MY FAVORITE MACULAE

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OptoWest 2016
California Optometric Association – Newport Beach

OPTICAL COHERENCE TOMOGRAPHY IMAGE / DATA ACQUISITION

- Time domain
  - A-scans make up the B-scan that is represented as X-sect sampling
  - Resolution approaching that of excisional biopsy and histopathology (~ 10 microns)

- Fourier or Spectral domain
  - Resolution 3-5 microns

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PVD W/ CONTINUED MACULAR TRACTION – CLINICAL TRANSLATION

- Fourier or Spectral domain
  - Resolution 3-5 microns
  - Volumetric data enables non-invasive depth-resolved functional imaging
PVD W/ CONTINUED MACULAR TRACTION – CLINICAL TRANSLATION

FIRST, LET’S REVISIT

Segment (x-sect) is one meridian of the cube of information (total data gathered).

NOTE THE PMB & THE “BLIP” @ PHOTORECEPTOR INTERFACE (LINE), PIL

Observe drusen

Case courtesy Diana Shechtman, OD

63 F C/O ↓ VA; 20/30

TD OCT

Normal foveal contour
Image RPE

SD OCT

How can this be the same eye?
Significant PVD
Intact IS/OS line (“P I L”)
Note visibility of drusen
VIT-MAC INTERFACE AND PVD AS THE ROOT OF ALL EVIL

55 BF presents for follow-up (x 4 mo.) [Macular hole]

VA 20/200 OD
20/25 OS

The left eye appears to be uninvolved

VA 20/200 OD
20/25 OS

GP 56 BF
The LEFT eye is normal except for vitreo-macular traction.

Note VMT

3D CUBE OD

POST-OP OD
Note:
• relatively normal macular contour & thickness
• But absence of PRL

MANAGEMENT AND FOLLOW-UP
• Visit of 14 March 2012
• VA
  • 20/80 OD!
  • 20/25 OS – no change in OCT
• Further update: seen 6/19/2012
• Scheduled for mac hole repair (OD)

GP: S/P VITRECTOMY, IOL (OD)
(9/18/12); VA = 20/400

High-definition images
Note absence of photoreceptor layer, but intact ELM
GP: S/P VITRECTOMY, IOL (OD) (9/18/12); VA = 20/400

High-definition images

Note absence of photoreceptor layer, but intact ELM

9/2015 VA: 20/40

HIGH-DEFINITION IMAGES

Fellow eye with remaining VMA but no retinal defect.

Contrast this to the next case with lamellar macular hole.

COMPARE THE PREVIOUS TO THIS CASE

65 WF

Referred for second opinion

VA 20/20- and 20/20

WHAT DOES THE OCT DEMONSTRATE?

HIGH-DEFINITION IMAGE SHOWS INTACT/CONTINUOUS OUTER RETINA AND PHOTORECEPTOR LAYER

NOTE: THICKENING @ ILM
COMPARE WITH UNINVOLVED OS
ABSENCE OF VITREOUS TRACTION & INTACT OUTER RETINAL LAYERS

OPTOMETRY TIMES DECEMBER 2013
- 51 WF with distorted VA X 3 Days
- 20/60 OD, 20/25 OS
- Non-contributory histories ...
Enzymatic Vitreolysis with Ocriplasmin for Vitreomacular Traction and Macular Holes

Peter Stalmans, M.D., Ph.D., Matthew S. Ritter, M.D., Arnd Gandorfer, M.D., Anselm Kumpf, M.D., Anja Graech, M.D., Stephen Pálás, M.D., and Julia A. Haller, M.D., for the MIVI-TRUST Study Group.

Resolution of VMT following intravitreal injection of ocriplasmin (1st endpoint).
(Follow the solid lines and note that "spontaneous" resolution occurred in about 12% of cases.)

61 B/M
- 12/02
- Followed X 12 years - Angioid Streaks
- 2° A1 Hemoglobinopathy
- VA 20/40

*Angioid streaks: PEPSI T
61 B/M
- 12/02
- Followed X 12 years - Angioid Streaks 2° A1 Hemoglobinopathy
- VA 20/25 (OS) – note less involvement

62 B/M (10/03)
- AS now symptomatic
- VA 20/30 (OD) note increased involvement
- RTC 4-5 mo.

11/03 – COMPARE RF AND CLINICAL (3 WEEKS LATER)

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11/03 – COMPARE RF AND CLINICAL (3 WEEKS LATER)
ANGIOID STREAKS - CNVM

- Late phase leakage consistent with CNVM
- Verteporfin

63 B/M (9/04)
- VA 20/80 (OD) note significant clinical picture consistent with remodeling following Verteporfin treatment

61 B/M (9/04)
- RF
- Note poor perfusion
- Lack of leakage indicating stable macula

48 SEC.
- Note lack of leakage (stabilized CNVM)

9:56 SEC
- Late-phase staining
- No leakage
OD – S/P VERTIPORFIN TREATMENT
VA = 20/200  7/08
VA = 20/200  8/09

OS – A DIFFERENT STORY
7/08 – sub macular fluid
VA = 20/80
Treatment with Avastin

OS 8/09
S/P 3 Avastin injections
VA = 20/40 with GA; flat macula

ANGIOID

CASE:  39 W/M
- Initial presentation:
  - Desires second opinion on treatment for retinal problem
  - History: taking clindamycin X 6 weeks PO
  - steroids PO X 3 weeks (D/C)
  - VA (OD) 20/200; mild vitritis, no A/C rxn, all else unremarkable; OS uninvolved

Questions
Comments
1/22/16

39 W/M
- OD Fundus appearance @ initial presentation

39 W/M
- Initial treatment
  - *Bactrim DS* (160 mg. Trimethoprim + 800 mg. Sulfmethoxizole) PO bid X 2 weeks
  - RTC X 2 wks

CASE: 39 W/M
- Fundus appearance at discharge visit (8 weeks following initial presentation): Note retinal vessel communicating with choroidal circulation (karyolysis retinalis)

39 W/M
- 2-week return visit (fundus appearance)
  - VA unchanged
  - Vitritis slightly diminished
  - Fundus appearance essentially unchanged
  - Options
    - Continue meds???
    - Change meds???
    - Add meds???
    - Refer???

39 W/M
- 4-week return visit
  - VA unchanged
  - Vitritis resolved; 1+ A/C reaction
  - Fundus appearance essentially unchanged
  - Options
    - Continue meds???
    - Change meds???
    - Add meds???
    - Refer???

Current recommended treatment when offered.
Lima GSC, Saravia PGC, Saravia FP. Current therapy of acquired ocular toxoplasmosis: a review. JOP 2015 (Sep) 811-817.
TREATMENT INDICATIONS
1. Involvement within the macular arcades
2. Proximity of lesion to optic nerve
3. Relative indications
   - Infection in immunocompromised patients
   - Marked vitreous reaction (posterior uveitis)
   - Active lesion in an only eye
   - Presentation in an elderly patient (shown to be more aggressive)

ADDITIONAL FEATURES
1. Punctate outer retinal toxoplasmosis
2. Retinal vasculitis
3. Retinal vascular occlusions
4. Rhegmatogenous RD with serous RD
5. Uveal pigmentary changes simulating RP
6. Neuroretinitis and other forms of optic neuropathy
7. Peripheral retinitis and scleritis
8. In children:
   - Cataract, CNV, glaucoma, RD
   - Has been associated with Fuch’s heterochromic iridocyclitis

OCT FEATURES OF TOXOPLASMIC RETINOCHOROIDITIS
- Reflective inner retina in active presentations
- Posterior hyaloid thickened and detached over the lesion
- Shadowing of the underlying choroid
- May have serous fluid

MANAGEMENT OF OCULAR TOXOPLASMOSIS
1. Clinical Presentation / Diagnosis
   - Observation of a yellowish lesion with overlying inflammatory cells is almost diagnostic
   - Vitritis/choroiditis may accompany
   - Blood tests are definitive (systemic) but not in ocular
     - Sabin-Feldman methylene blue dye test, IgG, IgM, ELISA
   - Treatment is indicated when the posterior pole is involved

OCULAR TOXOPLASMOSIS
- 30% - 50% of all cases of posterior uveitis
- Clinical presentation (same for cong. and acq.)
  - Focus of necrotizing retinitis
  - Moderate to severe vitritis
  - In immunocompromised patients, there may be multiple foci or extensive necrosis

OTHER TOXOPLASMIC RETINOCHOROIDITIS RISKS
- Eating locally produced cured, dried, or smoked meat
- Working with meat
- Drinking unpasteurized goat’s milk
- Having 3 or more kittens
- Eating raw oysters, clams, or mussels was significant in a separate model among persons asked this question.
T. GONDII (MULTIPLE PATHWAY RISKS)

POST-INFLAMMATORY LESIONS

POST-INFLAMMATORY LESIONS ("CONGENITAL TOXO")

INACTIVE RETINOCHOROIDITIS

58 B/M presents with near complaints

VA – 20/20
RECURRENTS (RISK)

- While risk appears to decline with increasing time from most recent episode, risk may be accelerated by increasing age (interactive factors).


PROPHYLAXIS?

- Current guidance

QUIZ TIME

16 W/M
- Slight decrease in VA recently
- VA = 20/40 (OS), DK = ?
- Dx = ?

Active lesions are white and fluffy with vitritis. (a controversial indication to all orlta prednisone)

OCULAR TOXOPLASMOSIS

- Questions
- Comments

46 W F (CASE SHARED BY DR. H. BENAVIDES)

- C/O “central blindness” both eyes
- Released from hospital
  - Acute pancreatitis
  - Dyslipidemia
  - Uncontrolled systemic hypertension
  - Suspected to be 2° to alcohol abuse

46 W F “CENTRAL BLINDNESS”

- BSCVA: 20/25(OD) 20/30 (OS) [EV]
- Pertinent lab results
  - Triglycerides > 4000
  - CBC w/diff (EKG, CRP) (WNL)
  - Fundus showed cotton wool spots
• The optic discs are unaffected
• The foveal reflex is absent
• There is evidence of disruption at the level of the nerve fiber layer that is more evident in the right eye

Red-free images of the right and left eyes enhance infarcts of the nerve fiber layer.

FA study – Red Free

No leakage at 47 sec
Note: patchy choroidal filling
No leakage from retinal or choroidal vessels at 1:03 and 3:21
Patchy choroidal filling still evident

No late leakage in the right eye, either

46 WF WITH DECREASED CENTRAL VA AND ABNORMAL CHOROIDAL FILLING

Purtscher Retinopathy
PubMed search: "visual field defect," "cotton wool spots"

Meyer CH, Callizo J, Schmidt JC, Mennel S.
www.pubmed.gov

PURTSCHER RETINOPATHY

Initially associated with head trauma and crush injuries

Current thinking
Micro circulatory defects secondary to endothelial cell damage (ischemic macula)

SOME CASES ARE ASSOCIATED WITH TRAUMA

20 F ejected from automobile.

DCT of the right macula. Thickening of the inner nasal retina correlates with the location of cotton wool spots. Additional findings include shallow subretinal fluid and discontinuity of the photoreceptor layers.

Fluorescein angiogram of the right eye at 22 seconds. Peripapillary hypofluorescence and an enlarged foveal avascular zone are observed.
**Table 1: Patient characteristics**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Predisposing factors</th>
<th>Presenting features</th>
<th>Initial visual acuity</th>
<th>Final visual acuity</th>
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<tr>
<td>1</td>
<td>64</td>
<td>F</td>
<td>Acute pancreatitis</td>
<td>BCVA 20/20 right</td>
<td>20/20</td>
<td>20/20</td>
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<tr>
<td>2</td>
<td>58</td>
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<td>Acute pancreatitis</td>
<td>BCVA 20/20 left</td>
<td>20/20</td>
<td>20/20</td>
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<tr>
<td>3</td>
<td>55</td>
<td>M</td>
<td>Acute pancreatitis</td>
<td>BCVA 20/20 right</td>
<td>20/20</td>
<td>20/20</td>
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<tr>
<td>4</td>
<td>50</td>
<td>M</td>
<td>Chronic pancreatitis</td>
<td>BCVA 20/20 left</td>
<td>20/20</td>
<td>20/20</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
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<td>20/20</td>
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<tr>
<td>6</td>
<td>48</td>
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<td>20/20</td>
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<tr>
<td>7</td>
<td>52</td>
<td>M</td>
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<td>BCVA 20/20 right</td>
<td>20/20</td>
<td>20/20</td>
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<td>8</td>
<td>70</td>
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<td>20/20</td>
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<td>10</td>
<td>47</td>
<td>F</td>
<td>RTA - long bone fracture</td>
<td>BCVA 20/20 left</td>
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<td>20/20</td>
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<tr>
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<td>20/20</td>
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<tr>
<td>12</td>
<td>49</td>
<td>F</td>
<td>RTA - long bone fracture</td>
<td>BCVA 20/20 left</td>
<td>20/20</td>
<td>20/20</td>
</tr>
<tr>
<td>13</td>
<td>66</td>
<td>M</td>
<td>RTA - long bone fracture</td>
<td>BCVA 20/20 right</td>
<td>20/20</td>
<td>20/20</td>
</tr>
<tr>
<td>14</td>
<td>25</td>
<td>M</td>
<td>Road traffic accident</td>
<td>BCVA 20/20 left</td>
<td>20/20</td>
<td>20/20</td>
</tr>
<tr>
<td>15</td>
<td>37</td>
<td>M</td>
<td>Road traffic accident</td>
<td>BCVA 20/20 right</td>
<td>20/20</td>
<td>20/20</td>
</tr>
</tbody>
</table>

**PURTSCHER RETINOPATHY**

- **Questions**
- **Comments**

**46 WF PURTSCHER RETINOPATHY**

- Within 3 weeks VA 20/20 (OD) and 20/25 (OS)
- Fundus picture improved
- Medications include:
  - Diovan 160 mg po qd (Valsartan and Hydrochlorothiazide) for HBP
  - Pravachol 40 mg po qd
  - Zosert 80 mg po ad
  - Librax 2 tab po qhs (Librium (chlordiazepoxide hydrochloride) + the anticholinergic/spasmolytic effects of Quarzan (clidinium bromide))

**PL: HX: CSR (OS, 20/30) X 30 YRS; NOW MAC HOLE AND GA (1998)**

20/60

**OD (2012)**

20/20
LET'S LOOK AT AN ACUTE CASE

46 ASIAN MALE

- "blurry vision" 11/20/2012
- X 3 mo OS; began only last night OD
-Began new BP med last week
-Has never had eye exam
-Central blur in OS has improved somewhat
-+ floaters X 1 yr
- - flashes, discharge, pain

Previous ocular history is negative for refractive correction, injury, glaucoma, cataract, strabismus, amblyopia, etc.
-Family medical / ocular histories negative
-No known allergies
-Began lisinopril qD X 1 wk. [ACE inhibitor]
-BP 150/100

46 ASIAN MALE

-VA 20/40-20/400 (PHNI)
-RAPD
-IOP: 14/14
-No EOM restrictions
-Confrontation FTFC OD, OS
- -1.50 / -2.25 -0.50 X 070 VA NI
Anterior segment unremarkable OD, OS

11/20/12
46 A M WITH CSR, HR

- Initiated Nevanac bid (11/20/12)
- RTC X 1 wk
- Correspond with PCP

- @ 1-wk F/U (11/27/12)
  - BP = 138/92
  - VA 20/25 - 20/40 !!!
    - (-1.00 / -0.75 – 0.50 X 070)
  - Continue Nevanac bid

46 A M WITH CSR, HR

- Initiated Nevanac bid (11/20/12)
- @ 2-wk F/U (12/4/12)
  - BP = 140/92
  - VA 20/20 - 20/20 !!!
    - (refraction unchanged)
  - Continue Nevanac bid
  - RTC X 1 Wk
86 YO

- Presents with reduced VA OS
- POH: repaired peripheral retinal hole SN OS X 11 yrs
- Pseudophakic in each eye
- Medicated for HTn X 20 yrs

CSR

- Questions
- Comments
WHAT'S THE DIAGNOSIS AND MANAGEMENT?

Note retinal thickening with intact RPE.

Caliper to measure retinal thickness = 462 μ.

Note retinal thickening temporal + inferior to macula.
**RETINAL ANGIOMATOUS PROLIFERATION**

- Aka Type III neovascularization
- Management
  - Avastin injection ⇒ 20/40 @ 3 weeks

Neovascularization
- Type I – CNVM
- Type II – retinal teleangiectasia

**Management**

- Avastin injection

Neovascularization
- Type I – CNVM
- Type II – retinal teleangiectasia

**04/09/2008**

S/P 1 Avastin injection

**Note retinal thickness response**

276 μ

**ANOTHER EXAMPLE (STAGE II, W/PED)**

A. RAP with hyperfluorescent PED (arrows)
B. ICGA with RAP hot spot, and hypo fluorescent PED
C. OCT: intraretinal RAP (arrow), cystic spaces and PED
D. Another RAP with PED
E. Arrow indicates intraretinal lesion; cystic spaces overlay PED

A. ICGA showing communication among intraretinal, subretinal and choroidal NV.
B. OCT shows choroidal invasion into subretinal space; RAP not well defined.


A. RAP (note drusen)
B. OCT suggesting choroidal invasion of subretinal space


Questions
Comments

AMD PROGRESSION IN A LOW-RISK PATIENT
- TX W/F
- 20/25 OD
- Baseline

AMD PROGRESSION IN A LOW-RISK PATIENT X 2 YEARS
- TX W/F
- 20/40
- Receives Avastin OD – watch drusen
AMD PROGRESSION IN A LOW-RISK PATIENT X 3 YEARS

- TX W/F
- 20/40 OD
- S/P Avastin
- Note: drusen diminution & beginning GA

AMD PROGRESSION IN A LOW-RISK PATIENT

- TX W/F
- 20/25 OS
- Baseline

AMD PROGRESSION IN A LOW-RISK PATIENT

- TX W/F
- 20/25 OS
- S/P Avastin

DEALING WITH AMD

What interventions were available at the time to possibly alter the natural history of this AMD? Current regimen: Centrum Silver + 5 mg Lutein

Continue vitamin supplements
RTC X 1 year
01/09/2008 20/60
Positive Amsler (wavy lines temporal and inferior OD)
Continue vitamin supplements
RTC X 1 year

04/14/2009:
"I woke up in the middle of the night and I couldn’t see the middle number on the digital clock with my right eye."
20/40

MANAGEMENT & FOLLOW-UP

- Retinal consult for CNVM
- Avastin injection same day
- 05/09 2009
  - VA 20/60, 20/50 stable macula
  - Follow X 3 months

FAST FORWARD TO 3/21/13

S/P numerous IV Avastin injections
Could the conversion to WAMD have been averted?

Note spontaneous release of VMA

Table 1. Five-Year Rate of Developing Advanced AMD in AREDS Participants by Drusen Size and Degree of Pigmentary Abnormalities

<table>
<thead>
<tr>
<th>Drusen Size</th>
<th>Pigmentary Abnormalities</th>
<th>Pigmentary Abnormalities</th>
<th>Pigmentary Abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>None or small</td>
<td>4.6% (42/917)</td>
<td>9% (204)</td>
<td>12.3% (413)</td>
</tr>
<tr>
<td>Intermediate</td>
<td>2.3% (4/49)</td>
<td>5.6% (251)</td>
<td>12.8% (671)</td>
</tr>
<tr>
<td>Large</td>
<td>2.4% (4/177)</td>
<td>12% (225)</td>
<td>29.2% (255)</td>
</tr>
<tr>
<td>Large, one</td>
<td>9.3% (78/831)</td>
<td>10.1% (177/1761)</td>
<td>25.4% (197/773)</td>
</tr>
<tr>
<td>Large, both</td>
<td>1.3% (17/276)</td>
<td>17.8% (501)</td>
<td>47.3% (2102)</td>
</tr>
</tbody>
</table>

AMD = age-related macular degeneration. AREDS = Age-Related Eye Disease Study.


Figure 3. Graph showing the 5-year risk of developing late AMD for various risk groups. AREDS = Age-Related Eye Disease Study.

Figure 4. Graph showing the 10-year risk of developing advanced age-related macular degeneration (AMD) in eyes without large drusen or hard drusen for various risk groups.

FERRIS FL, ET AL. OPHTHALMOLOGY. 2013 APR;120(4):844-51

CAN'T FORGET ABOUT MACULAR EDEMA

BORN: 7 APRIL 1957 (S.T.)
- First seen 19 April 2012
- DIABETIC (insulin) / HTN X 20 yrs (2 meds)
- BS: 140-200; A1C is unknown

19 APRIL 2012 VA 20/25 OD = OS
- Note cotton wool spots
  - Esp.
- Note: macula is definable

LEFT EYE HAS CWS AS WELL

RIGHT EYE
- Normal macular contour
- No thickening centrally
LEFT EYE
Normal macular contour
No fluid or thickening

OD
Note mild retinal thickening outside the macula but absence of fluid

OS
Note mild retinal thickening (consistent w/CWS) surrounding macula but absence of fluid at the macula

HIGH-DEFINITION IMAGES
Mild retinal thickening without fluid accumulation

Each macula shows mild thickening
Note RNFL profile comparing OD and OS to normal
Overly thick RNFL = CWS areas
SUBSEQUENT VISITS

- VA 1 June 2012  No OCT or photos
  20/20-  20/40

- VA 21 June 2012  No OCT or photos
  20/25  20/25

Recommend retina specialist consult;
Treatment recommended; Pt. refused

17 JULY 2012  VA = 20/20  20/25 [DIFFUSE DME]
ST RNFL DEFECT

2 OCTOBER 2012 CSME  20/50
(REDUCED FROM 20/25 IN APRIL 2012)

RNFL defect
(not glaucoma)

And “muddy macula”

Left eye appears less involved
20/30

Except nasally . . .

Note NV and Pre-retinal heme
Nasal to ONH

Note fluid IT to macula = CSME
Macular thickening
Similar pattern but not as severe fluid accumulation OS
Macular thickening corresponds to CWS thickening

OS Macular thickening corresponds to CWS thickening

High definition images show CWS and macular fluid / thickening consistent with CSME (OD)
High definition images show CWS and macular fluid / thickening consistent with CSME (OD)

RNFL DEFECT FOLLOWING CWS APPEARANCE IN HYPERTENSION

45 F
- VA = 20/20
- Normal history
- Baseline photo 2000

52 W F
- Sudden onset of reduced VA (X 7 ½ yrs)
- 20/80 w/central disturbance
- What are you going to do?

*MOST PREVALENT COAGULATION AND ANTI-COAGULATION DISORDERS IN BRVO

- Resistance to activated protein C (especially factor V Leiden mutation)
- Protein C or protein S deficiency
- Deficiency of antithrombin III
- Genetic mutation in the prothrombin (factor II) gene
- Anti-phospholipid antibodies
- Hyperhomocysteinemia

9/ 4/ 2008
Involvement confined to the inner retina

9/ 9/ 2008
Cystoid macular edema; Started on Xibrom (bromfenac) qid

9/ 22/ 2008
VA 20/200; distinct macular involvement; Now what?
1/14/2009
Continued on Xibrom qid
Some resolution

1/19/2009
Continued on Xibrom qid

2/17/2009
Continued on Xibrom qid

2/16/2009
Uninvolved OS
Recommend anti-VEGF intravitreal injection
BUT WAIT, THERE'S MORE

VF 2/24/2009
And an Avastin injection
VA = 20/25!!!
Restoration of normal anatomy

*TREATMENTS FOR ME FOLLOWING RVO

- **SCORE 5**
  - CRVO - standard care = observation
  - Neither 1 mg nor 4 mg IVTA offered better outcome


- **SCORE 6**
  - BRVO - standard care = grid photocoagulation
  - Both 1 mg and 4 mg IVTA showed 15-letter gains in ~ 25% of eyes @ 12 mo.
  - Fewer IOP elevations and cataract in the lower dose

- **BUT WAIT! THERE'S MORE!!!**

CRUISE
- CRVO intervention for CME trial
  - 0.3 or 0.5 mg intravitreal ranimizubab (Lucentis)
  - 46.2 and 47.7% of eyes gained ≥15 letters @ 6 mo. (1.1 in the sham group)
  - Fewer IOP elevations and cataract in the lower dose

BRavo
- BRVO intervention for CME (same dosing as CRUISE)
  - 55.2 and 61.1% of eyes demonstrated ≥15 letters @ 6 mo. (1.9 in the sham group)

Retina Congress September 2009 NYC

FDA-APPROVED JUNE 27, 2010

For the treatment of CME secondary to BRVO/CRVO

Questions
Comments
Thank you, California Optometric Association and attendees!