International Journal of Pharmacology and Clinical Research

ISSN Print: 2664-7613
ISSN Online: 2664-7621
Impact Factor: RJIF 8
IJPCR 2024; 6(1): 33-38
www.pharmacologyjournal.in
Received: 16-03-2024
Accepted: 18-07-2024

Kumar Raja Jayavarapu
Professor and Principal, Mother Teresa Pharmacy College, Kothuru, Sathupally Mandal, Khammam, Telangana, India

D Praveen Kumar
Mother Teresa Pharmacy College, Kothuru, Sathupally Mandal, Khammam, Telangana, India

B. Srinivasa Rao
Mother Teresa Pharmacy College, Kothuru, Sathupally Mandal, Khammam, Telangana, India

B Sangeetha
Mother Teresa Pharmacy College, Kothuru, Sathupally Mandal, Khammam, Telangana, India

K Indravathi
Mother Teresa Pharmacy College, Kothuru, Sathupally Mandal, Khammam, Telangana, India

K Sahithi
Mother Teresa Pharmacy College, Kothuru, Sathupally Mandal, Khammam, Telangana, India

R Omsri
Mother Teresa Pharmacy College, Kothuru, Sathupally Mandal, Khammam, Telangana, India

Corresponding Author:
Kumar Raja Jayavarapu
Professor and Principal, Mother Teresa Pharmacy College, Kothuru, Sathupally Mandal, Khammam, Telangana, India

Opthalmic diseases and it's types

Kumar Raja Jayavarapu, D Praveen Kumar, B Srinivasa Rao, B Sangeetha, K Indravathi, K Sahithi and R Omsri

DOI: https://doi.org/10.33545/26647613.2024.v6.i1a.33

Abstract
As we know that the eye plays a huge role in daily life. Many factors may cause the eye diseases either by contact with many exposures. It may include environmental factors and diseases may occur by the genetics (DNA mutations) and by previous eye injuries. After age 40, your eye tear production tends to decrease, causing dry eyes. Based on web based survey in December 2022 by including 1076 adults in Poland the survey had been conducted. From survey the results are the 83.6 percent of people are infected with cataract disease. Whereas, People of about 80.7 percent effected with glaucoma and 73.8 percent with Hordeolum. It is assumed that more than 50 percent of the most enfeebling ocular diseases have their origin in the posterior segment.
The AMD is characterized by an array of features in the macular region. The various factors include blunt chemical injury. Acute eye infection, genetic reason may cause glaucoma. Harmone replacement therapy and UV radiation and hypertension and other minor factors may cause cataract and retinitis pigmentosa and some factors may cause inherited optic neuropathies and also Marfan syndrome. Finally revised disease diagnostic criteria have been proposed for treatment of various diseases based on careful observation by different diseases.

Keywords: Glaucoma, age related macular degeneration, risk factor, prevention, age knowledge, awareness

Introduction
The eye is the most important organ the body. According to worldwide more than 2 billion people have the vision impairment? There are different types of eye diseases which leads to vision loss [1]. They are glaucoma, myopia, cataract, age related macular degeneration, uveal melanoma, retinitis pigmentosa, Stargardt's disease, Marfan syndrome, inherited neuropathies [2]. The WHO estimates that between 2020 and 2030, the global number of people with glaucoma will increased by 1.3 times and the global number of people with age related macular degeneration (AMD) will increased by 1.2 times [3]. Age is the most important factor numerous eye disease [4].
Many eye disease (eg; Glaucoma) can be asymptomatic or mildly symptomatic for long time [5]. Education on common eye disease may increase the level of public knowledge on eye disease and change individual behaviours which may promote early detection and treatement of eye disease and encourage people at risk to seek regular eye care [6].

Anatomy of eye
Introduction of eye
The eye or the organ of sight is situated in orbital it cavity of the
Skull it is well protected by bony walls of the orbit also contains muscles of eye ball their nerves blood vessels lachrymal gland.

Structure of the eye ball
- Wall of eye ball.
- Light transmitting media.

Wall of the eye ball
- Outer layer (fibrous coat): sclera, cornea.

- Middle layer (vascular coat): choroid, ciliary body, iris.
- Inner layer (nervous coat): retina.

Outer layer
The gives shape of eye ball& contain scleria & cornea.

Middle Layer
It is 3 structure.

- The choroid, culinary body & iris together from the veal tract

Pupil is the small opening in the front of it.

Inner Layer
Retina is the light sensitive membrane.

Sclera & cornea

- Sclera or the white of elite of eye forms shape of eye ball.

Aqueous Humor
Aqueous humor is clear fluid fills the space between cornea and lens it is secreted by capillaries of ciliary process.

Functions

1. It helps maintain intraocular pressure thus maintains the shape of eye ball.
2. It is rich in ascorbic acids glucose amino acids.

Vitereous Humor
Vitereous humor or vitreous body is coloer less transparent jelly like substance fill the posterior segment of the eye it en closed in a delicate hyaloid membrane.

Functions

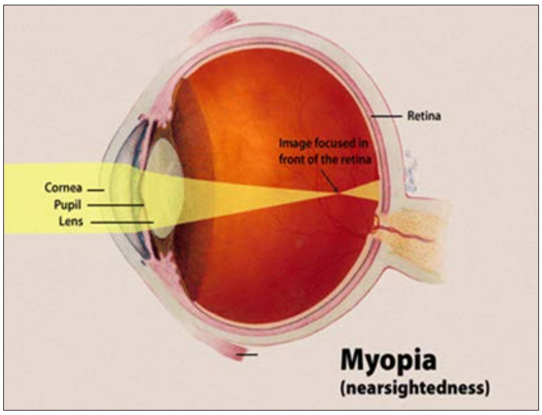
- It helps to preserve the spherical shape of the eye ball to support the retina.

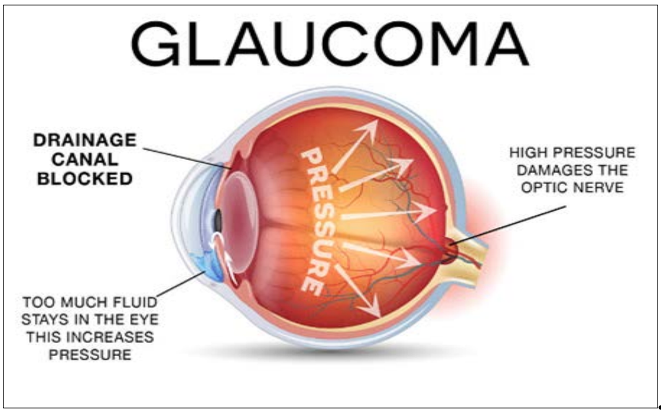
Lens

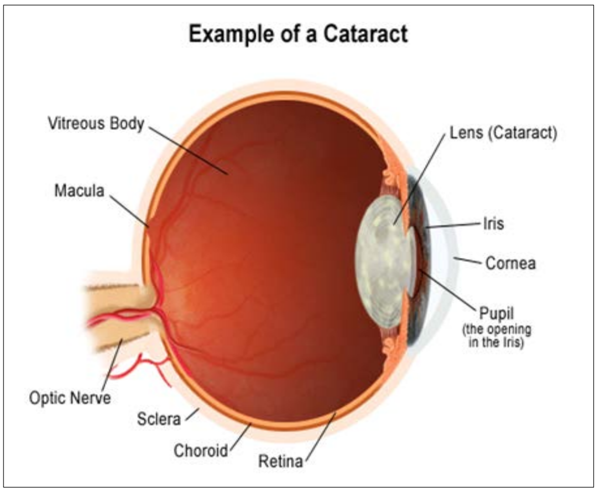
- The lens off the eye ball is crystalline in nature.
- It is situated behind the pupil
- Lens is supported suspensory ligaments which are attached with cilliary

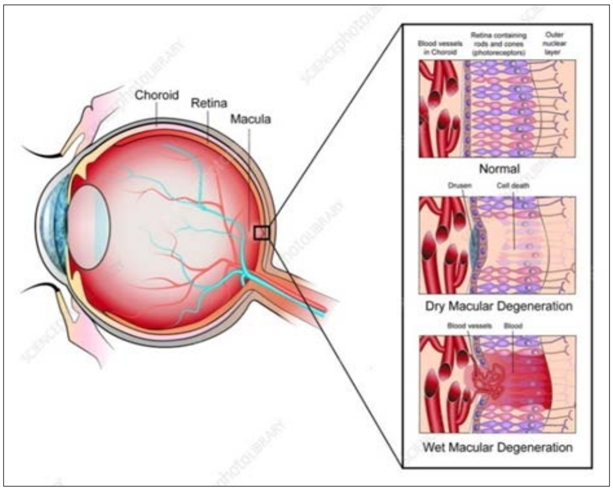
Pupil

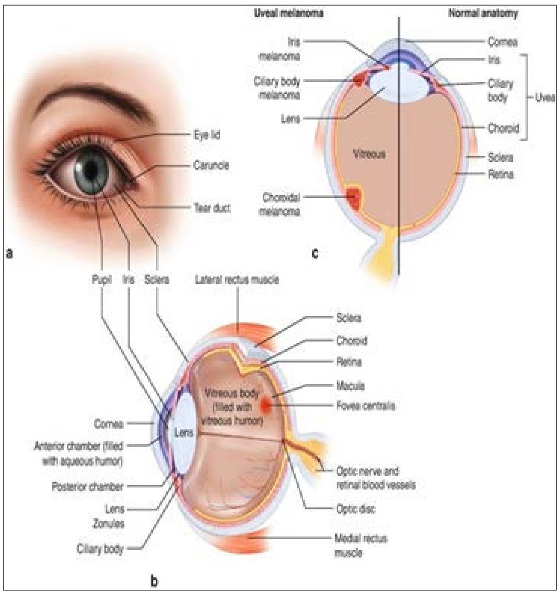
- Pupil is central round the aperture of the refractive system of the eye.
- Pupil in woman is larger than that in man.
- Two pupils are unequal then the condition [5].

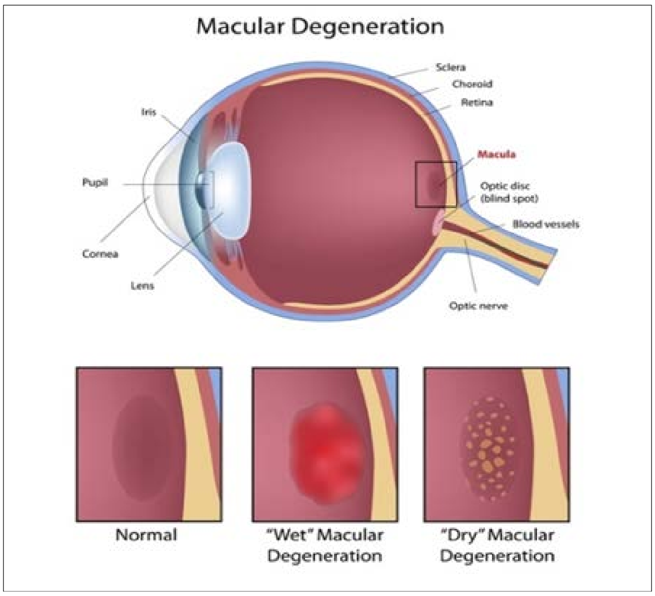


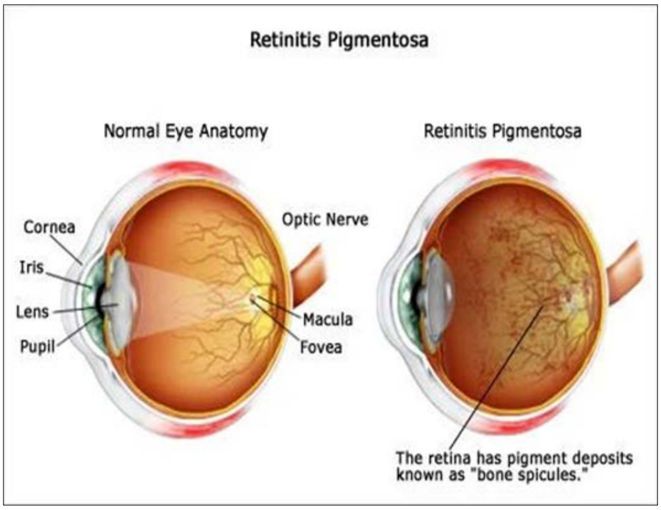


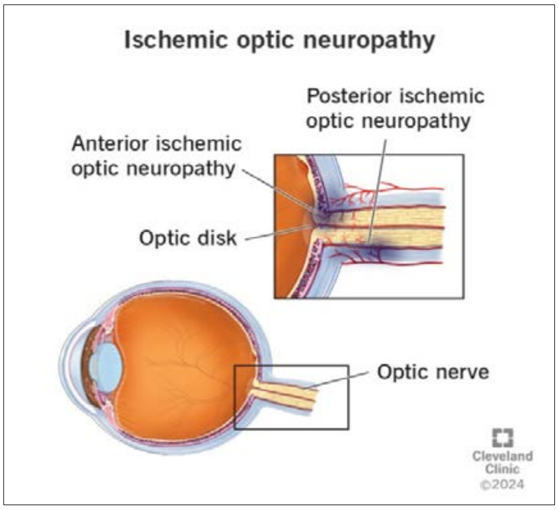


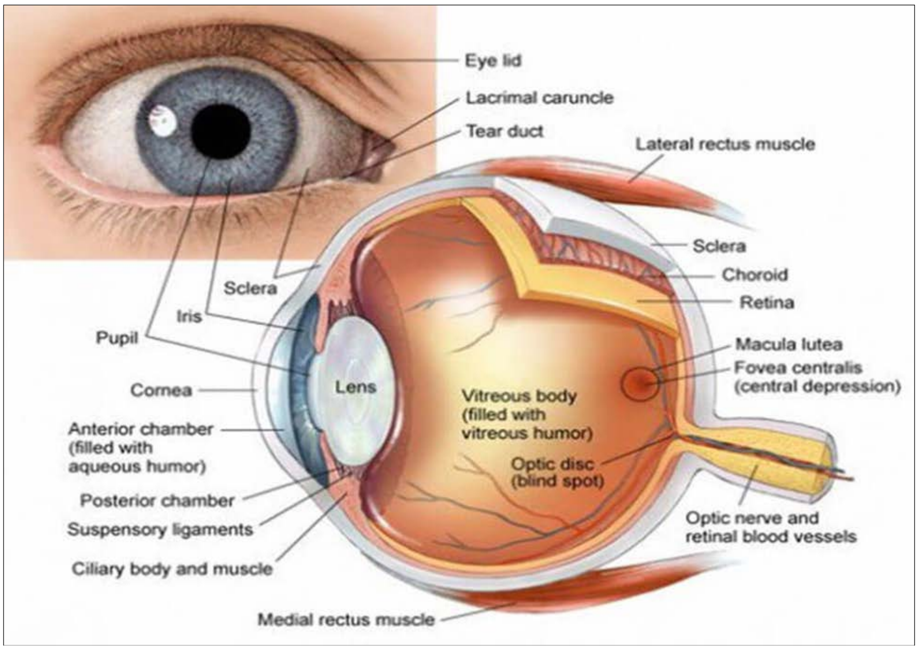



Uveal melanoma
Normal anatomy
Iris melanoma
Ciliary body melanoma
Lens
Vitreous
Choroidal melanoma
Cornea
Iris
Ciliary body
Uvea
Choroid
Sclera
Retina
Eye lid
Caruncle
Tear duct
a
c
Pupil
Iris
Sclera
Lateral rectus muscle
Sclera
Choroid
Retina
Macula
Fovea centralis
Vitreous body (filled with vitreous humor)
Lens
Cornea
Anterior chamber (filled with aqueous humor)
Posterior chamber
Lens
Zonules
Ciliary body
Optic nerve and retinal blood vessels
Optic disc
Medial rectus muscle
b


Macular Degeneration
Sclera
Choroid
Retina
Iris
Macula
Pupil
Optic disc (blind spot)
Blood vessels
Cornea
Lens
Optic nerve
Normal
"Wet" Macular Degeneration
"Dry" Macular Degeneration


Retinitis Pigmentosa
Normal Eye Anatomy
Retinitis Pigmentosa
Cornea
Iris
Lens
Pupil
Optic Nerve
Macula
Fovea
The retina has pigment deposits known as "bone spicules."


Ischemic optic neuropathy
Posterior ischemic optic neuropathy
Anterior ischemic optic neuropathy
Optic disk
Optic nerve
Cleveland Clinic
©2024


Eye lid
Lacrimal caruncle
Tear duct
Lateral rectus muscle
Sclera
Choroid
Retina
Sclera
Iris
Pupil
Macula lutea
Fovea centralis (central depression)
Cornea
Lens
Vitreous body (filled with vitreous humor)
Anterior chamber (filled with aqueous humor)
Optic disc (blind spot)
Posterior chamber
Suspensory ligaments
Optic nerve and retinal blood vessels
Ciliary body and muscle
Medial rectus muscle

Types: The eye diseases are of different types they are

1. Glaucoma
2. Myopia
3. Cataract
4. Age related macular degeneration
5. uveal melanoma
6. Retinitis pigmentosa
7. Stargardt's
8. Marfan syndrome
9. Inherited optic neuropathies

1. Glaucoma

Glaucoma is defined as the accumulation of fluids behind the eye due to this intraocular pressure increases [11]. This effects the optic nerve & include loss of retinal ganglion cells in a characteristic pattern

1. Glaucoma can often divided into 2 types:
   a) open angle glaucoma
   b) Closed angle glaucoma
2. Worldwide one of the main cause of blindness is glaucoma. But only 32.5% of peoples were identify it as a eye diseases [9].
3. This incidence of glaucoma is increased an increasing the age [10].

2. Myopia

1. It is also called short sightedness. Myopia is characterized as that a person can able to near objects but cannot identify the far or distant objects. Due to eye ball is elongated, So that parallel rays are focused in front of retina but divergent rays are focused on it [8].
2. The myopia is being a serious social problem. But effective treatment methods was not developed to rectify the error. A few methods were developed in management of myopia.
3. Due to incomplete understanding of pathogenesis of myopia, no effective method for treatment of myopia.
4. Despite myopia being a serious contemporary social problem no effective treatment method for this refractive error has been developed. Only few methods of myopia management have been developed so far. The lack of an effective method of myopia treatment is caused by the incomplete understanding of the pathogenesis of this disease [12].

3. Cataract

1. It is the condition which causes obstruction in the passage of light to retina [13].
2. The obstruction is occur due to the coagulated protein, calcium decomposition and denaturation.
3. This diseases can be treated by denaturation of proteins and has to remove surgically and to compensate for the loss of refraction [14].
4. In this condition the lens become cloudy due to changes in structural proteins.

4. Inherited optic neuropathies

1. Optic neuropathy and optic atrophy is defined as the damage or death of the optic nerve fibers. It is the most common cause of vision loss in children. In the UK about 28% of pediatric cases of severe sight impairment was identified [15].
2. Generally an optic nerve has a limited repertoire in expression of injury. All optic neuropathies will have some visual activity loss, atypical colour vision, visual field abnormalities and contrast sensitivity [16].
3. Since 30 years elapsed from the landmark in finding the first point mutation in mitochondrial DNA [mtDNA] which was associated with lebers heriditory optic neuropathy by " Doug Wallace" in 1988 [17].
4. The inherited optic neuropathies is a group of disorders because cell death which confined to the retinal ganglion cells [18].
5. The primary hereditary optic neuropathies comprise autosomal recessive and x-linked recessive and x-linked recessive optic atropy and the maternally inherited lebers hereditary optic neuropathy [19].

5. Marfan syndrome

1. This disease is develop due to denova mutation involving the gene (FBN_1) encoding the connective tissue protein fibrillin [20].
2. It is an autosomal dominant condition with a reported incidence of 1 in 3000 to 5000 individuals [22].
3. In 1996 as per ghant criteria, the diagnosis requires atleast presence of a major criterian in two different systems [21].
4. Marfan syndrome (MFS) is an autosomal dominant condition with a reported incidence of 1 in 3000 to 5000 individuals [22].
5. The incidence of classic MFS is about 2-3 per 1000 individuals, although this estimate depends on complete recognition of all affected and genetical predisposed individuals. Gray reported the incidence in Scotland as one in 9802 live births [23].
6. The disease occurs worldwide with no prediction for either sex. Tall sature with dolichostenomelia (long bone overgrowth) leads to increased incidence in certain athletes, including basket ball, volley ball players sometimes prompting a recommendation for screening by echocardiography [24].
7. The majority of medical therapy as it relates to MFS has been targeted at preventing cardiovascular compromise with beta blockers and afterload reducing agents used to reduse stress on the aortic root [25].

6. Uveal Melanoma

1. It is a life threatening ocular disease. It is developed from melanocytes located in the highly pigmented uveal tract. The main oxygen and nutrient provide from retina [26].
2. It is the most common primary eye cancer in adults. The both uveal and cutaneous melanomas originate from melanocyte's but the clinical behaviour differ significantly [27].
3. It is the common primary intraocular malignancy and it can be fatal in adults [27].
4. In the US, the incidence varies from 5.1 to 6 per million population per year, being highest in the southern latitudes. In Europe the incidence of uveal melanomas is much higher in caucians of northern European decent and significantly lower in Italy and spain [28].
5. Uveal melanoma is the most common primary cancer of the eye in adults, although both uveal and cutaneous melanomas both originate from melanocytes their underlying pathogenesis and clinical behaviour differ significantly [29].

6. The natural history of uveal melanoma often involves the development of metastasis, an event associated with poor prognosis. Therefore it is imperative that is treating physician be familiar with the clinical features of uveal melanoma allowing for early treatment and aversion of potentially life threatening metastasis [30].

7. Stargardt's disease

1. It was the first discovered by German opthamalogist "Karl Stargart" in 1909 with macular dystrophy surrounded by yellow white pisciform flecks [31].
2. It is a type of retinal disease with phenotype but eith an autosomal dominant inheritance [32].
3. It is the most common inherited mascular dystrophy identify in adults and childrens including autosomal recessive mode of inheritance which is associated with disease causing mutations in ABCA4 gene [33].
4. There slow progressive is loss of retinal function and structure over time ; however there is marked variability both within and between families, suggesting that other important factors influence phenotype including genetic modifiers and the environment [34].
5. With the emergence of ABCA4 specific molecular and genetic therapies it is essential that the clinical diagnosis of STGD1 is made promptly through the appropriate use of multimodal imaging to guide timely and accurate genetic analysis, allowing for the early environment of these patients into clinical trails of novel therapies [35].

8. Retinitis Pigmentosa

1. It is a group of eye disorders which cause the loss of vision also known as hereditary retinal dystrophy [36].
2. Vision loss is due to abnormalities of retinal photoreceptor cells these disease starts with damage and loss of rod cells (Nyctalopia) defective dark adaptation which were common in patients [37].
3. In 1984 the genetics of Retinities pigmentosa is varied after first report describing a linkage of an retinities pigmentosa locus to a DNA maker on human chromosome X [38].
4. The name rhetinitis pigmentosa described initially as a clinical entity in 1853 was not attached to the disease until 1857 [39].
5. The classic traid in retinitis pigmentosa consist of peripheral bone spicule pigment deposition, blood vessel attenuation and optic disc pallor. Bone spicule pigmentation usually develops in the midstages of the disease, starting at the mid periphery and moves towards the macula as the disease progresses [40].
6. Associates an usually severe retinitis pigmentosa (sometimes diagnosed as LAC) with nephronophtisis or sometimes a milder retinitis pigmentosa that is discovered late in life. At least four genes encoding nephrocystins are involved in this disease [41].

9. Age related macular degeneration (ADM)

1. It is the condition of retina related to age cause central vision loss this condition was observed at the age of 55 [42].
2. Advanced AMD accounts for an estimated 90% of cases of sever vision loss.[43]
3. It is a leading cause of visual impairment in both developed and developing nations, particularly in those aged 65 years and older. Projections indicate by that by the year 2040, the global population affected by AMD is expected to reach approx 288 million people [44].
4. Neovascular AMD can be treated effectively unusing antivascular endothelial growth factor agents, allowing patients to lead productive lives if diagnosed and treated early [45].

Symptoms and Causes

What are the symptoms of eye diseases?

There are many possible symptoms of eye diseases. Those symptoms usually happen in a few key ways:

- **Symptoms you can feel**: Examples include pain, irritation or eye tiredness or strain.
- **Changes in eye functions**. Examples include watery eyes (Epiphora) and trouble controlling how you blink your eyes.
- **Changes in the appearance of your eyes:** These changes include yellowing of the white of your eyes (scleral icterus), red eyes and pupils that are too narrow (miosis).
- **Changes in eye movement or alignment:** Examples include exotropia or esotropia.
- **Changes in how you see**: Some common examples are double vision (diplopia), blurred vision or tunnel vision.

What causes eye diseases?

Eye diseases can happen for many, many reasons. Some are direct causes while others are contributing or risk factors.

- **Genetics**. Your DNA is a key part of how your eyes develop and work, and DNA changes (mutations) can cause many eye conditions. The genetic form of color blindness is an example.
- **Differences in how your eyes form and develop**: These differences could have started while you were a developing fetus, or at some point during your childhood.
- **Environmental factors:** Some eye conditions develop because of things happening to or around you. Ultraviolet light exposure, humidity and temperature, dust or particles in the air, and many other variables can be a part of this.
- **Infectious Diseases:** Many types of germs and microbes (pathogens) can cause eye diseases. They include viruses, bacteria, fungi and parasites. These can infect your eyes directly or spread from elsewhere in your body.
- **Other Chronic Conditions**. Your eyes are vulnerable to many conditions that affect your whole body. Examples include high blood pressure (hypertension), Type 2 diabetes and several types of thyroid disease.
- **Previous eye injuries:** Some events that damage your eye can make you more prone to developing certain eye diseases or issues.
- **Idiopathic causes:** Idiopathic means the cause is unknown. That means there's no cause that experts can identify (Though this can sometimes change over time).

Diagnosis and Tests

How are eye diseases diagnosed?

An eye care specialist or other healthcare provider can diagnose eye diseases using a combination of methods. The first and foremost tool for diagnosing these diseases is an eye exam. While most people think of these as just checking how well you see (visual acuity), that's not always the case.

Eye exams can be routine and happen every one to two years like a regular wellness check. Providers can also do exams in a more specific way when you have signs or symptoms of an eye condition. Certain parts of an eye exam, like pupil dilation and a slit lamp exam, can be especially useful.

Your eye specialist may also recommend more specific eye tests. These include

- Fluorescein angiography.
- Tonometry.
- Retinal imaging.
- Corneal topography.
- Optical coherence tomography.

Many other tests that aren't eye-specific can help, too. They include

- Blood tests looking for specific chemical markers related to immune conditions, infections or to run DNA and genetic tests.
- Imaging tests like ultrasound, computed tomography (CT) scans and magnetic resonance imaging (MRI).
- Neurological tests like electroencephalogram (EEG), which analyzes brain activity.

Your eye care specialist or other healthcare provider can tell you more about any other tests they recommend, including how those tests work and how they may help you.

Diagnostic Services and Treatment Options

Our comprehensive ophthalmology care includes the latest diagnostic methods, medical treatments, and surgical techniques. These services include:

- Screening for common eye diseases, as well as treatment for glaucoma, cataracts, and diabetic retinopathy.
- Surgically implanted lenses to restore vision after cataract surgery.
- Diagnosis of and treatment for conditions such as dry eye syndrome and macular degeneration.
- General evaluation for patients with undiagnosed eye problems.
- Routine vision care such as comprehensive eye exams and vision testing.

Treatment for eye infections depends on what's causing your eye infection. It might include anti-infective medication in various forms, such as eye drops, ointments or tablets. It usually takes 24 to 48 hours for antibacterial medications to work.

You may have to let viral or allergic infections clear on their own, but you may be able to make yourself more comfortable with the following home remedies:

- Using damp, warm or cool compresses to make your eyes feel better.
- Using lubricating eye drops (artificial tears).
- Using over-the-counter (OTC) pain relievers.
- Avoiding allergens that caused the reaction in the first place.
- If routine testing indicates that you have a refractive error, conventional treatment calls for wearing corrective glasses or contact lenses. Over 150 million people in the US wear corrective lenses. In many cases, surgical correction of refractive errors is possible using modern surgical techniques such as LASIK.
- Conventional treatment for disorders such as nearsightedness, farsightedness, and astigmatism typically relies on corrective prescription lenses. Disorders such as cataracts, glaucoma, macular degeneration, and retinal detachment require advanced medical and surgical treatments. Enormous progress in eye surgery has been made over the past few decades. Many people with eye problems previously felt to be untreatable now enjoy improved eyesight and an improved quality of life. This applies to individuals of every age -- infants to senior citizens!
- To treat nearsightedness, your eye doctor will usually prescribe lenses to focus visual images correctly on the retina. Depending on the specifics of your eye exam, you may have a choice between wearing conventional eyeglasses and contact lenses.
- As an alternative to corrective lenses, surgery can sometimes be performed to treat nearsightedness. Excimer laser treatment (like LASIK) uses a laser beam to remove microscopic amounts of tissue from the cornea. This effectively flattens or steepens the cornea as needed so that light rays focus correctly on the retina. LASIK is safe and effective for most patients with nearsightedness, but only up to a certain level. Some patients with thin corneas, extreme nearsightedness, or significant dry eye may be better served with a variant of LASIK called PRK or they may not be good laser candidates at all. There are advanced options for these patients, including surgical correction with implantable contact lenses.
- To treat farsightedness that does not resolve itself naturally, glasses or contact lenses can be prescribed. People typically seek treatment for farsightedness when they begin to complain of eyestrain, especially at the end of the day, or when they have trouble focusing while reading. LASIK can also be used to treat lower levels of farsightedness. Higher levels of farsightedness can only be surgically corrected with a refractive lens exchange -- the natural lens with an intraocular lens that is more powerful, thereby focusing the light on the retina.

For macular degeneration, there is currently no cure for age-related macular degeneration (AMD), but new treatments are sometimes able to prevent severe visual loss. Treatment depends on the type of AMD, but may include:

- Injections of medication into the eye -- to prevent the formation of new, leaky blood vessels
- Laser therapy -- to destroy abnormal blood vessels
- Photodynamic therapy -- injection of medications that are activated by light exposure
- Submacular surgery (very rarely done) -- to remove blood under the retina, abnormal blood vessels or scar tissue
- Retinal translocation (very rarely done) -- surgical rotation of the damaged retinal tissue away from the macula
- Low-vision aids -- to help people with partial vision make the most of their remaining vision. TA wide range of aids are available including devices that have special lenses, electronic systems that magnify images so they can be seen by the parts of the retina that are still

functioning, reading prism, closed circuit television or video magnification, text reading devices and smart phone apps.

- Vitamins -- to help reduce or delay the risk of severe vision loss. Some evidence suggests that certain vitamins and antioxidants -- vitamins C and E, beta-carotene, and zinc -- may help. However, more current research replaced beta-carotene with lutein and zeaxanthin with similar results.

Other ways to protect your eyes from AMD include the following

- Eating vegetables high in carotenoids, such as spinach and collard greens
- Wearing sunglasses with an ultraviolet filter when outdoors
- Not smoking
- Having regular visits to an eye care specialist to detect AMD and other eye diseases in the early stages

For retinal detachment: Some cases can be corrected with laser surgery, which has a high rate of success. If the peeling or tearing of the retina is more advanced, more extensive corrective surgery may be necessary.

Treating glaucoma: Usually includes using a series of eye drops over time to keep the disease under control. In many cases of glaucoma, fluid in the eye fails to drain properly, causing pressure to build up. Laser surgery for this form of the disease, called chronic open-angle glaucoma, has been available since 1979, but ophthalmologists generally use it as an alternative or addition to drug therapy, not as the first line of treatment.

Related: Try This Test to See if You Need Reading Glasses

There is no single, surefire remedy for glaucoma. Much remains to be learned about this disease. Glaucoma therapies evolve and change continuously. One long-term study, however, indicates that treating people with laser surgery first works just as well, if not better, to keep glaucoma in check. But medication in the form of eye drops is still the primary and most common treatment of COAG.

Because the disease can strike people at any age and there is no cure, treatment for glaucoma may need to continue for several decades. Although some researchers express concern that the laser's effects may last only three to five years before the eyes need to be treated again, drugs may become less effective over time as well.

While people just diagnosed with glaucoma may be reluctant to try surgery first, there are good reasons to consider it. Some patients with heart problems or asthma may not be able to take topical beta-blockers, a commonly used glaucoma drugs, and drug treatment can be expensive for some medications, so surgery may be cheaper in the long run.

Conclusions

A low level of awareness of common eye diseases among adults in Poland was observed. Most adults in Poland were not aware of risk factors for age-related eye diseases, such as glaucoma or AMD. Gender, age, and the presence of chronic diseases were the most important factors associated with higher awareness of common eye diseases and risk factors for glaucoma or AMD. A future educational campaign should include these gaps, and personalized communication on eye diseases should be implemented. Findings from this study indicate the need to strengthen eye health knowledge among adults in Poland.

References

1. Flaxman SR, Bourne RR, Resnikoff S, Ackland P, Braithwaite T, Cicinelli MV. Global causes of blindness and distance vision impairment 1990-2020: a systematic review and meta-analysis. The Lancet Global Health; c2017 .p. 5.
2. World Health Organization. World report on vision. Geneva: World Health Organization; c2019.
3. Voleti VB, Hubschman JP. Age-related eye disease. Maturitas. 2013;75:29-33.
4. Nita M, Grzybowski A. Smoking and eye pathologies: a systematic review. Part 1: Anterior eye segment pathologies. Current Pharmaceutical Design. 2017;23:629-638.
5. Robinson EL, Harris JD, Mahanan CM, Liiakas SJ. Value of routine eye examinations in asymptomatic patients. Optometry and Vision Science. 2016;93:660-666.
6. Kirang N, Temel AB. The effect of an eye health promotion program on the health protective behaviors of primary school students. Journal of Education and Health Promotion. 2018;7:37.
7. Ross and Wilson. Anatomy and physiology in health and illness. Anne Waugh, Allison Grant, page numbers 195-197.
8. Jayavera KN, Vrushabendra Swamy BM. Human anatomy, physiology, and health education. p183.
9. Thapa SS, Berg RV, Khanal S, Paudyal I, Pandey P, Maharajan N, *et al*. Prevalence of visual impairment, cataract surgery, and awareness of cataract and glaucoma in Bhaktapur. BMC Ophthalmology; c2011.
10. American Academy of Ophthalmology. Preferred practice pattern: primary open angle glaucoma. Limited revision. San Francisco, CA; c2003.
11. Chatterjee D. Human anatomy and physiology, volume 2. Medical College Allied Agency.
12. Charman N. Myopia: its prevalence, origins, and control. Ophthalmic Physiological Optics. 2011;31:3-6.
13. Zepta C, Zejmo D, Mojsa A. Prevalence of myopia and hyperopia in a population of public school children. Ophthalmic Physiological Optics; c2007 .p. 27.
14. Jayaveera KN, Vrushabendra Swamy BM. Human anatomy and physiology and health education: recommended for JNTU as a textbook. S Chand; page 183.
15. Cungdon N, Vingerling JR, Kelin BE, West Friedman DS, Kempen J, *et al*. Prevalence of cataract and pseudophakia/aphakia among adults in the United States. Archives of Ophthalmology. 2004;122:487-494.
16. Rahi JS, Cable N. Severe visual impairment and blindness in children in the UK. The Lancet. 2003;362:251-252.
17. Biousse V, *et al*. Diagnosis and clinical features of common optic neuropathies. Lancet Neurology. 2016. Grenier J, *et al*. WFS in optic neuropathies: mutation findings in non-syndromic optic atrophy and assessment of clinical severity. Ophthalmology. 2016;123:6-14.

18. Wallace DC, Singh G, Lott MT, Hodge JA, Scherer TG, Lezzam, *et al*. Mitochondrial mutation with Leber's hereditary optic neuropathy. Journal of Medical Genetics. 2003;40:115-121.
19. Bristow EA, Griffiths PG, Andrews RM, Johnson MA, Turnbull DM. The distribution of mitochondrial activity in relation to optic nerve structure. Archives of Ophthalmology. 2002;120:791-796.
20. Wudunn D. Genetic basis of glaucoma. Current Opinion in Ophthalmology. 2002;13:55-60.
21. Dietz HC, Ramirez F, Sakai LY. Marfan syndrome and other microfibrillar diseases. Advances in Human Genetics. 1994;22:153-186.
22. Loeys BL, Dietz HC, Braverman AC, Callewaert BL, De Backer J, Devereux RB. The revised Ghent nosology for Marfan syndrome. Journal of Medical Genetics. 2010;47:476-485.
23. Robinson PN, Arteaga-Soils E, Baldock C, Collod-Béroud G, Booms P, De Paepe A. The molecular genetics of Marfan syndrome and related disorders. Journal of Medical Genetics. 2006;43:769-787.
24. Gray JR, Bridges AB, Faed MJ, *et al*. Ascertainment and severity of MFS in a Scottish population. Journal of Medical Genetics. 1994;31:51-34.
25. Kinoshita N, Mimura J, Obayashi C, Katsukawa F, Onishi S, Yamazaki H. Aortic root dilation among young competitive athletes: Echocardiographic screening of 1929 athletes between 15 and 34 years of age. American Heart Journal. 2000;139:723-728.
26. Lams HD. Diagnosis and management of Marfan syndrome. Current Sports Medicine Reports. 2010;9:93-98.
27. Maranduca MA, Branisteanu D, Serban DN, Branisteanu DC, Stoleriu G, Manolache N. Synthesis and physiological implications of melanic pigments. Oncology Letters. 2019;17:4183-4187.
28. Singh AD, Topham A. Incidence of uveal melanoma in the United States 1973-1997. Ophthalmology. 2003;110:15:956-961.
29. Virgili G, Gatta G, Ciccolallo L, Capocaccia R, Biggeri A, Crocetti B. Incidence of uveal melanoma in Europe. Ophthalmology. 2007;114:2309-2315.
30. Spagnolo F, Caltabiano G, Queirolo P. Uveal melanoma. Cancer Treatment Reviews. 2012;38:549-553.
31. Nathan P, Cohen V, Coupland SE, *et al*. Uveal melanoma UK national guidelines. European Journal of Cancer; c2015.
32. Tanna P, Strauss RW, Fujinami K, Michaelides M. Stargardt disease: Clinical features, molecular genetics, animal models, and therapeutic options. British Journal of Ophthalmology. 2017;101:25-30.
33. Kniazeva M, Chiang MF, Margan B, Anduze A, Zack DJ, Han M. New locus for autosomal dominant Stargardt-like disease maps to chromosome U. American Journal of Human Genetics. 1999;64(5):1394-1399.
34. Burke TR, Tsang SH. Allelic and phenotypic heterogeneity in ABCA4 mutations. Ophthalmic Genetics. 2011;32:165-74.
35. Lois N, Holder GE, Bunce C, *et al*. Phenotypic subtypes of Stargardt macular dystrophy (Fundus flavimaculatus). Archives of Ophthalmology. 2001;119:359-69.
36. Sears AE, Bernstein PS, Cideciyan AV, *et al*. Towards treatment of Stargardt disease: Workshop organized and sponsored by the Foundation Fighting Blindness. Translational Vision Science & Technology. 2017;6:6.
37. Boughman JA, Conneally PM, Nance WE. Population genetic studies of retinitis pigmentosa. American Journal of Human Genetics. 1980;32:223-35.
38. Fahim AT, Daiger SP, Weleber RG. Non-syndromic retinitis pigmentosa: Overview. GeneReviews; c2017 Jan 19 [cited 2021 Dec 21].
39. Bhattacharya SS, Wright AF, Clayton JF, Price WH, Phillips CI. Close genetic linkage between X-linked retinitis pigmentosa and restriction fragment length polymorphism identified by rDNA probe LI 28. Nature. 1984;253:255.
40. Verbakel SK, Van Huet RAC, Boon CJF, den Hollander AI, Collin RWJ, Klaver CCW. Non-syndromic retinitis pigmentosa. Progress in Retinal and Eye Research. 2018;66:157-186.
41. Hamel C. Retinitis pigmentosa. Orphanet Journal of Rare Diseases. 2006;1:1.
42. Saunier S, Salomon R, Antignac C. Nephronophthisis. Current Opinion in Genetics & Development. 2005;15:324-331.
43. Janoria KG, Gunda S, Boddu SH, Mitra AK. Novel approaches to retinal drug delivery. Expert Opinion on Drug Delivery. 2007;4:371-88.
44. Chappelow AV, Schachat A. Retinal pharmacotherapy: neurovascular age-related macular degeneration. Elsevier Health Sciences; c2010 .p. 128-132.
45. Wong WL, Su X, Li X, *et al*. Global prevalence of age-related macular degeneration and disease burden projection for 2020-2040: A systematic review and meta-analysis. The Lancet Global Health. 2014;2:106-16.
46. Finger RP, Daien V, Eldem BM, *et al*. Anti-vascular endothelial growth factor in neovascular age-related macular degeneration: a systematic review of the impact of anti-VEGF on patient outcomes and healthcare systems. BMC Ophthalmology. 2020;20:294.