

The carpal tunnel syndrome in diabetes: clinical and electrophysiological improvement after treatment with Palmitoylethanolamide.

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INTRODUCTION

Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy. Surgical and non-surgical treatments can induce excellent outcomes for patients. In general, the pharmacological treatments of CTS include non-steroidal anti-inflammatory drugs, corticosteroids administered either orally or through local injections. Surgery is needed for symptomatic and non responder. Thus, the need of having innovative therapeutic molecules, devoid of side effects and well tolerated remains still high. The most interesting molecules are those able to modulate the neuroimmune interactions. Among these, Palmitoylethanolamide (PEA) belonging to the family of N-acylethanolamines, a class of naturally occurring fatty acid amides, like endocannabinoids, is produced on demand within the lipid bilayer of cellular membranes and its signal is rapidly

terminated. When exogenously administered, PEA has been reported to exert anti-inflammatory and analgesic effects as well as neuroprotection. In particular, PEA is able to reduce pain intensity and functional disability in patients suffering from painful lombosciatalgia and to promote a reduction of anti-inflammatory and analgesic drugs in cases of neuropathic pain. Recent data have also shown the improvement induced by PEA on electrofisiological parameters such as the amplitudes of the action potentials measured by laser-evoked potentials in patients with peripheral neuropathy. Objective: to investigate the clinical and electrophysiological effects of conservative treatment with PEA in diabetic patients affected by the carpal tunnel syndrome.

PATIENTS AND METHODS

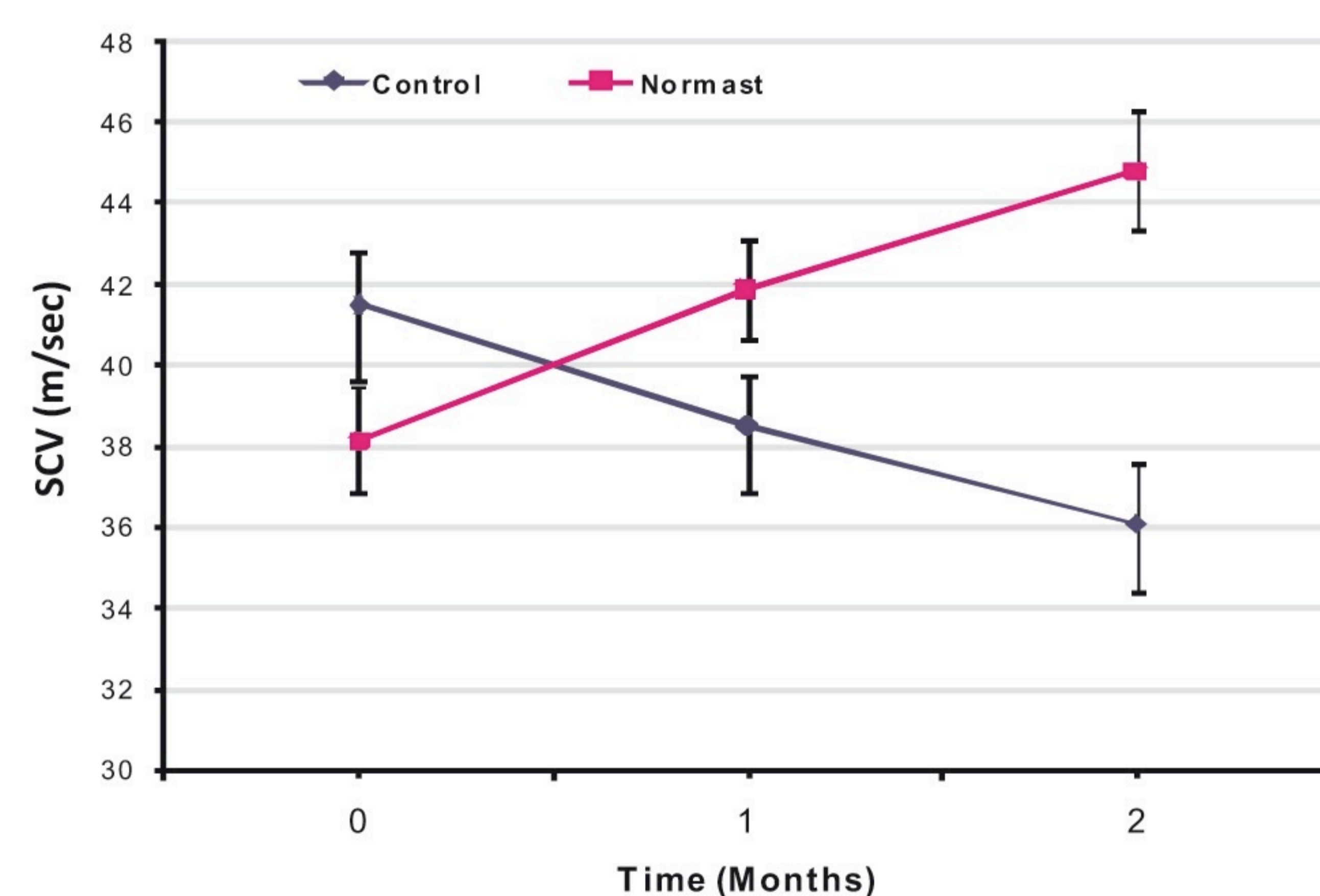
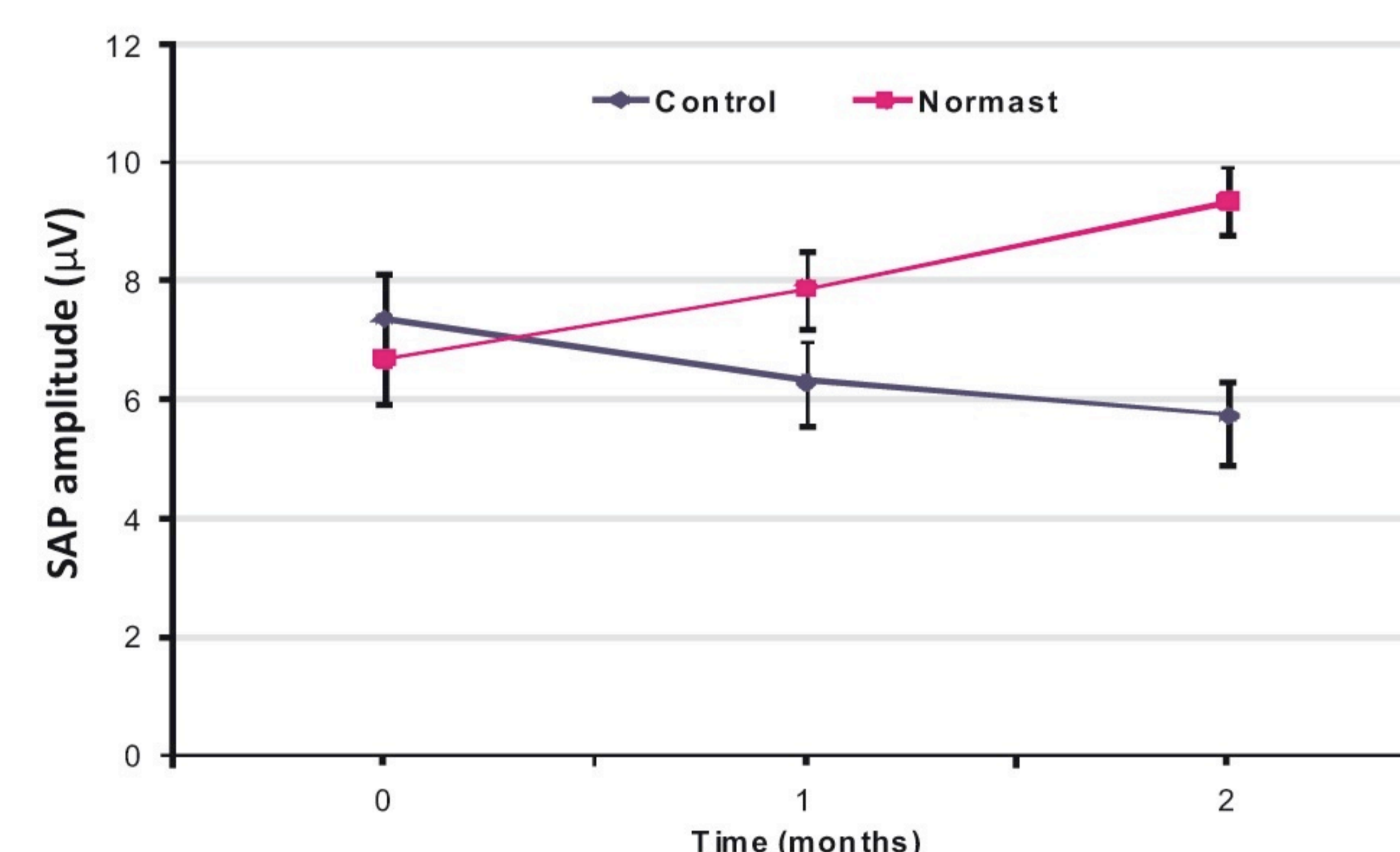
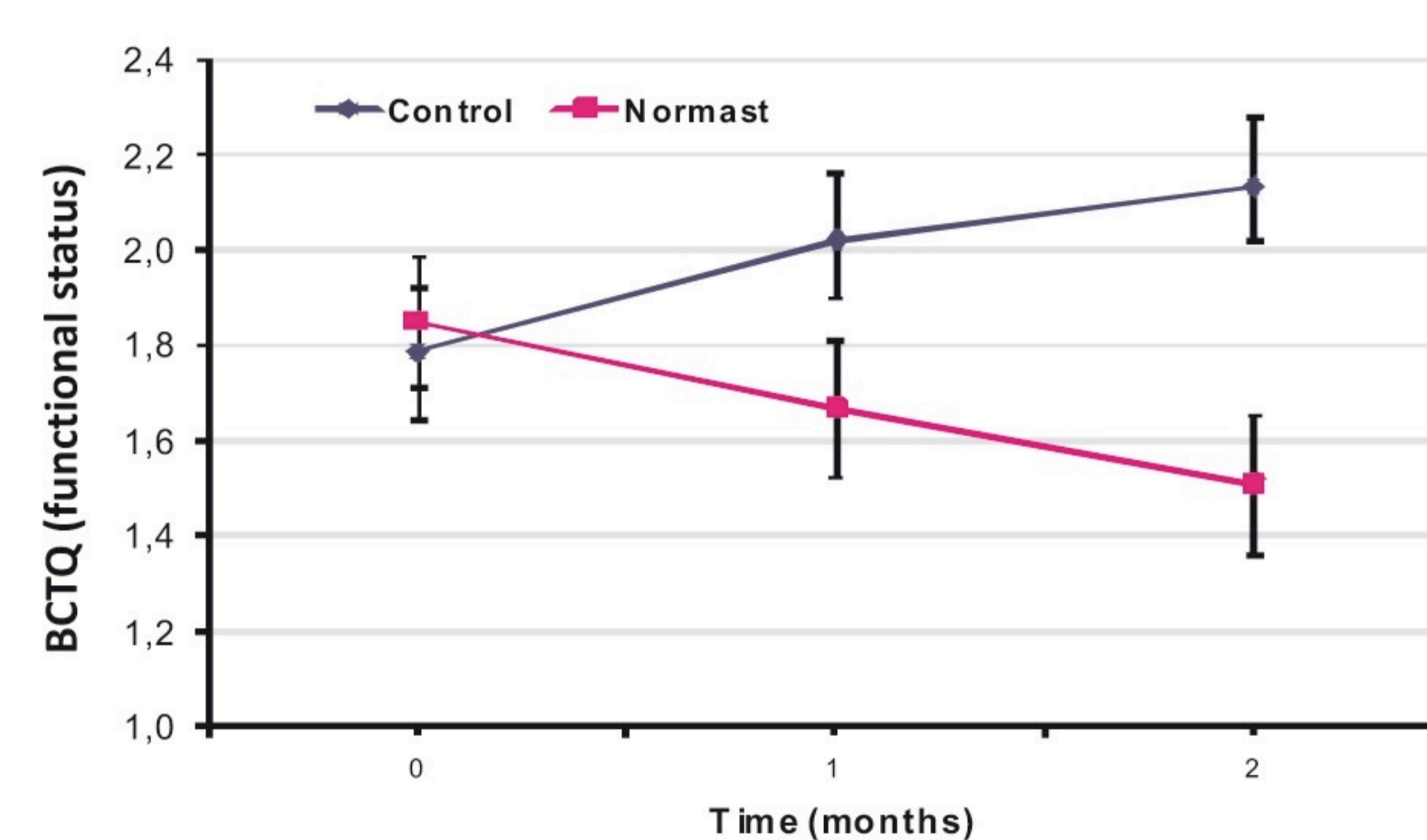
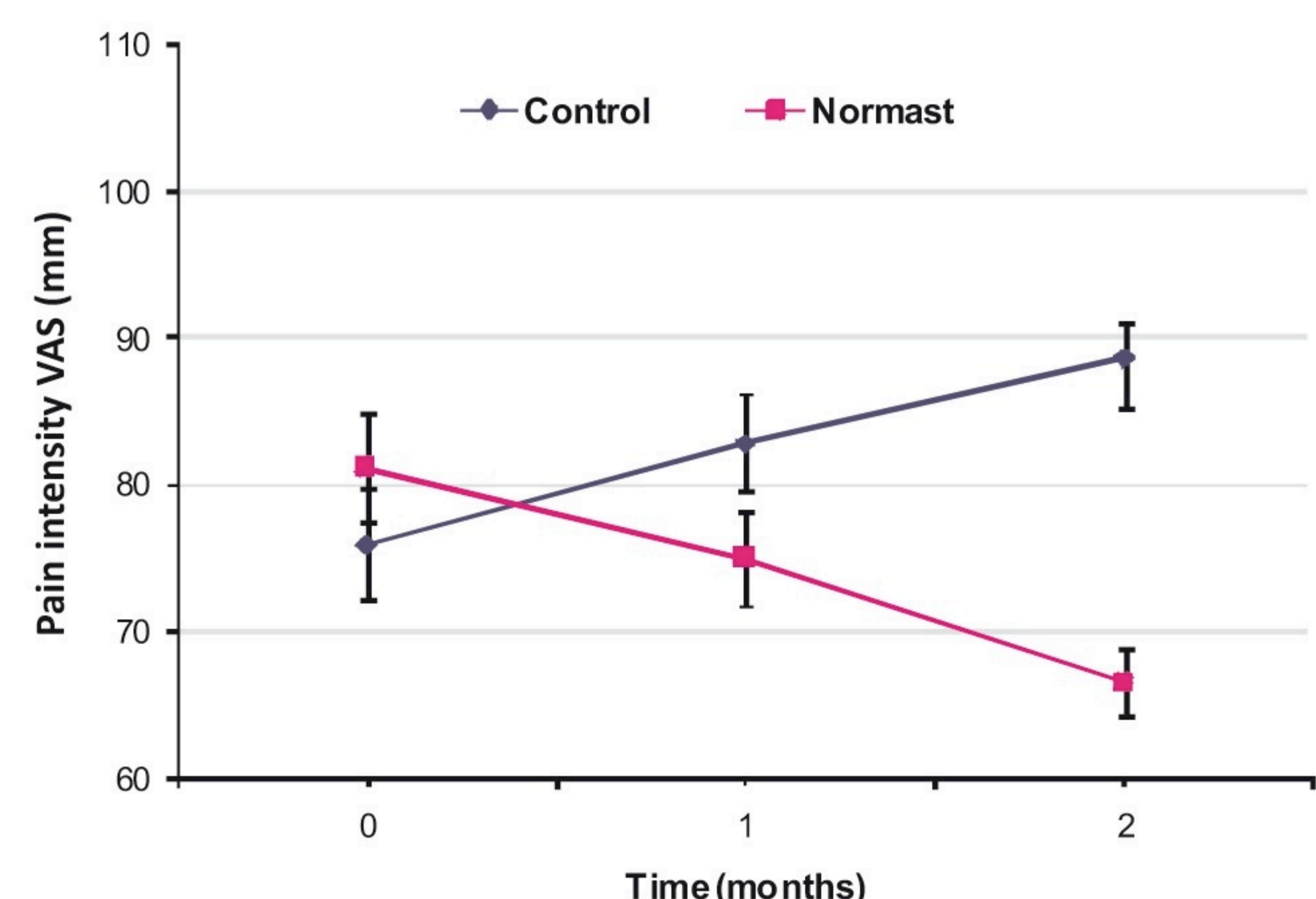
50 diabetic patients, of both sexes, having a clinical and electrophysiologically confirmed diagnosis of CTS, were considered; fourteen have a bilateral CTS; only the patients with mild/moderate CTS (degree ≤ 3) were considered. Patients were randomly assigned to PEA (n=25) or Control (n=25) group. PEA was administered twice a day (2x 2 tablets containing 300mg; total/day = 1200 mg) for two months. Controls did not received any treatment during the study period. Clinical and electrophysiological examinations were performed at baseline (T0), after 1 month (T1) and at the end of the treatment (T2). The outcome was evaluated using either clinical parameters, such as pain intensity by means of Visual Analogic Scale (VAS) and symptoms and functional status by means of "Boston Carpal Tunnel Questionnaire", either quantitative electrophysiological parameters, such as sensory conduction velocity (SCV), sensory nerve action potential amplitude (SAP) and nerve distal motor latency (DML) of the median nerve.

RESULTS

PEA induced a significant clinical and electrophysiological improvement in the analyzed patients; the effects were already evident on T1; however a more consistent effects were observed on T2. In fact, pain intensity mean scores were 81.1 ± 3.74 and 66.6 ± 3.36 at T1 and T2 respectively in PEA treated group while in the control group they were 75.9 ± 3.74 and 88.7 ± 2.33 at T1 and T2, respectively ($p \leq 0.0001$ at ANOVA analysis). In the "Boston Carpal Tunnel" questionnaire (part 2 functional status) the mean values were 1.9 ± 0.14 and 1.5 ± 0.11 , at T0 and T2, respectively, whereas they were 1.8 ± 0.14 and 2.1 ± 0.15 at T1 and T2 were control group ($p \leq 0.0001$).

A significant improvement induced by PEA treatment was also found by evaluating electrophysiological parameters, specifically in the sensory conduction velocity (SCV), that had a mean value of 38.1 ± 7.07 at T0 and 44.8 ± 6.52 at T2 in PEA treated group whereas in control group the mean value was 41.5 ± 4.65 at T0 and 36.1 ± 5.38 at T2 ($p \leq 0.001$); also the SAP amplitude in treated group significantly increased, in fact the mean value was 6.7 ± 0.76 at T0 and 9.4 ± 0.85 at T2, and 7.4 ± 0.76 at T0 and 5.8 ± 0.58 at T2 in control group ($p \leq 0.0001$).

	T0	T2	p
VAS (mm)	81.1 ± 3.74	66.6 ± 3.36	≤ 0.0001
BCTQ (part 2)	1.9 ± 0.14	1.5 ± 0.11	≤ 0.0001
SCV (m/sec)	38.1 ± 7.07	44.8 ± 6.52	≤ 0.0001
SAP Amplitude (μV)	6.7 ± 0.76	9.4 ± 0.85	≤ 0.0001



CONCLUSION

The present study suggests that treatment of carpal tunnel syndrome in diabetic subjects with PEA at dosage of 1200 mg/die allows an excellent improvement of clinical and electrophysiological parameters and confirms that it could be recommended as alternative to other conservative treatments for patients with mild/moderate degree of CTS.

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