2017 NORTHWEST RESIDENTS CONFERENCE
Pacific University College of Optometry – Jefferson 224
Friday, June 9 & Saturday, June 10, 2017

FRIDAY, JUNE 9, 2017		PAGES	
Kristin Yandrich, OD Lebanon VA Medical Center	**1:00-1:30**	**1-10**	Proliferative Retinopathy Secondary to Cryoglobulinemia Associated with Retinal Vasculitis in Hepatitis C
Katie Dailey, OD VA Puget Sound Health Care System	**1:30-2:00**	**11-29**	Acute Syphilitic Posterior Placoid Chorioretinitis: A Case Report and Disease Review
Timothy Mock, OD Mann-Grandstaff VA Medical Center	**2:00-2:30**	**30-39**	Spontaneous and Vision Threatening Choroidal Neovascular Nets: An In-Depth Presentation
Kerri Norris, OD Jonathan Wainwright Memorial VAMC	**2:30-3:00**	**40-52**	Who Turned Out the Lights: Embolic Causes of Transient Monocular Visual Field Loss and a Review of Most Valuable Diagnostic Testing
BREAK – **GRAB AND GO SNACK IN HALLWAY**	**3:00-3:30**		
Dayna Yim, OD VA Portland Health Care System	**3:30-4:00**	**53-62**	A Case Series on Neovascular Glaucoma
Prajkta Ingle, OD VA Roseburg Health Care System	**4:00-4:30**	**63-73**	The Effects of Vitreopapillary Traction at the Optic Nerve when Interpreting Optical Coherence Tomography
Dane Sultzer, OD Mann-Grandstaff VA Medical Center	**4:30-5:00**	**74-88**	Acquired Iris and Angle Abnormalities
Alanna Louie, OD VA Portland Health Care System	**5:00-5:30**	**89-98**	What “Liths” in the Lacrimal System?
Stephanie Ivor-Smith, OD ICON Surgical Center	**5:30-6:00**	**99-107**	Uveitis Savvy
Alise Gentry, OD VA Portland Health Care System	**6:00-6:30**	**108-114**	Ocular Cicatricial Pemphigoid: A Case Report and Review

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SATURDAY, JUNE 10, 2017		PAGES	
Emily Bucher, OD Jonathan Wainwright Memorial VAMC	**9:00-9:30**	**115-124**	Pathophysiology of Retinopathy – Beyond the Basics
Marc Harrie, OD Chu Vision Institute	**9:30-10:00**	**125-135**	IntraCorneal Inlays for Presbyopia Diplopia
Hannah Holtorf, OD (Peds/VT) Pacific University and Associated Clinics	**10:00-10:30**	**136-144**	Diplopia Management in Patients with Parkinson's Disease
Justin Burgerson, OD Mann-Grandstaff VA Medical Center	**10:30-11:00**	**145-153**	Asymptomatic VF Loss: The Case of a Failed DMV Test
Emily Korszen, OD (Cornea & CL) Pacific University and Associated Clinics	**11:00-11:30**	**154-161**	Do Modern Scleral Lenses Provide Adequate Oxygen to the Cornea?
Rebecca Lee, O VA Portland Health Care System	**11:30-12:00**	**162-169**	Prosthetic Contact Lenses: More than Cosmesis
BREAK – **GRAB AND GO LUNCH IN HALLWAY**	**12:00-12:30**		
Kelsey Sieg, OD Vision Northwest	**12:30-1:00**	**170-173**	Divergence Excess Treatment Approaches
Jamie Bergmark, OD Bright Eyes Vision Clinic	**2:00-1:30**	**174-185**	The Role of Vision Therapy in Neuro-Optometric Rehabilitation
Cara Sczepanski, OD Northwest Eye Care Professionals	**1:30-2:00**	**186-196**	Case Presentation and Management of Constant Alternating Exotropia with Vision Therapy
Josh Clermont, OD VA Puget Sound Health Care System	**2:00-2:30**	**197-215**	Peripapillary Retinoschisis: A Case Report and Review of the Literature
Sajal Sudhir Patel, OD VA Southern Oregon	**2:30-3:00**	**216-226**	Sickle Cell Retinopathy: A Case Study
Ben Jager, OD VA Portland Health Care System	**3:00-3:30**	**227-235**	Optical Coherence Tomography Findings in Acute and Chronic Branch Retinal Artery Occlusions in a Single Patient

Proliferative Retinopathy Secondary to Cryoglobulinemia Associated with Retinal Vasculitis in Hepatitis C

Kristin N. Yandrich, OD
Optometry Resident
Lebanon VA Medical Center
Lebanon, Pennsylvania

Learning Objectives

- Overview of Hepatitis C
- Define cryoglobulinemia
- Identify ophthalmic manifestations
- Review diagnoses and treatment

Hepatitis C

- Widespread infectious disease
- Blood borne virus affecting the liver
- 3.6 million in United States
- 170 million worldwide
- Ocular manifestations
  - Dry eye
  - Ischemic retinopathy
  - Vasculitis
  - Mooren's Ulcer

Hepatitis C Transmission

- Intravenous drug use
- Inadequate sterilization of medical equipment
- Transfusion of unscreened blood
- Mom to baby via birth

Hepatitis C Treatment

- No vaccine
- Chronic Hepatitis C has up to a 90% cure rate
- Therapy
  - Interferon
    - 6 months to a year
    - 40-50% cure rate
    - Side effects
  - Sovaldi (Sofosbuvir)
  - Olysio (Simeprevir)
  - Harvoni (Ledipasvir/Sofosbuvir)

Case Presentation

- 54 year old white male
- Presents for diabetic eye examination
- No symptoms

Ocular and Medical History

- Ocular history
  - Mild cataracts
- Recent lab work
  - Unremarkable
- Social history
  - Smoker
- Medical history
  - Type II Diabetes
  - Thrombocytopenic Disorder
  - Coronary Arteriosclerosis

- Current Medications
  - Atorvastatin
  - Aspirin
  - Lisinopril
  - Insulin
  - Glipizide
  - Metformin
- Successfully completed DAA treatment with Harvoni

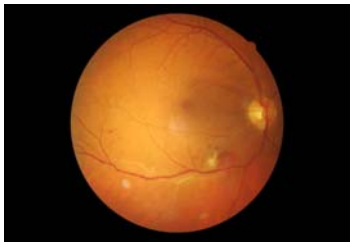
Examination

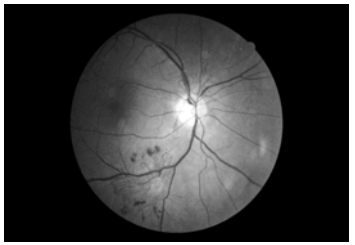
- SC 20/30 OD and 20/25 OS
- Entrance testing
  - Unremarkable
- Anterior segment examination
  - Unremarkable

Dilated Fundus Examination

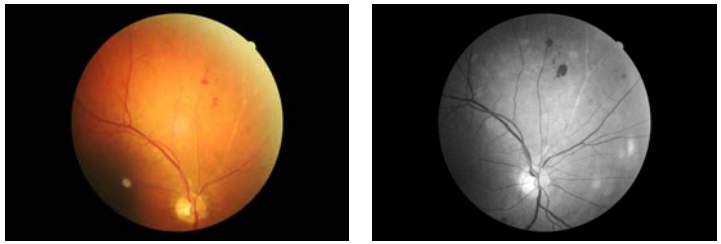
- Optic nerves
  - Perfused with distinct margins
- Macula
  - Clear and flat
- Periphery
  - Clear, no holes/breaks/tears

Right Eye

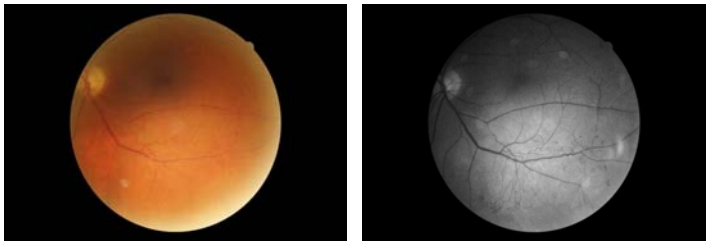




Right Eye



Left Eye



Differentials

- Diabetes
- Hypertension
- Carotid Blockage/OIS
- Toxoplasmosis
- Sarcoid
- Lupus
- Sjogren's
- Bechet's
- Syphilis
- Tuberculosis
- HIV
- Herpetic
- Wegener's Granulomatosis
- Cryoglobulinemia

One Month Follow Up

- Retina findings stable
- Patient remains asymptomatic
- Targeted lab work up ordered
- Rheumatology consulted
- Patient to follow up in one week for fluorescein angiography

Lab Testing

- HgbA1c Level
  - Mildly elevated
    - 7.5
  - Short duration
    - 2013
- Blood Pressure
  - 133/80
- Carotid Ultrasound and Cardiac Echogram
  - No significant stenosis
- Toxoplasmosis
  - Negative
- HIV
  - Negative

- Sed Rate
  - 18mm/hr (reference range 0-20)
- CRP
  - 0.063mg/dL (reference range 0-.747)
- Chest X-Ray
  - Unremarkable
- RPR and PPD
  - No syphilis or tuberculosis
- CBC
  - Lymphocytes, monocytes, neutrophils, eosinophils, basophils, WBC, RBC, platelets
- HCV RNA
  - Copy number by PCR was undetectable
  - No further antiviral treatment warranted

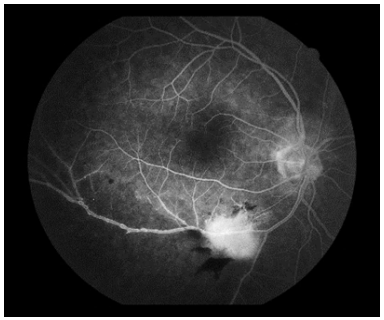
Rheumatology

- Ruled out by clinical exam
  - Wegener's
  - Lupus
  - Sjogren's
  - Bechet's

Pertinent Lab Findings

- Marginally elevated serum cryoglobulins
- Positive Rheumatoid Factor
- Positive Antinuclear Antibody
- Decreased Complement C4
- HCV RNA

IVFA Right Eye



Late phase fluorescein angiography showing nonperfusion of inferior temporal vessel with active neovascularization along vessel arcade

Vasculitis

- Sight threatening condition
- Inflammation of retinal vasculature
- Necrosis of endothelial layer
- Poor perfusion, ischemia and organ damage

Vasculitis in the Retina

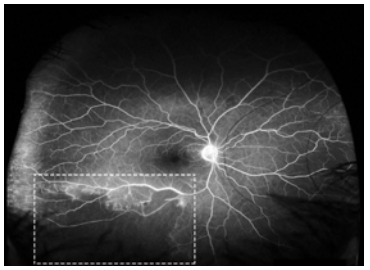
- Swelling
- Exudation
- Macular edema
- Ischemia
  - Cotton wool spots
  - Intraretinal hemorrhages
- Vein occlusion
  - Neovascularization
  - Vitreous hemorrhage
  - Tractional retinal detachment
  - Secondary glaucoma

- Presenting manifestations in the retina can be signs of active and potentially deadly concomitant systemic disease

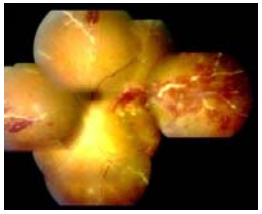
Causes of Vasculitis

- Idiopathic
- Neoplastic
  - Ocular Lymphoma
  - Acute Leukemia
- Infectious
  - Bacteria
    - Tuberculosis
    - Syphilis
    - Lyme
  - Rickettsia
    - Rocky Mountain Spotted Fever
  - Parasitic
    - Toxoplasmosis
  - Viral
- Autoimmune
  - Primary
    - Wegener's Granulomatosus
    - Polyarteritis Nodosa
    - Churg- Strauss
  - Secondary
    - Sjogren's
    - Lupus
    - Rheumatoid Arthritis
    - HLA-B27 associated uveitis

- Occlusive Vasculitis



- Inflammatory Vasculitis
  - Sheathing
  - Cuffing



Causes of Occlusive Vasculitis

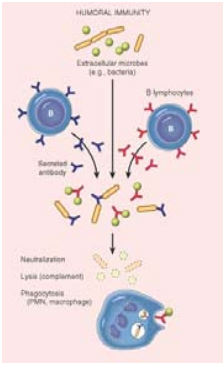
- Tuberculosis
- Eale's Disease
- Systemic Lupus Erythematosus
- Multiple Sclerosis
- Varicella Zoster Uveitis
- Cryoglobulinemia
- Syphilis

Symptoms of Vasculitis

- Blurring of vision
- Flashes
- Floaters
- Scotoma
- Altered color vision
- Metamorphopsia
- Pain

Review of Immunology

- Antibody or Immunoglobulin
  - Y shaped protein
  - Recognizes foreign material
- Antigen
  - Harmful agent
  - Bacteria or virus
  - Capable of eliciting an immune response
- Immune complex
  - Antibody binds with antigen
- Complement system
  - Cascade
  - Phagocytes
  - Clear materials



- Hepatitis C can generate cryoglobulins resulting in a vasculitis by way of immune complex deposition
- Immune complexes
  - Antigen/Antibody
  - Can cause illness when deposited in organs
  - Seen in vasculitis

Cryoglobulins

- Circulating immunoglobulins
  - IgG and IgM
  - Abnormal antibodies
- Precipitate in cold (<37°C)
  - Cold insoluble
  - Precipitate from the serum
  - Act as immune complexes
  - Deposit on endothelium causing a vasculitis
  - Resolubolize when warm

Cryoglobulinemia Vasculitis

- Affects small to medium sized vessels
- Characterized by the clonal expansion of RF –expressing B-cell lymphocytes
  - Liver
  - Lymph nodes
  - Periphery
- HCV is noted to be causative agent in over 80% of cases
- Lab work
  - Positive RF
  - Positive cryoglobulins
  - Low C4

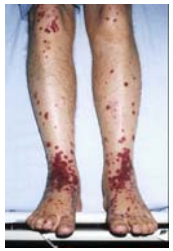
Subsets of Cryoglobulinemia

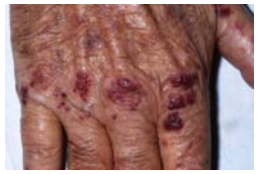
- Type I
  - Monoclonal cryoglobulinemia
  - Monoclonal IgG
  - Hematologic disorders
- Type II
  - Mixture of monoclonal IgM and polyclonal IgG
- Type III
  - Polyclonal
  - IgM and polyclonal IgG
  - No monoclonal component
- Mixed

Mixed Cryoglobulinemia

- Hepatitis C viral infection is the main cause
  - Found in 86% of people with cryoglobulinemia vasculitis
- Symptoms very greatly
  - Mild purpura
  - Life threatening glomerulonephritis and widespread vasculitis
  - Precipitated clumps can block blood vessels in extremities causing gangrenous

- Classic systemic triad
  - Palpable purpura
  - Arthralgia
  - Weakness





Back to the Case…

- Patient followed up 4 months later
- Diagnosis
  - Bilateral Proliferative Retinopathy
    - Secondary to occlusive vasculitis
    - Associated with HCV-induced cryoglobulinemia
- Treatment
  - Scatter laser to areas of ischemic retina OU
  - No macular edema
- No systemic treatment initiated
- Monitor

In Summary

- Occlusive Vasculitis + HCV Infection =systemic work up

- Or

- Purpura + Weakness + Arthralgia/Arthritis = dilated fundus examination

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Questions?

Acute syphilitic posterior placoid chorioretinitis in a heterosexual female: a case report and literature review

Kathryn Dailey, OD
VA Puget Sound Health Care System
American Lake Division

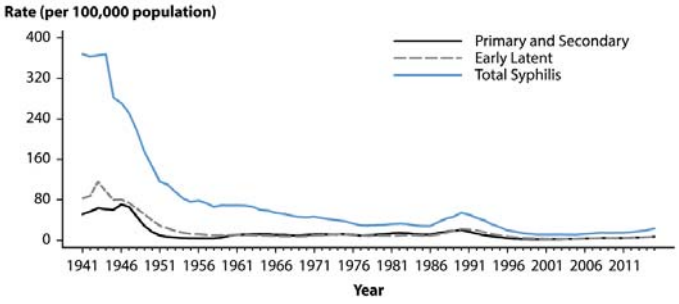
Syphilis

- Sexually transmitted infection caused by the spirochete bacterium *Treponema pallidum* that causes systemic and ocular disease

History of syphilis

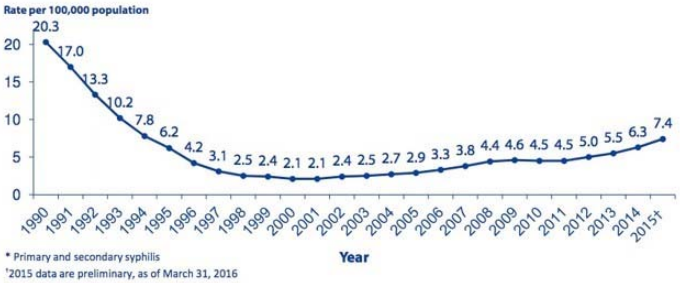
History of syphilis

Rates of Reported Cases by Stage of Infection, United States, 1941–2015[1]



Syphilis today

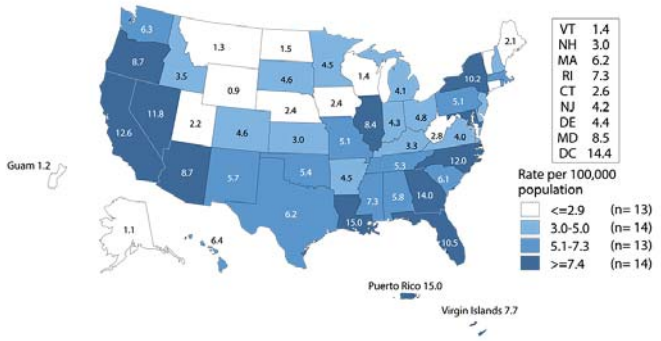
Rates of Primary and Secondary Syphilis, 1990-2015[1]



CDC reports highest prevalence of acquired syphilis in 2015 since 1994

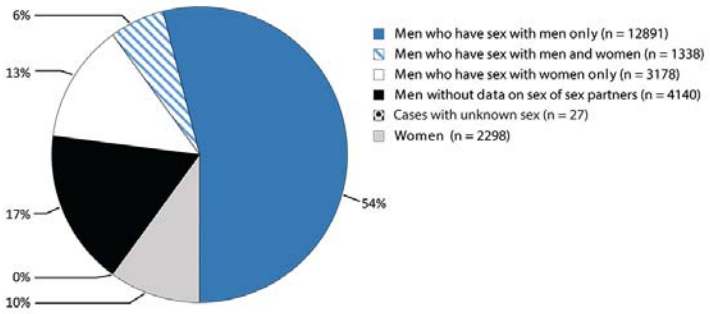
Syphilis today

Primary and Secondary Syphilis — Rates of Reported Cases by State, United States and Outlying Areas, 2015[1]



Syphilis today

Primary and Secondary Syphilis — Distribution of Cases by Sex and Sexual Behavior, 2015[1]



CDC Clinical Advisory

Clinical Advisory: Ocular Syphilis in the United States

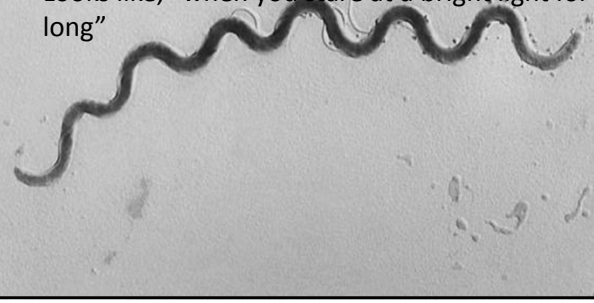
Updated March 24, 2016

Between December 2014 and March 2015, 12 cases of ocular syphilis were reported from two major cities, San Francisco and Seattle. Subsequent case finding indicated more than 200 cases reported over the past 2 years from 20 states. The majority of cases have been among HIV-infected MSM; a few cases have occurred among HIV-uninfected persons including heterosexual men and women. Several of the cases have resulted in significant sequelae including blindness.

- 12 cases of ocular syphilis in San Francisco and Seattle
- Mainly HIV+ MSM, but some HIV- men and women

Case Report

- 46yo white female
- CC: black spot in her central vision OS
  - Appeared upon awakening the day before
  - Looks like, "when you stare at a bright light for too long"



Case Report

- Ocular history: unremarkable
  - LEE one year prior at a different clinic
  - OTC readers only
- Other notable history
  - Swollen abdominal lymph nodes after a recent trip to Mexico
  - High levels of stress after recent death of her best friend
  - Denied recent cold/flu and neuro symptoms

Case Report

Medical history	
PTSD	Anxiety
Chronic neck pain	Gestational diabetes
GERD	

Current medications	
Methocarbamol	Omeprazole
Oxybutynin chloride	Venlafaxine

Case Report

- Entrance testing:

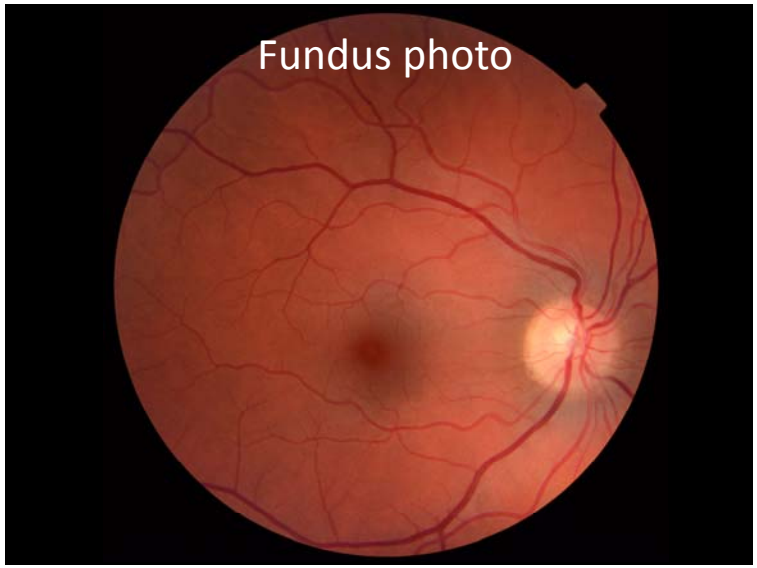
Test	OD	OS
Visual acuity (sc)	20/20	$20/70^{+2}$ PH 20/NI
EOMs	Full	Full
Pupils	RRL, (-) APD	RRL, (-) APD*
Confrontation VF	FTFM	FTFM
Amsler grid	Normal	Large central scotoma

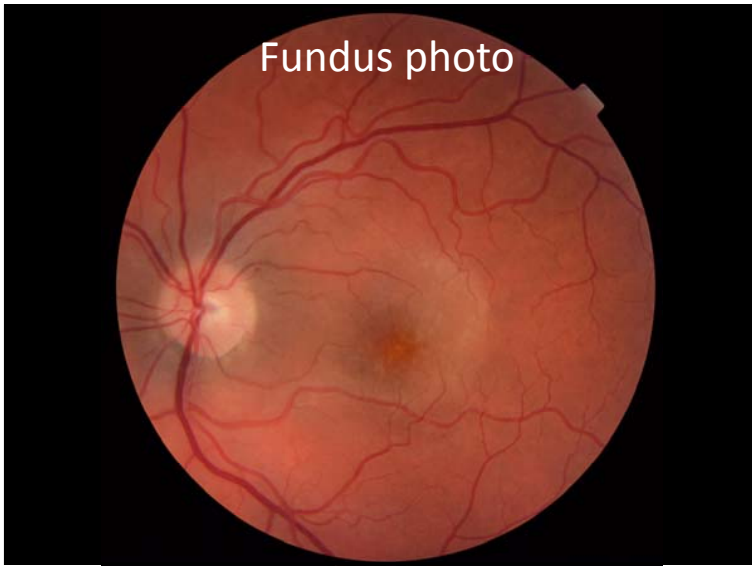
- IOP (OD/OS): 16/15mmHg via NCT
- Anterior segment: unremarkable with deep and quiet anterior chambers OU

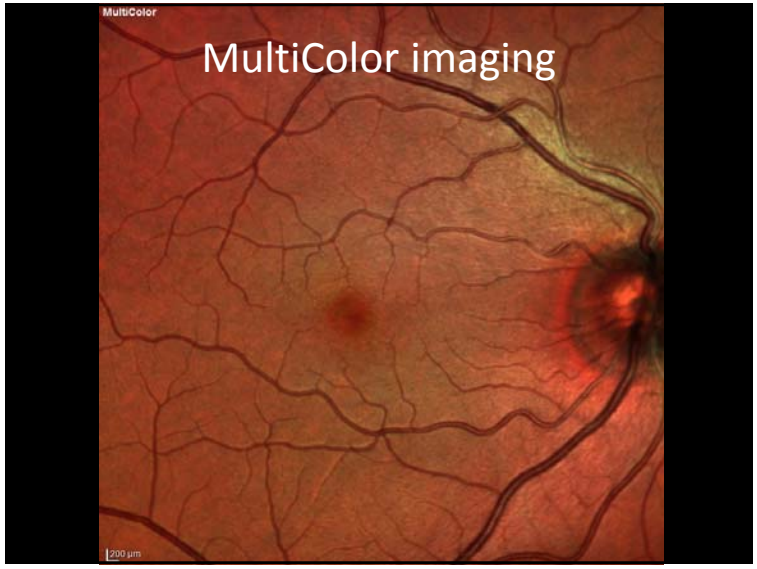
Case Report

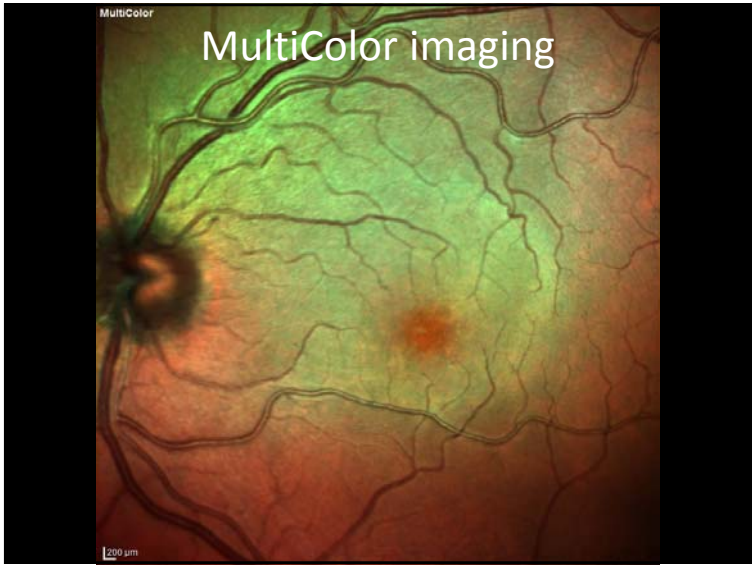
- Posterior segment

	OD	OS
Lens	Tr NS	Tr NS
Vitreous	Tr cell, (-) PVD	1+ cell, (-) PVD
Optic nerve	Distinct margins c/d 0.4R	Distinct margins c/d 0.4R
Macula	Flat and dry	Mottled with a deep, yellow, plaque-like lesion extending superiorly in to the posterior pole
Vessels	Unremarkable	Unremarkable
Periphery	Flat and intact 360	Flat and intact 360


Fundus photo


Fundus photo


MultiColor
MultiColor imaging
200 µm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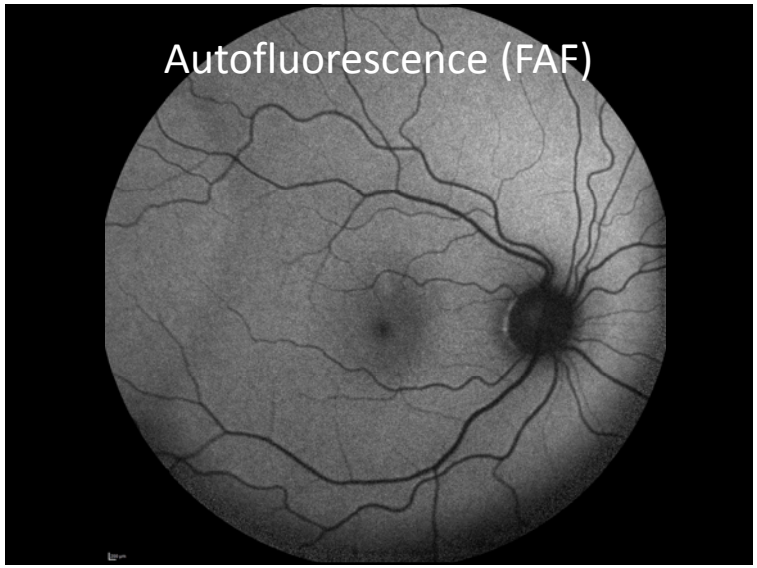
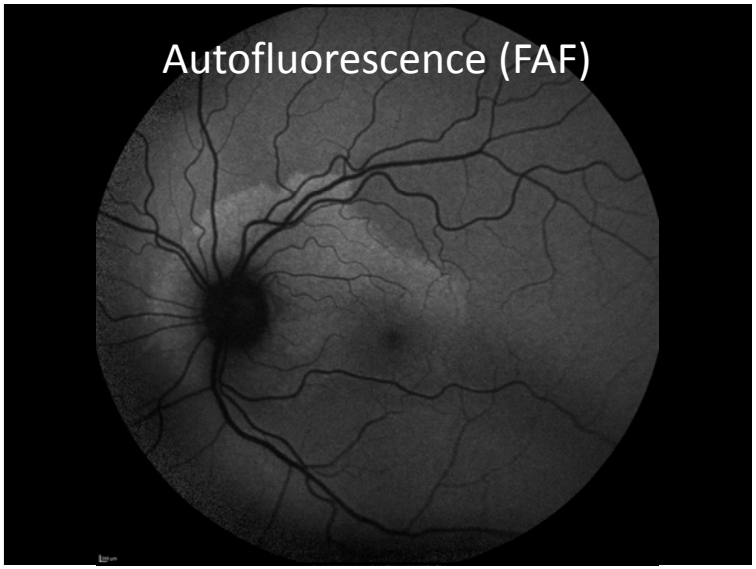
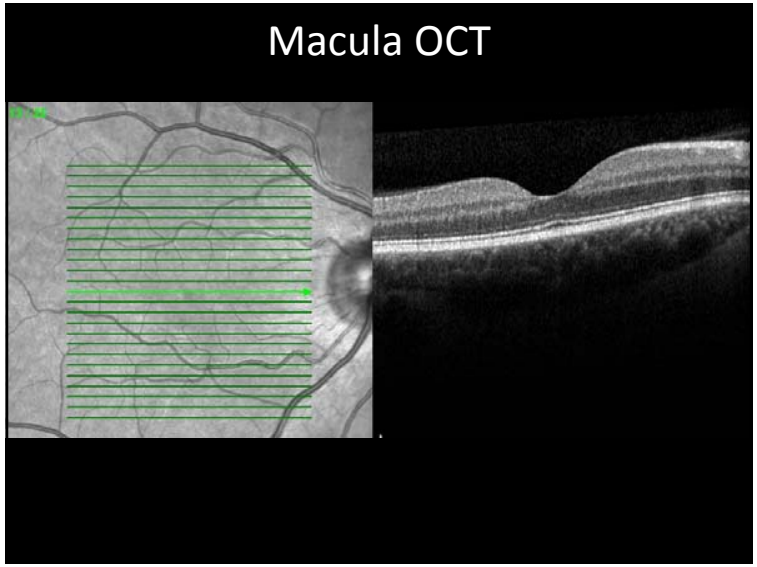
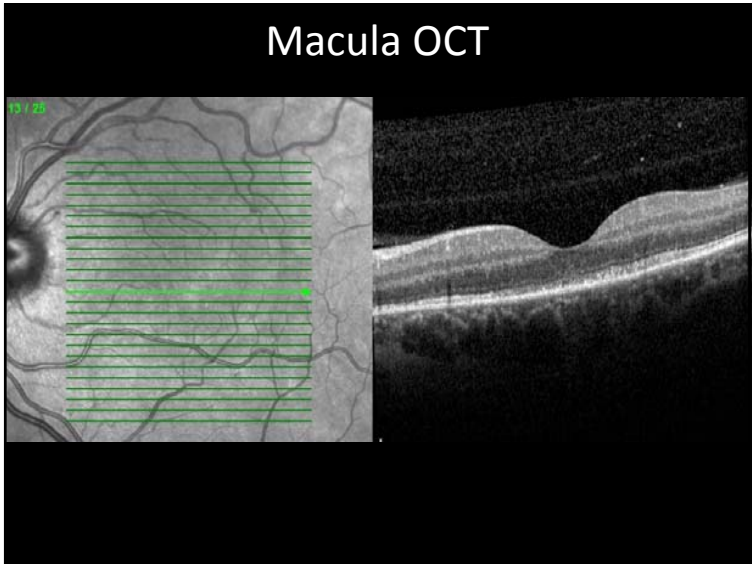

Autofluorescence (FAF)

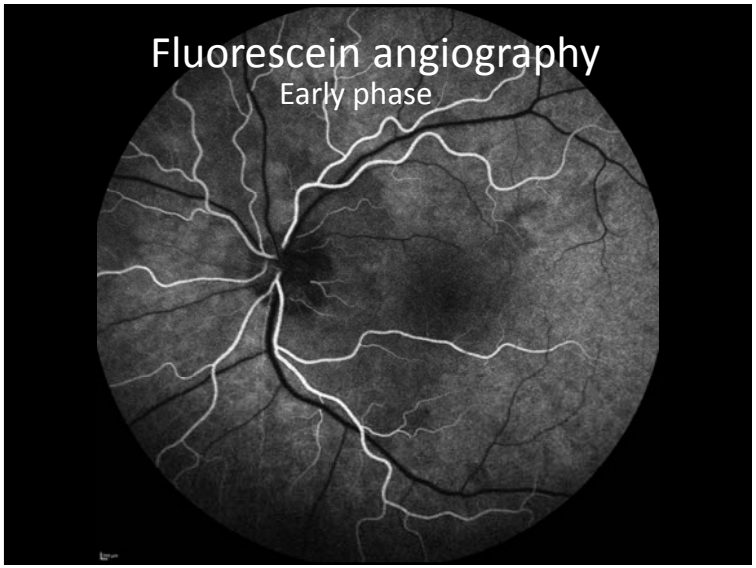

Autofluorescence (FAF)

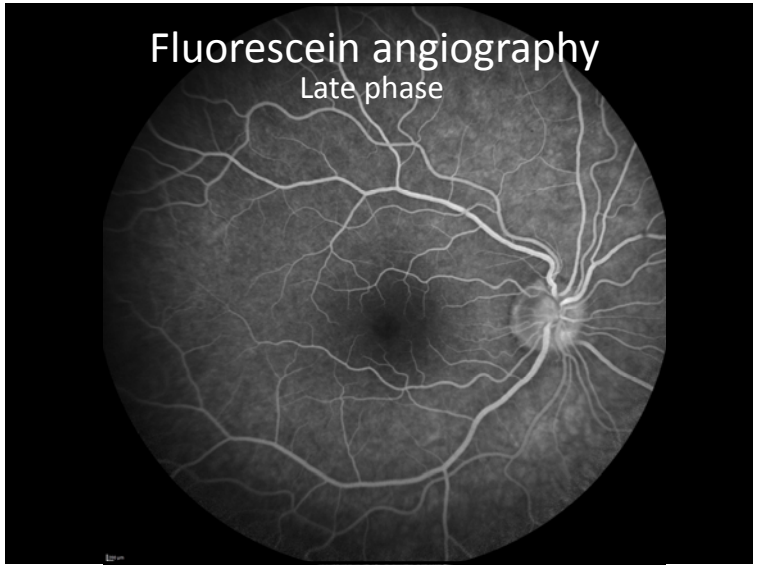

Macula OCT

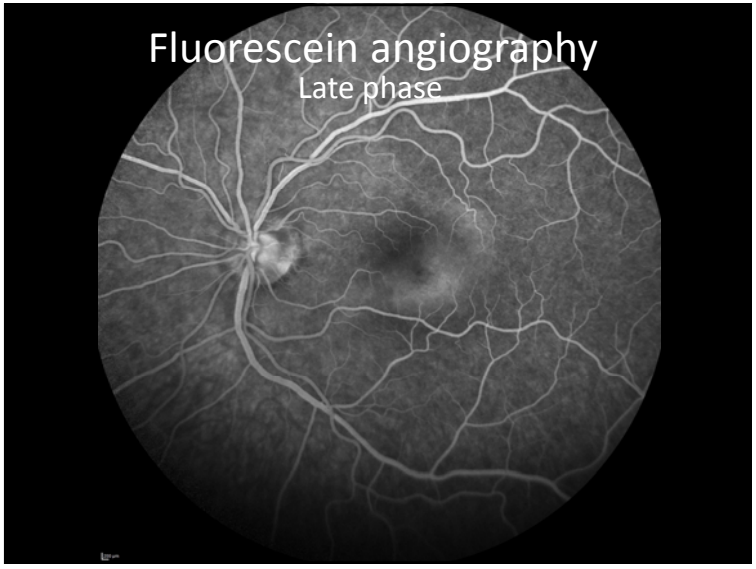

Macula OCT

Case Report

- Off to Retina…
- Additional history
  - Oral ulcers since August 2016







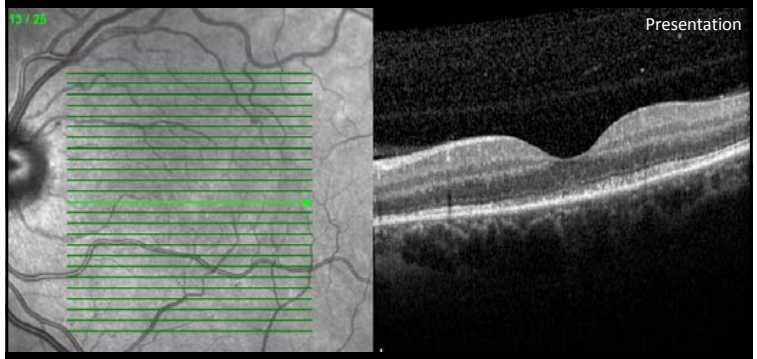
Management

- Ddx: posterior uveitis of unknown etiology
- Labs
  - CBC
  - Metabolic panel
  - Syphilis serology
  - QuantiFERON test for TB
  - ACE levels
  - Toxocariasis serology
  - Toxoplasmosis serology
  - Chest X-ray
- Follow-up scheduled for 3 days

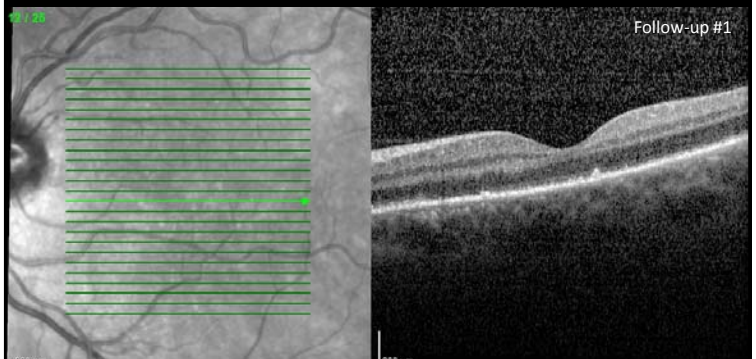
Follow-up #1

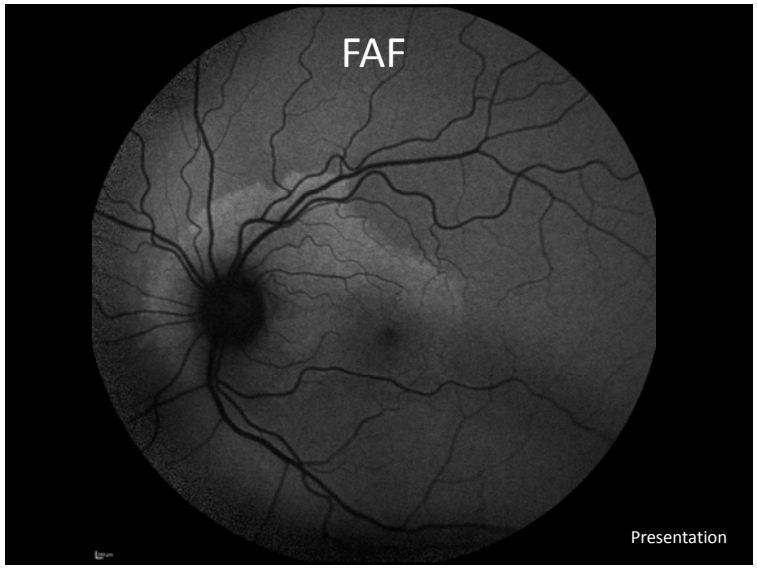
- CC: improved vision OS due to smaller black spot
- VA 20/30 OS
- No change in fundus appearance OU

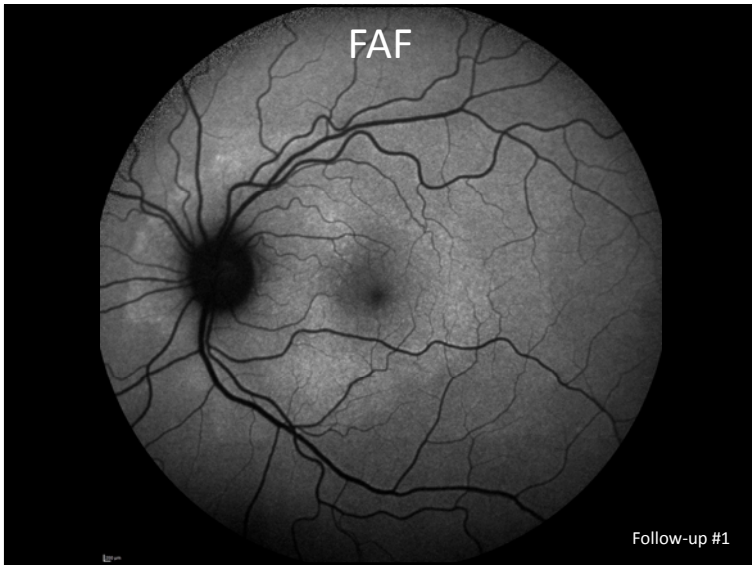
Macula OCT



Macula OCT







Lab results

- All normal EXCEPT syphilis serology
  - (+) Enzyme immunoassay (EIA) confirmed exposure
  - (+) confirmatory testing with Rapid Plasma Reagin (RPR) and *T. pallidum* particle agglutination assay (TPPA)
    - RPR titer of 1:512 confirms active disease
- Negative HIV, chlamydia, gonorrhea

Diagnosis

- Assessment
  - Ocular: Acute Syphilitic Posterior Placoid Chorioretinitis (ASPPC)
  - Systemic: Syphilis
- Plan

Ocular syphilis = neurosyphilis → Infectious Disease → Internal Medicine Lumbar puncture

Internal Medicine

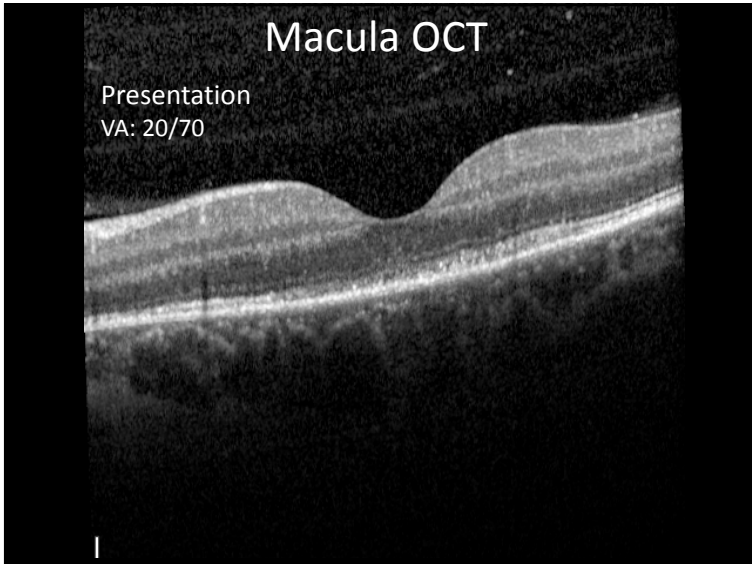
- Extensive social history gathered during examination prior to lumbar puncture:
  - Reported a current promiscuous sexual partner who had a small, round, painless lesion on his genitals for the past month
  - Several painful ulcers on her tongue and the inside of her lower lip
  - Intentional weight loss of 35 pounds by hypnosis

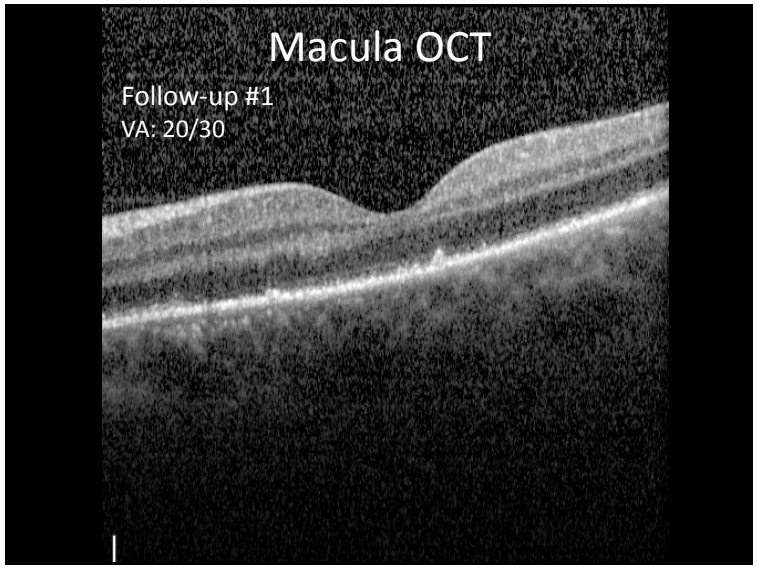
Treatment

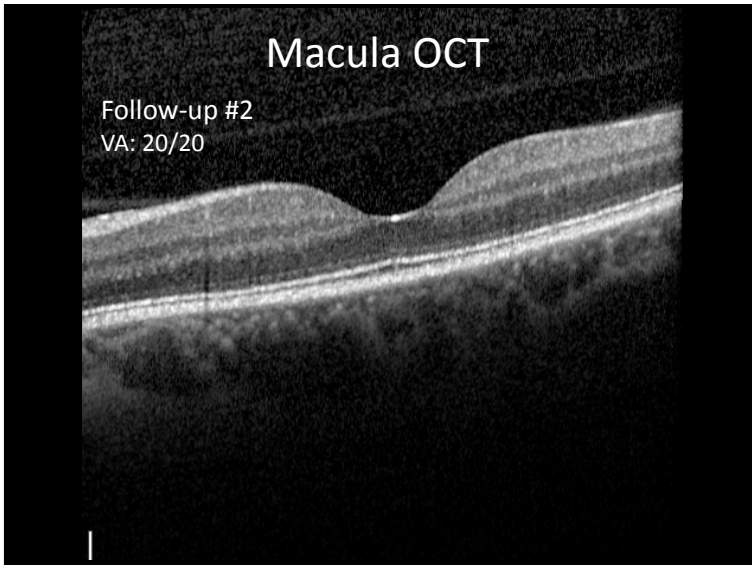
- (-) CSF-VDRL, but still considered neurosyphilis
- (+) history of severe penicillin allergy
- IV Ceftriaxone
  - 2g/day x 14 days via PICC line
- Observed in the hospital for her first treatment

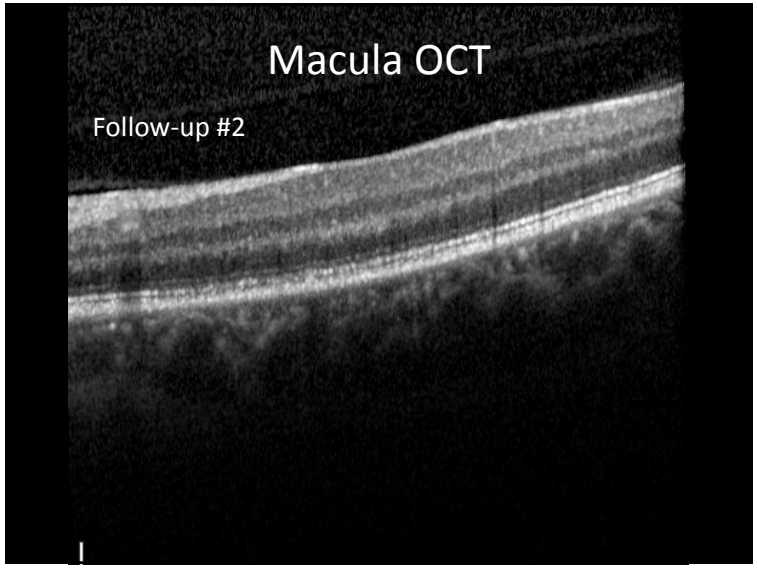
Follow-up #2

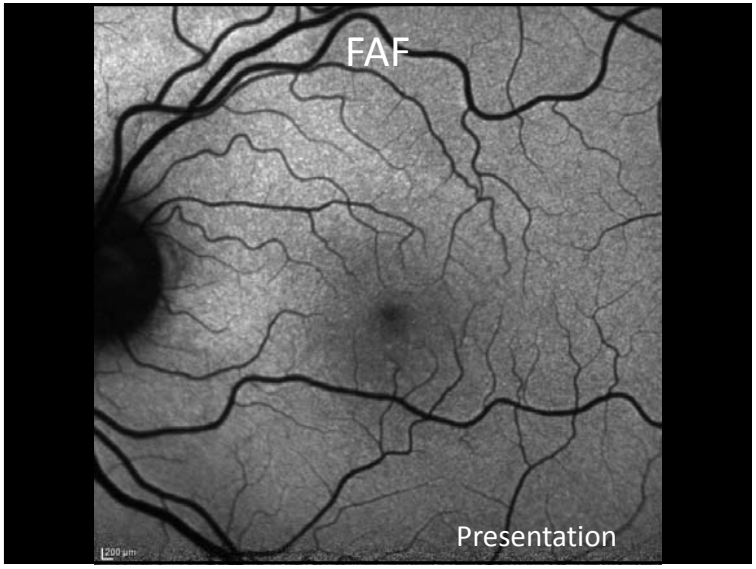
- 6 weeks after presentation
- CC: vision much better
  - VAsc 20/20 OD and OS
- Fundus
  - Resolved plaque, mild RPE mottling OS
- OD never became involved despite mild vitreous cell at presentation

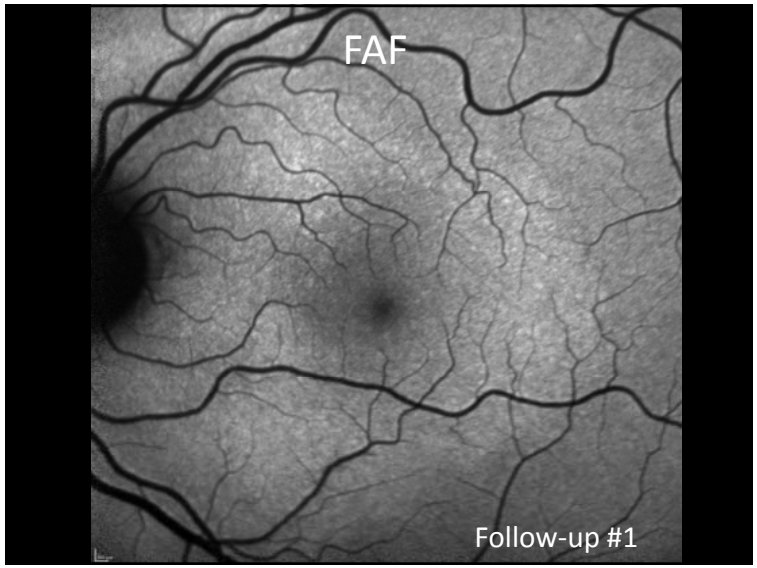


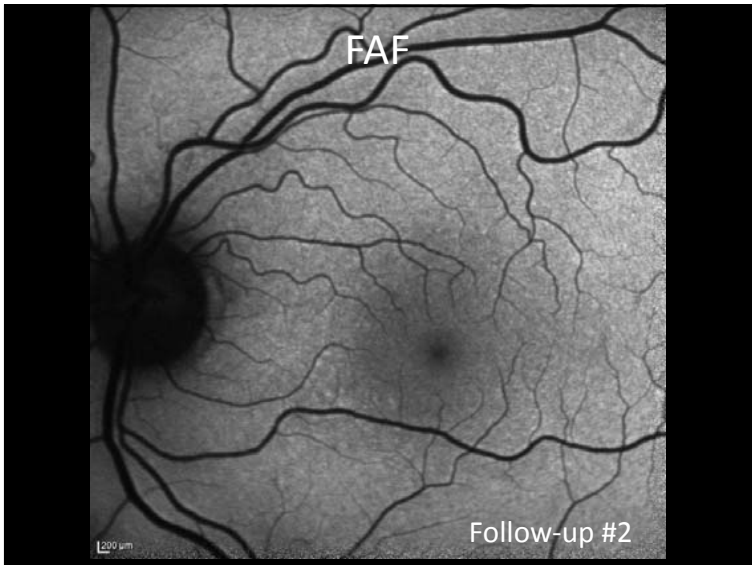

Macula OCT
Follow-up #1
VA: 20/30


Macula OCT
Follow-up #2
VA: 20/20


Macula OCT
Follow-up #2

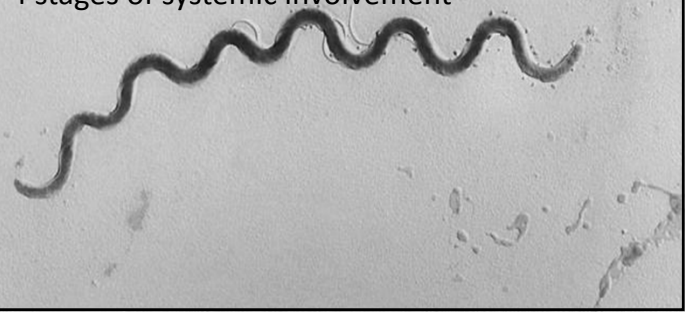

FAF
Presentation



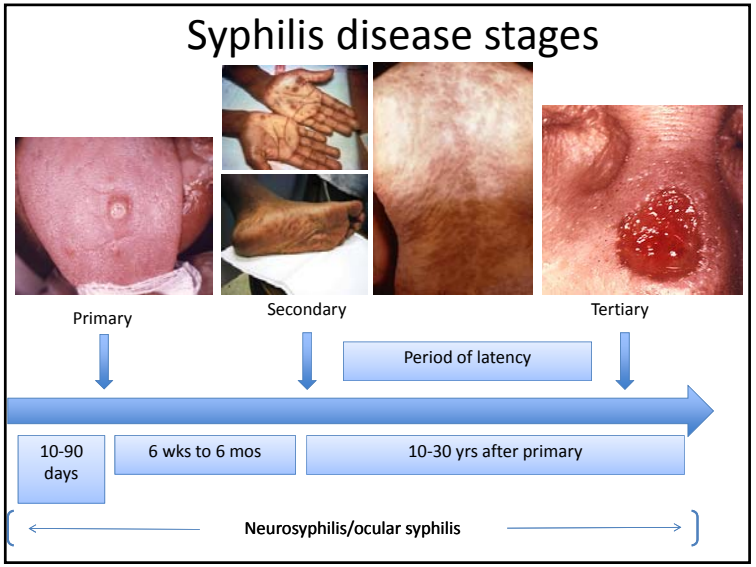


Acquired syphilis

- Transmission of *T. pallidum* occurs via direct contact with an infectious lesion
- 4 stages of systemic involvement

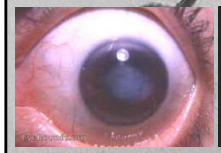


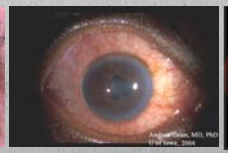
Syphilis disease stages

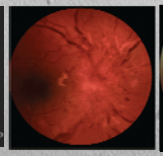


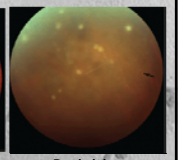
Ocular syphilis

- Notoriously difficult to diagnose due to ability to mimic other ocular inflammatory diseases and affect almost any part of the eye
- Cause of 1-2% of cases of ocular inflammation[2]
- ASPPC: 3% of cases of ocular syphilis[3]


Interstitial keratitis


Anterior uveitis


Papillitis


Retinitis

ASPPC

- Acute syphilitic posterior placoid chorioretinitis first coined by Gass et al in 1990[4]
- As of 2012, only 60 known cases[3]
  - 13% of those have occurred in females

ASPPC

Disease characteristics[3]

- 38% HIV co-infected
- Unilateral and bilateral
- Mean age of 40 at diagnosis
- Presenting VA: 20/20 to CF
- Most common associated systemic finding: mucocutaneous manifestations (~50%)
- Most common concurrent ocular finding: AC or vitreous inflammation

ASPPC

Pathophysiology[3-5]

1. Presence of spirochetes in the eye
2. Deposition of immune-complexes at the RPE-choriocapillaris junction

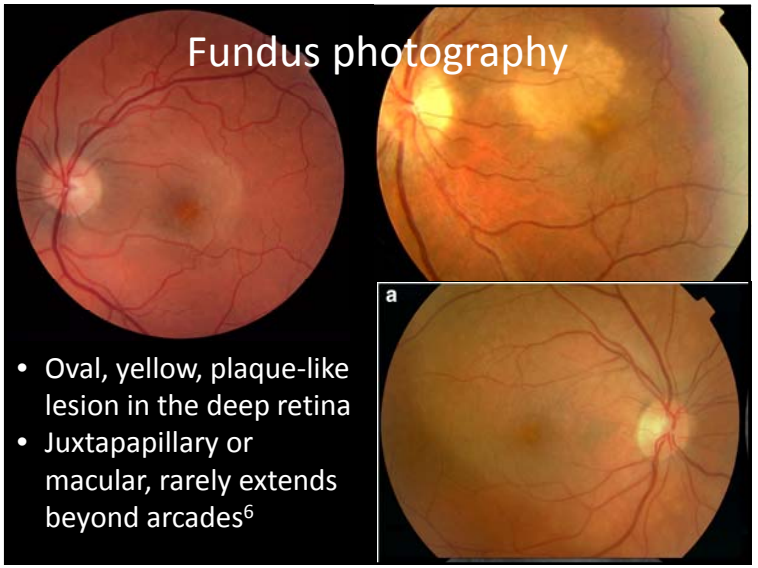
Importance of a modified immune response?

ASPPC
Imaging

Table 1: Imaging characteristics of ASPPC

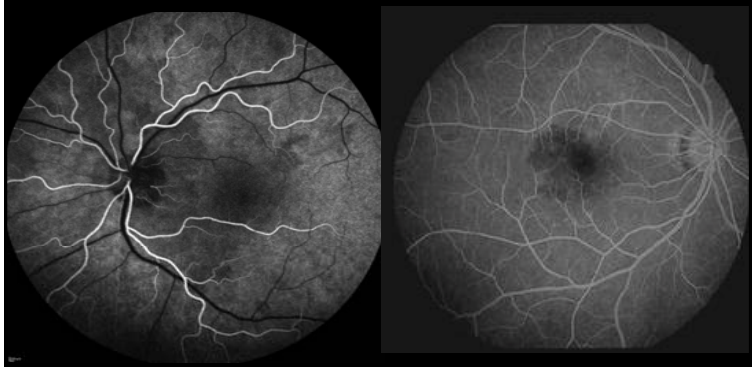
Imaging modality	Characteristic features
Fundus photography	Oval, yellow, plaque-like lesion in the deep retina Macular or juxtapapillary, rarely extending beyond arcades
Fluorescein angiography	**Early**: hypofluorescence in the area of the lesion **Late**: progressive hyperfluorescence with occasional "leopard spotting" areas of punctate hypofluorescence within the lesion
Fundus autofluorescence	Punctate hyperautofluorescence within the lesion during the active stage of the disease. Resolution of hyperautofluorescence occurs with elimination of inflammation.
OCT	**Acute phase**: thickening and hyper-reflective nodularity of the RPE with disruption of the overlying photoreceptors. Subretinal fluid is present in some cases. **Post-treatment**: resolution of RPE nodules. Photoreceptor disruption may resolve completely or remain to a mild degree indefinitely.
Multi-focal ERG	Reduced amplitude during the acute phase of the disease Improvement in mfERG amplitude is delayed as compared to acuity

Fundus photography



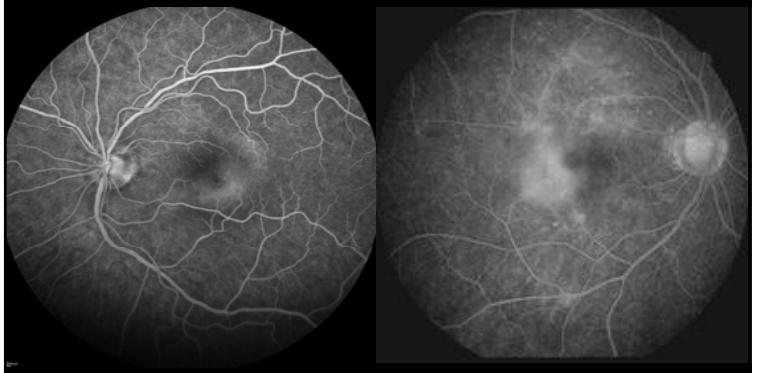
- Oval, yellow, plaque-like lesion in the deep retina
- Juxtapapillary or macular, rarely extends beyond arcades[6]

Fluorescein angiography



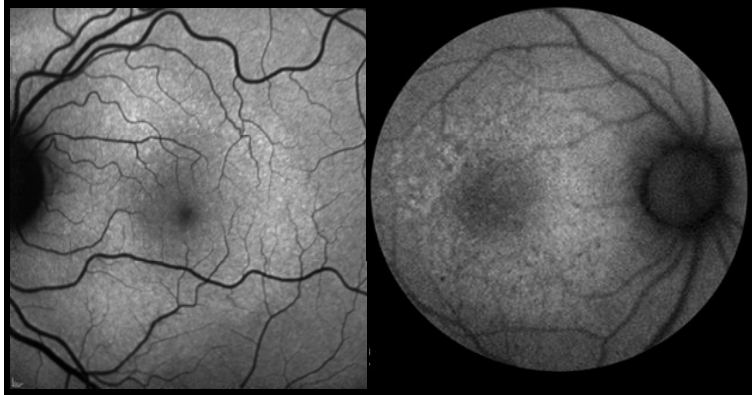
Early: Hypofluorescence in the area of the lesion[7]

Fluorescein angiography



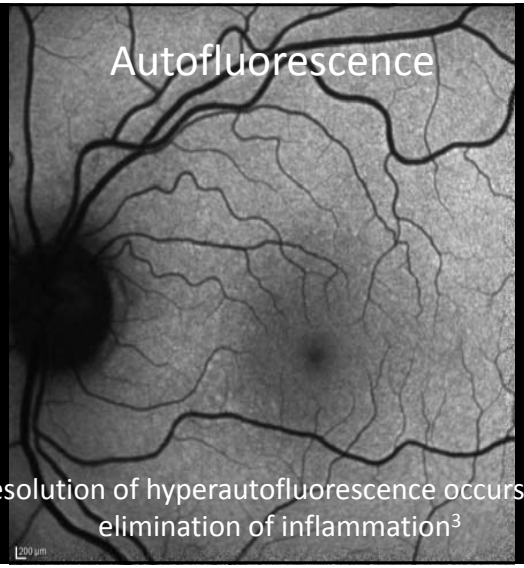
Late: Progressive hyperfluorescence with occasional "leopard spotting" within the lesion[7]

Autofluorescence



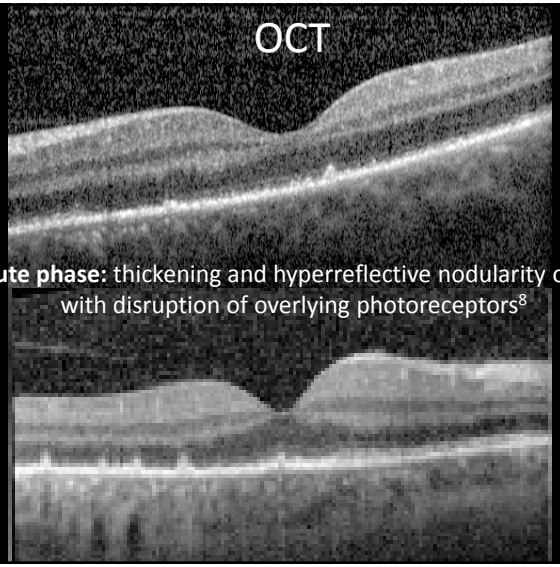
Punctate hyperautofluorescence within the lesion during active phase[3]

Autofluorescence



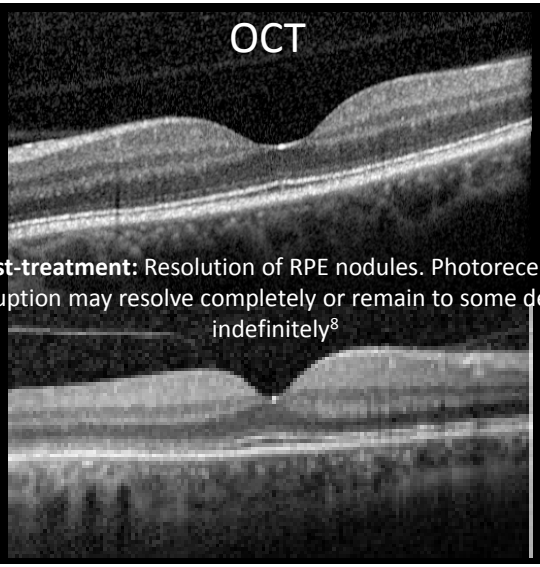
Resolution of hyperautofluorescence occurs with elimination of inflammation[3]

OCT



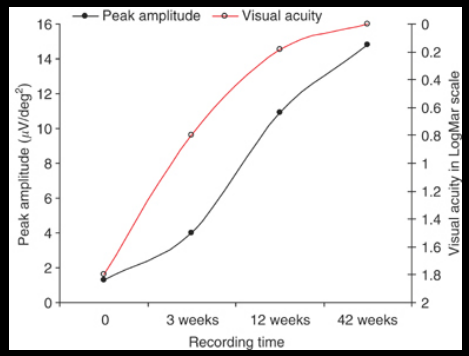
Acute phase: thickening and hyperreflective nodularity of RPE with disruption of overlying photoreceptors[8]

OCT



Post-treatment: Resolution of RPE nodules. Photoreceptor disruption may resolve completely or remain to some degree indefinitely[8]

Multi-focal ERG

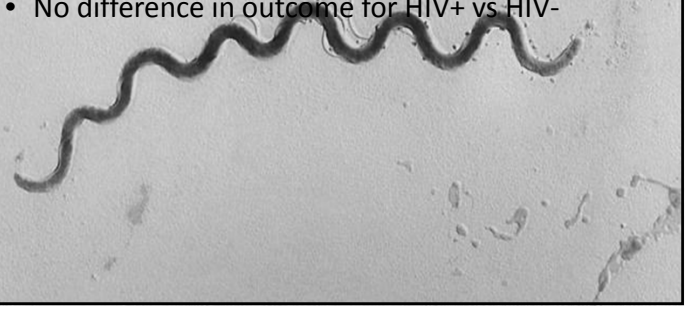


Joseph A, Rogers S, Browning A, et al. Syphilitic acute posterior placoid chorioretinitis in non-immunocompromised patients. Eye (Lond) 2007; 21:1114

- Reduced amplitude during acute phase
- Improvement in mfERG amplitude is delayed as compared to visual acuity[9]

Prognosis

- Good anatomic and visual recovery reported both with and without treatment
- VA 20/25 or better in ~90% of cases[3]
- No difference in outcome for HIV+ vs HIV-



Differential diagnoses

Table 2: Differential diagnosis of the posterior uveitides[10]

Cause	Clinical setting	Ancillary diagnostic testing	
		Ocular	Systemic
Idiopathic	Diverse	Diagnosis of exclusion	
Infectious			
Toxoplasmosis	Patch of white retinochoroiditis with speckled borders (next to a pigmented scar	**FA:** hypofluorescence in the area of an old chorioretinal scar, early hypofluorescence and late hyperfluorescence with persistent leakage during active infection	Serum antibodies. High frequency false positives.
CMV	Immunosuppressed patient with broad white patches of retinitis, often associated with hemorrhages	None	None required; HIV screening if no current diagnosis
Toxocariasis	Child with a white, round vitreous/inner retinal mass often extending from the optic nerve	None	Serum ELISA for anti-Toxocara antibodies
Tuberculosis	One or multiple slightly raised yellow, deep retinal patches with indistinct borders	None	PPD or Quantiferon gold
Syphilis	Any posterior uveitis	Varies depending upon presentation. See Table 1 for findings suggestive of ASPPC	VDRL, RPR, FTA-ABS
ARN	Patches of peripheral, white retinal necrosis	**FA:** peripheral areas of retinal arteriolar capillary nonperfusion	Aqueous PCR for VZV or HSV DNA
PORN	Immunosuppressed patient with deep, multifocal retinal lesions sparing retinal vasculature. Often bilateral.	**FA:** confirms lack of vasculitis	Aqueous PCR for VZV or HSV DNA
Inflammatory			
Sarcoidosis	Yellow-orange deep fundus lesions or larger peripheral full thickness white nodules. Sometimes accompanied by an exudative vasculitis	**FA:** hypofluorescence in area of ischemia, vascular leakage in area of inflamed vessels	ACE, chest x-ray
POHS	Classic triad of peripapillary atrophy, mid-peripheral histo spots, and macular lesions. Vitreous cell will be absent.	None	None
APMPPE	Creamy yellow deep retinal patches with indistinct borders	**FA:** early hypofluorescence with late hyperfluorescence in the area of active disease; **ICGA:** perfusion of the large choroidal vessels in the area of hypofluorescence in the early phase and marked choroidal hypofluorescence in the late phase	None
Serpiginous choroidopathy	Bilateral, well-circumscribed patches of yellow choroiditis that begin near the disc and extend in any direction.	**FA:** early hypofluorescence with later hyperfluorescence in the area of active disease. Hyperfluorescence begins at the margins of the lesion and spreads inward; **ICGA:** hypofluorescence through early and late phases. May be areas of hyperfluorescence outside the clinical lesion.	None
Birdshot retino-choroidopathy	Multiple white/yellow oval spots with indistinct borders; often mostly nasally	**FA:** lesions aren't visible until late in the disease process, when they stain; **ICGA:** late phase hypofluorescence. Detects a greater number of spots than seen clinically; **ERG:** delayed implicit time and b-wave amplitude	HLA-A29
PIC	Few clustered white spots with distinct borders, no vitritis, usually women	**FA:** punctate hyperfluorescent late staining	None
MEWDS	Multiple subtle, deep, gray spots	**FA:** early hyperfluorescence that persists into late phase; **ICGA:** hypofluorescence the area of the white dots, extensive area of hypofluorescence surrounding the optic disc; **VF:** enlarged blind spot; **ERG:** depressed A-wave, recovers during resolution of the disease	None
VKH Syndrome	Nonrhegmatogenous retinal detachments, often with deep, gray choroidal spots. Patients often note tinnitus or severe headaches.	**FA:** diffuse, pinpoint hyperfluorescent leaks at the level of the RPE, late pooling in the subretinal space; **ICGA:** multiple hypofluorescent spots; **Ultrasound:**	Often involves multi-system work-up including LP, audiogram, pathology, and genetic testing for HLA-DR4DS

Laboratory testing

- Serology=**presumptive** diagnosis

Table 3: Laboratory evaluation of suspected syphilis[11]

Category	Features	Specific tests
Non-treponemal	Non-specific Quantifiable as a titer Allows monitoring of treatment response	Venereal Disease Research Laboratory (VDRL) Rapid plasma reagin (RPR)
Treponemal	Treponemal specific Qualitative only	Fluorescent treponemal antibody absorption (FTA-ABS) *T. pallidum* particle agglutination assay (TPPA) *T. pallidum* enzyme immunoassay (TP-EIA)

Diagnosis requires a positive test from each category
Traditional testing algorithm=non-treponemal first, followed by confirmatory treponemal test
Consider additional screening tests for HIV and other sexually transmitted infections in all new cases of syphilis

Neurosyphilis

- Any CNS or ocular involvement → lumbar puncture
- CSF-VDRL
  - If positive, should be used to monitor treatment vs serology
  - Regardless of outcome, any ocular involvement w/ (+) syphilis diagnosis should be treated as neurosyphilis

Management

- Mandatory reporting
  - To state or local health department within 3 days of diagnosis
    - Syphilis diagnosis
    - Ocular disease
  - Often done by lab or medical facility
  - All partners for the past year require examination
- Referral to Infectious Disease for management

Treatment

Table 4: Treatment of confirmed, acquired syphilis in adults[11]

Disease stage	Drug	Route	Dose	Duration
Primary	Benzathine penicillin G	IM	2.4 million units	Single dose
Secondary	**Benzathine penicillin G**	**IM**	**2.4 million units**	**Single dose**
Latent				
Early	**Benzathine penicillin G**	**IM**	**2.4 million units**	**Single dose**
Late or unknown duration	Benzathine penicillin G	IM	2.4 million units	3 doses at 1 week intervals
Tertiary	**Benzathine penicillin G**	**IM**	**2.4 million units**	**3 doses at 1 week intervals**
Neurosyphilis	Aqueous crystalline penicillin G	IV	18-24 million units/day	10-14 days

- Treatment response: 4-fold decline in titer
- Alternative treatment options: tetracyclines or ceftriaxone
  - Limited data to support dosage, duration, and efficacy

Ocular syphilis conclusions

- ODs need to be aware:
  - Syphilis and ocular syphilis are on the rise
  - Syphilis can affect ANY part of the eye
  - Early detection and treatment
- Co-management partners:
  - Infectious disease/PCP
  - Retina
- Laboratory testing
  - Consider HIV/STD testing

Recommended reading

1. Moradi A, Salek S, Daniel E, et al. Clinical Features and Incidence Rates of Ocular Complications in Patients with Ocular Syphilis. Am J Ophthalmol 2015; 159:334.
2. Eandi CM, Neri P, Adelman RA, et al. Acute Syphilitic Posterior Placoid Chorioretinitis. Report of a Case Series and Comprehensive Review of the Literature. Retina 2012; 32(9):1915.
3. Jabs D and Busingye J. Approach to the diagnosis of the uveitides. Am J Opthalmol 2013; 156(2):228.
4. United States Centers for Disease Control and Prevention. 2015 Sexually Transmitted Disease Treatment Guidelines: Syphilis. Atlanta, GA: US Health and Human Services, CDC; 2016. https://www.cdc.gov/std/tg2015/syphilis.htm. (Accessed January 25, 2017).

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2. Moradi A, Salek S, Daniel E, et al. Clinical Features and Incidence Rates of Ocular Complications in Patients with Ocular Syphilis. Am J Ophthalmol 2015; 159:334.
3. Eandi CM, Neri P, Adelman RA, et al. Acute Syphilitic Posterior Placoid Chorioretinitis. Report of a Case Series and Comprehensive Review of the Literature. Retina 2012; 32(9):1915.
4.Gass JD, Braunstein RA, Chenoweth RG. Acute syphilitic posterior placoid chorioretinitis. Ophthalmology 1990; 97:1288.
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6. Joseph A, Rogers S, Browning A, et al. Syphilitic acute posterior placoid chorioretinitis in non-immunocompromised patients. Eye (Lond) 2007; 21:1114.
7. Ji YS, Yang JM, Park SW. Early resolved acute syphilitic posterior placoid chorioretinitis. Optom Vis Sci 2015; 92:S55.

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9. Joseph A, Rogers S, Browning A, et al. Syphilitic acute posterior placoid chorioretinitis in non-immunocompromised patients. Eye (Lond) 2007; 21:1114.
10. Nakamoto D and Gaudio P. Systemic diseases associated with ocular inflammation. In: Ocular Inflammatory Disease and Uveitis Manual: Diagnosis and Treatment, Huang JJ, Gaudio PA, Lippincott Williams & Wilkins, Philadelphia 2010. p.162.
11. United States Centers for Disease Control and Prevention. 2015 Sexually Transmitted Disease Treatment Guidelines: Syphilis. Atlanta, GA: US Health and Human Services, CDC; 2016. https://www.cdc.gov/std/tg2015/syphilis.htm.

Acknowledgments

- Judy Oh and Jeff Hiett

Questions

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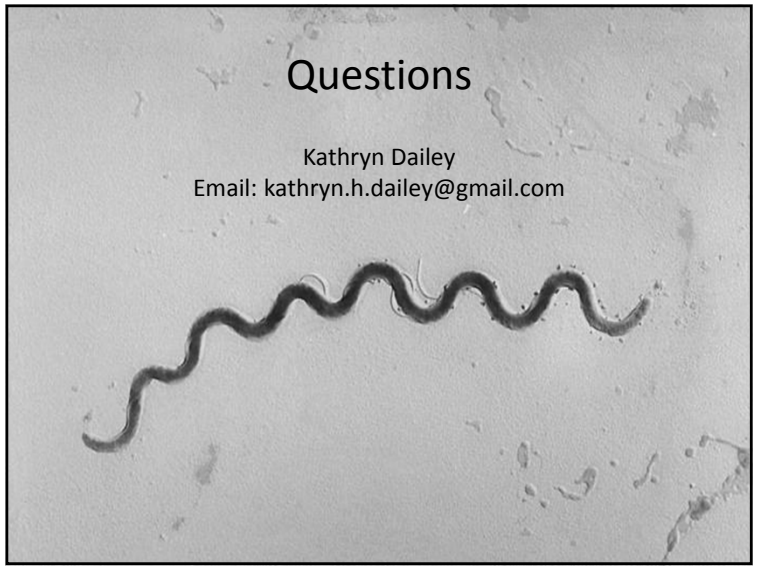
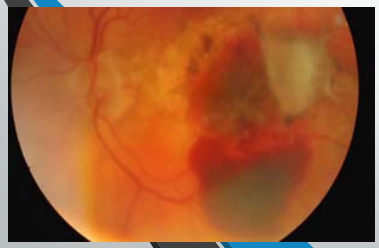


Spontaneous and Vision Threatening:

Choroidal Neovascular Nets

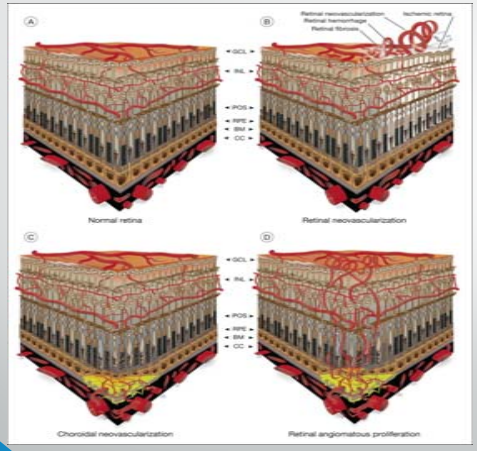
An In-depth Presentation



Timothy Mock, O.D.
06/09/2017
Spokane VAMC Resident

Choroidal Neovascularization

- Growth of new blood vessels from pre-existing choroidal vessels into the subretinal space
- Formation pattern:
  - Between choroid and retinal pigment epithelium (RPE)
  - Between RPE and neurosensory retina
- Retinal Angiomatous Proliferation
  - Initially neurosensory retina → later stage choroid space



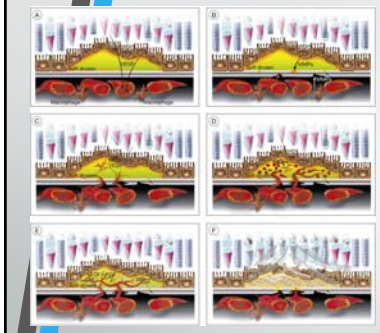
Retina (5th ed.), 2013

Common Conditions Associated with CNVM

- Age Related Macular Degeneration (AMD)
- Polypoidal Choroidal Vasculopathy (PCV)
- Ocular Histoplasmosis Syndrome (OHS)
- Angiod Streaks
- Pathologic Myopia
- Choroidal Rupture from trauma
- Idiopathic

Pathogenesis

- Hypoxia is primary stimulus for angiogenesis
- Response to tissue injury:
  - Damage to outer retina
  - Iatrogenic laser surgery
  - Traumatic choroidal ruptures
  - Outer retinal choroidopathies
- Free radicals



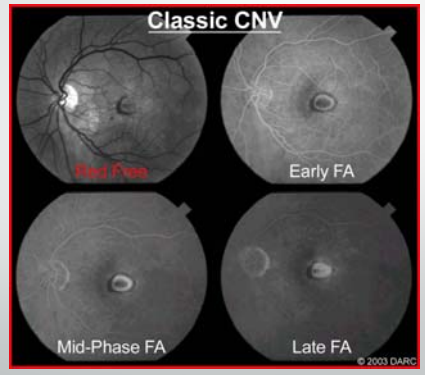
Retina (5th ed.), 2013

- Soft drusen, unlike hard is a common precursor
- Accumulation of deposits between RPE and Bruch's Membrane → obstruct diffusion of oxygen between choriocapillaris and RPE layers → VEGF
- Endothelial cells activated migrate through Bruch's

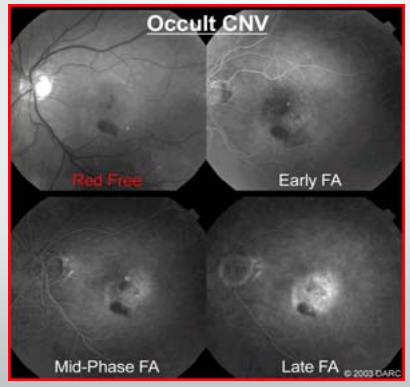
Types of Nets: Classic vs. Occult

- Understanding through:
  - Fluorescein Angiography
    - True extent of the net and fluid leakage
  - Optical Coherence Tomography

Classic CNVM



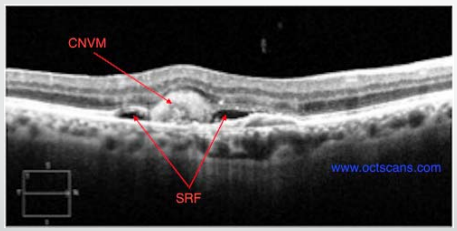
Occult CNVM



Features of a CNVM through OCT: Classic

- Classic CNVM:
  - thickening or disruption of the RPE-choriocapillaris complex
  - 20% incidence
  - Fibrovascular complex above the RPE layer w/adjacent SRF leakage

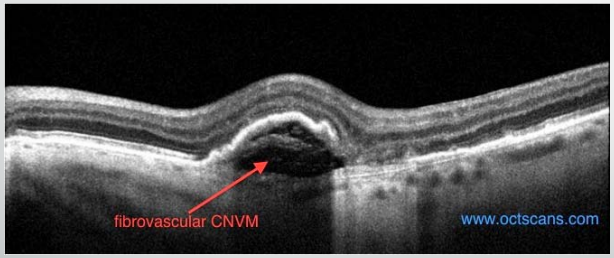
Classic CNVM



Features of a CNVM through OCT: Occult

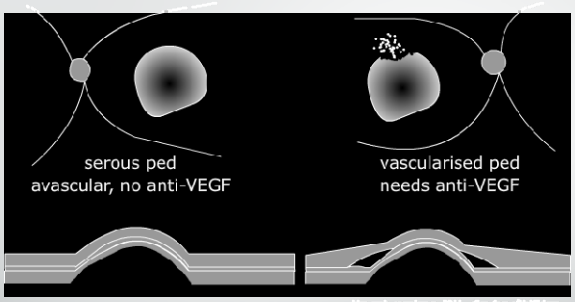
- Occult CNVM:
  - disruption of the RPE-choriocapillaris complex
  - most of the fibrovascular complex lies under the RPE; possible associated subretinal fluid and retinal edema
  - 60-80% incidence
  - Beneath the RPE layer and appear as a fibrovascular or hemorrhagic pigment epithelial detachment

Occult CNVM



PED Associated CNVM

- Occult CNVM:
  - A fibrovascular PED is seen as an elevation of RPE with some amount of reflectivity under it
    A haemorrhagic PED is seen as an elevation of RPE with shadowing of the choroidal layers
- Differs from serous PED

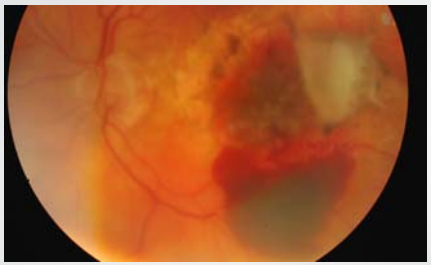


http://www.goodhopeeyeclinic.org.uk/oct.html

Types of CNVMs

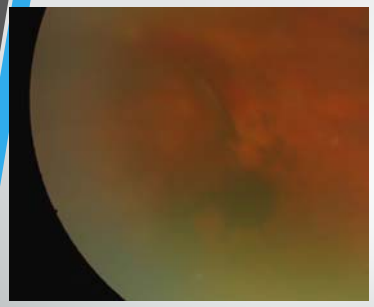
- Macular
- Peripheral
- Peripapillary

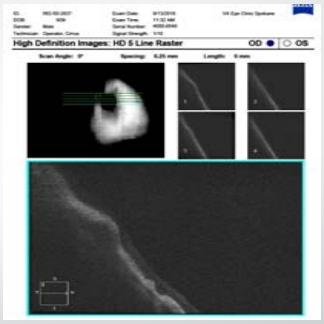
- CNVM is not restricted to any area in the retina
- Each should be considered it's own clinical entity



Macular CNVM

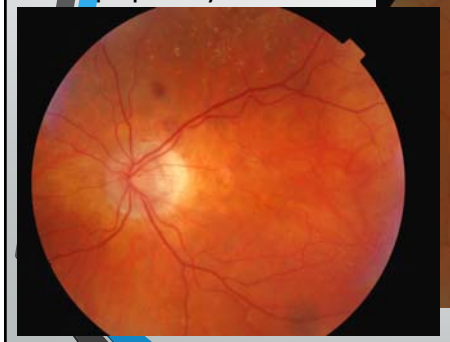
Disciform Scars

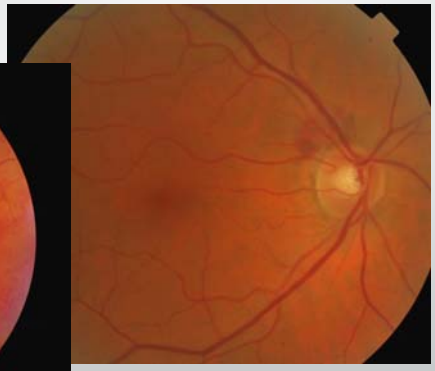




Peripheral CNVMs

Peripapillary CNVMs





In Yo Chair

- Determine the underlying cause
- Status of vision and symptoms
  - Reduced BCVA/Blur
  - Distortion
  - Central scotoma
- Potential threat to vision
  - Macular involvement: exudation and/or hemorrhage → increased CNVM/irreversible fibrosis
  - Vitreous hemorrhage

Continue care/Referral: Macular CNVM

- CNVM within the macular area warrants a referral to a retinal specialist:
  - Foveal
  - Perifoveal
  - Parafoveal
- Within 1 week for referral to retina

Treatment of Macular CNVM

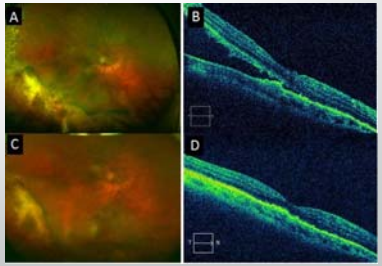
- Anti-VEGF therapy:
  - Lucentis (FDA Approved)
  - Avastin (non-FDA Approved)
  - Eylea (FDA Approved)
- Photodynamic therapy (PDT) – performed if other Tx options fail
- Thermal laser treatment – not performed

Treating Wet AMD: Anti-VEGF Therapy

- Monthly injections for first 3 months of outbreak
- Monthly F/U's with Macular OCT:
  - If macular is flat, extend time until next injection
  - If wet or no improvement, may perform same 3 month trial with Lucentis or Eylea

Continue care/Referral: Peripheral CNVM AKA Peripheral Exudative Hemorrhagic Chorioretinopathy (PEHCR)

- Bilateral 30%
- Associated with AMD and Polypoidal Choroidal Vasculopathy
- RPE detachment (serous or hemorrhagic), subretinal fluid or hemorrhage, subretinal exudation, and, rarely, vitreous hemorrhage
- Associated with extramacular involvement



Eyewiki.aao.org

Common Characteristics of PEHCR

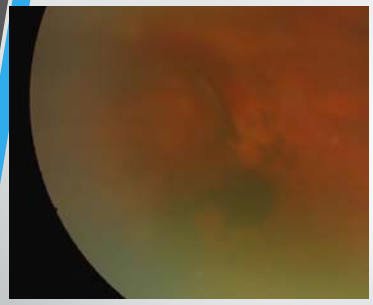
- Age (mean ~77)
- Women/Caucasians
- Associated with hypertension and anti-platelet/coagulation therapy
- Frequent pigment epithelial detachment
- Temporal retinal equatorial involvement
- Typically asymptomatic

Treatment of PEHCR

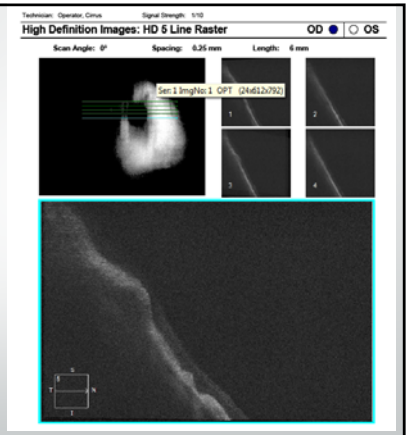
- Observation is standard of care for these lesions
  - 89% of reported cases regressed
- Vitrectomy if vitreous hemorrhage
- Consider Anti-VEGF Treatment and/or laser photocoagulation
- Macular involvement associated with PEHCR has been found to be successfully treated with Anti-VEGF therapy

Patient Case: #1

- 77 YOM
- ROS: HTN, RA, hyperlipidemia, anemia, carotid stenosis Class C R/L
- Ocular: Glx suspect, cataracts
- Asymptomatic



Inf temp lesion far periphery

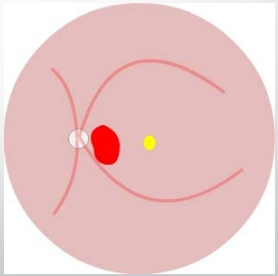


Continue care/Referral: Peripapillary CNVM

- Defined: CNVM within ~1 DD of the optic disc
- ~10% of total CNVMs
- Threat to vision – refer to retina
  - Macular involvement
  - Temporal in location to nerve
- Reduced threat to vision – monitor closely
  - Nasal, superior, or inferior in location

Treatment of Peripapillary CNVM

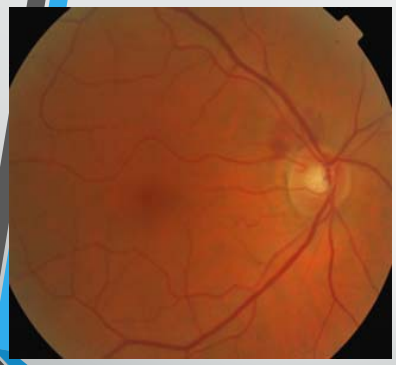
- Laser therapies not considered due to high risk of damage to papillomacular bundle → poor visual outcome
- Anti-VEGF treatment: not warranted until macular involvement (~750 microns)
  - No proven evidence tx works for non-macular nets
- Can be self-resolving

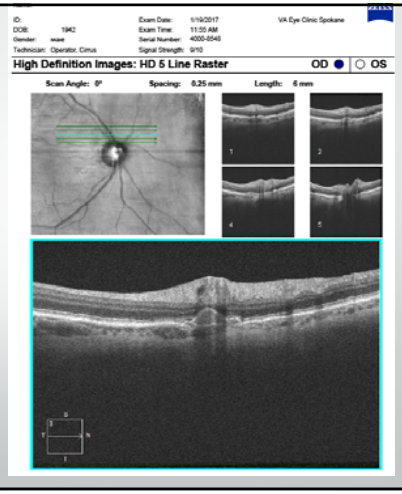


http://www.goodhopeeyeclinic.org.uk/armdperipapillary.html

Patient Case #2

- 74 YOM
- 6 week f/u for Symptomatic PVD
- ROS: Wolff-Parkinson-White Syndrome, tinnitus, osteoarthritis
- Symptoms: chronic floaters, no flashes





Low Vision Aids

- Referral to specialist
- Yellow tinted glasses
- CCTV
- Digital Magnifiers
- Improved home conditions

http://www.rnib.org.uk/

Take Home Messages

- When in doubt, refer out
- Location is everything
- Don't forget about low vision

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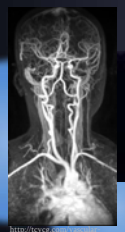
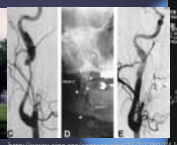
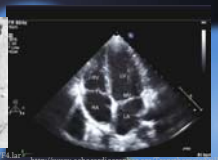
Who Turned Out the Lights:

Embolic Causes of Transient Monocular Visual Field Loss and a Review of Most Valuable Diagnostic Testing



Kerri Norris, O.D.
Jonathan M. Wainwright Memorial VAMC & PCLI Kennewick

Course Objectives

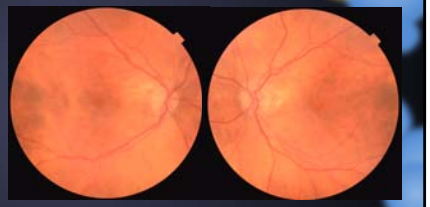
- Focusing on amaurosis fugax and identifying the most appropriate testing and imaging → the best possible patient care
  - What are we looking to rule out and what is the best test to find it?
    - Consider the yield, contraindications for, and availability of a test
  - What treatments are available depending on what we find?
    - Along the way we should always be asking if what we discover will change management or decrease morbidity
  - Don't forget about proper and prompt referral when called for

Case Presentation

- 89 yo W M with CC:
  - Painless graying/loss of vision described as a "shade" that falls down from top half to full vision OS
  - Happened 1-2 x month for the past 4 months, last event 3 weeks prior
  - Vision loss in seconds and lasted for about 5 minutes
  - Denied flashes, floaters, eye pain, jaw/temple pain
- Previous ocular history: unremarkable
- Medical history: atrial fibrillation
- Medications: had previously been on warfarin but self-d/c

Examination

- Fields full
- 20/20 OU
- SLE: unremarkable
- IOP 10 mmHg OU
- DFE: pre-surgical cataracts but otherwise normal



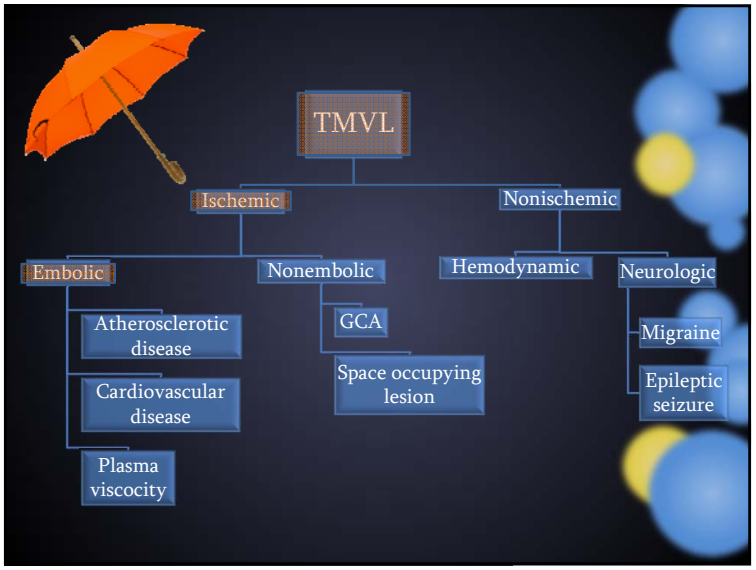
A & P

Asessment:

1. Transient monocular vision loss OS
   - Strong suspicion for amaurosis fugax
2. Exophoria
   - No tropia or vertical component noted during exam
   - Patient not experiencing diplopia during exam

Plan:

1. Patient educated on condition and importance of further testing and follow-up compliance. Ordered CBC c differential and PT/PTT bloodwork as well as carotid Doppler. RTC pending results.
2. No treatment currently indicated. Monitor PRN.



Differentials

TABLE 1

The Differential Diagnosis of Transient Monocular Blindness

Diagnosis	Age Group	Tests to Be Considered
Embolic		
Atherosclerosis	elderly	ultrasonography/angiography
Dissection	all ages	MRI
Cardiac thromboembolism	all ages	cardiac echography
Hypercoagulability	all ages	coagulation screen
Hemodynamic		
Primary postural hypotension	all ages	sitting/standing blood pressure, autonomic tests
Secondary postural hypotension	elderly	check blood pressure drugs
High blood viscosity	all ages	laboratory
Malignant hypertension	all ages	blood pressure
Reduced ocular perfusion	elderly	ultrasonography/angiography, central retinal artery perfusion pressure
Vascular		
Vasculitis (including giant cell arteritis as an important differential diagnosis in the elderly)	all ages	urgent ESR, laboratory tests, angiography, biopsy
Arteriovenous fistula	all ages	angiography
Vasospasm	all ages	funduscopy during attack
Optic disk, optic nerve, and brain		
Migraine	all ages	history & neurological examination
Epilepsy	all ages	neurological examination, EEG, brain imaging
Optic disk edema (causing visual obscuration)	all ages	ophthalmoscopy, VF, brain imaging, lumbar puncture + opening pressure
Optic disk anomalies (e.g., drusen)	all ages	ophthalmoscopy, retinal OCT
Gaze evoked	all ages	orbital imaging
Cortical ischemia	all ages	MRI brain
Miscellaneous		
Blowing the nose	all ages	—
Glaucoma	elderly	ophthalmoscopy, VF, intraocular pressure
Intraocular hemorrhages	all ages	ophthalmoscopy
Malaria	children, adults	blood smear
Pregnancy	fertile female	blood pressure, ophthalmoscopy, VF
Idiopathic	all ages	exclusion

EEG = electroencephalography; ESR = erythrocyte sedimentation rate; MRI = magnetic resonance imaging; OCT = optical coherence tomography; VF = visual fields.

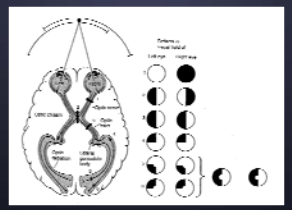
Pula J, Kwan K, Yuen C, et al. Update on the evaluation of transient vision loss. *Clin Ophthalmol.* 2016; 10:297-303. doi:10.2147/opth.s94971.

Pause for Vocab

- Amaurosis fugax: a sudden, transient loss of vision that can be either partial or total, lasting seconds to minutes before resolution, specifically due to an interruption of blood flow from retinal embolism
  - Vascular or cardiac thromboembolic cause
  - Monocular (although can switch eyes with repeat events)
  - A form of transient vision loss but terms not interchangeable!

TIA

- Transient ischemic attack or a "mini-stroke" is historically defined as interrupted blood supply which causes neurological dysfunction to the brain, spinal cord, or retina which recovers within 24 hours
  - Vertibrobasilar or hemispheric TIA would cause binocular hemifield loss
  - Could also be retinal (ex. affecting the ophthalmic artery) and therefore monocular vision loss
  - Cerebral transient ischemic attacks >> risk for future stroke than retinal TIA
    - 10-15% of these patients will have a stroke within 3 months
    - Greatest danger within 48 hours



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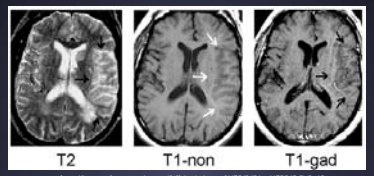
CVA



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- Stroke, or cerebrovascular accident, defined as brain injury caused by a sudden interruption of blood flow with symptoms that last for more than 24 hours

Importance of History

- Onset: sudden and painless monocular vision loss
- Frequency:
  - Wide range from single episode to many/day
  - Total number of attacks and how recently
- Systemic disease:
  - Thromboembolic conditions such as atrial fibrillation, carotid stenosis, cardiac valve issues

Duration of Vision Loss

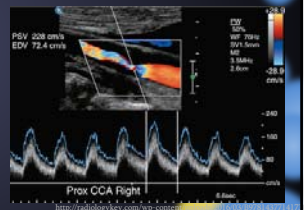
- Including speed of both onset and recovery
  - Onset: seconds
  - Recovery: complete and can clear in the opposite pattern of the vision loss
  - Length of vision loss:
    - <5 minutes associated with transient vision loss
    - 5-60+ minutes transient ischemic attack territory
- Patients who could not remember details about the mode of onset, disappearance, or duration of the attack were likely to have a normal internal carotid artery and nonembolic causes of vision loss

Associated Features/Provoking Factors

- Associated features: HA, temporal or jaw pain
  - Transient ischemic attack: patient likely to experience other neurological symptoms such as dysarthria or unilateral weakness
- Provoking/trigger factors:
  - Bright light
    - Retinal claudication a predictor for complete carotid occlusion
  - Exercise, postural change, cold, stress, gaze evoked amaurosis all less indicative of amaurosis

Embolic TMVL Trends

- Atherosclerotic lesions of the ipsilateral internal carotid artery or a cardiac source of emboli significantly associated with:
  - Attacks with an onset in seconds
  - Duration from 1-10 minutes
  - Provoked by bright light
  - An altitudinal onset or disappearance of field loss
  - The occurrence of 10+ attacks



http://radiologykey.com/wp-content/uploads/sites/3/2015/07/9781437714173.jpg

Pause

- Consider neurology referral for transient ischemic attack suspects:
  - Older patients especially 75+
  - Stoke risk factors:
    - H/o unilateral weakness/numbness, HA, dizziness, dysarthria (slurred or slowed speech), dysphagia (difficulty swallowing)
  - Duration of vision loss: >10 minutes
  - Diabetics, smokers, uncontrolled HTN
  - Patients with previously known thromboembolic conditions
- For those at high stroke risk may send directly to the ER:
  - Symptomatic within the last 72 hours
  - Duration of vision loss: >1 hour

Additional Risk Factors

TABLE 3

Risk Factors for Stroke in Patients with Atherosclerotic Carotid Stenosis

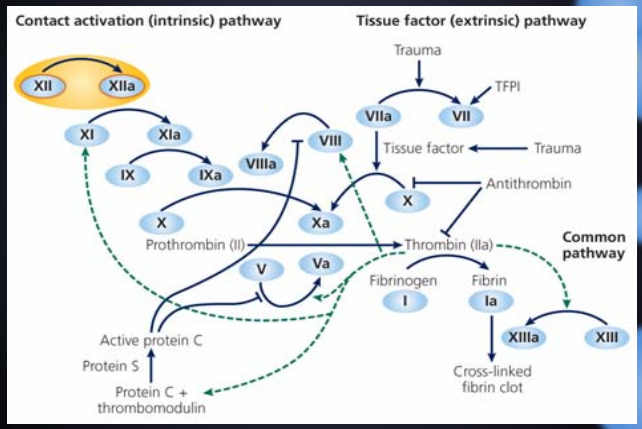
Risk Factor	Management Target
Absence of ECA-ICA collaterals	—
Adiposity	weight loss
Age	—
Atrial fibrillation	anticoagulation, cardioversion, anti-arrhythmic therapy
Cardiac valve problems	anticoagulation, surgery
Diabetes	fasting blood glucose levels < 126 mg/dl (7 mmoL/liter)
Elevated lipid levels	low-density lipoprotein < 100 mg/dl (2.6 μmoL/L)
Family history	find out about hereditary diseases which require targeted treatment
Hypertension	systolic blood pressure < 140 mm Hg and diastolic blood pressure < 90 mm Hg (for patients with diabetes, systolic blood pressure < 130 mm Hg and diastolic blood pressure < 85 mm Hg)
ICA stenosis	carotid endaterectomy and medical treatment
Intermittent claudication	exercise, flat shoes, aspirin, treatment of other risk factors
Male sex	—
Pharmacological contraception	consider alternative methods
Previous stroke	secondary prevention
Smoking	cessation

Pula J, Kwan K, Yuen C, et al. Update on the evaluation of transient vision loss. *Clin Ophthalmol.* 2016; 10:297-303. doi:10.2147/opth.s94971.

Bloodwork

- CBC with differential to rule out thrombus or severe anemia
- ESR/CRP to rule out giant cell/temporal arteritis
- Lipid panel to discern cholesterol levels
- If diabetic or unknown, blood sugar testing
- Prothrombin time/Partial Thromboplastin Time (PT/PTT)

Blood Clotting Pathway



http://www.rskmedical.com/z_medica_images/zmedica_pathways.jpg

Hypercoagulable States

- Clotting disorder, aka hypercoagulable state or thrombophilia
- Elevated odds ratio of transient vision loss, transient ischemic attack, or stroke:
  - Factor V Leiden
  - Hyperhomocysteinemia
  - Lupus anticoagulant
  - Antiphospholipid antibody syndrome

PT/PTT

- All fairly rare conditions and even then not overly increased risk of transient vision loss (more likely to manifest as central retinal vein occlusion)
  - Many easily treated with blood thinners like heparin/warfarin, but limited or even conflicting evidence of the *benefit* of initiating therapy
    - Either no established reduced risk of vascular events or at best contradicting studies
- Conclusion: testing for hypercoagulable states not recommended as part of routine work up in adults due to low yield
  - May consider for patients with personal or family history of thrombotic events, immobile patients, smokers, women on estrogen therapy, or those with other autoimmune disorders

Bloodwork Results

Collection Date/Time	Test	Result / Status	Flag	Units	Ref Range
Aug 22, 2016 14:34	NEUT #	68.7		%	44 - 74
	LYMP #	19.9		%	15 - 42
	MONO #	8.1		%	4 - 13
	EOS #	2.1		%	0 - 7
	BASO #	1.2		%	0 - 2
	WBC (TOTAL WBC COUNT)	4.7		K/cmm	3.3 - 10.6
	RBC	4.34		M/cmm	4.12 - 5.7
	HEMOGLOBIN	12.9		g/dL	12.9 - 17.1
	HCT	38.6	L	%	39 - 51
	MCV	88.9		cmu	81 - 102
	MCH	29.7		uug	27 - 31
	MCHC	33.5		uug	32 - 36
	RDW	13.3		%	11.2 - 16.2
	PLT	128	L	K/cmm	150 - 400
	PLT (ESTM)	ADEQ.			Ref: Adeq.
	NORMOCYTIC	YES			
	NORMOCHROMIC	YES			

Collection Date/Time	Test	Result / Status	Flag	Units	Ref Range
Sep 22, 2016 07:56	PROTIME	13.7		Sec.	11.4 - 14.9
	PARTIAL THROMBOPLASTIN TIME	39.1		Sec.	23.7 - 36.5
	INTERNATIONAL NORMALIZED RATIO	1.1	L		2.0 - 3.0

Carotid Doppler Imaging

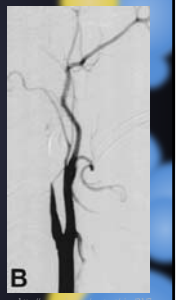
- One study found as high as 60% of transient monocular blindness cases are ultimately shown to be secondary to carotid artery stenosis
- The carotid can either be a source of emboli as a piece of the atherosclerotic plaque breaks off or the stenosis itself can lead to symptomatic retinal ischemia

Carotid Doppler Benefits

- Internal carotid artery the most common embolic source
  - Importance of bifucations as the most common site of atherosclerotic plaque buildup
    - This is due to increased turbulence and sheer stress at the forking of vessels
- Carotid ultrasound has 86% sensitivity and 87% specificity
  - Benefit: fairly cheap, noninvasive, widely available, safe!
  - No radiation, needles, nothing for the patient to have an allergic reaction to
- Results are straightforward:
  - Mild stenosis (16-49%), moderate (50-79%), severe (80-99%), or fully occluded

Internal Carotid Artery Dissection

- Another possible cause of carotid occlusion besides atheromatous plaquing
  - Vision loss in these patients often posturally or light-induced
  - Pain reported in up to 90% of cases
    - Carotid dissection rare in the first place and <4% have painless TMVL
- If your patient has painless TMVL and no neuro-ophthalmologic symptoms (Horner's, tinnitus, CN palsy, optic neuropathy), carotid artery dissection should be very low of your list of differentials *especially* if no h/o recent trauma



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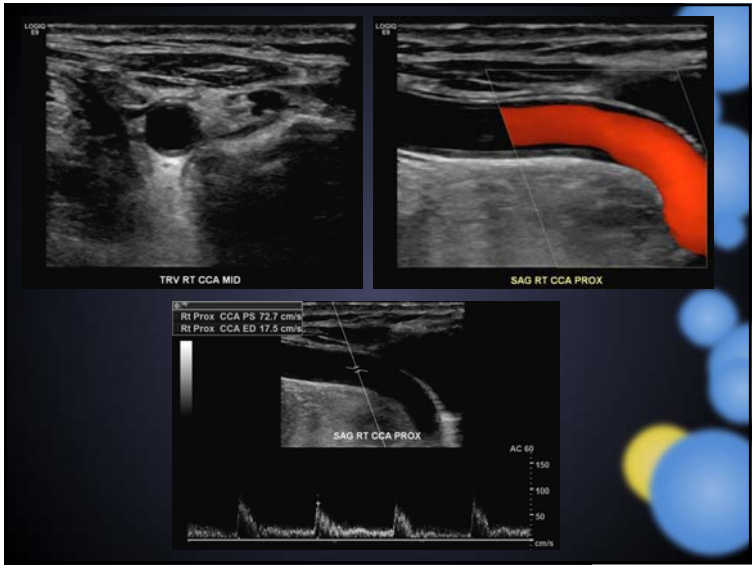
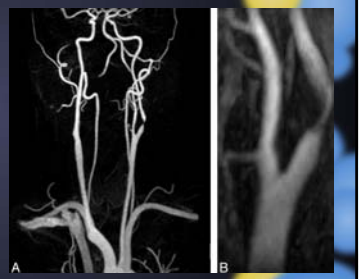


MRA + CTA

- Magnetic resonance angiography (MRA) or Computer tomography angiography (CTA)
  - Both have better discriminatory power than Doppler, detecting as high at 70-99% of carotid stenosis depending on the study
  - But maybe most importantly, they simply image MORE



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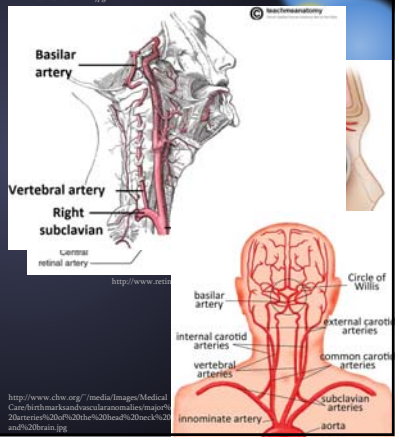
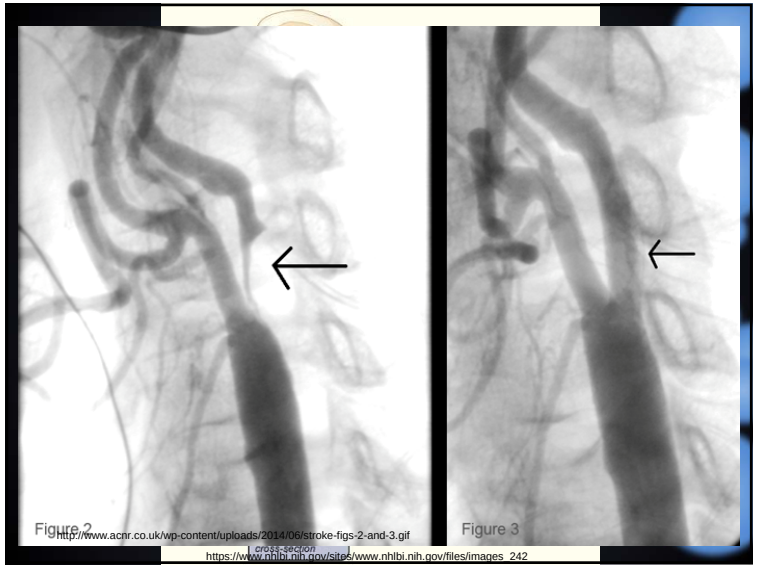
Ocular Blood Supply

- Just because the carotid doppler is clear doesn't mean there isn't an issue farther down the line of the vascular supply
  - For binocular vision loss skip Doppler and right to MRA/CTA imaging
- Always the possibility of artery stenosis distal to the extracranial carotid → responsible for 2% of transient monocular vision loss
  - Low prevalence of intracranial stenosis, however has been reported as high as 51-77% in Asian patients with h/o transient ischemic attack
    - Not to be ruled out



MRA	CTA
Pros:	**Cons:**
No ionizing radiation and can be performed without contrast media	Contrast agent contains iodine, radiation
	Pros:
Sensitivity of 95% and a specificity of 90% in detecting 70% to 99% carotid stenosis	Sensitivity and specificity for detecting >50% stenosis 97% and 99.5% respectively
Cons:	Better resolution than MRA
Patient must be able to sit in MRI machine (ex. no pacemaker, metal stents, obese, claustrophobic)	Good option for unstable patients ex. pacemakers or other MR contraindications
Must be able to tolerate contrast dye (good kidney function, no metformin for 48 hours)	Better availability
Testing can take 1-2 hours	Faster
Less sensitive to calcification/ossification	Better depiction of bones, plaques and landmarks (could be a negative if heavy calcification limits views)



http://www.acnr.co.uk/wp-content/uploads/2014/06/stroke-figs-2-and-3.gif

https://www.nhlbi.nih.gov/sites/www.nhlbi.nih.gov/files/images_242

Surgery Risk

- The North American Symptomatic Carotid Endarterectomy Trial (NASCET) assessed risk of perioperative complications, stroke, and death
  - Strokes were largely thromboembolic (released from the surgery itself) and most took place within 24 hours, especially 6 hours
  - Alternative to carotid endarterectomy is angioplasty/stenting → similar risks

http://radiologykey.com/wp-content/uploads/2016/03/B9781437714173000005X_f05-07-9781437714173.jpg

Risk vs. Benefit

- Certain cohort will not benefit from surgery because the risks of perioperative stroke may equal or exceed the risks of recurrent stroke on medical treatment
  - Strictly TMVL cases with stenosis tend to be "low risk" compared to cases of say TIA or stroke, and as such would be less likely to benefit from carotid endarterectomy
  - Lower stroke risk with medical treatment compared to Sx for many of these pts (ex. 4.1% 3 year risk of stroke vs. 9.4%)

Carotid Endarterectomy Indications

- Greater benefit in reducing risk of stroke:
  - Symptomatic (especially hemispheric transient ischemic attack)
  - If >70% carotid stenosis
    - Not indicated in cases of near or complete occlusion
  - Greater benefit if carried out within 1–2 weeks of the ischemic event
  - 75+ years
  - Male > female
  - No collaterals on angiogram
  - PVD

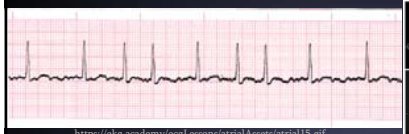
Treatment Options

- Medical:
  - Atherothrombotic TIA: long-term antiplatelet therapy
    - Ex. aspirin, clopidogrel, or aspirin with dipyridamole
- Controlling risk factors:
  - Hypertension:
    - Lower blood pressure to <140/90 mm Hg or <130/80 mm Hg for diabetics
  - Lipids:
    - LDL-cholesterol level <2.59 mmol/l (<100mg/dl)
  - Diabetes:
    - Fasting blood glucose goal <126mg/dl
  - Smoking cessation
  - Physical activity

Case Check-In

- CBC c diff
  - Not consistent with thrombus or severe anemia
- PT/PTT
  - Not consistent with clotting disorder/blood dyscrasia
- Carotid doppler
  - No evidence of flow-limiting stenosis of either ICA
- MRA of ophthalmic artery
  - Not performed

Cardiac Imaging



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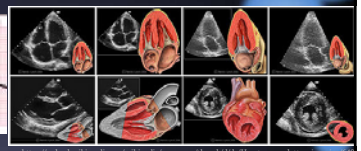

https://upload.wikimedia.org/wikipedia/commons/thumb/d/dc/Heart_normal_tte_views.jpg/640px-Heart_normal_tte_views.jpg

- Electrocardiogram (ECG)
  - Explore possible abnormal heart rhythms
  - Not performed, patient already known to have atrial fibrillation
- Echocardiogram (ECK)
  - Transthoracic (TTE) or transesophageal (TEE)
  - Cardiac sources of emboli and thrombi: ventricles, atria, septum, valves, even the aortic arch

Transthoracic ECK

- TTE: standard cardiac ultrasound
  - Imaging through the bony chest
  - Less invasive than transesophageal echocardiogram at the cost of decreased image quality
  - Suboptimal in patients with emphysema, chest deformities, even obesity

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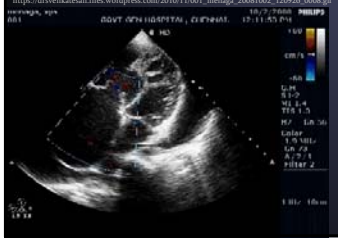

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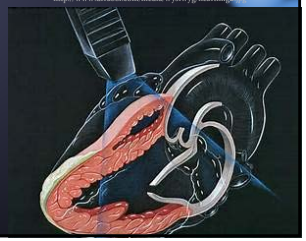
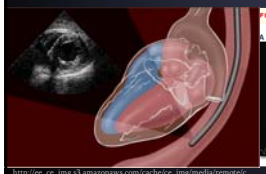


Transesophageal ECK

- TEE: higher spatial resolution = superior image quality than transthoracic echocardiogram
  - Higher resolution → more accurately measurement of lesion size which could change the treatment plan
    - Ex. determining if medical therapy vs. surgical intervention is indicated
  - Diagnostic accuracy 97% so why don't we always just order transesophageal ECK?



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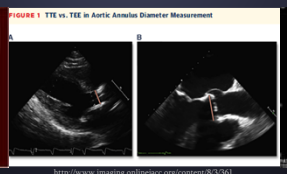

FIGURE 1 TTE vs. TEE in Aortic Annulus Diameter Measurement

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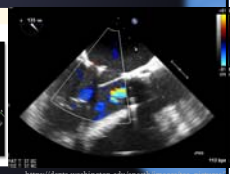

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ECK Comparison

TABLE 1 TTE Versus TEE in TAVR Guidance

	TTE	TEE
Procedure invasiveness	• Noninvasive	• Semi-invasive
Sedation requirement during TAVR	• Moderate sedation	• General anesthesia
Imaging advantages	• 2D & Doppler TTE is the primary means for quantitative and qualitative assessment of aortic stenosis and its impact on cardiac anatomy and function • Provides diagnostic, TAVR-relevant information with a potentially better safety profile compared with TEE	• Provides higher image resolution than TTE • 3D TTE has significant incremental value
Imaging disadvantages	• Quality of imaging determined by availability and location of imaging windows • Imaging may be limited by obesity, hyperinflation of lungs, chest deformity, and supine position • 3D TTE typically has limited incremental value • Shadowing of posterior PARs by TAVR prosthesis may occur	• TEE imaging may lead to injuries of oropharynx, esophagus, and the stomach
Potential for disruption of surgical field sterility	• Present but can be minimized with the use of sterile TTE probe covers	• Minimal
Impact of TAVR vascular access point to echocardiographic imaging	• Best suited for percutaneous transfemoral TAVR approach	• Can be provided with any TAVR access point

2D = 2-dimensional; 3D = 3-dimensional; PAR = perivalvular aortic regurgitation; TAVR = transcatheter aortic valve replacement; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography.

Kronzon I, Jelnin V, Ruiz C, et al. Optimal Imaging for Guiding TAVR: Transesophageal or Transthoracic Echocardiography, or Just Fluoroscopy? *JACC: Cardiovascular Imaging* 2015;8(3):361-370. doi: 10.1016/j.jcmg.2015.01.003.

Cardiac Embolic Sources

- Atrial fibrillation and left ventricular dysfunction the most common cardiac sources of emboli
  - Atrial fibrillation mechanism: pooling and stagnation of blood with subsequent thrombus formation
    - 5 fold increase risk of stroke
- Patent foramen ovale or atrial septal defect: holes between the chambers of the heart
  - Can lead to heart murmurs → increased turbulence → coagulation → thrombi
- Mass on the heart
  - Ex. myxoma: tumor of the wall between the two atria
  - Valvular abnormalities which disturb blood flow and increase risk of blood clot formation

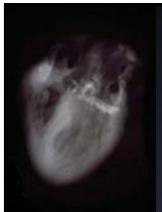
Cardiac Treatments

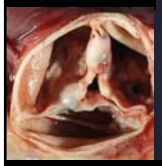
- Surgical intervention:
  - Tumor resection
  - Hole repair
  - Prosthetic heart valve
  - Valve leaflet debridement
- Medical therapy:
  - Anticoagulation therapy
    - Cardioembolic TIA: heparin or warfarin

Echo Results

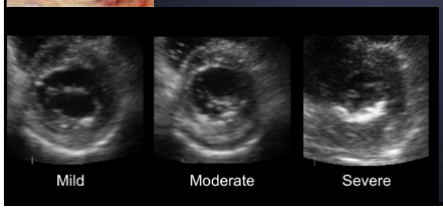
- Systemic history: atrial fibrillation, pt had self-d/c warfarin
- Newly diagnosed heart murmur secondary to leaky, stenosed, and calcified mitral valve
- Now learned that patient had h/o rheumatic fever as a child
  - Inflammatory disease caused by a group A strep throat or scarlet fever infection that is inadequately treated which goes on to affect connective tissues of the body
  - Known affinity of the auto-antibodies for the heart valves
    - Mitral valve is most often damaged with scar tissue → stenosis → calcification of valves later in life
    - More turbulent flow of blood across the valve → embolization of calcific plaque itself or thrombus formation → travels to ocular blood supply → amaurosis fugax
  - Accounts for <4% embolic TVL cases

Management





- Determined that the risk of heart valve replacement higher than medical therapy and close monitoring
  - Restarted on warfarin and followed closely by PCP



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https://academic.oup.com/eurheartj/article/29/12/1542/639769/Association-of-mitral-annular-calcification-and

Breakdown of TMVL Causes

- Prospective study of 2398 French patients with suspected TIA
  - "Major exam finding" such as embolus 28.1%
  - >50% to occlusion of extracranial stenosis 12.5%
    - Unexpectedly low compared to other studies which can show anywhere from 21-75%
  - Total cardiac source 8.6% → atrial fibrillation 5.7%, other cardiac sources of embolism 2.9%
    - As high as 21.9% in other studies
    - If internal carotid clear, cardiac source should be investigated
      - Higher statistical yield to move on to cardiac testing if carotid Doppler is clear than ordering MRA/CTA
  - Intracranial stenosis 2.1%
  - Extra- or intracranial dissection 0.4%
  - Blood dyscrasias not even considered in this study

Summary

- One retrospective study found that even after extensive testing, the source of embolism remains unclear in as high as 45% of patients
  - Another large study (n=1805), in about 27% the cause was not determined
- Even extensive testing can come back inconclusive
  - There's always the possibility of a plaque source without significant stenosis or in an area difficult to image

Wrap-Up

- Amaurosis fugax as a presenting symptom from valve stenosis is relatively uncommon, so an echo is not the first test on the list for a transient vision loss work-up
  - Proper clinically-based approach to testing, guided by a good patient history
  - Order your testing and imaging knowing what you're looking to rule out, not fishing for diagnoses
    - The most important tests are those which could rule out a vision- or even life-threatening condition
    - Will a result change management?
  - What is the screening power of a tests? What's the availability?
  - Consider your patient and any potential contraindications for a certain test
- Importance of patient education

Questions?

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A CASE SERIES ON NEOVASCULAR GLAUCOMA

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June 9, 2017

LEARNING OBJECTIVES

- Recognize the clinical symptoms and signs of neovascular glaucoma
- Understand the pathophysiology of ocular diseases that may result in neovascular glaucoma

CASE REPORTS

CASE 1 CB: 55 Y/O CAUCASIAN MALE

- 03/2016: NEW patient presents to clinic with mild periorbital/retrobulbar pain and fixed/dilated pupil with sudden blur x 5 days per PCP
- (-) Past Ocular Hx
- Medical History: Headache, HTN, carotid artery stenosis, hyperlipidemia, h/o amphetamine dependence and alcohol abuse

Last Carotid Ultrasound (08/2014):

- 1. Complete right internal occlusion (new since 2006)
- 2. Mild left internal carotid stenosis
- Plan: monitor w/o treatment due to complete occlusion

CASE 1 CB: CLINICAL EXAM

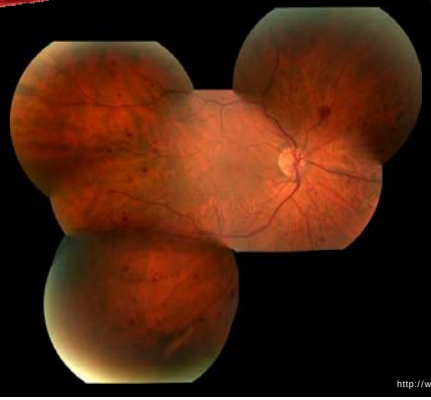
03/07/2016	OD	OS
Visual acuity	20/350 PH: 20/100-2	20/50+1 PH:20/20
Pupils	8.2 -8.0mm, sluggish (+) RIGHT APD	4.5-4.0mm, 3+ brisk
EOMs	Full	Full
Visual fields:	FTFC	FTFC
Lids/Lashes/Conjunctiva/ Cornea/Anterior Chamber	Normal	Normal
Iris	Diffuse sphincter atrophy (+) Perilimbal NVI	Normal
Intraocular pressure	26 mmHg	24 mmHg

CASE 1 CB: CLINICAL EXAM

Gonioscopy:

- OD: Open to SS in limited views, broad PAS ~270 degrees, extensive NVA in all 4 quadrants
- OS: Normal, no AR/PAS/NVA

CASE 1 CB: PHOTOS
(NOT ACTUAL PATIENT PHOTOS)



http://www.retinareference.com/diseases/c37843f6aa959e6f/images/c37843f6aa/

CASE 1 CB: SUMMARY OF SUBSEQUENT VISITS

- 03/2016: PRP was preformed 2 days after initial visit.
- 04/2016: Second session of PRP performed
- 05/2016: Vision and exam findings stable except for appearance of regression of NVA secondary to 2 sessions of PRP

Tonometry: 25/25

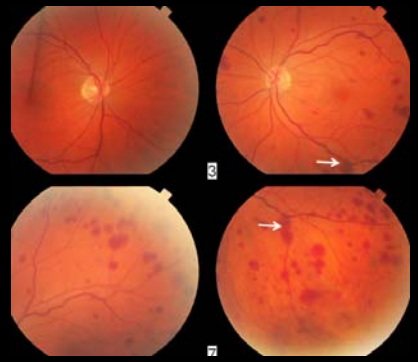
Gonioscopy

- OD: PAS in all quadrants with regressed NVA
- OS: CB inf, SS sup/temp/nas, with minimal pigment in all quadrants

CASE 1 CB: SUMMARY OF SUBSEQUENT VISITS

- Patient lost to follow up x 5 months w/ cc: fixed/milky right eye pupil and h/o severe perioribital pain 2 months ago, residual dull pain today
- Neovascularization of the iris and no views of the right posterior segment due to a mature cataract in the right eye
- IOP: 28/24
- Additional treatment since:
- Lowered IOP with brimonidine BID and dorzolamide BID OD
- Cataract surgery
- Diamox 250 mg BID s/p CE OD due to elevated OD IOP of 26

OCULAR ISCHEMIC SYNDROME



Variable vision (20/20 to NLP)

IOP may be elevated secondary to neovascular glaucoma

http://www.retinareference.com/diseases/c37843f6aa959e6f/images/b7265e4c8e/

CASE 2 ER: 79 Y/O CAUCASIAN MALE

- 09/19/2016: Pre-existing patient referred to clinic because a private doctor 3 weeks ago said a "blood vessel" burst in left eye due to uncontrolled hypertension
- (-) Ocular trauma or surgeries
- H/o POAG OU on Latanoprost QHS OU (poor compliance)
- Medical history:
- Uncontrolled hypertension, renal disease, colonic polyps
  - H/o poor compliance with medications prescribed by PCP

CASE 2 ER: CLINICAL EXAM

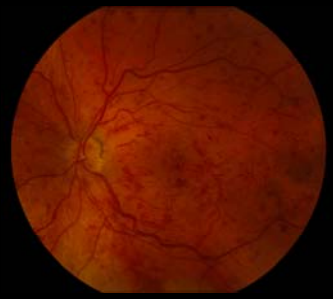
09/19/2016	OD	OS
Visual acuity	20/30	CF at 3 ft PHNI
Pupils	5/3 round, 2+ reactive	6/4 round, 1+ reactive 2+ Left APD
EOMs	Full and comitant	Full and comitant
Visual fields	FTFC	FTFC
Lids/Lashes/Conjunctiva /Anterior Chamber	Normal	Normal
Cornea	Clear	Mild edema, diffuse PEE
Iris	Normal (-) NVI	Normal, visible matrix vessels (-) NVI

CASE 2 ER: CLINICAL EXAM

Gonioscopy:
OD: Mildly narrow but otherwise normal, no AR/PAS/NVA
OS: Mildly narrow OS>OD but otherwise normal, no AR/PAS/NVA

- Tonometry (Goldmann):
- OD/OS: 28/42 @ 10:07
- OD/OS: 22/36 @ 10:35 (post gonioscopy)

CASE 2 ER: PHOTOS
(NOT ACTUAL PATIENT PHOTOS)



10/19/2016: Pt was seen in retina clinic but declined treatment

CASE 2 ER: CLINICAL EXAM 10/25/16

10/25/2016	OD	OS
Visual acuity:	20/30-2 PH:20/25-2	CF at 3 ft (stable to previous)
Pupils:	5/3 round, 2+ reactive	6/4 round, 1+ reactive 2+ Left APD
EOMS:	Full/comitant	Full/comitant
Visual fields:	FTFC OD	Temporal constriction OS (centrally blurry/distorted)
Lids/Lashes/Conjunctiva	Normal	Normal
Cornea	Normal	Central edema
Anterior Chamber	Moderate and Quiet	TR Cells GR 1 flare
Iris:	Normal	(+) NVI pupillary margin, dense at 3-6 oclock and 10-11 oclock

CASE 2 ER: CLINICAL EXAM 10/25/16

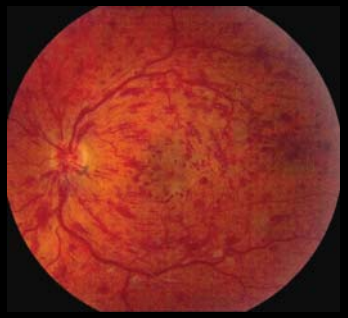
- OD/OS: 18/63 @ 9:46
- **Started pressure lowering therapy, 0.5% Timolol and 1drop brimonidine, 1 drop Cosopt, then1 drop of brimonidine w/ punctal occlusion, per discussion with Glaucoma specialist**
- OD/OS 16/38 @10:34 am (after drops instilled)
- Gonioscopy:
- OD: Convex approach but otherwise normal, no AR/PAS/NVA
- OS: Ant TM 360, trace pigment 360, very convex approach, NVA at 7:00, no AR/PAS (but hazy views)
- OU: Narrow approach 360

CASE 2 ER: SUMMARY OF SUBSEQUENT VISITS

- Patient was sent back to retina who had PRP but declined Avastin, pt then missed multiple scheduled follow ups and had difficulty instilling/remembering to put drops in eye at his retina and glaucoma follow up appointments

- Additional treatment:
- Anterior chamber paracentesis due to high eye pressure OS
- Cyclophotocoagulation
- Cataract surgery w/ Ahmed tube

CENTRAL RETINAL VEIN OCCLUSION



Variable vision loss
-worse vision suggests ischemic CRVO

Neovascularization occurs usually 1.5-6 months following ischemic event

Ocular Hypertension is a risk factor for CRVO

http://www.reviewofophthalmology.com/article/evaluation-and-management-of-retinal-vein-occlusion

CASE 3 DA: 64 Y/O CAUCASIAN MALE

- 04/08/2016: NEW patient referred from private doctor for sudden painless vision loss in the right eye

- (-) ocular trauma or surgeries (-) ocular medications
- H/o Mild NPDR OU

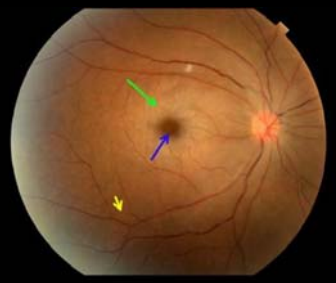
Medical History: DM 2 with renal manifestations, carotid artery disease, HTN, hyperlipidemia

Poor control of systemic conditions

CASE 3 DA: CLINICAL EXAMINATION

04/08/2016	OD	OS
Visual Acuity:	HM at 3 ft	20/30
Pupils:	OD Dilated 4+ APD	OS round, reactive
EOMs:	Full/comitant	Full/comitant
Visual fields:	HM in all 4 quadrants	FTFC
Lids/Lashes/Conjunctiva/Cornea/ Anterior chamber:	Normal	Normal
Iris:	Normal (-) NVI	Normal (-) NVI

CASE 3 DA: PHOTOS
(NOT ACTUAL PATIENT PHOTOS)



http://www.retinareference.com/diseases/f82527aa5cfac9f2/images/4cd4e945b1/

CASE 3 DA: CLINICAL EXAMINATION

- Tonometry (Goldmann):
- OD/OS: 22/25 @400p

05/05/2016 (1 month later): No changes to VA, health or slit lamp exam findings OU, IOP 17/20

- Gonioscopy OD: normal, no AR/PAS/NVA
  OS: normal, no AR/PAS/NVA
- Early NVD OD noted on dilated fundus examination

CASE 3 DA: CLINICAL EXAMINATION

06/09/16 (2 months since initial): No changes to visual acuity or health OU

- OD: conjunctival injection with 1+ cells/flare in AC (+) NVI
- Tonometry (Goldmann): OD/OS: 55/21 @ 2:48pm

- Gonioscopy:
- OD: +NVA 360 with PAS and angle closure in all 4 quadrants
- OS: Normal, no AR/PAS/NVA

CASE 3 DA: SUMMARY OF SUBSEQUENT VISITS

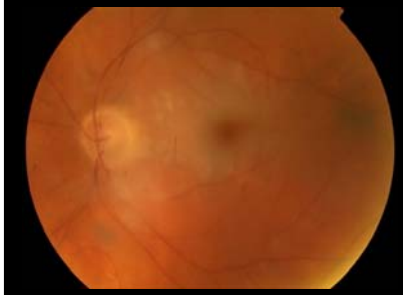
- 06/15/16: Patient sent to retina, PRP was withheld due to high IOP of 52/25, placed on Diamox 250 mg 35/25
- 06/23/16: PRP Performed OD

- Poor control of IOP and compliance with medication as directed

Additional treatment:

- Anterior chamber paracentesis
- Cyclophotocoagulation OD
- Currently on maximum medical therapy with better controlled pressures and compliance

CENTRAL RETINAL ARTERY OCCLUSION



Presents with poor vision, and APD

Loss of blood supply to inner retina causes chronic retinal ischemia from loss of perfusion.

Neovascular Glaucoma develops on average 3 months after event

http://www.retinareference.com/diseases/f82527aa5cfac9f2/images/713d294589/

OVERVIEW OF 3 CASES

- Ischemic conditions of the retina
- Poor visual outcome in affected eye due to pre-existing retinal conditions

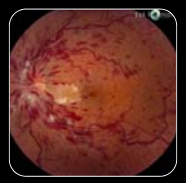
http://www.firstoasis.org/up-and-down/

- Poor compliance with medications (confusion/difficulty with instillation) and follow ups as directed by providers

NEOVASCULAR GLAUCOMA HISTORY AND DEMOGRAPHICS

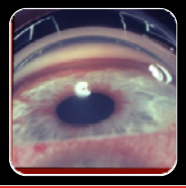
- Men > Women
- Mostly affects the elderly
  - 46.16% of patients between 60-79 years of age at onset
- Often requires medical/surgical intervention to help control IOP
- Associated with high rate of severe vision loss (HM or LP is not uncommon)

PATHOPHYSIOLOGY



Posterior Segment Ischemia

VEGF to anterior chamber



Neovascularization closes angle

IOP rises

Pain/corneal edema

- Blind/painful eye

http://shop.onjoph.com/catalog/images/atlas/RM702_1.jpg
http://webeye.ophth.uiowa.edu/eyeforum/atlas/pages/CRVO-w-CLRAO/index.htm
http://www.reviewofophthalmology.com/article/what-to-do-when-glaucoma-gangs-up-on-you

ETIOLOGY

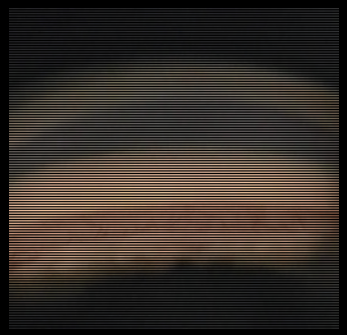
Ischemic	Retinal Detachment
Proliferative Diabetic retinopathy	Chronic Tractional RD
Central Retinal Vein Occlusion	Proliferative vitreoretinopathy
Ocular Ischemic Syndrome	Coat's Disease
Central Retinal Artery Occlusion	Retinoschisis
Sickle Cell Retinopathy	
Ocular Inflammation	**Intraocular Tumors**
Chronic Uveitis	Choroidal melanoma
Retinal Vasculitis	Iris Melanoma
Trauma	Retinoblastoma
Anterior Segment Ischemia	Metastatic Disease
Endophthalmitis	

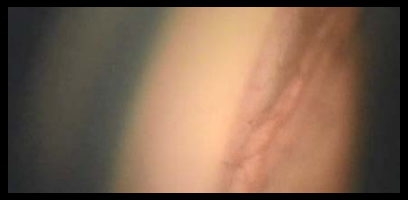
IMPORTANT CLINICAL EXAM FINDINGS

- Case History
- Review of Medical History
- Visual Acuity
- Pupils: APD?
- Slit Lamp Examination:
  Careful view of the cornea and iris
- Tonometry: Asymmetric or elevated IOP
- Dilated Fundus Exam: Neovascularization of the posterior segment

http://www.ttuhsc.edu/som/ophthalmology/eyeatlas/iris/extensive_iris_neovascularization_.aspx

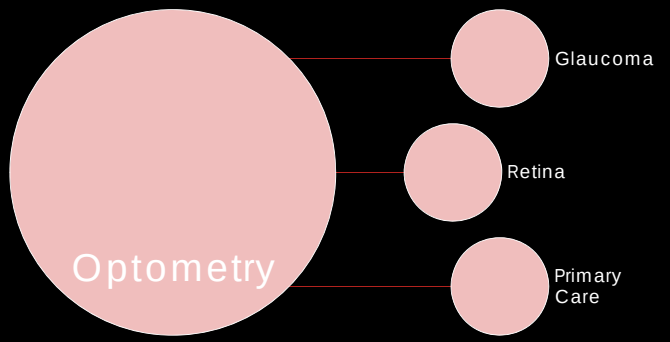
GONIOSCOPY





Gonioscopy.org

REFERRALS



MANAGEMENT/TREATMENT

Retina Specialist	Glaucoma Specialist
PRP and anti-VEGF to control posterior segment ischemia	Pressure lowering drops initially If IOP is very high: AC paracentesis (short term fix) Cyclophotocoagulation(CPC) AND or glaucoma shunt may be necessary

OUTCOME

- Poor prognosis for vision
- Keep patient comfortable (if possible) while preserving remaining vision, avoid "blind painful eye"
- Further treatment with enucleation may be necessary if eye becomes painful

CLINICAL PEARLS

- Serial gonioscopy and close follow up with dilated fundus examination on patients with new diagnosis of CRVO, CRAO and OIS
- REFER to the appropriate specialists when necessary
- Manage and communicate with PCP regarding systemic conditions

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QUESTIONS?

http://gentlemint.com/tack/4616/

Technology can be great, but…

A Review of Optical Coherence Tomography (OCT) of the Optic Nerve

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VA HEALTH CARE Defining EXCELLENCE in the 21st Century

Case 1: BW

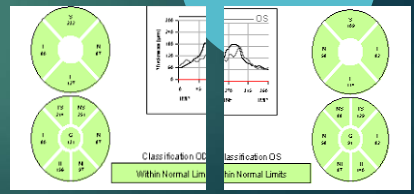
- 85 year old Caucasian male ---- 6 month follow-up
  - CC: no vision or ocular complaints
  - glaucoma-suspect OU

Case 1: BW

- Ocular medications: none
- Ocular history:
  - Mild non-exudative age-related macular degeneration OU
    - RPE mottling w/ scattered fine drusen OU
    - BCVA 20/25 OD and OS
  - Low myopia, astigmatism, presbyopia OU
  - Glaucoma-suspect secondary to physiological cupping OU
    - OD: 0.75 round – stable
    - OS: 0.70 round – stable
    - IOPs low/mid-teens OU and stable w/o treatment
    - Previous HVF shows no pattern defect OU

Case 1: BW

- OCT Optic Nerve rNFL analysis
  - "Within Normal Limits" OU
  - Impression:
    - OU: no rNFL thinning suggestive of glaucoma
- And "All Green is Good"… right?



Case 2: BP

- 62 year old Native-American male ---- annual eye exam
  - CC: Pt would like new bifocals

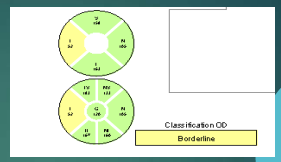
Case 2: BP

- Ocular medications: none
- Ocular history:
  - C/Ds 0.4 OU and stable
  - IOPs mid-teens OU and stable w/o treatment
  - Low hyperopia, astigmatism, presbyopia OU

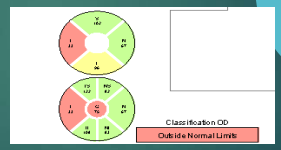
Case 2: BP

Right eye:

2014
"Borderline" Temporal



2017
"Outside Normal Limits" Temporal
"Borderline" Inferior



- Is this finding new glaucomatous thinning?
- Should treatment be initiated?

Are Case 1 and Case 2 straightforward… or is there more to the story?

CONSIDER:

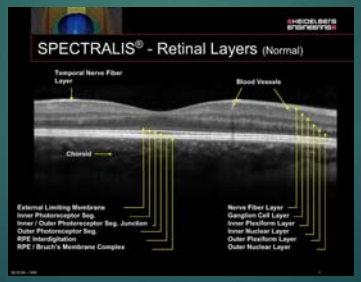
CASE 1 HAS LARGE CUP-TO-DISC RATIOS (GLAUCOMA-SUSPECT)

AND

CASE 2 SHOWS NEW & PROGRESSIVE THINNING OF THE RNFL

Overview of OCT

"Optical Ultrasound" - echo location technique, but uses light waves

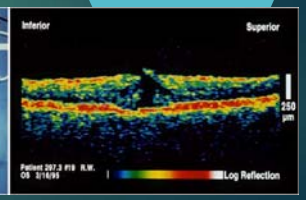


OCT basics

- Technology first introduced in 1991 by MIT: in vitro scan of human retina
- 1993: first in vivo scans of macula and optic disc
- 1996: commercially available as ophthalmology / optometry diagnostic tool

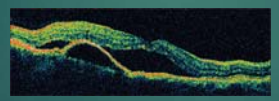
First OCT prototype

Scan of full thickness macular hole taken with original model



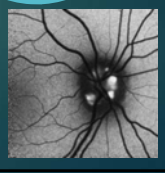
OCT basics

- Non-invasive, provides cross-sectional views of live retina
- Utilizes inferometry: near-infrared light (long wavelength)
  - Optical beam directed at tissue → small portion of light reflects back and collected
- Initially utilized for retinal pathology:
  - Macular hole, macular edema, epiretinal membrane, pigment epithelial detachment, central serous retinopathy



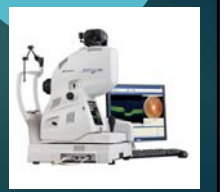
- Now commonly used for optic nerve pathology:
  - Glaucoma, optic neuritis, ONH drusen

Use of autofluorescence →



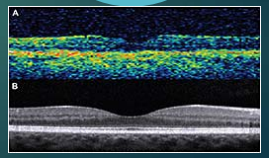
OCT basics: benefits

- Live surface/sub-surface images with great resolution
- Instant, direct imaging of tissue morphology
- No preparation of the sample or subject (no-contact)
- No ionizing radiation



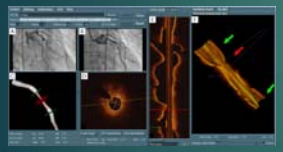
OCT basics

- Time-domain OCT
  - Original technology
  - Acquire ~400 scans per second using 6 radial slices oriented 30 degrees apart
  - Accurate to 10-15 microns
- Spectral-domain OCT
  - Acquire 20,000-40,000 scans per second
  - Increased scan rate reduces likelihood of artifact
  - Enhances the resolution
  - Decreases chance of missing lesions
  - Accurate to ~3 microns

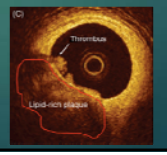


OCT basics: other uses

- Cardiology



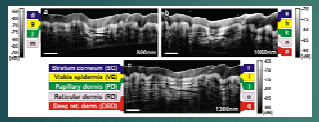
- Ultrasound vs OCT
- 3D coronary angiography: coronary artery disease



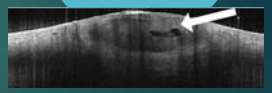
- Lipid-rich plaque

OCT basics: other uses

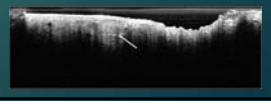
- Dermatology



- Non-melanoma skin cancer

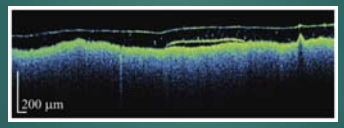


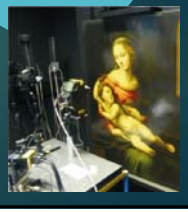
- Basil cell carcinoma with deeper tumor island



OCT basics: other uses

- Art world
  - Conservation and Restoration
    - Exfoliated varnish layer caused by incorrect ablation technique

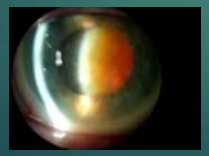


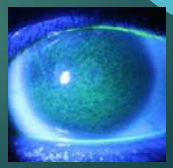


- Forgery Detection

OCT basics: Limitations

- Great for surface-level live tissue, however..
  - Images only 1-2 mm below the surface
  - Any deeper: portion of light that escapes w/o scattering is too small for detection
- High resolution imaging for transparent tissues, however..
  - Poor image quality and reliability with non-transparent tissue or media
  - Media opacities can interfere with imaging

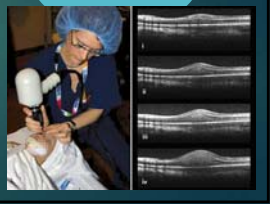




OCT basics: Limitations

- Central retinal structures can be scanned by OCT, however..
  - Peripheral structures much more difficult to scan reliably
- Good image quality relies on..
  - Patient's ability to fixate accurately
  - Patient's ability to position correctly for a small time

portable models available →

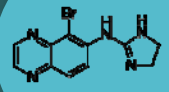


Case 3: RM

- 86 year old Caucasian male ---- 6 month follow-up
  - End-stage glaucoma OS, suspect OD

Case 3: RM

- Ocular medications: artificial tears prn OU, brimonidine BID OS
- Ocular history:
  - End-stage glaucoma OS, suspect OD
    - OD untreated IOP range: 9-18
    - OS untreated IOP range: 20-33 ---- OS treated IOP range: 10-22
  - Diabetes II w/o retinopathy or macular edema OU
  - Pseudophakia OU
  - Strabismic amblyopia OS (s/p EOM surgery age 3, prior BCVA 20/200)
  - Epiretinal membrane OD (BCVA 20/30)
  - Dry eye syndrome OU
  - Hyperopia, astigmatism, presbyopia OU



Case 3: RM

- Medical history:
  - Hypertension, high cholesterol
  - Diabetes II x 2003 controlled with metformin, A1C 6.0%
  - PTSD
- Family History:
  - (-) glaucoma / macular degeneration / other
- Social History:
  - (-) tobacco / recreational drugs
- Allergies:
  - Effexor (anti-depressant / nerve pain)

Case 3: RM

- Entrance skills:
  - cc OD 20/40+ PH:NI
  - cc OS 20/200 w/ EF PH:NI
  - Pupils: RRLA OD, (+)APD OS
  - EOMS: full OD, restricted adduction OS
  - CF: Full OD, restricted OS
  - CT: large XT OS
- Slit lamp:
  - Lids/lashes: Clear, incomplete blink OU
  - Conjunctiva: tr injection 360 OU
  - Cornea: clear OU
  - Iris: flat and intact 360 OU
  - A/C: Deep & Quiet OU
  - Lens: PCIOL, centered/clear OU

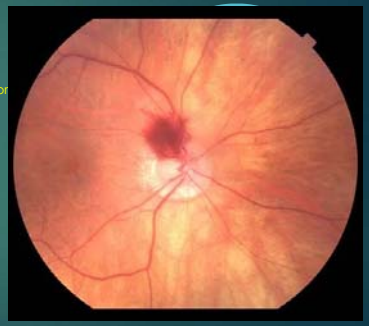
Current Rx: OD: +0.50-2.00x095
OS: balance lens
Near ADD +3.00

Manifest Rx: no change/improvement OU

Goldman Tonometry:
OD: 14 mmHg
OS: 15 mmHg (w/ treatment)

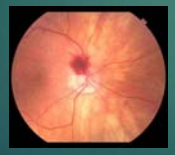
Case 3: RM

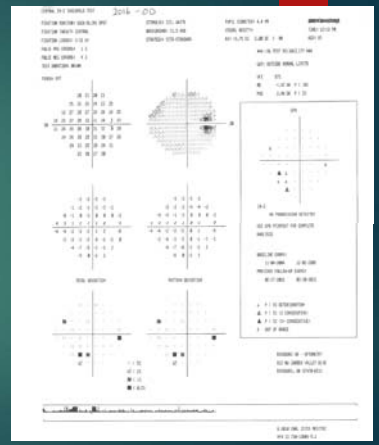
- Dilated Fundus Exam
  - C/D ratio
    - OD: 0.60, (+)PPA
      - round elevated vitreous heme over superior
    - OS: 1.0 cupped out
  - Macula
    - OD: mild ERM (-)CSME
    - OS: Clear (-)CSME
  - Vessels
    - OU: 2/3
  - Posterior Pole & Periphery
    - (-) holes / tears / detachments OU



Case 3: RM

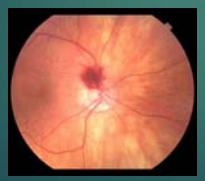
- HVF 24-2 SS Threshold Test
  - OD: last was early 2016
    - 3/15 FL, but no pattern defects
  - OS: unable due to fixation difficulty

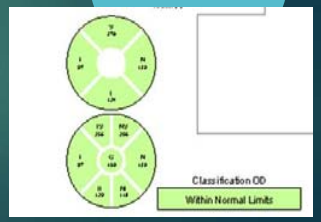




Case 3: RM

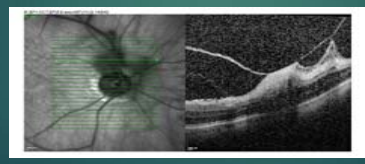
- OCT Optic Nerve rNFL analysis
  - OD: reliable
  - OS: unable due to fixation difficulty
  - "Within Normal Limits" and "All Green"…

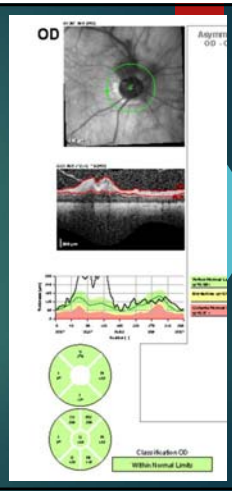




Case 3: RM

- Impression:
  - New hemorrhage obscuring true rNFL findings
    - ARTIFACT cause by hemorrhage
  - Excessive thickening superiorly from VPT
  - Causing shearing of nerve tissue

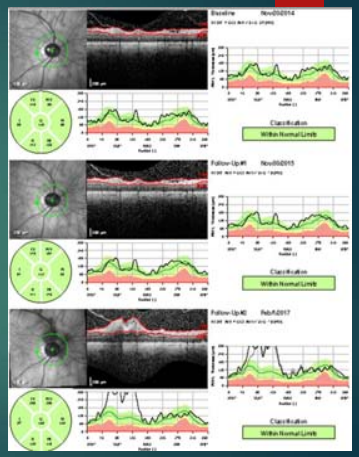




Case 3: RM

- OCT Progression Analysis
  - Nov 2014
  - Nov 2015
  - Feb 2017
  - VPT evident in previous scans…

But less obvious artifact than at present



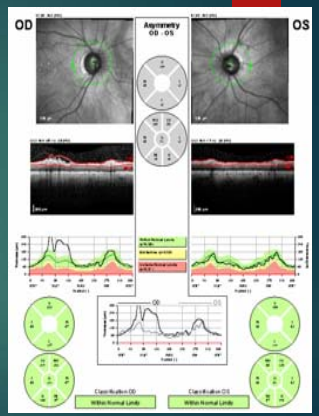
Quick Case Review

CASE 1…CASE 2…CASE 3

Case 1: BW

- 85 year old Caucasian male
  - 6 month follow-up
  - CC: no vision or ocular complaints
  - h/o glaucoma-suspect OU

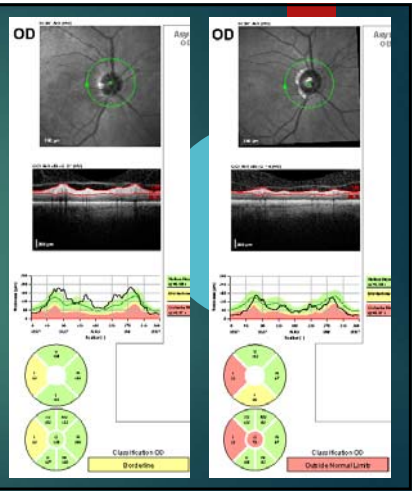
OD: VPT causing artifact on OCT



Case 2: BP

- 62 year old Native-American male
  - annual eye exam
  - CC: Pt would like new bifocals

2014 vs 2017
VPT caused artifact on OCT in 2014;
Perhaps still causing artifact in 2017?
- No other testing documented (HVF etc)

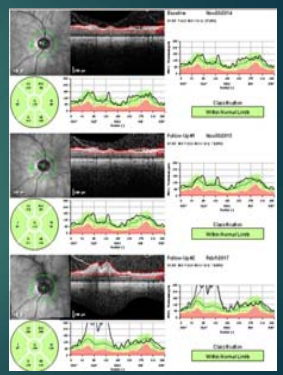


Case 3: RM

- 86 year old Caucasian male
  - 6 month follow-up
  - End-stage glaucoma OS, suspect OD
- OD: new vitreous heme at ONH
  - Drastically thickened rNFL measurements

Evidence of long-standing VPT causing artifact on all 3 scans

- Not noted in charts
- No change in follow-up or management/plan



Artifacts on OCT can interfere with interpretation and therefore management

STOPPING AT
"WITHIN NORMAL LIMITS" OR "ALL GREEN"
CAN MISS IMPORTANT OPTIC NERVE FEATURES ON OCT

Discussion

- Study by Liu et al, Harvard (2016): Review of OCT scans
  - 1070 out of 2313 OCT scans had at least 1 artifact --- 46.3%
    - Most common cause
      - de-centration error / posterior vitreous detachment
    - Increased prevalence of artifact
      - VAs <20/40 / moderate or severe cataract / advanced glaucoma
- Conclusion:

  "Clinicians should first assess scans for artifacts before making therapeutic decisions based on rNFL thickness measurements"

Discussion

- Chen and Kardon (2016): Pubmed literature review
  - Artifacts on OCT scans can lead to misinterpretations of:
    - rNFL thickness
    - Spatial distribution of rNFL bundles
    - Optic disc size and cupping
  - Factors that can confound rNFL analysis
    - Human error (eg. wrongly entered birthdate)
    - Poor signal strength at moment of capture
    - Peripapillary atrophy
    - Long or short axial lengths
    - "Ocular diseases that can cause an artifactual increase in rNFL thickness"

Discussion

- Chen and Kardon (2016): Pubmed literature review
  - Propose a systematic approach to OCT analysis
    - Similar to systemic approach recommended for Humphrey Visual Field analysis
    - Helps to identify and interpret ARTIFACTS to draw accurate conclusions from OCT scans
      - See next 3 slides
- Conclusion:

  "…we need to be cognizant of the artifacts that can occur with OCT. Failure to recognize some of these artifacts can lead to misdiagnoses and inappropriate investigations."

Systemic Approach to OCT analysis: Chen and Kardon (2016)

1. Confirm the name and age of the patient

   Measurements are made against age matched controls
2. Check signal strength

   Signal strength ≥7 out of 10 is preferable (for Cirrus OCT)
3. Check refractive error and, if available, axial eye length

   Axial eye length is particularly helpful for patients who are pseudophakic or who have had refractive surgery

Systemic Approach to OCT analysis: Chen and Kardon (2016)

4. Interpretation of the optic disc

Examine the thickness and probability retina maps for the presence of rectangular areas of absolute rNFL loss that do not match the anatomical distribution of rNFL arcuate bundles. Non-anatomical areas of loss typically indicate errors in segmentation

Compare the fundus image and thickness maps to ensure that the identification of the disc border and cup by OCT corresponds to the clinical estimation

Compare the OCT-based fundus image or en face image with the superimposed probability map for evidence of ocular torsion and to assess the angle of exit of the branches of the retinal arteries from the disc, which help predict the distribution of the arcuate nerve bundle locations

Examine the TSNIT rNFL plot and make note of whether the location of the peaks correspond to the peaks from the normative database. Common associations with discrepancy: long or short axial eye length, abnormal location of vessels as they exit the disc, and ocular torsion. Local areas with a thickness less than 40 µm are typically due to errors in segmentation unless the patient has longstanding severe optic neuropathy

Systemic Approach to OCT analysis: Chen and Kardon (2016)

5. Recognize that ocular disease can create errors in segmentation

The macular GCL-IPL thickness measurement is especially prone to segmentation error, especially in the presence of optic disc edema and outer photoreceptor disease (e.g., macular degeneration). A GCL-IPL thickness less than 40 µm is typically due to errors in segmentation unless the patient has longstanding severe optic neuropathy

6. Interpretation of subsequent scans for estimating progression of disease

Measurements from different brands of OCT machines cannot be directly compared

Ensure that the same scan location is compared between sequential scans

Conclusion

- Optical Coherent Tomography (OCT)
  - 21st century technology
  - Undeniable ability to detect subtle optic nerve disease
  - Failure to recognize ARTIFACTS or misinterpretation of ARTIFACTS can lead to errors in clinical judgement
    - False concerns
    - False reassurance – all green is not always normal – dig deeper

When interpreted correctly, OCT rNFL analysis is an invaluable diagnostic tool

References

Hwang Y, and Y Kim. "Peripapillary retinal fiber layer thickening associated with vitreopapillary traction." Seminars in Ophthalmology. 30.2 Mar 2015: 136-8.

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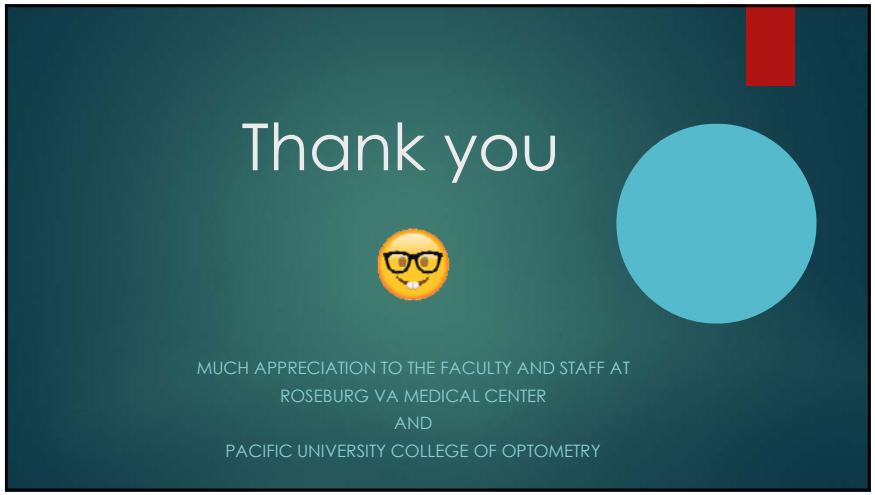
Brezinski, M, and J Fujimoto. " Optical coherence tomography: high-resolution imaging in nontransparent tissue." Journal of Selected Topics in Quantum Electronics. 5.4 Jul/Aug 1999: 1185-1192.

Chen, J and Randy Kardon. "Avoiding Clinical Misinterpretation and Artifacts of Optical Coherence Tomography Analysis of the Optic Nerve, Retinal Nerve Fiber Layer, and Ganglion Cell Layer." Journal of Neuroophthalmology. 36.4 Dec 2016: 417-438.

Liu, Y et al. "PatienAt Characteristics Associated with Artifacts in Spectralis Optical Coherence Tomography Imaging of the Retinal Nerve Fiber Layer in Glaucoma." American Jounal of Ophthalmology. 159.3 March 2015: 565-76.


Thank you
MUCH APPRECIATION TO THE FACULTY AND STAFF AT
ROSEBURG VA MEDICAL CENTER
AND
PACIFIC UNIVERSITY COLLEGE OF OPTOMETRY

Iris and Angle Abnormalities

COPE CATEGORY: TREATMENT & MANAGEMENT OF OCULAR DISEASE: ANTERIOR SEGMENT (AS)

Dane Sultzer, O.D.
Spokane VAMC Resident

Iris and Angle Abnormalities

- General information
- Few clinical cases
  - Assessment techniques
  - Treatment considerations

Iris and Angle Assessment

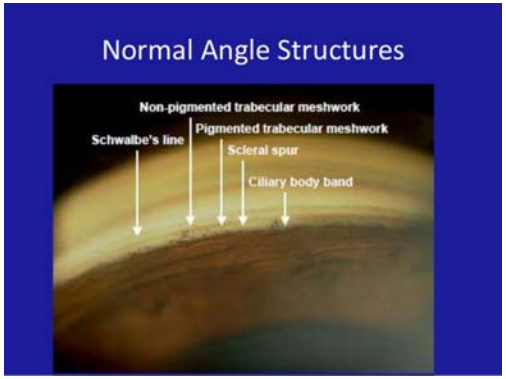
- Slit lamp
- Gonioscopy
- Optical Cohearance Tomography
- Photography
- High-resolution ultrasound biomicroscopy
- Iris FANG

Introduction

- Clinical Picture
  - Evaluate iris and angle for risk factors
  - Consider all of the data
  - Work to obtain additional information when necessary

http://www.flickriver.com/photos/cormackphotos/437848724/

Review



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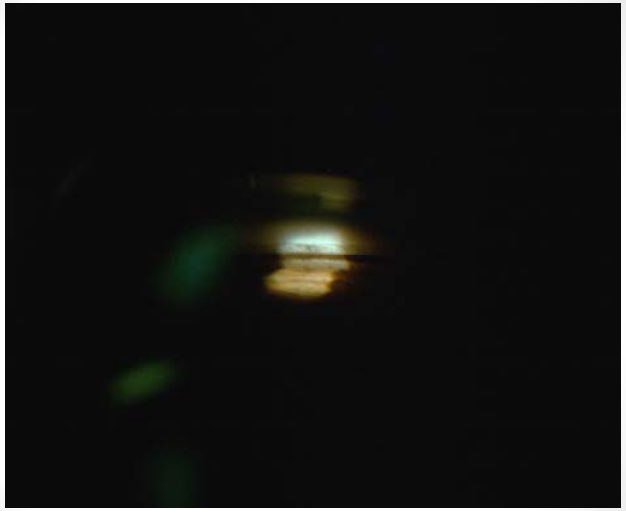


CASE 1

Chief Complaint:

- **68 year old** Caucasian male. Patient presents for a complete eye examination. No ocular complaints.

Ocular History:

- **Blunt trauma, OS,** 12-15 years ago.
- **Glaucoma suspect, OS**: No current treatment.

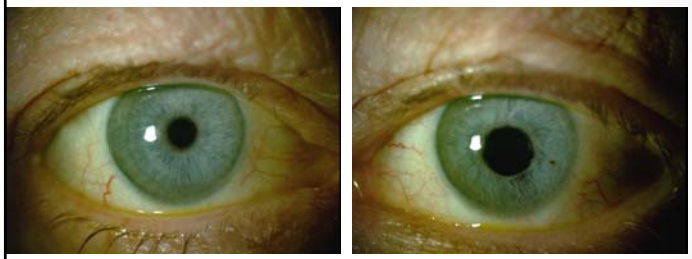
Examination:

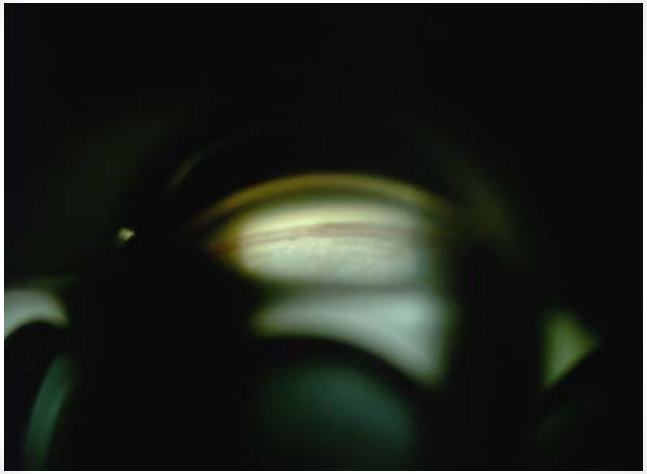
- **BCVA OD 20/20- OS 20/20-**
- **Anisocoria** (OS>OD). No APD.
- GAT: OD 13

  OS 17, OS Tmax is 21

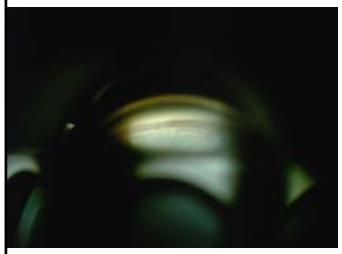
- All other findings are unremarkable.

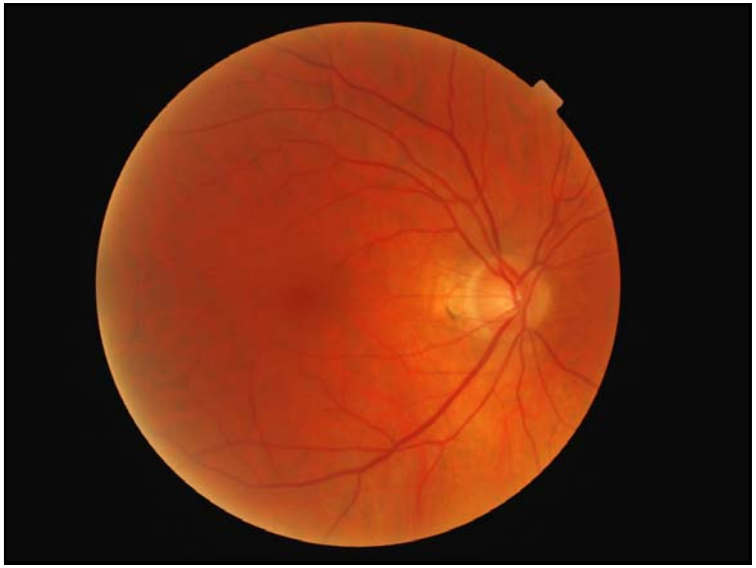
CASE1

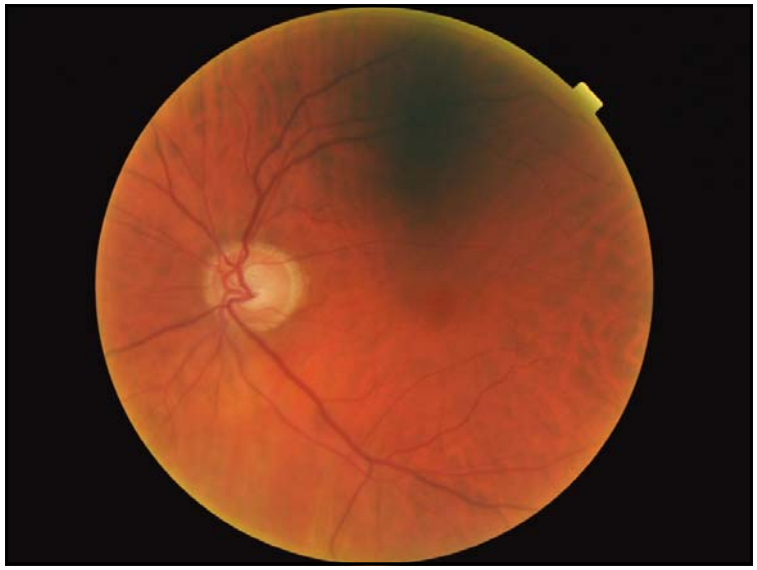


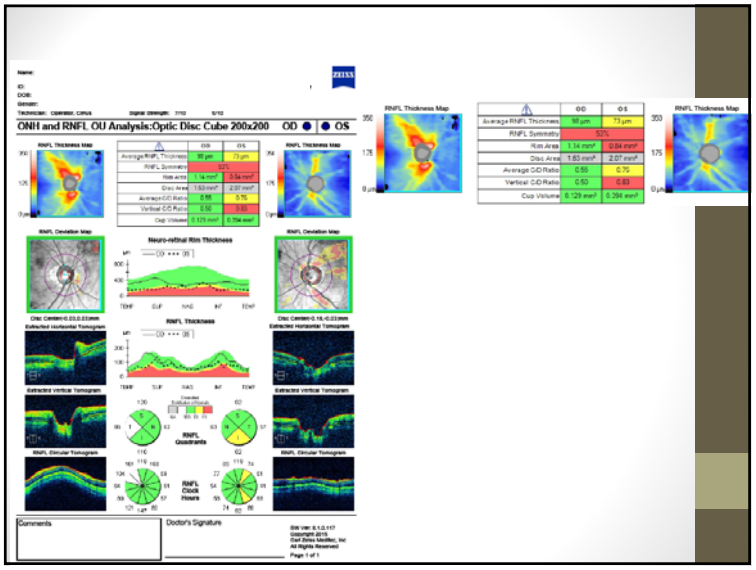


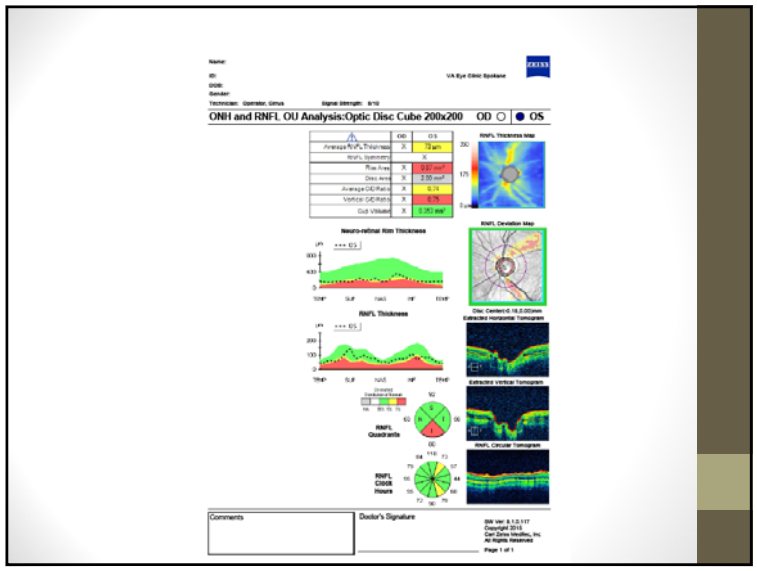
Case 1











Visual Field

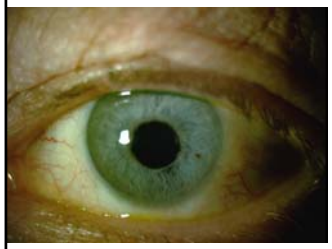
- Pre-perimetric Glaucoma: **presence of glaucoma** in **absence** of identifiable **functional loss** **on visual field**
  - Due to redundancy of visual system, a visual field defect is not needed to make the diagnosis of Glaucoma.
- A visual field without a glaucomatous defect can rule out severe glaucoma.

1. Litwak, Anthony B. *Glaucoma Handbook*. Boston: Butterworth-Heinemann, 2001. Print.
2. "PRE-PERIMETRIC GLAUCOMA." PRE-PERIMETRIC GLAUCOMA | American Academy of Optometry. N.p., n.d. Web. 21 Apr. 2017.

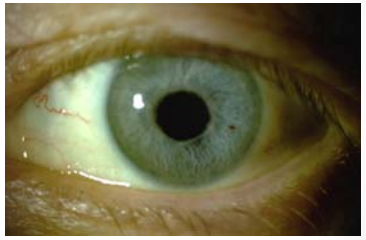
IOP: OD 12 OS 18

PRE TREATMENT OS

IOP 18
4.5 MM PUPIL



POST GTTS (SAME DAY) OS
BRIMONIDINE
IOP 11
4.0 MM PUPIL



Brimonidine BID OS only
RTC in 4-6 weeks for IOP/ONH at 4-6 pm

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1936355/

CASE 2

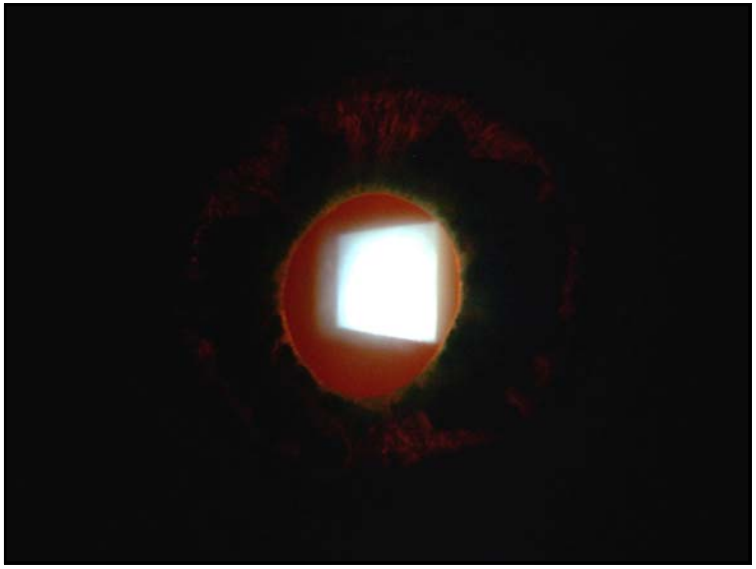
- **PIGMENT DISPERSION SYNDROME**
  - Pigment deposition on the corneal endothelium in a vertical spindle patter (**Krukenberg spindle**)
  - **Pigmented trabecular meshwork**
  - Mid-peripheral **iris transillumination defects**

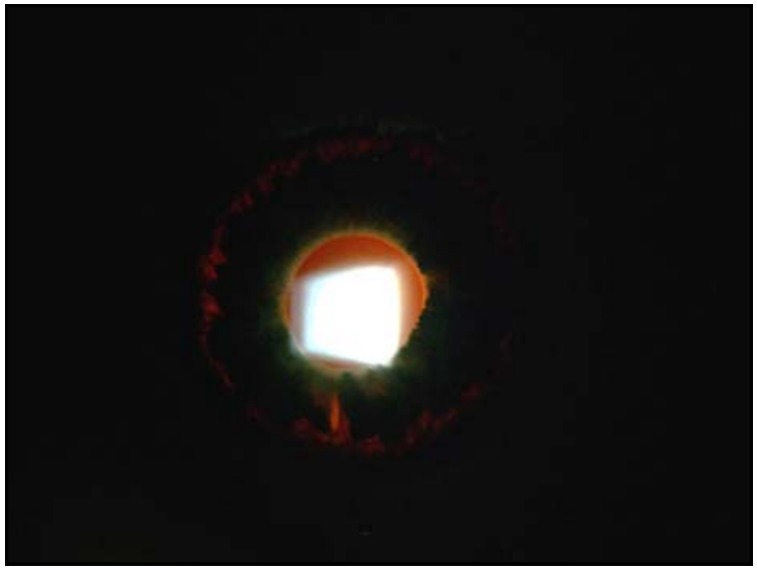
CASE 2

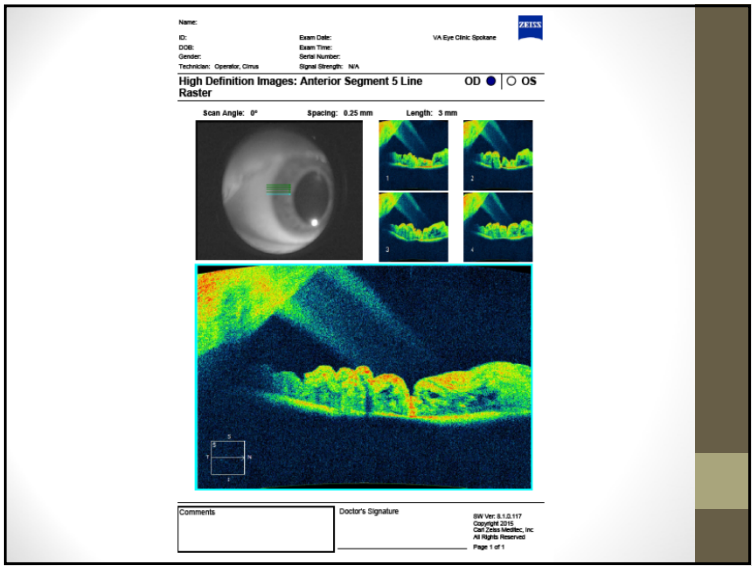
- **62 year old** Caucasian male. Patient presents for a complete examination. No ocular complaints.

Ocular History:
- **Pigmentary Glaucoma OU**
  - -Patient is on maximum topical therapy OU
  - -s/p SLT OU, FAILED BLEB OD

Examination:
- **BCVA** 20/30- OD, OS
  - Potential Acuity 20/20- OD, OS
- **IOP** 18/18
  - consistently in mid-high teens OU
- All other findings unremarkable.



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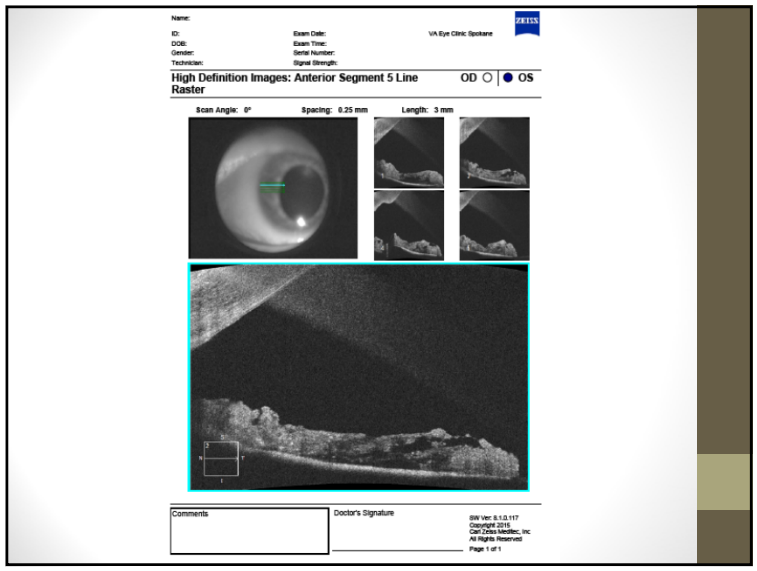
Doctor's Signature
Page 1 of 1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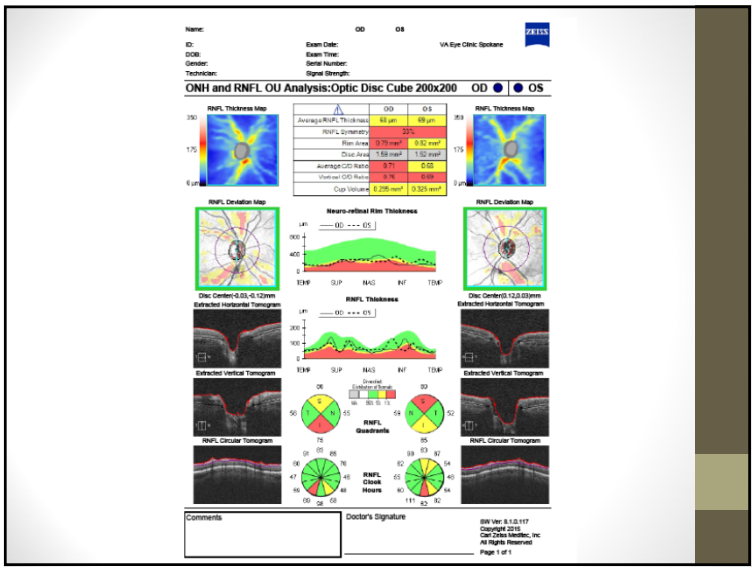
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VA Eye Clinic Spokane
ONH and RNFL OU Analysis:Optic Disc Cube 200x200
OD ● | ● OS
RNFL Thickness Map
RNFL Deviation Map
Neuro-retinal Rim Thickness
RNFL Thickness
Extracted Horizontal Tomogram
Extracted Vertical Tomogram
RNFL Circular Tomogram
RNFL Quadrants
RNFL Clock Hours
Comments
Doctor's Signature
Page 1 of 1

Pigment Dispersion

LPI should strongly be considered:

- **Younger patient**
- Patient with a **concave iris approach**

See reference slide for supporting studies

CASE 3

- **65 year old** Caucasian male. **New patient** presents for complete examination.

Chief Complaint:

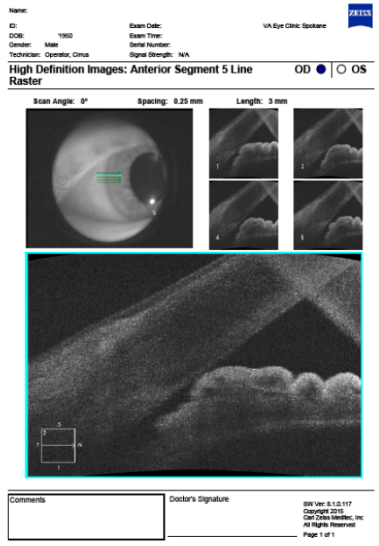
- The patient reports **blurry vision in both eyes, at distance and near**. The symptoms are **constant** , without relief. The symptoms have been present for about **"2 years."**

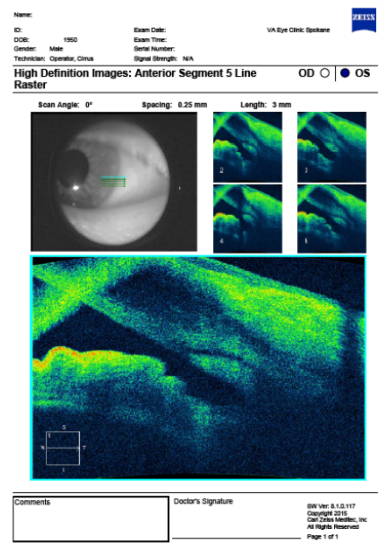
Ocular History:

- **LPI OU**

Examination:

- BCVA 20/30- OD, OS
  - Potential acuity 20/20- OD, OS
- IOP 19/19
- All other findings/history unremarkable





CASE 3

- Assessment:
  - Visually Significant Cataract OU
  - Plateau Iris Configuration OU
  - **Glaucoma Suspect OU** secondary to ONH appearance
- Plan:
  - **Cataract extraction OU**
    - Re-evaluate ONH s/p cataract extraction.

CASE 3

- PLATEAU IRIS CONFIGURATION
  - CONGENITAL, ANTERIORLY POSITIONED CILIARY PROCESS
  - SENILE CATARACT CAN FURTHER NARROW THE ANGLE

PEARLS

- Anterior OCT can aid in diagnosis
- Treatment: when necessary, aimed at pulling the iris away from the angle
  - **Laser peripheral iridoplasty**
  - **Iridectomy**: Effective
    - Studies show a few eyes will still develop chronic angle closure
- Imaging studies showing that anterior chamber depth increases after cataract extraction

CASE 4

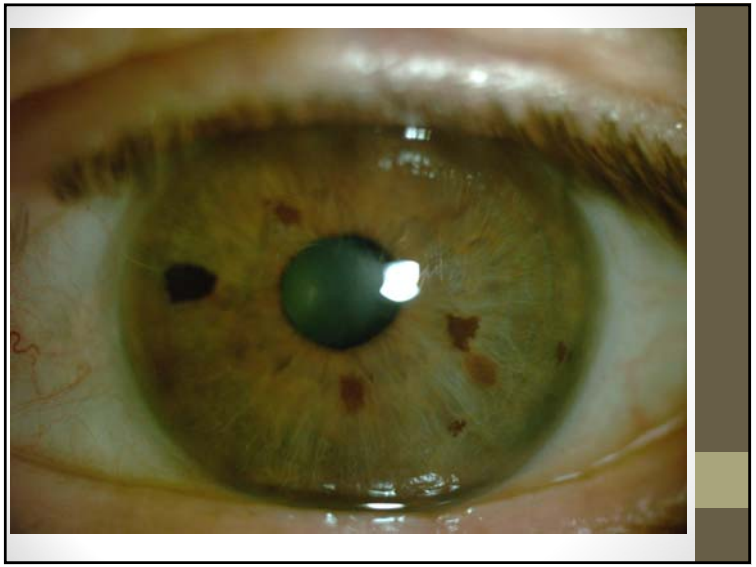
IRIS NEVUS

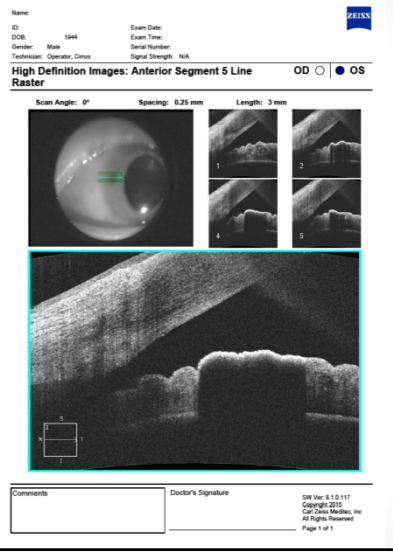
VS

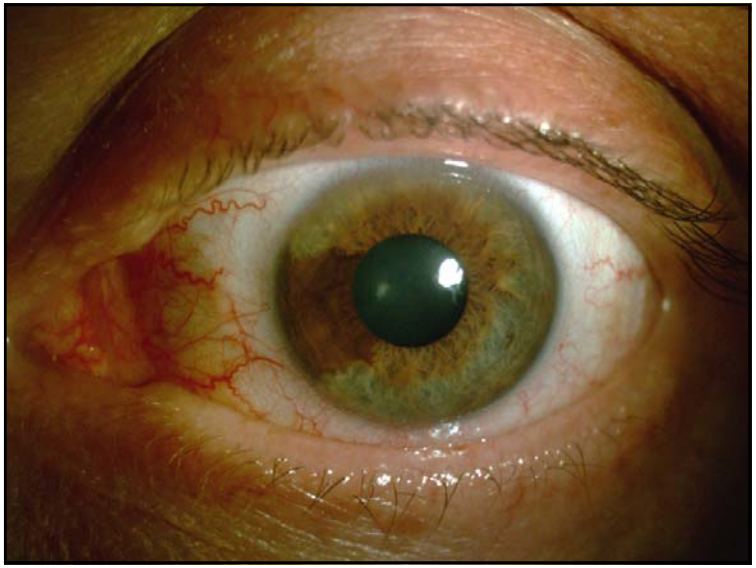
IRIS MELANOMA

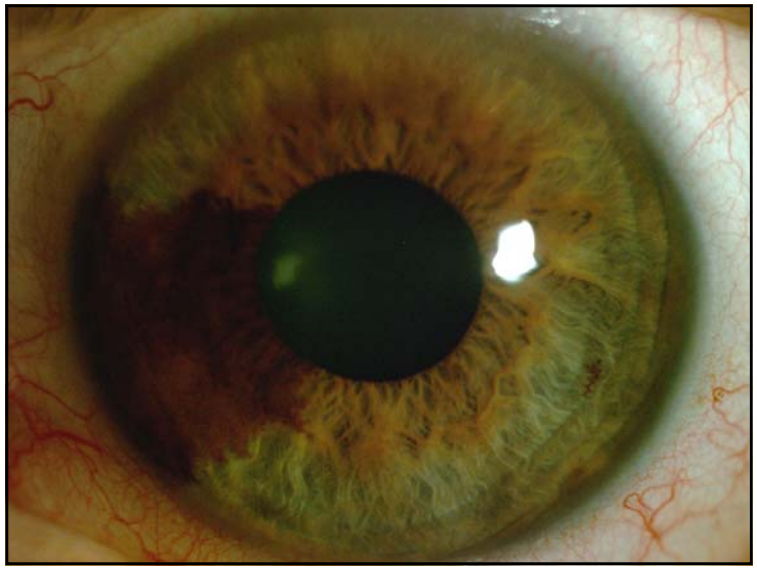
IRIS NEVUS	IRIS MELANOMA
FEATURES	
MINIMAL DISTORTION OF IRIS • MINIMAL/NO PUPIL IRREGULARITIES	PUPIL DISTORTION
DOES NOT INVADE OTHER STRUCTURES	INVADES OTHER STRUCTURES
NO GROWTH / SLOW GROWTH	GROWTH
LIMITED/NO VISIBLE VASCULATURE	SIGNIFICANT VASCULARIZATION CAN OCCUR • CAN CAUSE SPONTANEOUS HYPHEMA
NA	AVERAGE ONSET: 40-50 Y/O (~10-20 YEARS EARLIER THAN POSTERIOR MELANOMAS)
NA	RARELY METASTASIZE EXEPTION: **WHEN ENTIRE IRIS STROMA, ANGLE OR CB IS INVOLVED**
NA	TYPICALLY OCCUR INFERIORLY

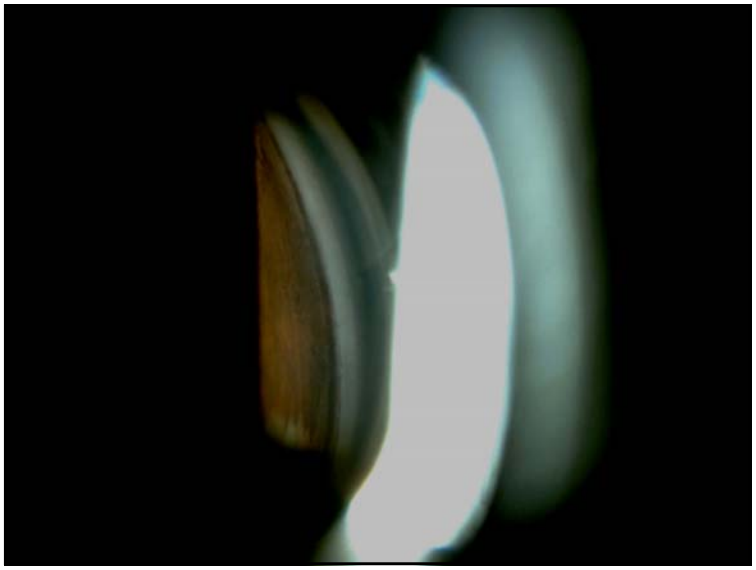
PRIMARY INFORMATION SOURCE: Ophthalmic Pathology and Intraocular Tumors: 2004-2005. San Francisco: American Academy of Ophthalmology, 2004-2005. Print.

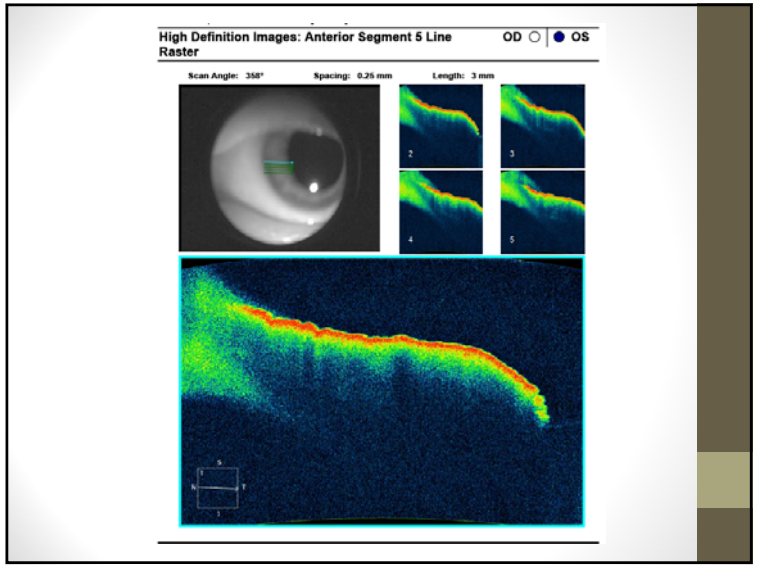








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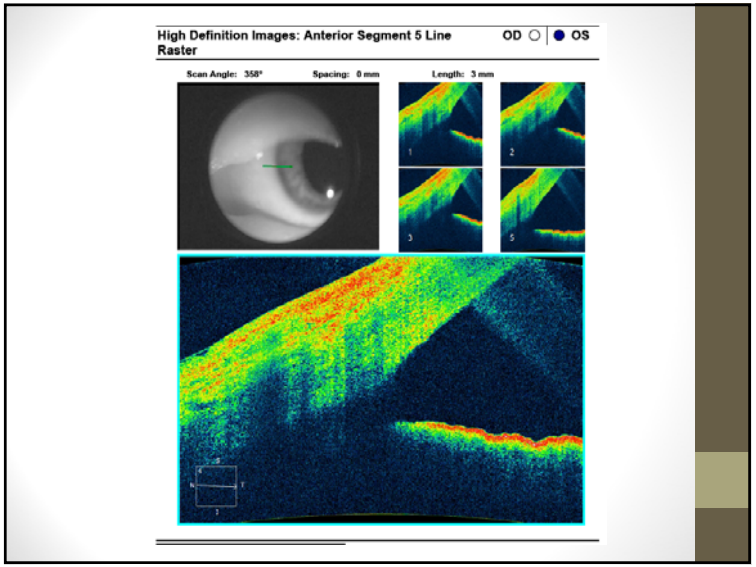
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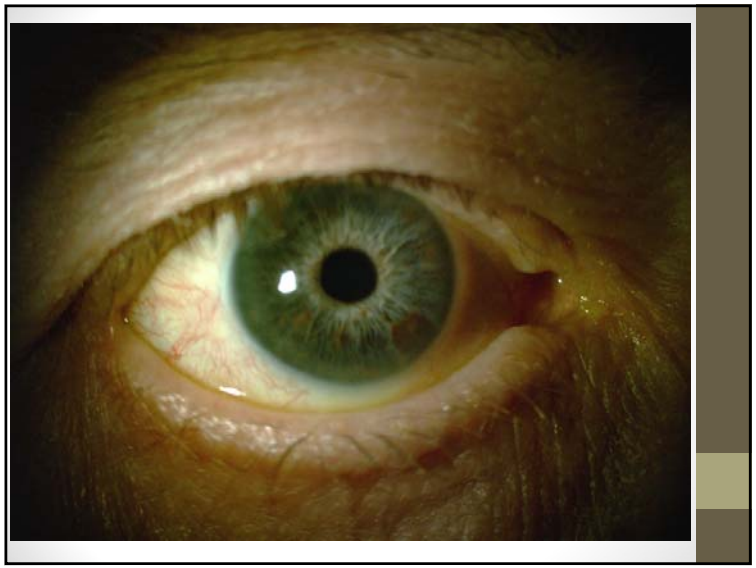
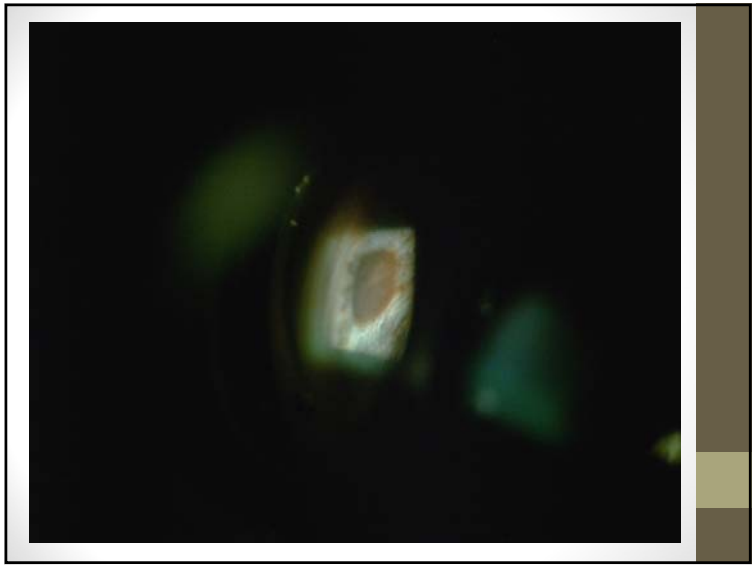
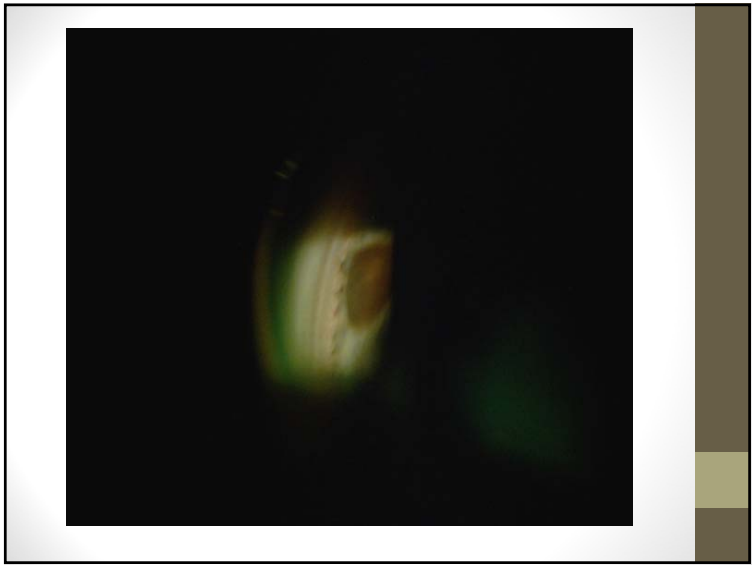
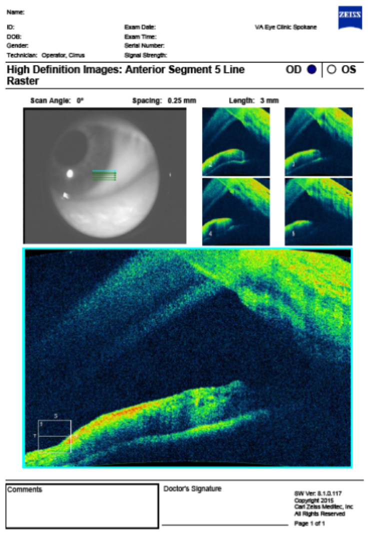
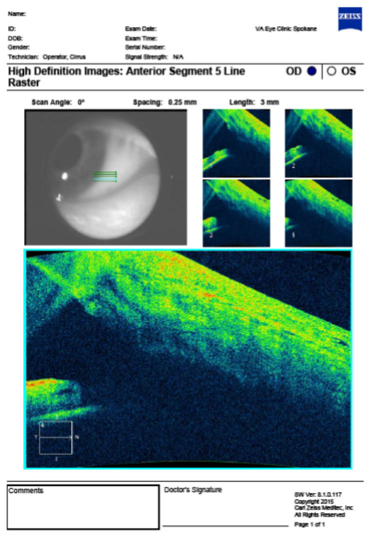
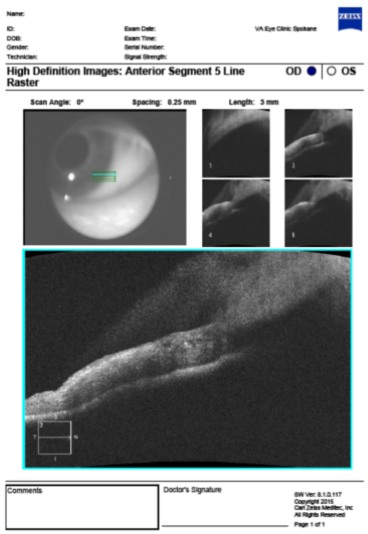




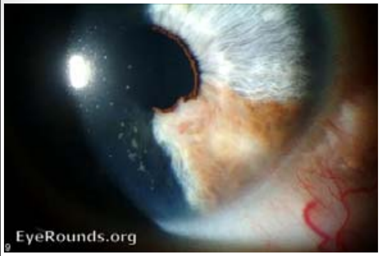




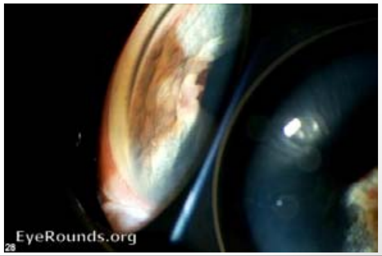




Iris Melanoma



http://webeye.ophth.uiowa.edu/eyeforum/atlas/pages/iris-melanoma.html



http://webeye.ophth.uiowa.edu/eyeforum/atlas/pages/iris-melanoma.html

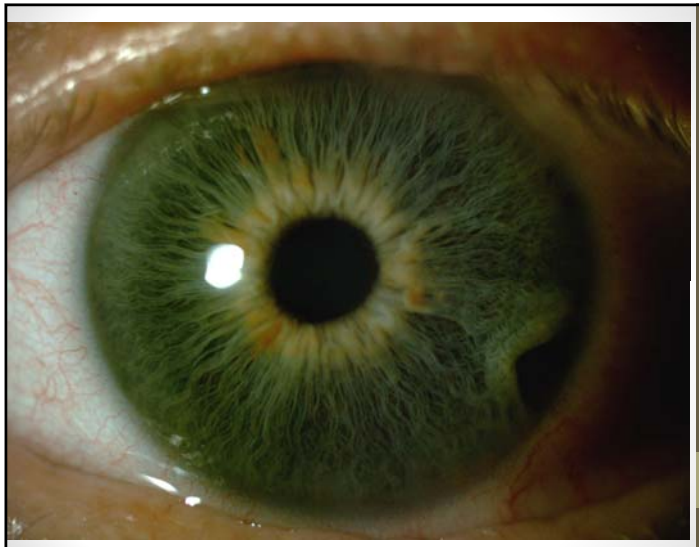
CASE 5

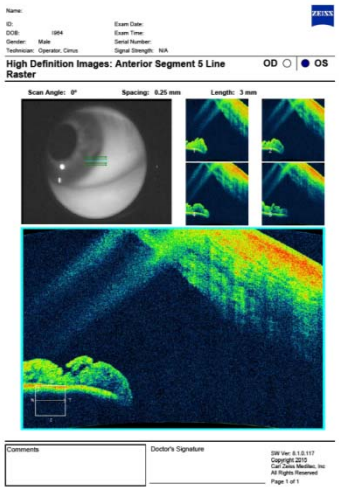
- 52 year old Caucasian male presents for complete exam

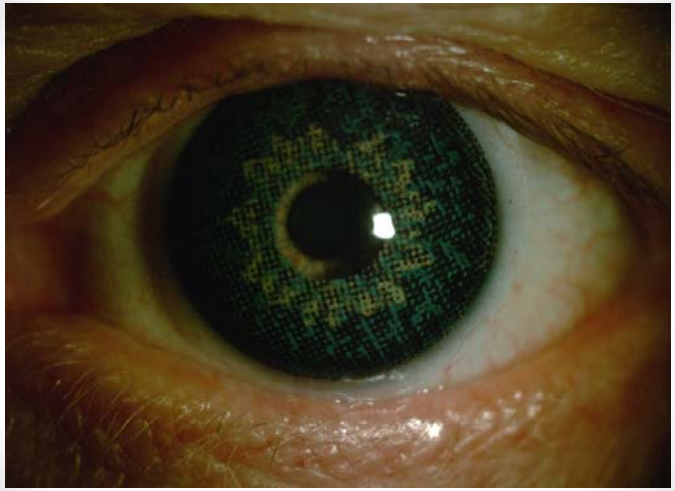
Chief Complaint:
- **Glare in the left eye**. The symptoms have been present for an unknown number of years. No relief. No associated factors.

Ocular History:
- of **blunt trauma, OS**, when 7 years old

Examination:
- BCVA 20/20- OD, OS
- **IOP 16/16**
- All other findings unremarkable



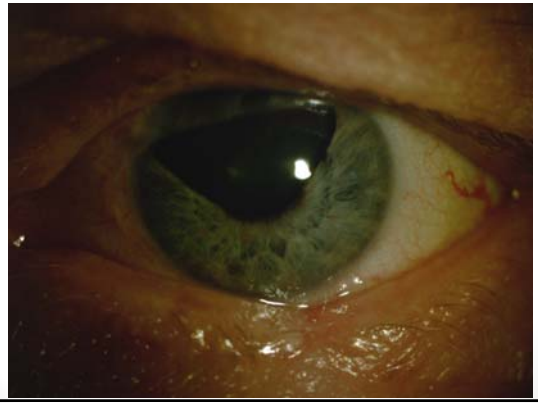


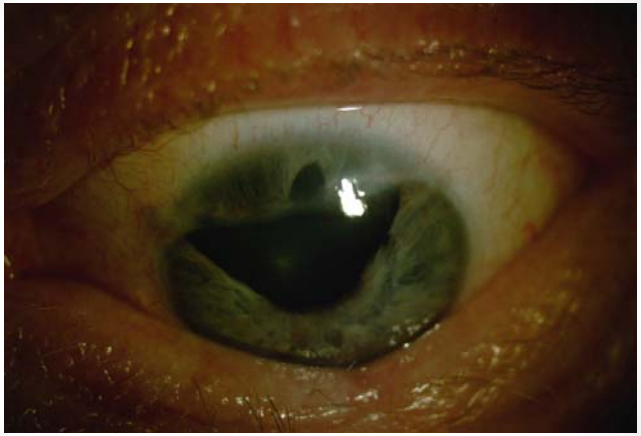


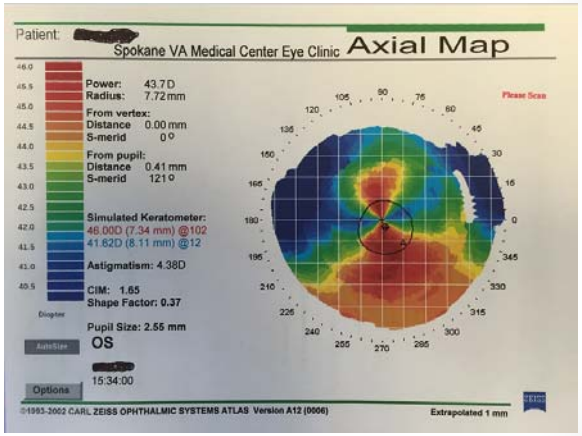
Case 5b

Chief Complaint:

- **Glare** symptoms for last **15 years**. Often resulting in an "**immediate headache.**"







CONCLUSION

- **Evaluate risk factors** and **obtain baseline information**
  - Aids in identifying progression early
  - Improves quality of care
- When necessary, use other instruments to **gain additional information**
- Always compare the two eye and **look for symmetry**
- When appropriate refer patient to specialist/colleagues

REFERENCES

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- "PRE-PERIMETRIC GLAUCOMA." PRE-PERIMETRIC GLAUCOMA | American Academy of Optometry. N.p., n.d. Web. 21 Apr. 2017.
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- https://www.ncbi.nlm.nih.gov/pubmed/17070597
- http://www.medical-hypotheses.com/article/S0306-9877(06)00855-3/pdf
- https://www.ncbi.nlm.nih.gov/pubmed/27373109
- https://www.ncbi.nlm.nih.gov/pubmed/25429743
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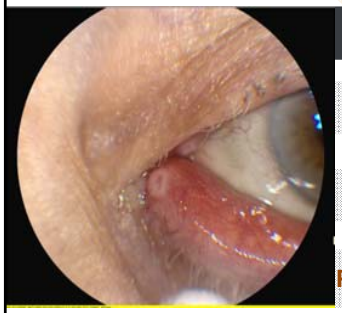
- https://www.ncbi.nlm.nih.gov/pubmed/24062148
- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1936355/

WHAT "LITHS" IN THE LACRIMAL SYSTEM

Alanna Louie OD, M.Ed.
Portland VA Health Care System

Northwest Residents Conference
June 9, 2017



OBJECTIVES

- Review of nasolacrimal system anatomy
- Recognition and management of lacrimal canaliculitis
- Review of dacryocystitis

ANATOMY OF THE NASOLACRIMAL SYSTEM

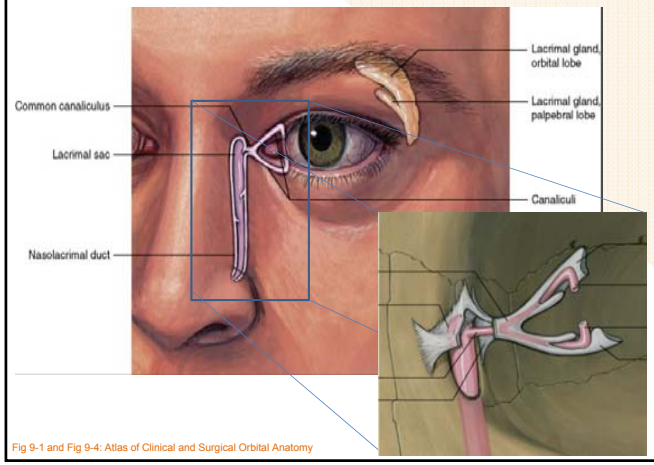
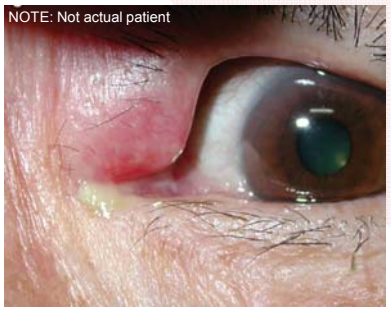


Fig 9-1 and Fig 9-4: Atlas of Clinical and Surgical Orbital Anatomy

CASE: 78 YO F



CC:
Pink bump on left upper eyelid, has been there for 1 year; itches without pain and is able to express white mucous discharge QID-BID. Uses Soothe XP for OS blurred vision, provides mild relief.

- H/o ?oral med, used for 2 days then d/c due to no improvement

CASE: 78 YO F

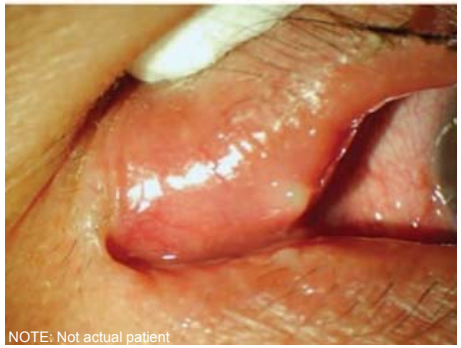
- **VA sc**:
  OD: 20/40+2 PH 20/20
  OS: 20/40 PHNI
- **Entrance Testing**:
  – PERRL (-)APD OD,OS
  – EOM: Full OD,OS
  – FTFC OD,OS

POHx
- LEE 03/2015: Dry eye syndrome OU with **focal edema OS nasal UL**
- Pseudophakia OU 2015

PMHx
- Cardiovascular: HTN, Hyperlipidemia
- Endocrine: DMT2

Medications: for above, names unknown; NKDA

CASE: 78 YO F



Biomicroscopy
OD: Unremarkable
OS: Nasal UL ~5mm focal edema and erythema +swollen punctum
- ~1mm pannus nasally
- Underlying palpebral conj injection

**White mucopurulent discharge +small white granular concretions expressed from superior punctum

CASE: 78 YO F

Assessment

Chronic lacrimal canaliculitis OS

- Prior oral therapy as recommended not beneficial, pt didn't adhere to tx plan as prescribed

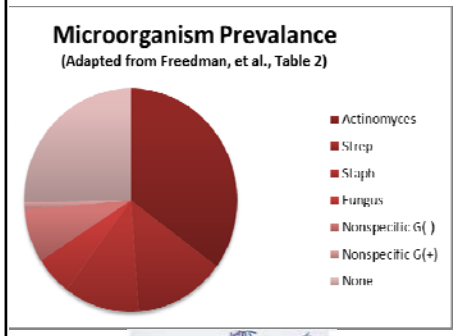
Plan

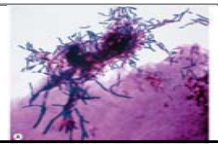
- Warm compresses and eyelid massage BID OS
- Tobradex QID OS x 10 days, shake bottle
- Cephalexin (Keflex) 500mg Q12H x 10 days
- RTC 2 weeks

FOLLOW-UP

2 week f/u	• **Improved "10 percent"** • **Persistent mucous discharge, size of area unchanged** • **Compliant with tx**	**• Discharge expressed today prior to instillation of 1 gtt TobraDex.** **• Continue tobradex QID x 2 weeks, precede each with warm compress/lid massage.** **• Continue Keflex 500mg PO BID x 10 days.** **RTC 2 weeks**
2 week f/u	• **"90 percent" resolution of itch** • **Amt of discharge decreased**	**Continue tobradex QID x 1 week** **RTC 4 weeks**
8 week f/u	**LUL still itchy and red** **Recurrence**	**Warm compresses BID then Bacitracin ung BID** **Refer to oculoplastics specialist @ Casey Eye Institute in 2-4 weeks**

LACRIMAL CANALICULITIS





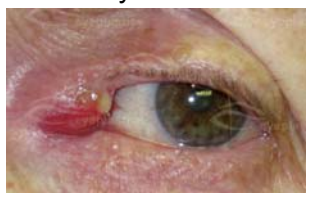
- Primary or secondary infection
- Inflammation of proximal lacrimal pathway
- 2-4% of lacrimal disease
  - MISDIAGNOSIS COMMON!
  - DIAGNOSIS DELAY!
- >50, ♀
  - Menopause?

CANALICULITIS: CAUSES

- PRIMARY: ***SPONTANEOUS***
  - LACRIMAL SYSTEM OBSTRUCTION PREDISPOSES ACCUMULATION OF BACTERIA
- SECONDARY: ***FOREIGN BODY***
  - **PUNCTAL PLUGS**
  - **INTRACANALICULAR PLUGS**

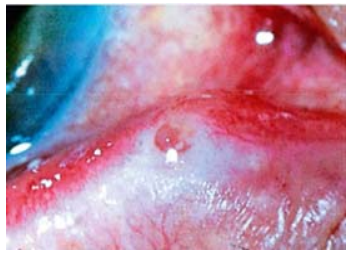
CANALICULITIS: SYMPTOMS

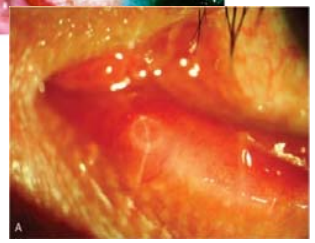
- <u>Acute</u>
  - Tenderness of medial canthus
  - Epiphora
  - +/- Discharge
  - +/- Eyelash mattering



- <u>Chronic</u>
  - Recalcitrant red eye
  - Focal swelling of medial canthus

CANALICULITIS: SIGNS

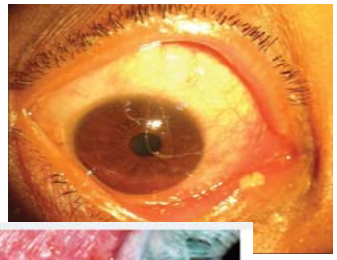


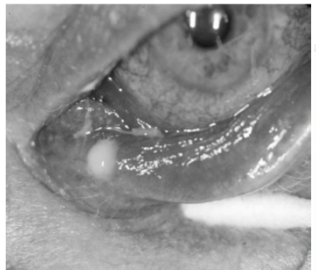


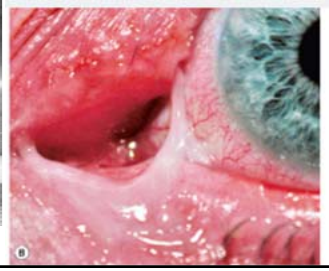
"Pouting punctum"
- Swollen
- Erythematous
- Dilated

CANALICULITIS: SIGNS

"Punctal Regurgitation"
- Mucopurulent discharge
- Expressible with **gentle** pressure

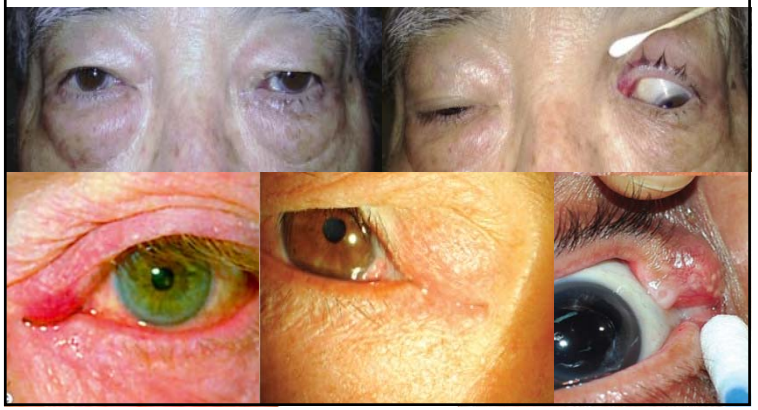




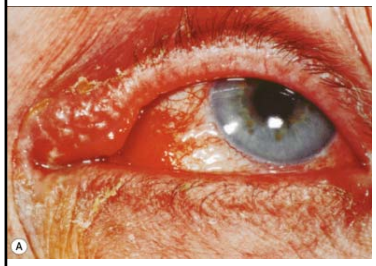


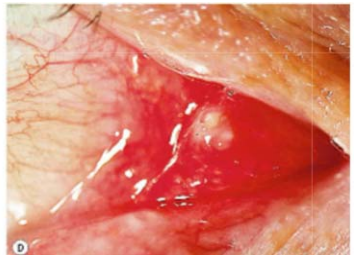
CANALICULITIS: SIGNS

Medial ¼ Eyelid swelling/thickening



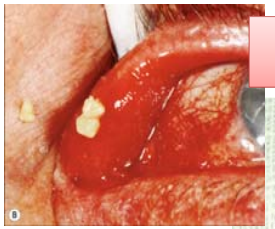
CANALICULITIS: SIGNS



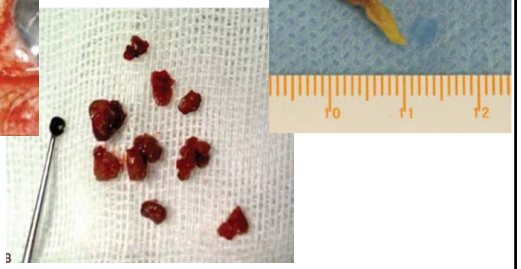


Conjunctival involvement

CANALICULITIS: SIGNS



Dacryolithiasis

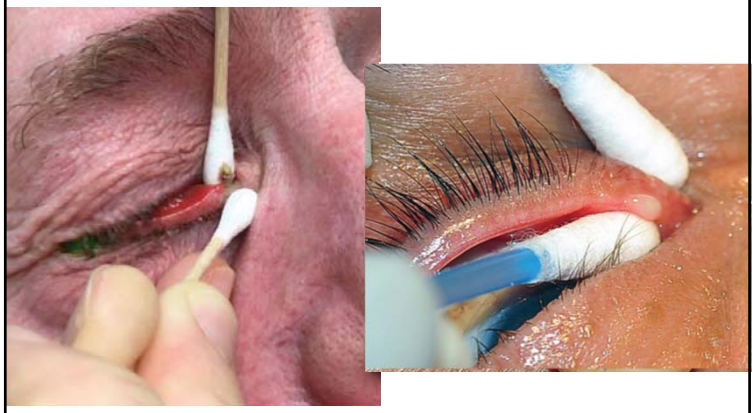


CANALICULITIS: TREATMENT

Goal: Re-establish tear flow without iatrogenic trauma

- Medical therapy alone
- Medical + Surgical intervention
- Surgical Intervention

EXPRESSION

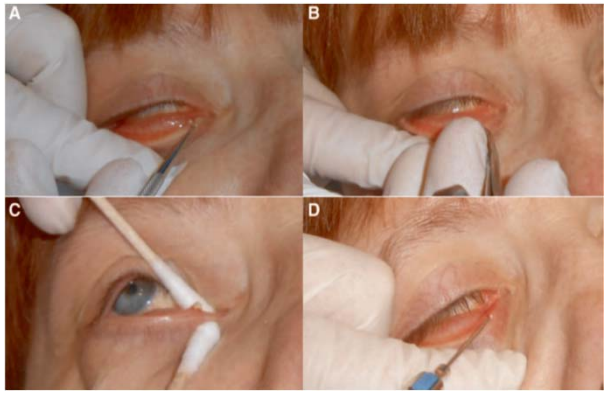


TREATMENT — Medical therapy alone

- **Warm compresses to the area & digital massage**
- **Topical antibiotics**
  - Broad spectrum until culture
    - Levofloxacin, moxifloxacin, PolyTrim or Tobramycin QID x 10 days
  - Actinomyces cultured?
    - Penicillin G ung, Neomycin, Polymyxin-B, Bacitracin
- **Oral antibiotics** x 10-14 days
  - Penicillin
  - Bactrim

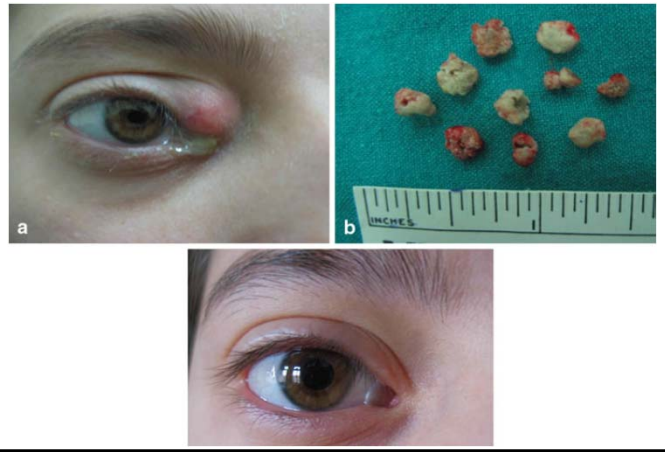
TREATMENT — Medical + Surgical intervention

Canaliculotomy with curettage



Medical + Surgical intervention

Canaliculotomy with curettage



TREATMENT

Surgical Intervention

- Curettage with punctal dilation
- Curettage with punctoplasty

Once resolved:

+/- Dacryocystorhinostomy (DCR)

Novel treatments?

Incision-sparing

- **Intracanalicular Ointment Filtration**
  - Anesthesia
  - Dilation of both puncta
  - Probing of canaliculi with expression
  - Irrigation using antibiotic (Tobradex, Cefazolin)
  - Repeat PRN

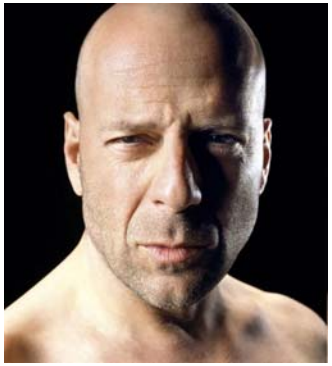
Hyperbaric O2 therapy

- 100% O2 6 days a week x 4 weeks
- 90 minute sessions

- Xu, J., et al (2015) Novel Therapy for Primary Canaliculitis: A Pilot Study of Intracanalicular Ophthalmic Corticosteroid/Antibiotic Combination Ointment Infiltration. Medicine, 94(39)
- DOI: 10.1097/MD.0000000000001611

Shauly, Y. Nachum, Z., Gdal-On, M., et al. (1993) Adjunctive hyperbaric oxygen therapy for actinomycotic lacrimal canaliculitis. Graefes Arch Clin Exp Ophthalmol; 231: 429-31

CASE: 59 YO M 2010



CC:
Mucous discharge OU AM>PM, treated 3 weeks ago with sulfacetamide 10% TID OU eyedrops (Rx from outside provider) without improvement (-)itch/burn/redness

http://www.dougrichardson.com/wp-content/uploads/2013/05/Bruce-Willis-hottest-actors-1083014_1020_1131.jpg

CASE: 59 YO M 2010

Assessment

H/o unspecified conjunctivitis, per pt report

- No evidence of mucous, foreign body, or blepharitis

Plan

- D/c sulfacetamide
- Initiate AT QID OU

RTC 1 year….

CASE: 59 YO M

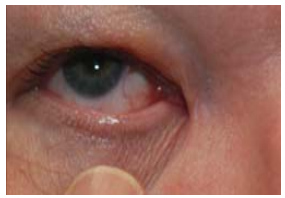
6 months **06/2010**	• **Consistent mucous discharge OU** • **Eyelids stick together x 6 months, similar symptoms for almost 1 year** • **(-)pain, moderate relief with hot water but mucous returns**	**1. Start erythromycin ung QHS** **2. Lid hygiene BID** **3. Artificial tears TID** **F/u in 4 weeks**
4 week f/u	Mild improvement	RTC 1 year for CVE
8 months 03/2011	• Green/yellow mucous discharge OU more in the AM but constant throughout the day • Redness and irritation OU, cleans eyes in shower QAM. • No relief with Refresh QD OU; x 3 months	A: Mucous fishing syndrome OU vs blepharoconjunctivitis OU with no signs of infection 1. D/c touching eyes 2. Warm compresses QAM OU 3. Tobradex QID OU RTC 1 week

CASE: 59 YO M

1 week	• **Discharge resolved**	**1. Taper Tobradex to BID OU x 1 week, then QD OU x 1 week** **RTC 1 year for CVE**
8 months 11/2011	• Yellow/green pus from the inner corners of his eyes, all day • Wakes up with eyelids stuck shut • Would like more "drops with the steroids	A: Mucous fishing syndrome OU with secondary chronic blepharoconjunctivitis OU 1. Start ketotifen, increase artificial tears 2. Don't touch eyes!
1 month	• No improvement, mucous returns 30 minutes after removal	

⏭ 10 visits/phone calls and 5 years later…

CASE: 59 YO M 08/2016



CC:
persistent mucus discharge OD>>OS green-yellow

- AM only OS, but OD has mucus discharge throughout the day (worse in AM);
- Does not think ophthalmic drops to date helped

CASE: 59 YO M

Assessment

Suspect Chronic Canaliculitis RLL

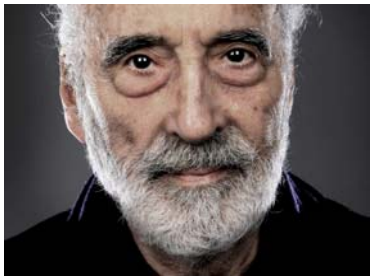
- Prior suspicon of mucous fishing syndrome with no subjective improvement with topical treatment

Plan

- Start Augmentin 500/125 TID po x 14 days
- Start Moxifloxacin 1gtt TID OD

RTC 2 weeks oculoplastics

CASE: 90 YO M



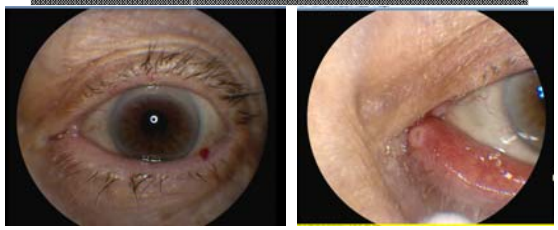
http://pixel.nymag.com/imgs/daily/vulture/2015/06/11/11-christopher-lee-3.w750.h560.2x.jpg

CC:
Mild irritation OS x 2-3 weeks with white discharge and redness

- No pain
- No eyedrop use

FOLLOW-UP

04/2016	**Dry eye syndrome OU**
07/2016	Bacterial conjunctivitis OS (treated at Kaiser)
07/2016	Presumed resolving bacterial conjunctivitis
07/2016	Chronic bacterial conjunctivitis OS
08/2016	**Suspected chronic canaliculitis LLL**



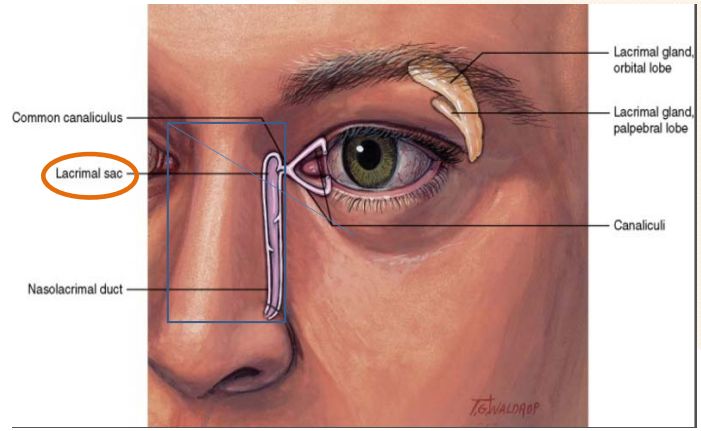
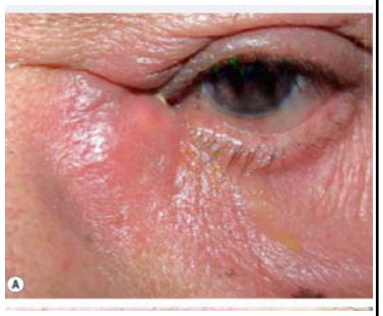


Fig 9-1 and Fig 9-4: Atlas of Clinical and Surgical Orbital Anatomy

DACRYOCYSTITIS

- Enlargement of lacrimal sac with discharge build-up
- Elevated mass under the skin
- Purulent discharge upon palpation of area over lacrimal sac
- Pain infero/nasal to the orbit



DACRYOCYSTITIS vs CANALICULITIS

- Enlargement of lacrimal sac with discharge build-up
- Elevated mass under the skin
- Purulent discharge upon palpation of area over lacrimal sac
- Pain infero/nasal to the orbit
- +/- Enlargement of upper or lower puncta
- Mucopurulent discharge with gentle pressure in nasal canthal area
- Tender

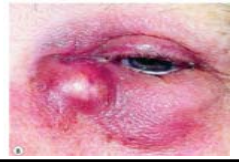
DACRYOCYSTITIS: TREATMENT

Medical/Conventional

- Oral antibiotic
  - Cephalexin 500mg QID x 7-10 days
  - Erythromycin 500mg QID x 7-10 days
  - Augmentin 500 mg Q8H
  - Pediatric → Augmentin
- Topical antibiotic
  - 4th gen FQL
- Warm compresses/lid massage

Surgical:

- If the acute infection progresses to a superficial pointing mass and the pt is uncomfortable, surgical drainage is appropriate



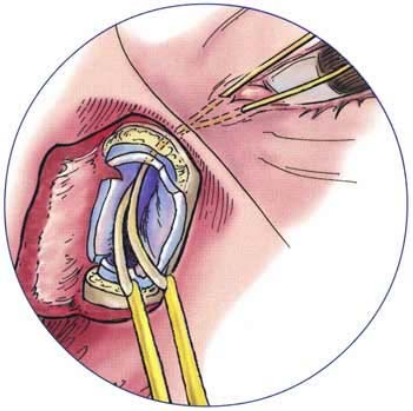
Case Video: 88 YO F

- q 6-8 month recurrence; treated with oral + systemic antibiotics with good response every time

Dacryocystitis_I_D.mp4

Dacryocystorhinostomy

- **External vs Endoscopic**



CONCLUSION

Resources and further reading

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Questions?

THANK YOU!

Uveitis Savvy

Clinical Findings to Improve Accuracy in the Diagnosis of Anterior Uveitis and Comfort in Management

Stephanie Ivor-Smith, OD
Resident, Icon Eye Care

Learning Objectives

- Identify and properly characterize uveitis based on the ocular signs using the Standardization of Uveitis Nomenclature (SUN) Project
- Create a list of differential diagnoses and appropriate tentative diagnosis
- Determine the appropriate testing, including imaging or lab testing and the expected histologic/pathologic findings
- Select and justify the most appropriate treatment/management for the different types of uveitides

Patient Presentation

- 33 yo WF CC constant, decreased vision, redness, light sensitivity, OD x 1 week, 1st time occurrence
- (-)Ohx, -(SHx), (-)FHx, no meds, NKDA
- ROS: (+) cough x 1year - Smoker
  (+) back pain x 20 years – swing accident

Examination findings

- VA sc OD 20/250 PH 20/100
  OS 20/50 PH 20/30
- IOP GAT OD 15mmHg, OS 15mmHg
- SLE – OS unremarkable
  - OD
    - 2+conj. Injection with diffuse ciliary flush
    - Clear corneal epithelium, 3+ stromal edema, 2+ endothelial folds, granulomatous KPs inferior endo.
    - AC 4+ cells and 1+ flare
    - No Iris TIDs or nodules
    - Posterior Synechiae from 4-9 o'clock, with fibrinoid material on the anterior lens
    - Poor views of the vitreous and posterior segment; (+) red reflex and retinal whitening

- Dx: Primary Granulomatous Iridocyclitis OD
- DDx Include:
  - Sarcoidosis
  - TB
  - Syphilis
  - Viral Etiologies
    - Herpetic or CMV

- Treatment
  - Begin topical steroids (Durezol) q1h
    - Atropine 1% BID
  - Ordered lab work up for
    - HLA-B27
    - Syphiilis IgG
    - ACE
    - Chest x-ray

Subsequent Visits

- 3 days later
  - Mild improvement in conj. injection and light sensitivity
  - Decreased VA and Corneal edema remained, along with endo folds and KPs
  - Added Valtrex 1g TID, continue current gtts
- Received lab results: All Negative
- 4 days later
  - VA improved to 20/80
  - IOP 12mmHg OU
  - Corneal edema and endo folds cleared
  - KP remained and AC 4+ cells, 1+ Flare
  - No iris TIDs, PS remained
  - Continue current regimen, RTC 1 week

Follow up visits cont.

- Encounter 4
  - VA 20/70
  - KPs reduced in size and number
  - AC rxn remained 4+ cells, 1+ flare, + synechiae
  - Added Prednisone 60mg qd
    - Tapered steroid QID
    - Atropine BID
    - Valtrex 1 gm TID

- 1 week later
  - VA 20/60
  - IOP 15mmHg
  - Further decrease in KPs
  - AC rxn improved to 2+ cell
  - Valtrex tapered to 1gm qd
    - Tapered Prednisone to 50mg qd x 1 week, 40mg qd x 1 week
    - Cont steroid qid, atropine bid

- 2 weeks later
  - Trace KPs remained
  - No AC rxn
  - Posterior synechiae broken
  - Cont. taper prednisone 30mg, 20mg,10mg by 1 week each
    - d/c atropine
    - Cont. Valtrex qd
    - Taper steroid TID

- Over the next 6 weeks the patient had no flare of symptoms, taper of topical drops and oral prednisone was continued until cessation.
  - She remained on Valtrex qd for 12 weeks and d/c with no problems

Signs and Symptoms of Ocular Inflammation

Patient Symptoms

- Pain
- Redness
- Photophobia
- Epiphora
- Decreased Vision

Clinical Signs

- Ciliary Flush
- Conjunctival Injection
- Scleritis
- Necrosis
- Cornea/Keratitis
  - Decreased sensation
- Anterior Chamber
  - Cells
    - Keratic Precipitates
      - Non-granulomatous
        - Fine/Small
        - Stellate
      - Granulomatous
        - Mutton Fat/Large – inferior endothelium
  - Hypopyon
  - Flare
  - Fibrin

Clinical Signs Continued

- Iris
  - Posterior Synechaie
  - Peripheral Anterior Synechaie
  - Iris Atrophy
  - Heterochromia
  - Nodules
    - Busaca
    - Koeppe
- Possible IOP elevation
- Posterior Chamber
  - Vitreous Cells – Snow balls
    - Snowbank formation – white exudative material over inferior ora serrata and pars plana
  - Choroidal Inflammation
  - Vasculitis – sheathing and exudates around vessels
  - Retinitis

How to classify uveitis?

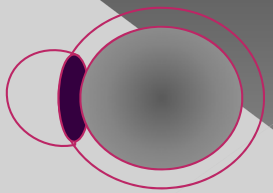
Standardization of Uveitis Nomenclature (SUN) Project

S.U.N. Classification

- Anatomic Location
- Course
- Laterality
- Morphology
- Infectious
- Host or Systemic Disease

Anatomic Location

- Where is the primary site of inflammation?
  - Anterior Segment
  - Intermediate – Vitreous
    - Pars Planitis
  - Posterior Segment
  - Panuveitis



Course

- Acute
  - Less than 3 months
- Recurrent
  - Active inflammation separated by a period of no inflammation
- Chronic
  - Insidious and long-lasting, > 3months

Laterality

- Unilateral
  - Right vs Left eye
  - Both →Alternating in recurrent cases
- Bilateral
  - Simultaneous symptoms in both eyes
  - Asynchronous symptoms

Morphology

- Posterior involvement
  - Retinitis
  - Choroiditis
  - Paucifocal vs Multifocal
    - Morphology of lesions

Infectious

- Toxoplasmosis
- CMV
- Herpetic
- Zoster
- Syphilis
- Lyme
- Bartonella

Host/Systemic Disease

- Child vs Adult
- Immunocompromised or Not

Differential Building

Ddx of Anterior Uveitis

- Infectious
  - CMV
  - HSV
  - HZO
  - **Syphilis**
  - Leprosy
  - **TB**
  - P.Acnes
- Systemic
  - HLA-B27
    - Ankylosing spondylitis
    - Reactive arthritis
    - Crohn disease
    - Ulcerative colitis
    - Psoriatic arthritis
  - JIA/JRA
  - Behcet disease
  - **Sarcoidosis**
  - MS
  - TINU
- No Disease
  - Fuchs Heterochromic Iridocyclitis
  - Trauma
  - Posner-Schlossman Syndrome
  - Lens induced
  - Idiopathic

Ddx of Intermediate Uveitis

- Infectious
  - **Syphilis**
  - Lyme
  - **TB**
- Systemic
  - MS
  - **Sarcoidosis**
  - Lymphoma
  - Inflammatory Bowel Disease

Ddx of Posterior Uveitis

- Infectious
  - Toxoplasmosis
  - Toxocariasis
  - CMV retinitis
  - ARN
  - PORN
  - DUSN
  - **Syphilis**
  - Lyme
  - **TB**
  - Bartonella Neuroretinitis
- Systemic
  - **Sarcoidosis**
- No Disease
  - White Dot Syndromes (6)

Ddx of Panuveitis

- Infectious
  - **Syphilis**
  - Lyme
- Systemic
  - Behcets
  - VKH
  - **Sarcoidosis**
- No Disease
  - Sympathetic ophthalmia

Lab Testing

Tests	Conditions/Comments
Angiotensin-converting enzyme	Sarcoidosis; may be elevated in children without sarcoidosis
Antiphospholipid Ab (lupus anticoagulant and anticardiolipin Ab)	Thrombosis, CNS disease, and spontaneous abortions in patients with systemic lupus erythematosus
ANA	Systemic lupus erythematosus and other rheumatic diseases
Antifungal Ab	Fungal disease
ANCA	Wegener's granulomatosis (cANCA)
Polyarteritis nodosa (pANCA)	
Antitoxoplasma Ab	Toxoplasmosis
Antiviral Ab	Viral infection
Calcium	Sarcoidosis
Chlamydia complement-fixation test	Chlamydia
C-reactive protein	Underlying inflammatory disease (i.e., rheumatic disease)
Cultures	Bacterial, fungal, mycobacterial, and viral diseases
Erythrocyte sedimentation rate	Underlying systemic diseases (i.e, rheumatic disease, malignancy)
Complete blood cell count	Underlying systemic disease
HIV ELISA	HIV
HTLV-1	HTLV-1 infection
HLA typing	(Specific HLA types associated with specific diseases)
Immune complexes	Rarely useful
Liver function tests	Sarcoidosis, hepatitis
Lumbar function for cell count	APMPPE, VKH, Infection, Malignancy
Lumbar puncture for CSF VDRL	Syphilis
Lumbar puncture for culture and Gram stain	Infection
Lumbar puncture for cytology	CNS lymphoma
Lyme serology	Lyme disease (be aware of false-positive results)
Rheumatoid factor (RF)	Rheumatoid arthritis; girls with JRA and uveitis often RF-negative but ANA-positive
Stool for ova and parasites	Parasitic disease
T-cell subsets	(Low CD4+ count predisposes patient for opportunistic infections)
Thyroid function tests	Increased incidence of thyroid disease in patients with uveitis
Urinalysis	(Blood suggests rheumatic disease)
VDRL/FTA-ABS	Syphilis

Nussenblatt RB, Whitcup SM. Uveitis: Fundamentals and Clinical Practice (4). St. Louis, US: Mosby, 2010

Let's look at some examples

Characteristic	Spondyloarthropathy-Associated	Juvenile Idiopathic Arthritis–Associated	Fuchs Uveitis Syndrome	Herpetic	Sarcoidosis
Course	Recurrent acute	Chronic	Chronic	Chronic	Chronic
laterality	Unilateral or unilateral alternating	Bilateral or unilateral	Unilateral	Unilateral	Bilateral or unilateral
Keratic precipitates	Fine	None; fine	Stellate	Fine; "mutton fat"	"Mutton fat;" fine
Anterior chamber	± Hypopyon; ± fibrinoid aqueous				
Iris	Posterior synechiae	Posterior synechiae	No posterior synechiae; heterochromia	Posterior synechiae; sectoral iris atrophy	Posterior synechiae; iris nodules
Other		Onset age ≤16 years		Keratitis	
Laboratory	HLA-B27	ANA		PCR anterior chamber tap for HSV or VZV	Chest radiograph, liver enzymes

ANA = antinuclear antibody; HSV = herpes simplex virus; PCR = polymerase chain reaction; VZV = varicella zoster virus.

Jabs DA, Busingye J. Approach to the Diagnosis of the Uveitides. *American Journal of Ophthalmology*, August 2013, Vol.156(2), pp.228-236.

- Test Everyone For **SYPHILIS** …
  - Regardless of age and what they tell you about sexual history
    - Patients will lie to you … or conveniently forget
  - And maybe Lyme in endemic areas

Types of Testing

- HLA- B27 – 20% of uveitis cases
  - Commonly undiagnosed at time of uveitis presentation
- Lyme disease
  - Antibody screening and Western Blot
- Syphilis
  - Specific tests
    - FTA-ABS
    - MHA-TP
    - Syphilis IgG Antibody
  - Non-specific tests
    - RPR
    - VRDL
- Herpes virus – 70% of the population will test positive!
  - PCR AC tap for HSV or VZV

Types of Testing

- Sarcoidosis – 5-10% of uveitis cases
  - Chest radiograph
  - Serum lysozyme
  - ACE
  - Query about skin lesion
- TB - <0.5% of uveitis cases
  - Chest x-ray
  - PPD
  - Quantiferon Gold
- JRA/JIA
  - ANA
- CMV
  - AC tap
- No Testing (clinical diagnosis)
  - Behcets
  - Fuchs

Treatment Options

- Cycloplegic
- Topical Steroid
- Oral Antivirals
- Oral steroids/NSAIDs
- Injections/Implants
- Immunomodulatory drugs

Management

- Follow up every 1 to 7 days
  - AC reaction should be evaluated
  - IOP measured
    - Pressure meds added if a steroid responder
    - Gonioscopy to check for PAS
- Always evaluate the posterior segment for signs of necrosis and vasculitis
- Once AC is quiet cycloplegic can be discontinued and steroid tapered
- Cataract evaluation
- Follow every 3 months to yearly, or sooner
  - Most patients will know if there is a flare up

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Ocular Mucous Membrane Pemphigoid: A Case Report and Review

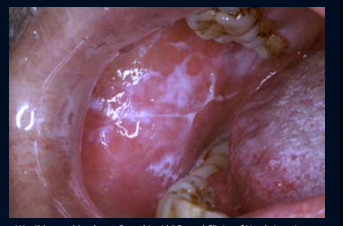
NORTHWEST RESIDENTS CONFERENCE
VA PORTLAND HEALTH CARE CENTER
ALISE GENTRY, O.D.
JUNE 9, 2017

Objectives

- By the end of this presentation, attendees will be able to:
  - Understand the pathophysiology of ocular mucous membrane pemphigoid
  - Identify ocular mucous membrane pemphigoid from differential diagnoses
  - Manage ocular mucous membrane pemphigoid appropriately

Introduction

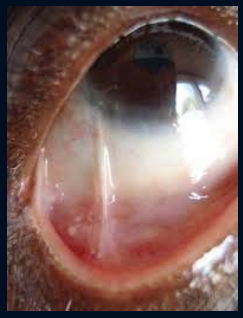
- Mucous Membrane Pemphigoid (MMP) : heterogeneous group of chronic autoimmune sub-epithelial blistering disease affecting mucus membranes
- Variable presentation and severity
  - Ocular MMP
  - Oral MMP
  - Other mucosa and skin diseases



Wu. "Mucous Membrane Pemphigoid." Dental Clinics of North America. (2013).

Epidemiology

- Rare
- 61% of newly diagnosed cicatricial conjunctivitis
- 0.8-2 patients per million per year
- No racial or geographic predilection
- 2:1 Women to men
- Onset 60-80 years old



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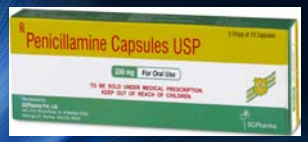
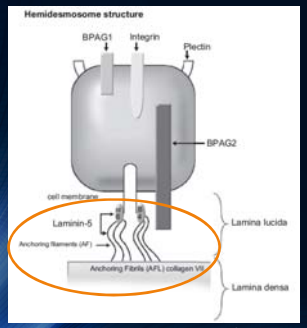
Etiology

- Genetic and environmental factors
- Histocompatibility complex (MHC) and human leukocyte antigen (HLA) association
- Triggered by medication or injury
  - Cause is usually unknown



Pathogenesis



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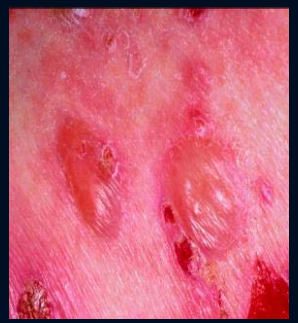
- Epitope spreading: prior inflammation exposes components to autoreactive T-cells
- Autoantibodies against adhesion molecules of epithelial basement membrane hemidesmosomes

Pathogenesis

- Complement process causes subepithelial detachment
  - Induces migration of lymphocytes, eosinophils, neutrophils and mast cells
- Activated fibroblasts leads to scarring



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Presentations and Complications

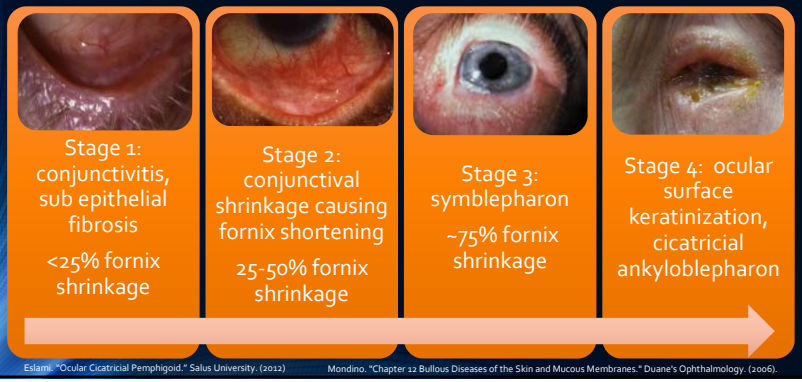
Site	Incidence	Presentation	Complication
Oral	85%	Desquamated gingivitis, ulceration	Pain, inability to eat
Ocular	65%	Conjunctivitis, symblepharon	Blindness
Nose	20-40%	Discharge, erosion, scarring	Difficulty breathing
Skin	25-30%	Erythematous plaque, scarring	Painful bullous eruptions
Anogenital	20%	Erosion, ulceration	Sexual dysfunction, dysuria, pain
Larynx	5-15%	Scarring, airway stenosis	Difficulty breathing, sleep apnea , asphyxiation
Esophagus	5-15%	Stricture formation	Dysphagia , death

Ocular MMP

- Bilateral chronic conjunctival inflammatory process with acute recurrent episodes
- Subconjunctival fibrosis
- Tear deficiency
- Rarely blisters
- Corneal neovascularization and scarring

Mondino. "Chapter 12 Bullous Diseases of the Skin and Mucous Membranes." Duane's Ophthalmology. (2012).

Tauber's Ocular MMP Stages



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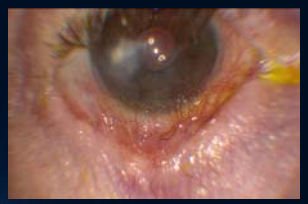
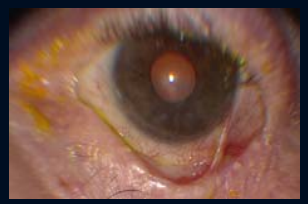
Case #1: 59 yo Caucasian Male

- CC: blur and irritation OU x 9 mo
- POHx: cataract surgery OD, RUL blepharoplasty, electrolysis/ cryotherapy BLL
- PMHx: HTN

	OD	OS
BCVA	20/100	20/30-
Anterior Segment	See photos	
Lens	PCIOL	2+ NS
Dilated exam	Unremarkable, C/D 0.35r	Unremarkable, C/D 0.35r

Case #1 : Presentation





Assessment:

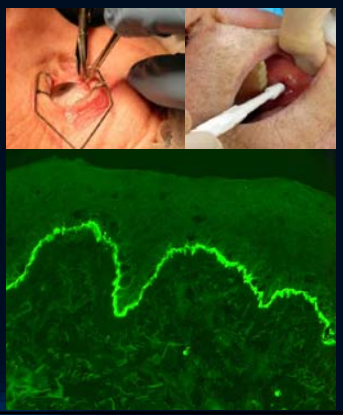
1. Symblepharon BLL OU, questionable ocular MMP
2. Corneal scarring OD>>OS
3. Trichiasis LUL

Cicatrizing Conjunctivitis Differentials

- Stevens-Johnson Syndrome
- Chemical burn
- Trauma
- Ocular Rosacea
- Pemphigus Vulgaris
- Atopic keratoconjunctivitis
- Epidemic keratoconjunctivitis
- Radiation
- Congenital
- Iatrogenic
- Medication
- Paraneoplastic pemphigus

Diagnostic Workup

- Oral mucosal biopsy recommended
- Direct Immunofluorescence (DIF) microscopy
  - Positive finding: linearly deposited IgG, IgA or complement component 3 at epithelial basement membrane



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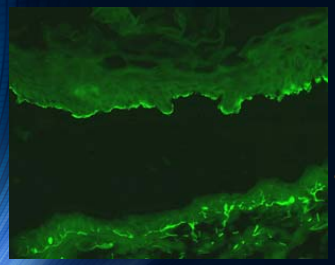
Diagnostic Workup



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- Indirect immunofluorescence (IIF) microscopy: identify presence and characteristics of serum autoantibodies
  - Salt-split divides autoantibodies into those targeting upper or lower lamina lucida antigens
- Enzyme-linked immunosorbent assay (ELISA)
- Immunonegative Ocular MMP

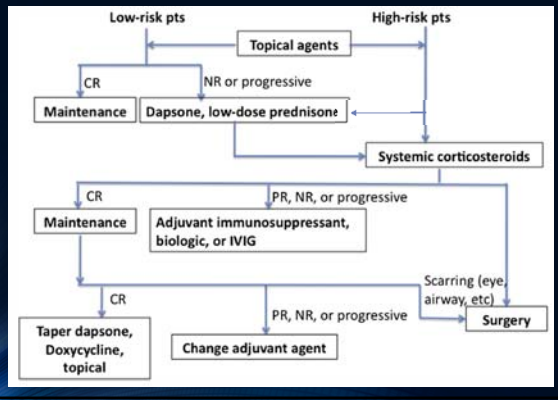
Case #1 : Workup

- Chlamydia trachoma: negative
- Vitamin A deficiency: within norms
- Conjunctival biopsy of inferior temporal fornix: negative for IgG, IgA, IgM and C_3
  - Assessment: Idiopathic subconjunctival fibrosis with symblepharon OU
  - Plan: lubricating ointment qhs OU and preservative free tears 4-6x/d OU

Case #1 : Workup

- Refer to dermatology: no other mucous membrane involvement
- Indirect immunofluorescence: negative
- Assessment: Presumed ocular mucous membrane pemphigoid (stage 3) OU
- Plan: lubrication and Dapsone 100mg qd

Treatment



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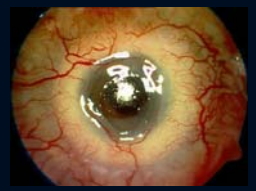
Additional Treatment

- Rituximab: monoclonal antibody that binds to molecule on surface of B-cells
- Intravenous immunoglobulin (IVIg): biologic immune modulatory agent
- Dry eye treatment
- No treatment to reverse scarring

Surgical Treatment

- Temporary symptom relief
  - Entropion surgery
  - Tarsorrhaphy
  - Mucous membrane or amniotic membrane transplantation
  - Boston keratoprosthesis
  - Osteo-Odonto-Keratoprosthesis



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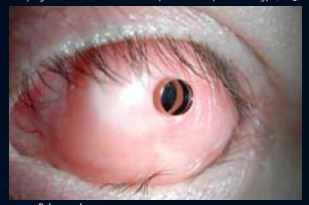

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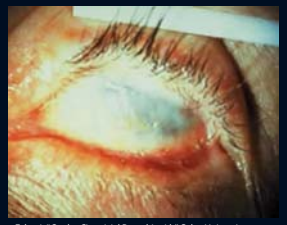
Prognosis

- Oral lesions only: excellent
  - Mild to moderate disease process
- Ocular, nasopharyngeal, esophageal and laryngeal: poor
  - 53% visual loss
  - 35% maintain reading vision
- IgG and IgA: more severe disease



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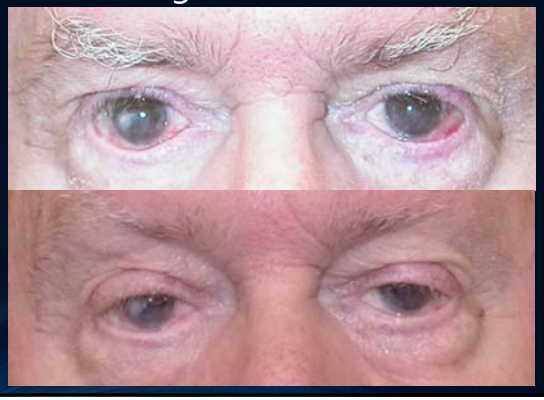
Case #1 : Treatment

- Treatment over next 9 years:
  - Dapsone → mild anemia but continue → ineffective → replace with Mycophenolate Mofetil → discontinue
  - Oral prednisone pulse x 2
  - Doxycycline
  - Azathioprine → discontinue
  - s/p 8 Rituximab infusions with great improvement but some side effects
  - Ocular lubrication

Case #1 : Prognosis

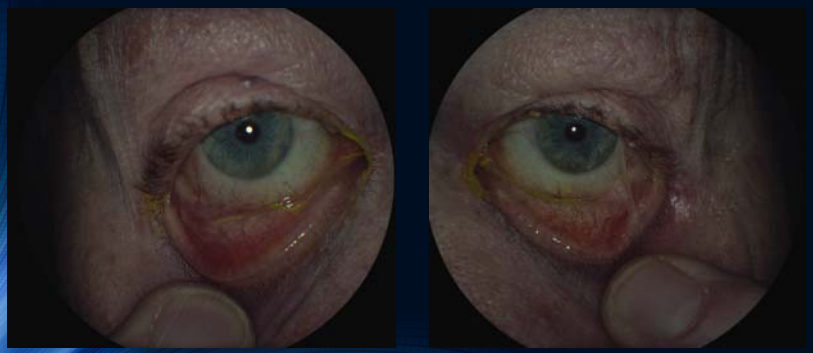


Case #2: 68 yo Caucasian Male

- CC: routine diabetic eye exam, LEE 1 year ago
- POHx: s/p cataract surgery OU
- PMHx: DM, HTN, hyperlipidemia, OSA, thrombocytopenia

	OD	OS
BCVA	20/20-	20/20-
Anterior Segment	See photos	
Lens	PCIOL	PCIOL
Dilated exam	Unremarkable, C/D 0.40r	Unremarkable, C/D 0.35r

Case #2 : Presentation



Conclusion

- Early detection and treatment is critical
- Multidisciplinary care
- Symblepharon ≠ Ocular MMP
  - As always, history is key!
- Long-term monitoring and treatment modification

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Questions?

Beyond the Basics: Diabetic Retinopathy

EMILY BUCHER, OD

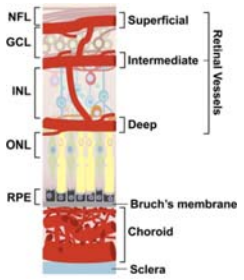
Objectives

- Understand the pathophysiology of retinal blood vessel deterioration in diabetes
- Recognize common presentation patterns/theories of clinical sings of diabetic retinopathy
- Differentiate vascular anomalies using the latest diagnostic techniques
- *Overall acquire a broader understanding of why diabetes presents as such in the eye*

Diabetic Retinopathy: Overview

Chronic hyperglycemia
- Altered retinal vasoregulation
- Increased blood viscosity
- Changes to local mediators/inflammation

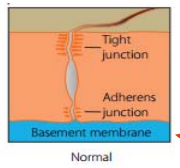
Damages all the major components of the retina
- Vascular Structures
  - *Pericytes*
  - Endothelial cells
  - Basement Membrane
- Glial Cells
- Oxidative Stress
- Leukocytes



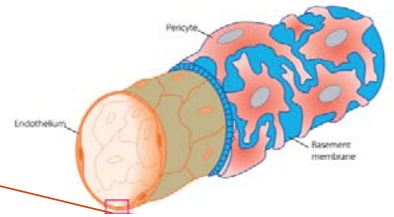
Microcirculation Review

Capillary unit→continuous **endothelium** surrounded by **BM** and **pericytes**
- Direct communication between endothelium and pericytes via gap junctions
- Exchange paracrine signals through shared basement membrane (BM)

Larger venules→ vascular smooth muscle cells



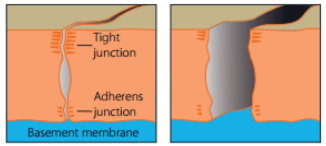
Normal



Role of Pericytes

Regulate blood flow through capillaries, along with smooth muscle on venules
- Contractile properties in the upstream precapillary arterioles
- Sensitive to and activate local mediators

Maintain inner BRB
- Promote tight junction and *non-fenestration* phenotype of vascular endothelium



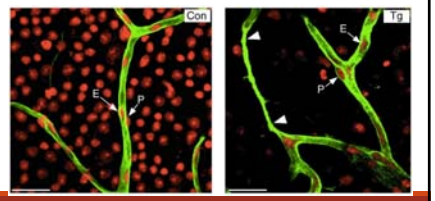
Pericytes and DM

Elevated glucose levels are toxic to pericytes within 96 hours

Pericytes more susceptible to mildly elevated glucose levels than endothelial cells
- Pericytes have almost no replicative capacity
- Endothelial cells have replicative capacity→ will be exhausted in time

Depletion of specific reliant growth factors

What does this mean for retinal vasculature?
- Disrupts autoregulation of downstream capillaries
- Alters vascular response to local mediators
- Alterations to inner BRB

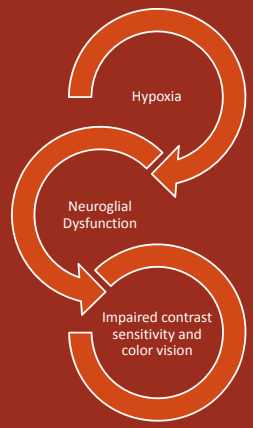


Basement Membrane Thickening

Basement Membrane→ Controls flow of local mediators in vessels

Thickening in diabetes→ predominately in capillaries **proximal to arterial circulation**
- Limits communication between endothelial cells and pericytes/VSMCs→ **autoregulation disruption**

Possible Etiologies for Thickening
- Response to increased flow and blood viscosity
- Sensitivity to concentrations of glucose and oxygen in vascular environments
  - **AGEs accumulation enhancing BM thickening?→ Possible treatment?**



Glial Cells

Excess glutamate is toxic to retinal neurons
- **Persistent inflammatory response**
- **Affects tone of retinal smooth muscle cells.**

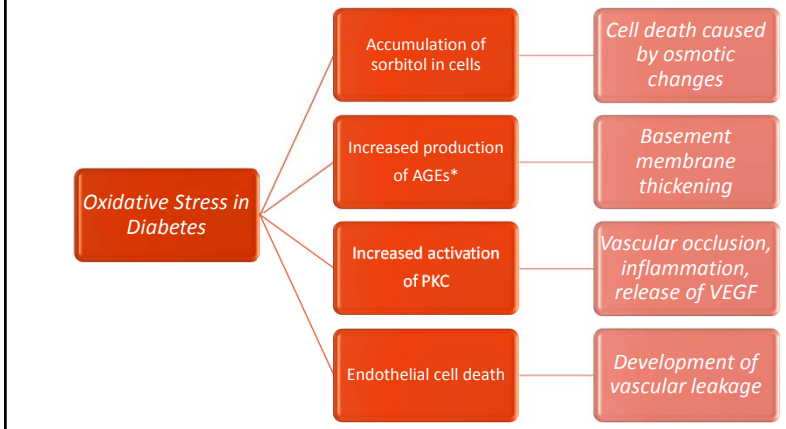
Tissue hypoxia early on→ neuroglial dysfunction

Oxidative Stress

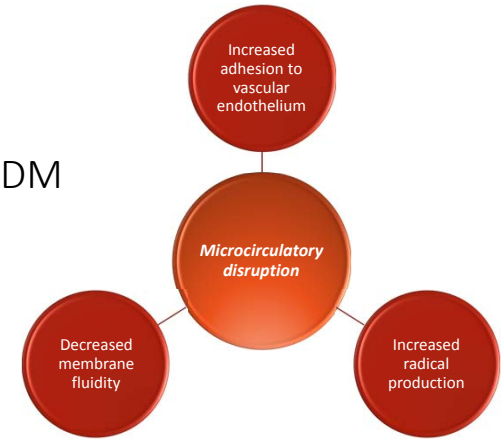
Oxidative stress →imbalance between the formation and destruction of free radicals→ excess free radicals

Oxidative stress toxicity

- Destroys cell membranes
- Disrupts intracellular energy forming molecules
- Damage to protein structures
- Damage to DNA structure



Leukocytes in DM



Leukocytes Role in Retinal Edema?

Increased vascular adhesion

- Disorganization of adherens junctions and tight junctions between endothelial cells
- Increase vascular permeability

Accumulation of leukocytes observed surrounding retinal edema

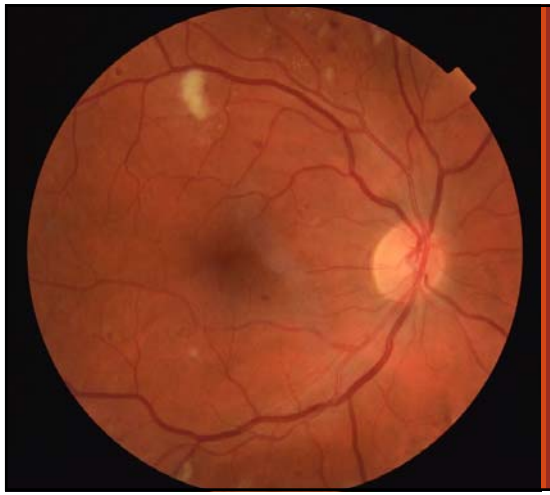
Inhibition of leukostasis→ therapeutic potential?
Pentoxifylline

What are our clinical findings in diabetic retinopathy?

Clinical Findings

Non-proliferative Diabetic Retinopathy
- **Microaneurysms (MA)**
- Dot/blot hemorrhages
- Cotton wool spots
- **Vasodilation**
- Exudates
- **Intraretinal Microvascular Anomalies (IRMA)**
- Macular Edema

Proliferative Diabetic Retinopathy
- **Neovascularization (NV)**
  - **NVE**
  - **NVD**
  - **NVI**
  - **NVA**
- Vitreous Hemorrhages
- Pre-retinal hemorrhages
- Macular Edema



Vasodilation

Vasodilation in DM

Reflects underlying arteriolar autoregulation dysfunction
- Affects from microvascular circulation dysfunction
- Fluctuating glucose levels → loss of venous tone→ chronic venous dilation

What are the consequences?
- Decreased resistance to flow→ more stress on microvascular system
- Increase hydrostatic pressure in capillaries → increase fluid movement into surrounding tissue→ **RETINAL EDEMA**

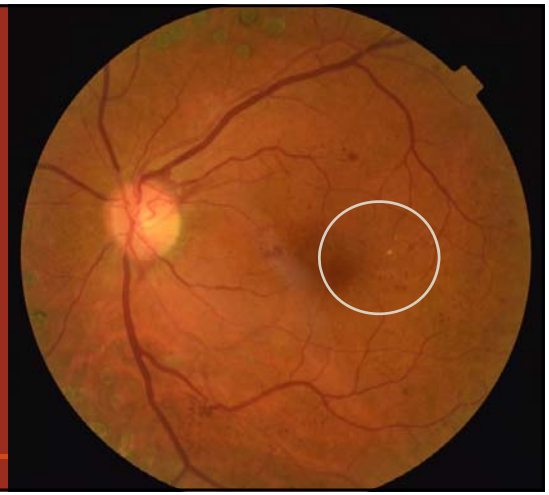
Vasodilation and patient education…

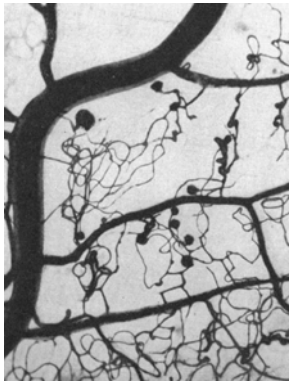
Can use as a clinical indication of progression or stage of chronicity

Consider stage of retinopathy
- Retinopathy with vasodilation→ higher risk for progression retinal/macular edema
  - Specifically with blood sugar fluctuations
- No retinopathy with vasodilation→ pre-diabetic retinopathy or hypertensive

Chronic venous dilation noted→ Possible long-term diabetic

Microaneurysms





Microaneurysms

Small dark red spots ~10-100microns in diameter

Arise only from vessels with normal endothelial lining and intact circulation
- Capillaries, terminal arterioles, or small venules

Selective loss of mural cells from the vessels→ weaken capillary wall
- Specific changes for diabetes

Capillary wall stretching at weak points→ uncontrolled hydrostatic pressure

Cogan D.G. Toussaint D. Kuwabara T. "Retinal Vascular Patterns." *Archives of Ophthalmology*. Sept. 1961;66:160-112.

Location of Microaneurysms

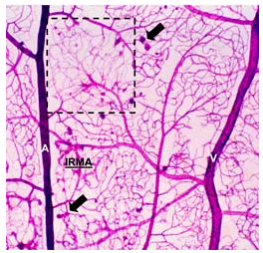
Mostly inner retinal capillary beds

Adjacent to arterial side of capillary bed

More prevalent in temporal than nasal retina
- Greater in superior temporal than inferior nasal quadrant

Often *downstream* from pre-capillary arteriolar smooth muscle cell loss and vasodilation

Often cluster *upstream* of large areas of **capillary nonperfusion**



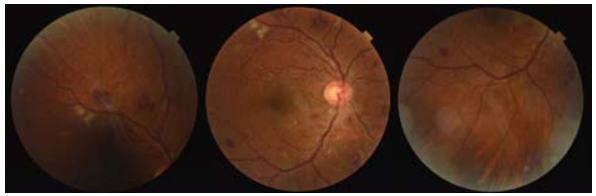
Gardiner T.A. et al. "Arteriolar Involvement in Microvascular Lesions of Diabetic Retinopathy: Implications for Pathogenesis" *Microcirculation*. 2007;14:25-38.

Capillary Closure/Nonperfusion in DM

Compressed by the swelling of the surrounding tissue, facilitated by the fall in capillary pressure resulting from the arteriolar occlusion

Most common mid-peripherally in diabetes

These areas protected from aneurysmal changes→ NO RETINOPATHY!

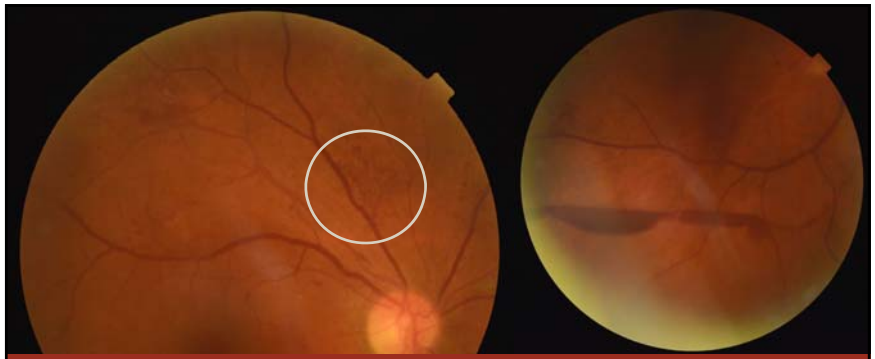


What does this mean for our patients?

Capillary nonperfusion is an irreversible vascular process
- *One of the best indicators of the past history*

More *recent* nonperfusion?
- Adjacent vessels may appear dilated
- Cluster arrangements of MAs bordering area (mid-peripheral?)
- CWS and IRMA→ increasing capillary closure?
- Important to educate patient on impending progression of retinopathy/neovascularization

Long-standing nonperfusion?
- Adjacent vessels have minimal reaction
- More progressed stages of retinopathy with neovascularization



Neovascularization

Neovascularization

Related to the degree of retinal ischemia to the site of the new vessel formation
- Growth triggered by vascular endothelial growth factor (VEGF)

VEGF
- Regulated by oxygen levels in retinal tissue and mediated by a vasoformative function
- Produced normally at low levels→ ischemic influences causes upregulation in pathology
- Produces increased vascular permeability
- Goal is to reduces the hypoxic stimulus for vessel formation

Ischemia and presentation of ocular neovascularization in DM
- NVI>NVD>NVE
- Extent of peripheral nonperfusion parallels severity of retinopathy

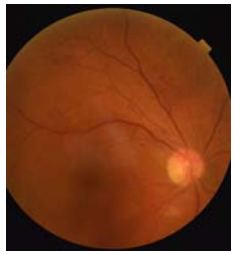
Neovascularization Elsewhere (NVE)

Derives its blood supply from *retinal* vasculature itself

Moderate midperipheral nonperfusion

Many originate along upper and lower vascular arcades
- Commonly along the proximal margin of nonperfused areas

Central fundus to the disc, the macula, and the vascular arcades often spared from neovascularization
- Area temporal to fovea involved to lesser degree



Neovascularization of the Disc (NVD)

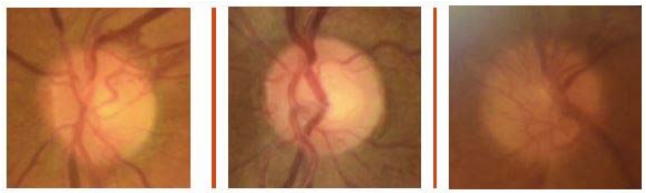
Current thought that NVD indicated a more advanced stage of PDR

Extensive midperipheral nonperfusion

Possibly derives part of its blood supply from *uveal* circulation
- Origin of NVD typically neuroretinal rim
  - Optic disc derives its blood flow from both retinal and uveal (choroidal or posterior ciliary) vessels
- Bruch's membrane is absent in the disc→ no separation between the choroidal and retinal vasculature
  - Under greater exposure to vasoformative substance than elsewhere

Location of NVD

Typically originates from neuroretinal rim/disc margin (<1DD from disc)
- Superior temporal sector most common
  - Temporal sector has fewer blood vessels than nasal
  - Temporal sector more frequently in a watershed zone



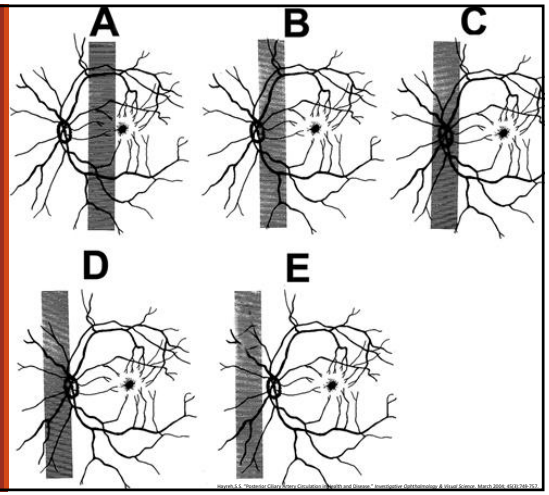
Watershed Zones and NVD?

Watershed zone= the border between the territories of distribution of any two end arteries
- Most vulnerable to ischemia

Present between the distribution of the various PCAs, between SPCAs, and between the anterior and posterior ciliary arterial circulation

PCAs represent the main blood supply to the ONH
- Range between 1-5 PCAs
  - 48% of cases there are only 2 PCAs and the resulting watershed zone is localized at the temporal part of the neuroretinal rim[31]

Watershed Zone Locations

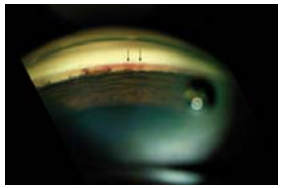


Neovascularization of the Angle (NVA)

Most occur with NVD

- 73.25% of eyes have areas of new vessel formation on the optic disc [17]

Extensive capillary nonperfusion throughout the whole recordable fundus except the central retina



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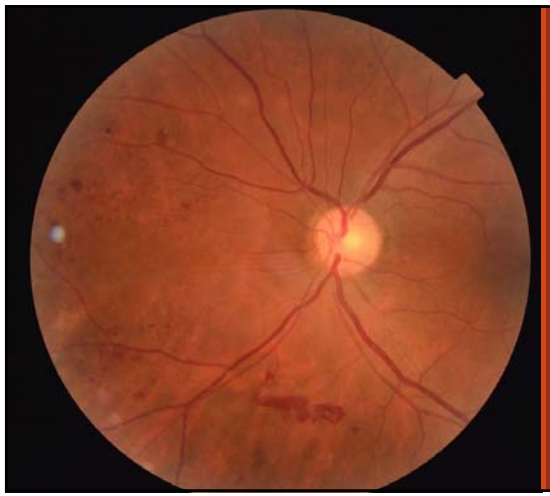


Neovascularization vs Intraretinal Microvascular Anomalies (IRMA)

IRMA	Neovascularization
Mid to late stages of NPDR	Proliferative lesions
Either dilation of pre-existing capillaries or actual growth of new blood vessels within the retina	From venous circulation→Areas of hypoxic retina, effort to compensate→ growing new vessels
Not "leaky"	"Leaky"→ delicate in structure
Deeper in the retina	Pre-retinal, break through ILM
Most common in midperiphery and arcades, not common around disc	Retina>optic disc>iris/angle
More burgundy in color	More red in color
Often cross over themselves	Tend to NOT cross over themselves
Do not cross over major blood vessels	Can cross over major blood vessels
Contorted with sharp corners, "spidery"; blurrier edges	Mature neo resembles "fronds", "cartwheel" or "umbrella"
Commonly adjacent to CWS	Initially lay flat, parallel with retinal surface
	Blossom like a flower bud→ outside of NV is more dilated than inner NV

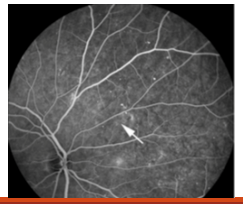
How do we typically differentiate between the two?

Fundus exam

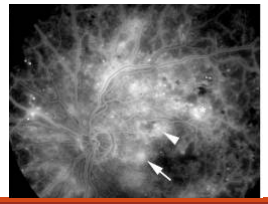
Fundus photos

Fluorescein angiography**

IRMA



Neovascularization



Lee,C. et al. "Reevaluating the Definition ...AM J Ophthalmol. Jan 2015; 159(1): 101–10.e1

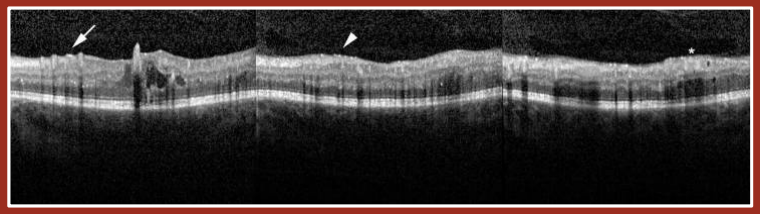
Differentiating IRMA from NV using OCT?

Can use the OCT to help to determine if the lesion is proliferative in nature
- Not diagnostic

Useful in patients who cannot tolerate FA or for quick screening of a suspicious vasculature.

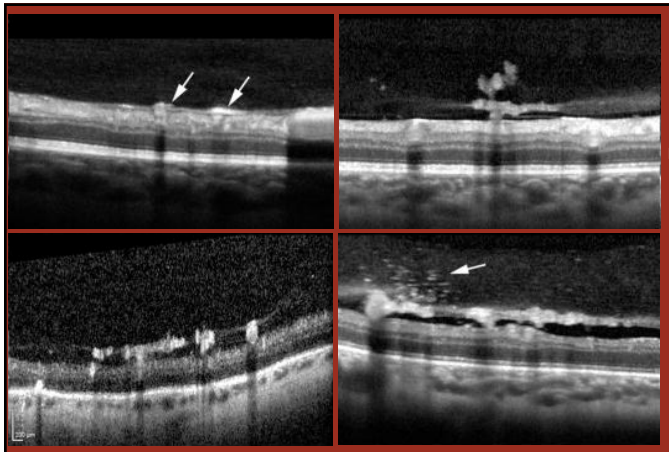
Main difference is whether the vasculature breaches the ILM.
- Growth into posterior hyaloid or pre-retinal surface is only seen in *neovascularization.*

SD-OCT provides no index of the activity of PDR disease



Does not project into vitreous
Outpouchings of internal limiting membrane
Hyper-reflective dots in the superficial inner retina

IRMA



Hyper-reflective complexes break through the ILM

Breach of the posterior hyaloid

Hyperreflective dots in the vitreous

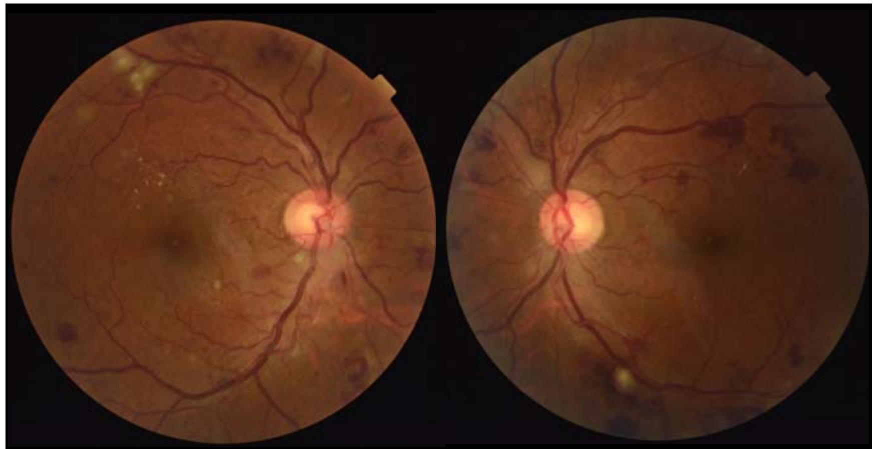
Neovascularization

Progression of Retinopathy

Tends to progress with time despite good glycemic control

- Supports existence of "metabolic memory"→ 2 manifestations of memory in the tissues
  1. **Salutary memory of good glycemic control**
     - Early implementation of good glycemic control may have a long-lasting protective effects on diabetic retinopathy
  2. **Damaging memory of hyperglycemia**
     - Induces changes in gene expression and cellular functions→ can persist in absence of hyperglycemia

**Important patient education tools



Questions?

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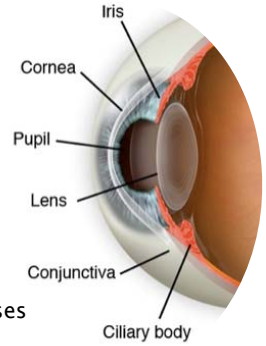
INTRACORNAL INLAYS FOR PRESBYOPIA

Marc Harrie, OD
Chu Vision Institute
Refractive and Ocular Surgery Resident

Accommodation

- Increase in eye refractive power to focus near objects
  - **Ciliary muscle**
    - Contraction of longitudinal fibers
      - Pulls choroid forward
    - Contraction of circular fibers
      - Draws ciliary body closer to lens
      - Releases tension on zonules
  - **Crystalline lens**
    - Thickens (diameter)
    - Anterior surface curvature increases



Presbyopia

- Inevitable loss of accommodation
  - Progressive & irreversible
  - Age-related condition
    - Usually manifests over the age of 40
- Pathophysiology is a topic of debate
  - Loss of lens elasticity (hardening)
  - Loss of capsule/zonular tension
  - Ciliary muscle dysfunction

Presbyopia

- Epidemiology
  - 1.37 billion people worldwide by 2020[1]
  - Age-related condition
    - Will continue to grow with an aging population

[1] *Arch Ophthalmol* 2008

Treatment Options

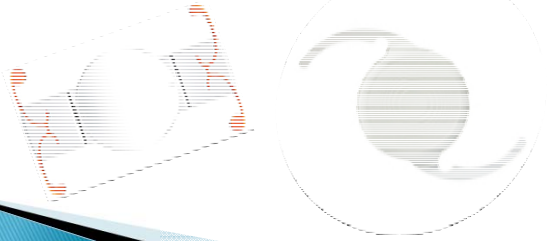
- Non-surgical
  - Glasses
    - Reading glasses
    - Bifocal / progressive lenses
  - Contact lenses
    - Monovision
    - Multifocal

Surgical Treatment Options

- **Laser Vision Correction (LASIK or PRK)**
  - Monovision
  - Multifocal LASIK (presbyLASIK)
    - Not FDA approved
    - Currently in trials

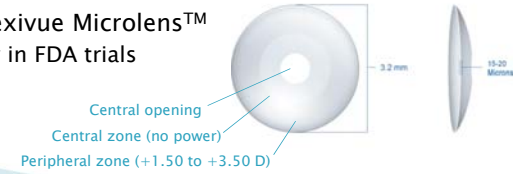
Surgical Treatment Options

- **Refractive Lens Exchange / Cataract Surgery**
  - Monovision
  - Multifocal IOL
  - Accommodating IOL (Crystalens™ & Trulign™)
  - Extended Range of Vision IOL (Symfony®)

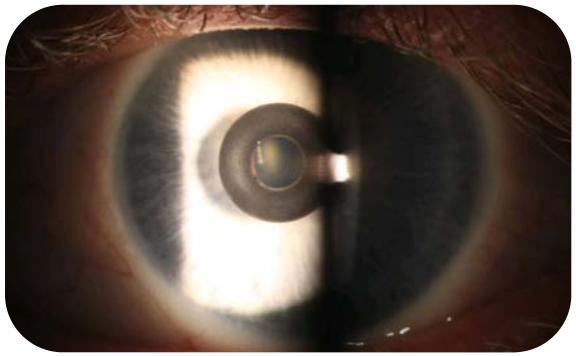


Surgical Treatment Options

- **IntraCorneal Inlays**
  - Two FDA-approved technologies available in U.S.
    - KAMRA™
    - Raindrop® Near Vision Inlay
      - Both implanted into non-dominant eye
      - Only additive technologies currently available
  - Presbia Flexivue Microlens™
    - Currently in FDA trials



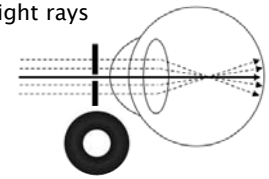
KAMRA™ (AcuFocus, Inc.)



3 month post-op

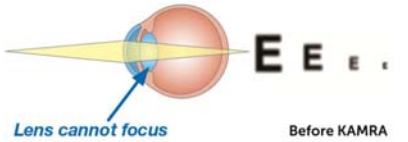
KAMRA™ (AcuFocus, Inc.)

- Optical design
  - Opaque, small-aperture inlay
    - 3.8mm diameter with 1.6mm central aperture
    - 6 microns thick
    - 8400 microperforations
    - Made of polyvinylidene fluoride
  - **Pinhole Effect**
    - Blocks unfocused peripheral light rays
    - Narrows blur circle
    - Extends depth-of-focus



KAMRA™ (AcuFocus, Inc.)

- Pinhole Effect



KAMRA™ (AcuFocus, Inc.)

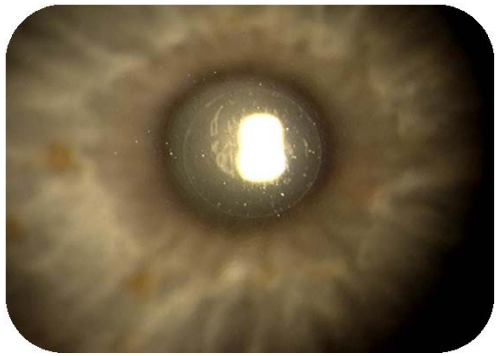
- Surgical technique
  - Implanted into corneal pocket
    - Non-dominant eye
    - Pocket created by femtosecond laser
- FDA approval
  - April 17, 2015 for 45-60 year old patients
    - Unable to focus on near
    - Do not need corrective lenses for distance

KAMRA™ (AcuFocus, Inc.)

- Clinical results
  - FDA Trial[2]
    - 478 subjects
      - 83.5% (400) achieved UNVA 20/40 or better
      - 12 month clinical trial
  - Seyeddain et al.[3]
    - 32 subjects
      - 97% (31) achieved UNVA 20/32 or better
      - 3 year trial

[2] *Data on File at AcuFocus, Inc.*
[3] *J Cataract Refract Surg* 2012

Raindrop® (ReVision Optics, Inc.)



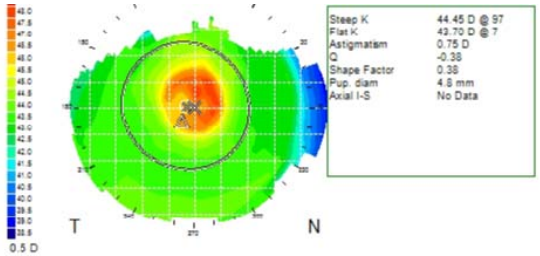
1 month post-op

Raindrop® (ReVision Optics, Inc.)

- Optical design
  - Clear, small-diameter inlay
    - 2.0 mm diameter with a thickness of 32 microns
    - No refractive power
    - Made of hydrogel
  - **Cornea Shape-Changing Effect**
    - Increases central steepening of anterior cornea
    - Produces variable power cornea
    - Variable power central to periphery

Raindrop® (ReVision Optics, Inc.)

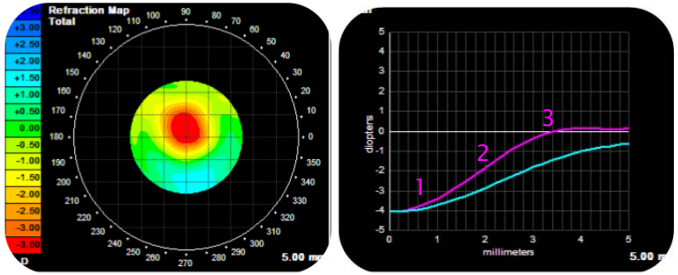
- Topography



Axial curvature 6 months postoperatively

Raindrop® (ReVision Optics, Inc.)

- Wavefront



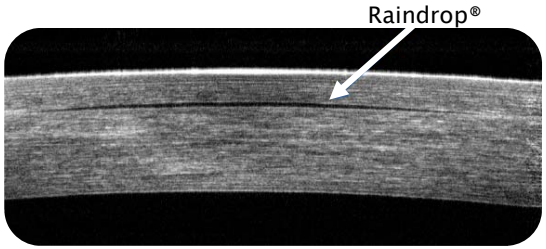
Tracey Wavefront 6 months postoperatively (right) with average radial refraction graph (left)

Raindrop® (ReVision Optics, Inc.)

- Surgical technique
  - Implanted under a corneal flap
    - Non-dominant eye
    - Flap created by femtosecond laser
- FDA approval
  - June 29, 2016 for 41-65 year old patients
    - Unable to focus on near
    - Do not need corrective lenses for distance

Raindrop® (ReVision Optics, Inc.)

- Optical coherence tomography



HD Anterior Segment OCT

Raindrop® (ReVision Optics, Inc.)

- Clinical results
  - FDA trial[4]
    - 364 subjects
      - 92.3 % (336) achieved UNVA 20/40 or better
      - 2 year clinical trial
  - Whitman et al.[5]
    - 30 subjects
      - 97% (29) achieved UNVA 20/32 or better
      - 12 month trial

[4] *Ophthalmol* 2016
[5] *J Cataract Refract Surg* 2016

Patient Selection

- Refractive error
  - **KAMRA™** MRSE: plano to -0.75D
    - Widely accepted ideal refraction: -0.75D
    - Pinhole effect at distance & near
  - **Raindrop®** MRSE: plano to +0.75D
- Off-label use
  - Both technologies can be performed with LASIK/PRK (simultaneously or consecutively)
  - Previous LASIK flap
    - KAMRA™ may be preferred inlay due to depth

Patient Selection

- Contraindications
  - Similar to LASIK
    - Corneal diseases (keratoconus, Fuch's dystrophy…)
    - Previous HSK
    - Thin pachymetry
  - Cataracts
  - Ocular surface disease (dry eye syndrome)
    - Relative contraindication
    - Ocular surface should be managed prior to inlay

Patient Selection

- Patient counseling
  - **CRUCIAL** to patient success
  - Setting expectations
    - Lifestyle/Hobbies (needlework, fine print, etc.)
    - Benefits/Limitations
      - Less dependence on "cheaters"
      - Improvement in functional vision
      - Not designed to bring accommodation back
      - Magnification
  - Patients with unreasonable visual demands or expectations should be avoided

Patient Selection

- Clinical simulation
  - KAMRA™
    - Pinhole over non-dominant eye
      - Increase in UNVA
      - Slight compromise in monocular distance vision

Patient Selection

- Clinical simulation
  - Raindrop®
    - Fit in a multifocal contact lens (non-dominant eye)
      - Usually PureVision2 High Add
      - Increase in UNVA
      - Slight compromise in monocular distance vision

Postoperative Care

- Medications
  - Similar to LASIK
    - Prophylactic antibiotic x 2 weeks
    - Anti-inflammatory (steroid) taper x months
    - Ocular surface management
      - Artificial tears, Xiidra, Restasis
      - Punctal plugs
- Optometry's role
  - Postoperative follow-ups
    - Patient compliance is very important
    - 1 day, 1 week, 1 month, 3 months, 6 months…
  - Co-management opportunity

Postoperative Care

- Healing time
  - Varies
    - Visual rollercoaster (months)
      - Physical healing
      - Neuroadaptation
    - Raindrop® tends to regain vision slightly quicker
      - Less sensitive to dryness?
- Side effects
  - Dryness is **most common** complaint
  - Blurred vision
  - Glare & halos

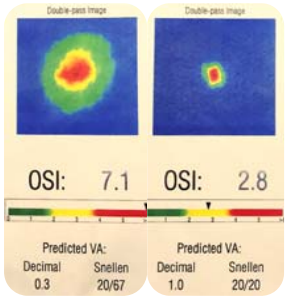
Postoperative Care

- Complications
  - Dryness
  - Flap/pocket complications
    - Dislocated or decentered inlay
      - Both most sensitive to superior decentration
    - Corneal abrasion
    - Diffuse lamellar keratitis (DLK)
  - Haze
    - Keratocyte activation; can lead to **corneal melt**
    - Important to recognize & manage appropriately
      - Should be treated aggressively with steroids

Dryness

−0.50+0.25x146
J1 UNVA

Not noticing any change in vision.



−0.50 DS
J1 UNVA

"Vision is great."
Patient happy.

AcuTarget OSI 1 week & 1 month Raindrop® postoperatively

Corneal Haze

- KAMRA™
  - Typically complain of reduction in near acuity
    Hyperopic shift; should be refracted using R/G
  - Fine dusting of white opacities at inlay interface
    Can be subtle or undetectable via slit lamp
  - Placido disc topography
    "Red ring" sign over inlay

Corneal Haze

- KAMRA™

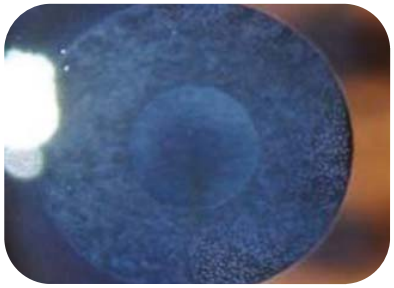


Image courtesy of AcuFocus, Inc.

Corneal Haze

- KAMRA™

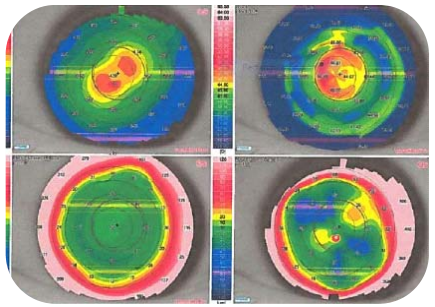


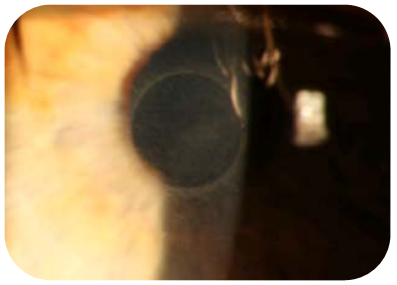
Image courtesy of AcuFocus, Inc.

Corneal Haze

- Raindrop®
  - Typically report closer near point
    - Myopic shift; diminished distance vision
  - Fine dusting of white opacities at inlay interface
    - More visible than KAMRA™
    - Can still be subtle or undetectable via slit lamp
      - Especially in early stages
  - Pentacam HR densitometry
    - Detects early haze development
    - Quantifies haze objectively

Corneal Haze

- Raindrop®



Grade 2+ haze

Case Study

- Chief Complaint
  - 50-year-old white male
    - Reports for Raindrop® post-op OD
      - 12 months s/p inlay implant
      - "Struggling with computer distance"
        - Also has to "hold things closer to read"
    - Eye is comfortable
- Ocular medications
  - Restasis BID
  - PF ATs (Oasis) BID to prn

Case Study

- Distance visual acuity (SC)
  - OD: 20/50-2 PH 20/20-2
  - OS: 20/20
- Near visual acuity (SC)
  - OD: J1+
  - OS: J6
- Refraction
  - OD: -1.00 DS 20/20-2
  - OS: Plano DS 20/20

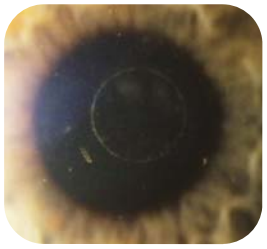
Case Study

- Anterior segment

OD		OS
Normal	**Adnexa**	Normal
W&Q	**Conj/Sclera**	W&Q
S/P Raindrop® Grade 1 haze	**Cornea**	Clear
Deep and Quiet	**A/C**	Deep and Quiet
Clear	**Lens**	Clear
Flat	**Iris**	Flat

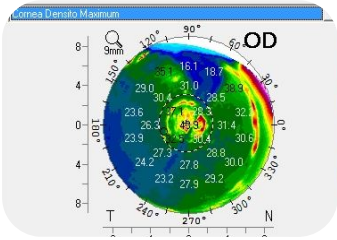
Case Study

- Anterior segment



Grade 1+ haze

- Pentacam HR

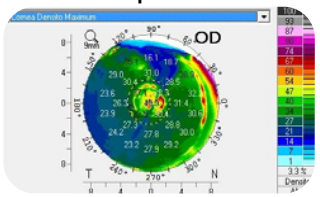


Case Study

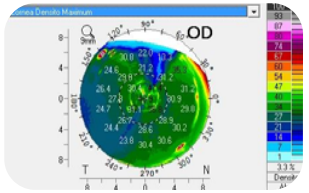
- Plan
  - Start Durezol taper OD
    - QID x 1 week
    - TID x 1 week
    - BID x 1 week
    - QD x 1 week
      - Lotemax taper following durezol
        - BID x 1 month
        - QD x 1 month
  - RTC 1 month for recheck or sooner prn

Case Study

- Follow-up visit



UDVA: 20/50-2
UNVA: J1+
Refraction: -1.00 DS



UDVA: 20/20-2
UNVA: J2+
Refraction: -0.25 DS
"Vision has improved."

Conclusions

- IntraCorneal inlays are **ADDITIVE** technology
  - Removability is attractive feature
    - Usually within ±0.75 D of preoperative refraction
    - Limited to no effect on BCVA
  - Explantation rate is low
    - Most common reasons:
      - Persistent corneal haze
      - Patient dissatisfaction
- Paradigm shift in refractive surgery
  - Success is most dependent on:
    - Patient selection
    - Proper postoperative management
      - Do not let your patients fall off the grid

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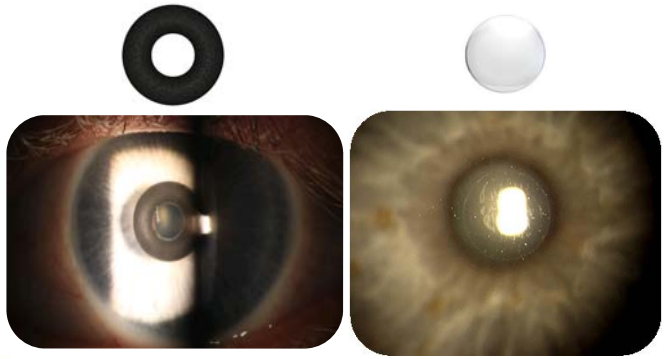
Questions?



Diplopia Management in Patients with Parkinson's Disease

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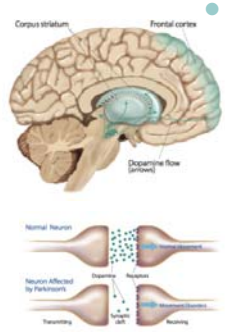
Course Description

- Parkinson's Disease Overview
- Ocular Effects of Parkinson's Disease
- Diplopia Management and Treatment Options
- Case Review

Parkinson's Disease Overview

- Prevalence
  - Every 1-2 people out of 1,000
  - 18 out of 1,000 over the age of 65
- Demographics
  - Mostly affects older people (males more than females), and typically begins after age 50

Parkinson's Disease Overview



- Neurology
  - Progressive, neurological disease
    - Pathology (see addendum for more detail)
  - Damages nerve cells that produce dopamine
  - Classified with other movement disorder diseases including:
    - Corticobasal degeneration
    - Multiple system atrophy
    - *Progressive supranuclear palsy
    - Lewy body dementia

Parkinson's Disease Overview

Signs

- Fine motor skills deteriorate and handwriting changes
- Loss of rhythm
- Lack of natural arm swing while walking
- Fixed facial expression
- Constipation
- Difficulty sleeping
- Mood swings

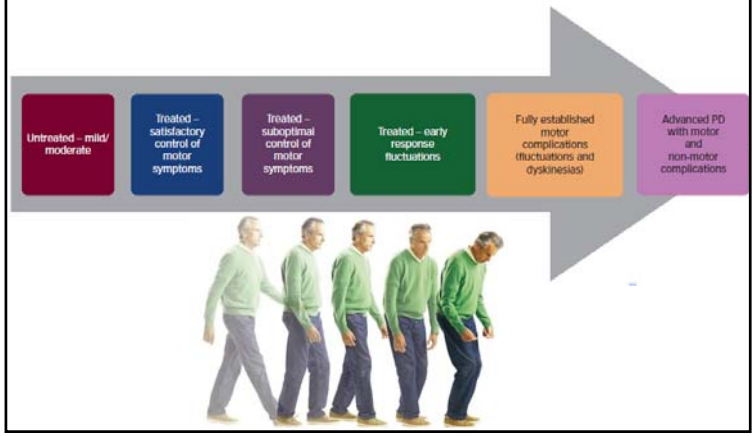
Symptoms

- *Bradykinesia/Akinesia: slowed movement*
- *Rigidity*
- *Tremor at rest*
- Speech difficulty
- Circulation problems
- Difficulty concentrating
- Depression
- Difficulty with mobility, increased risk of falls

Parkinson's Disease Overview

- Causes
  - Genetic vs. Environmental
- Diagnosis
- Treatment
  - Medications
  - Deep Brain Stimulation
  - Occupational Therapy/Physical Therapy

Parkinson's Disease Overview



Ocular Effects of Parkinson's Disease

Common

- Dry Eye
- Reduced Blink Rate
- Decreased Visual Acuity, Color Vision, and Contrast Sensitivity
- Diplopia
- Eye movement abnormalities
- Visual Hallucinations

Less Common

- Blepharospasm
- Apraxia of eyelid opening
- Pupillary abnormalities

Ocular Effects of Parkinson's Disease

- Visual testing can be useful in separating Parkinson's Disease from other neurological movement disorders

	Parkinson's Disease	Progressive Supra Nuclear Palsy
Saccades	Upward effected first	Downward effected first
Square Wave Jerks	On average 5x per minute	On average more than 30x per minute

Ocular Effects of Parkinson's Disease

Parkinson's Disease Medications and the Visual Side effects

Drug Class	Examples	Side Effects
Anticholinergic	Trihexyphenidyl, Benztropine	Large pupil, photophobia, poor focusing, dry eye, blurred vision, glaucoma risk
Dopamine Agonists	Bromocriptine, Pramipexole Ropinirole	May exacerbate visual hallucinations
Levodopa/Carbidopa	Sinemet, Stalevo, Parcopa, Rytary, Duopa	Pupil effects, twitchy/droopy lid, poor tracking
Monoamine Oxidase Inhibitors	Selegiline, Safinamide, Rasagiline	Blurred vision
Antiviral	Amantadine	Large pupil, poor focusing, dry eye, poor tracking

Diplopia & Parkinson's Disease

Diplopia & Parkinson's Disease

- Among the neurological conditions that can cause diplopia, Parkinson's disease is one of the most common
- Significant reduction in vision-related quality of life, especially with near activities not associated with visual acuity

Diplopia & Parkinson's Disease

- Most commonly due to Convergence Insufficiency
  - Increased exophoria at near with reduced BO vergence ranges and reduced NPC
  - Parkinson's Disease & Diplopia Retrospective Study By Frederick E. Lepore, M.D (2006)
  - Diplopia in Parkinson's Disease: Prevalence and associations with other motor and nonmotor symptoms by A. Sauebier (2013)

Diplopia & Parkinson's Disease

- Treatment options
  - Distance and Near spectacles
  - Vision Therapy
  - Prism
  - Monovision Spectacles
  - Central Stipple Clear Contact Paper Occlusion

Diplopia & Parkinson's Disease

Treatment	Pro	Con
Vision Therapy	- Good for patients in early disease stages - Helps maintain control without several spectacle options	- Temporary success as disease progresses - Extensive Time/Effort
Prism	- Successful when small amounts are required (less than 20 PD)	- Limitations for patients with large exo postures (greater than 20 PD) : - Poor cosmesis - Heavy - Poor Optics
Monovision Spectacles	- Successful for large deviations in which prism is not a great option	- Adaptation time - Limited if one eye has poor acuity - Limited if patient had cognitive difficulties
Central Stipple Occlusion	- Successful for patients with large deviations in which monovision is not a great option - Better cosmetically than black pirate patch - Maintains peripheral vision	- Poor adaptation in some patients

Other Ocular Motor Effects from Parkinson's Disease

- Fixation Abnormalities
- Saccadic Abnormalities
- Pursuit Abnormalities
- Eye Teaming Abnormalities

Case Examples

- Case #1: “Mr. Fast Cars and Freedom”
- 76 year old white male
- Chief Compliant:
  - Binocular diplopia at all distances
    - Most bothersome at near, control varies on the day
  - Difficulty tinkering with cars

Case Examples

- Medical History:
  - Parkinson’s Disease diagnosed in 2014
  - Cardiovascular disease
  - Depression
- Ocular History:
  - Dry Eye
  - Diplopia
    - Began in 2013, was diagnosed with PD in 2014
    - Wears spectacle Rx with prism with variable success

Case Examples

- Medications:
  - Amlodipine
  - Carbidopa
  - Carboxymethylcellulose Na 1% ophthalmic gel
  - Fluoxetine
  - Gabapentin
- NKDA

Case Examples

- Distance Visual Acuities:
  - OD: 20/25 $^{-2}$
  - OS: 20/30 $^{-2}$
- CVF: FTFC OD, OS
- Pupils: (-)APD OD, OS
- EOMS: -1 restriction in upgaze
- Pursuits: slightly unsteady; occasional catch up saccade
- Saccades: adequate latency, (+)Overshoots

Case Examples

- Habitual Glasses:
  - OD: +2.25 -4.00 x 095 BI: 5.5$^{\Delta}$BD: 4.25$^{\Delta}$
    OS: +2.75 -2.50 x 086 BI: 5.5$^{\Delta}$ BU: 4.25$^{\Delta}$
    ADD: +2.50
- Manifest Refraction:
  - OD: +2.50 -4.75 x 095
    OS: +2.50 -3.25 x 086
    ADD: +2.50

Case Examples

- Through Habitual Rx:
  - Near CT: 25$^{\Delta}$ alternating XT; OD fixation preference
  - Distance CT: 9$^{\Delta}$ intermittent alternating XT; POTS: 20%
  - NPC: 50 cm with effort

Case Examples

- Assessment:
  - Alternating exotropia secondary to Parkinson's Disease
- Plan:
  - Released Monovision Rx :
    - OD: +5.00 -4.75 x 095 (near eye)
      OS: +2.50 -3.25 x 086 (distance eye)

Case Examples

- Case #2: "Mr. Happy Plate"
- 70 year old white male
- Chief Compliant:
  - Binocular diplopia at intermediate distances and near
    - Bothersome while watching TV, no longer reads
  - Difficulty seeing plate while eating and using utensils
  - Increase in visual hallucinations

Case Examples

- Medical History:
  - Parkinson's Disease diagnosed in 2009
  - Wife reports significant progression over the past 6 months, patient not responding well to PD medications
  - Cardiovascular disease
  - Dementia
  - Depression
- Ocular History:
  - Dry Eye
  - Diplopia

Case Examples

- Medications:
  - Bupropion
  - Carbamide peroxide
  - Carbidopa
  - Carboxymethylcellulose Na 1% ophthalmic gel
  - Clindamycin
  - Clobetasol propionate
  - Hydrocortisone cream
  - Hydrophilic topical ointment
  - AREDS multivitamin

Case Examples

- Drug Allergies:
  - Sulfa Drugs
  - Atropine
  - Cephalexin
- Oriented to time and place but would often contradict himself due to his dementia

Case Examples

- Habitual Glasses:
  - Distance Glasses:
    - OD: +2.00 -1.50 x 007
      OS: +0.50 sphere
  - Intermediate Glasses:
    - OD: +3.50 -1.00 x 002
      OS: +2.50 -0.25 x 065
  - Near Glasses:
    - OD: +4.50 -1.50 x 180 PRISM: 8^{Δ} BO
    - OS: +3.50 -0.25 x 180 PRISM: 8^{Δ} BO

Case Examples

- Distance Visual Acuities:
  - OD: 20/40 $^{+2}$
  - OS: 20/25 $^{-2}$
- CVF: FTFC OD, OS
- Pupils: (-)APD OD, OS
- Fixation: Poor
- EOMS: -1 restriction in upgaze
- Pursuits: not smooth, series of saccades, slow and jerky, difficulty in upgaze
- Saccades: inaccurate, latent, upgaze more difficult than downgaze

Case Examples

- Distance CT: 3$^{\Delta}$ alternating XT
- Near CT: 20$^{\Delta}$ alternating XT
- Previously Evaluated at Annual Exam:
  - Anterior Segment:
    - Slowed blink response
    - Cornea: 2+ diffuse punctate epithelial errosions OD, OS
  - Posterior Segment:
    - Macula: Flat & Intact, pigmentary changes in both eyes

Case Examples

- Assessment:
  - Diplopia secondary to Convergence Insufficiency from Parkinson's Disease
- Plan:
  - Central stipple occlusion over OD
  - Discontinue use of near glasses
    - Did not release near spectacle Rx as patient denied near daily tasks and mostly relies on intermediate Rx
  - Advised patient to use breakfast tray on table to bring plate to higher level so he didn't have to look down as far

Thank you!

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Asymptomatic VF Loss: The Case of a Failed DMV Test

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AFFILIATIONS/ENDORSEMENTS

- None

LEARNING OBJECTIVES

Recognize the ophthalmic manifestations related to pituitary adenomas.

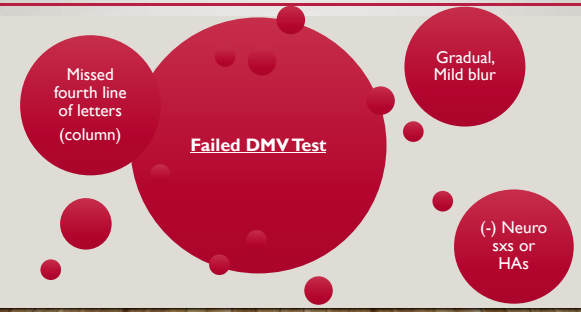
Develop a proficient clinical evaluation to aid in prompt diagnoses and treatment of Neuro-ophthalmic conditions.

Properly interpret and refer different neuro-ophthalmic conditions based on VF defects.

CASE HX

Age/Sex/Race	• 47 year old • Caucasian • Female	Medications	• Pro Air • Singulair • Qvar
Exam	• Comprehensive • SCL fitting	Ocular Meds	• None
Medical Hx	• Psoriasis • Asthma	FMHx	• Diabetes • HTN • Arthritis • Thyroid
Ocular Hx	• None	FOHx	• "Lazy eye"

CHIEF COMPLAINT



PERTINENT FINDINGS

Entrance Testing	Hab Rx	Manifest Rx

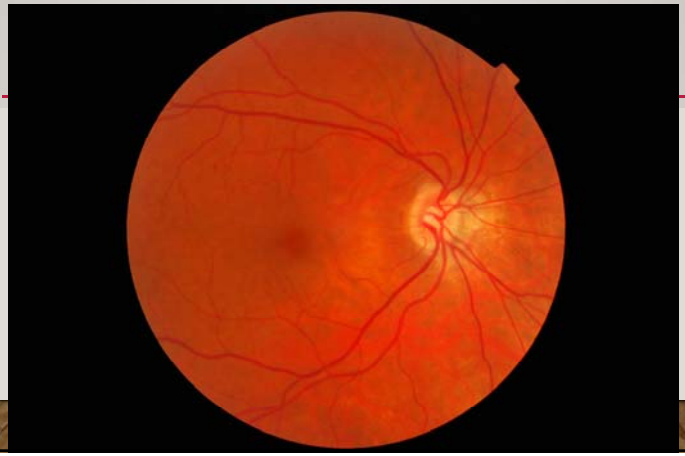
• Pupils: PERRL (-)APD
• CT: Phoric
• FDT: Mild temp misses (OD>OS)
• EOMs: full
	• OD: -0.75-0.75x071
	• OS: Plano-1.00x086
	• VA:
	  • OD: 20/30
	  • OS: 20/30
	  • OU: 20/25
		• OD: -0.75-1.00x072
		• OS: +0.50-1.25x080
		• VA:
		  • OD: 20/25-
		  • OS: 20/20

PERTINENT FINDINGS CONT…

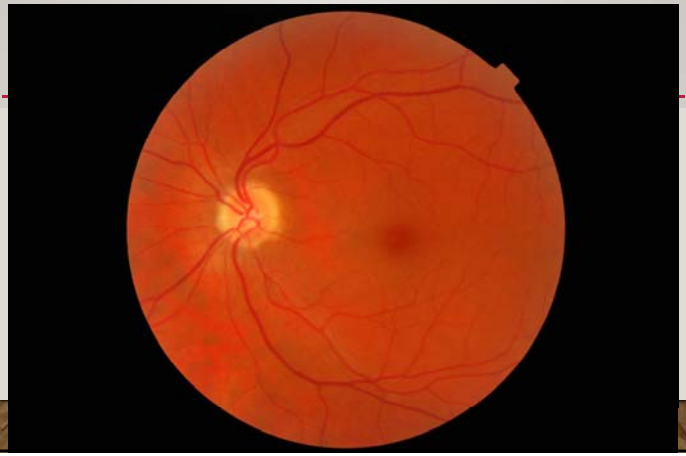
SLE	IOP	Fundus

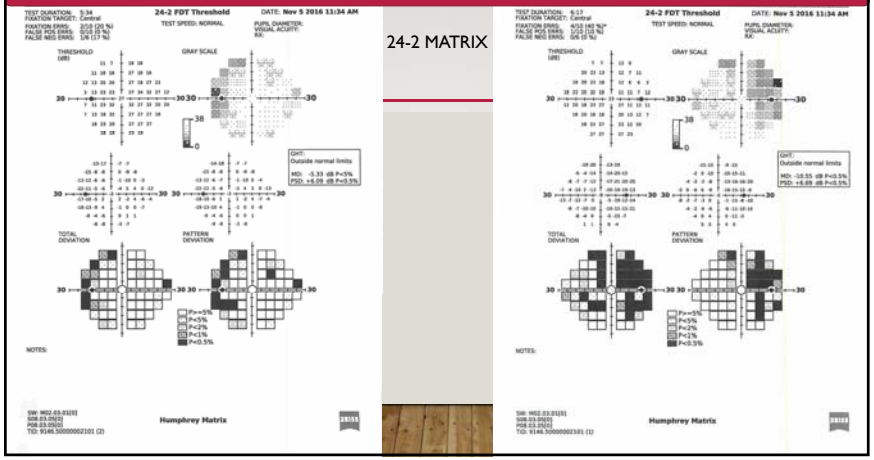
• WNL OU	• OD: 17
	• OS: 17
		• ONH: tilted, situs inversus, malinserted (OD>OS)
		• C/D:
		  • OD: 0.35r
		  • OS: 0.20r
		• Mottling OD

OD



OS





ASSESSMENT AND PLAN

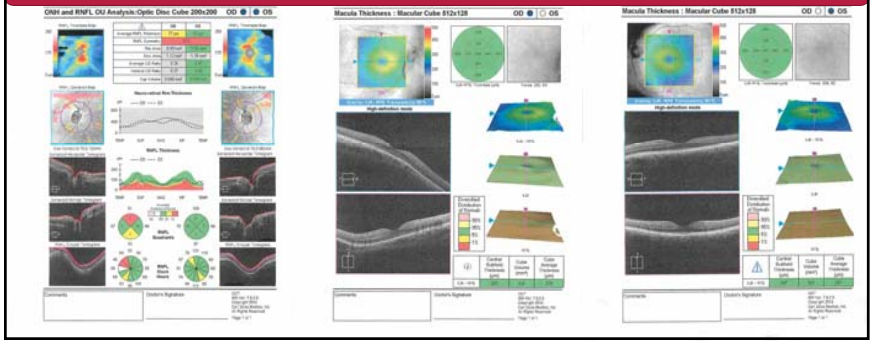
Bitemporal Hemianopsia

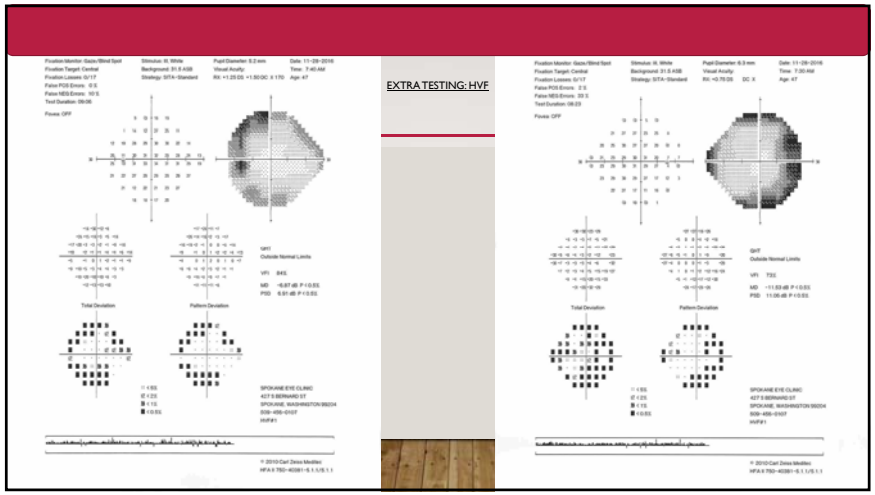
- Refer for OCT/HVF 30-2
- MRI pending results
- Pt ed on neuro s/sxs
  - not currently experiencing any

Presbyopia

- Pt ed on resolution of dec BCVA and VF defect prior to releasing new Rx.

EXTRA TESTING/IMAGING (SEC REFERRAL)



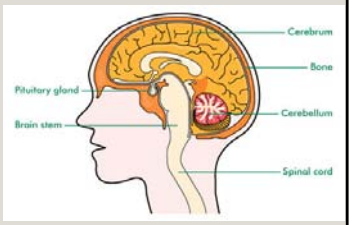


FOLLOW-UP CARE W/ OMD

- Dx w/ pituitary adenoma based off MRI
- Lesion too small for surgery per patient
- Patient to RTC Q3 mos to monitor for progression

BACKGROUND

- Pituitary Gland
  - Located inside anterior portion of the brain w/in the sella turcica, 10mm below optic chiasm
  - Responsible for secretion of many different hormones



PITUITARY TUMORS

- Incidence rate: 8/10,000 persons
- 10:1 female gender majority
- Results from single cell mutation followed by unregulated cell growth

PITUITARY GLAND HORMONES

Anterior Lobe
- HGH
- Follicle-stimulating hormone
- Luteinizing hormone
- Prolactin
- TSH
- Adrenocorticotrophic hormone beta-endorphin

Intermediate Lobe
- Melanocyte-stimulating hormone

Posterior Lobe
- Antidiuretic hormone
- Oxytocin

CLASSIFICATION

TYPE OF HORMONE SECRETED
Most common: Prolactinoma
2nd most common: GH-secreting

PRIMARY CELL ORIGIN
Prolactinoma= Prolactin-secreting adenoma originating from lactotroph cell

CLASSIFICATION

SIZE OF THE TUMOR
>10mm=Macroadenoma
<10mm=Microadenoma

PITUITARY APOPLEXY
Life-threatening hemorrhage or infarct

DDX

- Basilar Artery Thrombosis
- Brainstem Gliomas
- Cavernous Sinus Syndromes
- Cerebral Venous Thrombosis
- Dizziness, Vertigo, and Imbalance
- Glioblastoma Multiforme
- Intracranial Hemorrhage
- Low-Grade Astrocytoma
- Meningioma
- Primary CNS Lymphoma

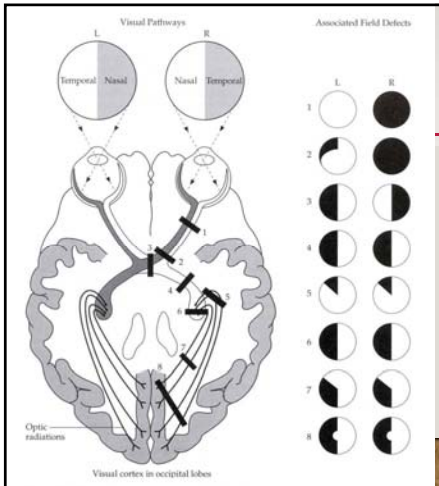
OCULAR DDX

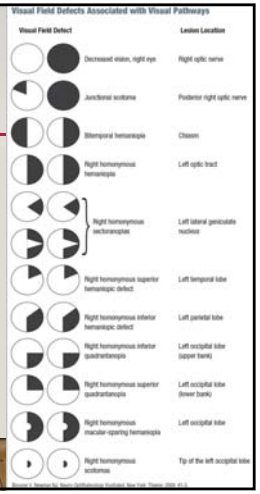
Glaucoma
- HVF defects
- Usually nasal
- Respects horizontal raphe

Tilted disc syndrome
- HVF defects
- Random

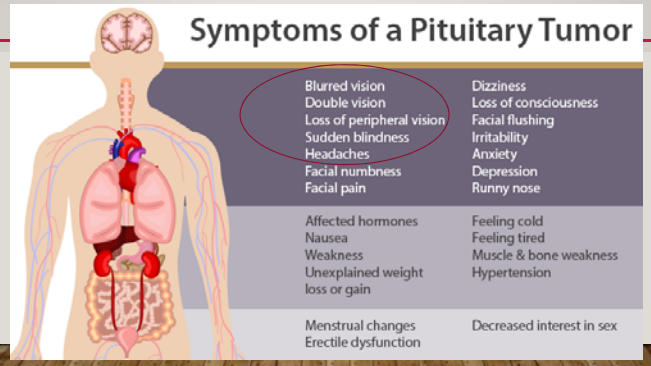
ONH Drusen
- HVF defects
- Random

DIAGNOSIS





SXS



CLINICAL SIGNS

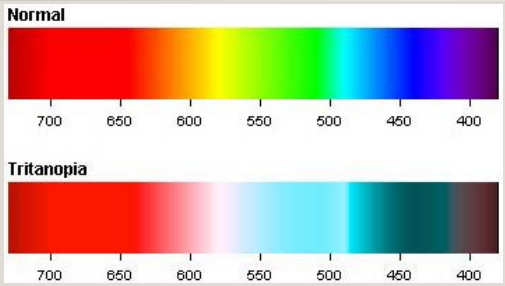
Optic Atrophy
- Chronic tumors
- Bow-tie pattern
- APD?
- CV issues (desaturation, tritan)

Papilledema
- Rare
- Increased cranial pressure due to enlargement of tumor

Nystagmus
- Very rare
- See-saw

DIAGNOSIS

DIAGNOSIS



TREATMENT/MANAGEMENT

Imaging referral
- MRI
- CT w/wo contrast

Neuro-OMD
- Continued management

Endocrinologist
- Hormone imbalance

TREATMENT/MANAGEMENT

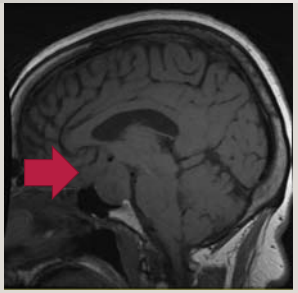
Dependent On The Type Of Tumor

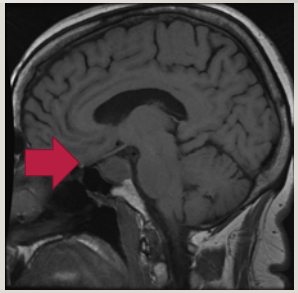
Hormone Therapy
- Prolactinoma: dopamine agonist to combat irregular increases prolactin production

Surgery
- Indicated for macroadenomas
- Trans-sphenoidal resection approach

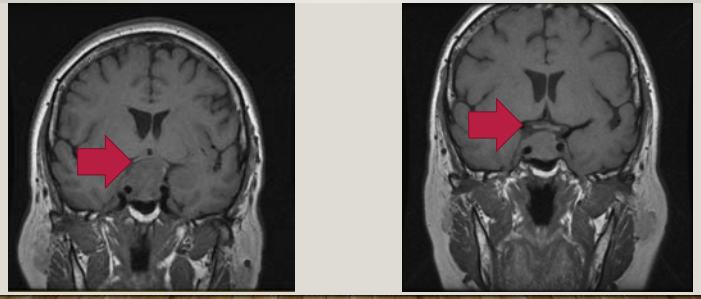
Recurrence
- 10-25% of pts w/in 4 yrs after initial tx.

RESOLUTION (57 YR OLD F; SAGITTAL CUT)



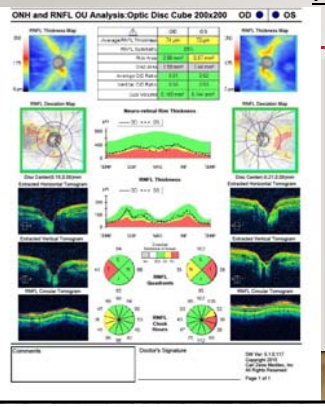


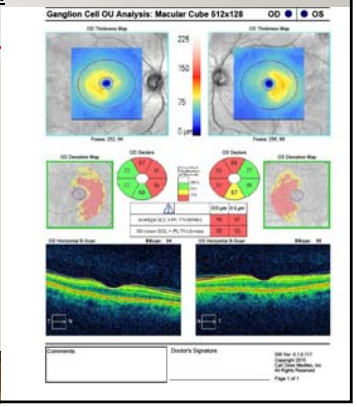
RESOLUTION (57 YR OLD F; CORONAL CUT)



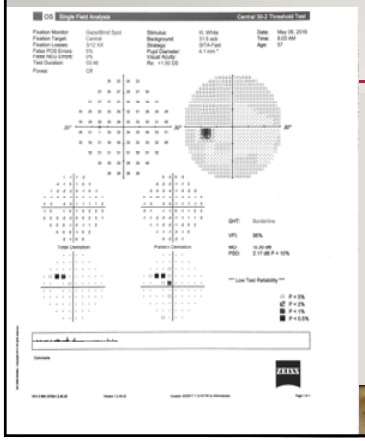
RESOLUTION

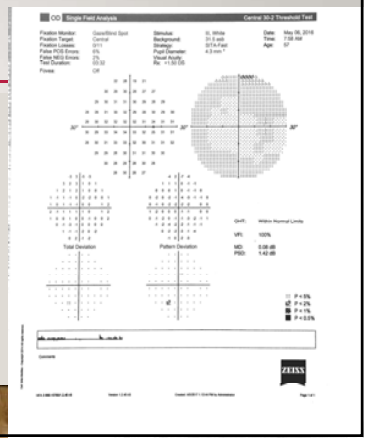
(57 YR OLD F)





HVF





PROGNOSIS

Dependent on severity of pre-treatment state

- Time frame for improvement: 1-4 mos; can take up to 3 yrs
- Most show improvement of VA and VF
- Radiation= slower recovery

CONCLUSION/CLINICAL PEARLS

- Pay attention to all complaints
- Ask about neuro sxs, hormonal changes and HAs
- Visual Fields are key in dx
- Unsure? REFER

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Do Modern Scleral Lenses Provide Adequate Oxygen to the Cornea?

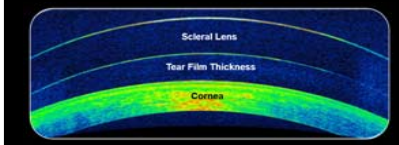
Emily M. Korszen, OD
Pacific University Cornea & Contact Lens Resident
Northwest Residents Conference
June 10, 2017

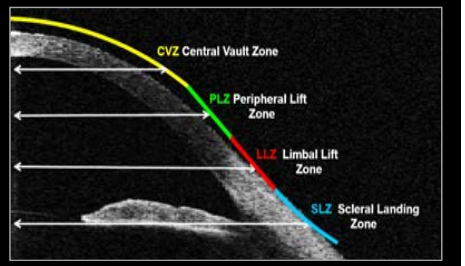
Acknowledgments

- *Prof. Patrick Caroline, FAAO*
- *Prof. Mark Andre, FAAO*
- *Dr. Matt Lampa, OD, FAAO*
- *Dr. Beth Kinoshita, OD, FAAO*

Scleral Lens Fitting Goals

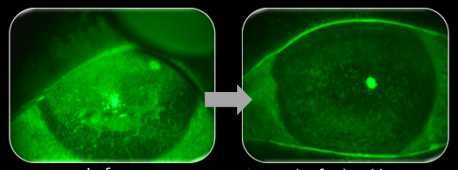
- Vault the cornea and limbus
  - Postlens tear film = clearance
- Land on the sclera (conjunctiva)
- Minimal tear exchange

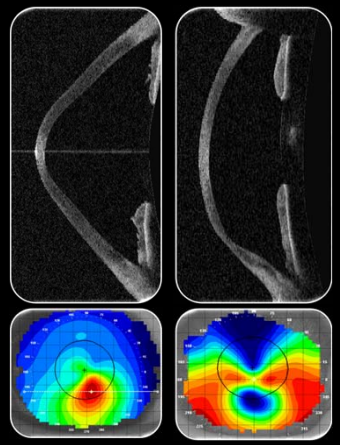




Scleral Lens Applications

- Irregular astigmatism
- Ocular surface disease
- Correction of refractive error in healthy patients?

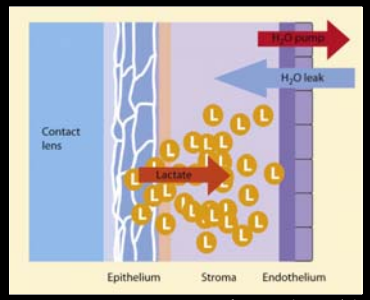

before *1 month of scleral lens wear*



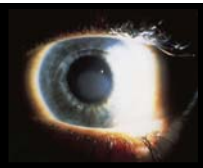
Corneal Oxygen Supply

- The avascular cornea relies on oxygen from:[1]
  - Open eye: primarily atmospheric O_2
  - Closed eye: palpebral capillaries, aqueous
- Oxygen is required for endothelial cell function
  - Maintain corneal transparency

Corneal Hypoxia



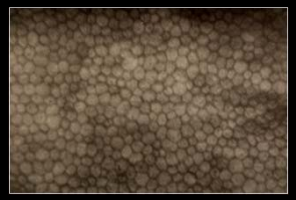
Efron, CL Complications 3rd Ed.

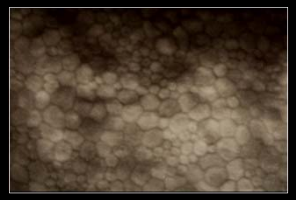


- Anaerobic metabolism → lactate production
- Accumulation of lactate in the stroma → influx of water into the cornea (osmotic forces)
- Corneal swelling: water enters the cornea faster than endothelial cells can pump it out[2]
- Physiological overnight swelling around 3-4%

Endothelial Cell Health

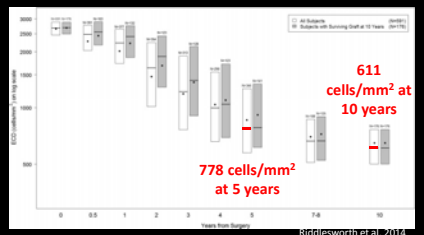
- Implications for successful contact lens wear
- Endothelial cell density and morphology
- Average endothelial cell density:[3,4]
  - At birth: 3000-4000 cells/mm^2
  - Middle age: 2500 cells/mm^2
- 500-1000 cells/mm^2 or less usually results in corneal edema



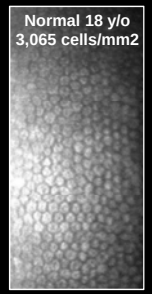


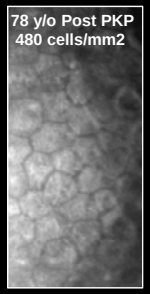
Endothelial Cell Health s/p PKP

- Decrease in cell density after penetrating keratoplasty:[5]

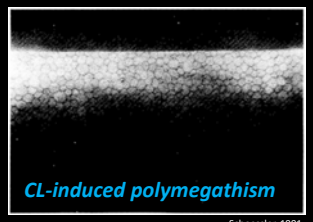


Riddlesworth et al. 2014





Endothelial Cell Health

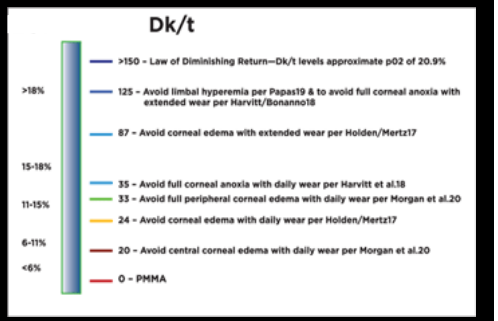


Schoessler, 1991

- Polymegathism and pleomorphism
- Polymegathism has been associated with long-term contact lens wear[6]
  - PMMA
  - Hydrogel soft lenses
  - Rigid gas permeable
  - Not found with silicone hydrogel soft lenses
  - Scleral lenses?

Oxygen Permeability and Transmissibility

- **Dk** Oxygen Permeability
  - Inherent to the material
- **Dk/t** Oxygen Transmissibility
  - Lens thickness taken into account

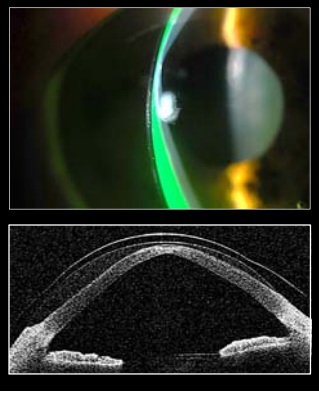


Zimmerman, 2014

How Much O_2 Do We Need?

- Holden & Mertz[8]
  - Daily wear: Dk/t of 24
- Holden et al./Brennan et al.
  - Tear pO_2 of 70-100mmHg
- Aim to prevent hypoxia-related complications:
  - Loss of corneal transparency
  - Endothelial cell damage
  - Neovascularization

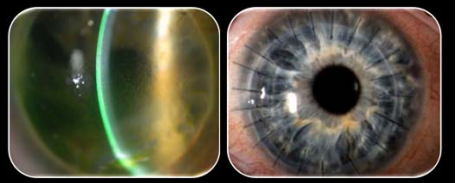
Lens Parameters Affecting Oxygen Delivery



- Lens material Dk: ranges from Dk of 100 to 163
- Lens thickness: center thickness (CT) ranges from 0.20 to 0.50mm
  - Flexure is more common in thinner lenses
- Clearance (postlens tear film): often ranges from 100 to 500µm
  - Areas of thick postlens tear film inevitable with irregular corneas
  - Dk of tears ~ 80

Other Factors Affecting Oxygen Delivery

- Wearing schedule
  - Daily wear vs. extended wear
  - Length of time worn, time lens is put on after sleeping
- Ocular pathology, especially conditions affecting the endothelium
  - Fuch's endothelial dystrophy
  - Post-penetrating keratoplasty
- Individual variance in corneal oxygen consumption



THEORETICAL STUDIES

Theoretical Model: Michaud et al.

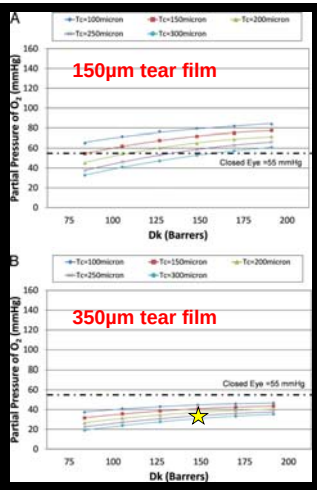
- Formula combining the contact lens and postlens tear film as resistors in series, to find **overall Dk/t of the system**[9]
- Varied: Lens material Dk, Lens thickness, Clearance
- Reference point:
  - Dk/t=24 central cornea, Holden & Mertz
  - Dk/t=35 limbal area, Harvitt & Bonanno

Dk=150	Clearance (μm)	100	150	200	250	300	350	400
Lens thickness (μm)								
250		34.3	28.2	24.0	20.9	18.6	16.6	15.0
300		30.8	25.8	22.2	19.5	17.4	15.7	14.3
350		27.9	23.7	20.7	18.3	16.4	14.9	13.6
400		25.5	22.0	19.3	17.2	15.6	14.2	13.1
450		23.5	20.5	18.2	16.2	14.8	13.5	12.5
500		21.8	19.2	17.1	15.5	14.1	13.0	12.0

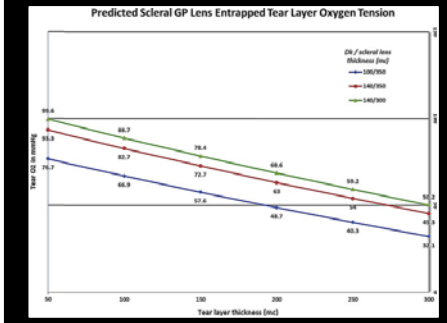
: satisfies HM criteria
: satisfies HM and HB criteria

Theoretical Model: Compañ et al.

- Diffusion equation used to calculate **partial pressure of oxygen** at the cornea under a scleral lens[10]
- Varied: Lens material Dk, Lens thickness, Clearance
- Lower Dk – larger effect from lens thickness and clearance
- Higher Dk – very little effect from lens thickness and clearance



Theoretical Model: Jaynes et al.



- Calculated **partial pressure of oxygen** across the cornea under a scleral lens, considered corneal oxygen consumption (Q)[8]
- Varied: Lens material Dk, Lens thickness, Clearance
- Reference point: tear pO_2 of 100mmHg to avoid edema

CLINICAL STUDIES

Clinical Study: Giasson et al.

- **Corneal relative pO_2** was measured under scleral lenses
- Compared Clearance values of **200µm** vs. **400µm**
- Dk 141, center thickness 0.30mm

RESULTS:[11]

200µm clearance: corneal pO_2 = **9.07%**
400µm clearance: corneal pO_2 = **6.19%**

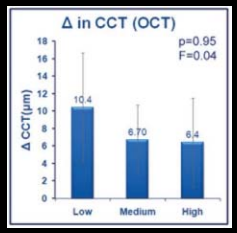
- 9.9% needed to avoid corneal edema
- Findings correspond to theoretical model
- Measured after 5 minutes of wear

Clinical Study: Esen & Toker

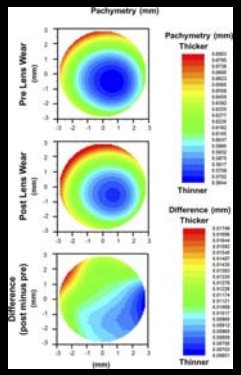
- Central corneal thickness (OCT) after scleral lens wear to estimate hypoxia-related edema
- Compared Clearance values of **100-200µm (low)** vs. **200-300µm (med)** vs. **>300µm (high)**
- Dk = 140, average center thickness = 0.366mm

RESULTS:[12] **Corneal swelling between the three groups did not differ significantly**

- Did not correlate well with theoretical models



Clinical Study: Vincent et al.



- Corneal thickness and posterior corneal curvature measured after 8 hours of scleral lens wear
- Center thickness 0.30mm, Dk 100, clearance 300-400µm

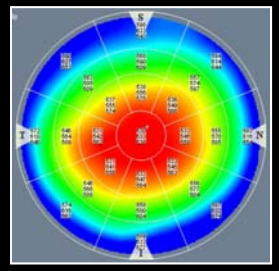
RESULTS:[13]

Average 1.70% corneal swelling

Stable posterior corneal topography

Clinical Studies: Pacific University

- Compare corneal thickness before and after lens wear to measure corneal edema after 8 hours
- Indirect measure of hypoxia
- Visante OCT – global pachymetry
- Evaluate effect of:
  - Clearance
  - Lens thickness
  - Lens Dk



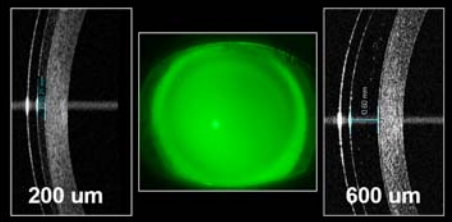
Pacific Lens Clearance Study

- **200µm** vs. **600µm** clearance
- Dk 125, center thickness 0.35mm
- N = 10

Average corneal swelling:

1.92% in the **200µm** group

2.59% in the **600µm** group



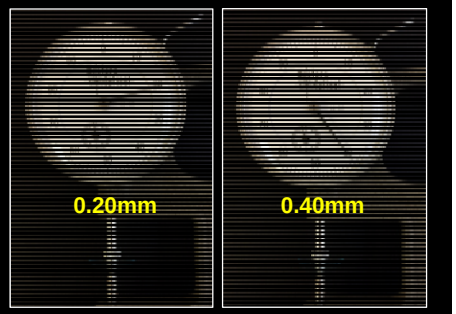
Pacific Lens Thickness Study

- **0.20mm** vs. **0.40mm** center thickness
- Dk 100, clearance 300µm
- N = 7

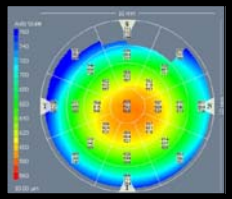
Average corneal swelling:

2.04% in the **0.20mm** group

2.23% in the **0.40mm** group



Pacific Lens Material Study



- **Dk 65** vs. **Dk 100** vs. **Dk 125**
- Center thickness 0.35mm, clearance 400µm
- N = 16

Average corneal swelling:

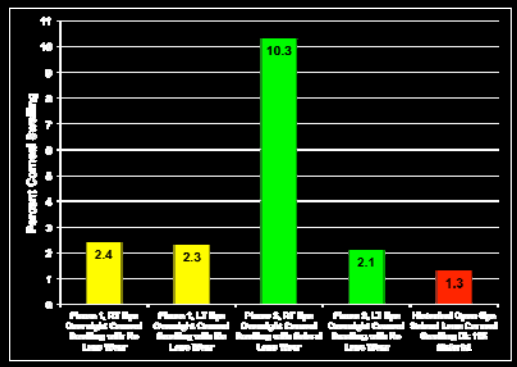
Contamac Comfort **(Dk 65): 2.27%**

Contamac Extra **(Dk 100): 1.54%**

Contamac Extreme **(Dk 125): 1.39%**

- Study in progress: Contamac extra (**Dk 100)** vs. Menicon Z **(Dk 163)**

Pacific Overnight Swelling Study



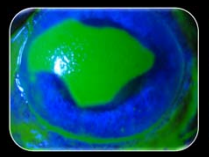
- Phase 1: baseline overnight corneal swelling for each subject with no CL wear.
- Phase 2: OD only 8 hour overnight wear of a scleral lens 0.45mm thick, plano power, Dk 141.
- Corneal thickness measured immediately upon awakening
- N = 10

Current Study Limitations

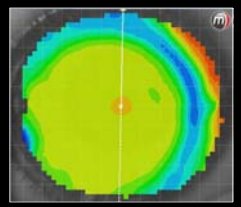
- Theoretical
  - Don't always match clinical findings
  - Don't account for individual corneal oxygen requirements
  - Lens power typically not accounted for
  - Tear exchange?
- Clinical
  - Normal eyes; no compromised corneas
  - Acute; no studies of long-term lens wearers

Optimum Parameters to Minimize Hypoxia

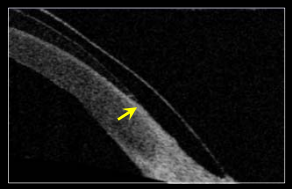
- **High Dk lens material**: at least 100 (higher?)
- **Thinner lens designs**: closer to 0.20 to 0.30mm when possible
- **Lower clearance**: aim for 150-200µm after settling if possible
  - Lenses settle average 100-200µm, patient dependent
  - Not an option with highly irregular corneas
  - Use caution with progressing ectasia
- No overnight wear unless medically necessary
  - Non-healing epithelial defects, keratoprosthesis

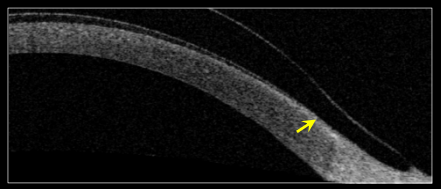


More Than Just Oxygen



- Considerations with small, thin lenses:
  - Clear the limbus!
  - Fully clear the cornea *after* lens settling
  - Check for flexure with keratometry/topography over the lens





Conclusion

- Risk vs. benefit ratio of fitting a scleral lens
  - Extra caution with low endothelial cell counts
  - Consider other options first?
- 6-7% and greater: corneal swelling clinically apparent
- Clinical experience: generally no visible swelling or neovascularization with scleral lens wear (up to 15 years)
- Further studies needed to understand the long-term effects of scleral lens wear on corneal physiology
- Future advancements: better oxygen delivery through postlens tear film?

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9. Michaud L, van der Worp E, Brazeau D, Warde R, Giasson CJ. Predicting estimates of oxygen transmissibility for scleral lenses, Contact Lens and Anterior Eye, Volume 35, Issue 6, 2012; 266-271, ISSN 1367-0484.
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11. Giasson CJ, Morency J, Melillo M, Michaud L. Oxygen Tension Beneath Scleral Lenses of Different Clearances. Optometry and Vision Science. 94(4):466-475, April 2017.
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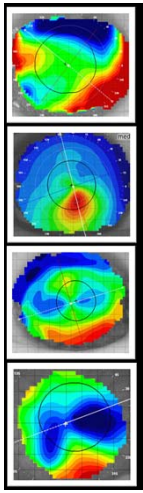
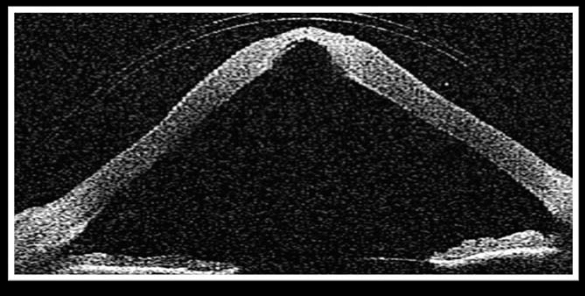
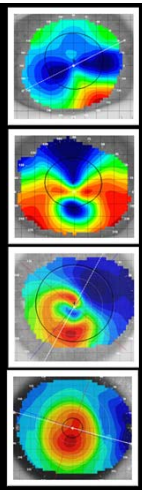
13. Vincent SJ et al. Hypoxic corneal changes following eight hours of scleral contact lens wear. Opt Vis Sci 2016;93(3), 293–299.



Thank You!

e.korszen@pacificu.edu





PROSTHETIC CONTACT LENSES: MORE THAN COSMESIS

Rebecca Lee, O.D.
VA Portland Healthcare System
Northwest Residents Conference
June 9, 2017

DISCLOSURES

- No financial disclosures

LEARNING OBJECTIVES

- Recognize ocular conditions that may benefit from prosthetic CL fitting
- Prescribe and fit prosthetic CLs to achieve optimal cosmetic and therapeutic benefit

CASE: 70yo Male

- **Chief complaint**: Severe photophobia OD since his retinal detachment in 2006 from round explosion, getting more bothersome
- S/p RD repair with scleral buckle
  - Buckle removed 1 year later due to visual distortions
- Pseudophakic OD
- Medical history and Medications: Non-contributory

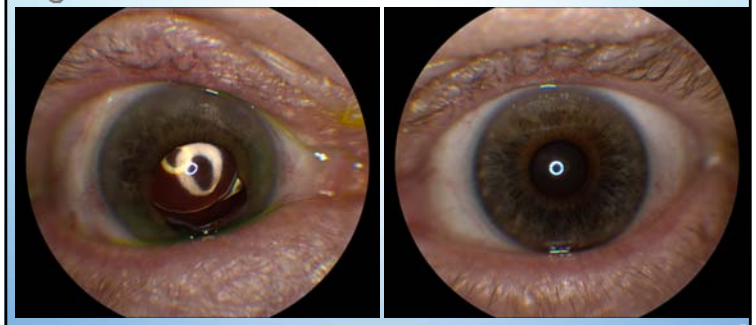
ENTRANCE EXAM

	OD	OS
BCVA	20/40	20/20
EOMs	Full and comitant	Full and comitant
CVF	Mild constriction 360	FTFC
Pupils	Non-reactive, irregular, +APD	No APD
IOP	20	20

SLIT LAMP EXAM

	OD	OS
Lids	Clear	Clear
K	Stromal scar, Mild neovascularization	Clear
Conj	Clear	Clear
AC	Deep & Quiet	Deep & Quiet
Iris	See photo	Clear

OD OS



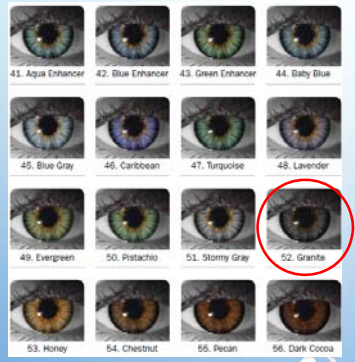
DILATED FUNDUS EXAM

	OD	OS
Lens	PCIOL	NS
Vitreous	PVD	Syneresis
Optic disc	0.25r, healthy	0.20r, healthy
Macula	Clear	Clear
Vessels	Normal	Normal
Periphery	Large area of chorioretinal atrophy inferonasal to ONH extending to periphery, extensive CRA and scarring 360; Retina attached	No holes/breaks/ tears, flat

WHAT NOW?

- Severe photophobia OD secondary to iris dysfunction and RD
- Former soft contact lens wearer 30 years ago
- Refer to contact lens clinic for prosthetic contact lens fitting

ORION BIOCOLORS



BioColors™ Tint Range

Choose from 16 best-in-class tint colors combined with your choice of starburst and/or limbal ring features.

Tint Specifications

Iris Diameter: 12.25

Limbal Diameter: 12.75

Clear Pupil: 2.8, 3.3, 4.2, 5.0, 5.5, 6.0 (4.2 standard)

Cosmetic Feature Options

- Starburst
- Limbal Ring
- Starburst and Limbal Ring *(shown here)*

ORION BIOCOLORS

Base Curve Selection Chart

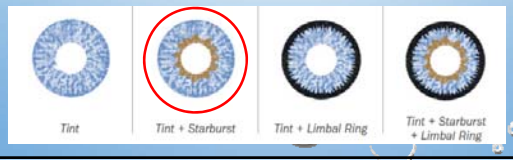
Average K	Lens Diameter: 13.5 & Smaller	14.0 & 14.5	15.0 & Larger
50	7.7	8.0	8.6
49			
49	8.0	8.3	
47			8.9
46		8.6	
45			
44	8.3		9.2
43			
42		8.9	
41			
40	8.6		9.5
39			
38			

Optional Prosthetic Features

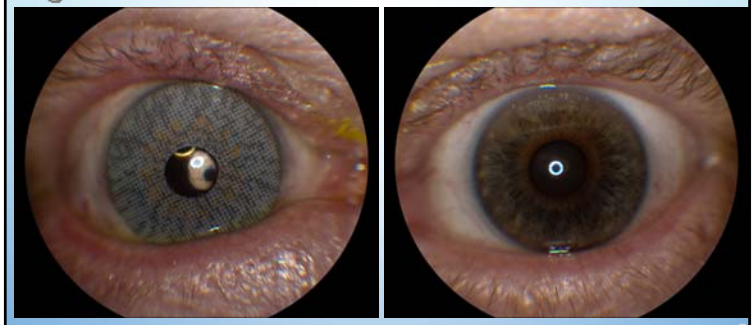
Occluded Pupil: 3.0, 3.5, 4.0, 4.5, 5.0, & 6.0mm

Underprint: Four underprint shades to select from

Total Occlusion available upon request



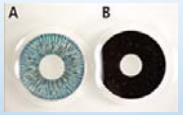
OD **OS**

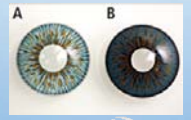


Manufacturer	Power	BC	Dia	Pupil	Color
Orion	Plano	8.6	14.5	3.3, Clear	Underprint U3, #52 Granite, Starburst

TYPES OF PROSTHETIC CONTACT LENSES

1. Transparent tint
2. Standard opaque design
3. Hand-painted lenses

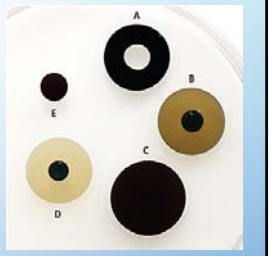




Lam, D. *Contact Lens Spectrum*. 2005.
Cassel, M. *Contact Lens Spectrum*. 2010.

IRIS AND PUPIL OCCLUSION

- Front surface: iris pigment
- Back surface: black backing
- Varying pupil diameters
- Clear/open pupil vs. Opaque/closed pupil



Lam, D. *Contact Lens Spectrum*. 2005.

INDICATIONS: Functional eyes

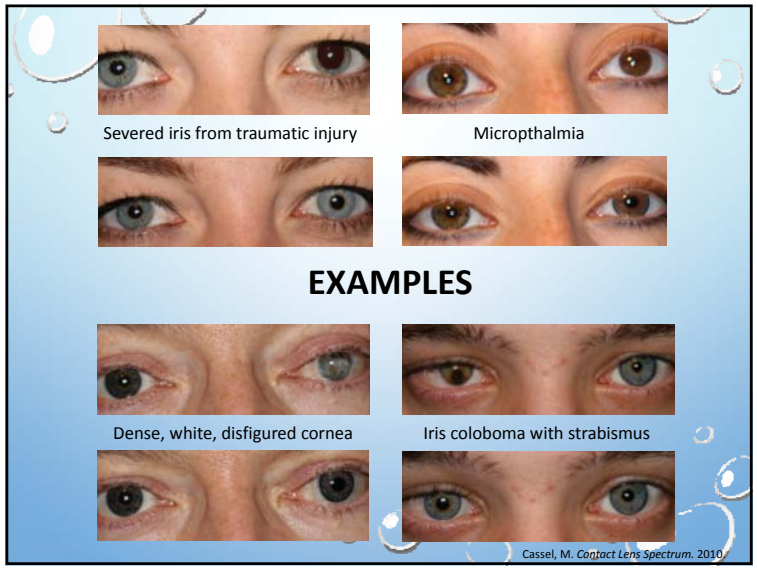
Location	Examples		Manifestation	Benefit
Cornea	Leukoma Bullous or Band keratopathy Microcornea Keratorefractive surgery		Opacity, edema, scars	Cosmesis
Lens	Aphakia Psudophakia Subluxated Lens		Photophobia	Light blocking, comfort
Iris	Aniridia Coloboma Polycoria Heterochromia	Iridectomy/ iridoplegia Posterior synechiae	Glare, photophobia, diplopia	Light blocking, comfort, cosmesis
Vitreous & Retina	Color deficiency Cone dystrophy Retinal Detachment		Photophobia, rod saturation	Light blocking/filtering, comfort
EOMs	Diplopia Amblyopia Strabismus		Diplopia, binocular rivalry	Occlusion, cosmesis
Inherited	Oculocutaneous albinism		Photophobia	Light blocking/comfort

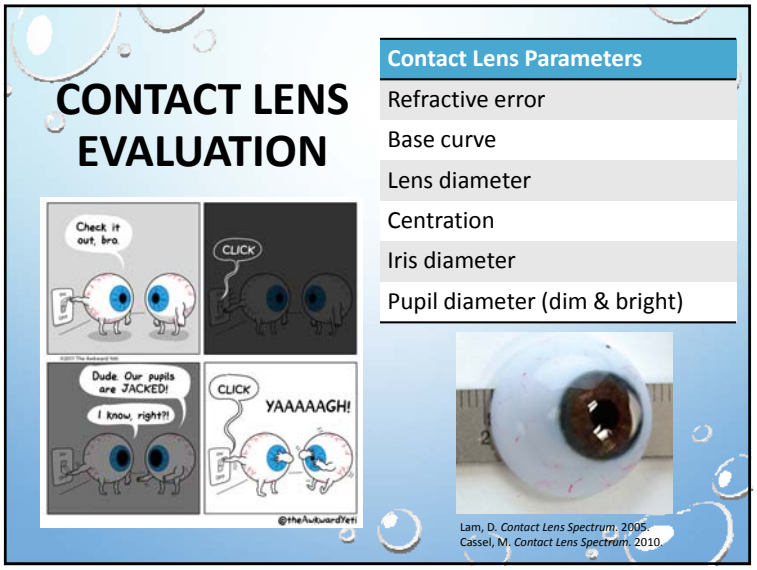
Bator, K.K. *Eyes & Contact Lens*. 2005.; Chan, S. [Powerpoint slides] 2016

INDICATIONS: Non-functional eyes

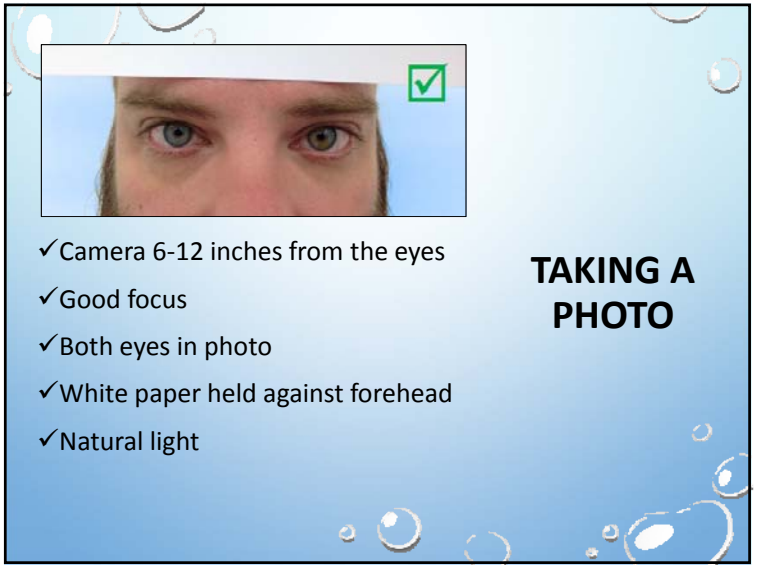
Location	Examples	Manifestation	Benefit
Globe abnormalities	Phthisis bulbi Buphthalmos Trauma	Opacification	Cosmesis
Cornea	Congenital leukoma	Opacification	Cosmesis
Lens	Dnese cataracts	Opacification	Cosmesis
Iris	Aniridia	Photophobia	Light blocking, comfort, cosmesis
Vitreous & Retina	Macular aplasia Hypoplasia Retinal Detachment	Photophobia	Light blocking, comfort

Bator, K.K. *Eyes & Contact Lens*. 2005.


Severed iris from traumatic injury
Micropthalmia
EXAMPLES
Dense, white, disfigured cornea
Iris coloboma with strabismus
Cassel, M. Contact Lens Spectrum. 2010.


CONTACT LENS EVALUATION
Contact Lens Parameters
Refractive error
Base curve
Lens diameter
Centration
Iris diameter
Pupil diameter (dim & bright)
Check it out, bro.
CLICK
Dude. Our pupils are JACKED!
I know, right?!
CLICK
YAAAAAGH!
@theAwkwardYeti
Lam, D. Contact Lens Spectrum. 2005.
Cassel, M. Contact Lens Spectrum. 2010.


CONTACT LENS EVALUATION
Color and Design
Take a photo!
Natural lighting
Dark vs. Light irides
Cosmetic color contact lenses
AIR OPTIX COLORS
Alcon
FRESHLOOK
COLORBLENDS
Lam, D. Contact Lens Spectrum. 2005.
Cassel, M. Contact Lens Spectrum. 2010.


✓Camera 6-12 inches from the eyes
✓Good focus
✓Both eyes in photo
✓White paper held against forehead
✓Natural light
TAKING A PHOTO

CONTACT LENS EVALUATION

- Consult laboratory with first step of lens order
- Order clear lens to confirm lens parameters
- Fitting guide and set
- Multipurpose solution or Hydrogen peroxide

Lam, D. *Contact Lens Spectrum*. 2005.
Cassel, M. *Contact Lens Spectrum*. 2010.

POTENTIAL PROBLEMS

- Lens overwear
- Lens tolerance
- Risk of tight lens fit

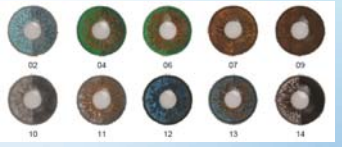
Astin, C.L.K. *Clinical Eye and Vision Care*. 1998.

MANUFACTURERS

Custom and prosthetic tinted lenses	
ABB Optical Group	(800) 772-3911
Advanced Vision Technologies	(800) 393-5374
Alden Optical	(800) 253-3669
Cantor & Nissel	+44 (0)01280-702002
Custom Color Contacts*	(800) 598-2020
Ocu-Ease Optical	(800) 521-8984
Orion Vision Group	(866) 300-6257
Tinting services only	
Adventures in Colors	(800) 537-2845
Crystal Reflections	(800) 807-8722
Specialty Tint	(800) 748-5500

Bator, K.K. *Eye & Contact Lens*. 2005
Cassel, M. *Contact Lens Spectrum*. 2010.

CANTOR PROSTHETIC



ALDEN ENHANCEMENT TINTS & HP PROSTHETICS

PATIENT EXPECTATIONS

- Motivation
- Perfect color match is rarely possible
- Glasses
- Time and money
- Co-management


Einstein discovers that time is actually money.

COST

	Cost to provider
Cosmetic	$40-54 per box *($80-208 for 1 year)*
Tinted	$100-250 per lens
Standard Opaque	$95-250 per lens
Hand painted	$560-680 per lens

Bator, K.K. *Eye & Contact Lens.* 2005
Cassel, M. *Contact Lens Spectrum.* 2010

	Orion Biocolors	Cantor & Nissel	Alden HP Prosthetic	Adventures in Colors
Cost	$95-250	$155	$125	$560-680
Solution	MPS	MPS	MPS	MPS or Hydrogen Peroxide
Replacement	Annually, quarterly	Annually	Annually, quarterly	Annual
Material	Polymacon 38%, Methalfilcon 55%	Filcon 1 38%	Hioxifilcon B, Polymacon 38% water, 9dK	Methafilcon A, 55% water
Diameter	13.0 to 22.0 mm	13.0 to 15.0	Any (13.0-14.5 suggested)	Depends on lens
Base Curve	7.7 to 9.5 mm	8.00 to 9.40 mm	Any (7.7-8.9 suggested)	
Sphere	+20.00 to -20.00	-30.00 to +30.00	+30.00 to -30.00	
Cylinder	Up to -10.00	-0.75 to -6.00	-0.25 to -10.00	
Axis	Up to 180˚	5˚ to 180˚	1˚ to 180˚	

PEARLS AND REFLECTIONS

- If you can fit soft contact lens, you can fit a prosthetic contact lens!
- Consult with manufacturer
- Take a photo!
- Patient education
- Can be life-changing for patient
- Co-management

ACKNOWLEDGEMENTS

- Molly Cardenal, O.D., F.A.A.O.
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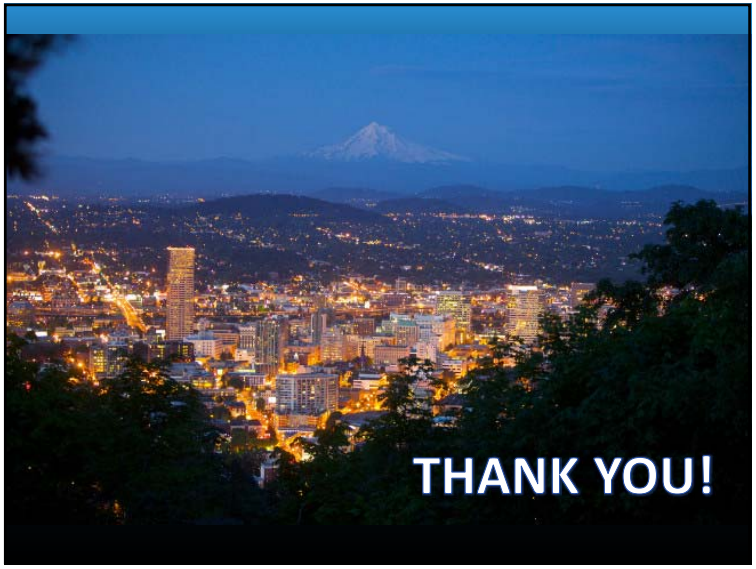


Divergence Excess Treatment Approaches

PRESENTER: KELSEY SIEG, OD

RESIDENCY PROGRAM: VISION NORTHWEST

RESIDENCY IN VISION THERAPY, PEDIATRICS AND NEURO-REHABILITATION

RESIDENCY SUPERVISOR: DR. CURTIS BAXSTROM

Divergence Excess (DE)

- Greater exo at distance than near (by 10 pd)
- Intermittent tendency, can vary with attention
- Normal visual acuity; absence of amblyopia
  - If amblyopic, more likely due to anisometropia than strabismus
- Normal stereopsis
- Calculated AC/A vs. Gradient AC/A
  - High vs. normal
  - Relaxation of accommodation → relaxed convergence vs. active divergence
- Comitant, usually 30 pd XT at distance
- Other names:
  - inattention strabismus
  - distance exotropia
  - divergence excess exotropia
  - periodic exotropia
  - exotropia of inattention
  - intermittent exotropia

Prevalence & Onset

- Strabismus ~5% of developmentally normal children
  - 3-7.5% of strabismics are exotropic
    - Intermittent exotropia 32.1/100,000 in children under 19 years old in the United States
- Women
- Asian and African ancestry more common
- Earliest manifestations around 12 months of age
- Etiology unknown, but theories of:
  - Anatomical, innervational, sensory, genetic

Manifestations and Clinical Characteristics

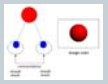
- One eye deviates outward when fixating at distance
  - worse when inattentive, when in bright sunlight, or when tired
- Closure of one eye in sunlight, light sensitivity, and panoramic viewing
- Usually asymptomatic
- Chief complaint: cosmesis
- Normal refractive error distribution
  - Some reports of higher incidence of myopia, especially in Asians with XT

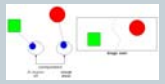
Differential diagnosis

- Infantile XT
  - Rule-out pathologic
    - Constant unilateral
- Basic exophoria/tropia
  - Distance and near equal
- Convergence insufficiency
  - near exo > distance
- True vs. simulated divergence excess
  - +3.00
    - Accommodation relaxed
  - 1 hr occlusion
    - Fusion disrupted

Theories for treatment

- Flax
  - Poor fusional skills due to near point strain and stress
    - Poor control of relaxation of accommodation and divergence
    - Sensory fusion developed; inconsistent motor alignment
- Cooper
  - Due to proximal convergence
    - Align eyes when advantageous – stereoscopic
    - Anomalous retinal correspondence only when eye deviated; aligned normal correspondence
      - During deviation extension of binocular field ie. panoramic viewing – dual retinal correspondence
        - variable





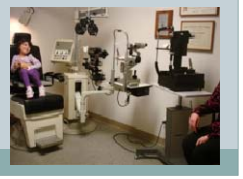
- Duane's
  - Active divergence
    - Lateral rectus increase firing rate during deviation and simultaneous decreased firing of medical rectus (Blodi and Van Allen)
      - Not relaxation of convergence or inability to converge

Treatment Options

- No Treatment/Observation
- Occlusion
- Lenses
  - Low plus at near
    - near vs. distance retinoscopy & accommodative testing
  - Overminus
    - Induce convergence
    - Not induce myopia
- Prism
  - BI
- Surgery
  - No evidence abnormal insertion of EOMs
    - Risks: secondary surgery needed, over/undercorrection, secondary hyper/cyclo deviation, diplopia worse after (effects of EF/AC), alter neuromuscular relationships (make VT harder), perforation of globe, infections, RDs, general anesthsia (long-term differences in language and cognitive function)
      - Success 40-60%
- Vision Therapy
  - Success 65-90%

Treatment Options: Vision Therapy

- Promote binocular vision
  - Third degree → first degree stereopsis
    - Stereoscopic → flat fusion → simultaneous perception (Flax)
  - Larger targets → smaller targets
  - Near space/centration point → distance space/free space
  - Accommodation rock
    - Monocular and binocular (Flax)
  - Smooth vergence → jump ductions → voluntary vergences
  - Bilateral integration
    - Angels in snow, chalkboard circles, etc.
  - Language
    - right/left hemisphere functionality
  - Central-Peripheral awareness and z-axis



Treatment Options: Vision Therapy

- Promote Binocular vision (distance and near)
  - Anaglyphs
  - Vectograms
  - Vertical prism dissociation
  - Bi-temporal occlusion at near
- Proprioceptive awareness
  - Eye control
- Diplopia awareness
  - Physiological diplopia training (Cooper)
- Visual-vestibular
  - linear acceleration → increase tone to all EOMs & optic flow
    - Swing, rocking chair, etc




Case Presentation: 8 y.o. Asian female

- Initial presentation exophoric equal at distance and near
- 1.5 years later presents IAXT
  - 25 IAXT 80% of time distance > 15 IAXT 20% of time at near
- Normal stereo at near, minimal Rx, NPC reduced mildly, W4D suppression at distance
- Vision therapy:
  - Physiological diplopia/diplopia awareness
    - Beads on the string
  - Stereovision in free space
    - Stick in the straw
    - Anaglyphs
    - Quoits
  - Monocular Fixations in a Binocular Field with Accommodative Rock
    - GTVT charts
  - Visual-vestibular input
    - Bean bag toss, turn and toss various distances, rotations with stick in the straw
  - Central-peripheral
    - Wayne saccadic fixator
- Improved magnitude, binocular at distance and near, NPC normal
  - 8 exophoria at near; 12 IAXT at distance


Case 2

- Vision therapy:
  - physiological diplopia/diplopia awareness
  - visual-vestibular tracking
  - MFBF distance and near
    - GTVT; Red pencil + red laser; Red pencil + green marker
- Decreased suppression, decreased frequency

Summary of Vision Therapy Approaches and Pearls

- Start at near (area of fusion) and work into distance/free space
- Stereoscopic targets (tertiary) → Flat fusion (primary)
- Smooth → voluntary vergences
- Bilateral integration
- Language component
- Central-Peripheral
- Visual-Vestibular

Summary of Treatment Approaches & Pearls

- It Depends!

- L.O.V.E.
  - Lots Of Varied Experiences

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The Role of Vision Therapy in Neuro-Optometric Rehabilitation

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Bright Eyes Vision Clinic

Disclosures:

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Acquired Brain Injury (ABI)

ABI: an umbrella term that is defined as "damage to the brain that occurs after birth and which is not related to congenital disorders, developmental disabilities, or processes that progressively damage the brain."

-Toronto Acquired Brain Injury Network

Types of Acquired Brain Injury (ABI)

- **Traumatic Brain Injuries**
  - Severity: Ranges from mild to severe; classified according to Glasgow Coma Scale
    - 75% mild (mTBI)
    - 25% moderate-severe
  - Common Traumatic Causes:
    - Falls
    - MVA
    - GSW
    - Blasts
    - Assaults
    - Spots Injuries
- **Non-Traumatic Brain Injuries**:
  - Stroke/CVA
  - Focal Brain Lesions
  - Tumors
  - Aneurysms
  - Vascular Malformations
  - Anoxia, often post-surgical complications
  - Inflammation or Infection
    - Lyme disease
    - Meningitis
    - Encephalitis

ABI Statistics

- Prevalence:
  - 10 million TBIs worldwide
  - 2.4 million ABIs annually in the United States
  - 795,000 non-traumatic ABIs
  - CDC: 1.7 million TBIs/year in US, likely under estimate
  - Most common cause of TBI is falls (28%)
    - 60% of TBIs in 65+ population
    - Close Second is MVAs (20%)
- Most common Sports related TBI in America is Football
- JAMA Pediatrics Study: risk of concussion in 1 season, July 2015
  - College: 5% risk
  - High School: 10% risk
- Cost of concussions in the US annually: $ 75 billion
- Incidence of concussions is increasing especially in middle school children

Significance

- 40-50% of brain involved in vision and/or visual processing
  - 30-40 brain areas
  - Majority of the cranial nerves (7/12)
- Many ABI patients report vision problems months to even years after an injury
- 40% of patients with ABI have a visual dysfunction complicating their recovery process
- Inability to integrate visual input with kinesthetic, proprioceptive, and vestibular input→ Sensory Mismatch
- Vision problems impair balance and coordination which increases fall risk and risk for a secondary brain injury

CDC Concussion Basics

- HEADS UP to Clinicians: Free Online Concussion Training for Health Care Providers
- Talks about IMPACT testing, Return to Play/School Decisions, recovery time, Sports Sideline Testing, etc.

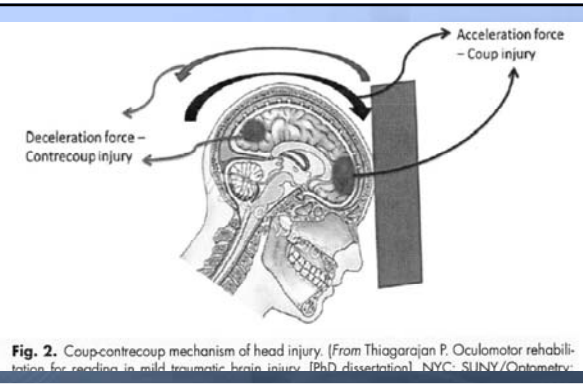


Fig. 2. Coup-contrecoup mechanism of head injury. (*From* Thiagarajan P. Oculomotor rehabili-

Injury Mechanism: Coup-Contrecoup

- Concussion: type of TBI that results from an injury to the head or body that causes the head and brain to move rapidly back and forth and cause damage to brain cells
- Coup-Contrecoup: Direct external force causes injury to opposite brain pole region
  - Damage caused by stretching of white matter fiber tracts and/or flexing/twisting of the midbrain which controls ocular motor center

Recovery Time

- More damage→ increased recovery time: ranges from few months to 1-2 years post-injury
- History of multiple concussions, migraines, mood disorders (depression/anxiety), developmental disorders (ADD/LD) prolongs recovery time
- CDC recommends PCP refer to concussion specialist if symptoms persist past 10-14 days
- Post-Concussion Syndrome: symptoms persist greater than several weeks/month
- Pathophysiology:
  - Concussion is a functional versus structural injury
  - Not always seen on MRI
  - Biochemical changes: neuro-metabolic/ chemical cascade causes axonal damage
- Timeline:
  - Initial Acute Phase: Hours after injury
  - Secondary Sub-Acute Phase: days to months after injury
  - Long term/Chronic Phase: 45 days post injury

Concussion: Signs and Symptoms

- Concussion Signs Observed
  - Can't recall events prior to or after a hit or fall.
  - Appears dazed or stunned.
  - Forgets an instruction, is confused about an assignment or position, or is unsure of the game, score, or opponent.
  - Moves clumsily.
  - Answers questions slowly.
  - Loses consciousness (even briefly).
  - Shows mood, behavior, or personality changes.

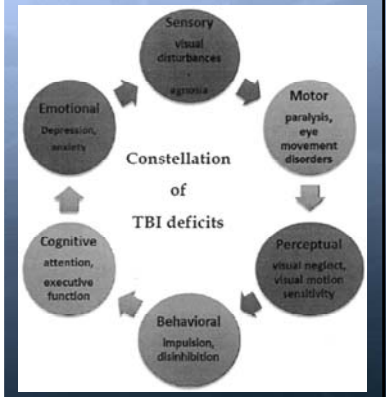
- CDC Concussion Basics
- https://www.cdc.gov/headsup/basics/concussion_symptoms.html
- Concussion Symptoms Reported:
  - Headache or "pressure" in head
  - Nausea or vomiting
  - Balance problems or dizziness, or double or blurry vision
  - Bothered by light or noise
  - Feeling sluggish, hazy, foggy, or groggy
  - Confusion, or concentration or memory problems
  - Just not "feeling right," or "feeling down"

Common Visual Symptoms

- Post Trauma Vision Syndrome (PTVS)- Padula:
  - Signs: exo deviation, accommodative dysfunction, convergence insufficiency, poor fixation and tracking, decreased blink rate/staring behavior, spatial disorientation, unstable ambient vision, Poor concentration, attention, visual memory, balance, coordination, and postural defects
  - Symptoms: Eyestrain, Diplopia, stable objects appear to move

ABI Signs

- Cognitive:
  - Attention
  - Executive function: difficulty following instructions
- Sensory:
  - Visual field defects
  - Light sensitivity
  - Dry Eye
- Ocular Motor:
  - Saccades/Pursuits
  - Vergence
  - Accommodation
- Visual Perceptual:
  - Hyper sensitivity to sensory input
  - Visual motion sensitivity
  - Figure ground, discrimination, memory
- Emotional:
  - Depression, fatigue, anxiety
- Behavior:
  - Impulsive, unreasonable expectations, disruptive



Ciuffreda et al. Traumatic brain injury, visual consequences, diagnosis, and treatment. Advances in Ophthalmology and Optometry 1 (2016) 307-333.

Common Visual Findings

- Binocular Vision Dysfunction:
  - Convergence Insufficiency- most common (47%)
  - Accommodative insufficiency- (42%)
  - Ocular-motor dysfunction (82%)
- Visual-Vestibular dysfunction (80% of TBIs)
  - Deficient VOR/Gaze Stabilization
  - Visual Motion Sensitivity (VMS) (40%)
  - Dizziness
  - Poor balance
- Visual Information Processing deficits (50% of TBIs)
  - Poor multi-tasking and poor reaction time
- Abnormal egocentric localization (30%)
  - Mismatch between subjective and objective sense of straight ahead

Ambient Vision Dysfunction

- Poor peripheral awareness/ poor spatial organization
- Poor magnocellular function, parvocellular and central details dominate.
- Patients report: Sensory Overload
  - Objects appear to move
  - Difficulty with over stimulating settings (crowds, noise, FL lights)
  - Easily overwhelmed by visual details (grocery stores, clutter, patterns)
  - Unable to ignore unimportant visual information
    - Causes Dizziness/ disequilibrium, or poor balance in visually busy environment because unable to suppress excessive visual movement in the background
- Low BI prism, plus lenses, and Binasal occlusion can reduce the effects of Ambient Dysfunction

Management: ODs are an important part of the care team!

- Treatment for ABI is an integrative approach with other rehabilitation professionals
  - Occupational Therapists
  - Speech/Language Therapists
  - Physical/Vestibular Therapists
  - Cognitive Therapists
  - Psychologists, Psychiatrists
  - Neurologists
  - Neuropsychologists
  - Physiatrists, Sports Medicine
  - PCP
  - Chiropractors
- Important to communicate findings with other providers in the patient's care team and refer for all non-visual based problems:
  - Depression/Anxiety
  - Fatigue/Trouble sleeping
  - Cognitive Impairments
  - Behavioral Problems
  - Postural Problems
  - Attention Problems
  - Neurological Problems

Role of Neuro-Optometrists

- Lenses:
  - Computer/reading lenses for Accommodative Dysfunction
    - Sensitive to small changes in refraction
    - No PALs (peripheral swim/distortion)
    - Least amount of add power for function is best
- Prism:
  - BI mini prisms 0.5-1 BI
  - Vertical prism :
    - Sensitive to small amounts of vertical misalignment
  - Yoked prism
- Tints
- Binasal occlusion
- Vision Therapy
- Education of patient and collaboration with care team

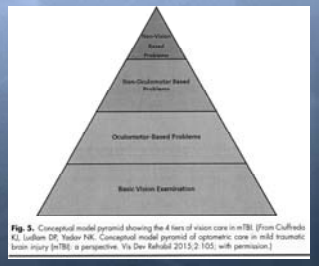


Fig. 3. Conceptual model pyramid showing the 4 tiers of vision care in mTBI. (From Ciuffreda KJ, Ludlam DP, Yadav NK. Conceptual model pyramid of optometric care in mild traumatic brain injury [mTBI]: a perspective. Vis Dev Rehabil 2015;2:105; with permission.)

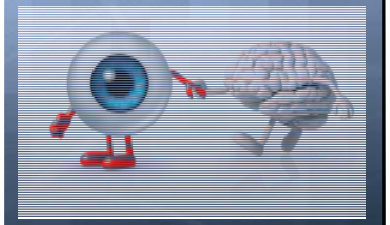
Ciuffreda et al. Traumatic brain injury, visual consequences, diagnosis, and treatment. Advances in Ophthalmology and Optometry 1 (2016) 307-333.

Studies Supporting VT for ABI

- To date, there is no RCT assessing the use of VT to remediate visual dysfunction in patients with ABI- Need large scale RCT with longer follow up time of 1-2 years
- 2016, VT for Binocular Vision Dysfunction Post ABI, Conrad et. al.
  - Prospective study n= 19
  - Home based computer vergence therapy for 12 weeks to treat BVD in adults 3 months+ after ABI
  - Results: Majority had meaningful improvements in signs (NPC, PFV, NFV, vergence facility) and symptoms (CISS) post-treatment
- 2016, VT for Post Concussion Vision Disorders, Gallaway et. al
  - Retrospective chart review, n= 218 concussion patients at two private practices
  - Results:, statistically significant changes in symptoms (CISS) and signs (NPC, PFV, and amplitudes of accommodation) after in office VT
    - 85% of patients w/ CI were successfully treated and 15% were improved
    - 33% of patients w/ AI were successfully treated and 47% were improved 67%?
    - 83% of patients with Saccadic dysfunction were successfully treated and 5% improved
- 2013-2014, Oculomotor Neuro-Rehabilitation for Reading in mTBI, Ciuffreda et. al.
  - Crossover interventional study design with 6 weeks/12 sess in office VT and placebo therapy, n=12
  - >80% improved after in office therapy (increased reading rate, increased amplitudes of accommodation and vergence, increased saccadic accuracy, decreased symptoms on CISS, and increased visual attention
  - No parameters improved with the placebo therapy

Case Report

- History
- Signs and Symptoms
- Exam:
  - Visual Efficiency Findings
  - Ocular Health
  - VF/VEP Testing
- Assessment/Diagnoses
- Management:
  - BI mini prisms
  - In-Office Vision Therapy
- Visual Processing Testing
- Vision Therapy
- Conclusion: End Results



ABI Case History

- Nature of ABI:
  - Location: determines areas of function affected
  - Date of Injury:
    - Acute vs. Chronic
  - Number of Injuries:
    - Multiple ABIs exacerbate visual dysfunctions
- Referral Source/reason for referral
- Types of rehab services received (inpatient and outpatient)
- Visual problems affecting performance of ADLs and progress in other therapies
- Personal and Family Medical and Ocular History
  - Medications/Allergies
  - Look for medications affecting vision/accommodation
- Determine Visual Needs and how patient responds to visual challenges (ie: avoidance, trying harder)
- Social History:
  - Occupation
  - Mobility
  - Driving ability
  - Support resources

Case Example SR, 45 YO WF

- Injury: Concussion 3 months ago from a fall off a truck onto pavement, no other injuries/previous concussions
- Complaints:
  - Blur and diplopia at near
  - Difficulty focusing between near and far distances
  - Transposing letters/#s/words run together, skipping lines, feels like "eyes bounce"
  - Reduced reading comprehension and memory
  - Dizziness and nausea with near work and vertigo
  - Headaches
  - Light sensitivity
  - Poor peripheral awareness and depth perception
- Referred By: Physical Therapist/Neurologist
- ROS: Epilepsy, seizures, depression
- Medications:
  - Gabapentin
  - Lamictal
  - Lexapro
  - Singulair
  - Vitamin D, Biotin, Folic Acid, Omega 3, Calcium

Vision Rehabilitation Examination

- Hab Rx:
  - -2.75-0.50X104 20/20
  - -2.75-0.75X80 20/25
- Ocular Health:
  - IOP 18/20
  - PERRL OU
  - EOMS FROM OU
  - CVF: FTFC OU
  - DFE: Ocular health WNL OU
- Visual Field: 30-2, reliable, full, no misses OU
- VEP: abnormal LX OU
  - Binocular Vision Dysfunction
  - Ambient Vision Dysfunction, improvement in amplitude/latency with BI prism/BNO (Padula protocol)
  - Improvement with BI prism
- Manifest:
  - -2.50-0.25x90 20/20
  - -2.50-0.75x75 20/20
- Prism testing:
  - 1 pd BI OU best
  - Gait Observations:
    - Walk Forward: Careful, right hand up, reports more relaxed and more stable w/ prism
    - Walk Backwards: unsteady, reports easier with prism
    - Tandem walk: very unsteady, improved and reports more steady w/ prism

Sensorimotor Exam

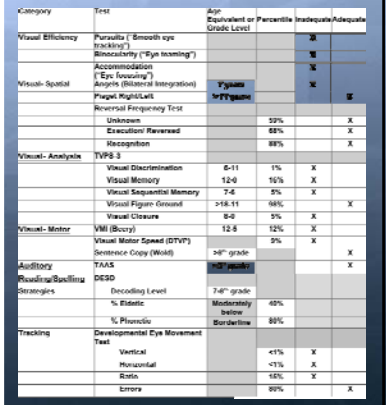
- Randot 6/10, 250'' Random Dot
- CT: ortho/8xp'
- NPC: 45 cm, +Strain
- Pursuits: smooth, +HA, +discomfort superior gazes
- Saccades: irregular/jerky, +HA, several undershoots
  - Feels eyes lag behind/shaking
- Near Prism Bar Ranges:
  - BO: 8/8 BI: 4/2
- W4D: fusion in light and diplopia at near in dark
- MR: aligned 1 BD OD
- VMS: over left ear, more aligned (over OS) w/ prism
- VOR: +Visual Motion Sensitivity
- NRA/PRA: +2.50/ -1.25

Assessment/Plan

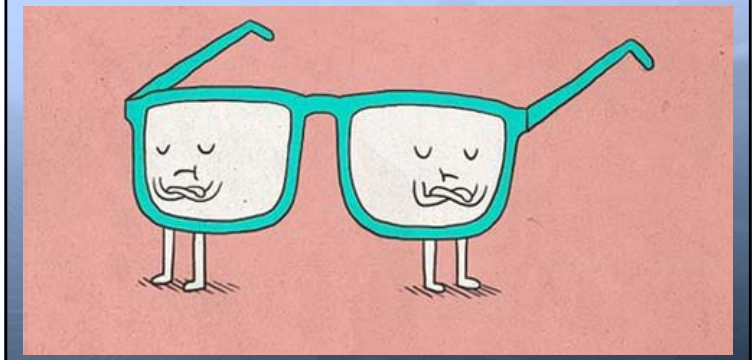
- Post Trauma Vision Syndrome (PTVS)
  - Convergence Insufficiency
  - Poor saccades/Pursuits
  - Headaches
  - Dizziness/Nausea
  - Visual Motion Sensitivity
  - Photosensitivity
  - Abnormal Egocentric Localization
  - Diplopia
- Benign Positional Vertigo (BPV) diagnosed by vestibular PT/neurologist
- Recommend prism glasses and in-office Vision Therapy

Visual Information Processing Evaluation

- Poor bilateral Integration
- Poor Visual Analysis: Visual Discrimination, Visual Memory, Visual Sequential Memory, and Visual Closure
- Poor Visual Motor
- Poor DEM scores



Category	Test	Age Equivalent or Grade Level	Percentile	Inadequate	Adequate
Visual Efficiency	Pursuits (" Smooth eye tracking")			X	
	Binocularity ("Eye teaming")			X	
	Accommodation (" Eye focusing")			X	
Visual- Spatial	Angels (Bilateral Integration)	[illegible]		X	
	Flegel Right/Left	[illegible]			X
	Reversal Frequency Test				
	Unknown		59%		X
	Execution/ Reversed		68%		X
	Recognition		88%		X
Visual- Analysis	TVPS-3				
	Visual Discrimination	6-11	1%	X	
	Visual Memory	12-0	16%	X	
	Visual Sequential Memory	7-4	5%	X	
	Visual Figure Ground	>18.11	98%		X
	Visual Closure	8-0	5%	X	
Visual- Motor	VMI (Beery)	12-8	12%	X	
	Visual Motor Speed (DTVP)		5%	X	
	Sentence Copy (Wold)	3rd grade			X
Auditory	TAAS	[illegible]			X
Reading/Spelling	DESD				
Strategies	Decoding Level	7-8th grade			
	% Eidetic	Moderately below	45%		
	% Phonetic	Borderline	88%		
Tracking	Developmental Eye Movement Test				
	Vertical		<1%	X	
	Horizontal		<1%	X	
	Ratio		16%	X	
	Errors		99%		X



Patient Therapy Goals: "Get Eyes To Work Together!"

- Reduce symptoms: headache, strain, nausea, double vision
- Improve short term memory
- Improve reading and reduce letters/eyes jumping around on the page
- Improve depth perception

Therapy Overview: 30 sessions

- Overall goal is to maximize visual function and reduce frequency and severity of symptoms
- Major areas of concern: tracking, vestibular symptoms, and poor ambient/peripheral awareness as well as processing deficits especially memory and poor automaticity
- Started with basic eye movements then added marsden ball/Greenwald Eye Movements to work on motion sensitivity
  - Incorporate eye movements with head/ body movement, walking, and turning/spinning to address vestibular symptoms
- Concurrently work on:
  - Monocular and binocular accommodation
  - Convergence gross, smooth, step- preference for of out of instrument, free space vergence activities
  - With all activities emphasize peripheral awareness!

ABI Vision Therapy Considerations

- Sequencing similar to learning/development cases but directed more heavily towards individual patient goals and can be limited by severe symptoms
  - Work first on areas of biggest concern/ greatest impact on ADLs
- Non-vision based issues contribute to symptoms and difficulty with activities: cognitive (poor memory and attention) and emotional issues (depression and anxiety)
  - ABI patients in therapy may need longer to complete program and more emotional support
  - Emphasize normal for activities to feel difficult initially and necessary to elicit more symptoms in order to desensitize and rehabilitate the system
- Associated symptoms of light, noise, and motion sensitivity as well as vestibular symptoms may necessitate shorter duration of activities and more breaks
  - May not be able to handle full VT program initially with severe symptoms in these areas, consider trial VT period

Additional considerations

- Note patients physical limitations
- Determine the patient's ability to understand instructions
- Try to use distraction free therapy zone (avoid visual/auditory clutter)
- Start below patient's ability level to build confidence
- Use positive reinforcement
- Be aware of patient's frustration level and change difficulty if needed also encourage patient's self awareness of symptoms and performance of activity
- Use Feedback to encourage proper performance of a skill such as double, blur, or suppression checks
- Encourage recognition of luster, localization, and kinesthetic awareness
- Be creative to help patients overcome obstacles

Outline

1. Ocular Motor activities
2. Visual- Vestibular Activities
3. Motor Activities - Bilateral Integration/midline
4. Central Peripheral Activities
5. Accommodative Activities
6. Vergence activities
7. Visual Processing activities

Oculomotor Activities

Goal: steady fixation and smooth saccades and pursuits without head/body motion or symptoms in any field of gaze + can do activities loaded

1. Greenwald Eye Movements/ Eye control
2. Chalkboard Circle Trace, incorporates peripheral awareness
3. Ball Games
4. Ball Circle Trace
5. Letter Tracking
6. Labarge
7. Pegboard rotator
8. Hart Chart Fixations/ 4 chart saccades/ Coding
9. Percons
10. SVI
11. Space Fixator
12. Incorporate metronome for automaticity and cognitive loading
13. Incorporate balance and vestibular demands to solidify skills

Ball Games

Goal: to improve visual spatial awareness, pursuits, vergence, and hand-eye motor coordination

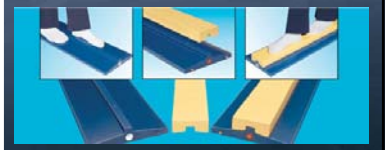
Patient and therapist pass marsden ball back and forth and alternate between catching with Fingertips, Palms, and Fists, can add calling out letter or number written on marsden ball as catch ball.

Visual-Vestibular Activities:

- Goal: reduce symptoms of vertigo, nausea, dizziness, and improve balance skills
  - Vestibular symptoms can significantly interfere with the success of VT if not addressed
  - Patient can become symptomatic during activities and may need frequent breaks/gradually work up to full activity, especially activities involving visual motion
- Start Where Able: laying down → sitting→ standing against wall→ standing, then incorporate walking, rotations, head movement, etc.
- Eye Control/ Ocular Calisthenics
- Greenwald Eye Movements
- Ball Circle Trace
- Chalkboard Circle Traces
- SEE Sickness Manual
  - Roving Eyes
  - Merry Go Round
  - Topsy Turvy
- VOR: Object Fixations moving head- "Gaze Stabilization"
- Midline Activities:
  - Pencil Saccades
  - Knee Taps
- Walking Rail
- Balance Sequence
- Balance Board
- Motor activities with Yoked Prisms

Visual-Vestibular Goals

1. Asymptomatic with pursuits and saccades while:
   1. Moving eyes only
   2. Moving head only
   3. Moving head and eyes
   4. In all fields of gaze
   5. All above on soft surface
      1. Foam Airex
      2. Cushion
   6. All above while balancing
      1. Walking rail
      2. Tandem stance
7. Asymptomatic with trunk rotations
8. No abnormal egocentric localization or visual neglect



Activities to Eliminate Abnormal Egocentric Localization

- Yoked Prisms with Motor Activities:
  - Bean bag toss
  - Ball games
  - Walking
  - Chalkboard Circles

Motor/Sensory Matching Activities

- Goal: remap sensory system and reintegrate senses (vision, proprioception, auditory, etc.)
- Incorporate multi-sensory (inter-modal) tasks:
  - Look (visual)
  - Touch (tactile)- extra sensory input reinforces visually obtained information
  - Listen (Auditory) i.e. metronome
  - Proprioceptive control i.e. Balance on cushion, walking rail, balance board, etc.
- Motor Match to Sensory-Mismatch:
  - Convergence/divergence SILO and localization with vectograms
  - Yoked prism lenses with bean bag catch: ocular motor match to optical shift of prism and motor response to recalibrate sensory-motor match
  - Red green or polarized glasses
- Loading activities: Incorporate motor movement with accommodative and tracking to Improve multi-tasking ability and develop strategies to deal with everyday tasks involving multiple sensory processes simultaneously (ie: visual, auditory, verbal, motor, cognitive requirements)
  - Example: pencil saccades/push-ups while walking or rotating in circles or on balance rail, or in different fields of gaze, or with prism glasses, etc.

Walking rail

Goal: improve balance and visual vestibular interaction (multi-sensory task)

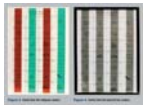
Walking forward
Walking backwards
Add loading:
Reading hart chart
Add metronome and chart
Arrow chart pointing
Marsden ball tracking and pointing
*Shoes off for better sensory feedback/proprioception

http://optometry.times.medicinenet.com/optometrytimes/news/vision-therapy-12-more-tools-your-practice?page=2

Addressing Ambient Vision Dysfunction

- Syntonics light therapy
  - Goal: Open up peripheral vision, reduce constricted visual fields
  - Secondary Effects: Improve sleep, headache symptoms, and anxiety

- Central peripheral activities:
  - Baseball Fixations
  - Peripheral Ball Circle Trace
  - Flashlight Pointing
  - MacDonald Card
  - Stick Pointing
  - SVI
    - Fine motor control
    - Visual scanning/saccades
    - Attention
    - Eye/hand coordination



Accommodative Activities

- Goal: emphasize physical near-far (look hard, look soft) response and awareness of periphery
- BAR 12 cpm at least with a +/- 2.00 flipper without suppression or symptoms

- Near-Far Hart chart
- Flippers MAR/BAR
- Focus Flexibility
- Monocular and binocular bullseye to encourage peripheral awareness
- Lens sorting/feeling
- Wach's mental minus
- Monocular minus lens distance rock

http://optometrytimes.modernmedicine.com/optometrytimes/news/vision-therapy-10-more-tools-your-practice?page=b

Vergence Activities

- Goal:
- Kinesthetic awareness of convergence/divergence (look hard, look soft)
- Gross convergence <2.5 cm from nose
- Smooth/jump vergence 35BO, 13BI
- Vergence facility 12 cpm on 12 BO/3 BI flipper
- Can perform loaded BIM/BOP, metronome

- Marsden Ball convergence
- Pencil push-ups
- Ruler Crawl
- Phys Dip
- Near-Far Pencil Jumps
- Brock string all positions
- Keystone fusion cards
- Vectograms or tranaglyphs
- Eccentric circles
- Lifesaver card / overlapping pictures
- BIM/BOP with Lifesaver or eccentric circles
- Dynamic convergence series

Visual-Perceptual Processing Goals:

- **Visual Spatial**
  - Eliminate abnormal egocentric localization- improve spatial localization
  - Normalize Bilateral Integration, localization, directionality
  - Activities: Floor Maze, Arrow Chart/Labarge, slap tap, midline activities
- **Visual Analysis**
  - Improve Perceptual span (amount of visual information acquired during a fixation)
    - Important in reading, driving, mobility, and visual search (visual attention/processing speed tasks)
    - Activities: Tachistoscope, VIPS, SVI
  - Visual Closure: improve recognition of objects with incomplete information and Improve cognition and processing speed
    - Activities: dot to dot, parquetry blocks, Visual Thinking 101, VIPS
  - Visual Figure Ground: develop organized search patterns to reduce visual confusion
    - Activities: hidden pictures worksheets, VIPS
  - Visual sequencing: improve working memory of sequences of visual information
    - Improve spelling, ability to follow written instructions, and transcribe data
    - Activities: parquetry blocks, tachistoscope, SVI, visual thinking 101, VIPS

Parquetry Blocks

- Direct Match
- Feely Box
- Build and Describe
- Memory
- Sequences
- Build in another place
- Flips and rotations

Visual Motor Integration (VMI)

- Goals:
- Improve reaction time and eye hand coordination
- Improve motor coordination of visual and proprioceptive information for planning and executing a movement
- Improve poor posture, balance, ambulation, navigation, reaching/grasping, and handwriting

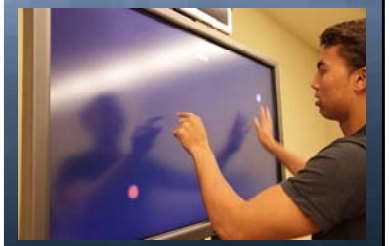
- Activities:
  - SVI
    - Proactive eye-hand
    - Reactive eye-hand
    - Hand speed
    - Rotator
    - Saccades
    - Verbal 1,2
    - Metronome
  - Saccadic Fixator
  - Rotating Peg boards
  - Tracing/drawing activities in perceptual workbooks
  - Integrate multi-sensory and motor-sensory mismatch activities
    - Add Yoked prisms to Activities

Visual Motor Integration (VMI)

Senaptic:
- Eye Hand Coordination and Reaction time

Right Eye:
- Go-No-Go
- Simple Reaction Time
- Choice Reaction Time
- Discriminate Reaction Time
- See more at: https://www.righteye.com/tests-therapies/vision-performance#sthash.jvYU3zVx.dpuf

RightEye



Conclusion: Results

Pre-VT

- CISS: 55
- NPC: 30/50 cm
- Pursuits: smooth +HA, strain in superior gaze
- Saccades: moderate undershoots, +HA
- Near Ranges: BO: 8/8 BI: 4/2
- VMS: Over left ear

After 30 sessions of VT

- CISS: 48
- NPC: 18/20 cm, 8 cm from nose
- Pursuits: smooth, no symptoms
- Saccades: smooth, no symptoms
- Near prism bar Ranges: BO: 20/12 BI: 14/12
- VMS: aligned
- CPM: 15cpm +/-2.00 flipper

Reading Resources:

- Suter P. Vision Rehabilitation. CRC Press 2011
- AOA Brain Injury Electronic Resource Manual Vol IA: Tramatic Brain Injury: Visual Dysfunction Diagnosis, Vol IB: Optometric Management
- CDC Heads Up On Concussion https://www.cdc.gov/concussion/headsup/clinicians/
- Neuro-Optometric Rehabilitation Association www.nora.cc
- Vision Related Literature on Acquired Brain Injury http://www.covd.org/?page=ABI
- Care of the Patient With Special Needs, Ch. 10 Acquired Brian Injury; Ciuffreda, Kenneth
- COVD Acquired & Traumatic Brain Injury, http://www.covd.org/?page=braininjury#hide1

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- Lighthill, Christina R., et al. "Coming into focus: brain injury and vision therapy: a clear vision of patient goals and evidence-based treatment reinforces efficient and effective outcomes." Rehab Management, Nov.-Dec. 2013, p. 20+. Academic OneFile.
- Acquired brain injury : an integrative neuro-rehabilitation approach. Jean Elbaum, 1960-; Deborah M. Benson; ©2007, Chapter 8: The Role of the Neuro-Rehabilitation Optometrist. pp 146-175.
- Thiagarajan P, Ciuffreda KJ, Capo-Aponte JE, et al. Oculomotor neurorehabilitation for reading in mild traumatic brain injury (mTBI): an integrative approach. NeuroRehabilitation 2014;34:129–46. [Context Link]
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- Conrad, Samuel, Joseph ; Mitchell, Lynn, G. ; Kulp, Taylor, Marjean. Vision Therapy for Binocular Dysfunction Post Brain Injury. Optometry and Vision Science, 2017, Vol.94(1), p.101-107 [Peer Reviewed Journal]
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Thank You!

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Case Presentation and Management of Constant Alternating Exotropia with Vision Therapy

Cara Sczepanski, OD
Northwest Eye Care Professionals

COPE and Financial Disclosure

- COPE approved course and in compliance with its Continuing Education (CE)
- I, Cara Sczepanski, OD, do not have any relevant financial relationships and/or commercial interest that could affect the content of the educational activity/lecture.

Case

- CC: 9 year old Hispanic female presented as a referral for poor tracking and eye turn
  - Eye turn occurs when reading and when tired; pt often sits very close to paper
  - No Has, no diplopia
  - Per mother: trouble with visual spatial orienting, delayed motor planning, poor handwriting, slow verbal response
- Ocular Hx: constant XTOU, patching Hx, no Hx of spectacle wear
- Medical Hx: Autism spectrum and ADHD, ADHD medication
- Sx Hx: previous strabismus sx for XT around 2.5 years of age
- Family Ocular and Medical Hx: unknown

Examination Findings, Diagnoses, and A/P

- 20/20 entering sc VAs
- WNL pupils, CVF
- EOMs (full, but jerky)
- Hirschberg: nasal displacement OD approx. mid iris
- Pursuits: 2/5, jerkiness
- Saccades: 2/5, overshooting
- CT: CAXT at distance and near approx. 20pd
- NPC: >1M
- No stereo detected
- Ret and Refraction: low plus with low cylinder OU
- Unremarkable ocular health OU
- Diagnosis: CAXT, diplopia, OMD saccades, OMD pursuits
- Plan: A spectacle RX with Fresnel prism was prescribed in addition to VT. 32+ VT sessions projected

Progress Evaluations

- First PE: reduced XT at distance (4pd XT), improved fixation and eye movement, pursuits 3/5
- Second PE: poor fixation during EOMs and CVF but otherwise WNL, 4/5 pursuits with little to no refixations, 4/5 saccades with mild undershoot, exophoria at near with CT, able to maintain fusion and control with 5BO and BI, DBI: 18/8, DBO 10/6, questionable accommodative responses
- Third PE: stable pursuits, saccades and CT, NBI: 24/6, NBO: 40/0, prism bar NBI: 12/8 and NBO: 6/4, NRA/PRA: +3.00/-5.00

Improvements Noted in VT and by Parents

- Davis: 15/66 to 23/66
- DEM Vertical: <1% to 33%, lack of automaticity
- DEM Horizontal: <1% to 7%, lack of automaticity
- VTS4 Motor fields: OU 10 eso to 12 exo, to OD 0.19 eso and OS 0.04exo
- VTS4 Saccades: 97% with 1.78 response time to >90% with 1.26 response time
- VTS4 Pursuits: previously unable to perform to 100% with 1.01 response time
- NPC: >1M to 5 cm
- Pt's mother noted that she is doing better in school; more focused and better at math, better cognition. In the past the pt would be "done" with school a couple months into the start of the new year and would not want to continue to do HW, but now she is remaining interested in school.

What is strabismus (exotropia vs esotropia)?

A. Right esotropia

B. Right exotropia

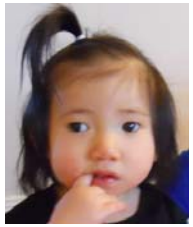
- When the visual axes of the eyes are not aligned toward the same object of interest; bi-foveal fixation is not present

Why does strabismus occur?

- Strabismus occurs due to a failure of the developmental process and learning how to coordinate the two eyes together.
- Eye muscles are normal in almost all patients with strabismus
  - Less than 5% of strabismus patients have an eye muscle problem

Prevalence of Exotropia

- Seen in 1% of the population
  - IXT is most common
- Female to male ratio: 2:1
- Less prevalent than ET (approx. ¼)
- More prevalent in Middle Eastern, Asian, African races
- Infantile XT is quite rare
- Most prevalent during the first decade of life



Characteristics of Exotropia

- Onset
  - Infantile: before 1 year of age
  - Non-infantile: 1.5 to 8 years of age
  - Late onset: associated with fatigue, decompensated phoria, systemic illnesses, trauma
- Constancy
  - Intermittent vs. Constant
    - Approx. 80% intermittent
- Left, Right, Alternating
- Periodicity: distance, near, or both
  - Amount of eye turn may differ with different distances

Subjective Signs and Symptoms of Exotropia

- Outward eye turn
- Closing one eye in bright light
- Head turn
- Decreased stereo

- Intermittent XT
  - Ocular discomfort
  - Eyes pulling
  - Blur
  - Headaches
  - Diplopia
  - For those without symptoms: suppression?, task avoidance
- Constant XT
  - Not many subjective symptoms
  - Poor cosmetic appearance

Types of Exotropia

- Divergence Excess Type (DEXT)
  - Distance deviation >Near deviation (>10pd)
  - 7-24% depending on the study
- Basic Type (BXT)
  - Distance deviation = Near Deviation (within 5-10 pd)
- Convergence Insufficiency Type (CIXT)
  - Distance deviation < Near deviation (>10pd)
  - Most common
  - 3-5% of US population

Divergence Excess XT

- Distance deviation >Near deviation (>10pd)
- 7-24% of cases depending on the study
- Clinical signs: distance vision suppression, normal NPC, normal or limited distance BI ranges, adequate distance BO ranges, poor response to 1st and 2nd degree fusion targets, appropriate response to stereo at near
- Symptoms: one eye turns out, occasional asthenopia, closes an eye in bright light, possible occasional diplopia, usually no HA/eyestrain/blur
- Treatment: refractive correction, VT

Basic XT

- Reduced distance and near BO
- Good prognosis with VT
- Distance and near posture within 5-10 prism diopters
- Possibly receded NPC
- Reduced BO vergence facility at distance and near
- Symptoms: eyestrain/HA, blur or diplopia at distance or near, fatigue, pulling sensation around eyes

Convergence Insufficiency XT

- Symptoms: near vision symptoms worse at end of day
  - Longstanding
  - Eyestrain/HAs/fatigue
  - Blur or diplopia at near
  - Pulling sensation around eyes
  - If no symptoms: near task avoidance, closing an eye, suppressing
- Treatment approaches: refractive correction, prism, VT (85-95% success rate)

Differential Diagnoses of Exotropia

- Pseudoexotropia (appearance of XT but no movement on CT)
  - Large positive angle Kappa
  - Wide PD
- Third nerve palsy with medial rectus weakness
- Duane's syndrome type 2 (no adduction)
- Orbital disease (ie tumor)
- Myasthenia Gravis
- Consecutive XT post strab surgery

Key Characteristics Affecting Prognosis

- Frequency: constant vs intermittent, D and/or N, dependent on energy level or visual tasks
- Magnitude:
  - Micro: 1-5 pd
  - Small: 6-20 pd
  - Moderate: 21-40 pd
  - Large: >40 pd
- Concomitancy: same size of deviation angle in all fields of gaze; monitor for changes during therapy
- Laterality: one eye or both eyes
- Age of onset: typical onset for XT is 18-28 months, better prognosis better than infantile ETs, be cautious with adult/sudden onset
- Duration: XTs of long duration may be successfully treated but expected results are less predictable, longer the duration the poorer the prognosis d/t potential for sensory adaptations
- Sensory Anomalies: suppression (most common), EF, AC
- Prior surgeries: post surgical XT (pre surgical XT with either an undercorrection or a regression of an XT posture post surgically); consecutive XT (pre surgical ET with an overcorrection to an XT post surgically); difficult to treat

Frequency

- Frequency
  - Constant vs Intermittent
  - At distance and near
  - Dependent on visual tasks
  - Dependent on energy level

Magnitude

- Micro: 1-5 pd
- Small: 6-20 pd
- Moderate: 21-40 pd
- Large: >40 pd

- Basic XT
  - Intermittent : 15-20 pd
  - Constant: 25-50 pd
- Convergence Insufficiency XT
  - Distance: ortho to 10 pd
  - Near: 10-20 pd
- Divergence Excess XT
  - Distance: 20-30 pd
  - Near: ortho to 10 pd

Magnitude Continued

- Differentiate a true DEXT from a Pseudo DEXT

- True DEXT
  - High AC/A ratio
  - Measured angles are the actual dissociated angles
- Pseudo DEXT
  - Not totally dissociated; the apparent deviation at near is smaller than the true deviation

- Differentiate by patching one eye for approx. 30 minutes to break down any spasm of fusional convergence

Concomitancy

- Size of angles in all fields of gaze
- Monitor for changes during therapy
- Common etiologies of non-concomitant deviations:
  - Obstetric trauma
  - Muscle anomalies (development, insertion, length)
  - Trauma
  - Vascular disease
  - Neoplasm
  - Viral disease
  - Chronic disease

Laterality

- Many constant XTs alternate fixation with each eye (but one eye may remain in the exo posture)
- Constant unilateral XTs may have deep amblyopia and eccentric fixation

Age of Onset

- Typical age of onset of XT: 18-28 months
- Intermittent divergent position: common in early infancy; typically replaced by stable fixation by 2-3 months of age
- Prognosis better than infantile ETs
- Preschool XTs: difficult to teach to voluntarily converge
- Adult: large XP decompensates into an intermittent or constant XT

Duration

- XTs of long duration may be successfully treated but expected results are less predictable
- Longer the duration the poorer the prognosis; more likely chance of suppression and AC
- Recent onset XTs of any age: evaluate carefully for potential of systemic etiology
  - Co-manage with other physicians
  - Initial management goal to resolve/minimize symptoms, BI prism

Sensory Anomalies and Adaptive Conditions

- Suppression: most common anomaly
  - All or part of the visual field of one eye is not perceived under simultaneous stimulation of both eyes
  - To prevent diplopia, confusion
  - Prevent a poor quality/blurry image from allowing a clear, single image
- Amblyopia: relatively normal VAs in the majority of XTs
- Eccentric fixation: monocular condition in which a non-foveal part of the retina is used for fixation
- Anomalous correspondence: binocular condition in which the two foveas do not form a pair of corresponding points
  - Foveal point in the fixating eye and a non-foveal point in the non-fixating eye with both eyes open
  - Cortical phenomenon; not a retinal phenomenon
  - Most IXTs have HAC resulting in single vision and some binocularity
  - CXTs: other types of AC found resulting in diplopia unless suppression is present
- All 3 anomalies may be found in pts with XT

Prior Surgeries

- Two types
  - Post surgical XT (pre surgical XT with either an undercorrection or a regression of an XT posture post surgically)
  - Consecutive XT (pre surgical ET with an overcorrection to an XT post surgically)
    - Difficult to treat
- Post surgical XTs: some have original deviation reduced, but still have sensory or motor anomalies
  - May need second surgery
- Convergence therapy prior to surgery
  - Some surgeons feel it results in overcorrection of the XT angle
  - Recommendation: VT pre surgery

Treatment and Management Options

- Optical Correction
- Occlusion
- VT
- Surgery

Optical Correction

- Goal: reduce binocular competition and improve binocularity
- Correct significant refractive error
  - Astigmatism
  - Anisometropia: consider CLs for significant anisometropia
- BI prism
  - Consider for initial RX and/or if patient has severe symptoms
- Over minus: least minus with maximum benefit of less XT
  - Minus will stimulate accommodation and make use of convergence to help reduce XT

Optical Correction Approach for IXTs

- Undercorrect hyperopia/overcorrection of myopia (good for preschool patients who may resist convergence training)
- Rule of Thumb: use 1D of over minus correction for each 12 pd of XT (make adjustments as needed)
- Near ADD may be required for school age patients
- CIXT: ametropic RX
- BXT: minus over correction at both far and near
- DEXT: minus correction at far and plus ADD for near

Occlusion

- Bitemporal vs. full
- Previously taught: Part time: 2-6 hours/day
  - Method to disrupt suppression and create diplopia awareness
  - Promote alignment
  - Break suppression
  - 37% success rate
- Dr. Sanet from COVD: full patching with opaque patch only during VT
  - Reduces changes for equalization of binocularity
  - Prefers MFBF activities
- Recommendation: use with other forms of treatment

Vision Therapy

- Preferred treatment if less than 20-25 pd; but improvements have been gained with up to 60pd
  - Depends on maturity of patient
  - Patient motivation is crucial
  - 59-92% success rate
- Goals: phoria at distance and near instead of tropia, normal fusional ranges, improved stereo, no suppression
  - Long term follow up: 63-86% retained phoria up to 2.5-3.0 years
- Can be combined with occlusion therapy
- Great success with dedicated patients

VT Strategy

- Stimulate strong AC/A response
- Teach eye movement awareness
- Stabilize voluntary convergence control
- Establish sensory fusion
- Teach diplopia awareness when eye drifts out
- Teach fast voluntary convergence recovery of IXT
- Teach accommodative accuracy
- Teach fusional convergence accuracy
- Stabilize efficient binocular vision in open visual space

VT Strategy

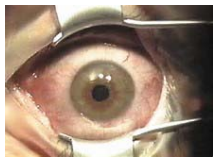
- Work from outside to in (Peripheral to Central)
- Improve cosmetic appearance
- Improve life performance based on patient goals
- Improve egocentric and oculocentric awareness and integration with primitive reflexes, body work, bilateral integration/balance
- Accommodation
- Free space procedures
- Central information processing demand with peripheral awareness and central-peripheral integration
- Stereo
- Diplopia awareness training as feedback mechanism

Visual Goals of Exotropia and Lack of Intervention

- Meet patient goals
- Clear, single, comfortable, cosmetically acceptable, efficient binocular vision at all distances and gazes, normal NPC, stereo, normal vergence ranges, comfortable vision, less symptoms
- Decreased time or episodes of XT
  - Generally, the larger the deviation, the more frequent the intermittent episodes
- Cosmesis
  - Important consideration even if binocular vision cannot be achieved
- Lack of Intervention: diplopia, poor cosmesis, poor self esteem, poor binocularity, poor body coordination

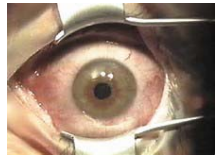
Surgery

- For deviations >25 pd
  - Some recommend IXTs >25 pd (at D or N) or CXT >20 pd
  - Success rate depends on type of XT and magnitude
  - Typically there is a recession of the lateral rectus and/or a resection of the medial rectus insertion
- Patients frequently regress to their previous deviation (multiple surgeries needed)
- Constant XT more likely to require surgery that IXT
- Cosmetic success rate: <15 pd has 70-95% success
- Post operative ET is associated with amblyopia and may precipitate loss of stereo



Surgical Disadvantages

- Surgical risks: (slipped or lost muscles, RDs, globe perforation, infections)
- Over/under correction, hyper/cyclo deviations, consecutive strabismus (4-20% with 42% occurring within 4 yrs post-op)
- Neural/spatial adaptations possibly stronger after sx
- Diplopia
- Nerve/muscle fiber severing leading to altered neuromuscular relationships thereby making VT more difficult
- Emotional trauma to child/patient
- Language, learning, cognitive problems later in life due to early childhood exposure to anesthesia



Lack of Intervention

- Diplopia
- Poor cosmesis
- Poor self esteem
- Poor binocularity

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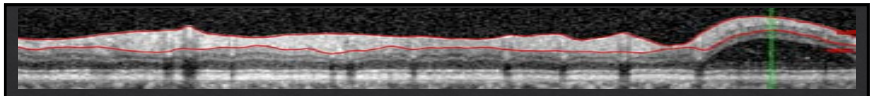
- "It is often stated that humans with amblyopia cannot be treated beyond a certain age; however, a review of the literature suggests otherwise. There is no clear upper age limit for recovery of visual acuity, and there are many studies of improvement in acuity of older people with amblyopia…New clinical and experimental studies in both animals and humans provide evidence for neural plasticity beyond the critical period." Dr. Dennis Levi, Dean, Berkeley University College of Optometry
- "Neural wiring in the visual system is not dismantled by visual deprivation. Neural wiring in the visual system is merely deactivated and is capable of being rapidly restored" Krahe, Medina, et, al. Neuron, Vol. 48, 329-343, October 2005



Peripapillary Retinoschisis in a Patient with an Acquired Pit of the Optic Nerve

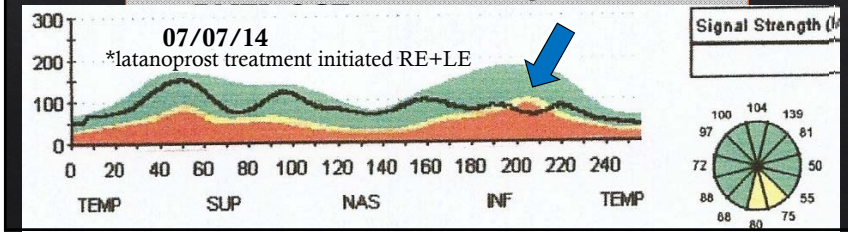
Joshua Clermont, O.D.
VA Puget Sound Healthcare System
American Lake Division
June 10, 2017

Case History

- 74 year old African American male
- H/O normal tension glaucoma (NTG) in his left eye
- CC: glaucoma f/u
- Ocular meds: latanoprost 1gtt QHS OU
- Personal medical hx: (+)Irritable bowel syndrome
- Family ocular hx: (+)glaucoma (mother, father, and two sisters)

History of Present Illness

- NTG suspect OS from 2010-2014 w/o tx
- Thin CCT
- (+) Strong FHx
- Borderline inferior thinning OS on



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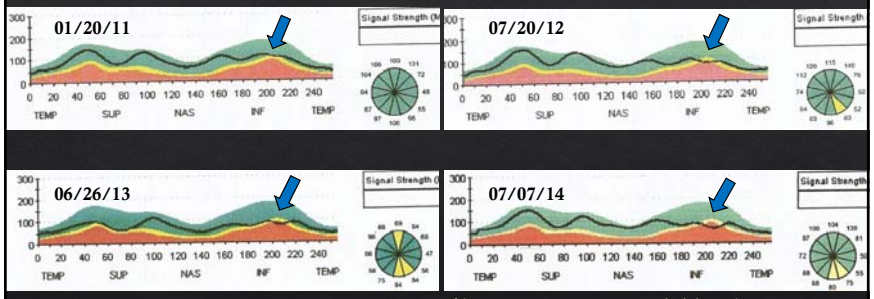
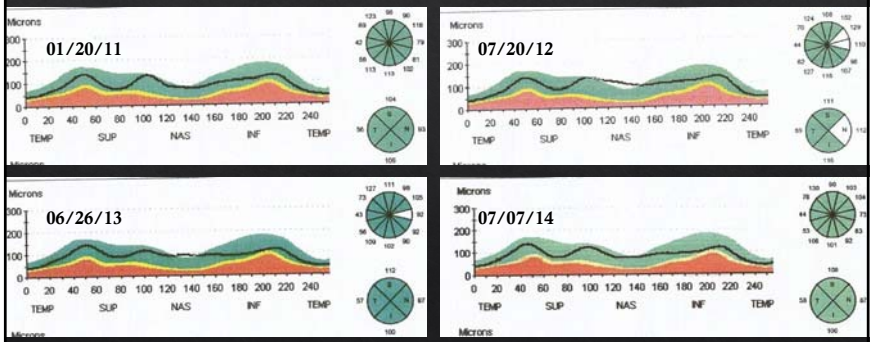


*latanoprost treatment initiated RE+LE

RNFL OCT OD



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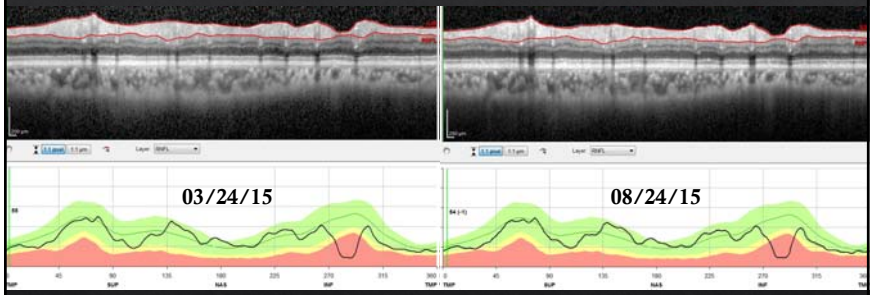
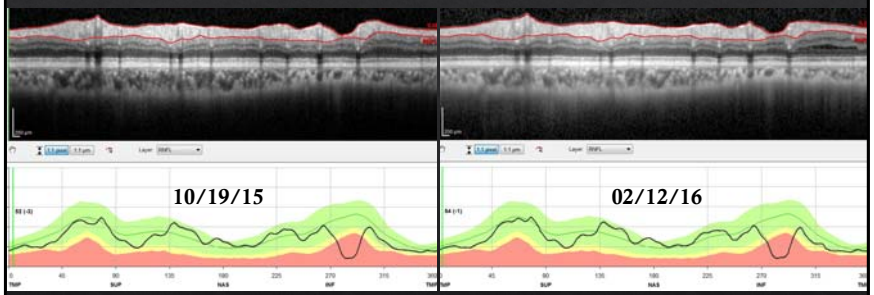


*Baseline Heidelberg Spectralis

RNFL OCT OS



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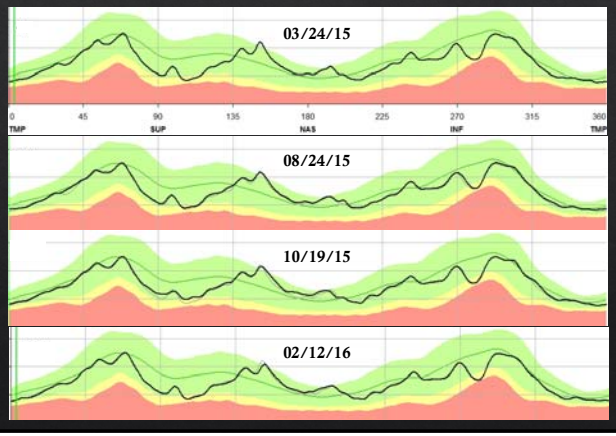
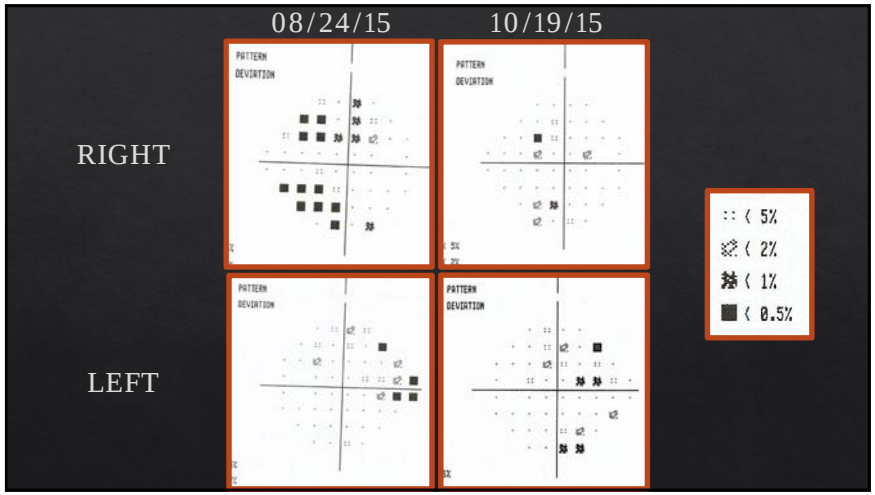


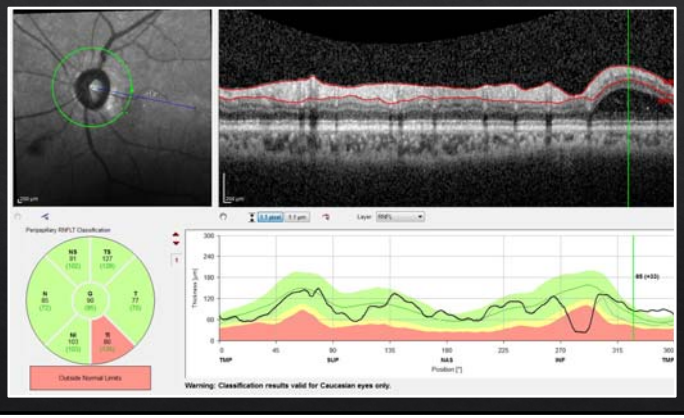


DATE	OD	OS	TIME	TX	OTHER TESTING	NOTES
04/28/10	12	12	0822	NONE	OCT	WNL 360 RE; Borderline inferior thinning LE
05/24/10	18	18	1359	NONE		
01/20/11	18	16	1107	NONE	OCT	Stable thinning LE
09/30/11	17	16	0853	NONE		
04/19/12	15	16	1402	NONE		
07/20/12	13	12	0910	NONE	OCT	Progressed inferior thinning LE
06/26/13	18	18	1326	NONE	OCT; FDT	Progressed inferior thinning LE; No FDT defects R+L
09/26/13	22	20	1049	NONE		
07/07/14	19	18	1029	NONE	OCT; FDT	Progressed inferior thinning LE; No FDT defects R+L
10/19/14	18	18	0900	Latanoprost QHS OU		
12/04/14	17	17	0815	Latanoprost QHS OU		
03/24/15	14	14	0820	Latanoprost QHS OU	OCT	Stable R+L
08/24/15	21	21	1100	Latanoprost QHS OU	OCT; 24-2 HVF	Stable OCT; unreliable HVF
10/19/15	16	17	1308	Latanoprost QHS OU	OCT; 24-2 HVF	Stable OCT; reliable superior paracentral VF defects

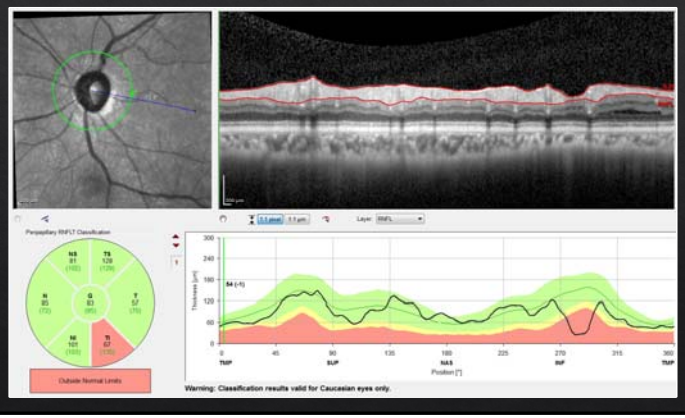
Initial exam-07/28/16

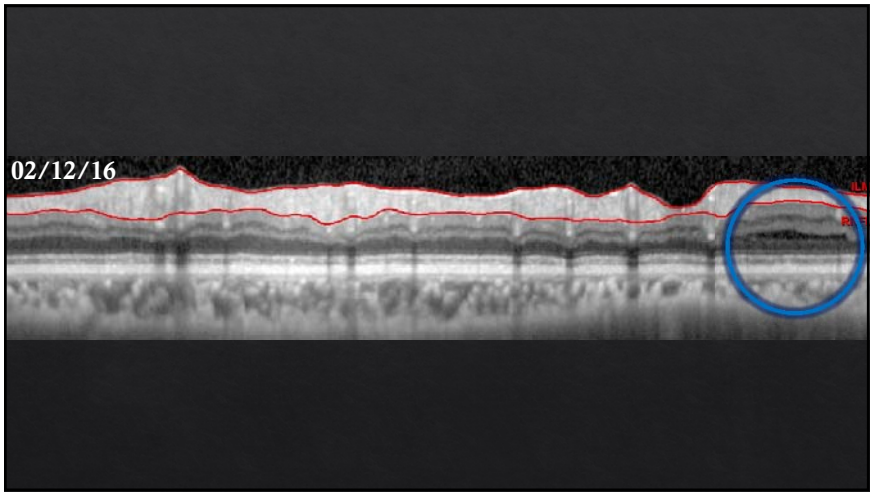
	OD	OS
DVAcc	20/30	20/30
Distance spec Rx	+0.50-0.75x035	+0.75-0.75x135
Anterior Segment	Mild MGD/dry eye	Mild MGD/dry eye
IOP	19mmHg	18mmHg

RNFL OCT OS - 07/28/16

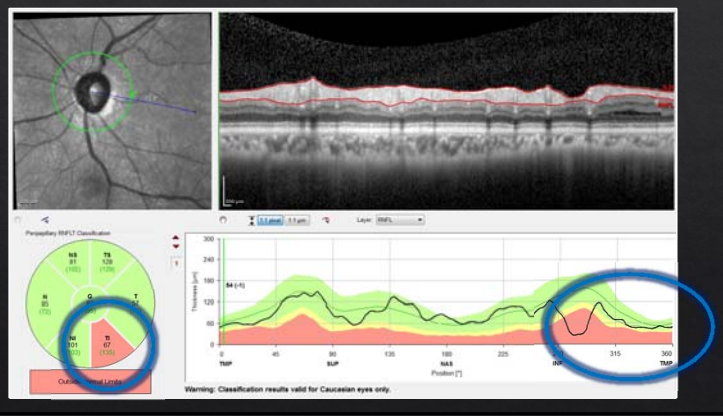


RNFL OCT OS – 02/12/16 (most recent RNFL scan)





RNFL OCT OS – 02/12/16 (most recent RNFL scan)



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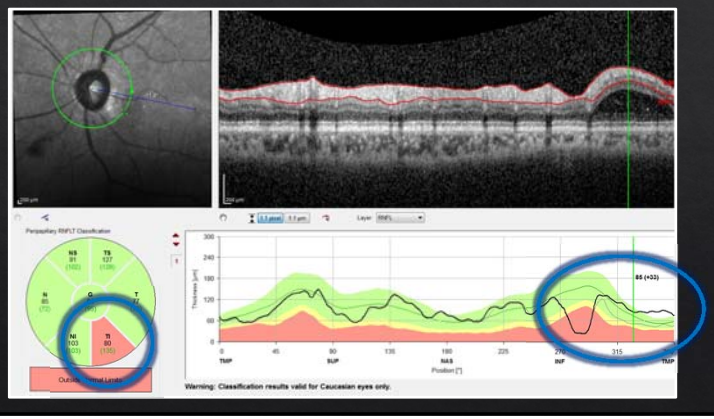

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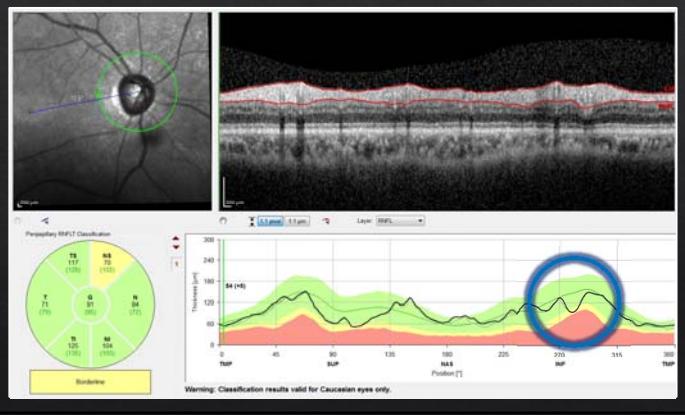
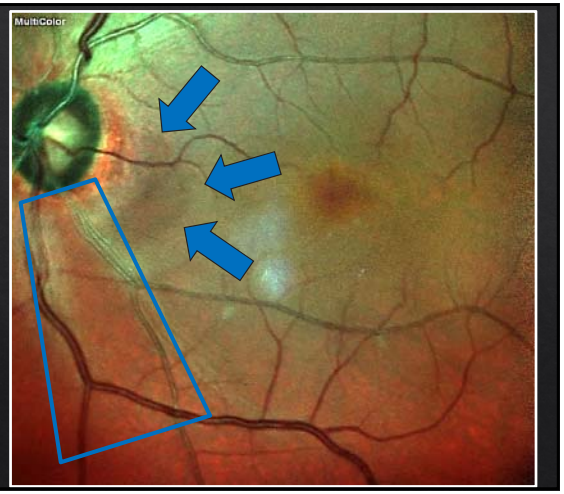


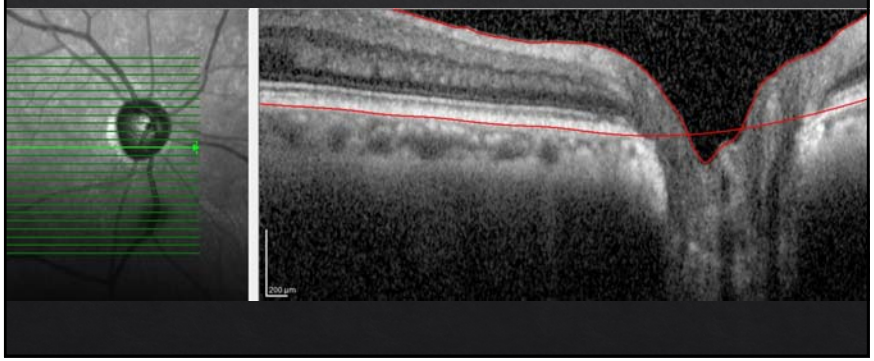
MultiColor OS 07/28/16

MultiColor OS 07/28/16

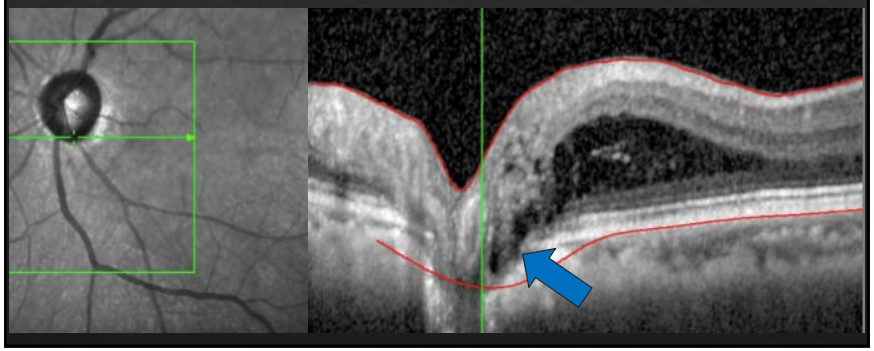


MultiColor OD 07/28/16

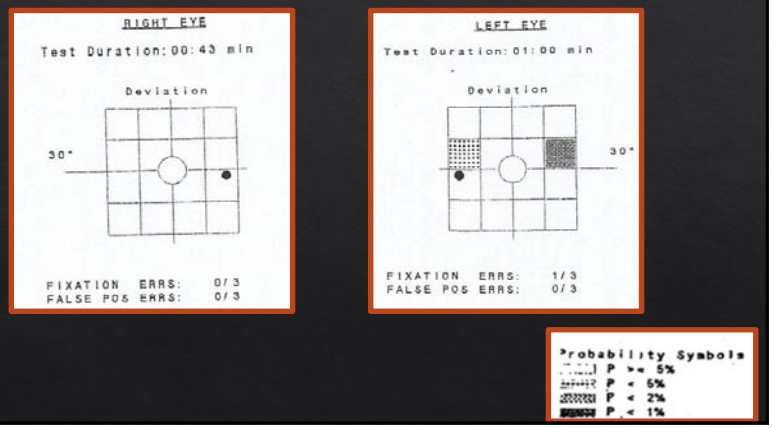
LINE SCAN THROUGH ONH OD 07/28/16



LINE SCAN THROUGH ONH OS 07/28/16

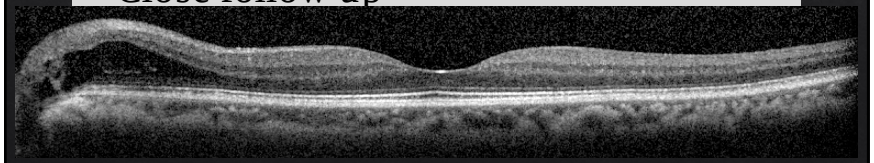


FDT 07/28/26



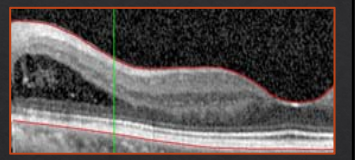
ASSESSMENT / PLAN

- Peripapillary Retinoschisis 2' glaucomatous nerve damage / optic nerve pit
- Consulted retina specialist[1]
- Close follow up



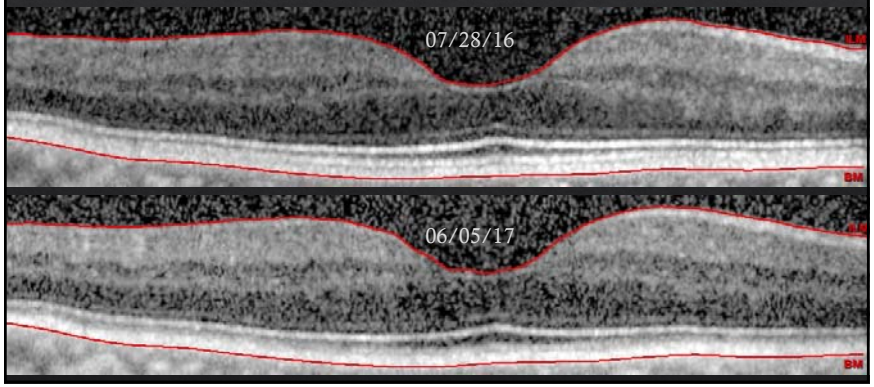
FOLLOW-UP

- Followed over next 9 months q6-10 weeks for DFEs, VFs, and IOP checks

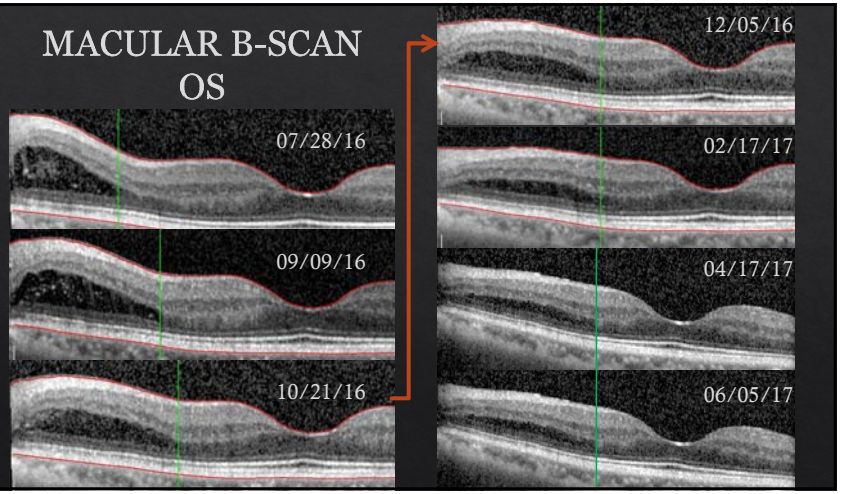


DATE	OD	OS	TIME	TX
09/09/16	20	19	1509	Latanoprost QHS OU
10/21/16	21	17	0812	Latanoprost QHS OU
12/05/16	17	17	1508	Latanoprost QHS OU
02/17/17	15	15	1444	Latanoprost QHS OU
04/17/17	19	17	1417	Latanoprost QHS OU
06/05/17				

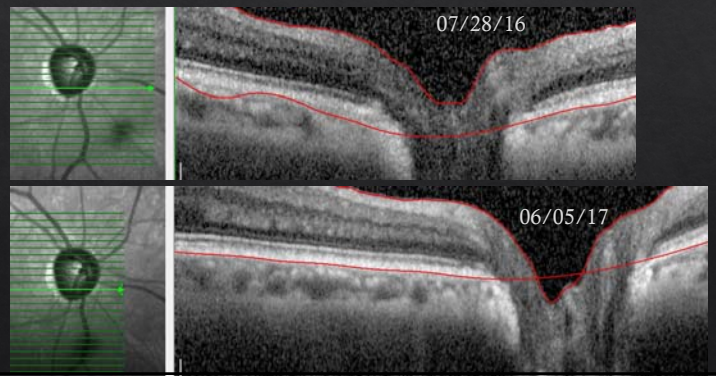
MACULAR B-SCAN OD

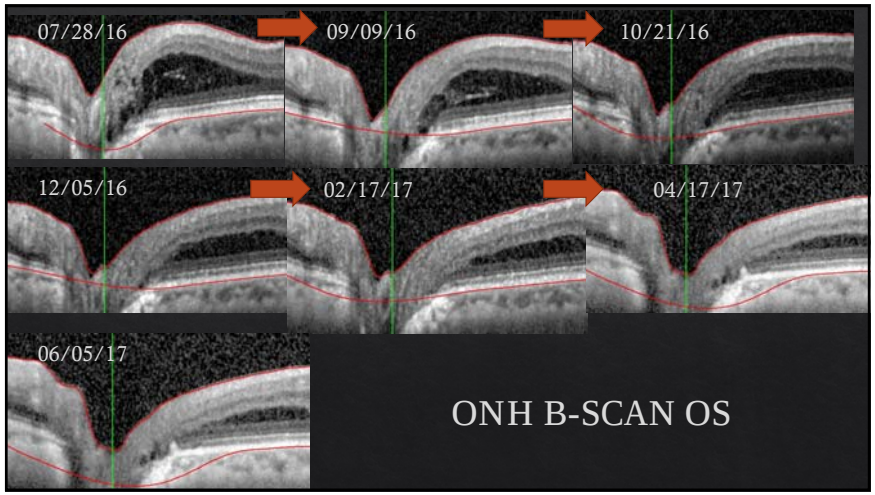


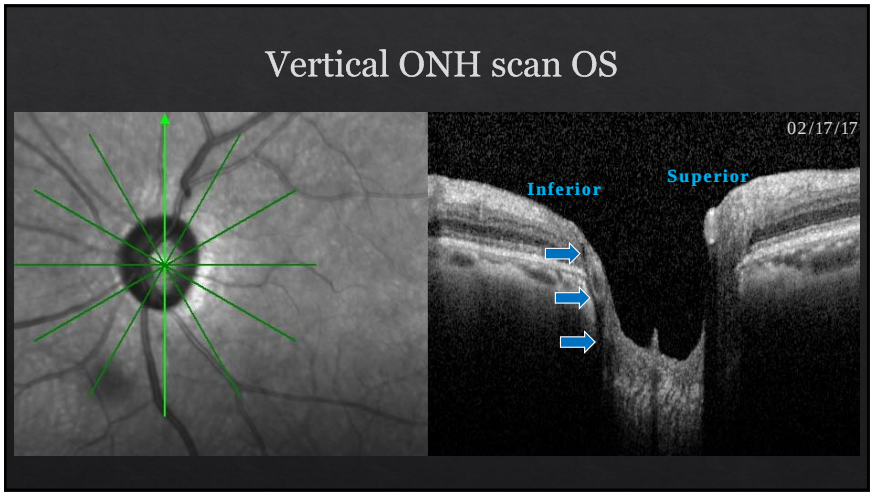
MACULAR B-SCAN OS

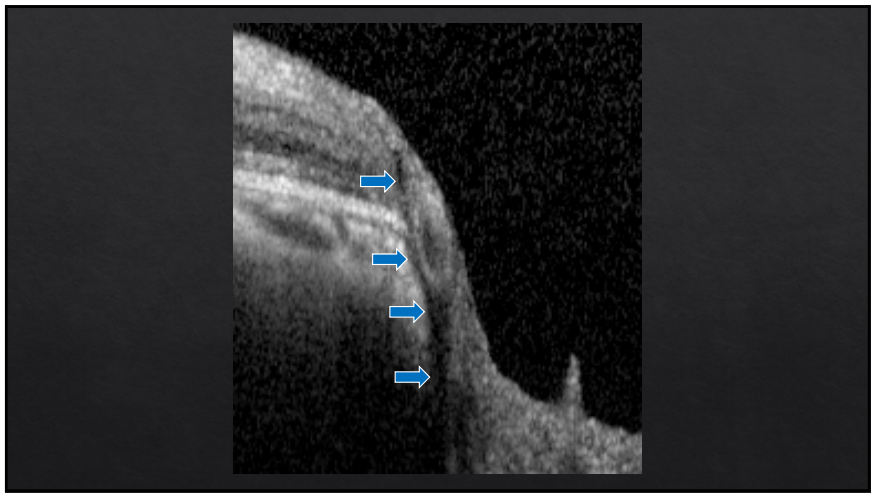


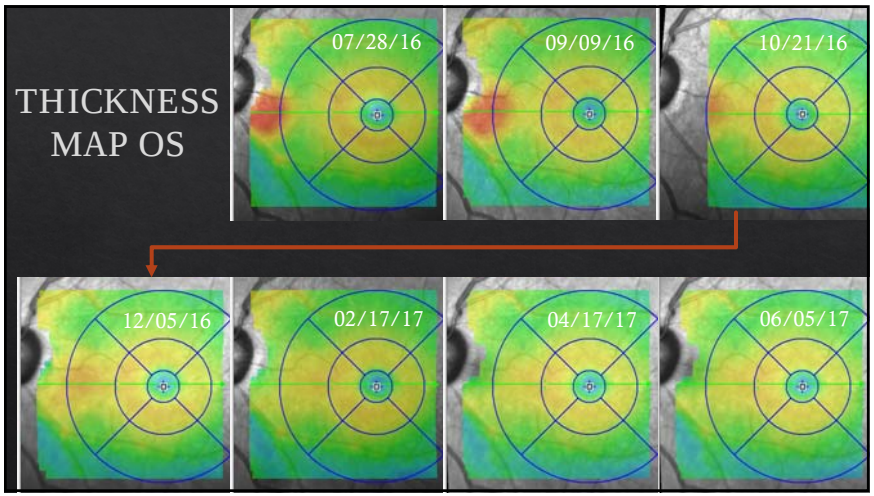
ONH B-SCAN OD




07/28/16
09/09/16
10/21/16
12/05/16
02/17/17
04/17/17
06/05/17
ONH B-SCAN OS

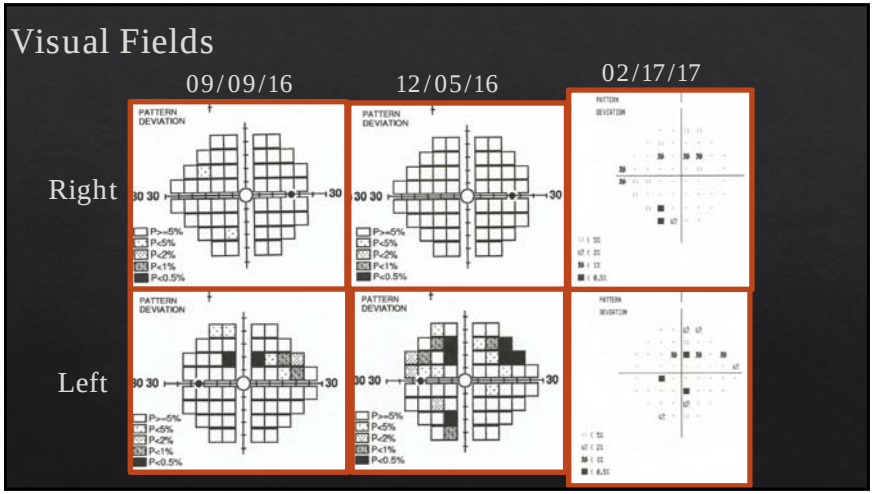

Vertical ONH scan OS
02/17/17
Inferior
Superior



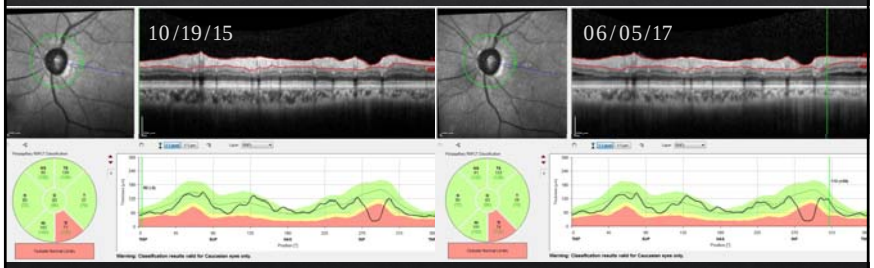

THICKNESS MAP OS
07/28/16
09/09/16
10/21/16
12/05/16
02/17/17
04/17/17
06/05/17

MultiColor OS

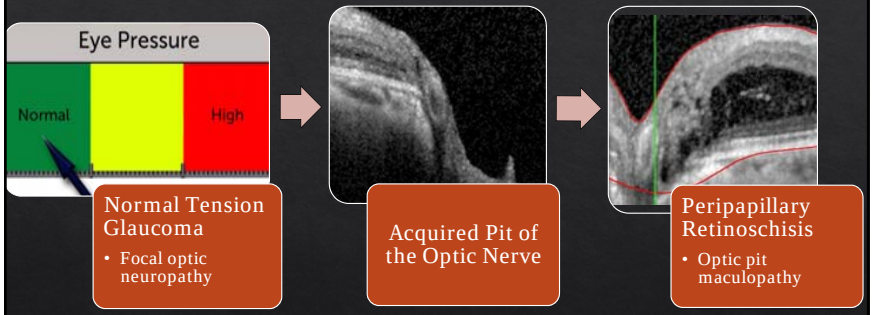
Visual Fields



RNFL OCT OS

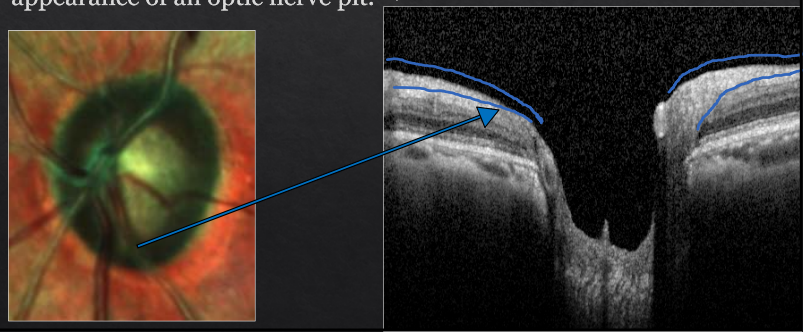


DIAGNOSIS



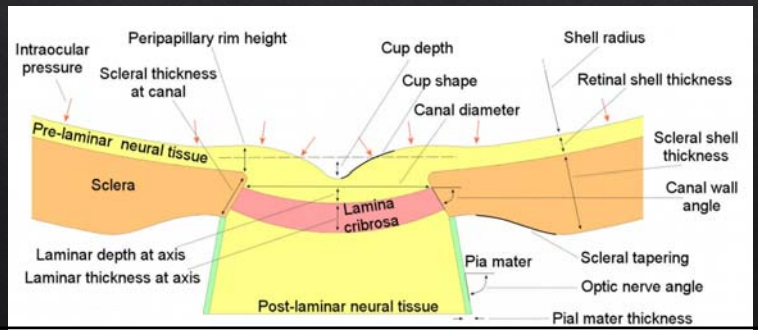
Acquired Pit of the Optic Nerve (APON)

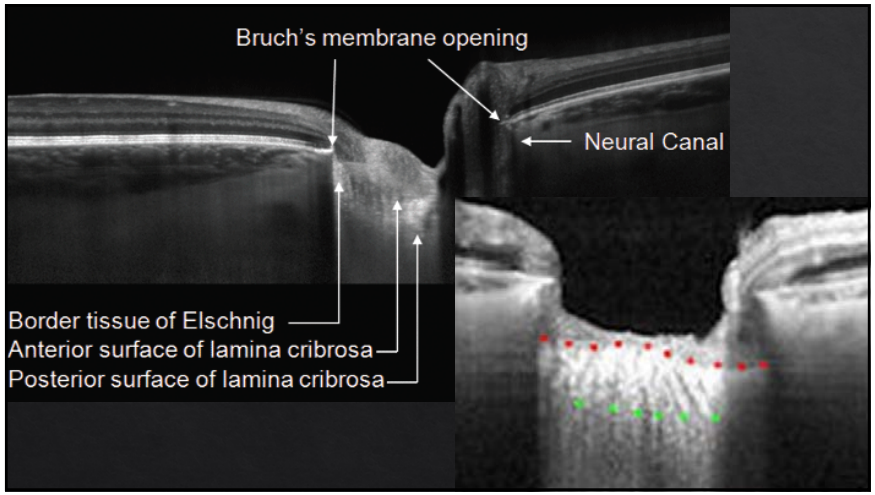
- Focal glaucomatous loss of neural rim tissue associated with pronounced excavation and loss of lamina cribrosa, giving the appearance of an optic nerve pit.[2-4]



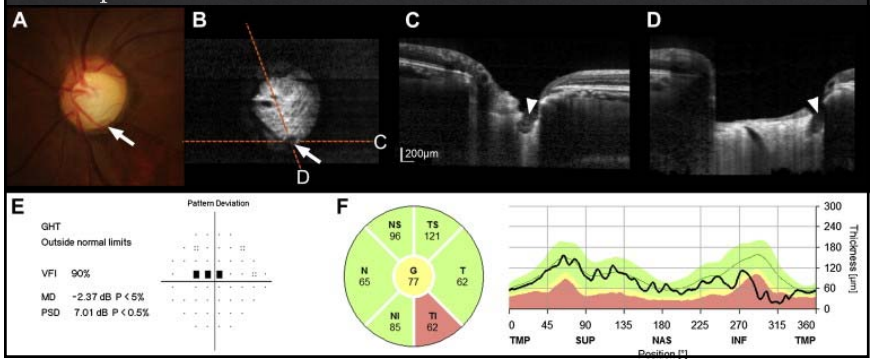
LAMINA CRIBROSA

- A mesh-like section of the sclera that allows optic nerve fibers to pass through it as they move posteriorly. It is made up of a multi-layered network of collagen fibers.[5]

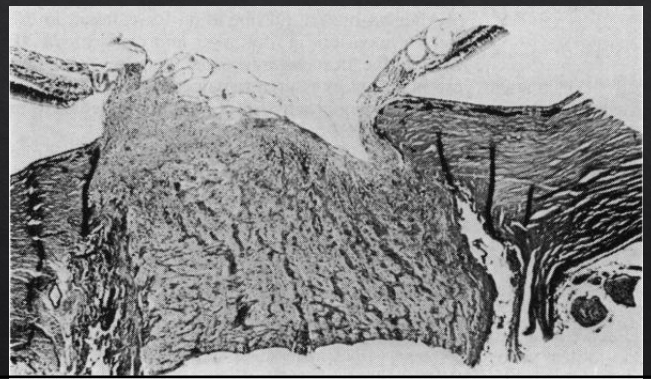




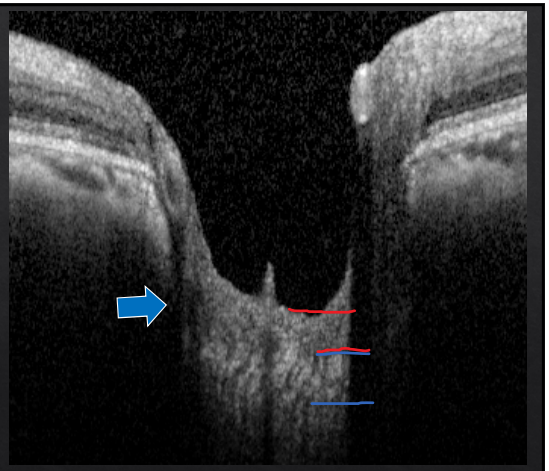
- Choi *et al.* used enhanced depth imaging (EDI) SD-OCT to show that optic pits associated with glaucoma progression (acquired) presented as alteration of the lamina cribrosa or prelaminar tissue or both.[6]



- Damage to the lamina cribrosa represents a focal and mechanical susceptibility to raised intraocular pressure.[6-9]
- More common in the peripheral lamina cribrosa.[6]

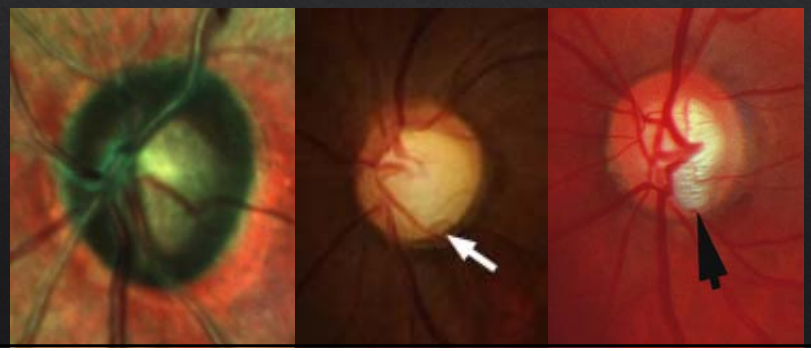


My patient
02/17/17



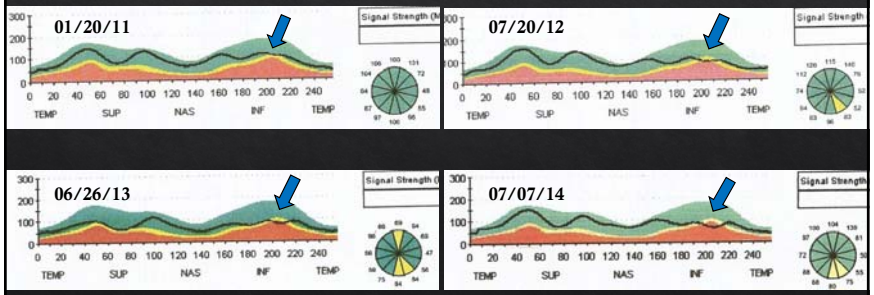
Characteristics

More often located at the inferior optic disc than superior disc.[4,7-11]



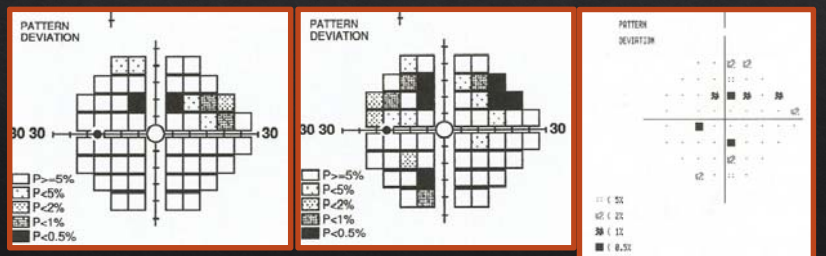
Characteristics

Associated with focal, progressive RNFL loss, which is more episodic than in most glaucomas, with periods of deterioration and stability.[10]



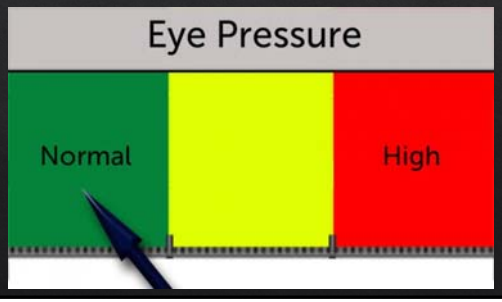
Characteristics

The visual field defect produced by an APON has been described as a deep, steep-margined scotoma, which approaches and sometimes involves fixation.[4,12,13]



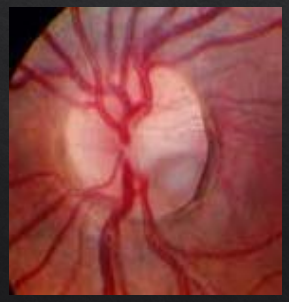
Characteristics

Multiple studies have found that there is an increased prevalence of APON in patients with low-tension glaucoma.[8,14-15]



Similar Optic Nerve Anomalies

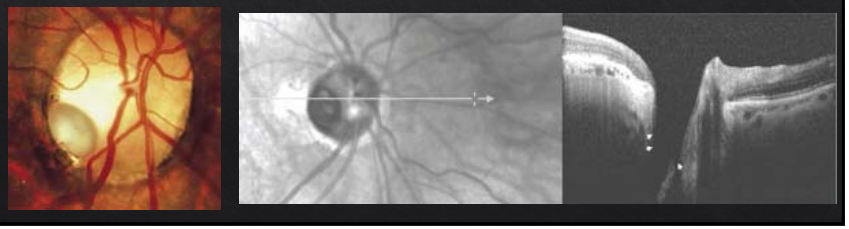
Congenital Optic Nerve Pit



Optic Nerve Coloboma

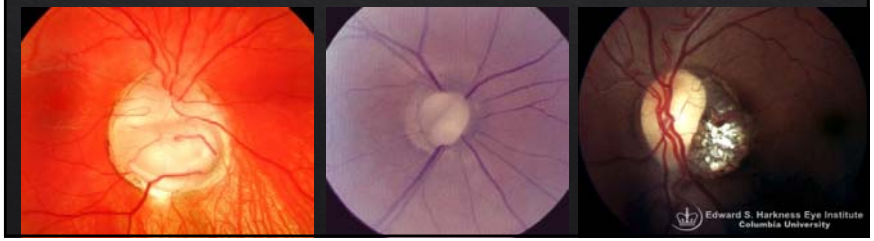
Congenital Pit of the Optic Nerve (CPON)

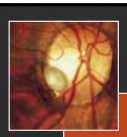
- Congenital optic anomaly of the optic disc caused by incomplete closure of the embryonic fissure.
- Round or oval, gray, white, or yellowish depression in the optic disc. Typically unilateral
- Congenital visual field defects (arcuate or enlarged blind spot)[16,17]



Coloboma

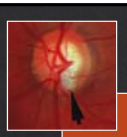
- Congenital eye abnormality also caused by an incomplete closure of the embryonic fissure.
- Typically bilateral with large discs
- Found at the inferior portion of the disc[17]





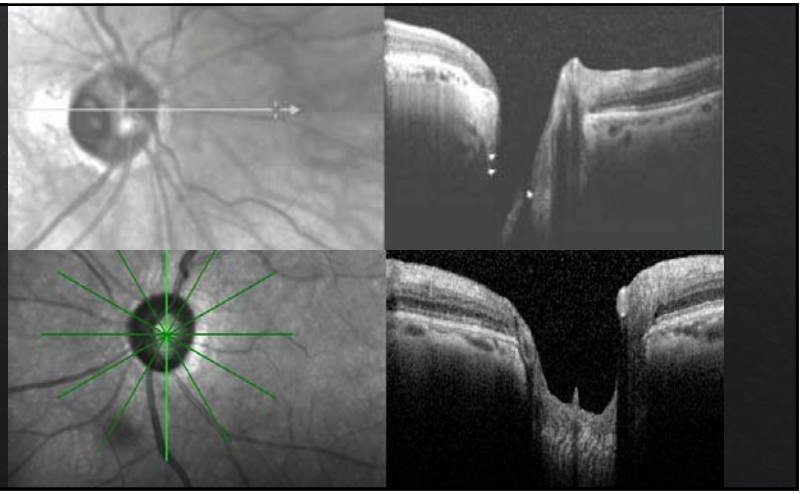
CPON

- Congenital
- Stable, congenital visual field defects / RNFL thinning
- Typically larger in size and at the temporal aspect of the disc[16,17]

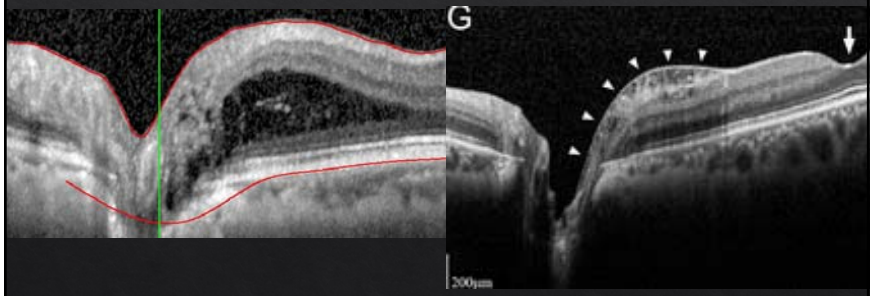


APON

- Acquired
- Progressive visual field defects / RNFL thinning
- Inferior disc

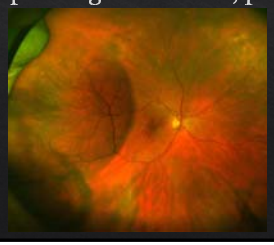


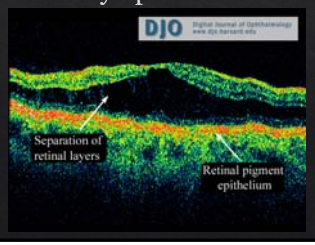
A common complication of both congenital and acquired optic pits is peripapillary retinoschisis.



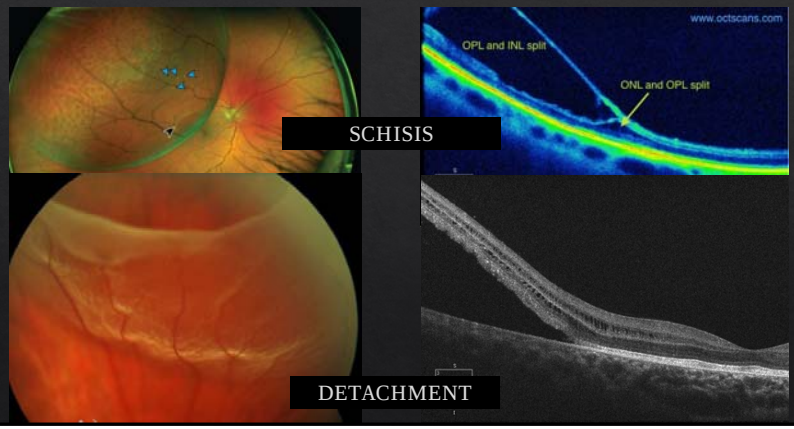
Retinoschisis

- Splitting of the retina's neurosensory layers, usually in the outer plexiform layer
- Can be found anywhere in the retina with the inferior temporal region being most common
- Depending on location, patients can be symptomatic[17]

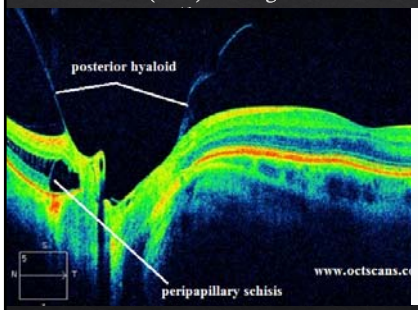


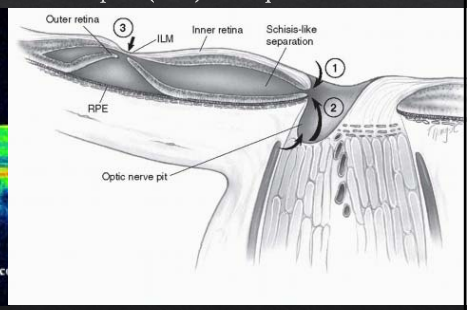


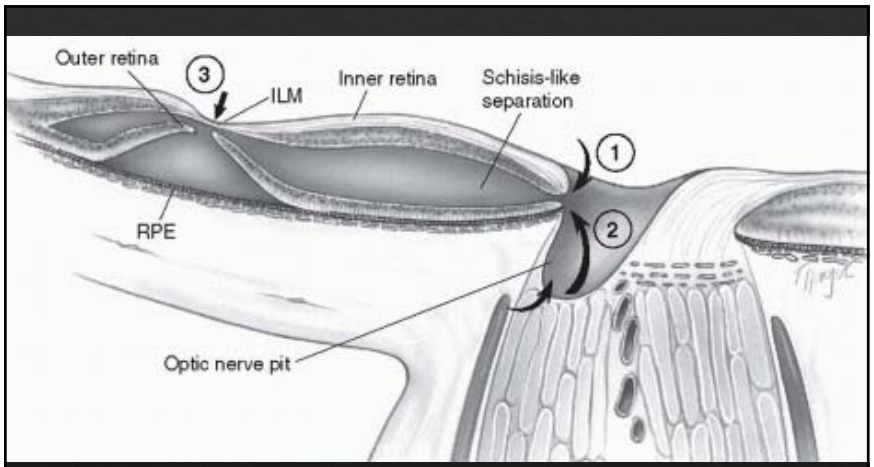
vs. Retinal Detachment

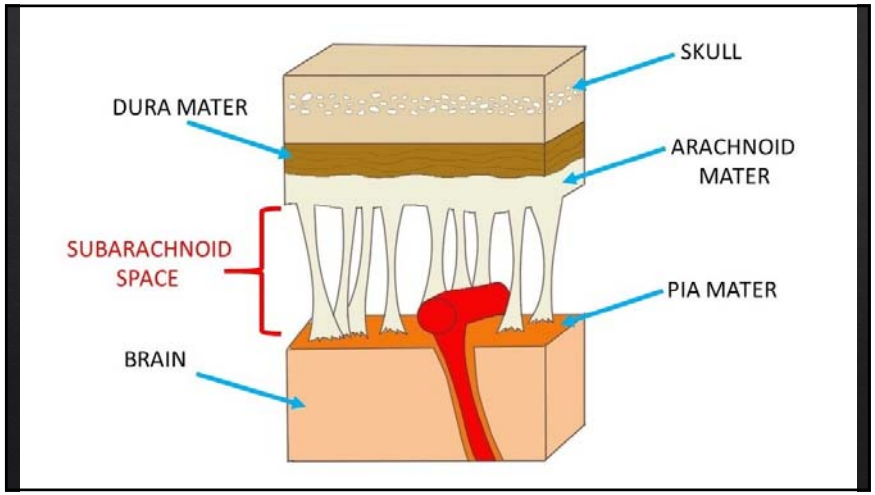


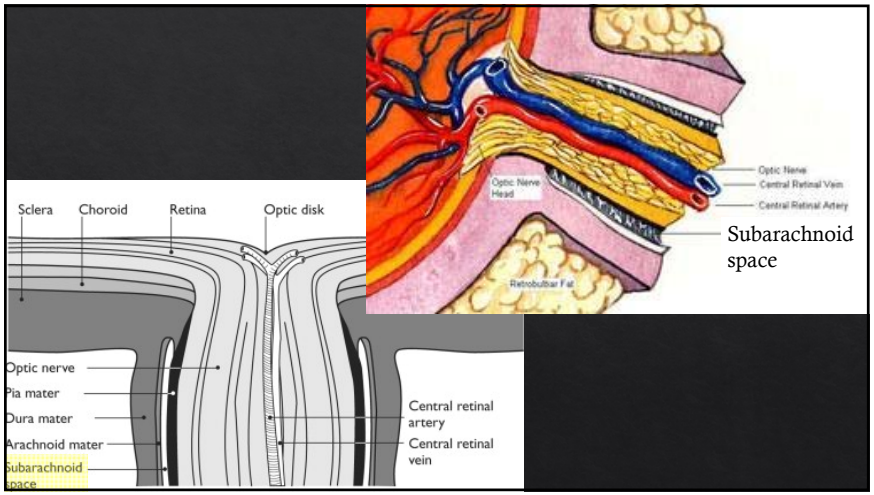
- The pathophysiology of peripapillary retinoschisis is still controversial but two leading theories for the source of the subretinal fluid are cerebrospinal fluid (CSF) coming from the subarachnoid space (SAS) and liquefied

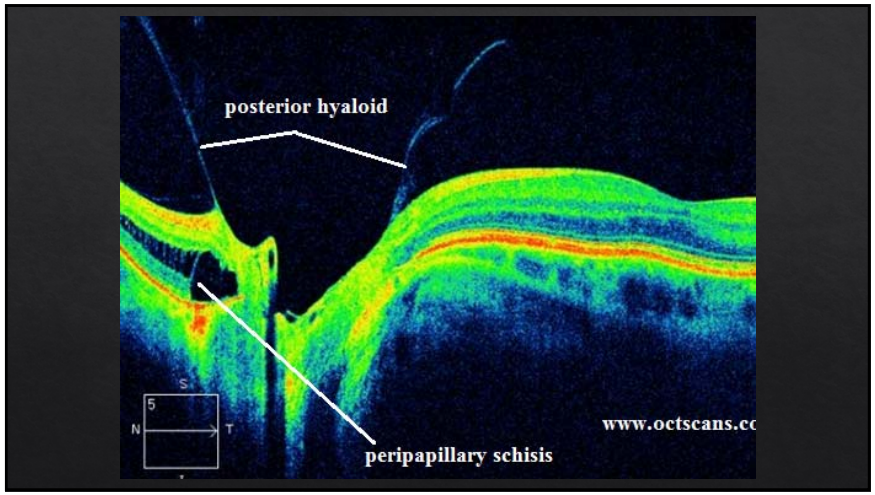


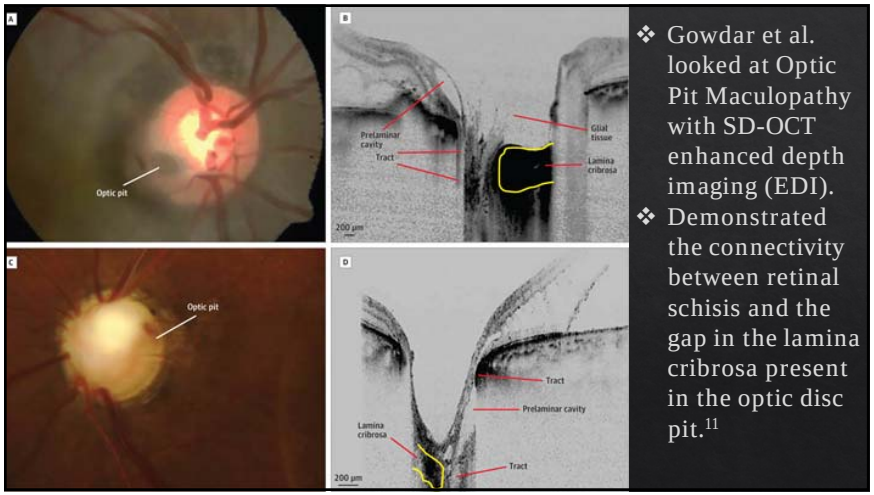




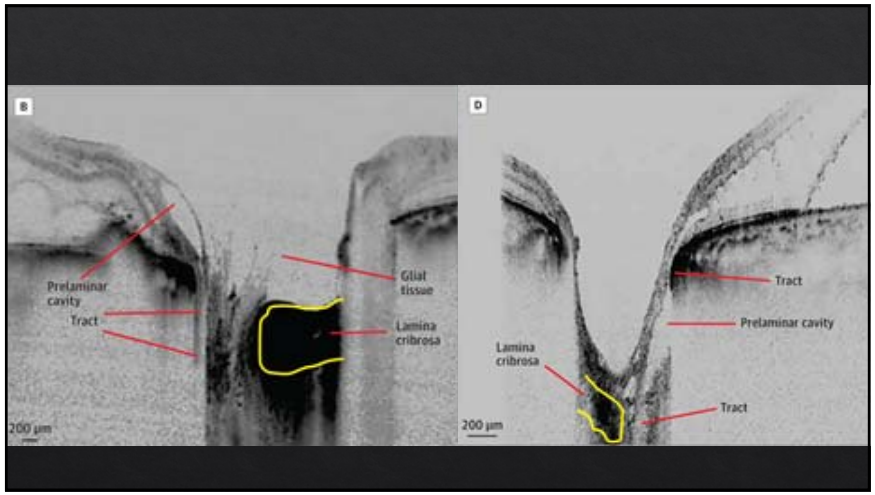


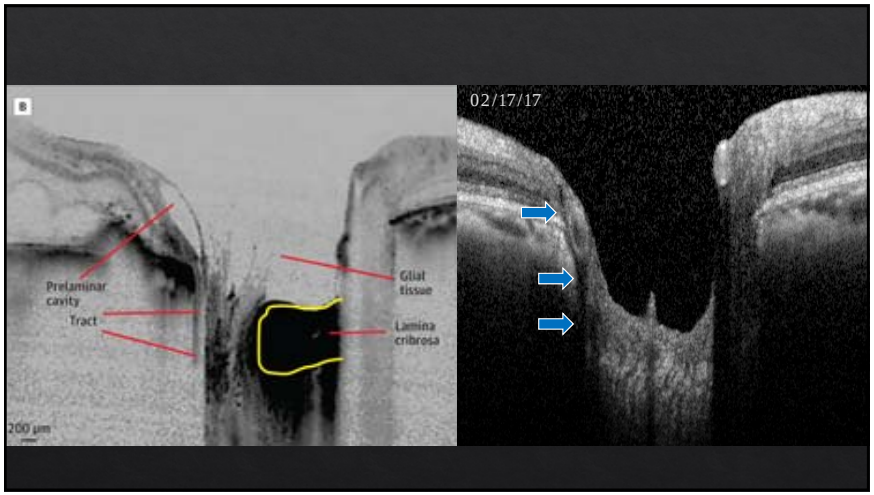






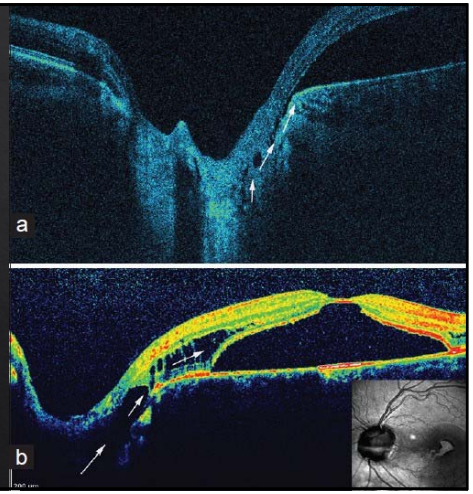
- Gowdar et al. looked at Optic Pit Maculopathy with SD-OCT enhanced depth imaging (EDI).
- Demonstrated the connectivity between retinal schisis and the gap in the lamina cribrosa present in the optic disc pit.[11]

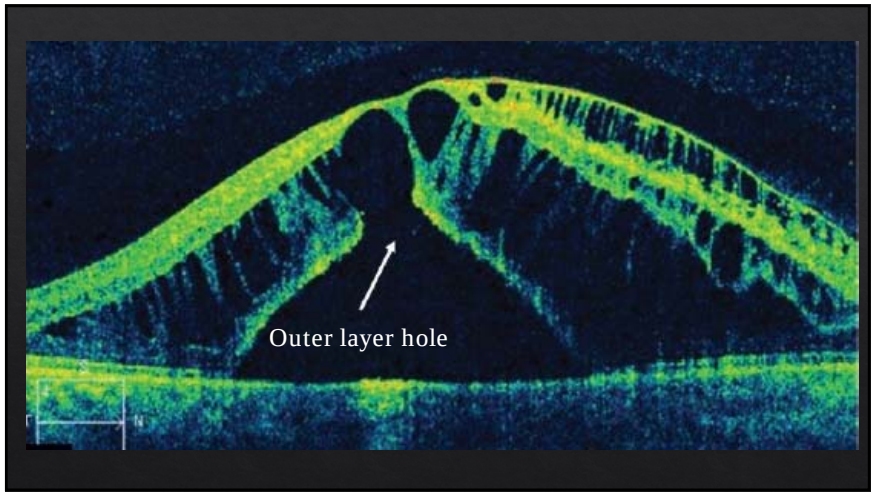




It is unclear the etiology of resolution of peripapillary retinoschisis, though a reduction in IOP has been suggested[8,20]

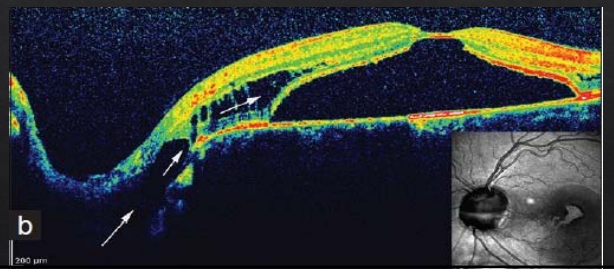
Optic Pit Maculopathy
OPM



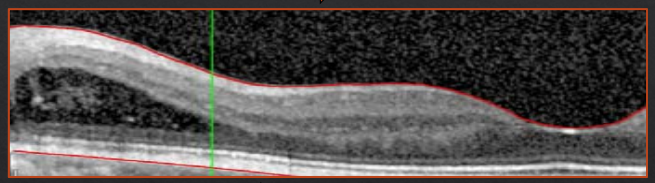


Prognosis

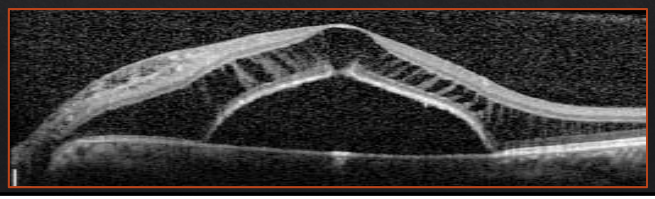
- Optic pit maculopathy is more commonly associated with congenital pitting of the optic nerve. Rates vary from 25-75%.
- Studies have shown that acquired pitting of the optic nerve is more commonly associated with peripapillary retinoschisis that waxes and wanes with a much better visual prognosis.[19]



Peripapillary Retinoschisis ➡ close follow-up

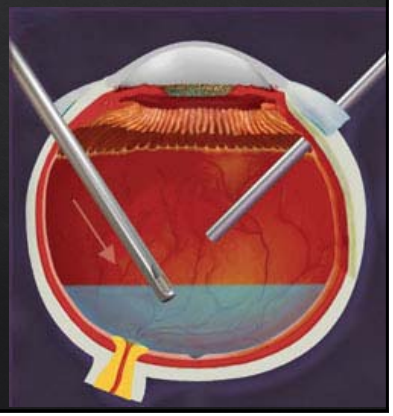


Optic Pit Maculopathy ➡ Treatment



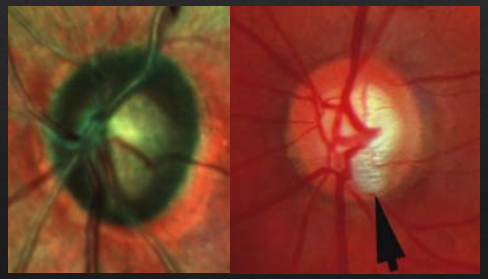
Treatment for OPM

- Primarily PPV w/ gas tamponade
- Blocks passage of fluid through the optic disc pit.
- Invasive surgery w/ risk of further vision loss.
- Visual recovery is a slow and long process.[19]



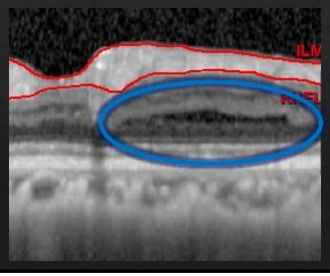
Conclusions

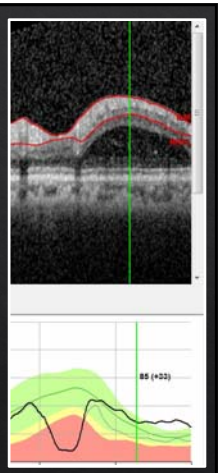
- This case study highlights the importance of optic nerve head evaluation in patients with normal IOP.



Conclusions

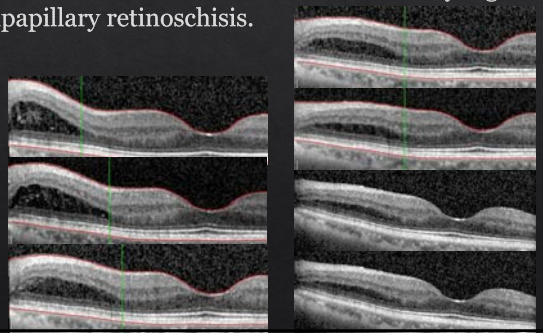
- Watch for peripapillary retinoschisis in patients with focal notching of the optic nerve head rim tissue.
  - Don't confuse with artificial improvement in RNFL thinning





Conclusions

- Close observation warranted for non-visually-significant peripapillary retinoschisis.



Thank You

- Jeffery Hiett, O.D., VA Puget Sound – American Lake Division
- Judith Oh, O.D., VA Puget Sound – American Lake Division

- QUESTIONS?

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Sickle Cell Retinopathy: A Case Report and Review

BY: SAJAL S. PATEL, O.D.

Case History

54yo African American Male

Chief Complaint: distorted vision OS for a few weeks but worsening the past few days, light sensitive, eyes feel sore, floaters OS > OD

Ocular History
- Glaucoma suspect OU

Ophthalmic Meds: artificial tears PRN

Family Ocular History:
- (+) glaucoma: mother

Case History

Medical History
- Major depressive disorder
- Stimulant dependence
- Chronic Lower Back Pain
- Hyperlipidemia
- Chronic PTSD
- Cocaine Dependence in remission
- Substance Abuse

- Pt mentions have sickle cell trait but does not recall being told he has sickle cell disease.

Case History

Medications:
- Acetaminophen
- Atorvastatin
- Bupropion HCL
- Carboxymethylcellulose Oph Sol
- Cholecalciferol (Vit D3)
- Fish oil
- Naproxen

NKDA

Exam Findings

Confrontation testing
- Pupils: PERRL, (-) RAPD OU
- EOMS: full OU
- Visual Field: FTFC OD/OS

BCVA SC:
- OD: 20/25+2
- OS: 20/30 PHNI

Exam Findings

Slit lamp exam
- Lids: clear OU
- Conjunctiva: tr injection OD, gr 1 diffuse injection OS
- Cornea: clear OU
- Angles: 4x4 OU
- Ant Chamber: deep and quiet (-) cells, flare
- Iris: flat and even
- Lens: tr NS OU

Tonometry: GAT
- 20/12 mmHg @ 1425

Exam

Dilated Fundus Exam
- Vitreous:
  - OD: clear
  - OS: hazy view in
- Optic discs: pink rim with distinct margins
  - OD: 0.50/0.50
  - OS: 0.55/0.50
- Vessels: normal course and caliber OU
- Post Pole: clear OU
- Macula: flat and even OU
- Periphery: no holes, breaks, tears OU
  - OD: temporally ghosting of vessels and attenuation
  - OS: 1:00- 3:00 small vitreous hemorrhage, obscuring underlying vasculature

Recent Lab Findings

As of July 2016
- Glucose: 117 (Ref: 71-109)
- Cholesterol: 239 (Ref: <=200)
- LDL-C: 173 (Ref: <=129)
- CBC: wnl

Differential Diagnoses

Intermediate uveitis

Proliferative Diabetic Retinopathy

Syphilis

Tuberculosis

Sarcoidosis

Lyme Disease

Multiple sclerosis

Adult Onset Coats Disease

Retinal artery macroaneurysm

Plan

- Refer to Ophthalmology for retinal consultation
- Order labs
  - Hemoglobin S
  - CBC
  - C reactive protein
  - ACE
  - RPR
  - ANCA
  - Glucose
  - A1C

Lab Results

As of September 2016
- Sickle Cell Prep: positive (Ref: negative)
- CBC
  - Hemoglobin: 13.5 g/dl (Ref: 13-18)
  - HCT: 38.8% (Ref: 41-51)
- C reactive protein: 0.5 mg/dL (Ref: <=3.0)
- RPR : nonreactive
- Glucose: 99 mg/dL (Ref: 71-109)
- C-ANCA: <1:20 (Ref: Neg: <1:20)
- P-ANCA: <1:20 (Ref: Neg: <1:20)
- ANA: negative
- ACE: 80 (Ref: 14-82)
- A1C: 5.9% (Ref: 4.4-6.4)

Sickle Cell Review

Most prevalent amongst African Americans
- 1 in ~ 500 African Americans
- 1 in ~ 1,400 Hispanic Americans

10% incidence in US

Most common inherited blood disorder in the US

Life expectancy is 40-60 years

Sickle Cell Review

Genetic mutation in beta subunit of adult hemoglobin → erythrocyte deformation in low oxygen environment
- Valine replaces glutamic acid
- Sickle hemoglobin can polymerize → sickle shape
- Decreased cell survival
- Vasco-occlusion

Distal tissue ischemia
- Pain crises
- Anemia
- Infections

11% of patients suffer stroke by 18yo

Treatment
- Red blood cell transfusions
- Hydroxyurea
- Allogeneic hematopoietic stem cell transplantation (HSCT)

Types of Sickle Cell

Normal hemoglobin: trait A
- Sickle Cell Trait (AS)
- Hemoglobin C Trait (AC)
- Hemoglobin D Trait (AD)
- Alpha Thalassemia Trait
- Beta Thalassemia Trait

Sickle Cell Disease
- Sickle Cell Anemia (SS)
  - Substitution of valine for glutamic acid
- Sickle Hemoglobin C Disease (SC)
  - Substitution of lysine for glutamic acid
- Sickle Hemoglobin D Disease (SD)
- Hemoglobin C Disease (CC)
- Sickle Beta Thalassemia Disease

SS vs SC

Most common to affect systemically is homozygous SS disease (only 3% proliferative retinopathy)

Genotypes with higher ophthalmic manifestations are heterozygous SC (33%) and Sthal (14%)

SS	SC
• 1 in 500 • Systemic complications more common • More severe anemia • Painful episodes more often • Age specific death rate: 2-3 upto the age of 30 • Overall worse prognosis	• 1 in 833 African Americans • Systemic complications less common • Milder anemia • Painful episodes occur less often • Age specific death rate: less than 1 upto the age of 30 • Overall better prognosis

Pathophysiology

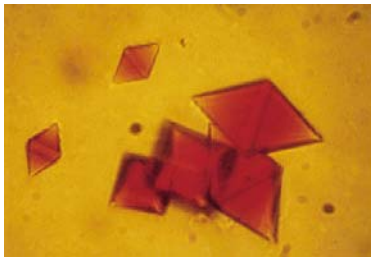
Paradox of SC disease: exact mechanism is unknown

Thought that HbC exemplifies characteristics of HbS pathology but is still milder than sickle cell anemia. The following have equal/higher prevalence:
- Retinopathy
- Osteonecrosis
- Acute chest syndrome

Suspected that higher Hct, increased cell density and greater blood viscosity in HbSC accounts for higher likelihood of retinopathy
- Sickle cell anemia, peripheral retinal microvasculature are completely occluded early so further retinal damage can not occur
- In HbSC, high circulatory competence so retinal circulation still occurs causing continuous retinal ischemia resulting in development of proliferative lesions.

Laboratory Diagnosis

Distinctive feature: intracellular HbC crystals



Laboratory Diagnosis

To determine the relative levels of HbS and HbC
- Hb electrophoresis
- Isoelectric focusing
- HPLC
- Capillary electrophoresis

Normal – slightly elevated leukocyte count

Platelet count is lower than in sickle cell anemia

Symptoms

Eye pain

Redness

Decreased VA

Floaters/flashes

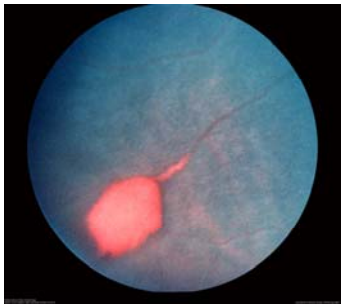
Peripheral vision loss

Asymptomatic

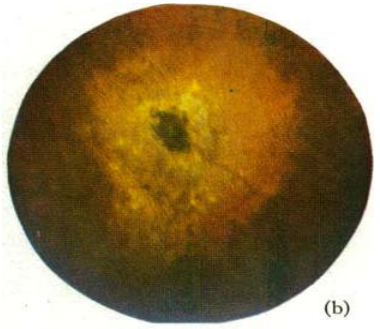
Signs

Nonproliferative retinopathy
- Venous tortuosity
- Silver wiring of arterioles
- Salmon patches
- Macular depression sign
- Macular arteriolar occlusion
- Acute CRAO
- Retinal Vein Occlusion
- Angioid streaks

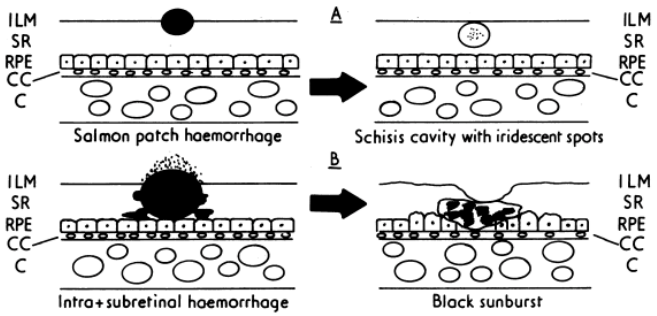
Salmon Patch Hemorrhage



Black Sunburst



Schematic



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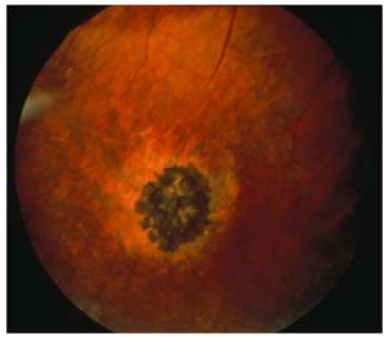


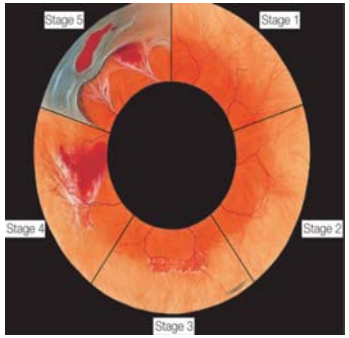
Signs

Proliferative retinopathy

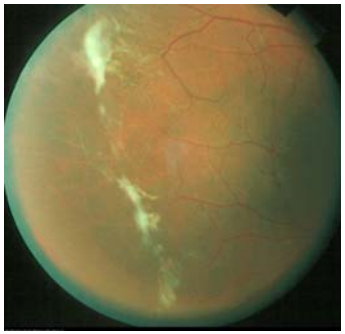
The 5 Stages

- 1. peripheral arterial occlusions
- 2. peripheral arteriovenous anastomoses
- 3. neovascular and fibrous proliferations
- 4. vitreous hemorrhage
- 5. retinal detachment

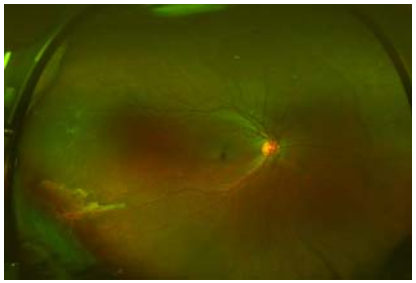
Stages



Sea Fan Neovascularization



Example 1



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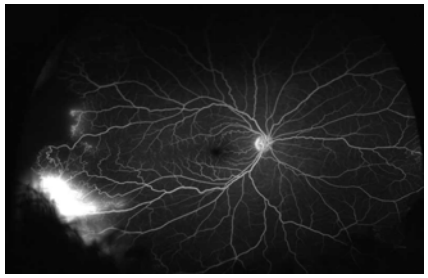
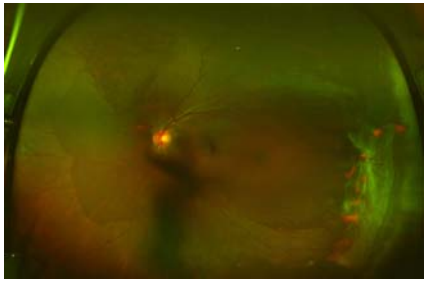


Example 2



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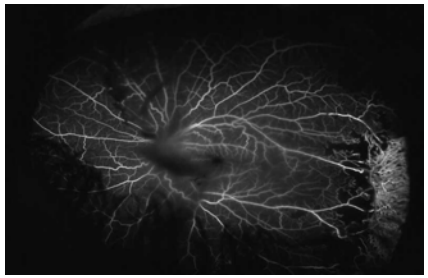
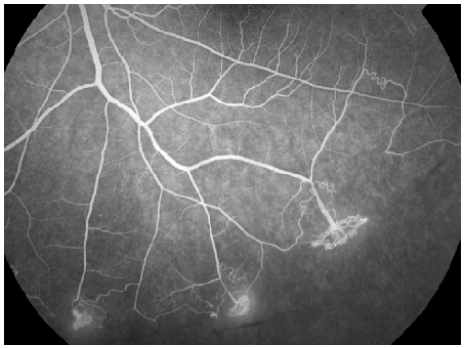


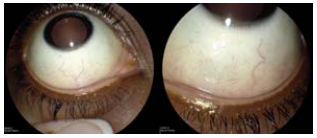
FA

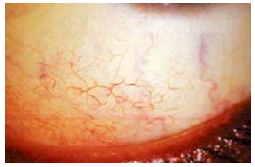


Anterior Segment Findings

Conjunctival sign
- "comma" sign
- Linear capillary segments that are comma shaped
- Inferior bulbar conjunctiva

Iris: circumscribed area of ischemic atrophy
- Uncommon



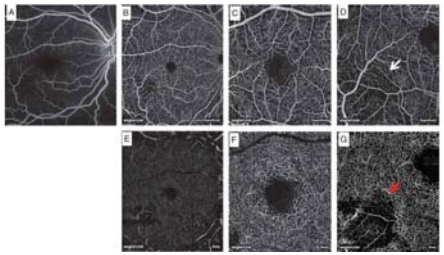


Sickle Cell Maculopathy

Hairpin venular loops

Foveal avascular zone irregularities

Microanerysmal dots



Additional Differential Diagnoses

Proliferative Diabetic Retinopathy

Talc Retinopathy

Eales Disease

Retinopathy of Prematurity

Familial Exudative Vitreoretinopathy

Treatment

No prevention treatment exists.

Scatter laser photocoagulation
- Decrease VEGF stimulus
- Decrease overall oxygen requirement

Pars plana vitrectomy for persistent vitreous hemorrhage / retinal detachment

Anti-VEGF agents

Management

Retinal specialist examinations recommended in patients beginning at 10yo.

Nonproliferative
- Monitor q6months

Regular follow up care with PCP and/or hematologist

Avoid treating large areas of peripheral non perfusion in one session → anterior segment ischemia

Back to Our Case…

Pt has stage 4 PSR OS

Ophthalmology findings
- PRP OS 10/12/16, 10/28/16, 12/12/16 – vitreous hemorrhage has improved
- PRP OD 11/11/16

As of 02/01/17…

Pt diagnosed with DM2, taking insulin. Hospitalized 1/19/17 for new onset diabetes (BS 800s, A1C 9.0)
- No diabetic retinopathy and no CSME noted upon examination.
- Vitreous hemorrhage OS has nearly resolved

Patient Update as of 5/10/17

History: persistent flashes OS, not changing.
- A1C lowered to 7.5
- Taken off insulin

Findings:
- BCVA: stable 20/25 OD/OS
- IOP wnl
- No sickle cell maculopathy
- Periphery OD: ghosting and attentuation. Temp PRP
- Periphery OS: heme almost resolved, some vascular abnormality temporal to NV OS, PRP

Plan: PRP OS in 1 month.

HbC Prevalence

HbC common in malarious areas of West Africa
- HbC associated with 29% reduction in risk of clinical malaria in HbC heterozygotes and 93% reduction in HbC homozygotes
- Long term in absence of malaria control, HbC will replace HbS in Central West Africa

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THANK YOU!

Optical Coherence Tomography Findings in Acute and Chronic Branch Retinal Artery Occlusions in a Single Patient

Benjamin Jager, OD
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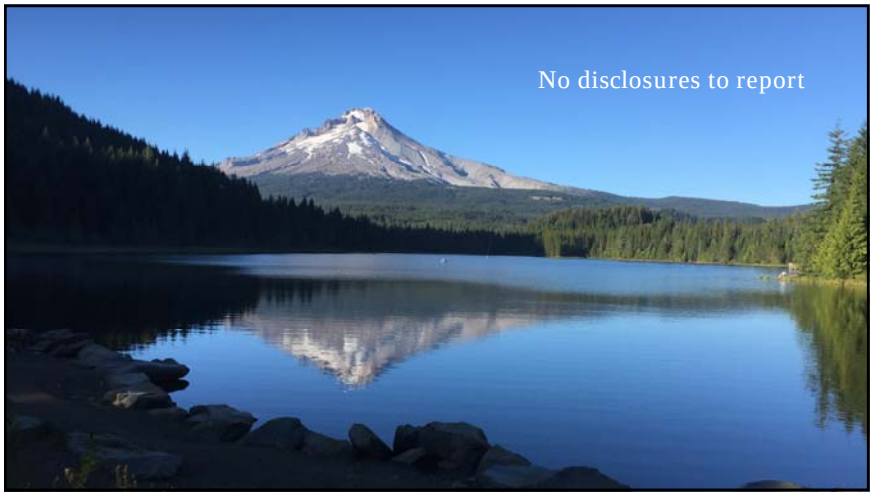
June 10, 2017



LEARNING OBJECTIVES

By the end of this presentation, attendees will be able to. . .

1. Understand the presentation of retinal anatomy on optical coherence tomography
2. Distinguish an adequate quality optical coherence tomography scan from one that is poor
3. Recognize the signs of an acute retinal artery occlusion on optical coherence tomography
4. Recognize the signs of a longstanding retinal artery occlusion on optical coherence tomography

CASE REPORT

BS, 69 Year Old Caucasian Male

"It's like someone put a piece of gray tape in my vision"

DIFFERENTIAL DIAGNOSIS

Giant Cell Arteritis
Non-Arteritic Anterior Ischemic Optic Neuropathy
Pre-Retinal Hemorrhage
Retinal Artery Occlusion
Retinal Detachment
Retinal Vein Occlusion
Vitreous Hemorrhage

PERTINENT MEDICAL HISTORY

Systemic Disorders

- Type II DM x 2013
- HTN
- History of CVA x 04/26/14
- Coronary Artery Disease
- History of MI x ~1995
- Carotid Artery Stenosis
- Peripheral Vascular Disease
- Hyperlipidemia
- Depression
- Erectile Dysfunction

Medications

- Insulin (Aspart/Glargine)
- Metformin
- Lisinopril
- Plavix
- Baby Aspirin
- Atorvastatin
- Sildenafil
- Tiotropium Inhaler
- Albuterol
- Nitroglycerin

OCULAR HISTORY

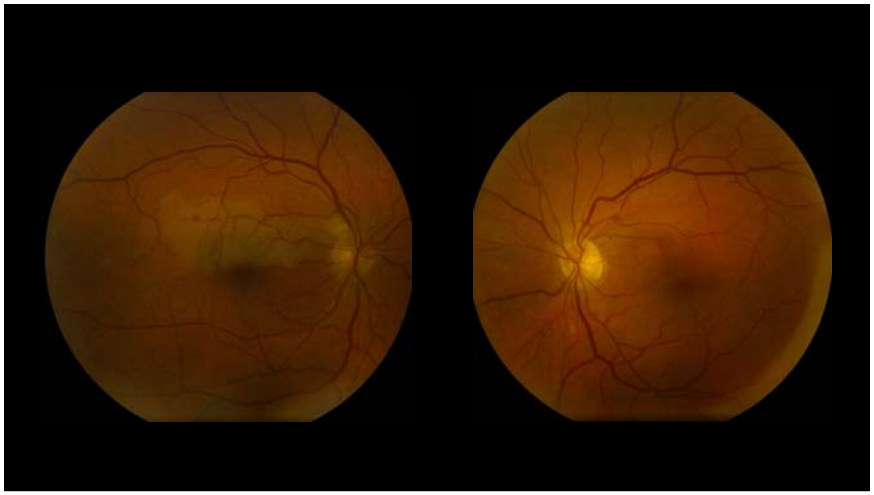
- Mild Non-Proliferative Diabetic Retinopathy OU
- History of Intra-Arterial Plaque OS
- Mild Hypertensive Retinopathy OU
- Mild Cataract OU
- Mild Macular Mottling OU
- Chorioretinal Scarring OS presumed secondary to Trauma
- History of Migraine Headache with Visual Aura
- Refractor Error and Presbyopia

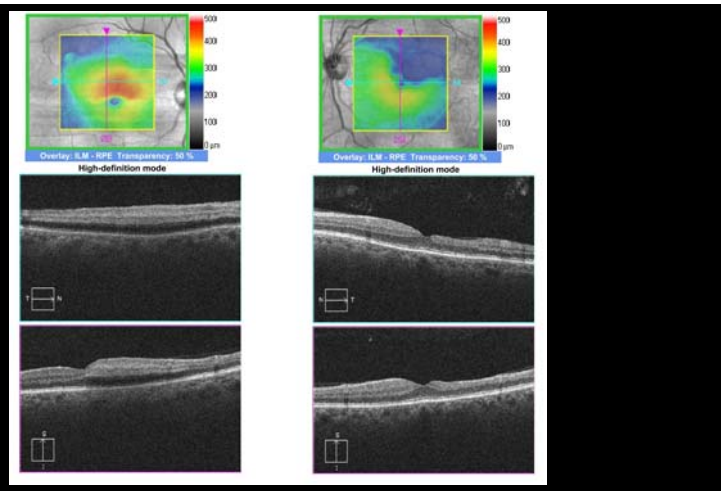
CLINICAL FINDINGS

	OD	OS
BCVA	20/30-1	20/20-1
EOMs	Full and unrestricted	Full and unrestricted
Pupils	No APD	No APD
Confrontation VF	Scotoma just inferior to fixation	FTFC
Anterior Segment	WNL	WNL
IOP	14	16

CLINICAL FINDINGS

	OD	OS
Lens	1+ NSC, vacuoles	1+ NSC, vacuoles
Vitreous	Syneresis	Syneresis
Optic Nerve	Healthy; (-) edema/pallor	Healthy; (-) edema/pallor
Posterior Pole	Area of retinal whitening extending from ONH temporally through macula just superior to fovea with 2 intra-arterial plaques in affected area	Mild macular mottling, Hollenhorst plaque at a bifurcation of superotemporal arcade
Periphery	WNL	CR scarring temporal





RADIOLOGICAL & LAB STUDIES

A1c
- 06/23/14 – 9.6%
- 08/10/16 – 8.3%

CBC
- 06/23/14 – WNL
- 08/10/16 – WNL

Carotid Duplex
- 09/29/15 – 80 to 99% right ICA stenosis; ~60% left ICA stenosis
- 08/05/16 – 80 to 99% right ICA stenosis; ~70% left ICA stenosis

ASSESSMENT	PLAN
Acute Branch Retinal Artery Occlusion OD; Suspected Old Branch Retinal Artery Occlusion OS	Monitor in 1-2 months with repeat DFE. Notify patient's PCP of findings, recommend restarting blood thinner and repeating carotid duplex

THE AFTERMATH

SUBSEQUENT FOLLOW-UP

DATE	SUMMARY	RESULTS
07/29/16	Initial optometry visit	Monitor in 1-2 months. PCP restarted Plavix and reordered vascular consult
08/19/16	Vascular follow-up	Recommended right carotid endarterectomy
09/01/16	Right carotid endarterectomy	No intraoperative complications
09/02/16 – 09/11/16	ICU neurovascular exams	Diagnosed with perioperative right MCA infarct. Recommended OT, PT, speech therapy

SUBSEQUENT FOLLOW-UP

DATE	SUMMARY	RESULTS
10/14/16	Optometry follow-up	Stable vision, resolved retinal edema, (-) neovascularization
11/22/16	---	Discharged from inpatient care
02/03/17	Optometry follow-up w/ planned visual field	Stable findings. No field due to staffing shortage
03/17/17	Vascular follow-up	Suspected residual common carotid stenosis. No additional surgery recommended at this point, repeat carotid imaging in 3-4 months

Retinal Artery Occlusions

A Brief Review

ETIOLOGY

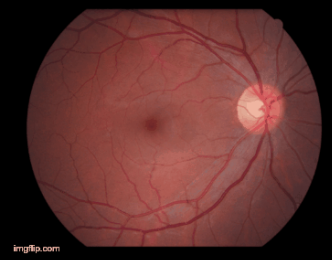
Blockage of central retinal artery or tributary retinal artery

↓

Retinal hypoxia in affected area

↓

Retinal ischemia



CLINICAL CHARACTERISTICS

Central Retinal Artery

- Retinal whitening and edema
- Cherry red spot
- APD
- Emboli
- Arteriole boxcarring

Branch Retinal Artery

- Retinal whitening and edema
- Narrowed retinal artery
- Emboli
- Arteriole boxcarring
- Cotton wool spots

DIAGNOSIS AND MANAGEMENT

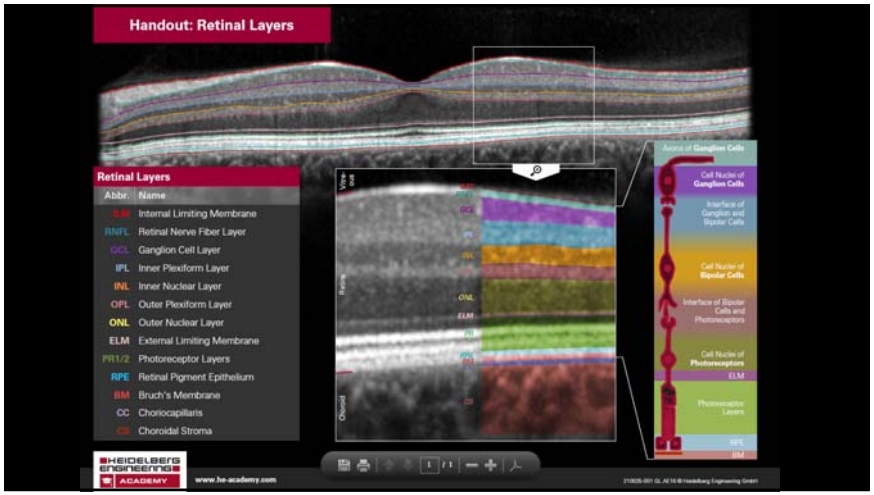
Diagnosis

- Often clinical, but may be subtle
- Optical coherence tomography helpful

Management

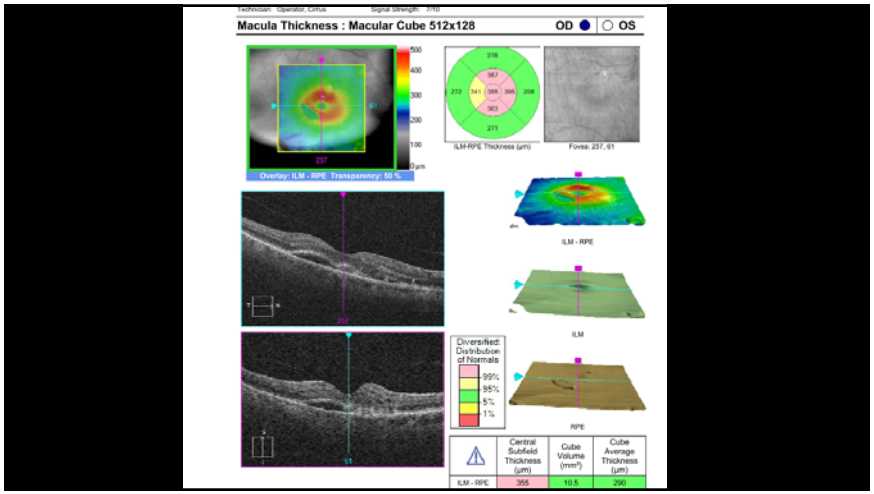
- No treatment has proven effective
- Investigate underlying cause
- Monitor for and treat any neovascularization

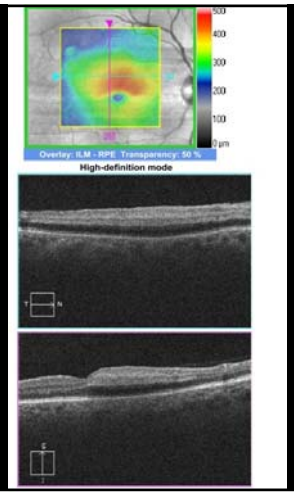
OPTICAL COHERENCE TOMOGRAPHY 101



ANALYZING SCAN QUALITY

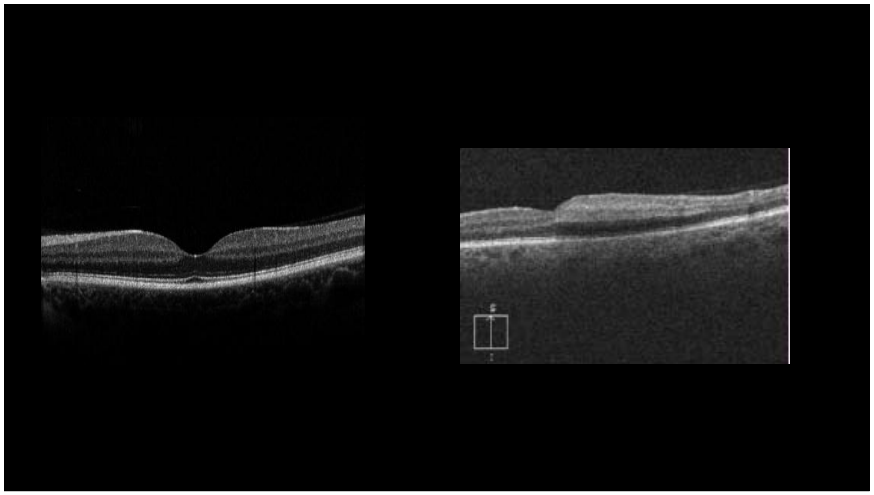
- Develop a systematic approach
- Does your OCT have a scan quality indicator?
- Scan area
- Look for artifacts
- Centration

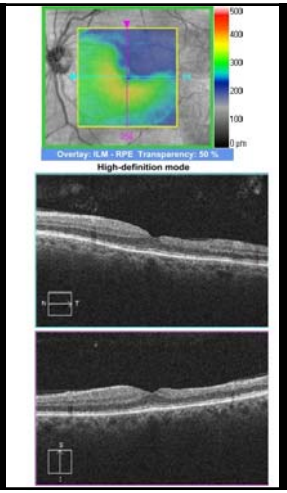




Acute BRAO

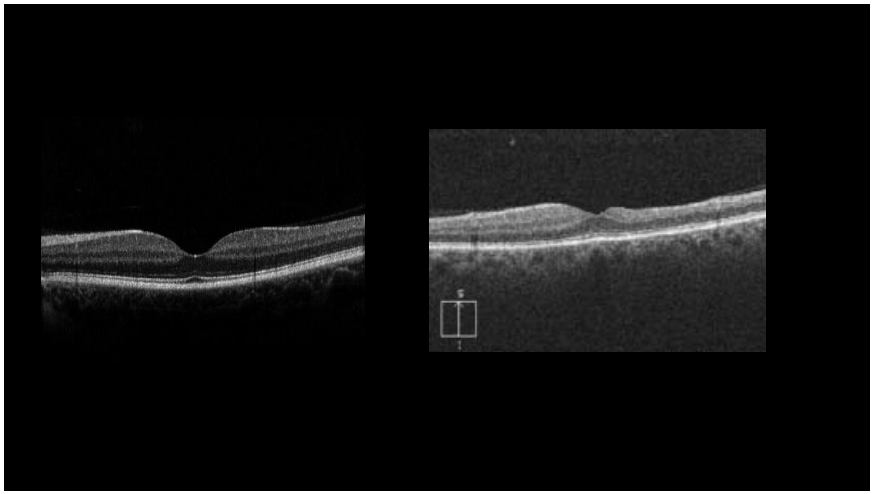
- Edematous inner retina
- Outer retinal shadowing

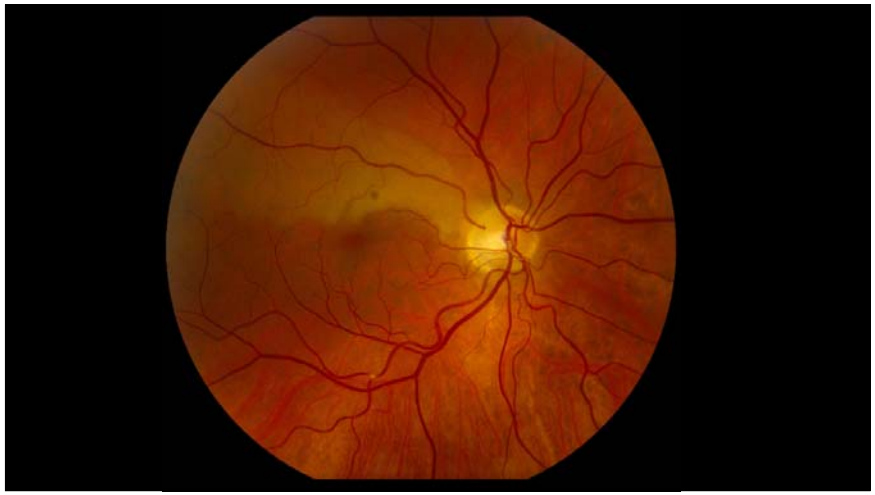


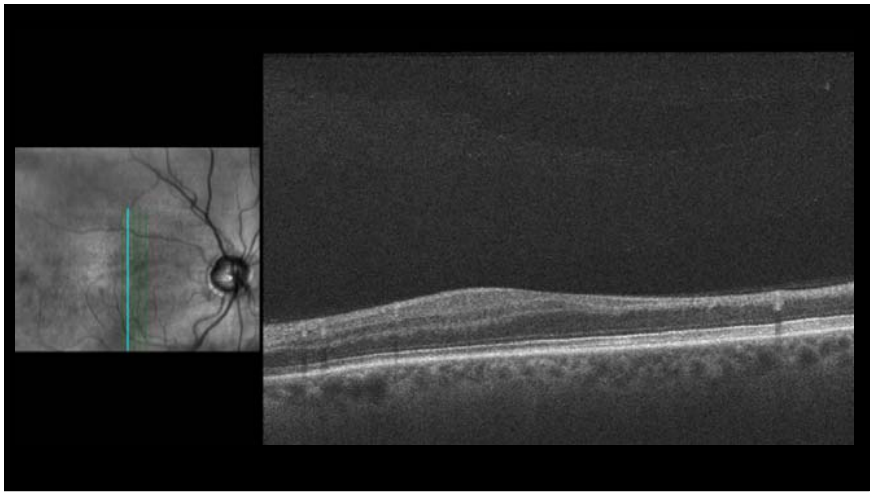


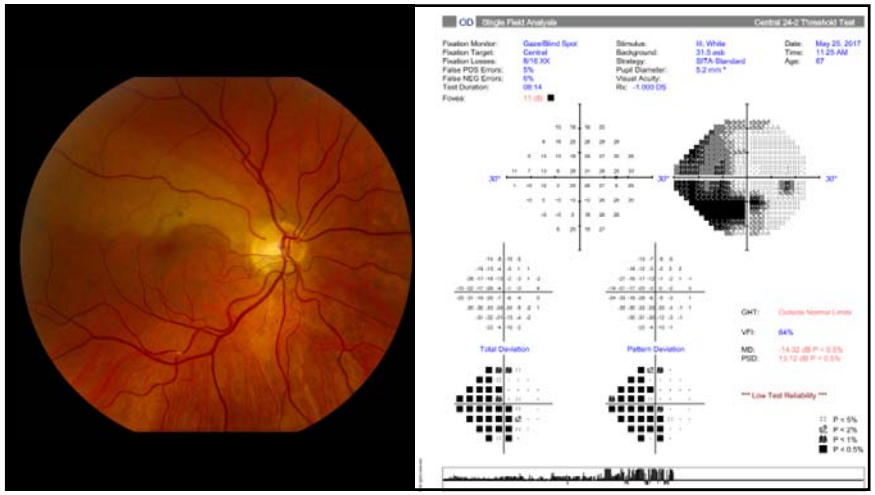
Old BRAO

- Atrophic inner retina
- Intact outer retina









SUMMARY OF OCT FINDINGS

Acute BRAO	→	Old BRAO
Inner Retinal Edema		Inner retinal atrophy
Shadowing of outer retina		Intact outer retina

CLINICAL PEARLS

- Develop a systematic approach to OCT analysis
- Use the tools that are available to aid in your diagnosis
  - Presentation of retinal artery occlusions can be subtle!
  - OCT findings in the acute phase are nearly pathognomonic
- Get your patient's other health care providers involved

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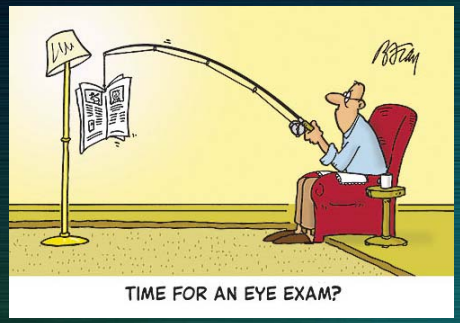
QUESTIONS?



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