Glaucoma
(γλαυκώμα)

Jorge L. Fernandez Bahamonde, MD.
Basics

- Glaucoma is a large group of disorders that share:
  - Progressive deterioration of the optic nerve and the visual field.
    - 25-38% treated patients keep losing VF if IOP drop to “normal” range.
    - 27% may go blind in one eye after 20 years (treated).
  - If not treated will lead to blindness.
    - Second cause of blindness in US.
      - 80,000 blind.
    - First among blacks.

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1 O’Brien, et al, IOP and the rate of of visual field loss, AJO 1991; 111: 491-500
Classification.

- Etiology.
  - Primary.
    - Not associated with obvious ocular or systemic disorders.
    - Bilateral, genetic basis.
  - Developmental.
  - Secondary.
    - Associated with an underlying disorder.
    - Unilateral or bilateral.
    - Genetic origin or acquired.
Classification.

- **Mechanism.**
  - Open angle.
    - Increased resistance of aqueous outflow.
      - Juxtacanalicular tissue of the TM.
  - Closed angle.
    - Outflow is blocked by the root of the iris.
- **Evolution.**
  - Acute, Subacute, Chronic.
Primary Open Angle Glaucoma: 

**Definition.**

- Chronic progressive anterior optic neuropathy:
  - not associated to other ocular pathology.
  - Intra-ocular pressure (IOP) is a prominent risk factor.
- Characteristic optic nerve and field changes.
- Bilateral, although often asymmetric.
Primary Open Angle Glaucoma: 
Etiology.

- Preferential loss of magnocelullar retinal ganglion cells.
  - Mechanical.
    - Damage at lamina cribosa.
  - Vascular.
  - Axoplasmic flow.
    - Secondary degeneration.
      - Elevation of glutamate in vitreous samples ³.
      - Future aim of therapy.
    - Apoptosis.
      - Elevation of Nitric Oxide Synthase (NOS) in optic nerve head.

RGC path to death?

Elevated IOP

Initial damage in susceptible individuals

Glutamate

Overstimulation of NMDA receptors

Nitric oxide and superoxide anions

Na influx

Secondary Phase

Ca influx

Cytotoxic enzymes

Recovery

Cell death
POAG: Epidemiology.

- Most common type of glaucoma in whites.
  - 1-3%, prevalence increase with age.
    - 8.8% in West Indies > 30 yr.
    - 0.11% per year for whites 55-74.
  - 60 million in the world.
    - 10% blind.
  - 2-3 million in the US. (second cause of blindness).
    - Half of them unaware.
    - 30,000 in PR?
    - 7,000,000 visits/year.

- Very aggressive in blacks.
  - Chronic angle closure plays a significant role.
POAG: Major Risk Factors.

- Intraocular pressure.
  - Great individual variation in susceptibility to damage.
    - 1/6 have IOP < 21.
    - Peaks & Diurnal Curve.

- Race.
  - Less common in China, rare in South Pacific.
    - China PACG > POAG.
  - Blacks more susceptible, 4 to 5x.
    - More prevalent, early appearance and blindness.
    - How about Hispanics?

- Age.
POAG: *Minor Risk Factors.*

- Family history.
  - 3.7 x, relative risk in siblings (Baltimore Eye Study).
  - Pos. family history 13-25%.
  - AD & AR, but probably is multifactorial.

- Vascular disease.
  - Hypertension & Hypotension.
    - Positive association of low diastolic perfusion pressure and/or wide pulse pressure with high tension POAG. \(^1\)
  - Migraine.

- Diabetes Mellitus
  - 3x increase POAG for DM, 6-11% incidence of DM in POAG. Protection against PDR?

\(^1\) Blood Pressure and Glaucoma, Archives June 2007
POAG: Minor Risk Factors

- Gender: OHT greater in females, POAG greater in males.
- Myopia.
  - 3-18%.
  - P < .001 if myopia > 10 diopters. Blue Mountain Eye Study, Australia.
- Thyroid associated orbitopathies.
- Immune?, RA, thyroid, migraine, Raynaud’s.
- Metabolic?, statins lower risk of POAG in cardio-pts.\(^4\)

POAG: Genetics.

- Familial association.
- Potential Glaucoma Loci.
  - GLC1: POAG
  - GLC2: PCAG
  - GLC3: Congenital Glaucoma
  - Add letters to above prefixes.
- Eight genes already.
  - GLC1A to F
  - GLC3A & B
POAG: Genetics.

- GLC1A.
  - 1q21-q31, 3-cM region.
  - Juvenile Open Angle Glaucoma.
    - Myocillin or TIGR (TM-induced glucorticoid response protein)

- GLC1F.
  - 7q35-q36.

- Changes in mitochondrial DNA (mtDNA).
  - Mitochondrial dysfunction as a risk factor in POAG.¹

¹ Investigative Ophthalmology & Visual Science, June 2006
POAG: *IOP*.

- Prevalence of POAG is proportional to IOP level.
- Animal models.
  - Identical to human expression of damage.
- Past damage predicts future damage, unless IOP is lowered.
- Damage in one eye is associated to increased risk in the other, unless IOP is lowered.
POAG: *IOP is not alone.*

- Great individual variation in susceptibility.
  - 10% or less of OHT develop POAG in 10 yrs.
  - 15-20% of POAG have IOP < 21 mm Hg.
- Progression may occur in the “normal” range.
  - If damage is severe IOP need to be in the 10-12 mm Hg range.
  - Positive association of high diastolic perfusion pressure and NTG. 

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1 Blood Pressure and Glaucoma, Archives June 2007
POAG: *Race & Age.*

- Prevalence in 5 x greater in blacks.
- Blindness 4-8 x more common.
- Disease more aggressive.
  - High IOP resistant to medical or laser therapy.
  - Filtering surgery fails even with anti-metabolites.
- Age. Expression of vascular compromise?
  - IOP increase with age as well as POAG.
  - Life expectancy reduced with high IOP or presence of glaucoma. 

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POAG: Evaluation.

- History.
  - Present ocular and systemic history.
    - Usually asymptomatic, but:
      - Loss of vision, haloes, difficult driving.
      - Frequent change of glasses, rare: pain.
  - DM
  - HBP, OHD, ASHD.
    - Systemic beta-blockers.
  - Bronchial Asthma, COPD.
- Ocular medications.
  - Steroids
  - Anti-histaminics.
POAG: *Evaluation.*

- **Past history.**
  - Ocular surgeries.
  - Experience with previous ocular medications.
    - Glaucoma meds?.

- **Family history.**
  - Severity in other members strongly suggest a more aggressive approach to therapy.

- **Social history.**
  - Smoking, alcohol.
  - Impact of present or potential disability.
POAG: Evaluation, Exam

- IOP.
  - Applanation.
    - Dicey if cornea is thin.
      - Pachymetry.
      - Goldmann assumes 520 nM. OHT cut is 550 nM.
    - NCT not for diagnosis or follow up.
  - Different time of the day.
    - Diurnal curve?
  - Forget about normal IOP.
    - Easy when:
      - < 12 mm Hg or
      - > 30 mm Hg.
POAG: *Evaluation.*

- **Exam.**
  - **External.**
    - Blepharitis. (problems with future surgery).
  - **Anterior segment**
    - Cornea
      - Guttate, deposits (pigmented, KP’s).
    - A/C depth and clarity.
    - Iris.
      - Transillumination.
      - Rubeosis.
POAG: Evaluation, Exam

- **Pupil.**
  - Symmetry, response.
  - RAPD.

- **Lens.**
  - Cataracts.
  - Deposits, (PXS).

- **Gonioscopy.**
  - R/O angle closure or secondary causes.
POAG: *Evaluation, Exam: Gonioscopy*

Trust only Schwalbe’s line (reflection where light from Descemet's membrane and Bowman meet) and the Scleral Spur.
POAG: *Evaluation, Exam: Gonioscopy*

- Describe what you see.
  - Degrees of iris-cornea angle.
    - 0- closed.
    - Slit
    - I-II, narrow.
    - III open.
    - IV wide open.
  - Insertion of iris.
  - PAS, PIGM, Rubeosis, Clefts, Recession, etc.
POAG: *Evaluation, Exam: Gonioscopy*

- Closed Angle
- Indentation in a narrow angle
- Brownish pigmentation of the normal angle
- PXS with Sampaolesi's line
POAG: Evaluation, Exam

- Optic nerve and nerve fiber layer.
  - Magnified stereo view.
    - Slit lamp plus a 90 diopter or superfield lens.
    - Search for other fundus abnormalities.
      - 20 or 30 diopter indirect ophthalmoscopy.

- Documentation
  - Stereo Photographs.
  - HRT-II or similar.
    - Just beginning.
POAG: *Evaluation, Exam*

- Optic nerve, normal.
  - Round and symmetric excavations.
  - Well preserved rim and NFL.
  - Smooth vessels path.
  - No hemorrhages or notches.

*Image of a retinal examination showing a normal optic nerve with round and symmetric excavations, well-preserved rim and NFL, smooth vessels path, and no hemorrhages or notches.*
POAG: *Evaluation, Exam*

- Suspicious disk.
  - Larger cup.
    - Saucer shape.
  - Vessel path at greater angle.
  - Asymmetry.
- Correlate with
  - Fields.
  - Fellow eye.
POAG: *Evaluation, Exam*

- Glaucoma disk.
  - Large cup.
  - Asymmetric enlargement.
  - Notch.
  - Sharp turn of vessel.
  - Nasalization.
- NFL loss.
- Hemorrhages.
- Peripapillary atrophy.
Optic Nerve Changes

Thinning of disc rim superiorly and increased saucerization of disc rim inferiorly
Optic Nerve Changes

Focal thinning of disc rim from approximately 5:30 to 6:00 o'clock; sectoral loss of nerve fiber layer and disc hemorrhage
Optic Nerve Changes

Generalized thinning of disc rim, most notably inferiorly; generalized and focal arteriolar narrowing
Optic Nerve Changes

Thinning of disc rim inferotemporally; vessel that traverses disc margin at 7:30 o'clock changes its course. NFL hemorrhage at 7:00 o'clock
POAG: *Evaluation, Exam*

- HRT-II
  - Rim volume.
  - Cup shape.
  - Rim area.
- Depends on examiner demarcation of optic nerve borders.
- Room for improvement.
POAG: Evaluation, Exam HRT-II

Stereometric Results 0° - 360°

- Disk Area: 1.799 mm²
- Cup Area: 0.616 mm²
- Cup/Disk Area Ratio: 0.343
- Rim Area: 1.183 mm²
- Height Variation Contour: 0.497 mm
- Cup Volume: 0.099 cm³
- Rim Volume: 0.383 cm³
- Mean Cup Depth: 0.201 mm
- Maximum Cup Depth: 0.691 mm
- Cup Shape Measure: -0.045
- Mean RNFL Thickness: 0.320 mm
- RNFL Cross Section Area: 1.526 mm²

Az [mm] 0.00 0.10 0.20 0.30 0.40

Tilted, relative
HRef = 0.496 mm
αH = 0.2°
αV = 0.3°
POAG: Evaluation, Exam HRT-II, obvious damage.
GDx

- Lack of hourglass pattern.
- Deviation from normals.
- Flattening of curve.
- Poor numbers, high NFI
Gdx: Early detection OD.

Courtesy of Dr. Naoya Fujimoto Chiba University, Japan
OCT for Glaucoma.

- Optic nerve analysis report.
  - Topographic view of the optic nerve head.
    - Looks similar to HRT-II

- RNFL thickness report.
  - Thickness of NFL around optic nerve.
    - Looks similar to a GDx.
Optic nerve analysis report
RNFL thickness report

**Chart Details:**
- **Scan Date:** 03/08/2004
- **Scan Length:** 10.87

**Table:**

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<th>OS (N=2)</th>
<th>OD-OS</th>
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<tr>
<td>Avg Thick</td>
<td>76.73</td>
<td>32.82</td>
<td>43.91</td>
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</table>

**OCT Image and Fundus Image**

**Thickness Chart**

- **RNFL Average is:** 72
- **RNFL Thickness is:** 42 microns at A-scan
- **Caliper Length is:** OFF
- **SNR:** 41.0
- **Accepted A-scans %:** 100.0

**Signatures:**
- Physician: Luis DeCorra M.D.
POAG: *Evaluation, Exam*

- Visual Fields.
  - Computerized static perimetry.
    - Full Threshold.
    - Humphrey VF analizer.
      - SITA.
  - Useful additions.
    - SWAP.
    - High pass resolution.
POAG: *Evaluation, Exam*

- Defects.
  - Nerve fiber bundle defects.
- Initial may be non-specific.
  - Generalized depression.
    - Miosis.
    - Refractive error.
    - Cataract.
POAG: *Evaluation, Exam*

- NFBD.
  - Bjerumm (10-20 degrees from fixation)
  - Arcuate scotomas.
  - Paracentral defects.
  - Nasal Steps.
  - Altitudinal.
    - Superior.
  - Temporal wedge.
NFBDs Gray Scale

- Superior Bjerrum
  10-20 degrees from fixation

- Nasal Step, Paracentral defect & early Bjerrum

- Inferior Arcuate

- Temporal Wedge
NFBDs examples

- Which test was performed?
- Is the patient's demographic and clinical information correct?
- Is the field reliable?
- Is the field abnormal?
- What is the pattern of the abnormality?
- Is the current examination different (worse) than previous examinations?
- Is the abnormality or worsening due to disease or artifact?
POAG.

Diagnosis and course of action.

- Combination:
  - History.
  - Optic nerve, fields.
  - Time.

- Best course.
  - Follow AAO guidelines &
    - OHTS.
    - CIGTS.
    - AGIS.
    - CNTGS.
POAG.
Key studies.

- **OHTS. AJO 120 No.6 June 2002**
  - Risk of developing glaucoma halved in susceptible individuals when IOP:
    - \( \leq 24 \text{ mm Hg} \) or 20% reduction from baseline average (min. 6 years follow up).
    - Significant reduction of risk if CCT > 550.
  - “Susceptible individuals predictors”.
    - older age.
    - larger vertical or horizontal cup-disc ratio.
    - higher intraocular pressure.
    - greater pattern standard deviation.
    - thinner central corneal measurement.
      - 3x/5 yrs risk of POAG if IOP > 25.75 mm Hg and CCT < 555.
CCT: IOP correction models.

- Imbert Fick.
  - \[ P = \frac{F}{\text{Area flattened}}. \]
  - Assumes.
    - Sphere.
    - Dry.
    - Flexible, elastic
    - Infinite thin.
- Goldmann’s
  - 520 microns.
  - Particular.
    - Rigidity.
    - Curvature.
    - Elasticity.

Table

<table>
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<tr>
<th>K Thickness ((\mu\text{m}))</th>
<th>IOP (+/- mm Hg)</th>
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<td>445</td>
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<tr>
<td>475</td>
<td>+4</td>
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<tr>
<td>595</td>
<td>-6</td>
</tr>
<tr>
<td>605</td>
<td>-7</td>
</tr>
</tbody>
</table>

For a corneal measurement of 10 \(\mu\text{m}\) to 20 \(\mu\text{m}\) thicker or thinner, IOP is adjusted by adding or subtracting 1 mm Hg.

POAG.
Key studies.

- CIGTS. OPH Vol. 108 Nov 2001
  - Newly diagnosed patients.
    - 4-5 years FU. Randomized to medical Rx or Trab (with or without 5FU).
    - IOPs
      - Medical Rx: 17-18 mm Hg (drop of 35% from baseline).
      - Surgery: 14-15 mm Hg (drop of 48% from baseline).
  - Outcome.
    - Similar VFs preservation.
    - VA worse in surgery group (Cataract).
POAG.
Key studies.

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POAG.
Key studies.

- Early Manifest Glaucoma Trial. Arch Oph 2002:120(10); 1268-1279
  - First to offer a control group with glaucoma.
    - Rx (medical) vs observation in early glaucoma.
      - 360º ALT + Betaxolol for 25 % IOP drop.
    - Sweden & US.
  - At 48 months of follow up:
    - 30% progression in treated.
    - 49% progression in control.
POAG.
Key studies.

- Early Manifest Glaucoma Trial.
  - Median time for progression.
    - 48 months in control.
    - 66 months in treated.
  - This persistent separation between groups is damped by intervention over control group when progression is found.
  - 1 mm Hg drop = 10% lowering risk.
POAG.
Key studies.

  - Advanced POAG, 7 + years FUP.
  - Disease progression close to zero only if:
    - IOP below 18 mm Hg at all visits first six years of FUP.
    - Mean IOP during this period 12.3 mm Hg.
POAG.

Key studies.

  - One eye treated, fellow eye control.
    - 7 + years FU.
    - 30 % drop from baseline for treated.
    - Deterioration ratio. Control vs. Treated, 3:1.
Primary angle closure glaucoma: 

*History*

- Acute congestive glaucoma.
  - Main recognized form of glaucoma, late 19\textsuperscript{th} century and early 20\textsuperscript{th} century.
- Curran 1920, role of PI.
- Barkan 1930’s gonioscopy.
  - Fail to recognize pupillary block mechanism.
- Rosengren 1958.
  - Two populations.
    - OAG & those with shallow A/C very high IOP
Primary angle closure glaucoma: *Definition.*

- Appositional or synechial closure of the TM caused by pupillary block in the absence of other causes of angle closure.
  - Hyperopia.
- Mechanism. “Crowded A/C”
  - Excessive iris-lens contact.
  - Decrease flow from posterior to anterior chamber.
  - Forward bowing of peripheral iris.
  - Appositional angle closure.
PACG: Differential Diagnosis.

- Secondary angle closure.
  - NVG: PDR, RVO.
  - Aphakic or pseudophakic pupillary block.
  - Uveitis. Secondary block or PAS.
  - ICE.
  - Ciliary block.
  - Post retina sx.
  - Lens induced (lens swelling, subluxation, nano)
  - Posterior segment tumors.
  - Iris cysts.
- Plateau Iris.
DD: NVG.

- Steamy cornea, difficult to evaluate affected eye.
  - Drop IOP with glyrol.
  - Glycerine over cornea to dehydrate it.
- Take a look at the other eye.

Regression of vessels after Avastin injection.
PACG: *Classification.*

- Acute.
- Sub acute or intermittent.
- Chronic.
- Residual or mixed mechanism.
- Suspect.
PACG: Epidemiology.

- Peaks sixth decade.
- Prevalence general population.
  - Whites 0.09-0.17%.
  - Eskimos 2.12-2.9%. (only 0.01-0.4% POAG).
  - Asians intermediate.
- Sex.
  - Greater in females (except in blacks, prevalence is similar).
- Family history.
  - Non-predictive.
PACG: Epidemiology

- Importance of PACG.
  - 60 millions with Glaucoma.
  - 1/3 have PACG.
  - Blind form glaucoma: 10 million.
    - 6 millions have PACG
  - Chronic angle closure.
    - Not recognized and mistreated as POAG.
PACG: *Acute.*

- Sudden closure.
  - Severe elevation of the IOP.
- Symptoms.
  - Hours, or few days in evolution.
  - Pain, blurred vision, halos
  - Red eye.
  - Nausea, vomiting.
PACG: *Acute*

- Findings.
  - High IOP.
  - Conjunctival and Ciliary injection.
  - Corneal edema.
  - Shallow A/C.
  - Closed angle.
  - Mid-dilated pupil.
  - Iris atrophy.*
  - Glaukomflecken.*
PACG: Acute. Diagnosis & Course of action.

- **Sum of symptoms and signs.**
  - History.
  - IOP.
  - SLE exam, Gonio OU.

- **Immediate Rx**
  - Medical Stabilization
  - Then Laser PI.
PACG: Indentation gonioscopy.

- Zeiss, Posner or Sussman.
- Push gently central to open angle.
  - PAS vs just appositional closure.
  - Therapeutic and Prognostic value.
PACG: Acute.
Course of action, Medical stabilization then LPI.

- Topical beta-blockers.
  - If systemic conditions allow.
- 4 oz Glyrol (or Isosorbide for DM).
- 500 mg diamox.
- When IOP < 35-40 mm Hg.
  - Pilocar 2%.
- Avoid Iopidine or Alphagan until miosis.
PACG: Acute. LPI and later.

- LPI.
  - Wise or Abraham’s.
  - Argon chipping followed by Nd:YAG.
    - Blue-green, 0.02, 50 microns, 1500 mW.
    - Punch out, 70-80% stroma. 20-40 shots.
    - 3-3.5 mJ single shot dead center, do not enlarge it through the center.

- Careful post-op follow-up.
  - Even with significant early drop in IOP, may rebound dramatically when production of aqueous resumes.
  - Steroids, aqueous suppressants, LPI fellow eye.
PACG: Sub-Acute

- Intermitent or self-limited attacks.
- Eventually will evolve into:
  - Acute exacerbation.
  - Chronic.
- Key:
  - Presence of peripheral anterior synechiae.
- Laser PI.
PACG: *Chronic.*

- Essentially asymptomatic.
- Progressive narrowing of the angle.
  - PAS.
  - Slow increase of IOP.
  - Deterioration of control.
- **Key:**
  - Gonioscopy.
  - LPI, convert to OAG
PACG: *Suspect.*

- Narrow angle with high IOP.
- Narrow angles, pupil dilation needed.
- Documented progressive narrowing.
- Documented PAS.
- Fam. Hx of PACG.
- Usually tough call.
Plateau Iris.

- Primary angle closure not relieved by a patent iridotomy.
  - All eyes should have frequent gonioscopies after LPI to r/o plateau iris.

- Anatomy.
  - Flat iris plane.
  - A/C depth adequate, peripheral iris contour is convex and lies against TM.
  - Abnormally anteriorly placed ciliary processes.
Plateau Iris.

- Be suspicious if PACG occurs in young or myopic patients.

- Treatment.
  - Chronic miotic therapy.
    - Pilocar 1% bid.
    - Rev Eyes (Dapiprazole, alpha adrenergic blockade).
  - Laser iridoplasty.
    - Need to be repeated.
      - 500 microns, 0.5 sec, 200-400 mW, 25-30 burns, no overlap.
Primary Infantile Glaucoma.

- 55% of all glaucoma in infants and children.
- Rest:
  - Aniridia.
  - Anterior cleavage.
  - Sturge-Weber.
  - Trauma.
- Prompt glaucoma specialist evaluation.
  - Rx mandatory.
Primary Infantile Glaucoma.

- Fortunately rare.
  - Few stats.
    - Gen OPH should see one or two in his life.

- Patterns.
  - 25% dx. at birth, 60% by 6 months.
  - Usually AR, however 65% males.
  - OU in 75% (incomplete forms in fellow eye).
Primary Infantile Glaucoma.

- Symptoms & Signs:
  - Epiphora.
  - Photophobia.
  - Blepharospasm.
  - Corneal enlargement.
  - Corneal edema.
  - Tears.
  - Deep A/C
  - Corneal Haze.
Primary Infantile Glaucoma.

- Evaluation under anesthesia.
  - Elevated IOP.
  - Corneal enlargement.
  - Edema, Tears.
  - Angle abnormalities.
  - Optic nerve atrophy.

- Rx: Surgery.