Course # 120

SECO2026
THE EDUCATION DESTINATION™

# Vascular Disorders and Vision

Kelsey Moody Mileski, OD

*Please Silence All Mobile Devices.*

attendseco.com

1

SECO2026
THE EDUCATION DESTINATION™

Kelsey Moody Mileski, OD

Disclosure statements:

*No financial relationships with ineligible companies to disclose.*

*All relevant relationships have been mitigated.*

2

## Vascular Disorders and Vision

Kelsey Moody Mileski OD, FAAO
Atlantic Eye Institute
Jacksonville Beach FL

3

- No financial disclosures

4

## Course Outline

- Discussion on stroke
- Blood flow to the brain with clinical correlates
- Clinical cases
- Blood flow to the eye
- Ocular manifestations of vascular disorders
- Clinical cases

5

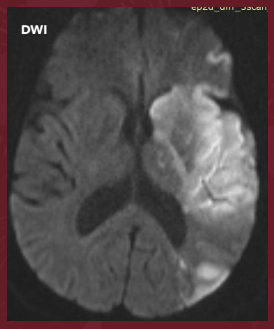
## Stroke

- Blood vessel to the brain is blocked or ruptured creating lack of blood flow to brain tissue
- #5 leading cause of death and leading cause of disability in US
- **Ischemic**
- **Hemorrhagic**
- **Cryptogenic**
  - Cause of the stroke is unknown

6

## Stroke

- **Ischemic**
  - 87% of all strokes
  - Thrombosis- blood clot in vessel
  - Embolism – wandering clot
    - Carotid arteries
    - Cardiac
      - Atrial fibrillation is common cause
  - Treatment: dislodge blockage and restore blood flow
    - **Timing matters!** – ideally within 3.5-4 hours (stroke window expanded to 6 hours)
    - IV-TPA
    - Mechanical thrombectomy
    - Often placed on ASA and cholesterol medication afterwards



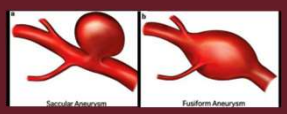

7

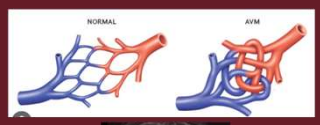
## Stroke

- **Hemorrhagic**
  - Aneurysm or AVM rupture
    - Intracerebral hemorrhage
    - Subarachnoid hemorrhage
      - Remember **Terson's syndrome**!
  - Uncontrolled hypertension #1 cause
  - Treatment:
    - Control BP and brain swelling
      - Rule out **papilledema**
    - Surgery to treat blood vessel issue and evacuate hemorrhage

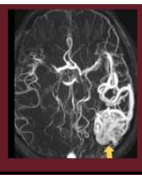
8

## Aneurysm and AVM



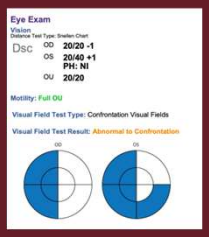




9

## 48 year old female with sudden loss of peripheral vision

- Surgical tech
- Started 2-2.5 hours ago
  - Walking into walls, dropping things, feeling clumsy



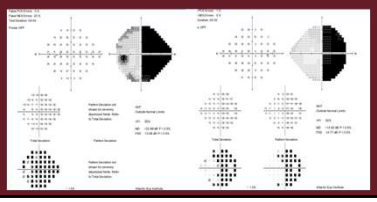

10

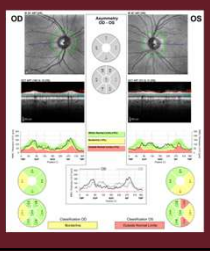
- CT/CTA head
  - 3 cm mass in the suprasellar cistern likely ICA aneurysm
  - Subarachnoid blood adjacent to the mass extending in the the sylvian fissure
  - Large left internal carotid artery aneurysm arising just distal to the left ophthalmic artery origin measuring 2.1cm x 2cm x2.6 cm. The neck of the aneurysm measures 7mm

11

## 2 months later

- S/P coil embolization ,EVD placement and 2 stents
- BCVA 20/20 OD and 20/50 OS
- Trace left RAPD






12

## Stroke Symptoms

**BE FAST**

- Balance
- **Eyes**
- Face drooping
- Arm weakness
- Speech Difficulty
- Time to call 911


13

## Stroke Prevention

**80% of strokes are preventable!**

**Modifiable risk factors**

- Hypertension
- Diabetes
- High cholesterol
- Obesity
- Smoking
- Heart disease – atrial fibrillation common

**Non-modifiable risk factors**

- Older age
- Pregnancy
- Race –African American and Hispanic more common
- Family history and genetics
- Aneurysm or AVM
- Inflammatory disease
- Viral illness

14

## Transient Ischemic Attack

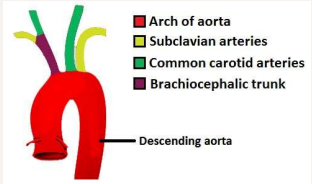

- Often called 'mini stroke' but really should be warning sign
  - Typically foreshadow stroke
    - 15% of patients
    - 1/3 of patients have a severe stroke within the first year
- Temporary blockage of blood vessel
  - Symptoms resolve within 24 hours however blockage is shorter

15


16

## Blood Flow to the Brain

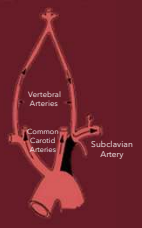
- **Heart → Aorta**
  - Ascending aorta
    - Arises from left ventricle
    - Gives rise to left and right coronary arteries that supply myocardium
  - **Aortic arch**
    - Brachiocephalic trunk
      - **Right common carotid and right subclavian artery**
    - Left common carotid artery
    - Left subclavian artery
  - Thoracic (descending)
  - Abdominal aorta



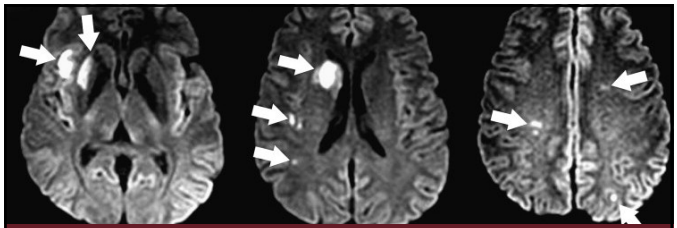

17

## Subclavian Steal Syndrome

- Proximal occlusion of the subclavian artery and reversal of flow in the ipsilateral vertebral artery away from the **brainstem, cerebellum and occipital lobe**
- **Left side is more common**
- Majority of patients are asymptomatic – symptoms can worsen with arm movements
- **Ocular symptoms can include transient visual field loss, nystagmus and diplopia**
- The presence of an arm pressure difference of greater than 20 mmHg has been proven to be a sensitive test
- The most common cause of this syndrome is artherosclerosis




18



## Clinical Correlate!

- **STARTS WITH THE HEART- CARDIOEMBOLIC STROKE**
  - Cause of ischemic stroke
  - Symptoms often sudden and maximal at onset
  - ½ involve **multiple territories**

19

## Clinical Correlate!

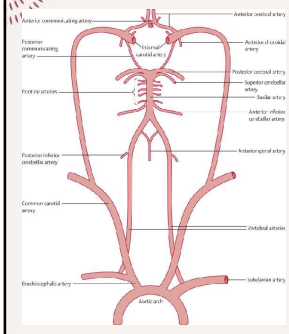
- **Atrial fibrillation**
  - Disorder of heart rhythm
  - Increases risk of stroke by 3-5 fold
- **Systolic heart failure**
- **Recent myocardial infarction**
  - 2.5% of patients have stroke after MI
- **Patent foramen ovale**
  - Affects 25% of population!
  - More common in younger patients
  - Passageway for paradoxical embolism from venous to arterial circulation
- **Aortic arch atheroma**
  - 45% of individuals over 45
  - Large, ulcerated, noncalcified or mobile are more often implicated with stroke
- **Prosthetic heart valves**
  - Treatment for moderate to severe valvular disease
  - 4.0% increased risk of stroke which decreases to <1% with anticoagulation
- **Infective endocarditis**
  - 1 in 5 cases are complicated by stroke
  - Bacteremia also implicated

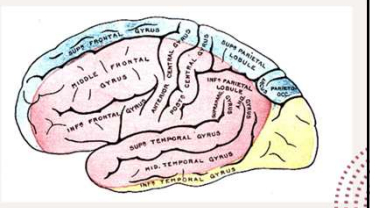
20

## Blood Flow to the Brain

| ANTERIOR CIRCULATION | POSTERIOR CIRCULATION |
|---|---|
| • Common carotids bifurcate in the neck to create external and **internal carotid artery**<br>• **Anterior cerebral artery**<br>• **Middle cerebral artery** | • **Vertebral arteries** arise from the subclavian arteries<br>• Come together at the pons to form the midline basilar artery<br>• Gives rise to posterior cerebral artery |

21



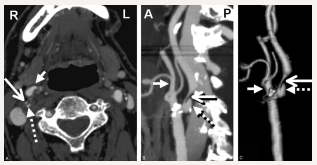



- Blue = ACA
- **Red = MCA**
- **Yellow = PCA**

22

## Clinical Correlate!

- **Carotid artery stenosis**
  - Blockage of the carotid artery secondary to plaque
  - Increases risk of stroke
  - Prevalence of 5% in general population in US
  - Diagnostic tests
    - Bruit
    - Carotid ultrasound or duplex
    - CTA/MRA
    - Cerebral angiography




23

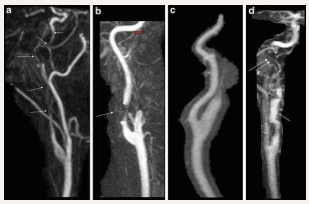
## Clinical Correlate!

- **Carotid artery stenosis**
  - Classification
    - Mild - <50%
    - Moderate- 50-79%
    - Severe- 80-99%
  - Treatment with endarterectomy
    - Patients with 50-99% or more stenosis with signs/symptoms
      - Ideally within 2 weeks of symptom onset
    - Patients with 70% or more stenosis with or WITHOUT signs/symptoms
  - Carotid artery stenting better for patients with increased comorbidities

24

## Clinical Correlate!

- **Carotid dissection**
  - Layers of the carotid artery are mechanically separated
  - Can occur extra or intracranially and lead to stroke or hemorrhage
    - Most common cause of stroke in younger patients
      - 20% of all strokes in younger patients
  - Can occur with blunt (MVA) or mild trauma (neck manipulations)
  - Increased risk with connective tissue disorders
  - Asymptomatic or symptomatic




25

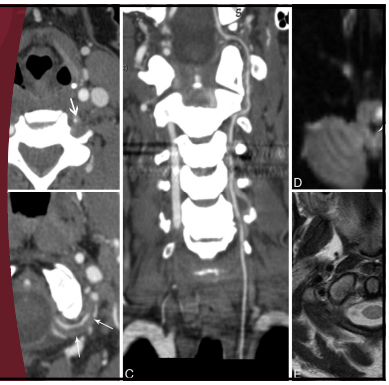
## Clinical Correlate!

- **Carotid dissection**
  - Symptoms
    - Headache
    - Facial or eye pain
    - Neck pain
    - HORNERS syndrome!
  - Treatment
    - Antiplatelets or anticoagulation to prevent stroke
    - Endovascular stenting

26

## Clinical Correlate!

- **Vertebral dissection**
  - Layers of the vertebral artery are mechanically separated
  - Majority are intracranial
  - Rare cause of stroke but more common in younger patients
  - Typically have **mild trauma**
    - Coughing, vomiting, chiropractic procedures and blunt trauma most common
  - Patients with connective tissue disorders at increased risk




27

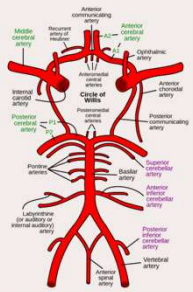
## Clinical Correlate!

- **Vertebral dissection**
  - Symptoms
    - Acute, severe neck pain and/or headache
    - Neurologic symptoms are often delayed
      - Lateral medullary syndromes and cerebellar infarctions are most common
        - Nystagmus, ataxia, loss of taste, tongue deviation towards side of lesion, INO and skew deviation, Horners syndrome
  - Treatment
    - 10% of patients die however prognosis is good for those that live
      - Extracranial more severe
    - Goal is to prevent stroke with anticoagulation

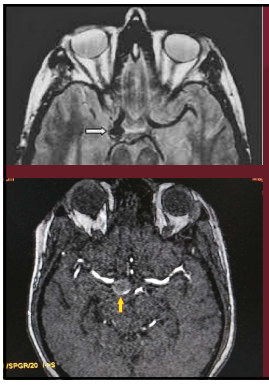
28

## Blood Flow to the Brain

- **Circle of Willis**
  - Arterial ring at the base of the brain
    - Around level of hypothalamus/cerebral peduncles
  - Connection of anterior and posterior circulatory systems




29



## Clinical Correlate!

- **Posterior communicating artery**
  - Connects anterior and posterior system
  - Runs medial to CN III
  - Aneurysm here pushes on medial aspect of CN III
    - May impact pupillary fibers first
    - RULE OF THE PUPIL
      - Complete CN III palsy with pupil involvement = aneurysm until proven otherwise
    - **MOST COMMON SITE OF ANEURYSMS**
      - 45.9% of all aneurysms with high risk of rupture

30

## Blood Flow to the Brain

- **Posterior circulation**
  - Posterior cortex = occipital lobe
  - Brainstem
    - Midline arteries supply midline structure
    - Lateral arteries supply lateral structures
    - Dorsal-lateral arteries supply dorsal-lateral structures and cerebellum
      - **Anterior inferior cerebellar artery**
      - **Posterior inferior cerebellar artery**

31

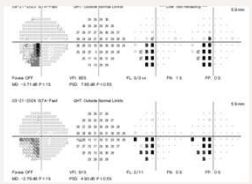
## Clinical Correlate!

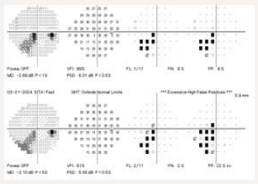
- **Occipital lobe stroke**
  - Area of most congruity
  - Macular sparing visual field defects
    - Due to dual blood supply
  - Typically vision is only symptom
    - Often complain of vision loss in one eye (eye with temporal field loss)
    - Homonymous hemianopsia most common but other presentations possible
      - Visual hallucinations
      - Prosopagnosia
        - Difficulty recognizing faces
        - Typically bilateral stroke>right sided stroke
      - Visual neglect

32

## 73 year old female with spots/images in her vision

- History of stroke 2 years prior with loss of peripheral vision – MRI w/right occipital lobe encephalomalacia
- New floaters/spots but sometimes formed images on the left side of vision
  - Dog, faces/people
  - Water-like movement
  - One episode of worsening peripheral field loss

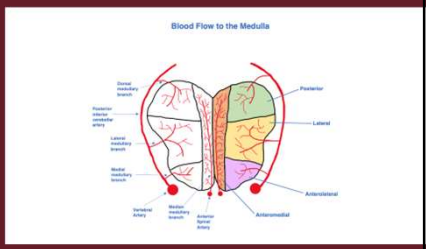




33

## Clinical Correlate!

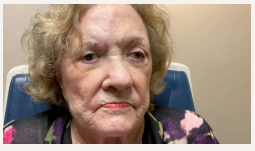
- **Wallenberg syndrome**
  - Damage to the lateral aspect of the medulla
  - Common from infarction of posterior inferior cerebellar artery
    - Horner's syndrome
    - Nystagmus
    - INO/Skew deviation

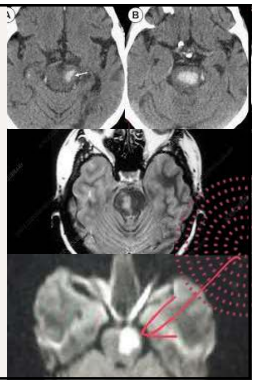



34

## Clinical Correlate!

- **Brainstem stroke!**
  - Remember INO, skew deviation, gaze palsy
  - Central nystagmus





35

## Blood Flow to the Brain

- **Anterior circulation**
  - **Anterior cerebral artery**
    - Supplies frontal, pre-frontal and supplementary motor cortex
    - ACA infracts are rare due to collateral circulation from anterior communicating artery
  - **Middle cerebral artery**
    - Most common affected blood vessel in the brain for stroke
    - Supplies a large area of the brain
      - 4 segments M1-M4

36

## Clinical Correlate!

- **Middle cerebral artery stroke!**
  - Possible stroke symptoms:
    - Contralateral sensory loss of legs, arms and lower 2/3 of face
    - Contralateral motor loss of legs, arms and face
    - **Contralateral gaze palsy** (frontal cortex – Broadmann area 8)
    - **Contralateral homonymous hemianopsia**
      - **Visual neglect** possible in right sided stroke
    - **Broca's aphasia**
      - Left sided stroke - superior division
        - Limited language – short sentences
    - **Wernicke's aphasia**
      - Inferior division
        - Jumbled words

37


38

## Case 1

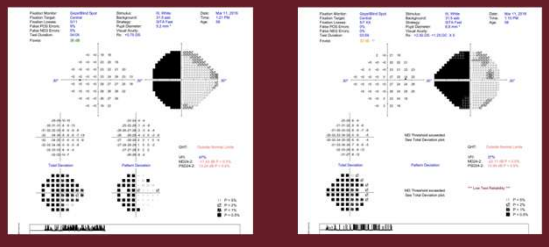
- 58 year old female with sudden vision loss to left eye
  - Woke up and only able to see half of computer screen
  - Present for 6 hours
  - Systemic history
    - Small cell carcinoma of lung – currently undergoing chemotherapy
  - Ocular history
    - PCO OS – scheduled to get YAG
    - Fuch's

39

## Case 1

| | OD | OS |
|---|---|---|
| BCVA | 20/50 | 20/70 |
| Pupils | 4.00→2.00 (-)RAPD | 4.00→2.00 (-)RAPD |
| Extraocular motility | Full and smooth | Full and smooth |
| Color vision | 14/14 | 14/14 |
| CVF (see VF) | See VF | See VF |
| IOP | 12 | 13 |
| Anterior segment | 3+ guttata, PCIOL | 3+ guttata, PCIOL, 2+ PCO |
| Posterior segment | Unremarkable | Unremarkable |

40



41

## Case 1

- Left homonymous hemianopsia
  - NEW
  - Send to ER for further work-up for stroke

42

## Case 1

- **Right occipital stroke**
  - Work-up revealed endocarditis
    - Started on IV antibiotics

43

## Case 2

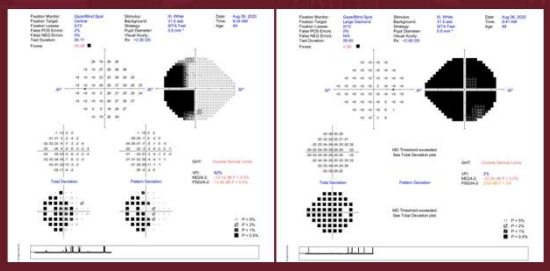
- 49 year old female with left-sided vision loss
  - Found on the ground after vomiting x 2
  - Unable to move left side of body
  - Taken to ER and CT demonstrated SAH and right temporal ICH
    - CTA demonstrated multiple ruptured right MCA aneurysms
      - Underwent coil embolization of aneurysms + R craniectomy with > 10 cm evacuation of hematoma and partial temporal lobectomy + EVD placement

44

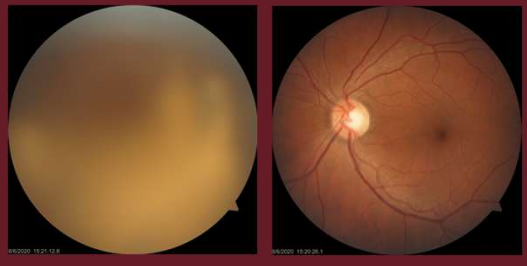
## Case 2

| | OD | OS |
|---|---|---|
| BCVA | CF @ 3 ft w EF | 20/30 |
| Pupils | 4.00→2.00 (-)RAPD | 4.00→2.00 (-)RAPD |
| Extraocular motility | full | full |
| Color vision | Unable to see plates | 14/14 |
| CVF (see VF) | See VF | See VF |
| IOP | 8 | 9 |
| Anterior segment | unremarkable | unremarkable |

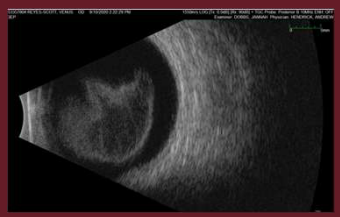
45



46



47



48

- S/P PPV MP
  - Vision improved to 20/25 OD

49

## Case 3: History

- 60 y.o male
- CC: Distortion in left eye while reading on the left side. Asymptomatic OD
  - Blinking and squinting helps slightly
  - (-)double vision
  - Started after aneurysm rupture/hospitalization
- 4 months prior
  - Presented to ED- AMS, lethargy, slurred speech S/P fall
  - CT head - extensive SAH
  - CTA head/neck - 6.4x5.6 saccular aneurysm at origin of left PCOM artery
    - S/P coil embolization + EVD
      - 2 weeks later EVD removed and pipeline flow diversion

50

## Case 3 : Clinical Evaluation

| | OD | OS |
|---|---|---|
| BCVA | 20/20 | 20/25 |
| Pupils | 4.00-->3.00mm (-)RAPD | 4.00-->3.00mm (-)RAPD |
| Confrontation Fields | FTFC | |
| Color Vision | 14/14 | 14/14 |
| IOP | 12 mmHg | 14 mmHg |
| Motility | Full (1XP all positions) | |
| Refraction | -1.50 +0.75 x 160 ADD: +2.00 | -1.25+1.00x026 ADD: +2.00 |

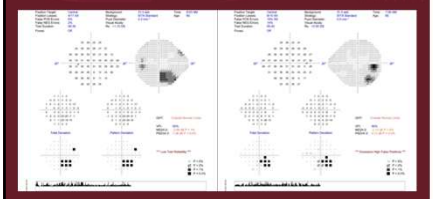
51

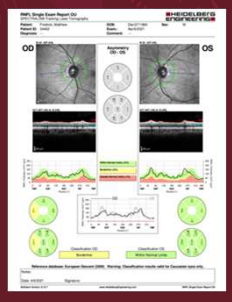
## Slit Lamp Evaluation

| | OD | OS |
|---|---|---|
| Lids/Lashes | clear | clear |
| Conjunctiva/Sclera | white | white |
| Cornea | clear | clear |
| Iris/Anterior Chamber | Flat/deep | Flat/deep |
| Lens | 1+ NS | 1+ NS |
| Posterior Segment | Trace pallor | Trace pallor |

52

## Case 3






53

## Case 3: Assessment/Plan

- **Right Homonymous Hemianopsia**
  - Likely left optic tract lesion 2/2 SAH from ruptured aneurysm
  - Recommend single vision glasses for distance and near work
  - F/U in 3 months to evaluate for visual improvement

54

## Case 3: Follow-Up

- Follow-up 1 month earlier due to new left ptosis
  - 1 week earlier- admitted for left craniotomy for residual PCOM aneurysm clipping
    - Ptosis and ghosted image since procedure
    - Numbness on top of his head
    - (-)eye pain

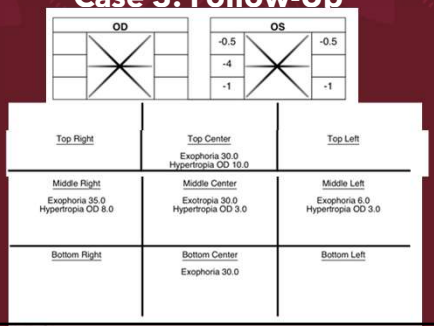
55

## Case 3: Clinical Evaluation

| | OD | OS |
|---|---|---|
| BCVA | 20/20 | 20/25 |
| Pupils | 4.00-->3.00mm (-)RAPD | 5.00-->3.50mm (-)RAPD |
| Confrontation Fields | FTFC | |
| Color Vision | 14/14 | 14/14 |
| IOP | 12 mmHg | 14 mmHg |
| Motility | Full | See next slide |
| External | Normal | Ptosis OS |

56

## Case 3: Follow-Up



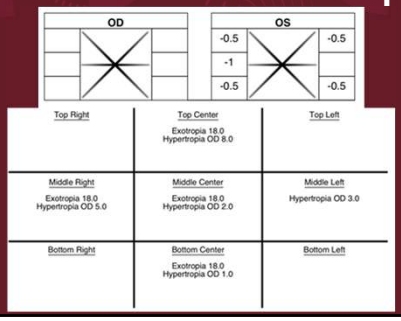

57

## Case 3: Assessment/Plan

- **Left CN III palsy**
  - 2/2 left PCOM aneurysm repair
  - 20 BI fresnel
- **Right Homonymous Hemianopsia**
  - Likely left optic tract lesion 2/2 SAH from ruptured aneurysm
  - Recommend single vision glasses for distance and near work
  - F/U in 1 month to evaluate for visual improvement and to re-evaluate ocular motility

58

## Case 3: 1 Month Follow-Up



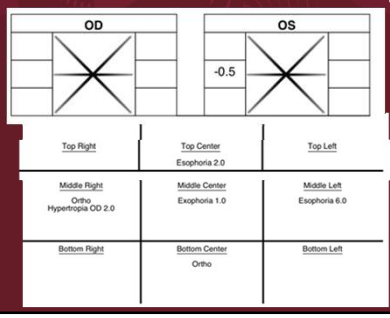

59

## Case 3: 2 month follow-Up

- Double vision resolved for past 3 weeks
- Returned to work 2 weeks ago
  - Working 4 hours with breaks every 20 minutes
  - Wants to increase
  - Heavy computer demand

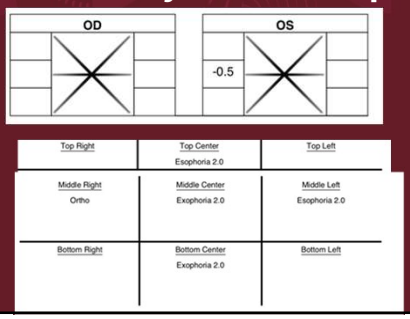
60

## Case 3: 2 Month Follow-Up



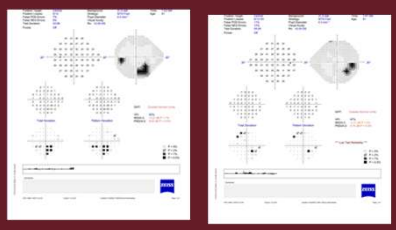

61

## Case 3: 1 year Follow-Up



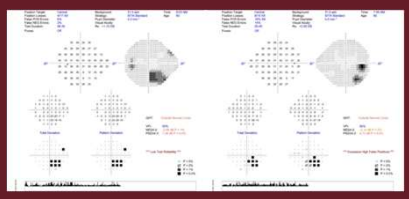

62

## Case 3: 1 year follow-up



63

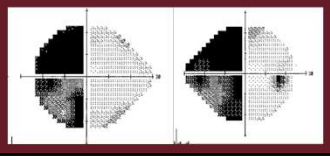
## Case 3: Initial Visual Field



64

## Case 4

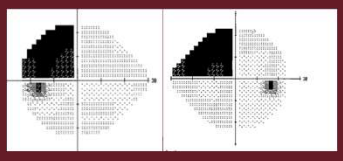
- 67 year old male presents 1-month post-stroke- right PCA involving the right occipital lobe and temporal lobe
- Notices vision is overall darker and hard to see to the left
- Also struggling with color perception



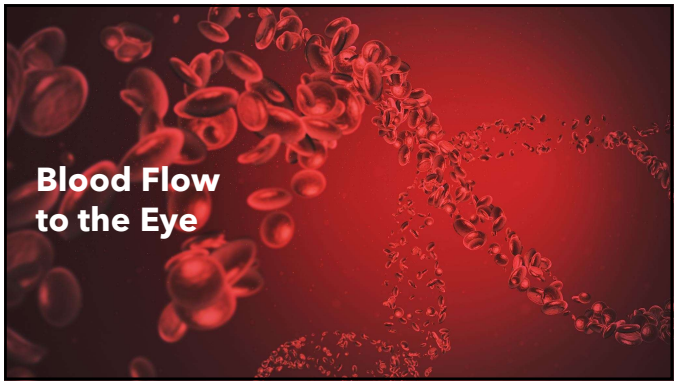
65

## 3-month follow-up

- Vision overall improved and brighter
- Colors now seem normal



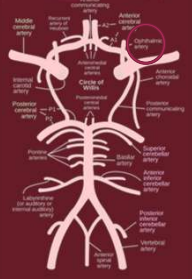
66




67

## Blood Flow to the Eye

- **Ophthalmic artery**
  - 1st major branch of internal carotid artery
  - Gives rise to:
    - **Central retinal artery**
      - Primary source of blood to the retina
        - Inner 2/3
    - **Posterior ciliary arteries**
      - Choriocapillaris
        - Outer 2/3 retina
      - Cilioretinal artery
    - **Anterior ciliary arteries**
      - Iris
      - Ciliary body



68

## Amaurosis fugax

- Fleeting or transient monocular blindness
- Sudden
- Often described as a shade or curtain over vision
- Visual loss can be altitudinal, peripheral or central
- As opposed to migraine there is no positive visual phenomenon
- Typically last 10 minutes or less and are painless

69

## Amaurosis fugax

- Associated with increased risk of stroke on ipsilateral side
- Carotid or cardiac sources should be investigated
- Rule out GCA >50
- EMERGENCY → SEND TO ER


70

## Artery Occlusions

- Occlusion of central retinal artery
- Most occur secondary to **emboli**
- **Carotid artery disease** is the most common embolic cause, followed by aortic arch atheroma and cardiac emboli
  - Emboli more commonly seen on exam with BRAO
- Other significant risk factors include hypertension, hyperlipidemia, tobacco use, diabetes mellitus, atrial fibrillation, ischemic heart disease and valvular disease
- New vascular risk factors often found after work-up

71

## Artery Occlusions

- Types of emboli
  - **Calcific**
    - a large solid, yellow white lesion
  - **Cholesterol**
    - smaller yellow refractive crystal
  - **Platelet-fibrin**
    - dull, gray-white, mobile materials that break apart as they travel through vasculature
    - commonly seen in transient non-arteritic CRAO's

72

## Artery Occlusions

- In the patient population in the European Assessment Group for Lysis in the Eye (EAGLE) trial, they found that 40% of all patients with CRAO had carotid artery stenosis of at least 70% at presentation.
- Although carotid artery stenosis is prevalent in patients with CRAO, the most important carotid abnormality to cause an embolus is the presence of plaque (~66%) compared to the amount of stenosis (~30%).
  - **Most emboli that cause CRAO are microemboli!**

73

## Artery Occlusions

- **Thrombus**
  - Occurs less often than emboli
  - **Hypercoaguable states**
    - Hematologic abnormalities
      - Familial and acquired thrombophilia (protein C deficiency and lupus anticoagulant)
      - Antiphospholipid antibodies
      - Homocysteinemia
  - **Vasculitis**
    - GCA most common
      - In a study by Hayreh et. al, of 170 patients with temporal artery biopsy confirmed GCA, 50% presented with ocular involvement. Of these, CRAO was present in 18%, cilioretinal artery occlusion in 25% and ocular ischemia in 1%
    - Lupus, sarcoidosis, granulomatosis with polyangiitis, etc.

74

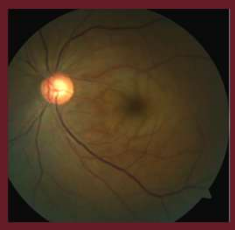
## CRAO Overview

- Considered a rare event with an incidence of 1 per 100,000 people
  - increases with increasing age
- Presents with sudden, painless loss of central and/or peripheral vision due to sudden deprivation of the retinal blood supply
  - Visual acuity loss can be variable, but is often count fingers or worse.
- Associated dyschromatopsia and an RAPD
- Visual field defect
  - Central scotoma (44.8%)
  - Paracentral scotoma (14.5%)

75

## CRAO Overview

- **Retinal findings:**
  - Retinal edema/whitening due to swelling of the retinal ganglion cells
  - Cherry red spot in the fovea due to the presence of the underlying choroidal circulation
  - Retinal arteriolar attenuation and box-carring of the retinal arterioles
  - The optic nerve usually appears normal unless the ophthalmic artery or PCA is occluded, however there may be a visible embolus on the optic disc



76

## CRAO: Time to Retinal Death

- The retina has the potential to recover if the offending agent is dislodged in a timely manner.
- Hayreh et al. investigated this in 38 elderly, atherosclerotic and hypertensive rhesus monkeys.
  - No detectable damage with CRAO for 97 minutes or less
  - Irreversible massive retinal damage at about 240 minutes
  - Between 97 minutes and 240 minutes, there was a variable degree of damage.
- **Hayreh also found that in eyes where retinal circulation was restored to normal after a CRAO of more than two hours, but less than four hours, retinal function did not show improvement until many hours or days after restoration of circulation**

77

## CRAO Entities

1. **Non-arteritic**: ~67%
   - Classic fundoscopic appearance without evidence of GCA
   - Average age of onset: 68 years old
   - Slightly more common in men (~57%)
2. Transient non-arteritic: ~16%
3. Non-arteritic with cilioretinal artery sparing: ~14%
4. Arteritic: ~4.5%

78

## CRAO Entities

1. Non-arteritic: ~67%
2. **Transient non-arteritic**: ~16%
   - On acute presentation there is vision loss and fundus findings similar to a non-arteritic CRAO, but there is normal retinal circulation on initial fluorescein angiography
   - Best visual prognosis
   - Average age of onset: 63 years old
   - Slightly more common in men (~54%)
3. Non-arteritic with cilioretinal artery sparing: ~14%
4. Arteritic: ~4.5%

79

79

## CRAO Entities

1. Non-arteritic: ~67%
2. Transient non-arteritic: ~16%
3. **Non-arteritic with cilioretinal artery sparing**: ~14%
   - There is perfusion of a small or large segment of the retina that is supplied by the cilioretinal artery
   - This will usually be evidence on a visual field test as an island of vision
   - Average age of onset and sex predilection similar to non-arteritic CRAO
4. Arteritic: ~4.5%

80

80

## CRAO Entities

1. Non-arteritic: ~67%
2. Transient non-arteritic: ~16%
3. Non-arteritic with cilioretinal artery sparing: ~14%
4. **Arteritic**: ~4.5%
   - Rarely affects the CRA alone – commonly presents with an associated anterior ischemic optic neuropathy.
   - Classical retinal findings of a CRAO with or without optic disc edema
   - Should be considered in all patients over 50 years of age with or without symptoms of GCA
   - Average age of onset: 74 years of age
   - Slightly more common in women (~63.6)

81

81

## CRAO Acute Treatment

- Goal:
  - Dislodge embolus and improve vision
- Reality:
  - There is no beneficial acute treatment for a CRAO that differs from the natural history of visual improvement
  - Attempted treatments include ocular massage, laser embolectomy, intraocular pressure lowering medications, anterior chamber parencentesis, hyperventilation or inhalation of carbogen, sublingual dinitrate and pentoxifylline to induce vasodilation, hyperbaric oxygen and thrombolysis including, intravenous or intra-arterial tissue plasminogen activator (tPA).
- Future:
  - There is still hope for thrombolysis in the future if treatment is initiated within 4-6 hours of symptoms

82

82

## CRAO Treatment: Prevention of Future Ischemic Events

- The most important management choice for patients presenting with a CRAO/BRAO is emergent referral to a stroke center of a hospital as a **CRAO/BRAO in itself is a stroke**

83

83

## CRAO Treatment: Prevention of Future Ischemic Events

- In 2013, the Stroke Council of the American Heart Association (AHA) and American Stroke Association (ASA) convened to develop a new definition of stroke for the 21st century. The new definition of stroke is 'brain, spinal cord or **retinal cell death** attributable to ischemia based on neuropathological, neuro-imaging and/or clinical evidence of permanent injury

84

84

## CRAO Treatment: Prevention of Future Ischemic Events

- **All patients with a CRAO, BRAO or transient ischemic attack (TIA) should be referred for evaluation of stroke, specifically urgent brain imaging with diffusion weighted imaging (DWI).**
  - Multiple studies have demonstrated that approximately one in four (~24%) patients with an acute CRAO or BRAO have acute brain infarcts on DWI
  - Majority of these brain infarcts were on the same side as the affected eye and in the anterior circulation. The infarcts were usually multiple and small without other neurologic symptoms
- Park et al. found that patients with an acute CRAO were significantly more likely to have a second stroke or myocardial infarction within the first 30 days with a peak during the first 1-7 days compared to controls.

85

85

## CRAO Treatment: Prevention of Future Ischemic Events

- After evaluation for acute stroke, the underlying cause of the embolus or thrombus must be investigated

86

86

## CRAO Treatment: Prevention of Future Ischemic Events

- In patients >50 years of age, GCA must be ruled out
  - Blood work: ESR, CRP, CBC with platelets
  - Temporal artery biopsy
- In patients under 50 years of age or >50 with normal GCA laboratory testing:
  - Carotid Doppler or angiography
  - Echocardiography, particularly transesophageal
  - Hypercoaguable/autoimmune work-up
- In patients with severe facial pain and an ipsilateral Horner's syndrome, a carotid artery dissection must be ruled out with a CTA of the head and neck

87

87

## Visual Prognosis

- Vision loss in CRAO is usually permanent and poor, however it can be variable and improve depending on multiple factors:
  - Time of retinal ischemia
    - <97 minutes
  - Location of embolus
    - More posterior to allow for collateral circulation
  - Type of CRAO
    - Transient has best visual potential

88

88

## Ocular Ischemic Syndrome

- Rare, vision threatening condition associated with severe carotid artery occlusive disease leading to ocular hypoperfusion
- Asymptomatic of symptomatic

89

89

## Ocular Ischemic Syndrome

- Symptoms
  - Visual loss
    - Wide range: 20/20-CF
  - Pain
    - Present 40% of time
    - 2/2 increased IOP or ischemia
    - May improve with lying down

90

90

## Ocular Ischemic Syndrome Signs

| Anterior Segment | Posterior Segment | Orbit |
|---|---|---|
| Conjunctival injection | Narrowed retinal arteries | Orbital pain |
| Corneal edema | Dilated retinal veins | Anterior or posterior segment ischemia with hypotony |
| Corneo-scleral melting | Mid-peripheral hemorrhages | Ophthalmoplegia |
| Corneal hypoesthesia | Cotton wool spots | Ptosis |
| Spontaneous hyphema | CRAO | |
| Anterior or posterior synechiae | Neovascularization | |
| Uveal ectropion | Vitreous hemorrhage | |
| Rubeosis iridis | Emboli | |
| Neovascular glaucoma | Ischemic optic neuropathy | |
| Iridocylitis | | |
| Asymmetric cataract/arcus | | |

91

## Ocular Ischemic Syndrome

- Atherosclerosis of the carotid vascular system most common
  - Typically >90% stenosis
  - OIS initial manifestation in carotid artery disease in 70% of patients
- Treatment:
  - Treat ocular sequelae:
    - IOP, intravitreal injections, etc.
  - Surgical treatment: carotid artery endarterectomy

92

## Case 5

- 48 year-old African-American female presents with a 6-hour history of reduced vision OS upon awakening
  - Stable since onset
  - Feels like she is looking through a large purple spot
- Mild discomfort around left eye/sinus area
- Pertinent negatives: headache, facial pain, nausea, vomiting, fever or recent illness

93

93

## Case 5

- Ocular History:
  - Patient at our clinic for last 18 years
    - Myopia: BCVA of 20/20 OD/OS
    - Contact lens abuse: corneal ulcer OS
- Systemic History:
  - Type 2 NIDDM x 5 years
    - LBS: 150 mg/dL; HbA1c: ?
  - Hypertension x 5 years
    - Blood pressure in office: 120/84 bpm RAS
    - Pulse: 96 bpm
  - Hypercholesterolemia x 5 years
- Medications: had not taken for last 2 weeks
  - Amlodipine, Glipizide, Lisinopril, Atorvastatin, Metformin
- NKDA

94

94

## Case 5

- Surgical history:
  - Sinus surgery x 2 years
- Social history:
  - Smoking: ¾ pack per day x20 years
  - Alcohol use: social
  - Drug use: none
- Family history:
  - Father: suffered a ruptured brain aneurysm
  - Mother: passed away 9 days following unknown complications after child birth
- Oriented to time, place and person

95

95

## Case 5

| | OD | OS |
|---|---|---|
| BCVA | 20/20 | Count Fingers at 3 feet |
| Color Vision | 14/14 | 0/14 |
| Confrontation Fields | Full to finger count | Large central scotoma and peripheral constriction greatest inferior nasally |
| Pupils | Isocoric, reactive with a >1.8 log unit APD OS | |
| Extraocular motility | Full and smooth | |
| Cover testing | Unable, due to poor acuity OS, but appeared aligned by Hirschberg reflex | |
| Exophthalmometry Base 102 mm | 24mm | 24mm |

96

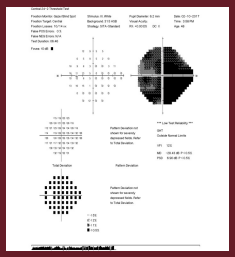
96

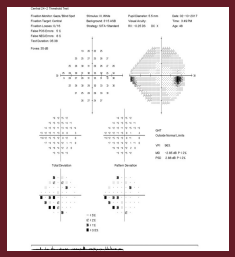
## Case 5

| | OD | OS |
|---|---|---|
| Lids/Lashes | xanthelasma | xanthelasma |
| Conjunctiva | Flat (racial) melansosis | Flat (racial) melanosis |
| Cornea | Clear and intact | Nasal stromal corneal scar |
| Iris | Flat and intact; (-)neo | Flat and intact; (-)neo |
| Anterior Chamber | Deep and quiet<br>VH: 4 | Deep and quiet<br>VH: 4 |
| Lens | Trace NS | Trace NS |
| Intraocular Pressure | 16 mmHg | 16 mmHg |

97

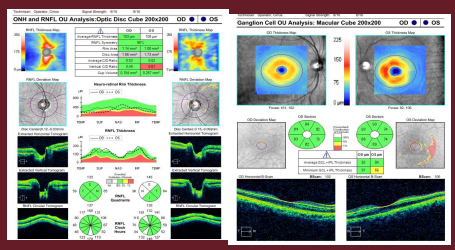
## Case 5





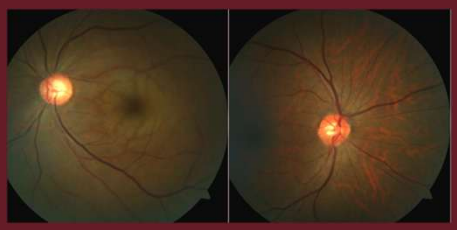
98

## Case 5




99

## Case 5



100

## Case 5

- Assessment:
  - CRAO OS
- Plan:
  - Referred emergently to the hospital for a stroke work-up Educated on the low chance of visual recovery and the importance of controlling all stroke risk factors and urged to stop smoking.
  - Follow-up in 3-4 weeks to re-evaluate her visual progression.

101

## Hospitalization

- Admitted to the hospital x 4 days
  - CT of the brain: normal
  - CTA of the head and neck: normal.
  - Transthoracic echocardiogram: normal.
  - Blood work:
    - Total cholesterol: 256 mg/dL
    - Triglycerides: 99 mg/dL
    - HDL 34 mg/dL
    - VLDL 20 mg/dL
    - LDL: 202 mg/dL
  - Discharge:
    - Increased atorvastatin dosage
    - Started on 325 mg of aspirin
    - Educated to follow-up with neurology and optometry

102

## Follow-up: 1 Week

- Returned earlier than initially recommended, as she wanted to update her glasses, as she was nervous to return to contact lens wear
- Improvement!
  - Two days after her initial vision loss, her vision started getting a little better.
  - On the third day, she noticed even more improvement and she felt like she was seeing more light than before.
  - Stopped smoking and had not had a cigarette for the last week.
- Blood pressure: elevated at 150/100 mmHg

103

103

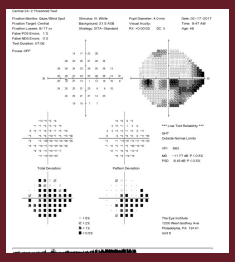
## Follow-up: 1 Week

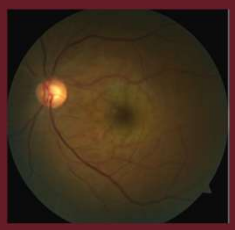
| | OD | OS |
|---|---|---|
| BCVA | 20/20 | **20/250 (inferior EF)** |
| Confrontation Fields | Full to finger count | **Central scotoma** |
| Pupils | Isocoric, reactive with a **1.5 log unit** APD OS | |
| Extraocular motility | Full and smooth | |
| Refraction | compound myopic astigmatism with presbyopia | |
| Anterior Segment | Unchanged; **(-)iris neovascularization** | |
| Intraocular Pressure | 20 mmHg | 20 mmHg |

104

104

## Follow-up: 1 Week





105

105

## Follow-up: 1 Week

- Assessment:
  - CRAO OS with mild improvement
- Plan:
  - Updated glasses prescription with polycarbonate lenses for protection
  - Prescription for blood work for hypercoagulable and hyperviscosity syndromes including:
    - CBC, ESR, CRP, ANA, platelets, PT/PTT, homocysteine, protein C/protein S, prothrombin gene mutation, anti-thrombin III, factor V leiden mutation, beta glycoprotein, anti-cardiolipin antibody and lupus anticoagulant.
  - Scheduled follow-up in three weeks.
  - Scheduled for an appointment in neurology in one week

106

106

## Follow-up: 1 Month

- Improvement!
  - Vision subjectively better
- New glasses made her feel funny
- Neurology consult:
  - Switched her from 325 mg to 81 mg of aspirin due to stomach problems
  - Changed her blood pressure medication from lisinopril to losartan
  - Ordered an MRI of the brain
- Restarted smoking: only one cigarette per day compared to ¾ of a pack.
- Had not completed the blood work given to her at the last exam.
- Blood pressure:126/92 mmHg

107

107

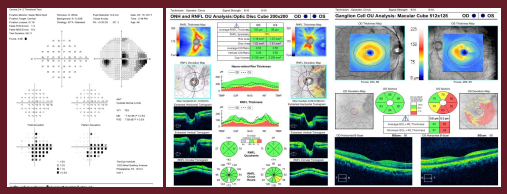
## Follow-up: 1 Month

| | OD | OS |
|---|---|---|
| BCVA | 20/20 | **20/30**<br>**PH: 20/20** |
| Color Vision | 14/14 | **14/14** |
| Confrontation Fields | Full to finger count | **Small central scotoma** |
| Pupils | Isocoric, reactive with a **0.9 log unit** APD OS | |
| Extraocular motility | Full and smooth | |
| Refraction | compound myopic astigmatism with presbyopia<br>**Refraction improved vision to 20/20 OD and OS** | |
| Anterior Segment | Unchanged; **(-)iris/angle neovascularization** | |
| Intraocular Pressure | 14 mmHg | 14 mmHg |

108

108

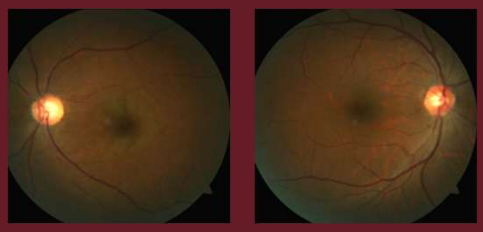
## Follow-up: 1 Month



109

109

## Follow-up: 1 Month



110

110

## Follow-up: 1 Month

- Assessment:
  - CRAO OS with significant improvement; likely transient
- Plan:
  - Re-educated on the importance of further stroke prevention and continued care with her doctors.
  - Re-educated on the importance of performing the blood work given to her at her last visit and to perform the MRI as recommended by her neurologist.
  - Updated the glasses prescription due to the change OS
  - Follow-up in 1 month to monitor for neovascularization.

111

111

## Additional Testing

- Blood work
  - ANA: 1:640
  - Anti-cardiolipin antibodies (IgM): 39
- MRI
  - Scattered areas of small vessel ischemic disease

112

112

## Case 6

- 58 year old male with transient dimming of vision when he looks upward OD
  - Present for last 2 weeks
  - No symptoms OS
  - No eye pain
- Systemic history: hypertension, hypercholesterolemia
- Ocular history: presbyopia OU

113

113

## Case 6

| | OD | OS |
|---|---|---|
| BCVA | 20/20 | 20/20 |
| Pupils | 4.00→2.00 (-)RAPD | 4.00→2.00 (-)RAPD |
| Extraocular motility | full | full |
| Color vision | 14/14 | 14/14 |
| CVF | full | full |
| IOP | 10 | 14 |
| Anterior segment | unremarkable | Unremarkable |
| Posterior segment | Peripheral hemorrhages superior, temporal and nasal | Unremarkable |

114

## Case 6

- **Concern for OIS**
  - Order carotid ultrasound

115

## Case 6

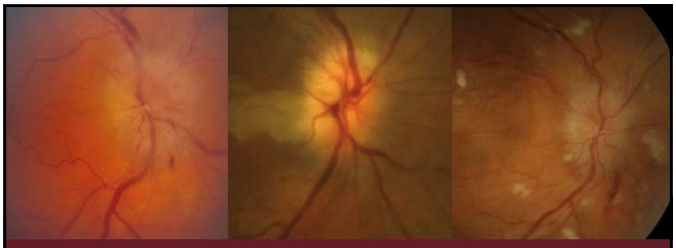
- **Concern for OIS**
  - Order carotid ultrasound
    - Returned normal
  - Order CTA head and neck
    - Returned normal
  - **Angiography**
    - **>90% occlusion of right internal carotid artery**

116

## Case 6

- **Concern for OIS**
  - Order carotid ultrasound
    - Returned normal
  - Order CTA head and neck
    - Returned normal
  - **Angiography**
    - **>90% occlusion of right internal carotid artery**
  - **Treatment**
    - **Carotid endarterectomy**
    - **Symptoms resolved**

117



## Ischemic Optic Neuropathy

118

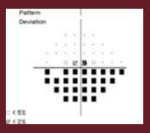
## Ischemic Optic Neuropathies

- Acute, presumably vascular optic neuropathy
- Sudden and severe vision loss
- Typically in older patients with or without vasculopathic risk factors
- **Two types:**
  - **Non-arteritic**
  - **Arteritic**

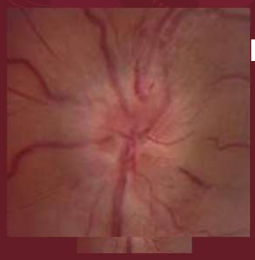
119

## Non-Arteritic Anterior Ischemic Optic Neuropathy (NA-AION)

- Acute, painless vision loss
- Males=females
- More common in Caucasian population
- 50+ with mean age of onset between 57-65
- Visual acuity is variable (not typically worse than HM)
  - 20/30 or better in 49% of cases
- RAPD
- Dyschromatopsia
- **Visual field loss follows RNFL**
  - Inferior altitudinal and arcuate the most common



120



## NA-AION

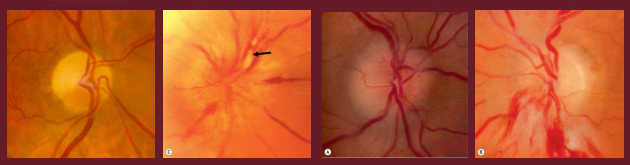
**Optic nerve appearance**

- Sectoral (25%) or diffuse (75%) disc edema
- Often has hemorrhages
- Disc may appear hyperemic with capillary dilations

**Fellow eye**

- Disc at risk = small nerve and cup

121



122

## NA-AION Work-Up

- Associated systemic risk factors – think **microvascular compromise**!!
  - Hypertension, diabetes, hypercholesterolemia
  - Smoking, sleep apnea, nocturnal hypotension, anemia, hypercoaguable states, optic disc drusen, ocular and non-ocular surgery
- Associated medications
  - Phosphodiesterase type-5 inhibitors (debatable)
    - Erectile dysfunction medications
  - Amiodarone
  - Semaglutide

123

Popular Prescription Weight Loss Drugs Linked to Uncommon Blinding Condition

*Jul 3, 2024*

Nutrition & Weight Management | Ophthalmology | Research

**The News Feed**

Published May 16, 2024 • By Staff
DIABETES, NEWS

Risk of NAION Higher in Patients on GLP-1 Drugs for Diabetes or Weight Loss

**Exposure to agonists such as Ozempic and Mounjaro was associated with 3.24-fold higher likelihood.**

Original Investigation FREE

July 3, 2024

**Risk of Nonarteritic Anterior Ischemic Optic Neuropathy in Patients Prescribed Semaglutide**

Jimena Tatiana Hathaway, MD, MPH[1,2,3]; Madhura P. Shah, BS[2,3]; David B. Hathaway, MD[4]; et al

Author Affiliations | Article Information

*JAMA Ophthalmol.* 2024;142(8):732-739. doi:10.1001/jamaophthalmol.2024.2296

124

**Results** Among 16 827 patients, 710 had T2D (194 prescribed semaglutide; 516 prescribed non–GLP-1 RA antidiabetic medications; median [IQR] age, 59 [49-68] years; 369 [52%] female) and 979 were overweight or obese (361 prescribed semaglutide; 618 prescribed non–GLP-1 RA weight-loss medications; median [IQR] age, 47 [32-59] years; 708 [72%] female). In the population with T2D, 17 NAION events occurred in patients prescribed semaglutide vs 6 in the non–GLP-1 RA antidiabetes cohort. The cumulative incidence of NAION for the semaglutide and non–GLP-1 RA cohorts over 36 months was 8.9% (95% CI, 4.5%-13.1%) and 1.8% (95% CI, 0%-3.5%), respectively. A Cox proportional hazards regression model showed higher risk of NAION for patients receiving semaglutide (hazard ratio [HR], 4.28; 95% CI, 1.62-11.29); $P < .001$). In the population of patients who were overweight or obese, 20 NAION events occurred in the prescribed semaglutide cohort vs 3 in the non–GLP-1 RA cohort. The cumulative incidence of NAION for the semaglutide vs non–GLP-1 RA cohorts over 36 months was 6.7% (95% CI, 3.6%-9.7%) and 0.8% (95% CI, 0%-1.8%), respectively. A Cox proportional hazards regression model showed a higher risk of NAION for patients prescribed semaglutide (HR, 7.64; 95% CI, 2.21-26.36; $P < .001$).

125

## NA-AION Work-Up

- **NA-AION IS A DIAGNOSIS OF EXCLUSION!**
- Need to rule out GCA! (THIS IS THE MOST IMPORTANT)
  - Presentation can be very similar
  - ESR/CRP/platelets
  - If suspicion high = TAB
- Need to rule out compressive mass/atypical optic neuritis
  - MRI brain/orbits W/W/O contrast
  - Particularly if edema persists > 4 weeks

126

## NA-AION Pathogenesis

**Unknown/unproven → Likely multifactorial**

- Transient disruption in the circulation of the optic nerve head
  - →Hypoperfusion + ischemia
- **Why disc at risk?** = localized swelling in a small, fixed space = arteries become more easily compressed = anterior compartment syndrome

127

## NA-AION Prognosis

- No known beneficial treatment
- Vision typically stabilizes within 2-3 months
- **Vision may improve by up to 3 lines in 43%**
- VF defects less likely to improve
- 5% risk of reoccurrence in same eye
- 15% chance of involvement in fellow eye in 5 years

128

## Arteritic Ischemic Optic Neuropathy (AION)

- Acute, **painful** vision loss
- Most commonly from giant cell arteritis
- Females>males
- More common in *Caucasian* population
- 50+
- **Profound vision loss (HM to NLP)**
- RAPD
- Dyschromatopsia
- Visual field loss
- May have associated headache, jaw pain, scalp tenderness

129

## Giant Cell Arteritis (GCA)

- Granulomatous medium and large-vessel vasculitis
- Most common vasculitis in US adults
  - Most common affected arteries include:
    - **Temporal** – this is where we biopsy – superficial
    - **Ophthalmic**- choroid + optic disc/retina/cilioretinal artery
    - **Posterior ciliary arteries** – AION
    - **Vertebral arteries** – stroke!
- **Medical emergency!**

130

## Giant Cell Arteritis

- Systemic features
  - New onset headache, scalp tenderness, jaw claudication, fever, fatigue, malaise, weight loss and polymyalgia*
    - 40-60% of GCA patients also have PMR; 16-21% of PMR patients have GCA
    - Region over temporal artery may be sensitive to touch
    - Temporal artery abnormalities
- Visual features
  - **Temporary or permanent**
    - Double vision
    - Amaurosis fugax
    - AION
    - Ophthalmic artery occlusion

131

## AION

- Optic disc features
  - **Chalky pale disc edema**
  - May have associated CWS
  - May have associated cilioretinal artery occlusion or choroidal ischemia

132

133

## GCA Work-Up

- If suspicion is high – **send to the ED** for prompt initiation of steroids (even before diagnosis is confirmed)
- Serologic studies
  - ESR (elevated)
    - Normal values:
      - Women: (Age +10)/2
      - Men: Age/2
  - CRP (elevated)
  - Platelets (elevated)
- Imaging
  - Cranial ultrasound – not used often as sensitivity is low
    - Hypoechoic ring – non-compressible ring around lumen that represents thickening due to inflammation
- **Temporal artery biopsy** – gold standard
  - False negatives can occur due to skip lesions – specimens should be >2 cm to avoid this

134


## GCA is a clinical diagnosis

- Normally: Disease → Diagnose → Treat
- **GCA:** Disease → **TREAT** → Diagnose
- Initial Treatment is high dose **CORTICOSTEROIDS**
  - No vision loss:
    - Oral Steroids 1 – 1.5 mg/Kg
  - Vision loss, amaurosis fugax, or disc edema:
    - **IV methylprednisolone**

135

## GCA Treatment

- Recommend management per rheumatology!
- Goal – prevent further vision loss and stroke
- If vision loss is present need pulse dose of IV corticosteroids 0.5-1.0 g methylprednisolone for 3-5 days
  - Followed by very slow oral taper – typically over 1-2 years
    - Oral also given if no visual symptoms
  - Inflammatory markers/symptoms will need to be monitored during taper
    - Relapses are common - 34-62% of patients
    - Adverse effects of long-term glucocorticoids
      - GI
      - Osteoporosis
      - Hyperglycemia
      - Immunocompromised
      - Psychiatric

136

## GCA Treatment

- **Tocilizumab (Actemra)**
  - FDA approved in 2017 for GCA
  - GiACTA trial
  - Studied the effect of tocilizumab on rates of relapse during glucocorticoid tapering in 251 GCA patients over the course of 52 weeks.
    - Tocilizumab weekly plus prednisone, with prednisone tapered over 26 weeks
    - Tocilizumab every other week plus prednisone tapered over 26 weeks
    - Placebo plus prednisone tapered over 26 weeks
    - Placebo plus prednisone tapered over 52 weeks.

137

## GCA Treatment

- **Tocilizumab (Actemra)**
  - FDA approved in 2017 for GCA
  - GiACTA trial
  - At 52 weeks, the rates of sustained remission were:
    - 56% with tocilizumab weekly
    - 53% with tocilizumab every other week
    - 14% with placebo plus 26-week prednisone taper
    - 18% with placebo plus 52-week taper.

1. The cumulative dose of prednisone in tocilizumab recipients was significantly less than in placebo recipients.
2. Rates of adverse events were similar.
3. Either weekly or every other week, was more effective than prednisone alone at sustaining glucocorticoid-free remission in patients with GCA.

138

## AION Prognosis

- Highly dependent on when steroids were started
  - If ischemic optic neuropathy already occurred- risk of improvement is very low
  - Vision generally stabilizes once steroids are started but may progress up to 5 days
  - 80% of patients LP/NLP
- Risk to fellow eye
  - 9% on steroids
  - 20-62% of untreated patients

139

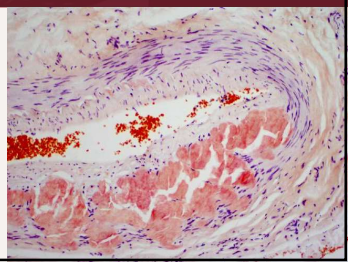
## GCA Mimicker: Amyloidosis

- Systemic or local disorder caused be deposition of fibrils made up of proteins throughout the body. Can affect any organ system and/or can circulate in the blood and deposit in vessels
- Often associated with plasma cell dyscrasias
- Ocular amyloidosis usually presents as conjunctival lesions and periorbital purpura however it can also manifest in the orbit, retina, vitreous and cornea
- Amyloid can also deposit in the temporal artery and can mimic GCA symptoms but can also cause ischemic optic neuropathy

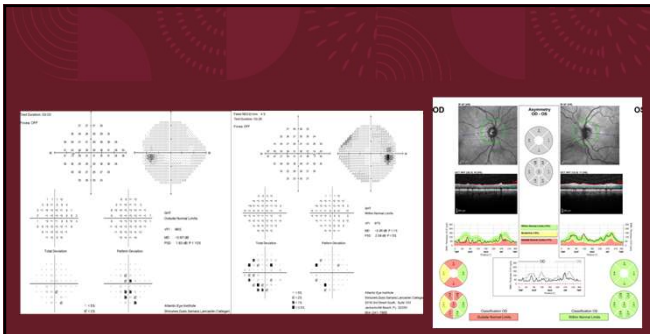
140

## Amyloidosis

- More common in men>females
- Age of diagnosis ~72
- Abnormal ESR/CRP
- Temporal artery biopsy is diagnostic
  - Both will show giant cells
  - Congo red stain can differentiate and will be positive in amyloidosis



141



142

## Case 7

- 71 year old male with double vision
- Onset: 2-3 weeks ago – resolves when he covers either eye
- Associated symptoms:
- Vision OD went 'out like a white screen' for 10-15 minutes 3 weeks ago – has happened 2-3 times total since then
- Headache/eye pain: frontal and behind right eye
- LEE: 30+ years ago – wears reading glasses
- Systemic history: HTN, hypercholesterolemia
- Medications: amlodipine, enalapril, lovastatin
- Social history: unremarkable

143

## Case 7

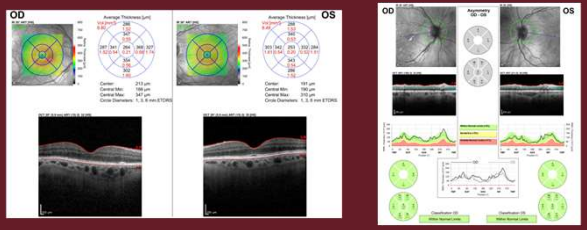
| | OD | OS |
|---|---|---|
| **BCVA** | 20/80 PD: 20/40 | 20/40 PH 20/30 |
| **Pupils** | 4.50→3.50mm (-) RAPD | 5.00→4.00mm (-) RAPD |
| **Confrontation Fields** | Full to finger count | |
| **Color vision** | 14/14 | 14/14 |
| **IOP** | 14 mmHg | 14 mmHg |
| **Motility** | -1.5 abduction | full |
| **Slit lamp exam** | 3+ CS, 2+ NS | 3+ CS, 2+ NS |
| **DFE** | 0.15, drusen nasal macula | 0.20 |

144

## Case 7


145

## Case 7




146

## Case 7

- Right abduction deficit + transient visual loss OD
- Concerning for unifying systemic cause
- Labs: CBC, ESR, CRP, BUN, creatinine
  - STAT: Going to PCP today
- MRI brain and orbits W/W/O contrast + MRA

- Cataracts- visually significant – will address later

147

## Case 7

- Work-up:
- **ESR: 41**
- **CRP: 160.5**
- **Platelets: 586**

148

## Case 7

- **Work-up:**
- **ESR: 41**
- **CRP: 160.5**
- **Platelets: 586**
- **Sent to ER**
- **MRI: 2 small acute strokes**
- **TAB: positive for Giant Cell Arteritis**
- **Started on IV steroids – discharge on oral steroids + Actemra**

149

## Case 7

- Follow-up: 3 months
- Taking Actemra weekly + 15 mg prednisone
- Double vision resolved
- One episode of vision dimming that lasted 2 seconds weeks ago and nothing since

150

## Case 7

| | OD | OS |
|---|---|---|
| **BCVA** | 20/50 PD: 20/25 | 20/30 PH 20/25 |
| | | |
| **Pupils** | 4.50→3.50mm (-) RAPD | 5.00→4.00mm (-) RAPD |
| **Confrontation Fields** | Full to finger count | |
| **Color vision** | 14/14 | 14/14 |
| **IOP** | 18 mmHg | 19 mmHg |
| **Motility** | full | full |
| **Slit lamp exam** | 3+ CS, 2+ NS | 3+ CS, 2+ NS |
| **DFE** | 0.15, drusen nasal macula | 0.20 |

151

## Case 7


152

## Case 8

- 49 year old female with blurry vision OD started 2-3 weeks prior with colorful visual aura episodes
- No eye pain or headaches
- Vision has worsened since the onset
- Systemic history: anxiety disorder, migraines
- Ocular history: LASIK OU
- Medications: sertraline
- Social history: Occasional alcohol usage

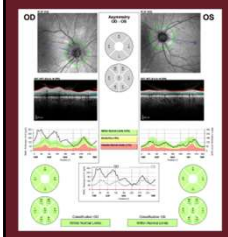
153

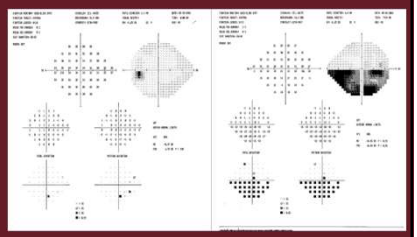
## Case 8

| | OD | OS |
|---|---|---|
| **BCVA** | 20/400 | 20/20 |
| **Pupils** | 4.00→3.00mm (+) RAPD | 4.00→3.00mm (-) RAPD |
| **Confrontation Fields** | Restricted inferior OD, Full to finger count OS | |
| **Color vision** | 0/14 | 14/14 |
| **IOP** | 15 mmHg | 15 mmHg |
| **Motility** | full | full |
| **Slit lamp exam** | unremarkable | unremarkable |
| **DFE** | Frisen grade 2 disc edema | normal |

154

## Case 8



155

## Case 8

- Assessment/Plan
- **Optic disc edema OD**
  - No pain, but concerning for atypical optic neuritis
  - Send to ER for MRI brain and orbits W/W/O contrast, blood work for AQP4 and MOG antibodies, RPR, FTA-ABS, ANA, SSA, SSB, ACE, quantiferon gold, RF and neurology consultation

156

## Case 8

- **Hospital course:**
- MRI – unremarkable
- Lumbar puncture – unremarkable
- MRI spine- unremarkable
- Bloodwork- unremarkable
- Started on IV steroids for ONTT to see if any improvement in vision

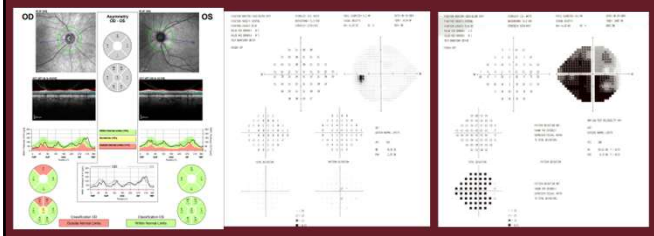
157

## Case 8

- 1- month follow-up
- Currently tapering prednisone, vision improved somewhat and then plateaued
- BCVA OD: 20/200, OS: 20/20
- Endorses taking Wegovy from June to February but stopped shortly before this event occurred

158

## Case 8




159

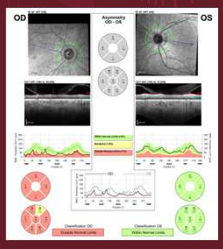
## Case 8

- Assessment/Plan
- **Optic disc edema OD**
  - Unremarkable work–up
  - Likely NA-AION from Wegovy vs undiagnosed OSA – recommend sleep study
  - Recommend 2nd opinion with neuro-op due to unusual presentation

160

## Case 8

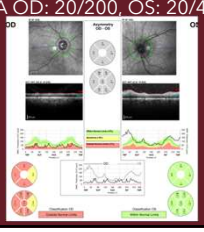
- 3-month follow-up
- Vision unchanged
- Waiting to get sleep study scheduled
- Stared ginko biloba by neuro-OMD
- Vision 20/100 OD and 20/20 OS

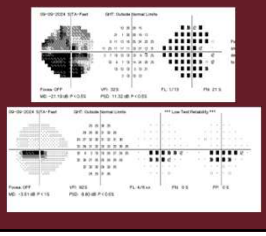



161

## Case 8

- Presents urgently 2 weeks later for changes to vision OS
- BCVA OD: 20/200, OS: 20/40





162

## Case 8

- **Assessment/Plan**
- Bilateral NA-AION
  - Completed sleepy study: diagnosed with moderate OSA
  - Started on 80 mg prednisone by neuro-OMD
  - Low vision consult

163

## Stroke and Ocular Examination

- **Homonymous visual field defects**
  - MAY improve within the first year – depends on severity and duration of ischemia
    - Typically within the first 3-6 months
  - Need to consider impact with driving
    - Varies by state
- **Diplopia**
  - More likely to improve than visual fields
  - Can exchange Fresnel prism every few months
- **Retinal artery occlusions**
  - Least likely to improve
  - Monitor for neovascularization, particularly within the first 3 months
- **GCA**
  - Consider in any patient >50
- **NA-AION**
  - Take a good history: high risk meds, vascular risk factors – big 5

164

## Questions??

- Kelsey.leigh.m@gmail.com

165