CLINICAL PRESENTATION
OF GLAUCOMA

DR. FAISAL ALMOBARAK
ASSISTANT PROFESSOR AND CONSULTANT
DEPARTMENT OF OPHTHALMOLOGY
COLLEGE OF MEDICINE AND KING SAUD UNIVERSITY
SAUDI ARABIA
Glaucoma is a heterogenous group of diseases with characteristic ONH damage and VF changes.

IOP is the single factor to be controlled.
AQUEOUS HUMOR

Produced by the non-pigmented ciliary epithelium

- Active secretion:
  1. Na/K ATPase
  2. Cl secretion
  3. Carbonic anhydrase
- Passive secretion
  1. Ultrafiltration
  2. Diffusion

Production rate is 2-3 µL/min
INTRODUCTION

AQUEOUS HUMOR

Aqueous outflow
INTRODUCTION

CLINICAL ASSESSMENT

- VA
- IOP
- SLE
- Gonioscopy
- ONH assessment
- Diagnostics: VF, OCT, Pachymetry…
**INTRODUCTION**

**IOP**

Aqueous secretion = Aqueous outflow

\[ Po = (F/C) + Pv \]

<table>
<thead>
<tr>
<th>Symbole</th>
<th>Means</th>
<th>Value</th>
<th>Measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Po</td>
<td>IOP</td>
<td>10-21 mmHg</td>
<td>Tonometry</td>
</tr>
<tr>
<td>F</td>
<td>Aqueous production</td>
<td>2-3 µL/min</td>
<td>Flurophotometry</td>
</tr>
<tr>
<td>C</td>
<td>Outflow facility</td>
<td>0.22-0.28 µL/min/mmHg</td>
<td>Tonography</td>
</tr>
<tr>
<td>Pv</td>
<td>Episceral venous pressure</td>
<td>8-12 mmHg</td>
<td></td>
</tr>
</tbody>
</table>
INTRODUCTION

IOP Measurement

Applanation
• Imbert-Fick principle: \( P = \frac{F}{A} \)
  1. Goldmann
  2. Perkins
  3. Tonopen
  4. Pneumotonometer

Indentation
• Schiotz

Noncontact
• Air puff
INTRODUCTION

Gonioscopy
INTRODUCTION

Gonioscopy

IS THE IRIS

Covering TM

Not covering TM

CLOSED

OPEN
INTRODUCTION

Scheie:
1. Grade 1: CB seen
2. Garde 2: SS seen
3. Grade 3: ATM seen
4. Grade 4: TM not seen

Shaffer
1. Grade 1: 10% open
2. Garde 2: 20% open
3. Grade 3: 30% open
4. Grade 4: 40% open
INTRODUCTION

Spaeth

1. Iris insertion: A, B, C, D, E

2. Iridocorneal angle width in degrees (5-45)

3. Peripheral iris configuration: r, s, q

4. TM pigmentation: 0-4
INTRODUCTION

ONH complex evaluation

- Disc margin and disc diameter
- Neuroretinal rim
- Cup/disc ratio
- Disc size
- PPA
- NFL defect
- Optic disc haemorrhage
INTRODUCTION

ONH patterns

• **Focal**: areas of localized loss of rim in superior and/or inferior poles, the remaining rim relatively well preserved.

• **Diffuse**: concentric enlargement of the cup with no localized loss. Small PPA can be present.

• **Sclerotic**: saucerized, shallow cup with pale rim and moth-eaten appearance. PPA surrounding most of the disc.
Classification of Glaucoma

Aetiology

Primary
- No detectable reason
- Often bilateral

Secondary
- Predisposing factor
- Often unilateral

Angle

Open

Closed

Combined Mechanism
Primary Open Angle Glaucoma

- Progressive bilateral asymptomatic disease. Vision later!
- Risk factors:
  - Age
  - Race
  - Family history
  - DM
  - Low perfusion pressure
  - Myopia
  - Retinal diseases

IOP > 21 mmHg

IOP ≤ 21 mmHg (NTG)
**Primary Open Angle Glaucoma**

---

**Single Field Analysis**

<table>
<thead>
<tr>
<th>Stimulus</th>
<th>White Background:</th>
<th>Pupil Diameter: 5.3 mm</th>
<th>Date: 16-01-2006</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixation Monitor:</td>
<td>Gaze/Blind Spot</td>
<td>Fixation Target:</td>
<td>Fixation Losses:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fixation:</td>
<td>C/17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Strategy: SITA-Standard</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visual Acuity: 6/6</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time: 10:18 AM</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age: 44</td>
<td></td>
</tr>
<tr>
<td>Test Duration:</td>
<td>03:35</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Fovea:** 37 dB

---

**Outside of Normal Limits**

- VFI: 60%
- MD: >10.19 dB P < 0.5%
- PSD: 12.77 dB P < 0.5%

---

**Visual Function:**

- Tolerated Well

---

© 2010 Carl Zeiss Meditec
HFA II 750-8404-3.5/5.0
Secondary Open Angle Glaucoma

Pre-Trabecular
(Membrane on TM)
- Fibrovascular
- Endothelial
- Epithelial
- Fibrous
- Inflammatory

Trabecular
(Particle clogging TM)
- RBC, degenerated
- WBC
- Proteins
- Pigments
- PXF
- OVD-Silicon
- Lens material
- Vitreous
  (TM changes)
- Edema (uveitis)
- Tear, scar
- Toxicity
- Laser

Post-Trabecular
(Increased ESVP)
- SVC obstruction
- C-C fistula
- Sturge-Weber syndrome
- Thyroid eye disease
PXF Glaucoma

• Unilateral (mostly) or bilateral
• 6th-7th decade
• asymptomatic, later vision drops.
• PXF more in females
• Males are at higher risk for glaucoma
• Fibrillogranular, extracellular matrix material
• Higher initial IOP than POAG
• Can have spikes despite open angle
PXF Glaucoma

- Cornea: guttata, pigments (krukenberg)
- AC: mild flare
- Iris: PXF on pupil margin, moth-eaten TID
- Gonioscopy:
  I. Open angle: patchy, dark hyperpigmented TM, dandruffs, Sampaolesi’s line
  II. Closed angle: combined mechanism
**Pigmentary Glaucoma**

- Bilateral
- White, myopic males (M:F, 2:1)
- 3\(^{rd}\)-4\(^{th}\) decade
- Reverse pupillary block: mechanical rubbing of posterior pigment layer of iris against the zonules.
- Pigments obstructs TM, denudation, collapse and sclerosis
- Sudden liberation of pigments cause halos, corneal edema, pain
- Unstable IOP with wide fluctuations
Pigmentary Glaucoma

- Cornea: krukenberg’s spindle
- AC: very deep, pigments
- Iris: peripheral TID, pigments
- Asymmetrical pupils
- Gonioscopy: widely opened with hyperpigmented TM all over
Steroid Induced Glaucoma

- Risk factors:
  - Open angle glaucoma
  - Family history of glaucoma
  - DM
  - High myopia
- Topical steroids have greater IOP rising effect than systemic steroids
  - High IOP
  - Open angle
  - Glaucomatous disc damage
Red Cell Glaucoma

- Trabecular blockage by RBCs
- Usually follows trauma
- Sicklers at higher risk of complications
- The larger the size, the higher the incidence of glaucoma:
  I. 27% risk with \( \frac{1}{2} \) AC hyphema
  II. 52% risk with total hyphema
- Need to R/O angle recession
- **GONIOSCOPY**
Angle Recession Glaucoma

- Tear between longitudinal and circular fibers of ciliary muscles
- Breaks in posterior TM result in scarring
- 60-90% of traumatic hyphema
- 5% develop glaucoma
  - High IOP
  - Open angle, enlarged CB band, torn iris processes
  - Glaucomatous disc damage
Primary Angle Closure Glaucoma

PAC
- Anatomically predisposed eye

PACG
- PAC
- ONH damage

Anatomic factors
- Anterior location of iris-lens diaphragm
- Shallow AC
- Narrow angle
- Short AL
- Hyperope
- Small corneal diameter
- Lens size
Primary Angle Closure Glaucoma

Classification

- **Angle closure suspect**
  - Asymptomatic
  - Anatomically predisposed eye
  - Shallow AC
  - Appositionally closed angle, open with indentation

- **Intermittent angle closure glaucoma**
  - Rapid closure of angle with pupillary block and high IOP
  - Spontaneously relieved
  - Transient blurry vision and halos
  - No redness
Primary Angle Closure Glaucoma

- Acute angle closure glaucoma
  - Visual loss with sudden pain and redness
  - Nausea and vomiting
  - High IOP
  - Ciliary flush
  - Corneal edema
  - Shallow AC with peripheral IC contact
  - AC cells and flare
  - Fixed, mid-dilated pupil
  - Closed angle. **GONIOSCOPE THE OTHER EYE**
Primary Angle Closure Glaucoma

- Primary (chronic) angle closure glaucoma
  - Asymptomatic
  - Gradual closure of angle cause slow IOP rise
  - Have large VF loss
  - Gonioscopy: variable amount of angle
  - ONH damage (pallor!)
Plateau Iris

**Configuration**
- Anterior position of CP results in:
  - Deep AC
  - Narrow angle
  - Flat iris plane

**Syndrome**
- Younger age than pupillary block ACG
- Acute angle closure post pupil dilation or spontaneously
- Punched up peripheral iris after dilation and closing TM:
  - Patent PI
  - Deep AC
  - Flat iris
Uveitic Glaucoma

- IOP rise: transient Vs. persistent
- Chronicity and severity of disease
- Topical steroids role !!
- IOP fluctuation is significant
- CB shutdown: especially with acute exacerbation of chronic anterior uveitis. Permanent angle damage
- Miotic pupil and media opacities affect disc assessment
Inflamed iris easily stick to pupil causing 360° posterior synechia
Anterior bowing of peripheral iris (iris bombé) cause the peripherally inflamed iris to easily stick to TM and cornea and the development of PAS

- Uncommon
- IOP mostly normal (CB shutdown)
- iris bombé
- Shallow AC
- Gonioscopy: PAS
- ONH damage
Uveitic Glaucoma

Uveitic Angle Closure Glaucoma Without Pupillary Block

- Deposition of inflammatory cells and debris in angle
- Contraction of inflammatory membrane will pull peripheral iris over TM and cause progressive PAS
  - Deep AC
  - Gonioscopy: extensive PAS
  - ONH damage
**Uveitic Glaucoma**

Uveitic Open Angle Glaucoma

---

**Acute Anterior Uveitis**

- CB shutdown
- IOP is usually normal
- Steroid effect
  - Trabecular obstruction:
    - inflammatory cells and debris
    - Increased aqueous viscosity
  - Acute trabeculitis:
    - Inflammation and edema of TM

**Chronic Anterior Uveitis**

- Chronic trabeculitis cause trabecular scarring/sclerosis
  - Gelatinous exudate in angle
  - Might have PAS later
Uveitic Glaucoma

- Posner-Schlossman syndrome
  - Recurrent attacks of unilateral, acute high IOP with mild uveitis
  - Acute trabeculitis. ?? Viral
  - More in males
  - IOP rise hours to days
  - May shift to chronic course
  - Mild discomfort, halos and blurry vision
  - Corneal edema
  - High IOP (> 40 mmHg)
  - White KPs
  - Few cells and flare
  - Gonioscopy: open angle
Uveitic Glaucoma

- Fuchs uveitis syndrome
  - No posterior synechia
  - Stellate KPs
  - Mild uveitis
  - Gonioscopy
  - Fine radial vessels
  - Small irregular PAS
  - Membrane covering the angle
Neovascular Glaucoma

- Sever, chronic retinal ischemia produce VEGF in an attempt to re-vascularize ischemic areas
- VEGF diffuse to AC
- Causes:
  - DR
  - Ischemic CRVO
  - Chronic RD
  - Chronic inflammation
  - CRAO
  - Carotid occlusive disease
  - Intraocular tumors
**Neovascular Glaucoma**

**Rubeosis irides**
*(Early stage)*
- Tiny capillary tufts at pupil margin
- Grows toward the angle
- Normal IOP
- Open angle
- RVO, CRAO: might have NVA without NVI

**2°OAG**
*(Intermediate stage)*
- NV grows over iris root
- FV membrane over CB and scleral spur to angle
- FV membrane block TM
- High IOP
- Open angle

**2°ACG**
*(Advance stage)*
- Contraction of FV membrane cause PAS
- Progress in zipper-like fashion
- Sever visual loss
- Pain and redness
- Very high IOP
**Lens Related Glaucoma**

**Lens protein**
- Hypermature cataract
- HMW protein leak through intact capsule + macrophage containing lens proteins blocks TM
- Pain, already poor VA
- Corneal edema
- Deep AC with white particles and pseudohypopyon
- Open angle

**Phacomorphic**
- Increased size of intumescent cataract
- Pupillary block
- Symptoms like AACG
- Same findings as AACG + intumescent cataract

**Phaco-anaphylactic**
- Leaking lens material through opened lens capsule