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ARTIGO ORIGINAL

Evaluation of the neurological involvement and prevalence of the carpal tunnel syndrome in patients with type-2 diabetes mellitus

Avaliação do comprometimento neurológico e da prevalência da síndrome do túnel do carpo em pacientes portadores de diabetes mellitus tipo 2

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RESUMO

Objetivo: Determinar a freqüência da síndrome do túnel do carpo (STC) em pacientes diabéticos tipo 2, verificar se está associada com a neuropatia diabética (ND) e identificar formas de evidenciar ambas com o exame dos membros superiores. Método: Os pacientes foram submetidos à anamnese, levantamento das queixas, avaliação da sensibilidade tátil e vibratória, estudo da condução nervosa sensitiva e motora (ECSM) e teste de Phalen (TPH). Considerou-se como critério diagnóstico de STC isolada: presença de alterações no ECSM, queixas de parestesias na área do nervo mediano e ausência de alterações sensitivas ou motoras na área do nervo ulnar e nas extremidades inferiores. Resultados: Entre os 94 pacientes estudados, 60 apresentaram parestesias. O ECSM detectou alteração em 88 pacientes e foi o que apresentou maior sensibilidade. No teste de discriminação de dois pontos estáticos (D2PE) observou-se alteração em 47 pacientes e, com os monofilamentos de Semmes-Weinstein, em 11. Com o bioestesiômetro, detectou-se alteração em 72 pacientes e, com o diapasão, em 4. A positividade do TPH ocorreu em 33 pacientes. Na correlação dos resultados observou-se que 92/94 pacientes apresentaram alteração nervosa, 11 no nervo mediano e 81 combinada nos nervos mediano e ulnar. Somente quatro apresentaram STC sem neuropatia subjacente. Conclusão: Os instrumentos mais sensíveis foram o bioestesiômetro e o D2PE. O exame neurofisiológico demonstrou a presença de neuropatia subjacente à STC. Apresentaram critérios clínicos e neurofisiológico para STC 31,91% dos pacientes: 27,66% com sinais de neuropatia subjacente e 4,25% sem neuropatia diabética. Os critérios clínicos devem ser considerados com preponderância sobre os demais testes e o neurofisiológico para se caracterizar a síndrome do carpo no paciente diabético.

PALAVRAS-CHAVE

diabetes mellitus tipo 2, síndrome do túnel carpal, neuropatias diabéticas, condução nervosa

ABSTRACT

Objective: to determine the frequency of the carpal tunnel syndrome (CTS) in patients with type 2 diabetes mellitus, verify whether it is associated with diabetic neuropathy (DN) and identify ways to recognize both by examining the upper limbs. Methods: The patients were submitted to anamnesis; the complaints were verified, tactile and vibratory sensitivity was evaluated, sensitive and motor nerve conduction (SMNC) was studied and Phalen's test (PHT) was performed. The criteria for isolated CTS diagnosis were: alterations in the SMNC, complaints of paresthesia in the median nerve area and absence of either sensitive or motor alterations in the area of the ulnar nerve and lower extremities. Results: Of the 94 patients studied, 60 presented paresthesia. The SMNC study detected alterations in 88 patients (93.6%) and it was the most sensitive test. At the test to discriminate the two most static points (D2SP) we observed alterations in 47 patients and with the Semmes-Weinstein test detected alterations in 11 patients. Alterations were detected in 72 patients (76.6%) using the bioesthesiometer and in 04 patients using the diapason. PHT was positive in 33 patients. The correlation of the results showed that neural alterations were present in 92/94 patients; 11 patients presented alterations only in the median nerve and 81 patients presented combined alterations in the ulnar and median nerves. Only 4 patients presented CTS without subjacent neuropathy. Conclusions: The most sensitive tools were the bioesthesiometer and the D2SP. The neurophysiological examination demonstrated the

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presence of neuropathy subjacent to CTS. Clinical and neurophysiological criteria for CTS were presented by 31.91% of the patients; 27.66% with signs of subjacent neuropathy and 4.25% without diabetic neuropathy. Clinical criteria should be preponderant over the remaining tests and the neurophysiological test in order to characterize the carpal tunnel syndrome in the diabetic patient.

KEYWORDS

diabetes mellitus type 2, carpal tunnel syndrome, diabetic neuropathies, neural conduction

INTRODUCTION

Diabetes mellitus is an important public healthcare concern in Brazil and is associated to complications that impair patients' productivity, quality of life and survival.

One of the most common complications of diabetes is the peripheral neuropathy. According to the basic guide for the diagnosis and treatment of diabetes mellitus,[1] it can be observed in 40% of the cases. In patients with type-2 diabetes, the neuropathy can be observed in 8 to 12% of the cases and 50-60% of the patients after 20-25 years of the disease.

The diabetic neuropathy has several forms of presentation, predominantly impairing a modality (neurovegetative, sensitive or motor) or encompassing several modalities: localized or focal (mononeuropathies, multiple mononeuropathy, dorsal and lumbar-sacral radiculopathies) or generalized (sensitive-motor distal symmetrical polyneuropathy).

Among the focal neuropathies (mononeuritis and compression syndromes), the carpal tunnel syndrome is often observed in the diabetic patient, not only due to alterations in the synovial tissue surrounding the nerve, but also because the nerve presents alterations secondary to elevated glycemia.

The physiopathological basis of the carpal tunnel syndrome is the median nerve compression when it passes through the carpal tunnel. This compression can occur due to any tenosynovial proliferation, wrist joint abnormality, tumor or muscular anomaly[2], producing a clinical picture that includes hand pain, paresthesias and hypoesthesias. At advanced stages, it can cause thumb paralysis and loss of sensibility.

Several studies in the literature have reported the incidence of carpal tunnel syndrome in patients with diabetes mellitus,[2-15] which can be asymptomatic in 20 to 30% of the cases.

The aims of this study were: to determine the frequency of carpal tunnel syndrome (CTS) in patients with type-2 diabetes, to verify whether it is associated with diabetic neuropathy and identify ways to confirm both through the assessment of the upper limbs.

METHODS

A total of 94 consecutive patients with type-2diabetes mellitus were assessed; these patients were being treated at the Service of Endocrinology of SUS (Brazilian Public Health Agency), at the Specialty Outpatient Clinic and at the Division of Rehabilitation of the "Lauro de Souza Lima" Institute in Bauru – SP. The time of disease evolution was above 10 years.

In the present study, 92 patients (97.9%) presented hand abnormalities: 47 patients (50%) with less than 10 years of disease evolution and 45 patients (47.9%) with more than 10 years of disease evolution presented these abnormalities.

Glycemia was assessed in all patients on the same day the tests were performed. The mean glucose level was 184.1 mg, which demonstrated an inadequate control of the glycemia.

The presence of paresthesias in the median nerve was investigated and quantitative tests were applied to assess: tactile sensibility, muscular strength and the velocity of sensitive and motor conduction in the median and ulnar nerves. The patients were also inquired about the presence or not of tingling or numbness in the hands.

Regarding Phalen's test, the patients were advised to make the volar flexion of wrist for 60 seconds. The test became significant when the symptoms of paresthesia appeared within 60 seconds, being considered positive.[15,16]

To evaluate the skin sensitivity of the hand, two instruments were used: Semmes-Weinstein nylon monofilaments and the Disk-Criminator™.[17]

Six monofilaments were used, which exert forces of 0.05g, 0.2g, 2.0g, 4.0g, 10.0g and 300g.[18] The Semmes-Weinstein nylon monofilaments were applied on the palmar region, on the sensitive distribution of the median and ulnar nerves. To evaluate the static two-point discrimination (STPD), the stimulus was applied longitudinally, on the distal pulp of the index and little fingers, specifically in the ulnar and radial half,[19] to prevent the crossing of overlapping digital nerves. The evaluation of results was based on the amount of right or wrong answers supplied by the patient. The result was considered positive when, in a total of ten, seven correct answers were given for the discrimination between two points. If the patient could not distinguish seven of 10 stimuli correctly, the distance between the two points was increased. The test was interrupted at 15 mm, when the response was not discriminatory.[19] At the evaluation of results, a threshold < 6 was considered normal for the finger pulp.[20]

The vibratory sensitivity test was carried out with the tuning fork handle of 256 cps.[21] The sensitivity was tested on the distal pulp of the index finger and the little finger. The results of the assessment were registered as follows: present vibratory sensitivity or absent vibratory sensitivity.

To quantify the vibratory sensitivity, the bio-thesiometer (Bio-Thesiometer, Bio-Medical Instruments, Newbury, Ohio).[19, 22] The vibratory sensitivity was tested on the distal pulp of the index finger and little finger, with a fixed frequency of 120 cycles per second. The results were registered in volts. Three ascending and three descending measurements were carried out. The lowest ascending measurement and the highest descendent measurement were discarded. The mean was calculated based on the four remaining measurements. The result was considered normal up to 5.0 V in the median nerve and 6.5 V in the ulnar nerve and was considered altered when it was above these values.

Such values were established after a study of 94 individuals wi-

thout peripheral neuropathy, paired by age and sex with the diabetic population. This study was necessary because most of the patients assessed was older than 50 years and the table that accompanies the instrument presents normal values for a population of young adults. At the statistical analysis, the control population obtained 5% and 95% percentiles.

To study the velocity of conduction of the median and ulnar nerves, a Keypoint electromyograph (Dantec, 1997) was used. The electrodes used were also manufactured by Dantec and the neuroconduction techniques strictly followed those described in the literature (Methods In Clinical Neurophysiology, published by Dantec Medical S/A).[23,24,25]

The measurements of motor and sensitive conduction of the median and ulnar nerve were assessed in both upper limbs. For the sensitive nervous conduction, the orthodromic technique was chosen.[23]

In the median nerve, the conduction of the II finger-wrist was investigated. In case of normal results, the double innervation fingers, I (median and radial) and IV (median and ulnar) were assessed to detect early signs of CTS, as well as the potential of sensitive action with double peak, which characterize incipient alterations of conduction through the carpal tunnel.[26] The palm-wrist conduction, upon stimulation with a fork electrode on the palm and wrist uptake at a distance of 8 cm, was also used to detect early alterations.

The sensitive conduction in the ulnar nerve was also assessed by the orthodromic technique, with stimuli applied to the V finger and wrist uptake at a distance of 12 cm between the cathode of the stimulator and the active recording electrode.[23]

Regarding the motor nervous conduction, the motor action potentials were recorded with two surface electrodes: the *active* electrode, on the muscle belly and the *reference* electrode on a neutral point (bone or tendon). The muscles used were the *Abductor Pollicis Brevis*, for the conduction in the median and the *Abductor Digiti Minimi,* for the ulnar nerve. The stimuli were supramaximal and carried out with bipolar fork electrodes, with a fixed inter-electrode distance. The median nerve was stimulated on the wrist – with the cathode at a standardized distance of 8 cm from the active uptake electrode – and proximally, on the inner side of the tendon of the *Biceps Brachii* muscle.[24] The distal motor latency through the carpal tunnel was compared with the reference values by Stalberg & Falck.[24] The velocities of motor conduction in the median nerve were measured along the forearm; for the ulnar nerve, they were measured along the forearm and through the elbow, with a 30º flexion.[24]

The diagnostic criteria for isolated CTS were: presence of alterations in the study of sensitive and motor conduction; complaints of paresthesia in the area of the median nerve and absence of sensitive or motor alterations in the area of the ulnar nerve; and normal clinical assessment in the lower extremities.

For the diagnosis of diabetic neuropathy, the diagnostic criteria were the presence of alterations in the study of sensitive and/or motor conduction in the ulnar nerve and clinical alterations in the foot.

For the foot evaluation, the medical files of the patients suspected to have carpal tunnel syndrome were reviewed and the presence of claw toes, plantar ulcers, alterations in the tactile sensitivity (Semmes-Weinstein monofilaments) and motricity (test of manual muscular strength with scores going from 0 to 5) were assessed.

Considering that the present study is a descriptive one, the information was processed in categories, in absolute and relative values, which dispensed with the statistical analysis as it was not a comparison of samples.

RESULTS

The results of the tests performed are depicted in Tables 1 and 2. The main symptom of CTS is hand paresthesia, especially in the region of the median nerve, divided in this study as the more specific complaints of tingling or numbness. A total of 60/94 patients (63.82%) presented hand paresthesia: 5 (5.32%) presented numbness; 16 (17.02%) presented tingling and 39 (41.48%) presented both symptoms.

The electrophysiological study detected an alteration in 88/94 patients (93.61%). Alterations in the median nerve were observed in 50 patients (53.19%) and a combined alteration in both median and ulnar nerves in 38 patients (40.42%).

Regarding the tactile sensitivity, at the static two-point discrimination (STPD) test, alterations were observed in 47/94 patients (50%). A combined alteration in both median and ulnar nerves was observed in 28 patients (29.79%); in the median nerve, 2 patients (2.13%) and in the ulnar nerve, 17 patients (18.08%). When using the Semmes-Weinstein monofilaments, alterations were detected in 11/94 patients (11.70%). A combined alteration in the median and ulnar nerves was observed in 5 patients (5.32%), 5/11 (5.32%) in the median nerve and 1/11 (1.06%) in the ulnar nerve.

With the bio-thesiometer, alterations in the vibratory sensitivity were detected in 72/94 patients (76.59%). A combined alteration in the median and ulnar nerves was observed in 49 patients (52.13%); only in the median, in 14 patients (14.89%) and only in the ulnar, in 9 patients (9.57%). With the tuning fork, alterations in the vibratory sensitivity were detected in only 4/94 patients (4.25%). A combined alteration in the median and ulnar nerves was observed

Table 1
Findings regarding the presence of alteration at the tests of tactile sensitivity, vibratory sensitivity, nervous conduction study, median nerve, ulnar nerve and combined median/ulnar nerves.

Tests		Patients with nerve alterations							
		Median		Ulnar		Median/Ulnar		Total	
		n	%	n	%	n	%	n	%
SMSC		50	56,8	0	0,0	38	43,2	88	93,6
Tactile sensibility	D2PE	2	4,2	17	36,2	28	59,6	47	50,0
	SW	5	45,5	1	9,0	5	45,5	11	11,7
Vibratory sensibility	Bio	14	19,4	9	12,5	49	68,1	72	76,6
	Diapasão	2	50,0	1	25,0	1	25,0	4	4,2

SMSC: Study of the Motor and Sensitive Conduction; SW: Semmes-Weinstein; STPD: static two-point discrimination; SW: Semmes-Weinstein; Bio: Bio-thesiometer; TF: tuning fork

Table 2
Findings regarding alterations at the nervous conduction study, Phalen, paresthesias, tactile and vibratory sensibility

Patients	SMSC		Phalen	Parestesias		Tactile sensitivity				Bio-thesiometer		Tuning fork	
						SW		STPD					
	Median	Neuropathy		Tingling	Numbness	Median	Ulnar	Median	Ulnar	Median	Ulnar	Median	Ulnar
1	alt	n	+	s	s	nl	nl	nl	alt	alt	nl	nl	nl
2	nl	n	+	s	s	nl	nl	nl	nl	alt	nl	nl	nl
3	alt	s	+	n	n	nl	nl	nl	nl	alt	alt	nl	nl
4	nl	n	+	s	n	nl	nl	alt	alt	alt	alt	nl	nl
5	alt	s	+	s	s	nl	nl	nl	nl	alt	alt	nl	nl
6	alt	s	-	s	n	nl	nl	alt	alt	alt	alt	nl	nl
7	alt	s	+	s	s	alt	alt	alt	alt	alt	alt	nl	nl
8	alt	n	+	s	s	nl	nl	alt	alt	alt	alt	nl	nl
9	alt	n	+	s	s	alt	nl	alt	alt	alt	alt	nl	nl
10	alt	s	+	s	s	nl	nl	alt	alt	alt	alt	nl	alt
11	alt	n	+	s	s	alt	nl	alt	alt	alt	alt	nl	nl
12	alt	n	-	s	n	nl	nl	nl	nl	alt	alt	nl	nl
13	alt	n	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
14	nl	n	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
15	alt	n	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
16	nl	n	-	n	n	nl	nl	nl	nl	alt	nl	nl	nl
17	alt	s	-	s	s	nl	nl	nl	nl	alt	alt	nl	nl
18	alt	s	-	s	s	nl	nl	nl	alt	alt	alt	nl	nl
19	alt	n	+	s	s	nl	nl	alt	nl	alt	alt	nl	nl
20	alt	n	+	n	n	nl	nl	nl	nl	alt	nl	nl	nl
21	alt	s	+	s	s	alt	nl	alt	alt	alt	alt	alt	nl
22	alt	n	+	s	s	nl	nl	alt	alt	alt	alt	nl	nl
23	alt	n	+	n	s	nl	nl	nl	nl	alt	alt	nl	nl
24	alt	n	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
25	nl	n	-	n	n	nl	nl	nl	nl	nl	nl	nl	nl
26	alt	n	+	s	s	nl	nl	nl	nl	alt	alt	nl	nl
27	alt	s	+	s	s	alt	alt	alt	alt	alt	alt	nl	nl
28	alt	n	-	n	n	nl	nl	nl	alt	alt	nl	nl	nl
29	alt	s	+	s	n	nl	nl	nl	nl	nl	nl	nl	nl
30	alt	s	-	s	s	nl	nl	alt	alt	alt	alt	nl	nl
31	alt	s	+	s	n	nl	nl	nl	nl	alt	alt	nl	nl
32	alt	s	-	s	s	nl	nl	alt	alt	alt	alt	nl	nl
33	alt	n	-	s	s	nl	nl	alt	alt	alt	alt	nl	nl
34	alt	s	+	s	s	nl	nl	nl	alt	alt	alt	nl	nl
35	alt	s	+	n	n	nl	nl	alt	alt	alt	alt	nl	nl
36	alt	s	+	s	n	alt	nl	alt	nl	alt	nl	nl	nl
37	alt	n	+	s	n	nl	nl	alt	alt	alt	nl	nl	nl
38	alt	n	+	n	n	nl	nl	nl	nl	nl	nl	nl	nl
39	alt	n	-	s	n	nl	nl	nl	alt	nl	nl	nl	nl
40	alt	n	-	s	n	nl	nl	nl	nl	alt	alt	nl	nl
41	alt	n	+	s	s	alt	alt	alt	alt	alt	alt	alt	nl
42	alt	s	-	s	s	nl	nl	alt	alt	alt	alt	nl	nl
43	alt	n	-	s	s	nl	nl	alt	alt	alt	alt	nl	nl
44	alt	s	+	s	s	nl	nl	alt	alt	nl	nl	nl	nl
45	alt	n	-	n	s	nl	nl	nl	nl	nl	nl	nl	nl
46	alt	n	-	s	n	nl	nl	nl	alt	alt	nl	nl	nl
47	alt	n	-	s	n	nl	nl	alt	alt	nl	nl	nl	nl

SMSC: Study of the Motor and Sensitive Conduction; SW: Semmes-Weinstein; STPD: static two-point discrimination

(continues)

Table 2
Findings regarding alterations at the nervous conduction study, Phalen, paresthesias, tactile and vibratory sensitivity (continuation)

Patients	SMSC		Phalen	Parestesias		Tactile sensitivity				Bio-thesiometer		Tuning fork	
						SW		D2PE					
	Median	Neuropathy		Tingling	Numbness	Median	Ulnar	Median	Ulnar	Median	Ulnar	Median	Ulnar
48	alt	n	-	n	n	nl	nl	nl	alt	nl	nl	alt	alt
49	alt	n	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
50	alt	n	-	s	s	nl	nl	nl	nl	nl	alt	nl	nl
51	alt	n	+	s	n	nl	nl	nl	alt	nl	alt	nl	nl
52	alt	n	+	s	s	nl	nl	nl	alt	nl	nl	nl	nl
53	alt	n	-	s	n	nl	nl	nl	nl	nl	alt	nl	nl
54	alt	s	-	s	s	nl	nl	nl	alt	alt	alt	nl	nl
55	alt	n	-	n	n	nl	nl	nl	alt	nl	nl	nl	nl
56	alt	n	+	s	s	nl	nl	nl	nl	alt	alt	nl	nl
57	alt	n	-	s	s	nl	nl	nl	alt	nl	nl	nl	nl
58	alt	n	-	n	n	nl	nl	nl	nl	nl	nl	nl	nl
59	alt	n	-	n	n	nl	nl	nl	nl	nl	nl	nl	nl
60	alt	n	+	s	s	nl	nl	nl	alt	nl	nl	nl	nl
61	alt	n	-	s	s	nl	nl	nl	nl	alt	nl	nl	nl
62	alt	n	-	n	n	nl	nl	nl	alt	nl	nl	nl	nl
63	alt	n	+	s	s	nl	nl	nl	nl	alt	alt	nl	nl
64	alt	n	-	n	n	nl	nl	nl	nl	nl	alt	nl	nl
65	alt	n	-	s	s	nl	nl	nl	nl	nl	nl	nl	nl
66	alt	s	-	s	s	nl	nl	nl	nl	alt	alt	nl	nl
67	alt	n	-	s	s	nl	nl	nl	nl	alt	nl	nl	nl
68	nl	n	-	n	n	nl	nl	nl	nl	nl	nl	nl	nl
69	alt	n	-	n	n	nl	nl	nl	alt	alt	nl	nl	nl
70	alt	s	-	n	n	nl	nl	nl	alt	nl	nl	nl	nl
71	alt	s	-	n	n	nl	nl	nl	nl	nl	nl	nl	nl
72	alt	s	-	n	s	nl	nl	nl	nl	alt	alt	nl	nl
73	alt	s	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
74	alt	s	-	s	s	alt	nl	nl	nl	nl	nl	nl	nl
75	alt	n	-	n	n	nl	nl	nl	nl	nl	alt	nl	nl
76	alt	s	-	n	s	nl	nl	alt	alt	alt	alt	nl	nl
77	alt	n	-	n	n	nl	nl	nl	nl	alt	nl	nl	nl
78	alt	s	-	s	s	nl	nl	alt	alt	alt	alt	nl	nl
79	alt	s	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
80	alt	s	+	n	n	nl	nl	alt	alt	alt	alt	nl	nl
81	nl	s	-	n	n	nl	nl	nl	nl	alt	nl	nl	nl
82	alt	s	+	s	s	nl	alt	nl	nl	nl	alt	nl	nl
83	alt	n	-	n	n	nl	nl	nl	nl	nl	nl	nl	nl
84	alt	s	-	n	s	nl	nl	alt	alt	alt	alt	nl	nl
85	alt	s	-	s	n	alt	alt	alt	alt	nl	alt	nl	nl
86	alt	s	-	s	s	alt	alt	alt	alt	alt	alt	nl	nl
87	alt	n	-	n	n	nl	nl	nl	alt	nl	nl	nl	nl
88	alt	n	-	n	n	nl	nl	nl	nl	nl	alt	nl	nl
89	alt	s	-	n	n	nl	nl	nl	nl	nl	alt	nl	nl
90	alt	n	-	n	n	nl	nl	alt	alt	alt	nl	nl	nl
91	alt	s	-	s	n	nl	nl	alt	alt	alt	alt	nl	nl
92	alt	n	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
93	alt	s	-	n	n	nl	nl	nl	nl	alt	alt	nl	nl
94	alt	s	-	s	n	nl	nl	nl	nl	alt	alt	nl	nl

ECSM: Estudo da Condução Sensitiva e Motora; SW: Semmes-Weinstein; D2PE: Discriminação de dois Pontos Estáticos

in 1 patient (1.06%); in the median, in 2 patients (2.12%); and in the ulnar, in 1 patient (1.06%).

The positivity at Phalen's test was observed in 33/94 of the patients (35.11%).

DISCUSSION

The most common symptoms of carpal tunnel syndrome are the paresthesias in the distribution of the median nerve in the wrist, varying from 96% to 100%.[3,26-30] In this study, paresthesias (numbness and tingling) were observed in 60 patients (63.82%).

For Seror,[16] the positivity at Phalen's test can differ between examiners and the author attributed such differences to three factors: the 60-second period with the wrist in flexion was not respected by some examiners; the patient's response might be incorrectly interpreted when induced by a single sensation; the maximum wrist flexion was not ascertained during the test. The author also referred that 34% of the patients with a diagnosis of CTS presented negative Phalen's test and that 20% of the individuals in the control group presented a positive Phalen's test.

In this study, a positive Phalen's test was observed in 33 patients (35.10%). Edwards [31] reported that, in the diabetic population, Phalen's test is not an indicative of CTS.

Gelberman et al[32] evaluated, through many tests, the controlled external compression of the median nerve in the carpal tunnel (at a level of 40, 50, 60 and 70 mm Hg). They observed that the tactile sensitivity assessed by Semmes-Weinstein monofilaments correlated accurately with the symptoms of nervous compression.

In this study, the percentage of alterations in tactile sensitivity (11.70%), assessed by Semmes-Weinstein monofilaments, were lower than those found in literature (83% to 91%).[22,32-34] Alterations in the static two-point discrimination (STPD) test were detected in 50% of the patients, whereas in the literature about CTS, the alteration varied from 14.4% to 51.1%.[32-35] The sensitivity test with Semmes-Weinstein monofilaments was more sensitive than the STPD test in the evaluations of chronic compressive neuropathies.[22] Callahan,[19] in his study, concluded that the abnormalities in STPD and MTPD? are late findings in compressive neuropathy.

The STPD test, performed in this study, detected more alterations than Semmes-Weinstein monofilaments, contrarily to the results found in the literature regarding CTS. These findings must be due to the characteristics of this sample, in which the patients presented a time of diabetes evolution > 10 years.

To evaluate the vibratory sensitivity, the tuning fork of 256 cps was used, which provided qualitative results: it detected alterations in 4 patients (4.2%). The small differences of perception on the intensity of vibratory stimuli were not quantified, and thus, the results were lower than those found in literature regarding CTS.[21, 22, 30]

According to Dellon,[37,38] Williams was the first to observe the decrease in the vibratory perception in diabetic neuropathies and diabetes mellitus was the first disease that had vibratory stimuli studied. For the author, the use of the tuning fork presents two disadvantages: the amplitude of the stimulus is not controlled and varies according to the force applied by the examiner when hitting it; hence, the results are expressed qualitatively, only.

The total number of patients with alterations in vibratory sensitivity evaluated with the bio-thesiometer was 72 (76.59%). It was observed in the present study that the bio-thesiometer detected more alterations than the tuning fork, as it provides quantitative data; the same findings were observed by Szabo.[39]

The alterations found in the test of vibratory sensitivity with the bio-thesiometer were higher than those found at the STPD test, which is in accordance with the literature.[22,38,35] According to Mirsky,[40] the alteration in the threshold of vibratory perception in diabetic patients is higher when compared to a non-diabetic person.

The study of nervous conduction, in addition to being quantitative, does not present the subjectivity of the other tests; therefore, it is considered to be the gold-standard for the diagnosis of peripheral neuropathies.[40] In this study, alterations were observed in 88 patients (93.61%). The review of the patients' medical files showed that the neurophysiological findings in the ulnar nerve were in accordance with the presence of clinical alterations in the lower limbs, and thus, its presence was considered a sign of systemic diabetic neuropathy.

The abnormalities found at the electrodiagnostic study are considered important for some authors,[41] but others[42,43] went as far as stating that the electrophysiological study is neither necessary nor sufficient to diagnose CTS, when the symptomatology is specific. Some of them reported CTS cases without electromyographic abnormalities and concluded that, regarding the neuropathy of the median nerve, the electrodiagnostic evidence is not enough to attain a diagnosis in the absence of symptoms. However, Tountas et al[44] reported that only 15% of the patients with CTS presented a normal electrophysiological study. As for the electrophysiological study reported in the literature, the alterations found in the median nerve with a clinical suspicion of CTS vary from 24% to 100%.[7, 18, 19, 23, 24]

Currently, the most widely accepted concept among researchers on the diagnosis of CTS is that of the concordance between the clinical signs and the electrophysiological alterations.[25, 42, 44-48]

The prevalence of carpal tunnel syndrome in the general population is 1% to 3%. In the diabetic population, according to Chamas[11] and Vinik[49], this prevalence is 11%-23%. For other authors, this incidence varies from 4.4% to 8.0%.[2,6,7] Phalen[4] reported that, in the diabetic population, the median nerve might be more susceptible to compression inside the carpal tunnel, when compared to non-diabetic patients.

To establish the differences between the isolated CTS, CTS associated to diabetic neuropathy and diabetic neuropathy without CTS, several tests were applied, which allowed the identification of patients presenting these alterations. If the diabetic patient has the potential to develop alterations in the peripheral nervous system, when alterations are found in the ulnar nerve region, one must suspect that they caused by the diabetic process.

At correlation of the results, it was observed that 92/94 (97.87%) of the patients presented confirmed peripheral nervous disorders: 11 (11.70%) of them presented alterations in the median nerve only and 81 (86.17%) presented combined alterations in the median and

ulnar nerves.

The criteria for the diagnosis of CTS were the presence of paresthesias (tingling or numbness) and the decrease in the velocity of sensitive or motor conduction in the median nerve; when the results were correlated, it was observed that among the 11 (11.7%) patients that presented alterations only in the median nerve, 4 (4.25%) had carpal tunnel syndrome (CTS) without subjacent neuropathy and 7 (7.45%) presented alterations in the median nerve without clinical signs of CTS. No foot alterations were found in the medical files of these patients.

Among the 81 (86.17%) patients with combined alterations in the median and ulnar nerves, 55 (58.51%) presented diabetic neuropathy with no clinical signs of CTS and 26 (27.66%) presented CTS + diabetic neuropathy.

Considering the 92/94 patients with confirmed peripheral nerve disorders, 30 of them (26 with CTS + diabetic neuropathy and 4 with isolated CTS) presented the clinical criteria defined for CTS and alterations in sensitive and motor conduction in the median nerve through the carpal tunnel, reaching a frequency of 31.91% of CTS.

On the other hand, all patients who presented electrophysiological alterations in the ulnar nerve also presented abnormalities at the clinical-neurological examination of the feet, showing a strong correlation between these findings in this sample of patients, with long-term evolution of diabetes.[50]

When the upper limb is evaluated electrophysiologically in search of CTS, the assessment of the ulnar nerve showed to be essential for the diagnosis of diabetic neuropathy subjacent to the carpal syndrome.

CONCLUSION

In the present study, the most sensitive tools used to detect neurological alterations were the bioesthesiometer and the STPD test.

The sensitivity tests are good options for the screening of diabetic patients with hand paresthesias, before submitting the patients to more complex procedures, as they can indicate the presence of localized neuropathy and confirm suspected systemic neuropathy.

The neurophysiological assessment of the upper limbs in diabetic patients, with the presence of ulnar nerve alterations – sensitive in the V finger as well as motor along the nerve – showed a correlation with the presence of systemic neuropathy subjacent to median nerve impairment or CTS.

A total of 31.91% of the patients met the clinical and neurophysiological criteria for CTS: 27.66% with subjacent neuropathy and 4.25% without diabetic neuropathy.

These data suggest that the clinical criteria must be considered predominant when compared to the other tests and that the neurophysiological assessment must be used to characterize CTS in the diabetic patient.

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REFERENCES

1. Brasil. Ministério da Saúde de Assistência à Saúde. Departamento de Assistência e Promoção à Saúde. Coordenação de Doenças Crônico - Degenerativas. Diabetes mellitus: guia básico para diagnóstico e tratamento. Brasília: MSAS; 1996.
2. Kouyoumdjian JA. Síndrome do túnel do carpo: aspectos clínico-epidemiológico em 668 casos. Arq Neuro-Psiquiatr. 1999;57(2):504-12.
3. Leach RE, Odom JA. Systemic causes of carpal tunnel syndrome. Postgrad Med. 1968;44(2):127-31.
4. Phalen GS. The carpal tunnel syndrome-clinical evaluation of 598 hands. Clinic Orthop. 1972; 83: 29-40.
5. Gamstedt A, Holm-Glad J, Ohlson CG, Sundström M. Hand abnormalities are strongly associated with the duration of diabetes mellitus. J Intern Med. 1993;234(2):189-93.
6. Stevens JC, Beard CM, O'Fallon WM, Kurland LT. Conditions associated with carpal tunnel syndrome. Mayo Clin Proc.1992;67(6):541-8.
7. Dobyns JH. Carpal tunnel release in patients with peripheral neuropathy. In: Gelberman RH. Operative nerve repair and reconstruction. Philadelphia: J B Lippincott Company; 1991. p. 963-5.
8. Clayburgh RH, Beckenbaugh RD, Dobyns JH. Carpal tunnel release in patients with diffuse peripheral neuropathy. J Hand Surg [Am]. 1987;12(3):380-3.
9. Posch JL, Marcotte DR. Carpal tunnel syndrome: an analysis of 1.201 cases. Orthop Rev. 1976; 5:25–35.
10. Comi G, Lozza L, Galardi G, Ghilardi MF, Medaglini S, Canal N. Presence of carpal tunnel syndrome in diabetics: effect of age, sex, diabetes duration and polyneuropathy. Acta Diabetol Lat. 1985;22(3):259-62.
11. Chammas M, Bousquet P, Renard E, Poirier JL, Jaffiol C, Allieu Y. Dupuytren's disease, carpal tunnel syndrome, trigger finger, and diabetes mellitus. J Hand Surg [Am].1995;20(1):109-14.
12. Perkins BA, Olaleye D, Bril V. Carpal tunnel syndrome in patients with diabetic polyneuropathy. Diabetes Care. 2002;25(3):565-9.
13. Stamboulis E, Vassilopoulos D, Kalfakis N. Symptomatic focal mononeuropathies in diabetic patients: increased or not? J Neurol. 2005;252(4):448-52.
14. Schiavon F, Circhetta C, Dani L. The diabetic hand. Reumatismo. 2004;56(3):139-42.
15. Phalen GS. Spontaneous compression of the median nerve at the wrist. J Am Med Assoc. 1951;145(15):1128-33.
16. Seror P. Phalen's test in the diagnosis of carpal tunnel syndrome. J Hand Surg [Br]. 1988;13(4):383-5.
17. Crosby PM, Dellon AL. Comparison of two-point discrimination testing devices. Microsurgery. 1989;10(2):134-7.
18. Lehman LF, Orsini MB, Nicholl AR. The development and adaptation of the Semmes-Weinstein monofilaments in Brazil. J Hand Ther. 1993;6(4):290-7.
19. Callahan A.D. Sensibility testing: clinical methods. In: Hunter JM, Schneider LH, Mackin EJ, Callahan AD. Rehabilitation of the hand: surgery and therapy. 3th ed. St Louis: Mosby; 1990. p. 594-610.
20. American Society for Surgery of the Hand. The Hand: Examination and diagnosis. Aurora, CO: American Society for Surgery of the Hand; 1978.
21. Dellon AL. Clinical use of vibratory stimuli to evaluate peripheral nerve injury and compression neuropathy. Plast Reconstr Surg. 1980;65(4):466-76.

22. Szabo RM, Gelberman RH, Dimick MP. Sensibility testing in patients with carpal tunnel syndrome. J Bone Joint Surg Am. 1984;66(1):60-4.
23. Falck B, Stålberg E, Christian B. Sensory nerve conduction studies with surface electrodes. Methods Clin Neurophysiol. 1994;5(1):1-20.
24. Stålberg E, Falck B. Clinical motor nerve conduction studies. Methods Clin Neurophysiol 1993;4:61-80.
25. Garbino JA. Avaliação eletroneuromiográfica da neuropatia diabética. In: Kuhn P. O pé diabético. São Paulo: Atheneu; 2006. p. 159-67.
26. Kouyoumdjian JA. Síndrome do túnel do carpo. São José do Rio Preto: Editora e Informática Rio Preto; 2001.
27. Barbieri CH, Cisneros RR, Audi A. Síndrome de túnel do carpal. Rev Brasil Ortop. 1985;20:255-60.
28. Clayburgh RH, Beckenbaugh RD, Dobyns JH. Carpal tunnel release in patients with diffuse peripheral neuropathy. J Hand Surg [Am]. 1987;12(3):380-3.
29. Phalen GS. The carpal-tunnel syndrome. Clinical evaluation of 598 hands. Clin Orthop Relat Res. 1972;83:29-40.
30. Borelli G, Stadler G, Abreu J, Medina E, Benzano D. Valor diagnóstico de los sintomas y signos clínicos en el síndrome tunelar carpiano. Med Reabil. 2006;25(2):35-9.
31. Edwards A. Phalen's test with carpal compression: testing in diabetics for the diagnosis of carpal tunnel syndrome. Orthopedics. 2002;25(5):519-20.
32. Gelberman RH, Szabo RM, Williamson RV, Dimick MP. Sensibility testing in peripheral-nerve compression syndromes. An experimental study in humans. J Bone Joint Surg Am. 1983;65(5):632-8.
33. Gellman H, Gelberman RH, Tan AM, Botte MJ. Carpal tunnel syndrome. An evaluation of the provocative diagnostic tests. J Bone Joint Surg Am. 1986;68(5):735-7.
34. Szabo RM. Carpal tunnel syndrome – general. In: Gelberman RH. Operative nerve repair and reconstruction. Philadelphia: J.B. Lippincott Company;1991. p.869-87.
35. Segre NG. Avaliação pós-operatória do tratamento da síndrome do túnel do carpo pela via de acesso unal [dissertação]. São Paulo: Universidade Federal de São Paulo, Escola Paulista de Medicina; 1996.
36. Lundborg G, Lie-Stenström AK, Sollerman C, Strömberg T, Pyykkö I. Digital vibrogram: a new diagnostic tool for sensory testing in compression neuropathy. J Hand Surg [Am]. 1986;11(5):693-9.
37. Dellon AL. Evaluation of sensibility and reeducation of sensation in the hand. Baltimore: Williams & Wilkins; 1991.
38. Dellon AL. The vibrometer. Plast Reconstr Surg. 1983;71(3):427-31.
39. Szabo RM, Gelberman RH, Williamson RV, Dellon AL, Yaru NC, Dimick MP. Vibratory sensory testing in acute peripheral nerve compression. J Hand Surg [Am]. 1984;9A(1):104-9.
40. Mirsky IA, Futterman P, Brohkahn RH. The quantitative measurement of vibratory perception in subjects with and without diabetes mellitus. J Lab Clin Med. 1953;41(2):221-35.
41. Dyck PJ, Hughes RAC, O'Brien PC. Quantitating Overall Neuropathic Symptoms, Impairments, and Outcomes. In: Dyck PJ, Thomas PK, editors. Peripheral neuropathy. Part IX. Quantitating symptoms, impairments, and outcomes. 4th ed. Philadelphia: Elsevier Saunders; 2005. p. 1031-52.
42. Katz JN, Stirrat CR, Larson MG, Fossel AH, Eaton HM, Liang MH. A self administered hand symptom diagram for the diagnosis and epidemiologic study of carpal tunnel syndrome. J Rheumatol. 1990;17(11):1495-8.
43. Amadio PC, Silverstein MD, Ilstrup DM, Schleck CD, Jensen LM. Outcome assessment for carpal tunnel surgery: the relative responsiveness of generic, arthritis-specific, disease-specific, and physical examination measures. J Hand Surg [Am]. 1996;21(3):338-46.
44. Tountas CP, MacDonald CJ, Meyerhoff JD, Bihrle DM. Carpal tunnel syndrome. A review of 507 patients. Minn Med. 1983;66(8):479-82.
45. Spinner RJ, Bachman JW, Amadio PC. The many faces of carpal tunnel syndrome. Mayo Clin Proc. 1989;64(7):829-36.
46. de Krom MC, Knipschild PG, Kester AD, Spaans F. Efficacy of provocative tests for diagnosis of carpal tunnel syndrome. Lancet. 1990;335(8686):393-5.
47. Atroshi I, Gummesson C, Johnsson R, Ornstein E, Ranstam J, Rosen I. Prevalence of carpal tunnel syndrome in a general population. JAMA. 1999;282(2):153-8.
48. Franzblau A, Werner RA. What is carpal tunnel syndrome? JAMA. 1999;282(2):186-7.
49. Vinik A, Mehrabyan A, Colen L, Boulton A. Focal entrapment neuropathies in diabetes. Diabetes Care. 2004;27(7):1783-8.
50. Llewelyn JG, Tomlinson DR, Thomas PK. Diabetic neuropathies. In: Dyck PJ, Thomas PK, editors. Peripheral neuropathy. 4th ed. Philadelphia: Elsevier Saunders; p.1951-91.
50. Llewelyn JG, Tomlinson DR, Thomas PK. Diabetic Neuropathies. In: Dyck PJ, Thomas PK. Peripheral Neuropathy. K. Neuropathy Associated with Systemic Disease. 4th. Philadelphia: Elsevier Saunders; 2005. p. 1951-91.