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Primary Open-Angle Glaucoma and Degenerative Changes in the Central Parts of the Visual Analyzer

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Abstract: The purpose of the article is to study the morphological changes in the central parts of the visual analyzer in glaucoma.

Keywords: open-angle glaucoma, optic nerve, neurodegeneration, chiasma.

INTRODUCTION

In recent years, primary open-angle glaucoma (POAG) has been classified as a neurodegenerative disease. The main cause of the development of neuroopticopathy in glaucoma is considered to be intolerant intraocular pressure. The disease develops with age and is characterized by a progressive course even against the background of a normal level of ophthalmotonus [1, 2]. The mechanism of death of retinal cells and optic nerve axons in glaucoma, as in all neurodegenerative disorders, is physiologically programmed apoptosis [3].

MATERIALS AND METHODS

At the same time, there are increasingly more works, especially in foreign literature, that indicate the relationship between POAG and neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. Undoubtedly, there is much in common between them: an increase in incidence with age, selective damage to one type of neuron, the same mechanism of nerve cell death. It is possible that with POAG, pathological changes do not end in the optic nerve head, but spread throughout the entire pathway of the visual analyzer.

The main task that confronts the doctor and the patient after the diagnosis of primary open-angle glaucoma is made is the most complete and long-term preservation of visual functions. However, even under the most favorable conditions, it is practically impossible to solve this problem. Thus, with long-term observation of more than ten years, negative dynamics were noted in 86.0% of cases [1], and the remaining 14.0% of cases can be explained by overdiagnosis or benign hypertension.

RESULTS AND DISCUSSION

Apparently, the only possible answer to this question may be that there is some unstudied factor that was beyond the scope of our attention and was not taken into account when prescribing treatment. Such a factor, in our opinion, may be mitochondrial pathology - a disease of cellular energy. Mitochondrial pathology is neither an etiological nor a pathogenetic factor in the development of POAG; however, by worsening the trophism of CNS cells, it contributes to the progression of optic neuropathy, accelerating the decay of visual functions.

A pathological examination was carried out in two people whose death was not associated with diseases of the central nervous system. As indicated in the outpatient records, they had suffered from

POAG for 8 to 10 years, and the diagnosis of advanced glaucoma was established through the targeted use of appropriate techniques and documented.

Macroscopic examination revealed severe atrophy of the optic nerve with the loss of a significant number of axons, as well as the loss of a significant number of neurons in the lateral geniculate body. Microscopic examination showed a decrease in the thickness of the cell layer of the visual cortex, shrinkage of the radius of neurons and their nuclei, lumpy, granular cytoplasm, and the presence of lipofuscin in large quantities, which indicated an atrophic process.

In both cases, neurodegeneration processes were identified in deceased patients who suffered from POAG during their lifetime. All levels of the central part of the visual analyzer were involved in the degenerative process, but most noticeably the area of the visual cortex in the area of the calcarine sulcus. Particularly noteworthy is the fact that amyloid plaques and bodies were found in the optic nerve and layers IV–V of the cerebral cortex (Fig. 1, 2).

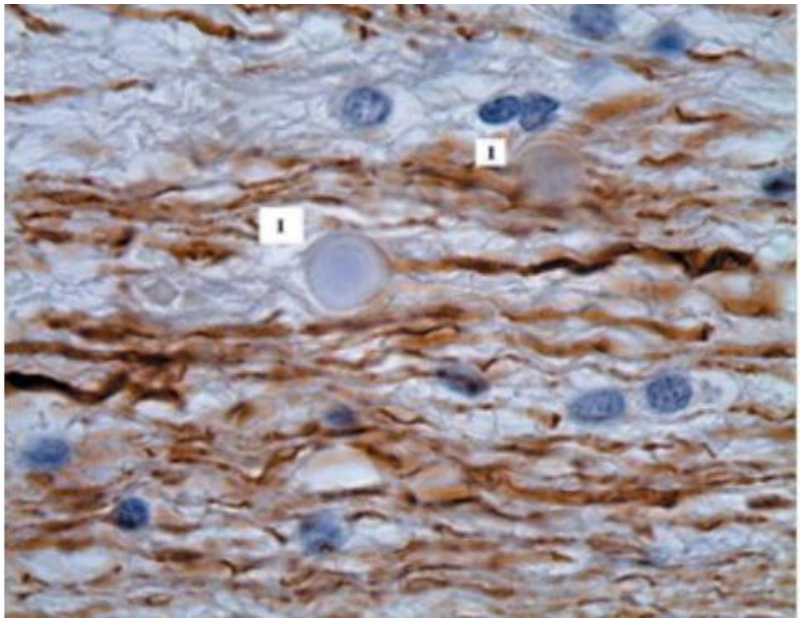

Fig 1. Autopsy. Amyloid bodies (1) in the optic nerve in glaucoma. Immunohistochemical study

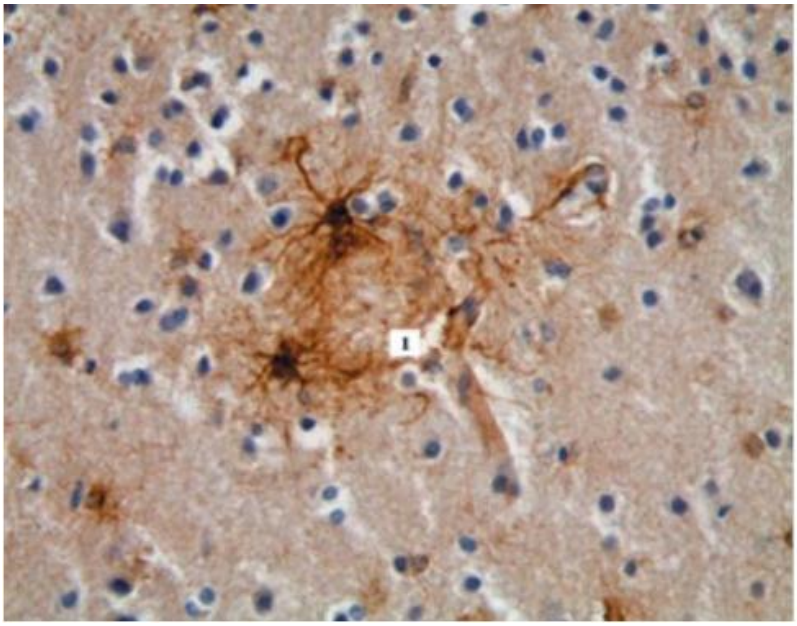

Fig 2. Autopsy. Amyloid plaque (1) in the cerebral cortex in glaucoma

As is known, beta-amyloid is a marker of neurodegenerative diseases, and its presence indicates a pathogenetic connection of POAG with Alzheimer's disease. The neurodegenerative process in the cerebral cortex is also indicated by the torsion of individual arteries of the cortical region, which is a

consequence of a decrease in the thickness of the cortex while maintaining the length of the vascular bed. In this case, the radial arteries of the cortex fold and twist within the vascular space. The signs of astrogliosis revealed by microscopy can be regarded as a consequence of neurodegeneration, death of neurons and oligodendrocytes, and their replacement by immature, functionally defective astrocytes.

It can be considered established that with POAG, a neurodegenerative process develops, which involves not only the peripheral part of the visual analyzer, but also the pathways and the central part, i.e., the visual pathway as a whole.

Two circumstances require separate consideration. This is, firstly, the presence in the brain tissues of people suffering from POAG, beta-amyloid, a generally recognized marker of neurodegeneration, a characteristic morphological sign of Alzheimer's disease. Secondly, the process of neurodegeneration is accompanied by astrogliosis, i.e. the death and replacement of brain cells with young, functionally immature astrocytes that are unable to carry out supporting, protective, trophic and other seemingly auxiliary functions.

CONCLUSION

So, with POAG, as established by autopsy, both the retinal ganglion cells and optic nerve fibers, as well as the tissues of the visual analyzer pathways, up to the cerebral cortex, undergo degenerative changes. This indicates the pronounced neurodegenerative nature of POAG, which is confirmed by the presence of such generally accepted criteria for the neurodegenerative process as astrogliosis and the presence of beta-amyloid accumulations in the cerebral cortex and optic nerve. This pathology is similar to other neurodegenerative diseases such as Alzheimer's disease or Parkinson's disease.

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