OCULAR EXAMINATION
Front to Back
Secondary Open Angle Glaucoma(s)
Angle Closure Glaucoma
The Big “G”

• Open Angle Glaucoma
  – Primary
  – Secondary
    • Steroid induced
    • Angle recession
    • Pseudoexfoliation
    • Pigment dispersion
  – Accounts for 90% of glaucoma in US

• Angle Closure Glaucoma
  – Acute
  – Chronic
    • Chinese >> C = African-American
  – Accounts for 10% of glaucoma in US
Open Angle Glaucoma

• Primary (POAG)

• Diagnosed
  – Presence of a normal appearing open angle
  – Multi-factorial optic neuropathy that is chronic and progressive
    • Characterized by acquired loss of optic nerve fibers, leading to peripheral vision loss consistent with glaucoma on a visual field test
Primary Open Angle Glaucoma

What is missing from the definition of POAG?
Open Angle Glaucoma

• Primary (POAG)
• Diagnosed
  – Presence of a normal appearing open angle
  – Multi-factorial optic neuropathy that is chronic and progressive
    • Characterized by acquired loss of optic nerve fibers, leading to peripheral vision loss consistent with glaucoma on a visual field test
• Intraocular pressure
  – Ocular hypertension is major risk factor

Where did normal come from???
IOP Studies

• IOP measured by Schiotz tonometer
• 10,000 people tested
• NON-Gaussian curve slightly skewed to higher IOPs
• Mean was 15.5 ± 2.57 mm of Hg
• Two SD above mean is 20.6 (21) mm of Hg
Angle Recession

- Abnormally wide CB
- Blunt trauma history
  – Years earlier

Secondary Open Angle Glaucoma

Ultrasound biomicroscopic evaluation of the traumatized eyes
M P Ç Özdal, M Mansour and J Deschénes
Secondary Open Angle Glaucoma

Pigment Dispersion

- Onset usually less than 40 years old
- Males > Females
- Affects Caucasians almost exclusively
- Glaucoma
- Myopes with nice open angles

Pigment on endothelium or Krukenberg’s spindle
Pigment Dispersion

Sampaolesi's Line
Secondary Open Angle Glaucoma

Pseudoexfoliation

- Flaky white material on anterior lens capsule
- Difficult to treat
Angle Closure Glaucoma

• Develops due to apposition of the iris to the trabecular meshwork

• Two broad mechanisms
  – Pushing forward of iris to angle
  – Pulling forward of iris to angle
Causes of Angle Closure Glaucoma

- Iris Pushed Forward
- Iris Pulled Forward
- Pupillary Block
- Plateau Iris
- Rubeosis Iridis
- Inflammation
- Tumors
- Iridocorneal-endothelial syndrome
- Choroidal Hemorrhage
Pupillary Block Glaucoma

• Narrow angles
• Shallow anterior chamber depth
  – < 1.5 mm ≈ 75% of acute ACG cases
• Thickening lens
• Iris insertion
• Iris configuration
Pupillary Block

- Completely blocks AH escape
- Pressure rise is immediate and drastic
What happens when IOP goes from 14 to 41 (or higher) in minutes?

- What are the patient’s symptoms?
Symptoms

- Unilateral ocular injection
- Ocular and facial PAIN
  - Frontal HA
- Unilateral Blurred vision
- Epiphora
- Colored Halos around lights
- Nausea and vomiting
Signs

• Unilateral

• Lid edema

• Conjunctival injection, most prominent at the limbus

• Corneal edema (microcystic or cloudy cornea)

• Narrow angle/shallow AC
  – Compare to other eye
Signs

• Fixed, mid-dilated pupil
• Shallow anterior chamber, often with inflammatory reaction
  — Iris bombe
• Reduced Best Corrected Visual Acuity (BCVA)
  — Often 20/80 or worse
• Extremely elevated IOP
• Glaukomflecken
  — Evidence of past attack
Fixed dilated pupil
Glaukomflecken

- Clinically as flecks of patchy anterior subcapsular opacities associated with severe elevation of intraocular pressure
- focal epithelial infarct from past acute angle closure glaucoma
Cup asymmetry 6 months after acute angle closure

OD: flat, diffuse optic atrophy

OS: unaffected eye
Acute (Pupillary Block) Angle Closure Glaucoma

- Pressures can reach 60-80 mm Hg
- Causes damage to the ENTIRE EYE
  - The longer the pressures are elevated the worse the damage.
- Ocular emergency
Management

• Goals of treatment
  – Reduction of IOP
  – Suppression of inflammation
  – Reversal of angle closure
  – Evaluation of fellow eye for treatment
Peripheral Laser Iridotomy

• 2-7 days after breaking attack
• IOP elevated at 4-6hr marks
  – Schedule emergency surgical iridectomy

When is the BEST time to get the LPI?
BEFORE the Acute Attack

• Prophylactic LPI

LPI Video
In your practice......

Convincing an asymptomatic patient that they need prophylactic laser iridotomy is one of the hardest things to do

– Approximately a 10% risk of angle closure each year
– Risk is cumulative
– After 5 years, their risk is 50%
Chronic Angle Closure Glaucoma

• Slow, gradual, insidious process
• One of two forms:
  – Chronic appositional closure (without PAS), which will open upon with indentation gonioscopy
• Synechial closure with broad areas of peripheral anterior synechiae
  – Superior and temporal > nasal > inferior
What happens when IOP rises gradually over a long period of time?

• What are patient’s symptoms?
Symptoms

• None, until late in disease process
• Usually these patients present for wellness ocular examinations
  – Condition then “discovered”
Diagnosis Made

- Based on “suspect” optic nerve appearance
  - Will talk about in ONH section
- Visual field changes
- Slit lamp examination
  - Anterior chamber quiet
  - No secondary glaucoma signs
  - Pupils normal
- IOP usually 40 mm Hg or less
  - May be normal in early stages of disease process
  - If long-time patient, may see a gentle rise in IOP over the years
- Know your patients at risk
  - Asian (Chinese)
  - Family history
Diagnosis Made

• Gonioscopy
  – Confirm narrow angle
  – Blotches of pigment on the meshwork or “black” pigment in lightly pigmented eyes

• Very often “confused” with primary open angle glaucoma
Severe, Irreversible Vision Loss

• Don’t forget LOW VISION training
  – Advanced training in the field
  – Clinical experience

• Agencies that can help
  – Counseling services
  – State and Federal for the blind services