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ELECTRO-NEUROPHYSIOLOGICAL CRITERIA OF EFFECTIVENESS OF PATHOGENETIC THERAPY OF POLYNEUROPATHY

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Abstract
The problem of diabetic distal polyneuropathy (DDPN) is preconditioned by its great prevalence, according to specialized studies data, late diagnostics and serious consequences. The importance of DDPN diagnostics at its early stage doesn't generate doubts: just at this period disturbance of peripheral nerves is of reciprocal nature and the therapy is effective to the maximum degree. But the objectification of the DDPN diagnosis is difficult: the data of neurologic and electrophysiological tests often do not correspond to the clinical symptoms and signs as these methods evaluate lesions of large nervous fibers whereas in DDPN it's small fibers that are damaged in the first place. Use of skin biopsy and confocal corneal microscopy for evaluation of small fibers is extremely restricted in a clinical practice. Possibilities of electroneuromyography (ENMG) are analyzed as an option: parameters with their subjective peculiarities, selection of nerves and their number for DDPN diagnostics as a context of topographic neurology and neurophysiology of distal fibers.

Keywords: Diabetic polyneuropathy, benfotiamine, electroneuromyography, prediabetes.

Introduction

Peripheral nerve damage in diabetes mellitus (DM) is the earliest and most common of all complications. According to various authors, up to 90% of patients with DM of varying duration have this complication [1]. The basis of the pathogenesis of diabetic polyneuropathy (DPN) is a combination of microvascular disorders, endothelial dysfunction and pathological metabolic processes that lead to demyelination and degeneration of peripheral nerve fibers [2]. in particular, The process is characterized by latency, a long absence of clinical manifestations, due to which polyneuropathy is not diagnosed in a timely manner, and, as a result, the patient does not receive the necessary therapy. Conducting neurophysiological studies, electroneuromyography, allows diagnosing subclinical manifestations of diabetic polyneuropathy. Subclinical manifestations can be detected in patients with type 2 diabetes mellitus already at the stage of establishing the main diagnosis, and in some cases, polyneuropathy is the first manifestation of diabetes mellitus. When conducting a targeted examination of patients with newly diagnosed type 2 diabetes, polyneuropathy is diagnosed in 14-20% of patients [3]. capillaries to hyperglycemia [4]. Most often, thick myelinated fibers are affected in type 2 diabetes, which is clinically manifested by

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symptoms of distal symmetric sensorimotor polyneuropathy. The following stages of DPN are distinguished: - stage I (subclinical) - clinical signs of polyneuropathy are absent, changes in the peripheral nervous system are recorded only by special neurological tests.- stage II - clinical symptoms of polyneuropathy are verified by altered neurological tests.- stage III - severe defects in nerve function leading to severe complications. And Stage I II does not have pronounced neurofunctional disorders, stage III implies the presence of pronounced functional and organic disorders. This distinction is extremely important for clinical practice, since the patient's treatment tactics largely depend on the stage of DPN [4]. One of the important components of pathogenetic therapy of diabetic polyneuropathy is the use of a fat-soluble form of thiamine – benfotiamine. Evaluation of the efficacy and safety of benfotiamine in the treatment of complications of type 2 diabetes mellitus has been carried out in several large studies, including those using placebo control and neurophysiological criteria [5-8]. The basis of the pathogenesis of diabetic polyneuropathy (DPN) is a combination of microvascular disorders, endothelial dysfunction and metabolic processes that lead to demyelination and degeneration of peripheral nerve fibers [2]. pathological And Recently, the pathogenetic significance of immunological processes in the formation of DPN has been discussed. This theory is confirmed by the detection of antibodies to components of the sympathetic nerve ganglia, vagus nerve, and adrenal glands in patients with diabetes. The relatively early development and rapid progression of DPN and other micro- and macrovascular complications of diabetes may be due to genetic predisposition. It is possible that genetic differences are based on the peculiarities of proteoglycan metabolism, which increase the sensitivity of the basement membrane The main therapeutic effect of Benfotiamine is due to its ability to activate the enzyme transketolase, due to which the main mechanisms of hyperglycemic tissue damage are blocked [9]. The use of benfotiamine in sufficient dosage (at least 300 mg per day) and for sufficient duration allows for the alleviation of such subjective manifestations of diabetic polyneuropathy as pain and paresthesia, as well as objective ones – improvement of neurophysiological parameters, improvement of sensitivity [10]

The study involved three groups of patients divided by the duration of the underlying disease - type 2 diabetes mellitus (T2DM). Group 1 (n=20) - patients with a duration of T2DM from 1 to 3 years (from the time of diagnosis). Group 2 (n=20) - patients with a duration of T2DM from 3 to 7 years. Group 3 (n=20) - patients with a duration of T2DM from 7 to 10 years. The study included patients with a primary diagnosis of T2DM, with clinical and electrophysiological manifestations of distal polyneuropathy, in the absence of pathogenetic therapy in the last 6 months. The study did not include patients younger than 40 and older than 70 years, patients with somatic diseases that can cause Table 1 Age characteristics of groups Group Data SD experience, years Floor Age, years 1 N=20 1-3 M – 12 (60%) 46-54 Avg.49.8±2.78 standardized F – 8 (40%) All patients underwent a clinical and neurological examination at the initial appointment, assessment of vibration sensitivity with a tuning fork, electroneuromyography of the nerves of the lower extremities with an assessment of conduction along the motor and sensory branches of the peroneal and tibial nerves. After the examination, all patients were prescribed the same course of pathogenetic therapy, including alpha-lipoic acid (Thiogamma) - 600 mg by intravenous infusion for 10 days, then in tablets - up to 3 months + Benfotiamine 300 mg (Milgamma® Mono 300) - daily for 3 months. Control visits were conducted after 6 and 12 weeks of therapy. The criteria for

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effectiveness were: 1. Change in ENMG indicators – increase in the amplitude of evoked sensory potentials and conduction velocity along the nerves of the lower extremities (peroneal, tibial); 2. Reduction of the vibration sensitivity threshold; 3. Evaluation of the effectiveness of therapy by patients general impression scale. Statistical The processing was carried out using the statistical functions option of Microsoft Office Excel. The average indicators of the sample (group), standard square deviation, minimum and maximum values of the feature in the sample were calculated. The assessment of statistical reliability in groups was carried out using the Student's t-test for related samples. development of polyneuropathy (vitamin B12 deficiency anemia, paraneoplastic processes, regular alcohol consumption, etc.), patients with complicated forms of diabetic polyneuropathy (ulcer defects, non-traumatic amputations in the anamnesis.

The criteria for effectiveness were: 1. Change in ENMG indicators – increase in the amplitude of evoked sensory potentials and conduction velocity along the nerves of the lower extremities (peroneal, tibial); 2. Reduction of the vibration sensitivity threshold; 3. Evaluation of the effectiveness of therapy by patients general impression scale. Statistical The processing was carried out using the statistical functions option of Microsoft Office Excel. The average indicators of the sample (group), standard square deviation, minimum and maximum values of the feature in the sample were calculated. The assessment of statistical reliability in groups was carried out using the Student's t-test for related samples

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