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We have eliminated all paper session evaluation forms. Please be sure to complete your electronic session evaluations online when you login to request your CE letter for each course you attended! Your feedback is important to us as our Conference Advisory Board considers content and speakers for future meetings to provide you with the best education possible.



Practical Approach to Uveitis Management

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Statement of Financial Disclosures

- Alcon-Consultant, Speaker Bureau
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Case Study

- › 23yo Caucasian Female
- › **CC:** painful left eye
- › **HPI:** started 3 days ago
Sudden, onset with no improvement with ATs
(+)photophobia
(+)mild blurry vision

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History

- › **Medical history:** unremarkable
- › **Medications:** birth control
- › **Ocular history:** unremarkable
LEE 2 years ago
- › **Social history:** (-)EtOH, nonsmoker

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Entrance Testing

- › **BCVA:** 20/20 OD; 20/25 OS
- › **Pupils:** PERRLA, (-)APD
- › **Confrontational VF:** full
- › **EOMs:** Full & Smooth OU
- › **IOP:** 12mmHG OD, 13mmHG OS

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Slit Lamp Findings

	OD	OS
Cornea	WNL	WNL
A/C	Quiet	1+ cells
Iris	Blue, (-)TIDs, (-)synechiae	Blue, (-)TIDs, (-)synechiae
Lens	Clear	Clear



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Posterior Pole Findings

	OD	OS
Vitreous	Quiet-no cells	Quiet-no cells
Optic nerve	Pink, healthy rim 0.3/0.3 C/D ratio	Pink, healthy rim 0.3/0.3 C/D ratio
Macula	Flat & clear	Flat & clear
Retina	No breaks/tears	No breaks/tears



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What's your diagnosis?

Acute, anterior, non-granulomatous uveitis OS



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Uveitis

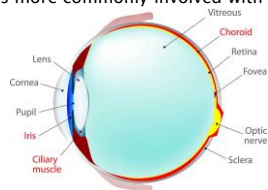
- ▶ Common cause of red & painful eye
- ▶ 10–20% of blindness in US
- ▶ 3rd leading cause of blindness in developed countries
- ▶ Important associations with systemic disease



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What is Uveitis?

- ▶ Inflammation of the Uveal Tract
- ▶ Highly vascularized tissues more commonly involved with inflammation
- ▶ Uveal Tract:
 - Iris
 - Ciliary Body
 - Choroid



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Inflammation Basics

- ▶ "Inflammation" (Latin, *inflammatio*, to set on fire)
- ▶ Inflammatory response Events:
 - (1) vascular changes: blood flow increases and fluid and plasma proteins leak into the inflamed tissue
 - (2) cellular infiltration: leukocytes adhere to vascular endothelium and migrate through the endothelial layer to gain access to surrounding tissue
 - (3) chemotaxis: leukocytes follow a chemical gradient to the site of insult and unleash potent killing mechanisms.



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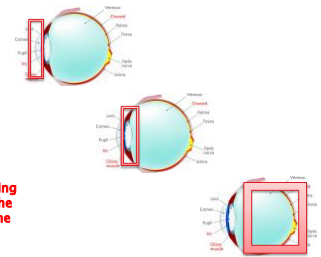
Uveitis Classification

1. Anatomical=What structures are involved?
2. Time-Course of Disease =When did it occur?
3. Pathophysiology=What type of inflammation?
4. Laterality=Which eye?

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Anatomical Classification

- ▶ Anterior Uveitis (AU)
- ▶ Intermediate Uveitis (IU)
- ▶ Posterior Uveitis (PU)
- ▶ Panuveitis



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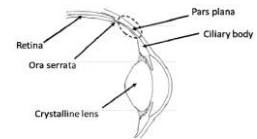
Anterior Uveitis

- ▶ Inflammation of anterior segment (iris & anterior ciliary body)
- ▶ Inflammation from breakdown of blood-aqueous barrier
- ▶ Most common form (50–60% of all cases)
- ▶ 30–50% have underlying systemic etiology

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Intermediate Uveitis

- ▶ Inflammation of the pars plana, vitreous, and peripheral retina
- ▶ "pars planitis" =subset category & only used if there is an absence of associated infection (i.e. idiopathic)
- ▶ Etiology
 - 70% Idiopathic
 - 22% Sarcoidosis
 - 8% Multiple Sclerosis
 - 1% Lyme Disease



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Posterior Uveitis

- ▶ Inflammation of the posterior pole (retina, choroid, optic nerve)
- ▶ Etiology
 - 25% toxoplasmosis
 - 13% Idiopathic
 - 12% CMV
 - 8% Sarcoidosis
 - 8% SLE

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Panuveitis

- ▶ Inflammation of anterior chamber, vitreous, retina, and/or choroid
- ▶ Etiology
 - 22% Idiopathic
 - 14% Sarcoidosis
 - 12% Bechet's
 - 5% Syphilis
 - 2% TB

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Clinical Classification

- › Acute: sudden onset <3mo
- › Chronic: >3months
 - Can be a "white eye"
- › Recurrent: repeated episodes separated by period of inactivity without treatment for 3+mo



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Pathophysiology Classification

- › Non-granulomatous
- › Granulomatous



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Non-granulomatous

- › Inflammation causes protein & WBC to enter aqueous humor
- › Inflammatory cells: lymphocytes, plasma cells, macrophages
- › More common in non-infectious etiologies
- › Can be acute or chronic
- › Predilection for iris & ciliary body



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Granulomatous

- › Inflammatory cells: macrophages, epithelial cells, multinuclear giant cells
- › Usually infectious, toxic, autoimmune, or neoplastic etiologies
- › Predilection for posterior segment
- › Mutton fat KPs, Koeppe/Busacca nodules, Vitreous precipitates



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Helpful Documentation

- › Acute non-granulomatous anterior bilateral uveitis
- › Chronic non-granulomatous anterior uveitis OD
- › Acute, granulomatous bilateral panuveitis



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Etiology Detective!

Key to determining the cause of uveitis:

- › 1) Accurate & thorough history
- › 2) Perform a thorough ophthalmic exam



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What is the Etiology?

- Lab tests are NOT a substitute for history & exam
- However lab tests are crucial for proper management
- History first → then laboratory testing/tissue biopsy may confirm etiology spectrum

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Exam=History

- › Past Medical History
 - Medication history
 - History of autoimmune disorders
 - Prior ocular surgery or trauma
- › Complete Review of Systems
- › Family History
- › Social History: smoking, travel, occupation, drug use

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Exam

- › Visual Acuity
- › Pupillary function
- › Motility, confrontation fields
- › IOP
- › Slit Lamp
- › Dilated Fundus Exam

Goal=Determine infectious vs non-infectious etiology

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Common Symptoms

- › Eye pain or ache
- › Photophobia
- › H/x of recurrent red eyes
- › Recent ocular trauma
- › Injection
- › Lacrimation
- › Decreased vision
- › Floaters

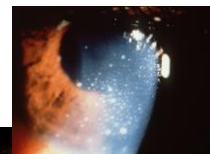
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Anterior Segment

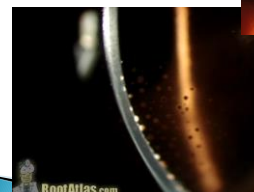
- › Conjunctiva
 - circumcorneal injection
 - conjunctival nodules/granulomas
- › Cornea
 - Epithelium dendrites
 - Stroma edema
 - Endothelium: keratic precipitates (KPs)
 - Size: small, large, non-granulomatous, granulomatous
 - Color: white, pigmented
 - Distribution: Arlt's triangle, diffuse

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New KPs=white with smooth borders



Chronic KPs=pigmented with irregular borders



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Anterior Segment

- ▶ Anterior Chamber
 - Depth–Shallow? Angle Closure?
 - Cells–Hypopyon? Hyphema? Pigmented cells?
 - Flare
 - Structural Changes–rubeosis?



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Anterior Segment

- ▶ Anterior Chamber: cells & flare
- ▶ Grading Cells (SUN)

Grade	Cells In Field
0	<1
0.5+	1–5
1+	6–15
2+	16–25
3+	26–50
4+	>50

Field Size 1 mm X 1 mm slit beam



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Anterior Segment

- ▶ Grading Flare (SUN)

Grade	Description
0	None
1+	Faint
2+	Moderate (iris & lens details clear)
3+	Marked (iris & lens details hazy)
4+	Intense (fibrin or plastic aqueous)

- ▶ Indicative of chronic inflammation
- ▶ Flare possibly predictive of adverse vision impairment



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Anterior Segment

- ▶ Iris
 - Miosis
 - Atrophy
 - Sectoral–VZV/HSV
 - Color–heterochromia?
 - Look before dilated
 - Contour



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Anterior Segment

- Nodules
 - Koeppe nodule=pupillary margin
 - Both granulomatous & non-granulomatous
 - Busacca nodules=on surface
 - Granulomatous
 - Nodules resolved when inflammation resolves
- Synechia
 - PAS (Peripheral Anterior Synechia)=irido–corneal adhesion
 - PS (Posterior Synechia)=iris–lens adhesion
 - Document location (quadrant/clock hour)



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Intraocular Pressure

- ▶ Variable
- ▶ Usually low in acute phase b/c of hypotony of CB
- ▶ Usually Elevated in chronic phase b/c of TM saturation with inflammatory material
- ▶ Herpetic Etiology: trabeculitis affects outflow→ elevated IOP



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Posterior Pole Findings

- ▶ Vitreous=cells vs. spillover
- ▶ Macula-Atrophy? CME? Thinning?
- ▶ Vessels-sheathing? Vascular occlusion?
- ▶ Retina-whitening? Edema? Inflammation?
- ▶ Choroid-old or active scars?
- ▶ Optic nerve- edema? NVD?
- ▶ Peripheral -RD? neovascularization? Exudates?

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Non-Ocular Clinical Clues

- ▶ Cutaneous
 - Vesicular or dermatomal rash? HSV/VZV
 - Erythema chronicum migrans? Lyme borreliosis
 - Psoriasis



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Non-Ocular Clinical Clues

- ▶ Glandular Inflammation=Sarcoidosis?
 - Dacryoadenitis
 - Parotitis
 - Heerfordt's Syndrome



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Laterality

- ▶ Unilateral Cases: HSV, VZV, CMV, Toxos
 - IOP elevation (Herpetic or toxoplasmosis)
 - Sectoral iris atrophy-VZV, HSV
 - Corneal scarring/edema-VZV, HSV
- ▶ Not always helpful
 - HLAB27 & scleritis often unilateral

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Common Complications

- ▶ Cataracts: from chronic inflammation or use of corticosteroids
 - Inflammation causes breakdown of protein in the crystalline lens
- ▶ Band Keratopathy
 - Inflammation causes alkalosis that favors precipitation of calcium & phosphate
- ▶ Glaucoma
 - Inflammation can cause pigment & WBC to clog the TM
- ▶ Cystoid Macular Edema
 - Inflammation causes breakdown of blood-retinal barrier → increased vascular permeability

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Suspect Systemic Disease if...

- ▶ Mutton Fat KPs
- ▶ Chronic & Recurrent
- ▶ Bilateral or Alternating
- ▶ Unresponsive to treatment

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Uveitis Etiology

- › Traumatic - will often resolve on own
- › Idiopathic - most common
- › Non-Infectious="auto-immune"
- › Infectious



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Non-Infectious Etiology

- › Seronegative spondyloarthropathies = 50% cases
- › Sarcoidosis
- › Systemic Lupus Erythematosus
- › Juvenile Idiopathic Arthritis
- › Behcet's disease



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Seronegative spondyloarthropathies

- › Family of rheumatologic disorders (back pain, uveitis, GI symptoms, rashes)
- › Ankylosing spondylitis (AS)
- › Psoriatic arthritis (PsA)
- › Inflammatory bowel disease (IBD) associated arthritis



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Sarcoidosis

- › Multisystem disease of unknown origin
- › Predominantly affects the lungs
- › 50% of cases have ocular involvement
- › Non-caseating granulomas composed of epithelioid & giant cells → secrete ACE



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Juvenile Idiopathic Arthritis

- › Accounts for 20-40% of pediatric uveitis patients
- › Many different subcategories
- › Often arthritis manifests before uveitis is detected



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Systemic Lupus Erythematosus

- › Multi-system autoimmune disease
- › Normal immune systems protect against pathogens (virus/bacteria), but ANA attacks cell nuclei triggering inflammation
- › ANA levels elevated in 97% of SLE patients
- › Can cause anterior uveitis



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Behcet's Disease

- ▶ Triad
 - Oral ulcers
 - Genital ulcers
 - Ocular inflammation
- ▶ Hypopyon common finding



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Hypopyon



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Infectious Etiology

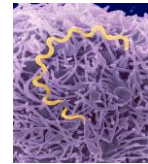
- ▶ Bacterial
 - Syphilis, Lyme, Tuberculosis, Endophthalmitis
- ▶ Viral
 - Herpes Simplex (HSV), Varicella Zoster (VSV), Cytomegalovirus (CMV)
- ▶ Fungal
 - Candida, Aspergillus, Histoplasmosis
- ▶ Protozoal
 - Toxoplasmosis
- ▶ Helminthic
 - Toxocariasis



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Syphilis

- ▶ **Systemic disease:** *Treponema pallidum*
 - Incubation period 2–4 wks
- ▶ **Eye:** "Great Imitator"
- ▶ **Epidemiology (from CDC):**
 - 46,042 new USA cases since 2011
 - 11.1% increase annually



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1^o & 2^o Syphilis—CDC Rates by State, United States, 2012

Remains major health problem in South & Urban areas of US



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Syphilis

Transmission:

- Congenital or Acquired
- Direct contact with 1^o or 2^o lesions

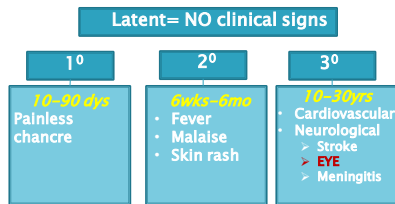
Risk factors:

- High-risk sexual activities
- Coexisting HIV infection
- IV drug use



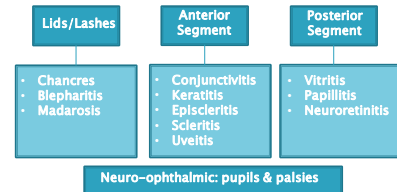
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Acquired Syphilis Stages:



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Ocular Manifestations:



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Syphilitic Uveitis

- ▶ **Neurosyphilis**
- ▶ Bilateral or Unilateral
- ▶ Non-granulomatous or Granulomatous
- ▶ Anterior, posterior, or both
 - Posterior=vitritis, retinitis, chorioretinitis
 - Posterior=55.2% cases
- ▶ HIV co-infection (60%)

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Neurosyphilis Tx (2010 CDC Guidelines)

Ocular disease = Neurosyphilis

1. Aqueous PCN G 18-24 million units/day IV x 10-14 days
 - Alt: Procaine PCN 2.4 million units/day IM x 10-14 days **PLUS** PO Probenecid 500 mg QID x 10-14 days
2. CSF examination & HIV testing
3. Repeat LP Q6mo X 2 yrs

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Lyme

- ▶ multisystem infection caused by the spirochete bacterium *Borrelia burgdorferi* -typically transmitted through the blacklegged tick
- ▶ Deer tick = transmit Lyme
 - Ixodes scapularis=NE, Mid-Atlantic, north-central
 - Ixodes pacificus=pacific coast
- ▶ Ticks not known to transmit Lyme disease:
 - Lone star ticks (*Amblyomma americanum*)
 - American dog tick (*Dermacentor variabilis*)
 - Rocky Mountain wood tick (*Dermacentor andersoni*)
 - Brown dog tick (*Rhipicephalus sanguineus*)

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Lyme

- ▶ On the rise: 30,000/yr reported cases to CDC
 - True # is like 10X this
- ▶ Illness consistent with Lyme reported in Europe as early as 1883
- ▶ Most-common vector-borne disease in both Europe & North America
- ▶ 22 variants of tick bites

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Lyme & the Eye

- › Keratitis, Follicular conjunctivitis, scleritis, uveitis, CN palsies, optic neuritis, papilledema
- › Non-ocular symptoms can vary
- › 2017 Retrospective study: no response to any type of corticosteroid (Ab only)

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Tuberculosis

- › caused by *Mycobacterium tuberculosis*
- › Can occur from active infection or secondarily as a result of immune reaction to the mycobacterium
- › Dx of intraocular TB is presumptive (after excluding other possible uveitis entities)
- › Beware of TB Treatment ocular side effects
- › Ethambutol: optic neuritis, color vision deficiencies, central scotomas, & retinal edema

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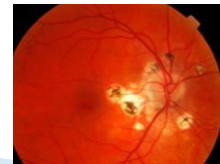
Cytomegalovirus (CMV)

- › Double stranded DNA virus in Herpesviridae family
- › Associated with HIV/AIDS (immunocompromised patients)
- › Transmitted by saliva, breast milk, sexual contact, organ transplantation
- › Anterior Uveitis, Retinitis, Corneal Endotheliitis
- › MOA theories: reactivation is primary cause of inflammation – or– reactivation occurs as secondary consequence of macrophage/dendritic cell activation

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Presumed ocular histoplasmosis syndrome (POHS)

- › Secondary infection with the yeast form of *Histoplasma capsulatum*
- › Triad:
 - Atrophic chorioretinal scars
 - Peripapillary atrophy (PPA)
 - Absence of vitritis



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Toxoplasmosis

- › *Toxoplasma gondii*: single-cell protozoan parasite
- › Hosts: **cats**, birds, reptiles, mammals
- › Congenital or acquired
- › Transmitted via direct contact with
 - Direct contact with contaminated food, soil, sand or cat litter
 - Consumption of raw or undercooked meat
- › White, chorioretinal, inflammatory lesion with an overlying vitritis

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Toxocariasis

- › Rare infection caused by roundworms (*toxocara canis* or *toxocara cati*)
- › *Unilateral 90% of the time*
- › Vitritis, leukocoria, endophthalmitis, central posterior or peripheral **granuloma**
- › Ultrasound biomicroscopy (UBM)=detection of granulomas



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Fuch's Heterochromic Iridocyclitis

- › Chronic, low grade unilateral non-granulomatous anterior uveitis
- › 2-3% of all uveitis cases
- › Asymptomatic
- › KPs have stellate appearance
- › Iris atrophy: decrease stromal melanocytes
- › Heterochromia ("moth eaten appearance") lighter iris → involved eye
 - Reversed Heterochromia → dark iris is involved eye

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Fuch's Heterochromic Iridocyclitis

- › Unclear etiology: Viral? Associated diseases?
- › To treat or not to treat
 - Few cases require therapy
- › Beware of complications:
 - Cataract
 - Glaucoma
 - Complications with cataract surgery (iris not dilated as well, acrylic vs silicone IOL)

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When to order Laboratory Testing

- › Recurrent or Chronic
- › Bilateral
- › Posterior pole findings
- › Unusual severity
- › Poor response to treatment

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Lab Tests for Uveitis

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Complete Blood Count (CBC)

- › Order with differential
- › Full panel describing blood makeup (erythrocyte, leukocyte, neutrophil, plasma count, etc)
- › Evaluates general health status
- › Viral, bacterial, leukemia
- › Helps differentiate between noninfectious & infectious

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Erythrocyte sedimentation rate (ESR)

- › Nonspecific for inflammation
- › Measures how quickly erythrocytes settle at bottom of test tube
- › Protein is produced by liver & increases with inflammation; RBC settle at a faster rate in the presence of increased proteins
- › Normal values: 0-13mm/hr males, 0-20mm/hr females
- › >50mm/hr → giant cell/temporal arteritis

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C-Reactive Protein

- › Blood test marker for inflammation in body
- › CRP produced in liver & sent into bloodstream in response to inflammation
- › Normal <10 mg/L
- › >10 mg/L → serious infection, trauma, chronic disease



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Antinuclear antibody (ANA)

- › Plasma cells produce antibodies directed against the body's tissues
- › Positive values:
 - SLE
 - TB
 - Hepatitis
 - Lymphoma
 - Sjogren's
 - JRA
 - Scleroderma



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Human Leukocyte Antigen (HLA-B27)

- › Presence of this antigen is indicative of the patient being at risk for having an autoimmune disorder
- › Ankylosing spondylitis
- › Reiter's Syndrome/Reactive Arthritis
- › Psoriatic Arthritis
- › Inflammatory Bowel Disease Arthritis

**Beware of false positives



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Angiotensin-Converting Enzyme (ACE)

- › Enzyme produced from granulomatous cells
- › Serum levels reflect total amount of granulomatous tissue in body
- › Sarcoidosis=75% sensitivity, 95% specific
- › False Positives: TB, Lymphoma, leprosy
- › Biopsy required for dx of Sarcoidosis



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VDRL, RPR, FTA-ABS

- › Syphilis
- › VDRL or RPR=Initial screening
- › FTA-ABS=confirm the diagnosis



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Tuberculosis

- › Purified protein derivative (PPD)
- › Quantiferon Gold
- › Chest X-ray



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Rheumatoid Factor (RF)

- Helps differentiate RA from other chronic conditions
- Can be (+) in Sjögren's syndrome, SLE, syphilis, sarcoidosis, & liver disease



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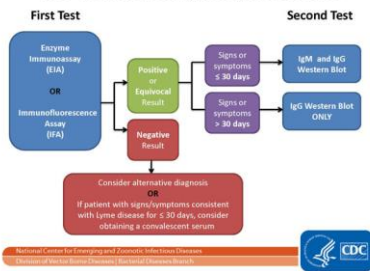
Enzyme-linked immunosorbent assay (ELISA)

- Detects antibodies in blood
- Helpful in identifying toxoplasmosis, toxocariasis, or Lyme



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Two-Tiered Testing for Lyme Disease



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Always Rule Out

- Tuberculosis
- Syphilis
- Sarcoidosis



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Lab Form



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Treatment Goals

- Preserve Vision
- Reduce Pain
- Eliminate inflammation
- Identify Source
- Prevent Synechia
- Manage IOP



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Treatment=Topical Steroids

- Concentration
 - 0.1% dexamethasone vs 1% prednisolone acetate
- Corneal penetration
 - Prednisolone greater than dexamethasone
 - 0.05% difluprednate = synthetic fluorinated pred derivative
 - Greater glucocorticosteroid receptor binding
 - Greater corneal penetration (addition of acetate ester at C-21)



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Treatment=Other Steroids

- Medrol Dosepak = oral methylprednisone
- Subconjunctival corticosteroid
- Dextenza plug=sustained release dexamethasone 0.4mg, Ocular Therapeutix (only approved for postoperative pain & inflammation so may not work alone for primary uveitis)



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Treatment

- Be aggressive
- Dosing
 - QID to q1hr
- Taper
 - Maintain steroid qd for additional 5 days after cells/flare resolve as it can sometimes take 3–5 days after inflammation for blood–aqueous barrier to restore
- Watch IOP spike



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Treatment

- Cycloplegics/Mydriatics
 - Improves comfort
 - Reduces leakage from Iris & CB
 - Prevents & breaks posterior synechiae
 - Avoid atropine if possible → slow acting can cause synechia lock
- Glaucoma Medication
 - Avoid prostaglandin
- Oral Steroids, steroid injections, immunosuppressive therapy, implant devices



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Appropriate Follow-up

- Mild: 4–7days
- Moderate: 2–4 days
- Severe: 1–2 days
- Resolved: q1–6mo



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When to Refer

- Infectious →STAT
- Intermediate, Posterior, Panuveitis
- Non-infectious w/systemic involvement → work with PCP, rheumatology, or appropriate provider



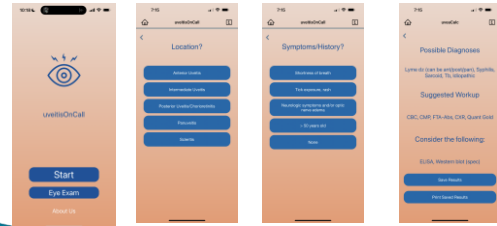
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Collaborative Care Steps

- › Report to the State/CDC
- › Letter to PCP/appropriate specialist
- › Make patient aware they may need specialist depending on clinical outcome

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New Technology: App called uveitisOnCall



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Case 1 Flashback–Treatment

- › 23yo acute, anterior, non-granulomatous uveitis
- › 1% pred acetate q2hr OS
- › 1 wk Follow-up Appointment
 - Complete Resolution
 - IOP stable
 - Tapered to BID X 3 days then d/c
 - No flare-up since

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Case Study 2

- › 25 yo AA Male
- › **CC:** “ blurry vision with black spots”
- › **HPI:**
 - Sudden, painless decrease in vision OS
 - Onset: 4 days, upon awakening
 - (+) flashes of light and floaters X 4 days OS

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History

- › **Medical history:** (+) epilepsy, (-)STDs, (-)inflammatory conditions
- › **Medications:**
 - Visine BID OU
 - 100 mg phenytoin sodium TID PO
- › **Ocular history:** Blind OD (2009 eye trauma)
- › **Social history:** (-)smoking, EtOH, drug use

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Entrance Testing

- › **BCVA:** NLP OD; 20/200 NIPH OS
- › **Pupils:** fixed, miotic OD; round, minimal reactivity OS
- › **Confrontational VF:** I & T constriction OS
- › **EOMs:** Full & Smooth OU

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Slit Lamp Findings

	OD	OS
Cornea	WNL	Edema 3+ guttata inferior KPs
A/C	Quiet irido-corneal touch	4+ cells/3+ flare (-)hypopyon
Iris	Atrophy	I, IT, ST synechiae
Lens	Displaced w/PS	4+ pigment AC
Vitreous	No view	(+)cells-hazy view of post pole

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Clinical Exam

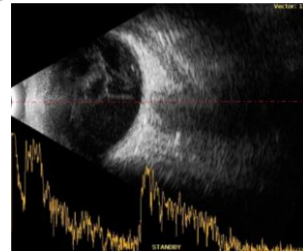
- ▶ **IOP:** 7 OD/10 OS (mmHG)
- ▶ **Gonioscopy:** PAS OS
- ▶ **DFE:**
 - OD: no view (dense cataract)
 - OS: photo

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B-Scan: OD



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Differential diagnosis

- ▶ Sympathetic ophthalmia
- ▶ Sarcoidosis
- ▶ Syphilis
- ▶ Tuberculosis
- ▶ HIV/AIDS

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Case Management

- ▶ 1% PA Q1hr & 1% Atropine BID OS
- ▶ Lab Work-up
- ▶ Uveitis specialist referral

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Diagnostic Testing

- ▶ **Labs:**
 - FTA-ABS (inconclusive)
 - (+)RPR, TP-PA
 - (-) HIV, HSV I IgG, HSV II IgG, HSV IgM, Lyme, EBV, WNV, Quantiferon
 - Unremarkable CSF & CXR



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Diagnosis

Syphilitic Panuveitis



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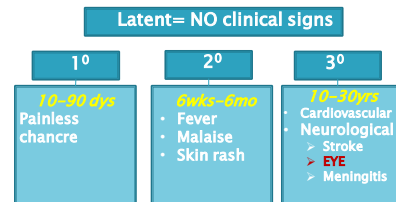
Treatment

- ▶ Hospitalized
- ▶ 60mg prednisone PO QD
- ▶ 24 million units/day IV aqueous PCN X 10 days



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Acquired Syphilis Stages:



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Neurosyphilis Tx (2010 CDC Guidelines)

Ocular disease = Neurosyphilis

1. Aqueous PCN G 18–24 million units/day IV x 10–14 days
 - Alt: Procaine PCN 2.4 million units/day IM x 10–14 days **PLUS** PO Probenecid 500 mg QID x 10–14 days
2. CSF examination & HIV testing
3. Repeat LP Q6mo X 2 yrs



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Post-Ab Treatment

- ▶ VA 20/100, PH 20/50 OS
- ▶ Essential Resolution of Uveitis & Vitritis
- ▶ F/U on going



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Case Study #3

- › 53yo Caucasian Female
- › **CC:** irritated left eye
- › **HPI:** started a month ago, previous provider treating for dry eye but sxs not improving
- › (+) watering, photophobia, FB sensation



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History

- › **Medical history:** joint pain, no diagnosis of arthritis
- › **Medications:** none
- › **Ocular history:** glasses, dry eye



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Entrance Testing

- › **BCVA:** 20/30 OD; 20/30 OS
- › **Pupils:** PERRLA, (-)APD
- › **Confrontational VF:** full
- › **EOMs:** Full & Smooth OU
- › **IOP:** 14mmHG OD, 13mmHG OS



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Slit Lamp Findings

	OD	OS
Conj	injection	injection
Cornea	Fine, white inferior KPs	Fine, white inferior KPs
A/C	1+ cells	2+ cells
Iris	(-)TIDs, (-)synechiae	(-)TIDs, (-)synechiae
Lens	Clear	Pigment on AC



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Posterior Pole Findings

	OD	OS
Vitreous	Quiet-no cells	Quiet-no cells
Optic nerve	Pink, healthy rim 0.3/0.3 C/D ratio	Pink, healthy rim 0.3/0.3 C/D ratio
Macula	Flat & clear	Flat & clear
Retina	No breaks/tears	No breaks/tears



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Diagnosis

- Bilateral acute anterior non-granulomatous uveitis
 -no posterior pole involvement
 -remnants of posterior synechiae OS only



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Treatment

- ▶ 0.05% difluprednate qid OU
- ▶ Due to bilateral presentation: CBC with diff, ANA, HLA-B27, PPD, CRP, VDRL/FTA-ABS, RF, and ACE analysis
- ▶ RTC 1 wk for f/u



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Follow-up

- ▶ 1 week=inflammation still present, CPM
- ▶ 2 week=resolved, taper started
 - Lab work unremarkable
- ▶ 4 week=resolved, d/c topical steroid



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4mo later

- ▶ Decreased vision OS, pain & irritation OU
- ▶ No changes to medical history
- ▶ VA 20/30 OD, 20/100 OS
- ▶ IOP 13mmHG OD, 15mmHG OS



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Slit Lamp Findings

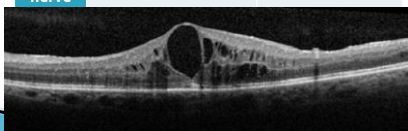
	OD	OS
Conj	injection	injection
Cornea	Fine, white inferior KPs	Fine, white inferior KPs
A/C	1+ cells	1+ cells
Iris	(-)TIDs, (-)synechiae	(-)TIDs, (-)synechiae
Lens	Clear	Pigment on AC



118

Posterior Pole Findings

	OD	OS
Vitreous	Quiet-no cells	Quiet-no cells
Optic nerve	Pink, healthy rim 0.3/0.3 C/D ratio	Pink, healthy rim 0.3/0.3 C/D ratio



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Treatment

- ▶ Start 0.05% difluprednate qid OU, 0.5% ketorolac TID OS
- ▶ Referral to uveitis specialist
- ▶ 2 wks later:
 - resolved CME
 - resolved inflammation →steroid taper
 - IOP spike → Combigan (Rx'ed uveitis specialist)



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2 years later

- › Frequent recurrent uveitis flare-ups
- › IOP controlled
- › Repeated Lab work: still negative

What now?



121

Treating Idiopathic Etiology

- › Still treat the current inflammation
- › Chronic inflammation can damage structures
- › Lab work



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Uveitis Clinical Pearls

- › Be a detective & find the cause
- › Be aggressive when initiating topical steroid treatment
- › Don't taper too soon
- › Optometrists play a key role



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Questions?



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Thank you!

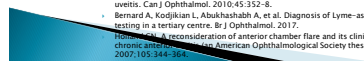
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