Diagnosing and Managing Ocular Emergencies and Urgencies: OD and MD Perspective
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Eye Care and the Emergency Department

What Classifies an Emergency?
- Any condition in which the patient has the potential for:
  - vision loss,
  - currently experiencing vision loss,
  - permanent structural damage,
  - pain or discomfort,
  - or is an "emergency" for the patient.
- It is important to be able to triage a walk-in patient and, more importantly, a call-in patient.

Visual Loss
- Visual loss varies greatly in meaning from patient to patient
  - ranging from blur to complete blindness and may affect one or both eyes
- Components include:
  - acuity,
  - visual field,
  - color and brightness may be affected jointly or separately
- Detailed history and extent of vision loss crucial

Acute Vision Loss
- Loss of vision is usually considered acute if it develops within a few minutes to a couple of days.
- May affect one or both eyes and all or part of a visual field
- 3 general causes:
  - Opacification of normally transparent structures (eg, cornea, vitreous)
  - Retinal abnormalities
  - Abnormalities affecting the optic nerve or visual pathways

What questions to ask?

<table>
<thead>
<tr>
<th>Onset</th>
<th>suddenly noticed or sudden onset?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual Loss</td>
<td>any loss of vision?</td>
</tr>
<tr>
<td></td>
<td>loss vs. blurry vision</td>
</tr>
<tr>
<td></td>
<td>one eye or both</td>
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<tr>
<td></td>
<td>part of visual field or all</td>
</tr>
<tr>
<td></td>
<td>transient vs. permanent</td>
</tr>
<tr>
<td>Pain</td>
<td>is there pain? constant? scale (1-10)</td>
</tr>
<tr>
<td>Redness</td>
<td>is there any redness? location?</td>
</tr>
<tr>
<td>Associated Factors</td>
<td>contact lens wear? trauma? discharge?</td>
</tr>
<tr>
<td></td>
<td>photophobia? medical history (eg, DM)</td>
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</tbody>
</table>

Eye Care and the Emergency Department:
Non-injury related ocular ER visits comprised 51% of ocular-related visits).
Only 3% of ocular-related ER visits required hospitalization.
75% of the time, there was a clinically significant change in the diagnosis when care was first delivered at the ED or PCP and then followed up by a visit to an eye care specialist.

Acute Vision Loss

- The most common causes of acute loss of vision are:
  - Vascular occlusions of the retina (CRAO, CRVO)
  - Ischemic optic neuropathy
  - Vitreous hemorrhage (caused by diabetic retinopathy or trauma)
  - Trauma

Profound Loss of Vision

- Referring to a complete or greatly diminished vision affecting the whole field
- Common causes of severe vision loss:

<table>
<thead>
<tr>
<th>Vascular</th>
<th>Inflammatory</th>
<th>Infiltrative</th>
</tr>
</thead>
<tbody>
<tr>
<td>central retinal vein occlusion,</td>
<td>central retinal artery occlusion,</td>
<td>optic neuropathy</td>
</tr>
<tr>
<td>central retinal artery occlusion,</td>
<td>vitreous hemorrhage</td>
<td></td>
</tr>
<tr>
<td>vitreous hemorrhage</td>
<td></td>
<td>Mechanical</td>
</tr>
<tr>
<td></td>
<td></td>
<td>retinal detachment</td>
</tr>
</tbody>
</table>

Monocular vs. Binocular

- Ocular or optic nerve pathology causes monocular vision loss
- Lesion at or posterior to chiasm causes binocular vision loss
  - VF defects become more congruous the further back in the visual pathway
  - Homonymous VF defects noted posterior to chiasm
- Difference between mono vs. bino usually straightforward, keeping the following in mind:
  - Patients occasionally mistake homonymous hemianopsia (similar loss of visual field in both eyes) for a monocular loss

Visual Defects

Monocular

- Differentiate between eyes that have lost all useful vision and those that have blurred vision
- Blurring of vision is not localized and may be caused by pathology anywhere from cornea to optic nerve
- Need to get anatomical diagnosis first before considering the cause

Case Example

- 48 yr old white female presents with acute loss of vision in her right eye and decreased vision in her left
  - She was scheduled 2 weeks previously for a diabetic eye exam on a referral from her PCP but had fallen and was unable to make that appointment
  - Was diagnosed with diabetes 1.5 years ago
    - BS control has been erratic with range between 120-240
    - Last A1C: 9.1
Entrance Skills/Health Assessment

- VA: OD: finger count
- OS: 6/12 (20/40)
- CVF: OD: unable to assess
- OS: temporal hemianopsia
- Pupils: sluggish reactivity with a 2+
- RAPD OD
- SLE: corneal arcus noted, no other significant findings
- IOP: 16, 16 mmHG OD, OS
- DFE: see photos

Note: not patient photos

Physical Presentation

- Upon entering the room I noted that her right hand was twitching
  - I asked her how long that had been going on and she said about 2-3 weeks
  - I asked her if she experienced headaches, to which she said she had bad headaches that even woke her up at night

Imaging/Surgery Referral

- MRI revealed large mass in her brain
  - Patient was diagnosed with a Craniopharyngioma
  - She was referred for immediate surgery
  - Neurosurgeon reported that she removed a tangerine sized Craniopharyngioma
  - was the largest tumor she has ever removed

Note: not patient MRI
http://neurosurgery.ucla.edu/images/Pituitary%20Program/Craniopharyngioma/Cranio_Sag_Preop_fullylabeled.jpg

Our Patient

- Patient had a complete resection of the tumor in addition to radiation therapy
- She developed several significant perioperative complications:
  - Leakage of CSF which resulted in her having to have a shunt
  - She subsequently developed an infection post surgically
  - She is NLP in her right eye, but did regain 6/12 (20/40) vision in her left eye
  - Retains a temporal hemianopsia OS
  - Diabetes control became erratic and was put on several hormone replacement medications

Pituitary Adenomas

- Pituitary adenomas are common benign tumors of the pituitary gland
- Secreting tumors:
  - About 50% of adenomas produce too much of one of the hormones
- Non-functioning or endocrine-inactive pituitary tumors

Pituitary Adenomas

- The most common symptoms include:
  - Headaches
  - Vision problems that cannot be easily explained
  - Menstrual cycle changes in women
  - Mood swings or behavior changes
  - Erectile dysfunction
  - Weight change
Ocular Manifestations of Pituitary Adenoma

- Patients will present with a bilateral temporal hemianopsia
- Patients will typically have bilateral swollen optic nerves heads (papilledema) which can eventually develop optic atrophy/pallor

Papilledema: Causes

- Brain tumor or abscess
- Cerebral trauma or hemorrhage
- Meningitis
- Arachnoidal adhesions
- Cavernous or dural sinus thrombosis
- Encephalitis
- Idiopathic intracranial hypertension (pseudotumor cerebri), elevated CSF pressure and no mass lesion

Papilledema: Testing

- If papilledema is suspected clinically, MRI with gadolinium contrast or CT with contrast is done immediately to exclude causes such as an intracranial mass.
- Lumbar puncture with measurement of CSF pressure and analysis of CSF should be done if a mass lesion has been ruled out.
- Lumbar puncture in patients with intracranial mass lesions can result in brain stem herniation.

Epidemiology

- Nonarteritic: usually seen in younger patients
  - Fellow eye involved in 25-40% of cases
  - Associated with hypertension and diabetes

Case

- 56 YOBM reports decreased vision and some peripheral field loss OD
- VA: OD: 20/70 OS:20/20
- Right APD 2+
- IOP: 19, 20
- Fundus: see photo

Epidemiology

- Arteritic: usually seen in >55 yrs old (mostly over 70)
  - Fellow eye involved in 75% of cases within 2 weeks without treatment
Arteritic vs Non

<table>
<thead>
<tr>
<th>ARTERITIC AION</th>
<th>NON ARTERITIC AION</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td>70 years</td>
</tr>
<tr>
<td>GENDER</td>
<td>Female in male</td>
</tr>
<tr>
<td>ASSOCIATED SYMPTOMS</td>
<td>Headache, scalp tenderness, jaw claudication</td>
</tr>
<tr>
<td>VISUAL ACUITY</td>
<td>&lt;40 in 70% patients</td>
</tr>
<tr>
<td>ESR</td>
<td>&lt;10 (usually raised)</td>
</tr>
<tr>
<td>FIA</td>
<td>Chronic (&gt;30 – 60%) and disc filling delay</td>
</tr>
<tr>
<td>NATOMES</td>
<td>Poor progression for recovery</td>
</tr>
<tr>
<td>THERAPY</td>
<td>Urgent administration of corticosteroids</td>
</tr>
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Erythrocyte Sedimentation Rate
This measures the height of RBC's settling out of plasma per hour

<table>
<thead>
<tr>
<th>ESR</th>
<th>Males: Age/2</th>
<th>Good sensitivity but poor specificity. Takes time for the levels to become detectable</th>
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<tbody>
<tr>
<td></td>
<td>Females: (Age +10)/2</td>
<td>High: Indicative of giant cell arteritis but normal levels do not exclude GCA as a diagnosis</td>
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C-Reactive Protein

- C-Reactive Protein
  - Normal = no CRP
  - Abnormal serum glycoprotein produced by liver during acute inflammation

There are two different tests that measure CRP and each test measures a different range of CRP level in the blood for different purposes:

- The standard CRP test measures markedly high levels of the protein to detect diseases that cause significant inflammation. It measures CRP in the range from 10 to 1000 mg/L.
- The hs-CRP test accurately detects lower levels of the protein than the standard CRP test and is used to evaluate individuals for risk of CVD. It measures CRP in the range from 0.5 to 10 mg/L.

Giant Cell Arteritis

- Vessels most often involved are the arteries of the scalp and head, especially the arteries over the temples, which is why another term for GCA is “temporal arteritis.”
- Symptoms, such as fatigue, loss of appetite, weight loss or a flu-like feeling
  - There may be pain in the jaw with chewing
  - Sometimes the only sign of GCA is unexplained fever
  - Less common symptoms include pains in the face, tongue or throat.
- GCA is a clinical diagnosis!
- If patient meets criteria of clinical symptoms then treatment will be started regardless of whether lab test or biopsy are positive
- Treatment should be started before lab results are back.
Symptoms
• Acute visual loss (arteritic>non)
• dyschromatopsia
• Arteritic may also have associated:
  – Headache, fever, malaise,
  – weight loss, scalp tenderness, jaw claudication,
  – amaurosis fugax, diplopia, and eye pain.

Ocular Signs
• Sudden, unilateral, painless decreased vision and color vision
• Positive RAPD
• Altitudinal visual field defect (usually inferior and large)
• Swollen optic disc
• Fellow nerve often crowded with small or absent cup (“disc at risk”)

Additional Testing
• Lab tests:
  – STAT ESR (rule out arteritic form)
  – CBC (low hematocrit, high platelets)
  – Fasting blood sugar
  – C reactive protein,
  – RPR or VDRL/FTA-ABS or TPPA
  – ANA
• Check blood pressure

“Traditional” Management
• Arteritic:
  – Systemic steroids to prevent fellow eye involvement
    • methylprednisolone 1 g IV qd in divided doses for 3 days then,
    • prednisone 60-100 mg po qd with a slow taper
  – Check PPD, blood glu and chest radiographs before starting systemic steroids
• Non-arteritic:
  – Consider daily aspirin

GCA Treatment Update!!
• May 22, 2017:
  – FDA expanded and approved the use of subcutaneous Actemra (tocilizumab) to treat adults with giant cell arteritis.
  – First FDA approved therapy, specific to this type of vasculitis
  – Primary endpoint was sustained remission and was defined as the absence of symptoms of giant cell arteritis, normalization of inflammatory laboratory tests, and tapering the use of prednisone
  – Patients were placed on subcutaneous Actemra and standardized prednisone compared to placebo and standardized prednisone treatment

13 YR Female
CC: noticed that her left eye became blurry and objects were “wavy” a couple of days ago. Sudden onset and she had experienced a headache over the left eye just prior to the vision going blurry.
Ocular Hx: she currently wear glasses for distance
Medical Hx: she is currently not diagnosed with any health problems and is not taking any medications
Entrance Skills

VA with current Rx: 20/30 OD and 20/30 OS
Entrance skills unremarkable
Amsler: metamorphopsia OS
BCVA: 20/20 OD with increased minus, no improvement possible in the left eye
IOPs: 13 mm Hg OD and OS

Fundus Photos

From the Experts

• Vogt-Koyanagi-Harada (VKH) disease is a multisystemic disorder characterized by granulomatous panuveitis with exudative retinal detachments that is often associated with neurologic and cutaneous manifestations.
• VKH disease occurs more commonly in patients with a genetic predisposition to the disease, including those from Asian, Middle Eastern, Hispanic, and Native American populations.

OCT

Retina Consult

• Referred patient to retina and they confirmed the diagnosis of VKH.
• She was begun on oral prednisone 60 mg per day and she was re-evaluated in 1 week.
• At the follow up, there was reduction in her serous retinopathy and vision was improved.

From the Experts

• VKH:
  – Patients have no prior history of ocular trauma or surgery
  – Patients have no evidence of another ocular disease based on clinical or laboratory evidence
  – Patients have bilateral ocular involvement.
From the Experts

 VKH:
- The neurologic and auditory signs include the following:
  - Malaise, fever, headache, nausea, abdominal pain, stiffness of the neck and back, or a combination of these factors; headache alone is not sufficient to meet the definition of meningitis
  - Tinnitus
  - Cerebrospinal fluid pleocytosis
- Integumentary signs include the following:
  - Alopecia: loss of body hair
  - Poliosis: loss of pigment in hair
  - Vitiligo: loss of skin pigmentation in blotchy pattern

VKH Treatment

- For most patients with bilateral serous detachments and severe visual loss, begin therapy with systemic prednisone (1-2 mg/kg/day).
- The length of treatment and subsequent taper must be individualized for each patient.
  - Most patients require therapy for 6 months and occasionally up to 1 year before successful tapering of systemic corticosteroids.
  - Systemic therapy should not be discontinued during the 3 months following the onset of the disease because of the risk for recurrence.

Case

- 30 WM presents with 2 weeks worsening vision OS
  - Was seen by neurologist 2 years previously for flashes, head CT was normal
  - Flashes continued for the two years
  - History of color blindness
  - Patient presents with pressure behind the eye and tightness with left eye movement for the past week
  - No vision changes with activity or movement
  - Denies history of trauma, redness, discharge or headache
- VA: 6/6 (20/20) and 6/9 (20/30)
- External exam reveals no ptosis or resistance to retropulsion

Case

- PERRL with a left APD
- Hertel: Base of 102 and measurements of 19 and 18
- EOM: FROM though notes tight feeling/pain in OS abduction
- IOP: 15 OU
- DFE: normal ONH appearance and fundus unremarkable
- HVF: inferior altitudinal defect OS

Case

- One week f/u:
  - Reports continued decreasing vision OS
    - Now 6/120 (20/400)
  - Increased left APD and more pain on eye movement
  - Increased visual field defect
  - ONH swelling OS

Optic Neuritis

- Optic neuritis typically presents with 2 main symptoms:
  - loss of vision (>90%) and eye pain (92%)
  - Color vision defects noted in 88% (Ishihara) 94% with 100 hue
  - 1/3rd patients had ONH swelling, 2/3rd have retrobulbar and normal fundus
- The initial attack is unilateral in 90% of adult patients and bilateral in 10% (typically in younger patients 12-15).
Optic Neuritis Treatment Trial (ONTT)

- The mean age of onset of optic neuritis is in the third decade of life.
- Five-year incidence of clinically definite MS is 30% following a first episode of idiopathic demyelinating optic neuritis. The cumulative incidence increased to 40% at 12 years and 50% at 15 years.
- The median time to diagnosis of MS was three years.
- Presence of characteristic demyelinating lesions on MRI is a strong predictor of developing MS.
  - The risk of MS after 15 years was 72% among those with one or more lesions on MRI versus 25% among those with no lesions.

Case

- 78 yr old Asian male presents for an eye exam with a complaint of decreased vision in both eyes.
  - Last eye exam was 1.5 years ago
    - Vision was correctable to 20/25 OD, OS
    - IOP's: 21, 18
    - C/D: 0.5/0.5, 0.5/0.5
  - Medical history:
    - Hypertension for 10 years

Case: Management

- We instilled every 15 minutes for first hour:
  - 1 drop of Azopt
  - 1 drop of Combigan
- 2 x 250 Diamox po
- IOP check every 15-20 minutes for first hour
  - At 1 hour time period patient's IOP had dropped to 28
- Referred to glaucoma specialist who performed a LPI and then referred to a cataract surgeon for extraction/PCIOL.

PAC/AAC Treatment

- Untreated, can cause severe visual loss and eventual blindness (PACG)
- Conventional treatment has utilized the use of both topical and systemic IOP lowering medications:
  - Immediate treatment includes oral acetazolamide 500 mg (2 x 250 mg, don't use sequels)
  - Topical B-blockers, topical lopidine (any other glaucoma meds you have available) q 15 min X 2 and then BID.
  - Topical steroid (Pred Forte 1% q 15 min X 4, then q 1 h)
  - Pilocarpine (1-2% QID) ?????
  - If no response consider use of hyperosmotic (eg oral glycerol)
**Acetazolamide (Diamox)**

- Reduces rate of aqueous humor formation by direct inhibition of enzyme carbonic anhydrase (CA) on secretory ciliary epithelium, causing, in turn, a reduction in IOP.
- May reduce IOP by 40-60%. Effects are seen in about an hour, they peak in 4 h, and trough in about 12 h.

**Extent of PAS?**

- Eyes with PAS affecting more than a critical extent (initial threshold of 180° of the angle) are more likely to require a filtration procedure
  - if the angle is entirely closed by PAS, a filter is likely needed
  - if the entire angle can be indented open, iridoplasty or cataract extraction are likely to be sufficient.

**PAC Treatment**

- As soon as IOP controlled and sufficient corneal clarity is established
  - Laser peripheral iridotomy (LPI) is the established first-line treatment for angle closure
  - relieves the pupil block component present in the condition and may be beneficial in PACS eyes to prevent Acute Angle Closure and also a chronic rise in IOP

**Case**

- 20 year old male presents with a red painful eye
  - Started that morning when he woke up
  - reports a watery discharge, no itching, and is not a contact lens wearer
- SLE:
  - See attached image with NaFl stain

**Herpes Simplex Keratitis: Clinical Features**

- Characterized by primary outbreak and subsequent reactivation
- Primary outbreak is typically mild or subclinical
- After primary infection, the virus becomes latent in the trigeminal ganglion or cornea
- Stress, UV radiation, and hormonal changes can reactivate the virus
- Lesions are common in the immunocompromised (i.e. recent organ transplant or HIV patients)
Pediatric HSV Keratitis

- Pediatric herpes simplex keratitis has an 80% risk of recurrence, a 75% risk of stromal disease, and a 30% rate of misdiagnosis.
- 80% of children with herpes simplex keratitis develop scarring, mostly in the central cornea.
  - Results in the development of astigmatism.
  - 25% of children have more than 2 D of astigmatism, most of which is irregular.
- Consider pediatric HSV when a patient has unilateral recurrent disease in the anterior segment.

HSV Stromal Disease

- HSV stromal disease is an immune-mediated disease.
- Increased risk of scarring and high risk of poor visual prognosis.
- Requires corticosteroids (HEDS: corticosteroid reduced risk of progression by 68%)
  - Without epithelial defect: corticosteroids and prophylactic anti-viral dosage.
  - With epithelial defect: active infection anti-viral dosage with judicious corticosteroids.

How much to dose steroid?

- HEDS used QID of prednisolone phosphate.
- Current Recommendations:
  - Mod – severe (especially with neo): 1% Prednisolone or Lotemax QID to 6x/day.
  - Want the lowest dose needed to control the inflammation.
  - AAO EBM Treatment Guideline 2014
    - Topical steroid for 10 weeks (this is based on HEDS results) with oral antiviral.

Herpes Simplex Keratitis Management

- Topical:
  - Viroptic (trifluridine) q 2h until epi healed then taper down for 10-14 days.
  - Viroptic is toxic to the cornea.
  - Zirgan (ganciclovir) available, use 5 times a day until epi healed then 3 times a week (US only).

Anti-Viral Medication

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Bioavailability</th>
<th>Dosing</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acyclovir</td>
<td>Inhibits DNA synthesis in inhibiting viral replication.</td>
<td>10-30% gets absorbed.</td>
<td>10-30% of patients</td>
<td>Nausea, vomiting, headache, diarrhea, confusion.</td>
</tr>
<tr>
<td>Zidovudine</td>
<td>Equivalent to acyclovir but better for pain management</td>
<td>95% converted to aciclovir</td>
<td>Better bioavailability and longer 1/2 life.</td>
<td>Same as acyclovir.</td>
</tr>
<tr>
<td>Famciclovir</td>
<td>Inhibits DNA chain elongation.</td>
<td>Superior to acyclovir</td>
<td>Superior to aciclovir.</td>
<td>Same as acyclovir.</td>
</tr>
</tbody>
</table>
Herpes Simplex Epithelial Keratitis

• Treatment Regimen:
  – Zirgan 5x/day until the ulcer heals, then 3x/day for one week
  – Oral Valtrex 500 mg 3x/day for 7-10 days
  – Artificial tears
  – L-lysine 2 grams daily?
    • Proven to “slow down” and retard the growth of the herpes virus and inhibit viral replication
    • Prior to topical antiviral therapy debridement was treatment of choice
    • Generally try to avoid use of sharp instruments and use of cotton swab and anesthetic

• RTC 1 day, 4 days, 7 days

Herpes Simplex Keratitis

• Prophylactic Treatment:
  – Reduces the rate of recurrence of epithelial and stromal keratitis by ≈ 50%
    • Acyclovir 400 mg BID
    • Valtrex 500 mg QD
    • Famvir 250 mg QD
  – L-lysine 1 gram/day;
    • Proven to “slow down” and retard the growth of the herpes virus and inhibit viral replication
    • Frequent debilitating recurrences, bilateral involvement, or HSV infection in a monocular patient

Prophylaxis??

• Pitfalls to Prophylaxis:
  – Reduction of recurrence does not persist once drug stopped
  – Resistance????
    • van Velzen, et. al., (2013) demonstrated that long-term ACV prophylaxis predisposes to ACV-refractory disease due to the emergence of corneal ACVR HSV-1.

Flashes and Floaters

• Patients often present complaining of “spots” or “cobwebs” in front of their eyes
• Causes of floaters include: posterior vitreous detachment (PVD), retinal tear, vitreous heme, uveitis.
• Since PVD and retinal tears present the same way, a RT has to be eliminated
• Ask the patient whether spots move with eye and continue to move after the eye has stopped
• Large spots could be blood clots

Posterior Vitreous Detachment (PVD)

Retinal Tear
Flashes and Floaters

- Sudden onset typically means a PVD, retinal tear or heme
- If the spots appear after flashing light, then retinal tear must be eliminated
- Myopes tend to have floaters and will notice them for a long time
- Key is to rule out potentially sight threatening condition for the floaters, ie retinal tear.
- Patients with retinal condition such as lattice degeneration and myopes need to be educated about S&S of RD (flashes and floaters)
  - 8-11% population has lattice
  - Risk of RD with lattice is <1%
  - 30-50% of patients with a RD have lattice

Flashes and Floaters: Management

- A patient who presents with a sudden onset PVD without retinal breaks or hemorrhage requires repeat peripheral examination in six weeks, as the risk of retinal complications is highest within the six weeks following vitreous detachment.
- If no retinal breaks are seen at that point, routine yearly examination is all that is needed

Recent Day in Clinic

- 50 yr old White male
  - Needs his drivers license form filled out as he failed his vision screening
- Vision: 20/100 (6/30) and 20/200 (6/60)
- Eye pressure:
  - 15 and 11

Management

- Diagnosis:
  - Retinal detachment with macula off
- Management:
  - Contacted retinal specialist and requested the patient be seen that day because we were concerned that he might not show up for an appointment if he didn’t go over immediately
  - Was seen by retina and scheduled for retinal repair the following day

Patient B-Scan Ultrasound

Retinal Detachment (RD)

- RD is the separation of the neurosensory retina from the underlying retinal pigment epithelium (RPE), resulting in loss of the corresponding visual field in the affected eye.
- Rhegmatogenous retinal detachment is the most common type
  - necessary for a rhegmatogenous retinal detachment are a full thickness retinal break and vitreous liquefaction.
- Failure to diagnose retinal detachment remains the second-most-common malpractice claim against optometrists.
RD Risk Factors

- Risk factors for rhegmatogenous RD include advancing age, previous cataract surgery, high myopia, focal retinal atrophy (lattice degeneration) and trauma.
- Lifetime risk for RD without lattice degeneration in an emmetrope is 0.3% while in a myope over -5.0 diopters life time risk is 2.2%.
- Individuals with myopia exceeding -5.0 diopters and have associated LD, the life time risk for RD increases to 35.9%.

Retinal Detachment

- A key indicator for visual outcome is the preoperative status of the macula.
  - Retention of good central visual function is expected if the macula is attached at the time of surgery
  - <75% of patients will achieve 20/40 or better once the macula detaches
  - rapid surgical intervention in the presence of a detached macula does not improve the visual prognosis.

RD: Symptoms?

- Evidence suggests that symptoms alone may not be reliable indicators of RD, and additional modifiers such as age, history of intraocular surgery, myopia and previous retinal pathology are required
  - isolated symptoms of flashes, floaters, progressive field defect, ocular pain or dyschromatopsia can equally be associated with other pathologies (e.g. AION)

RD: Symptoms?

- Predisposing event for RD is the development of a PVD
- An Australian study:
  - 90% of RD patients reported a variety of symptoms, with more than half (54.1%) reporting a combination of symptoms.
  - the single commonest symptom was loss of vision
  - patient self-awareness of an RD does not appear to be greater in that cohort who has previously experienced an RRD in either the presenting or fellow eye

RD: Treatment and Management

- If high risk retinal breaks are detected early, outpatient laser retinopexy or cryopexy therapy can be successfully accomplished in the clinic setting
  - treatment is over 95% effective in preventing progression of a retinal tear to RD
- if an RD has developed, surgical reattachment will be required.
  - Surgical correction of the RD aims to relieve vitreoretinal traction, close retinal tears and holes, remove subretinal fluid and reattach the retina.
  - Scleral buckling techniques achieve reattachment in over 90% of cases

Retinal Detachment Treatment
RD: Treatment and Management

- Reattachment may also be aided by incisional drainage of subretinal fluid, and/or using expansile gases or silicone oil to push the retina back into place.
- Although surgical treatment can result in 90% anatomical cure (permanent reattachment), visual outcomes can vary based on the etiology, length of time of detachment, and involvement of the macula.

Vision Loss Without Pain: Artery Occlusion

- Primarily embolic in nature from cholesterol, calcifications, plaques.
- Usually occurs in elderly associated with:
  - hypertension (67%),
  - carotid occlusive disease (25%),
  - DM (33%) and
  - cardiac valvular disease.
- Sudden loss of unilateral, painless vision
  - defect dependent upon location of occlusion

Vision Loss Without Pain: Artery Occlusion

- BRAO typically located in temporal retinal bifurcations.

CRAO

- CRAO has profound vision loss with history of amaurosis fugax.
  - Vision is usually CF (count fingers) to LP (light perception) with positive APD.
  - Diffuse retinal whitening with arteriole constriction, cherry red macula.

Ophthalmic Emergency

- Treatment is controversial due to poor prognosis and questionable benefit.
- Treat immediately before workup, if patient presents within 24 hours of visual loss:
  - Digital ocular massage,
  - systemic acetazolamide (500 mg IV or po),
  - topical ocular hypertensive drops (lopiol, B-blocker),
  - anterior chamber paracentesis,
  - consider admission to hospital for carbogen Tx (high carbon dioxide)