Journal of Pharmaceutical Research International

33(39A): 35-40, 2021; Article no.JPRI.71577
ISSN: 2456-9119
(Past name: British Journal of Pharmaceutical Research, Past ISSN: 2231-2919, NLM ID: 101631759)

Age-related Macular Degeneration: Simple Review

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JPRI/2021/v33i39A32138
Editor(s):
(1) . R. Deveswaran, M.S. Ramaiah University of Applied Sciences, India.
Reviewers:
(1) Niina Bhattarai, University of Eastern Finland, Finland.
(2) Bhandari Akshay Jawahirlal, Pravara Institute of Medical Sciences, India.
Complete Peer review History: https://www.sdiarticle4.com/review-history/71577

Review Article

Received 20 May 2021
Accepted 26 July 2021
Published 28 July 2021

ABSTRACT

Age-related macular degeneration (AMD) is a common, chronic, and innovative degenerative disease of the macula that affects the elderly, with significant loss of imagination and foresight due to abnormalities within the photoreceptor, retinal pigment epithelium, Bruch's membrane, and the choroidal complex. AMD is currently being elucidated through molecular dissection of histopathological samples and genetic coupling. Threat factors for AMD can be broadly based on character elements (e.g. age, gender, race / ethnicity, heredity, and socioeconomic reputation) and environmental factors (e.g. consumption and alcohol consumption). Signs and signs of AMD are: Druids. This is one of the early symptoms and symptoms of AMD and the presence of neovascularization in the macula in humid conditions. Form of AMD A medical examination is usually sufficient for the installation of an AMD analysis, but the use of additional examinations

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such as fundus vehicle fluorescence, optical coherence tomography, fluorescein angiography and inexperienced angiography with indocyanine for diffuse macular anomalies makes sense. unimaginative and prophetic cure cases can help.

Keywords: Macular degeneration; aging; macula; degeneration; central vision.

1. INTRODUCTION

The macula is an oval region near the center of the retina, which is more manageable at a length of 5.6 mm. it is the most sensitive part of the retina. the fovea is present in the middle of the macula. In addition to color vision, the macula is responsible for high visual acuity. The macula is yellowish due to macular pigments [1].

Age-related macular degeneration (AMD) is a common, chronic, progressive degenerative disease of the macula that affects the elderly and is accompanied by critical visual impairment due to abnormalities in the photoreceptor, the epithelial pigment structure of the retina, Bruch's membrane and the choroidal complex. they often cause geographic atrophy and / or neovascularization [2]. (AMD) goes through numerous stages that can be described as early, intermediate, and advanced. The first signs and symptoms to develop are yellowish deposits called drusen on the retina. Then the retinal tone abnormalities expand: there are paler areas called hypopigmentation and darker areas called hyperpigmentation. Dry and wet despite the fact that dry AMD makes up the majority of accurately diagnosed cases, wet AMD is responsible for the majority of the rather extreme loss of imagination and foresight and usually occurs over weeks and can be extended to months. Although neovascularization is the leading cause of excessive vision loss, geographic atrophy, the most advanced form of dry AMD, can also result in significant vision loss [3].

2. EPIDEMIOLOGY

AMD is the third leading cause of blindness worldwide after cataract and glaucoma. Most of the men and women affected remain in developed international locations. In general, advanced AMD is rare before age 56 and is more common in people aged 75 and over [4]. The prevalence of neovascular AMD and geographic atrophy appears to be variable among certain races and ethnic groups anywhere in the world. The prevalence of advanced AMD increases with age from 2.2% in 65 to 70 year olds, 7.5% in 80 to 85 year olds and 16% in> 85 year olds in every decade after age 50 it is associated with the highest occurrence [5].

3. RISK FACTORS AND ETIOLOGY

Risk factors for AMD can be broadly divided into personal or environmental factors (e.g., smoking, sun exposure, and nutritional elements such as micronutrients, fish consumption, and alcohol consumption) [6].

Personal factors can similarly be broken down into sociodemographic elements (e.g. age, gender, race, ethnicity, inheritance and socio-economic status), eye factors (e.g. iris color, macular pigment, density optics, cataracts and their companions) be subdivided. . surgical operation, refractive errors and cup / disc ratio) and systemic factors (e.g. cardiovascular problems and their threatening elements, reproductive and associated elements, dermal elastic degeneration and antioxidant enzymes).

There are numerous risk factors associated with choroidal neovascularization progression including: presence of 5 or more drusen, hyperpigmentation, systemic hypertension, one or more large drusen (> 62 µm in the largest linear size), Caucasian race, and smoking [7].

Studies have found that women are at increased risk of developing AMD. However, the association is not very consistent. Both early and late AMD are known to be prevalent among non-Hispanic whites compared to blacks and Hispanics.Socio-economic factors such as education, employment status, income, or marital status are not related to the prevalence or stage of maculopathy [8]. The role of other lifestyle factors, such as obesity and physical activity, in the progression of AMD is still uncertain.

The Age-Related Eye Diseases Study (AREDS) documented that supplementation with antioxidants and zinc reduced the risk of AMD progression and vision loss [9]. A mild to moderate association between high blood pressure and AMD has been documented. The

risk of late AMD. There is no consistent association between cholesterol levels and AMD. Further study is needed to better define the mechanisms by which HDL intervenes in AMD [10]. There is no significant association between diabetes and AMD.

Hormone replacement therapy or estrogen therapy in postmenopausal women has potential protective effects. Studies have shown that inflammation plays a role in the pathogenesis of AMD [10].

3.PATHOGENESIS

The cause of AMD is currently being clarified by molecular dissection of histopathological samples and genetic linkage analysis in different populations. At the beginning of the disease process, lipids are deposited on Bruch's membrane, possibly due to the RPE's inability to process cell debris associated with the turnover of the outer segment. These lipids encapsulate these cell debris due to an inflammatory process that leads to the formation of drusen [11]. It is believed that the inflammatory process is due to the alternative complement pathway. Only later in the course of the disease are drusen visible. A visible clinical sign of AMD. Drusen are small yellowish or whitish clusters of extracellular material that collect between Bruch's membrane and the RPE. Analysis of drusen shows that they contain lipids, amyloid, complement factors, and additional cellular components [12].

The appearance of drusen is accompanied or preceded by a thickening of the collagen layers of the Bruch's membrane, a degeneration of collagen and elastin within the Bruch's membrane with membrane calcification, increased levels of advanced glycation end products and the accumulation of exogenous proteins and lipids [12] These changes can act as a hydrophobic barrier to impede the passage of fluids and nutrients between the choroid and the outer retina, resulting in relative ischemic changes. The subsequent growth of a choriocapillary neovascularization can occur through fractures of the Bruch's membrane [13]. These drusen can be visualized ophthalmoscopically in early AMD patients with several small clusters of drusen or some medium-sized drusen; vision may not be impaired at this stage. Those with intermediate AMD have either numerous medium-sized drusen or one or more large drusen [14].

At this stage, vision problems such as blurred vision can occur. In addition to accumulations of drusen, patients with advanced 'dry' AMD (late type) have a breakdown of photoreceptor cells and supporting tissue in the part of the central retina, which is why it is called "geographic atrophy". Eyesight may be impaired in these patients, but changes can take years to develop, these people can adapt very well and remain symptom-free until they develop severe visual disturbances [15].

4. EYE MANIFESTATION

4.1 Dry AMD

In clinical practice, typical drusen are whitish-yellow foci deep in the retina. Typical drusen deposits lie under the retinal pigment epithelium and Bruch's membrane and vary greatly in number, shape, size and distribution. Most drusen are 21,100 μm in size and are characterized by being hard or soft [16].

Hard drusen that are round and inconspicuous with yellowish-white spots are often identified in many populations. They are not age-related and do not pose a high risk of developing neovascularization. On the opposite side, the soft drusen are diseased. Sharp, with non-discrete edges, from 64 μm. Various studies and studies have shown that large, soft, and confluent drusen are age-related and associated with an increased risk of developing advanced AMD with neovascularization [17].

Geographical atrophy is easily recognized by clinical examination as it appears as a well-defined area of reduced retinal thickness compared to the surrounding retina, with a relative change in color that allows for better visibility of the underlying choroidal vessels. found either hyperpigmentation or hypopigmentation, surrounding macular atrophy.

If the foveal center is retained, good visual acuity can be if the fovea is retained, good vision can be maintained, but reading vision will still be impaired due to the narrowing of the central visual field [18].

4.2 Wet AMD (new blood vessels)

Wet AMD is characterized by the presence of new blood vessels in the macula. Choroidal neovascularization is the growth of new blood vessels from choroidal capillaries by

destroying the outer surface of Bruch's membrane to the pigmented epithelial space. , Yellow-green or gray discoloration or plaque membrane, RPE detachment, RPE rupture as the disease progresses, neovascularization leads to atrophic fibrovascular or macular scars and subsequent irreversible damage to central vision [19].

Pigment epithelial shedding can be caused by the fusion of serous, fibrovascular tissue, hemorrhage, or drusen under RPE. Serous PED is characterized by dome-shaped RPE detachment, accompanied by bright diffuse high fluorescence, and gradually accumulates in a given space. Hemorrhagic PED is a dark increase in RPE due to the presence of underlying blood, and it shows blocked fluorescence throughout all phases of angiography [20].

5. DIAGNOSIS

Clinical examination is usually sufficient to make a diagnosis of AMD, although mild macular abnormalities are best detected by complementary tests such as fundus autofluorescence, fluorescein angiography, optical coherence tomography, and indocyanine green angiography.

Optical coherence tomography (OCT) is a useful additional test for every stage of AMD. In patients with dry AMD, high-resolution B-scans are beneficial to assess the ultrastructure of the drusen and examine the adjacent layers of the retina, which may be compromised as the disease progresses [21].

Severe forms of AMD, such as B. geographic atrophy can be controlled by OCT. High definition B-scans can be used to identify some of the characteristics of wet AMD, such as: [22].

6. MANAGEMENT

6.1 Dry AMD

Visual aids are helpful in treating early "dry" AMD, but once early AMD has reached advanced stages, no treatment can prevent vision loss. Treatment and monitoring can delay and potentially prevent intermediate dry AMD. progress to advanced AMD. The Age-Related Eye Disease Study (AREDS) found that taking high doses of antioxidants and zinc significantly reduced the risk of advanced AMD and the associated loss of vision. However, the use of antioxidants and zinc cannot help prevent AMD [23]. In addition, beta-carotene supplements are only recommended for those who have never smoked, as current or former smokers who take beta-carotene supplements are at a much higher risk of developing lung cancer [24].

6.2 Wet AMD

Recent advances in AMD have been linked to the treatment of neovascular macular degeneration. Prior to 2000, laser therapy and / or surgical procedures were the only available treatments for neovascular macular degeneration [25]. Thermal laser therapy has been used to photocoagulate CNV, which invariably caused damage to the overlying retina, while surgical techniques attempted to remove CNV under the macula or to relocate the macula to an area of the fovea without choroidal neovascularization. The results of both types of treatment were suboptimal, associated with poor visual results and higher recurrence rates [26].

Photodynamic therapy with verteporfin has been used to treat certain types of neovascular choroidal membranes since 2000. Verteporfin (intravenous) is a photosensitizer that is activated when exposed to low-intensity light of a certain wavelength. free radicals, which damage endothelial cells and cause secondary platelet adhesions, degranulation and thrombosis with subsequent occlusion of abnormal blood vessels. This deliberately destroys the choroidal neovascular membrane without damaging the overlying retina [27].

7. CONCLUSION

Risk factors for AMD can be broadly divided into personal factors (e.g., age, gender, race / ethnicity, heredity, and socioeconomic status) and environmental factors (e.g., smoking, sun exposure, and dietary factors including micronutrients, edible fish). Alcohol Consumption and Consumption) Signs of AMD are: drusen, one of the first signs of AMD, and the presence of neovascularization within the macula in wet AMD. The clinical examination is usually sufficient to make a diagnosis of AMD, but with additional tests. Fundus autofluorescence, optical coherence tomography, fluorescein angiography, and indocyanine green angiography, it is useful for subtle macular abnormalities. There is no form of treatment that can prevent vision loss; treatment can be helpful for poor eyesight.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle4.com/review-history/71577