Facial Nerve Conduction in Diabetic Neuropathy

C. Irkeç[1], B. Nazliel[1], I. Yetkin[2], B. Koçer[1]

[1]Department of Neurology and Endocrinology and [2]Metabolism, Gazi University Faculty of Medicine, Ankara, Turkey

————————

Abstract

Diabetes mellitus (DM) has a severe influence on the nervous system and it is more likely to occur on the nerves of the upper and lower extremities than on the cranial nerves. According to the statistics, the incidence of cranial nerve involvement ranges anywhere from 3% to 14%.

The aim of this study is to perform facial nerve conduction studies in diabetic patients with peripheral neuropathy, confirmed by electrophysiological methods, to determine the frequency of affection of a cranial nerve conduction in a neuropathy which mainly occurs in a distal, symmetric fashion.

The study was conducted in a group of 20 diabetics who had electrophysiologically confirmed polyneuropathy. All of the patients had type 2 DM. Sixteen of the patients were receiving insulin therapy and 4 were treated with oral hypoglycaemic agents only. We found prolonged facial nerve distal latency at least on one tested side in 70% of patients. Distal latency and amplitudes of muscle responses to facial nerve stimulation showed a statistically significant difference from controls (p < 0.001).

This study has shown that proximal nerves like cranial nerves are affected in a high proportion of cases in a neuropathy which mainly occurs in a distal symmetric fashion. The facial nerve is one of the most easily accessible nerves in the proximal part of the body (head-face) and makes it suitable for routine evaluation.

We believe this conduction abnormality may give us the chance to classify these neuropathies as more severe than the ones that only have limb conduction abnormalities. Further studies should be performed in order to confirm these findings.

Key words : Diabetes mellitus ; facial conduction time ; polyneuropathy.

It is a well known fact that diabetes mellitus (DM) has a severe influence on the nervous system and it is more likely to occur on the nerves of the upper and lower extremities than on the cranial nerves. According to the statistics, the incidence of cranial nerve involvement ranges anywhere from 3% to % 14 (Mazzotta *et al.*, 1988).

The most common disturbance is an isolated third nerve lesion, the sixth nerve being affected less frequently. It is a well recognized fact that the pupillary innervation is frequently unaffected in diabetic third nerve palsy. The fourth nerve is rarely involved alone, but sometimes in combination. Other cranial nerves may be affected but less frequently than those to the external ocular muscles. Apart from third, fourth and sixth nerves the seventh is involved with the greatest frequency (Thomas *et al.*, 1993).

Electrophysiological testing of cranial nerves in DM has rarely been performed. The aim of this study was to perform facial nerve conduction measurements in diabetic patients with peripheral neuropathy confirmed with electrophysiological methods to determine the frequency of affection of a cranial nerve conduction in a neuropathy which mainly occurs in a distal symmetric fashion.

Materials and methods

The study was conducted in a group of 20 diabetic patients, (16 females, 4 males) in the age range of 45-77 (mean : 63.1 ± 13.4) years. The mean duration of DM was 169 ± 75 months (mean : 12-264). All patients had type 2 DM according to WHO criteria (The Expert Committee, 2000). Sixteen of the diabetic patients were receiving insulin therapy and four were treated with oral hypoglycaemic agents only. The mean glycosylated haemoglobin value was 7.6 ± 2.0% (normal values : 4.4-5.7%).

Diabetic patients were included if they had symptoms or clinical signs of diabetic neuropathy. Physical examination included evaluation of muscle atrophy, tendon reflexes, and sensory deficit. Sensory deficit evaluation included touch, vibration position, pain, aching, numbness, cramps paraesthesia and definable complaints such as restless legs, faintness on standing, frequent episodes of intermittent diarrhea, and hesitancy before micturition.

Patients with earlier cranial nerve lesions, stroke, alcohol abuse, chronic renal failure, clinical or electrophysiological evidence of a hereditary or acquired neuromuscular disease, patients with oedematous limbs which could make recording or stimulation difficult during nerve conduction

Table 1

Facial nerve mean conduction time in normal subjects
and in diabetics

	Normal (n : 15)	Diabetics (n : 21)	< p
Age (mean and range)	61.75 (40-68)	63.1 (45-77)	
Mean duration of DM (months)		169 ± 75	
Conduction time of n.VII (ms/8.0 cm)	3.0 ± 0.3	4.1 ± 0.7	0.001
Mean CMAP amplitude of n. VII (mV)	1.7 ± 0.6	1.0 ± 0.5	0.001

CMAP : compaired muscle action potential

studies and patients treated with drugs recognised as potentially causing neuropathy were excluded.

All tests were performed at least 3 hours after the last insulin injection. Venous blood was taken for glycosylated haemoglobin (HPLS method) at the end of testing.

Nerve conduction studies were performed in a warm room, with extremity skin temperature of 32°C or above, at the side where nerve conduction velocity measurement (NCV) was done. Median motor and sensory nerve conduction velocities were obtained in one upper extremity, posterior tibial, peroneal motor conduction velocities in one lower extremity and sural nerve sensory conduction in both lower extremities were performed by the method described by Oh (1984). Abnormality was defined as slowed velocity or an absence of response in at least two nerves. Only patients with abnormal results were included in the study for the evaluation of distal latency of muscle responses to facial nerve stimulation (DML VII).

For the facial nerve, an active electrode was placed over the midpoint of the lower portion of the orbicularis oculi and a reference electrode was placed above the eyebrow along the same vertical plane of the active electrode. The zygomatic branch of the facial nerve was stimulated anterior and inferior to the tragus of the earlobe with a standard distance of 8 cm. Both sides were tested consecutively. The latency was measured from the stimulus onset to the first deflection of the compound muscle action potential (CMAP). Amplitudes were measured from peak to peak.

Only delays above the average latency ± 3 SD (standard deviation) of the mean of control subjects, or the absence of CMAP was considered abnormal.

The control group consisted of 15 subjects, (12 male, 3 female), age range 40-68 (mean 61.75 ± 6.06) years who were attending the EMG laboratory for non-specific complaints. Subjects with central or peripheral nerve diseases, or those treated with drugs recognised as potentially causing neuropathy were excluded. All subjects had a normal neurological examination.

The statistical evaluations were performed using nonparametric Mann-Whitney test and Pearson and Sperman's correlation coefficient when appropri-

ate. The software used for all statistical evaluations was SPSS 8.0 statistical package program.

Results

Table 1 presents DML VII in normal subjects and in diabetics. In control subjects, DML VII was 3.0 ± 0.3 ms with a range of 2.4-3.4 ms, and was 4.1 ± 0.7 ms with a range of 2.6-5.4 ms in the diabetics. The difference between the two groups was statistically significant ($p < 0.001$). No correlation was found between DML VII and duration of diabetes ($r = 0.24$, $p = 0.888$) and no correlation was found with DML VII and HbAlc values ($r = 0.171$, $p = 0.333$) either.

In control subjects, the mean amplitude of muscle responses to facial nerve stimulation was 1.7 ± 0.6 mV with a range of 0.50-3.80 mV ; it was 1.0 ± 0.5 mV in diabetics with a range of 0.44-2.40 mV. The difference between the two groups was statistically significant ($p < 0.001$). No correlation could be found between amplitudes of muscle responses to facial nerve stimulation and duration of DM ($r = 0.212$, $p = 0.202$), or with HbAlc values ($r = 0.42$, $p = .812$)

No correlation was found between DML VII and sural nerve sensory conduction velocity ($r = 0.258$, $p = 0.148$) or between DML VII and peroneal nerve motor conduction velocity ($r = 0.228$, $p = 0.201$).

Based on these data, criteria were established for abnormal response using the mean latency plus 3 SD of the upper limit of normal : a facial conduction time with a latency greater than 3.9 msec is defined as abnormal. Fourteen (70%) of our patients had an abnormal response at least on one tested side.

Amplitudes of muscle responses to facial nerve stimulation in both groups varied greatly. Therefore, the amplitude of muscle responses to facial nerve stimulation were not used as an index of abnormality.

Six (30%) diabetics demonstrated no abnormalities.

Discussion

The possibility of subclinical involvement of the facial nerve in different generalised neuropathies

was reported in the past by some authors. The degree of involvement depends on the type of neuropathy. The correlation between the degree of involvement of the facial nerve and peripheral nerves varies greatly in different types of neuropathies. The most pronounced prolongation of DML VII was found in demyelinating hereditary sensorimotor neuropathy (HSMN I) (Hausmanowa-Petrusewicz *et al.*, l987).

Neuropathies can occur in mild diabetics of recent onset and may be independent of other types of diabetic complications. There is only a small number of studies on the frequency of clinically apparent cranial nerve lesions associated with diabetes mellitus. Large retrospective series revealed 0.97% incidence of oculomotor and facial nerve palsies in diabetic patients over a 25-year period which was 7.5 fold more frequent than in the non-diabetic control group (Urban *et al.*, 1999).

Johnson and Waylonis (1964) stressed the fact that, even though the conduction of limb nerves were unaffected, subclinical involvement of the facial nerve was present in a group of known diabetics (Johnson *et al.,* 1964 ; Waylonis *et al.,* 1964).

Seventy % of our patients had abnormal facial latencies at least on one tested side. Johnson *et al.*, (1964) demonstrated a highly significant prolongation of DML VII in 59 diabetics, 76% of whom were insulin-dependent. Urban *et al.* (l999) reported that 77.5% of their diabetic patients demonstrated a significant prolongation of DML VII.

Hausmanowa-Petrusewicz *et al.* (l987) found no changes of the distal motor latency of the facial nerve in 22 diabetics, who were not further characterised, but showed only very mild signs of neuropathy. But they stated that the negative results they reported may be due to mild nature of DM in their patients.

This procedure evaluates polyneuropathy, not mononeuropathy, which develops with acute onset possible due to vascular involvement of the facial nerve. In view of length-related involvement of polyneuropathy, facial nerve conduction may be less impaired than limb nerve conduction. Although rarely, some patients with severe lower limb oedema necessitate the use of needle electrodes instead of surface recording techniques, which is hard and sometimes unbearable for the patient. We believe that under these circumstances, this non-invasive conduction abnormality will give us additional diagnosic information.

Our findings indicate that subclinical facial nerve involvement is not unusual in DM, although it is significantly less frequent than the involvement of limb nerves. This study has revealed that the fasial nerve is affected in a high proportion in a

neuropathy which mainly occurs in a distal symmetric fashion. Of course, this test is not a gold standard for the evaluation and confirmation of a neuropathy. But the facial nerve is one of the most easily accessible nerves in the proximal part of the body (head-face) suitable for routine evaluation.

In demonstrating the sensory disturbances of diabetic neuropathy, Thomas and Tomlinson (1993) reported that only in most severe instances of "length-related" progression of the distal symmetric neuropathic forms the vertex of the head may be reached.

So we believe this conduction abnormality may give us the chance to classify these neuropathies as more severe than the ones that only have limb conduction abnormalities. Further studies on this subject with more patients should be performed in order to confirm these findings.

REFERENCES

Hausmanowa-Petrusewicz I., Ryniewicz B., Rowinska-Marcinska K., Emeryk B., Kopec A. The subclinical facial nerve involvement in generalized neuropathies. *Electromyogr Clin Neurophysiol*, 1987, **27** : 229-234.

Johnson E. W., Waylonis G. W. Facial nerve conduction delay in patients with diabetes mellitus. *Arch. Phys Med Rehabil*, 1964, **45** : 131-139.

Mazzotta G., Del Gatto F., Firenze C., Gallai V. The blink reflex in diabetic patients. *Electromyogr Clin Neurophysiol*, 1988, **28** : 291-294.

Oh S. Anatomical guide for common nerve conduction studies. In : *Clinical Electromyography : Nerve conduction studies*. Park Press, Baltimore, Maryland, 1984, pp. 65-85.
and mononeuropathy multiplex in diabetes mellitus. *N Engl J Med*, 1968 : 17-22.

The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the expert committee on the diagnosis and classification of Diabetes Mellitus. *Diabetes Care*, 2000, **23** : s4-s15.

Thomas P. K., Tomlinson D. R. Diabetic and hypoglycemic neuropathy. In : *Peripheral Neuropathy*. Dyck P. J., Thomas P. K. (eds). 3rd ed. Philadelphia, WB Saunders, 1993, pp. l219-1250.

Urban P. P., Forst T., Lenfers M., Koehler J., Connemann B. J., Beyer J. Incidence of subclinical trigeminal and facial nerve involvement in diabetes mellitus. *Electromyogr Clin Neurophysiol*, 1999, **39** : 267-272.

Waylonis G. W., Johnson E. W. Facial nerve conduction delay. *Arch Physi. Med Rehabilit*, 1964, **45** : 539-547.

B. Nazliel,
Sedat Simavi sok no : 17/32,
B blok Çankaya-Ankara (Turkey).