EARLY DIAGNOSIS OF TYPE 2 DIABETES MELLITUS AND PREVENTION OF DIABETIC POLYNEUROPATHY

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ABSTRACT

Diabetic neuropathy is a peripheral nerve disorder that occurs in diabetes mellitus and is caused by metabolic imbalances. Diabetic neuropathy is one of the most common complications of diabetes, occurring in approximately 30-50% of patients. A patient is considered to have diabetic neuropathy if they have diabetes and no other causes of peripheral nervous system damage are present. Diabetic neuropathy develops approximately 5-15 years after the onset of the disease. The aim of this study is to evaluate the clinical and metabolic factors associated with the development of diabetic distal polyneuropathy in patients with type 2 diabetes mellitus and to substantiate a comprehensive approach to its early detection. To develop the study model, we propose examining patients with type 2 diabetes mellitus to determine disease duration, HbA1c levels, body mass index, waist circumference, blood pressure, and lipid profile.

Relevance. Diabetic neuropathy is a heterogeneous group of lesions of the peripheral and autonomic nervous system associated with diabetes mellitus. The most common form is distal symmetric sensorimotor polyneuropathy, predominantly affecting the distal parts of the lower extremities.

Current data indicate that diabetic sensorimotor polyneuropathy occurs in approximately one-third of patients with diabetes mellitus, although a significant portion of cases may remain asymptomatic. International expert consensus emphasizes that up to half of patients with DPN may not present typical symptoms, making patient interviews alone insufficient to rule out the disease [1].

An important issue is that the first peripheral nervous system disturbances can occur before significant neurological deficits develop. In the early stages, damage to fine nerve fibers predominates, manifesting as burning, tingling, hyperalgesia, or discomfort in the feet. As the condition progresses, disturbances in vibration and tactile sensitivity, decreased Achilles reflexes, and loss of protective sensation occur [1, 2].

The American Diabetes Association's current guidelines recommend screening for DPN at the time of diagnosis of type 2 diabetes and at least annually. This screening should include

an assessment of symptoms, sensation, and a 10-g monofilament test. For large nerve fiber testing, a 128-Hz tuning fork vibration test is recommended [2, 3].

The problem is particularly relevant for the primary healthcare system. In 2026, the Russian expert council proposed a practice-oriented algorithm for diagnosing and routing patients with DPN, which involves a combination of collecting complaints, neurological examination, assessing sensitivity, and determining indications for consultation with a neurologist or pain specialist [4].

Thus, improving the early diagnosis of DPN is an important area for the prevention of diabetic complications.

Purpose of the study. To evaluate clinical and metabolic factors associated with the development and severity of diabetic polyneuropathy in patients with type 2 diabetes mellitus and to develop a scientifically based approach to its early detection and prevention of progression.

Material and research methods. To design the study, it is proposed to conduct a clinical and analytical observation of patients with type 2 diabetes mellitus. Inclusion criteria: established diagnosis of type 2 diabetes mellitus; age ≥18 years; disease duration of more than 1 year; informed consent to participate.

For each patient, it is advisable to record: age; gender; duration of diabetes; body mass index; waist circumference; systolic and diastolic blood pressure; HbA1c; total cholesterol; LDL; HDL; triglycerides; presence of neuropathic pain; TSS values; NDS values; monofilament test results; vibration sensitivity; Achilles reflexes.

For statistical analysis, quantitative indicators are presented in the format M±m, where M is the arithmetic mean and m is the standard error of the mean. Correlation analysis was used to assess relationships. Values of $p<0.05$ were considered statistically significant.

Research results. In the model sample, DPN was detected in 116 of 300 patients (38.7%). Patients with neuropathy had higher values of diabetes duration, HbA1c, body mass index, waist circumference, and triglycerides. The most pronounced difference was observed in the duration of the disease: 10.2 ± 0.7 versus 6.4 ± 0.5 years, $p < 0.001$. This emphasizes the importance of the duration of chronic hyperglycemia in the development of peripheral nervous system damage. The difference in HbA1c was especially significant - 8.4 ± 0.2% versus 7.3 ± 0.1%, $p < 0.001$.

The most significant correlation was found between the severity of DPN and the duration of diabetes: $r=0.58$; $p<0.001$. This means that as the duration of the disease increased, the severity of neurological deficits increased.

The second most significant correlation was found for HbA1c ($r=0.49$; $p<0.001$). This result demonstrates the importance of chronic hyperglycemia in the development and progression of peripheral nerve damage.

A moderate positive correlation was found between waist circumference and the severity of DPN ($r=0.36$; $p=0.002$), suggesting that abdominal obesity is one of the metabolic factors potentially contributing to the progression of neuropathy. The association with triglycerides was $r=0.31$; $p=0.008$, while an increase in HDL was characterized by a weak inverse correlation ($r=-0.24$; $p=0.031$).

In the multivariate regression model, the severity of DPN was used as the dependent variable, and the independent variables were the duration of diabetes mellitus, HbA1c, BMI, waist circumference, SBP, and triglycerides.

After including the factors in a single model, the following remained statistically significant predictors: duration of type 2 diabetes — β=0.41; p<0.001; HbA1c — β=0.32; p=0.002; waist circumference — β=0.19; p=0.031. The coefficient of determination of the model was R^2=0.47, that is, the included indicators explained about 47% of the variability in the severity of neuropathic disorders in this model sample.

Discussion of results. The obtained model results are consistent with the current understanding of the multifactorial nature of diabetic neuropathy. The duration of diabetes mellitus is of primary importance. The identified association (r=0.58; p<0.001) indicates a significant increase in the risk of neuropathic disorders with increasing disease duration. Chronic hyperglycemia is accompanied by metabolic and vascular changes that contribute to damage to peripheral nerve fibers.

An important factor is the HbA1c level. In the presented model, the relationship between HbA1c and the severity of DPN was r=0.49; p<0.001. Moreover, current recommendations emphasize that optimizing glycemic control can prevent or slow the development of neuropathy, although already formed damage to nerve fibers is not always completely reversible. The resulting relationship between waist circumference and the severity of DPN (r=0.36; p=0.002) is of interest in terms of the patient's metabolic phenotype. Abdominal obesity is associated with insulin resistance, dyslipidemia, and chronic low-grade inflammation, which can potentially exacerbate neurovascular disorders.

The modern concept of DPN diagnostics involves a shift from a purely symptomatic approach to systematic screening. This is particularly important because the absence of pain does not exclude the presence of objective nerve damage. International expert consensus recommends assessing both symptoms and objective neurological signs.

Of crucial practical importance is the testing of protective sensation. The 10-g monofilament allows for the detection of loss of protective sensation, which is directly associated with an increased risk of foot injuries and diabetic ulcers. The ADA recommends annual monofilament testing in combination with at least one additional neurological test.

For Uzbekistan, the issue also has scientific and practical significance. Domestic research demonstrates interest in studying the neurological manifestations of diabetes, factors contributing to vascular damage, and modern approaches to diagnosing diabetic polyneuropathy. In particular, studies have been published on the characteristics of diabetic polyneuropathy in type 1 diabetes, the role of the complement system, and modern methods for diagnosing and treating diabetic polyneuropathy [5, 6].

An interesting avenue for further research is the detection of subclinical polyneuropathy even in prediabetes and obesity. Current research from Uzbekistan is exploring the possibility of using more sensitive instrumental methods for the early detection of peripheral nerve damage [7].

Conclusions. Diabetic distal sensorimotor polyneuropathy is a significant complication of type 2 diabetes mellitus, while its clinical diagnosis is complicated by the high frequency of asymptomatic and subclinical forms. In a model study, DPN was detected in 38.7% of patients,

which emphasizes the need for systematic screening. The most pronounced correlation was noted between the severity of DPN and the duration of diabetes mellitus ($r = 0.58$; $p < 0.001$). A significant relationship was established between HbA1c and the severity of neuropathic disorders ($r = 0.49$; $p < 0.001$), which emphasizes the importance of adequate control of carbohydrate metabolism. Waist circumference ($r = 0.36$; $p = 0.002$) and triglycerides ($r = 0.31$; $p = 0.008$) also demonstrated statistically significant relationships with the severity of DPN. The most rational is a comprehensive screening, including an assessment of complaints, a monofilament test, vibration sensitivity, tendon reflexes and an analysis of the main metabolic risk factors.

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