Clinical diagnosis of distal diabetic polyneuropathy using neurological examination scores: correlation with nerve conduction studies

Shereen R. Kamel^a, Mona Hamdy^a, Hanaa A.S. Abo Omar^a, Amal Kamal^b, Lamia H. Ali^c, Ahmed H. Abd Elkarim^a

Departments of aRheumatology and Rehabilitation, ^bInternal Medicine, ^cClinical Pathology, Minia University, Minya, Egypt

Correspondence to Shereen R. Kamel, MD, Minia University Hospital, Minya 6111, Egypt Tel: 01065800025; fax: +20862342503; e-mail: sh_rr70@yahoo.com

Received 09 March 2015 Accepted 09 July 2015

Egyptian Rheumatology & Rehabilitation 2015, 42:128–136

Aim

The aim of this study was to diagnose diabetic sensorimotor polyneuropathy using neurological examination scores and to correlate the findings with nerve conduction studies (NCS). **Patients and methods**

Thirty patients with type 2 diabetes were included in the study. Detection and grading of neuropathy were carried out based on the Diabetic Neuropathy Symptom (DNS) Score, modified Neuropathy Symptom Score (NSS), Diabetic Neuropathy Examination (DNE), and modified Neuropathy Disability Score (NDS). For the NCS, amplitudes, velocities, and latencies of seven nerves — that is, four motor (median, ulnar, tibial, and common peroneal) and three sensory (median, ulnar, and sural) nerves — were recorded. If the patient had two or more abnormal findings in any of these nerves, the patient was diagnosed as having peripheral sensorimotor neuropathy. Thereafter, the sensitivity, specificity, and diagnostic efficacy of each neurological score were recorded taking NCS as the gold standard.

Results

Diabetic sensorimotor polyneuropathy was diagnosed clinically and electrophysiologically in 17 patients (56.7%). However, there were nine cases (30%) of subclinical neuropathy. Neurological examination scores were significantly correlated with each other and with individual variables of NCS and the nerve conduction sum score. Taking the NCS as gold standard, DNS, modified NSS, DNE, and modified NDS had 65.4, 61.5, 30.8, and 61.5% sensitivity and 100, 75, 100, and 100% specificity, respectively. Their diagnostic efficacies were 70, 63.3, 40, and 66.7%, respectively.

Conclusion

Neurological examination scores can detect and grade neuropathy in the majority of cases. However, NCS was accurate for detection of diabetic sensorimotor polyneuropathy, especially for the subclinical neuropathies.

Keywords:

distal diabetic polyneuropathy, nerve conduction studies, neurological examination scores

Egypt Rheumatol Rehabil 42:128–136 © 2015 Egyptian Society for Rheumatology and Rehabilitation 1110-161X

Introduction

Distal symmetric polyneuropathy or diabetic sensorimotor polyneuropathy (DSPN) is a frequent complication of both type 1 and type 2 diabetes [1]. DSPN encompasses a group of clinical and subclinical syndromes with varied etiologies and clinical and laboratorial manifestations, defined by the progressive diffuse or focal degeneration of peripheral somatic and autonomic nerve fibers [2].

Consensus definitions for DSPN consistently recommend a combination of neuropathic symptoms and signs, in addition to specific abnormalities in nerve conduction studies (NCS), as criteria for diagnosis [3,4]. The absence of symptoms should not be equated with the absence of neuropathy; up to 50% of patients with diabetic polyneuropathy may be asymptomatic but are still at risk of foot ulcers. Therefore, monitoring for

neuropathy should be a regular part of the clinical care of patients with diabetes [5].

Several clinical scores have been developed to assess diabetic neuropathy, including the Diabetic Neuropathy Symptom (DNS) score [6], Neuropathy Symptom Score (NSS), Diabetic Neuropathy Examination (DNE), and Neuropathy Disability Score (NDS) [7].

Glycemic control may be considered as an auxiliary measure for predicting chronic diabetes mellitus complications, including DSPN. The clinical

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

The aim of the present study was to diagnose DSPN clinically in patients with type 2 diabetes using neurological examination scores, which are easy to perform, and to correlate these scores with electrophysiological measurements.

Patients and methods Study population

Thirty patients diagnosed with type 2 diabetes (according to the American Diabetes Association criteria) [9] were included in the study. Patients were recruited from the Rheumatology and Rehabilitation department and diabetes outpatient clinic of Internal Medicine department. Ten healthy age-matched and sex-matched participants served as the control group for electrodiagnostic studies. Informed consent was obtained from all participants in the study. The study was approved by the ethics committee of the Faculty of Medicine.

Patients having any evidence of autoimmune diseases (such as rheumatoid arthritis, systemic lupus erythematosus, and scleroderma), cervical and/or lumbar disc lesions, neuropathies of other etiologies (such as heredofamilial polyneuropathy, exposure to neurotoxic drugs, infections, or neuropathies due to other causes or renal failure) were excluded.

Neurological examination scores

We selected four scores on the basis of ease of performance and common use. The scores were DNS, modified NSS, DNE, and modified NDS [6,7].

Diabetic Neuropathy Symptom score

All participants were questioned as regards the presence of symptoms, either positive or negative, suggesting the presence of neuropathy.

The questions were answered with a 'yes' (positive: one point) if a symptom had occurred several times a week during the last 2 weeks, or with a 'no' (negative: no point) if it did not. The patients were questioned as regards the presence of following symptoms:

- (1) Symptoms of unsteadiness when walking.
- (2) Burning, aching pain, or tenderness in legs or feet.
- (3) Pricking sensations on legs and feet.
- (4) Regions of numbress on legs or feet.

Maximum score: 4 points; 0 points, polyneuropathy (PNP) absent; 1–4 points, PNP present [6].

Modified Neuropathy Symptom Score

Patients were questioned about the presence or absence of numbness, abnormal hot or cold sensations, tingling sensations, burning pain, irritation from bed clothes in the lower legs and feet, and nocturnal exacerbation of muscular cramps and whether maneuvers could reduce the symptoms. One point for the presence of each of these symptoms was assigned. For the first five symptoms one extra point was added if nocturnal exacerbation was present. The maximum score is 10 points. A score of more than 1 point is defined as positive for PNP [10] (Table 1).

Diabetic Neuropathy Examination score

The score contains two items for muscle strength, one pertaining to reflexes and five pertaining to sensation (eight total items). Each item is scored from 0 to 2 (0 is normal and 2 severely disturbed). The maximum score is 16 points. A score greater than 3 points is defined as positive for PNP [11] (Table 2).

Modified Neuropathy Disability Score

The modified NDS can be easily performed in the clinical setting and takes only a minute or two to complete and provides an assessment of the risk for neuropathic ulceration. The score is based on vibration perception, pin-prick sensation, temperature perception, and ankle

Table 1 Modified NDS scor	е
---------------------------	---

Symptomatology: foot/lower leg	Yes	No
Burning sensation	2	0
Numbness	2	0
Paresthesia	2	0
Feeling of weakness (fatigue, exhaustion)	1	0
Cramps	1	0
Pain	1	0
Localization		
Feet	2	
Lower leg	1	
Elsewhere	0	
Exacerbation		
Present at night	2	
Present during day and night	1	
Only present during the day	0	
Patient is awakened from sleep by the symptoms	1 ac	bb
Symptom improvement		
Walking	2	
Standing	1	
Sitting or lying down	0	
	Tota	l scor

In each point column, the maximum score can be given only once; 3–4, mild symptoms; 5–6, moderate symptoms; 7–10, severe neuropathic symptoms.

(Achilles) reflexes. The maximum deficit score is 10, which would indicate complete loss of sensation to all sensory modalities and absent reflexes. A score of 6 or more has been found to indicate an increased risk for foot ulceration [7,12] (Table 3).

The patients were diagnosed as having clinically detectable neuropathy (group I) if DNS was 1 or more, modified NSS was greater than 1, or DNE was greater than 3. Undetectable neuropathy (group II) was

Table	2	DNE	score
-------	---	-----	-------

Muscle strength
Quadriceps femoris: extension of the knee
Tibialis anterior: dorsiflexion of the foot
Reflex
Ankle reflex
Sensation: index finger
Sensitivity to pinpricks
Sensation: big toe
Sensitivity to pinpricks
Sensitivity to touch
Vibration perception
Sensitivity to joint position
Only the right leg and foot are tested
If the right leg is amputated, then the left leg is tested
Scoring from 0 to 2
0 = Normal
1 = Mild/moderate deficit
Muscle strength: MRC scale 3-4
Reflex: decreased but present
Sensation: decreased but present
2 = severely disturbed/absent
Muscle strength: MRC scale 0-2
Reflex: absent
Sensation: absent

Table 3 Modified NDS

Components	Assessment	Right	Left
Vibration perception threshold: 128-Hz tuning fork placed on the apex of the big toe: normal = can distinguish the presence or absence of vibration	Normal = 0; Abnormal = 1		
Temperature perception on the dorsum of the foot: Use a tuning fork with a beaker of ice/warm water			
Pin-prick: Apply a pin proximal to the big toe nail just enough to deform the skin; Trial pair = sharp, blunt; Normal = can distinguish sharp and blunt			
Achilles reflex	Present = 0; Present with reinforcement = 1; Absent = 2		
	Modified neuropathy disability score total out of 10		

diagnosed if DNS was less than 1, modified NSS was 1 or less, or DNE was 3 or less. Modified NSS and modified NDS were used to quantify the severity of the neuropathy [13].

Nerve conduction studies

NCS were performed to all patients and controls at a room temperature of 23 ± 2°C. Nihon Kohden MEB-9400K NeuropackS1equipment (Nihon Kohden Corporation 1-31-4 Nishiochial, Shinjuku, Tokyo 161-8560, Japan) was used by the same electromyographer. Motor and sensory distal latencies, amplitudes, and nerve conduction velocities were measured in the upper and lower limb nerves. The simplified NCS protocol was followed to record the NCS of the patients [14].

Five nerves were tested (median, ulnar, tibial, common peroneal, and sural nerves). The NCS were used to identify normal or affected nerves. The patients were diagnosed as having polyneuropathy if the value of two or more parameters was abnormal in one nerve, or one parameter was abnormal in any two nerves. Amplitudes, velocities, and latencies of seven nerves — that is, four motor (median, ulnar, tibial, and common peroneal) and three sensory (median, ulnar, and sural) nerves — were recorded. An overall Nerve Conduction Sum score was defined as the number of these five nerves with an abnormal conduction velocity, ranging from 0 (all normal) to 7 (all abnormal) [15].

Grouping of patients on the basis of neurological score and nerve conduction studies

For each neurological score, patients were divided into four groups: true positive, false positive, false negative, and true negative. If neuropathy was present by both clinical examination and NCS, the patient was included in the true-positive group, in the false-positive group if it was present on clinical examination but was absent on NCS, in the false-negative group if it was present on NCS but was absent on clinical examination, and in the true-negative group if it was absent on both testing methods. The sensitivity and specificity of each score were calculated taking NCS as the gold standard. Data presented in Table 8 were used to calculate test performance characteristics. Diagnositic efficacy of each test was derived from the same data.

Statistical analysis

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) 16.0. Descriptive statistics were carried out using number and percentage, as well as mean and SD. The ANOVA test was used to compare the difference between more than two group means in interval and ordinal variables. Correlations Sensitivity(%) = patients with true-positive neuropathy/ patients with true-positive neuropathy + patients with false-negative neuropathy ×100.

Specificity(%) = patients with true-negative neuropathy/ patients with false-positive neuropathy + patients with true-negative neuropathy ×100.

Positive predictive value (%) = patients with true-positive neuropathy/patients with true-positive neuropathy + patients with false-positive neuropathy ×100.

Negative predictive value (%) = patients with truenegative neuropathy/patients with false-positive neuropathy + patients with true-negative neuropathy ×100.

Diagnostic efficacy (%) = patients with true-positive neuropathy + patients with true-negative neuropathy/ all patients ×100.

Results

The patient characteristics are presented in Table 4. The ages of the studied patients ranged from 32 to

Table 4	Patient	characteristics
---------	---------	-----------------

variables	
Sex	
Male	2 (6.7)
Female	28 (93.3)
Age (years)	50.6 ± 11.8
Duration of diabetes (years)	4.6 ± 4.7
Fasting plasma glucose (mg/dl)	146.4 ± 60.9
2 h postprandial glucose (mg/dl)	240 ± 86.6
Glycated hemoglobin (HbA1c) (%)	9.9 ± 2.8
Mode of treatment	
Oral hypoglycemic drugs	20 (66.7)
Insulin	10 (33.3)

Data are presented as means \pm SD or n (%).

Table 5 Neurological examination	scores for the studied patients
----------------------------------	---------------------------------

70 years, and the duration of diabetes ranged from 0.5 to 15 years.

Results of neurological examination scores for all patients

The scores (mean \pm SD) of all patients, the percentage of patients with abnormal scores, and grading of neuropathic symptoms and deficits are presented in Table 5. Out of 30 patients, nine (30%) had a modified NDS of 6 or more, which indicates an increased risk for foot ulceration.

There were significant correlations between the DNS and modified NSS, DNE, and modified NDS (r = 0.81, P < 0.001; r = 0.67, P < 0.001; r = 0.7, P < 0.001, respectively), as well as between DNE and modified NSS, and modified NDS (r = 0.58, P = 0.001; r = 0.84, P < 0.001, respectively). In addition, a significant correlation was found between modified NSS and modified NDS (r = 0.64, P < 0.001).

There was a significant correlation between DNS, modified NSS, DNE, modified NDS, and disease duration (r = 0.65, P < 0.001; r = 0.6, P < 0.001; r = 0.59, P < 0.001; r = 0.71, P < 0.001, respectively), as well as between the DNE and HbA1c (r = 0.41, P = 0.008). There was no significant correlation between used scores and patient's age.

Severity of neurological symptoms and deficits, graded using modified NSS and modified NDS, was significantly correlated with the disease duration (r = 0.59, P < 0.001; r = 0.66, P < 0.001, respectively), as well as with HbA1c (r = 0.48, P = 0.002 for both).

Results of nerve conduction studies

Comparison between patients with clinically detectable and undetectable neuropathy and the control group showed significant difference between the three groups as regards all parameters of motor NCS except for

Scores	Range	Mean ± SD	Classified as polyneuropathy	l	Patients [N (%)]	
DNS	0–4	1.6 ± 1.5	DNS ≥1point	17 (56.7%)		
Modified NSS	0—9	3.4 ± 3.2	NSS >1point	17 (56.7)	Mild	2 (11.8)
					Moderate	10 (58.8)
					Severe	5 (29.4)
DNE	0–10	2.4 ± 3.1	DNE >3 points	8 (26.7)		
Modified NDS	0–10	3.4 ± 3.7	NDS >1 point	16 (53.3)	Mild	7 (43.8)
					Moderate	4 (25)
					Severe	5 (31.2)

Modified NSS: 3–4, mild symptoms; 5–6, moderate symptoms; 7–10, severe neuropathic symptoms; Modified NDS: 3–5, mild neuropathic deficits; 6–8, moderate neuropathic deficits; 9–10, severe neuropathic deficits; DNE, diabetic neuropathy examination score; DNS, diabetic neuropathy symptom score; NDS, neuropathy disability score; NSS, neuropathy symptom score.

ulnar conduction velocity and tibial and peroneal distal latencies (Table 6).

Similarly, comparison between patients with clinically detectable and undetectable neuropathy and the control group showed significant difference between the three groups as regards all parameters of sensory NCS (Table 7).

Comparison of neurological scores with nerve conduction studies

The comparison of neurological scores with NCS is presented in Table 8. According to the results of NCS, 26 patients (86.7%) had polyneuropathy. However, according to the neurological examination scores, 17 patients (56.7%) had polyneuropathy, which means that there were nine cases (30%) of subclinical polyneuropathy.

DNS was found to be the most sensitive test (65.4%), and DNS, DNE, and modified NDS had equal specificity (100%). DNS and modified NDS had a better diagnostic efficacy (70 and 66.7%, respectively) as shown in Table 9.

Relation between clinical scores and different components of nerve conduction studies

DNS significantly correlated with median, ulnar, and tibial motor latencies and with median, ulnar, and sural sensory latencies (r = 0.42, P = 0.007; r = 0.38, P = 0.01; r = 0.38, P = 0.01; r = 0.46, P = 0.003; r = 0.47, P = 0.002; r = 0.52, P = 0.001, respectively).

Modified NSS significantly correlated with median and ulnar motor latencies and with median, ulnar, and sural sensory latencies (r = 0.35, P = 0.02; r = 0.45, P = 0.004; r = 0.53, P < 0.001; r = 0.35, P = 0.02; r = 0.55, P < 0.001, respectively).

DNE significantly correlated with median and ulnar motor latencies, ulnar amplitude, tibial motor conduction velocity and motor latency, peroneal motor conduction velocity and sensory latency, and sural sensory latency (r = 0.39, P = 0.01; r = 0.31, P = 0.04; r = 0.36, P = 0.02; r = 0.38, P = 0.01; r = 0.39, P = 0.00; P = 0.00;

Table 6 Comparison between patients with clinically detectable and undetectable neuropathy and the control group as regards parameters of motor nerve conduction studies

Nerve	Parameters	Clinically detectable neuropathy $(N = 17 \text{ patients})$	Clinically undetectable neuropathy $(N = 13 \text{ patients})$	Control group (N = 10)	F	Р
Median	MCV (m/s)	46.5 ± 6.2	51.9 ± 6.5	58 ± 7.1	9.9	<0.001*
	DML (ms)	4.3 ± 1.2	3.9 ± 1	3 ± 0.5	4.5	0.02*
	Amp. (mV)	3.5 ± 1.4	3.7 ± 1.9	6.3 ± 1.3	10.7	<0.001*
Ulnar	MCV (m/s)	55.1 ± 11	59.6 ± 7.3	58 ± 7.1	0.9	0.4
	DML (ms)	3.7 ± 0.8	3.1 ± 0.8	3 ± 0.5	3.7	0.04*
	Amp. (mV)	3.4 ± 0.9	4.4 ± 1	6.3 ± 1.3	22.6	<0.001*
Tibial	MCV (m/s)	43.4 ± 12.4	47.2 ± 10.4	56.7 ± 6.8	4.9	0.01*
	DML (ms)	5.1 ± 1.4	4.6 ± 1	3.9 ± 0.6	3	0.06
	Amp. (mV)	2.5 ± 2.2	2.8 ± 1.3	6 ± 0.7	15.3	<0.001*
Peroneal	MCV (m/s)	42.5 ± 12	45.7 ± 16.8	6.8 ± 2.1	3.9	0.03*
	DML (ms)	6 ± 2.1	7.8 ± 9.9	3.9 ± 0.6	1.3	0.3
	Amp. (mV)	1.4 ± 1.7	1.2 ± 1.3	6 ± 0.7	42.4	<0.001*

By one-way analysis of variance test; Data are presented as means ± SD; Amp, amplitude; DML, distal motor latency; MCV, motor conduction velocity; *Significant *P*-value < 0.05.

Table 7 Comparison between patients with clinically detectable and undetectable neuropathy and the control group as regards	
parameters of sensory nerve conduction studies	

Nerve	Parameter	Clinically detectable neuropathy $(N = 17 \text{ patients})$	Clinically undetectable neuropathy $(N = 13 \text{ patients})$	Control group (N = 10)	F	Р
Median	SCV (m/s)	37.2 ± 9.8	50.9 ± 8.2	53.5 ± 3.4	16.5	<0.001*
	DSL (ms)	4.2 ± 1.3	3.4 ± 1.5	2.6 ± 0.5	5.4	0.009*
	Amp. (μV)	6.5 ± 6.2	13.2 ± 5.9	21 ± 3.7	21.2	<0.001*
Ulnar	SCV (m/s)	39.9 ± 7.3	50.7 ± 12.5	53.5 ± 3.4	9.6	<0.001*
	DSL (ms)	3.9 ± 1.04	3.5 ± 0.9	2.6 ± 0.5	6.8	0.003*
	Amp. (μV)	6.8 ± 4.6	14.3 ± 7.3	21 ± 3.7	22.2	<0.001*
Sural	SCV (m/s)	33.2 ± 5.3	42.8 ± 4.7	50.5 ± 4.4	40.7	<0.001*
	DSL (ms)	5.5 ± 1.01	4.6 ± 0.9	4 ± 1	7.7	0.002*
	Amp. (μV)	5.2 ± 3.9	9.4 ± 2.3	17.5 ± 2	50.9	<0.001*

By one-way ANOVA test; Data are presented as means ± SD; Amp, amplitude; DSL, distal sensory latency; SCV, sensory conduction velocity; *Significant *P*-value < 0.05.

Modified NDS significantly correlated with median, ulnar, and tibial motor latencies and with median, ulnar, and sural sensory latencies (r = 0.49, P = 0.001; r = 0.45, P = 0.004; r = 0.44, P = 0.005; r = 0.44, P = 0.004; r = 0.47, P = 0.002; r = 0.44, P = 0.004, respectively).

Relation between clinical scores and nerve conduction sum score

In all patients, the nerve conduction sum score ranged from 1 to 7 according to the involved nerves.

There were significant correlations of the DNS, modified NSS, DNE, and modified NDS with the nerve conduction sum score (r = 0.71, P < 0.001; r = 0.58, P < 0.001; r = 0.66, P < 0.001; r = 0.73, P < 0.001, respectively) (Fig. 1). It can be interpreted that the more the neurological scores had deteriorated, the higher were the chances of further deranged nerve conduction sum score.

Relation between disease duration, HbA1c and different components of nerve conduction studies

Disease duration significantly correlated with median, ulnar, and tibial motor latencies (r = 0.43, P = 0.006;

Table 8 Comparison of neurological scores with nerve conduction studies

Scores findings	Neuropathy on NCS		
	Present	Absent	
Neuropathy on DNS			
Present	17	0	
Absent	9	4	
Neuropathy on modified NSS			
Present	16	1	
Absent	10	3	
Neuropathy on DNE			
Present	8	0	
Absent	18	4	
Neuropathy on modified NDS			
Present	16	0	
Absent	10	4	

DNE, diabetic neuropathy examination score; DNS, diabetic

neuropathy symptom score; NCS, nerve conduction studies;

NDS, neuropathy disability score; NSS, neuropathy symptom score.

 Table 9 Test performance characteristics of the neurological scores as compared with nerve conduction studies

Characteristics	DNS	Modified NSS	DNE	Modified NDS
Sensitivity (%)	65.4	61.5	30.8	61.5
Specificity (%)	100	75	100	100
Positive predictive value (%)	100	94.1	100	100
Negative predictive value (%)	30.8	23.1	18.2	28.6
Diagnostic efficacy (%)	70	63.3	40	66.7

DNE, diabetic neuropathy examination score; DNS, diabetic neuropathy symptom score; NDS, neuropathy disability score; NSS, neuropathy symptom score.

r = 0.45, P = 0.004; 0.42, P = 0.007, respectively), as well as with median and ulnar sensory latencies (r = 0.36, P = 0.02; r = 0.39, P = 0.01, respectively).

HbA1c significantly correlated with tibial motor latency and median sensory latency (r = 0.48, P = 0.002; r = 0.36, P = 0.02, respectively).

Both disease duration and HbA1c significantly correlated with the nerve conduction sum score (r = 0.59, P < 0.001; r = 0.32, P = 0.04, respectively) (Figs 2 and 3).

Discussion

The disease process of diabetes causes alterations in the normal nerve functions, which can be reflected either when performing neurological examination, or during electrophysiological testing of the patient. The neurological scores and the electrophysiological studies are used for the diagnosis of the sensorimotor neuropathy. The relations between physiology and pathophysiology emphasize the close interdependence between electrophysiological studies and clinical findings [16].

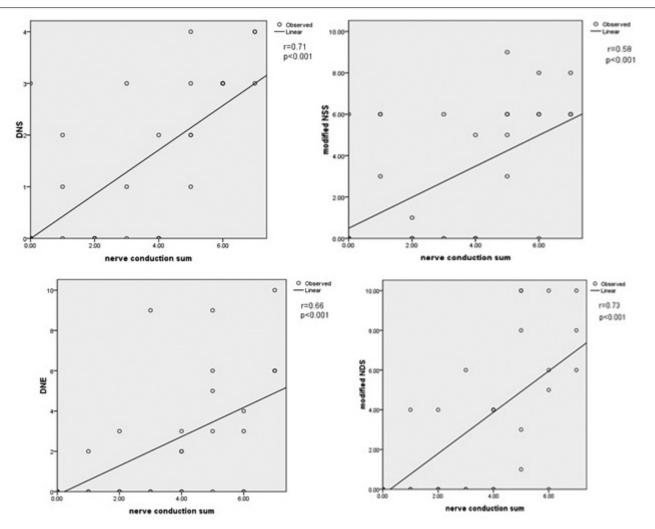
In the present study, we selected the four neurological scoring systems (DNS, modified NSS, DNE, and modified NDS), which were common and easy to perform, and compared them with NCS, which also has similar advantages. Out of 30 patients with type 2 DM, DSPN was diagnosed clinically (using neurological examination scores) and electrophysiologically (using NCS) in 17 patients (56.7%). However, there were nine cases (30%) of subclinical neuropathy. Subclinical neuropathy indicates the state of electrophysiologically verified neuropathy with the absence of subjective and objective neurological signs. It occurs in about 20% of patients with diabetes [17].

Studies on prevalence of neuropathy in type 2 diabetes had widely differing results, varying from 15 to 50%. The wide variability was attributed to differences in patient sample, diagnostic methods, and criteria adopted for diagnosis [18,19]. Studies that used NCS as a diagnostic marker also reported higher prevalence of neuropathy [20].

The higher prevalence of neuropathy in the present study may be due to the use of NCS for diagnosis of neuropathy, which is a more sensitive method.

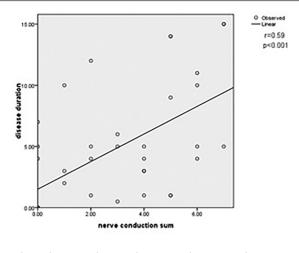
In diabetic patients, correlations between various neuropathy tests and scores have previously been reported [13,21]. An association between NSS and NDS has been observed [22].

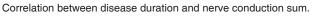




Correlation between neurological examination scores and nerve conduction sum.

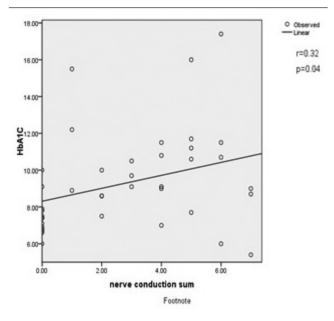
Figure 2





In the present study, the neurological examination scores significantly correlated with each other, which were similar to results of Meijer *et al.* [6]. At the





Correlation between glycosylated hemoglobin (HbA1c) and nerve conduction sum.

same time, the neurological examination scores were significantly correlated with individual variables of NCS. This is in accordance with the study by Meijer *et al.* [23], in which both scores strongly correlated with electrodiagnostic studies . In this study, we added the nerve conduction sum score, which ranged from 1 to 7 based on the involved nerves, and there were significant correlations with DNS, modified NSS, DNE, and modified NDS (r = 0.71, P < 0.001; r = 0.58, P < 0.001; r = 0.66, P < 0.001; r = 0.73, P < 0.001, respectively).

On comparing NCS with neurological examination scores in each group, it was found that NCS detected more cases of neuropathy (86.7%) compared with neurological examination scores (56.7%). The results showed that both clinical tests and NCS have a role in detecting cases of peripheral neuropathy. The NCS, however, is accurate in the detection of neuropathy as NCS is helpful in detecting subclinical neuropathies as well. Similar results have been obtained by most of the studies. A study by Asad et al. [24] supports the fact that subclinical neuropathy can be detected with NCS. They proved that NCS was more accurate in the detection of neuropathy compared with clinical examination, especially in the subclinical group, although the latter also has its role in the detection of neuropathy.

In Pakistan, Niazi *et al.* [25] evaluated diabetic polyneuropathy by performing electrodiagnostic study on 41 patients. Although clinical examination was carried out in detail, no statistical comparison was made between the clinical findings and NCS. However, it was suggested that these studies are capable of diagnosing diabetic neuropathy even before clinical manifestations, which has been proved in our results.

We assessed the sensitivity and specificity of the four scores taking NCS as the gold standard. DNS was found to be the most sensitive test (65.4%), and DNS, DNE, and modified NDS had equal specificity (100%). DNS and modified NDS had a better diagnostic efficacy (70 and 66.7%, respectively). According to Asad et al. [26], NDS was found to be the most sensitive test (92%) and DNE had the highest specificity (81%). NDS and NSS had a better diagnostic efficacy (76.67%), but DNS sensitivity was 64%, which was similar to that reported in our result. This difference may be due to different geographic area and different races being examined. Meijer et al. [6] also assessed the validity of DNS score against NSS. They did not use NCS. Their results as regards sensitivity and specificity were different from ours. They found high correlation between the two testing methods [6].

The results of both neurological scores and NCS in the present study confirmed the previous findings of greater involvement of the peripheral nervous system in diabetic patients with prolonged disease duration and elevated HbA1c [27]. Moreover, the severity of neurological symptoms and deficits was significantly correlated with the disease duration (r = 0.59, P < 0.001; r = 0.66, P < 0.001, respectively), as well as with HbA1c (r = 0.48, P = 0.002 for both).

In conclusion, neurological examination scores can detect and grade neuropathy in majority of cases. However, NCS was accurate for the detection of DSPN, especially subclinical neuropathies. Therefore, we can use each, NCS or neurological examination scores, for detecting and grading diabetic neuropathy and using both gives us a better chance for earlier diagnosis.

Financial support and sponsorship Nil.

Conflicts of interest

There are no conflicts of interest.

References

- Dyck PJ, Lais A, Karnes JL, O'Brien P, Rizza R. Fiber loss is primary and multifocal in sural nerves in diabetic polyneuropathy. Ann Neurol 1986; 19:425–439.
- 2 Greene DA, Sima AA, Stevens MJ, Feldman EL, Lattimer SA. Complications: neuropathy, pathogenetic considerations. Diabetes Care 1992; 15:1902–1925.
- 3 England JD, Gronseth GS, Franklin G, Miller RG, Asbury AK, Carter GT, et al. Distal symmetric polyneuropathy: a definition for clinical research: report of the American Academy of Neurology, the American Association of Electrodiagnostic Medicine, and the American Academy of Physical Medicine and Rehabilitation. Neurology 2005; 64:199–207.
- 4 Dyck PJ, Albers JW, Andersen H, Arezzo JC, Biessels GJ, Bril V, et al. Diabetic polyneuropathies: update on research definition, diagnostic criteria and estimation of severity. Diabetes Metab Res Rev 2011; 27:620–628.
- 5 Barbano R, Hart-Gouleau S, Pennella-Vaughan J, Dworkin RH. Pharmacotherapy of painful diabetic neuropathy. Curr Pain Headache Rep 2003; 7:169–177.
- 6 Meijer JW, Smit AJ, Sonderen EV, Groothoff JW, Eisma WH, Links TP. Symptom scoring systems to diagnose distal polyneuropathy in diabetes: the Diabetic Neuropathy Symptom score. Diabet Med 2002; 19:962–965.
- 7 Dyck PJ. Detection, characterization, and staging of polyneuropathy: assessed in diabetics. Muscle Nerve 1988; 11:21–32.
- International Expert Committee. International Expert committee report on the role of the A1c assay in the diagnosis of diabetes. Diabetes Care 2009; 32:1327–1334.
- 9 American Diabetes Association. Standards of medical care in diabetes-2014. Diabetes Care 2014; 37 (Suppl 1):S14–S80.
- 10 Young MJ, Boulton AJ, MacLeod AF, Williams DR, Sonksen PH. A multicentre study of the prevalence of diabetic peripheral neuropathy in the United Kingdom hospital clinic population. Diabetologia 1993; 36:150–154.
- 11 Meijer JW, van Sonderen E, Blaauwwiekel EE, Smit AJ, Groothoff JW, Eisma WH, Links TP. Diabetic neuropathy examination: a hierarchical scoring system to diagnose distal polyneuropathy in diabetes. Diabetes Care 2000; 23:750–753.
- 12 Boulton A. Management of diabetic peripheral neuropathy. Clin Diabetes 2005; 23:9–15.

136 Egyptian Rheumatology & Rehabilitation

- 13 Dyck PJ, Karnes JL, O'Brien PC, Litchy WJ, Low PA, Melton LJ. 3rd. The Rochester diabetic neuropathy study: reassessment of tests and criteria for diagnosis and staged severity. Neurology 1992; 42:1164–1170.
- 14 Albers JW, Brown MB, Sima AA, Greene DA. Nerve conduction measures in mild diabetic neuropathy in the Early Diabetes Intervention Trial: the effects of age, sex, type of diabetes, disease duration, and anthropometric factors. Tolrestat Study Group for the Early Diabetes Intervention Trial. Neurology 1996; 46:85–91.
- 15 American Diabetes Association, American Academy of Neurology. Report and recommendations of the San Antonio Conference on Diabetic Neuropathy (Consensus Statement). Diabetes Care 1988; 11:592–597.
- 16 Krarup C. Nerve conduction studies in selected peripheral nerve disorders. Curr Opin Neurol 2002; 15:579–593.
- 17 Kronenberg HM, Melmed S, Polonsky KS, Larsen PR, (editors). Williams textbook of endocrinology. chapter 2. 11th ed. London: Saunders; 2007.
- 18 Franklin GM, Shetterly SM, Cohen JA, Baxter J, Hamman RF. Risk factors for distal symmetric neuropathy in NIDDM. The San Luis Valley Diabetes Study. Diabetes Care 1994; 17:1172–1177.
- 19 Mythili A, Kumar KD, Subrahmanyam KA, Venkateswarlu K, Butchi RG. A Comparative study of examination scores and quantitative sensory testing in diagnosis of diabetic polyneuropathy. Int J Diabetes Dev Ctries 2010; 30:43–48.
- 20 Kumar S, Ashe HA, Parnell LN, Fernando DJ, Tsigos C, Young RJ, *et al.* The prevalence of foot ulceration and its correlates in type 2 diabetic patients: a population-based study. Diabet Med 1994; 11:480–484.

- 21 Dyck PJ, Bushek W, Spring EM, Karnes JL, Litchy WJ, O'Brien PC, Service FJ. Vibratory and cooling detection thresholds compared with other tests in diagnosing and staging diabetic neuropathy. Diabetes Care 1987; 10:432–440.
- 22 Dyck PJ, Karnes JL, Daube J, O'Brien P, Service FJ. Clinical and neuropathological criteria for the diagnosis and staging of diabetic polyneuropathy. Brain 1985; 108 (Pt 4):861–880.
- 23 Meijer JW, Bosma E, Lefrandt JD, Links TP, Smit AJ, Stewart RE, et al. Clinical diagnosis of diabetic polyneuropathy with the diabetic neuropathy symptom and diabetic neuropathy examination scores. Diabetes Care 2003; 26:697–701.
- 24 Asad A, Hameed MA, Khan UA, Butt MU, Ahmed N, Nadeem A. Comparison of nerve conduction studies with diabetic neuropathy symptom score and diabetic neuropathy examination score in type-2 diabetics for detection of sensorimotor polyneuropathy. J Pak Med Assoc 2009; 59:594–598.
- 25 Niazi PHK, Ahmad K, Hussain A, Butt AW, Alam A. Electrodiagnostic evaluation of diabetic polyneuropathy. Pak Armed Forces Med J 2001; 51:75–77.
- 26 Asad A, Hameed MA, Khan UA, Ahmed N, Butt MU. Reliability of the neurological scores for assessment of sensorimotor neuropathy in type 2 diabetics. J Pak Med Assoc 2010; 60:166–170.
- 27 Perkins BA, Zinman B, Olyleye D, Bril V. Simple screening tests for peripheral neuropathy in the diabetic clinic. Diabetes Care. 2001; 24:250–256.