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ORIGINAL ARTICLE

Ultrasound-guided insulin injection for carpal tunnel syndrome in type 2 diabetes mellitus patients

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Abstract

Objective To compare effectiveness of ultrasound-guided local insulin injection, local steroid injection, and local steroid followed by insulin injections in treating mild to moderate carpal tunnel syndrome (CTS) in type 2 diabetes mellitus (DM).

Method Study included 60 patients with electrophysiologic evidence of mild to moderate CTS. They were randomly divided into three groups: group I received insulin injection locally into the affected carpal tunnel at first visit and a similar dose after 2 weeks; group II received single injection of 40 mg methylprednisolone acetate injection; and group III received steroid injection then followed by insulin injection twice after 2 and 4 weeks. All injections were performed with ultrasonographic guidance. All patients were assessed by modified Boston Carpal Tunnel Questionnaire (FD score), CTS severity score (SS score), and neurophysiological and ultrasonographic assessments at baseline and 10 weeks after treatment.

Results A significant improvement in mean FD score, SS score, DML (distal motor latency), SNCV (sensory nerve conduction velocity), PSL (peak sensory latency), Samp (sensory amplitude), and CSA (cross-sectional area of median nerve) observed in all groups (with exception of mean DML and Samp in the second group and mean Samp in the third group). Group III showed significant improvement in CSA especially when compared to group II by post hoc analysis ($P = 0.005$).

Conclusions Local insulin injection is as effective as steroid in treating mild to moderate CTS in type 2 DM and is a safer alternative. Adding insulin injections after steroid shows more sonographic improvement than steroid alone.

Key Points

- *Local insulin injection is as effective as steroid in treating mild to moderate CTS in type 2 diabetic patients.*
- *Measuring CSA of median nerve at CT inlet by US is a better tool for monitoring median nerve changes after treatment.*
- *Adding insulin injections after steroid has more sonographic improvement than steroid alone.*

Keywords Carpal tunnel syndrome · Local insulin injection · Type 2 diabetes mellitus · Ultrasound guided

Introduction

Carpal tunnel syndrome (CTS) is the most well-known and frequent variety of median nerve neuropathy [1]. It influences 4–5% of general population particularly between the ages of 40–60 years. The prevalence rate is higher among females (9.2%) than among males (6%) between the ages of 45–60 years [2]. In diabetic patients, the rate of CTS has been reported to be as much as 15–33% [3].

The most reliable method to confirm clinical diagnosis of CTS is electrophysiologic testing [4]. Ultrasound (US) is being increasingly used as a primary imaging study to assist in the diagnosis of various neuromuscular diseases and entrapment neuropathies including CTS [5–7]. In comparison with nerve conduction study (NCS), ultrasonography has many

To our knowledge, this is the first study that used ultrasonography in the evaluation of the median nerve after insulin injection, via measuring CSA at the inlet of CT.

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advantages like lower cost, shorter examination time, and possibility of sonography-guided injection [4, 6].

Ultrasound measurement of the cross-sectional area (CSA) of the median nerve at the level of the carpal tunnel inlet was found to be an accurate and cost-effective confirmatory test for a clinical diagnosis of CTS [8]. One of the novel techniques is the use of doppler US to assess the intraneural vascularity of the median nerve for detecting the inflammatory process that precedes demyelination [9].

The pathophysiology of diabetic-induced neuropathies reaches far beyond the neurotoxic effects of hyperglycemia alone. Cellular changes related to diabetes mellitus (DM) will harm the nerve in numerous ways, including vascular changes, membrane damage by reactive metabolic intermediates, organelle damage, and reduced axonal transport [10]. Moreover, DM causes enlargement of the peripheral nerves, in a similar manner to Charcot–Marie–Tooth disease and chronic inflammatory demyelinating polyneuropathy [11]. Results of Chen et al. [12] showed that, the median nerve CSAs were significantly larger in DM patients with preexisting CTS than those with only DM.

Synthesis of growth factors particularly nerve growth factors and insulin-like growth factor-1 are reduced in target cells of diabetic sympathetic sensory and motor neurons [13] and axonal transport impairments, which are thought to occur in DM, will also limit delivery of growth factors to the cell body, further adding to axonal degeneration and limiting the regenerative capacity [14].

Conservative, non-operative therapy has been urged as being appropriate, at least initially, for most patients with mild to moderate CTS [15]. Established therapy has usually been decompression of the nerve, like splinting and steroid injection into the carpal tunnel [16]. However, corticosteroids create a theoretical risk to patients with DM. Evidence exists that locally administered corticosteroids could have a systemic effect, which, in patient with DM, could lead to elevated blood glucose levels [17].

It has been suggested that insulin has an effect on nerve regeneration like that of nerve growth factor. Local insulin treatment may be of incredible potential advantage in the improvement of nerve functions in type 2 DM patients with mild to moderate CTS [18]. Our study aimed to compare the effectiveness of ultrasound-guided local insulin injection, local steroid injection, and local steroid followed by insulin injection in treating mild to moderate CTS in patients with type 2 DM.

Patients and methods

Patients

Seventy patients with type 2 DM (according to The American Diabetes Association criteria) [19] and complaining of clinical symptoms and signs of CTS were enrolled to the study. The clinical diagnostic criteria of CTS were based on the American Academy of Neurology [20]. To confirm the clinical diagnosis of CTS, electrophysiological assessment was done. For severity grading, both electrophysiological and ultrasonographic assessments were done. One hand was selected from each patient; in patients with bilateral involvement, the more severe side was selected, or the dominant side was selected if of equal severity. Informed consent was taken from all participants in the study. The study was approved by the ethics committee of the faculty of medicine.

Diabetic patients with mild or moderate CTS "Grades 1, 2 and 3 electrophysiologically" [21], whom plasma glycosylated hemoglobin (Hb_{A1c}) level $\leq 8\%$, were included. All patients were under treatment with oral hypoglycemic medications. Excluded from the study are patients with severe CTS either detected by electrophysiologic study [21] or neuromuscular US [22]; patients with previous carpal tunnel surgery or previous course of CTS therapy; patients with CTS due to underlying systemic causes other than type 2 DM; and patients with various other disorders resembling CTS such as cervical radiculopathy, brachial plexopathy, pronator teres syndrome, and polyneuropathy.

Clinical examinations

The patients underwent a full clinical examination, including Tinel's, Phalen's test, and reverse Phalen's test. Modified Boston Carpal Tunnel Questionnaire (FD score) was used for the evaluation of the functional disability [23]; and CTS severity score (SS score) was used for the evaluation of the symptoms severity [24].

Electrophysiological assessment

The electromyographic apparatus used was EMG/NCS unit (Nihon Kohden MB9300). Electrophysiological assessment for the patients was performed using standard techniques of supra-maximal cutaneous stimulation and surface electrode recording [25]. Based on report of the American Association of Electrodiagnostic Medicine, American Academy of Neurology, and the American Academy of Physical Medicine and Rehabilitation [26], the electrophysiological evidence of slowing of distal median nerve conduction includes prolongation of peak sensory latency (PSL) and/or distal motor latency (DML) of median nerve action potential and delayed sensory nerve conduction velocity (SNCV) ± reduced sensory amplitude (Samp) of the median nerve.

Neuromuscular ultrasound assessment

All US scans were performed using Siemens ACUSON P300 Ultrasound System (Siemens Healthcare, Boulevard, and

Malvern, USA) with multi-frequency 10–18 MHz linear transducer. A dedicated protocol with optimization of scanning parameters (depth, focal zone, frequency, and color Doppler settings for low-flow vessels) had been preprogrammed for the purpose of this study to ensure consistency of results obtained. The carpal tunnel was scanned to identify the carpal tunnel contents as well as structural abnormalities and anatomic variations that may affect the procedure. The full course of the median nerve in the carpal tunnel was evaluated in both transverse and longitudinal scans. The CSA of the median nerve was calculated automatically by tracing the inner margin of the epineurium of median nerve [27]. The CSA of the median nerve was measured at the tunnel inlet [28] (Fig. 1a). The blood flow in the median nerve sheath was then detected 2 cm proximal to the carpal tunnel using power doppler US [29].

Study design and treatment

A single-blind randomized controlled trial was carried out. The included patients were block randomized in a 1:1 ratio into three groups via computer based "random number generators" using the GraphPad QuickCalcs Website: https://www.graphpad.com/quickcalcs/randomize1/Group I received 10 IU of Neutral Protamine Hagedorn insulin (NPH insulin) locally at baseline; 2 weeks later, group II received 40 mg of methylprednisolone locally, serving as a (positive control group), while group III received 40 mg of methylprednisolone locally at first visit followed by 10 IU of NPH insulin after 2 and 4 weeks from the baseline. All injections were performed with US guidance using the ulnar approach (Fig. 1b) described by Smith et al. [30]. Additional treatments such as splint, oral steroids, diuretics, and surgery were not allowed during the study. All patients were assessed at baseline and 10 weeks later.

Statistical analysis Data were analyzed by SPSS 19.0. Descriptive statistics were done by number and percent as well as mean and SD. The paired samples *t* test was used to compare the difference between two group means (patients at base line and at follow-up) in interval and ordinal variables. The Pearson's χ^2 test was used to compare qualitative variables. The McNemar's χ^2 test assesses the difference between paired proportions. Analysis of variance (ANOVA test) was

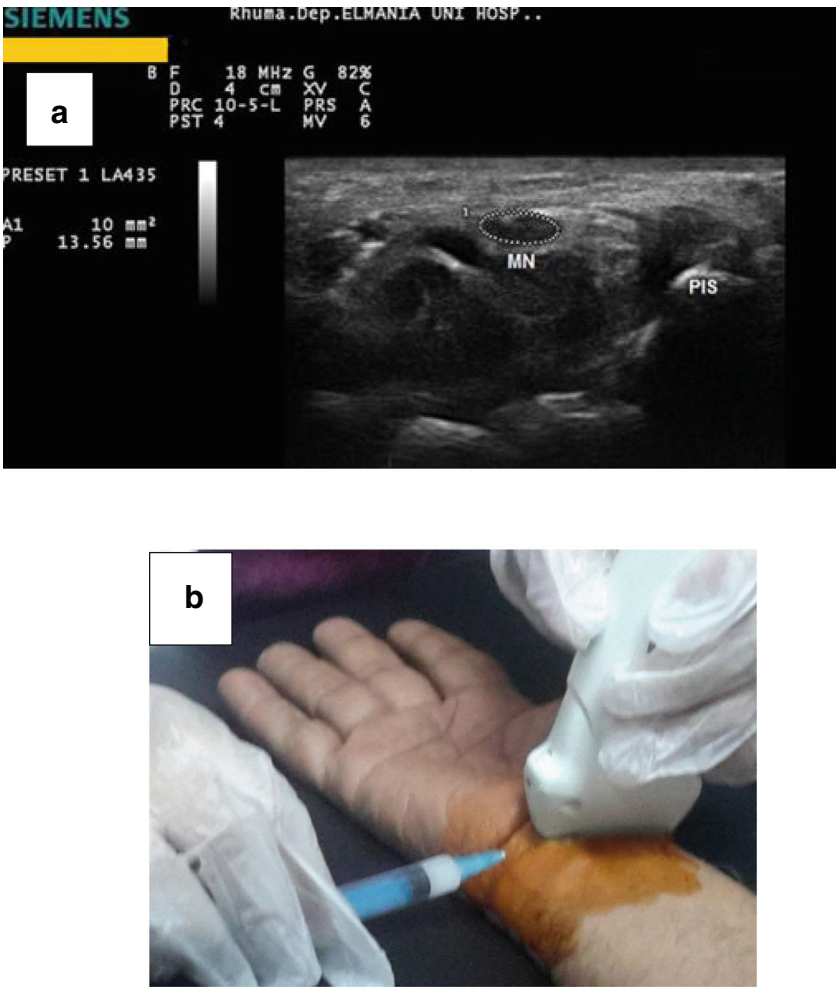

Fig. 1 **a** Transverse US image of median nerve (outlined) at carpal tunnel inlet; the CSA is 10 mm^2. MN, median nerve; PIS, pisiform bone. **b** Transducer position and needle approach for an ulnar-sided carpal tunnel injection

used to compare the difference between more than two groups as regards quantitative variables. When there is a statistically significant difference, post hoc test by Tukey's HSD (honest significant difference) method was used to confirm where the differences occurred between groups. The level of statistical significance was set at a $P < 0.05$.

Results

Ten of seventy patients were subsequently excluded from the study (3 patients with severe CTS, 1 pregnant patient, 2 patients had thyroid disease, and 4 patients received previous treatment). Consecutively, 60 patients (60 hands) completed the course of the study. Forty patients (66.7%) have bilateral CTS and 20 patients (33.3%) have unilateral CTS. By electrophysiological assessment, 15 patients (25%) had mild CTS and 45 patients (75%) had moderate CTS. By ultrasonographic assessment, four patients (6.7%) had normal CSA (less than 10mm^2 but had mild CTS with NCS), 24 patients (40%) had mild CTS (CSA 10.0–12.9 mm^2) and 32 patients (53.3%) had moderate CTS (CSA 13.0–15.0 mm^2).

The characteristics of the three groups of patients are shown in Table 1 with no significant differences between them.

Tables 2, 3, and 4 showed baseline and follow-up characteristics of the three groups. There were no major adverse effects caused by the medication in any group.

After 10 weeks, by comparing the improvement in clinical and electrophysiologic assessment between the three groups, there were no statistically significant differences. While the change in the level of Hb_{A1c} and CSA of median nerve after 10 weeks from the baseline showed a statistically significant differences ($P = 0.002$ for each one).

Post hoc comparisons using Tukey's HSD test was done for the significant different variables as shown in Table 5, there was a statistically significant difference in Hb_{A1c} level between group I and group II ($P = 0.001$); and there was a statistically significant difference in CSA between group II and group III ($P = 0.005$).

Discussion

Local insulin injection in CTS has anti-inflammatory and anti-edematous effects [31]. Ultrasound-guided injections result in better symptom relief and increased therapeutic duration compared to non-ultrasound-guided injections [32].

The present study evaluates the effectiveness of US-guided local insulin injection, compared to local steroid injection, and to local steroid followed by local insulin injections in the treatment of mild to moderate CTS in patients with type 2 DM.

In the literature, there were few studies that used insulin injections in treating CTS, both diabetic and idiopathic, with different doses and intervals [18, 31, 33–35].

Sixty adult patients with mild to moderate CTS were included in the present study; they were randomized into three groups equally. All injections were performed with ultrasonographic guidance.

There were no significant differences in mean of age of patients, duration of DM, duration of CTS, BMI, clinical severity, NCS and US assessment, and glycosylated hemoglobin between patients in the three groups. This indicates that these parameters were not a confounding factor in the present study.

Follow-up of three groups of patients showed that there was a statistically significant reduction in Phalen's test, Tinel's sign, and reversed Phalen's test, and there was a statistically highly significant reduction in the mean values of SS score and FD score. However, when comparing the difference of improvement between the three groups, there was no statistically significant difference.

Thus, insulin was as effective as steroid in improving CTS clinically in type 2 DM patients, with no additional benefit observed from further insulin injection after the steroid injection after 10-week duration. This was in accordance with Ozkul et al. [18] and Ibrahim and Hussein [34].

Comparing NCS before and after treatment in the insulin group, we found a statistically highly significant improvement in DML, SNCV, and PSL ($P < 0.001$ for each) and a statistically significant improvement in Samp ($P < 0.05$); while in the steroid group, there was a statistically highly significant improvement in PSL ($P < 0.001$) and a statistically significant improvement in SNCV ($P < 0.05$). In the third group (steroid followed by insulin), there was a statistically highly significant improvement in DML, SNCV, and PSL ($P < 0.001$ for each), while no statistically significant difference was found in Samp ($P \geq 0.05$).

However, when we compared the NCS improvement between the three groups, there was no statistically significant difference between them. This is similar to Abu-Zaid et al. [35] who found significant improvement in electrophysiological parameters such as DML and SNCV of median nerve 2 and 4 months after treatment ($P < 0.01$), but the difference between results in both groups were statistically non-significant. Also, Ibrahim and Hussein [34] found a statistically significant improvement as regards mean value of DML and DSL of median nerve 1 month after the treatment in both steroid and insulin injection groups ($P < 0.01$), with no significant difference between them.

We used US for staging of CTS (CSA of median nerve at the inlet) [22], detection of hypervascularity [29], and guidance of the local injection [30]. As regards severity of CTS, there were 24 patients (40%) who had mild CTS and 32 patients (53.3%) had moderate CTS. However, US did not show increased CSA in 4 patients (6.7%) (CSA less than 10 mm^2),

Table 1 Characteristics of the patients

	Group I ($n = 20$)	Group II ($n = 20$)	Group III ($n = 20$)
Age (years)	24–72 (40.7 ± 13.5)	22–70 (44.7 ± 10.2)	20–58 (38.3 ± 10.4)
Sex, male/female	3/17	3/17	2/18
BMI (Kg/m^2)	21.1–40.2 (28.5 ± 6.4)	21.4–35.2 (28 ± 4.3)	20–33.9 (27.6 ± 4.5)
Duration of DM (years)	1–15 (4.3 ± 3.1)	1–11 (2.9 ± 2.5)	1–20 (4.3 ± 4.6)
Duration of CTS (months)	1–36 (8.5 ± 7.6)	1–24 (8.1 ± 6.7)	2–48 (7.5 ± 10.1)
FD score	2.5–4.5 (3.61 ± 0.58)	1.75 ± 4.38 (3.18 ± 0.68)	2.38–4.5 (3.61 ± 0.53)
SS score	4.45–8.18 (6.41 ± 1.02)	4.45–8.82 (6.23 ± 1.36)	3.54–8.36 (6.82 ± 1.2)
Hb_{A1c} (%)	6.1–7.9 (7.09 ± 0.67)	6.5–7.9 (7.1 ± 0.4)	6–7.9 (7.2 ± 1.5)
NCS			
DML (ms)	3.75–6.2 (4.7 ± 0.7)	3.7–7.6 (5 ± 0.8)	3.9–5.8 (5 ± 0.6)
SNCV (m/s)	20.2–46.9 (37.3 ± 6.5)	16.6–47.8 (37 ± 6.7)	26.8–47 (36.5 ± 5.3)
PSL (ms)	2.5–6.3 (4.1 ± 0.7)	2.5–7.8 (4.29 ± 1.06)	2.5–5.5 (4.2 ± 0.75)
Samp (mV)	13.1–49 (20.3 ± 10.8)	10–38.7 (17.3 ± 6.6)	8–56 (26.4 ± 12.4)
MSUS			
CSA (mm^2)	9–15 (12.4 ± 1.8)	8–14 (11.7 ± 1.8)	10–15 (12.9 ± 1.3)
+ve PD	8 (40%)	6 (30%)	13 (65%)

Values are labeled as range (mean ± SD) or *n* (%)

BMI, body mass index; *DM*, diabetes mellitus; *CTS*, carpal tunnel syndrome; *FD score*, functional disability score; *SS score*, symptom severity score; *Hb_{A1c}*, glycated hemoglobin; *NCS*, nerve conduction studies; *MSUS*, musculoskeletal ultrasonography; *DML*, distal motor latency; *SNCV*, sensory nerve conduction velocity; *PSL*, peak sensory latency; *Samp*, sensory amplitude; *CSA*, cross-sectional area; *PD*, power doppler

No statistically significant differences between-groups ($P > 0.05$)

and this could be explained by when the median nerve is analyzed at a single location only (i.e., the tunnel inlet); occasionally, the nerve caliber may be within normal limits at the inlet and yet unduly swollen proximal to the inlet or at the outlet of the carpal tunnel [36], that agreed with Rao et al. [37] who found that US did not show increased CSA in 6 out of 30 (20%) neurophysiologically diagnosed CTS patients.

To our knowledge, no other studies have used US in evaluating median nerve after insulin injection. The median nerve was evaluated via measuring CSA at the inlet of CT, and with

Table 2 Baseline and follow-up characteristics of group I (insulin group)

		Before injection	After injection	*P* value
+ve Tinel		12 (60%)	4 (20%)	0.01*
+ve Phalen		14 (70%)	2 (10%)	0.002*
+ve reversed Phalen		13 (65%)	4 (20%)	0.008*
SS score		4.45–8.18 (6.41 ± 1.02)	1.18–4.5 (2.55 ± 0.86)	< 0.0001*
FD score		2.5–4.5 (3.61 ± 0.58)	1.13–3 (1.79 ± 0.5)	< 0.0001*
Hb_{A1c} (%)		6.1–7.9 (7.09 ± 0.67)	6–7.8 (7.06 ± 0.63)	0.249
NCS	DML (ms)	3.75–6.2 (4.69 ± 0.71)	3.66–5.7 (4.5 ± 0.66)	< 0.0001*
	SNCV (m/s)	20.2–46.9 (37.25 ± 6.46)	33.5–50.2 (44.27 ± 5.56)	< 0.0001*
	PSL (ms)	2.5–6.3 (4.05 ± 0.71)	2.92–5.1 (3.68 ± 0.45)	< 0.0001*
	Samp (mV)	13.1–49 (20.32 ± 10.82)	14–50 (24.84 ± 9.28)	0.006*
MSUS	CSA (mm^2)	9–15 (12.4 ± 1.79)	8–14 (11.05 ± 1.82)	< 0.0001*
	+ve PD	8 (40%)	0 (0%)	0.01*

Values are labeled as range (mean ± SD) or n (%)

SS score, symptom severity score; *FD score*, functional disability score; *Hb_{A1c}*, glycated hemoglobin; *NCS*, nerve conduction studies; *MSUS*, musculoskeletal ultrasonography; *DML*, distal motor latency; *SNCV*, sensory nerve conduction velocity; *PSL*, peak sensory latency; *Samp*, sensory amplitude; *CSA*, cross-sectional area; *PD*, power doppler

*Statistically significant ($P < 0.05$)

Table 3 Baseline and follow-up characteristics of group II (steroid group)

		Before injection	After injection	*P* value
+ve Tinel		17 (85%)	8 (40%)	0.003*
+ve Phalen		13 (65%)	3 (15%)	0.004*
+ve reversed Phalen		12 (60%)	2 (10%)	0.004*
SS score		4.45–8.82 (6.23 ± 1.36)	1.5–4 (2.76 ± 0.61)	< 0.0001*
FD score		1.75–4.38 (3.18 ± 0.68)	1–2.75 (1.83 ± 0.53)	< 0.0001*
Hb_{A1c} (%)		6.5–7.9 (7.1 ± 0.44)	6.6–8.3 (7.3 ± 0.47)	0.0009*
NCS	DML (ms)	3.7–7.6 (4.97 ± 0.8)	3.7–7.7 (4.8 ± 0.93)	0.2
	SNCV (m/s)	16.6–47.8 (37 ± 6.69)	19–62 (41.06 ± 9.16)	0.005*
	PSL (ms)	2.5–7.8 (4.29 ± 1.06)	2.68–7.6 (3.89 ± 1.06)	< 0.0001*
	Samp (mV)	10–38.7 (17.34 ± 6.56)	11.4–44 (21.16 ± 9.55)	0.07
MSUS	CSA (mm^2)	8–14 (11.7 ± 1.81)	8–13 (10.9 ± 1.71)	< 0.0001*
	+ve PD	6 (30%)	0 (0%)	0.04*

Values are labeled as range (mean ± SD) or *n* (%)

SS score, symptom severity score; *FD score*, functional disability score; *Hb_{A1c}*, glycated hemoglobin; *NCS*, nerve conduction studies; *MSUS*, musculoskeletal ultrasonography; *DML*, distal motor latency; *SNCV*, sensory nerve conduction velocity; *PSL*, peak sensory latency; *Samp*, sensory amplitude; *CSA*, cross-sectional area; P*D*, power doppler

*Statistically significant ($P < 0.05$)

the presence of hypervascularity with power doppler, there was a statistically highly significant improvement in CSA in all groups ($P < 0.001$); also, there were not any active doppler signals observed at the follow-up in all patients. Group III showed the most improvement in CSA especially when compared to steroid group by post hoc analysis; this indicates that addition of insulin to steroid reduces the nerve edema. This may be due to the additional anti-edematous effect of insulin as postulated by Abu-Zaid et al. [31].

In the follow-up measurements of Hb_{A1c} level, we found that, only the steroid group had statistically highly significant difference in the Hb_{A1c} level; the mean Hb_{A1c} had risen from 7.1 ± 0.44 to 7.3 ± 0.47%, while the other two groups had no statistically significant difference at follow-up measurements. When comparing the Hb_{A1c} level difference between the three groups, there is statistically significant difference between the insulin and the steroid groups, yet when comparing both with the third group, there was no statistically significant difference

Table 4 Baseline and follow-up characteristics of group III (steroid and insulin group)

		Before injection	After injection	*P* value
Tinel		11 (55%)	4 (20%)	0.02*
Phalen		17 (85%)	0 (0%)	0.0001*
Reversed Phalen		14 (70%)	1 (5%)	0.0009*
SS score		3.54–8.36 (6.82 ± 1.2)	1.32–4.3 (2.93 ± 0.9)	< 0.0001*
FD score		2.38–4.5 (3.61 ± 0.5)	1.25–3 (1.94 ± 0.5)	< 0.0001*
Hb_{A1c} (%)		6–7.9 (7.18 ± 1.5)	6–8 (7.26 ± 0.62)	0.159
NCS	DML (ms)	3.9–5.8 (5 ± 0.55)	3.7–5.4 (4.6 ± 0.5)	< 0.0001*
	SNCV (m/s)	26.8–47 (36.5 ± 5.3)	33.5–50.8 (41.6 ± 5.4)	< 0.0001*
	PSL (ms)	2.5–5.5 (4.2 ± 0.75)	2.9–4.6 (3.8 ± 0.4)	0.001*
	Samp (mV)	8–56 (26.4 ± 12.4)	15.3–56 (27.4 ± 11.2)	0.4
MSUS	CSA (mm^2)	10–15 (12.9 ± 1.3)	9–14 (11.2 ± 1.8)	< 0.0001*
	+ve PD	13 (65%)	0 (0%)	0.0009*

Values are labeled as range (mean ± SD) or *n* (%)

SS score, symptom severity score; *FD score*, functional disability score; *Hb_{A1c}*, glycated hemoglobin; *NCS*, nerve conduction studies; *MSUS*, musculoskeletal ultrasonography; *DML*, distal motor latency; *SNCV*, sensory nerve conduction velocity; *PSL*, peak sensory latency; *Samp*, sensory amplitude; *CSA*, cross-sectional area, *PD*, power doppler

*Statistically significant ($P < 0.05$)

Table 5 Comparison of the significantly different variables between the groups

	Compared groups		*P* value
Hb_{A1c}difference	Group I	Group II	0.001*
	Group I	Group III	0.07
	Group II	Group III	0.07
CSA improvement	Group I	Group II	0.06
	Group I	Group III	0.31
	Group II	Group III	0.005*

Hb_{A1c}, glycated hemoglobin; *CSA*, cross-sectional area
*Statistically significant ($P < 0.05$)

between them. Insulin seems to affect the blood glucose the least in contrary to steroid.

There was no difference between groups as regards the presence of power doppler signals; all the patients who had positive signals improved after receiving the injections. This was in accordance with El Miedany et al. [38] who found improvement in doppler signals in median nerve after local steroid injection within 1 week. They concluded that measuring CSA at CT inlet was found to be a better tool for monitoring median nerve changes after treatment than nerve conduction testing.

We did not register our study in any public data base which can be considered as a limitation.

In conclusion, local insulin injection is as effective as steroid in treating mild to moderate CTS in type 2 diabetic patients and is a safer alternative. Moreover, adding insulin injections after steroid shows more sonographic improvement than steroid alone. Further studies are needed including larger number of patients, longer duration for treatment and follow-up, and different doses of insulin. More researches are suggested to assess the possibility of using insulin in treatment of other diabetic nerve diseases and other entrapment neuropathies.

Compliance with ethical standards

Disclosures None.

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