

Blood in the Eye...Now What?

Dave Trujillo
Toledo 2020

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Goal

- Convince everyone that blood does not lie
- Review common etiologies of hemorrhages in the anterior and posterior chambers through a presentation of cases
- Feel comfortable in managing these "hemorrhagic" patients

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Financial Disclosure

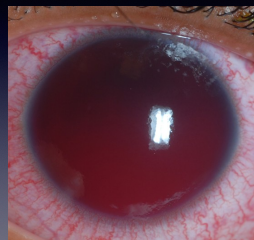
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Patient A - "I was hit in the eye"

- 22 year old white male
- Using a grinding wheel
- No pain, just can't see (HM)



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Patient B - "My eye hurts"

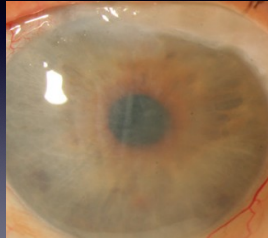
- 80 year old white female
- Pain 10/10 and she didn't sleep
- Her daughter was worried because it looked RED



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Patient C - I had cataract sx last week

- 78 year old white female
- Cataract Sx OD looked great...the problem was this was her left eye
- IOP at 73 - she was feeling great



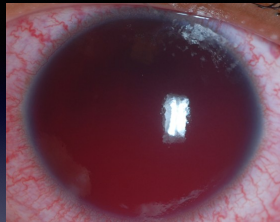
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Patient D - Vision was blurry when I woke up

- 58 year old white male
- "Vision is dim and foggy"
- No pain
- No trauma



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Patient A

Hit in the eye

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"I was hit in the eye"

- 22 year old white male
- Complete hyphema
- IOP 22 (18 OS)

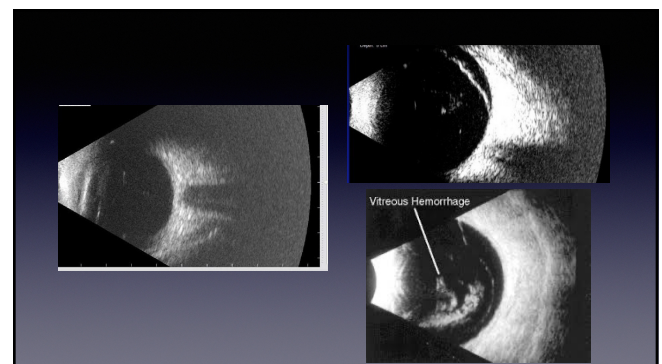


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Traumatic Hyphema What's Next??

- Control IOP
- Start Atropine tid for comfort and to reestablish blood aqueous barrier
- Prednisolone qid- q2hr for comfort and inflammation
- No aspirin or NSAIDs
- What about an ultrasound or gonioscopy??

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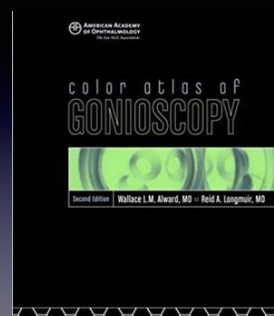
Gonioscopy

AKA: paperweight collecting dust

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AAO

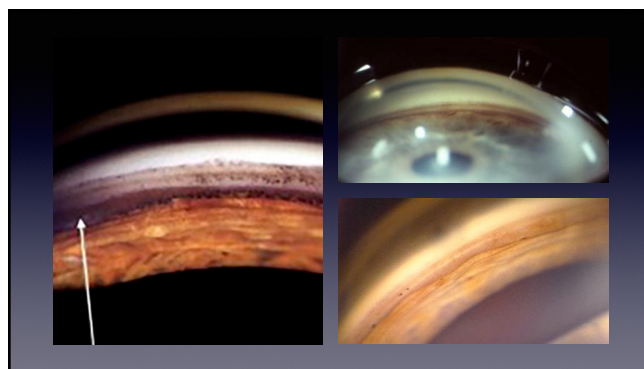
www.gonioscopy.org
University of Iowa



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Angle Recession

- Common manifestation of blunt ocular trauma
- Involves rupture of the ciliary body face, resulting in a tear/separation of the ciliary muscle
- 20-94% of eye
- Hyphema results from breaking of the anterior ciliary arteries
- 5-20% of eye with angle recession develop angle recession glaucoma
- Degeneration and scarring lead to aqueous outflow obstruction

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Follow up

- See 1-2 days until improvement
- 4-6 weeks for dilated exam
- Trauma: Risk of cataract, glaucoma and retinal detachment

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Pearls - Trauma

- Treat elevated IOP
- Control pain and inflammation
- Follow daily until improvement
- Monitor for retinal detachment, glaucoma, and early cataract

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Patient B

Painful red eye

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“My eye hurts”

- No light perception ++APD
- Pain 10/10
- In the ED last night and was heavily sedated from morphine
- No trauma



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What am I NOT telling you?

- IOP at 62
- Controlled hypertension and hypercholesterolemia
- No diabetes
- History of stroke and CRAO

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Let's look closer

- Rubeosis iridis or Neovascularization of the iris (NVI)
- Small fine blood vessels develop on the anterior surface of the iris
- Results from retinal ischemia
- Fragile and tend to bleed



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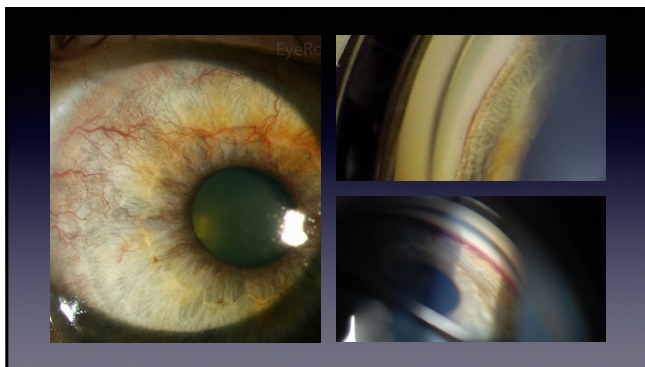
NVI following CRAO

- Korean Journal of Ophthalmology
- Retrospective study 2009-2015 of 214 patients
- 10.9% of patients developed NVI
- Mean time of NVI diagnosis was 3 months

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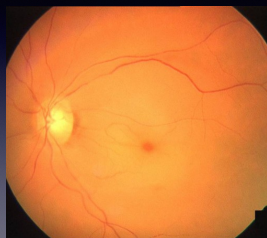
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What's next?

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Look at the retina

- * NVI results from:
 - Diabetic retinopathy
 - Central and branch vein occlusion
 - Central retinal artery occlusion
 - Ocular ischemic syndrome
 - Chronic retinal detachment



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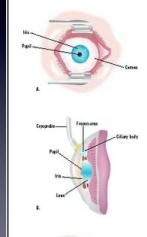
What's Next?

- Control IOP
 - Beta blocker, alpha agonist, carbonic anhydrase inhibitors
 - Prostaglandins??
 - PRP, cyclocryopexy, trabeculectomy, tube shunt/ahmed valve
- Control Pain
 - Topical steroid and cycloplegic

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Cyclocryopexy

- Retrobulbar or peribulbar block
- Probe is applied to the sclera above the ciliary body at -112 degrees Fahrenheit
- Repeated several times in a clockwise fashion
- Initial treatment is usually 180 degrees



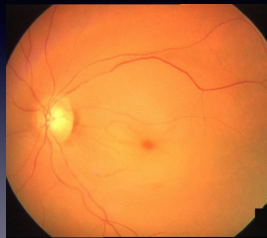
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Cyclocryopexy

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CRAO

- Stroke evaluation
- Nonarteritic vs Arteritic
- 2-3 month f/u to rule out NVI

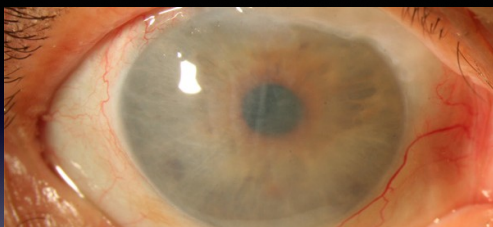


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Pearls

- Retinal ischemia including CRAO
- Monitor for NVI, NVD, NVE
- NLP eye - make comfortable
- Will need surgical treatment
- Cyclocryopexy - patient will have pain following

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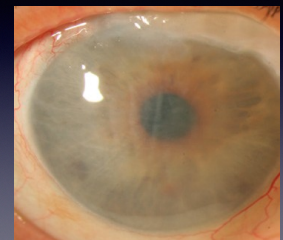


Patient C
One week after cataract Sx

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Lost vision 5 months ago

- IOP in the 70s
- NLP
- No pain
- Rubeosis??
- She has no clue what happened to her vision
- What's next??



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Look at the retina

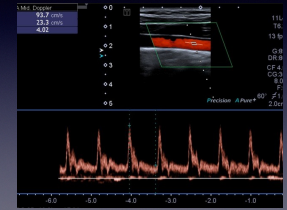
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What's Next

- Carotid Doppler
 - Possible endarterectomy
 - Waiting on results
- Lower IOP
 - Cyclocryopexy??
 - NLP
 - No pain



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Pearls - OIS

- Neovascularization from retina ischemia
- Control IOP
 - Alpha agonist, beta blockers, CAs
 - Surgical treatment Make
- the eye comfortable
- Keep the cornea as clear as possible
- Talk with PCP

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Patient D

Foggy VA upon waking

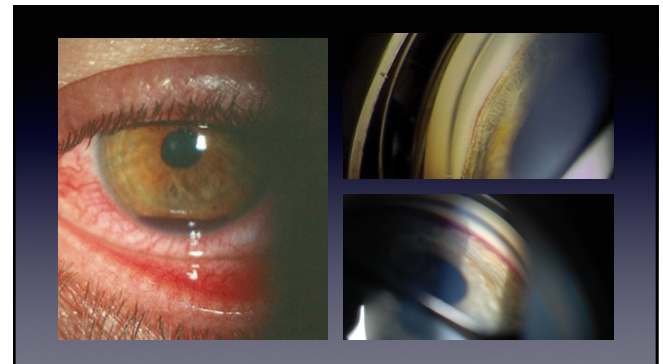
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Woke up with foggy vision

- 20/50
- IOP at 50
- 1-2 mm hyphema
- Insulin dependent ~25 years
 - HA1C 7.5
 - Until recently, hasn't been well controlled



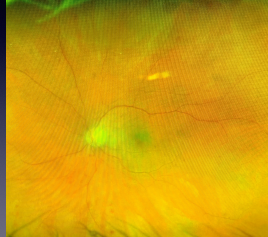
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Look at the Retina

- NVI results from:
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 - Chronic retinal detachment



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What's Next?

- Fluorescein angiography
- Neovascularization
- Non perfusion
- Anti-veg treatment
- Panretinal photocoagulation



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Retinal Hemorrhage

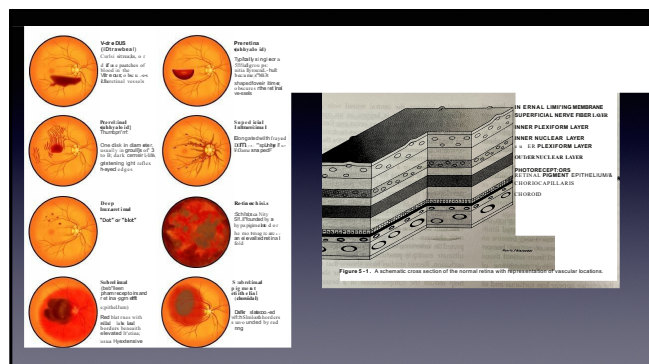
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third edition

PRIMARY CARE ^{of} THE
POSTERIOR SEGMENT

• Larry J. Alexander •

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Preretinal and Subhyaloid hemorrhage

• "D" or boat shape with distinct horizontal line at the top (controlled by gravity)

• Block underlying retinal features

• Think neovascularization of the nerve/retina or traction from posterior vitreal separation

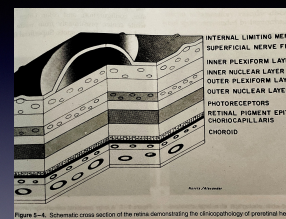
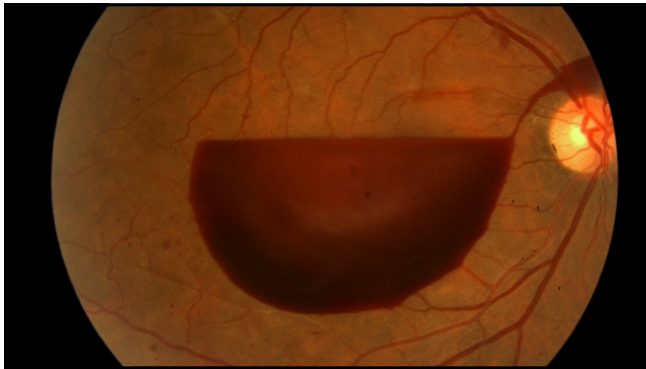
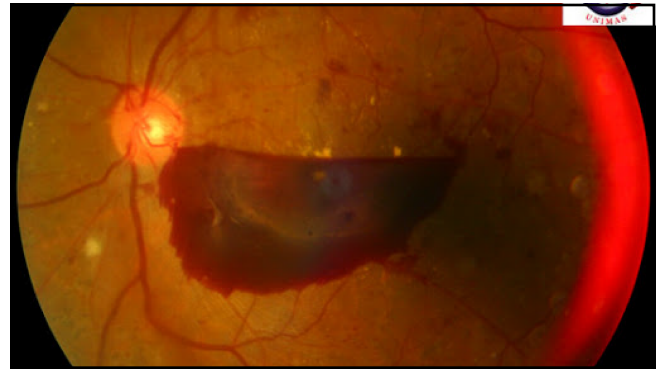


Figure 5-4. Schematic cross section of the retina demonstrating the clinical pathophysiology of preretinal hemorrhage.

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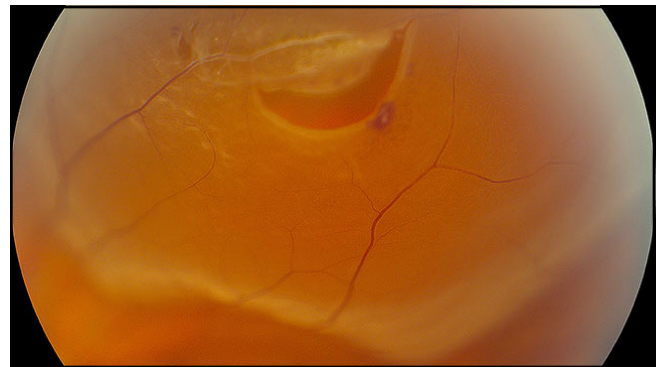
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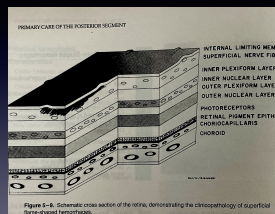
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Flame-shaped hemorrhage

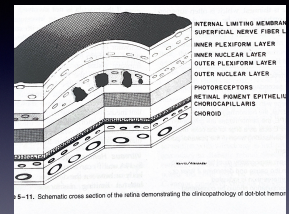
- Typically thin or elongated shape (feather, flame or brush-stroke)
- Located in the ganglion cell and nerve fiber layer
- Think artery based diseases



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Intraretinal "dot and blot"

- Displaces normal retinal tissue, forming round and uniform hemorrhages
- Located within the inner nuclear and outer plexiform layers
- Think vein based diseases



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Subretinal and Sub-RPE

- Broad with indistinct borders VS well defined borders
- Anterior to RPE VS Posterior to RPE
- Overlying vasculature can be visualized
- Think CNVM...but rule out trauma or tumor

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Back to Patient C

- * Ocular ischemic syndrome
- Hypoperfusion to the eye most commonly on account of carotid artery stenosis
- Mid peripheral heme in mid retina (venous)
- 2000-2500 cases in the US/year
- Men twice as likely
- Easily mistaken for diabetic retinopathy

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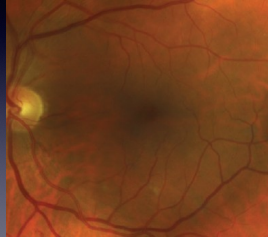
Patient D

Bain, SC, Klufas, MA, Ho, A, Matthews, DR. Worsening of diabetic retinopathy with rapid improvement in systemic glucose control: A review. *Diabetes Obes Metab*. 2019;21:454-466

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Mild NPDR

- Microaneurysms (MAs) only
- Monitor 1 year



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Moderate NPDR

- At least 1 hemorrhage or MA and/or 1 or more of the following
- Retinal hemorrhage
- Cotton wool spot
- Hard exudates
- Venous beading
- Monitor 6 months



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Severe NPDR

- 4 - 2 - 1 rule
- >20 intraretinal hemorrhages in all 4 quadrants
- Venous beading in at least 2 quadrants
- Intraretinal microvascular abnormality (IRMA) in at least 1 quadrant
- Retina referral



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Proliferative Diabetic Retinopathy

- Neovascularization
- Preretinal/vitreous hemorrhage
- Retina referral



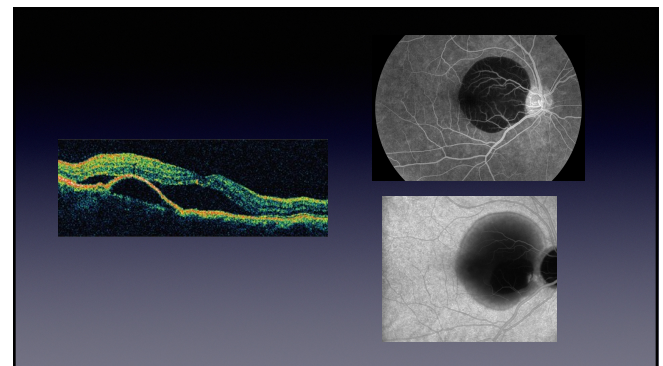
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Patient E

- Woke up with sudden loss of vision (HM)
- Sub RPE with PED
- Everything else is unremarkable OU



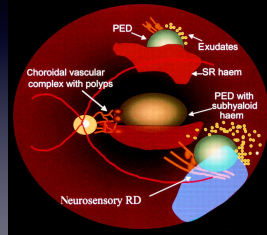
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Idiopathic Polypoidal Choroidal Vasculopathy

- Pachychoroid disease spectrum
- Abnormal choroidal vasculature
- Permanent choroidal thickening
- Similar to AMD
- No drusen
- CNVM not typical
- Anti-vegf vs PDT



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Retina Pearls

- Affected layer will determine diagnosis
- Vitreous, subhyaloid and preretinal — Neovascularization or Traction
- NFL — Artery based (HTN). Intraretinal — Venous based (DM)
- Subretinal and sub RPE — choroidal neovascularization/ Pachychoroid
- Retinal ischemia leads to NVI

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Thank You

dtvuiillo@specialtyveveinstitute.com

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