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Prevention of Age-Related Macular Degeneration

Gazizova L. K
Fergana Medical Institute of Public Health Fergana, Uzbekistan

Ermatova B. O
Fergana Medical Institute of Public Health Fergana, Uzbekistan

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ABSTRACT

Age-related macular degeneration (AMD) is a common eye disease that leads to a significant decrease in vision and threatens with disability. According to official data from the WHO Center for the Prevention of Avoidable Blindness, the prevalence of this pathology is 300 per 100,000 people. The disease occurs in 40% of cases among people over 40 years old, in 58-100% - over 60 years old.

INTRODUCTION

GassJ.D. presented age-related macular degeneration (AMD) as a chronic degenerative disease with a predominant lesion of the choriocapillary layer, Bruch's membrane (MB) and retinal pigment epithelium (RPE) with subsequent involvement of photoreceptors [2].

MATERIALS AND METHODS

This provision is valid and relevant today. According to modern scientists, AMD is a progressive disease that manifests itself as a chronic degenerative process in the RPE, MB, and the choriocapillary layer of the macular area, leading to a gradual decrease in central vision [1].

In the structure of primary disability according to AMD, patients of working age make up 21%, and in retirement age - 32% [6]. To date, AMD is the main cause of deterioration in the quality of life, disability and blindness in people over 50 years of age in economically developed countries [3]. Currently, AMD is a serious medical and social problem that attracts the attention of many researchers. It is known that 30-50 million people on the globe suffer from AMD, of which about 1.5 million have an exudative form. About 600 thousand new cases of the disease are registered annually worldwide [5].

RESULTS AND DISCUSSION

According to the WHO, by 2050 the number of patients with AMD will triple [2]. This is due to an improvement in the quality of life and its duration, with the availability of medicine, and with improved methods for diagnosing pathology.

Age-related macular changes of varying severity are found in more than 10% of the population aged 65–74 years and in 25% of people over 74 years of age [1]. It has been found that if vision is already lost in one eye as a result of AMD, then the risk of developing blindness in the other eye within 5 years is approximately 12% [2].

There are various classifications of AMD. The International ARM Epidemiological Study Group distinguishes 2 main forms of the disease: non-exudative (“dry”) and exudative (“wet”). This division is quite convenient for use in the practice of ophthalmology. In addition, this classification also distinguishes the early stages of the disease, terminologically emphasizing the implicitness of changes (age-related maculopathy) (AMD) [4]. Approximately 80% of cases of the disease occur in the "dry" form of AMD, the remaining 20% - in the "wet" (exudative) AMD [3]. The etiopathogenesis of this disease has not yet been fully elucidated. Domestic and foreign ophthalmologists consider AMD as a multifactorial disease, the main risk factors of which are age, light color of the iris, smoking, overweight, etc. It has been proven that smokers have a 2.4 times higher risk of developing advanced stages of AMD than non-smokers [3].

Oxidative stress plays a significant role in the pathogenesis of AMD. It is known that the retina, being the most highly differentiated human nervous tissue, is especially susceptible to oxidative stress, hypoxia, and ischemia [5].

According to the latest data, one can find information about the dominant role of autoimmune inflammation in the genesis of the disease [3]. Several components of the immune system have been identified: cytokines, macrophages, C3 component of complement, etc., which form a membrane attack complex and, through the activation of a cascade of complement components, lead to the onset of autoimmune inflammation and the development of degenerative changes in RPE, MB and the choriocapillary layer [4] .

In addition, genetic factors play a significant role in the pathogenesis of AMD. Since 2005, at least 20 genes have been identified and studied, the presence of which is associated with the risk of developing AMD: PLEKHA1, SOD2 ABCA1, ARMS2, CFH, C2/CFB, CFI/Y402H, CX3CR1, C3/CFD, CFI/R1210C, CYP24A1, HTRA1 , GSTM1, and others [3].

Mutations of these genes can lead to a chronic disruption of the interface in RPE and MB, leading to the clinical manifestations of AMD.

But individual changes in the macula refer to the processes of physiological aging and the accumulation of metabolic products in the cells of the pigment epithelium, changes in collagen fibers, an increase in density and thickening of the Bruch's membrane, and in the choroid, age-related changes in the walls of blood vessels. It has been recorded that one of the early manifestations of the disease is the accumulation of lipofuscin (“ageing pigment”) in RPE, the main component of lipofuscin, N-retilidineN-retinylethanolamine, which disrupts RPE functions, causing its apoptosis with subsequent development of GA [5]. Also, the accumulation of N-retilidine-N-retinylethanolamine in RPE cells has been found to increase the risk of neovascular AMD [4].

RPE is a polyfunctional cellular system [6], the most important properties of which include: formation of the hematoretinal barrier; synthesis and accumulation of vitamin A; phagocytosis of the outer segments of photoreceptors; light absorption; synthesis of cytokines; antioxidant protection against free radicals, lipid peroxidation, toxic effects of heavy metals, certain medicinal substances, etc. [5]. In addition, such carotenoids as lutein, zeaxanthin and mesoseaxanthin also have an antioxidant effect. These carotenoids accumulate in the cells of the outer plexiform layer of the fovea and create macular pigment (MP). MP is responsible for central vision, absorbs the blue part of the spectrum and is a powerful neutralizer of free radicals [6]. According to some authors, MP density decreases with age, which is accompanied by a

decrease in the concentration of carotenoids in the macula.

To determine AMD, traditional ophthalmological examinations are used: history taking, determination of visual acuity with maximum correction far and near, Amsler test, perimetry, ophthalmoscopy with pupil dilation. In addition, a number of ophthalmic reliable high-precision objective research methods are currently used: optical coherence tomography (OCT), fluorescein angiography (FA) of the retina, digital photography of the fundus. These methods make it possible to determine the structure of the retina online, and in addition, coherence tomography makes it possible to qualitatively and quantitatively determine the thickness of the retinal layer and assess the level of pathological changes in three dimensions.

There are several scientific studies in different countries of the world, leading the study of the properties of vitamin D and its role in the course of CM. It was found that it is able to reduce the proliferation of T-helpers and cytotoxic cells; vitamin D reduces the production of C-reactive protein, a marker of systemic inflammation, and pro-inflammatory agents such as IL-2, IL-6, IL-8, IL-12 [4].

In addition to anti-inflammatory activity, vitamin D has an antianginal property: it reduces the proliferation of endothelial cells. Angiogenesis is the basis of many physiological and pathological processes (atherosclerosis, psoriasis, diabetic retinopathy). The main mediator of angiogenesis is vascular endothelial growth factor. It also activates the migration, proliferation, differentiation of endothelial cells, through stimulation of the tyrosine kinase receptor, promotes the formation of neovascularization. Experiments in vivo and in vitro in the presence of vitamin D have shown that it can regulate the production of phospholipase C, which is involved in the activation of the tyrosine kinase receptor [6].

It was noted that in the treatment of a malignant tumor with the use of vitamin D, the formation of vessels of large diameter does not occur. Also, it was found that vitamin D is able to suppress the expression of anti-apoptotic proteins that are induced by vascular endothelial growth factor. The possible role of vitamin D in the pathogenesis of AMD is supported by the presence of receptors in the outer and inner segments of photoreceptors, ganglion cells, and pigment epithelium. In connection with the antioxidant function, manifested by the secretion of the reactive form of oxygen and nitrogen, vitamin D is able to prevent and slow down the development of the advanced stage of AMD.

The synthesis of vitamin D begins with the stimulation of ultraviolet rays on the skin, which promotes the conversion of 7-dihydrocholesterol into vitamin D. Vitamin D is activated in the liver and its release into the blood of its 25-hydroxycalciferol proactive form. According to scientists, in older people with blood levels of vitamin D less than 25 nmol/l, there will be a deterioration in health and the manifestation of diseases that can play an important role in the development of AMD.

CONCLUSION

Therefore, we can assume that vitamin D affects the course and development of AMD, due to the existence of numerous mechanisms that prove its effect on the inflammatory process in the retina. Previously, vitamin D was considered as a bone-forming component. Considering that AMD is a multifactorial disease; hypovitaminosis of vitamin D stimulates its development. Our aim is to determine the role of blood vitamin D concentration in the course and development of AMD.

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кизи/Tolipova Gulhayo–магистр; 3Уразалиева Ильмира Равкатовна/Urazalieva Ilmira–ассистент, Школа общественного здравоохранения Ташкентская медицинская академия, г. Ташкент, Республика Узбекистан. EUROPEAN RESEARCH: INNOVATION IN SCIENCE, EDUCATION AND TECHNOLOGY, 109.

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