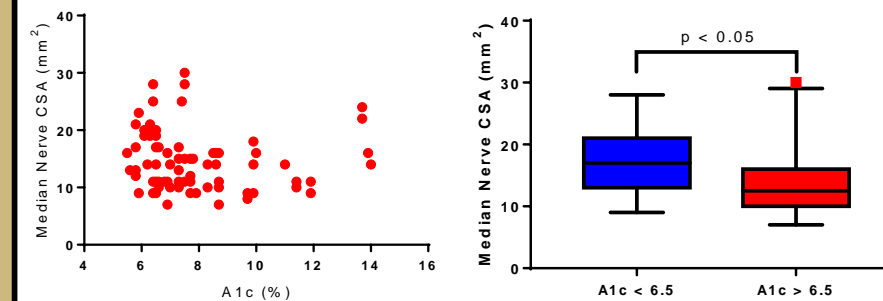


Influence of Diabetic Pharmacotherapy and Disease Control

No effect of polypharmacy nor insulin therapy on median nerve CSA



Poor diabetic control (A1c > 6.5%) **reduces** median nerve CSA



Conclusions

- While patients with diabetes exhibit larger median nerve CSA than patients without diabetes, CTS disease severity is similar
- Diabetic control and pharmacotherapy may influence median nerve CSA
- Influence of diabetes on median nerve CSA should be when utilizing ultrasound for diagnosis of CTS or for the evaluation of disease severity

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