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MODERN METHODS OF THERAPY OF VARIOUS FORMS OF OPTIC NERVE ATROPHY

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Abstract. *Optic nerve atrophy (ONA) can be considered as the outcome of diseases of the optic nerve of various origins. Decreased visual function in patients with ASD persists for many years and significantly impairs the patient's quality of life. Various methods of treating subatrophy or atrophy of the optic nerve (ON) are aimed at the rehabilitation of patients with this pathology. Currently, ASD is one of the severe diseases of the optic nerve, which leads to irreversible low vision and blindness. The level of disability in patients with AD is on average 13.24% of the total pathology of the organ of vision [1]. The importance of the problem is due to the general increase in the number of patients with atrophies of various origins, given that in most cases they occur in young patients [2]. In most patients with ON pathology, there is a significant progression of the pathological process, as well as an increase in the severity of the disease, which subsequently leads to irreversible changes in visual functions.*

Keywords: *ADHD is a degenerative disease in which damage to the retina occurs at the level of ganglion cell axons, as well as destruction of the myelin sheath of the ON to the level of the lateral geniculate body, which leads to impaired conduction of impulses to the brain [3, 4].*

Etiology , forms, severity of ASD

The vascular component plays an important role in the pathogenesis of the disease. In the work of F. Rose, it was revealed that a long-term violation of blood circulation in the vessels subsequently causes ischemia and damage to the optic nerve. Subsequently, in the ON, the damaged nerve fibers are replaced by neuroglia, which directly contributes to the development of AD. Due to the replacement of individual nerve fibers with connective tissue, abundant infiltration and proliferation of glial cells occurs, which can lead to damage to the myelin sheath of the optic nerve itself [4].

Another factor causing the development of AD is hypoxia. In the work [5], K. Golnik studied the histological state of the nerve against the background of oxygen starvation that occurs in AD and revealed the expansion of pial septa and subarachnoid space of the dura mater. NP Lyamina and BG Pilyavsky [6] described the metabolic processes that can contribute to the disruption of the transmission of nerve impulses and the development of AD in hypoxia. An important factor in the development of nerve pathology is the degeneration of the optic nerve fibers. So, in the work of G.N. Kryzhanovsky, it was shown that in the presence of a defect in the myelin sheath, the conduction of a nerve impulse is disrupted and areas of demyelination appear, which become places of ectopic excitation [7].

Traumatic damage to the optic nerve can also lead to AD. Compression most often occurs at the level of the exit of the optic nerve into the cranial cavity; chiasm is also a dangerous place [8].

AD can occur against the background of atherosclerosis of the carotid arteries due to malnutrition of the ON. As a result of changes, an ischemia focus is formed, around which growth of glia and connective tissue and thinning of the nerve fibers of the ON are noted. In the study of

patients with sclerosis of the internal carotid artery, changes in visual fields are revealed by the type of nasal hemianopsia and central scotoma [9].

In recent years, there has been an increase in the number of vascular lesions of the ON and retina. Common causes of AD are acute anterior and posterior ischemic optic neuropathies, primary open-angle glaucoma, and occlusion of the central retinal artery [10, 11]. Optic nerve atrophy can develop as a result of systemic diseases of the cardiovascular system, diseases of the central nervous system, intoxication of various etiologies, congenital and hereditary diseases, as well as as a result of traumatic brain injury and orbital injury [12–14].

Clinical Manifestations, Classification and Diagnosis Features of ASD of Different Genesis

The main manifestations of optic nerve atrophy are decreased visual acuity, changes in the fundus, and narrowing of the visual fields. In most cases, patients with AD have impaired color perception, mainly in the green-red part of the spectrum [15].

Patients often complain of vision loss with segmental or diffuse blurring of the visual field. To make a diagnosis, attention should be paid to the history of the disease, as well as to the data of physical and instrumental examination. It is recommended to clarify the family history, as well as the presence of injuries, systemic and chronic diseases.

Depending on the etiology of the optic nerve lesion, visual acuity can either remain unchanged or decrease up to absolute blindness. In patients with ADN, with damage to peripheral nerve fibers, vision do not deteriorate or deteriorates slightly, while damage to the papillomacular bundle leads to a pronounced visual impairment.

One of the methods for diagnosing the disease at an early stage is computerized perimetry. As a rule, changes in visual fields depend on the level of damage to the ON and have a certain localization. Thus, if the papillomacular bundle of the nerve is damaged, central scotoma occurs, if there is a defect at the level of the chiasm, bitemporal hemianopia is noted, and if the visual tracts are damaged, homonymous hemianopsia is noted [16]. Narrowing of the visual fields by the type of hemianopsia also occurs when the intracranial part of the optic nerve is damaged [17].

In addition to functional indicators of the state of the optic nerve, the anatomical structures of the anterior segment of the eye are assessed during the diagnosis of AD. Biomicroscopy reveals an afferent pupillary defect on the side of the lesion (decrease in direct pupillary reaction to light while maintaining a consensual pupillary reaction) [18]. Depending on the degree of damage to the ON, one of three variants of the ophthalmoscopic picture can be observed: with initial atrophy, the optic disc (ON) has a pale pink color and clear boundaries; with incomplete atrophy, blanching of the optic nerve is visualized with a decrease in the number of vessels passing through the edge of the ON (symptom of Kestenbaum), and with complete atrophy, there is a total blanching of the optic nerve, vasoconstriction, while Kestenbaum's symptom is pronounced [19].

To clarify the diagnosis and further follow-up of patients with AD, in addition to standard examination methods, it is possible to use special techniques, such as optical coherence tomography, Heidelberg retinal laser tomography, fluorescein angiography, and electrophysiological studies (visual evoked potentials).

The work of R. Burk and H. Völcker revealed that in patients with AD there is a thinning of the nerve fiber layer of the ONH, a decrease in the area and volume of the neuroretinal rim of the ON, and an increase in the area of ON excavation [20].

When performing fluorescein angiography in patients with AD in the arterial phase, hypofluorescence of the ONH is noted due to insufficient blood supply, while in ischemic lesions, narrowing of the arteries and a decrease in the number of capillaries of the ONH are noted.

To date, there is no generally accepted unified classification of ASD. Approaches to the treatment and assessment of the severity of AD are also heterogeneous. The following provides information on several ADS classifications.

The causes of AD can be divided into several groups:

1. Congenital optic neuropathies. These include dominant and recessive optic atrophy, Leber's hereditary optic neuropathy, Beer's hereditary atrophy.

2. AD associated with systemic disease or neurological conditions.

3. AD that occurred against the background of external compression of the ON due to pituitary adenoma, intracranial meningioma, aneurysms, craniopharyngioma, mucocele and papillomas, as well as metastasis in the brain.

4. AD resulting from tumors of the optic nerve, such as optic nerve glioma, optic nerve sheath meningioma, and lymphoma.

5. Vascular diseases: anterior and posterior ischemic optic neuropathy, central retinal artery occlusion, carotid artery occlusion, giant cell arteritis.

6. Inflammatory diseases: demyelinating optic neuritis (multiple sclerosis, Devic's disease), sarcoidosis, systemic lupus erythematosus, polyarthritis nodosa, Churg-Strauss syndrome, meningitis, orbital cellulitis.

7. ADS against the background of infectious diseases such as syphilis, tuberculosis, Lyme disease, aspergillosis, cryptococcal infection, chicken pox, measles, mumps.

8. Toxic optic neuropathies, as well as neuropathies due to nutritional deficiency: toxic amblyopia, endocrine ophthalmopathy, juvenile diabetes, smoking and alcohol, as well as atrophies that occur against the background of vitamin B deficiency. acute onset and symmetry of the process.

9. AD that occurred against the background of a traumatic impact on the ON: these are hematomas of the optic nerve sheaths, damage due to a fracture of the orbit, or the presence of a foreign body in the orbit.

10. ADS against the background of retinal diseases and conditions resulting from optic nerve edema: retinitis pigmentosa, age-related macular degeneration.

Also, ADS are divided depending on the severity of the process. The criteria for assessing the condition of the ON and dividing patients into stages are the visual acuity and field of view, and the ophthalmological picture is also of no small importance. According to the classification proposed by L.F. Linnik (1994) [16, 21], deterioration in visual acuity and narrowing of visual fields can be divided into five stages. The initial stage is characterized by a visual acuity of at least 0.4 with a relative decrease in photosensitivity in the central zone within 20°. With the progression of AD, visual acuity decreases up to the complete absence of visual functions, and a concentric narrowing of the visual fields occurs with the preservation of residual fragments of the peripheral visual field.

According to the topical level of the lesion, ASD is divided into ascending and descending. In the case of a pathological process at the level of the retinal ganglion cell layer, one speaks of an ascending character, and in case of damage to the optic nerve, of a descending one [23].

Optic nerve atrophy resulting from damage at different levels relative to the chiasm is divided into peripheral (before the chiasm), central (after the chiasm), and total — with damage to the chiasm and optic tracts [24].

In 2002 A.G. Duginov, E.E. Ioileva proposed a classification of the etiology of AD. According to the results of the work, the pathologies of the ON are divided into: AD that occurred against the background of CNS dysfunction (brain tumors, congenital atrophies, consequences of traumatic brain injury, orbital trauma), AD in the pathology of the retina (retinal abiotrophy and macular degeneration), AD due to vascular pathology eyes (occlusion of the central vein or artery of the retina and their branches), as well as acute neuropathies (anterior ischemic neuropathy, etc.) [25].

At present, great importance is attached to the clarification of the nosological cause of the occurrence of AD in order to determine the subsequent correct tactics of pathogenetically substantiated treatment.

Medications for the treatment of AD and their results

Recently, various methods have been used to treat patients with diseases of the optic nerve. Taking into account the current possibilities of diagnosing various types of AD, in most clinical cases it is possible to create a pathogenetically substantiated treatment regimen.

The main goal of treating patients with AD is to improve the blood flow of the optic nerve and activate the conduction of nerve fibers. In order to treat patients with AD, drugs of various pharmacological groups are used: neuroprotective and vasodilating types, nootropic effects, antioxidants, B vitamins, as well as drugs that affect the rheological properties of blood.

A study by R. Sandyk revealed that a combination of drugs from different pharmacological groups can influence the cause of optic nerve atrophy even in the early stages [26].

Depending on the cause of AD, the therapy of patients may be different. In the inflammatory genesis of the disease (anterior and posterior ischemic optic neuropathy, optic neuritis), corticosteroids are used. In 2015, R. Gal et al. published positive results in the treatment of patients with multiple sclerosis and optic neuritis. Patients in the observation groups with pathology of the optic nerve received glucocorticosteroids (GCS) intravenously (1000 mg) for 3 days, followed by a transition to oral administration (1 mg/kg) for 11 days. Based on the treatment, an accelerated recovery of vision was noted, but the functional results did not improve. Treatment of patients with multiple sclerosis and optic neuritis showed that doses of corticosteroids administered intravenously or orally had the same therapeutic efficacy and led to recovery in a short time [27].

Currently, considerable attention is paid to the treatment of hereditary atrophy of the optic nerve, in particular Leber's atrophy. In their work [28], J. Weiss et al. consider that stem cell treatment is of key importance in the treatment of AD. The researchers found that stem cell precursors, when injected into the vitreous cavity, are able to integrate into the layer of retinal ganglion cells, and then migrate to the optic nerve to stimulate the regeneration of nerve fibers.

Currently, neuroprotective drugs have found wide application in the treatment of patients with AD. The principle of neuroprotection implies an improvement in the conduction of impulses along the optic nerve, as well as a slowdown in the process of neuronal apoptosis in the early stages of the onset of AD.

In the work of E.M. Kolomoytseva and co-authors under observation were patients with advanced and advanced stages of glaucoma, who were prescribed neuroprotective and vascular

therapy. As a result of the use of complex therapy, after 3 months, an improvement in overall photosensitivity was revealed, as well as an expansion of the field of view, while the positive therapeutic effect of the treatment persisted for 6 months of observation [29]. N.I. Kurysheva and co-authors, I.B. Alekseev et al. studied the effectiveness of neuroprotection and concluded that the use of drugs of this group in the treatment of patients with glaucoma is aimed at slowing down the distant mechanisms of death of nerve fibers of the ON [30, 31]. The authors believe that the use of neuroprotective therapy has a beneficial effect on the state of visual functions in patients with glaucoma and AD.

In the treatment of patients with AD, nootropic drugs have good therapeutic efficacy.

In the work of G.S. Polunin and co-authors examined patients with AD of various etiologies (inflammatory, toxic-allergic, vascular origin), as well as with partial atrophy of the optic nerve. Researchers used a 0.1% solution of Semax to treat patients by intranasal instillation and endonasal electrophoresis [32–34]. According to the results of the study, an improvement in visual functions was revealed, as well as an acceleration of the recovery processes of the optic nerve.

Based on the data of published scientific works, it can be concluded that complex conservative treatment of patients with ON atrophy has a high but short-term effectiveness. To achieve a more stable result, many authors have proposed the combined use of physiotherapeutic methods and complex therapeutic treatment.

L.V. Zamaraeva and S.A. Gibadullin conducted a complex treatment of patients with AD and tapetoretinal abiotrophy using electrical nerve stimulation. Patients in the observation groups after the received therapy showed a significant improvement in the blood supply to the optic nerve [35]. According to a study by T.G. Kamenskikh, in addition to electrical nerve stimulation, restoration of ON blood supply improves with helium-neon laser stimulation of the ON in combination with vascular therapy [36].

Thus, to date, a number of physiotherapeutic methods for the treatment of AD have been developed, which can be used in combination with vascular and nootropic therapy. In most cases, complex therapy has a positive effect, although in a number of patients it is not possible to establish the etiology of AD and conduct pathogenetically substantiated therapy. Therefore, further study of the etiology and methods of treatment of ASD remains an urgent problem.

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