Optometric Education Consultants

The Spectrum of Angle Closure Glaucoma

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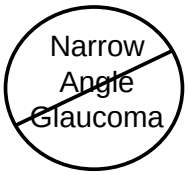
Angle Closure Glaucoma

- A severe type of glaucoma caused by the apposition of the iris to the trabecular meshwork, obstructing the outflow of aqueous, due to forces acting at four successive anatomic levels: the iris (pupillary block), the ciliary body (plateau iris), the lens (phacomorphic glaucoma), and vectors posterior to the lens (malignant glaucoma).

Polling question 1: The patient's IOP is 28 and gonioscopically you see no structures for 180^0 and posterior TM for 180^0. Diagnosis?

- Open angle glaucoma
- Closed angle glaucoma
- Narrow angle glaucoma
- I'm not sure. That's why I'm here.



Current Terminology

- Primary angle closure suspect
- Primary angle closure
- Primary angle closure glaucoma
- Primary angle closure attack

Primary Angle Closure Suspect

- Pigmented trabecular meshwork blocked by iris
  - Extent of blockage not clear
- No PAS
- Disc and IOP normal
- Probe for symptoms of intermittent closure
- Not clear if LPI or observation is better

Primary Angle Closure

- Pigmented TM is blocked by iris for 180^0
- Have either PAS or elevated IOP
- No disc damage or field loss
- Considered pathologic
- LPI recommended

Primary Angle Closure Glaucoma

- Pigmented TM is blocked by iris for 180^0
- Have either PAS or elevated IOP
- Glaucomatous neuropathy and field loss
- LPI recommended

Primary Angle Closure Attack

- Near complete apposition of iris to pigmented TM
- Classic signs and symptoms
  - Injection, vision loss, nausea, emesis, halos, corneal edema, elevated IOP, inflammation, mid-dilated fixed pupil
- Medical therapy, iridotomy, iridoplasty, trabeculectomy
  - Lens extraction?

Name	Irido-trabecular contact (> 180)	Increased IOP	PAS	GON	Acute Attack
PACS	+	-	-	-	-
PAC (CAC)	+	+/-	+/-		-
PACG (CACG)	+	+/-	+/-	+	-
AAC	+	+	+/-	+/-	+

UCLA Stein Eye Institute | DOHENY EYE INSTITUTE Emanual, Parrish, Gedde; 2014

Angle Closure Glaucoma

- Second leading cause of blindness worldwide
- Vision loss more common in ACG than POAG
- 7 million blind from glaucoma worldwide
  - 4 million blind from ACG
- More malignant
- By 2020, PACG will affect 20 million people, and 5.3 million will be blind.

Angle Closure Glaucoma: Patient Profile

- White > Black
  - Angle closure is uncommon in patients of African descent
- Asian: ACG > POAG
- Females > males
- Older > younger
- Hyperopes > myopes
- Inuit population has the highest incidence of angle closure

Classes of Angle Closure

- Primary angle closure glaucoma (ACG) with pupil block
  - Acute
  - Subacute (intermittent)
  - Chronic
- Primary angle closure without pupil block
  - Plateau iris syndrome
- Secondary angle closure with pupil block
- Secondary angle closure without pupil block

Classes of Angle Closure

- Primary angle closure glaucoma (ACG) with pupil block
  - **Acute**
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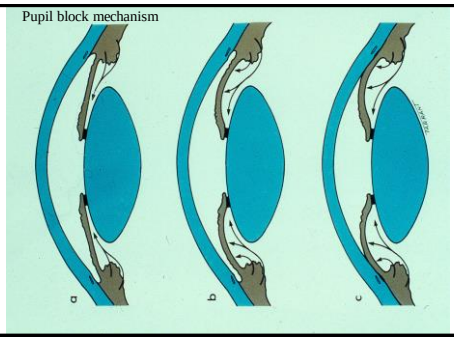
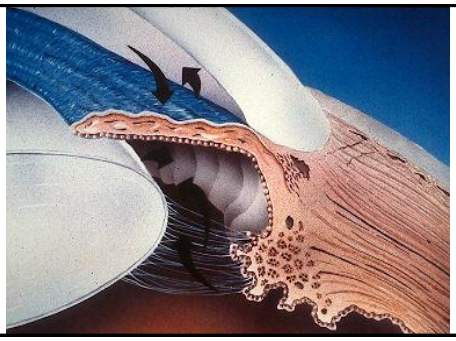
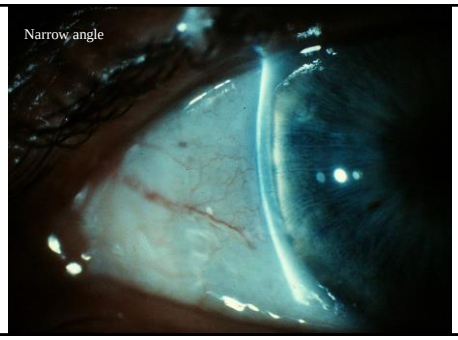
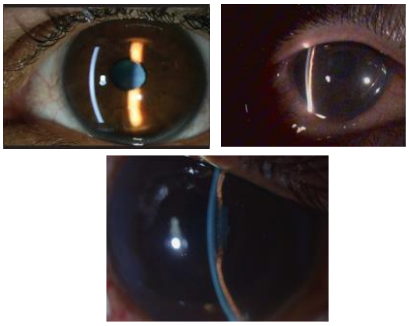
Primary Angle Closure With Pupil Block

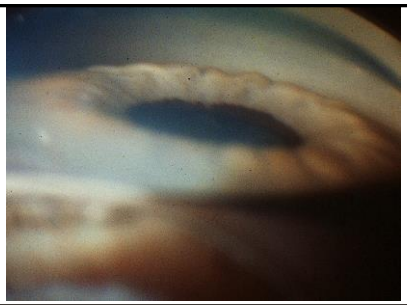
- Irido-lenticular apposition
- Mid dilated state causes most problems
  - Pupil block is normal physiology
  - Some develop a pathological problem
- Absent egress of aqueous to anterior chamber
- Pressure buildup
- Iris bombé: bowing forward of iris due to posterior pressure buildup.
- Irido-corneal apposition
- Closure of angle

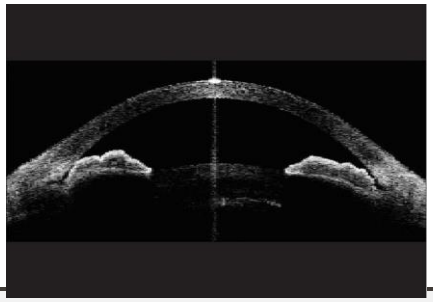


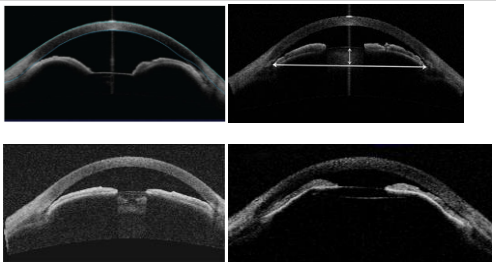












Primary Angle Closure With Pupil Block

- Permanent synechial closure if contact remains too long
- Miosis has long been the standard to pull the iris out of the angle, but anything that alleviates the irido-lenticular apposition will benefit.
  - Very few doctors will dilate a patient in angle closure
  - Atropine may be a medication of choice in some cases

Primary Angle Closure With Pupil Block

- IOP rise (40-70 mm hg or higher)
  - Possible vascular occlusion due to elevated IOP?
- Peripheral anterior synechiae (PAS) formation
  - Permanent

Primary Angle Closure With Pupil Block

- Anatomic features:
  - Small corneal diameter
  - Small axial length
    - Axial length 5% shorter
  - Moderate hyperopia
  - Thick lens
    - 7% thicker
  - Greater propensity for lens to move forward

Primary Angle Closure With Pupil Block

- Shallow anterior chamber
  - AC depth > 2.5 mm-almost never see ACG
  - AC depth 2.0-2.5 mm-sometimes see ACG
  - AC depth < 2.0 mm-frequently see ACG
  - 75% of ACG has AC depth < 1.5 mm
- Anterior chamber 24% shallower than controls
- Anterior chamber volume 37% less than controls

Primary Angle Closure Glaucoma: Classes

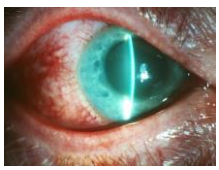
- Acute
- Subacute
- Chronic

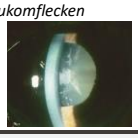
Acute Primary Angle Closure Glaucoma: Symptoms

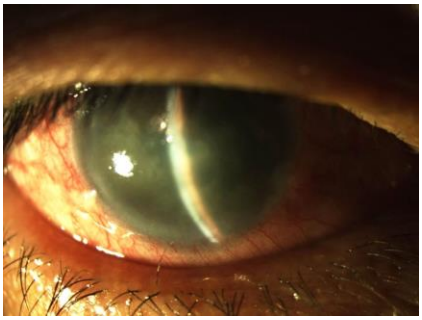
- Red eye
- Photophobic
- Halos- corneal edema
- Blurred vision
- Nausea/emesis

Acute Primary Angle Closure Glaucoma: Signs

- Elevated IOP
- Mid-dilated pupil
- Mild anterior chamber reaction
- Cloudy, corneal edema
- *Glaukomflecken*







Acute Primary Angle Closure Glaucoma: Acute

- Entire angle involved
- Vision loss in days
- Profound si/sx
- IOP 45 mm Hg (or greater) common
- Corneal edema
- PAS forms quickly
  - May result in chronic IOP elevation after breaking attack and curing angle closure due to TM damage.

Acute Primary Angle Closure Glaucoma: Management

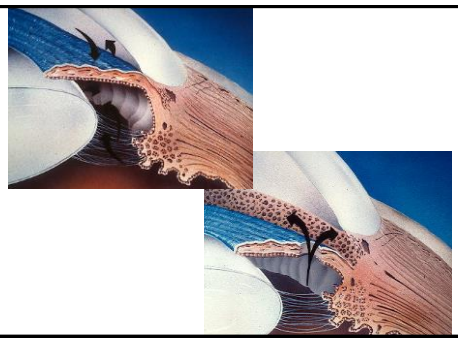
- Beta blocker (i gtt, no more than 2 drops)
- Pilocarpine 2% (if IOP < 40 mm hg).
  - Be sure that your diagnosis is correct before you start pouring pilocarpine into the patient's eye
  - This is becoming a dangerous strategy because too many doctors have a knee-jerk reaction to elevated IOP and pour in the pilocarpine when the diagnosis isn't even angle closure.
  - Concentrations greater than 2% should be avoided as it may increase ciliary body congestion and lead to greater pupil block

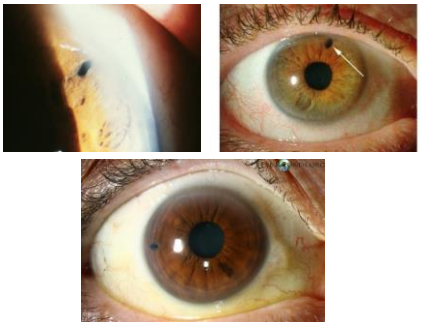
Acute Primary Angle Closure Glaucoma: Management

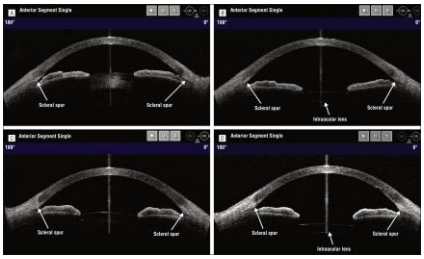
- Iopidine vs Alphagan
- Pred forte (or other steroid) 2-3 times
- PGA works too slow
- CAI
  - Diamox 500 mg
  - Topical may be used if oral not available or contraindicated.
- Oral osmotics?

Acute Primary Angle Closure Glaucoma: Management

- Laser PI
- Argon laser iridoplasty
- Trabeculectomy and tubes
- Iridotomy or trabeculectomy
  - High incidence of flat chambers after trab
  - Iris and lens move forward
- Phaco lens removal
  - After attack broken







Primary Angle Closure Glaucoma: Subacute

- Recurrent attacks
- Subsides spontaneously
  - Opens during sleep with miosis
- Lesser symptoms
  - Episodic blurred vision & halos
- Partial angle closure
  - PAS, particularly superiorly
- Cataract
- Incorrectly called narrow angle glaucoma
  - Angle chronically narrow
- Incorrectly confused with NTG or POAG- do gonio

Primary Subacute Angle Closure Glaucoma: Management

- Laser PI or lens removal
- Long term medical management alone not appropriate
  - False security-allows PAS to form
- Trab or tube surgery
  - Again, risk of malignant glaucoma

59-year-old asymptomatic white female

- Presents with complaints of mildly blurred vision, mild discomfort in both eyes
- BVA: OD 20/20, OS 20/20
- 38 mm Hg OD, 42 mm Hg OS
- Slit lamp: extremely narrow angles OU
  - Gonioscopy: Bare TM noted inferiorly only OU

Polling question 2: What is this patient's diagnosis?

- Open angle glaucoma
- Acute closed angle glaucoma
- Chronic angle closure glaucoma
- Narrow angle glaucoma
- I'm not sure. That's why I'm here.

Primary Angle Closure Glaucoma: Chronic

- Most common form of primary angle closure glaucoma
  - 80% of PACG is chronic, 20% acute
- Asymptomatic
- PAS - zippering shut of angle, especially superior angle
- Discovered on routine exam
- Mistaken for POAG- do gonio

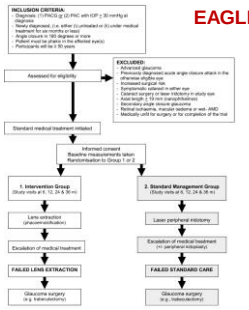
Primary Angle Closure Glaucoma: Chronic

- Multimechanism: The pattern of angle closure appears to mainly be creeping closure.
  - Pupillary block: caused by iris bombé due to pupillary block with acute or subacute attack.
  - Non-pupillary blocking: usually have a deeper anterior chamber, and tend to be younger (below 40 years of age).
- Angle closure in this form of chronic ACG is caused by: iris crowding mechanism or/and anteriorly positioned ciliary body against iris root to angle.

Primary Angle Closure Glaucoma: Chronic

- Iridotomy first, then filtering surgery if not controlled
- Often will require topical medications following iridotomy to control IOP
- Stable visual fields and good long term IOP control were seen in 90% of chronic primary angle closure glaucoma eyes on medical/surgical therapy
- Lensectomy is being investigated as a primary treatment for chronic angle closure glaucoma and may become the preferred treatment. Currently a very viable option.
  - The role of clear lensectomy (i.e. extraction of the non-cataractous lens) in patients with PCACG is unclear. The Effectiveness in Angle Closure Glaucoma of Lens Extraction (EAGLE) study, a prospective, randomized clinical trial now underway, will compare the safety and effectiveness of LPI and medical therapy to clear lens extraction for patients with newly diagnosed PCACG.

EAGLE Study



EAGLE Study

- Removal of clear lenses in eyes with PACG with IOP > 21 mm or eyes with PAC (without glaucoma) and IOP > 30 mm. Findings included:
- Patients undergoing phaco lens extraction had far fewer IOP controlling meds
- Only 1 patient needed trabeculectomy after phaco whereas 24 patients in the LPI group needed trabeculectomy
- Far fewer meds after phaco than LPI

Azuara-Blanco A, Burr JM, Cochran C, et al. Effectiveness in Angle-closure Glaucoma of Lens Extraction (EAGLE) Study Group. The effectiveness of early lens extraction with intraocular lens implantation for the treatment of primary angle -closure glaucoma. (EAGLE):The Lancet. Volume 388, No. 10052, p1389–1397, 1 October 2016.

Surgical treatment for PACG

- Phaco-goniosynecialysis
  - Lens is removed and PAS is surgically cut open
    - May be as effective as traditional glaucoma surgery (trabeculectomy) but with fewer complications. Avoids malignant glaucoma
    - Attempts to restore function to a previously closed angle
    - Factors determining whether or not PAS release will lower IOP include duration of angle closure, extent of PAS, function of proximal and distal outflow pathways prior to closure, secondary functional changes to trabecular meshwork and Schlemm's canal following closure
  - Theoretically goniosynechialysis makes sense, but few actually perform the procedure

Polling question 3: Can you dilate a patient with chronic angle closure?

- Yes
- No
- I'm not sure. That's why I'm here

Case

- Patient with CACG undergoes LPI successfully
- Angles open to scleral spur OU
- IOP drops to 19 mm Hg w/o meds
- At 3 mos F/U – IOP 27 mm Hg OU
- At 6 mos F/U – IOP 35 mm Hg OU

What's happening here?

Polling question 4: What is happening with this patient?

- The LPI is not patent.
- The angle closed again.
- The patient has 'mixed-mechanism' glaucoma
- None of the above
- I'm not sure. That's why I'm here.

Clinical Pearl:

After successful laser treatment for angle closure glaucoma, the IOP may still be elevated. The cause typically is compromise (damage) to the angle structures (meshwork) from the angle closure. Many doctors don't realize this and use the term; "mixed mechanism" glaucoma to denote a case where the patient has been successfully treated with laser for angle closure, yet still has elevated IOP. The doctor who uses the term, "mixed mechanism glaucoma" is really saying, "The pressure is high and I don't know why".

Primary Angle Closure Glaucoma: Prophylaxis

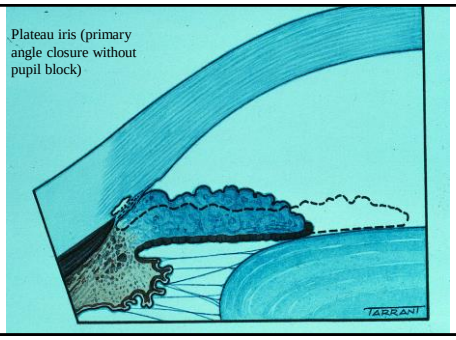
- Laser PI
  - Especially if AC < 2.0 mm
- Gonio to identify areas of reversible closure
- Gonio is actually poor predictor of risk of ACG
  - Static view of dynamic phenomenon
- 10 people have small eyes – 1 will develop ACG
  - Factors other than small size involved
    - Choriodal expansion
- Iridotomy speeds cataract

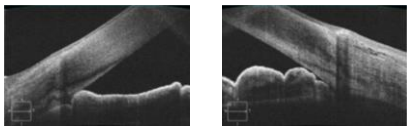
Classes of Angle Closure

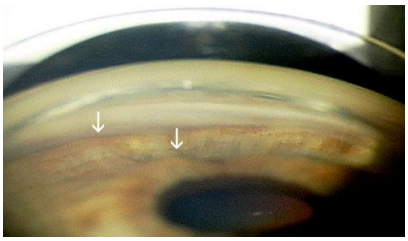
- Primary angle closure glaucoma (ACG) with pupil block
  - Acute
  - Subacute (intermittent)
  - Chronic
- Primary angle closure without pupil block
  - **Plateau iris syndrome**
- Secondary angle closure with pupil block
- Secondary angle closure without pupil block

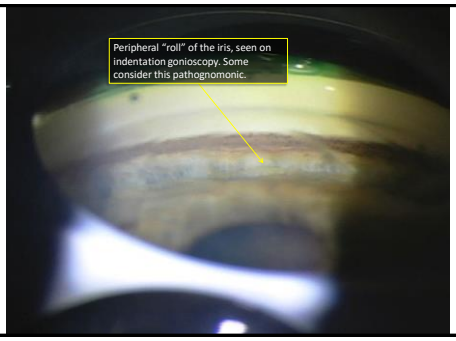
Primary Angle Closure Without Pupil Block

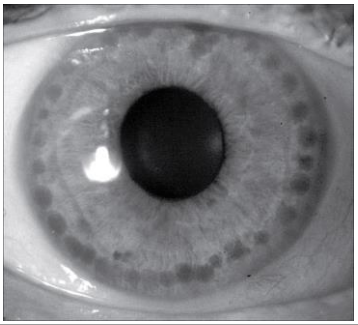
- Plateau iris
  - Gonioscopic description of any eye with deep anterior chamber and narrow angle due to large last role of the iris
- Laser PI
  - Occasionally helps, but mechanism unknown
- Argon laser iridoplasty
- Affected by dilation-crowds angle
- Permanent PAS may form

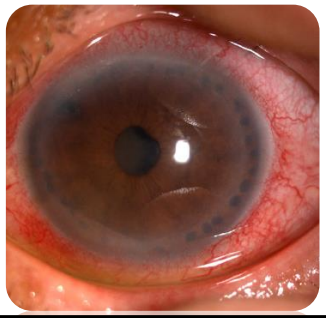












ZAP study

Design and methodology of a randomized controlled trial of laser iridotomy for the prevention of angle closure in southern China: the Zhongshan angle Closure Prevention trial.

Jiang Y, Friedman DS, Ha M, Huang S, Kong X, Foster PJ

Author information

ABSTRACT

PURPOSE: To summarize the design and methodology of a large-scale trial in southern China, the Zhongshan Angle Closure Prevention (ZAP) trial. This trial will determine if laser iridotomy (LI) is superior to no treatment for managing Chinese people who are Primary Angle Closure Suspects (PACS). In this trial, PACS was defined as having 6 or more clock hours of angle circumference in which the pigmented trabecular meshwork was not visible under static gonioscopy in both eyes without elevated intraocular pressure, peripheral anterior synechiae or glaucomatous neuropathy.

METHODS: Subjects were recruited from an urban district in Guangzhou. The target sample size was 870. Persons 50 years of age and older with 2Q-4Q or better vision in both eyes identified as having 6 or more clock hours of angle circumference in which the pigmented trabecular meshwork was not visible under static gonioscopy in both eyes were enrolled. Each subject was randomized to undergo LI in one eye with the fellow eye left untreated. Follow up is planned for a minimum period of 3 years. Baseline examination included tonometry, limbal chamber depth grading, gonioscopy, fundus photography, anterior segment coherence tomography, ultrasound A scan, ultrasound biomicroscopy, specular microscopy, and dark room provocative testing. Endpoints for the study include developing elevated intraocular pressure, peripheral anterior synechiae or experiencing acute primary angle closure.

CONCLUSION: The ZAP trial will determine if LI is safe and effective at preventing pathological angle closure in asymptomatic eyes with narrow angle configurations on gonioscopy. It will also provide data on what happens to untreated eyes in PACSs. Data collected at baseline will also help identify those at high risk for developing primary angle closure and primary angle closure glaucoma.

ZAP results

- 889 angle closure suspects
  - One eye received LPI and the other observation
- Outcomes at 72 months:
  - IOP > 24 mm; development of at least 1 clock hour of PAS, or acute attack.
- Results:
  - Outcome in 4.19 per 1000 eyes/yr in treated and 7.97 per 1000 eyes/yr (19 treated eyes and 36 untreated eyes)
  - Prophylactic LPI statistically significantly reduced incidence of ACG, but the actual event was very infrequent and hard to justify widespread use.
  - Very low rate of angle closure in suspect eyes (<1%/yr); prophylactic LPI did confer 47% risk reduction

The impact of pharmacological dilation on intraocular pressure in primary angle closure suspects

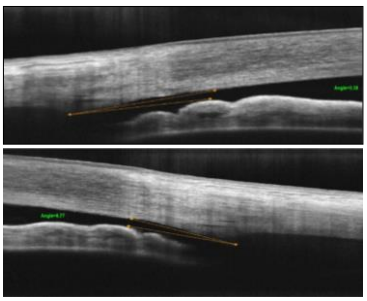
Conclusions: Post-dilation IOP elevation is similar among treated and untreated eyes, and the risk of developing AAC is very low even among PACS. Routine LPI before pupil dilation for PACS people is not recommended.

ZAP 14-year results

- Approximately 43% in each group lost to follow up
- 33 LPI-treated eyes and 105 control fellow eyes reached an endpoint (AAC attack or PAC- elevated IOP or PAS)
- 12 eyes had true disease (AAC or PACG)
  - AAC- 1 LPI treated eye; 5 control fellow eyes
  - PACG- 2 LPI-treated eyes; 4 control fellow eyes
- LPI treated eyes had worse cataract, higher IOP, and greated chamber depth.
- Eyes at most risk post LPI as those that still had persistent angle narrowing detected by anterior segment optical coherence tomography (AS-OCT) or gonioscopy. This emphasizes the concept that LPI is not always an angle-closure panacea and that eyes may still be at risk even after the procedure.

74 YOF

- CC: Blurred vision OU
- BVA: +5.25-1.75x145 20/60; +5.50-0.25x45 20/20
- PERRL(-)RAPD
- Nuclear sclerotic cataracts OD>OS
- IOP 30 mm OD, 25 mm OS
- Narrow angles
- Gonio: No structures OD; ATM nasal and temporal OS-otherwise no structures seen
- Fundus: no view undilated



Assessment and Plan?

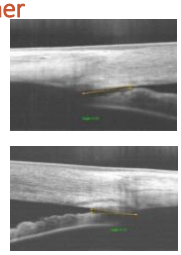
74 YOF

- Diagnosis: Primary chronic angle closure (glaucoma?)
- Plan: sampled PGA and set for cataract consult
- IOP at consult: 17 mm OD, OS
- Surgical measurements made (no dilation)- planned cataract extraction basic emme OD, then OS; CPM
- Pt cancelled surgery twice- reasons unknown.

You can lead an angle closure to osmoglyn, but you can't make him drink

Backed into a closure corner

- 30 YOF
- 2018: Referred for narrow angles
- BVA: +2.00 DS 20/20; +1.25 DS 20/20
- Gonio: "slit OU" Grade 1 OU
- IOP 18 mm OU
- Dx: PACS OU
- Plan LPI OU



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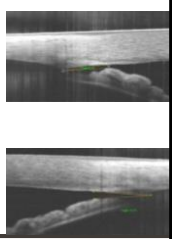
- Follow up (2018)
- No appreciable change after LPI
- Gonio: grade 1; no PAS, double hump sign
- Dx: plateau iris syndrome
- Plan: Discussion iridoplasty, pilocarpine, lens extraction
- Observation recommended
- Other glaucoma specialists may have different approach
  - welcome to second opinion
- Do not start any new medication without clearance
  - Cold and allergy meds



Backed into a closure corner

- 2022: Emergently presents with migraine aura
- Records reviewed
- No resolution to issue
- Forgot about the medication admonition
- Has been told that she can never be dilated
- She is worried and doesn't know what to do
- So, what do we do?

Backed into a closure corner

- Can this 30 YO go the rest of her life without dilation?
- Really no great options (Pilo? Iridoplasty? Lens extraction at 30 years old?)
- Hasn't had an attack yet
- Harry Quigley, MD, "*You just don't know, so sometimes you gotta bite the bullet, dilate, and see what happens. But you don't do it on Friday at 4 pm. You do it Friday at 9 am and tell them that they will be here until lunch time*"

Backed into a closure corner

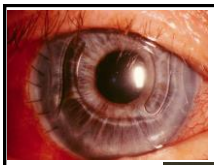
- Returns 8:30 am Tuesday
- IOP: 22 mm OD, 22 mm at 8:30 am; pt informed of risks; dilated 0.5% tropicamide
  - Diamox and Combigan ready
    - It works- trust me
- IOP: 22 mm OD, 22 mm OS at 9:30 am
- IOP: 22 mm OD, 23 mm OS at 1:15 pm; pupil in mid-dilated state
- Fundus normal OU; C/D 0.2 OU
- Pt educated si/sx AACG
- Will follow annually

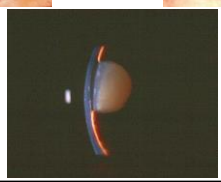
Classes of Angle Closure

- Primary angle closure glaucoma (ACG) with pupil block
  - Acute
  - Subacute (intermittent)
  - Chronic
- Primary angle closure without pupil block
  - Plateau iris syndrome
- Secondary angle closure with pupil block
- Secondary angle closure without pupil block

Secondary Angle Closure With Pupil Block

- Uveitic
- Phacomorphic
- Aphakia
- Vitreous prolapse
- Pseudophakia
- Reverse pupil block with AC lens
- Subluxated lens

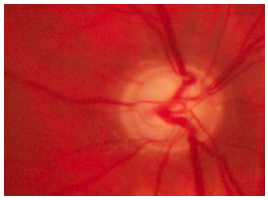
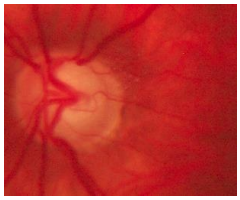




The Case of the Non-Routine Routine Eye Exam

- 50 YOM- CEE
- 20/40 OD, OS
- Rx: (-) 18.00 – 2.50 x 180 OU
- IOP: 42 mm Hg OU
- Constricted visual fields and advanced glaucomatous disc damage OU

What Next?

Polling question 5: What is the diagnosis?

- Primary angle closure
- Open angle glaucoma
- Phacomorphic glaucoma
- Some weird syndrome that I learned in school but can't remember right now.
- I'm not sure. That's why I'm here.

The Case of the Non-Routine Routine Eye Exam

- Gonioscopy: Chronic angle closure OU
- Non-myopic fundus
- Lens protrudes slightly into A/C
- Diagnosis: Chronic angle closure secondary to phacomorphic glaucoma secondary to isolated microspherophakia
- Management: LPI OU followed by topical glaucoma meds
  - Then things got complicated…

Phacomorphic Glaucoma

- Phaco=lens; morph=shape
- Secondary angle closure with pupil block
- Most common lens-induced glaucoma
- Unilateral or asymmetric cataract associated with asymmetric shallowing of the anterior chamber not explained by other factors
- Difficult to differentiate from primary angle closure
- Acute to intermittent red, painful eye, typically at night
- May present asymptomatically with chronic angle closure

Phacomorphic Glaucoma

- Typically, vision is greatly reduced (<20/400) from the cataract
- Due to increasing lens thickness: irido-lenticular apposition from growth of the lens cortex and intumescence of the lens.
- May be associated with short globe axial length
- Occasionally, phacomorphic glaucoma will occur not due to mature cataract formation, but due to microspherophakia (often associated with Weill-Marchesani syndrome)
  - Presents as acute or chronic angle closure in eyes with high myopia.

Phacomorphic Glaucoma

- Beta-blockers, alpha-2 adrenergic agonists, topical corticosteroids, topical or oral carbonic anhydrase inhibitors may be all systematically employed.
- An exceptional effect of prostaglandin analogs in managing the IOP of patients with chronic angle closure glaucoma both before and following LPI has been reported.
- Pilocarpine 2% and corticosteroids can also be used.
- RhoKinase inhibitors questionable.
- LPI
- Lens extraction

Classes of Angle Closure

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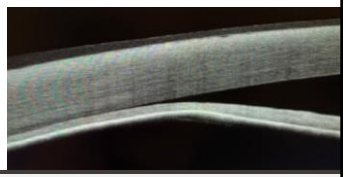
- Secondary angle closure with pupil block
- Secondary angle closure without pupil block

Secondary Angle Closure Without Pupil Block

- Either the peripheral iris is pulled or pushed into the cornea.
  - Drug induced choroidal expansion
  - Neovascular glaucoma
  - Neoplastic disease
  - Some inflammatory cases/ uveitic
  - Ciliary block

Glaucoma Upgrade

- 76 YOM
- Just released from hospital
  - BP 220/140
- Had Xen < 2 weeks ago
  - PXE glaucoma
  - On Durezol, moxi
- Painful OD
- Fixed mid-dilated pupil
- IOP 60 mm



Polling question 6: What is the genesis of the problem?

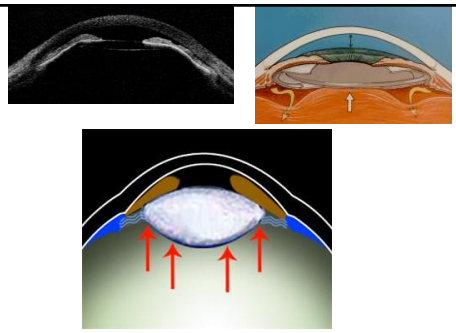
- IOP elevation due to malignant hypertension
- Pupil block angle closure
- Steroid induced pressure elevation from Durezol
- Malignant glaucoma
- I'm not sure. That's why I'm here.

Ciliary Block Glaucoma

- Also known as malignant glaucoma and aqueous misdirection syndrome
- Anterior displacement of the iris and ciliary body
- Due in part to swelling of the ciliary body, annular detachment of the ciliary body, and anterior vitreous displacement.
- Disparity in AC depth between eyes
- Miotics will aggravate, or even cause this condition
- No benefit from iridotomy

Ciliary Block Glaucoma

- Frequently caused by misdirection of aqueous into the vitreous and posterior chamber with anterior displacement of structures of the eye. Abnormally impermeable vitreous face. This most typically occurs following ocular surgery for angle closure glaucoma. Can occur immediately post-op or much later.
- Unresponsive to conventional medical and surgical glaucoma therapies

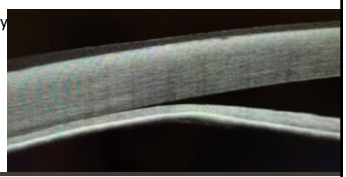


Ciliary Block Glaucoma

- Best managed with atropine 1% BID, phenylephrine 10% BID, and acetazolamide 1 gm PO QD. Eventually, everything but atropine is tapered (atropine is continued indefinitely).
- Surgical options:
  - Vitrectomy and lensectomy
  - Nd:YAG disruption of capsule

Malignat glaucoma: This patient

- Kept on meds
- Added topical IOP meds
- Added Diamox 1000 mg/day
- 24- hours- IOP 32 mm
- Underwent PPV
- Vision improved
- IOP 17 mm

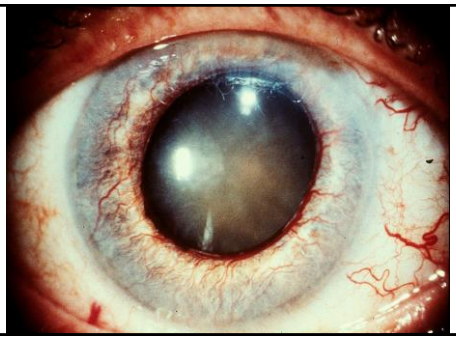


Neovascular Glaucoma

- Neovascularization of the iris and angle
- Causes
  - CRVO
  - Diabetic retinopathy
  - Carotid artery disease (ocular ischemic syndrome)
  - BRVO, HRVO
  - CRAO
  - Giant cell arteritis

Neovascular Glaucoma Pathophysiology

- Hypoxia
- Vasoproliferative substance diffuses to viable tissue
- Neovascularization
- Rubeosis
- Angle neovascularization
- Vessels bridge scleral spur and arborize on trabecular meshwork
- Fibrovascular membranes



Neovascular Glaucoma Pathophysiology

- Synechial closure of angle
  - Tent-like PAS initially, later broad areas of angle closure
- Inflammation and high IOP
- Poor prognosis
- Poorly responsive to medical treatment
- Mechanism is secondary angle closure without pupil block

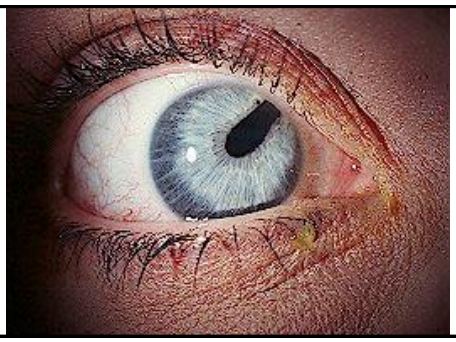
Neovascular Glaucoma Management

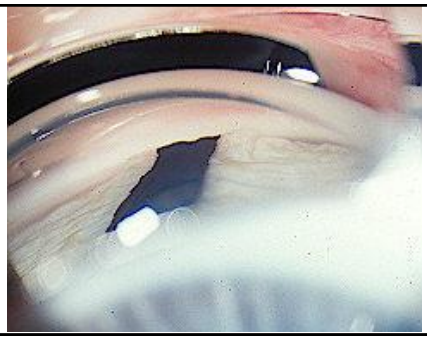
- Medical TX: atropine and Pred forte used for inflammatory component. May also temporarily use aqueous suppressants.
  - Generally, you do not medically treat this type of glaucoma.
- Trabeculectomy if not too much of the angle is compromised
- Anti-VEGF injections
- Pan-retinal photocoagulation (PRP) to destroy the ischemic retina and reduce the vasoproliferative substance and induce regression of neovascular vessels.

"What's wrong with your eye?"

A 28 year old white female presents emergently following a co-worker's remark about a grossly visible ocular abnormality. The patient reports that previous exams have been normal, and she feels that her eyes and vision have always been normal. There is no history of trauma. She had not noticed anything recently.

- BVA is 20/15 OD, OS.
  - Confrontation fields and EOM's are normal and her pupils react to light without afferent defect.
  - IOP is 15 mm Hg OU and discs are 0.4/0.4
  - However, the right pupil is misshapen



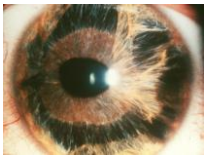


ICE Syndromes

- Iridocorneal endothelial syndromes (ICE): syndromes where corneal endothelial cells over-secrete leading to Descemet's membrane migrating and extending over the trabecular meshwork. As this membrane contracts, PAS forms. Theses are typically unilateral and more commonly affect women.

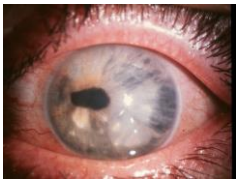
ICE Syndromes

- Essential iris atrophy- gonioscopy shows progressive angle closure by PAS. The pupil is displaced towards the PAS and the iris shows mild-to-moderate ectropion uveae, stromal atrophy, and full thickness iris hole formation opposite the PAS



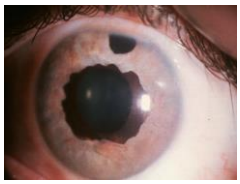
ICE Syndromes

- Chandler's syndrome- changes in the iris are mild to absent while corneal edema presents at normal IOP level



ICE Syndromes

- Iris nevus (Cogan-Reese) syndrome- the angle changes are the same as in essential iris atrophy, except that an iris nevus covers the anterior iris



ICE Syndromes

- Treatments include filtering surgery and often penetrating keratectomy. Medical therapy tends to work for a short while and LTP is ineffective
- Frankly may never develop glaucoma

ACG: Role of the Choroid

- Choroid expansion likely contributes heavily to ACG
- Choroidal expansion takes up volume
- Choroidal expansion by 20% takes up 96 uL
  - Normal anterior chamber is only 100 uL
  - IOP from 20 mm Hg to 60 mm Hg instantaneously

ACG: Role of the Choroid

- Choroidal expansion leads to forward movement of lens and iris and chamber flattening with ACG
- Change in choroidal vessel permeability leads to choroidal expansion
  - Proteins, fluid, blood moves into extracellular/ extravascular space in choroid

Factors Affecting Choroid

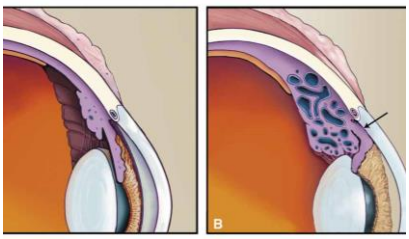
- Associated with ACG in otherwise normal eyes:
  - Hypotony
    - Choroidal detachment
  - Carotid Cavernous Sinus Fistula
  - Suprachoroidal hemorrhage
  - Scleritis
  - Choroidal tumors
  - PRP
  - Drug-induced choroidal effusions (sulfa based)

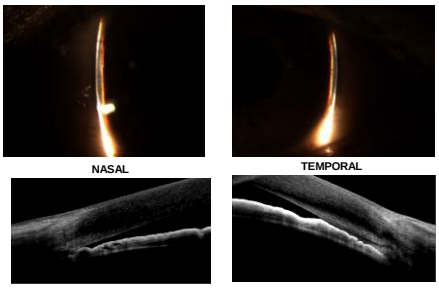
Drugs Causing ACG

- Acetazolamide
- Hydrochlorothiazide
- Trimethoprim-sulfamethoxazole
- Indapamide
- Promethazine
- Spironolactone
- Isosorbide dinitrate
- Viagra
- Bromocriptine
- Tetracycline
- Corticosteroids
- Penicillamine
- Quinine
- Metronidazole
- Isotretinoin
- Aspirin
- *Topiramate**

Ciliary Effusion

- Anterior Rotation of the Ciliary Body
  - Reduces tension on the zonules
    - Lens Thickening
      - Induces myopia
  - Iris-Lens diaphragm shifts anteriorly
    - Induces myopia by changing effectivity
  - Shallowing of Anterior Chamber
    - Potential for angle closure





Case

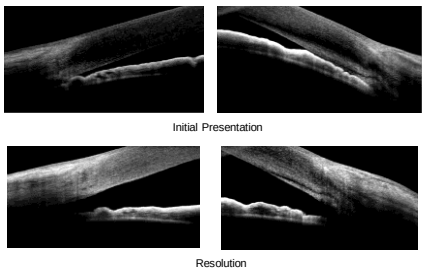
- 39 YOF
- Recently started on Topamax for migraine
- Sudden onset blurred vision and eye pain
- Formerly emmetropic, now (-) 6.00 DS
- IOP 44 mm Hg

Polling question 7: What is the best management?

- Cycloplegic and topical steroids
- Oral Diamox
- Cosopt and Lumigan
- Immediate LPI
- I'm not sure. That's why I'm here.

Case

- 39 YOF
- Recently started on Topamax for migraine
- Sudden onset blurred vision and eye pain
- Formerly emmetropic, now (-) 6.00 DS
- IOP 44 mm Hg
- D/C Topamax; add PF Q1H, scopolamine BID, beta blocker BID

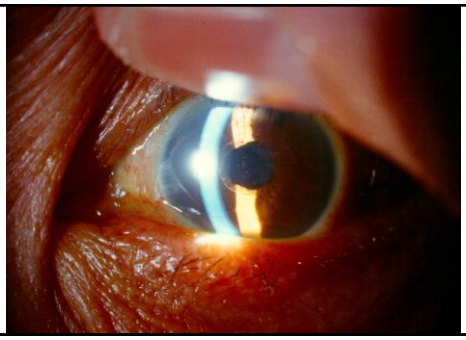


Choroidal Involvement in ACG

- Drug-induced choroidal expansion
- Choroidal expansion in ACG associated with shallowing of chamber
- Malignant glaucoma may not be aqueous misdirection, but poor fluid permeability and choroidal expansion
- Atropine may work by moving ciliary body and improving forward diffusional area for fluid
  - Atropine may be a better choice than pilocarpine

Case: *The fist and the eye*

- 75 YOAM: medical hx unremarkable
- (+) Pseudophakia OS
  - Previously examined and fine
- Struck accidentally by grandchild
- Presents 2 weeks later: (+) pain/photophobia/lacrimation/redness
- IOP 35 mm OS (17 mm hg OD)



Case: *The fist and the eye*

- Diagnosis: vitreous prolapse with pupil block and secondary angle closure
- Management:
  - Tropicamide 1%, phenylephrine 2.5%
  - 30 minutes:
    - Angle opens
    - Chamber deepens
    - IOP 22 mm Hg
  - Plan: Homatropine 5%, PF, beta blocker, LPI

POAG and Primary Angle Closure Glaucoma

- Changing angle configuration
- Gonio on a 2-3 year basis on all POAG patients to determine if a conversion from open to closed angle is occurring.
- Laser PI for occludable angles
  - It depends
- Narrow angle glaucoma is an antiquated term. Better to speak in terms of chronic angle closure, etc.

Persistent angle closure after LPI

- Not pupil block mechanism
  - Choroidal effusion, plateau iris, etc
- Significant PAS
- LPI not complete/ patent