CLINICAL DISCUSSIONS IN GLAUCOMA

JESSICA STEEN, OD
JOSEPH SOWKA, OD

FINANCIAL DISCLOSURES

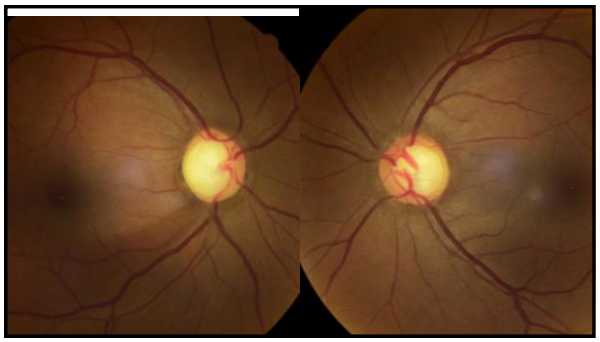
- Jessica Steen- Advisory board: Zeiss, B+L Santen (past 24 months)
- Joseph Sowka- Advisory board/ speaker bureau B+L; Zeiss; Visus (past 24 months)
  - All relevant relationships have been mitigated

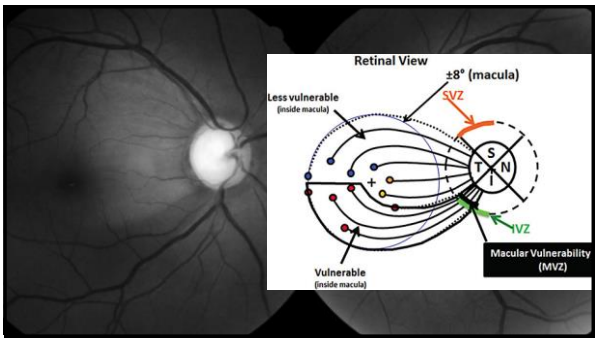
Please text us your questions

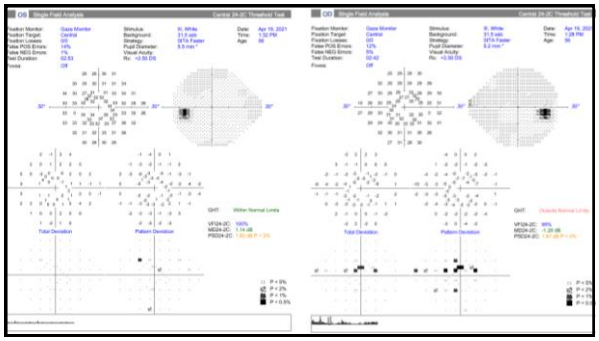
Joe: 954-298-0970

56 YEAR OLD AFRICAN AMERICAN FEMALE

- 56 year old African American female referred for evaluation due to suspicion of glaucoma secondary to optic disc appearance
  - No family history of glaucoma
  - No systemic diagnoses; no systemic medications





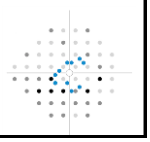


A Comparison of the Visual Field Parameters of SITA Faster and SITA Standard Strategies in Glaucoma

- Removes 'dead time' during the test
- No blind spot, no false negatives
  - Gaze monitoring and false positives
    - Unless you manually adjust settings
- Slightly increased overall threshold sensitivity (is this bad?!)
- More difficult testing situation vs.'positive start bias' of SITA Standard
  - No 'easy' answers
- *Clinically* equivalent to SITA Standard(?)

Lavanya R. et al.A Comparison of the Visual Field Parameters of SITA Faster and SITA Standard Strategies in Glaucoma. J Glaucoma. 2020 Sep;29(9):783–788.

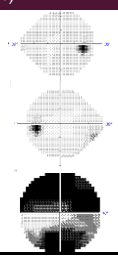
Thulasidas M, Patyal S. Comparison of 24-2 Faster, Fast, and Standard Programs of Swedish Interactive Threshold Algorithm of Humphrey Field Analyzer for Perimetry in Patients With Manifest and Suspect Glaucoma. J Glaucoma. 2020 Nov;29(11):1070–1076.

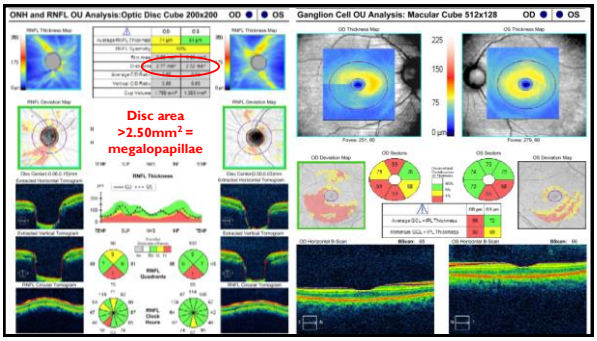


24-2C Testing pattern: an additional 10 points in the paracentral area overlaid on the the 24-2 pattern

QUICK NOTE ON STAGING OF GLAUCOMA (ICD-10)

- **Mild stage**
  - Optic disc abnormality consistent with glaucoma
  - *No visual field abnormality on standard white-on-white automated perimetry*
    - Allows for abnormality on FDT or SWAP
- **Moderate stage**
  - Optic disc abnormality consistent with glaucoma
  - Glaucomatous visual field abnormality in one hemifield not within 5 degrees of fixation
- **Severe stage**
  - Optic disc abnormality consistent with glaucoma
  - Glaucomatous field abnormality in both hemifields and/or loss within 5 degrees of fixation
- **Indeterminate stage**
  - Fields not yet performed;or poor quality fields





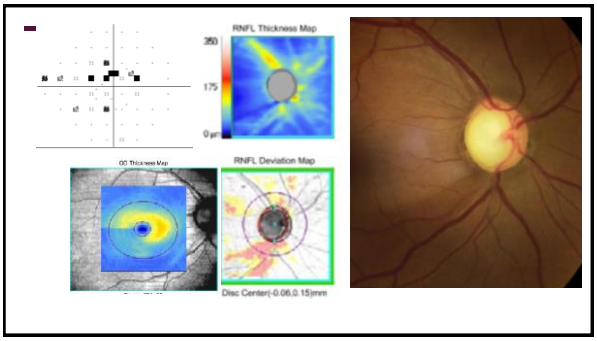
Angles
OD: 4+Nasal: 4+
OS: 4+Temp: 4+

Gonio Examination:

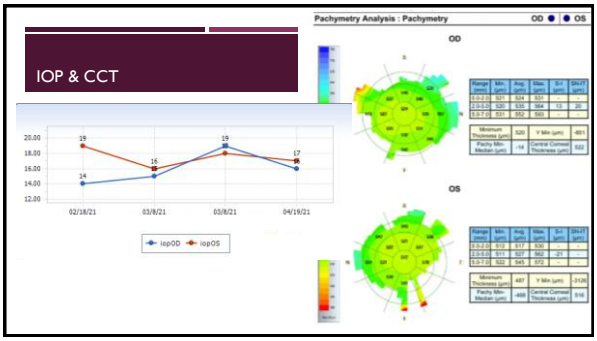
OD
Superior: OD SUP: open to CBB
Nasal: OD NAS: open to CBB
Temporal: OD Temp: open to CBB
Inferior: OD INF: open to CBB

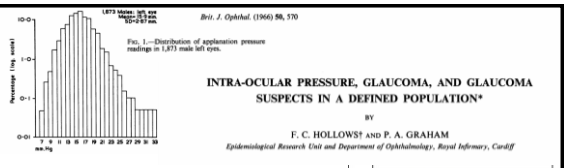
OS
Superior: OS SUP: open to posterior TM
Nasal: OS NAS: open to CBB
Temporal: OS TEMP: open to CBB
Inferior: OS INF: open to CBB

Comment: OD: iris processes temporal
(-) NVA/AR/PAS 360 degrees OD and OS
tr to 1+ TM pigment OD and OS
Flat iris approach OD and OS

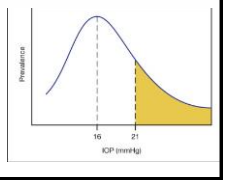


IOP & CCT





- "Average" intraocular pressure is 15-16mmHg (SD = 2.5mmHg)
- "Normal" range 11-21mmHg
  - Based on a population-based study in England of nearly 2000 white males over 40 years of age



- **Diagnosis?**

Impression/Plan

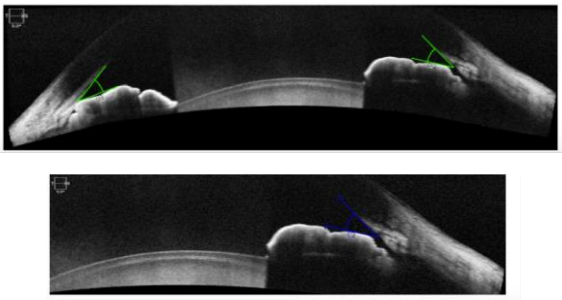
#	Detail Type	Description
1.	Assessment	Primary open angle glaucoma (POAG) of right eye, severe stage (H40.1113).
2.	Assessment	Open angle with borderline findings of left eye (H40.012).
	Impression	(1) Primary open angle glaucoma (POAG) of right eye, severe stage: H40.1113. (2) Open angle with borderline findings of left eye: H40.012. Slightly thin CCT OD and OS underestimates true IOP
	Plan	Discussed diagnosis in detail with patient. Discussed treatment options with patient including laser, medication, and observation. After detailed discussed regarding risks and benefits of condition and treatment options; patient opts for medical therapy at this time. Emphasized and explained compliance. Reassured patient of current condition and treatment. Will continue to observe condition and or symptoms. Will continue to monitor IOP. Poor adherence can lead to blindness. Discussed risks of progression. New medication(s) Rx given today. Discussed expected effects of medication including redness and mild irritation.

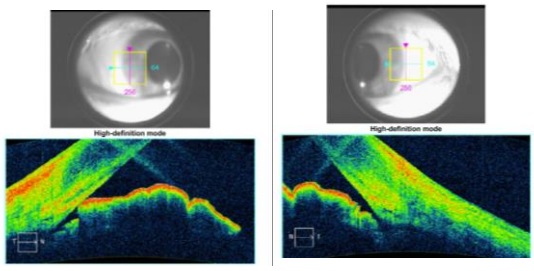
41 YEAR OLD FEMALE

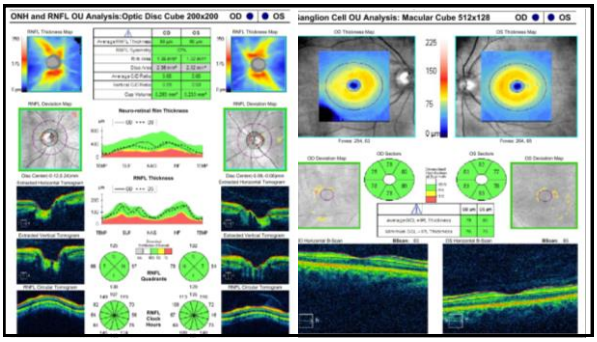
- Referred for evaluation of suspicion of glaucoma due to optic disc appearance and narrow angles
- Comprehensive eye examination:
  - HPI:
    - 1) Blurred vision
    - 2) Halos at night
    - 3) Redness (bilateral, relatively constant)
    - 4) Headache (2-3 times per month)
  - +0.75-1.00x170
  - +0.25-0.75x015
  - IOP 18/19mmHg

41 YEAR OLD FEMALE

- Pinhole VA 20/20 OD and OS
- IOP 18/19mmHg
- Gonioscopy
  - OD: No structures seen superior and temporal, anterior trabecular meshwork nasal and inferior
  - OS: Anterior trabecular meshwork 360
  - ***Convex iris approach, no PAS, NVA, AR 360 OD and OS (with compression)***







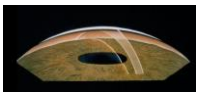
TERMINOLOGY

- **1) Primary angle closure suspect**
- 2) Primary angle closure
- 3) Primary angle closure glaucoma
- 4) Acute angle closure crisis

Either open or closed
There is no such thing as "narrow angle glaucoma"

PRIMARY ANGLE CLOSURE SUSPECT

- AKA "anatomical narrow angle"
- The pigmented trabecular meshwork is blocked by the iris 180 degrees or more by ***gonioscopy***
  - Without compression
    - No peripheral anterior synechiae
- **Disc is normal; IOP is normal**
- Ask the patient about symptoms of intermittent closure
  - Especially when the pupil is dilated (i.e. at night)
- ***LPI or observation?***
  - Stop going to movies, stop going to restaurants at night, stop using anti-allergy or cold medications…



Glaucoma

Progression of Primary Angle Closure Suspect to Primary Angle Closure and Associated Risk Factors: The Handan Eye Study

Ye Zhang,[1] Ravi Thomas,[2] Qing Zhang,[1] Si Zhen Li,[4] and Ning Li Wang[1,3]
[1]Beijing Tongren Eye Center, Beijing Key Laboratory of Ophthalmology & Visual Science, Beijing Tongren Hospital, Capital Medical University, Beijing, China
[2]Beijing Institute of Ophthalmology, Beijing, China
[3]University of Queensland, Brisbane, Australia
[4]Nanjing Tongren Hospital, Jiangsu, China

526 patients (111 male, 415 female)
32 progressed to angle closure (31 PAC, 1 PACG) in 5 years = **6%**

CLINICAL SCIENCE

Five year risk of progression of primary angle closure suspects to primary angle closure: a population based study

R Thomas, R George, R Parikh, J Muliyil, A Jacob

Br J Ophthalmol 2003;87:450–454

Southern India: ¼ PACS subjects developed PAC

TO ZAP OR NOT TO ZAP…THAT IS THE QUESTION

ZAP STUDY

Ophthalmic Epidemiol. 2010 Oct;17(5):321-32. doi: 10.3109/09286586.2010.506303

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[1], Friedman DS, He 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 670. Persons 50 years of age and older with 20/40 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)
    - Acute angle closure: 5 patients untreated, 1 treated (3 control eyes and one LPI eye were after dilation)
  - 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
  - Authors determined that laser peripheral iridotomy was not justified

Am J Ophthalmol. 2021 Jun 28;S0002-9394(21)00343-3. doi: 10.1016/j.ajo.2021.06.018. Online ahead of print.

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

Lanhua Wang[1], Wenying Huang[1], Xiaofang Huo[1], Chimei Liao[1], Ling He[1], Mingguang He[2]

Affiliations + expand

PMID: 34197780 DOI: 10.1016/j.ajo.2021.06.018

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.

to 70 years with LPI in one randomly selected eye and a fellow untreated eye were included. All participants underwent comprehensive examinations before and at 2 weeks, 6 m, 18 m, 36 m, 54 m, and 72 m after LPI. IOP was measured using Goldmann applanation tonometry before and 1 hour after pharmacological dilation.

Results: The mean pre-dilation IOP in the untreated eyes was 14.8±2.7 mmHg, which increased to 16.4±2.7 mmHg after pharmacological dilation (p<0.001). The treated and untreated eyes had similar pre-dilation and post-dilation IOP (all p>0.05). The average post-dilation IOP elevation was 1.5 mmHg in the treated eyes and 1.6 mmHg in the untreated eye without significant differences (p=0.802). Lower pre-dilation IOP (p<0.001), smaller AOD500 (p<0.001), smaller ARA500 (p=0.030), smaller TISA500 (p=0.040), and larger lens(p<0.007) were associated with post-dilation IOP elevation 5 mmHg and greater. Three untreated (1.04 per 1000 pupil dilations) and one treated eye (0.34 per 1000 pupil dilations) developed acute angle-closure (AAC) after dilation during the 72 m follow-up.

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.

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Anatomic Changes and Predictors of Angle Widening after Laser Peripheral Iridotomy

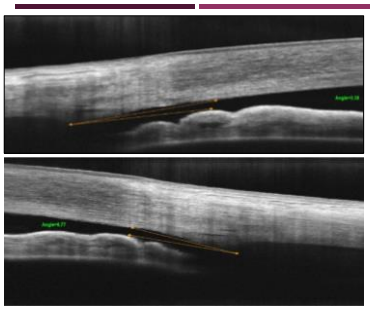
The Zhongshan Angle Closure Prevention Trial

Benjamin Y. Xu, MD, PhD,[1] David S. Friedman, MD, PhD,[2] Paul J. Foster, FRCS(Ed), PhD,[3] Yu Jiang, MD,[4] Anmol A. Pardeshi, MS,[1] Yuzhen Jiang, MD, PhD,[4] Beatriz Munoz, MS,[2] Tin Aung, FRCS(Ed), PhD,[5] Mingguang He, MD, PhD[4]

Conclusions: Superior LPI location results in significantly greater angle widening compared with temporal or nasal locations in a Chinese population with PACS. This supports consideration of superior LPI locations to optimize anatomic changes after LPI. *Ophthalmology 2021;■:1–8 © 2021 by the American Academy of Ophthalmology*

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?

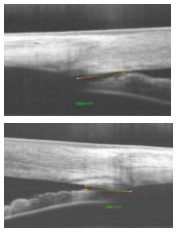
74 YOF

- 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

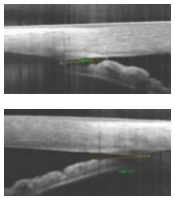
BACKED INTO A CLOSURE CORNER

- 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



BACKED INTO A CLOSURE CORNER

- 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

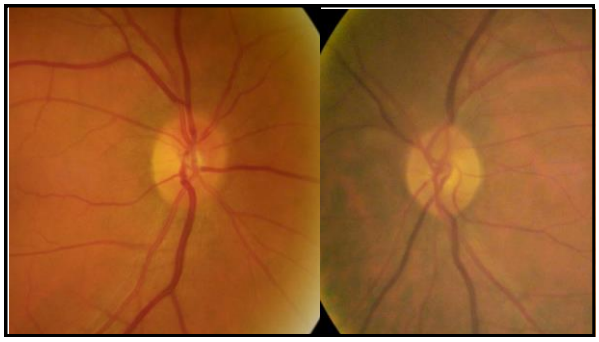
- Can this 30 YO go the rest of her life without dilation?
- Really no good options (Pilo? Iridoplasty? Lens extraction at 30 years old?)
- Hasn't had an attack yet
- Harry Quigley, MD, "*You just don't know, so sometimes you gotta bite the bullet, dilate, and see what happens. But you don't do it on 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

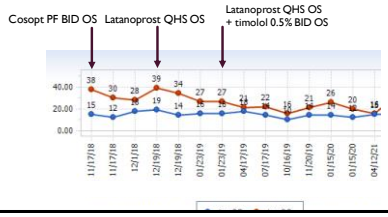
- Returns 8:30 am Tuesday
- IOP: 22 mm OD, 22 mm at 8:30 am; pt informed of risks; dilated 0.5% tropicamide
  - Diamox and Combigan ready
    - It works- trust me
- IOP: 22 mm OD, 22 mm OS at 9:30 am
- IOP: 22 mm OD, 23 mm OS at 1:15 pm; pupil in mid-dilated state
- Fundus normal OU; C/D 0.2 OU
- Pt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

CHARACTERISTIC FEATURES OF PIGMENTARY DISEASE

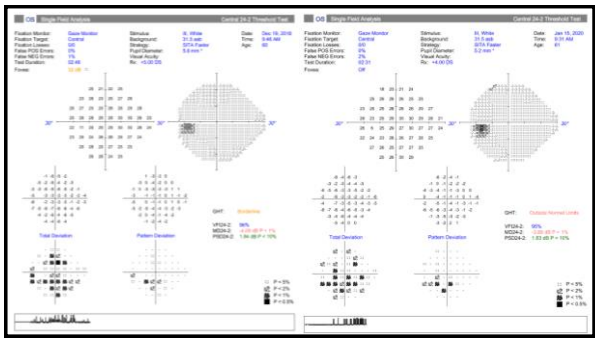
- Vertical deposition of pigment on the endothelium
- Heavily pigmented posterior trabecular meshwork
- Sampaolesi line
- Characteristic posterior bowing of the iris
- Midperipheral transillumination defects
- Scheie's line
  - Pigment deposited at the junction of the posterior zonules and the vitreous face

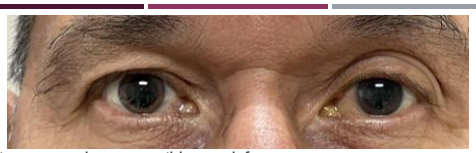


PIGMENT DISPERSION SYNDROME



CCT 584/588um





- Pigmentary glaucoma, mild stage, left eye
- Patient develops cosmetic concern of **"droop of LUL"**
  - Expected hyperemia with PGA-tolerable
- *Did we do this?*
  - *Pseudoenophthalmos due to loss of periorbital fat*
- ***Now what?***

PIGMENTARY GLAUCOMA, MILD STAGE

d/c latanoprost
(timolol 0.5% BID + brimonidine 0.2% BID)

PERTINENT POINTS

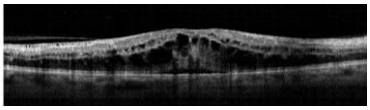
- Glaucoma is a chronic progressive optic neuropathy
- Take the time that you need to establish a diagnosis
- The goal of treatment is to *slow* the progression of disease to preserve functional vision—and therefore quality of life
  - **At a reasonable cost**
  - Ensure no duplications of medications when prescribing more than one bottle
    - ~~Simbrinza + brimonidine~~
    - ~~Cosopt + timolol~~
    - Vyzulta + brimonidine

67 YEAR OLD FEMALE; GLAUCOMA SUSPECT

- **What changed?**
  - Multiple IVI (approximately 6-9 per year) between 2014 and 2016 for macular edema secondary to CRVO
    - Avastin, Ozurdex

HOW *COULD* IVI AND VITRECTOMY CAUSE ELEVATED IOP?!

- Widening indications for vitrectomy
- Anti-VEGF injections are the cornerstone of medical retinal treatment
- **Long and short term IOP rise possible**
- Development of OAG & progression of OAG

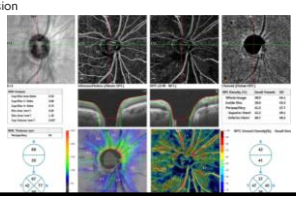


SHORT TERM

- We tend to think about the greatest risk of IVI to be endophthalmitis (1/2659)
- Immediately after injection: IOP rise to up to **87mmHg**
  - Most patients increase approximately 20mmHg-35mmHg
  - *Do most surgeons measure IOP after injections?*
- How does this happen?!
  - Increased intravitreal volume
    - 4-4.4mL average volume; most injections 0.05mL

WELL THAT CAN'T BE GOOD

- Risk of retinal artery occlusion (as high as 1/1389 Gao et al 2019)
- Repeated, sudden, **significant** IOP spike and temporary loss of perfusion



HOW CAN WE FIX THIS?

- **Treatment for elevated IOP vs. IOP spike-prevention**
  - Role of pre-procedure IOP lowering medication
  - Paracentesis
    - 32 gauge needle
    - Fluid balance

Anterior chamber paracentesis during intravitreal injections in observational trials: effectiveness and safety and effects

Sandeep Saxena[1], Timothy Y. Lai[2], Hideki Koizumi[3], Michel E. Farah[4], Daniela Ferrara[5], David Pelayes[6], Tomohito Sato[7], Carsten H. Meyer[8], Timothy Murray[9] and for the International Pharmacokinetic Collaboration

International Journal of Retina and Vitreous

ALL ABOUT OUTFLOW

- Reduced trabecular outflow:
  - 1) Direct toxicity of medication
  - 2) Inflammation
    - Trabeculitis
  - 3) Aggregation of particles
    - Silicone, protein in the TM
  - 4) Nitric oxide reduction

SILICONE?

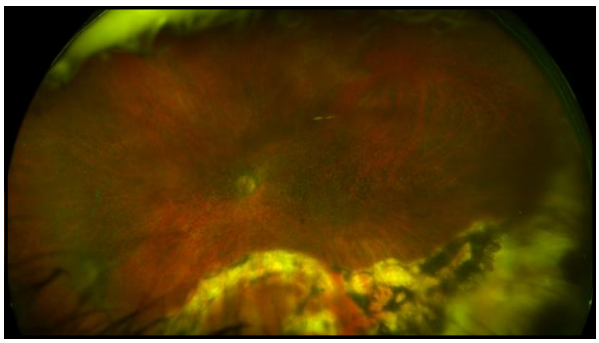
- Medical grade silicone oil droplets
  - Barrel of the syringe
  - Hub of the needle
  - Tip of the plunger
  - Stopper of the medication vial
- *Silicone oil has the potential to be pro-inflammatory*

NITRIC OXIDE

- Nitric oxide is involved in the signaling pathway which leads to relaxation of trabecular beams
  - Leads to increased trabecular outflow
    - Latanoprostene bunod
      - Latanoprost acid + butanediol monohydrate
        - NO is a gas, so must be attached to another molecule
- VEGF upregulates nitric oxide synthase = increased nitric oxide
- *Effect of **anti-VEGF** medications?*

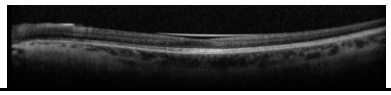
SO WHO IS MOST AT RISK?

- Greater number of injections (20+)
- Higher frequency of injections (7/year +)
  - Eadie et al 2017
- Younger patients
- Patients with shorter axial length



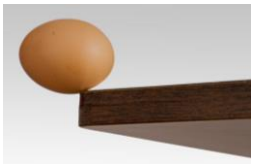
VITRECTOMY & TAMPONADE AGENTS

- Long term potential for IOP rise
  - Oxidative stress-fluid/air exchange
- Tamponade agents
  - Sulfur hexafluoride (SF_6)
  - Perfluoropropane (C_3F_8)
  - Silicone oil-greatest risk of IOP elevation-as high as 40%



BOTTOM LINE

- **Monitor intraocular pressure in patients undergoing IVI or who have a history of PPV**

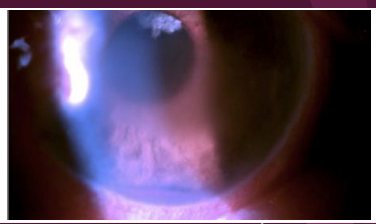


THE CASE OF THE DISAPPEARING DIABETIC

- 82-year-old Hispanic male presents for IOP check
- Chief complaint: Pt reports FB sensation, mild ocular eye-pain and redness of left eye that started 15 days ago
- Past Ocular History: POAG OS, severe stage
- LEE: 11/19/2018 – lost to follow-up
- Medical History
  - Diabetes Mellitus Type 2 - Glyburide 5mg Tablet QD po
- Ocular Medications
  - Latanoprost qhs OU

THE CASE OF THE DISAPPEARING DIABETIC

- BVA 20/25 OD; NLP OS
- Cornea: Normal cornea OD, diffuse PEK, microcystic corneal edema OD
- Iris: diffuse NVI at the pupil margin OS
- Anterior Chamber: deep & quiet OD; 1 hyphema with RBCs in anterior chamber OS
- Lens: PCIOL in good position OD; limited views OS
- IOP: 23 mm OD, 62 mm OS
- Gonio: NVA; PAS; hyphema



THE CASE OF THE DISAPPEARING DIABETIC

- NVG MOA
- Management straightforward
  - Atropine 1% BID
  - Pred forte QID
  - Diamox
  - Aqueous suppressants
  - Anti-VEGF
  - PRP (ultimate treatment)

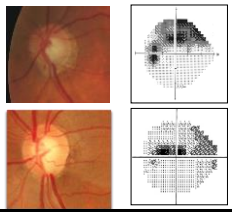
Are we setting target pressures too high?

INFORMATION FROM MAJOR STUDIES

Study	IOP reduction	Progression
OHTS	20%	Yes
EMGT	25%	Yes
CNTGS	30%	Yes
CIGTS (med)	35%	No
CIGTS (Surg)	48%	No
AGIS	< 18 all visits	No

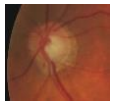
WHICH IS BETTER? ONE OR TWO?

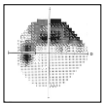
- Pt 1: treated 20 mm
- Pt 2: treated 15 mm

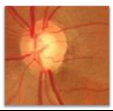


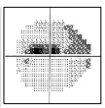
WHICH IS BETTER? ONE OR TWO?

- Pt 1:Ta max 42 mm- treated 20 mm
- Pt 2:Ta max 20 mm- treated 15 mm









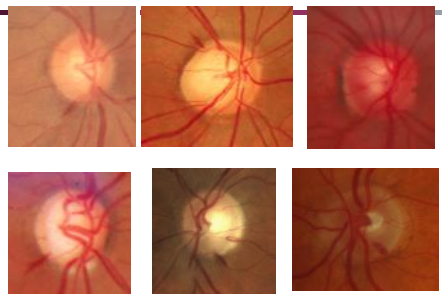
WHAT DO YOU DO WHEN YOU SEE A DISC HEMORRHAGE?

Not all hemorrhages of the disc are disc hemorrhages.

RISK FACTORS: DISC HEMORRHAGES

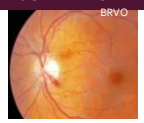
- Inferior, inferior temporal, superior, and superior temporal regions of the disc are most susceptible and account for virtually all true glaucomatous disc hemorrhages
- Typically occurs where notches and RNFL defects occur

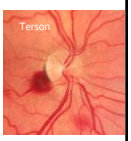
Hemorrhages at other areas of the disc (nasal and temporal) tend to not be associated with glaucoma.



OTHER CAUSES OF 'DISC' HEMORRHAGES

- PVD
- HTN
- Anemia
- Diabetes
- Vascular occlusion
- Subarachnoid bleed
  - Terson syndrome
    - Subretinal and intraretinal
    - May be juxtapapillary





Not all hemorrhages of the disc are disc hemorrhages. Make sure that the glaucomatous characteristics are there.

ARE DISC HEMORRHAGES A RISK FACTOR FOR PROGRESSION OR ACTUAL PROGRESSION?

EARLY MANIFEST GLAUCOMA TRIAL

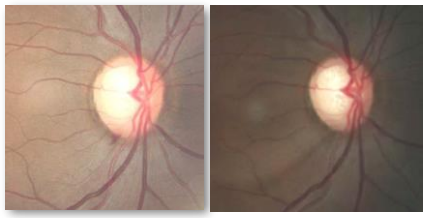
- Disc hemorrhages- predictive of progression
- Treatment was unrelated to the presence or frequency of disc hemorrhages.
  - Disc hemorrhages were equally common in both the treated and untreated groups of patients.
  - Disc hemorrhages don't occur in all glaucoma pts.
- Disc hemorrhages cannot be considered an indication of insufficient IOP-lowering treatment,
  - Glaucoma progression in eyes with disc hemorrhages cannot be totally halted by IOP reduction.

OCULAR HYPERTENSION TREATMENT STUDY

- The occurrence of a disc hemorrhage increased the risk of developing POAG 6-fold in a univariate analysis and 3.7-fold in a multivariate analysis that included baseline factors predictive of POAG
- Occurrence of an optic disc hemorrhage was associated with an increased risk of developing a POAG end point in participants in the OHTS
  - However, most eyes (86.7%) in which a disc hemorrhage developed have not experienced a POAG end point to date
- (OHTN defined as 21 mm Hg or more)

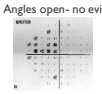
DISC HEMORRHAGE

- Ischemic, vascular, both, or neither?
- EDI OCT shows changes in lamina commensurate with disc hemorrhage
  - Thus it would be considered progression due to mechanical change
- If ischemic, would occur everywhere on the disc and be more bilateral



ALL GLAUCOMA IS NOT CREATED EQUAL

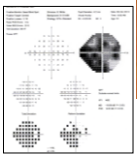
- 71 YOF
- Diagnosed POAG OU 2009- treated with Travatan Z will good response (IOP drops to 18 from 28)
- CCT: 579, 583
- Transfers care for convenience
- Angles open- no evidence of secondary glaucoma



ALL GLAUCOMA IS NOT CREATED EQUAL

- 2012: 20/30 OD, 20/400 OS
- SLT OU x2
- Meds: Lumigan, Combigan, Azopt
- Hx: Used oral CAI 3x/day- hands and feet hurt too much to continue
- Used pilocarpine- motion sickness
- IOP- 22 mm OD and 38 mm OS

Now What?



ALL GLAUCOMA IS NOT CREATED EQUAL

- Visit 2/14
- Not seeing OS since 9/13
- 20/50 OD, LP OS
- IOP 36 mm OD, 30 mm OS
- Now What?
- Declines surgery again and again

ALL GLAUCOMA IS NOT CREATED EQUAL

- N/S until 2/15
- Did request med refills throughout, however
- Using Combigan only- ran out of Azopt and Travatan
- 20/60 OD, NLP OS
- IOP 46 mm OD and 72 mm OS
- Refill all meds
- Declines surgery again

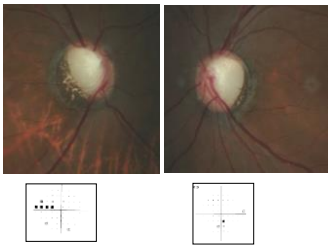
ALL GLAUCOMA IS NOT CREATED EQUAL

- Visit 6/15
- Using meds regularly, but was confused when to use Travatan so didn't use it in past week
- Vision unchanged
- IOP: 40 mm OD and 53 mm OS
- New views on surgery

NOW HOW WOULD YOU HANDLE THESE? DON'T WORRY…

THE CASE OF THE FAILED LASIK SCREENING

- Referred for glaucoma eval in 2002 after failing LASIK screening
- Had been treated since mid 20s for glaucoma
- IOP in mid-upper teens off meds
- CCT: 459 OD; 469 OS
- Anomalous nerves with mild field loss

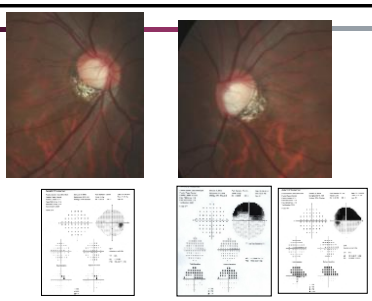


THE CASE OF THE FAILED LASIK SCREENING

- Congenitally anomalous nerves with field loss
- Monitored for 11+ years
- Field changes late
- Pt now treated with IOP 09 mm OD; 10 mm OS
- Pt had/had congenitaloma and now has glaucoma
  - Doubloma

THE CASE OF SO SIMILAR…YET SO DIFFERENT

- 45 YO Japanese Female
- Referred for glaucoma evaluation
- IOP never exceeds mid-teens
- CCT: 554 OU
- Marginal effect of meds

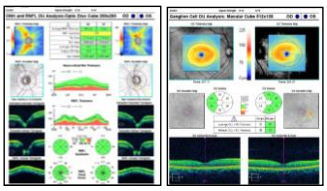


THE CASE OF SO SIMILAR…YET SO DIFFERENT

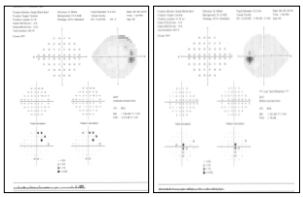
- Field loss due to anomaly, glaucoma, or both?
- Progressive or congenital?
- Mid-teen IOP and poor medical response
- Treatment or observation?

LOOK AT *ALL* OF THE DATA

- 62 YOF- glaucoma suspect
- 20/20 OD, OS
- CCT: not done yet
- IOP: 17-18 mm Hg OU – multiple occasions
- Biomicroscopy normal OU
- Angles open OU



Normal? Abnormal? Borderline? Suspicious?



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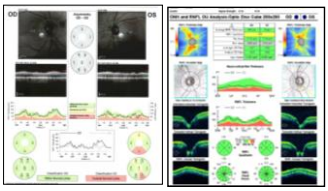
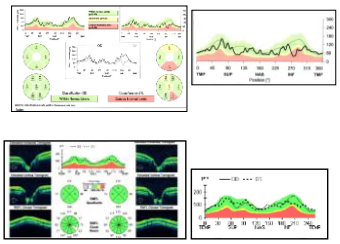
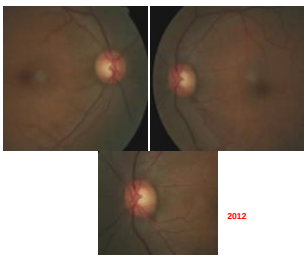


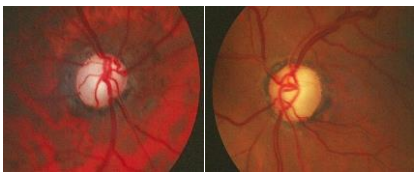


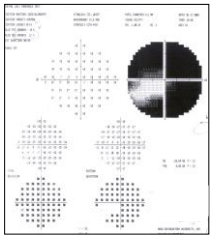


2012

AS GOOD AS IT GETS?

- 63 YOBM
- Knows he has POAG – doesn't follow through with treatment
  - Poor care in Caribbean
- IOP 43 mm Hg OD; 60 mm Hg OS
- Angles open by gonio OU
- Hand Motion OD, 20/40 OS
  - Small temporal island of vision OS



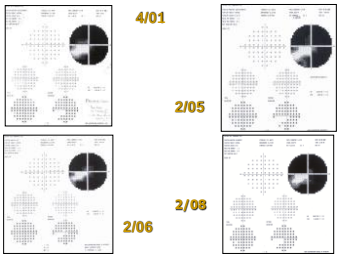


So, who wouldn't want this patient in their practice?

What are the options?

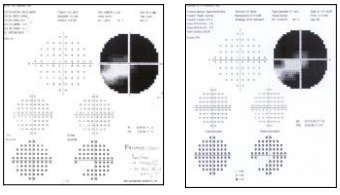
AS GOOD AS IT GETS?

- 63 YOBM - POAG
- Medications:
  - Timolol/brimonidine FC, brinzolamide, travoprost OS; travoprost OD
- IOP: 29-34 mm Hg OD, 10-13 mm Hg OS
- Never misses appointment
- Thankful things are as goods as they are



HANGING ON

- January 2009
- Reports vision slowly getting worse OS
  - 20/200
- "We had a good run, Joe"
- Is it cataract or glaucoma?



- Cataract surgery obtained on humanitarian basis
  - Nobody wanted to do surgery
  - 20/150 1-day post op
  - "Seeing beautifully"
  - 20/70 dist; 20/50 near
  - Final outcome?