Yonsei Medical Journal
Vol. 41, No. 1, pp. 123-127, 2000

Asymptomatic Electrophysiologic Carpal Tunnel Syndrome in Diabetics: Entrapment or Polyneuropathy

Woo-Kyung Kim[1], Soon-hee Kwon[2], Soong-Hyun Lee[1], and Il Nam Sunwoo[1]

Abstract

Electrophysiologic carpal tunnel syndrome (CTS) is common and is frequently asymptomatic in diabetics. In order to evaluate the clinical significance of asymptomatic electrophysiologic CTS, the nerve conduction studies (NCS) of 48 diabetics with asymptomatic electrophysiologic CTS were compared with those of 56 age and gender-matched controls, as well as 50 patients with symptomatic CTS without diabetes. Nerve conduction velocities of the ulnar, peroneal, and posterior tibial nerves were significantly slower in diabetics with asymptomatic electrophysiologic CTS than in normal controls. Compared to symptomatic non-diabetic CTS, there was also significant slowing of the median and ulnar nerve conduction velocities in asymptomatic diabetic CTS. However, in diabetics with asymptomatic CTS, abnormalities of the distal segment of the median NCS were more prominent compared with those of all the other tested nerves. These findings suggested that asymptomatic electrophysiologic CTS in diabetics is a manifestation of increased vulnerability to the entrapment of the peripheral nerve.

Key Words: Carpal tunnel syndrome, diabetic polyneuropathy, nerve conduction study

INTRODUCTION

Peripheral neuropathy is a common complication of diabetes. Its prevalence has been reported as having a range from less than 5% to nearly 60% by various investigators.[1] The most common form of diabetic neuropathy is distal symmetric sensory-motor polyneuropathy followed by carpal tunnel syndrome (CTS), autonomic neuropathy, and other varieties.[2] It is well known that diabetic neuropathies are frequently asymptomatic.

Nerve conduction studies (NCS) are well established and are considered to be the most sensitive, reliable, and objective means for studying and diagnosing diabetic neuropathies.[3-6] The high reproducibility of NCS and their correlations with nerve fiber loss and structural insults make these tests sensitive indicators of the presence of diabetic neuropathies. Sometimes the NCS are abnormal, even in completely asymptomatic diabetic patients. Although it is well known that asymptomatic electrophysiologic CTS is common in diabetic patients, the clinical significance is not clear.[7,8] For future treatment planning, it is very important to determine whether this finding is an entrapment of the median nerve under the transverse carpal ligament, or if it is an early manifestation of diabetic polyneuropathy.

To determine the clinical significance of asymptomatic electrophysiologic CTS in diabetes, we compared the NCS of diabetic asymptomatic electrophysiologic CTS patients with those of age- and gender-matched normal controls and symptomatic CTS patients without diabetes.

Received November 12, 1999
Accepted December 27, 1999
[1]Department of Neurology, Yonsei University College of Medicine, Seoul, [2]Department of Neurology, Pundang Jesaeng General Hospital, Sungnam-Si, Kyungki-Do, Korea
Address reprint request to Dr. I. N. Sunwoo, Department of Neurology, Yonsei University College of Medicine, C.P.O. Box 8044, Seoul 120-752, Korea. Tel: 82-361-5463, Fax: 82-393-0705, E-mail: sunwooin@yumc.yonsei.ac.kr

MATERIALS AND METHODS

Subjects

During the study period (Jan 1993-Feb 1996), all diabetic patients who registered at the diabetes special clinic of Severance Hospital were advised to take NCS for the baseline data regardless of suspected neuropathy. Therefore, many diabetic patients absolutely

free of neuropathy were administered a NCS. About 700 diabetic patients were studied during this period. Among them, 48 cases (37 females and 11 males) were studied. The inclusion criteria were: 1) no evidence of neuropathy as well as retinopathy or nephropathy in case history and upon physical examination; and 2) median nerve NCS compatible with electrophysiologic criteria of CTS. The electrophysiologic criteria of CTS in this study were an abnormal sensory NCS of the index finger-wrist and/or palm-wrist segments, with or without prolonged terminal latency in the median nerve. To avoid being compounded by other factors, we excluded diabetic patients having any abnormal NCS on all the other tested nerves according to the normative data of our laboratory. The average duration of diabetes was 61.2 months with a range of 1−372 months.

For comparison, we had recruited 50 symptomatic CTS patients without diabetes. They were all female and the NCS were performed in the involved arms only. In addition, we had performed the NCS in 56 age- and gender-matched normal healthy controls (40 females and 16 males). The mean age was 52.6 years for the asymptomatic diabetic CTS, 53.6 years for the symptomatic CTS without diabetes, and 51.3 years for the control group. The height was 158.8±6.6 cm (mean and standard deviation), 155.7±4.2 cm, and 158.9±7.5 cm respectively. There was no statistical difference between these groups.

Nerve conduction study

Nerve conduction studies were performed with conventional techniques of supramaximal percutaneous nerve stimulations and surface recordings in one upper and one lower limb (Excel apparatus, Cadwell,

Table 1. Results of Nerve Conduction Studies

Nerve			Diabetics with Asymptomatic Electrophysiologic CTS (n=48)	Control (n=56)	p-value
Median	TL (msec)		3.89±0.46*	2.91±0.41	.000
	Motor NCV (m/sec)		54.4±2.91*	58.9±4.37	.000
	CMAP (mV)		13.7±4.14	13.2±3.32	.482
	Mixed NCV (m/sec)	W-E	53.7±2.82*	56.8±3.12	.000
	Sensory NCV (m/sec)	F-W	34.3±5.79*	46.8±4.16	.000
	MNAP (uV)	W-E	35.7±12.6*	47.8±19.5	.000
	SNAP (uV)	F-W	24.0±11.7*	33.7±17.6	.001
Ulnar	TL (msec)		2.44±0.30*	2.24±0.33	.002
	Motor NCV (m/sec)		55.7±4.24*	61.4±4.21	.000
	CMAP (mV)		14.2±2.41	14.0±2.39	.594
	Mixed NCV (m/sec)	W-E	52.2±3.16*	56.0±3.53	.000
	Sensory NCV (m/sec)	F-W	43.4±3.29*	45.7±4.19	.002
	MNAP (uV)	W-E	37.9±17.7	44.5±16.9	.053
	SNAP (uV)	F-W	19.0±8.24	22.6±9.98	.051
Peroneal	TL (msec)		3.71±0.70	3.49±0.56	.077
	Motor NCV (m/sec)		45.1±2.54*	48.2±4.07	.000
	CMAP (mV)		5.76±3.17	6.40±4.89	.434
Post.tibial	TL (msec)		3.86±0.65	3.89±0.68	.840
	Motor NCV (m/sec)		46.7±4.02*	49.3±4.48	.002
	CMAP (mV)		18.1±5.95	19.2±7.20	.397
Sural	Sensory NCV (m/sec)		41.4±4.32	42.2±4.53	.388
	SNAP (uV)		21.2±10.2	22.5±10.6	.506

TL, terminal latency; CMAP, compound muscle action potential; MNAP, mixed nerve action potential; SNAP, sensory nerve action potential; W-E, wrist-elbow; F-W, finger-wrist.
* $p<0.05$ for differences between two groups (Student t test).

Table 2. Comparison of the Median and Ulnar Nerve Conduction Studies

Nerve			Diabetics with Asymptomatic Electrophysiologic CTS (n=37)	Non-diabetic Symptomatic CTS (n=50)	Control (n=40)	p-value *	p-value †	p-value ‡
Median	TL (msec)*†‡		3.90±0.50	5.77±1.34	2.82±0.37	.000	.000	.000
	Motor NCV(m/sec)*‡		54.5±2.97	54.9±6.74	59.1±4.61	.001	.920	.002
	CMAP (mV)†‡		13.2±4.02	9.29±4.12	12.7±3.02	.821	.000	.000
	Mixed NCV (m/sec)	W-E	54.0±2.79	53.3±9.89	56.8±2.93	.194	.883	.051
	Sensory NCV (m/sec)*†‡	F-W	33.7±6.42	21.2±14.0	46.1±3.69	.000	.000	.000
	MNAP (uV)*‡	W-E	36.6±11.7	31.0±20.5	47.0±17.8	.036	.344	.000
	SNAP (uV)*†‡	F-W	25.3±12.5	9.50±7.97	35.4±19.4	.007	.000	.000
Ulnar	TL (msec)*		2.41±0.28	2.27±0.34	2.18±0.32	.007	.118	.404
	Motor NCV (m/sec)*†		56.2±4.44	60.8±4.91	61.7±4.05	.000	.000	.618
	CMAP (mV)		14.0±2.17	13.6±2.71	14.0±2.16	.999	.674	.690
	Mixed NCV (m/sec)*†	W-E	52.8±3.14	55.8±4.28	56.6±3.51	.000	.001	.611
	Sensory NCV (m/sec)*	F-W	43.6±3.26	45.0±4.13	45.9±3.84	.036	.234	.573
	MNAP (uV)	W-E	36.0±16.6	38.5±14.3	42.7±16.6	.179	.755	.465
	SNAP (uV)	F-W	20.1±8.11	22.1±8.79	23.1±8.06	.292	.541	.855

Significant differences among three groups were determined by multiple comparisons (Scheffe method, $p<0.05$).
* for difference between diabetic asymptomatic CTS and control groups, † for difference between diabetic asymptomatic CTS and non-diabetic symptomatic CTS groups, and ‡ for difference between non-diabetic symptomatic CTS and control groups.
TL, terminal latency; CMAP, compound muscle action potential; MNAP, mixed nerve action potential; SNAP, sensory nerve action potential; W-E, wrist-elbow; F-W, finger-wrist.

WA, USA). Results were interpreted as abnormal when the nerve conduction velocity (NCV) was slower by more than 2 standard deviations of the normal means of our laboratory.

Statistical analysis

Statistical analysis was done using the statistical package SPSS-PC. Student *t* test and multiple comparisons were performed for comparisons of measures. The level of statistical significance was set at a p value of 0.05. Data were presented as means and standard deviations.

RESULTS

The results of nerve conduction studies of diabetic asymptomatic electrophysiologic CTS and the normal controls are shown in Table 1. There was significant slowing of NCV in asymptomatic diabetic CTS in the ulnar, peroneal, and posterior tibial nerves. The comparison of NCS of the ulnar nerve among three

Table 3. Results of Median/Ulnar Ratio of the Nerve Conduction Studies

Ratios	Diabetics with Asymptomatic Electrophysiologic CTS (n=48)	Control (n=56)	p-value
TL*†	1.450±0.446	0.675±0.391	.000
Forearm motor NCV	0.982±0.076	0.962±0.089	.240
Mixed NCV (W-E)	1.033±0.075	1.018±0.075	.313
Sensory NCV (F-W)*	0.792±0.141	1.030±0.118	.000
MNAP (W-E)	1.063±0.417	1.189±0.583	.206
SNAP (F-W)	1.413±0.744	1.666±0.975	.146

* $p<0.05$ for differences between two groups (Student *t* test).
† Difference between the TL of the median and TL of the ulnar nerves.
TL, terminal latency; MNAP, mixed nerve action potential; SNAP, sensory nerve action potential; W-E, wrist-elbow; F-W, finger-wrist.

groups of female diabetic asymptomatic CTS, female normal controls, and symptomatic CTS without diabetes confirmed the slowing of NCV in diabetics only (Table 2). In spite of diffuse slowing, the slowing of NCV was more prominent in the distal segment of the median nerve in diabetic patients. The median/ulnar ratio of sensory' NCV between the finger and the wrist was significantly reduced in diabetic patients compared with normal controls, whereas median/ulnar ratio of mixed NCV between the wrist and the elbow was no different between diabetic patients and normal controls (Table 3). The difference between the terminal latency of the median and the ulnar nerve was significantly increased in diabetic patients, whereas forearm motor NCV was no different (Table 3).

DISCUSSION

Diabetic neuropathy is one of the most common types of neuropathy. Early detection of the neuropathy is important to reduce the morbidity in diabetes, and NCS is a valuable tool for this purpose.[9] The cross-sectional study of diabetic neuropathy reported by Dyck et al. found polyneuropathy to be the most common form of diabetic neuropathy followed by CTS.[2] With regard to CTS, Ozaki et al. reported CTS was more common in diabetic individuals.[10] They postulated a diabetic nerve was generally vulnerable to extraneural pressure and more susceptible to entrapment. Dyck et al. found that approximately one quarter of patients with diabetes had electrophysiologic abnormalities characteristic of CTS without any symptoms of CTS.[2] Other authors have reported that 14.3% of patients with diabetes without neuropathy symptoms had asymptomatic electrophysiologic CTS.[11] In our study, approximately 6.8% of the diabetic patients had asymptomatic electrophysiologic CTS. The reason for less frequent asymptomatic electrophysiologic CTS in our study may relate to narrower inclusion criteria than others.

The aim of this study was to investigate whether asymptomatic CTS in diabetic patients was a manifestation of early polyneuropathy or whether it was an entrapment neuropathy itself. We found that nerve conduction was slower in diabetic patients with asymptomatic electrophysiologic CTS. Some previous studies have recognized that nerve conduction velocities were slower in diabetic patients without evidence of neuropathy than in the non-diabetic population.[12] Several investigators have hypothesized that an endoneurial edema initiates the deterioration in nerve electrophysiology which is followed by abnormal findings on neurological examinations and precedes the patient's final perception of symptomatic stocking-glove peripheral neuropathy.[13,14] Unfortunately, the above question has not been answered because our cross-sectional study has a clear limitation in showing progressive deterioration. However, we found that conduction delay in the distal segment of the median nerve was more remarkable than that in the distal segment of the ulnar nerve in diabetic patients with asymptomatic CTS. Therefore, our results may suggest that asymptomatic CTS in diabetic patients is related to an increased vulnerability to entrapment at the carpal tunnel.

Among diabetic patients with electrophysiologic abnormalities suggesting CTS, less than 30% of patients were symptomatic and little is known about this absence of typical entrapment symptoms.[2] One of the possible factors is an increased sensory threshold in diabetic patients. It is also possible that asymptomatic patients with diabetes have less severe electrophysiologic abnormalities compared with symptomatic CTS patients. Table 2 shows that asymptomatic CTS patients with diabetes have better electrophysiologic findings compared with symptomatic CTS patients without diabetes in our study. Diffuse conduction slowing in diabetic patients with asymptomatic CTS may explain a high sensory threshold and less pain in association with CTS. Further investigations such as a quantitative sensory testing are needed to confirm this assumption.

Based on these findings, we can postulate that asymptomatic CTS in diabetic patients is related with peripheral nerve vulnerability to entrapment rather than an early polyneuropathy. Again, further assessments such as a follow-up NCS or quantitative sensory testing might help to explain these manifestations.

REFERENCES

1. Thomas PK, Tomlinson DR. Diabetic and hypoglycemic neuropathy. In: Dyck PJ, Thomas PK, Griffin JW, Low PA, Poduslo JF, editors. Peripheral Neuropathy. 3rd ed. Philadelphia: Saunders; 1993. p.1219-50.
2. Dyck PJ, Kratz KM, Karnes JL, Litchy WJ, Klein R, Pach

JM, et al. The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: The Rochester diabetic neuropathic study. Neurology 1993;43:817-24.
3. Dyck PJ, Bushek W, Spring EM, Karnes JL, Litchy WJ, O'Brien PC, et al. Vibratory and cooling detection thresholds compared with other tests in diagnosing and staging diabetic neuropathy. Diabetes Care 1987;10:432-40.
4. Claus D, Mustafa C, Vogel W, Herz M, Neundörfer B. Assessment of diabetic neuropathy: definition of norm and discrimination of abnormal nerve function. Muscle Nerve 1993;16:757-68.
5. Bril V. Role of electrophysiologic studies in diabetic neuropathy. Can J Neurol Sci 1994;21:S8-S12.
6. American diabetes association. Report and recommendations of the San Antonio conference on diabetic neuropathy. Muscle Nerve 1988;11:661-7.
7. Albers JW, Brown MB, Sima AAF, Greene DA. Frequency of median nerve mononeuropathy in patients with mild diabetic neuropathy in the early diabetes intervention trial (EDIT). Muscle Nerve 1996;19:140-6.
8. Hansson S. Segmental median nerve conduction measurements discriminate carpal tunnel syndrome from diabetic polyneuropathy. Muscle Nerve 1995;18:445-53.
9. Johnson EW. Sixteenth annual AAEM Edward H. Lambert lecture. Electrophysiologic aspects of diabetic neuropathies: entrapments. Muscle Nerve 1993;16:127-34.
10. Ozaki I, Baba M, Matsunaga M, Takebe K. Deleterious effect of the carpal tunnel on nerve conduction in diabetic polyneuropathy. Electromyogr Clin Neurophysiol 1988; 28:301-6.
11. Çeliker R, Basgöze O, Bayraktar M. Early detection of neurological involvement in diabetes mellitus. Electromyogr Clin Neurophysiol 1996;36:29-35.
12. Diabetes control and complication trial (DCCT) research group. Effect of intensive diabetes treatment on nerve conduction in the diabetes control and complication trial. Ann Neurol 1995;38:869-80.
13. Eaton RP, Qualls C, Bicknell J, Sibbitt WL Jr, King MK, Griffey RH. Structure-function relationships within peripheral nerves in diabetic neuropathy: the hydration hypotheisis. Diabetologica 1996;39:439-46.
14. Hendriksen PH, Oey PL, Wieneke GH, Bravenober B, van Huffelen AC. Subclinical diabetic polyneuropathy: early detection of involvement of different nerve fibre types. J Neurol Neurosurg Psychiatry 1993;56:509-14.