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Opinion

Age Related Macular Degeneration: Where are we now? -

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Age-Related Macular Degeneration (AMD) is a progressive late onset disease affecting central vision, representing the leading cause of irreversible blindness among older adults aged over 50 years old and affecting approximately 11 million individuals in the United States and 170 million worldwide [1]. The latest findings evidenced increased incidence of AMD not only in the industrialized world, but also in Africa [2]. With the aging population the problem is heightening, thus underscoring not only medical, but at the same time a big socio-economic challenge [3].

The estimated number of patients suffered from age-related macular degeneration will increase and reach 22 million by the year 2050 in US and 288 million by the year 2040 worldwide [4].

At present effective treatment for AMD or for arresting its progression in its earliest phases does not exist. Likewise, management of this visually debilitating condition poses challenge taken into account its complicated etiopathogenesis. Despite improved treatment with intravitreal injections of antiangiogenics, AMD is still associated with nonresponders, tachyphylaxis, rebound phenomen, high re-injections rate, which may represent also a rising economic burden, nonfeasibility of endless injections, thus emphasizing the importance of addressing new approaches to formulate treatment strategies.

Tissue hypoxia due to drusen accumulation, causing disconnection between the Retinal Pigment Epithelium (RPE) and the choroidal blood supply is the most common driver of Vascular Endothelial Growth Factor (VEGF) synthesis in neovascular AMD that is why suppression of over expressed VEGF by anti-VEGF agents underscored their predictably rational use, specifically directed to prevent formation of new vessels [5].

Aforementioned initiated a common use of intravitreal injection of vascular endothelial growth factor inhibitors for neovascular AMD in recent years. Despite this impactful intervention, currently available findings obviate that pharmacotherapy by short acting antiangiogenics reached a physiological "ceiling" effect and symptomatic treatment intended to suppress just one chemical substance is inadequate to successful therapy of advanced AMD.

Ophthalmologists face a host of new challenges in the management of AMD as the natural history of the disease becomes better understood and new technologies become available. Scientific understanding of AMD continues to develop, and recent changes have been made to underscore the other game players in complex biological process taken a place in AMD, beyond anti-VEGF, which remains the important, but not the only process-driving factor.

There is a growing body of evidence of multifactorial pathogenetic origin of disease. Recently, it was highlighted to be caused by different processes, such as: oxidative damage, local inflammation, complement activation, disregulation of lipids intra and extracellular transport through RPE with accumulation similar to atherosclerotic plaques, neurodegeneration of macula [6-9].

This suggests a therapeutic potential of agents with multiple mechanism of actions, such as anti-inflammatory, neuroprotective.

Today, there is also a need to focus not only on achieving the maximal efficacy of therapy, but at the same time to improve safety of proposed interventions: noninvasive approaches such as lacrimal plug, reduction of toxicity, ocular and systemic side effects; and likewise to increase retinal bioavailability of novel pathogenetically tailored therapeutic agents.

Developing an understanding of the pathophysiological mechanisms of AMD will certainly allow the formulation and implementation of new, more effective and safe therapeutic procedures, to provide novel treatments to our patients. Having an awareness of cutting-edge advances in the field can be key to millennial-minded approach for AMD management.

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