

Acute Primary Angle Closure

An Ocular Emergency

Gloria P. Fleming, M.D.

Assistant Professor of Clinical Ophthalmology
Glaucoma Division
Havener Eye Institute
The Ohio State University

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

Introduction

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

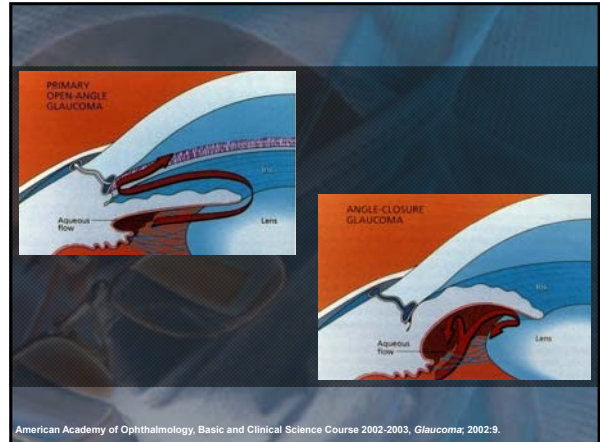
Introduction

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

Quigley H., et al. Br J of Ophthalmology. 2006;90:262-7.

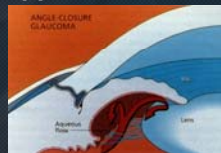
Etiology

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



Pathogenesis

- The pathological insult is anatomical-
 - Iridotrabecular apposition



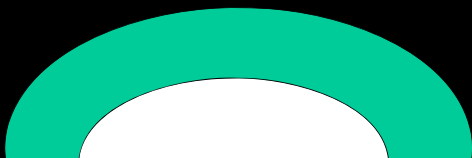
- Peripheral iris interferes with aqueous outflow

American Academy of Ophthalmology, Basic and Clinical Science Course 2002-2003, Glaucoma, 2002:9.

Pathogenesis

- In the “narrow angle” patient, any area of apposition of iris to the TM is abnormal.

The Narrow Angle Patient



Anatomic Narrow angles

Acute angle closure

- 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.

Nomenclature

- Acute angle closure
- Primary angle closure
- Acute angle closure glaucoma
- Primary angle closure glaucoma

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

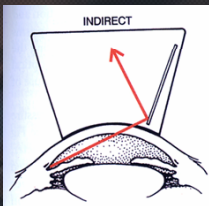
Gonioscopy Grading Systems



Courtesy of eyetext.net

Gonioscopy

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



Source: American Academy of Ophthalmology



Medical Treatment

- Initiate topical hypotensive agents:
 - Alpha₂-adrenergic agonists
 - Beta-blockers
 - Carbonic anhydrase inhibitors
 - Miotics

Classification of the Narrow Angles

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

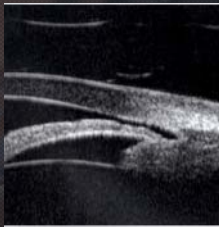
Narrow- occludable

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

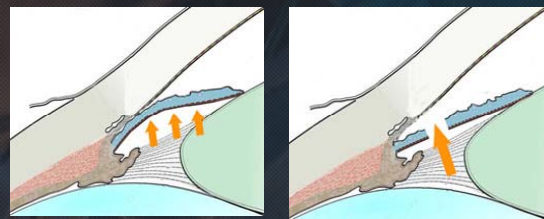


Narrow – not occludable

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



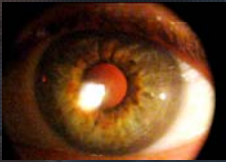
LPI



Courtesy of eyetext.net

LPI

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



- Pre-op miotic
- Pre/post-op α_2 agonist
- Post-op IOP check
- Topical steroids 5-7days
- Post-op visit must include repeat gonioscopy!
- Don't forget the other eye.

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

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

Iyad Azrak, MD
Clinical Assistant Professor
Glaucoma Division
Havener Eye Institute
Ohio State University

Glaucoma

- Definition:
 - ✓ Glaucoma
 - Clouded
 - Blue-green hue

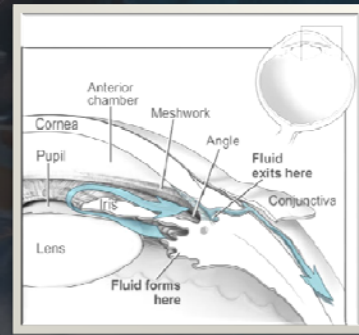
Glaucoma

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

American Academy of ophthalmology BCSC Section 10, Glaucoma 2004 P 3

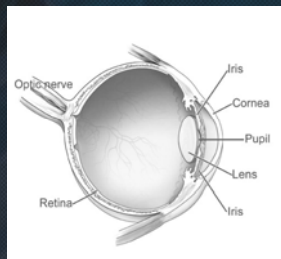
Glaucoma

- Classification

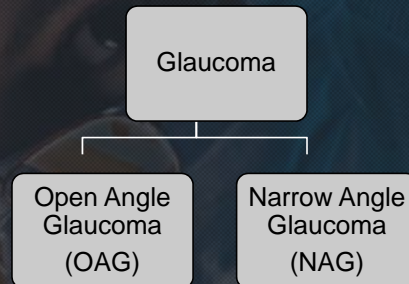


Glaucoma

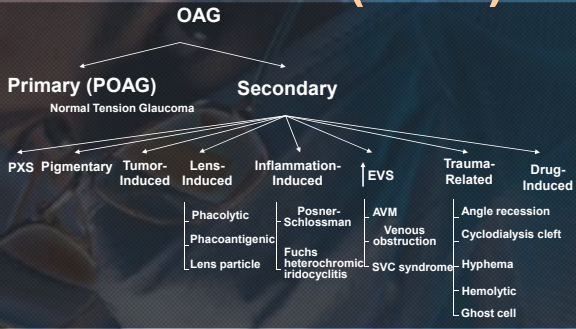
- Glaucoma:
 - ✓ Loss of ganglion cells
 - ✓ Thinning of retinal nerve fiber layer RNFL
 - ✓ Optic nerve cupping



Glaucoma



Open Angle Glaucoma (OAG)



POAG: Prevalence

Age	Whites ¹	African-American	Hispanics ²
40s	0.9%	1.2%	0.5%
80+	2.2%	11.3%	12.6%

1-Tielsch JM, Sommer A, Katz J, et al. Racial variations in the prevalence of primary open-angle glaucoma. JAMA. 1991;266:362-374.
2-Ganguly HA, West S, Rodriguez J, et al. The prevalence of glaucoma in a population-based study in Hispanic subjects. Proyecto VER. Arch. Ophthalmol. 2001;119:1819-1828.

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

1- Leske MC. The epidemiology of open-angle glaucoma: a review. Am J Epidemiology 1983;118:166-191
2-Wensor MD, McCarty CA, Stanislavsky YL, et al. The prevalence of glaucoma in the Melbourne Visual Impairment Project. Ophthalm 1996; 108:733-739

Glaucoma Risk Factors

	Open Angle
Intraocular Pressure	+
Central Corneal Thickness	+
Age	Older
Race	AA>W
Family History	+
Diabetes Mellitus	+
Sex	no

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 120:704-713, 2002

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

IOP/Aqueous Dynamics

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

Intraocular Pressure (IOP)

$$\text{IOP} = \frac{\text{Aqueous Formation Rate } (\mu\text{L}/\text{min})}{\text{Outflow Facility } (\mu\text{L}/\text{min}/\text{mmHg})} + \text{Episcleral Venous Pressure (EVP)}$$

IOP/Aqueous Dynamics

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

IOP/Aqueous Dynamics

- 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

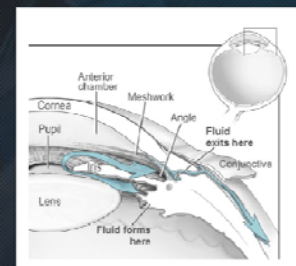
IOP/Aqueous Dynamics



Courtesy of <http://www.atlasophthalmology.com>

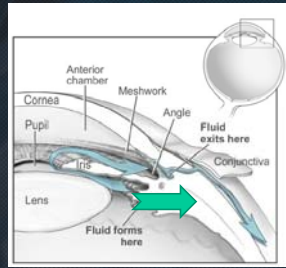
IOP/Aqueous Dynamics

- 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



Brubaker RF. Measurement of uveoscleral flow in humans. J Glaucoma. 2001;10(5 Suppl 1):s35-448

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



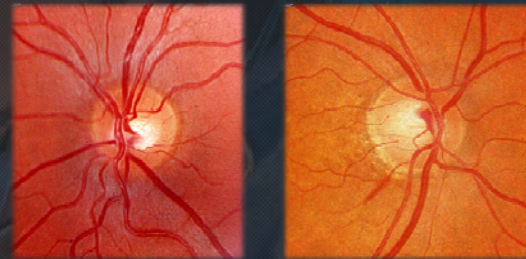
Clinical Evaluation

- Physical Exam

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



Clinical Evaluation



Courtesy of <http://www.atlasophthalmology.com>

Clinical Evaluation

- Optic Nerve Head

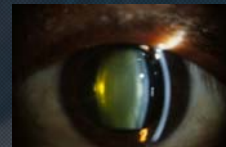
- ✓ Glaucomatous cupping
 - Starts at level of the lamina
- ✓ Peripapillary atrophy
 - Often associated with Glaucomatous optic neuropathy
 - Location may correlate with VF changes

American Academy of ophthalmology BCSC Section 10, Glaucoma 2004 P 50-55

Clinical Evaluation

- Ophthalmic Exam

- ✓ Slit Lamp Exam
- ✓ Gonioscopy



Clinical Evaluation

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



Dielemans I, Vingerling JR, Wolfs RC, et al. The prevalence of primary open-angle glaucoma in a population-based study in The Netherlands. The Rotterdam Study. Ophthalmology 1994;101:1851-5.

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'



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

1- Leske et al. Arch Ophthalmol 120: 1268-1279, 2002

2- EMGT Group Leske et al. Ophthalmol. 114 (11) cases 1965-1972, Nov 2007

Clinical Evaluation

- Optic nerve head stereophotographs
 - ✓ Gold standard to monitor progression

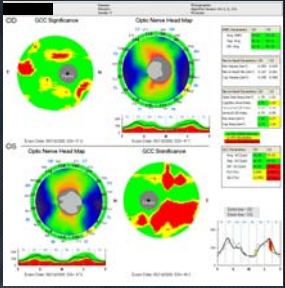


Courtesy of <http://www.atlasophthalmology.com>

American Academy of Ophthalmology practice guidelines

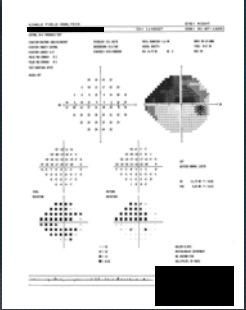
Clinical Evaluation

- Retinal Nerve Fiber Analysis
 - ✓ Diagnosis
 - ✓ Follow up



Clinical Evaluation

- Automated visual field testing
 - ✓ 50% of RNFL loss prior to detect visual field loss¹



¹Quigley HA, Addicks EM, Green WR. Optic nerve damage in human glaucoma. II. Quantitative correlation of nerve fiber loss and visual field defect in glaucoma, ischemic neuropathy, papilledema, and toxic neuropathy. Arch Ophthalmol 1982;100:133-46.

Treatment

- Medical
- Laser
- Surgery

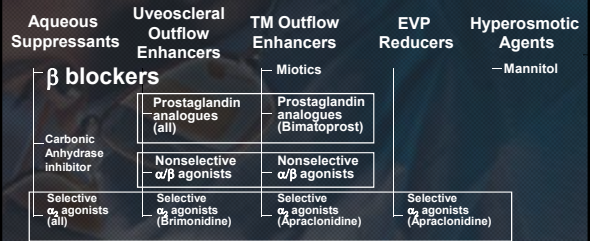
Treatment

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

Collaborative Normal-Tension Glaucoma Study Group. The effectiveness of intraocular pressure reduction in the treatment of normal-tension glaucoma. *Am J Ophthalmol* 1998; 126:498-505.

Glaucoma Medications

$$IOP = \frac{\text{Aqueous Formation Rate } (\mu\text{L}/\text{min})}{\text{Outflow Facility } (\mu\text{L}/\text{min}/\text{mmHg})} + \text{Episcleral Venous Pressure (EVP)}$$



Treatment

- Medical



Faisal A. Adatia, MD, MSc Karim F. Damji, MD, FRCS VOL 8: SEP 2005 G Canad Fam Physician

Glaucoma Medications

- β blockers
 - ✓ Ex: Timolol, carteolol, betaxolol
 - ✓ Average ~25% reduction in IOP
 - ✓ Inhibit cAMP in CB → reduced aqueous secretion
 - ✓ Can aggravate myasthenia
 - ✓ Betaxolol:
 - ❖ β_1 selective → safer in pulmonary disease

Glaucoma Medications

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

Glaucoma Meds

- Carbonic Anhydrase Inhibitor
 - ✓ Side effects
 - Dose related
 - Paresthesias
 - Fatigue
 - Weight loss
 - Hypokalemia..

Glaucoma Meds

- Carbonic Anhydrase Inhibitors
 - ✓ Topically...
 - Dorzolamide
 - Brinzolamide
 - ✓ Systemically (PO/IV)
 - Acetazolamide
 - ✓ Average ~15% reduction in IOP
 - ✓ Direct antagonist to enzyme CAII in CB epithelium

Glaucoma Meds

- α_2 agonists
 - ✓ Average IOP reduction: ~25% (peak), 15% (trough)
 - ✓ Prevent norepinephrine release at neuron terminal
 - ✓ Relative contraindication:
 - MAO-Inhibitors
 - Tricyclic Anti-Depressants

Glaucoma Meds

- α_2 agonists:
 - ✓ *Apraclonidine* :
 - Clonidine derivative
 - α_2 selective (but with significant α_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
- ← Limits long-term use

Glaucoma Meds

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

Glaucoma Meds

- α_2 agonists:
 - ✓ *Brimonidine*
 - Much more highly α_2 selective than lopicidine
 - In addition to decreasing aqueous production...
 - Improves uveoscleral outflow
 - Compared with Apraclonidine
 - Much less allergenic (15%)
 - Much less tachyphylaxis

Glaucoma Meds

- *Prostaglandin analogues/hypotensive lipids*
 - ✓ 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

- ✓ Parasympathomimetics (miotics)
 - Three types:
 - Direct cholinergic agonists (Pilocarpine)
 - Indirect-acting anti-AChE agents (not often used)
 - Mixed (direct and indirect) agents (Carbachol)
 - Rarely used

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 Surgery

- Incisional Surgery
 - ✓ Trabeculectomy
 - ✓ Tube Shunt
- Angle Surgery
 - ✓ Trabectome
 - ✓ Canaloplasty

Glaucoma Lasers

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