

OUR EXPERIENCE IN CARPAL TUNNEL SYNDROME THERAPEUTIC EFFECTIVENESS EVALUATION

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Abstract: Objectives: Analyze the literature data on the incidence and manifestation of carpal tunnel syndrome (CTS), as well as the pathogenesis and treatment options. Perform a specially designed, validated test - Pain Detection. Evaluate the objective state of patients with CTS - sensory impairment, compression test. Assess patients' neurologic and neurophysiologic data before and after the blockade and evaluate its effectiveness. Perform a specially designed, validated test - Patients' Global Impression of Change scale (PGIC) one month after corticosteroid injection (CSI).

Methods and Materials: The study includes an analysis of 55 arms of patients of different age with mild and moderate CTS who came for a neurological examination at the Neurology Outpatient Department of the Pauls Stradiņš Clinical University Hospital during the period of 01.08.2018 – 01.01.2019. All patients were analyzed clinically and neurophysiologically before CSI and one month after CSI. A Pain Detect scale, PGIC scale, compression tests and sensory tests were used for the evaluation of clinical symptom. A median nerve sensory and motor nerve conduction study was performed.

Results: According to the Pain Detect scale, 60% of patients showed neuropathic pain before CSI, and 78% of patients presented clinical effectiveness after CSI. 98% of patients present clinical effectiveness after CSI in the PGIC scale. 85% of patients had improvement in neurophysiological studies – motor distal latency decreased after CSI. Before CSI, the average motor distal latency was 5.7ms (range 4.5-12.9ms SD±1.5), which was on average 130% from the maximal norm (range 102-293 SD±36). After the CSI, the average motor distal latency was 5.2ms (range 3.8-10.7ms SD±1.3), which was on average 120% from the maximal norm (range 88-243 SD±30). We didn't find any significant correlation between the improvement of the patient's clinical condition and the improvement of electrophysiological outcomes.

Conclusions: The study concludes that the Pain Detect sensitivity for neuropathic pain evaluation of patients with CTS is 60%. Patients show clinical and neurophysiological improvement after CSI, but there is no correlation between neurophysiological and clinical improvement. The study concludes that the PGIC scale can be used to quickly assess the effectiveness of therapy.

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Introduction: Carpal tunnel syndrome (CTS) is the most common compression neuropathy (Atroshi et al., 1999; Blanc et al., 1996; Patterson, 2002; Katz et al., 2002). "Carpos" in Greek means a palm. "Tunnel" we define as a carpal tunnel. Median Nerve Compression Syndrome was first reported in medicine in 1854, but only a century later it was defined as CTS which we now use (Stecco et al., 2008). It is possible to diagnose CTS at a very early stage of the disease development and it tends to progress over time. CTS is a very topical problem because the disorder it causes can have a significant impact on the ability to work, social activity and care.

Literature review: The name of carpal tunnel syndrome is mainly based on the anatomy of the palm. Nervus medianus at the wrist level pass through a soft tissue canal called the wrist or carpal tunnel. CTS is median nerve damage, caused by nerve compression, which in results can manifest as a tingling sensation, sensation of numbness in the thumb, index finger, middle finger and partly also in the IV finger, which are more pronounced at night. At the late stages it manifests with muscle weakness, which can result in weakness in gripping objects and pain in the palm of the hand or wrist.

The disease can appear regardless of age. However, carpal tunnel syndrome is most commonly diagnosed between 40 to 60 years old and more often affects women (Atroshi et al., 1999). There are two peak frequencies - one between 45 and 59 (75% women) and the other between 75 and 84 (64% women) (Chammas et al., 2014).

The exact mechanisms and causes of carpal tunnel syndrome have not been fully understood. Damage occurs when, for some reason, the size of the tendons or the size of the muscles and connective tissue around the tendons increases as a result of an edema or infection, and the nerve is pressed. The pressure causes disturbances in the microcirculation of the nerve and if it is permanent and persistent, the myelin sheath can gradually disappear from nerves and the axon can be damaged. The available literature describes a number of combinations of pathophysiological mechanisms of CTS. The most

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common causes are trauma, endocrine diseases, and tissue metabolism disorders such as pregnancy and menopause, diseases of the connective tissue system, tumors and hygroma. This also includes patients with frequent and prolonged forced palm and wrist movements (e.g., packers) or prolonged tight work (e.g., work with a computer). Often, CTS occurs idiopathically (Chammas et al., 2014).

There is still no consensus on the standards for the diagnosis of carpal tunnel syndrome. The description of a set of symptoms, clinical findings, electrophysiological testing may be used. Clearly, correct diagnosis involves identifying the symptoms if they correspond to the median nerve projection area, which usually does not include the fifth finger. Historically, the diagnosis was based on anamnesis, clinical pictures and physical examination, but nowadays, with the advancement of technologies, electrophysiological methods are becoming increasingly important. Electrophysiological Studies (Robinson, 2007; Stevens et al., 1997), including Electromyography (EMG) and Neurography (ENG), are first-line selection studies recommended for the diagnosis of carpal tunnel syndrome (Chang et al., 2008). The ENG can measure the speed of motor and sensory nerve impulse control to determine nerve fiber damage, its nature and level (Preston et al., 2013; Jablecki et al., 1993). Mild and moderate nerve compression cause focal damage of the myelin sheath and are neurophysiologically manifested as a delayed distal latency of the motor nerve and the slowing in conduction velocity of the sensory nerve. Based on ENG, CTS is usually classified into mild, moderate and severe grades, but these evaluation criteria can differ in various laboratories. Severe nerve compression causes axonal damage and results in motor nerve amplitude reduction. Sensory fibers are more sensitive to compression than motor fibers. As a result, sensory fibers usually show earlier changes in ENG compared to motor fibers. In general, patients with mild carpal tunnel syndrome have only sensory disorders in electrophysiological tests, and patients with sensory and motor abnormalities have moderate carpal tunnel syndrome. However, any evidence of axonal loss (such as reduced or unresponsive response of a sensor or motor in a carpal tunnel area to ENG) is classified as severe carpal tunnel syndrome (Preston et al., 2013).

Steroid injections for CTS treatment are debatable and their effectiveness is assessed differently in different literature sources. CSI can only be made by a specialist. This treatment method is designed to relieve discomfort of patients with carpal tunnel syndrome, as it was able to relieve nerve edema. The goal is to administer the drug only intrasynovially, as there are many publications available in the literature that confirm the anabolic effect of steroid epineural injection (Shishido et al, 2002). The main thing is that the medicine comes under the fascia and synovial tissues (Babae - Ghazani et al, 2018). Steroid injections are not suitable for long-term treatment. In general, local steroid injections can only be used until the initial treatment has been effective, but the final therapy options have not yet been reached (Padua et al. 2016). In contrast, several other studies have shown that injecting steroids into the wrist is often successful. They may cause a temporary worsening of symptoms but may result in complete or significant reduction of pain from weeks to several years in 60 to 70% of cases (Mc Grath, 1984; Gelberman et al. 1980; Green, 1984; Giannini et al. 1991).

Data and methodology: This is a prospective case control study. The study includes the analysis of 55 arms of patients of different ages with a confirmed diagnosis of mild or moderate CTS, who came for a neurological examination at the Neurology Outpatient Department of the Pauls Stradiņš Clinical University Hospital during the period of 01.08.2018 – 01.01.2019. Written and verbal consent was obtained from each subject prior the injection and the ethical committee of the institute has approved the study protocol. The age of the patients was 18 to 90 years and with both genders. All patients were examined using Dantec Keypoint G4. Patients with median nerve motor distal latency less than 4.4 ms after nerve conduction, were not included in the analysis. We did not include pregnant women and patients with trauma of the affected hand requiring surgery or immobilisation from the previous 6 months. All patients were analyzed clinically and neurophysiologically prior and one month after CSI. At first, we performed nerve conduction studies (NCS) on the affected wrists. The studies were performed with the participant in a supine position using standard surface and ring electrodes. Then for clinical symptom evaluation we used a Pain Detect scale, PGIC scale, compression tests, sensory tests. The Pain Detect scale consists of seven questions that showed the quality of neuropathic pain symptoms. Five questions scored from 0 to 5 (never = 0, hardly noticed = 1, slightly = 2; moderately = 3, strongly = 4, very strongly = 5). The sixth question scored from – 1 to 2, The seventh question scored as 2 or 0. The final score was between 1 and 38, and indicates the likelihood of a neuropathic

pain component. A score of less than 12 points indicates that pain is nociceptive or unlikely to have a neuropathic pain component (< 15%). A score of 19 and more suggests that pain is likely to have a neuropathic component (> 90%). A score between these the values 13 – 18 indicates that the result is uncertain, and a more detailed examination is required to ensure a proper diagnosis. However, a neuropathic pain component can be present (Freyenhagen, 2006). After that CSI was performed with the arms of patients extended and wrists rested on a hard-flat surface. We used a sterile technique. The affected wrists were first cleaned with Betadine 10% solution and superficially injected with a 1 ml of solution Lidocaine 1%. Then a 23-gauge (0.6mm) 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 subcutaneous solution of 4 mg/ml dexamethasone was injected into the area surrounding the median nerve. Patients were advised to wait for 15 min following the injection and to rest the injected arm till the next day. One month after injection, we repeated the median nerve sensory and motor nerve conduction study. In the study we agree that mild CTS did not have any motor disturbance (motor distal latency is less than 4,4 ms), only sensory involvement. Moderate CTS – motor distal latency is $\geq 4,4$ ms, but motor nerve amplitude is not changed ($>4 \mu\text{A}$). Severe CTS – reduces motor nerve amplitude ($< 4 \mu\text{A}$) (axonal lose). One month after, patients filled out the PGIC scale. The PGIC scale consists of 7 scores, where 1 – means very much improved, 4 – no change and 7 – means very much worsening. The study also shows an analysis of the duration of the patient's illness and its association with neurophysiologic data and therapeutic effectiveness.

All statistical analyses were performed with *Microsoft Excel 8.0* and *SPSS (Statistical Package for the Social Sciences) for Windows 10.0*. The data was expressed as mean and standard deviation or median and range, as appropriate, for parametric variables and as numbers and percent for non-parametric variable.

Results: Demographic and clinical features of the CTS patients are summarized in Table 1.

	Means	SD
Age (years)	64.2	± 11.4
Sex (female/male)	(43/12)	-
Duration of illness (years)	9.0	± 8.9

Source: Authors

Fifty-six patients with CTS (43 women and 12 men) were enrolled. The mean age of patients with CTS was 64.2 (range 34 – 86 years, $\text{SD} \pm 11.4$), the mean disease duration was 9.0 (range 0.6 – 30 years, $\text{SD} \pm 8.9$) years. CTS are more common for women than men (43/12). There were 55 patients who were right-hand dominant and in 52 patient cases from 55 the dominant hand was affected (92.8%). There were 11 of 55 (19.6%) patients who suffer from diabetes mellitus type 2. There were 19 of 55 (33.9%) patients who received CSI before and 4 of 55 (7.1%) patients who had had surgical treatment before. Only 6 of 55 (10.7%) patients underwent ultrasound of the carpal tunnel prior to blockade.

CTS patients clinical finding are summarized in Table 2.

	Before CSI (n=55)	After CSI (n=55)
Day time pain	55 (100%)	17 (30%)
Night time pain	54 (98%)	13 (23%)
Positive sensory symptoms	55 (100%)	10 (18%)
Positive Phalen test	47 (85%)	7 (12%)
Positive Tinel sign	45 (82%)	4 (7%)

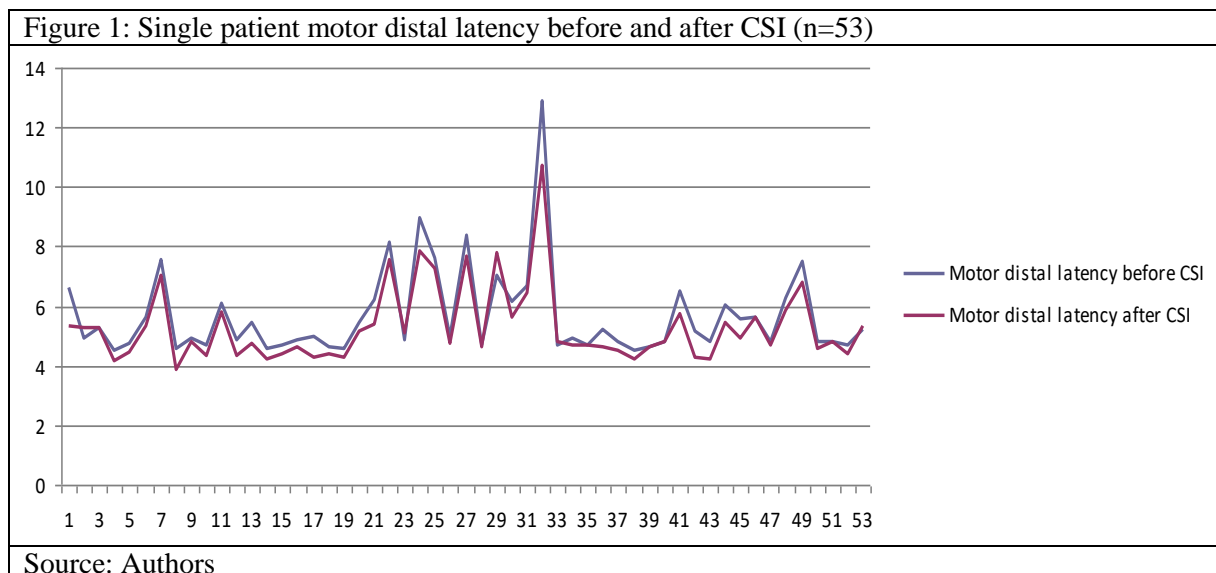
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Baseline clinical findings (before CSI). There were 55 of 55 (100%) patients who had sensory symptoms. The Phalen test was positive for 47 of 55 (85%) patients and Tinel sign positive for 45 of 55 (82%) patients. There were 55 of 55 (100%) patients who suffered from day time pain and 54 of 55 (98%) patients who suffered from night time pain.

One month after the CSI, patients had clinical improvement. There were 10 of 55 (18%) patients who had sensory symptoms after CSI. The Phalen test was positive for 7 of 55 (12%) patients and Tinel sign positive for 4 of 55 (7%). There were 17 of 55 (30%) patients who suffer from day time pain and 13 of 55 (23%) patients who suffer from night time pain one month after CSI.

According to the Pain Detect scale 33 of 55 (60%) patients showed neuropathic pain before CSI, 36 of 55 (78%) patients present clinical effectiveness after CSI. 54 of 55 patients (98%) presented clinical effectiveness after CSI in the PGIC scale.

45 of 53 (85%) patients had improvement in neurophysiological studies – motor distal latency decreased after the CSI. Before the CSI, the average motor distal latency was 5.7 ms (range 4.5-12.9ms SD±1.5), which was on average 130% from the maximal norm (range 102 – 293 SD±36). After CSI, average motor distal latency was 5.2 ms (range 3.8-10.7ms SD±1,3), which was on average 120% from the maximal norm (range 88-243 SD±30). There were 26 of 53 (57%) patients which NCS data one month after CSI showed improvement less than 10% compared to the single patient baseline condition, 16 of 53 (36%) showed improvement from 10 to 20% and only 3 of 53 (7%) patients show 20 to 30% improvement after CSI. There were 8 patients whose NCS data after CSI showed a little worsening. In our study, there were 2 of 55 patients who had extremely severe CTS and their median nerve motor distal latency was not registered neither in baseline, nor after CSI, that is why we cannot analyze their nerve conduction study results. Each patients` motor distal latency before and after CSI is shown in Figure 1.



Discussion

The present study included 55 CTS patients, mostly women. Many other studies also show that women are more prevalent than men (Atroshi, 1999; Chammas et al., 2014; Akalin et al., 2002; Baysal et al., 2006). There are many hypotheses and suggestions that the higher incidence of CTS of women may be partly due to hormonal factors (Ferrero et al, 2001; Wilgis, 2002). Many other studies show that CTS can be related to a higher frequency of musculoskeletal problems among women (McDiarmid et al., 2000; Shabir, 2004). In this study, 52 of 55 patients had their right hand affected which is their dominant hand. Bland et al. (2003) had a similar result - the most and the hardest suffering is the right hand because it is the dominant hand. This study shows better clinical outcomes in sensory symptoms (negative in 83% cases), Phalen's test (negative in 88% cases) and Tinel's sign (negative in 93% cases). In Agarwal et al.'s (2005) study they found that none of the 48 patients in their study had a positive Phalen's test or Tinel's sign after CSI and only two patients continued to complain of paraesthesia after steroid injection.

Neuropathic pain is a common clinical presentation. In our study, according to the Pain Detect scale, 33 of 55 (60%) patients show neuropathic pain. The Pain Detect questionnaire is a useful tool for clinicians, to screen for neuropathic pain in patients. Its sensitivity and specificity are high (Freynhagen, 2006).

The neurophysiological parameters (median nerve motor distal latency, motor amplitude) also showed improvement following steroid injection. Similar electrodiagnostic findings have been reported previously following steroid injection for CTS. The improvements of nerve conduction study parameters independently support the effectiveness of steroid injection therapy in CTS (Hagebeuk et al., 2004). In our study before CSI the average motor distal latency was 5.7 ms and after CSI the average motor distal latency was 5.2 ms. These results are particularly similar to Mangonon et al.'s (2014) study who found similar results. Their study enrolled one hundred forty-five patients with suspected CTS, and before injection the mean distal motor latency was 5.01 ms, but after injection the mean distal motor latency was 4.82 ms (Mangonon et al., 2014). We didn't find any sensory conduction velocity or sensory nerve amplitude improvement after CSI, even for patient who had significant motor distal latency improvement.

There was no statistically significant correlation between CTS patients' disease duration, NCS data and therapeutic effectiveness. We didn't find any significant correlation between the improvement of the patient's clinical condition and the improvement of electrophysiological outcomes. Pain is a subjective parameter and it is very difficult to measure it, and maybe this is also one of the reasons why our data didn't correlate. Coghill et al.'s (2003) research found that more sensitive individuals activated greater and more often regions of the brain– the primary somatosensory cortex, the anterior cingulate cortex. In earlier studies they analyzed the differences in individual pain perceived that effect these regions' activation which results in the increase of these regions stimulation intensity and increase of the intensity of pain perception (Coghill et al, 1999). Consequently, these differences in the primary somatosensory cortex and the anterior cingulate cortex are in line with the differences in the intensity of individuals' pain intensity and underline the fact that some differences in pain are real. To achieve statistically significant results, the study will be continued to collect a larger patient pool.

Conclusions

The treatment with steroid injection is effective for patients with CTS. We inferred that the PGIC scale can be used to quickly assess the effectiveness of the therapy. This study shows that the Pain Detect sensitivity for neuropathic pain evaluation for patients with CTS is in 33 of 55 (60%) cases. Patients showed clinical and neurophysiological improvement after CSI, but there is no correlation between neurophysiological and clinical improvement. In moderate and severe CTS, analyses of motor distal latency is more sensitive than sensory distal latency.

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