Interactive Therapeutics: My Most Challenging Cases

Bruce E. Onofrey, RPh, OD, FAAO
Professor, University of Houston

Questions??

EYEDOC3@AOL.COM
Bruceonofrey.com

“My doctor told me to get my vision checked”

• 29 Y/O Chinese female presents with CC: “My vision isn't right” - “IT’S BLURRY”
• Feels like its getting worse for the last 3 months
• My Doctor told me to have my eyes checked because of my medications

DISCLOSURE #1

• I’m a suffering CUBS fan

POSTER FROM 1923

My doctor told me to get my vision checked

• 29 Y/O Chinese female presents with CC: “My vision isn't right” - “IT’S BLURRY”
• Feels like its getting worse for the last 3 months
• My Doctor told me to have my eyes checked because of my medications

Medical HX Doctor #1

• “Lung infection” TX X 6 months
• No other significant HX
• MEDS: rifampin, ethambutol, clarithromycin
• Allergies: None
• Fm HX: Type II DM-F
• Social HX: NEG
• Fm Oc HX: NEG
The exam 10/2011
• BVA: 20/25-2 OU
• Current RX; (+) 0.50 OU
• OD: 20/20-2 OS 20/25-1
• Refrac. +0.75-0.25 X 165 20/20-2
• +0.75-0.25 X 10 20/25 -1
• Pupils: PERRLA (-) APD, IOP 12 OU
• DFE: WNL  C/D: 0.3/0.3 OU
• DX: HYPEROPE-told everthing is OK
• Gave new RX

DR. #2 4/2012
• HX and complaint: see Dr. #1: My vision is getting worse and my doctor told me to get an eye exam
• Acuity with RX: 20/25-2 OU
• Refraction: (+) 150- 0.50 X  10 20/25
• (+) 150 – 0.25 X 160 20/25
A/P: Hyperope/ made new RX

2 weeks later: Doctor #3
• MY new glasses don’t work-they make me blurrier
• HX: Dr. #3 has records from #2
• BVA: Best with original RX
• OD 20/30
• OS 20/30
• Amsler grid: (central blur OU)
• A/P: “Bilateral macular holes”: “see an ophthalmologist”-no appt made for patient

2 weeks later: Back to Dr. #1
• Same complaint with HX of other visits
• BVA: OD 20/50
• OS 20/80
• Macular OCT performed
• Result: NML
• A/P: Diabetic macular edema
• Consult to a PCP for undiagnosed diabetes

1 week later: PCP report:
• NO DIABETES
• Repeats OCT; NML
• Refers to RETINA: Appt made-non-emergent referral

WHAT DO YOU THINK?
• 1. LATENT HYPEROPIA
• 2. AMD
• 3. TOBACCO/ALCOHOL AMBLYOPIA
• 4. ETHAMAMBUTOL TOXICITY
• 5. BILATERAL MACULAR HOLES
• 6. LOW TENSION GLAUCOMA
• 7. MS/ optic neuritis
WHAT DO YOU DO?

• 1. Lower IOP at least 30%
• 2. Get MS consult
• 3. Advise pulmonologist to DC ethambutol
• 4. Advise patient to start drinking and smoking
• 5. Start ARED’s vitamins
• 6. Increase her plus at near and start VT

CC: “A big black spot in the right eye X 3 days”

• 26 y/o female
• MVA, head trauma/concussion X 7 years
• Fall down stairs/concussion X 1 year
• Med HX (-)
• Eye HX (-)
• Meds: NONE
• Allergies: NONE
• Manifest PL OU/ 20/20 OD/OS
• NO HX of migraine or other HA’s

Simple case #2:
The “atypical” red eye, I think we need a corneal consult”

conjunctivitis

Visit #1: AT COMMUNITY CLINIC

• 60 Y/O HF
• CC: “MY EYES ARE RED, I SEE DOUBLE AND I HAVE HAD DAILY SEVERE HEADACHES FOR THE LAST (5) MONTHS

Doctor #1: I think you have…….

• ATYPICAL CONJUNCTIVITIS
• DIPLOPIA: UNKNOWN ETIOLOGY
• TX: PRED FORTE BID OU
• REFER TO UEI FOR EVALUATION

Visit #2: UEI

• CC: Referred from community clinic for red eye X 5 months and diplopia X 1 month
• Using pred forte BID X 2 weeks-no help
• Med HX: Type II DM X 4 yrs and hypertensive
• Hospitalized 2 months earlier for BP
Visit #2 (cont’d)

- Meds: Olmesartan (BP) and metformin (DM)
- NKDA
- FM HX: (-)
- Social HX: (-)
- BVA: 20/20 – OD/OS Hyperopic/astig
- BP: 136/77

Visit #2 (cont’d)

- IOP: 17/16
- CF’s and pupils: Normal
- SLE: (+) 4 conjunctival hyperemia OU
- (+) 1-2 chemosis OU
- (-) C or F
- Cornea clear
- EOM’s” Mild? Restriction in (L) gaze
- Lids: Ptosis OS

Visit #2 (cont’d)

- HVF-30-2:
- OD Scattered defects, judged unreliable
- OS: Scattered defects, judged reliable
- DR# 2: I THINK YOU HAVE:
- DX: Unspecified conjunctivitis/
- (L) 6th nerve palsy secondary to BP/DM
- PLAN: DC PRED, start artificial tears
- Wear eye patch for diplopia/RTC if worsens
- Refer to cornea specialist for red eye (3) weeks

Visit #3 UEI 6/26

- Reason for visit: Recheck red eyes
- CC: “HA’s, red eyes and diplopia are worse”
- SLE: Corkscrew vessels OU
- IOP: 32/24
- EOM: Bilateral 6th N palsy
- CF/Pupils: normal
- DFE: Venous congestionOU

Assessment and Plan (take your pick)

- 1. Really bad conjunctivitis / Restart Pred forte
- 2. Episceritis / Restart pred forte
- 3. Scleritis / restart Pred forte
- 4. Angle closure GLC/ Diamox , oral glycerin, and topical brimonidine STAT
- 5. Immediate referral to corneal specialist STAT
- 6. Needs MRI/MRA/MRV STAT

Simple Case #3

I see black spots
Differential DX

1. PVD
2. RD
3. Optic neuritis
4. BRVO
5. CRVO
6. Cranial Mass

TESTS?

- MRI
- CT
- VF
- Amsler
- OCT
- Color vision

Test results

- Amsler: Normal
- Color vision: Normal
- Macular OCT: Normal
- Visual field:

Visual Fields

Time for an MRI/MRA?

- Her insurance carrier says WHY?
- Homonymous VF defect
- Tests scheduled for 1 week
- 5 days later-”I’ve got a new spot in my right eye”

Re-dilated eye

- DFE: Normal
- MRI/MRA in 2 days
- IT was NEGATIVE
- Now what?
Consult-Yes
BUT-Who gets it?

• 1. Retina
• 2. Neuro
• 3. Medicine
• 4. None of the above
• 5. All of the above

Ever heard of the …… Syndrome?

• 1. Neuro says ignore the other defect, unless it enlarges
• 2. May have cancer
• 3. May have Lymes disease
• 4. May have chorioretinal disease
• 5. Everybody gets a piece of the action

The “Simple” glaucoma case 12/07/2001

• 80 year-old female presents for general exam-new patient
• Last exam 1 year ago- told she had “cataracts”
• Had “dizzy spell” in Oct-Since then, decreased VA OS

Medical HX 12/07/2001

• Hypertension
• Meds: Premarin/cardiazem
• Allergies: Penicillin, sulfa, novacaine, ASA
• (-) family HX of significant eye disease
• (+) family HX of systemic hypertension

Clinical findings 12/07/2001

• VA: 20/30 OD No improvement
  20/60 OS- BVA = 20/30-2 OS
• IOP: 21mm OU
• SLE: Unremarkable
• Pupils: equal, rd, reactive-(-)APD
• Lenses: (+) 2 NS OU
• C/D: 0.5/0.5 OD
  0.6/0.6 (+) 2 pallor OS

Vision improves with refraction-Anything else? 12/07/2001

• 1. No-Pay your bill and leave-please
• 2. ESR/CRP
• 3. Visual field
• 4. MRI
• 5. MRA
Your game plan
12/07/2001

• 1. Monitor IOP and discs for changes-no TX now, recheck in 3 months
• 2. TX with glc med and recheck in 4-6 weeks-Monitor IOP, discs and VF
• 3. Order ESR/CRP
• 4. Order MRI
• 5. Refer to Ophthalmologist

A TALE OF TWO BLEEDERS

• 50 y/o hispanic male presents to acute care eye clinic with complaint of “bleeding eyes”
• VA w/o correction 20/25 OD, OS
• IOP 16 OU
• Pupils: 4mm, +3RX, RD, (-) APD

Bleeders (cont’d)

• Ecchymosis of upper lids
• When asked displays large bruise on upper arm
• 3 episodes of epistaxis in last month
• (-) Hx trauma
• (-) hx of ASA or NSAID or anticoagulant therapy
• BP RA 135/87

Bleeder (cont’d)

• DFE: 2 dot heme OD, NML OS
• Discs: No papilledema or pallor

Lab Tests??

1. PT, PTT
2. CBC
3. CBC, INR, HEPATIC PANEL
4. ESR, CRP, ANA, HLA-B27
5. FASTING GLUCOSE & LIPID PANEL

Bleeder #2

• 76 y/o white male with periocular bleed OS
• Does not drink
• GLC patient
• HX of recent, recurrent bruises on arms and epistaxis
• Normal DFE, IOP
• In spite of being married, denies trauma
**Current Meds**

- 1 325mg ASA/D
- Timoptic 0.25% BID OU
- Trusopt TID OU
- Zantac
- Glucosamine
- Vitamin E 400U BID
- Vitamin C
- Multivitamin

**Bleeder #2**

- BP: 140/68
- CBC: WBC: 1.1VL, RBC: 2.77VL, HCT: 29VL, HGB: 9.9VL, PL 57K VL, Neut: 0.3VL, Lymph, 0.7VL, Mono: 0.1VL, Neut: 18%VL, Lymph 61%H
- PT, PTT: Both normal
- Renal and Hepatic: Normal
- Glucose: 97nml

**Cause of hemorrhage?**

1. Hepatic failure
2. Reduced platelets
3. Renal failure
4. Leukemia
5. Drug induced

**Which meds can make you bleed?**

1. ASA, Zantac
2. ASA, Vit E, Trusopt
3. ASA, Vit E, Timoptic
4. Glucosamine + Tylenol
5. ASA + Glucosamine

---

**Fatal aplastic anemia following topical administration of ophthalmic chloramphenicol.**

Fraunfelder FT, Bagby GC Jr, Kelly DJ

A 73-year-old woman died of aplastic anemia less than two months after undergoing cataract extraction and beginning topical therapy with chloramphenicol. The first signs of pancytopenia began within one month of the surgery. The pattern of the aplastic anemia was associated with an idiosyncratic response to chloramphenicol. This was the second report of fatal aplastic anemia after topical treatment with chloramphenicol for ocular conditions, although two cases of reversible bone marrow hypoplasia have also been reported. Any other suspected cases of ocular toxicity associated with topically applied chloramphenicol should be reported to the National Registry of Drug-Induced Ocular Side Effects, Oregon Health Sciences University, Portland, OR 97201.

PMID: 7072998, UI: 8217020

---

**Drug-induced fatal aplastic anemia following cataract surgery.**

McWhae JA, Chang J, Lipton JH

Department of Surgical Oncology, Princess Margaret Hospital, Toronto.

Aplastic anemia attributed to medications used in ophthalmology is rare. We report a fatal case that developed in a 73-year-old woman 7 weeks after cataract extraction performed under local anesthesia. Postoperative medications included a chloramphenicol-containing ointment, flurbiprofen sodium drops, prednisone acetate drops and orally given acetazolamide. It was felt that the aplastic anemia was related to therapy with chloramphenicol or acetazolamide or both. We recommend that the course of prophylactic antibacterial therapy after intraocular surgery be kept short, regardless of the preparation used. We urge caution in the choice and use of drugs known to be associated with aplastic anemia and recommend close monitoring of the hemogram.

PMID: 1451021, UI: 93082573
Acetazolamide-associated aplastic anaemia.

Keisu M, Wiholm BE, Ost A, Mortimer O

Department of Drugs, National Board of Health and Welfare, Uppsala, Sweden.

Eleven cases of acetazolamide-associated aplastic anaemia were reported in Sweden during a 17-year period. There were six women and five men with a median age of 71 years (range 63-85 years). The median dose of acetazolamide was 500 mg, and the median duration of treatment was 3 months (range 2-71 months). Ten of the eleven patients died, all within 8 weeks after detection of their aplastic anaemia. The relative risk of developing aplastic anaemia when taking acetazolamide was 13.3 (95% confidence limits (CI), 6.8-25.3). The estimated incidence of reported acetazolamide-associated aplastic anaemia is approximately one in 18,000 patient years. The results strongly indicate that acetazolamide treatment is associated with a substantial increase in the risk of developing aplastic anaemia.

Case 2: The “Simple” Conjunctivitis Case

• 28 YO WT male with C/O red, painful OD X 1 month-first occurrence
• TX by primary care doctor with gentamycin drops QID
• Told to use till gone
• Told he has “pink eye”

HISTORY (Cont’d)

• BVA CF’s at 3 feet OD/20/20 OS
• A/C Deep with +3 cell and flare OD
• Post-synechiae 270 degrees OD
• IOP OD 2mm hg/ 17mm Hg OS
• (+) Hx lower back pain

Differential Diagnosis?

• 1. EKC- Adenoviral conjunctivitis
• 2. Acanthamoeba keratitis
• 3. Anterior uveitis
• 4. Spondylarthropathy induced uveitis
• 5. Possner-Schlossman Glaucomocyclitic-crisis

Tests?

• 1. HLA-B27 and Spinal and chest x-rays
• 2. ESR, ANA and RF (rheumatoid factor)
• 3. RPR-VDRL
• 4. All of the above
• 5. None of the above

Initial TX?

• 1. Scopolamine 0.25% BID & Inflamase forte 1% q IH
• 2. Voltaren QID and phenylephrine 2.5% TID
• 3. Ciprofloxacin QID and Voltaren TID
• 4. Homatropine 5% TID-NO STEROIDS
• 5. Viroptic 5X/day and acyclovir 800mg 5X/day PO
When should a DFE be performed?

- 1. A DFE is unnecessary when managing anterior uveitis
- 2. After the eye is “quiet”
- 3. ASAP

The cause of the uveitis is:

- 1. Pars planitis
- 2. Cytomegaloinclusion virus (CMV)
- 3. EKC adenovirus
- 4. Detached retina
- 5. Toxoplasmosis

Watch Out for Masquerade Syndromes

In Uveitis management know your adjectives
- Anterior vs Posterior
- Recurrent vs initial
- Granulomatous vs Non-granulomatous
- Idiopathic vs secondary
- Acute vs Chronic
- Acute anterior non-recurrent secondary non-granulomatous uveitis (WOW)

Case 2 Cont’d

- Prioritize your treatment
- Make sure Hx is complete - i.e. trauma, systemic disease
- Must break synechiae
- Get view of posterior pole
- Prednisolone acetate 1% every hour
- Scopolamine 1/4% BID

“The X-mas tree attacked me”

- 56 Y/O male trimming the Xmas tree when a branch springs back and “pokes” him in the eye (OS)
- Treated at UC (EYE DOCS PHONE MESSAGE TOLD HIM TO GO TO ER) with pressure patch and erythromycin ointment for 2 days
- Returns to UC with increasing symptoms of pain and blurred vision
- Given Neodecadron drops QID X last 7 days
- Initially felt better, but now pain and vision are “much worse”

1st visit to the eye doc: FRIDAY

- VA: 20/30 OD 20/20 OS
- SLE: 1mm infiltrate/ epith. Defect/Shallow ulcer
- + 1 FL, + 1 C
- +1-2 Bulbar injec.
- Pain: 7/10
Initial DX

- I: Bacterial keratitis
- P: DC'd Neodecadron
- Cultured on blood agar
- TX: Vigamox q 1/2h today, then q 2H ATC overnight, then q 1H till recheck on MONDAY (Doc didn’t have Saturday hours)

THE BAD NEWS

- VA CF 5ft OD
- Mucopurulent plaque
- Central lesion with satellites
- Hypopion
- Initial lab: + growth-Probable S. aureus
- Patient referred to Univ. cornea specialist
- Scraped and stained, incl. Gr stain
- DX: Filamentous septate fungi, probably Fusarium

Classic fungal presentation

- VA 20/20 / CF 3 feet NIPH
- 1- ring infiltrate/(+) C and F
- 2- Hypopion
- 3 – Satellite lesion
- 4 – Suppuration = heavy inflammation

The Disease-Mycology

- Fungi, like human cells are eukaryotes- They have a membrane bound nucleus
- Bacteria are prokaryotes-Unbound nucleus
- Because human and fungal cells are more alike than bacteria, there is greater cross toxicity with these agents

Classification of Fungi

Filamentous: The hyphae of these multi-cellular fungi branch and form a tangled mass on the culture plate called mycelium
- Septate: Hyphae divided by cell walls into single or multi-nucleated compartments
- Non-septate: No compartments, long, multi-nucleated tubes
Yeasts: Unicellular with buds or pseudohyphae ex. Candida
Diphasic fungi: Yeast-like in tissues and mycelial structure when cultured

Filamentous sp./ Eye Disease

Septate- #1 cause of keratitis@@@@
- Fusarium solanae: Major cause of keratitis in Florida and south, Most pathogenic of fungi
- Acremonium: Keratitis,-Produces proteolytic enzymes
- Aspergillus: More common in the north, but ubiquitous- lacrimal sac infections, endophthalmitis, orbital cellulitis and keratitis
- Cladosporium
- Penicillium
- Paecilomyces
The Patient at Risk

- Hx of injury from organic material
- "scratched eye with tree branch", then steroid treated with long term antibiotic/steroid combo
- Immunocompromised - Local vs systemic
- Diabetics/systemic disease
- HIV
- Chemotherapy
- Post-organ transplant
- Oral steroid user

Filamentous VS Yeast Signs

- Filamentous
  - Outdoor activity
  - 24-48 H after injury
  - Mild to severe inflammation
  - Feathery edge with satellites - "scattered lesions represent hyphae in corneal stromal tissue"
- YEAST
  - Host is commonly immunocompromised
  - Focal lesion
  - More suppuration
  - Perforation common
  - "Looks more bacterial"
  - No feathery edges or satellites

Lab Tests

- Giems Stain: Indirect view of fungal walls by staining interior
- Gram Stain: Direct view of fungal structure
- Gomori methenamine silver technique - Specific for fungi - Sharply delineates hyphae as black structures on a green background

Common Ophthalmic Drugs

- Amphotericin B (AMPHO-TERRIBLE)
  - Class: Polyene
  - Mechanism: Binds to ergosterol in cell membrane and increases permeability
  - Very toxic to human red cells and renal tubular cells
  - Toxic to conjunctiva/corneal epithelium
  - Produces conjunctival necrosis - NO SUBCONJ INJECTION
  - Dose: topical 2mg/ml q ½ - 1 H

- Natamycin (Pimaricin 5%)
  - THE ONLY APPROVED TOPICAL ANTIFUNGAL AGENT
  - Polyene
  - Increases cell membrane permeability
  - More stable and less toxic than amphotericin
  - "Sticks well" to ulcer site - least toxic of all topicals
  - Used q 2-4 hours
  - Some epithelial toxicity
  - Effective against Fusarium, Aspergillus and Candida

When do you start steroids?

NEVER !!!??
Corticosteroids

• Absolutely contraindicated prior to control of fungal infection
• Judicious use to control inflammatory damage/scarring only after clinical improvement with anti-fungal agent(s)
• Should only be used by someone with intimate/extensive fungal therapy mgmt expertise
• Initial anti-inflammatory TX with cycloplegics preferred

I Just want to die

• 22 year old male presents on consult from PCP for ocular consult
• C/O decreased VA OU
• Consumed approximately one pint of automobile antifreeze solution in a suicide attempt 3 weeks ago
• 1 prior failed suicide attempt 3 months earlier with tranquilizers

Clinical findings

• BVA: 20/100 OD
  20/200 OS
Pupils: Equal, RD, Sluggish, (-) APD
C/D: 0.3/0.3 OU (-) papilledema or pallor
Macula: NML
Color: missed 4/12 plates OD, 6/12OS

Additional tests

• 1. Phoria’s
• 2. MRI
• 3. ESR
• 4. CBC
• 5. VF’s

Treatment

• 1. Urgent case: Refer immediately for anti-freeze poisoning management
• 2. Oral steroids
• 3. Must have IV steroids ASAP
• 4. Refer to neuro-ophthalmologist
• 5. Get a guide dog

Case management

• The GOOD news:
• Your vision will not get worse
• The BAD news:
• It is too late-the damage is done-Won’t get better
• Timing is everything
Acute methanol poisoning

• Emergent
• Get to ER ASAP
• TX with IV ethanol and IV bicarbonate to inhibit metabolism of methanol to formaldehyde and control acidosis

Case:

• 48 YOHM CC: 3 month old glasses are the pits
• Can’t see side-view mirrors when driving-has to turn head-getting progressively worse

Case

• Glasses are PAL’s-first pair
• Acuity 20/20
• RX is correct
• Bases curves /PD,s/Segment Heights are perfect

The Balls in Your Court

1. Bend the glasses and tell him to adapt
2. Remake the glasses as separate SV far and near glasses or as a non-progressive.
3. Repeat the dilated fundus exam
4. Order a visual field
5. Immediate CT Scan

Additional Tests

• DFE: Cupping 0.3/0.3 OU Three months ago
• Today: 0.5/0.7 OD and 0.7/0.7 OS
• Low tension glaucoma??
• Tests?

My Husband Needs and emergency Diabetes check

• Woman calls and states that she is a nurse practitioner and states that her husband has an enlarged, non-reactive, left pupil and experiencing vertical diplopia.
• She demands that we order a fasting glucose and a hemoglobin A1-C
• She agrees to bring in husband for evaluation
Clinical data

- 80 year-old retired radiologist
- BVA: 20/40 OD   20/50 OS
- Meds: None
- Med Hx: Neg
- Pupils: 4mm, (+) 3RX OD/ 7mm, min RX OS X 2 days > light
- Monocular diplopia OU

Tests?

- 1. FBS /A1-C / ESR ASAP
- 2. MRI/MRA
- 3. Visual field
- 4. Weak pilocarpine test
- 5. None of the above
- 6. All of the above

0.125 Pilo test results

- Pupil does NOT constrict

DX/management

- 1. Aneurism–Get immediate MRI
- 2. Adies tonic pupil–Counsel patient
- 3. Pharmacologic effect
- 4. Diabetic pupilopathy–Refer to diabetologist
- 5. None of the above

Denies putting any dilating drops in eyes

Case: All Teenagers are Normal

- 16 y/o wm presents to clinic without parent CC: Blurred VA
- BVA: 20/20 OU-emmetrope
- Color VA Normal
- Pupils: Normal
Case

- Slit Lamp: Normal
- Pupils: PERRLA (-) APD
- DFE: Normal
- NOW What??

Additional Tests

- 1. Contrast sensitivity
- 2. Versions and ductions
- 3. Facility of accommodation
- 4. Visual field
- 5. MRI

Time to Tell Mom

- Told mom he needs MRI-Mom freaks out
- Said he was seen just 6 months earlier by a specialist-Nothing wrong
- Differential includes cranial Mass

The Diagnosis

- MRI shows craniopharyngioma
- Removed successfully
- VF’s normalize

The Big Surprise

- Mom is ever grateful
- Saved her son’s life and sight
- BUUUUTTTTTT………..

Case: The Case of the Stubborn Patient

- 74 y/o black itinerant preacher with c/o decreased VA
- Said he had a “touch of glaucoma” but stopped his drops 5 years ago
- BVA: 20/70 OD 20/200OS
- IOP’s: 33/38
Case

- C/D: OD 8/9 OD .95 OS
- VF’s: Tunnel OD, complete loss OS
- He was advised of his precarious condition of certain loss of vision without tx of his glaucoma
- Said if it is God’s will he accept his blindness” - Refused TX

Management

- 1. Document, document, document
- 2. Get specialist consult
- 3. Speak with relatives
- 4. Agree with patient that he should go blind
- 5. Tell him a story

Bruce Fifteen: Are you the same person I saw 5 years ago?

- 46 y/o hf General exam-No problems
- Last visit 5 years ago
- BVA: 20/20 OU
- Meds: None
- PERRL (+) 2 APD OS
- SLE: NML DFE: C/D: 8/8 OD 9/9 OS

Continued

- IOP’s 14 OU
- Med HX: “I get tired easily, particularly when I try to exercise”
- No visual disturbances

QUESTION: Differential Diagnosis?

- 1. COAG
- 2. Optic neuritis
- 3. LTN glaucoma
- 4. ION
- 5. MS

QUESTION: Additional Tests?

1. Order an immediate MRI
2. Serial Visual fields
3. Serial IOP’s
4. Need more information
5. Both #3 and #4 are correct
Test Results

- Serial IOPs never over 16mm Hg
- No history of blood loss or low BP

QUESTION: Diagnosis?

1. Idiopathic LTN GLC
2. COAG
3. Optic neuritis
4. MS

QUESTION: Management?

1. There is no TX for MS induced optic atrophy.
2. Medically treat as LTN glaucoma patient
3. Needs immediate filtration surgery
4. Needs Tx with high dose steroids-injectable

QUESTION: Best Medical Therapy?

1. Pilocarpine and timolol
2. Xalatan and Brimonidine
3. Betaxolol and Brimonidine
4. Dorzolamide and timolol (Cosopt)
5. Both 2 and 3 are correct

Imune-related disease and normal-tension glaucoma. A case-control study [see comments]

- Abstract: We reviewed the charts of 67 patients with the diagnosis of normal-tension glaucoma listed in the Bascom Palmer Eye Institute computer database. These patients were matched with respect to age, race, and sex with an equal number of patients having ocular hypertension. All medical diagnoses in the charts for both groups were tabulated and classified as either immune-related or non-immune-related. Twenty (30%) patients with normal-tension glaucoma had one or more immune-related disease(s) compared with five (8%) patients in the comparison group (P = .00134, McNemar statistic with continuity correction).

- Author: Cartwright MJ

Retrobulbar arterial hemodynamic effects of betaxolol and timolol in normal-tension glaucoma.

- CONCLUSIONS: These results suggest that, in patients with normal-tension glaucoma, selective beta-adrenergic blockade (betaxolol) may have ocular vasorelaxant effects independent of any influence on intraocular pressure, whereas nonselective blockade (timolol) lowers intraocular pressure without apparently altering orbital hemodynamics.

- Author: Harris A
ABC Drugs of Multiple Sclerosis

Multiple Sclerosis
Introduction

- 250,000 Americans affected
- Affects women 2x more than men
- Strikes between 20-40 years (30 peak)
- Caucasians (Northern European, northern latitudes)
- Autoimmune disease
- Affects myelin – interrupts nerve conduction
- Attack typically lasts 6-8 weeks

Patterns of Disease

<table>
<thead>
<tr>
<th>Course</th>
<th>Character</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign</td>
<td>Abrupt onset</td>
<td>About 20%</td>
</tr>
<tr>
<td></td>
<td>Few exacerbations</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No permanent disability</td>
<td></td>
</tr>
<tr>
<td>Relapsing-remitting</td>
<td>Abrupt onset</td>
<td>20-30%</td>
</tr>
<tr>
<td></td>
<td>Partial or total remission</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inactive for months or years</td>
<td></td>
</tr>
<tr>
<td>Relapsing-progressive</td>
<td>Abrupt onset</td>
<td>40%</td>
</tr>
<tr>
<td></td>
<td>Remissions initially</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Progressive disability later</td>
<td></td>
</tr>
<tr>
<td>Chronic-progressive</td>
<td>Slow onset</td>
<td>10-20%</td>
</tr>
<tr>
<td></td>
<td>Progressive disability</td>
<td></td>
</tr>
</tbody>
</table>

Signs and Symptoms

- Optic neuritis
- Brainstem dysfunction
- Diplopia
- Dysarthria
- Vertigo
- Cerebellar dysfunction
- Tremor
- Incoordination, especially gait
- Weakness or paralysis of limbs
- Numbness or pins and needles (paresthesias)
- Bowel or bladder dysfunction
- Fatigue
- Psychological changes
- Depression
- Personality changes
- Mood changes

Diagnosis

Poser criteria – clinically based
- 2 separate CNS lesions
- Symptoms occurred in ≥2 separate episodes
- Symptoms must involve white matter (tracts, pathways, axonal projections)
- Neuro exam shows objective abnormalities
- Patient between ages 10-50, preferably 20-40
- No other disease-causing symptoms

Laboratory support
- Spinal fluid protein and WBC elevated
- Increase in IgG level and synthesis rate
- Oligoclonal banding under electrophoresis

McDonald criteria – MRI based
- 83% accuracy for MS
- Doubled rate of diagnosis within 1 year of presentation with a clinically isolated attack
Optic Neuritis Treatment Trial Findings

- Untreated optic neuritis pt have a high-spontaneous visual recovery rate
- Treatment with oral prednisone alone is to be avoided
- IV methylprednisolone speeds visual recovery but does not improve visual outcome
- IV methylprednisolone delays onset of MS for 2 years in pt with abnormal MRIs
- 52% chance of developing MS over a 5-yr period if ≥3 MRI lesions; if no lesions on MRI – only 16% chance of MS
- Decreased incidence of MS in African-Americans
- Viral infection
- Neurorretinitis

Treatment Recommendations

- Patient counseling
- No treatment
- Treat with 250 mg IV methylprednisolone every 6 hours for 3 days, then oral prednisone (1 mg/kg/day) for 11 days
- Modifications of treatment
  - 1 g methylprednisolone IV infusion as outpatient for 3 days
  - Taper oral prednisone for 5 days

MS Therapeutic Agents ABC Drugs

A – Avonex (interferon beta-1a)
- Glycosylated – same as natural interferon
- 30 micrograms IM injection once a week
B – Betaseron (interferon beta-1b)
- Nonglycosylated
- 0.25 mg SC injected every other day
C – Copaxone (glatiramer)
- Polymer of basic amino acids
- 20 mg SC injected every day

MS Therapeutic Agents ABC Drugs

Novantrone (mitoxantrone)
- Antineoplastic agent
- 12 mg/m² IV infusion every 3 months
- Combination therapy with Betaseron
  - Relapses reduced by 64%
  - New lesions reduced by 88%

CHAMPS trial results

- 383 patients with first acute clinical demyelinating event & at least 2 clinically silent brain MRI lesions
- IV steroid (per ONTT) then Avonex or placebo
- 3-year follow-up

CHAMPS (Controlled High-Risk Avonex MS Study)

- Steroid and Avonex therapy reduced MS by 44%
- MRI findings
  - 91% reduction of T2 lesion volume
  - 57% reduction in new or enlarging T2 lesions
  - 67% fewer gadolinium-enhancing lesions
- Placebo treated had 82% new but silent MRI lesions within 18 months

Studies
Studies

CHAMPIONS (Controlled High-Risk Avonex MS Prevention Surveillance)

• Study long-term effects and factors associated with development of MS