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Importance of pain evaluation for more accurate diagnosis of painful diabetic polyneuropathy

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Key words: *glycemic control (HbA1c); quantitative sensory testing; painful diabetic neuropathy; superficial pain; deep pain.*

Summary. *Pain is a common problem in diabetic neuropathy, but relatively little has been published regarding the extent to which it needs to be addressed in clinical practice.*

Objective. To assess neuropathic pain profile and its association with quantitative sensory testing in painful diabetic polyneuropathy.

Material and methods. Altogether, 61 consecutive diabetic inpatients with symmetric neuropathic complaints were enrolled. Clinical neurological examination and quantitative sensory testing (QST) were performed. Patients were interviewed using the Neuropathic Pain Scale (NPS) and filled in the McGill Pain Questionnaire (MPQ).

Results. Of all patients, 49 (80.3%) had clinical diabetic polyneuropathy. Only 17 of these patients complained of lower extremity pain on an initial interview, while 27 marked it in the MPQ. The intensity of deep and superficial pain did not differ, but patients rated deep pain as more unpleasant than superficial (6.27±2.37 vs. 4.30±1.42 on the NPS, P=0.034). Superficial pain NPS items tended to correlate with QST results, while deep pain items did not. Only female gender (OR=7.87) and lower glycosylated hemoglobin level (OR=0.65) were predictive of pain in case of diabetic neuropathy.

Conclusions. Standard pain questionnaires were useful in identifying pain sufferers. At the same intensity, deep neuropathic pain was more unpleasant than superficial. Pain manifestation was associated with female gender and lower level of glycosylated hemoglobin.

Introduction

Diabetes mellitus (DM) is a chronic noninfectious metabolic disease with a rapidly increasing prevalence reaching epidemic levels. It is estimated that by the year 2030, 366 million people will have DM worldwide (1). One of the most common DM complications is DM-related neuropathy. A consensus statement produced by an international meeting on the diagnosis and management of diabetic neuropathy defined it as "the presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes after the exclusion of other causes" (2).

Diabetic peripheral neuropathy (DPN), also known as chronic sensorimotor distal symmetrical polyneuropathy, accounts for 80% of DM-related neuropathies (3). The prevalence rates of DPN reported by different epidemiological studies vary from 26% to more than 50% (4–6). DPN is diagnosed to as much as 44% of the patients with 5-year duration of DM in Lithuania (7).

Being one of the most serious and costly complications, it predisposes DM patients to symptoms, such as unremitting pain and unsteadiness, and leads to disabling end-stage complications of foot ulceration (experienced by 15% of patients [5]) and amputation. Different authors report varying prevalence rates of pain in DPN patients. One study reported a prevalence rate of 7.5% (one-third of all subjects with diabetic neuropathy) for painful lower limb symptoms in diabetic patients (6). The other study observed that pain in the feet and legs occurred in 11.6% of patients with insulin-dependent diabetes and in 32.1% of patients with non–insulin-dependent diabetes (6). It is estimated that 10% to 20% of patients with sensorimotor neuropathy experience painful symptoms at any one time (2, 8).

Chronic pain has an impact on many aspects of the sufferer's life. Studies of DPN have showed that symptoms of chronic, unremitting pain, nonhealing foot ulcers, and amputations are associated with

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worsened physical and psychosocial functioning (9). The painful symptoms of chronic sensorimotor neuropathy tend to last for years although the severity may vary (2). The improvement of these symptoms, however, may actually represent a worsening or progression of the neuropathy, resulting in the development of the insensitive foot at risk of ulceration (2). DPN accounts for more admissions to hospital than all other diabetic complications combined and is responsible for 50–75% of nontraumatic amputations in the United States, most of them occurring due to loss of nociceptive sensation (10).

Unfortunately, although it is acknowledged that pain is a common problem in some persons with diabetic neuropathy, relatively little information has been published on the nature and scope of this pain condition. Such information is necessary in order to determine the extent to which DPN pain needs to be addressed in clinical practice.

Therefore, the aim of the present study was to assess multiple aspects of pain by means available in clinical practice.

Material and methods

Participants. The study was conducted from February 1, 2007, to April 30, 2007. All consecutive diabetic inpatients who had symmetric neuropathic complaints on admission were included into the study. Complaints defined as neuropathic were burning sensation, paresthesia, loss or disturbance of sensation, cramps, pain, numbness, and feeling of weakness in lower extremities. The patients older than 70 years, suffering from other conditions, or using drugs potentially inducing neuropathy were excluded from the study as well as those who refused to participate. Other causes of neuropathy used as exclusion criteria were as follows: other metabolic disorders, neoplastic diseases, systemic diseases, autoimmune diseases, HIV infection, psoriasis, nutritional deficiencies, substance abuse, vertebroneurologic syndromes, demyelinating diseases of the nervous system, lesions of the central nervous system, use of neurotoxic drugs (cytotoxic drugs, isoniazid, metronidazole, chloramphenicol, nitrofurantoin, amiodarone, phenytoin, simvastatin).

The protocol used in this study was approved by the Kaunas Regional Bioethics Committee, and all the participants of the study signed their informed consent.

Measures. *Personal and medical data.* Data were collected by trained neurologists who reviewed previous medical records and who interviewed and examined patients by means of standardized methods and instruments during the period of hospitalization. The following data were collected: age, sex, history of diabetes mellitus (type, duration, treatment, glycosylated hemoglobin [HbA1c]), complications including previously diagnosed diabetic neuropathy, history of arterial hypertension, dyslipidemia.

Clinical neurological examination. The patients were interviewed for their complaints, and clinical neurological examination was carried out by a single neurologist (J.S.). The following neurological tests were used for clinical examination of lower extremities: sensation of pain with a toothpick; sensation of touch with a cotton swab; sensation of pressure and touch with a 10-g monofilament; temperature perception with Tip-Therm; joint position sensations; vibratory sensibility with a 128-Hz tuning fork on the big toe joint and medial malleolus; muscle proprioceptive reflexes (ankle and knee jerk reflexes); muscle strength (MRC classification; [2]). The severity of DPN was estimated based on the findings of clinical examination (ankle jerk reflexes, vibratory sensibility, pain sensation, and temperature perception) using the Neuropathy Deficit Score (NDS) (11).

At least 2 symmetric neuropathic impairments found on clinical examination was a diagnostic criterion for DPN.

Pain assessment. Patients were asked to fill in the Lithuanian version of the McGill Pain Questionnaire (MPQ) (12), which was developed to use by Pakula (13, 14). The Lithuanian version of MPQ contains 54 pain descriptors divided into 14 subscales (8 sensory and 6 emotional). Each descriptor has its nominative index, and adjusted intensity index is calculated. This allows evaluating both sensory and emotional components of pain. As it was important to select patients with typical polyneuropathic complaints, pain drawing was added to the MPQ to evaluate the distribution of painful symptoms.

All the patients were interviewed about lower extremity pain using the Neuropathic Pain Scale (NPS) (15). The Lithuanian version of this instrument was validated in 2004 (14). The NPS is one of the most widely used instruments for evaluation of different symptoms of neuropathic pain. It includes 10 descriptors that allow discriminating and quantifying clinically important aspects of neuropathic pain as well as to monitor effects of therapy on it.

Quantitative sensory testing. Quantitative sensory testing (QST) was performed by two trained medical students using NervScan 2000 (Neurotron, Inc., USA, 1994) (16). NervScan2000 electrodiagnostic examination employs a standardized automated procedure to generate objective quantitative measures of the conduction and functional integrity of sensory nerve fibers. The unit emits nonaversive transcutaneous electrical stimuli through a pair of gold-plated electrodes to quantify the values of neuroselective painless current perception threshold (CPT) (16). The procedure included testing

the patient at two sites of each lower extremity (big toe joint and dorsal ankle) with electrical stimuli of three different sinusoidal frequencies. Each frequency – 2000 Hz, 250 Hz, and 5 Hz – evokes a response from a different subpopulation of sensory nerve fibers, i.e., large myelinated, small myelinated, and small unmyelinated fibers, respectively. Abnormally low CPT values indicate a hypersensitive nerve function, reflecting hyperesthetic condition. Abnormally elevated CPT values indicate a loss of nerve function reflecting a hypoesthetic condition.

Statistical analysis. Statistical analysis was performed using the statistical package SPSS 12.0. Due to small sample and group sizes, nonparametric criteria were used in statistical analysis. Differences between groups were checked using the Mann-Whitney *U* (for continuous data) and chi-square tests (for categorical data). Associations between diagnostic measures and QST were evaluated using Spearman's correlation. Reliability analysis for the NPS questionnaire was performed: Cronbach α was 0.876; removal of any of the items did not change this value markedly. Factor analysis of the NPS questionnaire was performed, and pain predictors were determined using binary logistic regression analysis (forward stepwise method). The level of significance was set at $P<0.05$. The results of descriptive statistics are presented as mean (standard deviation).

Results

Altogether, 61 patients (41 males and 20 females) were examined. Of them, 49 (80.3%) were diagnosed with DPN. On an initial interview, 17 (34.7%) patients complained of pain in lower extremities, while 27 (55.1%) of them marked lower extremity pain in the MPQ. Among these patients, none had marked pain that had other than "glove-stocking" type of distribution.

Patients were divided into the painful and nonpainful DPN groups according to the results of the MPQ, as it is a standardized method that identified more pain sufferers. No significant differences, except for gender and HbA1c, were found between two groups of the patients in respect of the variables analyzed (Table 1). Pain was experienced by more than 80% of females and less than half of males. The painful DPN group had lower levels of HbA1c ($P=0.026$).

The most prevalent pain descriptors marked in the MPQ were tingling (n=11), cramping (n=12), gnawing (n=14), rushing to change position (n=15), keeping awake (n=13), and unpleasant (n=17).

Factor analysis of the NPS questionnaire was performed. Depending on the sample size and data, the initial eigenvalue of the extracted components was expected to be >1.8. Only one component exceeded this value (4.9), and it included all the items of the questionnaire.

All the deep pain descriptive NPS items correlated positively with number and adjusted intensity index of sensory pain descriptors from the MPQ, and many of them also correlated with emotional pain descriptors, while superficial pain NPS items did not tend to correlate with number and adjusted intensity index of both sensory and emotional pain descriptors from the MPQ (Table 2).

The skin sensitivity score from the NPS correlated significantly with QST results for 2000-Hz frequency (Aβ fibers); the superficial pain NPS score had significant correlations or tended to correlate with QST results for all frequencies (Table 3). None of QST results had any correlations with either number or adjusted intensity index of the pain descriptors from the MPQ.

According to NPS results, patients who experienced purely deep (n=11) and purely superficial (n=10) pain were separated out. Although there was no significant difference in the pain intensity score (on the NPS) between the groups, the unpleasant pain scores of the deep pain group (mean 6.27±2.37)

Table 1. Personal data for painful and nonpainful groups with diabetic peripheral neuropathy

	Painful DPN (n=27)	Nonpainful DPN (n=22)	*P* value
Gender, n (%)			
Male	14 (51.9)	19 (86.4)	**0.015**
Female	13 (48.1)	3 (13.6)	
Age, mean (SD), years	50.52 (11.54)	45 (15.16)	0.2
DM type, n (%)			
Type 1	12 (44.4)	10 (45.5)	1.000
Type 2	15 (55.6)	12 (54.5)	
DM duration, mean (SD), years	15.33 (8.42)	14.82 (10.18)	0.6
HbA1c, mean (SD), %	8.3 (1.4)	9.94 (2.55)	**0.026**
DPN severity, n (%)			
Mild, NDS <6	5 (18.6)	6 (27.2)	0.8
Moderate, NDS from ⩾6 to <9	11 (40.7)	8 (36.4)	
Severe, NDS ⩾9	11 (40.7)	8 (36.4)	

DPN, diabetic peripheral neuropathy; DM, diabetes mellitus; NDS, Neuropathy Deficit Score.

Table 2. Correlation between items of the Neuropathic Pain Scale questionnaire and pain descriptors in the McGill Pain Questionnaire

NPS items		Pain descriptors (MPQ)			
		Sensory		Emotional	
		Number of descriptors	Adjusted intensity index	Number of descriptors	Adjusted intensity index
Intensity	*r*	**0.580**	**0.544**	**0.491**	**0.482**
	P value	**0.000**	**0.000**	**0.000**	**0.000**
Sharp	*r*	**0.448**	**0.465**	**0.330**	**0.332**
	P value	**0.001**	**0.001**	**0.020**	**0.020**
Hot	*r*	**0.335**	**0.349**	0.188	0.204
	P value	**0.019**	**0.014**	0.195	0.160
Dull	*r*	**0.431**	**0.450**	0.207	0.212
	P value	**0.002**	**0.001**	0.154	0.144
Cold	*r*	0.268	0.247	**0.396**	**0.323**
	P value	0.063	0.087	**0.039**	**0.024**
Sensitive	*r*	0.198	0.202	–0.003	–0.002
	P value	0.172	0.163	0.938	0.991
Itchy	*r*	0.263	**0.288**	0.064	0.067
	P value	0.068	**0.045**	0.664	0.645
Unpleasant	*r*	0.279	**0.294**	0.180	0.186
	P value	0.052	**0.041**	0.216	0.200
Deep	*r*	**0.425**	**0.447**	0.262	0.276
	P value	**0.002**	**0.001**	0.069	0.055
Superficial	*r*	0.092	0.097	0.010	0.010
	P value	0.529	0.506	0.945	0.043

NPS, Neuropathic Pain Scale questionnaire; MPQ, McGill Pain Questionnaire.

Table 3. Correlation between items of the Neuropathic Pain Scale questionnaire and results of quantitative sensory testing

NPS item		QST (NervScan 2000)		
		2000 Hz (Aβ fibers)	250 Hz (Aδ fibers)	5 Hz (C fibers)
Intensity	*r*	0.213	0.163	0.012
	P value	0.138	0.257	0.936
Sharp	*r*	0.009	0.064	0.078
	P value	0.950	0.657	0.589
Hot	*r*	0.168	0.197	0.193
	P value	0.244	0.170	0.180
Dull	*r*	0.215	0.243	0.147
	P value	0.134	0.089	0.307
Cold	*r*	0.071	–0.047	0.050
	P value	0.625	0.746	0.731
Sensitive	*r*	**0.324**	0.114	0.230
	P value	**0.022**	0.429	0.108
Itchy	*r*	0.206	0.113	0.136
	P value	0.152	0.435	0.346
Unpleasant	*r*	0.155	0.397	0.175
	P value	0.282	0.397	0.225
Deep	*r*	–0.091	–0.097	–0.132
	P value	0.531	0.504	0.361
Superficial	*r*	**0.310**	0.265	**0.373**
	P value	**0.028**	0.062	**0.008**

NPS, Neuropathic Pain Scale questionnaire; QST, quantitative sensory testing.

on the NPS were significantly higher than the scores of superficial pain group (mean 4.30±1.42; t=2.28, df=19, P=0.034). Both the number and adjusted intensity index of sensory pain descriptors from the MPQ were significantly higher in the deep pain group (t=2.48, df=18, P=0.023 and t=2.59, df=19, P=0.018, respectively).

We tried to identify factors that would influence the lower extremity pain in DM patients. Binary logistic regression analysis was performed, using the

Table 4. Pain predictors in patients with diabetic peripheral neuropathy

	Variable	Odds ratio (95% CI)	*P* value
Step 1	HbA1c	0.640 (0.439–0.932)	0.020
Step 2	Gender (female)	7.871 (1.219–50.812)	0.030
	HbA1c	0.650 (0.453–0.932)	0.019

forward stepwise method, and pain was selected as an outcome variable. The initial model included the following factors: gender, age, DM type, DM duration, severity of DPN (according to NDS), HbA1c level, and QST results. After two steps, the stepwise procedure selected only two statistically significant factors, i.e., female gender and HbA1c level (Table 4). Females were more likely to experience pain than men (OR=7.87). A 1% increase in HbA1c level decreased the odds of experiencing pain by 35%. The overall accuracy of this model in predicting a rise in pain in diabetic patients was 65.9%. These factors predicted 35% of lower extremity pain dispersion in the study sample (R^2=0.35).

Discussion

In our study, the use of pain questionnaires helped to identify more patients with painful lower extremity symptoms. Female patients were more likely to experience pain than males, and higher levels of HbA1c were related to lower odds of experiencing pain. The patterns of pain differed between the deep and superficial pain groups.

Pain patterns in patients with diabetic peripheral neuropathy. It is well recognized that pain is the most distressing symptom of DPN and the main factor that prompts the patient to seek medical advice (6). However, many patients with painful DPN do not report their symptoms until pain becomes severe (17). Patients often find it very difficult to describe the symptoms because they are different from other types of pain they have previously experienced (18). These observations of other authors were confirmed by our findings. Only about one-third of the examined patients complained of lower extremity pain on the initial interview, while nearly half of them marked lower extremity pain in the MPQ. In order not to miss symptoms, which may not be considered as pain and are difficult to describe by patients, physicians should employ standard questionnaires at interview. Taken into the account that the diagnosis of neuropathic pain relies a lot on specific pain characteristics (e.g., Boureau et al. [19] found that they could differentiate neuropathic from nonneuropathic pain with a 66% accuracy, based solely on patient descriptors), standard questionnaires would also serve as a useful tool for revealing these characteristics and differentiating neuropathic pain from other types of pain.

The results of factor and reliability analyses of the NPS showed its integrity and relevance for the study sample. Although it proved to be homogenous, two different types of pain may be separated out according to its correlations with other diagnostic instruments. The NPS items, which could be attributed to deep pain characteristics (sharp, hot, dull, and deep) tended to correlate positively with the number and adjusted intensity index of both sensory and emotional pain descriptors, while this tendency was not observed for the items, which characterize superficial pain (itchy, sensitive to touch, and superficial). Furthermore, superficial pain items (sensitive and superficial) were observed to have a tendency of correlation with QST results, while deep pain items were not. This finding, in our opinion, may be due to the nature of the QST method used, in which electric impulses evoke direct response from nerve fibers of the superficial skin layers. Further investigation should be performed addressing the issue of unnatural pathways (omitting receptors) of this QST method.

The above-mentioned findings attracted our attention to different patterns of deep and superficial pain. Therefore, we identified patients with pure deep and pure superficial pain and sought for further differences. A significantly higher number and adjusted intensity index of sensory pain descriptors in the MPQ of the deep pain group suggest a greater variety of deep versus superficial pain mechanisms in DPN patients. No differences were observed in the overall pain intensity ratings in the NPS; however, deep pain sufferers rated their pain to be more unpleasant, suggesting that even at the same pain intensity, spatial characteristics have an impact on emotional pain evaluation (perception). The affective component of pain is influenced not only by the intensity of the pain sensation. Other factors, such as the meaning of pain and the context in which pain is experienced, also contribute to the unpleasantness of pain (20). Although research demonstrates consistently strong associations between measures of global pain intensity and pain interference (21), differences between deep and superficial pain according to its impact on different aspects of individual's life have not been widely studied. Neurophysiologic research shows considerable evidence for different deep and superficial pain circuits in the brain (22). It is believed that deep pain evokes passive emotional coping that includes quiescence and vasodepression. In contrast, cutaneous pain evokes an active emotional coping: the fight or flight response (22). A study by Jensen et al. (23) indicated that measures of pain quality and perceived depth were significantly associated with pain interference with functioning, independently of global pain intensity and unpleasantness. Practically, this proves the need of assessment of pain characteristics in addition to

its overall intensity in order to understand its impact on a patient's life. Knowledge about pain quality and perceived depth could potentially help to differentiate between different types of pain (i.e., nociceptive vs. neuropathic) and also could be used to help in selecting the treatment or intervention that is most appropriate or effective for a particular type of pain (23).

Pain-associated factors in diabetic peripheral neuropathy. Although many authors agree on risk factors for DPN, there is no consensus on what predisposes pain in DPN patients at present time. Some authors report that neither clinical data nor neurophysiological parameters enable to predict pain in DPN (24, 25). Others propose some factors possibly influencing painfulness of DPN. One of the main contradictions in this matter concerns structural changes in nerve fibers to be associated with pain in DPN. The abnormal nature of neuropathic pain means that the pain is often removed from any area of tissue damage or injury, and the degree of pain often does not correlate with the apparent extent of peripheral tissue damage (26). This is supported by some studies where no significant correlations were found between the structural changes in nerve fibers and presence of pain (24, 27). However, other authors have proposed a relationship between pain in DPN and peripheral nerve tissue damage (24, 25). Nerve fiber dysfunction may be indirectly demonstrated by changes in QST results. Some authors indicate correlations between deterioration of cold detection thresholds and intensity (25) or presence of pain (27) in painful DPN. In this study, QST results were not predictive of pain.

The relationship between pain and alterations in blood glucose levels in DPN patients has been widely studied. Hyperglycemia was found to reduce the pain threshold and tolerance to electrical stimulation (28). Spontaneous discharges in potentially nociceptive C fibers and occasionally in Aδ fibers were more common in experimental hyperglycemic animals (2). In a number of studies, improved glucose control was associated with relief of painful symptoms in DPN patients (2, 17, 27, 28). However, even in patients with long-term excellent glycemic control (HbA1c, <8%), the lifetime incidence of painful DPN remained 20% (17). Fluctuations in blood glucose levels were suggested to be other factor predisposing painful symptoms in DPN patients (2, 27). It has also been observed that a rapid marked decrease in blood glucose levels precipitated the onset of acute neuropathic pain (28).

Other factor, which was found to have an impact on pain presence in DPN studies, is disease duration, as some authors report that the natural course of painful DPN has a tendency of spontaneous improvement and resolution of pain (27, 28). Yet, this should not be equated to improvement of neuropathy but rather to the deterioration of peripheral nerve function (11, 27, 28). However, in other studies, no trends toward pain resolution over time were noticed (28). Moreover, some authors suggest that painful DPN is significantly more likely to occur with increasing severity of neuropathy (29).

Ziegler et al. (30) reported association between the type of DM and pain presence. However, other factors, such as disease duration and age of a patient, might have influenced the results as these measures differed significantly between both DM type groups.

Genetic factors, such as family history (8) and some specific genes responsible for chronic pain (25), may also have a role in pain rise in DPN patients.

Despite all the above-mentioned possible pain-associated factors, some authors report that different factors may only modify pain intensity or pain-coping strategies but not predict the presence of pain itself (25). Gender is reported to be one of such factors possibly modifying pain perception. According to some authors, females tend to be more sensitive to pain than males, while other studies do not report such differences (31).

Our findings do not support many of the above-mentioned hypotheses. The predictive value of female gender may be partly explained by the hypothesis that women tend to be more sensitive to pain than men. Therefore, we suggest that some of the unpleasant symptoms (dysesthesia) experienced by female patients might be referred to as pain while men indicate them as nonpainful sensations.

The influence of HbA1c on a rise in pain in our sample is contradictory to previous studies. We suppose this discrepancy may be attributed to the fact that our sample had a poor long-term glycemic control (mean HbA1c, 9.32%) suggestive of progressive DPN. In accordance to this, 38.8% of the DPN patients had a severe form of DPN (based on the NDS). This would favor the hypothesis of pain loss with progression of DPN.

Several limitations of our study should be mentioned. Firstly, the study included only inpatients, meaning that the majority of them had a poor long-term glycemic control and progressive DM complications (including DPN). Long mean DM duration in the study sample (approximately 15 years) also may lead to advanced-stage complications. Although there was no significant difference in DPN severity groups between painful and nonpainful DPN, we think that a large number of moderate-to-severe neuropathy cases (nearly 80%) might have biased the results and contributed to contradictory findings of the analysis of pain predictors. Secondly, we think that some correlations between QST results and pain assessment questionnaires might have

failed to reach statistical significance due to a small sample size. In order to make sure that some of our findings were not incidental (e.g., differences in patterns of deep and superficial pain), further investigation must be carried out with a larger number of patients.

Conclusions

In order not to miss painful symptoms, failed to be identified by patients themselves, standard quantitative pain questionnaires should be employed in the assessment of patients with diabetic peripheral neuropathy. Different qualities of pain may have different affective impacts; therefore, these characteristics should be taken into account while evaluating patients with painful diabetic peripheral neuropathy. Physicians should also be aware that some factors might predict a rise in pain in patients with diabetic peripheral neuropathy. In our sample, these proved to be female gender and lower levels of HbA1c.

Skausmo vertinimo reikšmė tikslesnei skausminės diabetinės neuropatijos diagnostikai

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Raktažodžiai: glikemijos kontrolė (HbA1c), kiekybinis jutimų tyrimas, skausminė diabetinė neuropatija, paviršinis skausmas, gilus skausmas.

Santrauka. Skausmas yra dažna pacientų, sergančių diabetine neuropatija, problema, tačiau duomenų, kaip jis turėtų būti vertinamas kasdienėje klinikinėje praktikoje, nepakanka.

Tyrimo tikslas. Ištirti pacientų, sergančių diabetine polineuropatija, patiriamo neuropatinio skausmo pobūdį ir jo ryšį su kiekybinio jutimų tyrimo rodmenimis.

Medžiaga ir metodai. Tyrime dalyvavo 61 stacionare gydytas pacientas, kuris turėjo neuropatijai būdingų simptomų. Tyrimo protokolą sudarė pacientų apklausa, skausmo įvertinimas pagal neuropatinio skausmo klausimyną NPS10 ir Pakulos-McGill klausimyną, klinikinis neurologinis ištyrimas ir kiekybinis jutimų tyrimas.

Rezultatai. Diabetinė neuropatija kliniškai nustatyta 49 pacientams (80,3 proc.; kriterijus – du ir daugiau simetrinių neurologinių sutrikimų). Kojų skausmą apklausos metu nurodė 17 pacientų (34,7 proc.), o Pakulos-McGill klausimyne kojų skausmą pažymėjo 27 (55,1 proc.). Nors pacientų patiriamo giluminio ir paviršinio skausmo intensyvumas reikšmingai nesiskyrė, giluminis skausmas buvo nemalonesnis už paviršinį (atitinkamai – 6,27±2,37 ir 4,30±1,42 balo pagal NPS10). NPS10 įverčiai, apibūdinantys paviršinį skausmą, reikšmingai koreliavo su kiekybinio jutimų tyrimo rodmenimis. Skausmo pasireiškimas, sergant diabetine neuropatija, buvo susijęs su moteriškąja lytimi (ŠS=7,87) ir mažesniu glikuoto hemoglobino kiekiu (ŠS=0,65).

Išvados. Vertinant skausmą pagal standartizuotus klausimynus, nustatyta daugiau jį patiriančių pacientų. Tokio paties intensyvumo giluminis skausmas buvo nemalonesnis už paviršinį skausmą. Skausmo potyris buvo susijęs su moteriškąja lytimi ir mažesniu glikuoto hemoglobino kiekiu.

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