The Case of Wow, How did that Happen!

- 57 y/o W male with h/o sudden LOV OD x 24 hours, with pain!
- Previous h/o Severe EKC 1 year ago OU.
- Medical history positive for 35 lbs weight loss over last 6 months.
- Meds: Lipitor, BB
- VA: LP OD / 20/20 – OS
- Ext: 2 mm fixed pupil OD/ OS & mobil.
- SLE: 4+ cell OD with 1/8 chamber hypopyon. Total secluded pupil with dense fibrin plaque. 4+ MF KP. SPK OD.
- Tn: 11/18.
- DFE: OD no view / OS wnl.

Additional Diagnostic testing
- Laboratory testing
  - CBC with Diff
  - FTA-ABS/VRL
  - FBS
  - ACO
  - Chest Rx
  - HLA B27
  - HLA B27
- Treatment
  - Durezol q1hr OD
  - Blasting Mixture @ office
  - Homatropine 5% OD OD
  - Systane Ultra q4 hrs.
The Case of “How Could I ever be Mad at You”

J. James Thimons, O.D., FAAO
Chairman, National Cornea & Anterior Segment Society

“How could I ever get mad at you”

- GS a 33 y/o Caucasian female presented with a complaint of discomfort, watering and light sensitivity following blunt trauma.
- PEX:
  - VA: 20/20 OD – 20/30 OS
  - SLE: 2 mm area of epithelial damage with staining at 12:00
  - Occasional A/C cell
  - 2+ injection

“How could I be mad at you”

- Treatment:
  - BCL
  - 4th Generation FQ
  - Acular PF
- Symptoms resolved after 1 week of Tx
- Patient dismissed with instructions and Systane q4 hours
- Muro 128 Unguent hs

Symptoms resolved after 1 week of Tx
Patient dismissed with instructions and Systane q4 hours
Muro 128 Unguent hs
“How could I be mad at you”

- Patient returned to office 10 weeks later with c/o AM pain and return of symptoms.
- D/C gtts after 4 weeks
- PEX:
  - VA: 20/30
  - SLE: As shown
  - 2+ injection

Recurrent Erosions

- Pathophysiology - basal epithelial basement membrane misdirection results in:
  - Thickened basement membrane
  - Reduplicated basement membrane
  - Intraepithelial pseudocysts
  - Lack of hemidesmosomes

Recurrent Erosions

- Medical Management
  - Nocturnal lubrication
  - Nocturnal hypertonic saline
  - Bandage contact lens
  - Treat underlying conditions

Recurrent Erosions

- Contributing Factors
  - Dry eyes
  - Blepharitis
  - External disease / tear film abnormalities
**Dry Eye**

- Early stage disease affects an estimated **20-30 million** in the U.S.
- Moderate to severe dry eye disease affects another **9 million** Americans.

*With an aging demographic, environmental changes and increasing visual tasking demands, dry eye remains one of the greatest unmet needs for your patients!*

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**Recurrent Erosions**

- **Surgical Management**
  - Epithelial debridement
  - Chalazion curette
  - 57 Beaver Blade
Recurrent Erosions

- Anterior Basement Membrane Puncture
  - 20-gauge needle

Recurrent Erosions

- Excimer Laser Phototherapeutic Keratectomy (PTK, Group II)
  - Approved for epithelial dystrophies and recurrent erosions
History

- 31 yo white female
- Presented with decreased VA at near with current spectacles and black spots in periphery
- Admitted to hospital in a coma:
  - Kidney failure
  - Liver transplant 4 weeks ago
- Meds: Prednisone, oral cyclosporin + other immunosuppressants
- Patient noticed curtain of vision temporally OU 2w/3 after transplant

Findings

- Habitual prescription -1.50 DS OU
- VA OD 20/20-2 OS 20/25-2
- No improvement with PH
- EOM's: full and smooth
- Pupils: 6/6+/4/-MG
- FC VF: restricted 360 degree field OU
- Amsler grid: no distortion but restricted FOV OU

Further findings

- SLE showed normal anterior ocular health
- Goldmann Tonometry: OD 19 OS 22 mmHg
- DFE: CD 0.1 with flat maculae OU
- OCT: healthy optic discs and maculae OU (within normative ranges)
Immediately contacted transplant surgeon
  – Patient presented in a coma to emergency after suffering a severe stroke and kidney failure
  – Then received the liver transplant
  – Stroke secondary to kidney disease?

Referred for MRI

Prognosis that visual field loss may not be permanent and can be possibly recovered

Bilateral stroke or multiple emboli is very uncommon.

Cyclosporin is the most common causative agent of neurological symptoms such as headache and stroke after organ transplantation. Cortical blindness is possible but neurological symptoms usually resolve upon cessation of the drug.

Macular sparing results from specific occipital infarctions in the distribution of the posterior cerebral artery but also incomplete damage from strokes involving the anterior portions of the extrachiasmatic visual pathways. Aneurysms of the distal PCA exhibit specific clinical features and have favorable prognosis.

Permanent blindness has not been previously reported. Prognosis depends on the cause, it is much better in patients following surgery or cerebral angiography rather than following a stroke.

At 2 week review, improvement noticed in visual field results with enlarged functional retina with increased viewing width

Results as follows:
References


6. Luu S.T., Lee A.W., Chen C.S., 'Bilateral occipital lobe infarction with altitudinal field loss following radiofrequency cardiac catheter Ablation' BMC Cardiovascular Disorders 2010, 10:14


The Case of not everything goes as planned

20 y/o male with severe Keratoconus OS. OD
- Failed CL wear x 3 over 2 years
- OS vision was deteriorating and topography showed progression
- Patient did not want PK.

The Case of not everything goes as planned!

- Patient reports severe pain s/p PRK with Riboflavin @ day two
- Day 1 post-op uneventful
- SLE: Anticipated PRK epi-defect / negative AC reaction
- Meds: Zymar 4/0, PF4/0
- Tx?

Incidence of Post-LASIK Keratitis

- Atypical Mycobacteria
  - M. chelonae/abscessus
  - M. fortuitum
  - M. mucogenicum

- S aureus
  - Fungi
    - Curvularia
    - Fusarium
    - Aspergillus

- S pyogenes
- S viridans
- Nocardia
- Mixed infection
- Herpes simplex (1%)
- P. aeruginosa (1%)
- Culture negative


The Case of not everything goes as planned

- Day 3: Pain severe with mild lid edema
- SLE: infiltrates centrally with several satellite lesions
- Dense apical scar with microstriae
- AC: occasional cell
- VA: 20/ 100 ph no change
- Dx?
- Tx?
The Case of not Everything goes the Way it was Planned!

- C&S Positive for MRSA
- Tx:
  - Vigamox q1
  - Vancomycin q1
  - Systane Ultra q3
  - Systane Unguent hs after PM loading dose of meds

What a Nice Guy!

- Chief Complaint
  - Visual Disturbance OU continuing after LASIK OU in 1999
  - DESx x 4 years
- What are typical post-LASIK complaints potentially related to BV?

Clinical Characteristics of Aqueous Deficient Dry Eye

Meibomian gland dysfunction (posterior blepharitis) results in a decrease in lipid volume and is a leading cause of evaporative dry eye disease.

Clinical Characteristics of Evaporative Dry Eye

- Lid Margin neovascularization
- Squamous metaplasia of meibomian gland orifices

Clinical Signs

- Lid Margin Neovascularization
- Blink Rate
- Schirmer Testing
- Rose Bengal
- Lissamine Green
- Osservarity

Remainder of History

- Personal History: Hypercholesterolemia
- Medication
  - Lipitor
- Allergies - seasonal
- Family History: COAG (father)
**Initial Presentation**

- VA: OD 20/60   OS 20/50
- Tonometry: OD 16mm  OS 15 mm
- SLE: OD and OS WNL
- DFE: Unremarkable

<table>
<thead>
<tr>
<th></th>
<th>OD</th>
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<tr>
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<tr>
<td>DFE</td>
<td>See photo</td>
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**Initial Fundus Photos**

**OD**

![Initial Fundus Photo OD](image)

**OS**

![Initial Fundus Photo OS](image)

**Initial Visual Field**

Reliable OU, Bitemporal Hemianopsia

**Endocrinology Report**

- Pt sent to Yale Medical Group for imaging and endocrinology assessment
- Prolactin Level= 27,000 mg/ml (normal level: 3451 +/- 1111 mg/ml)
- IGF-1 Level=305 (normal: 121-237)
- MRI revealed 2 cysts measuring 2cm as well as soft tissue densities
**Treatment**
- Bromocriptine
  - Dose 2.5 mg po qam, 5mg po qhs
- F/U MRI scan
  - shows tumor significantly shrunk
  - Optic Chiasm decompressed
  - Suprasellar mass still present

**Visual Field Following Bromocriptine TX**

**Anomalous Optic Nerves or Adolescent Glaucoma**

Case Presentation
By: Mitch Ibach, Optometric Intern, Ophthalmic Consultants of Central Illinois
Patient MK- January 2013

- 14 year old male, middle eastern ethnicity, presents for routine eye exam.
- CC: Needs new eyeglasses.
- Entrance Testing:
  - Pupils: PERRLA OU, (-) APD OU
  - CVF’s: FTFC OU

Assessment and Plan

- Assessment:
  1). Glaucoma Suspect
  2). Myopia
  3). Astigmatism
- Plan:
  1). Pt. RTC for full glaucoma work-up including-
     Cirrus OCT, Disc Photos, Humphrey Visual Field,
     and Ocular Glaucoma Work Up- Jan. 15th 2013

- 14 year male returns to clinic for Glaucoma work-up. No chief complaint.
- Angles: Open to Von Herrick angles.
- Corneal Pachymetry: OD-488 microns, OS-490 microns

Central Corneal Thickness (CCT) Factor.

- The Ocular Hypertension Treatment Study (OHTS) – Found two major findings for CCT
  1). The Five major risk factors for progression of Ocular Hypertension to Glaucoma were – Increasing Age, Vertical Cup/Disc Ratio, Pattern Deviation on Visual Field, IOP, and Central Corneal Thickness.
  2). Independent of IOP, a thin cornea was 3 times more likely to
Comparison Vs. Normative Data for 69 year old patients.

- **Parameter**
  - Normal Range*
  
  - **Average RNFL Thickness**: 75.0 - 107.2
  - **RNFL Symmetry**: 76% - 95%
  - **Rim Area**: 1.015 - 1.615
  - **Average C/D Ratio**: 0.618 - 0.169
  - **Vertical C/D Ratio**: 0.594 - 0.165
  - **Cup Volume**: 0.288 -

**Notice:** Mild optic nerve tilt in both eyes.

**OD:** Shallow cup, thin temporal rim, doesn't follow ISNT rule (nasal is thickest).

**OS:**

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**Visual Field - Right Eye**

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**Visual Field Left Eye**

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**Effect of Corneal Hysteresis**

In a study by Mangouritsas, Morphis, Mortzoudis, and Feretis they studied corneal hysteresis in patients diagnosed with glaucoma vs. non-glaucomatous eyes: **Results**

1. Non-glaucomatous eyes CH: 10.97
2. Glaucomatous eyes CH: 8.95

In a study at the Wilmer Eye Institute at Johns Hopkins they studied which influenced glaucoma progression more CCT of CH: **Results**

A lowering of the corneal hysteresis was more closely related to visual field progression than central corneal thickness.
Adding It All Up

Glaucoma Risk Factors:
- **IOP**: Initial visit high, normal 2nd visit
- **CCT**: Thin Pachymetry readings OU
- **Vertical C/D**: Abnormal for Age
- **Age**: 14 → Low for Glaucoma
- **Humphrey VF**: Abnormal

Final Assessment/Plan

**Assessment**
1. Glaucoma Suspect secondary to Anomalous Optic Nerves
2. Compound Myopic Astigmatism.

**Plan**
1. Return in 3 months for repeat Humphrey Visual Field.
2. Perform baseline gonioscopy.

References


The Case of the Disappearing Cup

Patient History

- 50 Year old White female
- Patient was referred by another Optometrist for c/o Headaches with near vision and questionable optic nerve status
- C/o poor vision at near, recent onset
- Meds:
  - Cymbalta, Atacano, Levothyroxin, Xanax, Frova
- Medical History:
  - Patient has a history of Lyme’s Disease.
  - Patient has a history of Rise in Blood Pressure in the last two years
- Family History:
  - Hypertension, Diabetes, Cancer, Heart disease and Cataracts

Patient complained of:
- **Headache**
  - Started two months ago, on the left side, pain and pressure feeling described as being moderate, patient used excedrin for relief
- **Dry Eyes**
  - OS>OD, noticed once a day, feeling described as being mild, patient used Visine to relieve.
Clinical Findings

- Visual Acuity (uncorrected): 20/20 OU
- Pupil: Normal (+) RAPD
- Versions: Smooth and Full
- CT: 6M 10exoph/ 40cm 17 exoph
- Confrontation: FTFC OU
- Color Vision 7/7 OU Ishihara Color Plates
- Today's Blood Pressure:
  - 150/90
  - 141/94
- SLE: Normal
- DFE: 0.3 OU question ONH edema

Patient Examination

- Diagnosis:
  - Optic Nerve Edema based on Decreased Cup/Disc
  - Convergence Insufficiency
- Treatment:
  - Patient was referred for a MRI with and without contrast.

MRI Findings:

- On coronal STIR imaging there is evidence of increased fluid signal intensity in the nerve sheaths of the optic nerve bilaterally.
- This is a non-specific finding but can be seen in intracranial hypertension such as found in pseudotumor cerebri. There is partially empty sella demonstrated with thin pituitary tissue seen in the inferior aspect of the sella turcica. This is likely an incidental finding No obvious abnormality of the optic chiasm is seen on this examination.
- There is no evidence of a mass lesion impinging upon the chiasm. Limited post contrast imaging through the remainder of the brain shows no evidence of abnormal post contrast enhancement.

Conclusion

- There is edematous change seen within the optic nerves bilaterally on coronal STIR imaging. In addition to this, there is partially empty sella as well as possible flattening of the sclera posteriorly.
- These findings can all be seen in idiopathic intracranial hypertension and further clinical evaluation with lumbar puncture may be helpful. Limited evaluation through the remainder of the brain shows no evidence of significant abnormal post contrast enhancement.

CASE #5

- A 37-year-old white male was referred s/p lasik x 3 weeks with h/o decreased acuity OU (OD>OS).
- Onset was gradual but patient has been symptomatic since Tx with pain/ou.
- Surgical history revealed bilateral abrasions at the time of the procedure involving the inferior half of the cap and bed.
- Ophthalmic had two lifts and scrape.

Physical exam revealed BVA 20/200 OD 20/100 OS.
- External shows minimal injection & OU.
- SLE revealed 2+3 epithelial irregularities/OU with 3+4 cystic changes, pseudodendrites and frank defects.
- Ta-18/20 @ 10
Case #5

- Additionally the patient demonstrated 3+ interface haze OU contiguous with and extending beyond the area of epithelial change.
- AC occasional cell.
- Remainder of exam WNL.
- Current medications: Pred Forte q3hour

IOP after DSEK

- Price, FW AJO 2008
- 50 eyes/38 patients
- Mean CCT 701 microns
- Pneumotonometry: 20.3 mmHg +/- 4.5 mmHg
- Pascal: 19.8 mmHg +/- 4.4 mmHg
- Goldmann: 15.9 mmHg +/- 4.9 mmHg
- If IOP is elevated with Goldmann it is probably real

Tell me that Again

- Patient MG - Male Age 52
  - accompanied by wife (who is a pharmacist)
- Referred by O.D. for management of diplopia
- Chief Complaints
  - Double vision
  - Vision seems to keep changing
  - Only relief is covering one eye
Remainder of History

- Ocular History
  - Myopia/Presbyopia
  - February 2009 onset of blur and diplopia D/N
  - Separate Rx for D/N each with differing BO^ from PC-OD
- Medical History
  - Hypercholesterolemia
  - Jun 2009 developed anxiety attacks and depression
- Medications
  - Prior Xanax, Paxil, Prozac, Elavil, Androgel (Testosterone)
  - Now Remeron, Lunesta

Initial Presentation (July 2009)

- Ocular Motility: no restrictions or nystagmus
- Binocularity: 16^ Alt ET/ 11^ Alt ET - Comittant
- Tonometry: OD 16 mm OS 15 mm
- VA: OD 20/20 OS 20/20
- SLE: OD and OS WNL
- DFE: OD and OS WNL
- TVF: normal but multiple fixation losses

Reduced Blink Rate

No Corneal Staining

Dry Palpebral Conjunctiva

Disease Rule-Out

- MRI
  - Normal brain and pituitary gland results (r/o hypogonadism)
  - Normal orbits
- BP 130/84, pulse 80, regular
- Blood workup negative
  - IFE & PE Serum, ANA Direct, RA Factor, Sed Rate, Vit B12, Antithyroglobulin, Lyme

Vision Therapy Implemented

- Emphasis on improving divergence ability
- Accompanied by wife each visit
  - he becomes anxious when left alone
- Progress evaluation shows mild progress
  - Fixation maintenance is difficult
  - Gait is suspect
- Other tests?
  - Visual Evoked Potential -> clearly delayed latency

Visual Evoked Potential (VEP)
N 100 = 140 msecs
Neuro-Compatible Diagnosis?

- Fixation Tremor on Visual Field
- Diplopia
- Dry Eyes
- Delayed VEP Latency

Early Signs of Parkinson’s

- Rigidity
- Bradykinesia
- Postural Instability
- Generalized Fatigue
- GI Problems
- Personality Change
- Anxiety/Depression
- Executive Dysfunction
- Sleep Disturbances

“Facial Mask”
- Loss of Dopamine
- Reduced facial muscles
- Decreased blink rate
- Flat affect/emotionless

Impotence

Trial of L-Dopa/Agonists

References for Parkinson’s


Pathophysiology of Signs/ Symptoms

**PRIMARY:**
- Repeated eversion of lid during sleep abrades conjunctiva and cornea on bedding

**SECONDARY:**
- "Rough" conjunctiva abrades bulbar surface and cornea
- Distracted lid unable to blink tears across eye
- Ptotic lashes +/- lid blocks superior vision

Management Options

1. Conservative
   - Eye shield to the affected side at bedtime (approximately 1/3rd of patients may be sufficiently treated by a shield alone)
   - Nightly ointment

2. Surgical
   - Various methods of eyelid tightening procedures

Surgery for FES

- **Classic Approach =** Eyelid tightening:
  - Wedge excision
  - Lateral tarsal strip
  - Medial plication

- **Disadvantage:**
  - Lid continues to have tendency to evert and stretch over time
  - High reoperation rate
To Sleep Perchance to Dream!
The Role of Sleep Dysfunction in Glaucoma

Obstructive Sleep Apnea
- Bendel, R et al. (Mayo Clinic, Jacksonville)
- OAS - Repeated apnea episodes
- Daytime symptoms
  - Daytime sleepiness
  - Chronic fatigue
  - Decreased cognitive function
- Etiology
  - Collapse of the pharyngeal airway
  - Last 10-60 seconds

OSA
- Diagnosis
  - Overnight polysomnography
  - EEG, EMG, EOG, EKG, Nasal buccal airflow, and pulse oximetry (arterial oxygen)
- Respiratory Disturbance Index 10 >= OASS
- 83 patients with apnea
- Outcomes
  - Median age 62
  - Median RDI 37
  - Median IOP 16mmHg

OSA
- Outcomes
  - 2.4% patients with OHTN
  - 33% COAG
  - No relation to gender, age, or BMI
  - Relation between IOP increase and BMI level

Sleep Apnea & NTG
- Mojon DS et al; Ophthalmologica 2002
- 16 patients with NTG had PSN
- RDI > defined as mild
  - < 45 - 0%
  - 46-64 - 50%
  - 65 & > 63%
Sleep Apnea: The Silent Assassin

- Co-Morbidities of Sleep apnea
  - Increased risk of CVA
  - Irregular Menstrual Cycles (40%)
  - Children May exhibit “Failure to Thrive”: T & A removal
  - Psychologic Dysfunction (32%)

How Low Can You Go!

- SM a 40 y/o white male was referred for evaluation
- VA 20/20 OD/OS
- Ta 12/12 @ 10
- SLE: wnl
- DFE: 0.7 OD / 0.9 OS with Optic Pit
- VF: Altitudinal loss OS
- Gonioscopy: CB 360 OU
- Medical Hx: BMI 38, BP 140/85
- Family Hx: Negative

Visual Field Loss