Clinical Decisions in Retina

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Mark Dunbar: Disclosure

- Consultant for
  - Allergan Pharmn
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  - Allergan
  - Carl Zeiss Meditec
  - Inspire
  - Alcon Nutritional Advisory Board
  - Macula Risk

- Speakers Bureau
  - Allergan
  - Carl Zeiss
  - Inspire
  - VSP

Mark Dunbar does not own stock in any of the above companies

Examining the Retina

- Don’t forget to look at the anterior vitreous
- Needs to be done on every dilated patient
- Done at the slit lamp, looking posterior to the lens
- Retroillumination may help if you suspect vitreous cell
  More on this later

Examining the Retina

- Assess the optic nerve
  - What is the cup to disc ratio
  - Is there good coloration and perfusion
  - Is it flat
  - Choroidal or scleral crescent

Examining the Retina

What is the caliber of the retinal vessels

- Make sure you look and consciously take not of what the caliber is
- Narrowing of the vessels requires checking the blood pressure
- Normal A/V ratio is 2/3, ¾
- What about the arterial light reflex?

Examining the Retina

The Macula

- Is there a foveal light reflex (FLR)?
- Is it flat?
- Is there any fluid, hemorrhage, or exudate
- Presence of drusen
- RPE mottling
Examining the Retina

The peripheral retina
- It has to be done through a dilated pupil
- Don’t substitute imaging for indirect ophthalmoscopy
  - Use Imaging as a compliment, but not substitute
- Be systematic in your examination
- You should be able to see ora on “all” gazes
  - It’s all about technique

59 y/o White Male

CC of new onset flashes RE X 3 wks
- See’s them only at night
- Does not seem to affect is vision
- VA: Best-corrected 20/20 (-3.50 OU)
- Motility, CVF, Pupils – all normal
- Anterior Segment – unremarkable
- Posterior Segment -

59 y/o White Male

What are you suspicious of?
What are you looking for?
Posterior Vitreous Detachment

PVD

- 65% of individuals > 65 have PVD
- More common in women
- More common following intraocular surgery
- More common following inflammation
- More common in aphakes

Exam of a Pt with Symptomatic PVD

- Should have a high suspicion of detecting Weis ring
- Should have a high index of suspicion of a possible retinal break
- Clinical exam should be conducted with these suspicions

PVD

- Retinal tears occur 8-15% of eyes with symptomatic PVD
  - 90% are superior
- VH occurs in 13-19% of symptomatic PVD’s
- VH + PVD -> 70% will have a retinal break
- PVD No VH -> 2-4% will have retinal break
Clinical Exam of a Patient with A Symptomatic PVD

- All the testing and procedures that you would normally do with any patient
- Dilated fundus exam
- Look specifically at the anterior vitreous
  - Note presence or absence of pigment or cells in the anterior vitreous → tobacco dust, Schafer’s sign
- Peripheral extended ophthalmoscopy including scleral depression

PVD is Seen

What is your management? Do you bring him back for follow up?

Management of Acute PVD

No Symptoms

- Educate about the Si/Sx of RD
- Return in 1 yr

With Symptoms

- Educate about the Si/Sx of RD
- Return in 4-6 weeks, then 3-4 months, then annually

Management of Acute PVD

PVD NOT Seen

but has symptoms…

What is your management?

Return with in 3-4 weeks

Lattice Degeneration as a Routine Finding?

Is this any cause for concern? How do you manage it?
Lattice Degeneration

- Present 5-20% of the general population
- Localized area of retinal thinning associated with a fluid pocket in the overlying cortical vitreous

Lattice Degeneration and Risk of RD

- RD develop in 0.7% of eyes with lattice degeneration followed for 10.8 yrs
- Eyes with lattice that developed tractional retinal tears
  - 40% occurred in areas not associated with lattice...normal-appearing retina

Byer NE. Ophthalmology. 1989; 96:1401-1402

Indications for Prophylactic Treatment of Peripheral Retinal Tears and Holes in Symptomatic Patients

<table>
<thead>
<tr>
<th>Treat</th>
<th>Horseshoe tears</th>
<th>Dialysis</th>
<th>Operculated holes</th>
<th>Atrophic holes</th>
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</thead>
<tbody>
<tr>
<td>Phakic</td>
<td>Yes</td>
<td>Usually</td>
<td>Rarely</td>
<td>No</td>
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Indications for Prophylactic Treatment of Peripheral Retinal Tears and Holes in Asymptomatic Patients

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<td>Rarely</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Flap Tears</td>
<td>Sometimes</td>
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48 y/o Asymptomatic Pilot

- VA 20/15 OU
- Anterior Segment: Unremarkable
- Fundus

Diagnosis?

- Choroidal Nevus
- Choroidal Melanoma
**Choroidal Nevi**
- < 3 mm elevation
- < 3 DD in size
  - 95% are less than 2 DD
- Slate gray
- Drusen
- SRF associated with drusen
- CNVM

**Features Suggesting Nevi**
- Drusen
- Overlying neurosensory detachment
- Choroidal neovascular membrane
- Circinate exudate
- Bony pigment spiculing
- Zones of RPE atrophy

**Choroidal Melanoma**
- > 3 mm elevation
- Variable pigment
- Multiple areas of orange pigment (lipofuscin)
- Serous fluid (detachment) in absence of drusen
  - Unequivocal evidence of growth

**Management**
- Flat choroidal nevi: follow yearly
- Suspicious nevi:
  - photo
  - follow in 6 wks, 3 mo, then 6 mo
  - evidence of growth → early melanoma
- Lesions > 3 mm thickness: probably early melanoma

**Isolated/Flame Hemorrhages**

**Isolated Cotton Wool Spot**
26 y/o Asian Female

- 2nd Year Med Student
- Healthy
- Routine exam – no complaints
- 6 mo ago, nonspecific episodes weakness and not feeling right
- Myopic – 20/20 OU

Assessment and Plan?

What is your management of an isolated CWS in absence of anything else?

Are you obligated to work it up?

Cotton Wool Spots


24 consecutive patients presenting with multiple or single CWS were worked up - Known diabetic patients excluded

- 5 patients were found to be undiagnosed diabetes
- 5 patients HTN
- 2 pts with cardiac valvular disease
- 2 pts with radiation ret
- 2 pts with severe carotid artery obstruction
- 1 Dermatomyositis

- 1 SLE
- 1 Polyarteritis nodosa
- 1 Leukemia
- 1 AIDS
- 1 Purtscher's retinopathy
- 1 Metastatic carcinoma,
- 1 IV drug abuse,

Systemic workup failed to reveal an underlying cause in only 1 patient

The presence of even 1 CSW spot in an otherwise normal fundus necessitates an investigation to ascertain systemic etiologic factors

Basic Work Up for CWS

- HTN
- Fasting blood glucose
- CBC
- ANA
- HIV testing

The Moral of the Story

- What’s common…is common
- Make sure you LOOK at the retinal vessels
  - Note the caliber and the presence of crossing changes
- Blood pressures are important
- You have the potential to save lives
- You would be surprised how many people are out there with dangerously high BP
The Diabetic Patient

What are the questions that you ask yourself when examining a diabetic?

- Look at the disc – specifically look for subtle NVD
- Are there hemorrhages or microaneurisms?
- Exudated, cotton wool spots?
- Look for the presence of NVE, traction, or VH
- Macular involvement?

What is the extent of the involvement?
That is the basis for classification

Diabetic Retinopathy Classification
Mild to Moderate Nonproliferative (NPDR)
- Hemorrhages, microaneurysms
- Hard exudate
- Cotton wool spots (CWS)
- Minimal venous beading/IRMA
- Macular edema

Severe Nonproliferative Diabetic Retinopathy

4-2-1 Rule
- Hemorrhages & Mac in 4 quadrants – or –
- Significant venous beading in 2 quadrants – or –
- IRMA in 1 quadrant

Risk for Developing PDR in 1 yr
- Mild NPDR: 5%
- Moderate NP: 12%
- Severe NPDR: 52%
- Very Severe NPDR 72%
Is there Macular Involvement?

- Are there hemorrhage or exudates in close proximity to the macula?
- Is it within 500 µ (1/3 DD)?
- Is there associated retinal thickening?
- By definition – that is clinically significant macular edema (CSME)

CSME

- Retinal thickening within 500 microns from the center of the FAZ
- Hard exudates associated with retinal thickening 500 microns from center of FAZ
- Zones of retinal thickening > 1 DD in area, any part of which is 1 DD from the center of the fovea

How Does Macular Edema Manifest Itself?

In what forms can macular edema present?

Diabetic Retinopathy

Pathologic process

- Microaneurysms
- Vascular permeability
- Ischemia
- Proliferation
- Cicatriziation

Diabetic Retinopathy

- Clinically significant macular edema (CSME)
  Retinal thickening which involves or threatens the center of the macula
**Diabetic Macular Edema**

It can’t be diagnosed with a direct ophthalmoscope. You need a 3D view of the macula.

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**Is there CSME?**

If yes – referral to retinal specialist is recommended.

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**Optometric Management of Diabetic Patient**

- **No diabetic retinopathy**
  - Educate and follow yearly
- **Early or moderate NPDR**
  - Establish presence of CSME
    - If CSME refer to retina specialist
  - No CSME
    - Educate
    - Follow yearly

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**Optometric Management of Diabetic Patient**

- **Severe NPDR**
  - Follow every 4 months
- **PDR:** refer to retina specialist

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**Proliferative Retinopathy (PDR)**

- Vitreous hemorrhage
- NVD
- NVE
- Fibrovascular proliferation
- Retinal detachment

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**Asymptomatic Hollenhorst Plaques**

When do you initiate a work up?

1. Never
2. Always
3. Only when symptoms such as TIA’s or amaurosis fugax
Asymptomatic Hollenhorst Plaques

Important Questions to Ask

- Any TIA’s?
  - Occurs in 30-50% of pt with severe CAD
  - 50-75% of stroke pts have had TIA’s
- Any amaurosis fugax?
  - Retinal TIA’s
  - Lasts up to 2 minutes (can be up to 2 hrs)
- Numbness/weakness in parts of their body

Work Up of Retinal Plaques

Progress in Retinal Eye Research 24 (2005) 493-519

Misconception: That Asymptomatic plaque(s) in retinal arteries do not require a detailed evaluation

SS Hayreh:
“This misconception may result in a patient later developing retinal artery occlusion and visual loss, or a cerebral vascular accident. Thus it is prudent to evaluate the source of all asymptomatic plaque(s)...”

Unexplained Vision Loss

Causes

- Refractive
- Functional
- Lens Opacities
- Neurologic
- Occult Retinal/Macular Disease

It has to make sense...

Unexplained Vision Loss

Obeying the Fundamentals of the Complete Eye Exam is Important

Its not always what you see, but more what you don’t that makes it difficult....
When do you refer?
When should you refer?

Retinal Imaging and OCT has really helped us figure out many of these cases of unexplained vision loss.

Optical Coherence Tomography (OCT)
- Non-contact, non-invasive imaging device
- Produces high-resolution images of the posterior segment
  - Optical biopsy
- Images are objective and quantifiable

Advantages of OCT
- Quick – takes less than five minutes to obtain images of both eyes
- Non-invasive and well tolerated by patients
  - No injection
  - No biohazard or blood-related risk
  - No medication reactions
- More readily interpreted and understood by patients

Main Clinical Utilities of OCT
- High resolution evaluation of retinal anatomy
- Diagnosis of macular conditions difficult to establish with biomicroscopy
- Quantitative assessment of retinal anatomic alterations
- Quantitative assessment of vitreoretinal interface
- Objective means for monitoring disease progression and/or therapeutic response

Central Retinal Vein Occlusion (CRVO)
- Common retinal vascular disorder with potentially blinding complications
  - Macular edema
  - Neovascularization leading to NVG
- Intraretinal hemorrhages in all 4 quadrants
- Dilated venous system
- Generalized disc edema
What is the pathophysiology of a CRVO?
1. Thrombin at the level of the lamina
2. Emboli at the level of the lamina
3. Hyperviscosity of the blood
4. Spasm of the central retinal vein

What is the difference between these two clinical presentations?
1. Only difference is severity – #2 is much worse
2. They are two different diseases: #1 has ocular ischemia, #2 has CRVO
3. #2 is ischemic CRVO, #1 is nonischemic CRVO
4. #2 has a combined CRVO and CRAO

CRVO: Ischemic vs. Nonischemic
30% 70%
- Two distinct clinical entities
- Nonischemic -> relatively benign
- Ischemic -> seriously blinding disease with a high risk of neovascularization
  - Little chance of visual improvement
  - Poor visual outcome

Site of the CRVO...
Is it the lamina cribrosa?
- This information is based on histopathology of eyes enucleated for NVG
  - Eye with Ischemic CRVO
- Eyes with nonischemic CRVO do not get enucleated -> no histopathology to base where the site of the thrombosis occurs
- Nonischemic CRVO occlusion is likely posterior to the lamina –
  - More collateral circulation available to shunt blood

For patients with CRVO – where are they likely to develop neovascularization?
1. They don’t develop neovascularization
2. Anterior segment
3. Posterior segment
4. Can be either anterior or posterior segment

What is the risk for developing neovascular complications with an ischemic CRVO?
1. Less than 10%
2. About 34%
3. More than 50%
4. About 75%
Central Retinal Vein Occlusion Study (CVOS)

**Purpose:**
- To determine whether photocoagulation can help prevent iris neovascularization in eyes with CVO and evidence of ischemic retina
- To assess whether grid-pattern photocoagulation will reduce loss of VA due to macular edema
- To develop new data describing the course and prognosis for eyes with CVO

CVOS:
- 728 eyes from 725 patients
- Randomized to 4 study groups
  - Perfused
  - Nonperfused
  - Indeterminate perfusion
  - Macular edema

CVOS: Early Results
- 46 eyes randomized to indeterminate group
  - 38 of 46 eyes (83%) went on to nonperfusion
- 547 eyes randomized to perfused group
  - 4 months: 16% (81 or 522) developed evidence of ischemia (10 DD of nonperfusion or NVI/NVE)
- Risk factors for ischemia
  - Duration < 1 month
  - VA < 20/200
  - 5 to 9 disc areas of nonperfusion in retina

CRVO Study: 3 Yr Natural History
- VA outcome dependent on initial acuity
  - Initial ≥20/40: 65% maintained
  - VA < 20/200 initial: 80% chance VA ≤ at final
  - VA 20/50 to 20/200: 19% improved, 44% stayed in this range, 37% worse than 20/200

CRVO Study: 3 Yr Natural History
- 34% initially perfused converted to ischemic
- INV/ANV developed in 16% of all eyes (117/714)
  - 56/117 were initially perfused
- 35% of nonperfused eyes developed INV/ANV

Suspicious Optic Nerves

Pseudoedema vs. True Optic Nerve Edema

What goes into your decision making process when you decide if this patient has true disc edema vs. anomalous nerve?
**The Typical Scenario…**

- Disc elevation….looks like disc elevation
- Blurring of the disc margins
- No optic nerve dysfunction
- Often bilateral…but could be unilateral

How do you differentiate?

**Optic nerve head swelling is a common, but nonspecific, neuro-ophthalmic sign**

It may present as either as true edema vs. pseudoedema

True edema may arise from a variety causes

**True Disc Edema vs. PseudoEdema**

**True Disc Edema**
- Absent SVP
- Hyperemia of the nerve – heightened reddish hue
- Absent hyperemia
- Absent dilation of microvasculature on the surface of the nerve
- Dilation/telangiectasis of the microvasculature and surface capillaries

**PseudoEdema**
- SVP Present
  - Absent in 20% of normals
  - Anomalous branching pattern of the retinal vessels
  - Yellow hazy appearance in deep peripapillary tissue
  - Obscuring border b/w disc and retina

**True Disc Edema vs. PseudoEdema**

**True Disc Edema**
- Blurred disc margins
- Occurs at the level of RNFL
- Obscuring retinal vessels and junction b/w myelinated and nonmyelinated RNFL

**PseudoEdema**
- Blurring disc margins result from changes occurring at the RPE
- Thus retinal vessels are clearly seen as they cross the disc margin
Papilledema
- Intracranial mass
- Hydrocephalus
- Idiopathic intracranial hypertension
- Meningitis or encephalitis
- CNS granulomatous or malignant transformation

Optic Disc Edema with Optic Nerve Dysfunction
- AION
- Optic neuritis
- Leber’s hereditary optic neuropathy
- Intraorbital optic nerve compression
- Infiltrative optic neuropathy
- Toxic optic neuropathy

Buried Disc Drusen
- Scalloped appearance to the disc margin
- Disc is not hyperemic
- No microvascular blood vessel abnormalities on the surface of the nerve

Buried Disc Drusen
- Anomalous branching retinal vessels
  - Loops, trifurcations and increased branching
- Gray/black change deep around the nerve

Hemorrhage or Subretinal Hemorrhage in the Macula?
What are the implications?
What clinical decisions do you make with regards to etiology?

72 y/o White Male
Blurred VA with Glasses R > L
RE: 20/30, LE 20/25
72 y/o White Male
Blurred VA with Glasses R > L
RE: 20/30, LE 20/25

Diagnosis
◆ Age-Related Macular Degeneration

What are the questions that you ask yourself when you examine the macula of a patient like this?

The Questions
◆ Is there fluid in the macula?
◆ Do you see subretinal hemorrhage or exudate?
◆ Is the macula flat or is there any elevation?

The Questions
◆ If you can confidently answer No to these questions…and you have explained their acuity (20/30) your job is done
◆ If the answer is Yes to any of these questions, then you need to conduct a work up
  ♦ FA and OCT

Age-related Macular Degeneration (AMD)
◆ Degenerative disorder that affects the macula
◆ Leading cause of legal blindness in people > 65 yo
◆ 90% of vision loss is 2° to CNV

Dry ARMD
◆ Earliest clinically detectable feature
◆ Lie between BM of RPE and Bruch’s
◆ Hard drusen: smaller, calcified or ossified
◆ Soft drusen: ill-defined, larger, coalesce, resemble small serous detachments

Choroidal Neovascularization (CNV)
◆ Growth of new blood vessels originating from the choroid
◆ Growth under the sub-RPE or subretinal space
Type I vs. Type II

Classification CNV
- Classic - well defined on FA
- Occult - represent 70% of CNV
  - Poorly defined by FA – nondistinct borders
  - Stippled hyperfluorescence, with late leakage
- Mixed

Classification CNV
- Occult CNV
  - Predominantly Classic
    - Area of classic CNV ≥ 50% of the lesion
  - Minimally Classic
    - Area of classic component < 50% of the lesion
  - Occult-only
    - No classic component

Disorders assoc w/ CNV
- AMD
- Ocular Histoplasmosis
- High Myopia
- Idiopathic
- Others:
  - Inflammatory, angioid streaks, etc
Diagnosis: Pathologic Myopia

- Myopic Degeneration

- What are the questions that you ask yourself when examining the retina of a patient with high myopia?
- What should you be specifically looking for?

Myopic Degeneration

- Peripapillary scleral crescent
- Tilted optic nerve
- Thinning of the sensory retina/RPE
- Staphyloma
- Fuch’s spot
- Lacquer cracks
- CNV
- Peripheral retinal degeneration
- Retinal Detachment

Myopic Degeneration

- Do you see any gray/green changes that could be consistent with CNV?
- Is there fluid in the macula?
  - Subretinal hemorrhage or exudate?
- Is the macula flat or is there any elevation?

CNV & Degenerative Myopia

- 5-10% with high myopia, axial length > 26.5 mm
- Incidence of CNV w/ high myopia 40.7%
  - Hotchkiss/Fine AJO 1981
- Bilateral CNV or Fuch’s spot
  - 12% Hotchkiss/Fine
  - 18% Curtin (Arch Ophthalmol 1963)
  - 24% Fuchs
  - 28% Campos

CNV & Degenerative Myopia

- Typically Type II – grow under the retina
- Up to 74% subfoveal
- < Incidence w/ ↑ post staphyloma
  - Suggests may need preserved CC for CNV to develop
- Exhibits different dynamics w/ age
  - Less leakage in younger patients
  - More leakage in older patients

51 yo Hisp Female

Wants to change her CL’S

51 yo Hisp Female

Wants to change her CL’S