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EFFICIENCY OF MICHIGAN NEUROPATHY SCREENING INSTRUMENT AND NERVE CONDUCTION STUDIES FOR DIAGNOSIS OF DIABETIC DISTAL SYMMETRIC POLYNEUROPATHY

Cristina Muntean [1,✉], ***Bogdan Cătălin*** [2], ***Valerica Tudorică*** [1], ***Maria Moța*** [3]

[1] Department of Neurology, University of Medicine and Pharmacy of Craiova, Romania
[2] Department of Physiology, University of Medicine and Pharmacy of Craiova, Romania
[3] Department of Diabetes, Nutrition and Metabolic Diseases, University of Medicine and Pharmacy, Craiova, Romania

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Abstract

Background and Aims: *Little data regarding distal symmetric polyneuropathy (DSP) prevalence in Romania is available. The aim of the present study was to assess the prevalence of DSP in our cohort, to characterize it depending on glycemic control, and also to find an easy-to-apply method for DSP screening which could be used in Romania.* ***Material and Methods:*** *We performed a cross-sectional study enrolling 51 patients followed in the Diabetes, Nutrition and Metabolic Diseases Clinic, Clinical County Hospital of Craiova, Romania. A complete evaluation protocol consisting in clinical examination and Michigan Neuropathy Screening Instrument (MNSI), together with nerve conduction studies were applied for evaluation.* ***Results:*** *Among the type 2 diabetic patients investigated, 72.54% had DSP. Three-quarters of them had poor glycemic control (HbA1c ≥7%). Mean HbA1c level was 9.17%. Poor glycemic control led to a more severe DSP form as proven by nerve conduction studies and clinical examination. Allodynia and motor deficit were predominantly found with HbA1c ≥7%. Mean MNSI score for the group was 2.55, strongly correlated with nerve conduction studies.* ***Conclusion****: MNSI is a simple and validated diagnostic tool for DSP with a strong correlation to electrophysiological parameters. Therefore, its daily implementation in clinical practice could help identify and follow patients at risk for DSP.*

key words: *distal symmetric polyneuropathy, glycemic control, Michigan Neuropathy Screening Instrument, nerve conduction studies, type 2 diabetes.*

1. Introduction

Distal symmetric polyneuropathy (DSP) is, by far, the most frequent type of neuropathy found in association with diabetes [1,2]. Approximately 50% of patients with diabetes develop DSP during disease progression, while 20% present symptoms of DSP at diagnosis [3].

The classical progression of DSP is insidious, starting with symptoms affecting the distal lower limbs, and then slowly progressing proximally. Usually, patients symptoms are either mild or moderate, although some reports claim that up to 60% of patients with DSP can present with a pain predominant form [4]. Often

✉ No. 1 Tabaci Street, 200642 Craiova, Romania; *corresponding author e-mail*: cristinamuntean29@yahoo.com

damage to small myelinated and thinly myelinated fibers leads to complaints such as burning or aching pain, "asleep numbness" or prickling located predominantly in the heels - 32%, plantum of foot, calves - 37%, hands - 39%, dorsum of foot - 54%, toes - 67%, balls of feet - 69%, and feet - 96% [5]. Moreover, as a sign of nerve hyperexcitability [6], muscle cramps are frequently found in diabetic patients, having a prevalence of around 75.5% for type 2 diabetes, and around 57.5% for type 1 diabetes [7]. Motor involvement, consisting in muscle weakness and atrophy of ankle plantar and dorsal flexors [8] seems to be less important than sensitive damage, and it is closely related to signs and severity of DSP [9]. Furthermore, only 16-20% patients with DSP show signs of cardiovascular damage (cardiac arrhythmia, silent myocardial ischemia, orthostatic hypotension), gastric motility dysfunction, abnormal pupillary reaction, erectile dysfunction, and sudomotor dysfunction [10].

Criteria for the diagnosis of DSP were established, based on clinical history, physical examination and nerve conduction studies [2]. According to the "so called" Toronto Diagnostic Criteria [2], confirmed DSP was considered when symptoms (tingling, stabbing, burning pain, decreased sensitivity) or signs of neuropathy (distal symmetrical hypoesthesia or diminished/abolished ankle reflexes) were associated with abnormal results in nerve conduction studies. Probable DSP was diagnosed only in the presence of symptoms and signs of neuropathy. Presence of either symptoms or signs of neuropathy establishes the diagnosis of possible DSP and, finally, if only nerve conduction studies were altered (without symptoms or signs), a diagnosis of subclinical DSP was made.

Several composite scores have been proposed for screening and quantification of DSP severity, and some of them have already been validated [11]. One such tool is the Michigan Neuropathy Screening Instrument (MNSI) [12,13] that has been widely used in large clinical trials [14,15]. It consists of a 15-item self-administered questionnaire regarding sensory complaints, and a lower limb examination that includes foot inspection, vibratory sensation and ankle reflexes assessment [13]. A cutoff point for MNSI questionnaire ≥4 [16] or for MNSI examination score >2 [12,17,18] is considered abnormal.

There is a paucity of reports on DSP in Romania, with little data regarding its prevalence available at the moment. For this reason, the aims of our study were firstly to assess the rate of DSP in a group of diabetic subjects from Craiova, Romania, secondly to characterize it and to find differences in our group depending on glycemic control, and thirdly to find an easy and fast method for DSP screening.

2. Material and methods

2.1 Patients

Inclusion criteria. The study was conducted in the Diabetes, Nutrition and Metabolic Diseases Clinic, Emergency Clinical County Hospital of Craiova, Romania between 2012-2013. Fifty-one type 2 diabetic patients, 28 women and 23 men, were randomly chosen to be enrolled in the study, after having given written consent. The only inclusion criterion was for patients to be diagnosed with type 2 diabetes according to current criteria [19-21].

Exclusion criteria. We excluded from this study type 2 diabetic patients working in toxic environments, with chronic alcohol abuse, and also those using neurotoxic medications [22]. Patients presenting conditions that could be associated with neuropathy, such as end stage terminal diseases (chronic kidney disease,

hepatic dysfunction, cancer, hematological disease) or vitamin deficiency (B1, B6, B12, E, folic acid) [23] were excluded from this study.

Toronto Diagnosis Criteria [2] were applied to established DSP prevalence in our cohort.

2.2 Clinical evaluation. All patients underwent a thorough clinical examination for symptoms and signs of neuropathy. Data related to age, gender, diabetes duration from diagnosis, age at onset of neuropathic symptoms, type and location of sensory and motor symptoms, as well as time-course of the disease and other possible causes of peripheral nerve damage, were recorded. Neurological examination included muscle and tendon reflex testing, evaluation of light touch, pinprick, vibratory, temperature sensation and position sense. Weakness was assessed bilaterally in 14 muscle groups of the upper limbs (shoulder abductors, elbow flexors and extensors, wrist flexors and extensors, finger flexors and extensors, first dorsal interosseus muscle, abductor pollicis brevis) and of the lower limbs (hip flexor, knee flexors and extensors, ankle dorsiflexors and ankle plantar flexors). Muscle status was quantified on a scale from 0 (no contraction) to 5 (normal movement) on the Medical Research Council (MRC) grading system [24].

Deep tendon reflexes were scored as absent, present but decreased, normal, increased or increased with clonus [25]. Light touch was evaluated using a cotton wisp and 10-g Semmes-Weinstein monofilament (the latter by applying pressure over the plantar surface of the foot) [26]. Additionally, vibration perception threshold using a 128-Hz tuning fork, and joint position sense at the great toe were tested. Vibration and joint position were also evaluated more proximally if distal impairment was noted. Pinprick sensation was tested by applying a sterile safety pin on the surface of the skin and asking the patient, with eyes closed, to identify the sensation as sharp or dull or whether they felt it at all. Temperature sensation was evaluated using tubes filled with warm or cold water that were gently applied on patients skin. For both pinprick and temperature sensations testing started on the plantar surface of the foot, slowly moving towards more proximal parts until stimuli were correctly identified by the patient. Each patient had also a general examination notably looking for skin lesions and abnormal pulses in the lower extremities.

Patients were examined for autonomic symptoms defined as any of the following: symptomatic postural hypotension, abnormalities of sweating, gastrointestinal symptoms and erectile dysfunction.

MNSI questionnaire and MNSI examination were used for the overall evaluation of neuropathy severity of the entire group.

- MNSI questionnaire consisted in a self-administered test with 15 yes/no questions. “Yes” responses to questions 1–3, 5–6, 8–9, 11–12, 14–15 were each counted as one point: presence of numbness in the feet/legs=question 1, presence of burning sensation in the feet/legs=question 2, presence of increased sensitivity to touch in the feet=question 3, presence of prickling sensation in the feet/legs=question 5, presence of pain elicited by bed covers touching the skin=question 6, history of open sore in the foot=question 8, diabetic neuropathy already diagnosed=question 9, night aggravation of symptoms=question 11, pain in the feet elicited by walk=question 12, presence of dry skin=question 14, history of amputation=question 15. “No” responses to question 7 and 13 were also counted as one point: ability to distinguish cold from

hot water=question 7, ability to sense the feet during walking=question 13. Question 4 (evaluating the presence of cramps) and question 10 (evaluating presence of general weakness) considered to be a measure of peripheral vascular disease and general asthenia respectively are not included in the scoring algorithm [12]. In a recently published algorithm a new cutoff point for abnormal MNSI questionnaire ≥4 [16] is considered abnormal.

- MNSI examination was performed by a health professional. Presence of either deformities, dry skin, calluses, infections, fissures and ulcers, was scored 1 point for each foot. Ankle reflexes were examined and scored as follows: 1 point for each absent ankle reflex, 0.5 point for each ankle reflex present only with reinforcement (after Jendrassic manouver) or 0 point for a normal ankle reflex. Vibration sensation was tested in the great toe with a 128-Hz tuning fork and the score was designated as follows for each foot: 1 point for absent sensation in the toe, 0.5 point when the examiner felt the vibration for more than 10 s after the patient ceased to feel it at the great toe and 0 point when the examiner sensed the vibration for less than 10 s compared to the patient. A score >2 was considered abnormal [16].

2.3 Laboratory tests. Fasting blood glucose and glycated hemoglobin (HbA1c), renal and liver function tests, vitamin B12, thyroid hormones, protein electrophoresis with immunofixation, together with other routine blood tests (complete blood count, erythrocyte sedimentation rate) were assessed in all patients. In selected cases, hepatitis panel, HIV serology and antinuclear antibodies were pursued. Retinopathy was evaluated through a fundoscopy exam by an ophthalmologist.

2.4 Electrophysiological assessment. Routine electrophysiological studies were performed in all patients using standard clinical electromyography machine (MEB 9100 provided by Nihon Kohden). Skin temperature was measured and values were adjusted to a standard of 34°C. Bilateral testing of four motor nerves (median, ulnar, fibular and tibial) and three sensory nerves (median, ulnar and sural) was performed for each patient. Motor distal latency (DL), compound muscle action potential (CMAP), amplitude (A) and nerve conduction velocity (NCV) were recorded for each motor nerve. For sensory stimulation, the A and DL of the sensory nerve action potentials (SNAP) were recorded and NCV was calculated. Patients results were compared with results obtained in the control group, represented by 20 healthy volunteers. To exclude autonomic dysfunction, sympathetic skin response and heart rate variation during deep breathing were measured.

2.5 Statistical analysis

Statistical analysis was performed with GraphPad Prism version 6.0. Data are expressed as mean±standard deviation (SD) and statistical comparisons were determined using t-test, where <0.05 level of probability was considered statistically significant. Further regression analysis (linear, Spearman's rank correlation) was applied to assess the correlation between specific variables.

3. Results

3.1 General characteristics of the study group are given in Table 1. Mean age of the group was 60.23±13.40 years; more than half of the patients reported a disease progression of at least 3 years, with a mean diabetes duration of 3.78±3.53 years. Thirty-one patients (60.78%) were treated with oral antidiabetic agents, insulin

alone or combined therapy (insulin and oral agents). The other 20 were non-treated patients, 15 (29.41%) of them having recently been diagnosed with diabetes and the remaining 5 patients (9.80%) having interrupted their therapy. Approximately half of the group received concomitant antihypertensive treatment (53%).

Table 1. General characteristics of diabetic patients.

	Total group N=51	HbA1c <7% N=14	HbA1c ≥7% N=37	P[#]
Age at inclusion, (years)*	60.23 ± 13.40	64.78±16.69	58.51±11.73	0.21
Diabetes duration, (years)*	3.78 ± 3.53	1.57±2.65	4.62±3.49	0.004
Treatment, N (%):				
• Oral antidiabetics	21 (41.17)	4	17	0.27
• Insulin	5 (9.80)	0	5	0.15
• Oral antidiabetics + insulin	5 (9.80)	0	5	0.15
• No treatment	20 (39.21)	10	10	0.004
Neuropathy:				
• Duration of symptoms, (years)*	2.47±2.17	2.35±1.98	2.51±2.26	0.81
• Referral symptoms, N (%):				
o numbness	28 (54.90)	8	20	0.84
o muscle cramps	19 (37.25)	6	13	0.61
o tinglings	19 (37.25)	6	13	0.61
o burning sensations	18 (35.29)	5	13	0.96
o allodynia	6 (11.76)	1	5	0.04
• Clinical examination, N (%):				
o motor deficit	10 (19.60)	0	10	0.03
o ataxia	29 (56.86)	8	21	0.98
o ankle reflex	29 (56.86)	9	20	0.51
o tactile hypoesthesia	30 (58.82)	10	20	0.51
o vibratory hypoesthesia	33 (64.70)	10	23	0.54
• MNSI questionnaire*:	2.47±1.94	2.28±1.58	2.54±2.07	0.64
• MNSI examination*:	2.55±2.12	2.14±1.56	2.71±2.29	0.31

* =mean±standard deviation; N=number of subjects; [#] poor control vs. good control group

Anamnesis and clinical examination revealed signs and symptoms of neuropathy with a length-dependent distribution in 45 patients (88.23%), with a mean duration of symptoms of 2.47±2.17 years. The most common referral symptoms were numbness, cramps, tingling and burning sensations. Vibratory sensation measured at the great toe was diminished in 33 patients (64.70%), and tactile hypoesthesia was present in 30 cases (58.82%). Mild decreased tactile sensation not exceeding toes was found in another 5, while one patient complained of dysesthesia in the feet. Temperature discrimination and monofilament test were in the normal limits for all subjects in the study group. Mild ataxia was found in 29 patients (56.86%).

Mean MNSI examination score for the studied cohort was 2.55±2.12 (range 0-9), while mean MNSI questionnaire score was 2.47±1.94, being significantly correlated with neuropathic symptoms duration (R=0.403, p=0.003), with these patients having a higher sum of sensory symptoms. When applying MNSI examination score, we found a DSP prevalence of 50.98%. For MNSI questionnaire DSP prevalence was lower: 3.92% when keeping the old threshold, and 23.52% when using the modified threshold.

Motor evaluation revealed that only 10 patients (19.60%) had symmetric mild motor deficit located at toe and foot extensors (scoring 4 points out of 5 on the MRC score). Ankle reflexes were either absent or diminished in 56.86% of the group. Inspection of the feet revealed dry skin, calluses and fissures in 11 patients. Importantly, none of the patients had signs or symptoms of dysautonomia.

Concerning glycemic control, mean HbA1c level of the entire group was 9.17±2.60%. Depending on HbA1c values we identified 2 subgroups: "poor control" subgroup, with HbA1c ≥7%, and "good control" subgroup, with HbA1c <7%.

Characteristics of the "good control" subgroup (Table 1). Fourteen patients (27.45%) had a good glycemic control (HbA1=6.40±0.39%), mean diabetes duration prior to inclusion being 1.57 years. About half of the group (57.14%) had been diagnosed with type 2 diabetes during the current year of the study. All but 2 patients had sensory signs and symptoms consistent with DSP, with duration of 2.35±1.98 years. No motor deficit was identified. Mean MNSI examination score was 2.14±1.56, therefore compatible with DSP diagnosis; results at MNSI questionnaire above the target limit were found only in 2 patients.

Characteristics of the "poor control" subgroup (Table 1). Approximately three-quarters of the group had poorly regulated glycemia, with mean HbA1c levels of 10.21±2.29%. Mean age at inclusion was 58.51±11.73 years, while type 2 diabetes duration was 4.62 years, therefore significantly longer than that of the "good control" subgroup (p=0.004). Duration of neuropathic symptoms was similar to that of the previously described subgroup (2.51±2.26 years). Allodynia was more frequently found in this subgroup (5 patients reported allodynia in the "poor control" group compared to 1 in the "good control" group) (p=0.047). Motor deficit and diabetic retinopathy were particularly found in these patients. Mean MNSI examination score was 2.71±2.29, while mean MNSI questionnaire score was 2.71±2.29.

3.2 Electrophysiological results. All patients underwent nerve conduction studies, and their results were compared with those obtained by the control group. Motor nerve conduction studies revealed significantly longer distal latency in the median nerve (p=0.008) and important reduction in ulnar nerve amplitude (p<0.0001). Amplitude was also reduced for fibular nerve (p=0.01). Importantly, motor response was elicited in all patients, contrary to sensory potentials, which were abolished in 16 patients. Sensory abnormalities were recorded for both upper and lower limbs, with important reduction of amplitudes and conduction velocities for all tested nerves. Amplitude was reduced to almost ¼ of the response recorded in the control group: 29.81% for the median nerve, 23.10% for the ulnar nerve, and 25.23% for the sural nerve. Conduction velocity was reduced by less than 30% compared to the values of the control group. Further details are given in Table 2.

When glycemic control was considered, it was found that values for both motor and sensory conduction were lower in the "poor control" subgroup. Furthermore, 15 patients with

absent sensory response in the lower limbs were part of this subgroup. However, significant reduction was only noted for ulnar and fibular motor amplitudes and median sensory amplitude (Table 2).

Table 2. Electrophysiological results.

		Patients N=51	**Control group** N=20	**p**	**HbA1c<7%** N=14	**HbA1c≥7%** N=37	**p**
		M±SD	M±SD		M±SD	M±SD	
Motor nerves							
Median nerve	DL (ms)	4.04±0.83	3.36±0.37	p=0.0008	3.71±1.04	4.17±0.73	0.08
	A (mV)	7.57±5.15	9.23±2.33	p=0.06	9.58±8.81	6.78±2.45	0.08
	CV (m/s)	47.54±5.72	49.29±0.43	p=0.17	47.54±5.08	46.97±5.90	0.25
Ulnar nerve	DL (ms)	2.65±0.36	2.53±0.43	p=0.2	2.73±0.39	2.62±0.34	0.35
	A (mv)	7.66±2.12	11.37±2.35	p<0.0001	8.66±2.10	7.27±2.02	0.03
	CV (m/s)	53.81±4.74	54.38±0.39	P=0.16	53.68±5.09	53.87±4.68	0.90
Fibular nerve	DL (ms)	4.05±1.18	3.57±0.54	p=0.08	4.17±1.14	4.06±1.20	0.64
	A (mV)	3.74±2.02	4.96±1.16	p=0.01	4.19±2.05	2.58±1.46	0.01
	CV (m/s)	43.46±6.71	41.15±0.26	p=0.13	41.52±9.28	44.21±5.38	0.20
Tibial nerve	DL (ms)	5.09±1.47	3.57±0.54	p<0.0001	5.17±1.65	5.06±1.42	0.81
	A (mV)	5.09±3.70	8.01±1.27	p=0.001	4.88±4.88	5.17±3.22	0.80
Sensory nerves							
Median nerve	A (μV)	11.52±6.30	38.64±4.95	p<0.0001	15.82±8.14	9.80±4.49	0.001
	CV (m/s)	41.43±8.24	55.34±0.43	p<0.0001	44±8.23	40.41±8.13	0.17
Ulnar nerve	A (μV)	8.40±4.74	36.36±7.29	p<0.0001	8.25±5.57	8.46±4.49	0.89
	CV (m/s)	47.69±7.22	55.34±0.40	p<0.0001	50.05±5.48	46.72-7.69	0.14
Sural nerve	A (μV)	4.77±4.30	27.78±5.76	p<0.0001	6.47±6.09	4.10±4.83	0.15
	CV (m/s)	28.03±20.64	45.07±0.09	p=0.0003	35.21±13.08	25.24±22.46	0.126

DL=distal latency; A=amplitude; CV=conduction velocity; N=number of subjects; M±SD=mean+standard deviation

In order to investigate the relationship between nerve conduction studies and the level of HbA1c, duration of type 2 diabetes and the severity of DSP (as provided by MNSI examination score), further correlation analysis was applied. Spearman's coefficient showed significant correlation between this score and some of the parameters of nerve conduction studies. Conduction velocities for motor ulnar, motor median and fibular nerve showed important inverse correlation with MNSI examination score (R=-0.482, p=0.004; R=-0.408, p=0.003; R=-0.294, p=0.038). At the same time tibial amplitude was also correlated with MNSI score (R=-0.375, p=0.007). Regarding sensory conduction, main correlations were with sural amplitude (R=-0.407, p=0.003) and conduction velocity (R=-0.330, p=0.019). In the upper limb only conduction velocity of ulnar sensory nerve was correlated with the MNSI score (R=-0.328, p=0.022).

Patients with higher HbA1c values had slower conduction velocities of motor fibers in the upper limbs, as recorded in ulnar nerve (R=-0.445, p=0.001), as well as diminished amplitude and conduction velocity in sural nerve (R=-0.420, p=0.002; R=-0.372; p=0.007). Sensory response for upper limbs did not seem to be influenced by poor glycemic control. Diabetes duration seemed to be related only to motor conduction in the lower limbs, represented by lower amplitude of fibular nerve in patients with longer diabetes duration (R=-0.509, p=0.0002).

4. Discussion

DSP has a high prevalence worldwide. According to literature it varies widely, between 10%-75%, depending on the chosen criteria for patient selection and on methods applied for their evaluation [9,27–29]. In our study we decided to perform a complete evaluation protocol consisting in clinical examination and clinical composite score, together with nerve conduction studies. Subsequently, when applying Toronto Diagnosis Criteria [2], we found a confirmed DSP prevalence of 72.54%, representing patients that had clinical and electrophysiological signs of diabetic neuropathy.

Our patients had classical DSP, and only 27.45% of patients met ADA glycemic goal [30]. Most frequently reported symptoms were numbness, cramps, tingling and burning sensation, about half of the group (54.90%) presenting more than 2 neuropathic symptoms. A small percentage of patients (11.76%) had a painful form, this form being more frequently found in those with poor glycemic control. Ataxia was found in all groups including patients with good glycemic control contrary to motor deficit that predominated in patients with HbA1c≥7%.

Most prominent clinical sign in our group was decreased vibratory sensation (64.70%), followed by tactile hypoesthesia (58.82%) and absent or lowered reduction of ankle reflex (56.86%). Pain and temperature examination is considered to be a better method than vibration for early detection of nerve fiber damage, and risk of developing diabetic foot [31]. None of our patients presented pain or temperature sensation impairment, hence explaining absence of severe trophic lesions in the foot. There was no statistically significant difference regarding DSP severity when measured with MNSI score in the 2 glycemic control subgroups. Nevertheless, diabetes complications, such as retinopathy, was associated with the low glycemic control subgroup, as confirmed in other studies [32].

Nerve conduction studies revealed motor and sensory conduction alteration in the upper and lower limbs, compared to control group.

These values were more reduced in patients with higher HbA1c levels, and mainly involved fibular, ulnar and median nerve. At the same time, sensory potentials were abolished in 40.54% patients with poor glycemic control. Patients with longer diabetes duration had a greater reduction of motor conduction in the lower limbs, while those with higher HbA1c levels had, apart from that, worse upper limbs motor and lower limbs sensory response.

Nerve conduction studies remain an expensive diagnosis tool for neuropathy assessment. That is why several studies tried to compare them with clinical examination scores. One such study that was conducted in Turkey and compared MNSI score to electroneuromyography, found a prevalence of 32.1% and 46.2% respectively [33]. Similar results were published in a recent Italian study that tested MNSI score on 3591 patients with type 2 diabetes, with a reported prevalence of DSP of 30% [34]. Differently from this data, our study recorded higher DSP prevalence (50.98%) with MNSI examination tool. At the same time we have proven that there is a strong relation between this score and some of the parameters tested by nerve conduction. Mainly sensory and motor conduction in the lower limbs seem to correlate with results recorded for the MNSI score.

Contrary to MNSI examination tool, MNSI questionnaire has a lower sensitivity. The highest prevalence of neuropathy diagnosed by this tool was 5% [12]. A large study conducted on type 1 diabetic patients suggested that a lower threshold to define abnormal MNSI questionnaire could significantly increase the accuracy of this test. They recommend a cutoff point of ≥4 instead of ≥7 for defining abnormal values [16]. In our group we found a 3.92% DSP prevalence when keeping the old threshold, and a 6-fold higher prevalence (23.52%) when applying the modified threshold.

Our study had some limitations. There was an unequivocal distribution of patients in the 2 glycemic control subgroups and patients were randomly recruited from the same diabetes clinic; they were thus representative for only one region in the country. Despite these limitations, the strengths of the study were the fact that all patients had a thorough clinical examination performed by the same examiner, and all patients underwent nerve conduction studies, allowing us to correctly establish the prevalence of DSP in our group.

5. Conclusions

Even if we cannot translate our results to other centers in Romania, we could expect to find high rates of DSP. A standardized protocol for diabetic population should be implemented, at least through a thorough examination with validated scales such as MNSI score. Despite the fact that this score dates back to 1994 and is less sensitive than nerve conduction studies, it has proven to be a simple diagnosis method that correlates with nerve conduction parameters, and could allow an objective evaluation and follow-up of patients with DSP associated to diabetes. A similar approach could be implemented in other hospitals in Romania in order to reinforce our results.

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