International Journal of Medicine and Pharmaceutical Research

Journal Home Page: www.pharmaresearchlibrary.com/ijmpr

Review Article **Open Access**

A Review on Glaucoma and Management

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A B S T R A C T

Glaucoma is an eye disease that gradually steals your vision. It is an optic neuropathy the second largest cause of blindness worldwide, many glaucoma patients continue to lose vision despite treatment to lower intraocular pressure. In addition to the loss of retinal ganglion cells in the eye, there is injury to major visual pathways of the brain along the retino-geniculo-cortical pathway. The intraocular pressure (IOP) level is the major risk factor, other issues such as the tolerance of retinal ganglion cells (RGCs) in the individual patient.

Keywords: Glaucoma, Intraocular Pressure, retinal ganglion cells.

A R T I C L E I N F O

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Article History: Received 12 August 2015, **Accepted** 15 September 2015, **Available Online** 10 October 2015

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Manuscript ID: IJMPR2726

PAPER-QR CODE

Citation: Sk. Meharunnisa, et al. A Review on Glaucoma and Management. *Int. J. Med. Pharm, Res*., 2015, 3(5): 1207-1211.

1. Introduction

Glaucoma, recognized as optic neuropathy is the second largest cause of blindness worldwide, Glaucoma refers to a group of conditions characterized by typical changes to the retinal nerve fiber layer and optic nerve head resulting in reduced visual field sensitivity. The “glaucoma” was derived from Greek word, “opacity of the crystalline lens”. It is a term describing a group of ocular disorder with multi factorial etiology united by a clinically characteristic intraocular pressure associated optic neuropathy. This can cause permanently damage vision in the affected eyes and lead to blindness if left untreated. It is normally associated with increased fluid pressure in the eye (aqueous humor).The term “ocular hypertension” is used for people with consistently raised intraocular pressure (IOP) without

any associated optic nerve damage. Glaucoma is characterized by the slow, progressive degeneration of retinal ganglion cells (RGCs) and optic nerve axons. This condition affects over 66 million people worldwide.

Definition: Glaucoma is an eye disease that gradually steals your vision. Often, glaucoma has no symptoms and can suddenly result in vision loss. Without proper treatment, glaucoma can lead to blindness. The good news is that with regular eye exams, early detection, and treatment, you can preserve your sight (6). Glaucoma can be roughly divided in two main categories, "open-angle" and "closed angle" (or" angle closure") glaucoma. The angle refers to the area between the iris and cornea, through which fluid must flow to escape via the trabecular meshwork. Closed angle glaucoma can appear suddenly and is often painful; visual loss can progress quickly, but the discomfort often leads patients to seek medical attention before permanent damage occurs. Open angle glaucoma, chronic glaucoma tends to progress at a slower rate and patients may not notice they have lost vision until the disease has progressed significantly. Glaucoma has been called the "silent thief of sight" because the loss of vision often occurs gradually over a long period of time, and symptoms only occur when the disease is quite advanced.

Different Types of Glaucoma: There are several types of glaucoma. The two main types are

Open-Angle Glaucoma:

Open-angle glaucoma, the most common form of glaucoma, accounting for at least 90% of all glaucoma cases. Is caused by the slow clogging of the drainage canals, resulting in increased eye pressure Has a wide and open angle between the iris and cornea Develops slowly and is a lifelong condition Has symptoms and damage that are not noticed. There are no early warning signs of open-angle glaucoma (also called primary or chronic glaucoma). It develops slowly and sometimes without noticeable sight loss for many years. Most people who have open-angle glaucoma feel fine and do not notice a change in their vision. That is why regular eye exams are so important. With early detection, open-angle glaucoma usually responds well to medication. However, it will be very important that you carefully follow your medication regimen to continually preserve healthy eye pressure and prevent vision loss. Open-angle is the most common type of glaucoma.

Angle-Closure Glaucoma:

Drainage Canals Drainage Canals Open Angle Lens Iris Fluid Flow Cornea 6 Angle-Closure Glaucoma The other main type of glaucoma is called angle-closure glaucoma. It is also called acute glaucoma or narrow-angle glaucoma. This type is caused by blocked drainage canals, resulting in a sudden rise in intraocular pressure Has a closed or narrow angle between the iris and cornea Develops very quickly Has symptoms and damage that are usually very noticeable Demands immediate medical attention. The closed angle prevents fluid from reaching the drainage canals. As a result, the intraocular pressure rises very quickly, causing a painful attack in the eye. The eye may appear red. You may have a headache, feel nauseous, feel intense eye pain, see rainbows around lights at night, or have blurred vision. If you have any of these symptoms, you should seek immediate medical attention to prevent serious eye damage. Treatment of angle-closure glaucoma usually involves either laser or conventional surgery to remove a small portion of the bunched-up outer edge of the iris. Surgery helps unblock the drainage canals so that the extra fluid can drain. If you have angleclosure glaucoma in one eye, doctors may go ahead and treat the other eye as a safety measure. In general, surgery for angleclosure glaucoma is successful and long lasting. Regular checkups are still important though, because a chronic form of glaucoma could still occur.

Other Types of Glaucoma

- Secondary Glaucoma.
- Exfoliative Glaucoma
- Pigmentary Glaucoma.
- Normal-tension Glaucoma.
- Congenital Glaucoma
- Neovascular Glaucoma,
- Uveitic Glaucoma.
- Traumatic Glaucoma.

2. Pathophysiology

The nerve fibres of the optic disc exit the eye posteriorly through a hole in the sclera that is occupied by a mesh-like structure called the lamina cribrosa, which is thought to maintain the pressure gradient between the inside of the eye and the surrounding tissues. The lamina cribrosa is more sensitive to pressure changes, which causes it to be displaced in high IOP, resulting in pinching of the optic nerve and blood vessels and possibly causing nerve damage. High IOP can also cause mitochondrial dysfunction in retinal ganglion cells in which energy demands are not met, leading to metabolic stress and nerve degeneration. Intra ocular pressure is determined by the balance between the production of aqueous humour and its drainage via the trabecular meshwork and uveoscleral outflow pathway. In OAG, there is a resistance to aqueous outflow via the trabecular meshwork, whereas in CAG the pathway to the drainage channel is obstructed, usually by the iris as seen in glaucoma. Aqueous humour is produced by the ciliary body and at an average rate of 2.5 microlitres per minute. It is secreted into the posterior chamber of the eye and passes through the pupil into the anterior chamber, and then to the trabecular meshwork in the anterior angle. The trabecular meshwork is composed of collagen and elastic tissue covered by trabecular cells that form a filter of decreasing pore size as the canal of Schlemm is approached [5]. Any condition that causes blockage of the trabecular meshwork (e.g. debris following cataract surgery) can cause secondary glaucoma [2]. The uveoscleral outflow pathway refers to drainage of aqueous humour from the anterior chamber to the anterior angle other than through the trabecular meshwork. Unlike the trabecular meshwork, the uveoscleral pathway is not a distinctive pathway with defined structures and channels, but a route whereby aqueous humour seeps around and between tissues. The uveoscleral outflow is thought to be driven by pressure gradients by movement of the ciliary muscle [6].

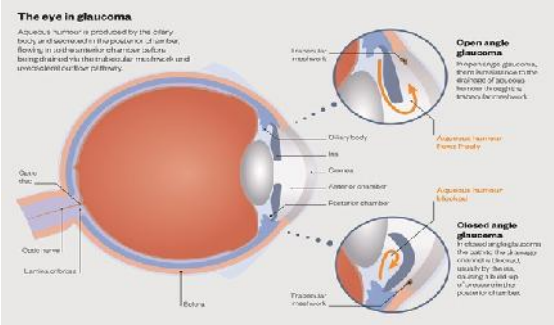

Figure 1: Pathophysiology of Glaucoma

Ocular hypertension is an elevated IOP above 21mmHg without disc or field changes, and is often a risk factor for glaucoma. The Ocular Hypertension Treatment Study (OHTS), which looked at patients with ocular hypertension, aimed to reduce IOP to less than 24mmHg or produce a decrease of IOP of at least 20% from baseline. After five years, 4.4% in the treatment group had developed glaucoma compared with 9% in the placebo group (50% relative risk reduction, hazard ratio 0.4, 95% confidence interval 0.27–0.59, $P<0.001$). After five years, the placebo group also started treatment to reduce IOP, and after 13 years, 22% of patients in the original placebo group had developed glaucoma compared with 16% in the original treatment group.

Glaucomatous optic nerve damage can occur in patients with IOP in the normal range. These patients may have an abnormally low cerebrospinal fluid pressure in the optic nerve subarachnoid space, resulting in a large pressure gradient across the lamina cribrosa. Conversely, there are a significant number of patients with an IOP above 21mmHg (ocular hypertension) who do not develop glaucoma despite 20 years of follow up. The National Institute for Health and Care Excellence (NICE) recommends that all patients with an IOP above 32mmHg are treated regardless of symptoms. Patients with a central cornea thickness (CCT) between 555–590µm should receive treatment if their IOP is more than 22mmHg, and patients with a CCT below 555µm are treated if their IOP is more than 21mmHg. Patients with type 2 diabetes are at increased risk of glaucoma as microvascular changes associated with diabetes are thought to lead to impaired microcirculation, which contributes to greater susceptibility of the optic nerve to damage. Patients with myopia are thought to have weaker scleral support at the optic nerve, which can also lead to nerve damage.

Primary open angle glaucoma

The most common form of glaucoma is primary OAG but it is unusual in patients aged under 50 years. It is a chronic progressive optic neuropathy, with a characteristic appearance of the optic nerve head and retinal nerve fibre layer in the absence of any other ocular disease or congenital abnormalities. The relationship between structural and functional loss is weak, especially in early glaucoma. In some patients structural changes are seen but no functional loss, and vice versa. However, in advanced glaucoma there is increased correlation. Disease progression depends on many factors, including age at time of diagnosis, difference between visual function at time of diagnosis and age-matched controls, and rate of progression (usually established in the first two years). Risk factors for OAG include having a first-degree relative with OAG, high myopia, and low CCT (one reason is that this may increase the risk of missed diagnosis, resulting in a delay to treatment). Other factors that lead to an increased risk are less clear cut and less certain, such as ocular perfusion pressure, diabetes and systemic hypertension (2).

Primary congenital glaucoma

It is a rare form of glaucoma that occurs in children. It usually presents from birth up to two years of age, although it can be diagnosed later. It is caused by incomplete development of the trabecular meshwork and has a strong genetic component. If diagnosis is delayed, extremely extended globes can occur. Treatment is surgical (2).

Secondary open angle glaucomas

Exfoliative (pseudoexfoliative) glaucoma is caused by abnormal fibrillo-granular proteins being produced in the ocular and extraocular tissues. This material, together with pigment granules from the iris, accumulates on the anterior lens capsule and the trabecular meshwork and inhibits the drainage of the aqueous humour. Exfoliative glaucoma usually occurs in patients aged over 60 years, has a strong genetic component and produces higher IOP than primary OAG. There is often significant damage by the time the disease is diagnosed and it progresses rapidly, even in patients treated with IOP-lowering medicines (2).

Corticosteroid-induced glaucoma

Corticosteroid-induced glaucoma results from changes in the extracellular matrix (glycoproteins) caused by corticosteroids, which lead to decreased outflow facilities in certain predisposed patients. The risk of increased IOP depends on the strength of the corticosteroid, dose frequency, duration of therapy and route of administration. Risk factors for developing raised IOP with corticosteroids include a family history of glaucoma, diabetes, myopia, and young or old age. Elevated IOP usually develops two to six weeks after initiating corticosteroid therapy, but can occur at any time (2). Obstruction of the trabecular meshwork can be caused by leaking lens material from a mature or hyper-mature cataract, traumatic lens injury or intraocular haemorrhaging. Uveitis can result in inflammatory cells, precipitates and secondary scarring, all of which can impede drainage of the aqueous humour.

Signs and Symptoms

- Hazy or blurred vision.
- The appearance of rainbow-colored circles around bright lights.
- Severe eye and head pain.
- Nausea or vomiting (accompanying severe eye pain)
- Sudden sight loss

3. Diagnosis

Early OAG can be challenging to diagnose as it is often symptomless until a large amount of neural damage has occurred, resulting in vision loss. A reduction in vision in

one eye may be missed due to compensation by the other eye (2). The main diagnostic tests are measurement of IOP, optic nerve assessment, and visual field measurement. NICE also recommends measurement of CCT (which can influence IOP results), peripheral anterior chamber configuration and depth assessment (which helps to establish if the patient has OAG or CAG). Examination of the optic nerve head and standard visual field testing are recommended to detect early disease. However, with the latter there needs to be a 30–50% retinal ganglion cell loss before changes can be detected [1, 2].

IOP:

It **is** measured using a Goldmann applanation tonometer mounted on the slit lamp biomicroscope. The cornea is anaesthetized, and the tonometer head is used to flatten the cornea by a fixed amount; the force required to achieve this level of flattening is measured. The measurements can be influenced by corneal thickness; a thin cornea produces an artificially low measurement, whereas a thick cornea can produce an artificially high result [2],[10].

Optic head examination:

Optic head examination looks at the appearance of the optic nerve head and the retinal nerve fibre layer. Glaucoma causes narrowing of the neuroretinal rim or localised notching, which leads to cupping of the optic disc. Glaucoma can be missed in patients with small optic discs because there is 'saucerisation' rather than cupping, whereas patients with large discs have a narrow neuroretinal rim normally, which may be recorded as a false positive.

Visual field testing

Visual field testing is important in the diagnosis of glaucoma but it is more important in monitoring disease progression and the efficacy of treatment. The preferred method is static automated perimetry, as it is less subjective, the results are numerical and tools for computer-based interpretation are available. If abnormal results are obtained, it is important to rule out other diseases or conditions that could influence these results (e.g. tumours, macular degeneration and diabetes). Ideally, all newly diagnosed glaucoma patients should have three standard automated perimetry tests per year during the first two years following diagnosis to identify eyes showing a fast rate of progression at an early stage. Rapidly progressing eyes need more aggressive treatment, a lower target IOP and more frequent monitoring (2).

Gonioscopy:

Gonioscopy is used to inspect the anterior chamber angle of the eye. Patients with narrow angles are at increased risk of acute angle closure which, if not treated promptly, leads to damage and development of glaucoma. OAG and CAG are treated completely differently so it is important to differentiate the conditions promptly. Optic disc haemorrhages are more common in glaucoma patients, but they are not unique and are often associated with disease progression (5).

4. Treatment

There are three types of treatment for glaucoma: medicine, laser surgery, and traditional surgery. None of these treatments will cure your glaucoma, but they can help stop the damage to your optic nerve.

Medicine is often the first treatment for glaucoma and works in one of two ways:

- Making your eyes produce less fluid.
- Increasing the outflow of fluid from inside your eyes.

Laser surgery works in one of two ways:

a. Helping your eyes drain fluid better.
b. Decreasing the amount of fluid that your eyes produce (this type of laser surgery is not discussed in this summary)

Traditional surgery works in one of two ways:

a. Redirecting fluid to bypass the part of the drainage system in your eyes that is not working properly.
b. Making new pathways for fluid to drain from your eyes.

Pharmacological Treatment:

The main aim of treatment is to lower the IOP and reduce inflammation so that laser iridotomy or surgical iridectomy (hole in iris) is possible. Corticosteroids (e.g. topical prednisolone 0.5% every five minutes for three applications, then four to six times a day) are used to reduce inflammation. Acetazolamide (orally or intravenously 10mg/kg), in addition to topical beta blockers and topical alpha$_2$-adrenoceptor agonists, are used to reduce fluid production in the eye. Pilocarpine 2% eye drops are used to constrict the pupil and open the anterior chamber angle. However, it is ineffective until a decrease in pressure has been achieved with other medicines. Once one eye has been treated, it is recommended that a prophylactic iridotomy is performed on the other, unaffected eye. Some patients who are considered at high risk of developing primary CAG are offered a prophylactic iridotomy to both eyes. Systemic osmotic diuretics (e.g. oral glycerol or intravenous mannitol) are used to withdraw fluid from the posterior chamber. Care needs to be taken in patients with kidney or heart disease as osmotic diuretics will initially increase blood volume.

Counseling a patient regarding usage of Eye drops:

Individual must Schedule medications around daily routines like waking and mealtimes. Remember that twice a day means every 12 hours, for example, 7 am and 7 pm. Keep your medications in a visible place at home and take them with you when you go out. Keep eye drops out of the reach of children and out of direct sunlight. Tell your doctor or pharmacist if your medications are causing reactions or if you are having any difficulty using them. It may be possible to change your medication program. Before using eye drops, wash your hands. Sit down and tilt your head back, or lie down and look at the ceiling.

Then follow these steps:

a. Make a pocket in your lower lid pulling down with your index finger.
b. Look up. Squeeze one drop into the pocket in your lower lid. Don't blink, wipe your eye, or touch the tip of the bottle to your eye or face.
c. Close your eye. Press the inside corner of the eye. (This stops the drop from draining into your throat.) Continue pressing for 2-3 minutes.

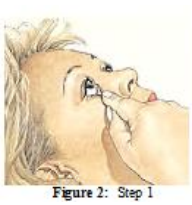
Figure 2: Step 1


Figure 3: Step 2

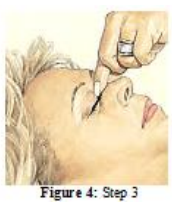
Figure 4: Step 3

Repeat steps 1 through 3 for each eye and each kind of drop you use. Wait 3 to 5 minutes between drops.

Non Pharmacological Treatment:

Types of Laser Surgery

Selective Laser Trabeculoplasty (SLT)

Selective Laser Trabeculoplasty (SLT) uses a laser that works at very low levels. Treats specific cells and leaves the mesh-like drainage canals surrounding the iris intact. For this reason, SLT may be safely repeated. May be an alternative for those who have been treated unsuccessfully with traditional laser surgery or with pressure lowering eye drops.

Argon Laser Trabeculoplasty (ALT)

Argon Laser Trabeculoplasty (ALT) used for primary open-angle glaucoma. Laser beam is aimed at the fluid drainage channels helping the drainage system work.

Micro pulse Laser Trabeculoplasty (MLT)

Micro pulse Laser Trabeculoplasty (MLT) provides the same pressure-lowering effects as SLT and ALT. Uses a specific diode laser to deliver laser energy in short micro bursts.

Laser Peripheral Iridotomy (LPI)

Laser Peripheral Iridotomy (LPI) used for angle-closure glaucoma where the iris in the eye blocks fluid drainage. Procedure makes an opening in the iris, helping the fluid to drain.

Laser Cyclo photocoagulation:

Used for those with very severe glaucoma damage that is not being managed by standard glaucoma surgery. Laser is used to treat the ciliary process to decrease the amount of fluid produced.

Microsurgery:

Microsurgery is also called conventional surgery because it is done in a hospital or surgery center and uses conventional but tiny instruments, along with a microscope. Microsurgery is successful with many types of glaucoma.

4. Conclusion

Glaucoma is a common eye disease that can cause irreversible blindness if left undiagnosed and untreated. This review concludes that, the glaucoma is a major disease affecting more no of people all over the world, Primary care physicians can play an important role in the diagnosis of glaucoma by referring patients with positive family history or with suspicious optic nerve head findings for complete ophthalmologic examination. Glaucoma is a common eye disease that is usually associated with an elevated intraocular pressure. Treatment options for patients with glaucoma include medications, laser therapy, and incisional surgery. The risks and benefits of each type of treatment must be carefully considered to maximize the treatment's benefits while minimizing adverse effects. The medication should be given to the patients through councelling. Glaucoma affects more than 70 million people worldwide with approximately 10% being bilaterally blind, making it the leading cause of irreversible blindness in the world.

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