

CLASSIFICATION CRITERIA FOR GLAUCOMATOUS OPTIC NEUROPATHY

¹Jalalova D.Z., ²Shernazarov Farrukh

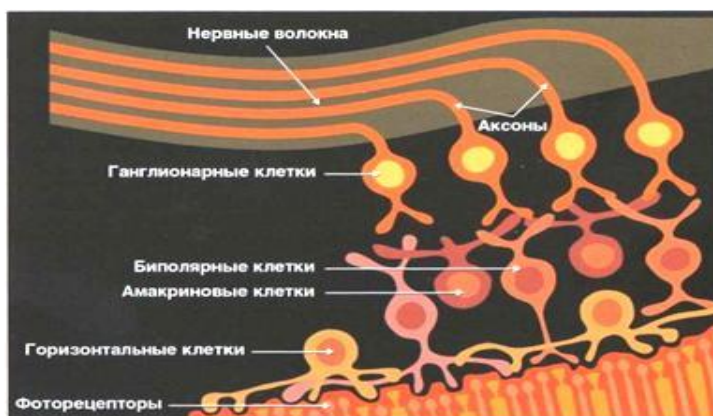
^{1,2}Samarkand State Medical University

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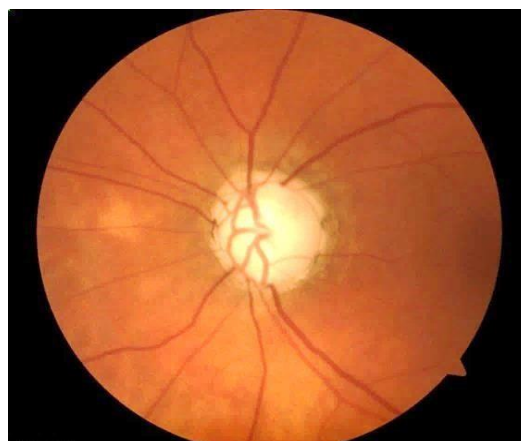
Abstract. GON characterized progressive metabolic violatedniyami in the ONH. Its development is based on apoptosis of GCS. Trigger factor this type of "programmed" cell death is an increase in IOP with subsequent development hypoxia, a decrease in the concentration in the cells neurotrophic factor of the brain and a local increase in con- concentration of glutamates [2]. GON is characterized by the absence of an acute onset and steady Among other links in the pathogenesis of GON, an important place is occupied by: thermal disorders, heat shock proteins, vascular pathology, mitochon- drial dysfunction, inflammation and immunity.

Keywords: key factor V pathogenesis glaucoma is damage GKS (rice. 1).

Rice. 1. Mechanism of damage to retinal ganglion cells (RN Weinreb,R. T. Khaw, 2004)



The progression of GON is accompanied by degeneration and death of GCS, What leads To increase perimetric losses (concentric narrowing of the visual fields, scotomas, decreased sensitivity) and the development



The retina contains many types of ganglion cells (GCs), appropriate for different populations. As is known, at the level of the retina from GC begin magnocellular, parvocellular and koniocellular pathways that form sensory input to the lateral geniculate bodies (LGBs) and then to the visual cortex. Numerous studies indicate that damage to GCs belonging to the parvo-, magno- and koniocellular system mothers, leads to a weakening of the function of the visual channels already at the source visual pathways — at processing information on level retina.

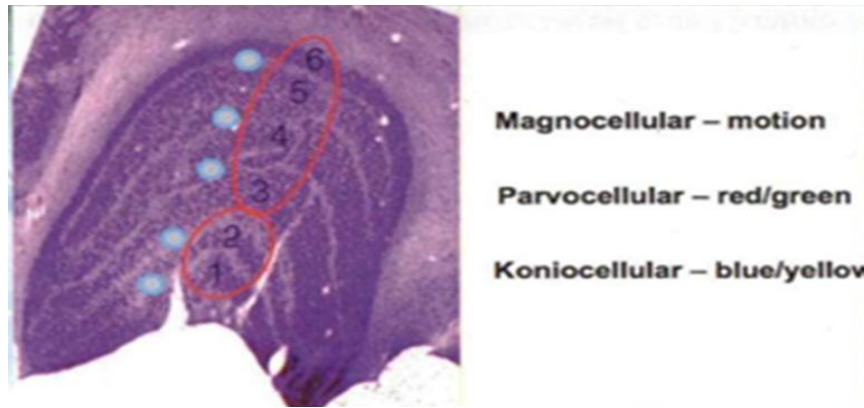
The parvocellular system, which makes up 80% of GCS, is represented by small GK, called X cells, beta or parvo-GK (P cells).

They form a tiny dendritic field, the size of which rarely exceeds but the size of their cell body. The maximum size of the dendritic field glacial GC on the periphery of the retina is 20 microns. pygmy gangli- ous neuron has an elongated body with a diameter of 8-12 microns and a single apical dendrite, which in the terminal region forms a dense a bunch of daughter branches with varicose thickenings in the form of a brush or a bouquet[3]. Small HAs are sensitive to color and provide visual acuity. Akso- us these neurons reach parvocellular layer LKT.

The magnocellular system, which makes up 15% of the ganglion cells current retina, present Y cells (umbrella, alpha-, magno cells, M cells). These are neurons with dense branching of varicose dendrites, whichrye propagate horizontally along the radii from the cell body, like disclosed Chinese umbrella, With diameter dendritic fields from 60 before 100 μm . M-type cells have an axon 1.5–2 μm thick [3]. M cells provide registration of the movement of objects, perception of spatial Noah Depth and Definition spatial relationships. The axons of these neurons reach magnocellular layer LKT. Parvocellular system, comprising 5% of ganglion cells retina, answers behind blue-yellow processing, sensitive To "blue on yellow." Damage to different types of cells in glaucoma occurs in different terms. With an acute rise in IOP, P-cells are predominantly affected.

This is explained by the fact that small parvo-HAs have a larger ratio of surface to volume than large magno-HA, low energy and oxygen requirements de. This makes parvo-HA more vulnerable to an acute rise in IOP - they have little available energy to resist stroke. On the contrary, at long-term rise in IOP in parvo-GC, a better survival is observed bridge than magno-GC, since they require less oxygen and ATP and therefore suffer less from a lack of blood supply [4]. Correspondingly, large magno-HAs have a lower surface-to-volume ratio, how at small parvo-GK, high need V energy and oxygen. Bigger than in P cells, intracellular reserves of ATP, oxygen and potassium, however, more she suffers from long-term rise in IOP from deprivation of their supply oxygen and ATP. Important Mark, What outdoor cranked body has layered structure, and in each layer there are neurons associated with a certain retinal neurons. Thus, each of the million neurons retina in its layers has its representation in the outer crankshaft those bodies.

For example, representatives of those neurons that respond and re- hysteryze movement, are located in the first layer, those that are responsible for the reproduction acceptance of green and red, make up the parvocellular pathway, and blue and yellow, the koniocellular pathway. In the fourth and fifth layers, the subcortical ganglia of those neurons that perceive blue on yellow (yellow background, blue stimulus on yellow background), which underlies blue-yellow perimetry [7] (rice. 3).



With glaucoma, degenerative changes develop in the LKT, wrinkling neuronal atrophy and atrophy in both the parvo- and magnosystems (YH Yucel et al., 2003 2006; N. Gupta et al., 2006), ectopia nuclei And nucleolus, pericellular edema, chromatolysis (V.P. Erichev et al., 2014). Reducing the size of the outer geniculate bodies in a patient with glaucoma is shown on the rice. 4, b (N. gupta, 2007) .For the first time, the external geniculate bodies and the cerebral cortex during ex- perimental glaucoma started research 15 years back Professor Y. Yucel And N. Gupta V Canada, V university Toronto on primates. Through 14 months at primates after raise IOP, quantity neurons V external cranked bodies responsible for movement (magnopath) decrease sewn twice. In the parvo path (perception of red and green colors) - also twice, but in the conio path (perception of blue on a yellow background) -almost V 5 times. With glaucoma, in addition to GCS, not only the outer knees are affected chat body, But and visual cerebral cortex brain (rice. 6).

Today it becomes obvious that age-related changes in HA, their sons, as well as central targets in the LCT and primary visual cortex underlie the involutionary decrease in visual functions. Aging the retina contributes to an increased vulnerability GC and visual pathways in degenerative diseases, including glaucoma, and age is one him from the most important factors risk occurrence primary open-angle no glaucoma. Against the background of nonspecific manifestations, each neurodegenerative Rative disease is characterized by certain symptoms: if process affects the motor zones more, then this is Parkinson's disease, if cognitive functions, then this is Alzheimer's disease, if there is a progressive visual field disturbance that is glaucoma. In the minds of most ophthalmologists, glaucoma is still associated first of all, with the level of IOP, to a lesser extent - with excavation ONH, even less often doctors think about damage to the inner layers of the retina ki, And almost never - About, What This CNS disease.

Treatment. Neurodegenerative diseases must start off how possible earlier. Clearly, a glaucoma treatment strategy aimed at exclusively for the normalization of IOP, unable to fully provide chit the desired effect, which led to the search for new directions of drug natural therapy glaucoma. Most promising from them turned out neuroprotection, designed to protect the neurons of the retina and nerve optic nerve fibers from the damaging effects of various factorstors (see the section "Neuroprotective approach to the treatment of opto-retinal Noah pathology").

REFERENCES

1. Касимова М.С., Махкамова Д.К., Жалалова Д.З. Эндотелин-1 ва гомоцистеин даражасини артериал гипертензия фонида тўр парда ўзгаришларида эндотелиал

- дисфункциянинг маркерлари сифатида текшириш Журнал «Биомедицина ва амалиёт». Тошкент - 2021, Том № 6, №5. С. 203-210
2. Жалалова Д.З., Махкамова Д.К Мультикомпонентный подход к диагностике изменений сетчатки при артериальной гипертензии Журнал «Проблемы биологии и медицины» – 2021. №5 С – 205-211.
 3. Жалалова Д.З., Махкамова Д.К.ОКТ- ангиография при оценке сосудистого русла сетчатки и хориоидеи Журнал «Проблемы биологии и медицины»– 2021. №6 С – 211-216.
 4. Zhalalova D.Z.The content of endothelin and homocysteine in blood and lacrimal fluid in patients with hypertensive retinopathy Web of Scientist:International Scientific Research Journal Volume 3,ISSUE 2,February-2022,С. 958-963
 5. Zhalalova D.Z. Modern aspects of neuroprotective treatment in hypertensive retinopathy Web of Scientist:International Scientific Research Journal Volume 3,ISSUE 2,February-2022,С. 949-952
 6. Zhalalova D.Z.Development of classification criteria for neuroretinal ischemia in hypertension Web of Scientist:International Scientific Research Journal Volume 3,ISSUE 2,February-2022,С. 972-978
 7. Жалалова Д.З.Классификационные критерии изменений сосудов сетчатки при артериальной гипертензии Журнал «Проблемы биологии и медицины» – 2022. №1 С – 50-53.
 8. Жалалова Д.З.Диагностические критерии оптической когерентной томографии с функцией ангиографии при ишемических заболеваниях органа зрения на фоне артериальной гипертензии Журнал «Проблемы биологии и медицины» – 2022. №5 С –73-78
 9. Жалалова Д.З.Оценка маркеров эндотелиальной дисфункции в слезной жидкости у пациентов с артериальной гипертензией Журнал «Биомедицина ва амалиёт». Тошкент - 2022, Том № ,№. С.
 10. Жалалова Д.З. ОКТ-ангиография в оценке ретинальной и хореоретинальной микроциркуляции у пациентов с неосложненной артериальной гипертензией Международный офтальмологический конгресс ИОС Ташкент 2021,С 95-96
 11. Жалалова Д.З. Современные аспекты нейропротекторного лечения при гипертонической ретинопатии Журнал ТМА – 2022. № 4 С 84-87
 12. Zhalalova D.Z.Magnetic Resonance Tractography as a Method of Choice for Neuroimaging in ocular ischemic syndrome against the background of hypertension Central Asian Journal of medical and natural sciences Vol 3 ISSUE 2, Mar-Apr 2022, С 207-210
 13. Zhalalova D.Z.Development of classification criteria for neuroretinal ischemia in arterial hypertension Central Asian Journal of medical and natural sciences Vol 3 ISSUE 3, May-Jun 2022, С 59-65
 14. F. Shernazarov РОЛЬ С–РЕАКТИВНОГО БЕЛКА В ПАТОГЕНЕЗЕ СОСУДИСТЫХ ЗАБОЛЕВАНИЙ ОРГАНА ЗРЕНИЯ У БОЛЬНЫХ АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ // SAI. 2022. №D8. URL: <https://cyberleninka.ru/article/n/rol-s-reaktivnogo-belka-v-patogeneze-sosudistyh-zabolevaniy-organa-zreniya-u-bolnyh-arterialnoy-gipertenzii> (дата обращения: 27.01.2023).

15. F. Shernazarov СОЧЕТАННАЯ СТОМАТОЛОГИЧЕСКАЯ И ГЛАЗНАЯ ПАТОЛОГИЯ // SAI. 2022. №D8. URL: <https://cyberleninka.ru/article/n/sochetannaya-stomatologicheskaya-i-glaznaya-patologiya> (дата обращения: 27.01.2023).
16. Farrukh Shernazarov, MICROCIRCULATION DISORDERS IN THE VASCULAR SYSTEM OF THE BULBAR CONJUNCTIVA IN THE INITIAL MANIFESTATIONS OF CEREBRAL BLOOD SUPPLY DEFICIENCY // SAI. 2022. №Special Issue 2. URL: <https://cyberleninka.ru/article/n/microcirculation-disorders-in-the-vascular-system-of-the-bulbar-conjunctiva-in-the-initial-manifestations-of-cerebral-blood-supply> (дата обращения: 27.01.2023).
17. F. Shernazarov, CAUSES, SYMPTOMS, APPEARANCE, TREATMENT OF VARICOSE VEINS // SAI. 2022. №D7. URL: <https://cyberleninka.ru/article/n/causes-symptoms-appearance-treatment-of-varicose-veins> (дата обращения: 27.01.2023).
18. F. Shernazarov, TYPES OF HEMORRHAGIC DISEASES, CHANGES IN NEWBOENS, THEIR EARLY DIAGNOSIS // SAI. 2022. №D5. URL: <https://cyberleninka.ru/article/n/types-of-hemorrhagic-diseases-changes-in-newboens-their-early-diagnosis> (дата обращения: 27.01.2023).
19. F. Shernazarov TINTING IN THE EAR CAUSES, DEVELOPMENT, TREATMENT AND PREVENTION OF NOISE IN THE EAR // SAI. 2022. №D8. URL: <https://cyberleninka.ru/article/n/tinting-in-the-ear-causes-development-treatment-and-prevention-of-noise-in-the-ear> (дата обращения: 27.01.2023).