# GEORGIAN MEDICAL MEWS

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# ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии საქართველოს სამედიცინო სიახლენი

# **GEORGIAN MEDICAL NEWS**

Monthly Georgia-US joint scientific journal published both in electronic and paper formats of the Agency of Medical Information of the Georgian Association of Business Press. Published since 1994. Distributed in NIS, EU and USA.

GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

# К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

- 1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра. Используемый компьютерный шрифт для текста на русском и английском языках Times New Roman (Кириллица), для текста на грузинском языке следует использовать AcadNusx. Размер шрифта 12. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.
- 2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.
- 3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

- 4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).
- 5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи. Таблицы и графики должны быть озаглавлены.
- 6. Фотографии должны быть контрастными, фотокопии с рентгенограмм в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста в tiff формате.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

- 7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.
- 8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform\_requirements.html В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.
- 9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.
- 10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.
- 11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.
- 12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

# REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

- 1. Articles must be provided with a double copy, in English or Russian languages and typed or computer-printed on a single side of standard typing paper, with the left margin of 3 centimeters width, and 1.5 spacing between the lines, typeface Times New Roman (Cyrillic), print size 12 (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.
- 2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.
- 3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

- 4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.
- 5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles. Tables and graphs must be headed.
- 6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

- 7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.
- 8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform\_requirements.html http://www.icmje.org/urm\_full.pdf
- In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).
- 9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.
- 10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.
- 11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.
- 12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

#### ᲐᲕᲢᲝᲠᲗᲐ ᲡᲐᲧᲣᲠᲐᲓᲦᲔᲑᲝᲓ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

- 1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში Times New Roman (Кириллица), ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ AcadNusx. შრიფტის ზომა 12. სტატიას თან უნდა ახლდეს CD სტატიით.
- 2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ,რუსულ და ქართულ ენებზე) ჩათვლით.
- 3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).
- 4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).
- 5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.
- 6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით tiff ფორმატში. მიკროფოტო-სურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სუ-რათის ზედა და ქვედა ნაწილები.
- 7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა უცხოური ტრანსკრიპციით.
- 8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.
- 9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.
- 10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.
- 11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.
- 12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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# NEW INSIGHTS INTO THE PATHOGENESIS AND TREATMENT ADVANCES OF AGE - RELATED MACULAR DEGENERATION

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#### Abstract.

Age-related macular degeneration (AMD) is a prevalent and vision - threatening disease among the elderly. This review article focuses on the latest understanding of its pathogenesis and the significant treatment advances. In terms of pathogenesis, new research reveals the complex interplay of genetic factors, such as complement pathway gene mutations, oxidative stress, chronic inflammation, and abnormal lipid metabolism in the development of AMD. The treatment landscape has also witnessed remarkable progress, with anti-vascular endothelial growth factor (VEGF) therapies revolutionizing the management of wet AMD, and emerging treatments like gene therapy, complement pathway inhibitors, and stem cell - based therapies showing great potential for both wet and dry AMD. This article aims to provide a comprehensive overview of these new insights and treatment options, which may contribute to better clinical management and future research directions for AMD.

**Key words.** Age-related macular degeneration, overview, clinical management.

#### Introduction.

Age - related macular degeneration (AMD) is a progressive neurodegenerative disease that primarily affects the macula, the central part of the retina responsible for sharp, central vision. It is a leading cause of severe visual impairment and blindness in individuals over the age of 50 in developed countries [1]. As the global population ages, the prevalence of AMD is expected to rise significantly, posing a substantial public health burden. Understanding the pathogenesis of AMD is crucial for the development of more effective treatment strategies. In recent years, there have been significant advancements in our understanding of the disease mechanisms, along with the emergence of novel treatment modalities.

#### Pathogenesis of AMD.

# **Genetic Factors:**

Genetic studies have identified numerous genetic variants associated with AMD. Mutations in genes involved in the complement system, such as complement factor H (CFH), factor B (BF), and complement component 3 (C3), have been strongly linked to AMD risk [2]. The Y402H polymorphism in the CFH gene is one of the most well -studied genetic risk factors. This polymorphism reduces the ability of CFH to bind to C - reactive protein and glycosaminoglycans, leading to dysregulation of the alternative complement pathway. As a result, there is over - activation of the complement system in the retina, causing chronic inflammation and damage to the retinal pigment epithelium (RPE) and choroid [3].

In addition to complement pathway genes, other genes like age related maculopathy susceptibility 2 (ARMS2) and high - temperature requirement factor A1 (HTRA1) have also been associated with AMD. The ARMS2/HTRA1 locus on chromosome 10q26 is a major genetic risk factor. Although the exact function of ARMS2 is still unclear, HTRA1 is a serine protease that may play a role in extracellular matrix remodeling. Abnormal expression of HTRA1 can disrupt the normal structure and function of the RPE - Bruch's membrane - choriocapillaris complex, contributing to AMD development [4].

#### **Oxidative Stress:**

Oxidative stress is considered a key factor in the pathogenesis of AMD. The retina is highly susceptible to oxidative damage due to its high oxygen consumption, abundant polyunsaturated fatty acids, and continuous exposure to light. With aging, the balance between reactive oxygen species (ROS) production and antioxidant defense mechanisms in the retina is disrupted. Mitochondrial dysfunction in RPE cells can lead to increased ROS generation. Excessive ROS can oxidize lipids, proteins, and DNA in the RPE and photoreceptor cells, causing cell damage and death [5].

Moreover, oxidative stress can up-regulate the expression of pro - inflammatory cytokines and chemokines, further promoting inflammation in the retina. Oxidized lipoproteins can accumulate in Bruch's membrane, forming drusen - like deposits. These deposits can interfere with the normal exchange of nutrients and waste products between the RPE and the choriocapillaris, ultimately leading to RPE atrophy and the development of AMD [6].

#### **Chronic Inflammation:**

Chronic inflammation is an important feature of AMD. The activation of the complement system, as mentioned above, is a major contributor to the inflammatory response in the retina. In addition to complement activation, there is also an increase in the expression of other inflammatory mediators such as interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) in the eyes of AMD patients [7].

Infiltration of immune cells, including macrophages and T lymphocytes, into the retina and choroid has been observed in AMD. Macrophages can phagocytose debris and apoptotic cells, but in the context of AMD, they may also secrete pro - inflammatory cytokines and contribute to tissue damage. T lymphocytes can recognize and attack self-antigens in the retina, leading to an autoimmune - like response that further exacerbates inflammation and tissue destruction [8].

# **Abnormal Lipid Metabolism:**

Recent research has highlighted the role of abnormal lipid metabolism in AMD pathogenesis. Lipid deposits are a characteristic feature of AMD, and drusen contain a variety of lipids, including cholesterol esters, triglycerides, and phospholipids. Abnormalities in lipid transport and metabolism genes, such as apolipoprotein E (APOE), have been associated with an increased risk of AMD.

APOE plays a crucial role in lipid transport and clearance. Different APOE isoforms (APOE2, APOE3, and APOE4) have different affinities for lipoprotein receptors. The APOE4 allele is associated with a higher risk of AMD, as it may lead to impaired lipid clearance and increased lipid accumulation in the retina. Additionally, dysregulation of lipid metabolism can lead to the formation of oxidized lipids, which can trigger oxidative stress and inflammation in the retina [9].

#### Treatment Advances of AMD.

# Anti - VEGF Therapies for Wet AMD:

Wet AMD is characterized by the growth of abnormal blood vessels (choroidal neovascularization-CNV) under the retina, which can leak

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blood and fluid, causing rapid vision loss. Anti-vascular endothelial growth factor (VEGF) therapies have revolutionized the treatment of wet AMD. VEGF is a key factor in promoting angiogenesis, and blocking its activity can inhibit the growth and leakage of CNV.

Currently, several anti-VEGF drugs are available, including ranibizumab (Lucentis), bevacizumab (Avastin), aflibercept (Eylea), and faricimab (Vabysmo). Ranibizumab is a humanized monoclonal antibody fragment that specifically binds to all active isoforms of VEGF - A. Bevacizumab is a full - length monoclonal antibody originally developed for cancer treatment but is also widely used off - label for wet AMD. Aflibercept is a recombinant fusion protein that binds to VEGF-A, VEGF-B, and placental growth factor (PIGF) with high affinity [10].

Faricimab is a novel bispecific antibody that targets both VEGF-A and angiopoietin-2 (Ang-2). By blocking these two key pathways involved in angiogenesis and vascular permeability, faricimab has shown the potential to provide longer - lasting efficacy compared to traditional anti-VEGF monotherapies. Clinical trials have demonstrated that anti-VEGF therapies can stabilize or improve vision in the majority of wet AMD patients. However, they require regular intravitreal injections, which can be inconvenient for patients and may be associated with risks such as endophthalmitis and retinal detachment [11].

#### Gene Therapy:

Gene therapy holds great promise for the treatment of AMD. One approach is to deliver genes encoding anti-VEGF proteins to the retina using viral vectors. For example, adeno - associated virus (AAV) vectors can be used to transfer the gene for a soluble VEGF receptor or an anti-VEGF antibody into RPE cells. Once transfected, these cells can continuously produce anti-VEGF proteins, providing a long - term solution to inhibit CNV growth in wet AMD.

Another gene - therapy approach is to correct genetic mutations associated with AMD. For example, in cases where AMD is caused by mutations in genes like CFH, gene - editing technologies such as CRISPR - Cas9 could potentially be used to correct the mutations in the relevant cells. Although gene therapy is still in the experimental stage, early results from pre - clinical and clinical trials are encouraging, and it may offer a more definitive treatment option in the future [12].

## **Complement Pathway Inhibitors for Dry AMD:**

Dry AMD, which accounts for about 80-90% of all AMD cases, is characterized by the presence of drusen and progressive atrophy of the RPE and photoreceptor cells. Currently, there is no cure for dry AMD, but recent research has focused on targeting the complement pathway, which plays a crucial role in its pathogenesis.

Pegcetacoplan (Syfovre) and avacincaptad pegol (Izervay) are two complement pathway inhibitors that have been approved for the treatment of dry AMD with geographic atrophy. Pegcetacoplan targets the C3 complement protein, while avacincaptad pegol inhibits the C5 complement protein. By blocking these key components of the complement cascade, these drugs can reduce the chronic inflammation and tissue damage associated with dry AMD. Clinical trials have shown that these drugs can slow down the progression of geographic atrophy, although they do not improve vision directly [13].

#### **Stem Cell - Based Therapies:**

Stem cell - based therapies are emerging as a potential treatment for AMD. The goal is to replace the damaged RPE cells with healthy cells derived from stem cells. Embryonic stem cells (ESCs), induced pluripotent stem cells (iPSCs), and adult stem cells, such as mesenchymal stem cells (MSCs), have all been investigated for this purpose.

ESCs and iPSCs can be differentiated into RPE - like cells in vitro and then transplanted into the retina. These transplanted cells may be able to integrate into the existing retinal tissue, replace the dysfunctional RPE cells, and restore normal retinal function. MSCs, on the other hand,

may exert their therapeutic effects through immunomodulation and secretion of growth factors that promote tissue repair and regeneration. Although there are still many challenges to overcome, such as ensuring the safety and long - term survival of transplanted cells, stem cell-based therapies offer new hope for AMD patients, especially those with advanced disease [14].

# Conclusion.

The pathogenesis of AMD is a complex process involving multiple factors, including genetic mutations, oxidative stress, chronic inflammation, and abnormal lipid metabolism. Our understanding of these mechanisms has significantly advanced in recent years, which has led to the development of novel and more effective treatment strategies. Anti - VEGF therapies have transformed the management of wet AMD, and new drugs with longer - lasting efficacy are emerging. For dry AMD, complement pathway inhibitors offer the potential to slow disease progression, and stem cell - based therapies and gene therapy hold great promise for the future. However, more research is needed to optimize these treatment modalities, improve patient outcomes, and ultimately find a cure for AMD. Future studies should also focus on early diagnosis and prevention strategies to reduce the burden of this devastating disease on the aging population.

#### Conflict of interest statement.

The authors declare that this research was conducted in the absence of any business or financial relationships that could be construed as potential conflicts of interest.

#### Data Availability.

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

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