GLAUCOMA

Dr. Sajid A l Assady

Meaning: ***hidden thief of vision.***

Definition: ***Glaucoma is a group of eye diseases characterized by damage to the optic nerve usually due to excessively high intraocular pressure (IOP).This increased pressure within the eye, if untreated can lead to optic nerve damage resulting in progressive, permanent vision loss.***

Aqueous secretion

Aqueous humour is produced in two steps:

1. **Active secretion** by the non-pigmented ciliary epithelium accounts for the vast majority that depends on several enzyme systems. (Carbonic anhydrate enzyme)
2. **Passive secretion** by ultrafiltration and diffusion, which are dependent on the capillary hydrostatic pressure, oncotic pressure, is thought to play a minor role.

Aqueous outflow

Anatomy

1. **The trabecular meshwork:** (trabeculum) is a sieve-like structure at the angle of the anterior chamber, through which 90% of the aqueous humour leaves the eye.

2. **Schlemm canal:** is a circumferential channel in the perilimbal sclera.

Physiology

Aqueous flows from the posterior chamber via the pupil into the anterior chamber, from where it exits the eye by two different routes:

1. **Trabecular** (conventional) route accounts for approximately 90% of aqueous outflow. The aqueous flows through the trabeculum into the Schlemm canal and is then drained by the episcleral veins. Trabecular outflow can be increased by drugs (miotics, sympathomimetics), laser trabeculoplasty and filtration surgery.

2. **Uveoscleral** (unconventional) route accounts for the remaining 10% in which aqueous passes across the face of the ciliary body into the suprachoroidal space and is drained by the venous circulation in the ciliary body, choroid and sclera. Uveoscleral outflow is decreased by miotics and increased by atropine, sympathomimetics and prostaglandin analogues.

Intraocular pressure

The IOP is determined by the balance between the rate of aqueous secretion and aqueous outflow. The rate of aqueous outflow is proportional to the difference between the intraocular and episcleral venous pressure.

Concept of normal intraocular pressure

The distribution of IOP within the general population has a range of 11–21 mmHg. Although there is no absolute pathological point, 21 mmHg is considered the upper limit of normal and levels above this are viewed with suspicion.

Fluctuation

Normal IOP varies with the time of day, heartbeat, blood pressure level and respiration. The diurnal pattern varies, with a tendency to be higher in the morning and lower in the afternoon and evening. Normal eyes manifest a mean diurnal pressure variation of 5 mmHg.

Classification of Glaucoma:

Glaucoma can be congenital (developmental) or acquired, further sub-classification in to open angle and angle closure types, it's also may be primary or secondary according to the absence or presence of associated cause for elevation of IOP.

Secondary open angle glaucoma: it can be sub divided on basis of aqueous out flow obstruction site in to:

1. Pretrabecular: in which aqueous outflow is obstructed by membrane cover the Trabeculum as in:

Neovascular glaucoma

Iridocorneal –endothelial syndrome

Epithelial ingrowth

2. Trabecular: in which the obstruction occur as a result of clogging up the trabeculum as in:

Pseudoexfolation glaucoma

Pigmentary glaucoma

Red cell glaucoma (RBC in the trabeculum)

Ghost glaucoma (degenerated RBC)

Phacolytic glaucoma (Macrophages and lens proteins in the trabeculum).

Scar in the Trabeculum (herpes zoster)

3. Post trabecular: in which the trabeculum is normal but the obstruction occur beyond it as in:

Carotid-Cavernous fistula.

Sturge Wiber Syndrome

Superior vena cava obstruction

Secondary angle-closure glaucoma: it occur due to impairment of aqueous outflow by apposition between the peripheral iris and the trabeculum by either anterior or posterior forces.

Primary open angle glaucoma (POAG)

Prevalence

POAG is the most prevalent of all glaucoma, affecting approximately 1 in 200 of the general populations over the age of 40 years .it's affected both sexes equally and more common in black than in white individuals.

Inheritance:

POAG is frequently inherited probably in multifactorial manner. First-degree relatives of patient with POAG are at increased risk of developing it. In general the approximated risk is 10% in siblings and 4% in offspring.

Risk factors:

Major risk factors for developing POAG are:

1. Elevated intraocular pressure : persistent elevated IOP is the most significant risk factor for development of glaucoma, the higher the level of IOP, the greater the risk of developing the disease normal IOP (11-21mmhg).

2. Race: the black compared to white, POAG is up to 5 times more prevalent and develops at an earlier age, and is more sever in black; blindness caused by POAG is up to 8 times more in black.

3. Family history of POAG: a family history of glaucoma particularly first degree relatives is an important risk factor for the development of glaucoma.

4. Increased age: the prevalence of glaucoma is increased with age. White person of age 80 years have up to 10% prevalence rate, white person of age 40 have 1-2%prevelance rate.

Person with any one or combination of these risk factors should be checked regularly for the development of glaucoma.

Minor risk factors: There is evidence for an association between POAG and:

- myopia
- diabetes
- hypertension
- migraine

Clinical features:

POAG is a chronic, slowly progressive, usually bilateral diseases with an insidious onset.

It's generally asymptomatic until it has caused significant loss of visual field, although some patients may become aware of early defects by chance, occasionally patients with very high IOPs may complain of ocular pain, headache and even haloes caused by transient corneal epithelial edema.

Signs:

- Glaucomatous optic disc changes (cupping , parapapillary changes, nerve fiber layer atrophy)
- Raise IOP.
- Characteristic glaucomatous visual field defects.

Management:

The goal of glaucoma therapy is to preserve vision by reducing intraocular pressure to a level thought to be safe for the optic nerve, while preserving patient quality of life. This can involve any or all of:

- 1.medical therapy
- 2.laser therapy
- 3.surgical therapy

Medical therapy:

Medications in the form of eye drops. In sever major pharmacological classes, are used to lower IOP. They act either by decreasing aqueous production in the ciliary epithelium, or to improve aqueous outflow via the trabecular meshwork or the uveoscleral route or combination of the above.

The most commonly used drugs are listed below:

1. β-adrenergic blockers:

Mechanism: decrease aqueous flow production

Examples: Timilol, levobunolol, Betaxalol (β1-selective receptors).

Major side effects cardiac failure, heart block and bronchospasm.

2. Cholinergic agonists:

Mechanism: improve trabecular outflow

Examples: Pilocarpine, Carbachol

Major side effect: miosis, browache and decrease vision (especially with cataract).

3. Carbonic anhydrase inhibitors:

Mechanism: decrease aqueous production

Example: Acetazolamide (oral), Dorzalomide (topical).

Major side effects: GI upset, malaise, renal stones ad aplastic anaemia (oral).

4. Alpha agonists:

Non selective//

Mechanism: decrease aqueous fluid production, increase uveoscleral out flow

Example: Epinephrine, Dipiverin (pro-drug)

Major side effect: pupil dilatation, macular edema, tachycardia

Selective//

Mechanism: decrease aqueous fluid production, increase uveoscleral outflow

Example: Apraclonidine, Brimonidine

Major side effect: contact dermatitis, hypotension in children<12 years.

Prostaglandin analogue:

Mechanism: improve uveoscleral outflow

Example: latanoprost

Major side effect: iris color changes, lash growth and trachiasis

Hyperosmotic for emergency use:

Mechanism: establishes a concentration gradient and draw fluid from the eye.

Example: Mannitol (IV), Glycerin (oral).

Major side effect: cardiovascular overload, renal insufficiency, stroke.

Laser therapy:

1. Argon laser trabeculoplasty (ALT): is considered when medical therapies fail to control IOP adequately. The laser beam is directed at the trabecular meshwork and the healing of the laser burn improve aqueous drainage through a mechanism that is still not well explained.

2. Laser cyclophotocoagulation: is considered for glaucoma that is resistant to standard medical and surgical treatment. Thermal energy applied to the ciliary body which causes destruction of the tissue, reducing aqueous production.

Surgical therapy:

Trabeculectomy is a surgical technique that establishes an escape route for the aqueous fluid to leave the eye while maintaining a system closed to the external environment.