3) Glaucoma – Dr. Tara

Introduction

- Glaucoma describes a number of ocular conditions characterized by:
  - Raised intraocular pressure (IOP).
  - Optic nerve head damage.
  - Corresponding loss of visual field (VF).
- IOP depends on the relationship between aqueous production and outflow.
- The normal ocular tension is between 10-21mm.Hg. There is a normal fluctuation in ocular tension of up to 3-5mm.Hg during the course of the day called diurnal variation.
- Glaucoma remains one of the principal causes of blindness throughout the world

Anatomy of the Drainage Angle

- *The anterior chamber* of the eye is that space, containing aqueous humour, which is bounded in front by the cornea and part of the sclera, and behind by the iris and part of the ciliary body. Its normal depth in adults varies from 2.5-3.5mm.
- *The angle of the anterior chamber* refers to that peripheral recess bounded posteriorly by the root of the iris and the ciliary body and anteriorly by the corneo-scleral junction or the limbus. Among the deeper lamellae of the limbus, there is an annular channel, called the canal of Schlemm. The canal is separated from the aqueous in the anterior chamber by the trabecular meshwork.
- *The trabecular meshwork* is made up of circumferentially disposed flattened collagenous bands which criss-cross, leaving numerous tortuous passages through which the aqueous humour drains from the anterior chamber to the canal of Schlemm.
- *The aqueous humour* is a transparent colorless fluid which fills the anterior and posterior chambers of the eye. Its chief site of formation is the processes of the ciliary body. The volume of aqueous in the anterior chamber of the human eye is 0.25 ml.

Classification of Glaucoma

G. is classified according to three principal parameters:

- **Angle**: Open, Narrow/Closed
- **Onset**: Acute, Chronic
- **Aetiology**: Primary, Secondary

Ocular Hypertension

- Ocular hypertension, is a risk factor for the development of glaucoma. Yet many individuals manifest this risk factor and never develop glaucoma. The prevalence of ocular hypertension approximately 3 to 8% of population exhibits intraocular pressures consistently above 22mm Hg, w/o concurrent optic nerve damage.

Primary Open-Angle Glaucoma

- Primary open-angle glaucoma (POAG) is characterized by the development of glaucomatous optic neuropathy in an eye with a normal-appearing anterior chamber angle and absence of other ocular or systemic disorders which may account for the optic nerve damage. Most cases of primary open-angle glaucoma are associated with statistically elevated intraocular pressure. Primary open-angle glaucoma is also called chronic open-angle glaucoma or simple open-angle glaucoma. POAG is the most common form of glaucoma. It is typically a bilateral disease but may be asymmetric.
Risk factors: age (>40 y): positive family history; diabetes; myopia.

Aetiology: Unknown. Theories suggest: functional inadequacy of TM drainage; hypoperfusion of optic N. head; and weakness of structural collagen in the angle and disc.

Symptoms: none until late, when considerable field loss has already occurred; not associated with pain, discomfort or redness; new cases are usually identified by screening.

Signs:

- **IOP:** in ocular hypertension IOP is elevated without disc or field changes. A proportion of eyes with ocular hypertension ultimately develop glaucoma.
- **VF:** up to 50% of ganglion cell axons entering the disc may be lost before disc and field changes are evident. Peripheral field is progressively lost, but central acuity is affected late.

The development of glaucomatous optic neuropathy in patients with statistically normal intraocular pressure results in a paradox if glaucoma is defined by elevation of intraocular pressure. Glaucomatous optic neuropathy occurring in an eye without intraocular pressure elevations is referred to as "low tension glaucoma." Many believe that the optic nerves of such individuals are more susceptible to pressure damage, even within the normal range. Therefore, the original definition of glaucoma is modified to accommodate the disease low tension glaucoma.

Pathophysiology

- Even today, much remains unknown about this disease.
- Elevated IOP almost certainly plays a significant role, but the process is poorly understood.
- According to the mechanical theory of POAG, chronically elevated IOP distorts the lamina cribrosa, crimping the axons of retinal ganglion cells as they pass through the lamina cribrosa and eventually killing the cells.
- The vascular theory suggests that with elevated IOP, reduced blood flow to the optic nerve starves the cells of oxygen and nutrients.

Clinical Testing & Examination Techniques in Glaucoma

**Tonometry**

- Indentation Tonometry: The Schiotz tonometer is the primary indentation tonometer used to measure intraocular pressure.
- Applanation Tonometry: Applanation tonometry uses a variable amount of force to produce a fixed amount of flattening of the corneal surface.

**Gonioscopy**

- Is an examination technique used to visualize the structures of the anterior chamber angle. Mastering the various techniques of gonioscopy is crucial in the evaluation of the pathophysiology of aqueous humor outflow obstruction and the diagnosis of the various glaucomas.

**Fundoscopy**

- Disc damage usually begins as an upper or lower temporal notch, giving rise to a nasal arcuate scotoma.
Management

- Disc cupping and field loss in POAG progress at a variable rate, leading in the most severe cases to profound field constriction and ultimately blindness. The aim of management is to lower IOP sufficiently to arrest progressive VF loss.
- **Medical treatment:** topical beta-blocker (timolol, carteolol,betaxolol) and pilocarpine, carbonic anhydrase inhibitor, prostaglandin analogue, alpha 1&2 agonist.
- **Surgical treatment:** Trabeculectomy provides a definitive and permanent reduction of IOP to within safe limits in the majority of cases.

Secondary Open-Angle Glaucoma

- Secondary open-angle glaucomas are characterized by glaucomatous optic neuropathy in eyes with elevated intraocular pressure, developmentally normal and open (not obstructed by the peripheral iris) anterior chamber angles, and the absence of systemic disorders which may account for the optic nerve damage. In secondary open-angle glaucoma, the aqueous outflow obstruction is the result of exogenous or intraocular factors, and not a primary abnormality of the trabecular meshwork, as is found in primary open-angle glaucoma.

Angle-Closure Glaucoma

**Primary Angle-Closure Glaucoma with Pupillary Block**

- Primary angle-closure glaucoma refers to an increase in intraocular pressure secondary to iris apposition to the trabecular meshwork. This apposition prevents aqueous humor outflow from the anterior chamber and may occur suddenly (acute angle-closure glaucoma),
- Slowly over time (chronic angle-closure glaucoma), or intermittently (subacute angle-closure glaucoma). Primary angle-closure glaucoma derives from relative pupillary block. Relative pupillary block is an increased resistance to aqueous humor flow from the posterior chamber into the anterior chamber, through the pupil. Relative pupillary block is dependent on the contact between the lens and iris and is increased in eyes with narrow anterior segments, such as hyperopic eyes.
- Eyes with short axial lengths
- Small corneal diameters
- Increased thickness of the crystalline lens are predisposed to increased pupillary block and angle-closure glaucoma.
- Relative pupillary block also increases with age as the lens thickens and the pupil becomes more miotic. Maximal pupillary block occurs in the middilated position; therefore, angle-closure may be exacerbated by pupillary dilation. In the middilated eye, the apposition between the lens and iris is substantial, and the peripheral iris is lax enough to be anteriorly displaced.
- Eyes in which the pupil has been pharmacologically dilated are most prone to increased pupillary block and acute angle-closure glaucoma as the medications begin to wear off and the pupil is in the mid dilated position. In the normal eye, there is apposition between the posterior surface of the iris and the anterior surface of the lens, but the resistance to aqueous humor flow into the anterior chamber is minimal.
Clinical features

- Visual reduction: due to corneal oedema and posterior segment ischaemia.
- Raised IOP: usually 40-70 mmHg.
- Coloured haloes around lights, due to diffraction of light through the oedematous corneal epithelium.
- Pain: may be severe, and associated with vomiting.
- Ciliary injection.
- Mid-dilated pupil, due to ischaemic iris atrophy.
- Corneal oedema: due to decoplensation of the corneal endothelial pump.
- Anterior chamber cell and flare due to anterior uveitis.

Treatment

- The IOP must be reduced medically as quickly as possible, using:
  - Diuretic: Acetazolamide to reduce aqueous production: 500 mg i.v immediately, 250-500 mg orally, t.d.s, if the pressure remain high, give an i.v mannitol infusion (200 ml 20% i.v over 1-2 hr.).
  - Miotic: Intensive pilocarpine therapy (every 10 min for 1 hr, then hourly for 6hr). Then maintain on pilocarpin 2% q.d.s.
  - Steroid: Topical steroid q.d.s during acute phase to reduce the accompanying inflammatory response.
  - Analgesia: Parenteral analgesia and antiemetic may be needed.
  - Surgical treatment: Peripheral iridectomy or iridotomy (YAG laser).

Examination of the fellow eye with gonioscopy should be done in all patients presenting with angle-closure glaucoma. Patients with a marked difference in the depth of the anterior chamber and the configuration of the anterior chamber angle between the two eyes should be suspected of having a secondary cause of angle-closure glaucoma.

Lens-Associated Glaucoma

1. Even in the healthy eye, cataractous change and enlargement of the lens (intumescent cataract), with increase in irido-lenticular touch and relative pupillary block, may result in angle-closure glaucoma (phacomorphic glaucoma).
2. Dislocated or subluxed lenses also may migrate forward to become entrapped in the pupil, leading to angle-closure glaucoma.
3. A spectrum of glaucomas associated with a permeable or "leaky" lens capsule have been described: These include (phacolytic glaucoma), which is a lens-induced uveitis with secondary glaucoma, and obstruction of the trabecular meshwork by lens protein or particles of lens material. Phacoanaphylactic endophthalmitis

Congenital, Infantile, Juvenile & Developmental Glaucoma

- Primary congenital glaucoma occurs at birth or within the first several months of life. It is characterized by severe elevations of intraocular pressure, which when left untreated will cause stretching of the various support tissues of the eye and enlargement of the globe (buphthalmos).
- The classic presentation of a child with primary congenital or infantile glaucoma is the triad of excessive epiphora, photophobia, and blepharospasm.