Lens-Associated Glaucomas

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Case History 1

- Phone Call from cataract surgeon
  - One month prior to exam, surgeon performed cataract extraction on this eye
  - Dropped lens material during I&A
  - “Positive” no nucleus remained
  - Piece of cortex “extremely small”
  - “Recovered well”, continues painfree
  - One month later, IOP 45 with corneal edema
  - Mild steroid dose, nonsteroidals. No IOP meds.
Case History 1, cont’d

- **Exam**
  - Vision 20/200
  - Conjunctiva white and quiet
  - Cornea = 3+ microcystic edema, no KP
  - AC: AC IOL, same position as intraop; rare cell; no lenticular precipitates
  - Iris: round pupil, patent generous PI, flat surface without bombe
  - No vitreous seen in AC
Case History 1, cont’d

- **Fundus:**
  - Retained cortical lens material
  - No nucleus seen
  - Retina attached
  - Some vitreous cell around remnant

- **Gonio:**
  - IOL well positioned, angle grade IV
  - No PAS, +fluffy white deposit inferior angle
Case History 1: Differential Diagnosis

- Angle closure glaucoma
  - Progressive angle “zip” as in PKP
  - Closed PI due to inflammation

- Angle Recession glaucoma
  - “Eventful” surgery with AC IOL

- Vitreous plugging meshwork
  - As in aphakic glaucoma
Case History 1: Differential Diagnosis 2

- Steroid Response Glaucoma
- Uveitis/glaucoma/hyphema
- Endophthalmitis
- Epithelial Downgrowth
- Sympathetic Ophthalmia
Case 1: Differential Diagnosis 3

- Lens-associated Glaucoma
  - Lens particle
  - Phacolytic
  - Phacoanaphylactic
  - Phacomorphic

- All have clinically obvious findings: lens material, shape, location or inflammation
Case 1: Evolution

- Removal of lens material
- Moderate-term anti-inflammatory treatment
- Gradually-tapering topical antiglaucoma treatment
- Did not need glaucoma surgery
Case 1: Take Home Messages

- Removal of lens material is key
- Hard to separate causes, “got to start somewhere”
- Debulk lens particles + control inflammation = not necessarily glaucomatous for life
- Intervene earlier than later if significant lens material remains and signs of inflammation
Case History: 2

- 67 year old retired bookkeeper, Diabetic with known BDR
- Presented to Urgent Care with 3 day history of red/painful left eye: Prior trabeculectomy 13 yrs prior (pre-antimetabolite era)
- No discharge/exudate
- No known trauma
- + Prior URI symptoms 1 wk prior, now resolving
- IOP had been well controlled without medications
- No outside notes of prior exams for comparison
Case 2 Physical Exam

- Best-Corrected Central Visual Acuity
  - 20/25 R, 20/60 Left

- EOM full, pupils ERRL

- IOP 18, 22

- 2+ Diffuse conjunctival injection, no purulence in tear film, old bleb site scarred flat, no focal injection

- Cornea clear, no infiltrates, no KP

- AC 1+ deep, no cell no flare

- Iris normal

- Lens 3+ Nuclear Sclerosis

- No view posterior pole, did not dilate
Case 2 Initial Treatment

- **Dx:** Viral Conjunctivitis
- **Rx:** Supportive care, return PRN
Case 2 Followup

- One week later, back as urgent care
- No improvement, now additional deep “boring” pain
- Vision was 20/60, now Light Perception
- No Afferent
- IOP 18/44
- Diffusely injected, clear cornea, AC 2+ deep, No cell, 1+ flare, Lens “fluffy material on surface”
Case 2 Next Steps

- Referred to UW
- Findings confirmed with addition of:
  - Fine KP
  - 1+ Cell in AC and 1+ Flare
- Gonio Grade III all quadrants, open trabeculectomy ostomy. No pigment.
- Lens = intumescent with fibrillar deposits on lens capsule and pupil edge
Case 2: Revised Dx & Rx

- Phacolytic Glaucoma
  - Inflammation, lens particles vs foam cells on Lens surface
  - Gonio Open

- Treatment Options:
  1. Immediate lens removal, then quiet inflammation
  2. Quiet inflammation, then remove lens
Case 2: Evolution

- 3 days later
  - IOP 22 on Timolol, Dorz, latanoprost, Pred Forte q2°
  - Much quieter, still flare but no cell, completely comfortable
  - Still LP vision

- 3 weeks later
  - IOP 18 on same meds
  - No inflammation remaining (lens capsule still dusted)
Case 2: Surgical Rx

- Phaco with internal bleb revision
- Huge inflammatory response despite continuing trade name Pred Forte q2° while awake (=8x/day) with AC full of fibrin
- IOP remained low 20s off IOP meds (cup 0.8)
- Followup for 10+ years
  - Bleb failed
  - IOPs upper teens on 2-3 meds
  - Best acuity 20/40, limited by chronic DME despite focal laser x 2.
Case 2: Take Home Messages

- Need to look carefully even if “just” conjunctivitis
  - Applies to eventual diagnosis of angle closure glaucoma, too!!

- Chronic/advanced inflammation + Diabetes:
  - chronic problems, particularly with angle, even if not synechially closed
  - Likely to worsen BDR/DME
Case 3

- 56 year old woman med school grant writer followed for 5-6 years for OHT, without meds, presents d/t insurance change
- 20/20 both eyes with mild hyperopic correction
- IOP 21, 21 (no pachymetry)
- Both eyes: AC “Shallow”; 1+ NS; C:D 0.3
- Referred to G Team
Case 3: Eval

- 20/20 with +1.25 and +1.00 corrections
- HVF full both eyes
- IOPs 22/21 Pachy 593, 598 (= quite thick, IOP lower than msmt)
- Pupils with full excursion, no synechiae, no APD
- AC 1+ by Van Herick, Gonioscopy slit to I all 8 quadrants
- 1+ Nuclear Sclerosis both eyes
- C:D 0.3 both eyes
- Assess: Occludable Angles OU
- Treatment: YAG laser peripheral iridotomy both eyes
Case 3: Discs
Case 3: Evolution

- Post PI, AC deepened to +2 both eyes
- 19 year followup:
  - Cataracts gradually worsened
  - Field Remained Full and normal
  - IOPs fluctuated greatly, required medications and lasers
Case 3: Right Eye Fields
Case 3: Left Eye Fields
Case 3: Fields VFI Plot
Case 3: IOPs
Case 3: Cataracts

- Eventually, both cataracts worsened till became visually significant (Dec 2010)

- Planned Phaco/Trabecs for both eyes, left eye first.

- On the table, phaco was extremely difficult to perform due to shallow AC (Axial length = 22.5 & 22.8mm)

- Once completed and IOL placed, AC *markedly* deeper

- Discussed with pt, chose to stop at phaco and not do trabec
Case 3: Evolution

[Graph showing eye pressure and medication usage over time, with specific dates and labels for medical procedures and measurements.]
Case 3: OS IOP Post Phaco

![Graph showing IOP and Meds OS over time](image-url)
Case 3: OD IOP Post Phaco

IOP OD
Meds OD
Case 3: Take Home Messages

- Not all changes of narrow angles/ anterior segment crowding disappear with iridotomy
- Relief of crowding, even quite “late” in process, can still have incredibly significant improvement in trabecular outflow/IOP
- Parallel: effect of pilocarpine to open meshwork
- Growing literature, particularly in exfoliation patients, of oft-dramatic effect of lens removal on IOP
- You CAN cure glaucoma!!!
Bibliography: IOP reduction after cataract surgery


- **Phacoemulsification versus trabeculectomy in medically uncontrolled chronic angle-closure glaucoma without cataract**; Tham CC, Kwong YY, Baig N, Leung DY, Li FC, Lam DS; Ophthalmology. 2013 Jan; 120(1):62-7

- **Reduction in intraocular pressure after cataract extraction**; the Ocular Hypertension Treatment Study; Mansberger SL, Gordon MO, Jampel H, Bhorade A, Brandt JD, Wilson B, Kass MA; Ocular Hypertension Treatment Study Group; Ophthalmology. 2012 Sep;119(9):1826-31


- Clinical outcomes **after lens extraction for visually significant cataract** in eyes with primary angle closure; Shams PN, Foster PJ; J Glaucoma. 2012 Oct-Nov;21(8):545-50
Differential Diagnosis Keys

- Steroid glaucoma:
  - No physical findings
  - Look in history for other eye, etc

- Angle recession:
  - Need to look very carefully at gonio
  - Ask surgeon about heme intraop
  - Compare to opposite eye
Differential Diagnosis Keys

- Vitreous - associated
  - Look very carefully for vitreous around pupil into AC
  - Should be fairly late process (years)
  - No inflammation
  - Intractable

- Endophthalmitis
  - Inflammation/hypopyon, pain, etc
Differential Diagnosis Keys

- Epithelial Downgrowth
  - Membrane +/- visible
  - Laser to detect membrane
  - History of open wound

- Sympathetic ophthalmia
  - Damage to uvea
  - Delay to symptoms
  - KP/retinitis
Differential Diagnosis Keys

- **UGH**
  - Uveitis: keratic &/or lenticular precipitates
  - Hyphema: Look for RBCs on endo, in angle, on IOL and/or iris
  - Remember: lens usually “propellors” in UGH - careful notes intraop/postop as to IOL position
  - Usually episodic
Phacomorphic Glaucoma

- **Strict Definition**
  - Acutely intumescent lens with swelling sufficient to close the AC

- **Working definition**
  - Slowly progressively growing lens causing shallowing of AC not relieved with PI and not having a plateau configuration
  - Just a Big Lens

- **NO inflammation**
Phacolytic Glaucoma

- Lens Capsule is intact to slit lamp exam
- Lens proteins leak out of capsule “pores”
- Lens proteins = antigens
- Antigens = immune response
- Macrophages “only”
- “Foam cells”
Phacolytic Glaucoma
Phacolytic Glaucoma
Phacoanaphylactic Glaucoma

- Lens capsule ruptured
- Time delay
- Multicellular inflammatory response surrounding lens material clumps
- Granulomatous, intractable
- Multinucleate giant cells may be present
- Key to Dx is DELAY
Phacoanaphylaxis
Lens Particle Glaucoma

- Lens capsule ruptured
- NO Delay
- Lens particles themselves clog meshwork pores
- IOP elevated without any inflammatory response
Lens Particle Glaucoma
Lens-associated Glaucoma: Further Workup

- B scan
  - Vitreous cells
  - Lens particle size/location
  - Retinal integrity

- AC Tap:
  - Lens material (birefringence)
  - Inflammatory cells
  - Culture
Our Case 1:

- Inflammation: yes
- Marked inflammation: no
- Delay to symptoms: yes
- Cortical remnants: yes
- Vitreous in AC: no
- Recession: no
- Hyphema/propelloring: no
- Downgrowth/SO: no
- Steroid use: yes
Treatment of Lens-Induced Glaucomas

- Phacomorphic: lensectomy
- Lens particle: lensectomy
- Phacolytic/phacoanaphylactic:
  - Control Inflammation
  - Control IOP
  - Delay lens removal if possible
  - Remove lens/fragment
  - ?Exchange AC IOL?
Treatment of Lens-Induced Inflammation

- Aggressive topical steroids
- ?Nonsteroidal role?
- Cycloplegia
Topical Treatment of Lens-Induced Glaucoma

- Pick class carefully
  - Prostaglandins: may increase CME and inflammation risk
  - Carbonic Anhydrase Inhibitors: may cause compromised cornea to further decompensate
  - Miotics: may increase inflammation
  - Classes remaining: beta blockers, alpha agonists
  - Add other classes if IOP recalcitrant
Lens-Associated Glaucomas: Take Home

- May be diagnosis of exclusion
- Careful differential steps
  - Especially Gonioscopy !!!!!!!
- Needs to occur to practitioner
- May occur after:
  - Trauma
  - Cataract surgery (= trauma)
  - YAG Capsulotomy
- If circumstances permit “luxury of time”, USE IT!
  - Control IOP/Cells first, then consider procedure
Phacolytic Glaucoma:


Lens Particle Glaucoma: Bibliography


Phacoanaphylactic Glaucoma: Bibliography


Phacomorphic vs \textit{phacomorphic}

- Phacomorphic:
  - Secondary angle-closure glaucoma due to lens intumescence
Phacomorphic vs *phacomorphic*

- *phacomorphic:*
  - Shallowing of anterior chamber due to advancing cataract
  - Same risk factors as primary angle closure
    - Age
    - Short axial length
phacomorphic Glaucoma
Iris Surface Contour: Diagnostic Significance

- Trace the iris contour in a linear section from pupil to iris root

- Where is the “highest” point?
  - At pupil
    - Shallow CENTRAL depth = iris is pushed forward by LENS, contour matches anterior capsular surface
  - At mid point
    - Iris pushed forward by FLUID= pupillary block
  - At iris root
    - Iris pushed forward by ciliary body root= plateau iris
Where’s the highest point?

Midpoint = pupillary block

Pupillary = lens pushed forward
Figure 23.1. Concept of ciliolenticular block as the mechanism of malignant glaucoma. Apposition of ciliary processes to the lens equator (arrows) causes a posterior diversion of aqueous (A), which pools in and behind the vitreous with a forward shift of the lens-iris diaphragm.
Phacolytic with Posterior Synechiae and secondary pupillary block
Aqueous Misdirection
Primary Angle Closure Glaucoma
Plateau Iris

Clinical Differentiation
- High Plateau
- High Lens Rise
  - Central AC depth may be normal or slightly shallow
  - "Double-hump sign"
  - Central AC depth is shallow
  - "Volcano sign"

Imaging Differentiation
- High Plateau
- High Lens Rise
  - Central AC depth is shallow or slightly shallow
  - Plateau angle
  - Anteriorly positioned ciliary processes
  - Large lens
  - Direct anterior lens rotation

Evaluation of Shallow Anterior Chamber

“Shallow” AC: Assess Central Depth (Cornea to Lens)

Center: Relatively Deep
Center: Shallow

Where is most anterior portion of Iris Contour? (gonio and SLE)

Pupillary
Mid Iris
Peripheral iris

Mild Posterior pushing: Iris Follows Anterior Shape of Lens

Source: Mild Posterior Pole mass effect
Source: Lens

Orbital mass
Hemorrhage
Tumor
Trauma/rupture
Cataract

Source: Cataract
Source: Mild Posterior Pole mass effect
Source: Lens
Evaluation of Shallow Anterior Chamber

Shallow AC: Assess Central Depth (cornea to lens)

Center: Relatively Deep

Center: Shallow

Where is most anterior portion of Iris Contour? (gonio and SLE)

Pupillary

Mid Iris: Pupillary Block

Primary Angle Closure: PI

Posterior Synechial Closure

Peripheral iris

Gonio: peripheral ridge: Plateau Iris

Neovascular

Inflammatory

etc
Evaluation of Shallow Anterior Chamber

Shallow AC: Assess Central Depth (cornea to lens)

Center: Relatively Deep = “Cornea Service Shallow”

Center: Shallow, nearly lens-cornea touch = Glaucoma Service Shallow

Severe Posterior Pushing

Lens
- Intumescence

Posterior Pole
- Choroidal (serous vs hemorrhagic)
- Gas bubble
- Tumor