Research Article

Clinical Utility of Facial and Dorsal Sural Nerve Conduction Studies in Patients With Early Stage Type II Diabetes Mellitus

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Summary

Objective: The aim of this study was to perform facial and dorsal sural nerve conduction in early stage diabetic patients with electrophysiological methods to determine the severity and the frequency of affection of cranial and distal nerve conductions.

Results: The mean distal latency of the dorsal sural nerve response was 2.9 ± 0.4 ms (range 1.79- 3.7), the mean amplitude was 8.3 ±3.15 (range 4.1-17.0) and the mean SCV was 32.9 ±3.8 (range 26-40) in our diabetic patients. In control subjects, facial nerve distal motor latency was 2.4± 0.2 ms with a range of 1.8-2.9 ms, and was 2.8± 0.3 ms with a range of 2.2-3.6 ms in the diabetics. Facial nerve distal latency delay was more significant than the dorsal sural nerve latency in the diabetic patients (p <0.001, p <0.01, respectively). Decrement of the dorsal sural amplitudes was significant than decrement of the facial nerve amplitudes in the diabetic patients (p < 0.01, p= 0.8).

Conclusions: The evaluation of dorsal sural nerve conduction and facial nerve distal latency may improve the diagnostic yield and it should therefore be included in the routine evaluation of patients with normal nerve conduction studies in diabetic patients.

Key words: Dorsal sural nerve, facial nerve, Diabetes mellitus, polyneuropathy

Erken Dönem Tip II Diabetes Mellituslu Hastalarda Fasial ve Dorsal Sural Sinir İletim Çalışmalarının Klinik Değeri

Özet

Amaç: Bu çalışmada amaç Tip II Diabetes Mellitus tanısı alan hastalarda erken dönemde kranial ve distal sinir tutulumlarının sinir iletim çalışmaları ile sıklığını ve değerini saptamak.

Materyal ve Metot: Bu çalışmaya 41 sağlıklı ve 41 erken dönem diabet tanısı alan hasta dahil edildi. Tüm hastalara geleneksel motor ve duyu iletim çalışmaları, fasial motor ve dorsal sural duyu iletim çalışmaları yapıldı ve sonuçları karşılaştırıldı.

Sonuçlar: Yapılan elektrofizyolojik değerlendirme sonucu diabetik grubta dorsal sural sinir latANSı 2.9 ± 0.4 ms (1.79- 3.7), amplitüdü 8.3 ±3.15 µV (4.1-17.0 µV) , iletim hızı 32.9 ±3.8 m/s (26-40) saptandı. Fasial sinir distal motor latansı kontrol grubunda 2.4 ± 0.2 ms (1.8-2.9 ms), diabetiklerde 2.8± 0.3 ms (2.2-3.6 ms) idi. Diabetik hasta grubunda fasial sinir distal latansındaki gecikme dorsal sural latansından daha anlamlıydı (p<0.001, p <0.01, sırasıyla). Amplitüdlere göre karşılaştırıldığında ise dorsal sural sinir amplitüdündeki azalmanın diabetik hasta grubunda anlamlı olduğu görülü (p < 0.01, p= 0.8).

Tartışma: Dorsal sural ve fasial sinir iletim çalışmaları diabetik hasta grubunda rutin elektrofizyolojik çalışmalar normalken erken dönem patolojiyi yansıttıları açıından değerlendirilir. Bu nedenle rutin iletim çalışmalarını normal olan hastalarda incelemeye mutlaka fasial sinir ve dorsal sural iletimleri de dahil edilmelidir.

Anahtar Kelimeler: Dorsal sural sinir, fasial sinir, diabetes mellitus, polinöropati
INTRODUCTION
Neuropathy is the most common complication of diabetes mellitus (DM)\(^6\). In the presence of moderate to severe disease, conventional nerve conduction studies (NCS) are generally reliable diagnostic methods for diabetic polyneuropathy\(^16,4\). Subclinical diabetic neuropathy or asymptomatic neuropathy has been defined as the presence of the nerve lesions associated with diabetes mellitus in the absence of abnormal clinical signs and symptoms\(^3\). The most distal sensory fibers in the lower extremities of diabetic patients are generally first to be affected, and routine NCS of sural and superficial peroneal nerves are commonly assessed for polyneuropathy. However, they remain limited in the evaluation of the distal parts of the feet\(^10\). Currently, the dorsal sural nerve is used to diagnose early stage polyneuropathy\(^10,17\). Because the dorsal sural nerve is the most distal and sensory nerve of the foot, it may be affected in the early or subclinical peripheral neuropathy\(^9,11,15\).

Cranial neuropathies usually present as mononeuropathies and are relatively rare. The incidence of cranial nerve involvement has been reported to range from 3% to 14%\(^12\). Diabetic cranial neuropathies usually have an acute onset, affect the oculomotor and facial nerves, and may or may not be accompanied by pain\(^19\).

Clinical utility of NCS of the dorsal sural and facial nerves in the early diagnosis of polyneuropathy has been shown by several studies\(^1,3,10,17\). This study aimed to perform facial and dorsal sural nerve conduction in early stage diabetic patients by electrophysiological methods to determine the degree to which the cranial and distal nerve conduction is affected.

MATERIAL AND METHODS
Forty-one patients (21 male, 20 female; mean age: 47.0 ±7.8 years; age range: 30-62 years) without clinical signs of cranial nerve involvement and 41 healthy controls (15 male, 26 female; mean age: 47.0 ±8.3 years; age range: 30-60 years) were included in the study. The patients had type 2 DM according to WHO criteria\(^5\). Hemoglobin A1c (HbA1c) measurements and electrodiagnostic studies were performed before the treatment.

All the patients had clinical evidence of polyneuropathy with sensory complaints of numbness, paresthesias, and tingling for more than 2 months. Physical examination included evaluation of tendon reflexes, muscle atrophy, and weakness or sensory deficit. Sensory deficit evaluation included pain, touch, aching, vibration, position, numbness, paresthesia, and cramps. The patients with a history of stroke, cranial nerve lesions, chronic renal failure, and alcohol abuse were not included in the study.

Nerve conduction studies were performed using the conventional procedure. All the tests were carried out with limb temperatures greater than 32 °C. Electrodiagnostic tests included motor conduction studies of the tibial, peroneal, median, ulnar, and facial nerves, determination of minimal F-wave latency of the tibial, peroneal, median, and ulnar nerves, sensory and mixed nerve conduction in the sural, median, ulnar and dorsal sural nerves, and needle examination of the gastrocnemius and tibialis anterior muscles. Recordings were made using a Medelec Synergy EMG machine. Ulnar, median, motor and sensory nerve conduction velocities in one upper extremity; peroneal, posterior tibial nerve motor conduction velocities in one lower extremity; and sural and dorsal sural nerve sensory conduction in both lower extremities were performed by the method described\(^13\). The median and ulnar nerve compound muscle action potentials (CMAPs) were recorded from the abductor pollicis brevis and abductor digiti minimi.
muscles, and stimulation was delivered at the wrist and at the elbow. The median and ulnar sensory nerve action potentials (SNAPs) were recorded with disc electrodes from digit II, palm-wrist, and digit V, and stimulation was applied at the wrist. Sural nerve conduction studies were performed recording the SNAP posterior to the lateral malleolus, and stimulation was carried out 8-10 cm proximally in the midcalf. Polyneuropathy was diagnosed on the basis of abnormal nerve conduction studies when abnormalities were found in at least two nerves. The dorsal sural sensory responses were obtained bilaterally with an antidromic technique, using surface electrodes, the stimulation being performed 10 cm proximal to the recording site; the amplitude was measured from peak to peak, and the distal latencies and sensory conduction velocities were determined from the peak latencies (Figure 1 and 2)\(^{(14)}\). For the facial nerve stimulation, 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 standard distance of the 8 cm. The latency was measured from the stimulus onset to the first deflection of the compound muscle action potentials (CMAPs). The amplitudes were measured from peak to peak. Both sides were tested consecutively.

Normative data for electrophysiological studies were derived from 41 healthy volunteers who had no risk factors for neuropathy, radiculopathy and no abnormalities in neurological examination.

The data are represented as mean ± SD (range). The differences between the groups were analyzed by student's t-test. Mann-Whitney rank sum test was performed when the normally test failed. p value below 0.05 was considered statistically significant.

Figure 1: Dorsal sural nerve conduction technique (S, stimulating electrode; R, recording electrode; G, ground electrode)

Figure 2: Dorsal sural SNAP recording from a control subject
RESULTS

Table 1 presents clinical and laboratory results of the diabetic patients. There was no difference between the two groups for gender and age (p= 0.145, p= 0.775). Forty percent of the patients complained of symmetrical painful dysesthesias including burning or lightening pain and numbness in the feet and lower parts of the legs. On neurological examination, 8 patients (19.5%) had reduced pinprick sensitivity and temperature loss. Vibration sensation was abnormal in 5 (12%) patients. Muscle stretch reflexes at the ankles were reduced in 16 (39%), and absent in 6 (14.6 %) patients. Fourteen of the diabetic patients were receiving insulin therapy. Fifteen patients were receiving oral hypoglycaemic agents only, and five patients were treated with insulin and oral hypoglycaemic agents. The mean glycosylated hemoglobin value was 6.6 ±1.8 % (normal values: 4.4-5.7%).

A comparison of the diabetic group with the control group showed abnormal nerve conduction to the sural nerve (reduced amplitude in 15%, prolonged latency in 23.3%, reduced sensory conduction velocity (SCV) in 25%), the median sensory fibers (reduced amplitude in 15%, prolonged latency in 23.3%, reduced SCV in 25%), prolonged median motor latency 18%, the ulnar sensory fibers (reduced amplitude in 5%, reduced SCV in 8%), peroneal motor nerve (prolonged F latency in 10%, reduced NCV in 5%), and prolonged facial motor latency 44%. None of these patients revealed the diagnosis of polyneuropathy criteria. The sixty-four percent of patients were diagnosed as having mild polyneuropathy by conventional conduction studies.

The clinical cranial nerve findings were unremarkable in all the patients. In the control subjects, the distal motor latency of facial nerve (DML VII) was 2.4 ± 0.2 ms with a range of 1.8-2.9 ms, and was 2.8 ± 0.3 ms with a range of 2.2-3.6 ms in the diabetic group. The difference between the two groups was statistically significant (p< 0.000). Table 2 presents facial nerve conduction in the healthy group and in the diabetic group. The DML VII and HbA1c values were correlated (p< 0.000), and the DML VII and disease duration were correlated (p= 0.02).

In the control groups, the mean amplitude of the muscle responses to facial nerve stimulation was 3.1 ± 0.8 mV with a range of 1.6-5.9 mV; in the diabetic group; the mean amplitude was 2.6 ± 0.7 mV with a range of 1.0- 4.1 mV. There was no difference in the amplitudes of the facial nerve between the two groups (r= 0.45, p= 0.69).

In the diabetic patients, the mean peak latency of the dorsal sural nerve response was 2.9 ± 0.4 ms (range 1.79- 3.7); the mean amplitude was 8.3 ± 3.15 µV (range 4.1-17.0 µV), and the mean SCV was 32.9 ± 3.8 m/s (range 26-40 m/s). In the control subjects, the mean peak latency of the dorsal sural nerve response was 2.1 ± 0.3 ms (range 1.79-3.4); the mean amplitude was12 ± 3.5 µV (range 5.2-42 µV), and the mean SCV was 37.5 ± 2.9 m/s (range 27.3-42.9). The difference between the two groups was statistically significant (p< 0.000). The dorsal sural nerve latency and HbA1c values were correlated (p< 0.000), and the dorsal sural nerve latency and disease duration were correlated (p<0.02). In the diabetic patients, the dorsal sural and facial nerve latency and amplitude were correlated (p< 0.000). Facial nerve distal latency delay was more sensitive than the dorsal sural nerve latency in the diabetic patients (p<0.001, p=0.01, respectively). Decrement of the dorsal sural amplitudes was significant than decrement of the facial nerve amplitudes in the diabetic patients (p< 0.01, p=0.8).
**Table 1:** Clinical and laboratory results of the diabetic patients

<table>
<thead>
<tr>
<th></th>
<th>Diabetics</th>
<th>Controls</th>
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</thead>
<tbody>
<tr>
<td>Gender (M/F)</td>
<td>21/20</td>
<td>15/26</td>
</tr>
<tr>
<td>Age (years), median (range)</td>
<td>47.0 ± 7.8 years</td>
<td>47.0 ± 8.3 years</td>
</tr>
<tr>
<td>Diabetes duration (months)</td>
<td>24±32 months</td>
<td></td>
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<tr>
<td>Neurological findings, n (%)</td>
<td></td>
<td></td>
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<tr>
<td>Abnormal pinprick sensitivity and temperature loss</td>
<td>8 (19.5 %)</td>
<td></td>
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<tr>
<td>Abnormal vibration or proprioception</td>
<td>5 (12%)</td>
<td></td>
</tr>
<tr>
<td>Loss of deep tendon reflexes</td>
<td>22 (53.6 %)</td>
<td></td>
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<tr>
<td>HbA1c</td>
<td>6.6 ±1.8%</td>
<td>4.4-5.7%</td>
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**Table 2:** Results of the dorsal sural and the facial nerve conduction studies in diabetic patients and controls.

<table>
<thead>
<tr>
<th></th>
<th>Diabetics</th>
<th>Controls</th>
<th>P</th>
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<tbody>
<tr>
<td><strong>Dorsal sural nerve</strong></td>
<td>N=41</td>
<td>N=41</td>
<td></td>
</tr>
<tr>
<td>Latency</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Mean ± SD</td>
<td>2.9 ± 0.4 ms</td>
<td>2.1± 0.3 ms</td>
<td>p&lt; 0.000</td>
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<tr>
<td>range</td>
<td>1.79-3.72</td>
<td>1.79-3.4</td>
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<tr>
<td>Amplitude (µV)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>8.3 ± 3.15 µV</td>
<td>12±3.5 µV</td>
<td>p&lt; 0.000</td>
</tr>
<tr>
<td>range</td>
<td>4.1-17.0</td>
<td>5.2-42.0</td>
<td></td>
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<tr>
<td>Peak NCV (m/s)</td>
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<tr>
<td>Mean ± SD</td>
<td>32.9 ±3.8 m/s</td>
<td>37.5± 2.9 m/s</td>
<td>p&lt; 0.000</td>
</tr>
<tr>
<td>range</td>
<td>26.0-40.0</td>
<td>27.3-42.9</td>
<td></td>
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<tr>
<td><strong>Facial nerve</strong></td>
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<tr>
<td>Latency</td>
<td></td>
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<tr>
<td>Mean ± SD</td>
<td>2.8± 0.3 ms</td>
<td>2.4± 0.2 ms</td>
<td>p&lt; 0.000</td>
</tr>
<tr>
<td>range</td>
<td>2.2-3.6</td>
<td>1.8-2.9</td>
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<tr>
<td>Amplitude (µV)</td>
<td></td>
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<tr>
<td>Mean ± SD</td>
<td>2.6±0.7 mV</td>
<td>3.1±0.8 mV</td>
<td>p= 0.69</td>
</tr>
<tr>
<td>range</td>
<td>1.0-4.1</td>
<td>1.6-5.9</td>
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DISCUSSION

The main aim of this study was to investigate the clinical utility of facial and dorsal sural NCS in the detection of early stages of the diabetic polyneuropathy. In the early stages of the diabetic polyneuropathy, many patients may have normal routine nerve conduction studies. Sural nerve conduction study is a routine procedure for the diagnosis of polyneuropathy. However, by using this nerve, the most distal fibers in the lower extremities cannot be evaluated\(^{(10)}\). Dorsal sural nerve conduction study was first performed by Burke et al. as an alternative to sural nerve conduction study\(^{(2)}\). Our study evaluated the value of the dorsal sural, and facial nerves in the diagnosis of early polyneuropathy in diabetic patients with normal routine nerve conduction studies. According to a study by Lee et al. in 1992, electrodiagnostic evaluation of the distal sural nerve was easily performed\(^{(11)}\).

Killian et al. reported that bilateral, but not unilateral, absence of the dorsal sural nerve action potential suggested the presence of distal sensory neuropathy\(^{(10)}\). In their study on diabetic children with no sensory signs or symptoms of peripheral neuropathy, Turgut et al. determined longer latency and slow conduction velocity in the dorsal sural nerve\(^{(17)}\). In the study by Balci et al., low SNAP amplitude was determined in adult diabetic patients with clinical symptoms and signs of neuropathy\(^{(1)}\).

Thus, dorsal sural nerve conduction study can evaluate the most distal segments of the extremities and can be considered an alternative method for the diagnosis of polyneuropathy in the early stage of diabetes mellitus\(^{(10,17)}\).

Patients with mild diabetes of recent onset may develop neuropathies, which may be independent of other types of diabetic complications. Electrophysiological evaluation of cranial nerves in diabetes mellitus is a rarely used procedure. Literature reveals only a small number of studies on the frequency cranial nerve lesions associated with diabetes mellitus. In diabetics, cranial nerve palsies most frequently involve the external ocular muscles. In the study by Waylonis and Johnson, although the nerve conduction of limb nerves was unaffected, a group of known diabetics suffered subclinical involvement of the facial nerve\(^{(20)}\). In a large retrospective series studied by Urban et al., the incidence of oculomotor and facial nerve palsies in diabetic patients over a 25-year period was 0.97\%, which was 7.5 fold more frequent than in the nondiabetic control group\(^{(18)}\). The incidence of peripheral facial palsy in diabetes has been reported to be 0.45\%\(^{(19)}\).

Forty four percent and twenty percent of our patients had abnormal facial and dorsal sural latencies, respectively, at least on one tested side. In 70\% of their patients, Irkeç et al found a significantly prolonged DML VII, while Waylonis et al reported that 59 diabetic patients had significantly prolonged DML VII\(^{(8,20)}\). The rate of prolonged DML VII was 17.5\% in the study by Urban et al\(^{(18)}\). On the other hand, Hausmonova – Petrusiewicz et al reported no changes of the distal motor latency of the facial nerve in 22 diabetics\(^{(7)}\).

In this study, dorsal sural nerve conduction and facial nerve distal latencies were the most sensitive methods for detection of early stage polyneuropathy in diabetes without abnormal routine nerve conduction studies. Even the dorsal sural nerve latency delay and the amplitude decrement were important for the evaluation of the neuropathy. The facial nerve distal latency delay was more sensitive according to our results. This evaluation is not a unique standard for the evaluation of a neuropathy. Nevertheless, the dorsal sural nerve and facial nerve are easily accessible nerves in proximal and distal part of the body, which renders them suitable for routine evaluation.
In conclusion, evaluation of dorsal sural nerve conduction and facial nerve distal latency may improve the diagnostic yield and thus, it should be included in the routine evaluation of diabetic patients with normal nerve conduction studies.

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