Acute Primary Angle Closure
An Ocular Emergency

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Introduction

- Glaucoma is the second leading cause of worldwide blindness
- 67 million patients with glaucoma
- 50% with angle closure glaucoma


Introduction

- Primary angle closure glaucoma-
  - Leading cause of bilateral blindness
  - Predominant form of glaucoma in East Asia
  - Responsible for 91% of bilateral blindness in China
  - In the US, < 10% of glaucoma cases


- Angle closure glaucoma in 2010
  - Estimated ~ 15.7 million people
  - 3.9 million will be bilaterally blind
Etiology

- The most common cause of primary angle closure is pupillary block.

Pathogenesis

- The pathological insult is anatomical -
  - Iridotrabecular apposition

- Peripheral iris interferes with aqueous outflow

Pathogenesis

- In the “narrow angle” patient, any area of apposition of iris to the TM is abnormal.
As a primary care provider, you serve an important role in recognizing patients at risk and referring them for urgent ophthalmologic evaluation.

An attack of acute angle closure is an ocular emergency.

Who is at risk?

Prevention of this disease starts with identifying those most at risk of it's development.

- History
- Exam
Risk Factors of PACG
- Race
- Age
- Gender
- Family History
- Refractive Error
- Eskimoans, E. Asians, Japanese
  - Prevalence increases with age
  - 2-4 x more common in women
  - Increased in 1st relatives
  - Hyperopes

Medication History
- Cold and allergy medications:
  - Antihistamines:
    - Inherent anticholinergic (parasympatholytic) activity
  - Decongestants:
    - Adrenergic (sympathomimetic)
- Topiramate
- Antidepressants

Symptomatic History
- Headaches
- Blurred vision
- Ocular pain
- Tearing
- Photophobia
- Halos around lights
- Nausea and vomiting

Pertinent Clinical Signs
- Red eye
- Mid-dilated pupil
- Elevated IOP
- Corneal edema
- Conjunctival vascular congestion
- Shallow Anterior Chamber
- Iris Bombe
Differential Diagnosis

- Conjunctivitis
- Corneal abrasion
- Ocular infections
- Orbital infections
- Ocular inflammation
- Secondary glaucomas

Work-up

- Visual Acuity
- Extra-ocular motility
- Pupil exam
  
  Ophthalmologic consultation!

Impostors

- “Migraine Headache”
- “Gastrointestinal disorder”

Treatment

- Preliminary intervention:
  ✓ Analgesics
  ✓ Antiemetics
  ✓ Avoid dark rooms
Ophthalmology Referral

• Confirm diagnosis
• Intraocular pressure reduction
• Break the attack
• Preserve vision
• Consider treatment of the other eye

Gonioscopy

• Indirect gonioscopy
  • Goldmann – 3-mirror
  • Zeiss, Posner, Sussman–4-mirror

Medical Treatment

• Initiate topical hypotensive agents:
  • Alpha,
  • Beta-blockers
  • Carbonic anhydrase inhibitors
  • Miotics
Classification of the Narrow Angles

- Narrow - not occludable
- Narrow and occludable
- Plateau iris configuration
- Creeping/Chronic angle closure

Narrow - occludable

- Treatment of choice - LPI
- Purpose is to equalize IOP between anterior and posterior chambers.

Narrow – not occludable

- Full width of the TM is visualized
- Asymptomatic
- Risk assessment
- Educate signs and symptoms of AAC
- Medication warnings
- ? LPI - clinical call

LPI

Courtesy of eyetext.net
LPI

- Treats Pupillary block
  - Argon or Nd:YAG or combination

Risks of LPI

- Elevated IOP
- Bleeding
- Inflammation
- Corneal abrasion
- Halos, glare
- Closure of iridotomy
- Failure to complete LPI

Iridoplasty

- Used to treat residual appositional angle closure
- Contraction burns to the peripheral iris to deepen the angle.
- Plateau iris

Pre-op miotic
- Pre/post-op $\alpha_2$ agonist
- Post-op IOP check
- Topical steroids 5-7 days
- Post-op visit must include repeat gonioscopy!
- Don’t forget the other eye.

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Summary
- Acute Angle closure can be a devastating condition
- Knowledge of risk factors and a thorough history and exam
- Ophthalmologic referral is imperative
- Gonioscopy is required to assess the filtration angle
- Narrow angle suspect – advise of symptoms of AAC attack
- Laser surgical options

Overview
- Definition
- Classification
- Epidemiology
- Intraocular Pressure and aqueous humor
- Clinical Evaluation
- Treatment

Glaucoma
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Glaucoma
- Definition:
  • Glaucosis
    • Clouded
    • Blue-green hue
Glaucoma

- Definition:
  - Progressive optic neuropathy
  - Peripheral visual field loss
    - Intraocular pressure (IOP) is the primary risk factor.

Glaucoma:
- Loss of ganglion cells
- Thinning of retinal nerve fiber layer (RNFL)
- Optic nerve cupping

Glaucoma

- Classification
  - Open Angle Glaucoma (OAG)
  - Narrow Angle Glaucoma (NAG)
Open Angle Glaucoma (OAG)

POAG: Epidemiology

- The Second leading cause of blindness in US¹
- Over 2 million affected¹
  - 2.25 million ≥ 45 years old in U.S.
  - 84,000 – 116,000 bilaterally blind (best VA ≥ 20/200 or field < 20°.
- 50% of cases are undiagnosed²
- Most Common cause of irreversible blindness in African American¹

POAG: Prevalence

<table>
<thead>
<tr>
<th>Age</th>
<th>Whites¹</th>
<th>African-American</th>
<th>Hispanics²</th>
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<tbody>
<tr>
<td>40s</td>
<td>0.9%</td>
<td>1.2%</td>
<td>0.5%</td>
</tr>
<tr>
<td>80+</td>
<td>2.2%</td>
<td>11.3%</td>
<td>12.6%</td>
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Glaucoma Risk Factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Open Angle</th>
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</thead>
<tbody>
<tr>
<td>Intraocular Pressure</td>
<td>+</td>
</tr>
<tr>
<td>Central Corneal Thickness</td>
<td>+</td>
</tr>
<tr>
<td>Age</td>
<td>Older</td>
</tr>
<tr>
<td>Race</td>
<td>AA&gt;W</td>
</tr>
<tr>
<td>Family History</td>
<td>+</td>
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<tr>
<td>Diabetes Mellitus</td>
<td>+</td>
</tr>
<tr>
<td>Sex</td>
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References:
Intraocular Pressure (IOP)

- IOP is the most important modifiable risk factor in patients with glaucoma
- 1 mmHg decrease of IOP will:
  - Decrease the relative risk of POAG progression by 10% (EMGT)¹
  - Decrease the relative risk of POAG Onset by 10% (OHTS)²

1-Leske et al., Arch Ophthalmol 120: 1268-1279, 2002
2-Kass et al., Arch Ophthalmol 120: 701-713, 2002

Intraocular Pressure (IOP)

\[ IOP = \frac{\text{Aqueous Formation Rate (μL/min)}}{\text{Outflow Facility (μL/min/mmHg)}} + \text{Episceral Venous Pressure (EVP)} \]

IOP/Aqueous Dynamics

- IOP
  - Normal range: 10-22 mmHg
  - Diurnal variation:
    - Normal: 2-6 mmHg
    - Higher IOP → greater variation
    - >10 indicative of glaucoma
  - IOP usually peaks in early AM

American Academy of Ophthalmology BCSC Section 10: Glaucoma 2004 P 25

- Factors affecting:
  - Time of day
  - Heartbeat/respiration
  - Exercise
  - Body position

IOP/Aqueous Dynamics
IOP/Aqueous Dynamics

**Aqueous Formation**
- Source: Inner nonpigmented epithelial cells of ciliary processes
- Decreases
  - Sleep
  - Advancing age
  - Trauma

**Aqueous Formation cont**
- Formation process involves:
  - Active transportation
    - ‘Secretion’ independent of pressure
    - Involves CAII
    - Accounts for majority of formation
  - Ultrafiltration
    - Down pressure gradient
  - Diffusion
    - Passive movement of ions across membrane due to charge

IOP/Aqueous Dynamics

**Aqueous Outflow**
- Aqueous enters posterior chamber, then flows through pupil into anterior chamber
IOP/Aqueous Dynamics

- Aqueous Outflow
  - Uveoscleral Outflow
    - Any non-TM outflow
      - Aqueous in AC enters CB exits eye across sclera
    - Is pressure independent
    - May account for 50% of outflow in young people


Clinical Evaluation

- History
- Physical Exam
  - Optic Nerve Function
  - Ophthalmic Exam
  - Ancillary Testing

IOP/Aqueous Dynamics

- Aqueous Outflow
  - Episcleral venous pressure EVP
    - Usual range 8-12 mmHg
    - Acute increase in EVP → 1:1 increase in IOP
    - Chronic increase in EVP → complex effect on IOP

Clinical Evaluation

- History
  - Symptoms
    - Early: Asymptomatic
    - Advanced: loss of peripheral vision

Clinical Evaluation

- History
  - Physical Exam
  - Optic Nerve Function
  - Ophthalmic Exam
  - Ancillary Testing
Clinical Evaluation

• Physical Exam
  - Afferent pupillary defect
    - Marcus Gunn pupil
  - Confrontational visual field testing
  - Optic nerve head ONH evaluation

Clinical Evaluation

• Optic Nerve Head
  ✓ Glaucomatous cupping
    • Starts at level of the lamina
  ✓ Peripapillary atrophy
    • Often associated with Glaucomatous optic neuropathy
    • Location may correlate with VF changes
Clinical Evaluation

- Ophthalmic Exam
  - Applanation tonometry
    - 50% of POAG patients will have IOP consistently < 22 mmHg


Clinical Evaluation

- Ancillary Testing
  - Central Corneal Thickness
  - Optic nerve head Photographs
  - Automated Visual Field Testing
  - Retinal Nerve Fiber Analysis

Clinical Evaluation

- Ophthalmic Exam
  - RNFL defects
    - GON associated with loss of axons in RNFL
    - Best seen in red-free light
    - May be diffuse or localized in specific bundles

[Image: Courtesy of http://www.atlasophthalmology.com]

Clinical Evaluation

- Central Corneal Thickness
  - Mean: 542μm
  - Strong predictor of development of glaucoma
    - RR 81% for every 40 μm Thinner (OHTS)¹
  - Significant risk factor for progression
    - Patients with higher baseline IOP²
  - Effect on IOP measurement

Clinical Evaluation

- Optic nerve head stereophotographs
  - Gold standard to monitor progression

Clinical Evaluation

- Retinal Nerve Fiber Analysis
  - Diagnosis
  - Follow up

Clinical Evaluation

- Automated visual field testing
  - 50% of RNFL loss prior to detect visual field loss

Treatment

- Medical
- Laser
- Surgery
Treatment

- Goal of treatment
  - Arrest or slow the progression of the visual loss
  - Lowering intraocular pressure


Glaucome Medications

\[
\text{IOP} = \text{Aqueous Formation Rate (μL/min)} + \text{Outflow Facility (μL/min/mmHg)}
\]

- Beta blockers
- Nonselective alpha beta agonists
- Selective alpha 2 agonists
- Prostaglandin analogues
- Mannitol
- Carbonic anhydrase inhibitors

- Beta blockers
  - Ex: Timolol, carteolol, betaxolol
  - Average ~25% reduction in IOP
  - Inhibit cAMP in CB \( \rightarrow \) reduced aqueous secretion
  - Can aggravate myasthenia
  - Betaxolol:
    - Selective \( \beta_1 \), safer \( \rightarrow \) pulmonary disease
**Glaucoma Medications**

- **β blockers**
  - Side effects
    - Fatigue
    - Depression
    - Sleep disturbance
    - Heart block
    - Syncope
    - Asthma
    - Decreased sexual ability

**Glaucoma Meds**

- **Carbonic Anhydrase Inhibitors**
  - Side effects
    - Dose related
    - Paresthesias
    - Fatigue
    - Weight loss
    - Hypokalemia...

- **α₂ agonists**
  - Average IOP reduction: ~25% (peak), 15% (trough)
  - Prevent norepinephrine release at neuron terminal
  - Relative contraindication:
    - MAO-Inhibitors
    - Tricyclic Anti-Depressants
Glaucoma Meds

- \(\alpha_2\) agonists:
  - Apraclonidine:
    - Clonidine derivative
    - \(\alpha_2\) selective (but with significant \(\alpha_1\) effects as well)
    - In addition to decreasing aqueous production...
      - Lowers EVP
      - Improves TM outflow
    - Proven effective in blunting IOP spikes when given pre- and post-operatively for LPI, ALT, YAG cap, CE
    - Notoriously allergenic (40%)
    - Significant tachyphylaxis

- Brimonidine
  - Much more highly \(\alpha_2\) selective than Iopidine
  - In addition to decreasing aqueous production...
    - Improves uveoscleral outflow
  - Compared with Apraclonidine
    - Much less allergic (15%)
    - Much less tachyphylaxis

- Prostaglandin analogues/hypotensive lipids
  - Ex: Latanaprost, travaprost, Bimatoprost
  - Average IOP reduction: 30%
  - Peak effect at \(~12\) hours

- Side effects:
  - Darkening of iris & periocular skin
    - More prevalent in darker eyes
  - Eyelash hypertrichosis (increased length/number)
  - Conjunctival hyperemia
  - Can exacerbate:
    - Uveitis
    - Cystoid Macular edema
    - HSV keratitis
**Glaucoma Meds**

- Prostaglandin analogues/hypotensive lipids
  - Side effects:
    - Upper respiratory tract infection/Sinusitis
    - Hypotension

**Glaucoma Meds**

- Parasympathomimetics (miotics) cont
  - Associated with retinal detachment
  - Cataractogenic
  - Ciliary body contraction→induced myopia
  - Ciliary body contraction→brow ache
  - Weaker formulations may help prevent pupillary block
  - Pull peripheral iris away from angle

**Glaucoma Meds**

- Hyperosmotic Agents
  - Ex: Mannitol, glycerin
  - Increase blood osmolality→osmotic gradient between blood and vitreous→water drawn from vitreous cavity→↓IOP
  - ↑dose→increased IOP-lowering effect
  - ↑rate of administration→increased IOP-lowering effect
### Glaucoma Meds
- Hyperosmotic Agents
  - Side effects:
    - Headache, confusion
    - Backache
    - Acute congestive heart failure, myocardial infraction
    - Glycerin is metabolized into sugar→hyperglycemia or even ketoacidosis in diabetics

### Glaucoma Lasers
- ALT
  - Argon laser trabeculoplasty
- SLT
  - Selective laser trabeculoplasty
  - Complications
    - Transient IOP spike
- CPC
  - Cyclophotocoagulation
  - Destruction of ciliary body

### Glaucoma Surgery
- Incisional Surgery
  - Trabeculectomy
  - Tube Shunt
- Angle Surgery
  - Trabectome
  - Canaloplasty