Chapter

Age-Related Macular Degeneration and Its Current Treatment Strategies: An Updated Review

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Abstract

The retinal pigment epithelium (RPE), which is crucial for good vision, supports the health and function of photoreceptors or Bruch's membrane (BM). The two most prevalent retinal vascular disorders that account for the majority of blindness in people in their working years and older are diabetic macular edema (DME) and neovascular age-related macular degeneration (nAMD). The blood-retinal barrier (BRB), cell differentiation, autophagy, growth factors (GFs), and other complex signaling pathways all play a role in maintaining morphology, and their disruption by harmful substances affects RPE function. It is urgent to gain a better understanding of the molecular mechanisms underlying the pathogenesis of AMD and identify potential targets as leads for creating potent therapies because there are currently no effective treatments for the early-AMD and late-AMD forms of the disease. For this reason, it is vital to identify molecular targets and therapies that can stop RPE deterioration in AMD and restore RPE function. Currently, the first-line treatment for nAMD and DME involves anti-vascular endothelial growth factor (VEGF) medications that inhibit VEGF family ligands, such as ranibizumab, bevacizumab (off-label usage), brolucizumab, and aflibercept. However, because nAMD and DME have complicated pathophysiological backgrounds, further research is still needed to determine the causes of non-response, resistance to anti-VEGF treatment, and disease relapses.

Keywords: age-related macular degeneration, oxidative stress, epithelial-mesenchymal transition, ranibizumab, aflibercept, brolucizumab, faricimab

1. Introduction

The main factor in blindness among the elderly in industrialized nations is age-related macular degeneration (AMD), which causes central vision loss. Choroidoidal neovascularization (CNV) is a hallmark of neovascular age-related macular degeneration (nAMD). In people with nAMD, a complicated mechanism involving the signal protein vascular endothelial growth factor A (VEGF-A) promotes the formation of new blood vessels. Ranibizumab, bevacizumab, and aflibercept are examples of anti-VEGF medications that inhibit this protein [1–3]. Retinal vascular disease has been