

## Introduction

- Glaucoma is an optic neuropathy associated with characteristic damage to the optic nerve head (cupping) and the visual field (nerve fibre bundle defects).
- It is a blinding disease where first the peripheral visual field becomes constricted, followed by loss of central visual acuity
- Glaucoma, if defined with either field or nerve criteria, has a prevalence of 5.6\%
- If defined with both field and nerve criteria, it has a prevalence of 2.4\%
- The appearance of the optic nerve head and visual fields are the major factors for a diagnosis of glaucoma


## Risk Factors

- Age
- Ethnicity Afirian-Americans
- Family History
- Intraocular pressure (IOP) is the most important risk factor $\begin{gathered}\text { Increased IOP + optic neuropathy = glaucoma } \\ \text { Increased IOP - optic neuropathy }=\text { Ocular HTN }\end{gathered}$
** In normal-tension glaucoma the optic nerve is vulnerable to lower IOP
- Trauma
- Eye surgery
- Drugs
- Refractive errors


## Non-IOP dependent risk factors

- Systemic Vascular Dysregulation
- Raynaud's, Prinzmetal Angina, Migraine
- Nocturnal Hypotension
- Sleep Apnea


## Intraocular Pressure IOP

What's considered necessary is the optic neuropathy

- An elevated eye pressure is neither necessary nor sufficient to make the diagnosis:
- in "normal tension glaucoma", the patient is never found to have a pressure over the normal limits
- in "ocular hypertension" the patient has high eye pressures but no signs of optic nerve or visual field damage
- The level of the intraocular pressure is the main risk factor, and is important in the monitoring of treatment
- Gonioscopy is of major importance in the classification of the glaucoma type

Gonioscopy is an eye test that checks for signs of glaucoma. It uses a special lens and slit lamp to evaluate
your eye's
drainage angle
(anterior
chamber angle).

- The extent of damage to the optic nerve and visual field determines the stage of the glaucoma
(Image of Iridocorneal Angle)




## Basic Principles-IOP

- Intraocular pressure (IOP) represents the equilibrium between the rigidity of the cornea and sclera, and the outward pressure of the ocular contents
- As the vitreous is of fixed volume, the most important variable is the amount of aqueous humour, which varies with respect to production and drainage.
- The pathology of elevated intraocular pressure is due to inadequacies of aqueous outflow rather than production
- The normal mean IOP is 15.5 mm Hg
- Range is $10-21 \mathrm{~mm} \mathrm{Hg}$
- Diurnal variations exist
- Many ways to measure it but standard is Goldmann Applanation Tonometry -GAT



## BIPRISM PROBE

> The two beam-splitting prism within the applanating unit optically convert the circular area of corneal contact into 2 semicircles

Side View Of Probe And Probe Carrier
Degrees Are From Zero To 180

$>$ The fluorescent semicircles are viewed through the biprism and the force against the cornea is adjusted until the inner edges overlap.


Applanation tonometry rings viewed through the Goldmann prism


## Basic Principles- Aqueous Humor

- The volume of the aqueous humour in the anterior segment is 0.25 cc or $250 \mu \mathrm{~L}$
- One quarter of this is in the posterior chamber and three quarters in the anterior chamber
- The ciliary body produces $2.5 \mu \mathrm{~L}$ per minute, with complete turnover of the aqueous in about 100 minutes


## Aqueous Humor

- It is produced by the non pigmented epithelium of the ciliary processes
- Produced by :
- Ultrafiltration
- Active secretion


## Aqueous Humor Pathway

- Aqueous humor passes from the posterior chamber between the iris and the lens through the pupil into the anterior chamber

AQUEOUS HUMOUR DYNAMICS


## Aqueous Humor Pathway

- It drains through 2 pathways:
- Conventional (80-90\%): trabecular meshwork, Canal of Schlemm, aqueous veins and episcleral veins impoved through $M$ agoniss
- Uveoscleral ( $10-20 \%$ ): Face of the ciliary body and iris to the supraciliary/suprachoroidal space.
Improved through prostaglandins' analogues (A contraindication to keep in mind : pregnancy)




## Classification

- Glaucoma is not a single disease, but a large number of similar conditions with factors in common.
- It is usually classified on the basis of the anatomy of the anterior chamber angle as open or closed, and each type has primary and secondary sub-categories.


## Glaucoma Classification



## Open Angle Glaucoma

- Primary Open Angle Glaucoma POAG:
- Idiopathic increase in outflow resistance
- Secondary Open Angle Glaucoma SOAG:
- Clogging of trabecular meshwork TM
- Increased episcleral venous pressure EVP
- Scarring of TM
- Increased TM resistance due to medications (steroids)


## POAG

- Most prevalent type
- Female = male
- More common in myopes

Structural vulnerability (increased risk of elevated outflow resistance)

- Asymptomatic till late in the disease
- IOP 20-40 mmHg


## SOAG

- Clogging:
- RBCs: Hyphema
- WBCs: Uveitis
- Pigment: pigment dispersion syndrome, melanoma
- Proteins: Pseudoexfoliation syndrome lens proteins


Gonioscopy


## ADDITIONAL I Pseudoexfoliation Glaucoma

** Pseudoexfoliation syndrome is a systemic disease with primarily ocular manifestations characterized by deposition of whitish-gray protein on the lens, iris, ciliary epithelium, corneal endothelium and trabecular meshwork. Although attempts at identifying the material have been unsuccessful, pathologic study has revealed that the lens epithelium, trabecular meshwork, iris, ciliary processes, conjunctiva and periocular tissue are its source. The material is insoluble and floats in the aqueous humor, where it is filtered and deposited in the trabecular meshwork. Meanwhile local production of the proteinaceous material by the trabecular endothelial cells continues. All of this accumulates in the trabecular spaces and focally collapses Schlemm's canal. This decreases aqueous humor outflow and increases IOP.
** It is important to note that not every individual with pseudoexfoliation syndrome will develop pseudoexfoliation glaucoma.
** Pseudoexfoliation glaucoma commonly presents unilaterally with IOP that tends to escalate faster than among patients with primary open-angle glaucoma (POAG). The higher IOP observed in pseudoexfoliation glaucoma can lead to more rapid optic nerve damage and visual field loss. When symptoms are present in one eye, the contralateral eye must be examined carefully and monitored, since pseudoexfoliation glaucoma will develop in the other eye of more than 40 percent of these patients.

## SOAG

- Increased EVEDPal
- Carotid cavernous fistula Episcleral hemangioma
- Sturg Weber Syndrome - SVC obstruction
- Scarring:
- Angle recession (trauma)

Angle recession is a common manifestation of blunt ocular trauma and involves rupture of the ciliary body face, resulting in a tear between the longitudinal and circular fibers of the ciliary muscle.



## Closed Angle Glaucoma

- Anatomic features predisposing to angle closure: shallow anterior chamber (e.g., hyperopia, short eye)
- Advanced age (>60 years).
- Female gender
- Inuit and Asian ethnicity
- Eye injury with scarring and adhesions
- Rubeosis iridis
- Drugs: Sulfonamides, TCA, MAOi, antihistamines
- Mydriasis
I. Drug-induced: mydriatics
II. Darkness
III. Stress/fear response


## Pathophysiology

- Blocked trabecular meshwork $\rightarrow$ decreased drainage of aqueous humor from the eye $\rightarrow$ sudden $\uparrow$ in IOP
A. Primary : the chamber angle is narrowed due to the peripheral iris obstructing the TM.
B. Secondary :

Peripheral anterior synechiae (PAS) refers to a condition in which the iris adheres to the angle.
A. Scarring: PAS or PS
B. Lens luxation/ large cataracts
C. Rubeosis iridis (neovascular glaucoma)

## Open vs Closed Angle



## Acute Angle Closure with pupillary Block

Associations: uveitis
Treatment: remove the block ( laser iridotomy )

## Pupillary Block



## Chronic Angle Closure-NVG/ Uveitis



## Clinical Presentation of Acute Angle Closure Attack

- Sudden onset of symptoms
- Severely painful eye (hard on palpation), redness
- Photophobia and excessive tearing Frontal headache
- Headache, nausea and vomiting
- Blurred vision and halos seen around lights
- Complications: irreversible damage of the optic nerve


## Diagnosis- Physical Examination

- Decreased visual acuity
- Non reactive, fixed oval pupil
- Cloudy edematous cornea
- Shallow anterior chamber
- Closed angle on gonioscopy
- IOP > 40 mm Hg


## Injected, Cloudy cornea, oval pupil

When the pressure rises quickly, the cornea becomes waterlogged ( corneal edema )


## Shallow A/C



## Management outline

- Lower IOP:
- Systemically : IV Acetazolamide/ Mannitol
oral Acetazolamide
- Topical Eye drops: B blockers, $\alpha$ agonists, Carbonic anhydrase inhibitors, pilocarpine
- Break the angle closure cycle:
- YAG laser Iridotomy/ Surgical iridectomy
- Examine second eye and treat prophylactically


## YAG Laser Iridotomy




## Glaucoma Diagnosis: Tips and Tools

- History
- Physical Examination
- Special Tests


## Tip One

- Every patient has glaucoma until proven otherwise


## What are glaucoma risk factors?

- History
-Family history -Refractive Error
-Race
-Age


## Ocular History

- Trauma
- Laser
- Surgery
- Other



## Medical History

- Diabetes
- Hypertension
- Asthma
- Other
- Known drug allergies/reaction


## Vision

Best spectacle-corrected visual acuity refers to the measurement of the best vision correction that can be achieved using glasses or contact lenses.

- BSCVA
- State refraction myope vs. hyperope
- RAPD



## What is an RAPD?

## No Light

Normal<br>Response to Light



Positive
RAPD of Right Eye


- Elicited during a swinging flashlight test
- Dilation of both pupils when the light is swung from the normal eye to affected eye


## Tip Two

- Intraocular pressure is neither necessary nor sufficient for the diagnosis of glaucoma
- Intraocular pressure is, however, the most important risk factor


## IOP

- The higher the pressure the higher the risk
- Goldmann technique preferred
- Tonopen, etc. if necessary



## What else is important?

- Slit lamp examination
-Classify type based on angle structures
- Look for signs of secondary glaucoma
- Optic nerve head examination
-Stage disease based on ONH damage
- Visual field examination
- Stage disease based on VF damage


## Slit lamp examination

- Lids/lacrimal
- Conjunctiva/
- sclera
- Cornea
- Lens

NEXT SLIDE

- Other
- PXE
- PDG
- NVI
- Iritis
- PI



## ADDITIONAL

(1) Pseudoxanthoma elasticum (PXE) is a rare genetic disease characterized by elastorrhexia, or progressive calcification and fragmentation, of elastic fibers primarily affecting the skin, retina, and the cardiovascular system.
(2) Pigment dispersion syndrome (PDS) is a common type of open-angle glaucoma which is relatively underdiagnosed. It is characterized by the spontaneous dispersion of pigment granules from the pigment epithelium of the iris, which gets deposited in the anterior segment.
(3) Neovascularization of the iris (NVI), also referred to as rubeosis iridis. ( explained earlier )

## Gonioscopy

- Always performed on any patient where glaucoma is a possibility
- Classify into open vs. narrow vs. closed angle

4. Schwalbe Line



## Gonioscopy- Look for secondaries

Sampaolesi line in pseudoexfoliation glaucoma


## Tool One

- Four mirror lens excellent for compression gonioscopy
- This differentiates between appositional and synechial closure
- Three mirror lens also fine



## Gonioscopy lenses/mirrors



## Tool Two

- Corneal thickness is becoming more and more important in glaucoma diagnosis
- Pachymetry is not, however, currently a part of the standard of care
- Prior LASIK will result in very thin central cornea

- Thin cornea can give a falsely low IOP reading
- Thick cornea can give falsely high IOP


## Optic Nerve \& Retina

- State C:D ratio
- Note other findings:
-Thin rim
- Notch
-Drance hemorrhage
- Peri papillary atrophy, $\alpha$ or $\beta$
- State relevant retinal findings:
-AMD, etc


## Vertical Cup to Disc Ratio

How to differentiate between physiological cupping \& glaucoma cupping?

1. Visual field ( most important )
2. IOP



Variants of Normal


A


## Challenging Nerves

## Optic Nerve Head Examination

- Look for: -Cupping
-Asymmetry
-Notching
-Hemorrhages
-ISNT rule
The ISNT rule is defined as the order of the neuroretinal rim width or RNFL
thickness that follows the pattern of $\mathrm{I}>\mathrm{S}>\mathrm{N}>\mathrm{T}$ ( broadest in the inferior
rim, followed by the superior and nasal rims, and thinnest in the temporal disc
region )

In severely advanced glaucoma with complete loss of retinal tissue, retinal vessels may disappear as they make a sharp turn into the cup, termed bayoneting or "bean-pot" cupping.


## Tool Three

- Stereoscopic viewing at the slit lamp with a 66D or 78D lens and a dilated pupil


## Tool Four Optic Nerve Head Imaging

- Computer aided imaging of optic nerve and/or nerve fibre layer
- Confocal scanning laser ophthalmoscopy / HRT
- Optical coherence tomography/OCT
- Polarimetry / GDx
- These are all commercially available - and costly!



## Tool Five Visual Field Examination

- Assess functional damage prior to patients perception of field loss
- Assess patient's performance in relation to age matched normal database (Statpack, SITA, Octopus)


Visual field within normal limits


Visual field outside normal limits

Test results from a normal visual field without vision loss (left) and a visual field with vision loss from glaucoma (right). Darker gray and black areas represent loss of vision in a visual field. The optic disc appears black in both fields since there is no vision there; this is normal.


## Tip Three

- One standard text book of glaucoma lists over 100 forms of glaucoma
Organize management plan by
deciding which subtype of glaucoma the patient has


## Treatment

- Optic nerve damage in glaucoma is irreversible
- Treatment is aimed at maintaining the residual optic nerve function
- Most modifiable risk factor is IOP
- Lowering IOP Increases the chances of slowing down or stopping nerve damage.


## Treatment Modalities

- Medical
- Laser
- Surgical


## Medical Treatment

- Prostaglandin analogues (PGAs):
- Once daily
- Increases uveoscleral outflow
- Proinflammation- causing hyperemia


## Side effects

## Ocular

- Conjunctival hyperaemia
- Eyelash lengthening, thickening hyperpigmentation
- Irreversible iris hyperpigmentation
- Periorbital fat loss
- deepening of the upper lid sulcus
- Hyperpigmentation of periocular skin - Common but reversible

- B blockers:
- Twice daily
- Decrease aqueous production
- Contraindicated in patients with bradycardia/ heart block/ asthma
- $\alpha_{2}$ agonists :
- Aqueous Suppressant
- Neuroprotective
- Can cause severe allergic reactions/ contact dermatitis

- Carbonic Anhydrase Inhibitors
- Systemic ( Acetazolamide)
- Topical
- Sulfonamide derivative/ watch out for allergy
- Aqueous suppressant
- Parasympathomimetic / Cholinergic agonists
- Pilocarpine
- Increase conventional pathway outflow
- Cause miosis, myopic shift
- May increase retinal detachment risk


## Laser Treatment

- Increase Outflow Facility
- Trabeculoplasty
- Iridotomy
- Decrease Aqueous production
- Cyclodiode laser


## Trabeculoplasty

- Series of laser burns at the TM to increase outflow facility



## Cyclodiode

- Transscleral Ciliary body Ablation to decrease aqueous production



## Surgery

- Trabeculectomy
- Glaucoma Drainage Devices


## Trabeculectomy

- A fistula between the anterior chamber and the subtenon space



## Glaucoma Drainage Devices

Ahmed Valve


## Summary

- All patients have glaucoma until proven otherwise
- Risk assessment is based on IOP, other risk factors
- Classify based on gonioscopy and other anterior segment findings
- Stage the disease based on optic nerve and field changes


## The End

