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● *Original Contribution*

IDIOPATHIC CARPAL TUNNEL SYNDROME: EVALUATION OF THE DEPTH OF THE CARPAL TUNNEL BY ULTRASONOGRAPHY

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Abstract—The objective of the work described here was to evaluate the depth of the carpal tunnel (DCT) in patients with idiopathic carpal tunnel syndrome (CTS) and healthy volunteers by ultrasonography (US), through measurement of the distance from the flexor retinaculum to the surface of the capitate bone at the carpal tunnel outlet, and compare it with other ultrasonographic and electrophysiologic parameters in CTS. The study was conducted in 60 non-diabetic patients with idiopathic carpal tunnel syndrome (unilateral n = 37, bilateral n = 23) evidenced by electrophysiologic diagnosis according to the criteria of the American Association of Electrodiagnostic Medicine (AAEM). Furthermore, 40 hands from 20 healthy volunteers were examined. Median nerve cross-sectional area (CSA); flattening ratio (FR), the ratio of the length to the width of the median nerve; and DCT at the canal outlet were measured for all participants. The mean age was 35.6 ± 9.48 y. The female-to-male ratio was 47:13 in the CTS patients. The sensitivity and specificity were 82% and 95% for CSA, 75% and 60% for FR and 75% and 87.5% for DCT, respectively. Differences between patients and healthy controls were significant for all three parameters, greatest for DCT, followed by CSA and then FR. We conclude that DCT increased in CTS and this new parameter is comparable in sensitivity and specificity to CSA and FR. DCT increased independently of the cause of the CTS (decrease in size of canal or increase in contents). (E-mail: Sarah.ohrndorf@charite.de)

Key Words: **Idiopathic carpal tunnel syndrome, Electrophysiology, Ultrasonography, Carpal tunnel depth.**

INTRODUCTION

Carpal tunnel syndrome (CTS) is the most common form of entrapment neuropathy. In Scandinavian countries, its prevalence ranges from 8% at ages 35–44 to 15.8% at ages 55–64 (Atroshi et al. 1999). It affects 3% to 6% of the adult general population (LeBlanc and Cestia 2011). Carpal tunnel syndrome is compression of the median nerve at the carpal tunnel and can result in sensory and motor disturbances in areas of the hand supplied by this nerve, leading to pain and loss of function (Werner and Andary 2002).

The exact pathophysiology of CTS is not fully understood. Two theories have been put forward to explain its etiology. According to the first theory, the increase in pressure on the median nerve leads to transient ischemic episodes linked to microvascular disorders without changing the size of the carpal tunnel or the volume of the nerve; according to the second theory, median nerve compression results in a reduction in tunnel volume or an increase in the volume of tunnel contents (Rossignol et al. 1998).

In this study our aim was to shed light on the second theory by measuring depth of the canal in non-diabetic individuals with CTS and its correlation to other ultrasonographic parameters in patients with electrophysiologically confirmed CTS (Visser et al. 2008). Evaluation of the dimensions of the carpal tunnel has been inadequately addressed in the literature on CTS, as most previous studies evaluated the dimensions of the carpal tunnel in normal individuals and not in CTS patients. These studies, which were performed on healthy patients or cadavers, employed either computed tomography (CT) or magnetic resonance imaging (MRI), or used a silicon cast to fill the carpal tunnel to measure these dimensions (Bower et al. 2006; Mani et al. 2011; Pacek et al. 2010; Widgerow et al. 1996).

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Ultrasonography is a useful non-invasive painless bedside test preferred by most patients. In many studies, it has been used to evaluate CTS through measurement of median nerve cross-sectional area (CSA) and calculation of the flattening ratio (FR) (Visser et al. 2008). Sonographic measures of CTS are usually obtained at the inlet level, as it is believed that measures at the outlet are technically more difficult and have less inter-rater reliability, though the sensitivity and specificity at both levels are comparable (Moghtaderi et al. 2012; Moran et al. 2009).

METHODS

All patients recruited into the study were informed about the methodology and goals and signed a written consent. The study protocol was approved by the local ethics committee of Sohag Faculty of Medicine, Egypt. Personal and medical information was kept confidential and was not made available to a third party. Patients clinically suspected of having CTS were recruited from the neurology and rheumatology outpatient clinics of Sohag University Hospital. They were then referred to the neurologist for clinical and neurophysiologic examinations and, then, to the rheumatologist for ultrasonographic examination. Only the rheumatologist was blinded to the patients' diagnosis.

A descriptive cross-sectional study was carried out on the hands of 60 non-diabetic patients (unilateral n = 37, bilateral n = 23) aged ≥20 y with uni- or bilateral carpal tunnel syndrome and 40 hands of 20 healthy volunteers in the same age group attending the rheumatology and neurology outpatient clinics of Sohag University Hospital from January to March 2014.

Carpal tunnel syndrome was diagnosed according to the criteria of the AAEM (Jablecki et al. 2002; You et al. 1999). These criteria included both clinical and electrophysiologic evidence of CTS. Clinical evidence of CTS included both major and minor criteria for CTS diagnosis. Major criteria include: (i) paresthesias of the hand in a median nerve, median nerve and ulnar nerve or glove distribution; (ii) paresthesias aggravated by activities such as driving, holding a book, holding a telephone and working with the hands raised; (iii) paresthesias and pain in the hand that awaken the patient from sleep; and (iv) paresthesias relieved by shaking the hand or holding it in a dependent position. The minor criteria for diagnosis of CTS were: (i) subjective weakness of the hand; (ii) clumsiness of the hand or dropping of objects; and (iii) positive Tinel or Phalen sign. The electrophysiologic evidence of slowing of distal median nerve conduction includes prolongation of distal sensory and/or motor latency of the median sensory nerve action potential (SNAP) and/or compound muscle action potential (CMAP) ± reduced SNAP/CMAP amplitude of the median nerve (Keith et al. 2009).

Patients with polyneuropathy were excluded from the study. Polyneuropathy was diagnosed according to the recommendation of the AAEM (England et al. 2005).

For motor nerve conduction, compound muscle action potential (CMAP) was recorded using (Ag/AgCl)

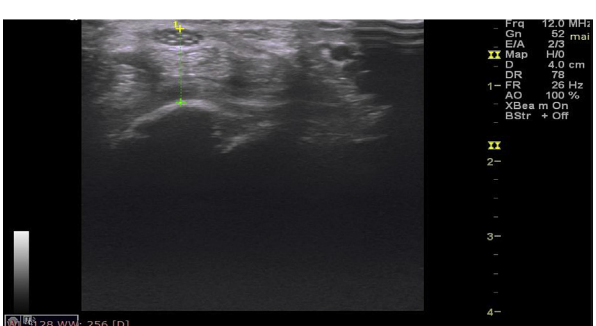

Fig. 1. Normal median nerve with fascicular pattern and transverse measurement of the depth of the carpal tunnel from the flexor retinaculum to the surface of the capitate bone.

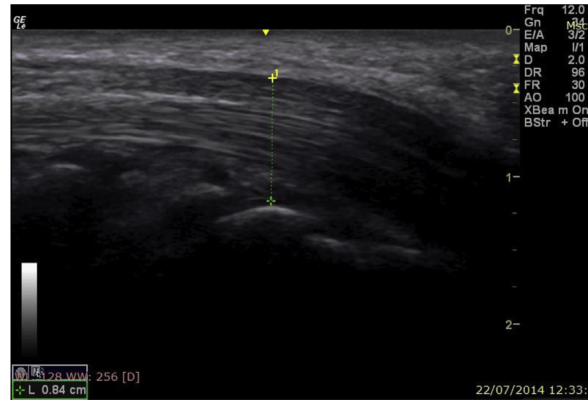

Fig. 2. Longitudinal measurement of the DCT from the surface of the median nerve perpendicularly to the capitate bone. DCT = depth of the carpal tunnel.

Fig. 3. Transverse measurement of the DCT from the flexor retinaculum perpendicularly to the capitate bone. DCT = depth of the carpal tunnel.