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A Review on Retinitis Pigmentosa (RP)

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Abstract: *Retinitis Pigmentosa (RP) is a group of inherited retinal disorders characterized by the progressive degeneration of photoreceptor cells (rods and cones) in the retina, leading to vision loss. The condition typically starts with night blindness and loss of peripheral vision due to the degeneration of rod cells, which are responsible for low-lig0ht vision. Over time, cone cells, responsible for central and color vision, also degenerate, causing further vision impairment, including loss of central vision and, in severe cases, complete blindness. RP is genetically heterogeneous, with mutations in over 80 different genes linked to the condition. It is commonly inherited in autosomal dominant, autosomal recessive, or X-linked patterns. Currently, there is no cure for RP, but ongoing research is exploring gene therapy, retinal implants, and pharmacological approaches to slow disease progression and restore vision. Early diagnosis, genetic counselling, and supportive interventions such as low-vision aids can help manage the disease and improve quality of life for affected individuals. Retinitis Pigmentosa (RP) is a group of inherited retinal disorders characterized by the progressive degeneration of photoreceptor cells (rods and cones) in the retina, leading to vision loss. The condition typically starts with night blindness and loss of peripheral vision due to the degeneration of rod cells, which are responsible for low-light vision. Over time, cone cells, responsible for central and color vision, also degenerate, causing further vision impairment, including loss of central vision and, in severe cases, complete blindness. RP is genetically heterogeneous, with mutations in over 80 different genes linked to the condition. It is commonly inherited in autosomal dominant, autosomal recessive, or X-linked patterns. Currently, there is no cure for RP, but ongoing research is exploring gene therapy, retinal implants, and pharmacological approaches to slow disease progression and restore vision. Early diagnosis, genetic counselling, and supportive interventions such as low-vision aids can help manage the disease and improve quality of life for affected individuals.*

Keywords: Retinitis Pigmentosa.

I. INTRODUCTION

The disease was not given the name retinitis pigmentosa (RP) until 1857, after it was first identified as a clinical problem in 1853.[1] The name retinitis is still used today, with the fact that inflammation has little to do with the disease's natural course and is generally thought to be a misnomer. RP is a collection of illnesses that cause progressive vision loss rather than a single condition. RP, often referred to as hereditary retinal dystrophy, is the most prevalent inherited retinal disease, affecting 1 in 4000 individuals in the US and about one in 5000 globally.[2]

The Initial sign of RP is typically nyctalopia, or loss of night vision, followed by a progressive narrowing of the visual fields, though it can present and proceed with a range of clinical symptoms. Tunnel vision or total vision loss may develop over time, based on the nature and rate of illness progression. Other symptoms, such as a loss of specific colour separation and ultimately a loss of visual acuity, may also appear as the illness develops. Even with late-stage RP, the macula will continue to function, allowing most patients to retain some light perception. The development of photopsia, or seen flashes of light, which is caused by sensory loss, is the most distressing late symptom of RP. [3] The condition may cause "no syndromic" RP, which is vision loss only. No syndromic cases consider for about 70–80% of RP patients. [1,] The term "syndromic" refers to RP that interacts with systemic illness. With neurosensory hearing loss and visual loss, Usher syndrome is the most common type of syndromic RP. [4,5]

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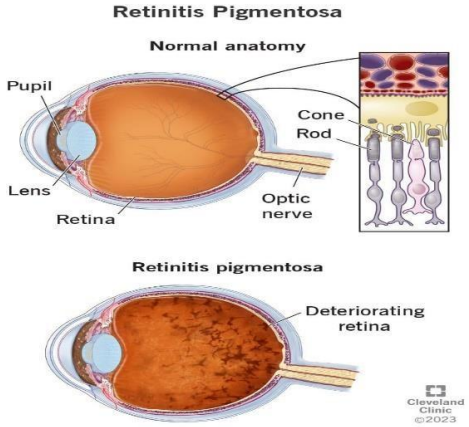

FIGURE NO 1. VERTUAL IMAGE OF RENITIS PIGMENTOSA

HISTORY

Complaints of visual abnormalities starting at age 20 are the typical presentation of RP. These problems typically involve poor vision in low light or circumstances requiring quick transitions from bright to dark settings. Some people report having trouble driving at night because it's hard for their eyes to adjust to the darkness after being exposed to bright light sources like headlights. In addition, it takes time for the visual fields to constrict, which is not immediately noticeable. Determining the sort of inheritance pattern implicated and aiding in diagnosis will require a genetic pedigree of the patient's family. To find more systems impacted and syndromic variations of RP, a thorough history and review of systems are required. Examine any potential exposure to poisons or infectious diseases that could cause a "mimic" of the illness. A fundoscopic examination reveals the "classic triad" of aberrant waxy pallor of the optic disc, vascular constriction, and bony spicule colouring. [6]

Early in the disease, these might not be noticeable, and the severity of the condition affects how severe the abnormalities occur. The onset of arterial attenuation is quite early. In certain situations, even though the electroretinogram indicates rod failure, the optic nerve may appear normal (retinitis pigmentosa sine pigmento). Macular oedema and posterior subcapsular cataracts, which affect up to 72% of patients, are possible additional physical findings. Patients with RP are more likely than the general population to develop this condition, even if external eye exams are typically routine. Keratoconus development is still extremely uncommon. [7]

PATHOPHYSIOLOGY

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The progressive degeneration of photoreceptor cells in the retina is a sign of RP, an inherited retinal disease that causes severe vision impairment. Investigating the processes behind photoreceptor degradation, the consequences of genetic alterations, and their impacts on visual function and quality of life are all essential to understand the pathophysiology of RP. [8] The main cause of photoreceptor degeneration in RP is apoptotic or programmed cell death, which is brought on by genetic abnormalities that impact the retina's rods and cones. Early symptoms including night blindness and peripheral vision loss are caused by rod photoreceptors, which are essential for low-light vision, being more impacted than cone photoreceptors. [9]

Cone cells degrade as the illness worsens, impairing colour perception and central vision. When harmful products accumulate inside photoreceptors, cellular stress and apoptosis occur, starting the degeneration process. Additionally, additional degeneration in nearby cells may result from this cell death, increasing retinal damage. Further complicating the pathophysiology of the disease, the distinctive "bony spicule" pigmentation seen in RP is caused by pigment accumulation in the retina and separation of retinal pigment epithelial cells. [10] The quality of life is impacted by this progressive loss of visual acuity, which increases psychological discomfort and dependence. Mobility, reading, and face

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recognition are major challenges for people with RP, which can lead to social isolation and a decline in wellbeing. The absence of standardised treatments highlights the urgent need for continued study and the creation of efficient therapies, even though rehabilitation programs and low-vision equipment can help with some difficulties. [11]

EPIDEMIOLOGY

Nonsyndromic RP has a worldwide prevalence of about 1 in 5000 individuals. [1] About 50% of instances of hereditary retinal disorders are caused by RP, and [8] impacts about 1.5 million individuals globally. [12] prevalence estimates start at 1 in 3026. [13]

The genetic type at play determines the typical age at which symptoms appear. Those who have autosomal dominant RP are unlikely to experience symptoms until well into their 20s, whereas those with autosomal recessive RP will exhibit symptoms in the initial stages of adolescence. By the time they are 30 years old, almost 75% of people with RP will have symptoms and be seen for a clinical assessment and diagnosis.[12] According to a Japanese study, the median age at diagnosis was 36.5 years, with an average of 35.1 years.[14]

SYMPTOMS

1) Early signs and symptoms of retinitis pigmentosa include:
• Problems with night vision.
• Problems seeing in dim light.
• Blind spots in peripheral (side) vision.
2) Later retinitis pigmentosa signs and symptoms may include:
• Having a sensation of twinkling or flashing light.
• Having tunnel vision (only central vision).
• Being sensitive to or uncomfortable in bright light (photophobia).
• Losing the ability to see color.
• Having very low vision.[16]

DIAGNOSTIC APPROACHES OF RETINITIES PIGMENTOSA

Retinal pigmentopathy (RP) is a genetic retinal abnormality characterized by progressive loss of vision, mostly affecting the retina's photoreceptors. For treatment measures and efficient management, an accurate diagnosis is necessary. Currently used methods for diagnosis include evaluating clinical symptoms, using diagnostic instruments, using imaging methods, and carrying out genetic tests. [17] Individuals diagnosed with RP often have a wide spectrum of symptoms that differ in intensity and timing. Night vision loss, this is frequently one of the first symptoms and makes it difficult for people to see in low light or become used to darkness, is one of the common clinical presentations. Patients eventually develop tunnel vision, an increasing of their viewing area brought on by a loss of peripheral vision as the condition harms.

[18] Considering the variation in Testing genes of RP, genetic testing is essential to the Undiagnosed and treatment of the disorder. More than 65 genes are believed to be linked to RP, and certain mutations can be found through genetic testing, which helps with diagnosis and prognosis. [19] Understanding genetic causes of RP is crucial for regulating expectations about the course of the disease and family planning in particular. Genetic counselling plays a key role in this regard. Genetic counsellors are a great source of knowledge regarding inheritance patterns and how they affect family members. Genetic testing will be essential for establishing eligibility for gene therapies targeting mutations when they become available. [20] The current methods for RP diagnosis are shown in Figure 2.

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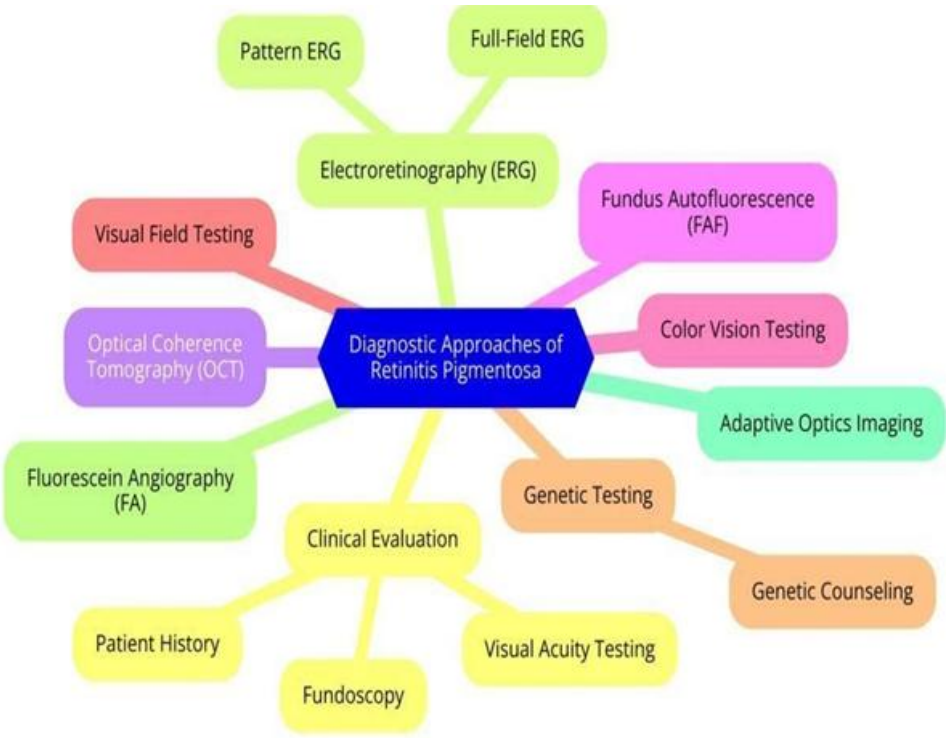

FIGURE NO 2: DIAGNOSTIC APPROACHES OF RETINITIS PIGMENTOSA

CONVENTIONAL APPROACHES TO MANAGEMENT

The management of RP mostly focusses on supportive care because there are currently few alternatives for reversing the illness. In addition, with vitamin A is one of the most thoroughly studied conventional approaches. Research has shown that in certain hereditary subtypes of RP, vitamin A nutritional supplements, especially in the form of palmitate, may reduce the r ate of vision loss. [21] Approximately 15,000 IU should be taken daily. However, in order to control dosage and prevent any toxicity brought on by excessive vitamin A levels, patients should speak with medical specialists. [22] Apart from vitamin supplements, the standard of life of patients with RP is markedly improved by diverse visual aids and methods that are adaptive. Telescopic lenses, electronic visual aids, and magnifiers are examples of low-vision aids that can help patients make the most of their remaining eyesight. In addition, the emergence of smartphone applications intended to help with reading and navigating has opened up new opportunities for freedom. [23] These applications frequently use audio feedback to improve user interaction with the surroundings. Furthermore, adjusting the knowledge in living areas can increase safety and visibility, allowing people to move around with more confidence. [24] Support services and treatment are essential for assisting people with RP in adjusting to vision loss. Using tools like a walking stick or other mobility devices, movement and orientation training teach people how to properly navigate the environment. [25]

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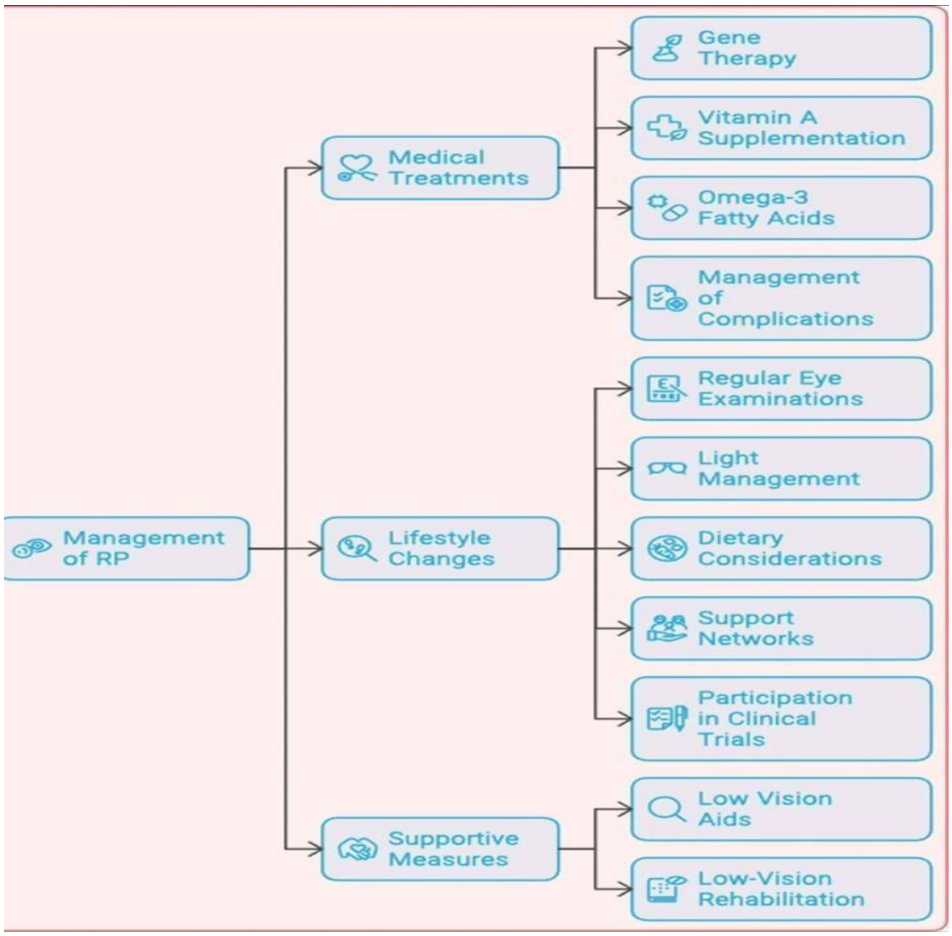

Figure no 3: management of Retinitis Pigmentosa

TREATMENT

Gene Therapy

The treatment of RP can be significantly improved by the developments in gene therapy, particularly when the phase I trial by Ghazi et al. (2016) is considered. This investigation was limited to individuals whose RP was brought on by changes in the c-mer proto-oncogene tyrosine kinase (MERTK) gene, which codes for a protein that RPE cells utilize to phagocytose photoreceptor outer segments. This trial demonstrated that this kind of genetic therapy was risk- free and did not have any negative effects on those receiving it. Additionally, there were some signs of good effectiveness, such as the patients' improved visual function following the application of the medication under analysis. This is particularly noteworthy in light of the fact that RP is a degenerative disease that causes a progressive loss of photoreceptor cells, which eventually results in blindness and total vision loss. However, the authors stressed the need for additional research to examine the therapy's long-term washout effects to determine its effectiveness in other hereditary subgroups of RP. RP is a recessive condition characterized by significant genetic variability, wherein the disease progression is associated with over 70% of identified mutations. If gene therapy is beneficial in treating other types of RP caused by different genes, more study will be required to verify conclusion. [26]

Cell-Based Therapy

The encouraging treatments for RP include cell-based therapies, especially those that use induced pluripotent stem cells (iPSCs) and stem cell transplantation. These methods attempt to bring back retinal function and provide fresh approaches to the treatment of this degenerative illness. [27] PSCs are a cutting-edge method in the field of regenerative

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medicine. iPSCs, which are calculated from somatic cells, have the ability to be reprogrammed to look like embryonic tissue, which enables them to develop into any type of cell, including retinal tissue. The capacity of iPSCs to produce patient-specific cells significantly lowers the danger of immunological rejection, which is one of its main advantages. [28] Strategies for RP stem cell transplantation centre on restoring injured retinal cells and promoting retinal tissue regeneration. The ability of various stem cell types, such as mesenchymal stem cells, embryonic stem cells, and induced pluripotent stem cells (iPSCs), to develop into retinal tissue forms, like as retinal pigment epithelium (RPE) and photoreceptors, is being investigated. These cells offer trophic support, which may aid in the restoration of injured retinal structures in addition to the capacity to substitute lost cells. [29]

Genome Surgery

In a study, Tsai et al. Investigated potential genome surgery based on clustered regularly interspaced short palindromic repeats (CRISPR) could be used in the case of adRP. Because of the study's favourable results, it is now possible to explore the possibility of correcting changes in genes in RP using genome surgery. Therefore, one mutation in a gene transmitted from one parent causes adRP, a type of the disease. Tsai et al. Investigated if CRISPR might be used to directly modify these mutations in particular photoreceptor cells. In order to prevent the degradation of photoreceptors and restore normal gene activity, the researchers tried to apply CRISPR particles to the retina as a means of treating inherited blindness. [30] Tsai et al.'s study shown that CRISPR-genome surgery may be effective in treating mutations in animal models of adRP. It showed that the treatment could be able to prevent or at least slow down the deterioration of vision. Thus, these results provide credence to the notion that the CRISPR/Cas system could function as a possible therapeutic in adRP. However, the writers also accurately highlighted the shortcomings and limitations of this particular technology. However, the research conducted by Tsai and colleagues represents a noteworthy advancement in the field of genome surgical applications for RP treatment. With the use of gene editing technology, researchers are currently working towards more customized care that would focus on the genetic causes of the illness. Therefore, the development of therapeutic techniques like genome surgery might be seen as a hope for improving the quality of life and restoring vision for those with this severe condition. [30]

RISK FACTOR

A) Inheritance patterns:

The influence of heredity patterns on the course of RP remains uncertain; yet mounting data indicates that inheritance patterns represent a significant risk factor. Due to genetic family history, patients with RP can be classified genetically into many inheritance patterns, including X-chromosome related, AD-RP, and AR-RP. A significant portion of patients is isolated cases. Xu and company. [31] evaluated throughout a period for follow- of up to 29 years (on average, 12 years), and it was discovered that the annual VF loss rate amongst the various genetic patterns did not differ statistically significantly. It is estimated that the annual incidence of AD inheritance is 2.7%, X-linked inheritance is 7.2%, and AR inheritance is 10.3%. Nevertheless, the data show that AR-RP has a propensity to give up VF faster than one of the two genetic variants. The earlier research of Sandberg et al. [32]

B) Genotype:

Several investigations have demonstrated the significance of genotype in the development of RP. Ninety-three genes and loci have so far been linked to RP; they are mostly connected to the phototransduction cascade, visual cycle, and photoreceptor structure (last updated September 29, 2021). Words In photoreceptor neurons, phototransduction is a biological process that translates light absorption into electrical activity. Several gene families, including cyclic nucleotide gated ion channels, transducing, and rhodopsin, have been shown to be involved in the biochemical mechanism. The viability of photoreceptors is highly susceptible to disruptions in phototransduction. Gene mutations encoding phototransduction proteins have the potential to degenerate photoreceptors, so impacting the phototransduction cascade and contributing to the gradual demise of photoreceptors. [33]

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C) Gene variants:

Analysis of the number of variations should be done in conjunction with the molecular genetic diagnosis of RP. There are numerous genes associated with RP; however, even in cases where the genes responsible for the disease are same, the disease's development mode and rate of degeneration may differ. The reason for this is that numerous genes have various variations that result in various pathogenic phenotypes. As an example, Jespersgaard et al. [34]

D) Phenotypic Risk Factors

a) Photoreceptor layer thickness:

The primary pathogenic characteristic of retinal pigment epithelium and photoreceptor modifications is retinal pigment epithelium complex; structural alterations will impact the associated functions, here referring to visual function. Nonetheless, a lot of research aims to elucidate the relationship between optical structure and its function in RP. Sandberg and colleagues. [35]

E) Demographic and Environmental Risk Factors

a) Gender: Similar to how age affects results, the vast majority of data show that gender has little bearing on the typical MD's declining rate, which denotes that RP advancement is independent of age. The influence of gender on the course of the disease was statistically significant among those who have legal blindness, a symptom of an advanced stage of the illness. Men have a risk ratio of 3.03 in comparison to women. [36]

II. CONCLUSION

RP management has witnessed remarkable advancements in recent years, driven by a deeper understanding of the genetic underpinnings and the development of innovative therapeutic strategies. From Groundbreaking genetic therapies and promising cell-based treatments to innovative optogenetics and Retinal prosthetics, these novel approaches offer hope for slowing disease progression, restoring visual Function, and improving the quality of life for individuals with RP. Despite these exciting developments, Challenges remain, including safety issues, accessibility, and the need for further research to validate long-term outcomes. As the field continues to evolve, ongoing collaboration and technological advancements will Be crucial in overcoming these hurdles and moving toward effective and personalized treatments. The Future of RP management holds great promise, with the potential for transformative therapies that could Significantly alter the trajectory of this debilitating condition and bring renewed hope to those affected.

REFERENCES

[1]. Verbakel SK, van Huet RAC, Boon CJF, den Hollander AI, Collin RWJ, Klaver CCW, Hoyng CB, Roepman R, Klevering BJ. Non-syndromic retinitis pigmentosa. Prog Retin Eye Res. 2018 Sep;66: 157-186.

[2]. Boughman JA, Conneally PM, Nance WE. Population genetic studies of retinitis pigmentosa. Am J Hum Genet. 1980 Mar;32(2):223-35.

[3]. Bittner AK, Diener-West M, Dagnelie G. Characteristics, and possible visual consequences of photopsias as vision measures are reduced in retinitis pigmentosa. Invest Ophthalmol Vis Sci. 2011 Aug 11;52(9):6370-6.

[4]. Wolfrum U, Nagel-Wolfrum K. [The Usher Syndrome, a Human Ciliopathy]. Klin Monbl Augenheilkd. 2018 Mar;235(3):273-280.

[5]. Mathur P, Yang J. Usher syndrome: Hearing loss, retinal degeneration, and associated abnormalities. Biochim Biopsy Acta. 2015 Mar;1852(3):406-20.

[6]. Tripathy K, Sharma YR, Chawla R, Basu K, Vohra R, Venkatesh P. Triads in Ophthalmology: A Comprehensive Review. Semin Ophthalmol. 2017;32(2):237- 250.

[7]. Shintani K, Shechtman DL, Gurwood AS. Review, and update: current treatment trends for patients with retinitis pigmentosa. Optometry. 2009 Jul;80(7):384-401. [PubMed]

[8]. Kamde SP, Anjankar A: Retinitis pigmentosa: pathogenesis, diagnostic findings, and treatment. Cureus.2023, 15: e48006. 10.7759/cureus.48006

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Volume 5, Issue 3, January 2025

[9]. Newton F, Megaw R: Mechanisms of photoreceptor death in retinitis pigmentosa. Genes (Basel). 2020,11:1120. 10.3390/genes11101120

[10]. Travis GH, Golczak M, Moise AR, Palczewski K: Diseases caused by defects in the visual cycle: retinoids as Potential therapeutic agents. Annu Rev Pharmacol Toxicol. 2007, 47:469-512. 10.1146/annurev.pharmtox.47.120505.105225

[11]. Cross N, van Steen C, Zegaoui Y, Satherley A, Angelillo L: Retinitis pigmentosa: burden of disease and Current unmet needs. Clin Ophthalmol. 2022, 16:1993-2010. 10.2147/OPTH.S365486

[12]. Cross N, van Steen C, Zegaoui Y, Satherley A, Angelillo L. Retinitis Pigmentosa: Burden of Disease and Current Unmet Needs. Clin Ophthalmol. 2022; 16:1993-2010. [PMC free article] [PubMed]

[13]. Hamilton R, Bees MA, Chaplin CA, McCulloch DL. The Luminance-esponse function of the human photopic elect-o-etinog-am: A Mathematical Model. Vision Res. 2007;47 (23):2968–2972. Doi: 10.1016/j.vis-es.2007.04.020.

[14]. Tsujikawa M, Wada Y, Sukegawa M, Sawa M, Gomi F, Nishida K, Tano Y. Age at onset curves of retinitis pigmentosa. Arch Ophthalmol. 2008 Mar;126(3):337-40. [PubMed]

[15]. Birch DG, Anderson JL, Fish GE. Yearly rates of rod and cone Functional loss in retinitis pigmentosa and cone-rod dystrophy. Ophthalmology 1999; 106: 258–68.17429-retinitis-pigmentosa

[16]. Nguyen XT, Moekotte L, Plomp AS, Bergen AA, van Genderen MM, Boon CJ: Retinitis pigmentosa: current Clinical management and emerging therapies. Int J Mol Sci. 2023, 24:7481.10.3390/ijms24087481

[17]. Retinitis pigmentosa. (2023). Accessed: August 2, 2024:

[18]. Neveling K, Collin RW, Gilissen C, et al.: Next-generation genetic testing for retinitis pigmentosa. Hum Mutat. 2012, 33:963-72. 10.1002/humu.22045

[19]. Genetic counseling. Understanding Genetics: A New York, Mid-Atlantic Guide for Patients and Health Professionals. Genetic Alliance, Washington (DC); 2009.

[20]. Parmeggiani F, Sato G, De Nadai K, Romano MR, Binotto A, Costagliola C: Clinical and rehabilitative Management of retinitis pigmentosa: up-todate. Curr Genomics. 2011, 12:250-9.10.2174/138920211795860125

[21]. Vitamin A and carotenoids. (2023). Accessed: August 2, 2024: https://ods.od.nih.gov/factsheets/VitaminAHealthProfessional/.

[22]. van Nispen RM, Virgili G, Hoeben M, Langelaan M, Klevering J, Keunen JE, van Rens GH: Low vision Rehabilitation for better quality of life in visually

[23]. impaired adults. Cochrane Database Syst Rev. 2020,1:CD006543. 10.1002/14651858.CD006543.pub2

[24]. Messaoudi MD, Menelas BJ, Mcheick H: Review of navigation assistive tools and technologies for the visually Impaired. Sensors (Basel). 2022, 22:7888. 10.3390/s22207888

[25]. Virgili G, Rubin G: Orientation and mobility training for adults with low vision. Cochrane Database Syst Rev. 2010, 2010:CD003925. 10.1002/14651858.CD003925.pub3

[26]. Ghazi NG, Abboud EB, Nowilaty SR, et al.: Treatment of retinitis pigmentosa due to MERTK mutations by Ocular subretinal injection of adeno-associated virus gene vector: results of a phase I trial. Hum Genet.2016, 135:327-43. 10.1007/s00439-016-1637-y

[27]. Chehelgerdi M, Behdarvand Dehkordi F, Chehelgerdi M, et al.: Exploring the promising potential of induced Pluripotent stem cells in cancer research and therapy. Mol Cancer. 2023, 22:189. 10.1186/s12943-02301873-0

[28]. Shi Y, Inoue H, Wu JC, Yamanaka S: Induced pluripotent stem cell technology: a decade of progress. Nat Rev Drug Discov. 2017, 16:115-30. 10.1038/nrd.2016.245

[29]. Nair DS, Thomas BB: Stem cell-based treatment strategies for degenerative diseases of the retina. Curr Stem Cell Res Ther. 2022,17:214-25. 10.2174/1574888X16666210804112104

[30]. Tsai YT, Wu WH, Lee TT, et al.: Clustered regularly interspaced short palindromic repeats-based genome Surgery for the treatment of autosomal dominant retinitis pigmentosa. Ophthalmology. 2018, 125:1421-30.10.1016/j.ophtha.2018.04.001

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[31]. Xu ML, Zhai Y, MacDonald IM. Visual field progression in retinitis pigmentosa. Invest Ophthalmol Vis Sci. 2020;61(6):56. [PMC free article] [PubMed] [Google Scholar]

[32]. Sandberg MA, Rosner B, Weigel-Difranco C, McGee TL, Dryja TP, Berson EL. Disease course in patients with autosomal recessive retinitis pigmentosa due to the USH2A gene. Invest Ophthalmol Vis Sci. 2008;49(12):5532–5539. [PMC free article] [PubMed] [Google Scholar]

[33]. Li BQ, Gografe S, Munchow A, Lopez-Toledano M, Pan ZH, Shen W. Sex related differences in the progressive retinal degeneration of the rd10 mouse. Exp Eye Res. 2019; 187:107773. [PMC free article] [PubMed] [Google Scholar]

[34]. Jespersgaard C, Bertelsen M, Arif F, Gellert-Kristensen HG, Fang MY, Jensen H, Rosenberg T, Tümer Z, Møller LB, Brøndum-Nielsen K, Grønskov K. Bi-allelic pathogenic variations in MERTK including deletions are associated with an early onset progressive form of retinitis pigmentosa. Genes. 2020;11(12):1517. [PMC free article] [PubMed] [Google Scholar]

[35]. Sandberg MA, Brokhurst RJ, Gaudio AR, Berson EL. The association between visual acuity and central retinal thickness in retinitis pigmentosa.

[36]. Invest Ophthalmol Vis Sci. 2005;46(9):3349–3354. [PubMed] [Google Scholar]

[37]. Xu ML, Zhai Y, MacDonald IM. Visual field progression in retinitis pigmentosa. Invest Ophthalmol Vis Sci. 2020;61(6):56. [PMC free article] [PubMed] [Google Scholar]