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APPROACHES TO THE DIAGNOSIS OF POLYNEUROPATHY CAUSED BY SOMATIC DISEASES

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Abstract: Syndromic diagnosis of polyneuropathy does not present any difficulty, but nosological diagnosis can be extremely difficult. The paper deals with the principles of diagnosis of polyneuropathy, giving the clinical picture of the most frequent forms of polyneuropathy caused by physical illness. The basis of treatment is that of neuropathies somatogenic underlying disease that led to the development of neuropathy. High doses of B vitamins that accelerate reparative processes in the nerve fiber can be beneficial in most forms somatogenic polyneuropathy.

Key words :polyneuropathic syndrome, diagnosis, diagnostic algorithm, treatment of polyneuropathy, intravenous immunoglobulin, intravenous immunotherapy, efficacy, safety, somatogenic polyneuropathies, B vitamins, neurobion.

Among the neurological complications of somatic diseases, polyneuropathic syndrome occupies a leading place. It is often the defeat of the peripheral nervous system that causes disability and disability of this category of patients and, as a result, a violation of the quality of life. The spectrum of the most common somatic diseases leading to damage to the peripheral nervous system.

The symptoms of polyneuropathies are caused by simultaneous damage to a larger or smaller number of peripheral nerves. Therefore, the symptoms of polyneuropathy consist of a common sensory and motor deficiency, loss of tendon reflexes and further muscular atrophy. In general, the complete polyneuropathic syndrome is a complex of sensory, motor and vegetative symptoms.

Diagnosis of somatogenically caused neuropathies.

The syndromic diagnosis of polyneuropathy does not present any difficulties. On the contrary, establishing the nosological affiliation of neuropathy can cause objective difficulties. Even with the most thorough examination in specialized clinics, the cause of polyneuropathy remains unknown in about 25% of cases, and in non-specialized nosological diagnosis 50% of patients do not have [1]. To help in establishing. A nosological diagnosis can be a correct descriptive syndromic diagnosis. It is useful to carry out a descriptive analysis of the clinical picture of neuropathy in the following three clinical categories:

According to the prevailing clinical signs, which depend on the predominant lesion of one or another type of fiber that makes up the peripheral nerve. The peripheral nerve consists of thin and thick fibers. All motor fibers are thick myelinated fibers. Proprioceptive (deep) and vibrational sensitivity are also conducted along thick myelinated fibers. On the contrary, fibers that transmit pain and temperature the feeling is unmyelinated and thinly myelinated. Both thin and thick fibers take part in the transmission of tactile feeling. Vegetative fibers are thin, unmyelinated.

•Damage to thin fibers can lead to selective loss of pain or temperature sensitivity, paresthesia, spontaneous pain in the absence of paresis and even with normal reflexes. An example of a relatively isolated lesion of thin fibers It is a diabetic symmetrical distal neuropathy with damage to thin fibers, manifested by an unusual combination of symptoms and characterized by an unfavorable prognosis. Clinically, this form of diabetic neuropathy usually manifests symptoms of vegetative insufficiency in combination with burning, shooting pain, hyperalgesia, paresthesia, decreased pain and temperature sensitivity, foot ulceration and decreased visceral pain sensitivity [2].

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•Neuropathy of thick fibers is accompanied by muscle weakness, areflexia, and sensitive ataxia. An example of this type of neuropathy is the easily recognizable diabetic distal sensorimotor neuropathy. Damage to vegetative fibers leads to the appearance of somatic symptoms. The involvement of all fibers leads to mixed sensorimotor and vegetative polyneuropathy. In addition, it is diagnostically important to determine the substrate of the lesion: axonopathy and/or myelinopathy. The lesion substrate is most accurately determined using electroneuronomyography. Most somatically conditioned neuropathies are axonopathies, although the involvement of segmental demyelination as an additional damage factor is also described.

According to the distribution of the lesion: distal/proximal and symmetrical/asymmetric limb lesion. In most cases, somatically caused polyneuropathies are manifested by distal symmetrical sensory or motor disorders of the extremities. Demyelinating neuropathies are characterized by a symmetrical predominantly proximal lesion of the extremities. Multiple mononeuropathy, on the contrary, is characterized by an asymmetric proximal lesion. Polyneuropathy is also distinguished with predominant involvement of the upper extremities and predominant involvement of the lower ones limbs. The latter option significantly prevails in frequency of occurrence among neuropathies associated with somatic diseases.

By the nature of the course: acute (the development of symptoms

occurs within a few days to 4 weeks); subacute (for several weeks); chronic (for several months or years). Recurrent polyneuropathies are chronic forms. The acute onset is characteristic of the toxic, vascular, or immune etiology of polyneuropathy. Most toxic and systemic diseases develop subacutely in for several weeks or months. Finally, some neuropathies of metabolic origin can develop extremely slowly (years).

Thus, the syndromic diagnosis of polyneuropathy should include the nature of the course, the distribution of the lesion and the predominant clinical symptoms, for example, "subacute symmetrical distal sensory neuropathy".

The algorithm of nosological diagnosis of polyneuropathic syndrome also includes electrophysiological examination and biochemical studies of cerebrospinal fluid, blood and urine. Stimulation electroneuromyography is the most informative. To determine the character (axonopathy or myelinopathy) and the level of peripheral nerve damage, it is important to study the rate of excitation along the motor and sensory fibers of peripheral nerves.

The most common somatic neuropathies

Diabetic neuropathies are the most common variant of somatic neuropathies. Damage to the peripheral nervous system in patients with diabetes occurs in 20-40%. As a rule, clinical symptoms of neuropathy develop 5-10 years after the onset of the underlying disease. But in at least 10% of patients, the diagnosis of diabetes is verified only after the onset of neurological deficiency.

The individual combination of clinical signs and symptoms in diabetic neuropathy varies widely. However, it is possible to group the symptoms into characteristic syndromes, which seems to be very justified for a better description of the clinical picture. The following clinical syndromes are most often distinguished:

• Distal symmetrical sensorimotor diabetic polyneuropathy.

• Proximal motor diabetic neuropathy (diabetic amyotrophy).

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• Mononeuropathy in diabetes.

• Cranial nerve neuropathy in diabetes.

• Damage to the autonomic nervous system in diabetes.

According to the algorithm presented above, all these syndromes can be divided into diffuse or symmetrical polyneuropathies (sensory, motor and vegetative) and focal neuropathies (mononeuropathies, multiple mononeuropathies, plexopathies, radiculopathies and cranial neuropathies). The importance of this approach is due to the difference in pathogenetic mechanisms and therapeutic approaches.

Distal symmetrical sensorimotor neuropathy is the most common variant of damage to the peripheral nervous system in diabetes. It usually develops several years after the onset of the underlying disease.

This form develops slowly (chronically), the first symptoms appear in the lower extremities, sometimes unilaterally. In some patients with predominant damage to the thin fibers of the peripheral nerves an excruciating pain syndrome develops. Severe forms of polyneuropathy occur in patients with early onset diabetes (juvenile forms) and poorly controlled diabetes. In the most severe forms of polyneuropathy, loss of proprioceptive sensitivity can lead to sensitive ataxia, a pseudotabetic form.

Unlike diffuse forms of neuropathy, focal forms develop acutely or subacutely, and the main damaging mechanism in these forms is ischemia. Among the cranial nerves, the oculomotor nerves, III and VI, are most often affected. Proximal asymmetric diabetic neuropathy is less common than distal forms. The clinical picture is characterized by an acute onset and dominance of pain symptoms, which often worsens at night. Usually, the pain is localized proximally and affects the lower extremities more than the upper ones. At the same time, muscle weakness occurs with subsequent atrophy, which makes it difficult for the patient to climb the stairs. Elderly people with type 2 diabetes are mainly affected, with the peak incidence occurring at the age of 65.

Vegetative disorders in diabetic patients are usually associated with other neurological deficits, but may be presented in isolation from other symptoms. The most common symptom is sphincter dysfunction, manifested by sphincter insufficiency or atony of the bladder, attacks of diarrhea, especially at night, and impotence. Other symptoms of peripheral autonomic insufficiency include tachycardia, orthostatic hypotension, swelling of the feet and joints, and dry skin.

Uremic polyneuropathy occurs in chronic renal failure. Predominantly sensory, symmetrical distal disorders are characteristic. The disease can debut with crampy and restless legs syndrome. Then dysesthesia, burning and numbness of the feet are added. Sometimes uremic polyneuropathy is called neuropathy with "hot feet" syndrome. There is a positive effect of hemodialysis on the course of neuropathy. At the same time, 25% of patients on dialysis have symptoms of neuropathy. Arteriovenous fistula associated with dialysis, It can lead to focal ischemic neuropathy of the median nerve.

Neuropathies in systemic diseases are primarily caused by vasculitis. Among the diseases of this group, neuropathies are most often found in nodular periarthritis (in 25% of patients), rheumatoid arthritis (in 10% of patients). Neuropathies associated with systemic vasculitis are usually sensory (with a pronounced pain component and/or spontaneous pain) mononeuropathies or asymmetric polyneuropathies with acute or subacute onset [3]. Symmetrical sensory or sensorimotor polyneuropathies are less common.

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Polyneuropathies caused by exogenous causes, They make up 1/4 of all neuropathies. Such causes may include stimulants, medicines, industrial poisons and other substances. The scope of this work does not allow us to consider the entire spectrum of exogenous neuropathies, so we will focus only on certain types.

Alcoholic polyneuropathy ranks 2nd in frequency after diabetic neuropathy among somatic neuropathies. Clinical manifestations of peripheral nervous system lesions in patients suffering from alcoholism (alcoholic polyneuropathy) occur, according to various authors, in 12.5–29.6% cases [4]. Previously, it was believed that the development of alcoholic polyneuropathy causes primarily an alimentary deficiency of vitamin B1 (thiamine), due to a monotonous, unbalanced, predominantly carbohydrate diet. However, the toxic effects of alcohol cannot be reduced to a single mechanism or process. Nevertheless, thiamine levels should be assessed in every patient with suspected alcoholic polyneuropathy.

Phenomenologically, alcoholic polyneuropathy is most often a usually symmetrical distal sensorimotor neuropathy, which is based on axonal degeneration. However, the spectrum of nerve fiber damage may include different patterns.

Alcoholic neuropathy can be aggravated by vitamin B12 deficiency. Cobalamin deficiency neuropathy usually occurs suddenly, and primarily affects the arms or arms and legs together. The defeat of the neuropathic process of the upper extremities should alert the clinician in relation to B1-, B12-deficient conditions. In B12-deficient conditions, polyneuropathy can be combined with myelopathy, which sometimes serves as a key to diagnosis. On the other hand, Clinically, it can be difficult to determine whether sensory symptoms are caused by myelopathy or polyneuropathy. An electroneurophysiological examination, including somatosensory evoked potentials, can be useful in differential diagnosis.

Many medications have neurotoxic side effects, among which the most common is damage to the peripheral nervous system (polyneuropathy). A classic variant of drug neuropathy It is an isoniazid polyneuropathy. This sensorimotor neuropathy occurs as a result of vitamin B6 deficiency caused by isoniazid in individuals with a genetically determined metabolic disorder of this vitamin. The administration of pyridoxine together with isoniazid made it possible to practically rid tuberculosis patients of this type of neuropathy.

Neuropathy can be a dose-limiting side effect of most drugs used in the treatment of life-threatening conditions such as cancer, HIV infections. Epidemiological studies confirm data from earlier reports that cytostatics are the cause of axonal sensorimotor neuropathy or, less often, damage to thin fibers in some patients [5]. The prognosis of drug neuropathies is unfavorable, since the withdrawal of drugs does not lead to an improvement in symptoms neuropathies. Antiviral drugs can lead to sensory neuropathy. A whole range of lesions of the peripheral nervous system is characteristic of HIV infection, it is caused by the immunodeficiency virus itself, metabolic disorders and the neurotoxic effect of antiviral therapy.

Neuropathies associated with HIV infection include: distal symmetrical sensorimotor polyneuropathy, toxic (drug) symmetrical sensory neuropathy, inflammatory demyelinating polyneuropathy (proximal symmetrical sensorimotor polyneuropathy), multifocal mononeuropathy and progressive polyradiculopathy [6].

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