RETINAL EMERGENCIES

THE URGENT & THE EMERGENT
Financial Disclosure

Speaker, Martin Pearlman, M.D. has a financial interest/agreement or affiliation with Lansing Ophthalmology, where he is employed as a retina specialist.
How rapid is emergent vs. urgent?

Emergent should be seen that day preferably within hours of onset.

Urgent should be seen within 48 hours.
In most of medicine the history is most important in making a diagnosis.

In ophthalmology, the physical exam is more important. Histories tend to be similar for multiple causes of vision loss.
There is an extensive differential diagnosis for acute loss of vision. Included are:

1. Central Retinal Artery Occlusion
2. Central Retinal Vein Occlusion
3. Branch Retinal Artery Occlusion
4. Branch Retinal Vein Occlusion
5. Ischemic Optic Neuropathy
6. Retinal Detachment
7. Vitreous Hemorrhage
8. Exudative (wet) Macular Degeneration
The only acute retinal emergency is:

Central Retinal Artery Occlusion. (CRAO)

We will concentrate on that entity first.
History:

• Acute, monocular, painless, loss of vision.
• Occurs over a few seconds.
• Sometimes preceded by amaurosis fugax.
Amaurosis Fugax

1. Fleeting loss of vision lasting a few seconds.
2. Usually represents an embolic phenomenon.
3. But only 1%/year of amaurosis fugax leads to a CRAO.
Visual Acuity

1. ranges from counting fingers (CF) to light perception (LP) in 74% to 90% of eyes

2. poorer vision at onset correlates with poorer visual outcomes.

3. Visual acuity may improve in 22% of patients, but only 10% report significant improvement.
External Examination

• Afferent pupillary defect develops immediately.
• Intraocular pressure is normal acutely.
• Anterior segment is normal.
• Only see rubeosis iridis (iris neovascularization) 4-5 weeks after an acute episode or if chronic carotid ischemia preceded the CRAO.
The fundus exam makes the diagnosis.

- Cherry red spot (choroid visible through the thin fovea.)
- Retinal whitening (ischemic damage to inner ½ of retina.)
- Box-carrying of retinal arteries and veins (segmentation of blood column)
- Retinal artery attenuation
- Optic nerve edema
- Optic nerve pallor
- Retinal emboli
- Retinal periphery is usually normal
CRAO
CHERRY RED SPOT
RETINAL WHITENING
BOX-CARRING
CRAO
HEMORRHAGE IS UNUSUAL
CHERRY RED SPOT
RETINAL WHITENING LESS PROMINENT.
Central Retinal Artery Occlusion Treatment

In primate experiments, photoreceptors begin to die 90 to 100 minutes after complete occlusion. By 4 hours there is massive photoreceptor damage.

However, it is our clinical impression that most central retinal artery occlusions are partial and tend to be transient.

Therefore, anytime in the first 24 hours after the event we attempt to intervene.

This is considered an ophthalmologic emergency. Call the ophthalmologist.
Central Retinal Artery Occlusion Treatment

As an emergency measure, compress the eye with the heel of your hand for 10 seconds and release for 10 seconds over a 5 minute period to attempt to displace the clot and re-establish blood flow.

Rebreath in a paper bag to raise the pCO2. This causes arteriolar dilatation.

Inhale 95% O2 with 5% CO2 to cause arteriolar dilatation.
CRAO TREATMENT
MASSAGE TO CAUSE EMBOLUS TO MOVE
PAPER BAG REBREATHEING RAISES $p \text{co}_2$ TO DILATE ARTERIOLES.
ANTERIOR CHAMBER PARACENTESIS

WILL ABRUPTLY LOWER INTRAOCULAR PRESSURE IN ATTEMPT TO MOVE EMBOLUS.

We endeavor to keep the intraocular pressure low to encourage arteriolar inflow. Diamox and glaucoma drops are used.
CRAO Treatment

• If the visual prognosis is so poor, why do we rush to see these patients?

• If the etiology of the CRAO is not embolic, we must not miss Giant Cell Arteritis (GCA) which can cause loss of vision in the other eye in as little as 24 hours after the CRAO.

• We are more likely to suspect GCA with a history of older age, temporal headaches, jaw and tongue claudication, and scalp tenderness.

• Nevertheless a stat Sed Rate and C-reactive protein should be obtained.
CRAO TREATMENT

• It would be logical to ask why not use t-PA?
• Intravenous thrombolytics are avoided because of the incidence of hemorrhagic stroke.
• EAGLE study in Europe showed similar clinically significant visual improvement comparing the group treated with intra-arterial t-PA to conventional therapy.
Central Retinal Vein Occlusion (CRVO)

- Loss of vision may be severe, but onset is usually sub-acute.
- Fundus picture includes disc edema, retinal hemorrhages, cotton wool spots, and macular edema.
CRVO
LOOKS LIKE A RIPE TOMATO WAS TOSSED AGAINST THE BACK OF THE EYE AND SPLATTERED EVERYWHERE.
CRVO
HEMORRHAGES LESS PROMINENT
ACCOMPANIED BY MACULAR EDEMA
CRVO
PROMINENT COTTON WOOL SPOTS DILATED TORTUOUS VEINS PROMINENT DISC EDEMA
CRVO TREATMENT

• REVOLUTIONIZED WITH ADVENT OF VASCULAR ENDOLETHIAL GROWTH FACTOR INHIBITORS (ANTI-VEGF)

• INJECTIONS ALSO PREVENT NEOVASCULAR GLAUCOMA DUE TO RUBEOSIS IRIDIS.

• VISUAL PROGNOSIS IS NOW MUCH IMPROVED.

• THIS IS URGENT AS THE VISUAL RETURN IS BETTER WHEN ANTI-VEGF TREATMENT IS BEGUN SOONER.
BRANCH RETINAL VEIN OCCLUSION (BRVO)

HISTORY: SUDDEN MONOCULAR VISION LOSS OFTEN WITH A SECTOR VISUAL FIELD DEFECT OCCURS AT ARTERY-VENOUS CROSSING WHERE A COMMON ADVENTITIAL SHEATH IS SHARED OFTEN SEEN WITH HTN, GLAUCOMA, DM
BRVO COMPLICATIONS

MACULAR EDEMA (SHOWN HERE)
MACULAR ISCHEMIA
RETINAL OR DISC
NEOVASCULARIZATION

PREVIOUS TREATMENT
PHOTOCOAGULATION
RECENT ADVANCE: GREAT WITH
ANTI-VEGF OR INTRAOCULAR
STEROIDS

URGENT BECAUSE BETTER VISUAL
RESULT IS OBTAINED WITH
PROMPT USE OF ANTI-VEGF
INJECTIONS.
BRVO COMPLICATIONS
DISC NEOVASCULARIZATION
BRANCH RETINAL ARTERY OCCLUSION (BRAO)

HISTORY: SUDDEN MONOCULAR VISION LOSS OFTEN RESTRICTED TO PART OF THE VISUAL FIELD.

MUST SEARCH FOR SOURCE OF EMBOLI

GCA LESS LIKELY, BUT URGENT TO RULE IT OUT.

COMPLICATIONS: RETINAL NEOVASCULARIZATION
ANTERIOR ISCHEMIC OPTIC NEUROPATHY (AION) HISTORY

HISTORY: MORE GRADUAL ONSET UNILATERAL LOSS OF VISION

USUALLY A VISUAL FIELD DEFECT RATHER THAN COMPLETE BLACK OUT.

PALE DISC SWELLING OFTEN WITH HEMORRHAGE.

MUST R/O GCA.
AION
WILL OFTEN RESULT IN
OPTIC ATROPHY
VITREOUS HEMORRHAGE (VH)

HISTORY: MAY REPORT FLOATERS PRIOR TO HEMORRHAGE

LOSS OF VISION MAY BE SUDDEN, BUT NOT COMPLETE

MAY HAVE A HISTORY OF POORLY CONTROLLED DM OR BVO

HEMORRHAGE MAY SHIFT WITH MOVEMENT
VH can also be seen with posterior vitreous detachment and retinal tear.
VH
URGENT TO R/O TORN RETINA OR INCIPIENT RETINAL DETACHMENT

IF DUE TO RETINAL VASCULAR DISEASE, OBSERVE FOR CLEARING AND DO PAN-RETINAL PHOTOCOAGULATION.
RETINAL DETACHMENT

HISTORY: USUALLY FLASHERS AND FLOATERS FOLLOWED BY VISUAL FIELD DEFECT.

CHOROIDAL MARKINGS ARE DIFFICULT TO SEE

REPAIR WITHIN 24-48 HOURS IF MACULA IS ATTACHED

REPAIR WITHIN 7 DAYS IF MACULA DETACHED

URGENT TO SEE AND TREAT TO PREVENT PROGRESSION.
RETINAL DETACHMENT

THIS IS A MACULAR HOLE WITH A DETACHMENT

NOTE THE PERIPHERAL CHOROIDAL MARKINGS WHICH ARE VISIBLE.
POSTERIOR VITREOUS DETACHMENT (PVD)

- HISTORY: FLASHES AND FLOATERS.
- SEEN IN 50% OF PEOPLE OVER 50
- 10% CHANCE OF RETINAL TEAR.
- CHANCE OF A RETINAL TEAR IS 25% WHEN HEMORRHAGE SEEN
- SEE WITHIN 24 HOURS TO R/O RETINAL BREAK
- A FRESH RETINAL TEAR HAS A 35% CHANCE OF CAUSING A RETINAL DETACHMENT.
The examination for retinal tears or retinal detachment is done with an indirect ophthalmoscope.
EXUDATIVE (WET) MACULAR DEGENERATION

• History: May have a history of “dry” macular degeneration

• Central visual loss is often gradual, but may be sudden.

• Peripheral vision is intact.

• Subretinal fluid and subretinal hemorrhage common.
Wet Macular Degeneration

- Confirm diagnosis with fluorescein angiogram and ocular coherence tomography (OCT)

- Old treatment: laser

- New treatment: Anti VEGF injections.
WET MACULAR DEGENERATION

Can be evaluated with optical coherence tomography (OCT)