

Challenging Cases: Front to Back

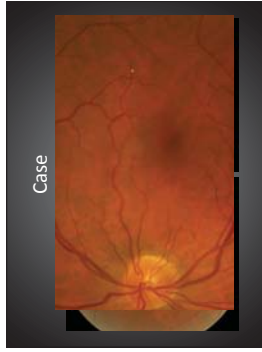
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Disclosure

- Presenter is on speakers panel of Alcon, Allergan, J.J. Bausch + Lomb, Tear Lab, Ocusoft, BVI, Bruder, Reichert
- AOA CE Chair
- OCCRS-Past President
- Presenter has NO financial interest in any products mentioned
- Except he does have stock in a certain coffee company...

Case SS

- 71 year old retired Military and secret service agent
- Hasn't had eyes checked in a few years
- VA 20/20 OU with low hyperopic/astigmatic RX
- SLE: mild bleph, trace NSC
- Posterior pole:



Case SS

- A: HH plaque OS
- P: refer for carotid doppler
- Labs
- refer to PCP for management of other risk factors
- Vascular clinic dependent on carotid study

Case SS: Labs

- Labs
- BP: 134/88
- Weight: 236
- BMI: 38.2
- A1c: 9.9 (H)
- Triglycerides: 173 (H)
- HDL: 31.2 (L)
- PCP: diet, education, start insulin

Case SS

- Carotid:
 - Right: non hemodynamically significant soft calcific plaque at left carotid bifurcation
 - Left: 50-69% ICA stenosis
- Vascular clinic:
 - Monitor left carotid q 6 mos. as no symptoms in last year
 - Start ASA therapy

Retinal Plaques

- Several different types of plaques can often be visualized in the retinal vasculature
- PIs typically elderly, has HTN, CAD, hypercholesterolemia/hyperlipidemia, and/or other ocular disease
- Often totally asymptomatic and found on routine exam

RISK FACTORS

- Age
- HTN
- Vascular disease
- Past vascular surgery
- SMOKING
- High TOTAL cholesterol
- Men > women

Prevalence

- Beaver Dam Eye Study: 1.3%
 - smoking, HTN and DM
 - 9x more likely after age 75 vs. 43-54
 - after 75, 3.1% prevalence
 - Equates to 1.2 million people with emboli 43-86
 - 450,000 are 75-95
 - Age structure is similar over 8 years in pfs with emboli, adjusting for other factors
- ODOS
- Bilateral very infrequently

Prevalence

- Blue Mountain Eye Study: 1.4%
 - HTN, smoking, vascular disease
- LA Latino Eye Study: 0.4%
 - smoking, CAD, h/o MI, HTN
- Singapore Eye Study: 0.6%
 - smoking, high cholesterol, h/o angina

Retinal Plaques

- May present with amaurosis fugax, transient episodes of monocular blindness
- Rarely, may report transient ischemic attack (TIA), which is above with hemiparesis, parasthesia or aphasia

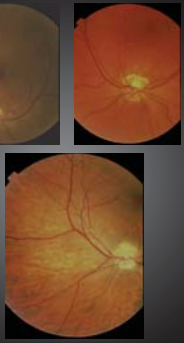
Retinal plaques

- Three different types of plaques, but all share strong association to significant cardiovascular disease
 - HH 80% > fibrino-platelet 14% > calcific 6%

Retinal Plaques

- Cholesterol (Hollenhorst) plaque
 - Most common
 - shiny yellow-orange in appearance
 - from plaque in the ipsilateral carotid artery
 - Rarely causes occlusion, unless multiple
 - Typically occurs at bifurcations
 - Mobile in nature

Cholesterol Plaques

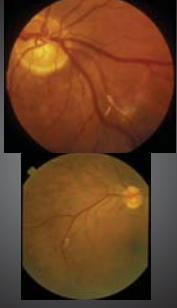


Retinal Plaques

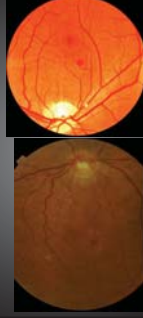
- Fibrino-platelet
 - Appear as dull white to gray, long plugs
 - Typically within arterioles, not at bifurcations
 - May break-up and dissolve with time
 - May lead to BRAO or CRAO
 - Often associated with carotid disease or mitral valve insufficiency



Fibrino-platelet Plaques



Calcific Plaques



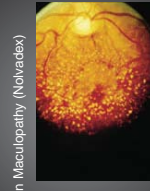
Retinal Plaques

- Calcific
 - Appears more whitish than HH
 - Dull, non-reflective, white
 - Classically within arteriole, not at bifurcation
 - Typically immobile
 - Most dangerous, as often cause BRAO
 - Often from cardiac atheromas of heart valves

Retinal plaques

- Talc retinopathy
 - Represents an exogenous plaques as opposed to others
 - Appears typically as multiple shiny yellow plaques within capillaries in posterior pole
 - Typically smaller than other plaques
 - Rarely cause complications, but reported cases of associated NV and occlusions

Others



- Tamoxifen Maculopathy (Nolvadex)

Talc Retinopathy





Retinal plaques

- No direct management of plaques is needed
- Management is aimed at discovering source of embolus to decrease risk of other emboli, occlusion, or stroke
- Pls need referral to internist for complete physical

Retinal Plaques

- Assess risk factors with POP
 - DN, HTN, lipid panels
- Carotid ultrasound
- MRA: non-invasive image with 2D/3D
- TEE: invasive, probe into esophagus to image heat valves
 - Helpful with calcific
- CTA: CT scan of arteries construct 3D images

Retinal Plaques

- <50-60% occlusion
 - ORAL TREATMENT
 - Anti-Platelet
 - ASA
 - Anti-coagulation
 - Comarin, plavix
 - Cholesterol meds
- >70-99%
 - SURGICAL TREATMENT
 - Carotid endarterectomy
 - Angioplasty
 - Reduces risk of future stroke!

Carotid Ultrasound

- First line screening test
- ORDER WITHIN TWO WEEKS!
- Identifies flow rate and % stenosis
- Common, internal, and external
- Only ~20% of asymptomatic emboli will have significant carotid stenosis

Is it worth working up these patients?

- 18% of pts with retinal emboli had internal or common carotid stenosis >75%
- Higher incidence of stroke
 - 8.5% with normal vs 0.9% per year
- Pls with cholesterol, any emboli have 15% mortality at 1 yr, 25% by year 3, and 54% by 7 years

43 yo female

- "It felt like something was stabbing my eye"
- "This happened another time and I had to use my eye at night"
- Type II Diabetes
- NKMA

"IT REALLY HURTS"

Retinal Plaques

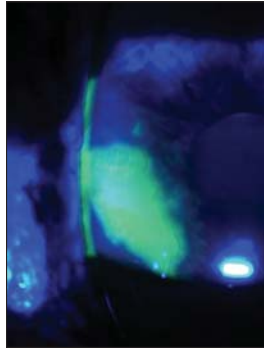
- After ruling out underlying etiology, see patient regularly, q 6-12 mos, to evaluate for additional plaques or other disease associated with vascular disease
 - BRVO/CRAO
 - NTG

THE "PREVENTED" CORNEAL EROSION

THE RECURRENT EROSION

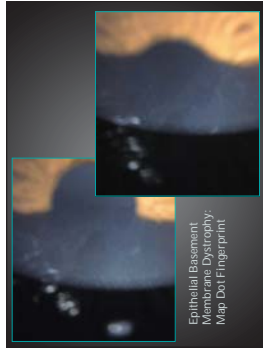
VA: 20/100 PH 20/30
SLIT-LAMP: SUPERFICIAL SUPERIOR ABRASION + STAINING

DX: CORNEAL EROSION

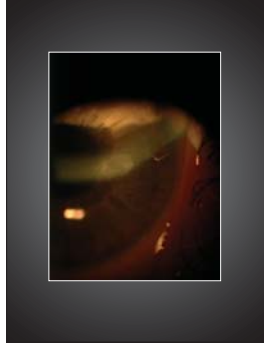
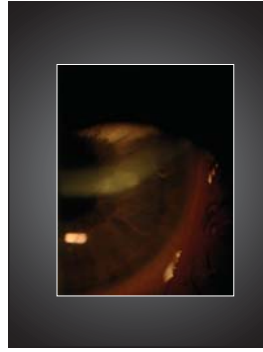
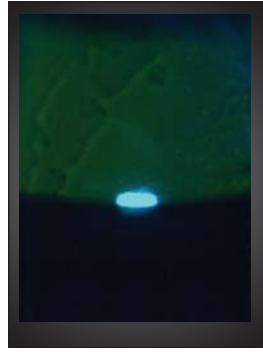


What are some leading causes of RCE?

EBMD



Epithelial Basement Membrane Dystrophy: Map Dot Fingerprint



87% of all RCE occurs in what part of the cornea?
Inferior Cornea
Reidy JJ, Pauli MP et al. Cornea 2000 Nov.

46% of all patients in this study had EBMD
• James Reidy et al. Recurrent erosions of the cornea: epidemiology and treatment. Cornea. 2000 Nov; 19(6): 767-71
• The remainder had trauma induced causes
– Fingernail
– Paper cut, etc.

Non-Treatment:
• **What medications should be avoided?**
• **Bland Artificial Tear Ointments**
Eke J. et al. Recurrent symptoms following traumatic corneal abrasion. Eye. 1999 June.

Passive Treatment Of RCE
• DEBRIDE
– Weck-Cel Spear
– Alger brush
• Bandage Lens
• Broad-spectrum AB

RECURRENT CORNEAL EROSION

Study	Patients	Prevalence	CF
1	100	100%	100%
2	100	100%	100%
3	100	100%	100%
4	100	100%	100%
5	100	100%	100%
6	100	100%	100%
7	100	100%	100%
8	100	100%	100%
9	100	100%	100%
10	100	100%	100%
11	100	100%	100%
12	100	100%	100%
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43	100	100%	100%
44	100	100%	100%
45	100	100%	100%
46	100	100%	100%
47	100	100%	100%
48	100	100%	100%
49	100	100%	100%
50	100	100%	100%

Active Treatment...Dry or Wet??

NON-HEALING ABRASION

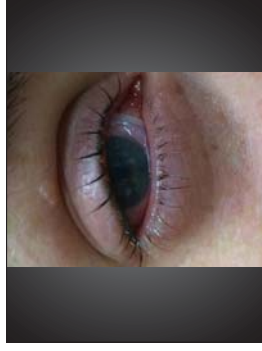
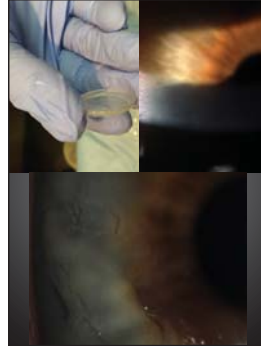
TREATMENT

STANDARD PROTOCOL:

- RCT
- ANTI-BIOTIC
- ANTI-INFLAMMATORY
- RTC
- NEW REGIMENT
- AMNIOTIC MEMBRANE
- LATERAL TARE TARSOGRAPHY
- PATIENT EDUCATION
- RTC 5 DAYS

Amniotic Membrane

- Amniotic membrane is the inner most lining of the placenta (fetus) and shares the same cell origin as the fetus
- Contains cytokines and growth factors
 - Anti-Apoptotic
 - Anti-Inflammatory (protease inhibitors)
 - Aids in rapid wound healing and re-epithelialization
 - Anti-Scarring

Long Term TX Regimen Resolchirant RCE

- FreshKote TID x 2 months
- Lotemax Gel QID x 2 weeks then BID x 6 weeks
- Doxy (20 or 50mg) BID x 2 months
- Restasis Bid!

5 DAY FOLLOW-UP

"THE PAIN IS GONE"
REMOVED PROKERA

- VASC: 20/15
- SLIT-LAMP: CORNEAL CLEAR

SLIT-LAMP: CORNEAL CLEAR

TX: RESTASIS BID
RTC 4-6 WEEKS



SF CASE

- 68 year old male
- Presents with c/o flashes floaters OD x 2 days
 - No pain
 - No change in acuity
- Med hx: Type 2 DM x 2 years, well controlled; HTN; ED
- Meds: Metformin, HCTZ, Lipitor, Viagra
- Oc Hx: Unremarkable

3 MONTH FOLLOW-UP

- "MY EYE FEELS GREAT"
- VASC: 20/15
- SLIT-LAMP: CLEAR CORNEA
- DX:

RCE (PREVENTED CORNEAL EROSION)
TX: CPM(RESTASIS)

SF CASE

- Entering VA: 20/25 OU
- SLE: WNL
- IOP 14 mm OU
- DFE:



SF CASE

- Assessment:
 - Acute PVD OD
- Plan:
 - Pt education
 - Signs/symptoms of RD
 - RTC when?

SF CASE

- Really no consensus
- Symptomatic PVD without retinal break
 - AAO: 1-2 weeks
 - AAO: depending on symptoms, risk factors and clinical findings:
 - 1-6 weeks
 - then 6 mo to 1 year
 - Cleveland Clinic: 4-6 Weeks
 - Others: If no hema or other issues, very low risk so no need to see to back.

PVD

- Floaters are typically most common symptom
 - Cobwebs
 - Flies
 - Hairs
- Flashes
 - Indicative of traction on retina, but not necessarily a tear or break



The Vitreous Humor

- Vitreous attached most firmly at:
 - Macula
 - WMT
 - Vitreous base
 - Around optic nerve head
 - Vessels, fling
 - Retinal traction on blood vessels
 - Vitreous

Physiologic Changes

- With age, **hyaluronan** due to reduction in hyaluronic acid causes loss of support.
- This process is referred to as **synchysis**.

Physiologic Changes

- Vitreous shrinkage, contraction and collapse can cause traction.
- This process is referred to as **synchysis**.

Incidence of PVD

Age	Incidence
<30	RARE
30-59	10%
60-69	27%
>70	68%
>80	79%

- 65%>65 HAVE A PVD

Incidence of PVD

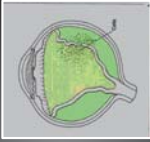
- Incidence may be accelerated by
 - Myopia
 - Trauma
 - Prior vitreoretinal disease
 - Surgery
 - Inflammation
- Symmetrical 90% of the time
- Happens to second eye with 1-2 years

PVDs

- Good News:
 - Retinal Tears/Breaks: Relatively uncommon
 - One study only 7.15% of symptomatic PVDs have a retinal tear
 - 8-26% acute PVDs have an associated RB/RO at the time they present (Ophthalmology AAO 2014)
- Bad news:
 - 1/3 have a retinal break
 - The chance of RB there after is <5%

Risk Factors

- Pigment
 - Schaeffer's sign
 - Indicates break is possible
- Hemorrhage
 - 90% have break
- Inflammatory cells



My recommendations

- DFE WITH XALATAN **CONSERVATION**
- DISCUSS SIGNS/SYMPTOMS OF RD
- RTC 6 WEEKS
- SEE UNTIL FLASHES SUBSIDE
- IF RISK FACTORS, CONSIDER REFERRAL TO RETINA
 - Vitreous hemorrhage
 - Pt is Lawyer/father-in-law, etc
 - Just doesn't feel right

Move...

Case

48 YO HF

- Diagnosed with POAG 1995
- Diagnosis made by ophthalmologist in Minnesota
- Relocated to Phoenix, assume care
- Untreated peak IOP
 - OD=27mm Hg
 - OS=29mm Hg

48 YO HF

Treatment History

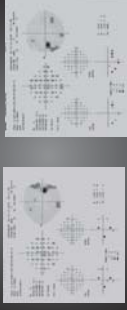
- Initial therapy, Timolol 0.5%
 - Discontinued after 2 months
 - Side effects of bradycardia & fatigue
- Current Medical Regimen:
 - Xalatan 0.01% x 2 yrs

48 YO HF

- Since starting Xalatan IOP readings:
 - OD=17-19mm Hg
 - OS=18-20mm Hg
- Previous doctor felt that patient was being "safely" treated at this IOP level.

48 YO HF

VF 2 yrs ago



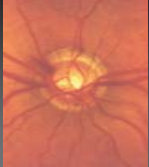

48 YO HF

Initial Exam in Phoenix

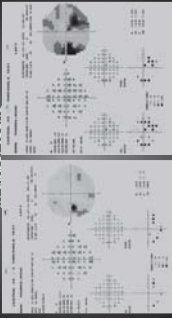
- BCVA 20/20 OU
- – RAPD
- IOP OD=17mm Hg OS=18mm Hg
- Subjectively, the patient
 - Reports excellent compliance
 - Denies any side-effects

48 YO HF

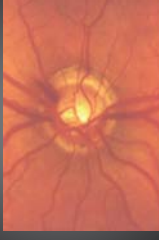
Left Eye: Phoenix ancillary tests

Visual Field Progression in upper teens



Disc hemorrhage suggests Upper teens are NOT low enough



WHAT WOULD YOU DO NEXT?

OBA Reicht: **MORE TESTING IS NEEDED**

Understand the Cornea, Understand the Pressure
Corneal Biomechanics and Accurate IOP in One Simple Instrument



Key Features

- OBA is the only device in the world capable of measuring Corneal Hysteresis (CH), which is an indicator of bio-mechanical properties of the cornea (its "spring" or "soft" nature)
- CH is independently predictive of visual field progression, which helps clinicians make better informed decisions about glaucoma treatment
- CH is superior to Humphrey CCT as a glaucoma risk assessment tool
- CH features an "invented" corneal model (OBA's) IOP measurement that is less influenced by corneal properties than other tonometers, including Goldmann
- This is superior to CCT based IOP adjustment, which has been discarded
- There are over 425 peer-reviewed publications about OBA in the literature
- OBA is fast, objective, non-contact, and operates flexibly



Hysteresis: Not a New Concept

Dr. James Alfred Ewing first introduced the term in 1890

A measurement that characterizes response to application and removal of force (load/unload)

- Force does not disappear but is slowly or dissipate a portion of the applied energy

More than 7000 papers published on hysteresis

The importance of corneal hysteresis in glaucoma

- Various factors affect hysteresis, time, stress, etc.
- Corneal hysteresis is a key factor in glaucoma

1. Ewing JA. *Philos Mag*. 1897;44:97-107. doi:10.1080/14746259708239128

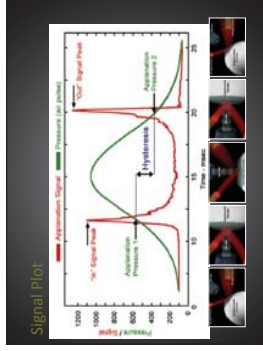
Basic Parameters

OBA

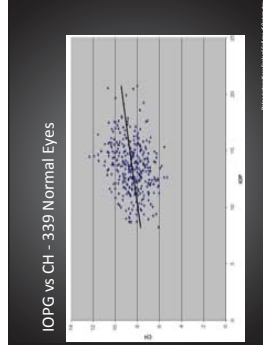
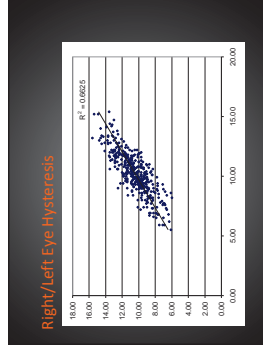
- IORe - Goldmann Correlated IOP
- IOAcc - Corneal Compensated IOP
- CH - Corneal Hysteresis
- CRF - Corneal Resistance Factor

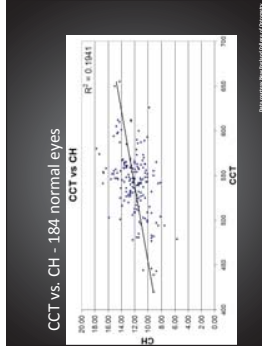
ZOE

- IORe - Goldmann Correlated IOP
- IOAcc - Corneal Compensated IOP



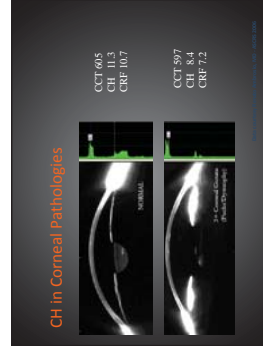
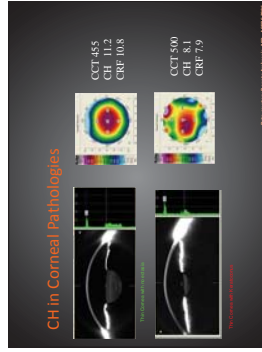
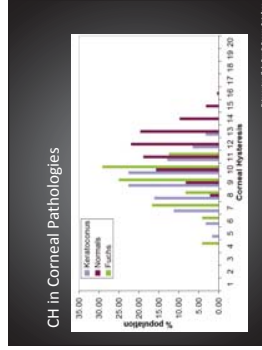
Corneal Hysteresis: A New Ocular Parameter



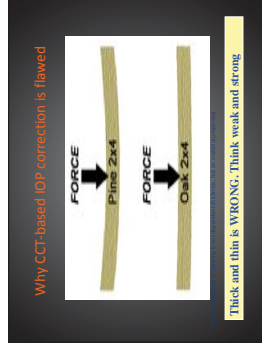
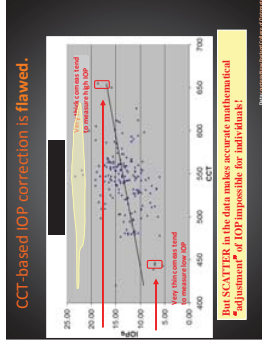
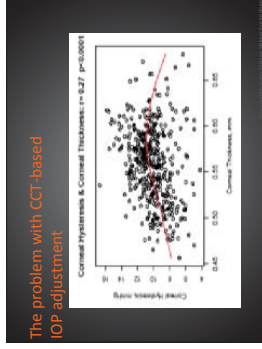


Normal CH values - Summary of published results

Author	Age (y)	CH (mmHg)
Alm & Chauhan (2000)	30-70	10.8 ± 1.5
Alm & Chauhan (2001)	30-70	10.7 ± 2.0
Alm & Chauhan (2002)	30-70	10.8 ± 2.3
Alm & Chauhan (2003)	30-70	10.7 ± 2.3
Alm & Chauhan (2004)	30-70	10.7 ± 2.3
Alm & Chauhan (2005)	30-70	10.7 ± 2.3
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Alm & Chauhan (2014)	30-70	10.7 ± 2.3
Alm & Chauhan (2015)	30-70	10.7 ± 2.3
Alm & Chauhan (2016)	30-70	10.7 ± 2.3
Alm & Chauhan (2017)	30-70	10.7 ± 2.3
Alm & Chauhan (2018)	30-70	10.7 ± 2.3



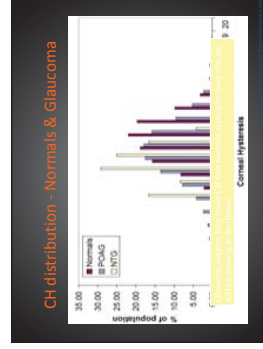
The Cornea, IOP, and Glaucoma



The Cornea and Glaucoma

Numerous studies, such as the Ocular Hypertension Treatment Study, have shown that Corneal thickness is an independent indicator of glaucoma risk.

More recent research has indicated that the Corneal Hysteresis measurement appears to be even more powerful in this regard.



Define & Describe IOPcc

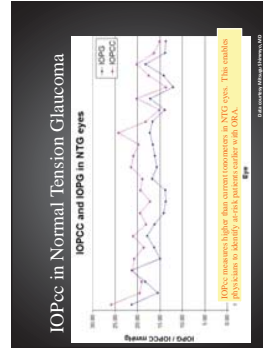
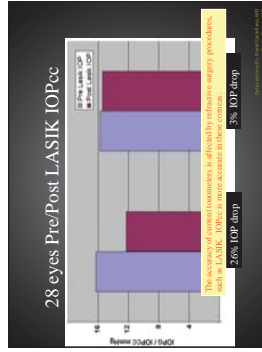
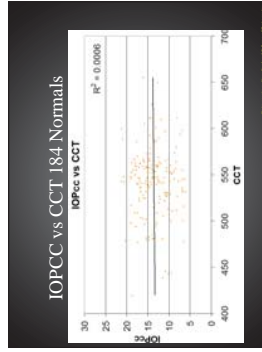
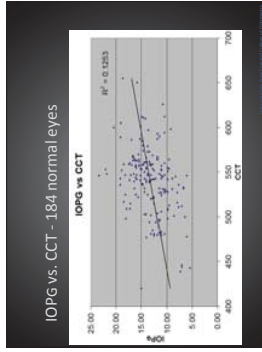
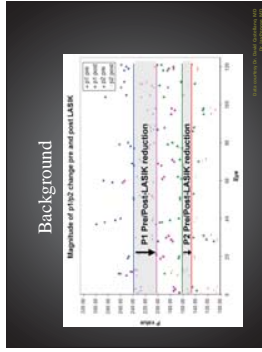
Corneal-Compensated Intraocular Pressure

An Intraocular Pressure measurement that is designed to match the geometry of the eye, and is not affected by corneal geometry (but is affected by corneal properties, including Corneal Hysteresis).

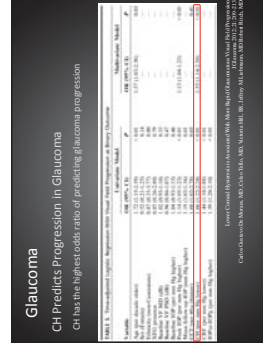
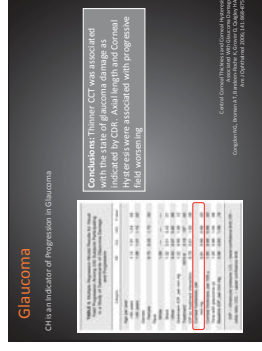
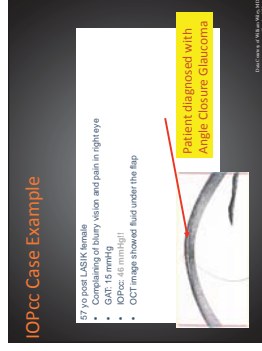
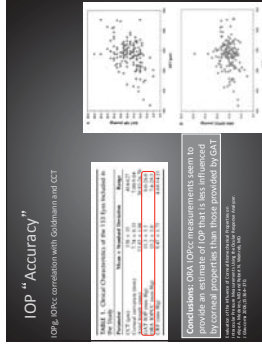
IOPcc has essentially zero correlation with CCT in normal eyes and stays relatively constant post-LDk.

IOPcc has been shown to be more associated with progressive field loss than CCT.

IOPcc = IOP - (0.43 * CH)



A Summary of ORA Clinical Findings



- ### 48 YO HF
- CH OD: 7.3 OS: 6.9
 - IOP G OD: 19 mm Hg IOPcc OD: 25 mm Hg
 - IOP G OS: 17 mm Hg IOPcc OS: 2
 - Patient switched from Xal to Lumigan
 - IOP at 2 wks after switch
 - IOPcc OD=12mm Hg
 - IOPcc OS=13mm Hg
 - IOP 3 months after switch
 - IOPcc OD=9mm Hg
 - IOPcc OS=11mm Hg

48 YO HF Summary

- AGIS 7 asserts that IOP reduction correlates with visual field preservation.
- Low teens preserve visual field better than upper teens
- Fewer medications improve compliance

NEXT ...

Fuch's Dystrophy

- Autosomal dominant inheritance
- Bilateral / Asymmetry
- Late onset > 50 y.o.
- Females affected 3 times more than males
- ~5.7% develop edema
- Characterized
 - Corneal guttata
 - Excessive accumulation of abnormal endothelial secretions
 - Appears in 3rd/4th year of life

Fuch's Dystrophy

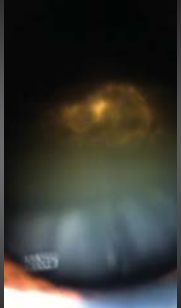
- Characterized
 - Corneal Guttata
 - Small, white, "peppercorn" deposits on corneal endothelium
 - Affects the "pump" action of the endothelium
 - Edema
 - Greater in the AM
 - Occurs as day gets on
 - Excessive fluid slowly lead to corneal swelling
 - RCG's common

Case Study

- 67 year old WM
- "My vision is not good...I have blurred vision. My eyes cry a lot too. They cry all the time."
- NIDDM (diet controlled 15 years)
- NKMA
- History of skin lesion removed from cheek

Case Study

- VACC
 - 20/30 (PH-20/20) OD
 - 20/100 (PH-20/70) OS
- SLEX
 - 2+ guttae-OU
 - Mild pigment on endo-OU
 - 1/2+ NSC/Tx; PSC-OD
 - 2+NSC/2+ PSC-OS



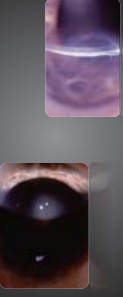
Cataract Surgery OS

- VASC
 - 20/100
 - 2-3+ Striae
 - 3+ POE
 - 1+ cells
 - Lens centered
 - IOP
 - * 14 mmHg (OIA)

Fuch's Dystrophy

- Symptoms vary with degree of guttata and compromise of the endothelial tissue
- Moderate guttata
 - May affect visual function
 - May induce mild-moderate edema
 - Hazy vision > a.m.
 - Halos around lights
- Severe guttata
 - Vision decreases
 - Possible bullous develops

FUCH'S DYSTROPHY



Fuch's Dystrophy

- Treatment
 - Early stages of disease
 - Increase artificial tears
 - Hyperosmotic q/s
 - RCL used if bullous is present
 - EDUCATION!
 - Visual function is significantly compromised
 - Penetrating keratoplasty
 - Deep Lamellar Endothelial Keratoplasty (DLEK)
 - Automated Stripping Automated Endothelial Keratoplasty (ASAEK)

Fuch's Dystrophy



Fuch's Dystrophy

- DLEK
 - recipient cornea is stripped of Descemet's membrane and endothelium
 - Transplantation of donor cornea through small incision
 - Results in
 - improve endothelial function, corneal clarity and restores vision
 - Minimally affects refraction
 - Can provide rapid visual recovery
 - Maintains structural integrity of the cornea

Case Study

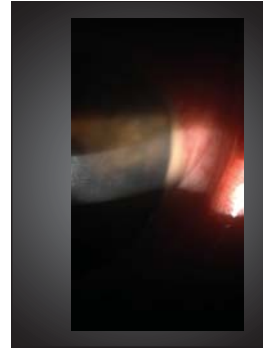
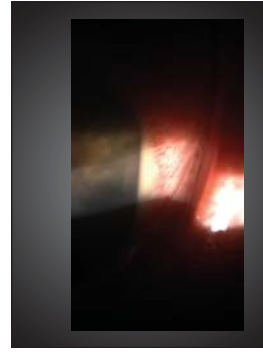


- 6 months PO
 - VACC
 - 20/30 OS
 - Slit
 - Well centered lens
 - 3+ guttata
 - Mild pigmentendo
 - IOP - 5 mmHg (OIA)

Case Study

- 13 months later
 - Patient calls....
 - "Sorry to bother you on a Sunday, but my eye is blurry today and it hurts a little."
 - "I have seen the corneal surgeon recently and he said my cornea looked good."
 - "I was 20/30. Today it is not so great."
 - SO.... I head into see patient...

CASE STUDY

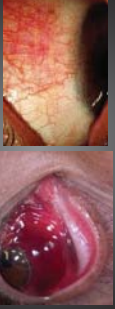
Case

I am seeing RED

Red Eye Day

- Case 2
 - 52 yo female
 - Contact lens wearer
 - Red eye x 3 hours
 - No discomfort, but feels dry
 - VA unaffected
 - No systemic meds other than OTC allergy
- 45 yo female
 - Uncomfortable eye
 - On and off x 8 months
 - Positive allergy history
 - No systemic meds other than OTC allergy
- Overall good health

What do these have in common?



Conjunctival Redundancy
Conjunctivochalasis

- Defined as a redundant, loose nonvascularized inferior conjunctiva
- Located between the globe and inferior eyelid
- Conjunctival folds lying along the inferior lid margin
- Some proposed causes:
 - Aging
 - Ocular surface inflammation
 - Dehydrated tear film (cause or symptom?)
 - More common in females
 - Co-existing pingueculae
 - More common in blepharitis patients
- Patients are usually asymptomatic, however correct diagnosis is crucial in symptomatic patients

Redundant Conjunctival Folds



Conjunctival Redundancy
Conjunctivochalasis

CONJUNCTIVOCALASIS: A Matter of Timing and Management.

Lee JF

JAMA Ophthalmology

Abstract

Conjunctivochalasis is a common ocular surface condition characterized by loose and redundant inferior conjunctiva. It is most likely a result of conjunctival aging, but the exact pathogenesis is unclear. It is often associated with conjunctivitis, dry eye, and contact lens intolerance. This is the first case report of conjunctivochalasis in a patient with a history of blepharitis. The patient presented with a history of conjunctivitis and dry eye. The patient was treated with topical antibiotics and artificial tears. The patient's symptoms improved with treatment. The patient's conjunctivochalasis was managed by thorough conjunctival debridement. The patient's symptoms improved with treatment. The patient's conjunctivochalasis was managed by thorough conjunctival debridement.

Key words: conjunctivochalasis, conjunctivitis, dry eye, blepharitis, conjunctival debridement.

Conjunctival Redundancy Conjunctivochalasis

Clinical Ophthalmology

Clinical characteristics of patients with conjunctivochalasis

DEFINITION
Redundancy of the conjunctiva, which may be associated with conjunctivochalasis.

ETIOLOGY
The etiology of conjunctival redundancy is unclear. It is thought to be a result of degenerative changes in the conjunctiva, possibly related to aging or chronic inflammation.

CLINICAL PRESENTATION
Patients with conjunctival redundancy may present with a variety of symptoms, including tearing, irritation, and blurred vision. The condition is often asymptomatic and may be discovered incidentally during an eye examination.

DIAGNOSIS
The diagnosis of conjunctival redundancy is based on a clinical examination of the eye. The characteristic finding is a redundant fold of conjunctiva that may or may not be associated with conjunctivochalasis.

TREATMENT
Treatment is typically conservative and includes artificial tears and lubricating eye drops. In severe cases, surgical resection of the redundant conjunctiva may be necessary.

Conjunctival Redundancy Conjunctivochalasis

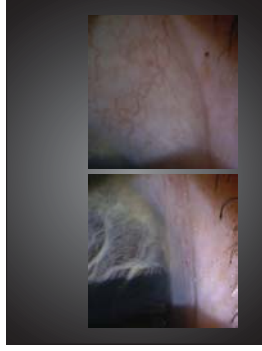
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ALL YOU SEE IS THE TIP OF THE ICEBERG

Ocular Surface

Eyelid & Fornix

Loose conjunctival tissue due to degenerated Tenon's

Anatomical Tear Reservoir

A TEAR RESERVOIR TRANSILATES TO A DRY OCULAR SURFACE

How Is Tear Flow Interfered by CCh?

Why are some CCh patients NOT symptomatic?

- 1. Location of the fold
- 2. Depth of the fold
- 3. Size of the fold
- 4. Shape of the fold
- 5. Thickness of the fold

Go to the Source:

Hydrodynamic Measurement that Defines the Ocular Surface

FORNIX

MENISCUS

OCULAR SURFACE

Conjunctival Redundancy Conjunctivochalasis

- Symptoms:**
 - Tearing/epiphora- especially with nasal folds due to blockage of punctum
 - Dryness*
 - FBS
 - Redness
 - Eye pain
 - Blurry vision, especially in down gaze

What Are Unique Clinical Features of Dry Eye Caused by CCh?

Distiguishing Feature	ATD Dry Eye	CCh Dry Eye
Symptoms	Worse in the morning	Worse throughout the day
Visual Disturbance	Symptoms improved	Symptoms worsened
Effect of Artificial Tears	Improvement	Prognosis worsened
Phenol/Alcohol Slit-Lamp	Low tear meniscus without conjunctivochalasis	Low tear meniscus with conjunctivochalasis
Slit-Lamp	Normal/Healthy	Frequently abnormal
Best Overall Finding	Epiphora zone	Non-epiphora zone
Effect of Punctal Occlusion	Symptoms improved	Symptoms worsened

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Conjunctival Redundancy Conjunctivochalasis

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**Conjunctival Redundancy
Conjunctivochalasis**

Conjunctival redundancy is a common clinical finding that may progress to conjunctivochalasis with age-related photorefractive changes.

Treatment:

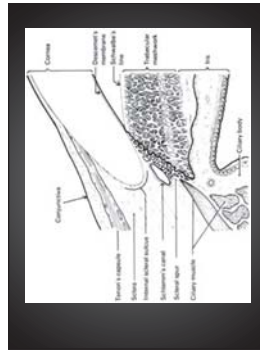
- Lubricants
- Antihistamines
- Topical Steroids
- Surgical resection of the conjunctiva

**Conjunctival Redundancy
Conjunctivochalasis**

- Treatment:
 - Lubricants
 - Antihistamines
 - Topical Steroids
 - Surgical resection of the conjunctiva

Why is CCh Refractory to Conventional Dry Eye Treatments?

- **Fornix Obliteration by CCh**
- Cannot hold patient's own tears
- Seldom benefit from artificial tears
- Easy to generate "overflow"
- Re-tastasis™ is not helpful



Conclusion

- CCh is an overlooked cause of dry eye.
- CCh dry eye differs from ATD dry eye in diurnal variation, symptoms changed by gazes and blinking.
- Symptomatic CCh is due to (1) interference of tear meniscus, (2) punctal drainage and (3) interference of tear flow from the fornix to the meniscus.
- Surgical treatment should be directed to deepening the fornix with AMT.

Key Surgical Steps
Conjunctivochalasis is a disease of the fornix.

- Key Characteristics of CCh:**
 - Observed and diagnosed by the fornix and its pocket
- Treatment Strategy:**
 - Deepen the fornix with one layer of Amniograft®
- Key Characteristics of CCh:**
 - Observed and diagnosed by the fornix and its pocket
- Treatment Strategy:**
 - Fornix excision to deepen the fornix and restore the normal anatomy of the fornix

