Everything Therapeutic: San Antonio

Course Notes

To view notes for a specific lecture, click the lecture title on the agenda page to instantly progress to the appropriate section.
# Everything Therapeutic
## San Antonio

**Program Location**
The Westin Riverwalk Hotel  
420 West Market Street, San Antonio, Texas 78205

### Saturday, November 23rd

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>lecturers</th>
<th>COPE ID#</th>
<th>CEE Available</th>
<th>D/T Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:00 am - 8:00 am</td>
<td>Registration, Continental Breakfast, &amp; Visit Exhibits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8:00 am - 9:45 am</td>
<td>Clinical Challenges in Uveitis: Tales from the AC</td>
<td>David Sendrowski, OD, MS, FAAO</td>
<td>65597-AS</td>
<td>CEE Available</td>
<td>2 D/T Hours</td>
</tr>
<tr>
<td>9:45 am - 10:15 am</td>
<td>Break &amp; Visit Exhibits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10:15 am - 11:05 am</td>
<td>Ocular Dermatology: Lids and Lesions</td>
<td></td>
<td>59971-AS</td>
<td>1 D/T Hour</td>
<td></td>
</tr>
<tr>
<td>11:05 am - 12:00 pm</td>
<td>Anterior Segment: Herpes Simplex Infection</td>
<td></td>
<td>61963-AS</td>
<td>1 D/T Hour</td>
<td></td>
</tr>
<tr>
<td>12:00 pm - 1:00 pm</td>
<td>Lunch &amp; Visit Exhibits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1:00 pm - 2:45 pm</td>
<td>Controversies in the Basic Glaucoma Evaluation</td>
<td></td>
<td>65610-GL</td>
<td>CEE Available</td>
<td>2 D/T Hours</td>
</tr>
<tr>
<td>2:45 pm - 3:15 pm</td>
<td>Break &amp; Visit Exhibits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3:15 pm - 4:05 pm</td>
<td>Pot Glaucoma Updates: What We All Should Know</td>
<td></td>
<td>52520-GL</td>
<td>1 D/T Hour</td>
<td></td>
</tr>
<tr>
<td>4:05 pm - 5:00 pm</td>
<td>2019 Professional Responsibility Course for Texas Optometrists</td>
<td></td>
<td>60627-EJ</td>
<td>1 GEN/PR Hour</td>
<td></td>
</tr>
</tbody>
</table>

### Sunday, November 24th

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>lecturers</th>
<th>COPE ID#</th>
<th>CEE Available</th>
<th>D/T Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:00 am - 8:00 am</td>
<td>Registration, Continental Breakfast, &amp; Visit Exhibits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8:00 am - 9:45 am</td>
<td>Rules and Exceptions in Neuro-Ophthalmic Disease</td>
<td></td>
<td>62267-NO</td>
<td>CEE Available</td>
<td>2 D/T Hours</td>
</tr>
<tr>
<td>9:45 am - 10:15 am</td>
<td>Break &amp; Visit Exhibits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10:15 am - 12:00 pm</td>
<td>Conversations in Optic Nerve and Retinal Vascular Disease</td>
<td></td>
<td>65612-PS</td>
<td>CEE Available</td>
<td>2 D/T Hours</td>
</tr>
<tr>
<td>12:00 pm - 1:00 pm</td>
<td>Lunch &amp; Visit Exhibits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1:00 pm - 2:45 pm</td>
<td>Depress Your Patient: Diagnosis and Management of Vitreoretinal Anomalies</td>
<td></td>
<td>65622-PS</td>
<td>2 D/T Hours</td>
<td></td>
</tr>
<tr>
<td>2:45 pm - 3:15 pm</td>
<td>Break &amp; Visit Exhibits</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3:15 pm - 4:05 pm</td>
<td>Preparing the Dry Eye for Contact Lens Wear</td>
<td></td>
<td>52613-AS</td>
<td>1 D/T Hour</td>
<td></td>
</tr>
<tr>
<td>4:05 pm - 5:00 pm</td>
<td>Managing Ocular Pain and Inflammation</td>
<td></td>
<td>65611-PH</td>
<td>1 D/T Hour</td>
<td></td>
</tr>
</tbody>
</table>
DISCUSSIONS IN NEURO-OPHTHALMIC DISEASE:
RULES, EXCEPTIONS TO THE RULES, AND EXCEPTIONS TO THE EXCEPTIONS TO THE RULES

Joseph Sowka, OD, Diplomate
Nova Southeastern University College of Optometry

THURSTON HOWELL III DOESN’T LIKE NEURO

"Neuro equals referral"

"Diagnose and adios!"

A personal case to prove my point

MANAGING PATIENTS WITH NEURO-OPHTHALMIC DISEASE

- Understanding of anatomy
- Following several fundamental principles
- Following several simple rules
- Developing a network of referral physicians
  - Neuroradiologist
  - Neurologist
  - Internist
  - Neurosurgeon
  - Rheumatologist

DISCLOSURE:
Joseph Sowka, OD is/has been a Consultant/ Speaker Bureau/ Advisory Board member for Novartis, Allergan, Glaukos, and B&L. Dr. Sowka has no direct financial interest in any of the diseases, products or instrumentation mentioned in this presentation. He is a co-owner of Optometric Education Consultants (www.optometricedu.com)

The ideas, concepts, conclusions and perspectives presented herein reflect the opinions of the speaker; he has not been paid, coerced, extorted or otherwise influenced by any third party individual or entity to present information that conflicts with his professional viewpoints.
A PERSONAL CASE TO PROVE MY POINT

You’re wife is going to kill you if she finds out!

RULE

Congenital optic nerve anomalies can have (sometimes dramatic) visual field loss

RULE

Don’t make diagnosis of immune disease in immunosuppressed patients
Now we are up to the audience participation part of this program.

**Rule**

Urgency of evaluation is dictated by duration of condition.

**46 YOM**

- Reports waking up 3 months ago not being able to see OD
- LP OD, 20/20 OS
- Disc pallor OD - no other concurrent findings
- Last medical exam unknown - no medical hx
- Resident gets nervous - sends to ER immediately
- How long do we have to get this worked up?

**Rules must be obeyed**

- 57 YOF
- Low risk OHTN OU
- GDx, OCT, ONH – perfectly normal OU

**Rule**

Chiasmal and retrochiasmal lesions have bilateral involvement.

Unilateral visual field loss reflects anterior visual pathway disease which will show something identifiable in the form of damage to the vision, disc, RNFL, dyschromatopsia or afferent pupil defect.
RULE

A patient can fake a field, but can't fake a retinal nerve fiber layer or pupil defect.

What is wrong with this picture?

59 YOM

- Routine exam- c/d 0.5/0.5 OU
  - IOP 20 mm Hg OU
- Returns 2 years later- slowly progressive loss of vision OD
- RAPD OD; 20/80 OD; 20/20 OS
- Superior altitudinal defect splitting fixation OD; mild inferior defect OS
- Disc pallor OD
- Dx: NAAION

What is wrong with this picture?

59 YOM

- Routine exam- c/d 0.5/0.5 OU
  - IOP 20 mm Hg OU
- Returns 2 years later- slowly progressive loss of vision OD
- RAPD OD; 20/80 OD; 20/20 OS
- Superior altitudinal defect splitting fixation OD; mild inferior defect OS
- Disc pallor OD
- Dx: NAAION

What is wrong with this picture?

RULE

Don’t make the diagnosis of NAAION in glaucoma patients

RULE

A diagnosis of exclusion (Adies tonic pupil, PTC, Bell’s palsy, NAAION, Tolosa Hunt syndrome) should your last diagnosis, not your first.
48 YOWM

Painless loss of visual field OS
- 20/20 OD, OS
- Noticed upon waking

Med Hx: Unremarkable, except for viral illness 3 weeks before

NAAION OS
Disc at risk OD

RULE

Pallor in excess of cupping indicates something other than, or in addition to, glaucoma

RULE

Nothing notches a nerve like glaucoma

IN THE AGE OF IMAGING, DO WE REALLY NEED FIELDS?
- 54 YO Nigerian man
- Referred for glaucoma management
- Told he had glaucoma 6 years earlier- no Tx
- 20/30 OD; HM OS
  - Vision loss from glaucoma- not coming back
- 30 mm Hg OD; 23 mm Hg OS
  - Lumigan- 17 mm Hg OD, 15 mm Hg OS
Diagnosis?

Plan?

Do we really need fields in this case?

Yes, we still need to do fields in the age of imaging.
Sometimes its not glaucoma

POAG GETS COMPLICATED?
- 70 YOWM
- POAG OU
- Auto accident with concussion
- Develops gaze induced amaurosis fugax
- Referred by PCP to neuro-ophthalmologist
- Complete evaluation with MRI- negative
- Psychological?
Sometimes it is glaucoma

ODE TO A CUPPED DISC

Oh, to have a cupped disc pink.
That my friend hath a glaucomatous stink.
But to have a cupped disc pale,
Call this glaucoma and you shall fail.
Disc and field damage that is one-sided
Simply cannot be abided.
It might be trauma, infarct or meningioma.
But if the rim is cut always remember,
Nothing notches a nerve like glaucoma.

Joseph Sowka, OD

CASE HISTORY 46 WM

- CC: Patient reports a "droopy left eye" which began about 6 weeks ago. Headache and numbness ipsilateral; hives
- ER diagnosed with "stye". Patient was referred in by a local optometrist.
- Past Ocular History: unremarkable
- Past Medical History: (+) Mitral Valve Prolapse, (+) GERD and recent weight loss of about 20 lbs. over the past 6 months or so.
- Medications: Prilosec, Metoprolol Succinate, Xanax, Prednisone, Lipitor, Claritin

PERTINENT FINDINGS

- BCVA 20/20 OD and 20/20 OS
- Pupils: unequal, round, reactive to light, No APD

<table>
<thead>
<tr>
<th>Bright Illumination</th>
<th>Dim Illumination</th>
</tr>
</thead>
<tbody>
<tr>
<td>OD: 4 mm</td>
<td>OD: 6 mm</td>
</tr>
<tr>
<td>OS: 3 mm</td>
<td>OS: 4 mm</td>
</tr>
</tbody>
</table>

- Motility and confrontation fields unremarkable
- Observation: LUL ptosis, Left miosis
- Intraocular pressure: 18 mmHg OD and 19 mmHg OS
- Fundoscopy-unremarkable

So, what do you think and what do you want to do now?
HORNER’S SYNDROME

- Etiology unclear based upon exam
- Headache, neuralgia and ‘hives’
  - Not consistent with cluster migraine
    - Dx of exclusion, not convenience
  - Hives- not consistent with HZO
- Unexplained weight loss concerning-relationship unclear
- Recommend medical eval by PCP
  - Additional testing dictated by PCP results

DISCUSSION

What is Horner’s Syndrome?

- a triad of clinical signs arising from disruption of sympathetic innervation to the eye and ipsilateral face that causes miosis, upper lid ptosis, mild elevation of the lower lid, and anhydrosis of the facial skin.

PHARMACOLOGICAL TESTING

- Cocaine
  - Horner’s pupil doesn’t dilate, normal pupil does
- Hydroxyamphetamine (Paredrine)
  - Differentiates post- from pre-ganglionic
  - Not available and doesn’t matter because bad stuff happens everywhere
- Apraclonidine 0.5% (Iopidine)
  - Denervation suprasensitivity
    - 36-72 hours from onset
  - Horner’s pupil dilates, normal doesn’t
    - Reversal more classic and diagnostic that cocaine
HORNER'S SYNDROME: ETIOLOGIES

First-order neuron disorder: Stroke (e.g., vertebrobasilar artery insufficiency or infarct); tumor; multiple sclerosis (MS), and, rarely, severe osteoarthritis of the neck with bony spurs.

Second-order neuron disorder: Tumor (e.g., lung carcinoma, metastasis, thyroid adenoma, neurofibroma). Patients with pain in the arm or scapular region should be suspected of having a Pancoast tumor. In children, consider neuroblastoma, lymphoma, or metastasis.

HORNER'S SYNDROME: ETIOLOGIES

Third-order neuron disorder: Headache syndrome (e.g., cluster, migraine, Raeder paratrigeminal syndrome), internal carotid dissection, herpes zoster virus, otitis media, Tolosa–Hunt syndrome, neck trauma/tumor/inflammation, prolapctinoma.

• Congenital Horner syndrome: Trauma (e.g., during delivery).
  • Facebook tomography
  • Other rare causes: Cervical paraganglioma, ectopic cervical thymus

MANAGEMENT

• Localizable-targeted workup
  • Neck and facial pain—carotid dissection
  • Facial paraesthesia—middle cranial fossa disease

• Necessary Work Up (non-localizable):
  • MRI of brain, orbits and chiasm with and without contrast, attention to middle cranial fossa.
  • MRA of head and neck—rule out carotid dissection
  • MRI of neck and cervical spine, include lung apex and brachial plexus
    • Horner’s syndrome patient needs to be imaged from chest to head—3 scans
    • Horner’s protocol
  • All imaging in patient unremarkable

CAROTID DISSECTION

- A 3rd-order Horner’s and ipsilateral head, eye, or neck pain of acute onset should be considered diagnostic of internal carotid dissection unless proven otherwise.

CAROTID DISSECTION

• Carotid artery dissection presents with the sudden or gradual onset of ipsilateral neck or hemicranial pain, including eye or face pain
• Often associated with other neurologic findings including an ipsilateral Horner's syndrome, TIA, stroke, anterior ischemic optic neuropathy, subarachnoid hemorrhage, or lower cranial nerve palsies
  • 52% with ocular or hemispheric stroke with 6 days
    • 67% within first week; 89% within 2 weeks; none after 31 days
  • Horner’s from suspected carotid dissection should go to ER

HORNER SYNDROME ALGORITHM

1. Confirm it is Horner syndrome
   • Apraclonidine; dilation lag
2. Determine if accidental or surgical trauma as cause
3. Urgent imaging
   • CT/CTA; MRI/MRA head and neck if present< 2 weeks
4. Image lung apex
**RULE**

Diagnosing Horner’s syndrome is insufficient. You must try to ascertain a cause and never assume that it is benign.

**CASE: 59 BF**

- Long time patient presents for her glaucoma f/u. She reports drooping in the right eye and smaller pupil for about 1 month. Symptoms were noticed at/ about time of dx of lung cancer and subsequent surgery.
  - She also reports scapular pain and weakness in the right hand.
- Past Medical History: (+) Lung Cancer, (+) Pancreatitis, (+) HTN and (+) Acid Reflux
- Social History: Smokes 1 pack per day for 45 years, Drinks a 6 pack of beer daily

**CASE: PERTINENT FINDINGS CONTINUED…**

- Pharmacological testing not done
- New onset of ptosis and miosis with dx lung cancer and h/o recent lung surgery
- Dx=Pancoast Syndrome

**PANCOAST TUMOR**

A Pancoast tumor is a lung cancer arising in the apex of the lung that involves structures of the apical chest wall.

**Treatment**
- Chemotherapy
- Radiation Therapy
- Surgery: lobectomy vs. wedge resection

Prognosis: 5 year survival rate is around 30%
- Not an emergency

**ODE TO HORNER’S SYNDROME**

When the lid is low and the pupil small,
Check to see the sweat don’t fall.
Cocaine is no longer universal,
Iopidine will cause reversal.
You have to scan head to chest,
And remember that MRA is best.
Pain in association, will surely cause commotion.
Send to the ER without correction,
Remember, it might be carotid dissection.

Joseph Sowka, OD
**RULE**

Suspect the worst

---

**63 YOIM**

- Long standing glaucoma patient
- Sudden onset of orbital pain x 3 days
- + DM; +HTN
- On coumadin
- Pacemaker
- No vision change
- Presents as walk-in emergency glaucoma eval

---

**63 YOIM**

- Pupil involved CN III palsy
- 3 days duration at least
- Most likely cause: intracranial aneurysm
- Sent to ED with detailed notes and recommendations
- Endovascular therapy with coils
- Hospitalized 23 days
Secondary aberrant regeneration

CN III Palsy Clinical Picture
- An eye that is down and out with a ptosis
- Adduction, elevation, depression deficits
- Isocoric or anisocoric

CN III Anatomy
- Vulnerable to compression from aneurysm in subarachnoid space
  - Posterior communicating artery (PCOM)
  - Junction PCOM and ICA
  - Tip of basilar artery

Still More Clues
- Pupil involved CN III palsy is PCOM aneurysm until proven otherwise
- Incomplete palsy is PCOM aneurysm until proven otherwise
  - Regardless of pupil
- 30% of CN III palsy are caused by aneurysm
- Pain is pain
  - Only helpful when not present
- Vasculopathic CN III will resolve in time
- Life threatening posterior communicating aneurysm will rupture in time

Still More Clues
- CN III palsy caused by aneurysm
  - 20% die within 48 hrs from rupture
  - 50% overall die
  - Average time from onset to rupture – 29 days
    - 80% rupture w/i 29 days
    - Many never make it to hospital
RULE

Never dilate a patient with cranial nerve III palsy

STILL MORE CLUES

- CN III palsy caused by aneurysm
  - 20% die within 48 hrs from rupture
  - 50% overall die
  - Average time from onset to rupture – 29 days
    - 80% rupture w/i 29 days
  - Many never make it to hospital
- Ruptured aneurysms
  - 5% surgical mortality
  - 60% functional impairment post-op
- Unruptured aneurysms
  - No mortality; 75% with normal outcomes; 50% with CN III recovery

RULES FOR CN III PALSY IMAGING

- High suspicion of aneurysm: DSA (gold standard)
- CT/CTA is preferred non-invasive imaging for CN III palsy
  - CT for SAH
- CTA requires contrast- renal impairment prefers MRI/MRA
- CTA superior to MRI when patient can’t have MRI
  - Pacemaker, claustrophobia
- MRI superior for non-aneurysmal causes (tumor)
  - MRA adds very little time to scan

A DIFFERENT PATIENT AND PROGNOSIS

- 63 YOF
- Diabetes and HTN
- Sudden onset retro-orbital pain

Complete CN III palsy with pupil sparing and vasculogenic risk factors
WHICH IS BETTER? ONE OR TWO?

Resolves over several weeks

Hospitalized 23 days with 2 neurosurgical procedures

SUSPECT THE WORST

- Optometrist sees patient with CN III palsy
- Referred to ophthalmologist next day
- Pt dies from SAH before consult

DOES PRESENCE OF VASCULOPATHIC RISK FACTORS HELP?

- Arteriosclerotic risk factors in elderly favors microvascular etiology but does not rule out aneurysm
- HTN, DM, atherosclerosis, hypercholesterol all common and don’t protect against aneurysm
- Answer: no, but makes me very nervous when NOT present

DOES ACUTENESS OF PRESENTATION HELP?

- Ans: Yes and No
- Aneurysm expansion usually produces acute manifestations, but chronic and evolving cases well known
- Acute is more worrisome
- Chronic and improving less worrisome but does not rule out aneurysm
- Resolved without recurrence reassuring

ANEURYSM RISK ASSESSMENT: ISOLATED CN 3 PALSY

- Isolated dilated pupil none
- Complete CN3-normal pupil low
- Partial CN3-normal pupil high
- Pupil involved CN3 emergency
NEVER OUT OF THE WOODS

- Pt develops CN III palsy from aneurysm
- Successfully treated with aneurysm clip
  - All coils are inert and MRI safe; not all clips are MRI safe
- Radiologic tech doesn’t verify type of clip
- Pt undergoes F/U MRI with non-MRI safe clip in major medical center
- Clip displaces during MRI
- Patient has fatal hemorrhage during procedure
- Patient survived disease…killed by follow up

ODE TO A THIRD NERVE

When the eye is down and out with ptosis,
You better hope for miosis.
If the palsy is total with pupil sparing,
In an Oldie it’s vascular and not too daring.
A partial palsy calls for double duty,
Because it’s probably an aneurysm going through puberty.
But if the pupil is dilated,
An aneurysm has violated.
No time for deferral and no time for referral.
Send to the ER without debate.
Remember, twenty percent will die within the first forty-eight

Joseph Sowka, OD

47 YEAR FEMALE

- CC: Horizontal double vision in far left gaze
- BVA: 20/20 OD, OS
- Medical Hx: newly diagnosed diabetes
- Left abduction deficit in far left gaze
  - Negative forced duction test
- Mild ocular injection OS
- IOP: 14 mm Hg OD, 16 mm Hg OS
- Fundus: normal OU

47 YEAR OLD BLACK FEMALE

- Presumptive diagnosis: Left vasculogenic CN VI palsy- monitor
- Returns 1 week with marked worsening of injection, diplopia and ophthalmoplegia
- IOP: 16 mm Hg, 26 mm Hg
- Fundus disc congestion and vascular tortuosity OS

What does she look like NOW?
What do you want to do NOW?

47 YEAR OLD BLACK FEMALE

CT scan:
CAROTID CAVERNOUS SINUS FISTULA

Cavernous sinus...
- Trabeculated venous cavern
- Houses CN III, IV, VI, V1, oculosympathetics, and ICA
- Drains eye and Adnexa via inferior and superior ophthalmic veins to petrosal sinuses and jugular vein

Fistula...
- Rupture of ICA or meningeal branches within sinus
  - Meningeohypophyseal, McConnell’s Capsular, Inferior Cavernous
  - Mixing of arterial blood in venous system

CAROTID CAVERNOUS SINUS FISTULA

Hemodynamic
- High flow vs low flow

Angiographic
- ICA vs meningeal branches

Etiology
- Spontaneous vs traumatic
CAROTID CAVERNOUS SINUS FISTULA
- Increased venous pressure
- Orbital congestion
- Proptosis (pulsatile)
- Corneal exposure
- Arteriolization
- Orbital bruit
- Myopathies and cranial neuropathies with diplopia
- Secondary glaucoma

CAROTID CAVERNOUS SINUS FISTULA
- Vision threatening – not life threatening
- Spontaneous etiology – spontaneous resolution
  - ICA compression with contralateral hand
- Traumatic – clipping and ligation
- Balloon or particulate embolization
- Manage glaucoma aggressively
  - Prostaglandin analogs

RULE: BEWARE THE CHRONIC RED EYE
- Dilated & tortuous episcleral vessels that go to the limbus and back (omega loops) Ω
- Intervening “clear conjunctiva”
- Red eye that doesn’t respond to any topical treatments
  - Bag-o-Meds
- Other non-red eye findings: Chemosis, IOP elevation, proptosis, ophthalmoplegia, ptosis, lid edema
ODE TO A FISTULA

Beware the chronic red eye
It isn’t infected, inflamed, or dry.
When corkscrew vessels makes the eye reds
And the patient has bag-o-meds.
The problem is deep
And arterial blood has begun to seep.
Your first fistula you will always miss
But on your second case you will never be remiss

Joseph Sowka, OD

CASE:
23 YEAR OLD WHITE FEMALE

What questions do you want to ask?
What tests do you want to order?

Additional questions to ask:
• Any double vision? No!
• Any use of ophthalmic pharmaceuticals? No!
• Any history of migraine headaches? Maybe...

Differential diagnosis?
Aneurysmal compression on CN III? No
Pharmacological misadventure? No

BENIGN EPISODIC PUPILLARY MYDRIASIS

Episodic unilateral mydriasis
• Lasts minutes to weeks
Accompanied by blurred vision and headache
Young, healthy females (may have migraine history)
Peculiar sensations about affected eye
• Often progresses to headache
• Not typical migraine
Defective accommodation
Lid and motility defects not present
Extensive medical testing unremarkable

CASE:
23 YEAR OLD WHITE FEMALE

• CC: Sudden onset pupil dilation with ipsilateral headache
• Medical Hx: normal
• BVA: 20/20 OD, OS
• Pupils:
  • 3 mm anisocoria, OS larger, anisocoria greater in bright illumination. Previously isocoric. (-) RAPD, (+) Accom
  • Remainder of exam normal
  • Similar incident 2 days antecedent, resolved within hours
• What does she look like?
**BENIGN EPISODIC PUPILLARY MYDRIASIS**

- Increased sympathetic activity?
  - Reverse Horner’s syndrome – not likely
- Pupil paralysis following migraine?
  - Tends to last longer – not likely
  - No ophthalmoplegia
- Spasm of segment(s) of iris dilator muscle?
  - Round pupil, so not likely
- Pharmacologically dilated?
  - Parasympatholytic – no light or near reactivity
  - Sympathomimetic – can mimic and must R/O

**PUPIL RULES**

- Anisocoria greater in dim = sympathetic dysfunction
  - Horner’s syndrome- look for dilation lag
  - Miotic use
- Anisocoria greater in light = parasympathetic dysfunction
  - CN 3 palsy
  - Tonic pupil
  - Pharmacologic or traumatic pupil
    - No reactivity?

**RULE: ISOLATED DILATED PUPIL IS ALMOST NEVER AN ANEURYSM**

Ambulatory patients with isolated dilated pupil more likely to harbor iris or ganglion (Adie’s) lesion or medication misadventure than CN 3 palsy

- Comatose patient is a different story
- Risk of angiography is much higher than risk of aneurysm in this setting

- No imaging needed for isolated dilated pupil

**WORLD’S BEST DISC HEMORRHAGE**

- 33 YOWM
- Occipital HA x 4 mos
  - Visual aura with HA
- Worsens when standing after sitting
- Relieved by sleep
- Denies vision loss, nausea, diplopia, pain on eye movement, behavioral changes

**BENIGN EPISODIC PUPILLARY MYDRIASIS**

- Anisocoria greater in bright than dim
- Parasympathetic dysfunction
  - Not an aneurysm
  - Edinger-Westphall lesion?
- Migraine variant – most likely etiology
- Treatment – none except to avoid unnecessary testing
WORLD'S BEST DISC HEMORRHAGE

- 20/20 OD, OS with myopic correction
- Pupils, EOMs, conf fields normal OU
- Biomicroscopy normal OU
- IOP 12 mm Hg OU
- Nasally obliquely inserted nerves

Now what?

WORLD'S BEST DISC HEMORRHAGE

- Co-manage with PCP/internist
- MRI w/o and w/ contrast of brain and orbits
- Complete blood work blood work up including FTA-ABS/RPR; Lyme titer; CBC w/differential
- Rule out mass lesion, infections, collagen vascular and autoimmune etiology.

WORLD'S BEST DISC HEMORRHAGE

- MRI
- Pt had MRI done and mass was identified in fronto/parietal region more toward right side
- Outcome?
CONVERSATIONS IN OPTIC NERVE AND RETINAL VASCULAR DISEASE

Joseph Sowka, OD, FAAO, Diplomate

Dr. Joseph Sowka is a member of the advisory boards for Novartis, Glaukos, Allergan, and B&L. Dr. Sowka has no direct financial interest in any of the diseases, products or instrumentation mentioned in this presentation. He is a co-owner of Optometric Education Consultants. [www.optometricedu.com](http://www.optometricedu.com)

28 YOF

- Presents with intermittent blurred vision & visual "blackouts", intermittent diplopia, and chronic headache steadily worsening X 2 weeks
- MHx: "white coat hypertension", shoulder injury X 6 mos
- Meds: Flexeril® 10 mg BID PRN
- Height / weight: 5'3", 220 lbs.
- VA: OD 20/20, OS 20/20
- Pupils & motility: normal

28 YOF

- Additional hx: Dull "ringing" in ears
- BP: 142/100
- SLE: unremarkable
- T₄: OU 16 mm Hg
- VF: blind spot enlargement & nasal step defect OU
- Serology Normal
- Imaging: small ventricles, otherwise normal
- LP: O.P. = 510 mm H₂O; all CSF studies normal
- DX: Pseudotumor cerebri (PTC)

DEFINITION:

**PAPILLEDEMA:** EDEMA OF THE OPTIC DISC, SPECIFICALLY RESULTING FROM ELEVATED INTRACRANIAL PRESSURE.
PAPILLEDEMA: SIGNS & SYMPTOMS

**Signs:**
- bilateral disc edema
- superior & inferior aspects of discs affected FIRST
- obliteration of optic cup
- hemorrhages common
- absence of SVP
- Paton's folds
- highly variable VF defects
  - enlarged blind spot (early)
  - arcuate defects and constricted (late)
- NO RAPD typically
- VA near normal

**Symptoms:**
- Visual:
  - transient visual obscurations
  - intermittent horizontal diplopia
- General:
  - headache common
  - nausea & vomiting
  - dizziness
  - tinnitus

PAPILLEDEMA TYPES:

- **Acute**
  - Hemorrhages, exudates, hyperemia, RNFL edema
- **Chronic**
  - Minimal hemorrhage/exudate. Collateral vessels may be present
- **Atrophic**
  - Eventually occurs if papilledema remains chronic. Optic disc pallor

PAPILLEDEMA PATHOPHYSIOLOGY

- Disc edema results from axoplasmic stasis
  - intracellular fluids, metabolic by-products accumulate and are regurgitated at the level of the optic nerve head
  - in papilledema, cerebral edema is effectively transmitted along the common meningeal sheaths of the brain and optic nerve producing an engorged, swollen disc.

PAPILLEDEMA PATHOPHYSIOLOGY

- Associated with intracranial abnormalities:
  - increased brain volume (intracranial mass lesion)
  - increased intracranial blood volume
  - increased CSF volume
    - Hydrocephalus
    - Ventricular blockage by mass lesion

PAPILLEDEMA MANAGEMENT

- Rule out “swollen disc masqueraders”
  - ultrasonography can be invaluable in differentiating ONHD
  - also consider color, margins, SVP, vasculature, etc.
- **Acute papilledema constitutes a medical emergency**
  - Immediate neuro-imaging to rule out an intracranial mass.
  - If imaging is normal, lumbar puncture to measure CSF pressure and exclude meningitis or other disease processes is necessary.
- **Atrophic papilledema with significant vision/field loss:**
  - urgent measures must be undertaken to prevent blindness
- **Papilledema accompanied by any neurologic abnormalities, fever or stiff neck:**
  - Possible serious underlying neurologic abnormality, intracranial infection or bleed requiring immediate medical attention.

PTC VS. IIH

- **Pseudotumor Cerebri (PTC)**
  - Increased intracranial pressure in the absence of an intracranial mass lesion
  - Many causative agents have been identified
- **Idiopathic Intracranial Hypertension (IIH)**
  - Increased intracranial pressure without an identifiable cause
  - Young, obese females are at risk
- **Primary PTC**
  - IIH
- **Poor CFS drainage**
PSEUDOTUMOR CEREBRI

DIAGNOSIS

- **Si/SX**: consistent with increased ICP
- **Papilledema**
- **Normal neurological examination**
  - except for cranial nerve abnormalities
- **Neuro-imaging**: Normal without evidence of hydrocephalus, mass, or structural lesion, thrombosis
- **Normal CSF composition**
- Elevated LP opening pressure
  - Adults: > 250 mm CSF
  - Children: > 280 mm CSF
  - > 250 mm CSF if not sedated/obese

PSEUDOTUMOR CEREBRI

MANAGEMENT

- **No visual loss**
  - Symptomatic headache therapy
  - Acetazolamide 500 mg tid
  - Weight reduction
- **Mild visual loss**
  - Acetazolamide 500 mg tid
  - Furosemide, Topiramate, Zonisamide
  - Weight reduction

PSEUDOTUMOR CEREBRI

MANAGEMENT

- **No/ Mild visual loss**
  - **Prognosis**
    - Excellent (all signs and symptoms, visual loss)
    - 6-9 months
  - Follow-up and visual fields
- **Role of weight loss**
  - Treat the primary problem
    - 10% weight loss
  - Prevent recurrence
    - Keep the weight down

33 YOF

- Horizontal diplopia
- Headache
- TVOs 20/day
- Denies OCP, tetracyclines, vitamin A
- Lost 10 lbs- headaches improved
- 118/72
- 5’5”; 160lbs; BMI 26.62
**PSEUDOTUMOR CEREBRI MANAGEMENT**

- Severe, or progression of visual loss
  - Optic nerve sheath decompression (ONSD)
  - High-dose IV steroids and acetazolamide
  - Lumboperitoneal shunt
    - Failed ONSD
    - Declined ONSD
    - Intractable headache

**REMEMBER**

- Not all elevated discs are swollen, not all swollen discs are edematous, and not all edematous discs are papilledema
- True papilledema is a medical urgency and should be treated as such with a search for the cause.
- Many conditions can present with papilledema, including intracranial mass lesion, hydrocephalus, VST, PTC
- PTC is a diagnosis of EXCLUSION.

**ODE TO A SWOLLEN DISC**

When you think the disc is swollen,
The vessels north and south will appear stolen.
Not all elevated nerves are edematous,
Just like not all snakes are venomous.
Your thoughts should go to papilledema,
But infection and inflammation should still be in your schema.
MRI, MRV and LP, are soon to be.
Remember, pseudotumor is a diagnosis of exclusion,
Female and firm does not make pseudotumor a forgone conclusion.
Brain tumors can exist when the pseudotumor profile is classic.
Do the evaluation so they don’t end in a casket.

Joseph Sowka, OD

**WHICH IS BETTER? ONE OR TWO?**

**48 YOM**

Painless loss of visual field OS
- 20/20 OD, OS
- Noticed upon waking

Med Hx: Unremarkable, except for viral illness 3 weeks before
74 YOM

- Presents with ‘worst headache of his life’
  - Sees: PA, ED physician; cardiologist; NP;
  - 3 week period
  - Histories: Eye ache; jaw pain, scalp pain, facial pain, somnolence; malaise; jaw claudication
  - Diagnoses: TMJ; Lyme disease
  - “vasculitis such as temporal arteritis highly unlikely”, “Not GCA”
    - However, ESR and CRP ordered and elevated- never reviewed
  - Ultimately OD makes diagnosis
  - End result?

ANTEOR ISCHEMIC OPTIC NEUROPATHY

- Hypoperfusion of the posterior ciliary arterial supply to the anterior optic nerve head.
- May be arteritic (AAION) or non-arteritic (NAAION)
- Mechanical factors and atherosclerotic disease play a role in the non-arteritic form while vasculitis contributes in the arteritic form.
- Unilateral presentation but high incidence of subsequent contralateral involvement
  - AAION

AAION VS NAAION

- Risk factors:
  - Hypertension, diabetes, atherosclerotic disease, small optic nerves
- Inferior field defects
- Hyperemic swollen nerve- disc at risk
- Progressive moderate vision loss with potential recovery
- Late 30s/ early 40s and beyond
- Painless
AAION

- Pallid optic nerve swelling with flame hemorrhages, arteriole attenuation and NFL infarcts
- Pain (of some sort)
- Severe optic nerve dysfunction
- Visual field defects
- Giant cell arteritis/ PMR- risk factors
- Typically 70s, uncommon under 60
- High risk bilateral involvement

DIAGNOSIS

- Careful history: Must directly ask about nonvisual symptoms
  - Headache (present in over 90%), scalp tenderness, jaw claudication (almost diagnostic), ear pain, arthritis, temple pain and/or tenderness, malaise, intermittent fevers
- Examination
- Laboratory studies
  - Erythrocyte sedimentation rate
    - Lowered by statins and NSAIDS
  - C-reactive protein
    - Not affected by statins and NSAIDS
  - Elevated platelet count

TREATMENT

- Prompt steroids and hydration
- Consider IV when vision loss present
  - Very effective in prevention of second eye
  - Occasionally restores vision

AAION VERSUS NAAION

- Think AAION>>NAAION
  - Systemic symptoms of GCA
  - TVOs/amaurosis
  - Elevated ESR/CRP
  - Platelets
  - AION + cilioretinal artery occlusion
  - Evidence of posterior ciliary artery occlusion on FA
  - Early massive vision loss
  - Chalky white optic disc edema

WHICH IS BETTER? ONE OR TWO?

- Bilaterally blind
- Residual field loss, but otherwise not bothered

ODE TO AN ISCHEMIC NERVE

When your patient’s optic nerve is ischemic
You better hope the disc is hyperemic.
In Non-arteritic no treatment is needed
And life will rarely be impeded.
But if the disc is swollen and pale,
And vision is an epic fail
If the patient is sixties, seventies or eighties
You will feel heat like in Hades
ESR and CRP are required
And steroids must be acquired
Remember, when you see a choked disc
Always assess the giant cell risk

Joseph Sowka, OD

29 YOF

- Referred for glaucoma evaluation due to suspicious cupping - no complaints
- IOP 12 mm OD, 13 mm OS

“Now let’s get serious”

- 20/15 OD, OS
- IOP: 12 mm Hg OD, 13 mm OS
- CCT: 493 OD, 488 OS
- Gonio normal OU
- +RAPD OS

Segmental disc pallor OS

OPTIC ATROPHY

- Primary optic atrophy
  - Uniform nerve fiber degeneration, resulting in glial replacement but no architectural alteration of the optic nerve head.
  - Disc appears chalky white but the margins remain distinct and retinal vessels appear normal.
    - Trauma and compression (e.g. tumor) causes
- Secondary optic atrophy
  - Results from pathological chronic disc edema
    - Malignant hypertension, papilledema, or infiltrative diseases like leukemia or sarcoidosis.

- Consecutive optic atrophy
  - Degenerative retinal conditions
    - Retinitis pigmentosa, pathological myopia and central retinal artery occlusion.
    - Pale, waxy disc, normal margins and marked attenuation of the arterioles.
- Temporal disc pallor
  - Toxic/nutritional (bilateral) or demyelinating optic neuropathy (optic neuritis)
**OPTIC ATROPHY**

- Numerous potential etiologies
  - Infarction, infection, infiltration, inflammation, trauma, toxicity, metabolic dysfunction or direct compression of the nerve or chiasm
- Evaluation:
  - MRI studies should be obtained of the orbits, the optic chiasm and the brain with and without contrast, fat suppression for orbits, in a high field scanning unit.
  - Contrast dye (gadolinium) is beneficial in discerning malignant lesions, demyelinating plaques indicative of multiple sclerosis.

All cases of optic nerve pallor/ optic atrophy must be investigated or explained.

**OPTIC ATROPHY**

- Systemic causes of optic atrophy
  - sarcoidosis, tuberculosis, Behçet's disease, lymphoma, leukemia, systemic lupus erythematosus, nutritional or metabolic disorder (e.g. pernicious anemia, folate deficiency), syphilis, Lyme disease, and antiphospholipid antibody syndrome.
- CBC, ESR, ACE, ANA, serum cardiolipin, serum homocysteine, serum B12 and folate levels, and rapid plasma regain (RPR) for syphilis.
  - Additionally, chest x-rays could prove helpful in suspected cases of TB or sarcoidosis.

---

**29 YOF - OUTCOME**

- MRI orbits- normal (limited/ poor study)
- Repeat MRI brain- no lesions
- Lupus panel, ANA, DS DNA, ESR, metabolic panel, Vit B12/folate- normal
- RPR, HIV- non-reactive

---

**A FAMILY AFFAIR**

- 56 YOBF
- Dx POAG OU 5 years ago
- Slowly progressive vision loss
- LP OD; 20/30 OS
- Used combo med- ran out months ago
- IOP: 19 mm OD, 18 mm OS
- CCT: 560; 544
54 YOM

- Referred for glaucoma management
- Told he had glaucoma 6 years earlier - no Tx
- 20/30 OD; HM OS
- 30 mm Hg OD; 23 mm Hg OS

Distinct rim pallor
OS
Cupping does not match vision

COMPRESSION OPTIC NEUROPATHY

- Results from compression of the optic nerve within orbit, at the orbital apex, chiasm-secondary to:
  - Space occupying orbital lesions, including tumor masses
  - Infiltrated extraocular muscles (Graves' ophthalmopathy) in thyroid disease (most common)
- Unilateral with orbital masses, can be bilateral in Graves’ disease
- Presents with slowly progressive, variable vision loss; variable proptosis and motility restriction

COMPRESSIVE OPTIC NEUROPATHY

- Optic nerve may be initially hyperemic with retinal edema, tortuous vessels, and associated hemorrhages; with prolonged compression, may see pallor and optic disc collateral vessels
- Visual fields consistent with papilledema in early stages, ischemic optic neuropathy/glaucoma in later stages
- Increased concentric ‘cupping’ can occur
  - Compression causes pallor; glaucoma causes notching
- Management involves orbital imaging and serum thyroid profile if Graves’ suspected

ODE TO A CUPPED DISC

Oh, to have a cupped disc pink.
That my friend hath a glaucomatous stink.
But to have a cupped disc pale,
Call this glaucoma and you shall fail.
Disc and field damage that is one-sided
Simply cannot be abided.
It might be trauma, infarct or meningioma.
But if the rim is cut always remember,
Nothing notches a nerve like glaucoma

Joseph Sowka, OD
42 YOF
- Sudden painless loss of vision OS x 1 week
  - getting worse, not getting better
  - began as dimming, then rapidly dropped off
- BVA: 20/20 OD; 20/400 OS
- PERRL (+) RAPD OS (mild)
- Conf. Fields: FTFC peripherally OD, OS
- Amsler: Central/ceccocentral scotoma OS
- SLE: normal OU
- IOP: 18 mm Hg OD, 19 mm Hg OS

42 YOF
- No known HIV risk
- Recent illness: Severe flu with malaise, fever, and lymphadenopathy 4 weeks antecedent.
- No tick bites or rashes
- Exposure to cats
- Serology:
  - FTA-ABS/RPR; HIV, Lyme, toxoplasmosis, toxocariasis, PPD: Negative
  - Bartonella henselae titers: positive
- Dx: Cat scratch neuroretinitis

INFECTIONOUS OPTIC NEUROPATHY
- Syphilis
  - Retrobulbar, papillopathy, neuroretinitis, perineuritis
  - Retrobular, bulbar: severe vision reduction
  - Perineuritis has normal vision, MRI optic sheath enhancement
- Lyme - mimics syphilitic optic neuropathy
  - Bite of mammalian deer tick- can cross react with syphilis
- Toxoplasmosis, HIV/AIDS, CMV
  - Destructive to vision
- Neuroretinitis
  - Typically benign lymphoreticulosis (cat scratch disease)

NEURORETINITIS
- Mild RAPD compared to vision loss
  - Vision loss more retinal than optic nerve
- Serous macular RD
  - OCT shows subretinal fluid between disc and macula in cases with disc edema only
- Macular star late finding

62 YOF
- ‘Strep throat’
- CF @ 8’ OD, 20/25 OS – antibiotics x 1 day
- RAPD OD
- Black spot and blurry vision 3 days
NEURORETINITIS

- Many potential etiologies
  - Toxoplasmosis, toxocariasis, measles, syphilis, Lyme disease, herpes simplex and zoster, mumps, tuberculosis, malignant hypertension, ischemic optic neuropathy, and leptospirosis, bartonella (most common). Fleas are vectors, thus no need for actual scratch.
  - Prognosis for visual recovery excellent, especially if the cause is cat scratch disease.
  - Most patients will have a return to normal or near normal vision without
  - Antimicrobial therapy may be used to hasten recovery.
    - Rifampin, doxycycline, and azithromycin for one month.
    - Fleas are vectors, thus no need for actual scratch.
  - While antibiotics are frequently used for cat scratch disease neuroretinitis, there are no controlled clinical trials that indicate a better clinical outcome from this therapy. The same can be said for the use of oral steroids and intravitreal anti-angiogenic medications.

ODE TO AN INFECTED NERVE

When the vision is poor and the APD mild,
It's often the bite of something wild.
If the disc is swollen and macular swelling great,
Its neuroretinitis and the star comes late.
Syphilis and Lyme are alike,
and can cause similar titres to spike.
One is transmitted sexually and the other not,
Unless the patient is weirder than you thought.

Joseph Sowka, OD

34 YOF

Patient referred by PCP for complete ocular evaluation
CC: sudden onset “foggy” vision and pain OS X 2 days
  - retrobulbar pain; exacerbated with eye movement
Medical Hx:
  - Hodgkin’s lymphoma X 2 years, currently in remission
  - most recent Gallium scan negative
  - (-) meds, (-) allergies

BVA: OD 20/15, OS 20/25
Confrontation Fields
  - full OD
  - dense constriction OS
Color Vision (Ishihara):
  - OD 8/8
  - OS 1/8

Biomicroscopy:
  • all external structures normal OD & OS
Tonometry (Goldmann):
  • 14 mm Hg OD
  • 14 mm Hg OS

Presumptive diagnosis?

Presumptive diagnosis: Left optic neuritis
Differential diagnosis: demyelinating, infiltrative, infectious optic neuropathy
Plans: Automated perimetry - patient scheduled to return in 16 hours
Refer for neuroimaging and hematological studies
34 YOF

- Patient returns for perimetry - acuity now NLP OS
- Patient immediately referred, admitted for evaluation:
  - MRI (with contrast, brain & optic nerves):
    - no UBO’s identified, no “meningeal enhancement”
  - HEMATOLOGY:
    - ESR = 37 mm/hr
    - ANA (+)
    - D/S DNA initially (+), then (-) on repeat testing
  - CSF STUDIES:
    - normal ICP (O.P. = 180 mm Hg)
    - normal cell count, no evidence of lymphoma

34 YOF

- Patient admitted to hospital for testing and therapy - I.V. corticosteroids
  - 250 mg methylprednisone sodium succinate QID
  - therapy was poorly tolerated; discontinued after 3 days - no oral steroids given
  - Patient discharged after four days
  - Subsequent evaluations (2 weeks, 4 weeks, 3 months, 6 months) demonstrated no improvement in visual acuity
  - Presumptive Etiology:
    - Infiltrative Optic Neuropathy Secondary to Lymphoma

INFLTRATIVE OPTIC NEUROPATHY

Disorders associated with infiltrative neuropathy:
- Sarcoidosis
- Systemic lupus erythematosus
- Leukemia
- Lymphoma
- Metastatic cancer
- Primary optic nerve tumors

INFLTRATIVE OPTIC NEUROPATHY:
MEDICAL MANAGEMENT

- Neuroimaging (MRI w/ & w/o gad preferred)
- LP (if neuroimaging is negative)
- Hematology and serology:
  - ANA, ACE, D/S DNA
  - histologic analysis for leukemic and lymphocytic cells
  - consider also FTA-Abs & RPR, ELISA-HIV
- Evaluation by GP

NOW FOR SOME RETINA...
CASE: I (DON’T) FEEL GOOD!

- 66 year old Black male
- CC: sudden, painless blurring OS x 3 days
- No previous eye or medical care
- Wants glasses to clear vision
- BVA OD 20/30, OS HM
- Pupils: ERRL (+) RAPD OS
- Good appetite, poor diet

CENTRAL RETINAL ARTERY OCCLUSION (CRAO)

- Painless, sudden loss of vision
  - < 20/400 in most cases
- Retinal edema and white fundus – hypoperfusion
  - Cherry red spot
- 60’s and above
- Early and late appearances
  - Initially normal fundus
  - Optic atrophy with attenuated vessels

CRAO: ETIOLOGY

- Emboli from heart or carotid lodging at lamina
- Intraluminal thrombosis
- Dissecting aneurysm
- Vasospasm
- Arteriolar necrosis
- GIANT CELL ARTERITIS!

CRAO: TREATMENT?

- Paracentesis
- Carbogen
- Digital massage
- Hyperventilation
- Urokinase/streptokinase
- 1-24 hr window of opportunity
- Does anything work?

CRAO: SYSTEMIC CONSIDERATIONS

- Atherosclerosis
- Carotid artery disease
- GCA
- Antiphospholipids ABS
- Infectious endocarditis
- Vasospastic disease
- Cardiac arrhythmia
- Clotting factor abnormalities
- Hypertension
- Diabetes
- Cardiac valve disease
- Cardiovascular disease
- Hyperlipidemia
- Disc drusen
- Mural thrombosis
- Hyperviscosity syndromes
CRAO: COMPLICATIONS
- CVA
- MI - Main cause of death
  - 9 yr mortality 56%
- Fellow eye involvement if GCA cause
- ESR and CRP for GCA
- Cardiology/ internal medicine referral
- Neo not common

JAMES’ OUTCOME
- Referred for medical care
- Diagnosed with hypertension, NIDDM, hypercholesterolemia
- Returns for ocular follow up 3 months later
  - “I’m scared”
- Several toes amputated from diabetes
- Passed away from MI within year

BRAO; CILIoretinal AO
- BRAO nearly always embolic
- Greater risk of cardiac mortality
- Cilioretinal AO- branch of PCA- high risk of GCA

Guidelines
- 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

All Patients with Acute Retinal Arterial Ischemia
- MUST have immediate brain imaging
  - Brain MRI with DWI >>> Head CT
- Including patients with transient visual loss (presumed of vascular origin)

Concurrent Acute Brain Infarcts in Patients with Monocular Visual Loss
- ¼ with acute retinal ischemia had acute brain infarction (anywhere) on brain DWI-MRI
- Infarctions often small, multiple, ipsilateral to retinal ischemia, asymptomatic

- DWI-MRI abnormal in:
  - 33% with CRAO/BRAO vs 18% with TVL
  - 28% with embolic vs 8% non-embolic retinal ischemia

Adapted from Drs. Nancy Newman and Biousse; 2015
ODE TO AN ARTERY OCCLUSION

When the vision is poor and the fundus is pale,
A branch or laminar emboli has caused the fail.
Heroic measures are rarely helpful,
And vision return is doubtful.
In an Oldie, always remember giant cell it may be.
Hurry and get an ESR and CRP.
The retina is infarcted and dead,
So neo you should not dread.
But here is where you must not choke,
Send them to the ER because they are having a stroke

Joseph Sowka, OD

THE CASE OF THE COLORED FLASHING LIGHTS

• 45 YOHF presented with colored “map-like” phosphenes and small black flashing spots OD x two weeks
• Noted that she had to “look between the lights” to see out of her right eye.
  - 20/20 OD, OS
• Medical history was unremarkable except for treated migraines
• Lost 1 pregnancy

Study #2

Co-occurrence of Acute Retinal Artery Occlusion and Acute Ischemic Stroke: Diffusion-Weighted Magnetic Resonance Imaging Study

JAEWOOD LEE, SULG CHOE, SE, DO-WOO, KIM, YOUNG DAE KI, AND BOURISSE

Am J Ophthalmol 2014; 157: 1231-1238

Adapted from Drs. Nancy Newman and Biousse; 2015

Tell the patient:

• “Go to the Emergency Department”
• “Tell them you had a retinal stroke”
• Do not send these patients to their PCP, cardiologist, neurologist, neuro-ophthalmologist
• Do not try to obtain the workup yourself

Co-occurrence of acute retinal artery occlusion and acute ischemic stroke: Diffusion-weighted magnetic resonance imaging study

• 33 patients with CRAO (48) and BRAO (25)
• Evaluated similarly to acute stroke patients (DWI)
• ¼ with acute retinal ischemia had acute brain infarction (anywhere) on brain DWI-MRI
  - 5/18 CRAO; 3/15 BRAO
  - Infarctions often small, multiple, ipsilateral to retinal ischemia, may be asymptomatic
  - Abnormal DWI-MRI strongly correlated with major cause of stroke (even when neurologically asymptomatic)

Adapted from Drs. Nancy Newman and Biousse; 2015

DWI in Acute Retinal TIA/Ischemia

• DWI-MRI identifies subgroup of patients at very high risk of major stroke
• DWI-MRI needs to be performed within 24/48 hours of visual loss to allow for effective prevention of recurrent stroke

Adapted from Drs. Nancy Newman and Biousse; 2015

THE CASE OF THE COLORED FLASHING LIGHTS

• 45 YOHF presented with colored “map-like” phosphenes and small black flashing spots OD x two weeks
• Noted that she had to “look between the lights” to see out of her right eye.
  - 20/20 OD, OS
• Medical history was unremarkable except for treated migraines
• Lost 1 pregnancy
CASE CONTINUED

- She returned four days later complaining of decreased vision in the right eye, which had reduced to counting fingers at ten feet.
  - Macular edema, more extensive hemorrhaging, cotton wool spots, disc edema and dilated vessels
- Underwent IV Kenalog injections and showed improved vision of 20/70 OD during follow up examinations.
  - Released by retinal specialist
  - No medical evaluation

CRVO: SYSTEMIC CONSIDERATIONS

<table>
<thead>
<tr>
<th>Hypertension</th>
<th>Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperviscosity</td>
<td>Syphilis</td>
</tr>
<tr>
<td>CV disease</td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Sickle</td>
<td>Leukemia</td>
</tr>
<tr>
<td>Polycythemia</td>
<td>Carotid artery disease</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>Sarcoid</td>
</tr>
<tr>
<td>Autoimmune factors</td>
<td>Clotting abnormalities</td>
</tr>
<tr>
<td>Homocysteine</td>
<td></td>
</tr>
</tbody>
</table>

TREATMENT & MANAGEMENT

- Referred blood work through PCP
  - DM, HTN, hypercoag, ANA, antiphospholipid antibodies, anticardiolipin, PT, PTT, ESR, CBC with diff
- Elevated erythrocyte sedimentation rate
- Mildly elevated cholesterol level.
- Elevated anti-cardiolipin IgM antibodies
  - Suggestive of antiphospholipid antibody syndrome
  - She was recommended for long term anti-coagulant therapy to prevent future thrombotic events, but patient never followed through.

Now
What?
Are there any tests that you would like to order?

So,
What’s your diagnosis?
Management...?
CASE CONTINUED
- Seven months later the patient returned with the same signs and symptoms in her right eye.
- At this time, the vision was markedly more decreased with more evidence of ischemia - CF @ 6'
- She was referred to a hematologist
- Now on anti-coagulation therapy

CENTRAL RETINAL VEIN OCCLUSION
- Thrombotic/atherosclerotic phenomenon
- Properties of blood and vein act in concert
- Vascular flow and vessel wall abnormalities
- Problem at lamina - Turbulent flow - Decreased luminal pressure - Thrombus
- Perfused; non-perfused; indeterminant
- Evolving condition

MANAGEMENT-CRVO
- Standard Care vs. Corticosteroid for Retinal Vein Occlusion (SCORE)
  - 1-mg of IV triamcinolone should be considered for one to two years to improve vision loss secondary to macular edema following a CRVO.
  - CRUISE Results
    - Demonstrated efficacy for Lucentis treatment

PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME
- Thrombotic disorder
- Secondary antiphospholipid syndrome
  - Associated several autoimmune diseases but most often systemic lupus erythematosus
- Primary antiphospholipid syndrome is not associated with further systemic disease
- Recurrent vascular thrombosis, pregnancy loss and positive anticardiolipin or lupus anticoagulant are all characteristics of this disorder

PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME
- The clinical criteria
  - One or more vascular thrombotic episodes of venous, arterial or small vessel thrombosis in any organ or tissue or spontaneous abortion.
- Laboratory testing must show persistently elevated anticardiolipin antibodies, IgG and/or IgM or lupus anticoagulant (inhibits the conversion of prothrombin to thrombin) at least six weeks apart
**PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME**

- Phospholipids are identified by the body as "foreign."
  - The antiphospholipid antibodies are produced against the "foreign" antigen.
- The antibodies appear to react with the cell membranes causing irritation or stimulation, thus disrupting the coagulation cascade
- This disruption leads to abnormal blood clotting and inhibits normal phospholipid binding.

- This abnormal or inhibition of proper phospholipid binding leads to a hypercoagulable state thus causing thrombosis.
- Propensity of clot formation is within the venous and arterial portions of the vascular tree, especially targeting the retinal vessels and placenta.

---

**MORAL OF THE STORY**

*Just because you refer somebody out doesn’t mean that everything will be addressed*

**50 YOIF**

- POAG OU x 10 years- medically controlled
  - PGA, beta blocker
- Hx CVA at age 17
  - No cause found
- N/S x 1 year
- Presents with sudden onset vision loss OD (6 hrs)
  - IOP 22 mm OU; using PGA, not using beta blocker
- 20/100 OD; 20/20 OS; 3+ RAPD OD
  - Never present before

**Ok, now that we have warmed up... Let’s see if we can figure this one out.**
So, What are your thoughts?

- Digital massage and combigan given
  - No improvement
- Recommend retinal consult for angiogram- pt initially declines
  - Pt ultimately sees retinal specialist next day
- Angiogram normal.
  - Normal arterial filling 'somewhat delayed' venous filling.
  - No evidence of edema or ischemia
  - pt released

- Pt returns 6 days later
- Some visual improvement
  - 20/60 OD
  - RAPD now grade 2
  - IOP 12 mm OU
  - Ischemia
  - f/u 1 mos

- Pt returns 3 weeks later
- Vision improved to 20/30
- RAPD diminished to grade 1

So, What are your thoughts?
CRVO? CRAO? Variant?
Reappointed for 1 month
Pt returns as scheduled- vision improved
- 20/25+
- RAPD disappeared

QUESTIONS
- Artery or vein occlusion?
- Why OCT and FANG normal?
- How does RAPD form and disappear over 2 months?

THE MEDICINE MAKES ME SICK
- 52 YOWF
- Medical history: hypertension x 10 years; NIDDM x 2 yrs
  - Medicines unknown
  - Poorly controlled
  - Pt non-compliant
  - "God will take care of me"
- BP: 157/109 RAS

So, What do you think?
**HYPERTENSIVE RETINOPATHY**

- Arteriolosclerotic vessel changes
  - Some classification schemes include vessel changes in hypertensive retinopathy and others don’t
- Elschning’s spots – subtle choroidal infarcts
- CWS
- Flame shaped hemorrhages
- Macular edema (rare)
- Macular star/ ring of exudates
- Disc edema

**NOW A TWIST**

- 47 YOBM
- Obese
  - 400 lbs (and that’s being kind!)
- Headaches x 3 months
- Vision reduction x 2 months
  - 20/50 OU
- BP: 212/155 RAS

**BLOOD PRESSURE**

- “Normal” blood pressure: ≤ 120/80 (systolic / diastolic) JNC 7, 2003
- Hypertension is defined as any elevation of blood pressure above the norm, as measured by sphygmomanometry on two separate occasions

| Prehypertension: 120-139 (S) and/or 80-89 (D) |
| Stage 1 hypertension: 140-159 and/or 90-99 |
| Stage 2 hypertension (severe): ≥160 and/or ≥100 |

**HYPERTENSIVE CRISIS**

- Hypertensive EMERGENCIES
- Hypertensive URGENCIES
HYPERTENSIVE EMERGENCY
• Severe Hypertension + End-Organ Damage
  - Examples of end-organ damage… hypertensive encephalopathy, intracerebral hemorrhage, acute myocardial infarction, left ventricular failure with pulmonary edema, acute coronary syndrome, dissecting aortic aneurysm, or eclampsia.
• Hypertensive EMERGENCIES require immediate BP reduction (not necessarily to normal ranges) to prevent or limit organ damage.
  - Patients with hypertensive emergencies are often admitted through the ER for aggressive treatment.

HYPERTENSIVE URGENCY
• Severe Hypertension + NO End-Organ Damage
  - Typically identified during routine evaluation
  - Usually represents chronic hypertension, nonadherence to drug therapy or inadequate treatment by the PCP.

INDUCTION OF ADVERSE EVENTS SECONDARY TO TOPICAL PHENYLEPHRINE
• Regarding 2.5% phenylephrine (PE), numerous reports suggest there is little concern over adverse responses:
  - Jennings et al (1986) – 252 patients (3 – 92 years); no significant changes in systolic or diastolic BP in patients dilated with 2.5% PE.
  - Malhotra et al (1998) – 54 consecutive patients undergoing cataract extraction; no sustained changes in BP or heart rate after 2.5% PE.
  - Lam et al (2003) – 217 consecutive patients undergoing phacoemulsification; no untoward cardiovascular effects with 2.5% PE.

• Pharmacologic dilation can help to identify target end-organ damage, particularly hypertensive encephalopathy (Stage 4 hypertensive retinopathy) and intracerebral hemorrhage (Terson’s syndrome). Therefore, in patients with significantly elevated BP, dilated funduscopy is of PARAMOUNT importance, but…

IS THERE A SUBSTANTIAL RISK TO THE PATIENT??
CONCLUSIONS & RECOMMENDATIONS

- Based on data submitted to the National Registry of Drug-Induced Ocular Side Effects:
  - 2.5% PE is recommended for routine pharmacologic dilation.
  - 10% PE should be avoided in the elderly, infants, and patients with cardiac disease, idiopathic orthostatic hypotension, hypertension, aneurysms, Type 2 diabetes, and advanced arteriosclerosis.
  - 10% PE should also be avoided in patients using MAO inhibitors, tricyclic antidepressants, reserpine, guanethidine, or methyldopa.

ARE THERE ANY QUESTIONS?

IMPENDING STROKE

- AF and TIA
  - hemiparesis, hemisensory loss, hemifacial weakness of upper motor neuron distribution, amaurosis fugax, and aphasia
    - < 1 hr; no changes on neuroimaging
    - AF, light-induced, gaze-induced
- Atherosclerosis with subsequent visible retinal emboli formation from hypertension
  - Cholesterol → fatty streak → atheroma → ulceration → thrombus → plaques → emboli

STAGES OF ATHEROSCLEROSIS

- Healthy artery
- Build-up of plaque
- Plaque bulging
- Plaque rupture
- Plaques, thrombus, emboli
**Visible Retinal Emboli**

- **Fibrin/platelet aggregate (Fisher plaque-carotid in origin, also walls of arteries and valves of heart)**
  - Dull gray or white
  - Readily migrate through vascular system producing symptoms (AF)

- **Hollenhorst-cholesterol (carotid in origin)**
  - Refractile, glistening, yellow
  - Most common (87%) of all emboli
  - Typically do not occlude artery
  - Malleable and allows for blood to pass through the artery may appear totally blocked
  - Will readily break up and move distally, so will not be seen typically in patients complaining of AF
  - Common cause of AF

- **Calcific (cardiac)**
  - Dull white and non-refractile
  - Usually from valvular calcification
  - Most likely to cause artery occlusion and stroke

**TIA**

- **Anterior circulation**
  - Internal carotid, middle cerebral, anterior cerebral arteries, and their tributaries.
  - Receives 80% of the cerebral blood flow and accounts for 80% of transient ischemic attacks and strokes.
  - Amaurosis fugax indicates an abnormality in the ophthalmic branch of the internal carotid artery distal to the bifurcation
    - CRA, BRA

**TIA/AF**

- **Emboli-forming conditions (other than HTN)**
  - Rheumatic heart disease
  - Prosthetic heart valves
  - Bacterial endocarditis
  - Indwelling catheters
  - Rhythm disturbance - Mitral valve prolapse

- **GCA**
  - Thrombus, not emboli

- **Vasospastic**
  - Non-embolic arterial narrowing
  - Vasospastic substance (Cocaine) use
  - Migraine?

- **Hematological**
  - Polycythemia (Too much hemoglobin)
  - Sickle cell (#1 hematological cause of transient vision loss)
  - Anemia (Too little hemoglobin)
  - Hypercoagulability states
  - Anti-phospholipid antibody syndrome

- **Possibility of a subsequent CVA**

- **TIA also indicates widespread vascular disease putting patients at risk of myocardial infarction and cardiac death**

- **Patients with amaurosis fugax as only manifestation of TIA are at lesser risk than pts. with hemispheric TIA**
  - Risk of arterial occlusion (with permanent vision loss), CVA, and MI.

- **Risk of future events for TIA dictated by cause and degree of carotid stenosis**
THANK YOU FOR YOUR ATTENTION.
ALWAYS REMEMBER TO RECYCLE AND
PROTECT THE PLANET THAT WE WILL
ULTIMATELY LEAVE TO KEITH RICHARDS
DEPRESS YOUR PATIENTS
DIAGNOSIS AND MANAGEMENT
OF PERIPHERAL RETINAL DISEASE

William D. Townsend, O.D.,
FAAO
Canyon, Texas
Adjunct Professor, UHCO

Resources
Goals of this Course
- Review normal vitreous & peripheral retina anatomy
- Understand normal interactions between vitreous-retina
- Recognize abnormal interactions between vitreous-retina
- Review non-threatening vitreo-retinal conditions
- Discuss sight-threatening vitreo-retinal conditions

The Vital Relationship Between Vitreous and Retina

Most retinal detachments are rhegmatogenous, i.e. caused by holes & tears (breaks)
- Retinal breaks typically result from anomalous attachments @ the vitreous-retinal interface
- Conditions that accelerate vitreous degeneration promote retinal breaks
  - High myopia
  - Trauma
  - Aging
  - Cataract surgery
  - Intraocular surgery
Vitreous Macrostructure
- 98%–99% water
- Vitreous cortex (posterior hyaloid)- peripheral layer of vitreous
  - Inserts into ILM of the retina
  - Dense structure due to increased number of collagen II fibers
- Central vitreous
  - Predominantly hyaluronic acid
  - Less dense due to low collagen content

Vitreous Anatomy: Child

Condensed Collagen Fibers
@ 4 years of age human vitreous overlying the macula starts to liquefy
By 14 -18 years of age, 20% of vitreous is liquid
80 - 90 years of age > 50% of vitreous is liquid
These are normal aging changes

Evaluated highly myopic children (> -6.00D)
46% had some form of peripheral lesion
- Lattice degeneration 20%
- With w/o pressure 11%
- Retinal hole w/ SR fluid 4%
- PVD 2%
- 39% disc findings, 17% macular findings
- 30% were normal
- Children w/ retinal detachment were less likely than adults to report symptoms

Bansal AS, Hubbard GB. Peripheral retinal findings in highly myopic children < 10 years of age. Retina 2010 Vol. 30; No 4
Retinal Detachment: What are the odds?
- Otherwise normal eyes - 5 in 100,000 per year
- Middle age & elderly eyes - 20 in 100,000 per year (4X)
- Highly myopic eyes - 5,000 in 100,000 per year (1,000X)
- Highly myopic eyes after cataract surgery - 7,000

Complications of Vitreous Degeneration
- Directly impact genesis of vitreo-retinal pathology
- Vitreous liquefaction & ensuing posterior vitreous detachment may lead to:
  - Retinal break, detachment
  - Vitreo-macular traction syndrome
  - Epiretinal membrane
  - Macular hole


But wait, there’s more!
- The extent of vitreous degeneration correlates with NS cataract formation
- POAG is associated with both premature vitreous liquefaction & lensectomy
- After vitrectomy incidence of glaucoma:
  - Liquified vitreous reduces O2 available to retina, increases oxidative stress to TM
  - Within 4 years post-vitrectomy 8% of eyes → glaucoma
  - During remaining lifespan, 15%-20% of eyes → glaucoma

Addressing PVD – Phone to Fundus

- Staff: give special attention to callers with flashes and floaters - “You will be dilated.”
- Symptoms: photopsia present? type of floater?
- Onset?
- History:
  - Fellow eye
  - FHx retinal detachment
  - Trauma
  - Onset flashes, floaters
- How soon should we see this patient?

Tools” For Evaluating The Fundus

- Slit lamp evaluation
  - Anterior chamber, vitreous
  - Three mirror fundus lens
  - Digital wide field (78 D)

- BIO
  - Dark room- dark adapted
  - Tilting exam chair
  - 20D or 30D lens
  - Scleral indenter

Bad Prognostic Indicators in PVD

- Photopsia
- Hemorrhage - R/O retinal break
- Pigment in vitreous- found 70% of retinal tears
  - Cellular or free melanin released from RPE via full-thickness retinal break
  - AKA “tobacco dust”
  - AKA “Schaffer’s sign”
- Pigment- anterior vitreous space
Where are vitreous and retina most and least adherent?

1. Macula
2. Optic nerve
3. Ora serrata
4. Vitreous base
5. General vitreous-retina interface

Is this really important?

Normal Vitreo-Retinal Attachments

1. Vitreous base  strongest
2. Optic nerve
3. Macula
4. Blood vessels  weakest
5. Vitreous-retinal interface

It's all about connections!
Vitreous base
Cloquet's canal
Vitreous-disc attachment

Anomalous vitreo-retinal attachments

Zones of the Peripheral Retina
Vortex vein
Pars Plana
Ora Serrata
Oral zone
Equatorial zone
Equator
Juxtabasal tears
Intrabasal tears here

Vortex vein ampulla
Short ciliary nerves
Long ciliary nerves
Peripheral senile pigment degeneration
Cystoid retinal degeneration
Oral pearls
Cobblestone (pavingstone) degeneration
Non-cystic retinal tufts
Congenital RPE hyperplasia
Retinal white without pressure

Appearance - areas of "salt & pepper" hyper-hypo pigmentation
Reticular form has net-like appearance
Histology - RPE degeneration w/ pigment migration
Occurrence - 20% of population > 40 years of age
Risk - None
Differential - Retinitis pigmentosa
Management - None
Peripheral Retinal Pigment Degeneration

- Appearance: Grayish/white retina with small red spots (cysts) located adjacent to ora serrata
- Histology: Thickened retina in which tissue loss has created cystic spaces
- Occurrence: Typical form - all individuals over 8 yrs. of age, seen in autopsy eyes of neonates
- Risk: None (precursor to retinoschisis)
- Differential: Retinal edema, white without pressure
- Management: None

Peripheral Cystoid Degeneration

- Appearance: Grayish/white retina with small red spots (cysts) located adjacent to ora serrata
- Histology: Thickened retina in which tissue loss has created cystic spaces
- Occurrence: Typical form - all individuals over 8 yrs. of age, seen in autopsy eyes of neonates
- Risk: None (precursor to retinoschisis)
- Differential: Retinal edema, white without pressure
- Management: None

Cystoid Degeneration

- Appearance: Grayish/white retina with small red spots (cysts) located adjacent to ora serrata
- Histology: Thickened retina in which tissue loss has created cystic spaces
- Occurrence: Typical form - all individuals over 8 yrs. of age, seen in autopsy eyes of neonates
- Risk: None (precursor to retinoschisis)
- Differential: Retinal edema, white without pressure
- Management: None
1. Lattice degeneration
2. Cystoid degeneration
3. Cystic retinal tuft
4. Pavingstone degeneration
5. Oral pearl
Pearls of the Ora Serrata
- Appearance: small glistening spheres located at or near a dentate process
- Usually white, can be brown or grey occasionally detach and float in vitreous
- Histology: Drusen-like structure adherent to basal lamina (may have pigment covering)
- Occurrence: 20% of eyes; inferior, nasal
- Risk: None
- Differential: R/O operculum- investigate all floaters- can mimic appearance of operculum
- Management: None

What is the name of this lesion photographed in the speaker's eye?
1. Laser scar
2. Coat’s disease
3. Pavingstone
4. Unspecified STD
5. Who cares?
Peripheral Chorioretinal Atrophy (Pavingstone Degeneration)

- Appearance: Yellow/white lesions 1-1.5 mm diameter, usually inferior fundus near ora
  - May be coalescent, in linear clusters that parallel the ora serrata
- Histology: Focal areas of RPE & receptor loss
  - Correspond to:
    - Areas of decreased perfusion in choriocapillaris
    - Pigment migration to margins of lesions

Occurrence: 27% of adult autopsy eyes, bilateral 38% of cases
- Increases w/ age, high myopes
- Most common in the inferior quadrants
- Risk: In retinal detachment, retina may tear at borders of pavingstone lesions
- Differential: Chorioretinal scars
- Mgt: None except in retinal detachment
In case you are wondering, it does look bad…..

Degenerated glial tissue
Vitreous traction invariably present

Appearance- appear as grey-white dots close to ora
Histology- composed of altered retinal tissue, degenerated glial tissue
Base diameter usually less than .1 mm
Occurrence- 72% of adults and bilateral in 50% of cases; vast majority are intrabasal
Risk- low to none - small size, intrabasal location
Differential- none
Management- none; rarely, small benign holes are noted at their base
Appearance- flat, round pigmented lesions, usually black or dark grey, distinct borders
Size- varies from small to several disc diameters
May have a surrounding area of decreased pigmentation (halo).
Histology- congenital hypertrophy of RPE cells- have larger than normal melanin granules
Occurrence- unknown; unilateral in 85% of cases
Risk- none
Differential- nevus, melanoma
RPE Hyperplasia & Hypertrophy

A. hyperplasia
B. hypertrophy
C. hypertrophy w/ halo of depigmentation

Retinal Pigment Epithelium Hypertrophy

Congenital RPE Hypertrophy
Retinal Pigment Epithelium Hyperplasia
- Appearance- flat, round pigmented lesions, usually black or dark grey, distinct borders
- Size- small to several disc diameters.
- Histology- acquired (hyperplasia) increased number of RPE cells
- May occur at site of vitreoretinal traction
- Occurrence
- Risk- if tractional, may cause break
- Differential- nevus, melanoma, assess for vitreoretinal traction

RPE Hyperplasia Associated w/ Traction vs. Congenital RPE Hypertrophy

Congenital RPE hypertrophy Multi-lesion
- Associated with Gardner Syndrome
- Familial colorectal polyposis
  - Autosomal dominant disease characterized by GI polyps, pigmented cutaneous, ocular lesions
  - Adenomatous polyps often progress to cancer
  - Onset in teens, often requires colonectomy
- Shields et al. found very little association between CHRPE & Gardner syndrome

Appearance- Whitish areas of translucent retinal tissue w/ ill-defined borders adjacent to or posterior to ora serrata.
- WWP is only seen w/ scleral indentation
- WWOP is seen without indentation.
- May surround an area of normal appearing retina giving the appearance of a retinal break
- Histology- Extended areas of abnormal vitreoretinal traction.
- Watzke reported RPE changes; disorganization, loss of retinal tissue.

Occurrence- 31% of the population, equal males & females,
- Increased in older individuals, strong association w/ lattice degeneration
- Risk- varies according to study; probably poses a minimal increase in risk for retinal tears
- Differential- retinal edema (post trauma)
- Dark without pressure can mimic retinal break, scleral indentation
- Management- monitor in patients w/ other risk factors

White Without Pressure
Where are we?

- Enclosed ora bays
- Meridional folds
- Cystic retinal tufts
- Zonular traction tufts
- Acquired peripheral retinoschisis
  - Typical
  - Reticular
- Atrophic retinal holes
- Operculated retinal breaks
- Lattice retinal degeneration

Two Breaks in WWOP

Peripheral Retinal Anomalies with Moderate Threat to Sight
Oral bay
Dentate process
Ora bay
Vitreous Base

1. Horseshoe tear
2. Retinal excavation
3. Subclinical detachment
4. Enclosed ora bay
5. Retinoschisis

Appearance- dark red or brown ovoid areas adjacent and perpendicular to ora serrata
Histology- areas of pars plana epithelium
- Lack overlying neural elements
- Partially/totally enclosed by normal sensory retina
Occurrence-
- Totally enclosed bays- 2%-5.2% of eyes, bilateral 25% of cases
- Partially enclosed bays- 0.6-5.0% of eyes
- Associated w/ meridional folds
Enclosed Ora Bays

- Risk: usually benign, retinal breaks may be noted at posterior margin of enclosed bays.
- Enclosed bays are a posterior extension of normally smooth contour of vitreous base
- May lead to a retinal tear when PVD tugs on the retina surrounding the bay

Enclosed Ora Bay

Appearance with different degrees of scleral indentation
Enclosed Ora Bays
- Differential - may mimic appearance of a retinal break; differential is scleral indentation
- Management -
  - Education
  - In the absence of retinal breaks, manage w/ yearly DFE
  - If small breaks posterior to ora bay are noted, follow more frequently.
  - In fresh PVD, scleral depress to R/O breaks, tears

Meridional Folds
- Appearance -
  - Rolls or folds of redundant white retinal tissue oriented radially and perpendicular to ora serrata
- Histology - elevated cystic retinal tissue with changes on the inner surface
  - Originate at dentate processes (82%) and ora bays (18%)
- Occurrence - 20%-40% of eyes; bilateral in 49%-55% of cases.
  - Most common superior-nasal quadrant near the horizontal meridian, more frequent in males.
  - Tend to be mirror symmetrical and associated w/ enclosed ora bays and retinal excavation.

Risk
- Temporal quadrant folds or folds on ora bays are considered pathological.
- 72% of pathological folds have retinal break at posterior end.
- Nasal folds & folds on dentate processes considered normal
- Differential - none
- Management - evaluate by scleral depression, r/o retinal break; patient education on flashes, floaters
Appearance- grey nodules of varying size, usually extrabasal (outside vitreous base)
Histology- degenerated, proliferated cystic tissue with some degree of vitreous traction 0.1 to 1 mm diam.
Occurrence- 6% of patients
Risk- varies depending on source
Byer: 10% of all retinal detachment caused by CRT
Foos: CRT cause 82% of opercula, 97% of tears
Management:
Patient education
Scleral indentation, yearly DFE
Do not underestimate the potential for harm
Appearance- Usually round, occasionally elongated red areas in peripheral retina. Often w/ cuff of subretinal fluid and subclinical detachment (30%). Opercula are almost universally seen in adjacent vitreous. Pigment surround common. Histology- Full thickness break in retina. Caused by traction on CRT, or area of abnormal vitreoretinal traction.

Operculation Retinal Holes
- Occurrence- 13.4% of retinal breaks, increased in frequency w/ age: 80% associated w/ PVD. Most common location extrabasal. Large breaks associated w/ lattice degeneration & PVD are rare. Reportedly associated w/ WWP & WWOP. Risk- Slight, increases w/ size. Differential- Atrophic hole, retinal hemorrhage, flap tear.
  - Management-
    - If old, asymptomatic, patient education, yearly DFE.
    - If fresh, or symptomatic, patient education, 6 week DFE.
    - If subclinical detachment > 2 dd refer.
Pigment is our friend!

Where are we?
**Zonular Traction Tufts**

- **Appearance:** Thin, elongated projections of grey/white retinal tissue extending from their base toward ora serrata. Often have RPE hyperplasia at base.
- **Histology:** Developing misdirected zonules attach to peripheral retina and lead to formation of ZTT.
  - Base shows cystic changes, high magnification shows zonule attached to tip of tuft.
  - Usually intrabasal, nasal (79%), and inferior (61%).
- **Occurrence:** 5% of population; bilateral in 15% of cases.
- **Risk:** Due to intrabasal location & small size, risk is minimal.
  - In aphakes, more of a concern; small holes nasal periphery most common cause of RD in aphakes.
  - PVD poses far less risk to patients w/ ZTT than those w/ CRT.
- **Differential:** Scleral indentation.
- **Management:** Yearly DFE unless retinal break; refer aphakes w/ nasal breaks.

In aphakes, more of a concern; small holes nasal periphery most common cause of RD in aphakes.
**Acquired Peripheral Retinoschisis**
- Caused by splitting of sensory retina into inner & outer layers
- Always associated with cystoid degeneration; advanced stage of same process
- Occurs in two forms, typical and reticular
- Found in 4% of general population & 7% of population over 40 years of age
- Round or ovoid in shape
- Bullous lesions more prone to progression or detachment

**Typical Peripheral Retinoschisis**
- Appearance- Round or ovoid shape with inner & outer layers separated by fluid.
  - Details of choroidal vasculature are obscured
  - White dots on inner surface (70%) of lesions.
  - Inner layer smooth, has white dots on inner surface (70% of lesions)
  - Outer layer has moth-eaten, pockmarked appearance. Inner or outer layer holes occasionally seen
- Visual field- causes absolute scotoma

**Typical Retinoschisis**

![Image of retinoschisis](image-url)
Histology: Splitting of neurosensory retina in region of outer plexiform layer.
- Inner layer is relatively thick & smooth in appearance. Supporting elements or pillars between layers at margins of lesions.

Occurrence:
- Found in .69% of eyes,
- Bilateral in 33%-64% of cases, most common in inferior temporal quadrant (70%-82%),
- Some studies indicate more prevalent in hyperopes.
Risk: Minimal; typical retinoschisis is less progressive than reticular retinoschisis. Outer layer holes infrequent.

Diff. Retinal detachment, melanoma

Visual field defect is absolute w/ sharp borders.
No folds, w/ scleral indentation distance between inner & outer layers is maintained
Tends to be less bullous than reticular form and lacks lines on inner layer surface

Mgt. Education
Yearly DFE, fundus photos, drawings

Appearance- Round or ovoid shape w/ inner & outer layers separated by fluid.
Details of choroidal vasculature are obscured; inner layer thinner than in typical form.
White dots and an arborizing pattern of white lines in inner layer
Outer layer has moth-eaten, pockmarked appearance.
Inner or outer layer holes more common.

Histology- Splitting of neurosensory retina in region of internal limiting membrane
Inner layer is relatively thin & smooth in appearance.
Peripheral Retinoschisis Reticular

- **Occurrence**
  - 0.95% of eyes, bilateral in 15.8% of cases:
    - found in 1.62% of patients.
  - Most common in inferior temporal quadrant;
  - Outer layer holes in 22.7% of eyes.
- **Risk**
  - More prone to progress past equator than typical form
  - More prone to cause retinal detachment, especially when outer layer holes are present
  - Greatest risk is when outer and inner layer holes are present.

Typical Retinoschisis

Peripheral Retinoschisis Reticular
**Peripheral Retinoschisis Reticular**
- Differential: Retinal detachment, melanoma
- In retinoschisis visual field defect is absolute w/ sharp borders,
- With scleral indentation distance between inner & outer layers is maintained.
- Reticular retinoschisis tends to be more bullous than typical form and has lines on inner layer surface.
- Presence of demarcation lines often heralds an underlying detachment

**Peripheral Retinoschisis Reticular**
- Management: yearly DFE if no outer layer holes noted, fundus photos, drawings
- Watch for development of inner & outer layer breaks, progression
- Scleral depression, give special attention to reticular lesions w/ outer layer breaks or those w. pigment demarcation lines.
- Refer if underlying detachment or if lesion encroaches on the macula

**B-Scan Retinoschisis**

*Courtesy Larry Alexander, OD*
Appearance - small (<.5 dd) round red areas in peripheral retina, often w/ surrounding cuff of fluid and subclinical detachment

Histology - full thickness break in retina.

Occurrence - 76% of all retinal breaks, occur in 2.4% of eyes, 4% of cases, 76% found w/in lattice lesions, associated w/ meridional folds. ZTT, CRT; increased in myopes, 80% have associated subclinical detachment
Atrophic Peripheral Retinal Holes

- Risk: minimal; 7% lead to clinical detachment
- Differential: tractional tear, retinal hemorrhage; scleral depression
- Management: document, yearly DFE, refer if clinical detachment > 2 dd noted
Lattice Retinal Degeneration

- Appearance
  - Varies widely; usually parallels the ora
  - White lines- 12%-15%
  - Atrophic holes- 18%-43%
  - Pigment- 83%
  - White spots- 80%
- Histology- thinned area of peripheral retina with overlying lacunae of liquified vitreous, vitreous traction on margins, especially posterior

The Perfect Storm

- To start a fire you need
  - A spark
  - Oxygen
  - Fuel
- To start a retinal detachment you need
  - Liquified vitreous
  - A break in retina
  - Access under RPE
**Lattice Retinal Degeneration**

- **Occurrence:** 8%-11% of individuals
- **Increased in myopic eyes**
- **Most common @ 12 o’clock & 6 o’clock**
- **Risk:**
  - 0.5% chance for detachment
  - 30% of all detachments associated with lattice
  - Greatest risk is large horseshoe tears

**The Many Faces of Lattice Retinal Degeneration**
Lattice Retinal Degeneration
- Occurrence: 8%-11% of individuals
  - Increased in myopic eyes
  - Most common at 12 o’clock & 6 o’clock
- Risk
  - 0.5% chance for detachment
  - 30% of all detachments associated with lattice
  - Greatest risk is large horseshoe tears

Peripheral Retinal Anomalies with Significant Threat to Sight
- Horseshoe tears
- Retinal dialysis
- Giant Retinal tears

Horseshoe or Flap Tears
- Appearance: red, U-shaped lesion with "horse running toward the posterior pole"
- Cystic retinal tuft may be seen at end of flap
Horseshoe or Flap Tears
- Histology- full thickness retinal break w/ attached flap of retina having traction from vitreous on apex of the flap
- Occurrence- 10% of all retinal breaks
- Frequently symptomatic due to traction. May be found at posterior border of
  - Lattice
  - Chorioretinal scars
  - CRT
  - Enclosed ora bays
- 81% occur in individuals over 40 years of age

Horseshoe or Flap Tears
- Risk - moderate to high because of the continuing presence of traction.
- Differential- atrophic hole, operculated tear, hemorrhage
- Management- Small (< 1/4 dd) and asymptomatic: patient education & 4 week DFE,
  Larger (> 1/4 dd) or symptomatic, especially if superior, or Hx detachment fellow eye: refer

Which is better, one or two
Appearance: large area of detached retina adjacent to ora serrata

Histology: Retinal separation at or near ora serrata

Occurrence: Most common superior-nasal quadrant.
- Over half are idiopathic.
- Remainder are secondary to trauma
- Development of detachment is usually slow.
- Average time from onset till RD occurs is 14 months

Horseshoe or Flap Tears

Retinal Dialysis
Risk factors
- Trauma: Retinal dialyses
  - Account for @y 75% of retinal breaks after trauma
  - Present in up to 85% of traumatic RD detachment
- Risk - very high
- Management - refer to retinologist;
- When detachment occurs, typical draining and buckle are indicated.

Retinal Dialysis

Retinal Dialysis B-scan

History
- Football injury (head shot)
- Unconscious for one week
- Remembers nothing
- Mom insisted he have eye exam
- Six months ago.....
- Symptoms none
- VA: 20/20 OU
- Confrontation, pupils NL
Patient Management & Education

- Annual DFE, fundus photos, drawings
- Giant retinal tears- similar to retinal dialysis, but extend greater than one quadrant.
  - Treatment depends on extent of retinal detachment.
  - Draining and scleral buckle
  - Cryo-treatment
- Retinal cysts- fluid filled, usually self-limiting and self-resolving over time
  - Chorioretinal pathology

Rhegmatogenous Retinal Detachment

- App. Elevated, usually bullous lesions w/ undulating surface.
  - Recent detachments are clear, but old detachments have white, fixed folds.
  - Demarcation lines may be seen anterior to the posterior-most point on the detachment.
  - Loss of underlying choroidal details may be only sign in fresh, shallow detachments

Rhegmatogenous Retinal Detachment

- Histology- Separation of inner layers of sensory retina from RPE.
  - Liquified vitreous fills the cavity formed by the detachment unless it is exudative.
  - Demarcation lines represent futile attempts by the eye to limit the detachment by RPE hyperplasia.
- Occurrence- 8.9 per 100,000 population.
- Highest risk is in myopes > 4.00 D, individuals over 50 year of age, and those w/ a RD in the fellow
Risk - Any patient w/ a fresh RD should be referred to a retinal specialist immediately.

Macula-on detachments require immediate retinal consult, since VA loss increases once macula is detached

Diff. Retinoschisis, choroidal detachment, exudative detachment,

Diagnosis is by scleral indentation

Management - Refer if fresh; if old detachment, carefully examine contralateral eye for breaks, tears,

Inform of increased likelihood (15%) for detachment in fellow eye
The relationship between retina and vitreous is vital in the genesis of retinal breaks & detachment.

Vitreous degeneration is a normal aging process that accelerates with specific conditions.

Clinicians should understand this relationship and be aware of common retinal degenerations.

Evaluate for other recently discovered complications of vitreous degeneration.
PREPARING THE DRY EYE FOR CONTACT LENSES

WILLIAM D. TOWNSEND, O.D., F.A.A.O.
ADVANCED EYE CARE, CANYON, TX
ADJUNCT PROFESSOR, UHCO

DISCLOSURES: MY LECTURES AND RESEARCH HAVE BEEN SUPPORTED BY THE FOLLOWING

- ALCON
- TEARLAB
- VALEANT (LACRISERT)
- ODYSSEY MEDICAL
- SCIENCE BASED HEALTH
- COOPERVISION
- TEAR SCIENCE
- MANY WINERIES
MOLINA K, GRAHAM AD, YEH T, ET AL. NOT ALL DRY EYE IN CONTACT LENS WEAR IS CONTACT LENS-INDUCED. EYE CONTACT LENS. 2019 SEP 10.

- Ninety-two subjects completed the.....
  - Berkeley Dry Eye Flow Chart without lenses
  - Ocular Surface Examinations
  - A battery of questionnaires.
- After evaluation of findings subjects were assigned to one of three study groups:
  - Asymptomatic contact lens wearers 37 subjects (40%) (ASYM)
  - Symptomatic CL wearers who become asymptomatic on lens removal (33%) (CLIDE)
  - Symptomatic CL wearers did not resolve on lens removal 5 (27%) underlying Physiologic DE.
- Conclusion: Many CL wearers w/ symptomatic dryness have underlying DED and will not respond to treatments aimed at changing lenses or solutions!

Over 25% of CL patients had symptomatic dry eye that had nothing to do with the fact that they were CL wearers!

IMAGINE..

It is 3:33 PM, and your staff just finished working up a new patient who wants to wear contact lenses. A quick review of the chart informs you that this thirty-seventy year old female has issues with ocular discomfort, especially later in the day. She is taking a diuretic and an antidepressant daily. Biomicroscopy reveals a very reduced tear film in addition to significant loss of meibomian glands. What is your next move?

1. Tell her there is no way she will ever be able to wear contacts.
2. Distract her by describing a new line of frames you’re sure she will love.
3. Refer her for refractive surgery.
4. Explain that treating her dry eye can improve her chances of successful contact lens wear.
MONUMENTAL STUDIES IN UNDERSTANDING DED

1995 - LEMP MA - REPORT OF THE NATIONAL EYE INSTITUTE/INDUSTRY WORKSHOP ON CLINICAL TRIALS IN DRY EYE

- Defined DED as "a complex multifaceted group of medical & ocular diseases due to" decreased tear production" and/or increased tear evaporation"

- First study that made many clinicians aware of multiple causes of dry eye and large number of affected individuals
TEAR FILM & OCULAR SURFACE SOCIETY (TFOS)

• The TFOS Mission
  • Advance research, literacy, and educational aspects of the scientific field of the tear film and ocular surface
  • TFOS DEWS I was established to achieve a global consensus: determine basic underlying pathology or pathologies

• The TFOS Goal
  • Achieve a global consensus concerning multiple aspects of DED. TFOS DEWS II sought to
    • Update the definition and classification of DED;
    • Evaluate critically the epidemiology, pathophysiology, mechanism, and impact of this disorder;
    • Develop recommendations for the diagnosis, management and therapy of this disease;
    • Recommend the design of clinical Aqueous Tear Deficiency (ATD)
  • Lipid anomaly dry eye (LADE) i.e. evaporative dry eye
  • Lipid surface/blinking anomalies dry eye (LSADE)

2007- FOULKS GN ET AL- INTERNATIONAL DRY EYE WORKSHOP (DEWS)

• Multifactorial disease of the tears and ocular surface

• Symptoms & Signs
  • Discomfort
  • Visual disturbance
  • Tear film instability
  • Potential damage to the ocular surface
  • Increased tear film osmolarity
  • Inflammation
2011-Nichols KK ET AL. INTERNATIONAL WORKSHOP ON MEIBOMIAN GLAND DYSFUNCTION

- Proposed Explanation of the Pathophysiology of MGD
- Outlined Details about Composition of MG Secretions
- Classified MGD Based on MG - Low Delivery vs. High Delivery
- Diagnostic Workup - Use of Standard Dry Eye Tests & Gland Expression - Expressibility, Meibum Quality
- Proposed a Staging System of MGD
- Proposed a Treatment Plan Based on the Disease Stage
- Concluded that MGD is the Leading Cause of DED
2017 TFOS DEWS II REPORT EXECUTIVE SUMMARY
CRAIG JP, NELSON JD, AZAR DT, ET AL

“Dry eye is a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles.”

2017 TFOS DEWS II REPORT EXECUTIVE SUMMARY
CRAIG JP, NELSON JD, AZAR DT, ET AL

“Dry eye is a multifactorial disease of the ocular surface”

• Loss of homeostasis of the tear film
• Ocular symptoms
• Tear film instability
• Hyperosmolarity
• Inflammation and damage
• Neurosensory abnormalities
2017 TFOS DEWS II REPORT EXECUTIVE SUMMARY
CRAIG JP, NELSON JD, AZAR DT, ET AL

Dry Eye Disease

Aqueous deficient
Mixed
Evaporative

Management to restore homeostasis

Dry Eye Workup

HISTORY

AQUEOUS DEFICIENCY
- Tear Meniscus
- Slit Lamp
- OCT
- Schirmer Test
- Phenol Red Test

COMBINED ETIOLOGY
- SCREENING TOOLS
  - Ocular Surface Disease Index (OSDI)
  - Standard Patient Evaluation of Eye Dryness (SPEED)
  - System Dry Eye Questionnaire (DEQ-5)
- VITAL DYES
  - Sodium fluorescein + NaFl,
    - @ 2% concentration = non-fluorescent
    - @ 0.1% concentration = highly fluorescent
  - Rose Bengal: toxic, and causes discomfort
  - Lissamine green: LG non-toxic, comfortable
- MMP-9

EVAPORATIVE
- Meibomian Gland Evaluation
  - % MG Present
  - Expression % MGYS Yielding Lipid Secretions
- IMAGING
  - Sl Photograph
  - Lipview
  - Infra-red Imaging
OCULAR SURFACE DISEASE INDEX (OSDI)

DEQ 5

1. Questions about EYE DISCOMFORT:
   a. During a typical day in the past month, how often did your eyes feel discomfort?
      - 0 = Never
      - 1 = Rarely
      - 2 = Sometimes
      - 3 = Frequently
      - 4 = Constantly
   b. When your eyes felt discomfort, how intense was this feeling of discomfort at the end of the day, within two hours of going to bed?
      - 0 = Never
      - 1 = Not at all intense
      - 2 = A little intense
      - 3 = Intense
      - 4 = Very intense

2. Questions about EYE DRYNESS:
   a. During a typical day in the past month, how often did your eyes feel dry?
      - 0 = Never
      - 1 = Rarely
      - 2 = Sometimes
      - 3 = Frequently
      - 4 = Constantly
   b. When your eyes felt dry, how intense was this feeling of dryness at the end of the day, within two hours of going to bed?
      - 0 = Never
      - 1 = Not at all intense
      - 2 = A little intense
      - 3 = Intense
      - 4 = Very intense

3. Questions about WATERY EYES:
   a. During a typical day in the past month, how often did your eyes leak or feel excessively watery?
      - 0 = Never
      - 1 = Rarely
      - 2 = Sometimes
      - 3 = Frequently
      - 4 = Constantly

Scores: 1a + 1b + 2a + 2b + 3m + Total
HISTORY

DRY EYE WORKUP

AQUEOUS DEFICIENCY
• Tear Meniscus
• Slit Lamp
• OCT
• Schirmer Test
• Phenol Red Test

COMBINED ETIOLOGY
• SCREENING TOOLS
  • Ocular Surface Disease Index (OSDI)
  • Standard Patient Evaluation of Eye Dryness (SPEED)
  • 5-Item Dry Eye Questionnaire (DEQ-5)

VITAL DYES
• Sodium Fluorescein• Nat.,
  • @ 2% Concentration• NON-fluorescent
  • @ 0.1% Concentration• HIGHLY fluorescent
• Rose Bengal• TOXIC, and causes discomfort
• Lissamine Green• LG NON-toxic, COMFORTABLE
• MMP-9 (Marker for inflammation)

EVPORATIVE
• MEBOMIAN GLAND EVALUATION
  • % MG Present
  • Expression [% MG]/LS YIELDING LIPID SECRETIONS

IMAGING
• SL Photograph
• LipView
• Infra-red Imaging
EVALUATING FOR DRY EYE

AQUEOUS DEFICIENCY

• Tear meniscus
  • Slit lamp
  • OCT
• Schirmer test
• Phenol red test

EVAPORATIVE

• Meibomian gland evaluation
  • % MG present
  • Expression (% MGYS yielding lipid secretions)
• Imaging
  • SL photograph
  • UPV
  • Infra-red imaging

WHY DO CONTACT LENS PATIENTS DO POORLY?

• Two primary reasons people drop out of their lenses are discomfort and dryness (Richdale et al., 2007)
• Patients over the age of 45 account for only 30% of all contact lens wearers (Nichols, 2015)
• 87% of all dry eye is partly or totally caused by meibomian gland dysfunction (DEWS Report 2011)
Managing Ocular Pain and Inflammation
William D. Townsend, O.D., F.A.A.O.
Advanced Eye Care    Canyon, Texas
Adjunct Professor, UHCO

Friday, 4:30 PM

When pain strikes......
Ultimately, patients may remember how well you managed or mismanaged their pain more than how you managed their disease.

You have a 12 hour window to be a hero or a heel. That is what this presentation is all about.
How Pain Happens - Reflex
- Nociceptors – (pain receptors) stimulated by chemical, thermal, or mechanical forces
- Signals rapidly course through the dorsal horn of the spine and cause a reflex response

How Pain Happens - Sensation
- Signals reach thalamus via the spinal-thalamic tract
- Pain perception occurs
- Thalamus signals the presence of pain to other parts of the body

Physical & Physiological Consequences of Pain
- Tachypnea
- Tachycardia
- Systemic hypertension
- Emotional distress
- Poor sleep
- Increased risk for complications in patients w/ cardiovascular disease
**Pain**

“...an unpleasant sensory and emotional experience arising from actual or potential tissue damage

**Inflammation**

“...the process by which white blood cells and fluid accumulate within a tissue, triggered by various stimuli (trauma, infection, or immunity)”

**Inflammation ≠ Pain**

**Why ODs Need To Manage Pain and Inflammation**
Common Misconceptions About Pain: Patients & Doctors

- Pain is to be expected
- I have no control over my own pain
- I should not ask for pain relief until I am desperate
  - All people experience pain the same way and to the same degree
  - Pain can only be managed with medications (narcotic analgesics)

General Principles Of Pain Management

- It is easier to prevent pain from escalating than to reduce existing pain levels
- Avoid treating pain without knowing or at least trying to determine its cause
- Obtain a careful, in-depth health & medications history
- Use safest, most efficacious analgesic
- Consider potential complications of proposed TX

How much does it hurt?

[Image of pain scale]
Case 1

Hx: A 43 year old female presents with a history of having struck herself in the right eye with a lampshade.
CC: moderate pain & photophobia, blurred vision, and lid edema.
General health HX: unremarkable
She takes no systemic medications
She has no medication allergies.

Case 1

- VA = OD 20/25  OS 20/20
- SLE:
  - OD: irregular shaped abrasion superior cornea
  - Gr. II+ injection
  - No flare or cells
  - Gr. II+ lid edema
  - OS:  NL
  - Pupils  NL
11/19/2019

Case 1
Assessment:
- Corneasal abrasion 2nd to trauma

Plan
- Pressure patch with:
  - 2 gtt Voltaren
  - 1 gtt tropicamide 1%
  - 1 gtt ofloxacin
- 200 mg Ibuprofen PO q 4 hours
- RTC: 1 day

Immediately after stain

5 minutes after stain
Case 1
Day 2: after removal of patch in office

- Subjective: “Shooting pains in OD, eye feels feverish, swollen"
- VA = OD 20/30  OS 20/20
- SLE:
  - Corneal abrasion 95% re-epithelialization- stains under the epithelium with Na FL
  - A/C: trace cells, flare, decreased injection
- Assessment: The eye looks better, but the patient is VERY unhappy
- What went wrong in our management of this case?

Corneal Abrasion: A Model for Pain & Inflammation Management

What went wrong?
- Cornea erosion ⇒ release of inflammatory mediators, decreased pain threshold
- Conjunctiva ⇒ vasodilation and increased vascular permeability causes injection, increased extracellular fluid, diapedesis
- Anterior chamber ⇒ increased vascular permeability, A/C activity with cells/flare
- Lids ⇒ release of inflammatory mediators causes lid tenderness and increased edema
- An unhappy patient
What Went Wrong On Day 2?

Corneal Abrasion: A Model for Understanding Pain & Inflammation

- Release of bradykinin
- Trauma
- Vasopermeability, Miosis
- Chemotaxis
- Vasodilation & Inflammation, IOP changes
- Pain out of proportion to injury

PMN migration

Phospholipase A

Prostaglandins

Leukotrienes

Bradykinin

Release of cell membrane phospholipids

Reduced pain threshold

Analgesics And Anti-inflammatory Agents For The Practicing Optometrist
Corneal Abrasion: A Model For Ocular Trauma

**OPPORTUNITIES FOR INTERVENTION!**

1. **Release of bradykinin**
2. **Reduced pain threshold**
3. **Release of cell membrane phospholipids**
4. **Conversion by:**
   - **Phospholipase A**
5. **Arachidonic acid**
6. **Prostaglandins**
7. **Leukotrienes**
8. **Cyclooxygenase**
9. **Lipoxygenase**

**Side Effects?**

- **PMN migration**
- **Release of bradykinin**
- **Reduced pain threshold**
- **Increased pain sensation**

**Side Effects?**

- **NSAIDS, Narcotics**
- **Increased vasopermeability, Miosis, Chemotaxis**
- **Vasodilation, IOP changes, inflammation**
- **PMN migration**

**Case 1 - Day 2**

- **New Plan**
  - **Debride loose corneal tissue to margins**
  - **Kimura**
  - **Voltaren gts**
  - **Better than pressure patch**
  - **Homatropine 5.0%**
  - **Much better than tropicamide**
  - **Ofloxacin**
  - **Today I might use moxifloxacin**
  - **RTC 1 day**

- **Long-term plan**
  - **Remove bandage CL**
  - **Continue Ocuflox Q 6 hours**
  - **Muro 128 ung hs x 60 days**
  - **Advise possibility of recurrent erosion**

**Corneal Erosion**

- **Etiology:** Painful separation of epithelium from basement membrane
  - Often secondary to trauma or epithelial BM dystrophy
- **Anatomy**
  - Epithelium bonded to underlying BM by hemidesmosomes and stroma by intermediate filaments
  - Hemidesmosomes anchored to stroma by anchoring filaments and anchoring plaques
- **Ripping or shearing injuries**
  - Damage ultrastructural connection between epithelium and underlying tissue
  - Common sequela in finger nail injuries and paper cuts
Corneal Erosion: A Bad Model For Ocular Pain Management

- 30 day rule: hemidesmosomes
- 60 day rule: anchoring filaments

Recurrent Corneal Erosion

- Once damage from trauma or dystrophy occurs
  - Epithelium becomes less firmly attached
  - May adhere to tarsal conjunctiva during sleep
  - Tissue may be torn from basement membrane during REM or upon awakening
  - Physiologic edema (nocturnal) contributes to development of recurrent erosions

Corneal Abrasion: A Model for Pain & Inflammation Management

What I learned from this case.....

- Preemptively Rx Topical + systemic NSAIDS reduce pain & inflammation
- Remove all damaged, loose tissue
- Cycloplegia to preemptively manage inflammation
- Bandage lenses to minimize discomfort & promote healing
- Consider an amniotic membrane
Amniotic Membranes

- Amniotic membrane - portion of the placenta closest to the baby
- Advantages: quicker healing, less pain, less scarring, less inflammation
- Available in frozen and non-frozen forms
- Frozen (Prokera) contained in plastic ring
- AmbioDisc (IOP Ophthalmics) is

Case X Day 1

- 67 y/o female presents w/ fb sensation & reduced VA OD
- Hx- DED, bilateral CE w/ IOL
- BCVA OD 20/40 OS 20/40
- SLE: SPK in dendritic pattern
- Corneal sensation: dental floss
  - OD –
  - OS +++
- Initiate antiviral therapy

Case X
Case Day 10
- BC VA = 20/40 OD 20/30 OS
- Patient denies discomfort
- Current Tx- non-preserved AT
- Non-healing epithelial defect
- Decision to insert amniotic membrane

Case X- Day 10

Case X- Day 15
Case X- Day 15
1 Day Post-removal of Prokera

Corneal Abrasion:
Considerations in Management

- Does this condition present with significant risk for....?
  - Recurrent erosion
  - Secondary infection
  - Uveitis
- What is the patient’s degree of pain
  - Presenting pain level may not correlate with severity of pain a few hours later
  - How will the patient feel in 3 hours?
  - How will the patient feel in 8 hours?

Corneal Abrasion
Management

Debride all loose epithelial tissue
Bandage CL in Corneal Trauma

- Currently approved bandage CL
  - Night & Day (lotrafilcon A, Alcon)
  - Purevision (balafilcon A, Bausch & Lomb)
  - Acuvue Oasys (Senofilcon A, Vistakon)
- Factors to consider:
  - How dry is the eye?
  - How long will this lens need to be in place?
  - Prescribing meds with the CL in place?
- Bill for it: 92071 with modifier

FDA Approved Bandage CLs

<table>
<thead>
<tr>
<th>Lens</th>
<th>Manufacturer</th>
<th>Dk</th>
<th>Base Curve</th>
<th>Diameter</th>
<th>Powers</th>
<th>Max wear time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air Optix</td>
<td>Alcon</td>
<td>140</td>
<td>8.4, 8.6</td>
<td>13.0</td>
<td>+6.00 to -10.00 D</td>
<td>30 days</td>
</tr>
<tr>
<td>Night and Day Aqua</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PureVision</td>
<td>Johnson &amp; Johnson</td>
<td>91</td>
<td>8.3, 8.6</td>
<td>14.0</td>
<td>+6.00 to -12.00 D</td>
<td>30 days</td>
</tr>
<tr>
<td>Acuvue Oasys</td>
<td>Vistakon</td>
<td>103</td>
<td>8.4, 8.8</td>
<td>14.0</td>
<td>+8.00 to -12.00 D</td>
<td>7 days</td>
</tr>
</tbody>
</table>

Contraindications for Bandage CL

- Active infection
- Poor compliance
- Poor access to follow-up care
- Poor CL hygiene
- Injury circumstances
- Vegetation involved
- Contaminants
Bandage Contact Lenses and Antibiotics

- Avoid BAK-preserved preparations
- Consider 4th generation fluoroquinolone
- My personal preference
  - Vigamox- (moxifloxacin) no BAK, efficacious
  - Cycloplegia pre-contact lens first 24 hrs.
- Daily follow-up until patient has re-epithelialized
- Stain with NaFl while lens is in situ

Corneal Abrasion Management: General Principles

- Larger lesions
  - Be proactive in preventing recurrent erosion
  - Use hyperosmotic agent @ HS for a minimum of 60 days
- Avoid aminoglycosides due to toxicity
- Use FDA approved CL rewetting drops
- Non-preserved unit dose artificial tears or Addipak (off label) in patients w/ dry eye

Corneal Abrasion

- Oral NSAID (OTC dosing usually adequate)
  - Ibuprofen gel caps 2 x 200 mg q 4 hr
  - More rapid onset of analgesia
  - Naprosyn sodium gel caps 220-440 mg q 12 hr
- Narcotic and narcotic like analgesics
  - Hydrocodone 5.0 or 7.5 mg w/ APAP or ibuprofen Q 4 hr
  - Tramadol (Ultram) 0.25%, 0.5%
  - Ask patient if he or she is a non-responder
Think Narcotic Analgesics are Foolproof?
- Evaluated 186 patients with moderate to severe pain; initially prescribed morphine.
- 74% responded well to morphine
- 26% did not achieve adequate relief on morphine:
  - Switched to another form of pain relief


Complications of Systemic Narcotic Analgesics- OIC
- Opioid-induced constipation OIC
  - Most common adverse effect of long-term opioid management of pain.
  - May occur within days of initial dose
  - Does not develop tolerance
- Preemptive management
  - Drink lots of water, and be active
  - Stool softener- Docusate
  - Osmotic laxative- Lactulose

Psychology of Pain Management: Suggestions and Placebos
Is this stuff all in your head?
- 35% of patients w/ severe pain reported significant relief after taking a placebo. Only 40%
- 75% of patients w/ severe pain reported significant relief after being given morphine.
- Placebo responses were
  - More evident in cases of severe pain than in cases of mild pain
  - Highest in patients experiencing high stress or anxiety
Narcotic Analgesics
Pharmacology
- Narcotic agonists bind to mu and kappa opioid receptors in dorsal root of spinal cord and produce analgesia
- NO anti-inflammatory activity!!!!!

Narcotic Analgesics
Pharmacology
- Narcotics appear to affect sensation (pain) and emotional impact (suffering)
- Most do not possess a "ceiling effect"
- With extended use, tolerance develops (tachyphylaxis)
- First-pass metabolism through liver requires larger oral doses than parenteral doses

Narcotic Analgesics
Side Effects
- Respiratory system
  - Depression, arrest, asthma exacerbation
- Gastrointestinal system
  - Constipation, emesis, nausea, dyspepsia
- CNS
  - Sedation, dizziness, somnolence, euphoria, addiction, impaired judgment
- Urinary
  - Retention
NSAIDS:
Non-steroidal anti-inflammatory drugs
- Discovered 1829 by Leroux (salicin, a precursor of salicyclic acid)
- Do not cause most of the undesirable side effects of corticosteroids, narcotics
- Block conversion of arachidonic acid to prostaglandins
- Systemic NSAIDS act centrally & peripherally to reduce pain, fever, inflammation

Acetylsalicylic Acid (Aspirin, ASA)
- Used for over 100 years
- Mechanism discovered in 1970’s
- Mild to moderate pain
- Available as tablets, enteric coated tablets, suppositories
- Adult dosage: 325-650 mg q 4h
- Always take with food
- Inexpensive, readily available
- Ceiling effect
- GI complications long term use

Acetylsalicylic Acid (Aspirin, ASA)

Side Effects
- Gastrointestinal disturbances
- Hemorrhage—a single therapeutic dose of aspirin inhibits platelet function for up to 8 days
- Reye’s syndrome
- Hypersensitivity
- Drug interactions – especially “blood thinners” Warfrin, Plavix, Xarelto
Non-Acetylsalicylic NSAIDs

- Used to manage mild to moderate pain
- Analgesia: compare well with many narcotic analgesics in relieving pain
- Anti-inflammatory agents- work peripherally and centrally
- CNS effect reduces recognition of pain
- Antipyrexic- (lower body temperature)
- Ceiling effect- dosing above given level does not further increase pain relief

Ibuprofen

- OTC in 200m tablets, capsules, gel caps
- Rx 400mg, 600mg, 800mg
- Gel caps most rapid relief of pain and inflammation
- Acts centrally and peripherally to reduce:
  - Sensation of pain
  - Inflammation
- Requires Q 4hr dosing
- Should be taken with food
- Pediatric syrup available (OK for adults)
- Ceiling effect!

Ceiling Effect Ibuprofen- Analgesia
Oral Ibuprofen vs. Tylenol No. 4

Ibuprofen 400 mg was significantly more effective than acetaminophen 300 mg with codeine 60 mg for every analgesic measure (P < .05).

In most instances you do not have to prescribe narcotic analgesics to manage pain well.

Diflunisal (Dolobid)
- Difluorophenyl derivative of salicylic acid
- Not metabolized to salicylate, so reduced toxic effects
- 3-4 x more potent than ASA as an analgesic and anti-inflammatory agent
- Not very good antipyrexic
- Fewer gastric, platelet effects than ASA
- For severe pain, 1000 mg loading dose, then 500 mg Q 12 hours
- Adult dosage: one 500 mg tablet BID or, in more severe cases of pain, TID
**NSAIDs**

**Contraindications**
- Active GI disease
- Asthma, nasal polyps, ASA sensitivity
- Bleeding disorders, anti-coagulant Tx
- Post surgical cases
- Lactating females
- Pregnancy (especially third trimester)

**NSAIDs**

**Side Effects**
- Adverse GI effects (up to 16% of patients)
  - Epigastric pain
  - Nausea, gastric and duodenal ulcer after long-term therapy
  - GI hemorrhage
  - Flatulence
- GI effects primarily due to inhibition of prostaglandin synthesis
- Reduced w/ COX 2 inhibitors

**Acetaminophen (APAP)**

**Pharmacology**
- Antipyresis through action on heat-regulating center in hypothalamus
- NO ANTI-INFLAMMATORY EFFECTS
- Exerts actions in CNS only
- No GI complications if taken short term
  - Long-term use may increase risk for ulcer
- Can damage or destroy liver tissue in compromised individuals
Acetaminophen (APAP)

Indications
- When ASA or NSAIDs are contraindicated
- Allergy to ASA or NSAIDs
- Gastric ulcer or other GI conditions
- Children and adolescents (no Reye’s syndrome)
- Pregnancy and lactation
- Asthmatics

Recommended doses
- 1,000 mg per single dose and up to 4,000 mg per day for adults
- Up to 2,000 mg per day if drinking alcohol
- Foremost cause of acute liver failure in the Western world
- Chronic use at standard dosing increases risk of major CV events
- Ceiling dose: 1000 mg every 4 to 6 hours

Acetaminophen (APAP)

Side Effects
- Allergy
- Hepatic toxicity!!!!
  - ETOH abusers
  - With overdose

Contraindications
- Hepatic impairment, damage (cirrhosis)
- Chronic ETOH abuse, alcoholism
- You must inquire about ETOH, liver
Topical NSAIDS

<table>
<thead>
<tr>
<th>Medication</th>
<th>Branded name</th>
<th>Manufacturer</th>
</tr>
</thead>
<tbody>
<tr>
<td>bromfenac 0.09% solution</td>
<td>Bromday, Xibrom</td>
<td>Ista</td>
</tr>
<tr>
<td>diclofenac 0.1% solution</td>
<td>Voltaren</td>
<td>generic</td>
</tr>
<tr>
<td>flurbiprofen 0.03%</td>
<td>Ocufen</td>
<td>generic</td>
</tr>
<tr>
<td>ketorolac 0.5% solution</td>
<td>Acular</td>
<td>Allergan</td>
</tr>
<tr>
<td>ketorolac 0.5% solution nonpreserved</td>
<td>Acular LS</td>
<td>Allergan</td>
</tr>
<tr>
<td>ketorolac 0.45% solution in single use dispensers</td>
<td>Acuvail</td>
<td>Allergan</td>
</tr>
<tr>
<td>nepafenac 0.1% solution</td>
<td>Nevanac</td>
<td>Alcon</td>
</tr>
</tbody>
</table>

What about 2019

- Nevanac
  - Nepafenac 0.1% suspension
  - Potent pro-drug converted by enzymes into amfenac
  - Penetrates anterior segment and posterior segment as well
  - Works well in pre & post foreign body removal, debridement

What about 2019?

- Bromday
  - Bromfenac 0.09%
  - Potent anti-inflammatory activity with QD dosing
  - Excellent penetration
  - Useful for CME (off label use)
  - Useful for pre and post foreign body removal
Rational Drug Therapy

- Know the three A’s
  - Autonomics
    - Does the medication have autonomic SE
  - Ancestry
    - Is there a family Hx of atopy
  - Allergy
    - Is there a personal Hx of allergy
    - Is it really allergy or just an expected SE
- Topical vs. systemic medications
  - Does the condition warrant systemic
  - Use topical when it is equal to systemic

Corticosteroids

- Work at cellular level to reduce inflammation by altering or inhibiting protein synthesis
- Block conversion of arachidonic acid by phospholipase A
- Inhibit PMN migration, initiation of inflammatory response
- Inhibit secondary leukocyte migration
- Inhibit fibroblast activity, scar formation

Teddy Roosevelt Philosophy of Steroid Therapy

- “Speak softly and carry a big stick!”
- Hit the inflammatory process hard
- Use a potent steroid to reduce tissue damage
- Taper the therapy as soon as it is practical
Topical Corticosteroids

Drugs With Maximum Clinical Action

- Prednisolone acetate 1% - (acetate always stronger) avoid using generic due to irritation
- Dexamethasone 0.1% - excellent for chronic uveitis
- Rimexolone 1% - essentially equal to prednisolone acetate with less IOP elevation
- Lotoprednol 0.5% - my DOC for steroid responders
  - Slightly less efficacious than pred. acetate 1%
  - Good for chronic tx
  - May still elicit IOP elevation

Potential Complications of Systemic Corticosteroids

- Avascular necrosis of the femoral head
- Precipitation or aggravation of diabetes
- Skin thinning resulting in easy bruising
- Redistribution of body fat: moon face, buffalo hump, and truncal obesity
- Osteoporosis especially in:
  - Smokers, postmenopausal women, elderly, those who are underweight or immobile, and patients with diabetes or lung problems

Corneal Erosion
Conclusion

- 50% of all hospital patients get inadequate pain relief
- Doctors often ignore the fact that pain perception is individual
- Patients often believe pain is unavoidable
- Some patients may not ask for pain relief until they are desperate
- It is easier to prevent pain escalation than to reduce pain once it is severe

Conclusion

- Do not deprive your patients of the pain relief that they need and deserve
- Use basic science knowledge to provide the best, least expensive, and safest pain relief possible
- If you question the wisdom of prescribing a given analgesic or antiinflammatory drug, consult the patient/s PCP or a trusted pharmacist

Conclusion

- Pain management cannot be done in a cookbook fashion.
- Each case is individual: art vs. science.
Conclusion

- Realize that what you say and how you say it may have as much impact on pain-management outcome as the medications and treatments you prescribe.
- Do not force your patient to seek help from another provider: you can manage pain associated with eye trauma and inflammation.