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AGE-RELATED MACULAR DEGENERATION (LITERATURE REVIEW)

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Annotation: Age–related macular degeneration (AMD) is a progressive disease that steadily leads to blindness and manifests itself as a chronic degenerative process in the pigment epithelium, Bruch's membrane and choriocapillary layer of the macular zone of the retina. Spectral optical coherence tomography (SOCT) is the most informative method of morphometric examination in patients with retinal diseases. Among the functional ones, static automatic perimetry (SAP) is the most informative, which makes it possible to evaluate the features of changes in the central field of vision, indicating the quantitative and percentage ratio of absolute and relative changes.

Key words: glaucoma, macular degeneration, optical coherence tomography, static perimetry.

Аннотация. Возрастная макулярная дистрофия (ВМД) – прогрессирующее заболевание, неуклонно ведущее к слепоте и проявляющее себя хроническим дегенеративным процессом в пигментном эпителии, мембране Бруха и хориокапиллярном слое макулярной зоны сетчатки. Наиболее информативным методом морфометрического исследования у пациентов с заболеваниями сетчатки является спектральная оптическая когерентная томография (СОКТ). Среди функциональных наиболее информативна статическая автоматическая периметрия (САП), позволяющая оценивать особенности изменений центрального поля зрения с указанием количественного и процентного соотношения абсолютных и относительных скотом.

Ключевые слова: глаукома, макулодистрофия, оптическая когерентная томография, статическая периметрия.

Visual impairments in the elderly are a global health problem that significantly affects the quality of life of millions of people worldwide [53, 167]. AMD is the main cause of severe and irreversible loss of central vision in developed countries [129]. It should also be noted that for many patients, visual impairments associated with AMD mean loss of independence, depression, and increased financial problems [55, 260]. According to a meta-analysis, by 2040, the number of people in the world with this disease will reach 288 million people [129]. The incidence of AMD in the early stages is estimated at 6.8%, while in the late stages it is 1.5% [230]. The annual incidence of advanced forms of AMD is 3.5 per 1,000 people over the age of 50, which means 293,000 new cases annually [123]. The rate and degree of progression from the initial stage of AMD to the late stage is highly variable. Thus, according to Beaver Dam Eye, in patients aged 43-86 years with signs of initial AMD in both eyes during 15 years of follow-up, the overall detection rate of GA was 13.5%, and HCV was 14.8% [115]. A combined analysis of demographic data on the prevalence of eye diseases in three racial populations from North America, Europe, and Australia showed the age-dependent prevalence of AMD. Overall, AMD was detected in 0.21% of the total population aged 55 to 64 years. This indicator increased to 13.05% in people aged 85 years

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and older. The prevalence of the wet form of AMD increased from 0.17% among subjects aged 55 to 64 years and to 5.8% among people over 85 years of age. The prevalence of GA increased from 0.04% to 4.2% in the same age groups [230]. 16 AMD is considered as a disease caused by the convergence of various risk factors. It was found that old age, smoking, low intake of antioxidants, increased body mass index, family history, hypertension, large soft druses and subretinal drusenoid deposits increase the risk of developing both HA and HCV [88, 230, 232, 233, 256, 258]. According to a number of authors, the interaction of metabolic and structural changes with genetic and environmental risk factors causes pathological changes that contribute to the development of phenotypic changes and as a result of an earlier onset of the disease [81, 125]. To date, 34 loci associated with AMD have been mapped, and according to various estimates, there are from 33 to 50 polymorphic genes that can influence the development of this disease. In addition, the following are conducted genetic studies aimed at identifying the features of various late-stage AMD phenotypes [12, 28, 150]. A special role in the development of AMD is assigned to genetic polymorphisms of genes encoding: protein inhibitor of the alternative pathway of complement activation - H (CFH), serine protease 1 (HTRA1), AMD hypersensitivity factor 2 (ARMS2), cell proliferation regulator, lymphocyte activator PLEKHA1 expressed in the macular region of the retina. Rare variants of polymorphisms in the CFH, CFI, and complement components (C9 and C3) genes are more often observed in patients with GA than in patients with HCV [8, 12, 28, 228]. According to clinical signs, AMD is classified into two forms: exudative/"wet" (or neovascular) and non-exudative/"dry" (or atrophic). The generally established and frequently used clinical classification proposed by the Age–Related Eye Disease Study (AREDS), according to which the early, intermediate and late stages of AMD were distinguished. The most pronounced changes are observed in the intermediate and late stages of AMD. The intermediate stage of AMD (category 3 AREDS) 17 is characterized by a large number of medium-sized druses, at least one large druse (diameter ≥ 125 microns) or atrophy of the RPE, which does not affect the central fossa. Mainly, severe vision loss is associated with the late stage of AMD (category 4 AREDS), manifested by the presence of one of the following signs: progressive atrophy of the RPE, photoreceptor layer and choriocapillaries with seizure of the central fossa, known as GA, and the growth of newly formed vessels and ingrowth through the Bruch membrane, known as CNV [85]. The modern classification of HCV is based on the origin of neovascularization and the level of damage: HCV type 1 (latent) vessels originate from the choroid and remain under the RPE; HCV type 2 (classical) vessels also originate from the choroid, but they break through the RPE and are located above it. In addition, a combination of these two types with the formation of a mixed type of HCV is often observed. Depending on the predominance of one component or another in the neovascular complex, "predominantly classical" and "predominantly latent" types are distinguished. HCV vessels of type 3, which are called retinal angiomatous proliferation (RAP), are formed from the retinal arteries and, sprouting towards the choroid, form a retino-choroidal anastomosis [147]. Yannuzzi et al. proposed that the source of HCV is the deep capillary plexus of the sensorineural retina with its expansion into the choriocapillary layer [100, 223]. Another clinical form of AMD is idiopathic choroidal vasculopathy, which is an anomaly of the choroidal vessels in the form of a branching vascular network and the presence of "polyp-like" vascular dilation [166, 206]. Although GA with capture of fovea and CNV are considered manifestations of late-stage AMD, they are not mutually exclusive and may be present in the same eye. [124, 126, 177, 228, 253]. Attention should be paid to the development of HCV, which occurs against the background of existing GA [126, 206, 228, 239]. 18 histopathological studies have

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demonstrated the coexistence of atrophy and CNV in the eyes, where the latter has not been clinically identified [253]. Both of these variants imply the existence of a third, mixed, late-stage AMD phenotype, which has been poorly studied and is of particular interest. The development of atrophy in the eyes with existing HCV has been actively discussed recently. Prolonged antiangiogenic therapy of the wet form of AMD is able to stabilize visual functions in the long term, but recent studies have raised concerns that treatment with angiogenesis inhibitors may accelerate the appearance and/or progression of atrophic changes with the development of the so-called "MA" [40, 123, 124, 138, 139, 172, 176, 177]. Despite the fact that this term is not clearly defined and is used as a synonym for "GA", in a number of published works it is used in the context of clinical research in the presence of atrophic changes against the background of HCV regression and/or antiangiogenic therapy, and in the presence of the final stage of progression of the dry form of AMD without signs of neovascular membrane, the term "GA" is used." [138, 139, 168, 260]. Early clinical manifestations of AMD are various types of cysts and/or pigmented changes. In most cases, there are hard or soft druses, as well as a combination of them in one eye. Solid druses, which look like round and discrete yellow-white spots, are commonly identified in many populations. They are not age-related and do not carry an increased risk of developing neovascularization. Soft druses with indistinct boundaries, measuring 63 microns or more, are associated with age and with a higher risk of progression to a late stage of AMD [12, 42, 101]. Reticular pseudodruzes (RPD) differ in their structure and subretinal localization. Their presence is associated with a higher risk of progression to advanced forms of AMD and the development of atrophy after intravitreal administration of angiogenesis inhibitors [87, 174, 231]. The late forms of AMD are still the subject of study by many domestic and foreign researchers. GA19 is easily recognized clinically, as a clearly demarcated area of retinal thinning with discoloration is identified, which makes it possible to visualize the underlying choroidal vessels. Clinical manifestations of neovascular AMD may include the following signs: edema, detachment of the neuroepithelium (ONE), hemorrhages, deposition of exudate, detachment of the pigment epithelium (OPE) [128]. Despite the accumulated experience in identifying AMD risk factors, the modern pathogenetic foundations of the development of this pathology have not been fully studied to date, which requires detailed research.

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