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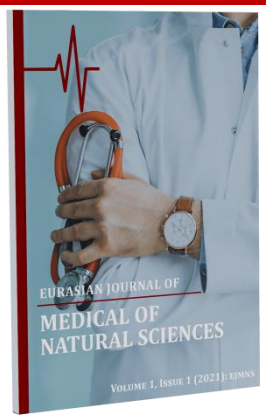

CLASSIFICATION AND CLINIC OF DIABETIC POLYNEUROPATHY

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ABSTRACT

The studies carried out show that only 25% of patients with Type 1 diabetes complain of neurological symptoms at the reception of a doctor. Patients diagnosed with DPN were observed in 46% of cases, and it often depended on the duration of the disease. In cases with a duration of Type 1 diabetes of less than 5 years, the prevalence of DPN was 2.4%, at a duration of 5-10 years - 28.2%, and when the disease lasted more than 10 years, the incidence of DPN was 69.4%. 10.8% of patients with Type 1 diabetes were found to have decreased vibrational sensitivity, and 31.1% to have decreased or lost Achilles reflex. The most common neurological symptom has been found to be cleavage, burns, and muscle strains of the calf muscles.

There are different variants of DPN, which vary depending on the clinic, pathogenesis, histological changes, treatment susceptibility and prognosis [1]. DPN is the most common complication of diabetes mellitus. 70% of the complications it gives to the peripheral nervous system correspond to distal symmetric polyneuropathy [7]. There are various clinical classifications of DPN: 1. Hyperglycemic polyneuropathy; 2. Distal sensor-autonomous polyneuropathy. 3. Acute polyneuropathy (amyotrophy, cranial mononeuropathy, compressive neuropathy, toracoabdominal neuropathy, Mucha mononeuropathy); 4. Mixed forms [1].

Focal neuropathies include mononeuropathy and tunnel syndromes. Mononeuropathy is often observed in older patients. Clinically, the symptoms are acute and develop rapidly. It usually occurs with a symptom of pain. The pathogenesis of the disease is manifested by infarction of nerve endings caused by thrombotic obstruction of the affected nerve interneural tissue [3]. Tunnel neuropathies in diabetes are usually observed with damage to the intermediate nerve, elbow nerve, wrist nerve, thigh nerve, ankle nerve. Damage to these nerves begins slowly and has a progressive character. Tunnel neuropathies can occur in patients with QD as a result of various trauma in the area of anatomical channels, metabolic disorders, nerve tissue inflammation, and causes such as edema [1].

Diabetic amyotrophy, Diabetic Neuropathic cachexia, son neuropathy are included in proximal motor neuropathies. They are often observed in older patients and have the character of progressing and starting. Patients complain of pain and muscle weakness that develop in the thigh, buttocks areas. When clinically examined, patients find it difficult to move from a sitting position to an upright position. The muscles were percussed spontaneous flights are detected in the thigh muscles. Proximal neuropathies develop with autoimmune disorders, an increase in antithelolar titer relative to nerve tissue, vasculites of an inflammatory nature, and demyelinating neuropathies [8].

The most commonly affected type of nervous system in QD disease is distally symmetric, senso motor – "classical" polyneuropathy. In this type of DPN, sensory and motor fibers with thin myelin, medium and large myelin are damaged. Damage to thin myelin-free fibers is damaged from the initial periods of the disease and can occur without clinical symptoms and ENMG changes. In the distal areas of the axon of the long nerves, the intraneural blood system circulates, resulting in the loss of sensory and motor nerve function [12]. In the DPN, sensory disturbances are usually observed in the distal part of the mucosa of the "clawed and sock-shaped" type. As DPN develops, individual types of sensation in the lower mucosa cause impairment and decreased reflexes. Initially, the sensory fibers are damaged. Movement disorders are manifested in the form of muscle atrophy, weakness in the arms and soles of the feet, and develop especially in the late stages of DPN. Damage to the small nerves occurs with pain sensations of a superficial nature, allodynia, painful burns, followed by symptoms of hypoalgesia. An early symptom may be a decrease in knee and Achilles reflexes and a decrease in vibrational sensation in the toe. As the disease progresses, symptoms such as paresthesia, sense of association, Frostbite of flies, followed by anesthesia and vegetative disorders continue to be added [16]. Such neurological stages can occur for several months. In some cases, DPN symptoms begin with intense intensity pain, dramatically reducing patients ' ability to work, disrupting daily activity and leading to insomnia [7]. The origin of pain is explained by the fact that peripheral nerves are affected by the nociceptive neurons of the dorsal nodes under hyperglycemic conditions for a long time [8]. In this case, the pain signal spontaneously generalizes to the entire nervous system and stimulates the system, resulting in intense intense pain [2]. In DPN, the pain becomes different. Most often, pain is characterized by constant burning, itching, Association. In rare cases there is a sharp, cutting, stinging character [2]. In studies, pain syndrome has been observed in 11.6% of cases in low-end flies, and in 7.1% of cases in high flies [2]. Due to the fact that usually the pain is accompanied by damage to the long nerve fibers, the heel spreads along the surface of the palm of the foot, which can then also spread to the proximal parts of the mucosa. The pain can be called or spontaneous. The pain called includes hyperalgesia and allodynia. Hyperalgesia occurs as a result of a painless effect over a long period of time. Allodynia usually occurs through an effect that does not call for pain (allodynia caused by cold or hot exposure). In this case, the skin is extremely sensitive and pain sensations can be triggered even with mild effects. The pain is usually felt a lot at a calm time and intensifies in the evening. Sleep is disturbed in patients due to nighttime pain [8]. Pain can sometimes disappear spontaneously for months. Such a condition can indicate the restoration of nerve function, but the opposite can also happen, that is, the development of a pathological

condition leads to the disappearance of the sensation of pain. The nature of the pain also depends on the pathological process lesion and the type and caliber of the affected nerve. Pain usually occurs in axonal neuropathy, when the fine nerves are damaged. Therefore, pain syndrome is often accompanied by disorders of pain and temperature sensation and thus vegetative-trophic lesions [2].

Diabetes mellitus is introduced into multi-factor polygenic diseases. Based on modern views, the role of metabolic disorders and the genetic factor in the development of QD is almost the same. Patients with Type 1 diabetes mellitus are considered to have a genetic predisposition that has an ethnic dependence on the development of DPN. The development of DPN among North African patients has been found to be more frequent and more severe than patients of European origin [3].

In recent years, progress has been made regarding the identification of candidate genes in the study of the genetic predisposition of microcirculation to various multifactorial diseases, including DPN.

If the pathogenetic mechanisms of the disease are accompanied by the participation of peptides, enzymes and structural proteins, then the candidate genes that encode these proteins are also responsible for these processes. Major advances in candidate gene studies have been associated with the introduction of polymerase chain reaction (PZR) (Polymerase chain reaction) technology to genetic research [8]. In 5% of cases in studies, blood vessels in patients without dependence on the stage of compensation complications have been identified. Based on genetic predisposition, 20-25% of patients have less pronounced complications. In most patients (70% - 75%), the genetic predisposition level changes indefinitely. It is in this group of patients that good control of carbohydrate metabolism inhibits the development and progression of diabetic complications [9]. Identifying genetic markers of susceptibility to various diseases is becoming the fastest growing field of Molecular Genetics. In this case, gene properties are studied and given different definitions of its properties: gene polymorphism is the evolutionary multivariate of the same gene, the qualitative and quantitative properties of its protein product can be manifested in the composition of the enzyme, its specificity, proximity to the substrate, activity and concentration; polymorphic genetic marker is a variable part of DNA that can be connected to; A candidate gene is a gene in which an expression product (enzyme, hormone, receptor, structural or transport protein) can be directly or indirectly involved in the development of the pathology under study; a genotype is a set of paired alleles of a gene or its marker; a disease phenotype is a set of external manifestations of clinical heterogeneous pathology. The fusion of the phenotype with the genotype is a significantly unequal distribution of the frequency of the appearance of the marker among individuals with different phenotypes. Among people with a certain pathology in relation to healthy people, a high prevalence of marker indicates a predisposition or increased risk, and a low one indicates a decrease in risk in relation to the disease.

Genetic predisposition to the development of DPN in Type 1 diabetes is due to the hereditary assignment of certain alleles of simple "healthy" genes. Sometimes these alleles that detect a predisposition to the development of DPN in Type 1 diabetes and are associated with the disease are called etiological mutations or variants. Etiological variants are common

in the population, but each of them does not lead to the development of the disease on its own. Only the presence of a certain combination of etiological options in a number of genes that determine the predisposition to the disease and its complications can lead to physiological diseases, which are manifested in the development of DPN in Type 1 diabetes mellitus.

Thus, for each multifactorial and Polygenic pathology, a certain range of candidate genes is determined, and the predisposition of the polymorphism of these genes or their protection against the development of any disease is studied [9]. The genotypes Z-2 allele, Z/Z — 2, and Z-2/Z-2 have been shown to positively associate the UK population with the development of DPN in the European population. At this time, DPN was less developed in Z±2 allele carriers [9]. In the Russian population with DPN-complicated QD in Moscow, the Pro72Arg and S(-594)SS genes TR53, t (- 78b)s gene NOS3, GPX3 of g4077a gene, GNB3 of s825t gene, G(-25)ANT1 of a gene, G(-605)T gene PEOl, t(-365)s gene POLG1, Val762Ala, and Leu54Phe gene the distribution of polymorphic markers and alleles of adprt1 genotypes has been studied [15].

In recent years, a number of new loci associated with Type 1 QD have been found, including complete genome searches using high - density microchips.

R53 protein plays an important role in maintaining genome stability and its regulation of transcription by interacting with many cellular proteins. It is discovered that DNA damage causes a large number of R53 protein folding. This protein blocks progression of the cell cycle in G1fase and prevents DNA damage from passing from replication to replication. If it is not possible to restore the lesion, that is, if no reporation is observed, the R53 protein triggers the mechanism of apoptosis (Kastan et al, 2001) in stresses and cell damage, the activity and amount of the R53 protein increases. At the same time, R53, which is in stressful conformation, mainly loses activity that promotes DNA recombination or recovery (Copnin, 2001, Chumakov, 2000) such protein r53 effectively activates or reverses much more stable and specific target genes, leading to cell cycle arrest or induction in abnormal cells of apoptosis. Oxidative stress plays a major role in the development of microvascular complications of QD. Antioxidant enzymes protect against the rapid development of diabetic polyneuropathy (DPN) by reducing oxidative stress. Thus, genetic changes affecting the activity or expression levels of antioxidant enzymes can lead to DPN.

Ala(-9)Val in the SOD2 gene and Arg213Gly polymorphic markers in the SOD3 gene have been examined indicating the likelihood of DPN origin in patients of Russian ethnicity with Type 1 QD.

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