Authors:

Reyhan Çeliker, MD
Şule Arslan, MD
Fatma İnanıcı, MD

Affiliations:

From the Department of Physical Medicine and Rehabilitation, Hacettepe University School of Medicine, Ankara, Turkey.

Disclosures:

Presented at the EULAR 2000 Congress, June 2000, Nice, France.

Correspondence:

All correspondence and requests for reprints should be addressed to Reyhan Çeliker, MD, Hacettepe Üniversitesi Tıp Fakültesi, Fiziksel Tıp ve Rehabilitasyon Anabilim Dalı, Ankara 06100, Turkey.

0894-9115/02/8103-0182/0
American Journal of Physical Medicine & Rehabilitation

Research Article

Corticosteroid Injection *vs.* Nonsteroidal Antiinflammatory Drug and Splinting in Carpal Tunnel Syndrome

ABSTRACT

Çeliker R, Arslan Ş, İnanıcı F: Corticosteroid injection *vs.* nonsteroidal antiinflammatory drug and splinting in carpal tunnel syndrome. *Am J Phys Med Rehabil* 2002;81:182–186.

Objective: To compare the efficacy of local corticosteroid injection to a nonsteroidal antiinflammatory drug and splinting for the treatment of carpal tunnel syndrome.

Design: This study was a prospective, unblinded, randomized clinical trial with an 8-wk follow-up. Thirty-three hands of 23 patients were randomly treated with acemetacine and splinting (group A) or with corticosteroid injection (group B). Clinical (symptom severity scale, visual analog scale, Tinel and Phalen tests) and electromyographic evaluations were performed on initial visit and after 8 wk.

Results: Clinical and electromyographic parameters, which were similar at baseline, were improved in both groups after treatment. Improvement was also similar when both groups were compared at 8 wk.

Conclusion: Both splinting combined with the use of a nonsteroidal antiinflammatory drug and steroid injection into the carpal tunnel resulted in significant improvement in carpal tunnel syndrome.

Key Words: Carpal Tunnel Syndrome, Corticosteroid Injection, Splinting, Symptom Severity Scale

The carpal tunnel is bordered posteriorly by the carpal bones of the wrist and anteriorly by the transverse carpal ligament. Because of these anatomic restrictions and the pressing role of repetitive use of the flexor and extensor tendons in the tunnel, carpal tunnel syndrome (CTS) is the most common entrapment neuropathy.[1] The prevalence is 9.2% in women and 0.6% in men.[2]

A hallmark of CTS is nocturnal pain. Most of the patients relate a history of wakening in the middle of the night with painful numbness in the hand.[2] The most common sensory and motor impairments are decreased tactile sensation and numbness in the first three digits, pain in the hand, forearm, elbow or even shoulder, and weakness of the hand. These impairments may cause a disability in the performance of activities of daily living.

Initial choice of treatment for CTS may be influenced by various factors, such as age, duration of symptoms, occupation, severity of clinical findings, severity of electromyographic findings, previous treatment, and opportunity for job modification.[3,4] Splints and nonsteroidal antiinflammatory drugs (NSAID) are the two most frequently used interventions.[5] The therapeutic effect of wrist splinting arises from minimizing carpal tunnel pressure, which increases with wrist positions away from neutral. Flexion of the wrist causes the flexor tendons of the fingers to be displaced against the palmar side of the carpal tunnel, increasing pressure on both tendons and the median nerve. Extension of the wrist causes the tendons to be displaced against the dorsal side of the tunnel and distal radius, again producing increased pressure.[1] Other treatment options include steroid injections, surgery, physical therapy and occupational rehabilitation. Many patients with CTS can be treated nonsurgically with satisfactory results. The aim of this study was to define and compare the role of steroid injection *vs.* splinting combined with NSAID use in the treatment of CTS.

SUBJECTS AND METHODS

This study was a prospective, unblinded, randomized clinical trial with an 8-wk follow-up. Consecutive patients were recruited for the study if electrodiagnostic tests confirmed unilateral or bilateral CTS. When bilateral CTS was present, measures were taken for each hand. No patient had previously received any treatment for CTS. There were no exclusion criteria other than thenar atrophy. Clinical examinations were performed on initial visit and repeated on second and eighth weeks. Tinel, Phalen, and reverse Phalen signs were tested. Phalen and reverse Phalen tests were performed for a minimum of 60 sec. Pain was also assessed by the Visual Analog Scale (VAS). Patients were given the self-administered Symptom Severity Scale.[6] It consists of 11 questions. The answers were rated from 1 to 5 points. The overall score was calculated as the mean. Electrophysiologic examinations were performed before treatment and on the eighth week. By using standard techniques, all electrodiagnostic tests were performed by the same physician with a Medelec (UK), Synergy Version 2.0, electromyography apparatus.

Electrophysiologic Studies. Median and ulnar sensory and motor nerve conduction studies were performed on both hands of all patients. The active surface electrode (E-1) was placed one-half the distance between the metacarpophalangeal joint of the thumb and the midpoint of the distal wrist crease for the measurement of median nerve motor conduction velocity and latency. The reference surface electrode E-2 was placed on the distal phalanx of the thumb. The ground was between the E-1 electrode and the stimulating electrode. Stimulation was applied with the cathode 8 cm proximal to E-1, between the flexor carpi radialis and the palmaris longus tendons. Proximal stimulation was applied in the medial aspect of the antecubital space, lateral to the brachial artery.[7] Sensory conduction studies were performed by using the antidromic technique.

For the measurement of median nerve distal sensory latency, ring electrodes were placed on the third digit. The E-1 and E-2 electrodes were placed 4 cm apart, with the E-1 electrode proximal at the base of the digits. The ground was placed between the E-1 electrode and stimulation cathode. Stimulation was applied 14 cm proximal to the E-1 ring electrode and over the median nerve between the tendons of the palmaris longus and flexor carpi radialis, and proximal stimulation was applied at the antecubital space.

The E-1 surface electrode was placed on the abductor digiti minimi on a point midway between the distal wrist crease and the crease at the base of the fifth digit and at the junction of the dorsal and palmar skin for the measurement of ulnar motor nerve conduction velocity and the distal latency.[8] The E-2 electrode was placed on the fifth digit. The ground was between the stimulating cathode and E-1 electrode. Stimulation was applied 8 cm proximal to the E-1 electrode just over the flexor carpi ulnaris tendon and over the ulnar groove at the elbow. Ring E-1 and E-2 electrodes were placed on the fifth digit, with at least a 4 cm separation, if possible. The E-1 electrode was proximal at the base of the digit. Stimulation was applied 14 cm proximally, radial to the flexor carpi ulnaris, and at the elbow in the ulnar groove.

Treatment. Patients were randomly assigned to one of the two groups by using sequentially numbered sealed opaque envelopes; the patients in

group A were treated with splinting and acemetacine 120 mg/day. Lightweight, neutral-positioned wrist splints were used just at night. Group B received 40 mg methylprednisolone acetate (1 ml) injected locally by the same investigator (R. Çeliker). For injection of steroid suspension, the patient sat with the forearm supported in full supination and the wrist extended. The point of entry was about 4 cm proximal to the wrist at the midline or just to the radial side of the palmaris longus. A 22-guage needle was angled almost horizontally and passed its full length into the carpal tunnel without piercing either the tendon or the nerve. If median paresthesias were elicited, the needle was repositioned, and then the steroid suspension was injected. The entire suspension was discharged under the transverse ligament.[9,10]

Statistical Analysis. SPSS 9.0 program (SPSS, Inc., Chicago, IL) was used for statistical analysis. χ^2 and Fisher's exact tests were used to compare the differences between groups for clinical assessment and physical findings. Pretreatment and posttreatment measures for VAS, Symptom Severity Scale, and median nerve motor and sensory nerve conduction studies were compared by using Wilcoxon test. Mann Whitney *U* test was used to compare these measures between groups.

RESULTS

A total of 23 patients were enrolled in the study. Fourteen (60.9%) patients had bilateral involvement, giving a total of 37 hands. Table 1 shows the baseline characteristics of the patients. No significant differences existed between groups for age, duration of the symptoms, VAS scores, and Symptom Severity Scale scores. All patients were right-handed. Five (45.5%) patients in group A and four (33.3%) patients in group B were in menopause. None of the patients had a history of diabetes mellitus, hypothyroidism, rheumatoid arthritis, acromegaly, or amyloidosis.

Phalen test was positive in 13 (81.3%) hands in group A and 14 (66.6%) hands in group B before treatment. Tinel sign was positive in 15 (93.8%) hands in group A and in 19 (90.5%) hands in group B. Reverse Phalen test was positive in 11 (68.8%) hands in group A and in 13 (61.9%) hands in group B. When tested after treatment, in group A, Tinel sign was positive in two (12.5%) hands and Phalen and reverse Phalen tests were negative for all hands. In group B, Phalen test was positive in three (14.3%) hands, Tinel sign in six (28.6%) hands, and reverse Phalen in one (4.8%) hand. There was not statistically significant differences for these physical findings between two groups ($P > 0.05$).

Pretreatment and posttreatment (at 8 wk) VAS scores were statistically not different between two groups ($P > 0.05$) but decreased significantly after treatment in both groups ($P < 0.05$, Table 2, Fig. 1). When evaluated with the Symptom Severity Scale, there was a significant improvement in both groups ($P < 0.05$, Table 2).

Motor and sensory distal latencies were also improved in both groups after treatment ($P < 0.05$). Table 3 shows data from the electrodiagnostic evaluation of the patients. In group A, sensory distal latencies did not change in three (18%) hands, and motor distal latencies decreased in all hands. In group B, sensory and motor distal latencies did not change in two (9.5%) hands. The difference between groups for unresponsiveness was statistically not significant ($P > 0.05$).

TABLE 1
Baseline characteristics of patients

	Group A $n = 11$	Group B $n = 12$
Mean age, yr ± SD	49.6 ± 15.3	46.9 ± 10.0
Sex, male/female	0/11	1/11
Bilateral CTS, *n*	5	9
Duration of symptoms, months ± SD (range)	6.9 ± 6.9 (1–24)	8.5 ± 16.4 (1–60)
VAS score, cm ± SD	7.9 ± 1.4	7.0 ± 2.2
Symptom severity scale, mean ± SD	3.5 ± 0.7	3.4 ± 1.1

CTS, carpal tunnel syndrome; VAS, Visual Analog Scale.

TABLE 2
Mean values of Visual Analog Scale and Symptom Severity Scale scores

	Group A $n = 11$	Group B $n = 12$
Visual Analog Scale Scores, cm		
Baseline	7.9 ± 1.4	7.0 ± 2.2
2nd wk	4.3 ± 0.9	3.1 ± 2.5
8th wk	1.7 ± 1.0	1.8 ± 1.9
Symptom Severity Scale		
Baseline	3.5 ± 0.7	3.7 ± 1.1
2nd wk	1.8 ± 0.5	1.8 ± 1.0
8th wk	1.3 ± 0.3	1.4 ± 0.7

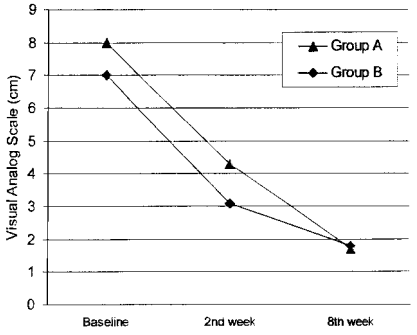

Figure 1: Visual Analog Scale scores before and after treatment.

TABLE 3
Mean values of motor and sensory distal latencies of median nerve

	Baseline	8th Wk	*P*	Improvement
Motor distal latency (ms)				
Group A ($n = 16$)	5.3 ± 1.0	4.2 ± 0.6	<0.05	0.9 ± 0.8
Group B ($n = 21$)	4.9 ± 0.9	4.1 ± 1.0	<0.05	0.8 ± 0.7
Sensory distal latency (ms)				
Group A ($n = 16$)	4.8 ± 0.9	3.9 ± 0.4	<0.05	0.6 ± 0.7
Group B ($n = 21$)	4.3 ± 0.6	4.0 ± 0.9	<0.05	0.5 ± 0.5

In patients with symptom duration less than 9 mo, splinting and steroid injection resulted in significant improvement in median nerve motor and sensory distal latencies ($P < 0.05$). The changes in median nerve motor and sensory distal latencies were not significant if the symptoms had lasted more than 9 mo. No complication or side effect was reported.

DISCUSSION

CTS is probably the most common syndrome and diagnosis seen in electrodiagnostic laboratories. In this study, two conservative treatment methods for CTS were compared. Both splinting combined with use of a nonsteroidal antiinflammatory drug and steroid injection into the carpal tunnel resulted in significant improvement as assessed by median nerve motor and sensory distal latencies, VAS scores, Symptom Severity Scale scores, and physical findings.

Beneficial effects of repeated steroid injections were reported in several studies, which date back many years. Phalen and Kendrick[11] and Foster[12] applied three to four injections at weekly intervals, and both noted that 80% of their patients showed definite improvement.

In a randomized, double-blind, placebo-controlled trial, Dammers et al.[10] found that a single injection of steroids close to the carpal tunnel may result in long-term improvement. Girlanda et al.[13] also reported that steroid injection was effective for the treatment of CTS. Maximum improvement of symptoms occurred at 1 mo after the first injection of steroid and concerned mainly nocturnal pain and paresthesia. When evaluated with VAS and Symptom Severity Scale scores, significant improvements were noted by the second week for both treatment groups in our study. The most common electrophysiologic abnormality seen in CTS is the increase in median nerve motor and sensory distal latencies.[14] In our study, steroid injection and splinting for CTS each resulted in a significant decrease of sensory and motor distal latencies. Giannini et al.[15] also found that abnormalities of sensory and motor distal latencies improve after steroid injection into the carpal tunnel. Electrodiagnostic studies were done at baseline and 45 days and 6 mo after treatment. Recovery of the function of the median nerve was found to last after the pharmacologic effect of the steroid ceased. Walker et al.[16] reported that symptoms, functional deficits, and median nerve electrodiagnostic abnormalities improve in patients with CTS who undergo 6 wk of neutral-wrist splinting.

In a prospective study, Gelberman et al.[17] compared steroid injection to splinting for the treatment of CTS. Patients with milder symptoms responded better to conservative treatment. Those with mild symptoms and physical findings were symptom free for longer than 12 mo. They reported recurrence of symptoms in most patients by 9–15 mo after steroid injection for CTS. Patients with severe symptoms of more than 1 yr duration received little long-term relief from injection and splinting.[17] In our study, patients with symptom duration more than 9 mo did not respond well to treatment in either group as assessed by median nerve motor and sensory distal nerve latencies.

In another study, Weiss et al.[18] reported that patients 40 yr of age or younger had a significant decrease in the rate of symptom resolutions when compared to patients over 40 yr of age. No differences were noted between patients having symptoms less than 1 yr and those having symptoms longer than 1 yr with regard to symptom resolution.

In conclusion, both treatments for CTS were found to be effective in our study. There was a significant symptom resolution after 2 wk. Both treatment options were safe and well tolerated by the patients. Because follow-up was limited to 8 wk, we cannot comment on recurrence rates or long-term results. Duration of symptoms may also play a significant role for the determination of treatment response. Those results are derived from an almost exclusively female population and may not apply to the patients with severe CTS because this study excluded patients demonstrating thenar atrophy.

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