



Can replace FA in many cases

OCT Technology: Caveats

- DOES NOT take place of clinical exam!
- DOES NOT take place of careful history taking
- DOES NOT replace FA in some cases!
- DOES NOT REPLACE COMMON SENSE!
- ONE MORE PIECE OF CLINICAL PICTURE
 - Not the end all be all!!
 - Not to be taken in vacuum

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Common Types of Vitreoretinal Pathology by Anatomic Location

- Retinal Surface Vitreous and ILM
- Inner Retina
- Outer Retina/RPE/Choroid

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Vitreous

- Collagen and hyaluronic acid gel matrix congenitally adherent to the retina and optic nerve
- Degrades over decades as gel matrix dehydrates and coalesced fluid pockets forms, ultimately resulting in separation of the vitreous from the posterior retina and optic nerve (posterior vitreous separation)
 - Accelerated by trauma, inflammation, hemorrhage, myopia, ocular surgery, degenerative retinal diseases
- Vitreous separation is a discrete window when retinal tears or retinal detachment typically occur (15% risk in general population)

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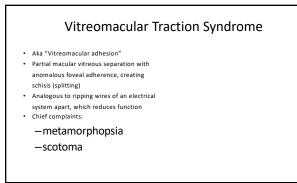
Vitreous Attachment/Detachment

- Vitreous consistency is like epoxy glue (sticky and stringy more than gooey)
- Pathologic states involve abnormal
- excessive adhesion between gel and retina • Symptomatic retinal breaks usually
- accompany PVD
- Completion of PVD significantly reduces
 risk for future retinal breaks
- Surgical approach for RD repair depends on PVD status

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Vitreoschisis

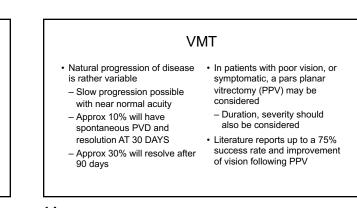
- The vitreous gel is arranged in layers, like skins of an onion
- The layers closest to the innermost layer of the retina (ILM) are tightly adherent and may strip away from the vitreous body in pathologic states



VMT

- More commonly encountered in older women - Can occur in either sex, and age, no apparent racial
- predilection Aphakia and pseudophakia are protective
- VAST STUDY
- 2,179 eyes, 1,120 asymptomatic pts>40 years of age
 - Mean age 59
 - 57% female 57% hyperopes, 35% myopes, 8% emmetropes
- VMA in 31% of eyes
 - Peak age 50-59
 - Less common in AA and HA

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Diabetic Tractional Retinal Detachment • Proliferative (neovascular) diabetic retinopathy thickens/toughens the ILMvitreous interface (adhesion) while prematurely aging the vitreous body (contraction) Degree of tractional forces depends on quantity (more) and quality (fibrotic) of new vessels 15

Full Thickness Macular Hole

- More severe form of VMT resulting in
- full thickness foveal defect Stages:
- 1 = outer retinal defect 2 = full thickness defect with vitreous
- traction on one edge
- 3 = operculum
- 4 = PVD
- Chief complaint: scotoma
- Treatment: Surgery, Intravitreal Ocriplasmin

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FTMH FTMH If pt has macular hole in one eye, 28-44% chance of macular hole in other eye w/o a PVD Definition: Macular hole that Small holes: <250 um affects all macular layers from - Small rate of spontaneous closure If PVD already, very little chance vs lamellar or partial - Very high surgical closure rate (almost 100%) Most common cause is idiopathic or "primary" – With advent of OCT realize "idiopathic" is due to vitreoretinal · Vision typically 20/80 to • Medium holes: 250um to 400um - High surgical closure rate (>90%) Highest incidence in 7th Large holes: >400 um traction Secondary causes include blunt trauma, severe myopia, solar retinopathy, CME - High surgical closure rate (75-90%) Women 2x as often as men 1/2 of all holes are large at time of diagnosis

ILM to RPE

20/200

decade of life

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Vitreous Opacities

- Opaque objects in the vitreous block the OCT's laser, producing **SHADOWING** of deeper layers
- Examples: Asteroid hyalosis (calcium), hemorrhage, inflammation, intraocular foreign bodies, vitreous opacities
- Treatment: Surgery

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Epiretinal Membrane

- Thickened mass of cells at the VR interface (hyaloid, reactive, RPE)
- Very common (25% after PVD), but most not visually significant
- Chief Complaints:

metamorphopsia,

blurred vision

Treament: Surgical Removal

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Lamellar Macular Hole

- Symptoms
 - mild metamorphopsia
 - limited acuity loss
 - stable vision
- Surgery is controversial
- 25% to 75% improved visual acuity
- Therefore, monitoring seems reasonable

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Myopic Macular Schisis

- Pathologic myopia disproportionately elongates posterior aspect of eye (eggshaped)
- Posterior hyaloid face acts like ERM and contracts against excessive posterior curvature of myopic macula
- Results in splitting (schisis)

Staphyloma

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Retinal Detachment

- Contraction of vitreous body, often with PVD, can result in retinal tears if VR traction exceeds retinal integrity
 Retinal breaks become an avenue for
- subretinal fluid and overwhelm the RPE pump, creating RD
- Macular status determines urgency of repair and prognosis about visual outcome

Inner Retina

- Responsible for processing light information from multiple photoreceptors and transmitting it through the optic nerve towards the brain
- Contains nerve fiber layer, ganglion cells; bipolar, amacrine, and horizontal cells; Müller cells
- · The retina receives oxygen from two sources
 - Inner 2/3 supplied directly by retinal blood vessels
- Outer 1/3 supplied by diffusion from the choroid
- Obstruction of the retinal circulation produces visible changes on OCT

Retinal Artery Occlusion

- Acute obstruction of a retinal artery results in ischemia (dysfunction) and ultimately infarction (death)
- Acutely, inner retinal edema seen clinically as retinal whitening
- Chief complaint:
 - -scotoma, blindness

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Treatment controversial: paracentesis, globe compression, YAG laser embolysis, aspirin

- Workup: R/O GCA, Carotids, Echo, Hypercoagulability workup,
- Vasculitis/Uveitis workup

Retinal Vein Occlusion

- Venous obstruction releases contents of the bloodstream (water, blood cells, and cholesterol) into the retina
- Plumbing problem
- Chief Complaints: blurred vision, scotoma, metamorphopsia
- Treatment: manage underlying vascular disease (HTN, DM, OSA) and/or glaucomas, retinal lasers, intravitreal anti-VEGF and/or corticosteroids

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Diabetic Retinopathy Diabetic Macular Edema

- · Diabetes preferentially damages capillary beds, creating microaneurysms and avascular zones
- Clinically edema, exudates, and hemorrhage are seen in varying degrees associated with vascular anomalies
- Similar to RVO, intraretinal hyperreflective areas and inner and outer retinal cystic hyporeflective cavities are seen on OCT

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Macular Telangiectasia Type 2

- · Idiopathic macular disease
 - associated with Retinal telangiectasia
 - Cystic macular degeneration
 - RPE hyperplasia

 - Intraretinal crystals

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Outer Retina/RPE/Choroid

- Outer Retina retinal photoreceptors are highly specialized neurons that generate electrical responses to light.
- RPE multipurpose layer needed to maintain outer retinal function and health
- Choroid high flow blood vessel layer that sustains outer retina by diffusion

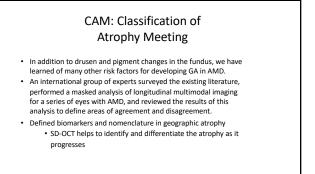


- the US in adults over 50 "Dry" = progressive loss of RPE/outer retinal function/cells (atrophy) with accumulation of drusen (hallmark lesion)
- "Wet" = development of new choroidal vessels outside the choroid that leak water, blood, or cholesterol into the subRPE or subretinal space
- Chief Complaints: Blurred Vision
 - Metamorphopsia Scotoma
- Treatment

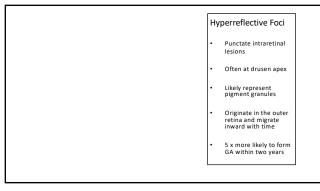
Drusen

- Accumulation of incompletely recycled visual cycle pigments that could not be returned to the photoreceptors in conjunction with complement cascade components/inflammatory debris
- Accumulate progressively, but distribution can fluctuate in some patients
 Loss of druson frequently
- Loss of drusen frequently accompanied by RPE atrophy (geographic atrophy)

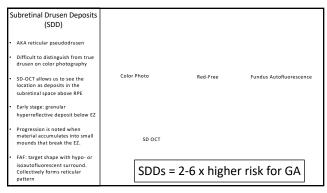
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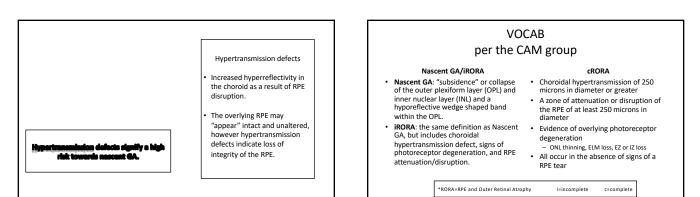
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Choroidal Neovascularization

- A break in Bruch's membrane and/or RPE associated with leaky vessels
- Type 1: SubRPE
- Wet AMD
 Type 2: Subretinal
- Inflammatory
- Pathologic MyopiaType 3: Retinal Angiomatous
- Proliferation
- Wet AMD
- Macular Telangiectasia

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Pathologic Myopia

- Axial myopia, commonly called "near-sightedness," in its extreme form
- Represents a connective tissue weakness
 - Elongates the eye, thinning the layers of the eye wall (cornea, sclera, choroid, RPE, retina)
- Degenerating the vitreous early (collagen and hyaluronic acid)
- Creating traction between the stretched retina and vitreous
- Predisposes to vitreoretinal interface disorders, retinal tears, and retinal detachment

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Myopic Macular Degeneration

- Thinning of the posterior eye wall layers
- Increased posterior curvature (staphyloma)
 - Diffuse or focal
- RPE thinning/atrophy (lacquer cracks, geographic atrophy)

Central Serous Retinopathy

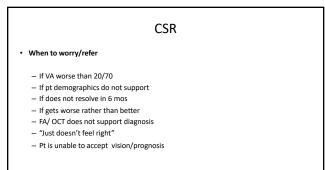
- Vision typically 20/30-20/70
- Typically self-limiting
 80-90% of pts will undergo
 - spontaneous resolution within 1-6 mos.
 - >60% resolve back to 20/20
 Rare to have vision remain < 20/40
 - $-\cong 40\%$ will get recurrence
- FA is helpful in providing definitive diagnosis

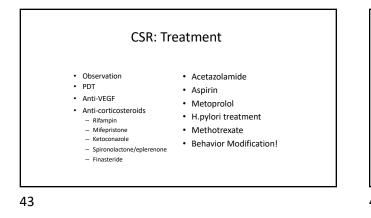
 Classic Smoke stack appearance (occasionally)
- Ink-blot appearance
- Not needed with advent of OCT/OCTA unless unusual presentation

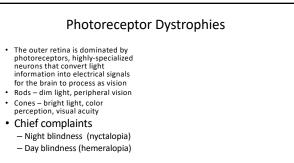
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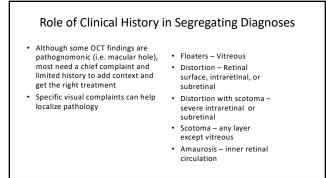
CSR: Risk Factors OTHERS TRADITIONAL • Male > Female 10:1 • Age: Peak 20-45 • Type A personality • Stress • Pregnancy • Signal Characteristic for the set of the set of







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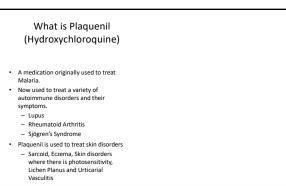
Patient presents for AMD evaluation...

- 75 year old white woman
- CC: Patient moved from California to Ohio. New to the area, the patient states that she was diagnosed with Dry AMD by her eye doctor in California. Patient states that her vision has been decreasing for several years now. She is having difficulty seeing details and no longer reads. In general, she feels her vision is keeping her from doing what she loves to do. She no longer drives.
- Amsler grid testing: Irregular in both eyes with central metamorphopsia OU
- · Pupils and slit lamp within normal limits OU

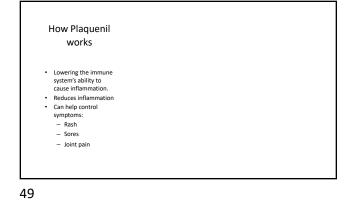


Let's take a look at our patient's medication list...

- Centrum Complete
- Vitamin D3
- Sertraline HCl
- Lisinopril
- Levothyroxine Sodium
- Diclofenac Sodium
- Hydroxychloroquine (Plaquenil) 200mg BID



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Possible Ocular Effects of Plaquenil (Hydroxychloroquine)

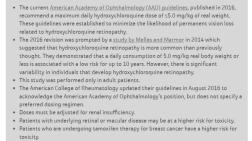
- A rare side effect of extended or over-dose Plaquenil use can be damaging, or toxic to the Retina.
- It is believed that Plaquenil binds itself to the Retinal Pigment Epithelium (RPE) and can cause damage to the photoreceptors.
- Typically asymptomatic in its early stages, but can lead to severe retinal damage, and permanent vision loss.
- Early signs include blurry central vision, losing the ability to read a digital clock, loss of color vision, and trouble seeing at night.

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Higher Risk Factors for Toxicity:

- Taking Plaquenil for 5+ years
- · Taking a higher than recommended dose
- Pre-existing Kidney or Liver Disease
- Pre-existing Retinal Disease
- Age 60 or older
- · Losing a significant amount of weight while taking Plaquenil without adjusting your dose
- May be more common than previously thought (≅7%)

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- toxicity.

mdcalc.com

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Important Exam Testing:

- HPI should include:
 - How long have you been taking Plaquenil?
 - What is your weight?
 - What is your dose?
 - Do you have a history of Kidney disease?
- Have you noticed any changes in your vision? Baseline testing should be completed before, or within the first year of, beginning Plaquenil.

 - Testing: 10-2 Humphrey Visual Field (HVF)
 - OCT Macula
 Fundus Autofluorescence (AF/FAF)
 - Color vison