That's Not Glaucoma:
Tales from a referral center
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Course Objectives
- better understand the “new” definition of glaucoma
- list and ID the characteristics of glaucoma
- Differentiate glaucomatous fields from neurological fields and other types of VF loss
- Identify the classic findings of the glaucomatous optic disc
- Become better adept at differentiating glaucomatous optic nerves from other ON pathologies/abnormalities

To differentiate what is not glaucoma one must first have a firm grasp of what glaucoma is…

Glaucoma Defined
A multi-factorial optic neuropathy characterized by loss of retinal ganglion cells and optic atrophy, resulting in visual field defects and changes in the optic nerve.
No IOP criterion

Hallmark Findings in glaucoma
- IOP elevation
- The Glaucomatous disc
- VF loss
- NFL loss

IOP: What is normal really?
- Normal IOP is not a number
- Could be defined as that pressure which does not lead to glaucomatous optic atrophy
  - All eyes do not respond the same to given pressure levels
- Numerous studies on measuring IOP
  - All show normal mean IOP ranges from 15-17 mm Hg
  - I tell patients: can be normal from 8-21 mm Hg
Ocular Hypertension Treatment Study (OHTS)

OHTS: What have We learned?

Ocular HTN is Defined As:
- IOP > 21 (AAO); IOP > 24 (OHTS)
- Without VF Defect
- Without ONH Abnormalities
- Without A/C Angle Abnormalities

OHTS

Mission:
- Evaluate the safety and efficacy of topical ocular hypotensive medication in delaying or preventing the development of primary open-angle glaucoma (POAG) in individuals with elevated IOP
- Identify baseline demographic and clinical factors that predict which participants will develop POAG

The OHTS Entry Criteria
- Aged 40 to 80 years
- Normal visual fields
  - Humphrey 30-2
- Normal optic discs
- Untreated IOP:
  - 24 to 32 mmHg in qualifying eye
  - 21 to 32 mmHg fellow eye

Summary
- Treatment produced about a 20% reduction in IOP.
- Treatment reduced incidence of POAG in OHTS participants by more than 50% at 5 years from 9.5% in the observation group to 4.4% in the medication group.
- Little evidence of safety concerns with Tx.

OHTS

Significant Baseline Predictive Factors
- Corneal thickness was the single most powerful predictor of progression in the OHTS.

*Dr. Heuer (OCHS investigator)*
Corneal Thickness (CCT)

- < 545 um greater risk
- > 545 um less risk

* 2.5 mm correction factor for every 50 um change in CCT (Doughty and Zaman survey, Ophthalmology 2000)

IOP Conversion

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<th>Corneal Thickness (µm)</th>
<th>Correction Value</th>
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Correction values depending on corneal thickness of 545 µm.

Misclassification Concerns

- This will have significant implications on everyday practice.
  - Misclassification based on measured IOP
  - Many may have inadequately controlled IOP
  - Many OHT’s may be over-treated
  - May mean we will set even lower TP’s
OHTS

Summary
• Not every patient with OH should be treated
• Consider measuring corneal thickness in all patients with OH or glaucoma
• Recommend treatment to OH patient at moderate to high risk, taking into consideration:
  ▫ Age
  ▫ Medical Status
  ▫ Life Expectancy
  ▫ Likely Treatment Benefit

LTG/NTG: Does it exist?
• Leaders in the field are not in total agreement!
• Most consider those with glaucomatous optic neuropathy with IOP never higher than 21 NTG
  ▫ Although IOP has significant influence on progression
  ▫ 30% reduction in IOP is assoc. with protection of VF and nerve status
• Other factors involved besides IOP

The Glaucomatous Disc

• rim thinning/notching
• drance hemes
• baring of the circumlinear vessels
• nasalization of vessels
• laminar baring

VF loss in glaucoma

OCT/ NFL in glaucoma

NFL analysis
Don’t get blinded by the Science
The most common things that will lead you to misdiagnosis………

- Abnormal disc appearance: pale, oblique/tilted, or otherwise anomalous disc
- Unexplained VF abnormalities
- #1 NFL abnormalities (GDx, OCT, HRT)
  - Don’t put all your eggs in that basket!

Advanced Optic nerve Assessment

Optic Disc assessment
(The Five R’s)

Five Rules for Assessment of the Optic Disc in Glaucoma

1. Observe the scleral ring to identify the limits of the optic disc and its size
Five Rules for Assessment of the Optic Disc in Glaucoma

1. Observe the scleral Ring to identify the limits of the optic disc and its size.
2. Identify the size of the Rim.
3. Examine the Retinal nerve fiber layer.
4. Examine the Region of parapapillary atrophy.
5. Look for Retinal and optic disc hemorrhages.

Rule #1
Observe the scleral Ring to identify the limits and the size of the optic disc.
Optic Disc Size

Measurement of optic disc size with direct ophthalmoscope

Small aperture (5 degree) of Welch-Allen direct ophthalmoscope

Size of light spot ~ size of average optic disc

Optic Disc Size

Measurement of optic disc size with biomicroscopy

Volk lens
Measure length of slit beam

Correction factors
Volk 60D – x 1.0
Volk 78D – x 1.1
Volk 90D – x 1.3

Avg vertical diameter: 1.8 mm
Avg horizontal diameter: 1.7 mm

Optic Disc Size

Size of cup varies with size of disc
Large discs have large cups in healthy eyes

Small disc: avg vertical diameter <1.5 mm
Large disc: avg vertical diameter >2.2 mm

Optic Disc Size

Small discs with glaucoma may have small cups

Rule #2
Identify the size of the neuroretinal Rim

Be cautious with myopic discs
**ISNT RULE**

Rim width
Distance between border of disc and position of blood vessel bending

ISNT rule
Inferior > Superior > Nasal > Temporal

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**Localized Rim Thinning/Notching**

Notching

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**Observe the color of the rim to identify pallor**

A pale rim increases the likelihood for a non-glaucomatous optic neuropathy

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**Pallor**

Diffuse pallor

Pallor > cup
Non-glaucomatous neuropathy

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**Rule # 3**

Examine the Retinal nerve fiber layer (RNFL)**
**RNFL Examination**

- Best performed using red-free light (red-free photographs or green light)
- Look at:
  - Striations
  - Brightness
  - Visibility of parapapillary retinal vessels
- Look for **diffuse** and **localized** RNFL loss

**RNFL Red-Free Photographs**

**Diffuse RNFL Loss**

- Diffuse loss of striate pattern + increased vialility of retinal vessel borders

**Localized RNFL Loss**

- Localized RNFL defect
- Wedge-shaped dark area

**Rule # 4**

*Examine the Region of parapapillary atrophy (PPA)*
Parapapillary Atrophy

**Alpha zone**
- Hypo- and hyper-pigmented areas
- Present in normal as well as in glaucomatous eyes

**Beta zone**
- Atrophy of the retinal pigment epithelium (RPE) and choriocapillaris
  - Large choroidal vessels become visible
  - More common in glaucomatous eyes

**Rule # 5**
*Look for Retinal and optic disc hemorrhages*

Optic Disc Hemorrhage

Indicative of glaucoma progression

**Optic Disc Hemorrhage**

Normally disappears after 2-6 months

Detection of disc hemorrhages requires careful optic disc examination
Theories of ONH Damage

### Mechanical Theory
- Abnormal levels of IOP cause direct damage on ON
- Compression of laminar sheets
- Distortion of laminar pores
- Crimp or pinch axons of GC causing blockage of axonal transport $\rightarrow$ apoptosis
- In LTG, if not purely due to vascular insufficiency, ON tissue must have some connective tissue abnormality/susceptibility

### Vascular Theory
- Assumes a decrease in blood flow in lamina cribrosa blocks axoplasmic flow by reducing energy available to keep system operational.
- Increase IOP causes a reduction of blood flow in intraocular vessels and prelaminar ONH
- True only of choroidal circulation
- Retinal circulation has “auto regulatory mechanisms”
- Auto-regulation of blood flow for blood vessels supplying ON (may be lacking)
  * Evidence - dramatic improvement of blood flow following adequate lowering of IOP.
The pale disc: (What glaucoma is not)

Differential Diagnosis
1. Compression
2. Ischemia
3. Disc Drusen
4. Disc anomalies

Clinical Findings/ Intro case
- 74/AA/Female referred in for floaters OS x 4 days.
- (-) decreased VA painless small spots comes and goes
- Med Hx: (+) HTN (-) DM (-) CVA
- Meds: Loratadine, Clonidine HCL, Benicar
- OCHx; (-) prior Hx glaucoma (-) trauma (-) injury
- (-) blood loss, hypotensive episodes, transfusions, Reynaud’s, migranes

Clinical Findings
- DVA cc OD 20/25 OS 20/20
- Color NML
- Pupils (-) APD
- Ext. Nml
- SLE; 1+ cs/2+ ns OU
- Ta OD 20 OS 20 @ 3:22 pm

Clinical Findings
- DFE: Vitreous syneresis OU, macula Cl OU ; Optic nerve; FPH OD; heme off nerve OS, C/D ratio .60/.80 OD OS .55/.60 vessels attenuated
- Periphery: (-) Tears (-) Holes (-) RD’s

Optic nerves: Initial visit

Diagnosis and Plan
- Diagnosis
  - COAG Suspect
  - HTR O.U.
  - IR Heme OS HTN vs Drance Heme
- Plan
  - Ordered blood work-up, ESR, CRP, CBC with diff.
  - Fasting Blood Glucose, HgA1C
  - Check HTN, BP with PCP, photo document
  - Order Carotid Duplex Study
Testing: Gonio / Pach

- PACH: OD 518 OS 521
- Gonio: open grade 3-4 360° OU plateau Iris OU

Visual Fields/ initial visit

Visual Fields / at Follow up

Case # 1 TM

- 51/AA/F seen in consultation for glaucoma / 2nd opinion
- Patient using Cosopt for years in OS BID
- Documented VF defect OS only since 11/04
- Non-progressive
Key questions to ask your glaucoma patients

- History of blood loss / i.e. surgery, MVA, pregnancy / delivery
- Blood transfusions
- Hypotensive episodes or crisis
- Migraines
- Reynaud's Phenomenon
- Ocular trauma / blunt

Medical hx.

- Med Hx: (-) DM, elevated cholesterol, (-) HTN, (-) MS, (+) history of epilepsy / blacked out two times in 2002, diagnosed as seizures, (+) Hx. hypothyroid
- Fibroids removed, no transfusions, or significant blood loss per patient
- Oc. Hx: treatment for “low tension glaucoma” for years OS, Cosopt BID OS
- Pretreatment IOP 11 OD 10 OS (-) injury, surgery
- Possible family hx. of glaucoma in (PGM)

Clinical findings / Case 1

- DVA cc: OD 20/20 OS 20/20
- Pupils: trace APD OS, color vision nml OU
- SLE: Cl cornea OU, (-) endopigment, A/C deep and quiet (-) PX, iris nml OU (-) TID and NVI. Lens trace CS/NS OU.
- Ta @ 10:01 OD 12 OS 12

Optic nerve photos / case 1 Tm

- Vitreous clear OU, macula clear, optic nerve enlarged cups with temporal sloping OU, C/D ratio .65 round OD, .70/.60 OS with pale rim from 9-5:00, Large ON Soc OU
- Vessels mild attenuation
- Periphery clear OU
TESTING: VF’s and Gdx/ NFL

- VF’s show a full and normal OD, OS inferior altitudinal VF loss
- Gdx. NFL analysis: Thin superior NFL OS

What do we have, let’s add it up!

- Large nerves with large cups
- Pale nerve OS, never worked up!
- Low IOP, on and off meds
- VF defect, long standing inferior altitudinal

Testing ordered

- CBC with Diff, ESR, CRP, ANA, ACE, Serum Lysozyme, clotting factors, serum protein S and C
- Carotid Doppler/ Duplex study
- Echo cardiogram

Testing results

- All normal; clean carotids and echo.
- Dx.: Ischemic event of unknown etiology vs, possible old MS?
- Plan: D/C Cosopt
  - F/u visits (3) show IOP’s between 09 and 13 OU
- Ordered MRI of head and orbits R/O compression/MS…….Normal
Follow-up visits
IOP has been 8-10 on three visits OS
off meds

Case # 2 MP
- Ocular Hx.: Possible AION OS, glaucoma suspect (poor compliance), no injury, cataract OU
  - Pretreatment pressures of 27 OD/OS
    - Previously treated with Lumigan, and Cosopt
    - Hx. Of non-compliance
    - Surgery; ALT OS 02/10/04, OD 03/05/04

Case # 2 MP: Clinical Exam (4/22/09)
- DVA cc OD 20/40 OS HM @ 3Ft.
- Pupils: 1+ APD OS; 50% desaturation to bright light
- External: NML
- SLE: microcystic degeneration, peripheral cornea OU, (-) endopigment. A/C (-) PX OU deep and quiet
  - Lens 2+ CS/NS OU

Case # 2 MP / 73yo Asian male
- Referred by PCP for reduced VA OS> OD progressive
- Previously Dx. of COAG and NAION OS
- Referred to PCP for "vascular w/up" 2002
- Patient was started on Xalatan therapy (09/19/05) but stopped med, lost to follow-up till (04/22/09).
- Medical HX.: +MI, +HTN, + elevated cholesterol
  - Meds: Flomax, HCTZ, amlodipine (Norvasc), Trazodone,
  - Bypass surgery in 2000
  - Intracranial surgery in 2008 (tumor removed)

Case # 2 MP: Clinical Exam
- DFE: Vitreous Cl OU, macula CL OU, vessels HTR I
  - Optic Nerves: large cupping .80/.70 OD, .85/.80 OS with pale temporal rim OU. (See Photo)
  - Periphery Cl OU, (-) tears, holes, detachments

Optic Nerve Presentation
- Ordered VF OD only today
  - Showed right homonymous VF loss
Analysis of Visual Fields

- 4/22/09
  - VA 20/40 OD

Diagnosis

- Bilateral Optic atrophy 2° to compressive lesion (old)
  - Pituitary adenoma
  - Progressive VF Loss OS>OD
  - Glaucoma suspect, Ocular HTN off treatment
  - Cataract
  - HTR OU

Retrospective analysis of Visual Fields

OS (9/19/05)  OD (9/19/05)

Points to consider/ Sins of Omission

- Patient seen originally on 03/20/02
  - Dx. NAION OS
  - No VF Done (Sin of Omission) on pale disc

- Found later to have VF loss OS (12/9/02)
  - New Diagnosis : HH
    - Failure to recognize need for neuro-imaging study with homonymous VF loss.
      - First head CT – Scan was done on 06/19/08

- DVA (03/20/02)  OD 20/25  OS 20/200
- Optic nerves
  - C/D .35 round OD, .50 round OS pale disc
The Sins of Omission/Commission

- 1st Diagnosis: AION Non-arteritic OS (3/20/02)
  - (no blood work-up) CRP and ESR
- 2nd Diagnosis: Right Homonymous hemianopia consistent with left occipital CVA (12/09/02)
  - (12/9/02) no CT was done until 06/08
  - Vision dropped to CF OS in 9 months from original visit
  - Tx. On 12/9/02.....Lamigan qhs OU
    - For a Pituitary adenoma

Head CT (6/19/08)

Large homogeneously enhancing sellar mass with suprasellar extension

Very well defined and does not invade surrounding structures

Does have mass effect, favor pituitary adenoma

MRI of head on 7/18/08

Plan

- Consider treatment of pressure if consistently above 23 with Trial of Cosopt

- Follow-up IOP in 1 month OD 17  OS 7

- Observe

Case #3: VW

- Medical Hx.: The triad; (+) HTN, (+) DM, and elevated cholesterol
  - Taking HCTZ, K+, No cholesterol meds at this time

- Ocular Hx.: (+) past history of glaucoma suspect on treatment.
  - (-) hx. of trauma, blood loss, migraines, transfusions, Reynaud’s, hypotensive episodes

Case #3 VW

51yo/ AA/F

Referred for 2nd opinion on glaucoma

Treated OS only for about a year: Timolol 0.5%

BID, Xalatan QHS, VF defect OS.
Clinical findings

- DVA cc: 20/20 OD  20/20 OS
- Pupils: 2+ APD OS
- EOM's: full without restriction pain or diplopia
- SLE: Cornea Cl (-) endo-pigment; A/C Deep and quiet, iris nml OU (-) TID (-) NVI;
  Lens NS 1+ OU
- TA @ 9:30 am OD 15 OS 16,
  * PACH 581 OD 591 OS, CTA 11 OD 12 OS

DFE

- Macula, vitreous Cl.
- optic nerves pink/healthy OD/pale disc OS (9-3)
- C/D .55/.45 OD, .60/.65 OS
- Vessels Attenuated

Optic Nerves

V.F./Initial/ 7-22-09

V.F./Follow Up/ 11/18/09

GDx
Dx

- COAG suspect
- Optic atrophy 2 to ischemia likely (HTN, DM, COD)
- Mild Cataract OU

Plan: order carotid duplex study, echocardiogram to search for embolic source.
- Blood work-up: ESR, CBC w/diff, ANA, ACE, sickle cell screen, C-RP, serum protein electrophoresis, D/C Timolol for now, continue Xalatan qhs OS

Follow-Up

- F/U visit
- D/C Timolol BID OS
- TA 20 OD 17 OS
- IOP changed only 1 point!

Test Results Show

- Echocardiogram: mild calcification of the mitral valve leaflets.
- Carotid Doppler: mild amount of smooth plaque in the proximal portion of the internal carotid arteries bilaterally.
- <50% stenosis
- Blood work-up all normal

Follow-Up Visit

- Px on Xalatan only OS qhs
  - (-) neurological symptom inventory.
- DVA OD 20/15 OS 20/15
- Pupils 2 + APD OS
- TA 15 OD 11 OS @ 8:18 am
- Corrected TA 11 OD 7 OS
- Externals: PA 7mm OU

Follow-Up Visit con’t

- Px spared MRI due to: Health Hx, DM, TTN, and elevated cholesterol
- DVA 20/15 OU
- Color vision normal OU
- VF’s stable over 3 visits
- (-) neuro symptom inventory
- Will follow closely for 1 year VF repeat 3 months

Follow-up VF/ No change
Follow-up

• Patient spared MRI due to:
  ▫ Health Hx.: DM, HTN, AND ELEVATED CHOLESTEROL
  ▫ DVA 20/15 OU
  ▫ Color vision Nml OU
  ▫ VF’s stable over 3 visits
  ▫ (-) neurological symptom inventory

• Will follow closely for 1 yr./ repeat VF 3 mo.

CASE # 4

Clinical Findings and History SJ

• 50/AA/F Referred for 2nd opinion/glaucoma
• Med Hx: (+) HTN, elevated cholesterol,
  ▫ (+) MS Dx 1 yr prior symptoms of tremor,
  ▫ MS confirmed w/ MRI Dec. 2009
• Meds: Lisinopril, Prevastatin, Rebif

History

• Fm Hx of Glaucoma – Father
  ▫ (+) HTN, (+) DM, mother deceased from MI
• OcHx: (-) Trauma/(-)Sx,
  ▫ (+) previous Dx optic neuritis OS, no previous Hx or prior tx for glaucoma

Clinical Findings:

• DVA with OD 20/20 OS 20/20
• Pupils: (-) APD
• SLE: clear cornea, (-)endo-pigment, A/C clear (-) PX, Iris nml, lens 1 + NS OU
• Ta 18 OD 21 OS Pach: 562 OD 551 OS

• Corrected TA OD 16 OS 19

DFE

• Vitreous and Macula CL OU, Optic nerves tilted and obliquely inserted OU, temp. sloping, questionable bilateral temporal pallor.
• periphery: Clear OU (+) T, H, RD’s
Optic Nerves/ OD

• **VF 24-2**: OD Superior Bjerrum, OS Inferior Nasal Loss, Superior/Temp para-central scotoma

• **OCT/NFL**: OD Inferior NFL thin, OS normal

Testing
• **VF 24-2**: OD Superior Bjerrum, OS Inferior Nasal Loss, Superior/Temp para-central scotoma

• **OCT/NFL**: OD Inferior NFL thin, OS normal

Dx
• COAG Suspect OD>OS
• VF defects
• Probable old optic neuritis with secondary OA and VF loss

Visual Fields

OCT/NFL
Clinical Findings and History

• 45/B/M – referred in for Glaucoma OS treated for years since 2005 for LTG OS only.
• IOP’s ranged from 10-15 mm Hg OU. Tried Lumigan an Xalatan in past OS only, well controlled IOP without meds now.
• Med Hx: (+) DM (NIDDM) (-) HTN (+) Elevated cholesterol
• Meds: Metformin, glipizide and Actos

Clinical Findings and History con’t

• Ocular Hx: ?LTG OS, (-) Hx trauma/eye, (-) Hx blood loss, transfusions, hypotensive episodes
• Fm Hx of glaucoma- father
• Clinical Findings: DVA cc OD 20/20 OS 20/25
• Pupils Trace APD OS
• SLE cornea clear OU, A/C D/Q (-) PX; iris Nml, Lens 1+ NS/Trace CS OD Trace CS/NS OS,

IOP

• TA OD 13 OS 14 PACH OD 508 OS 483 Corrected TA 14.0 OD 17.0 OS

DFE

• Vitreous/Macula Cl,
  • Optic Nerves C/D .5/.55 OD .60R OS – Temporal Pallor OS, Vessels nml OU,
  • Periphery Cl OU (-) T, H, RD’s OU

Optic Nerves
VF (old) 08/20/09

Visual Fields 2/15/10

OCT/NFL

Testing

• VF OD Full, OS stable inferior para-central scotoma, OCT/NFL slight NFL thinning OS superior

Dx

• COAG Suspect vs LTG suspect OS only
• VF defect OS
• Likely AION OS R/O compression
• NIDDM without DR
• Mild cataracts OU

Plan: PCP to recheck BP + cholesterol and glucose
Order MRI of Head/Orbits w/contrast (both come back negative)

CASE # 6
History (MG)

- 77/AA/F – Px previously treated for COAG (< 10 yrs) by another practice comes to us to get refill on Cosopt taking BID OU
  - pre-treatment IOP’s: 20 OD, 20 OS
    - (-) Complaints (-) Fm Hx Glaucoma
    - Med Hx: (+) HTN (+) arthritis (+) sinusitis chronic (-) Hx for CVA (-) cardiovascular disease
    - Meds: Terazosin, Metoprolol, Lisinopril, HCTZ

History con’t

- OcHx. (-) Hx trauma (-) CVA (-) eye sx
  - (-) blood loss, hypotensive episodes/crises (-) low BP, (-) migraines (-) Reynaud’s (-) transfusions

Clinical Findings

- DVA cc OD 20/40 OS 20/20
- Pupils: (-) APD
- SLE: Cornea Cl (-) endopigment, A/C D/Q OU
- (-) PX
  - SLE lens mixed combined form cataracts both eyes
  - TA OD 18 OS 20 @ 9:50 am

DFE

- Macula Cl OU: Optic nerve: PFH OU (-) Pallor; C/D OD .70 round OS .65/.60, vessels, A/V crossing changes OU with HTR II OU
- Periphery: flat intact 360° OU

VF Initial (12-10-07)

VF Follow-up (11-11-08)
OCT/NFL

Dx.
- COAG by Hx but VF does not match disc appearance
- Questionable VF possibly 2° to CVA of inferior occipital lobes
- Plan: MRI of brain with contrast
- Findings of MRI consistent with chronic ischemic/vascular change in the inferior occipital lobes bilaterally no acute CVA

Pearls you can take to the bank
- In glaucoma the VF should always match or be explained/supported by the disc/NFL findings
- Pallor can be a subtle but common finding but is not a sign of glaucoma but rather ischemia or compression
- If you note pallor/OA be ready to work it up!

Lastly, Don’t be Blinded by the Science!

Your diagnosis consists of 5 parts:
1. Careful Pointed Hx
2. IOP evaluation
3. VF assessment
4. Careful inspection of the optic nerve
5. And lastly NFL evaluation!

THANK YOU

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