NEURO-OPHTHALMIC MANIFESTATIONS OF STROKE
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Cost of Stroke

☐ To the US society: $68.9 billion per year
☐ Includes direct cost of care
☐ And indirect cost of lost productivity

STROKE is the leading cause of disability in the US, and the #5 cause of death in the US

Cost of Stroke

☐ To the individual: PRICELESS
☐ Loss of vision, diplopia, altered speech, paralysis, aphasia
☐ Loss of ability to drive, loss of independence, loss of life

Incidence of First-time Stroke

• > 800,000 per year
• Steadily increasing
• 1/5 die within a year of 1st stroke
Stroke Statistics

• More women than men have strokes
  • Men: 370,000/yr
  • Women: 425,000/yr

80% of strokes are preventable (NSA, 2009)

• Deaths from stroke are dropping (due to regional stroke centers)
• We still have a long way to go

Role of Optometrists

• Identify Acute Stroke / TIA
  • Get patient immediate (emergent) work-up/treatment as necessary
• Determine if Deficits are from old strokes or if they indicate a new stroke/other process
• Preventative Measures
  • Identify stroke risk factors
  • Educate patients
  • Refer as necessary
• Assess visual function after stroke
  • Improve visual function where possible
  • Comment on driving ability

STROKE

• CVA – Cerebrovascular accident

  • Etiology
    – Ischemic (80-85%)
      – Thrombosis
      – Embolism
    – Hemorrhage (15%)

Stroke Prevention

• Eat Healthy
• Do More Physical Activities
• Quit Smoking & Drinking
• Keep a tab on your health
Ischemic stroke from Emboli

- Mitral stenosis
- Endocarditis
- Myocardial infarction
- Patent Foramen Ovale – test with bubble study
- Congestive Heart Failure
- Atrial Fibrillation (irregular pulse)

Ischemic stroke from Thrombus

- Arterial stenosis - atherosclerosis
- Sickle cell anemia
- Protein C deficiency
- Hyperhomocysteinemia
- Other hypercoaguable states
Other Ischemic Stroke Risk Factors

- Hypertension
- Diabetes
- Hypercholesterolemia
- Tobacco Use
- Sleep Apnea
- Poor Diet
- Lack of exercise

Hemorrhagic Stroke

- Intracerebral Hemorrhage
  - Hypertension
- Sub-arachnoid Hemorrhage
  - Ruptured aneurysm
- More likely present with symptoms of increased ICP
  - Nausea
  - Vomiting
  - Headache
  - Change in consciousness

STROKE WARNING SIGNS

**Spot a stroke F.A.S.T.:**
- Face Drooping
- Arm Weakness
- Speech Difficulty
- Time to call 9-1-1

Hemorrhagic Stroke

- STAT head CT in ER to identify Hemorrhagic stroke
  - Treatment
    - Evacuation of the hemorrhage - Craniotomy
    - Observation (if small)
Beyond F.A.S.T. – Other Symptoms You Should Know

- Sudden numbness or weakness of the leg
- Sudden confusion or trouble understanding
- Sudden trouble seeing in one or both eyes
- Sudden trouble walking, dizziness, loss of balance or coordination
- Sudden severe headache with no known cause

Classic signs of a stroke. (Source: NINDS, 2013.)

Who Has A Stroke

- More common in those over age 65
- But, can happen at any age
- Even in children and babies

STROKES CAN HAPPEN AT ANY AGE
Pediatric stroke can happen in infants, children and even before birth.
Stroke in pts under age 50

- Blood dyscrasias (sickle cell, Factor V Leiden, Hyperhomocysteinemia, etc)
- Patent foramen ovale – bubble study
- Oral contraceptives (especially in women over age 35, with a hx of migraines and smoking)
- Drug use (Cocaine)
- NOT GCA

Who Has A Stroke

ACUTE STROKE (EMERGENCY)

- Must assume that every acute stroke is hemorrhagic
- CT is done immediately to r/o bleeding
- Blood is seen better on CT than on MRI
- If no blood is noted, and stroke is not seen, MRI is done with diffusion weighted imaging (DWI) to identify acute ischemic infarct

If you suspect an acute stroke

- Do not complete your whole eye exam
- Only do what you need to confirm your stroke suspicion
- Call 911 immediately
- Tell the dispatcher the patient has had an acute stroke

Time is Brain!
(for BOTH ischemic and hemorrhagic stroke)

With a stroke... time matters.
Get The Patient to the RIGHT ER

Stroke Team Stops Brain Loss

• Advanced Comprehensive Stroke Center**
  – Find out where the closest one to you is
  • This differs from an Advanced PRIMARY Stroke Center

Stroke Work-Up

- Acute Stroke
  - ER ASAP
  - CT – r/o hemorrhagic stroke
  - MRI with DWI (diffusion weighted imaging)
  - CTA
  - EKG
  - Echocardiogram
  - Trans-thoracic (TTE)
  - Trans-esophageal (TEE)
  - Carotid Doppler US

- Labs
  - CBC, chem panel, hypercoaguable states, lipid panel, homocysteine, ESR, C-reactive protein, platelet count, etc.

ACUTE ISCHEMIC STROKE

MRI with diffusion weighted imaging (DWI)

Look for bright areas of infarct

Time to brain death based on % normal blood flow

<table>
<thead>
<tr>
<th>% NORMAL BLOOD FLOW</th>
<th>TIME TO BRAIN DEATH</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO flow</td>
<td>10 minutes</td>
</tr>
<tr>
<td>&lt;30% normal flow</td>
<td>1 hour</td>
</tr>
<tr>
<td>30-40% normal flow</td>
<td>Hour to several hours</td>
</tr>
<tr>
<td>With collateral and residual flow</td>
<td>Up to 6 hours</td>
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</tbody>
</table>
rt-PA

National Institute of Neurologic Disorders and Stroke (NINDS)
1992: Recombinant tissue type plasminogen activator (rt-PA) study group. FDA approval in 1996.

Early administration of t-PA benefited pts with acute ischemic stroke (= MEDICAL EMERGENCY)

Initially benefit of t-PA thought to occur in 3 hour window from onset of stroke symptoms. More recently, window has been extended to 4.5 hours (8 fold improvement of ischemic stroke outcomes)

Risk of rt-PA: Hemorrhage (5.2%)

Pts excluded from expanded 4.5 hr t-PA window
- Pts over age 80
- Pts taking blood thinners, regardless of INR
- Pts with hx of stroke
- Pts with hx of diabetes

Actilyse [Alteplase] - TPA

Glycoprotein
Activates plasminogen
to plasmin.
Dissolution of the fibrin clot after binding it.
Metabolized by liver.
Half-life is 4-5 min.
Package: 50mg.

rt-PA

Effective in ¼ to 1/3 of patients

If patients are not candidates, the time frame has passed, or the treatment was ineffective, there is now another alternative treatment option........
The Stent Retriever

- Tiny wire cage
- Threaded through a catheter into a blood vessel in the groin
- Guided up to the blocked artery in the brain
- Cage opens up and captures the clot
- The stent, along with the clot, is removed
- Immediately blood begins flowing again to the brain

Updated Stroke Guidelines Back the Use of Stent Retrievers in Select Patients

- Key Points: AHA/ASA revise guidelines for early management of acute ischemic stroke
- Stent retrievers recommended after IV fibrinolysis in specific circumstances

2015 AHA/ASA Focused Update of the 2013 Guidelines for the Early Management of Patients With Acute Ischemic Stroke Regarding Endovascular Treatment

A Guideline for Healthcare Professionals From the AHA/ASA

The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists.

Stroke. 2015;46:3020-3035.

July 2015

(Landmark change in Stroke Care)

- American Heart Association and American Stroke Association issued guidelines recommending the use of the stent retriever in concert with tPA.
- Based on 5 studies in the New England Journal of Medicine found that stent retrievers:
  - reduced disability
  - improved neurological function
  - shortened recovery time
  - increased rate at which stroke survivors regain function.

ORIGINAL ARTICLE
Stent-Retriever Thrombectomy after Intravenous t-PA vs. t-PA Alone in Stroke
Jeffrey L. Saver, M.D., Mayank Goyal, M.D., Alain Bonafe, M.D., H...

June 11, 2015

Conclusions
In patients receiving intravenous t-PA for acute ischemic stroke due to occlusions in the proximal anterior intracranial circulation, thrombectomy with a stent retriever within 6 hours after onset improved functional outcomes at 90 days. (Funded by Covidien; SWIFT PRIME ClinicalTrials.gov number, NCT01607461.)
AHA / ASA Guidelines regarding Stent Retriever

- Brand of Stent Retriever
  - Medtronic’s Solitaire
  - Stryker’s Trevo ProVue

- Conditions for use
  - adult patient
  - treated within six hours of the onset of stroke symptoms
  - clot in a large artery (ICA or proximal MCA)
  - brain imaging shows the brain is not permanently damaged

Used primarily at advanced comprehensive stroke centers

Transient Ischemic Attack (TIA)

- Neurologic deficit that resolves within 24 hours
- 80% resolve within 1 hour
- 10% of pts with TIA go on to have a stroke in 90 days

- TIA = temporary (includes transient vision loss)
- Stroke = permanent (includes CRAO and BRAO)
- NEED TO BE TREATED THE SAME!!

(more on this later...)

RISKS FOR STROKE

- ELEVATED BLOOD PRESSURE
- IRREGULAR PULSE
  - Atrial fibrillation
  - Atrial flutter

IMPORTANT IN-OFFICE TESTS

- CHECKING BLOOD PRESSURE
  - MANUAL VS AUTOMATIC
- CHECKING PULSE
  - MANUAL VS AUTOMATIC
EXAMPLES OF END-ORGAN DAMAGE
- RETINOPATHY
- UNSTABLE ANGINA
- PULMONARY EDEMA
- STROKE
- INTRACRANIAL HEMORRHAGE

HOW DO YOU MANAGE ELEVATED BLOOD PRESSURE?

PULSE ASSESSMENT
• RADIAL PULSE (WRIST NEAR BASE OF THUMB)
• IF CAN’T FEEL RADIAL PULSE, TRY CAROTID PULSE (LATERAL NECK BELOW JAW)

WHY DO WE EVEN CHECK PULSE?

3 out of 4 people who have a stroke for the first time have hypertension.
Atrial fibrillation is present in 1 out of 5 strokes.

American Heart Association

RISKS FOR STROKE
• ASYMMETRIC BLOOD PRESSURE
• ASYMMETRIC PULSE
  – Subclavian Steal Syndrome

SUBCLAVIAN STEAL SYNDROME
• retrograde blood flow in the vertebral artery associated with proximal ipsilateral subclavian artery stenosis or occlusion
• usually in the setting of subclavian artery occlusion or stenosis proximal to the origin of the vertebral artery

Subclavian Steal Syndrome
• patients may develop upper limb ischemic symptoms due to reduced arterial flow in the setting of subclavian artery occlusion
• they may develop neurologic symptoms due to posterior circulation ischemia associated with exercise of the ipsilateral arm
Location of stroke (infarct)

- Anterior Circulation
  - Carotid artery
  - Anterior cerebral artery
  - Middle cerebral artery
- Posterior Circulation
  - Vertebro-basilar
  - Posterior cerebral artery

The location of the involved circulation/infarct determines the clinical presentation

Knowing the anatomy and its corresponding function is key to stroke localization

NEURO-OPHTHALMOLOGY OF STROKE (Anterior Circulation)

- AMAUROSIS FUGAX & RETINAL STROKE (CRAO, BRAO)
- OCULAR ISCHEMIC SYNDROME
- CAROTID ARTERY DISSECTION
- VISUAL FIELD NEGLCET
- Supranuclear gaze palsy/preference

NEURO-OPHTHALMOLOGY OF STROKE POSTERIOR CIRCULATION (Vertebral-basilar)

- BRAINSTEM MOTILITY DISORDERS
  - INTERNUCLEAR OPHTHALMOPLEGIA
  - SKEW DEVIATION
  - TOP OF THE BASILAR SYNDROME
    - (Dorsal Midbrain Syndrome)
- HOMONYMOUS HEMIANOPIA
- DISORDERS OF VISUAL ASSOCIATION CORTEX

APHASIA

- A defect in language processing caused by dysfunction of the dominant cerebral hemisphere
- Both spoken and written language can be affected
- **BROCA’S AREA**: Responsible for articulation of sounds that produce words and constitute speech
  - If damaged – expressive aphasia
- **WERNICKE’S AREA**: Enable particular sequences of sounds to be identified and comprehended as meaningful words
  - If damaged – receptive aphasia
Almost all areas of the brain, when damaged, result in visual manifestations:
- Occipital lobe – homonymous hemianopia
- Cerebellum – ataxia, nystagmus
- Brainstem – diplopia, skew deviation, INO, nystagmus
- Cerebrum– supranuclear gaze palsy

We will use cases to demonstrate multiple visual effects of stroke.

Case 1

- Complete vision loss of the right eye that she noticed upon waking this morning around 1:00 am.
- She reported that the vision began to improve around 6:00 am at which point she could see shadows and lights.
- She denies any ocular pain or GCA symptoms.

55 Year Old Woman

- Complete vision loss of the right eye that she noticed upon waking this morning around 1:00 am.
- She reported that the vision began to improve around 6:00 am at which point she could see shadows and lights.
- She denies any ocular pain or GCA symptoms.

Systemic History
- Hypercholesterolemia for 2 years (not treated)
- Iron deficiency anemia

Ocular History
- Mild cataracts and primary open angle glaucoma bilaterally

Medications
- Iron, fish oil, vitamin B-12, multivitamin.
- Given medication in the past for hypercholesterolemia, did not use it because of side effects.
- Her PCP apparently recently prescribed cholesterol mediation again, but the patient has not yet had it filled.

BCVA: HM at 3 feet OD and 20/20- OS
(+ ) RAPD OD >1.8 log
CF: Severe constriction OD, full OS
Ocular motility testing: normal
SLE: Mild lens changes OU
GAT: 14 mmHg OD and 13 mmHg OS
BP: 150/70 RAS
Pulse: 70 bpm (regular)
Retinal ischemia vs cerebral ischemia

- SAME MECHANISMS!
- SAME Guidelines for
  - CRAO
  - BRAO
  - Transient vision loss

Any patient with suspected TIA or those with acute retinal ischemia should be evaluated urgently in order to identify those at high risk of immediate cerebral infarction and cardiac ischemia.

10-15% of patients with acute retinal ischemia (even if transient) had an acute brain infarction on brain DWI-MRI. What's the hurry?
What needs to be done?

- DWI-MRI within 24-48 hours of vision loss
- Imaging (CTA) of cervical and intracranial vessels.
- EKG and echocardiogram
- Laboratory testing
  - CBC with platelets
  - Coagulation studies
  - Fasting lipid profile

How does this get done?

Do NOT send these patients to their PCP, cardiologist, neurologist, neuro-ophthalmologist, or retinal specialist.

Do NOT try to obtain the work-up yourself.

Send to an ED with an Acute Stroke Care Center!

Back to the Case

- IMMEDIATE HOSPITALIZATION
- echocardiogram (TTE, TEE)
- carotid Doppler/CTA/MRA
- MRI of brain (rule out acute stroke with DWI)
- Lab testing

Results

- High total cholesterol and high LDL
- Anemia
- mild stenosis of the proximal right internal carotid artery (no stroke on MRI DWI)
- TREATMENT: Crestor and aspirin 325 mg, as well as a multivitamin and iron.
• In terms of management:
  • Cerebral stroke = cerebral TIA = retinal stroke = retinal TIA

• So, even if this was a transient process, and vision had returned. We would still need to follow the same guidelines/protocol.
65 year old woman

- C/o red, painful eye x 3 days
- Pain is a “10”
- 3 days ago, episode of bilateral vision loss
  - Complete blackness
  - Lasted a few seconds
  - Associated dizziness

- Shortly after, OS became red
- Headache and left eye pain
- VA OS decreasing since then

- VA OD 20/40 and OS 20/60
- + red desat OS
- + decreased brightness sense OS (pupils dilated)
- CF: inferior temporal constriction OS
- TA: OD 12 OS 10
- BP: 129/72

Systemic Hx:
- HTN x 15 yrs – on Accupril
- Hypercholesterolemia – no tx / allergies
- + SOB / fatigue, had carotid US
  - Stenosis of left carotid artery
  - Saw vascular surgeon
  - Ordered CTA of neck and cardiology consult
  - Will F/U in one month
CT-angio neck

Report showed

- Severe stenosis (90%) of left subclavian artery
- Severe stenosis of mid-portion of left common carotid
  • Extends for 2.5 cm

Assessment / Plan

- Acute Ocular Ischemic Syndrome
- STAT hospital admission
- Carotid endarterectomy vs stent

DIAGNOSIS: Acute Ocular Ischemic Syndrome

- STAT hospital admission
- High-risk for stroke – pt not allowed to move
- Carotid endarterectomy was determined to be too risky
- Patient underwent endovascular stenting
- Patient did suffer a stroke during surgery
- Resulting in R hemiparesis and memory loss

FOLLOW-UP:

- Underwent motor and speech therapy
- Recovered well

Statistics Regarding Ocular Ischemic Syndrome:

- A 40% mortality rate has been reported in patients with OIS
- The most common symptom is slowly progressive vision loss, but 10% report sudden vision loss
- 40% present with pain
- 67-87% present with iris neovascularization
- 10-20% are asymptomatic at time of diagnosis
- 86% of patients are smokers
- Risk factors include diabetes, ischemic heart disease, cerebro-vascular disease, trauma, and vasculitis (need to R/O GCA)

NASCET

• NORTH AMERICAN SYMPTOMATIC CAROTID ENDARTERECTOMY TRIAL
SURGICAL TREATMENT (NASCET)

☐ SYMPTOMATIC with STENOSIS > 70%
CAROTID ENDARTERECTOMY IS RECOMMENDED

☐ SYMPTOMATIC with STENOSIS 50-70%
CAROTID ENDARTERECTOMY IS INDICATED

2.1% PERIOPERATIVE RISK OF STROKE AND DEATH

☐ SURGICAL TX of ASYMPTOMATIC CAROTID STENOSIS IS CONTROVERSIAL

SURGICAL TREATMENT

• PERICUTANEOUS TRANSLUMINAL ANGIOPLASTY
• INTERNAL CAROTID ARTERY STENT
• FOR PATIENTS UNABLE TO UNDERGO ENDARTERECTOMY

CASE 3

49 year-old woman

• At a party 2 days prior
• Laughing hard
• Felt something “pop” in her head
• Developed headache / pain behind left eye
• Noticing a difference in appearance between eyes
Diagnostic Test For Horner Syndrome

- 0.5% or 1.0% Apraclonidine (Iopidine)
- Alpha agonist
- Weak alpha 1 agonist
- No effect on normal pupil
- Dilates Horner pupil (supersensitivity)
- Look for reversal of anisocoria
- May not be positive in acute Horner Syndrome (up-regulation did not occur)

Carotid Artery Dissection

- Need to consider this diagnosis in EVERY PAINFUL HORNER’s
- Even if Apraclonidine test appears negative
- Can occur with or without trauma
- Medical Emergency
- Horner’s with eye, head, neck pain
  – Pt to hospital (MRI, MRA (head & neck), CTA, angiogram)

CAROTID ARTERY DISSECTION

- EPIDEMIOLOGY
  - ANNUAL INCIDENCE 2.5 – 3/100,000
  - 2% OF ALL ISCHEMIC STROKES
  - AFFECTS ALL AGE GROUPS INCLUDING KIDS
  - 10-25% OF ALL ISCHEMIC STROKES IN YOUNG AND MIDDLE AGED PATIENTS!
CAROTID ARTERY DISSECTION

- PREVENT THROMBOEMBOLIC COMPLICATIONS WITH ANTICOAGULATION
- IV HEPARIN; ORAL WARFARIN
- @3 MOS: MRA TO EVALUATE INTRA LUMINAL IRREGULARITIES
- @3 MOS: HIGH RATE OF RECANALIZATION
- HEAL SPONTANEOUSLY

CASE 4

53 year-old woman

- Left sided occipital headache x 5 days
  - Daily, every am, lasts about 1 hour
- Yesterday, upon waking, she noticed colored lights/shapes on the right side of her vision
  - Come and goes

- 2 years ago, she lost control of her left hand and had difficulty using 2 fingers
  - Saw a neurologist, found no problems, no additional testing
- Last year, she had an episode of walking crooked
- Previous episode of vertigo

- Diabetes x 8 months
- Hypertension x 30 years
- Past dx of hypercholesterolemia
- MEDS:
  - Atenolol
  - Lisinopril
  - HCTZ
  - Metformin
  - Not compliant with meds! Uses about 3 times per week

- VA 20/20 OD and 20/20 OS
- Color: 14/14 OD, 14/14 OS
- PERRLA (-) APD
- No ptosis or proptosis
- Normal ocular motility
- Normal slit lamp, and IOP OU
- Normal optic discs and maculae OU
- BP: 210/120
- Pulse: normal/regular
• Neurologic Exam:
  – Mild residual left upper extremity weakness (especially left hand)
  – Difficulty with tandem gait
  – Felt as if falling to the right on Rhomberg testing

• Admit to Hospital
  – Suspect a new stroke of left occipital lobe above calcarine fissure
    • Need to r/o hemorrhagic stroke
    • Need to control BP
    • Need to control other stroke risk factors

• Follow-up
  – Dx with old infarct in right basal ganglia
  – Lacunar right frontal subcortical infarcts
  – No acute infarct noted
  – BS was 336mg/dL in hospital
  – Her homonymous hemianopia was a TIA
  – She is no longer noticing the colors and shapes in her vision
  – She is back to driving and all other activities

Follow-up VFs (1 month later)

CASE 5
68 year-old woman

- Shadow on left side of vision x 2 days
- Headache and flashes of light for same amount of time
- 3 weeks ago, noted stiffness in left hand and foot

- Diabetes x 20 years
- Hypertension x 20 years
- Hypercholesterolemia
- Kidney cancer 5 years ago
  - Left kidney removed
- Breast cancer 5 years ago
  - Right breast removed
  - Chemotherapy
- Defibrillator

- Glaucoma
- Travatan (does not use due to cost)
- The patient does drive (drove to office today)

- VA: OD 20/25, OS 20/30
- Color: 14/14 OD 14/14 OS
- PERRLA (-) APD
- No ptosis or proptosis
- Normal ocular motility
- Slit lamp: cataracts OU
- IOP: OD 24 mm Hg, OS 21 mm Hg
- BP: 136/86
- Pulse: normal/regular
- DFE: asymmetric cupping OD > OS, otherwise unremarkable OU

- Neurologic Exam:
  - ? Left-sided weakness
  - Difficulty with tandem gait
  - Difficulty with rapid alternating movements of the left hand
• Admit to Hospital
• Rule out acute stroke vs. metastatic lesion
• Will need to have CT
• MRI contraindicated

• Pt told of confirmed stroke
• Underwent much testing
  — Carotid ultrasound
  — TEE
  — Cardiology consult
  — Nephrology consult

• Visual Field defect persists
• Pt will no longer be able to drive

• What is the optometrist’s role in assessing ability to drive?
  • Visual requirements vary by state
  • Some states mandate reporting if pt does not meet requirements

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• What is the optometrist’s role in assessing ability to drive?
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HOMONYMOUS HEMIANOPIA

• Can improve as edema resolves
  — 84% have some amount of recovery at 6 months
  — Allow 1 year to get to maximum recovery

» Usually not enough recovery to regain driving ability in those not meeting visual driving requirements right after stroke

Visual Driving Requirements by State

• Differ according to state
• VA and VF requirements in most states:
• 12 states require a minimum of 90-110 degrees
• 20 states require from 120 to as much as 140 degrees (ex: FL - 130, NY - 140)
• 16 states have NO VF requirements (ex: CA, NJ)
  — AZ: 70 degrees, plus 35 degrees on the opposite side of the nose, in at least one eye.
  — PA: The horizontal visual field requirement is at least 120 degrees (combined) in the horizontal meridian, excepting the normal blind spots.
  — KY: 35 degree horizontal, 25 degree vertical VF
• A few states, including NC will not issue a license to a pt with a homonymous hemianopia

HOMONYMOUS HEMIANOPIA

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CASE 6

HOMONYMOUS HEMIANOPIA

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CASE 6
52 year-old woman

• referred for VF defects
• noted change in VA on 2 yrs prior to presentation
• Went to ER 9/11 told of BP 200/140
• Still notes problems with superior VF OU
• Hx of HTN (23 yrs), heart murmur, hypercholesterolemia
• Procardia, multivitamins
• Denies eye or head pain, diplopia
• Notes 5 episodes of dizziness, transient blur OU
• 1 episode of L upper & lower extremity numbness x 15 min

VA: OD 20/20, OS 20/20
Pupils isocoric, (- ) RAPD
Color: OD 14/14 OS 14/14
Normal ductions, versions, OKN
SLE: xanthelasmas OU
Neurologic Exam: normal

• When you see BILATERAL altitudinal defects, in the setting of NORMAL optic nerves and retina
  – Right HH combined with Left HH
  – Localizes to OCCIPITAL LOBE
  – Shared posterior blood supply
Why Was This an Emergency?

Even though the VFs depict an old infarct, the patient was sent to the hospital because:
- increasing frequency of TIAs
- suggests increased risk of additional stroke
- need to control all stroke risk factors
- stroke in young requires work-up to rule out other causes
  - hypercoaguable states
  - patent foramen ovale
  - etc

67 year-old man

- History of blindness OD x 7 yrs (RD), HTN, high chol, gout
- Meds: Diovan, indomethacin (ASA d/c c indo)
- 1-2 wks ago noted sudden decreased vision OS
- Seemed to be temporally OS
- Some shortness of breath
- No associated pain or other neuro symptoms
- Vision improved somewhat in first day, but stable since
- Saw PCP – did EKG – normal
- Pt refused stress test

Work-up

- MRI / MRA brain
- Lab testing

CASE 7

67 year-old man

- History of blindness OD x 7 yrs (RD), HTN, high chol, gout
- Meds: Diovan, indomethacin (ASA d/c c indo)
- 1-2 wks ago noted sudden decreased vision OS
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Work-up

- MRI / MRA brain
- Lab testing

CASE 7

EXAM RESULTS

- VA OD NLP OS 20/25
- (+) RAPD OD
- BP 160/100
- Neuro-exam:
  - Finger-to-nose ataxia
  - Difficulty with tandem gait
Old lacunar infarcts in pons and thalamus

Hypertensive white matter changes

New right occipital lobe infarct

Lack of flow in right PCA

Lab Tests

- R/O GCA
- CBC
- Hemoglobin
- Platelets
- ESR
- C-reactive protein
- Homocysteine levels
- Methylmalonic acid levels
- PT/PTT
- Others

Results

- ESR mildly elevated at 39 (age 67)
- Homocysteine level elevated at 27
  - Normal level (4.0 – 15.4)
Homocysteine

Amino acid in the blood
Increased with vitamin B12 or folate deficiency, genetic causes or renal disease
Related to greater risk of cardiovascular disease, ischemic stroke, peripheral vascular disease
Elevated levels = atherosclerosis
Folic acid and vitamins B6 and B12 break down homocysteine in the blood
A 3 umol/L drop in homocysteine = 16% less chance of heart attack, 24% less chance of stroke, and 25% less chance of DVT
Levels above 14 umol increase risk of Alzheimer’s by 150%

CASE 8

76 year-old woman
• 4 day history of left-sided vision loss preceded by pain in temple
• Has felt “odd” x 10 days
• Tired, fatigued, memory problems
• Feels like heart is pounding at times
• Saw PCP 1-2 weeks ago

Treatment
• Folic Acid
• Re-initiate ASA 81 mg
• Stroke neurology consult

• HTN x 10 years
• Hypothyroidism x 10 years
• Glaucoma (s/p trab)
• Meds: synthroid, ? HTN med, Cosopt, Travatan

• VA OD 20/40 OS 20/25
• Color OD 7/7 OS 7/7
• PERRL (-) RAPD
• CF: dense R homonymous hemianopia
• Last HVF (6 mos ago) showed arcuate defects OU
• Normal EOMs
• TA: 12 mmHg OU
• BP: 150/84 RAS
• Pulse 92 bpm & irregular (periods of quick beat, then would slow down or stop and then get fast again)
• ON assessment: large optic disc cupping OU

• Due to new onset HH – suspect recent stroke
• Because of irregular pulse, need to consider A-fib
• Pt sent directly to ER
  □ Diagnosed with left occipital lobe stroke & atrial fibrillation
• Was put on Coumadin (anticoagulant - inhibits vit K dependent coagulation factors)
• Because of frequent falls and bleeding risk, was switched to Aggrenox (platelet inhibitor)

Atrial Fibrillation (AF)

• Associated with increased rate of ischemic stroke.
• 2 to 7 times the rate of stroke in patients without AF.
• With AF, the stroke risk averages 5% a year
• The risk increases with:
  — co-existent cardiovascular diseases
  — Increasing age
  • Risk is 1.5% for those aged 50-59 years
  • Risk approaches 30% for those aged 80-89 years

ALWAYS CHECK PULSE IN PT SUSPECTED OF HAVING STROKE
81 year old man with diplopia

- When he awoke this morning everything was double
- Feels “drunk”
- Balance difficulties
- Feels as if BP may be high
- Denies GCA symptoms

Initial Presentation

Follow-Up

Acute Skew Deviation
Sent Immediately to ED (stroke center)

- Transient cerebral ischemia
- Followed by neurology
- Meds: HCTZ, Lipitor, 325 mg ASA, niacin, metoprolol, Diovan

- Diplopia significantly improved, no longer has any dizziness or balance problems
- No new symptoms

INTERNUCLEAR OPHTHALMOPLEGIA

- **PONS**: long circumferential branches of basilar and anterior inferior cerebellar (AICA) arteries
  - Supplies MLF
  - Convergence not affected

- **MIDBRAIN**: paramedian branches from PCA and basilar bifurcation
  - Supplies both MLF & and CNIII
  - Center for convergence
  - INO with inability to converge

NEURO-OPHTHALMOLOGY OF STROKE

- POSTERIOR CIRCULATION (Vertebral-basilar)

- **BRAINSTEM MOTILITY DISORDERS**
  - **INTERNUCLEAR OPHTHALMOPLEGIA**
  - **SKEW DEVIATION**
  - **TOP OF THE BASILAR SYNDROME** (Dorsal Midbrain Syndrome)
  - **HOMONYMOUS HEMIANOPIA**
  - **DISORDERS OF VISUAL ASSOCIATION CORTEX**

CASE 10
Pt also has paralysis of left upper and lower extremity.

Frontal eye fields: control contralateral saccades

Parieto-occipito-temporal area: control ipsilateral pursuit (and contralateral eye movements)

Supranuclear control of eye movements

- Frontal Eye Fields – responsible for contralateral saccades
- Parieto-occipito-temporal area – responsible for ipsilateral pursuits and contralateral eye movements

Gaze palsy / gaze preference

- Cerebral infarct – supranuclear (anterior circulation)
  - Contralateral eye movements affected – eyes point toward side of lesion
  - Often associated with hemiparesis
  - Eyes pointing away from paralyzed side
NEURO-OPHTHALMOLOGY OF STROKE (Anterior Circulation)

• AMAUROSIS FUGAX & RETINAL STROKE (CRAO, BRAO)
• OCULAR ISCHEMIC SYNDROME
• CAROTID ARTERY DISSECTION
• VISUAL FIELD NEGLECT
• Supranuclear gaze palsy/preference

Differs from brainstem lesion

• CN VI nucleus or PPRF affected
  – Eyes do not look to the ipsilateral side
  – Eyes may point away from the lesion
  – Contralateral hemiparesis – corticospinals
  – Eyes point to the paralyzed side

  – BE SURE CLINICAL PRESENTATION MATCHES ANATOMIC LOCALIZATION OF STROKE (get past records if necessary)

CASE 11

75 year-old man

• History of stroke 3 years ago
  – Vision affected – difficult to see to one side
• No other known visual problems
• No new complaints, feels vision is stable
  – Systemic history:
    • + DM, HTN, hypercholesterolemia
    • s/p prostate cancer
  – Ocular history:
    • s/p bilateral cataract surgery
    • Denies any trauma

• VA OD 20/60  OS 20/30
• Color Vision: OD 6/14  OS 13/14
• PERRL (+) 0.6 log RAPD OD
• Normal Motilities
• No ptosis or proptosis OU
• Normal SLE, IOP OU
• Report of neuro-imaging
  – Right occipital lobe infarct (from 3 years ago)

- Are all findings from the same known process (stroke)?
  - Yes
    - Left Homonymous Hemianopia (occipital lobe – after LGN)
  - No
    - RAPD (before LGN)
    - Optic disc pallor (before LGN)
    - Dyschromatopsia (before LGN)
    - Overlying arcuate defects

- Additional work-up necessary for causes of optic neuropathy
- Labs
- Additional imaging

• It is just as important to known what features are secondary to a stroke as it is to known what features are not secondary to stroke
Optic Neuropathy Work-Up

- MRI – brain and orbits
- Lab testing

Anatomic Localization is essential
- Lesions anterior to the LGN
  - Can have RAPD, dyschromatopsia, optic disc pallor
- Lesions posterior to the LGN
  - Will NOT have RAPD, dyschromatopsia, optic disc pallor
  - ONE EXCEPTION: Central Achromatopsia

CASE 12

60 Year old man

- Difficulty reading
- Trouble with bifocals
- Holds bifocals up when reading

- HTN, hypercholestrolenia
- Hx of cocaine use

Exam Findings

BCVA: OD 20/20, OS 20/20
Pupils: PERRL (-)APD
Color: OD 12/14
      OS 11/14
PERRLA (-) RAPD
Normal motilities, SLE, IOP
Street Drug Use and Stroke

- Cocaine, methamphetamines, and other stimulants can cause stroke in two ways:
  - Increase blood pressure
    - Direct effect on the vessel walls, and the extra pressure can cause them to rupture (hemorrhagic)
  - Narrow the blood vessels
    - Cut off blood flow to parts of the brain and kill brain tissue (ischemic)

Risk of stroke from street drugs increases with other stroke risk factors.

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- Brainstem Motility Disorders
- Internuclear Ophthalmoplegia
- Skew Deviation
- Top of the Basilar Syndrome
  - (Dorsal Midbrain Syndrome)
- Homonymous Hemianopia
- Disorders of Visual Association Cortex

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**Disorders of the Visual Association Cortex**

- **Ventromesial Pathway** “What it is”
- **Association Cortices Beneath Calcarine Fissure & Adjacent Temporal Regions**
- **Visual Object Recognition, Reading, Color Vision**
- **Dorsolateral Pathway** “Where it is”
- **Association Cortices Above Calcarine Fissures and in Adjacent Parieto-Parieto-Occipital Regions**
- **Balint’s Syndrome, Akinetopsia**
CENTRAL ACHROMATOPSIA

- Acquired disorder of color perception involving all or part of the visual field
- Visual association cortices in the middle third of the fusiform and lingual gyrus
- Area V1 with underlying radiations are spared

ACHROMATOPSIA

- Can involve a quadrant, hemifield or entire visual field
- Describe affected vision as shades of gray
- Lesions of inferior occipito-temporal cortex
- Can occur with:
  - Superior VF defects
  - Alexia

VISUAL AGNOSIA

- Unable to recognize objects that are seen
  - **APERCEPTIVE AGNOSIA** – Image of the object is in some way distorted and cannot be recognized
  - **ASSOCIATIVE AGNOSIA** – Image is clear, but connections to association visual cortex are impaired not allowing recognition

PROSOPAGNOSIA

- Cannot recognize familiar & own face
- Bilateral occipital temporal lesions
- Damage to inferior longitudinal fasciculus near its occipital temporal origin
- Disconnection of occipitotemporal cortex specialized for facial recognition
ALEXIA WITHOUT AGRAPHIA

- CANNOT READ BUT CAN WRITE; "WORD BLIND"
- CAN READ NUMBERS
- CAN SPELL WORDS UPON REQUEST
- DIFFICULTY NAMING COLORS MAY HAVE RIGHT HOMONYMOUS, PROSOPAGNOSIA, OR VISUAL OBJECT AGNOSIA
- INFARCT OF LEFT (usually the dominant side) VISUAL ASSOCIATION CORTEX AND SPLENIUM OF CORPUS CALLOSUM; OCCLUSION OF PCA

ALEXIA WITH AGRAPHIA

- CANNOT READ, WRITE OR SPELL
- CANNOT RECOGNIZE A WORD THAT IS SPELLED
- CANNOT INTERPRET WORD PICTURES
- OK WITH SPOKEN LANGUAGE
- INFARCT IN THE ANGULAR GYRUS OF DOMINANT HEMISPHERE

DISORDERS OF THE VISUAL ASSOCIATION CORTEX

- VENTROMESIAL PATHWAY “What it is”
  - ASSOCIATION CORTEXES BENEATH CALCARINE FISSURE & ADJACENT TEMPORAL REGIONS
  - VISUAL OBJECT RECOGNITION, READING, COLOR VISION
- DORSOLATERAL PATHWAY “Where it is”
  - ASSOCIATION CORTEXES ABOVE CALCARINE FISSURES AND IN ADJACENT PARIETAL, TEMPORO-PARIETO-OCCIPITAL REGIONS
  - BALINT'S SYNDROME; AKINETOPSIA

AKINETOPSIA

- MOTION BLINDNESS
- AREA V5 OF ASSOCIATION VISUAL CORTEX
- POSTERIOR BANK OF SUPERIOR TEMPORAL SULCUS
- A "MOTION FUNNEL" – ALL INFORMATION RELATED TO MOTION IS RELAYED THROUGH V5
- RETINA MUST BE NORMAL
BALINT’S SYNDROME

- OPTIC ATAXIA: DEFECTIVE HAND CONTROL
- OCULAR APRAXIA: DEFECTIVE GAZE CONTROL
- SIMULTANAGNOSIA: UNABLE TO SYNTHESIZE ALL THE FEATURES IN AN ARRAY
- BILATERAL POSTERIOR PARIETAL OCCIPITAL INFARCTIONS

Simultanagnosia

- Core abnormality of Balint’s syndrome
- Impaired ability to perceive parts of a visual scene as a whole
- Focus on small isolated parts that shift unpredictably

Optic ataxia

- Inability to reach for or point to objects by use of visual guidance
- Can point or reach using proprioceptive or auditory cues
  - Cannot touch Dr's finger, but can touch own finger
- Once object is touched, can continue to reach for it even with eyes closed
- Differs from cerebellar ataxia because of smooth movements rather than shaking movements

Ocular apraxia

- Difficulty directing gaze toward objects in peripheral vision through saccades
- Move head to try to change gaze

Balint’s syndrome

- Often bilateral lesions of dorsolateral parieto-occipital cortex
- MCA-PCA watershed infarcts
- Often associated with
  - Inferior quadrant VF defects
  - Aphasia
  - Hemi-neglect
CASE 13

67 year old man

- History of congenital nystagmus
- Functioned fine in past – reading/driving
- Having difficulty within last year
- s/p cataract surgery OS – no improvement
- New glasses – no improvement

• VA OD 20/50   OS 20/50
• Color  OD 2/14  OS 1/14
• PERRLA (-) RAPD
• Nystagmus
• Visual fields:
  – CF: difficulty with all quadrants with finger counting
  – Better when using color caps
  – HVF: apparent left homonymous hemianopia

- Pt can read individual letters
  • Cannot make the letters into words
- Can write sentences
  • Unable to read them back
- Unable to process what is happening in a detailed photo

- Alexia without agraphia
- Simultanagnosia
- Left homonymous hemianopia
- Pt currently undergoing work-up

- Localization
  – Occipital lobe
  • Right side (maybe the dominant side)
VISUAL FIELD NEGLECT

An 80 year old woman eats only from the right half of the dinner plate. In order to find the food on the left side of her plate she rotates 360 degrees to her right until the food appears on her tray. She applies lipstick only to the right side of her mouth.

VISUAL FIELD NEGLECT

- NEGLECT OF THE LEFT VISUAL FIELD
- MAY HAVE A SUPERIOR LEFT HOMONYMOUS
- OFTEN ACCOMPANIED BY LEFT HEMISPARESIS & HEMISENSORY LOSS
- LARGE INFERIOR DIVISION MCA TERRITORY INFARCT OF RIGHT TEMPORAL LOBE

VISUAL FIELD NEGLECT

- ANOSAGNOSIA – no knowledge of the loss
- PATIENT IGNORES & NEVER LOOKS TO LEFT
- EATS ONLY FROM RIGHT SIDE OF PLATE
- USES MAKE-UP OR SHAVES ONLY THE RIGHT SIDE OF THE FACE
- WILL TURN IN A CIRCLE TO THE RIGHT TO FIND OBJECTS TO HER LEFT
- ANTON’S SYNDROME (denial of blindness)

VISUAL FIELD NEGLECT

- SUSPECT WITH A LEFT HOMONYMOUS VISUAL FIELD CUT
- DETECTED WITH:
  - SIMULTANEOUS FINGER COUNTING
  - CROSSING LINES SCATTERED ON A SHEET
  - BISECTING LINES
Almost all areas of the brain, when damaged, result in visual manifestations:
- Retina – CRAO, BRAO
- Occipital lobe – homonymous hemianopia
- Cerebellum – ataxia, nystagmus
- Brainstem – diplopia, skew deviation, INO, nystagmus
- Cerebrum – supranuclear gaze palsy

Role of Optometrists

- Identify Acute Stroke / TIA
  - Get patient immediate (emergent) work-up/treatment as necessary
- Determine if Deficits are from old strokes or if they indicate a new stroke/other process
- Preventative Measures
  - Identify stroke risk factors
  - Educate patients
  - Refer as necessary
- Assess visual function after stroke
  - Improve visual function where possible
  - Comment on driving ability

Thank you.