Journal of Advanced Zoology

ISSN: 0253-7214

*Volume **44** Issue **Special Issue-2 Year 2023** Page **3687:3691***

METHODS OF TREATMENT OF DIABETIC POLYNEUROPATHY

Xodjiyeva Dilbar Tajiyevna
Axrorova Shahlo Batirovna
Bukhara State Medical Institute named after Abu Ali ibn Sino, Uzbekistan

Article History
Received: 08July2023
Revised: 27 Sept 2023
Accepted: 29 Oct 2023

Abstract: *Normoglycemia is a key condition of DPN prophylaxis. Research has shown that against the background of 5 years of intensive insulin therapy, DPN is known to have a significant decrease in the frequency of development (by 64%), a decrease in the frequency of detection of nerve fiber conduction disorders (by 44%) and a decrease in the development of autonomic dysfunction (by 53%) [5, 11]. It should be remembered that each patient has a "glycemic ulcer". Pathological processes when the Anashu bulge increases cascade reactions are triggered and lead to the development of DPN [4, 6, 7]. But its duration is considered more important than the degree of hyperglycemia. Prolonged retention of normoglycemia in patients with severe levels of DPN has been found to cause some delay in progressive damage to the peripheral nerves, which is certainly very important, but does not contribute to the recovery of the damaged nerve [13, 14, 15]. Therefore, Additional symptomatic treatment is required to improve the quality of life of patients, especially in the presence of pain syndrome[9].*

Keywords: medications, treatment, regular glycemic control

.

There are many medications used to treat DPN, but despite this, treatment of most forms of neurological symptoms associated with DPN remains problematic [8, 9]. The DPN treatment strategy should be based on symptom relief and improving the quality of life of these patients. This is achieved through regular glycemic control, as well as pharmacological correction [6].

The most important and basic condition for treating polyneuropathy observed in diabetes is to achieve normal blood glucose levels using oral or insulin drug drugs that control blood glucose levels. The dose of the drug prescribed by the attending physician should always remain unchanged. Maintaining glucose levels in moderation is the most important factor in preventing the further development of polyneuropathy. Neuropathic pain that develops in diabetic neuropathy develops differently compared to pain in other nerve lesions. Therefore, the effect of commonly used pain relievers is not felt. Various groups of controlled, prescription drugs are widely used to reduce the degree and intensity of pain.

Symptom-altering drugs (do not affect the course of the pathological process)

Anticonvulsants are more effective in acute and severe pain, they stabilize the condition of the nerve fibers and reduce neurological pain. The means of this group have a complete analgesic effect in neurological lesions and are widely prescribed as first-line drugs for the treatment of neuropathic pain.

The mechanism of analgesic action of antidepressants is explained by the fact that it reduces serotonin levels in the central nervous system and is also likely to affect the body's opioid systems. Pain relieving plastics and topical gel and grease mazes can also be used as topical anesthetics. Narcotic analgesics have a positive effect on neuropathic pain, but for obvious reasons their recommendation is limited, with narcotic drugs being used in hospital settings when necessary. These drugs do not affect the course of the disease, they only help to reduce symptoms and improve the quality of life of patients with diabetic polyneuropathy. In addition to drug preparations with a symptomatic mechanism of action, there are several drugs that affect the mechanisms of development (pathogenesis) of diabetic neuropathy. The earlier treatment with them begins, the more accurate the result will be. Lipoic acid preparations are used for this purpose, which is an antioxidant and neuroprotective agent with a complex mechanism of action. Some other drugs (aldoreductase inhibitors, nerve tissue growth factors, linoleic acid and carnitine drugs) are currently in the clinical trials phase and have not yet entered wide practice.

Fibrates (phenofibrate), which represent hypolipidemic agents, affect lipid metabolism, normalize cholesterol fractions, and also reduce glucose concentration. Reduces the risk of developing diabetic neuropathy and prevents the development of pathology. Group v vitamin agents, pentoxifylline, and other conventional agents that affect the metabolism of nerve tissues are additional drugs used in the treatment of DPN.

Physiotherapy procedures are used at various stages of DPN to achieve analgesic, neuroprotective, regenerative effects, as well as improve blood circulation and restore nerve sensitivity (improve permeability), improve the nutrition of nerve fibers and surrounding tissues.

Electrotherapy is prescribed in order to improve tissue nutrition. Electrophoresis is also a physiotherapeutic treatment with the property of introducing drugs under the influence of direct current flow, which has a good analgesic effect. In diabetic polyneuropathy, drugs such as nicotinic acid, sodium thiosulfate, prozerin, vitamins V are used using electrophoresis treatment.

Today, combined physiotherapeutic methods are effectively used in photolaser and magnetolaser therapy. In diabetic polyneuropathy, magnetotherapy has analgesic, angioprotective and neuroprotective effects, helps restore nerve fibers and improves blood supply and nutrition of adjacent tissues. At the beginning of the course of therapy, the pain is significantly reduced, the sensitivity of the leg improves, and muscle activity increases. With this method, it is possible to achieve a significant improvement in the condition of the nerve fibers and accelerate the recovery processes. In addition to the effectiveness of the method, it should also be noted its high safety. Magnetotherapy diabetic polyneuropathy can also be used in complex treatment in patients of different ages, including children and adolescents, and older patients with many comorbidities.

Electrostatic and pneumatic massage of the lower mucosa, balneotherapy and acupuncture therapy are again common, among other non-predicamentous methods.

Diabetes and its complications (diabetic polyneuropathy and angiopathy, diabetic foot syndrome, retinopathy — retinal damage) are the most dangerous diseases today, which can not only reduce the quality of life, but also directly threaten it. With the help of modern methods of treatment of medicamentosis and nomedicamentosis, it is possible to achieve good results, control the disease and prevent serious consequences.

The study selected patients with Type 1 diabetes mellitus who received regular inpatient treatment in the Departments of the Endocrinology dispensary of the Bukhara region and were on account in the consulting Polyclinic. The examination included patients aged 18 to 60 years with Type 1 diabetes and a group of healthy volunteers. The average age of patients was 33.2 ±1.68. A total of 157 patients in our observation were studied in 4 groups, and the control group consisted of 40 volunteer people made up of practically healthy people. The examination group includes all patients with Type 1 diabetes, regardless of the presence or absence of positive neuropathic symptoms (Table 1). The average duration of the disease was 12.5±10.1 years (up to 43 years from newly diagnosed diabetes). The average level of glycosylated hemoglobin (na 1 s) was 8.3 ± 1.9% (4.2 to 13.8%).

Clinical-neurological examination methods

A number of clinical trials have been conducted to determine neurological deficits in patients.

Patient complaints, Anamnesis, assessment of the general condition of the lower mucosa (Status localis) (condition of the skin of the feet; areas of hyperkeratosis; deformity of the legs);

Neurological examination: determination of the surface and deep sensitivity bladder, pay reflexes, different muscle strength in the arms and legs.

To determine the clinical stages of diabetic neuropathy, Dyck P.J., Thomas P.We used the DPN weight class classification proposed by K (1999) [6].

Classification of the DPN by weight (Dyck P.J.)

DPN stages	Description
0 (DPN «-»)	Clinical and neurophysiological symptoms of DPN are not observed.
1 Subclinical stage (DPN 1)	Objective neurological signs and symptoms are not observed, but when enmg and quantitative autonomic tests are examined, different 2 different changes are detected.
2 Clinical stage (DPN 2)	DPN-specific complaints, sensory, movement and vegetative system disorders are observed. In patients, lower mucosa flexor muscle function may be impaired. The patient cannot stand on his heels.
3 Heavy stage (DPN 3)	There is a violation of labor capacity or social adaptation.

Neuropathic complaints were collected for the diagnosis of DPN symptoms. The level of temperature, vibration, tactile, pain sensitivity was assessed in the patient. Knee and Achilles reflexes were studied. Typical positive neuropathic symptoms included complaints such as numbness in the legs, burns, stinging pain, paresthesia. These complaints and symptoms developed mainly at night. To determine the characteristics of the pain, attention was paid to certain signs, such as the time of the appearance of pain, increased pain when moving ECI at a calm time. In DPN, pain appears in a calm state.

Subjective signs of DPN were evaluated using the TSS (Total symptoms Score) Scale (Table 2) [4, 14]. On this scale, each neuropathic positive symptom is assessed in intensity and frequency over the last 24 hours (stinging pain, Association, burns, parastezii).

1jadval. Positive neuropathic symptomlarni baxolash TSS (Total Symptoms Score) scales (1994).

Symptomlar frequencies	Symptomlar of the Intensive League (Score)			
	Symptoms are not observed	Light level	Middle level	Heavy level
Not observed	0	1	2	3
Quickly observed	0	1,33	2,33	3,33
Always observed	0	1,66	2,66	3,66

The intensity of each symptom can be manifested at 4 degrees: symptom intensity is not observed, mild intensity, moderate intensity, severe intensity. The frequency of symptoms is assessed in 3 criteria: poorly observed, rapidly observed, always observed symptoms. Each

symptom varies from 0 to 3.66 in points. All positive signs in patients were evaluated on a scale and the sum was calculated. The sum of points on the TSS scale was 15.44 points out of 0.

To assess negative neuropathic symptoms (neurological deficits), the NIS LL scale (Neuropathy Impairment Score Low Limbs - expression of neurological disorders in the lower mucosa) was used

Muscle strength was assessed as follows: 0 - normal muscle strength; 1 - muscle strength decreased by 25%; 2 - muscle strength decreased by 50%; 3 - muscle strength decreased by more than 50%; 4 - active movements are not observed, paralysis.

Reflexes were assessed as follows: 0-norm; 1-dropped; 2 - not called. When sensitivity was tested, 0-norm; 1 - decreased; 2-intuition was assessed as unobserved.

Surface sensitivity was examined in conditions where the patient was lying on his back, with his eyes closed, calm and muscles relaxed.

The vibration sensation was examined using neurologically calibrated Camerton (128 Hz). On both legs of the Camerton, there is a shaft from 1 to 8, with a light stroke, vibration is created, and the main phalanx of the thumb of the legs is touched to the dorsal area. The patient is asked for a time when the feeling of vibration is lost. The check was determined from 3 times on both sides, and the average indicator of the results was obtained. The tip of the pain sensation was examined using a blunt needle. Pain sensitivity detection was detected on the back of the legs in the area of the last phalanx of the thumb. Pain sensitivity is believed to be maintained if the patient feels the same sharp touch at the distal and proximal points of the foot. Pain sensitivity is considered to be reduced if the patient experiences less pain sensitivity at distal points. The inability to distinguish a sharp touch from a blunt touch was assessed as a lack of sensitivity.

Tactile sensitivity was tested using 10 g of monofilament. The examination was carried out in the area of terminal phalanges on the plantar surface of the legs. The Monofilament was affected by bending the Strand at a 90 degree angle. Tactile sensitivity was considered to be maintained if the patient felt touch at all points. If the patient made several mistakes, the sensitivity was considered reduced. If the patient did not feel the touch sensation, the sensitivity was assessed as absent.

Proprioreceptive intuition was examined with eyes closed in the lying position in patients, passive movements were performed in the terminal phalanges of the toes, the patient should describe in which finger what movement is taking place its direction (up or down). If the patient correctly described the action performed by the neurologist, the proprioreseptive – muscular joint sensation was considered intact. If the patient made several mistakes, deep intuition was considered reduced. Deep intuition was assessed as lost if the patient could not describe passive movements.

Pay reflexes were learned in the usual way. Knee reflexes were checked in the position where the patient was sitting: reflexes were called with a light tattoo under the patellar area with a neurological hammer when the legs were freely suspended from the calf area and placed at right angles to the thighs. In this, the intensity of the contraction of the four-headed muscle of the thigh was assessed.

The Achilles reflex was examined by striking Achilles Pai successively with a neurological hammer. To call the reflex, the patient sits on the chair with his knee, and his legs should be freely hung on the edges of the chair seat. In this, the intensity of the contraction of the calf muscle was assessed.

On the NIS LL scale, the sum of the scores of the right and left leg verification results was calculated. On a scale, the sum of negative neuropathic symptoms was estimated at up to 28 points.

In addition, temperature sensitivity was studied using a special instrument in the form of a cylinder (thermostesiometer, Germany), the ends of which are made of materials with different thermal conductivity (metal and plastic). In the norm, the metal side is felt cold, the plastic side is felt warm. The study shows that the skin of the feet and thumbs is alternately affected by the cold and hot tip. If the patient separates the cold and warm sensation at 5 points, the temperature

sensitivity is considered intact. If the error is 1 or more, the temperature sensation is reduced and the temperature sensation is considered lost if the patient is unable to distinguish 5 effects.

References:

1. Akmaev, I.G. Neuroimmunoendocrinnie vzaimodeystviya: experimentalnie I klinicheskie aspect / I.G. Akmaev / / sakharny diabetes. - 2012. - № 1. - S.2-9.
2. Alekseev, L.P. Immunogenetics saxarnogo diabeta pervogo tipa (SD - 1) / L.P. Alekseev, I.I. Dedov / / material IV Vseros. Kongr. endocrinologov. - Spb., 2011. - S. 10.
3. Alekseev, L.P. Mejpopulyasionny podhod v viyavlenii assosiasiy Genov HLA ClassA s insulinzavisimim sakharnim diabetom / L.P. Alekseev, A.B. Zilov, M.N. Boldireva [I dr.] / / Quickly. docle. 1-go Ros. diabetologicheskogo Kongr. - M., 2010. - S. 16.
4. Alekseeva, L.P. Hla - geni-marker insulinzavisimogo saxarnogo diabeta, ethnicheskie aspect / L.P. Alekseev, I.I. Dedov, M.N. Baldirev [I dr.] / / Immunology. - 2013. - № 5. - S. 308-311.
5. Albrant, E.V. Osobennosti immunnogo status I metabolism lymphositov krovi U detey I podrostkov s SD-1 / E.V. Albrant, A.A. Savchenko, V.T. Manchuk / / Pediatrics. - 2014. - № 3. - S. 19-22.
6. Ametov, A.C. Antioxidants therapy diabeticheskoy polyneuropatii / A.C. Ametov, I.A. Strokov, P.P. Samigullin / / Rus. med. magazine. - 2015. - № 6. - S. 3-7.
7. Antsiferov, M.B. Immunoglobulini I insulinsvyazivayutshaya sposobnost T-lymphositov pri SD 1 i 2 tipa / M.B. Antsiferov, M.I. Arbuzov, A.A. Pereligina [I dr.] / / Sov. media. - 2019. - № 7. - S.25-28.
8. Arion, V.Ya. Itogi nauki I technical / V.Ya. Arion / / Immunology. - 2014. - T.9. - S. 10-50.
9. Akhmedova, Sh.U. Sostoyanie pokazateley kletochnogo immunity U detey v debute saxarnogo diabeta 1 tipa i na fone insulinoterapii / Sh.U. Akhmedova, G.N. Rakhimova, D.A. Rakhimova [I dr.] / / Immunology. - 2013.- № 1.- S. 51-54.
10. Balabolkin, M.I. Sakharny diabetes / M.I. Balabolkin. - M.: Media, 2014. - 383 P.
11. Balabolkin, M.I. Pathogenesis sosudistix oslozhneniy sakharnogo diabeta / M.I. Balabolkin / / Fast. docle. 1-go Ros. diabetologiche-skogo Kongr. - M., 2018. - S.36.
12. Balabolkin M.I. Diabetology / M.I. Balabolkin-M., 2018. - 672 P.
13. Balabolkin M.I. Diabeticheskaya neuropathy / M.I. Balabolkin, V.M. Kreminskaya / / Journal neurologii I psychiatrist. - 2019. - № 10.- S. 57-64.
14. Bashina V.M. Povishenie urovnya autoantitel k faktoru rosta nervov v sivorotke krovi detey, bolnix schizophrenie / V.M. Bashina, I.A.Kozlova, T.P. Klyushnik [I dr.] / / Experimentalno - teoreticheskie problem. - 2017. - № 1. - S. 47-51.
15. Bogatova, O.V. Razlichnie assosiasii Mejdu na I insulinzavisi-mim sakharnim diabetom v dvux gruppax russkix iz razlichnix oblastey evropeyskoy chasti Rossii / O.V.Bogatova, I.A. Guskova, E.G. Grudakova [I dr.] / / Immunology. - 2012. - № 2. - S. 192.
16. Bondar, T.P. Laboratorno-Klinicheskaya diagnostics sakharnogo diabeta I ego oslozhneniy / T.P.Bondar, G.I. Cosines. - M.: Mia, 2013. - 87s.
17. Bursa, T.R. Kriterii diagnostic diabeticheskoy polyneyropatii pri populationnom issledovanii / T.R. Bursa, I.A. Strokov, M.V. Novosadova [I dr.] / / Problem endocrinologii. - 2014. - T. 50, № 1. - S.9-13.
18. Vartanyan, H.JI. Immunologicheskie marker v diagnostics saxarnogo diabeta 1 tipa / N.L. Vartanyan, T.A. Dubinina, N.B. Serebryanaya / / Med. immunology. - 2013. - № 3-4. - S. 248-249.
19. Vlasova, M.S. Osabennasti vegetativnay nervnay system u detey I padrostkov, bolnix SD tipa 1 / M.S. Vlasova, O.I. Wotyako - WA, A.I. Ribkin [I dr.] / / Quickly. docle. 3-go Vseros. diabetologicheskogo Kongr. - M., 2014. - S.530-531.
20. Vozianov, V.K. Tsitokini: Biologicheskie i protivoopuxolevie svoystva / V.K. Vozianov, K.P. Zack, A.K. Butenko. - Kiev: Naukova dumka, 2018.-312 P.