# Local corticosteroid treatment for carpal tunnel syndrome: A 6-month clinical and electrophysiological follow-up study

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**Abstract**. *Objective*: To evaluate the clinical and electrophysiological effects of local depo-methylprednisolone injection in patients with carpal tunnel syndrome (CTS) over a 6-months period.

Methods: Twenty one patients, of whom 7 were lost for follow-up (mean age  $57.9 \pm 8.4$ ) with clinical and electrophysiological evidence of CTS were treated by injection of depo-methylprednisolone 40 mg proximal to the carpal tunnel. Severity of pain (VAS), rates of numbness/paresthesias and nocturnal awakening, motor and sensory nerve conduction studies were used as outcomes. All tests were performed before, 1, 3 and 6 months after the injection.

Results: Severity of pain was significantly reduced at all follow-up time points (p < 0.001). Prior to injection all patients complained of night pain and awakening. On the first, third and sixth months, 0(0%), 4(29%) and 7(50%) of the patients, respectively, had night awakening. All patients complained of numbness before the treatment. This symptom disappeared in 81% of the patients after one month and reappeared in all after three months. Significant improvement was shown in the mean distal motor latency (DML) of the median nerve:  $5.2 \pm 0.9$  msec. before,  $4.6 \pm 0.6$  msec. and  $4.7 \pm 0.6$  msec. 1 and 3 months after the injection, respectively (p < 0.05). Mean values of motor muscle potential amplitudes, sensory latency and sensory amplitude did not change significantly after the treatment.

Conclusions: Local corticosteroid injection for the treatment of CTS provides significant symptom improvement for three months. No electrophysiological parameters were improved after injection, except the improvement in distal motor latency of the median nerve

Keywords: Carpal tunnel syndrome, conservative treatment, corticosteroid injections, electrophysiological studies

# 1. Introduction

Carpal tunnel syndrome (CTS) is the most frequent entrapment neuropathy. The direct medical costs associated with CTS were estimated in the early 1990s to be more than \$1 billion per year in the United States [24]. The diagnosis of CTS is based on clinical evidence of median nerve deficit distal to the wrist and confirmatory nerve conduction studies if the diagnosis is uncertain [23]. Carpal tunnel release is the most common surgical procedure performed on the hand, with over 200,000 procedures being performed each year in the United States [14]. Surgical decompression is effective in 75–99% and permanent complications occur in less than 1% [4,5,11]. However, there are still many reasons to choose conservative care for CTS over surgery. For example, patient's refusal for operation, its relatively high cost, and patients at high risk for general or local

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anesthesia, etc.

Non-surgical approaches, such as oral steroids, nonsteroidal anti-inflammatory drugs, diuretics, pyridoxine, wrist splints and physical therapy methods such as ultrasound and mobilization of the carpal joints have been studied in small, randomized trials, with no evidence of long-term efficacy [7–9,11,13,35].

Steroid injection into the carpal tunnel is safe, easy to perform, reduces symptoms of CTS significantly [1–3,10,12,15–17,19,21,29,34], and has been shown to be superior to placebo in randomized clinical trials [10, 15,17]. However, such injections carry a small risk of nerve damage [25,33]. Dammers et al. [10] suggested an injection of 40 mg methyl prednisolone proximal to the carpal tunnel as an alternative. In their randomized, double-blind, placebo-controlled trial, such injection was beneficial in 77% of the patients when evaluated after 1 month and had positive effects over 1 year in 50%. However, the main outcome measure in this study was the subjective patients' self report of improvement (defined as no or minor symptoms requiring no further treatment).

Most of the studies investigating the beneficial effect of corticosteroid injection for CTS used subjective measures, such as patients' appraisal of improvement, pain, Boston Carpal Tunnel Questionnaire, etc., as main outcome parameters. Objective neurophysiological measures were less often evaluated [1,3,15–18, 21,28,32], and even fewer studies evaluated long term effect of the corticosteroid injection for CTS.

The aim of the present study was to evaluate the clinical and electrophysiological effects of local depomethylprednisolone injection proximal to the carpal tunnel in patients with CTS over a 6-months period.

# 2. Methods

# 2.1. Patients

Patients referred to the neurologic or rheumatologic clinics at a local HMO clinic (Meuhedet, Ashkelon) with clinical signs of moderate to severe CTS were screened for eligibility criteria. Clinical diagnosis was made on a basis of the presence of pain and/or paraesthesias in the distribution of the median nerve, nocturnal exacerbation of symptoms, and positive Phalen's maneuver and/or Tinel's sign. Exclusion criteria included: diabetes mellitus; wrist trauma or deformity; evidence of polyneuropathy/radiculopathy or double-crush syndrome on electro-diagnostic studies; ad-

vanced CTS with thenar atrophy or marked weakness; marked axonal loss on nerve conduction study or non-stimulatable nerves; previous surgery or local injection for CTS; pregnancy; systemic disorders (rheumatoid arthritis, hypothyroidism, amyloidosis, etc.). Patients with prominent thenar atrophy were excluded and referred to a hand surgeon. Most patients had some degree of hand osteoarthritis. Twenty one patients (17 females, 4 males) were found eligible, signed an informed consent form and were included in the study. Clinical evaluation and electrophysiological studies were performed 2–3 days before, 1, 3 and 6 months after the injection. Only patients returning for the clinical and EMG follow-up evaluations were included in the final analysis.

### 2.2. Clinical evaluation

One study physician (T.R.) performed the clinical evaluation in all study patients during scheduled study visits. Severity of pain during the last week was assessed on a 10 cm visual analogue scale (VAS). Subjects were also asked about the presence of numbness/paraesthesias, nocturnal pain or awakening at any time during the last week. Their positive or negative answers regarding each symptom were recorded at preintervention and at each follow up visit.

# 2.3. Electrophysiological studies

Nerve conduction studies were performed according to the American Association of Electrodiagnostic Medicine guidelines for CTS [23] and consisted of electromyography examination of the abductor pollicis brevis and adductor digiti minimi muscles and motor and antidromic sensory conduction velocities of the median and ulnar nerves. One investigator (L.V.) carried out all neurophysiological studies on a Dantec Key-Point machine on the inclusion date 2–3 days before treatment and at 1, 3 and 6 months after injection. At followup visits, the investigator was no longer aware of the results of the previous neurophysiological studies. To enhance the reproducibility of the neurophysiological studies during follow-up, each (repeated) measurement was carried out using the same locations of the stimulating and recording electrodes. For example, the ring electrodes for sensory distal latencies of the median nerve were placed around the proximal interphalangeal joints of the second digit. The stimulating electrode was placed proximal at the wrist at a distance of 14-15 cm from the ring electrode of the second digit (de-

Table 1 Clinical data at baseline (0) and 1, 3 and 6 months after injection

Parameter	0	1 month	3 months	6 months
VAS for pain (Mean $\pm$ SD)	$9.3 \pm 0.5$	$2.0 \pm 0.5^*$	$2.9 \pm 0.6^*$	$6.1 \pm 0.6^*$
Night awakening (%)	100	0* *	29**	50**
Numbness (%)	100	19**	100	100

<sup>\*</sup>The results of comparison against baseline (t-test) are statistically significant at p < 0.001 level.

pending of the hand size of the patient). The distance between the stimulating and the recording electrodes were measured by a measuring tape and recorded. The same distances were used for repeated measurements. The same measures were applied for the ulnar nerve. To record the distal motor latency (DML) of the median nerve, surface recording electrodes were placed over the abductor pollicis brevis muscle 6–7 cm distal to the proximal stimulating electrode at the wrist (depending of the hand size of the patient). All studies were performed with the patient supine in a room with the temperature kept at  $25\,^{\circ}\text{C}$ .

# 2.4. Intervention

A single local injection of 1 ml containing depomethylprednisolone (40 mg) and lidocain hydrochloride (10 mg) was given by a rheumatologist (T.R.). The injection was given with a 2.5 cm long 0.6 mm needle at the volar side of the forearm 4 cm proximal to the wrist crease, between the tendons of the flexor carpi radialis and the palmaris longus muscles. The angle of introduction of the needle depended on the size of the wrist. In participants with a thin wrist the median nerve is close to the skin. In these participants the angle was  $10^{\circ}$ . The angle was larger, about  $20^{\circ}$ , in those with a thick wrist. In participants with well developed muscles, the pronator quadratus muscle may push up the median nerve, so in a thick muscular arm the angle of introduction was also flat, between 10° and 20°. After insertion the needle was moved up slowly for about 2 cm to a point as close to the wrist crease as possible. The needle was repositioned if the patient had paraesthesias or pain in the distribution of the median nerve in the hand. The injected fluid was massaged towards the wrist crease (procedure according to Dammers et al. [10]). In the case of bilateral symptoms only the more affected hand was treated.

### 2.5. Statistical analysis

As the VAS and electrophysiological parameters are continues variables and were approximately Gaussian distributed, we used parametric tests. A paired t-test was used to compare the baseline data with data at 1, 3 and 6 months follow-up. To compare the presence of numbness/paraesthesias and night pain or awakening complains at the baseline with data at 1, 3 and 6 months follow-up we used  $\chi^2$  test. All statistical analyses were performed using SPSS 15 software.

### 3. Results

Fourteen patients (12 females and 2 males, age 46–70, mean 57.9  $\pm$  8.4) with symptomatic idiopathic CTS returned for all follow-up clinical and EMG evaluations, and their results were included in the statistical analyses. Seven patients (5 females and 2 males, age 34–76, mean 59.3  $\pm$  14.9) were lost to follow-up after injection. Those patients were statistically not different (p>0.05) from those that completed follow-up evaluations in terms of the pre-treatment VAS for pain, rate of numbness and nocturnal awakening and electrophysiological studies.

The clinical parameters at baseline and 1, 3 and 6 months after injection are shown in Table 1. Severity of pain, evaluated by VAS, was significantly reduced during the follow-up period (p < 0.001). Although mean VAS score at month six was slightly higher than that at month three, it was still significantly lower than the VAS score at baseline. Prior to treatment all patients complained of night pain and awakening. One month after the injection no patient had night pain or awakening. After 3 and 6 months, only 4 (29%) and 7(50%) of the patients, respectively, had night awakening. All patients complained of numbness/paraesthesias before the treatment. This symptom disappeared in 81% of the patients after one month and reappeared in all after three months.

<sup>\*\*</sup>The results of comparison against baseline ( $\chi^2$  test) statistically significant at p < 0.005 level.

Table 2 Electrophysiological data at baseline (0) and 1, 3 and 6 months after injection

Parameter	0	1 month	3 months	6 months
DML, msec. (Mean $\pm$ SD)	$5.2 \pm 0.9$	$4.6 \pm 0.6^*$	$4.7 \pm 0.6^*$	$4.7 \pm 0.9^{\#}$
Motor amplitude, $\mu V$ (Mean $\pm$ SD)	$5.8 \pm 2.8$	$5.7 \pm 2.1^{\#}$	$5.9 \pm 3.2^{\#}$	$5.6 \pm 2.5$ #
SDL2, msec. (Mean $\pm$ SD)	$4.3 \pm 2.2$	$4.6 \pm 1.3^{\#}$	$4.7 \pm 0.9^{\#}$	$4.6 \pm 1.5$ <sup>#</sup>
Sensory amplitude, $\mu V$ (Mean $\pm$ SD)	$16.9 \pm 18.3$	$21.8 \pm 17.7$ #	$19.1 \pm 10.2$ #	$17.6 \pm 13.3 $ #

Abbreviations: DML - distal motor latency, SDL2 - sensory distal latency to the second digit.

Table 2 shows the electrophysiological data at baseline and 1, 3 and 6 months after injection. Distal motor latency (DML) generated by stimulating the affected median nerve at the wrist was prolonged in all patients (mean:  $5.2\pm0.9$  msec.). Significant improvement was observed in the mean values of DML:  $4.6\pm0.6$  msec. and  $4.7\pm0.6$  msec. 1 and 3 months after the injection, respectively (p<0.005). Although mean DML was  $4.7\pm0.9$  msec. six months after the injection, the difference did not reach statistical significance because of a high values of standard deviation. Mean values of motor muscle potential amplitudes, sensory distal latency of the second finger (SDL2) and sensory amplitudes did not change significantly after the treatment.

# 4. Discussion

Our results demonstrate that local corticosteroid injection for CTS results in marked symptom improvement for 3-6 months. At baseline, all patients complained of numbness in the hand and night awakening. One month after steroid injection, numbness/paraesthesias disappeared in 81% of the patients and none had night awakening. Mean VAS for pain decreased from  $9.3 \pm 0.5$  to  $2.0 \pm 0.5$ . In addition, electrophysiological studies showed a significant decrease in DML from  $5.2 \pm 0.9$  to  $4.6 \pm 0.6$ . This remarkable improvement is in line with previous evidence-based analyses of the effectiveness of local corticosteroid injection in CTS [27]. This improvement was still prominent after three months. By that time, mean VAS was 2.9  $\pm$ 0.6, no night awakening was still reported by 71% of the patients, and DML mean value was  $4.7 \pm 0.6$ . On the other hand, numbness/paraesthesias reappeared in 100% of the patients after 3 months. After six months the remaining effect was modest: VAS value increased to 6.1  $\pm$  0.6; night awakening reappeared in 50% of the patients and the DML was no longer different from baseline values. This limited long-term effectiveness of steroid treatment has been noted by many authors [15,

21,22,27,30]. This is probably due to temporary antiedematous effects of the injected steroids, without alteration of the original causes of CTS. Additional studies are needed to evaluate the effects of combined treatment of local steroid injection with evaluation and modification of occupational and extra-occupational factors that originally lead to CTS [6].

We used the method described by Dammers et al. [10], in which the injection is given proximal to the carpal tunnel. The advantage of this method is its lower risk of damage to the median nerve. No adverse effects or worsening in clinical or electrophysiological parameters were noted over 6 months in any of our patient. Therefore, the results of our study support the use of this method. In addition, our study confirms previous studies demonstrating that steroid injection is a safe, easy to perform and effective short-term treatment for CTS, suggesting that this treatment may be considered as an option before a decision on surgical treatment is made. Only one study [21] reported on changes in objective nerve conduction parameters after corticosteroid injection using this method of Dammers et al. [10]. In this study the authors found significant improvement in Boston Carpal Tunnel Questionnaire score and in electrophysiological parameters such as SDL4, DML and others even six months after the injection. However, the mean baseline electrophysiological parameters in Hagebeuk and De Weerd's study [21] were much lower than in our study. For example the baseline SDL2 in this study was 3.5  $\pm$  0.6 vs. 4.3  $\pm$ 2.2 in our study, mean baseline DML was  $4.5 \pm 1.3$  vs.  $5.2 \pm 0.9$  in our study. The improvement in subjective parameters in our study is in agreement with previous reports by Dammers et al. [10], and others who used the traditional method of injection [3,12,17,29]. In the present study we found improvement in DML that was still significant after 3 months, which is in agreement with previous reports [1,21,28]. On the other hand, contrary to Hagebeuk and De Weerd [21], Agarwal et al. [1] and others who found improvement in both motor and sensory nerve conduction parameters, we did

<sup>\*</sup>Statistically significant (t-test) from the baseline data at p<0.005 level.

<sup>#</sup>Not statistically significantly different from the baseline data.

not find any change in SDL2 or sensory amplitude after corticosteroid injection. The only electrophysiological change we found after steroid injection was the DML, indicating improvement in the demyelinating component of the damage to the motor nerve fibers, but not to the sensory nerve fibers which are affected earlier in the course of the disease. In our study, in contrast to other studies (e.g. Hagebeuk and De Weerd [21], Demirici et al. [12]), the sample comprised individuals with moderate to severe chronic CTS with very prominent clinical and electrophysiological signs, including significant prolongation of both SDL2 and DML. Gupta et al. showed that an early consequence of chronic nerve compression (CNC) is local demyelination, and therefore we hypothesize that anti-edematous effects of the steroid injection on the entrapped nerves [36] affect motor nerve fibers, which are richer in myelin than the sensory ones, to a greater extent. Additionally, there were several studies showed that corticosteroids have roles as neuroprotective agents against demyelination and augmentative agents for remyelination [37].

The main limitations of our study are the lack of a control or placebo group and the relatively high number of patients who did not return for follow up evaluations (7 of 21 or 33.3%), and, in general, relatively low sample size, which may potentially bias the results of our study. The effect of local steroid injection in comparison with placebo was already convincingly demonstrated by Dammers et al. [10]. Hagebeuk and De Weerd [21] in their study showed improvement in clinical and nerve conduction parameters after steroid injection proximally to the carpal tunnel. Therefore, we considered it unethical to use placebo as a control in our study. Moreover, two previous placebo controlled studies, one of hand brace [26] and the other one of ultrasound therapy [31] showed no significant changes in neurophysiological measurements in the placebo group after approximately 1 month.

The seven patients which were lost to follow-up did not differ from the 14 patients included in this study in terms of mean baseline VAS, numbness, nocturnal awakening and DML. It may be argued that different treatment outcomes in these patients may cause a bias in the results and our conclusions. However, because the recruitment of the patients to the study took place at a local HMO, which serves as a primary health care provider and the administrative "gate" for referral to secondary and tertiary medical centers, no-return of patients for follow-up or for additional treatment more likely indicates a good outcome and disappearance of symptoms rather that a poor outcome or the emergence of any complications.

### 5. Conclusions

This is one of the few studies that evaluate the short and long term changes in objective parameters after steroid injection for CTS. We used the proximal site for steroid injection and our study supports the idea that this is a safe site for this type of intervention. Local corticosteroid injection for the treatment of moderate to severe CTS provides significant improvement in symptoms for three months, although longer improvement in some parameters can be observed. We did not see a significant improvement in electrophysiological parameters, except for improvement in distal motor conduction of the median nerve. This short term benefit suggests that local injection of corticosteroid may be useful in CTS patients who refuse or cannot undergo surgical treatment, or as a conservative treatment option before considering surgery.

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