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CLINICAL AND DIAGNOSTIC ASPECTS OF DIABETIC POLYNEUROPATHY AND THE IMPLEMENTATION OF THERAPEUTIC MEASURES

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ABSTRACT

Diabetic polyneuropathy occupies one of the leading places among non-infectious diseases of mankind. According to the World Health Organization (who), more than 40 million people(1-4% of the population) suffer from diabetes around the world and 7-10% are aged 65 years and older people. Risk factors for diabetes are obesity, low physical activity, hyperlipidemia (abnormal increase in lipid levels), hypertension, decreased glucose tolerance, heredity for diabetes, and changes in pregnancy.

Keywords

Diabetes mellitus, immunological, cytogenetic,hematological methods, multisystems, hyperlipidemia, hypertension, hyperalgesia.

The phenomenon of neuropathic pain in DPN is explained both by the processes of direct damage to nerve fibers and by the interaction of two mechanisms at different levels of the nervous system: nociceptive (responsible for the perception of pain stimuli) and antinociceptive (painkillers). Much attention is paid to the study of central sensitization (increased sensitivity), as a result of which the phenomenon of neuropathic pain is formed and hyperpathy (increased reaction to stimuli) and allodynia develop [2].

Like other pathologies of the peripheral nervous system, all diabetic neuropathies can be conditionally divided into the following:

focal (with damage to individual nerves): mononeuropathy (damage to one nerve), radiculopathy (damage to spinal roots), plexopathy (damage to nerve plexuses);

multiple, or multifocal, neuropathies (damage to several individual nerves);

diffuse (polyneuropathies) — damage to all nerve fibers of any area of the body.

In diabetes mellitus, the most common form of damage to the peripheral nervous system is precisely polyneuropathy.

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DPN according to the topographic principle:

Distal polyneuropathy mainly affects the feet, and then the hands. This species also has several variants:

sensory (damage to sensitive nerve fibers);

motor (damage to motor neurons);

mixed (sensorimotor);

vegetative (affecting the nerves of various organs).

Proximal, predominantly asymmetric motor neuropathy. It mainly affects the muscles of the thighs and buttocks with the development of weakness and pain syndrome in them.

According to clinical manifestations:

Reversible polyneuropathy (transient hyperglycemic, acute pain, sensory);

Progressive neuropathy (distal sensorimotor polyneuropathy, proximal motor neuropathy, autonomic neuropathy) [8].

The stages of development of the most common chronic distal sensorimotor diabetic polyneuropathy are distinguished:

Stage 1 — subclinical neuropathy — is characterized by the absence of clinically pronounced symptoms, can be diagnosed in specialized neurophysiological centers.

Stage 2 — clinical neuropathy — clinical manifestations depend on the form of polyneuropathy and correspond to mild or moderate DPN. If we consider chronic DPN, then the most characteristic is the presence of symptoms that worsen in the evening: burning, tingling, acute or piercing pain, lack or impaired sensitivity, decreased or absent reflexes. In the case of acute DPN, the main symptom is pain, hyperesthesia, allodynia may be present, sensitivity disorders are minimal or absent. In the pain-free form, the absence of pain is combined with a decrease or absence of sensitivity and a decrease or absence of tendon reflexes.

Stage 3 — severe polyneuropathy — is characterized by a pronounced defect in sensory or sensorimotor functions (with the possible development of disability), the development of vegetative manifestations, severe pain syndrome and complications of DPN: trophic ulcers, neuroarthropathy, diabetic foot[10].

Осложнения диабетической полинейропатии

Complications of diabetic polyneuropathy include neuropathic deformity of the feet with the development of neuroarthropathic "Charcot's foot" and diabetic foot syndrome, complications of which may lead to the need for amputation.

Diabetic foot is characterized by dry skin, areas of hyperkeratosis and the development of painless ulcerative defects in areas of excessive stress pressure on the sole. Ulcerative defects develop due to a violation of pain sensitivity: minor injuries (for example, a violation of the integrity of the skin and the formation of

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calluses as a result of improper shoe selection) may go unnoticed, ulcers form in case of infection.

In the case of ischemic ulcers (due to insufficient arterial blood supply), the temperature of the feet changes, the pulse becomes asymmetric, skin peeling appears, hair loss, deformation and dullness of nails, intermittent lameness, the limb changes color depending on the position: it becomes pale when raised and cyanotic (cyanotic) when lowered [5].

In addition to ulcerative defects, a specific deformation of the fingers and joints (Charcot's neuroarthropathy) may form [3]. Diabetic Charcot's neuroarthropathy is manifested by the gradual formation of subluxations and dislocation of bones with subsequent healing. Its formation is associated with a violation of innervation (connection with the central nervous system), blood supply and the development of weakness of the ligamentous apparatus of the feet. In addition, Charcot's neuroarthropathy increases the likelihood of injury due to a violation of bone density and the formation of fragile, "defective" joints, unstable to repeated injuries. Most characteristic of diabetic osteoarthropathy is the lesion of the interphalangeal tarsal-metatarsal joints.

In the diagnosis of diabetic polyneuropathy, its detection at an early stage is important. In the future, this will improve the quality of life of patients and prevent early disability. The diagnosis of DPN consists in the collection of anamnesis, high-quality clinical examination and the use of modern instrumental examination methods.

Despite the wide possibility and accessibility of neurophysiological examination methods, special attention is paid to clinical examination. Do not forget that the absence of symptoms does not indicate the absence of neuropathy — DPN can often be asymptomatic in the early stages. An important role in the preclinical or early clinical stages is played by specific, special screening tests that exist to detect early manifestations of diabetic polyneuropathy:

The study of vibration sensitivity using a graduated 128 Hz tuning fork, which is applied to the bony part of the distal phalanx of the big toe and the inner ankle.

Research of tactile sensitivity using monofilament. This is a special tool with a retractable monofilament. To perform the test, the instrument is installed perpendicular to the surface under study (the area of the back of the toes, the area of the inner ankles, shins, knees). When touching the skin, the working monofilament should normally bend, which indicates the presence of a reaction. The absence of a reaction indicates a decrease in sensitivity.

Using scales and questionnaires to identify polyneuropathy. There are specific scales for studying muscle strength in the feet, Achilles reflex and pain sensitivity — NISS-LL (Neuropathy Impairment Score Low Limb — assessment of neuropathy

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in the lower extremities) and for determining the severity of pain, numbness and paresthesia — TSS (Total Symptom Score — general assessment of symptoms) [3].

When diagnosing the most common variant of DPN — diabetic distal symmetric sensorimotor polyneuropathy — a clinical assessment of existing symptoms is carried out:

disorders of surface sensitivity in the legs, which can manifest as pain, burning and numbness;

reduction of reflexes and disorders of deep sensitivity (vibrational and tactile) due to damage by the pathological process of thick myelinated fibers.

There are various instrumental techniques for studying nerve function:

Thick, fast-conducting nerve fibers and their function are examined using electroneuromyography (ENMG). With the help of ENMG, it is possible to detect a decrease in the speed of motor and sensory nerves in the early stages of the development of diabetic polyneuropathy. It is carried out by applying electrodes to the skin. Fine nerve fibers are examined using quantitative sensory and autonomic testing (CT and KW, respectively) [9].

All patients with diabetes mellitus should be examined for DPN as part of an annual screening, regardless of the presence and intensity of complaints. The examination includes a study of deep sensitivity using a tuning fork and an assessment of superficial, in particular pain and tactile, sensitivity using a monofilament. Screening examination helps to diagnose DPN at an early stage, which reduces the likelihood of complications and further disability of patients.

Differential diagnosis of DPN with other polyneuropathies (PNP), which may also be a symptom of other diseases, should be carried out:

Chronic inflammatory demyelinating polyradiculoneuropathy.

Alcoholic PNP.

Toxic PNP (including poisoning with heavy metal salts).

Medicinal PNP.

Amyloid PNP (associated with the deposition of amyloid protein).

Paraneoplastic PNP (damage to peripheral nerves on the background of malignant tumors).

Dysmetabolic (uremic, dystyroid, vitamin B12-deficient porphyriac) PNP and others. Treatment of diabetic polyneuropathy

In diabetes mellitus and the development of diabetic polyneuropathy, first of all, it is necessary to normalize the patient's blood glucose level. Currently, it has been established that normalization of glycated hemoglobin (HbA1) levels should precede drug therapy for DPN.

Non-drug therapy includes: dieting, therapeutic gymnastics and maintaining physical activity. In the complex of non-drug measures, the following are

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mandatory: correction of cardiovascular risk factors (hypertension, hypercholesterolemia, obesity, smoking), which can contribute to both the earlier development and more severe course of complications of diabetes mellitus.

Drug therapy includes symptomatic and pathogenetic therapies. Among the drugs for the symptomatic therapy of diabetic polyneuropathy, preference is given to drugs that relieve pain. The main groups of drugs are:

Anticonvulsants — pregabalin and gabapentin in appropriate analgesic dosages.

Antidepressants with analgesic effect. The serotonin and norepinephrine reuptake inhibitors (SSRIs), duloxetine and venlafaxine, have proven effectiveness. The drug amitriptyll (tricyclic antidepressant), despite a fairly wide range of contraindications and side effects, with proper dosing and titration of doses at the beginning of administration, has also shown its effectiveness in the treatment of neuropathic pain, which is noted in patients with DPN.

Among the drugs for pathogenetic treatment, according to most studies, alpha-lipoic acid preparations are the most effective. To achieve the maximum positive effect, two-stage treatment should be used:

At the first stage, alpha-lipoic acid preparations are administered intravenously for two to three weeks.

At the second stage, drugs of this group in the form of oral administration contribute to a prolonged therapeutic effect, which significantly reduces the clinical manifestations of diabetic polyneuropathy: both subjective in the form of regression of pain intensity, paresthesia and numbness, and objective in the form of improvement of vibration sensitivity and electrophysiological characteristics of nerve fibers [8]. Of the local anesthetics, transdermal therapeutic forms of 5% lidocaine are used to treat DPN pain syndrome.

High-frequency spinal cord stimulation (10 kHz) can relieve pain in patients with diabetic neuropathy who are not helped by drug therapy. Treatment reduced pain by an average of 77% in 86% of patients [6].

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