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FEATURES OF NEUROSENSORY DISORDERS IN PATIENTS WITH DIABETIC POLYNEUROPATHY AND THE POSSIBILITY OF NON-DRUG CORRECTION

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Abstract
The article presents the results of the rehabilitation of patients with diabetic polyneuropathy based on the complex use of electrical stimulation and vacuum exposure. The results of the study showed that the use of the developed rehabilitation technology improves the clinical effectiveness of the treatment of patients with diabetic polyneuropathy.

Keywords: diabetes mellitus, diabetic polyneuropathy, electrical stimulation, vacuum exposure.

Introduction

The social and economic significance of the problem of diabetic neuropathy can hardly be overestimated, since diabetes mellitus is one of the most common diseases of our time. The WHO estimates that more than 180 million people worldwide have diabetes and that number could more than double by 2030. Metabolic disorders in diabetes leads, one way or another, to the defeat of all organs and systems of the body. Most often, disability and reduced life expectancy in patients with diabetes are the result of late vascular complications, such as myocardial infarction, stroke, peripheral vascular disease (macroangiopathy), as well as retinopathy, nephropathy, neuropathy (microangiopathy). Diabetic neuropathy (the most common form of diabetic neuropathy is polyneuropathy) affects more than 50% of diabetic patients [1]. Polyneuropathies are characterized by diffuse damage to the nerve fibers that make up various nerves, and occupy a leading place among the neurological complications of somatic diseases. As a rule, clinical symptoms develop 5–10 years after the onset of the underlying disease; it is believed that at least 10% of patients have diabetes mellitus and are verified only after the manifestation of neuropathy [2]. Diabetic neuropathy usually presents with tingling, pain, numbness, or weakness in the arms and legs. Risk factors for the development of polyneuropathy in diabetic patients

include the duration of the disease itself, the level and significant fluctuations of HbA1c (glycosylated hemoglobin is an indicator of compensation for carbohydrate metabolism over the past 60-90 days) in the blood, dyslipidemia, high body mass index, albuminuria, hypertension and smoking [1].

Today, achieving stable normoglycemia is the first step in the treatment of diabetic neuropathy, which, nevertheless, is of great importance, as evidenced by the comparability of the incidence of neuropathy in patients with type 1 and type 2 diabetes. Thus, in the DCCT study (The Diabetes Control and Complications Trial Research Group, 2013), it was shown that adequate glycemic control led to a decrease in the incidence of new cases of polyneuropathy, and in patients with newly diagnosed polyneuropathy, against the background of stable glycemic control, regression was noted. clinical symptoms [1]. The subsequent study [2], which included the majority of DCCT participants, showed that previous long-term adequate glycemic control significantly improved the long-term prognosis, reducing the likelihood of developing polyneuropathy and other late complications of diabetes. However, in routine clinical practice, optimal and long-term carbohydrate metabolism compensation is achieved in a relatively small number of patients. Given the progressive nature of the disease, the possibility of using drugs that affect various parts of the pathogenesis of diabetic neuropathy is very relevant. In case of disorders leading to a decrease in the patient's quality of life, along with basic antidiabetic drugs, it is also recommended to use specific treatment of affected nerve fibers and microvessels. A typical lesion of peripheral nerves in diabetes is distal polyneuropathy. Metabolic changes predominantly affect sensory nerve fibers, resulting in paresthesia and pain. Patients are concerned about tingling, numbness, chilliness of the feet or a burning sensation, pain in the limbs. For several years, the painful symptoms appear mainly at rest, and then become more and more constant and intense. Usually, from the very beginning of the disease, it is possible to detect disturbances in pain, temperature and / or vibration sensitivity, decreased reflexes and movement disorders [3]. In addition to the fight against hyperglycemia, many authors associate certain prospects in the treatment of diabetic neuropathy with preventive therapy aimed at improving the metabolism of the nervous tissue. Actually metabolic therapy involves the use of drugs containing substances that are characteristic of the internal environment of the body and have a primary metabolic effect, i.e. affecting homeostasis, directly involved in biochemical processes as substrates, coenzymes, cofactors or other participants in metabolism, and not through regulatory mechanisms, like the vast majority of drugs. Usually, metabolic drugs are of auxiliary importance, but in neuropathies their role increases, since metabolic disorders are, in this case, an important link in

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pathogenesis [4]. Pathogenetically substantiated in the treatment of diabetic neuropathy is, therefore, the use of B vitamins, due to their specific neurotropic effect [2]. B vitamins are widely prescribed as a metabolic therapy to improve the function of peripheral autonomic nerve fibers, slow down the progression of complications and reduce the intensity of pain [1].

The following B vitamins are traditionally considered neurotropic - thiamine (B1), pyridoxine (B6) and cyanocobalamin (B12). These vitamins have a variety of metabolic and clinical effects, but they are united by a high significance for the normal functioning of the nervous tissue (Table 1).

Table. The main functions of thiamine (B1), pyridoxine (B6) and cyanocobalamin (B12) [4 each with changes]

metabolic processes	Neurotropic action	Other effects
Thiamine (B1)		
Participation in dehydrogenase complexes of the Krebs cycle. Dehydrogenation of branched-chain keto acids. Regulation of the activity of the pentose phosphate cycle.	Participation in the conduction of a nerve impulse. Security axonal transport, which determines the regeneration of nervous tissue. Modulation of neuromuscular transmission in n-cholinergic receptors. Regulation of "painful" activity of the nerve.	Antioxidant. Blockade glycation proteins. Immunomodulation due to lymphoprotective activity.
Pyridoxine (B6)		
Dez- and transamination amino acids. Decarboxylation amino acids. Phosphorylation glycogen. Participation in the metabolism of folic acid.	Ensuring synaptic transmission (participation in the synthesis of catecholamines, histamine). Ensuring the processes of inhibition in the central nervous system (participation in the synthesis of GABA).	Antiplatelet. Improving the absorption of magnesium from the gastrointestinal tract and its accumulation in the cell. Maintenance of hematopoietic processes.
Cyanocobalamin (B12)		
Stimulation of nucleic metabolism through the activation of folic acid. Participation in protein, carbohydrate and fat metabolism (indirectly). Hematopoiesis.	Participation in the synthesis of the myelin sheath. Reduction of pain associated with damage to the peripheral nervous system.	Activation clotting systems. Regulation of the function of the gastrointestinal tract.

When creating high concentrations of thiamine in the blood, its neurotropic effect is manifested by a decrease in pain associated with pathological processes in nerve fibers. Of particular importance in the treatment of complications of diabetes mellitus is the ability of high concentrations of thiamine to block protein glycation.

Vitamin B6 - pyridoxine - is a cofactor for more than 100 enzymes, affects the structure and function of the nervous tissue, primarily due to the ability to regulate the metabolism of amino acids, thus ensuring the normalization of protein metabolism and preventing the accumulation of excess amounts of neurotoxic ammonia. Optimization of the activity of the nervous system is additionally provided by the participation of pyridoxine in the synthesis of catecholamines, histamine and the inhibitory mediator of the central nervous system - gamma-aminobutyric acid (GABA). In addition, pyridoxine increases the intracellular reserves of magnesium, which plays an important role in metabolic processes and in the activity of the nervous system.

The number of patients with diabetes mellitus is progressively growing and in the Russian Federation is about 3 million [3, p.6]. One of the most common complications of diabetes is neuropathy and it is detected in 50% of patients. Almost all patients develop DPN at various times. The most common is chronic sensorimotor polyneuropathy, including 7-10% of patients with newly diagnosed type 2 diabetes [4, p.29]. The pathogenesis of DPN is multifactorial, one of the established factors is hyperglycemia, which has a damaging effect on the nervous tissue. Along with the metabolic theory, the vascular theory is also considered. Damage to the Vasa nervorum as a result of glycation of endothelial cells and impaired endoneurial circulation leads to a decrease in blood flow in the nerve, axonal atrophy, and degeneration of nerve fibers. Changes in the peripheral nerve are formed as a result of nerve ischemia, which develops in violation of the production of vasoactive relaxing agents (NO), endoneural hypoxia.

According to the WHO definition, DPN is a disease characterized by progressive death of nerve fibers, leading to loss of sensation and development of a foot ulcer [5, p. 98]. The main direction in the prevention and treatment of DPN is to achieve normoglycemia and maintain it for a long time. But this does not contribute to the rapid elimination of manifestations of DPN, and in order to improve the quality of life of patients, it is necessary to use complex rehabilitation, with the inclusion of non-drug methods of treatment. Physiotherapeutic factors are widely used, including electrotherapy. [1, p.244] Stimulation by low-frequency currents accelerates the regeneration of peripheral nerves and improves the functional properties of the

neuromuscular apparatus. A peculiar massaging effect of vacuum exposure promotes the release of toxic products from the intercellular space and reduces cellular hypoxia. Evaluation of the clinical effectiveness of including the technique of combined use of vacuum exposure and electrotherapy in the program of complex rehabilitation of patients with diabetic polyneuropathy was the purpose of this study.

For the correction of metabolic disorders that occur in diabetes mellitus, an anabolic function of the third neurotropic vitamin - cyanocobalamin (B12) plays an important role. In the treatment of diabetic neuropathy, the ability of vitamin B12 to restore the structure of the myelin sheath and reduce neurogenic pain is most significant. In the treatment of polyneuropathies, it is possible to use both each of the neurotropic vitamins individually and their complexes.

Vitamin Bi (thiamine). Like other water-soluble vitamins when taken orally, thiamine has low bioavailability [7], which cannot be compensated by increasing the dose, since the unique lipophilic substance with thiamine-like activity, benfotiamine, has the full effect of "saturation" [9, 10]. Benfotiamine, due to its lipid solubility, penetrates well through the blood-brain barrier, as well as through the lipophilic membrane of nerve cells. Inside cells, benfotiamine is rapidly converted to thiamine diphosphate, which promotes optimal glucose utilization. Thus, lipophilic benfotiamine has better pharmacokinetics, which determines its use with greater efficiency.

Vitamin B6 (pyridoxine). Pyridoxine is absorbed in the jejunum using a passive diffusion mechanism that does not have a saturation effect, and therefore the flow of pyridoxine into the blood depends on its concentration in the intestinal lumen.

Vitamin B12 (cyanocobalamin). For absorption in the intestines of doses of cyanocobalamin corresponding to the daily requirement, synthesized by the intestinal microflora and supplied with food, the internal anti-anemic factor of Castle is used. When higher than physiological concentrations of cyanocobalamin are created in the intestine, absorption is possible even without the participation of the Castle factor due to passive transport and pinocytosis. The long-term preservation of cyanocobalamin in the body is due, in particular, to the effect of the hepato-intestinal circulation.

Sensitivity indicators improved statistically significantly in the main group, which resulted in a change in temperature (from 2.0 ± 0.3 to 0.7 ± 0.2 points), pain (from 1.6 ± 0.3 up to 0.6 ± 0.2 points) and tactile sensitivity (from 0.7 ± 0.3 to 0.14 ± 0.1 points). In patients of the control group, there was a tendency to improve the index of temperature (from 1.2 ± 0.3 to 0.4 ± 0.1 points) and pain sensitivity (from 1.6 ± 0.4 to 0.6 ± 0.2 points) (Table 2).

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Table 2 Dynamics of indicators on the scale of the Neuropathic symptomatic score in patients with diabetic polyneuropathy (points)

Symptom	Main group n=21		Control group n=15	
	before	after	before	after
tingling	3,0 ± 0,34	0	6,0 ± 0,32	6,0 ± 0,31
burning	4,0 ± 0,33	0	6,0 ± 0,35	3,0 ± 0,33
numbness	21,0 ± 0,18	12,0 ± 0,23**	3,0 ± 0,15	0
pain	21,0 ± 0,2	3,0±0,18**	12,0 ± 0,18	9,0 ± 0,14
convulsions	18,0 ± 0,17	3,0 ± 0,33**	15,0 ± 0,67	6,0 ± 0,62**
DRR	3,2 ± 0,24	0,9 ± 0,21*	2,8 ± 0,22	1,6± 0,35

* - p<0.05; ** - p < 0.01; DRR - average total score.

With intramuscular administration of thiamine, pyridoxine and cyanocobalamin, their bioavailability is about 20%.

Wörwag Pharma presents on the market two highly effective neurotropic drugs that form a single course of treatment:

• Milgamma - an injectable preparation containing 2 ml (1 ampoule) 100 mg of thiamine and pyridoxine, as well as 1000 mcg of cyanocobalamin. The presence of 20 mg of lidocaine in the preparation and the small volume of the ampoule make injections practically painless, and the treatment is comfortable for the patient;

• Milgamma compositum is a combined preparation containing 100 mg of benfotiamine and 100 mg of pyridoxine in 1 tablet.

The course of treatment consists of 10 intramuscular injections of Milgamma and 6 weeks of oral administration of Milgamma compositum, 1 tablet 3 times a day. Preventive courses of Milgamma compositum are recommended to be carried out 2 times a year.

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