Possibilities of the BRT method in the treatment of chronic diabetic distal sensorimotor polyneuropathy in elderly patients

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Diabetic neuropathy is a dysfunction of the somatic and / or autonomic peripheral nervous system, caused by the actual diabetic pathophysiological and dysmetabolic processes [16]. The incidence of diabetic neuropathy, according to various authors, varies from 40 to 95% of all cases of diabetes mellitus (DM) [9]. The pathogenetic basis of the formation of the syndrome of diabetic neuropathy is oxidative stress, which develops as a result of the formation of a large number of free radicals against the background of insufficiency of its own antioxidant systems, due to the activation of the thiol pathway of glucose metabolism, an increase in the activity of protein kinase C, the formation and accumulation of end products of non-enzymatic interaction of glucose with proteins, nucleotides and lipids (advanced glycation end products, AGEs),

Diabetic neuropathy is a factor that not only worsens the quality of life of patients with diabetes mellitus, but also an unfavorable prognostic sign in relation to the risk of disability and a reduction in their life expectancy. This is especially true for sick older age groups.

The most common type of diabetic neuropathy is chronic distal sensorimotor polyneuropathy. [12]. With this form of diabetic neuropathy, pain syndrome is observed in a quarter of patients [5]. Chronic pain syndrome in diabetic polyneuropathy is a variant of neuropathic pain and is characterized by a combination of positive and negative sensory phenomena. Typical complaints of patients are tingling and numbness in the feet and legs, aggravated at night. In some cases, patients experience sharp, shooting, lancinating, pulsating and burning pains. Allodynia and hyperesthesia are noted in some patients. All of the above disorders are referred to as positive sensory symptoms of neuropathic pain. Negative symptoms include pain and temperature hypesthesia, which in the initial stages of the disease are presented moderately in the distal regions, but as they progress, they spread proximally and may involve the hands. Tendon reflexes are usually diminished, and muscle weakness is limited to the muscles of the foot [1].

Elderly patients with complex somatic burden often have atypical manifestations of pain syndrome. As a reaction to persistent pain in elderly patients, depression and anxiety, limited social contacts, and sleep disturbances are often observed [3].

Purpose of the study: To study the effectiveness of bioresonance therapy (BRT) in patients type 2 diabetes mellitus with chronic distal sensorimotor polyneuropathy.

Materials and research methods

The study involved 64 patients with type 2 diabetes mellitus (insulin-dependent) with diabetic polyneuropathy at the stage of clinical neuropathy in the form of chronic sensorimotor neuropathy (distal symmetric polyneuropathy) according to Thomas PK [15], which corresponds to the chronic pain form of the stage of clinical neuropathy DPN according to the classification of the International Group experts in diabetic polyneuropathy [6]. 32 patients made up the main group in the treatment scheme of which, along with standard drug treatment, BRT was included using hardware-software

complex "IMEDIS-EXPERT". The control group consisted of 32 patients who received only standard drug therapy.

Table 1

Clinical characteristics of patients

Index	Main group (n = 32)		Control group (n = 32)	
Age, years	63.8 ± 2.4		61.8 ± 3.1	
Gender, n (%)	wives	husband.	wives	husband.
	20 (62.5%)	12 (37.5%)	21 (65.6%)	11 (34.4%)
Duration of SD	10.8 ± 1.3		10.1 ± 1.5	
Duration taking insulin	8.1 ± 1.3		8.6 ± 1.5	

Patients' complaints (pain, burning, numbness, paresthesia) were assessed using the TSS (Total Symptom Score) scale, which makes it possible to quantify the severity of DPN. To quantify the neurological symptoms (pathological changes in muscle strength, reflexes, sensitivity) identified by the doctor, the NIS (Neuropathy Impairment Score) scale was used [4]. The overall assessment of the manifestations of diabetic polyneuropathy was expressed in points. In addition, a psychometric study of patients was carried out using the Hospital Anxiety and Depression Scale (according to Zigmond AS, Snaith RP, 1983; Wade D., 1993; Bevz I.A.)

BRT sessions were held every other day. The number of sessions was limited by regression of complaints and electropuncture diagnostics data. The number of BRT sessions was 12 ± 1.8.

results

In the course of treatment, the positive dynamics was more clearly manifested in the main group, where pain syndrome, paresthesia and dysesthesia regressed faster, the severity of motor disorders, as well as psycho-emotional disorders, decreased (Table 2).

table 2

Dynamics of clinical manifestations of diabetic polyneuropathy and psychometric indicators

Index	Main group (n = 32)		Control group (n = 32)	
Control time	Before treatment	After treatment	Before treatment	After 28 days
TSS scale	10.5 ± 0.62	1.3 ± 0.41	9.7 ± 0.74	4.1 ± 0.31 *
NIS scale	17.2 ± 0.53	2.8 ± 0.36 *	15.9 ± 0.8	5.7 ± 0.55 * #
Depression scale	11.2 ± 0.27	4.9 ± 0.22 *	10.3 ± 0.85	9.7 ± 0.91 *
Alarm scale	9.1 ± 0.58	2.3 ± 0.75 *	9.4 ± 0.47	8.7 ± 0.69 #

Note:

* - significant differences within the group ($p < 0.001$),

- significant differences between the main and control groups ($p < 0.01$).

conclusions

The inclusion of BRT in the complex treatment of chronic distal sensorimotor polyneuropathy in elderly patients with type 2 diabetes mellitus makes it possible to accelerate the onset of clinical improvement in the form of regression of subjective (complaints) and objective symptoms, to reduce the drug load by eliminating analgesics and psychotropic drugs, thereby reducing the risk of developing therapeutic iatrogenies. and significantly improve the quality of life of this category of patients.

Literature

1. Anisimova E.I., Danilov A.B. Neuropathic pain syndrome: clinical neurophysiological analysis. Journal. Nevrol. Psychiatrist. Them. S. S. Korsakova, 2003, 103 (10): 15–20.

2. Zotova E.V., Chistyakov D.A., Savostyanov K.V. et al. Study of the association of polymorphic markers Ala (-9) Val of the SOD2 gene and Arg213Gly of the SOD3 gene with diabetic polyneuropathy in patients with type 1 diabetes mellitus // Molecular Biology. –2003. No. 3. –S. 345-348

3. Levin O.S. Chronic pain management problems in the elderly. Journal. Treatment of diseases nervous system, 2010. No. 2 (4), 11–18.

4. Strokov I.A., Novosadova M.V., Barinov A.N., Yakhno N.N. Clinical assessment methods severity of diabetic polyneuropathy // Neurological journal. –2000. –№ 5. –C. 14-19.

5. Strokov I.A., Pegasova O.M., Morgoeva F.E. Clinic and treatment of pain forms diabetic polyneuropathy // Russian medical journal. –2004, 2, 23 - 27.

6. Boulton AJM, Gries FA, Jervell JA: Guidelines for the diagnosis and outpatient management of diabetic peripheral neuropathy. Diabet Med 15: 508-514, 1998.

7. Caselli A. Validation of the nerve axon reflex for the assessment of small nerve fiber dysfunction // J. Neurol Neurosurg Psychiatry. –2006; 77: 927-932.

8. Gorson KC, Ropper AH Additional causes for distal sensory polyneuropathy in diabetic patients. // J Neurol Neurosurg Psychiatry. - 2006; 77: 354-358.

9. Martin CL Neuropathy among the Diabetes Control and Complications Trial Cohort 8 years after trial completion. // Diabetes Care. - 2006; 29: 340-344.

10. Nosikov VV, StrokovI A., Nikitin A. G. et al. Poly (ADP-ribose) polymerase-1 gene (PARP1) involved in the genetic susceptibility to diabetic polyneuropathy in Russian patients with Type 1 diabetes mellitus // Abstr. of the 14th Annual Scientific Meeting of the Diabetic Neuropathy Study Group of the EASD (NEURODIAB). 2004. –P. 138.

11. Orstavik K. Abnormal function of C-fibers in patients with diabetic neuropathy. // J Neurosci. - 2006; 26: 11287-11294.

12. Oyibo S, Prasad YD, Jackson NJ, Jude EB, Boulton AJM: The relationship between blood glucose excursions and painful diabetic peripheral neuropathy: a pilot study. Diabet Med 19: 870–873, 2002

13. Ryle C, Donaghy M. Non-enzymatic glycation of peripheral nerve proteins in human diabetics. J Neurol Sci. Mar 1995, 129 (1), 62-8.

14. Tesfaye S., Chaturvedi N., Eaton SE (EURODIAB Prospective Complications Study Group). Vascular risk factors and diabetic neuropathy // N Engl J Med. - 2005; 352: 341-350.

15. Thomas PK: Classification of the diabetic neuropathies. In Textbook of Diabetic Neuropathy. Gries FA, Cameron NE, Low PA, Ziegler D, Eds. Stuttgart, Thieme, 2003, p. 175-177.

16. Vinik AI, Mehrabyan A. Diabetic Neuropathies. // Med Clin North Am. - 2004; 88: 947-999.

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