Ellerbrock Presents Grand Rounds, Part IV

Moderator: Gerald Selvin, OD, FAAO

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American Academy of Optometry 2012 Annual Meeting

Grand Rounds

Abstract:

20 year old Asian female presents with complaints of decreased visual field, and unilateral pain around OD. Examination reveals a right RAPD, high IOP, altitudinal field loss, and optic nerve pallor with cupping OD only.

I. Case History

- Patient demographics:
  20 year old Asian female presents to Ocular Health clinic at University of Waterloo School of Optometry with complaints of vision loss and headaches on the right side of her head that have been getting worse over the last 6 months
    - Symptoms started 7 mos ago with **unilateral headache in frontal/temporal** region on the right side
      - Minor vision changes over that time **especially at the time of the headaches**
      - Been told by a GP **that they were migraines**
      - Reported no history of pain on eye movements
      - **No nausea or vomiting** associated with headaches
      - Noted a **profound loss of vision** in the inferior field of the right eye
      - Progressively getting more severe on daily basis

- Ocular and Medical History:
    - Seen in the primary care clinic (2 weeks prior) and by another optometrist (1 month prior) who had diagnosed her with possible compressive optic neuropathy
        - Had reported (+) RAPD, symmetric IOPs (20 mmHg OU) and optic nerve pallor with cupping
    - Had sent report to the University walk-in clinic with request for STAT MRI and blood work but neither had been ordered.

- Had also requested ocular health records from previous examinations which were present at patients initial visit at the ocular health clinic
- Showed no optic nerve abnormalities and IOPs were wnl (from past exams in the last 10 years)

- Medications
  Birth control pill

- Other salient information
  Questionable history of trauma to the head secondary to abuse when younger.

II. Pertinent findings

- Clinical:
  - First visit (Nov.):
    - BCVA: 20/60 (PH 20/20) OD 20/20 OS
    - Pupils: (+) RAPD
    - EOMs: Unrestricted (-) pain, diplopia
    - Intraocular pressure: 45 mmHg OD; 22 mmHg OS
    - Anterior Segment:
      - External adnexa, lids/lashes, cornea, conjunctiva all within normal limits
      - Anterior chamber: (-) cells and flare, no signs of inflammation OU
      - Angles OPEN by Van Herrick (confirmed with gonio)
    - Gonioscopy: Angles open to ciliary body OU. No signs of angle recession, peripheral anterior synechia, or neovascularization
    - Posterior Segment
      - Lens: cl OU
      - Vitreous: cl OU (-) cells
      - **Optic nerve: 0.90 with slight pallor, deep cupping, positive arterial pulsation OD 0.60 healthy colour, rim tissue healthy, borders distinct OS.**
      - Macula: flat (+) foveal reflex seen OU
      - Vessels: 2/3 normal caliber OU, no change in vasculature around the lesions. (-) vasculitis
      - Periphery: (-) holes/tears/detachments 360 OU

    - Pachs: 515 um OD, 525 um OS
    - Imaging:
      - OCT
        - Very thin retinal nerve fiber layer over entire nasal rim OD
        - WNL OS
      - VF:
        - OD: superior and inferior altitudinal defect with residual inferior temporal island of vision
        - OS: no glaucomatous defects seen (MD and PSD wnl)

- Fundus Photos
- B-scan: revealed no optic nerve lesions

- Management:
  - **LOWER THE IOP:**
  - **4:40pm** 1 drop of Iopidine OD and 1 drop of Betagan OD
  - **5:00pm** IOP OD 42 mmHg
  - **5:05 pm** 1 drop of Iopidine and Azopt
  - **5:15 pm** Iopidine
  - **5:45pm** IOP 44 mmHg OD;
  - Iopidine and Azopt repeated
  - **Rx:**
    - 1 gt Combigan (brimonidine tartrate/timolol maleate ophthalmic solution 0.2%/0.5%) bid OD
    - 1 gt Trusopt (dorzolamide hydrochloride ophthalmic solution 2%) bid OD
    - 1 gt Xalatan (latanoprost ophthalmic solution 0.005%) qd OD
  - RTC the next day
  - Immediate referral made to local Ophthalmologist, reporting asymmetric probable juvenile open angle glaucoma with atypical appearance. Recommended referral for MRI is kept

- One day follow up:
  - BCVA unchanged
  - IOP: 21 mmHg, 22 mmHg
  - Management
    - Continue with combigan, trusopt, xalatan OD
    - Seeing ophthalmologist
  - Outcome:
    - Patients vision dropped to 20/200 OD, 20/20 OS
    - When patient saw ophthalmologist IOP was 44 mmHg (she reported good compliance on meds)
    - She was referred to glaucoma specialist who performed trabeculectomy surgery OD and decided to keep the MRI due to abnormal presentation of the case.
    - After trabeculectomy, patient was on xalatan OU because IOP in OS crept up to 28 mmHg. IOP OD 12 mmHg.
    - MRI results pending.

III. Differential diagnosis

- Primary:
  - Juvenile Open Angle Glaucoma
  - Cavernous sinus disease causing elevated IOP and optic neuropathy

- Given the apparent rapid increase in IOP OD and past history of a normal optic neuropathy and normal IOPs
- Cavernous sinus thrombosis can cause a sudden increase in IOP and intraocular pressure which leads to optic nerve disease
- May also cause an ischemic optic neuropathy
- Can be bilateral or unilateral

- Others:
  - Secondary Glaucoma:

    - Secondary glaucomas: Sturge-Weber, Axenfeld's anomaly, Rieger's anomaly and syndrome, Peter's anomaly, Peter's anomaly, pigmentary glaucoma, posttraumatic glaucoma. All have characteristic signs that can be picked up on slit lamp exam

IV. Diagnosis and discussion

Juvenile Open Angle Glaucoma

- Rare form of glaucoma (incidence of 1%)
- Differs from POAG in age of onset and magnitude of IOP elevation
- Onset is between 4 and 30-40 years of age
- Genetic link for patients with JOAG and is often passed as an autosomal dominance inheritance pattern therefore a strong family history usually present
- No racial predilection, equally effects males and females
- Early clinical signs are subtle, therefore diagnosis may be delayed
- Does not typically have signs of pain or ocular irritation, unless IOP is elevated
- Corneal edema, ciliary congestion and uveitis not usually seen despite elevated IOP
- IOP tends to be greater than 50 mmHg, usually bilateral
- Minimum vision loss until later in the disease
- Gonioscopy results have been described as: open angles with various gonioscopic fidings such as thickened, membrane-like trabecular meshwork, prominent iris processes that may cover the ciliary body band and scleral spur, and a high iris insertion
- This suggests an abnormal development of the trabecular meshwork

Treatment:

- Same medical and surgical therapies as POAG, however, medical therapies often have limited success in management
- Most require surgical therapy for adequate control of IOP
- ALT has a limited use
- Trabeculectomy has shown good results, has higher success rates than goniotomy. However, has less success in JOAG than POAG unless it is combined with MMC to prevent excessive wound healing response that occurs in younger patients
- Implantation of setons may be successful when trabeculectomy with MMC has failed

UNIQUENESS OF THIS CASE PRESENTATION:

- Tentative diagnosis of Juvenile Open Angle Glaucoma (JOAG) was made, however, the clinical findings are not all typical of JOAG.
- Signs consistent with JOAG: elevated IOP, cupping more pronounced than pallor, visual field defect matches the optic nerve appearance and the retinal nerve fiber layer defect
- One-fourth of primary JOAH patients present as a unilateral optic neuropathy with 60% of these having normal IOP in the fellow eye
- Primary JOAG may present with considerable asymmetry with a small proportion presenting as a unilateral disease
- JOAG tend to have severe optic neuropathy and dense visual field loss (~60% with worse than -12dB in worse eye)
    - IOP was elevated unilaterally
    - Optic nerve damage unilateral
    - Severe visual field damage OD
    - Onset of IOP elevation apparently sudden, as was the damage to the optic nerve
    - JOAG diagnosis should not be completely assumed until MRI is complete.

VI. Conclusion

- **Juvenile Open Angle Glaucoma can present unilaterally 25% of the time**
- **Need to rule out secondary glaucomas before diagnosis can be made**
- **Medical management is not typically effective because of uncontrollable IOP, most of the time trabeculectomy's or shunt surgery will be necessary**
- **MRIs are indicated for management when presentation of juvenile open angle glaucoma is atypical.**

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Priming the Pump: A Novel Therapeutic Use for Topical Dorzolamide

Wing Li OD, FAAO

Abstract: Juvenile X-Linked Retinoschisis is a rare hereditary retinal disease that cause decreased vision. Recent studies suggest benefits of treatment with topical anhydrase inhibitors; this case reviews its use on a young patient.

I. Case History
   A. 10 y/o Asian male
   B. No chief complaint but had failed vision screening at school. Went to several optometrist and ophthalmologist who were unable to determine cause for blur.
   C. Personal ocular history: unremarkable
   D. Family ocular history: unknown on mother's side, no significant ocular history on father's side. Older siblings never had an eye exam.
   E. Personal and Family medical history: unremarkable

II. Clinical Findings
   A. BCVA: 20/50-2 OD, OS
   B. Cycloplegic refraction: +1.25 – 0.50 x 090 OD, OS
   C. IOP: 16 OD, 17 OS
   D. Anterior segment: unremarkable OD, OS
   E. Posterior segment

1. Optic nerve: no nerve pallor, healthy rims, C/D of 0.35/0.35 OD, OS
2. Macula: stellate pattern radiating from middle of fovea with symmetry OD, OS
3. Peripheral fundus: unremarkable OD, OS

   F. OCT

1. Macular thickness: 259 microns OD, 554 microns OS
2. Cystic pockets in retina arranged parallel to each other around the fovea

III. Differential Diagnosis
   A. Primary: Juvenile X-Linked Retinoschisis

B. Secondary:

1. Goldmann-Favre Syndrome
2. Retinitis Pigmentosa
3. Ocular Albinism

IV. Diagnosis

A. Juvenile X-Linked Retinoschisis (JXLR)

1. Mutation in retinoschisin responsible for retinal layer adhesion
2. Average VA: 20/60 to 20/80
3. Retinal Presentation:
   a) Stellate pattern around macula
   b) Possibility of peripheral retinoschisis
4. OCT: Increased macula thickness with cystic pockets
5. VA stable for most of life but significant decline at 50-60 y/o

V. Treatment

A. Trusopt 1 gtt tid OS

B. Rationale for treatment

1. Standard option is to monitor and provide necessary services for patient
2. JXLR will cause significant vision loss due to macular atrophy at 50-60 y/o (Apushkin 2005, Gerth 2008)
3. Trusopt and other carbonic anhydrase inhibitors have been shown to reduce retinal fluid in retinal detachment, retinitis pigmentosa (Marmor 1982, Apushkin 2005, Wolfensberger 1999)
4. Recent research suggesting possible improvement in vision and prevention of macular atrophy (Apushkin 2005, Apushkin 2006, Bastos 2008, Genead 2010, Walia 2009)
5. Family insistent on attempting anything that may improve vision

C. Outcome

1. Intitial Visit
   a) Diagnosis made
   b) Treatment discussed and family wanted to initiate treatment
   c) f/u scheduled in 2 weeks
2. Follow-up Visit #1
   a) VA improved to 20/40+2 OS, stable OD
   b) OCT detected a decrease in macular thickness by 190 microns OS, stable OD
   c) f/u scheduled in 4 weeks
3. Follow-up Visit #2
   a) VA decreased slightly 20/40-2 OS compared to follow-up #1, stable OD
   b) OCT detected rebound in macular thickness by 61 microns OS, stable OD
   c) Suspicion for poor compliance
   d) f/u scheduled in 4 weeks
4. Follow-up Visit #3
   a) VA decreased to baseline VA 20/50-2 OS, stable OD
   b) OCT detected continued rebound in macular thickness by 35 microns, stable OD

c) Patient confirmed poor compliance
d) Discontinued treatment
e) f/u scheduled in 3 months

5. Follow-up Visit #4
   a) VA and macular thickness returned to baseline values

VI. Conclusion
   A. Case defines possible role of Trusopt as a treatment option for JXLR
   B. Important to get patient to buy into treatment or else it will fail from poor compliance

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Iris Mass with Angle Invasion and Sectoral Cataract
Denise Pensyl OD, MS, FAAO

Iris mass with angle invasion and sectoral cataract

I. Case history

A. Demographics: 58 yo WM
B. Chief concern: new patient, wants to transfer care for eye 'tumor'
C. Ocular history
    1. "Eye tumor" OS monitored x ~10 years by private practitioner; believes it may be increasing in size
    2. No h/o ocular surgery; reports 'radiation burns' OU at age 16
    3. No family h/o ocular or visual problems
    4. No ocular medications
D. Systemic medical history:
    Barrett's esophagitis, thyroid nodules, COPD, impaired fasting glucose, hyperlipidemia, peripheral vascular disease, smoker, atherosclerosis of aorta
E. Medications: tiotropium, omeprazole, multivitamin w/ iron, ASA, albuterol

II. Pertinent findings
A. Acuity: 20/20 OD,OS
B. Pupils, EOMs, CVF: unremarkable
C. Slit lamp: OS 3 x 2.5mm elevated iris mass inferior, unevenly pigmented
D. Gonioscopy: OS iris mass extending into inferior angle, no tumor seeding
E. GAT: 11, 12
F. Dilated fundus examination (w/ depression): OS sectoral cataract inferior
G. Scleral transillumination: no definitive ciliary body involvement
H. Orbital CT: unremarkable
I. Liver panel: unremarkable
J. PET scan: no definitive evidence of malignancy or metastastis

III. Differential diagnosis
A. Primary: iris melanoma
B. Other:
    1. Iris nevus
    2. Iris cyst (stromal or posterior pigment epithelial)
    3. Iris pigment epithelial tumor (adenoma or medulloepithelioma)
    4. Iris melanocytoma
    5. Iris leiomyoma
    6. Iris metastases
    7. Choroidal or ciliary body melanoma extension
    8. Lisch, Koeppe or Busacca nodules
    9. Iris nevus syndrome
    10. Iris foreign body
    11. Other heterochromia
        a. Congenital
        b. Fuch's iridocyclitis
        c. Prostagladin use
        d. Ocular hemosiderosis or siderosis

IV. Discussion
A. Presentation

1. Circumscribed (nodular) or diffuse
    a. Inferior location, generally pigmented
    b. > 6 clock hours, acquired heterochromia
2. Patient often asymptomatic
3. Rapid growth of pre-existing nevus
4. Associated glaucoma, ectropion uveae/corectopia, cataract, uveitis

B. Malignancy
1. Size: diameter >3mm and thickness >1mm
2. Documented growth
3. Glaucoma
4. Pigment dispersion
5. Invasion: angle, scleral
6. Prominent/intrinsic vascularity
7. Hyphema
8. Ring growth

C. Prevalence
1. Most common iris neoplasm
2. Least common (4%) uveal melanoma
3. Incidence <1/1,000,000
4. Caucasian, light irides
5. Onset 5^{th} to 6^{th} decade
6. Oculodermal melanocytosis increased risk
7. Chromosone 3 abnormalities

D. Diagnosis
1. Clinical
    a. Photographic monitoring
    b. Ultrasound biomicroscopy (UBM)
    c. Shields' criteria:
        1) >3mm diameter x >1mm thick
        2) Minimum of three:
            Photodocumented growth
            Secondary glaucoma
            Secondary cataract
            Prominent vascularity
            Ectropion uveae
2. Biopsy
    a. Fine needle aspiration (FNA)
    b. Excisional
    c. Histology: spindle B, epithelioid, mixed

E. Metastasis
1. 0.5% @ 3 years, 4% @ 5 years, 7% @ 10 years

2. Higher for choroidal and ciliary body melanoma
3. Liver and lungs most common sites
4. Risk factors
    a. Mixed or epithelioid tumor
    b. Angle or scleral invasion
    c. Increased IOP
    d. S/p glaucoma or intraocular surgery
    e. Older age
    f. Diffuse (vs. nodular)
    g. Iris root location
    h. Ciliary body invasion
    i. Extraocular extension

V. Management
    A. Observation
    B. Surgery
        1. Excision/resection
        2. Enucleation
    C. Plaque radiotherapy

VI. Conclusion
    A. More rare and generally better prognosis than other uveal tumors
    B. Workup: gonioscopy, scleral transillumination, DFE with depression, photography, UBM, liver panel/imaging, chest Xray
    C. Diagnosis: clinical, biopsy
    D. Treatment: based on clinical appearance/behavior
    E. Monitor: change/recurrence and systemic metastasis

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Severe Visual Field Restriction Secondary to Optic Nerve Head Drusen

Robert Wang OD, FAAO

78 y/o male reporting to the eye clinic with complaints of restricted field related to optic nerve head drusen. Patient displays a visual field that is restricted to less than 10 degrees in some axises.

- Case History
  - Patient demographics
    78 y/o white male
  - Chief complaint
    Patient is on consult from teleretinal imaging for optic nerve head drusen. Patient was told by a VA retina specialist that he was losing his vision and there was nothing that could be done. Patient is very frustrated that he still has to come to the VA if there is nothing that can be done.
    CC: patient is having trouble running into people or knocking items over because of his visual field constriction.
  - Ocular, medical history
    Ocular: Optic nerve head drusen followed by another VA hospital.
    Medical: GERD, atrial fibrillation, diabetes mellitus, essential hypertension, hypertrophy of prostate, chronic airway obstruction, dyslipidemia, congestive heart failure, pulmonary fibrosis.
  - Medications

Aspirin 325 mg	Carvdilol 6.25 mg	Doxazosin 8 mg
Enalapril M 20 mg	Furosemide 20 mg	Glipizide 10 mg
Metformin 1000 mg	Simvastatin 40 mg	Omeprazole 20 mg

Mometasone F 220 mg	Testosterone C 200 mg	Albuterol 90 mcg
Travaprost Z oph soln	Alphagan oph sol	Flunisolide nasal spray

  - Other salient information
- Pertinent findings
  - Clinical
    Entering VA: 20/25 OD, 20/30 OS
    BVA: 20/20 OD, 20/25+ OS
    ERRL No APD
    Confrontation fields:
      OD: centrally restricted sup/nasal and peripherally 360
      OS: peripherally reduced 360 with sup/temp restriction
    Posterior Segment:
      Patient declined dilation at this visit. Undilated no cup with multiple optic nerve head drusen present both eyes.
  - Physical
    Kinetic visual field finds restriction down to less than 10 degrees in portions of the field in both eyes.
  - Laboratory studies
  - Radiology studies
    B-scan
      OD: highly reflective ONH characteristic of ONH drusen
      OS: highly reflective ONH characteristic of ONH drusen
  - Others
- Differential diagnosis
  - Primary/leading
    Optic nerve head drusen
  - Others
    Disc Edema/Papilledema
    Astrocystic hamartoma
    Gliomas
    Meningiomas
- Diagnosis and discussion
  - Elaborate on the condition
    Optic nerve head drusen are defined as calcified laminate hyaline deposits anterior to the lamina cribrosa. Optic nerve head drusen appear as globular bodies contributing to a scalloped appearance of the disc margin. Over time they migrate to the surface of the optic nerve head. They are found usually bilaterally but with no sex predication. Visual acuity is usually preserved, but optic nerve head drusen are associated with visual field defects 24-87% of the time.
  - Expound on unique features
    Occurrence noted at 3.4 per 1000 adults or 4 per 1000 in children however, one histological adult autopsy study noted 20.4 per 1000. The location of visible optic nerve head drusen does not correlate with the location of visual field defects. With optic nerve head drusen occupying the disc the disc can become crowded creating

a disc at risk leading to an increase in vascular events (ION, arterial or venous occlusions or sub-retinal neo).

- Treatment, management
  - Treatment and response to treatment
    While several treatments have been proposed but no effective treatment has been found. Current guidelines for care include monitoring patient closely for progression of visual field defects and consider anti-glaucomatous therapy to prevent progressive field loss. A referral to the low vision specialist when appropriate will assist the patient greatly with the visual field that s/he retains. Patient was placed on Travoprost once daily OU prophylactically and was referred to the low vision program for assistance with low vision aides.
  - Refer to research where appropriate
  - Bibliography, literature review encouraged

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- Conclusion
  - Clinical pearls, take away points if indicated
    Optic nerve head drusen is a naturally occurring deposit on optic nerve head fibers. Optic nerve head drusen can result in visual field defects that can be progressive. At this time there is no effective treatment for optic nerve head drusen, however some are prophylactically placing patient on anti-glaucoma medication. While there is no effective treatment for visual field loss secondary to optic nerve head drusen our patient was very happy to be referred to the VA's low vision service where he was able to better utilize the vision he retained.

Neovascular Glaucoma in a Non-compliant Diabetic Patient
Andrea Murphy OD, FAAO

Neovascular Glaucoma in a Non-compliant Diabetic Patient
Andrea L. Murphy, OD
AAO 2012 Grand Rounds Case Report Submission
February 2, 2012

Abstract

Neovascular glaucoma is one of the most sight-threatening, late complications of diabetes. This case chronicles the struggles in managing such patients, as well as medical, social, and rehabilitation needs that may need to be considered.

I. Case History

- **Patient demographics**
  Patient AS is a 63 year old Caucasian Male who first presented to the White River Junction (WRJ) VAMC eye clinic as a triage patient on 12/23/2011.
- **Chief complaint**
  Presented to the WRJ ED with eye complaints and shortness of breath.
  Upon examination in the eye clinic, he reported redness OU, intermittent eye pain OU off and on for the last several months (although he denied being in pain at the time of examination), and declining vision OS>OD for about one year
- **Ocular history**
  Last eye exam was in February 2010 at the Manchester VA with the following findings:
  BCVA 20/50+ OD, 20/40- OS
  1) Proliferative diabetic retinopathy with NVD OU, vitreous hemorrhage OU, and pre-retinal hemes OU
  2) Rubeosis OU - (no NVA seen) with elevated IOP OS- patient was instructed to start dorzolamide TID OU and brimonidine TID OU
  He was referred to the ophthalmology department at the Jamaica Plain VAMC in Boston for further evaluation, however, he never showed for this visit or any other eye appointments until presenting in our clinic on 12/23.
- **Medical history**
  DMII x ~7 years; Arthritis; Hypertension; Insomnia; GERD; Depressive disorder; Prolonged PTSD; Hyperlipidemia; Treatment compliance problem
- **Medications**

(from last PCP note at Manchester VA, 11/2010): Glipizide 10 mg tab BID, Lisinopril 40 mg tab QD, Meloxicam 15 mg tab QD, Omeprazole 20 mg cap BID, Temazepam 30 mg QHS, Metformin Hcl 500 mg 2 tab BID.
Patient AS had self-discontinued all medications approximately 4 months prior to being brought to the ED.

- **Other salient information**

Patient was lost to follow-up with his PCP since November 2010 and the eye clinic since February 2010.
Social history: Patient was reportedly living alone with minimal to no contact with family, outside of occasional contact with his brother and sister. He reported having 3 daughters who he had not spoken with for nearly a decade after his wife's passing.

II. Pertinent findings

- **Clinical**

Visual Acuity: (without correction) OD: 2/300; OS: NLP, ? bare LP
Pupils: fixed OU, unreactive OU
Confrontation fields: unable OU
Orbits/Adnexae: normal OU

Slit Lamp Exam:
Lids/Lash: dermatochalasis OU
Sclera/Conjunctiva: 3+ diffuse injection OD; 4+ diffuse injection OS
Cornea: mild corneal edema OD; 2+ corneal edema OS; 1+ diffuse SPK OD; 1+ diffuse SPK OS; severe corneal neovascularization 360 OS extending ~3mm onto cornea, greatest superonasal & superotemporal
Anterior Chamber: deep and quiet OD; grossly normal OS
Iris: +NVA 360 OS>OD
IOP: OD: 55 OS: 70, mires distorted @ 328 pm with Goldmann
Instilled 1gtt dorzolamide OU, 1gtt brimonidine OU, 1gtt timolol OU @ 344 pm

3- mirror gonioscopy: OD: NVA visible in 3 quadrants with scattered PAS
OS: no views due to corneal edema/neovascularization

IOP: OD: 50 OS: 55 @ 4:10 pm, Goldmann
Instilled 1gtt dorzolamide, 1gtt brimonidine OU, 1gtt timolol OU @ 415 pm
Instilled 1gtt prednisolone acetate 1 % OU and 1gtt atropine OU @420 pm
Instilled 1 gtt 2.5% phenylephrine and 1 gtt 1% tropicamide @4:22 pm
Gave 2 250 mg tab acetazolamide po @ 425 pm

Contact with ophthalmology was made in the case that emergency glaucoma surgery would be needed.

IOP: OD: 32 OS: 45 @ 436 pm, Goldmann

IOP: OD: 28 OS: 40 @ 502 pm, Goldmann

Fundus exam:
Lens: OD 3+ NS, 2+ cortical; OS 4+ milky NS, 3-4+ cortical, no view posteriorly
Vitreous: hazy views, (-)vitreous hemes visible OD
Nerve: OD: C/D 0.35/0.35 (horiz/vert) +NVD, greater superior rim
Macula: extensive exudation with thickening throughout macula & extending into posterior pole OD
Posterior Pole: scattered dot/blot hemorrhages in all 4 quadrants
Periphery: poor peripheral views due to poor dilation, (-)tears/detachments evident
***NO VIEWS OS due to corneal/lenticular changes

Instilled 1gtt dorzolamide, 1gtt brimonidine OU, 1gtt timolol OU @ 530 pm
Instilled 1gtt prednisolone acetate 1% OU @530 pm

B-scan OS: vitreal debris, possible vitreous hemorrhage or detachment nasal

IOP: OD: 22 OS: 22,24 @7:10pm, Perkins, at bedside in ward
Instilled 1gtt dorzolamide, 1gtt brimonidine OU, 1gtt timolol OU @ 7:15 pm
Instilled 1gtt prednisolone acetate 1% OU @ 7:15 pm

Patient AS was admitted to the hospital for management of his uncontrolled diabetes and hypertension. Maximum medical therapy was recommended for controlling his IOP and ocular inflammation to include: dorzolamide/timolol combo TID OU, brimonidine TID OU, latanaprost qhs OU, prednisolone acetate 1% QID OU, atropine BID OU, and 2 250 mg tablets of acetazolamide BID po. Patient AS was monitored daily while in the ward (until he could be seen by ophthalmology the following week). His IOP's remained relatively stable in the mid-upper 20's.

- **Laboratory studies** (by medical team at admittance) CBC with differential normal except for low absolute basophils; Glucose 410 (measured as high as 493 later that evening); Hemoglobin A1c 15.4; LDL/Cholesterol/Triglyceride elevated; normal electrolytes and kidney/liver function; urine levels normal; TSH normal; MRSA negative
- **Radiology studies**: none
- **Other:**

  - Patient AS was examined by the Jamaica Plain VAMC retina clinic on 12/27/12 where an injection of Avastin was given. His IOP's on this day were 10 OD and 25 OS with Tonosafe.
  - During his admission at WRJ, patient AS was seen for initial low vision and blind rehabilitation services. Optical devices and glasses were ordered and assistive devices and techniques were discussed to help with his medication management and ADL's. A referral was placed to social work and VIST (Visual Impairment

Services Team Coordinator. Further evaluation of his depression was recommended, however, he declined at that time. He was discharged home with his sister on 1/30/12.

- He was seen for follow-up with JPVA retina the following week and PRP was performed OD. He was also evaluated by the glaucoma specialist who recommended cyclophotocoagulation OD. The patient was scheduled to return for this in 1 week, however, on the day of this appointment, 1/11/12, he returned to the WRJ emergency department with complaints of vomiting and diarrhea for which he was admitted for. Signs of depression were evident as well as reported suicidal ideations by the patient.
- It was suggested that he was suffering from gastroenteritis, probably viral, however, he was also showing signs of metabolic acidosis, therefore, the medical team discontinued his acetazolamide. Unfortunately, the eye clinic was not alerted of this until 2 days later and his IOP's were elevated again to 45 mmHg OD/OS. After discussion with the attending PCP at that time, it was agreed upon to re-start him on Methazolamide 100 mg TID po, due to its lower risk of side effects.
- Cyclophotocoagulation was finally performed OD on 1/18/12. Since that time, his IOP's have ranged from the mid-teens to low 30's OU.
- Once his medical condition was stable, he agreed to be transferred to the inpatient psychiatry unit for monitoring and management of his depression/suidical ideations and adjustment to vision loss. He is planned to be discharged home again on 2/6/12.

III. Differential diagnosis

- **Primary/leading**: Neovascular glaucoma OS>>OD secondary to proliferative diabetic retinopathy from uncontrolled diabetes mellitus II
- **Others:** (if open angle) inflammatory glaucoma, primary acute angle-closure glaucoma; (if closed angle) other causes of iris distortion and peripheral anterior synechiae, such as ICE syndrome, posterior polymorphous corneal dystrophy, Fuch's endothelial dystrophy, and old trauma or surgery; or development disorders including Peter's anomaly, sclerocornea, aniridia, and Axenfeld-Rieger syndrome.

IV. Diagnosis and discussion

- Neovascular glaucoma is considered a secondary angle-closure glaucoma and can develop as a result of a variety of retinal, inflammatory, and vascular disorders, or tumors, surgery, or irradiation, with diabetic retinopathy and central retinal vein occlusion being the leading underlying etiologies.
- It results from the growth of new blood vessels (neovascularization) on the iris (rubeosis iridis)- as well as in the anterior chamber angle.
- The prevalence of rubeosis iridis among patients with diabetes mellitus ranges from 0.25-20%, with neovascular glaucoma developing in approximately 13-41% of these patients.

- Neovascular glaucoma can develop within weeks to years after neovascularization of the iris is first noted, although diabetes usually exists for many years before rubeosis develops and concomitant proliferative diabetic retinopathy is usually found. It rarely occurs in patients with non-proliferative retinopathy.
- Theories behind neovascularization include: retinal hypoxia, angiogenesis factors, and vasoinhibitory factors.
- The pathophysiology of neovascular glaucoma can be described in 4 stages:
  1) Prerubeosis
  2) Preglaucoma where new blood vessels are present on the surface of the iris
  3) Open-angle glaucoma characterized by an increase in neovascularization and a fibrovascular membrane on the iris and in the angle with elevated IOP
  4) Angle-closure glaucoma where contracture of the fibrovascular membrane occurs, causing corectopia, ectropian uvea, flattening of the iris and peripheral anterior synechiae
- The risk of developing rubeosis iridis and neovascular glaucoma associated with diabetic retinopathy is greatly influenced by surgical interventions, including vitrectomy & cataract extraction.
- There is a high correlation between rubeosis iridis and optic nerve neovascularization.
- Clinicians must examine the pupillary margin of the iris closely in these high risk patients, as neovascularization can be subtle and is typically seen here first. Iris fluorescein angiography can also be helpful. Gonioscopy must be performed to rule out any involvement of the angle.

V. Treatment, management

- Includes treating the underlying etiology as well as the glaucoma.
- Tight metabolic control of blood glucose is recommended along with lipid control and effective treatment of arterial hypertension.
- Panretinal photocoagulation (PRP) is the first line of treatment for most cases of neovascular glaucoma. It can be used as a prophylactic treatment, but has also been shown to help lower IOP.
- Panretinal cryotherapy can be helpful to regulate IOP when media is cloudy and help reduce or abolish the neovascularization.
- Anti-VEGF agents, especially Avastin, are gaining more power as an adjunctive treatment of neovascular glaucoma.
- Medical management of IOP, inflammation, and pain with topical and oral medications to include: carbonic anydrase inhibitors (topical or oral), topical B-blockers and alpha-2-agonists, topical corticosteroids, and atropine. Prostaglandin analogues are generally not used due to ineffectiveness & possible exacerbation of inflammation. Miotics are usually avoided as they may increase inflammation and discomfort.
- Glaucoma filtration surgery can be performed when the IOP cannot be controlled with topical or orals medications and the neovascularization is inactive.
- When active neovascularization is present, a cyclodestructive procedure such as cyclocryotherapy, transscleral Nd:YAG cyclophotocoagulation, or diode laser

cyclophotocoagulation may be necessary. Tube-shunt procedures have also had encouraging results.

- Other treatment areas of study are: endoscopic cyclophotocoagulation, silicone oil injection during revision of vitrectomy, intravitreal injection of crystalline triamcinolone acetonide, and exposure to 100% oxygen under hyperbaric conditions.
- In patients who have suffered vision loss, a referral for low vision rehabilitation should be considered.
- As with this patient, concerns with self-neglect/non-compliance and depression should be addressed with referrals to mental health, diabetes education, and/or social work, for example.

VI. Conclusion

- Neovascular glaucoma is a highly complex condition that requires considerable care in establishing the etiologic diagnosis. Its management is often frustrating and prognosis poor, as the available treatment modalities are frequently unsuccessful. Timely diagnosis, patient education, initiation and compliance with treatment is prudent in preserving vision for these patients.

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An Atypical Melanoma Masquerading as a Benign Nevus
Thien Huong Nguyen OD, FAAO

An Atypical Melanoma Masquerading as a Benign Nevus

By Kim Thien Huong Nguyen, OD

A 68-year-old white male presents with a nevus located between his optic disc and macula. To date, it had appeared flat and stable; however, the most recent dilated exam reveals a subtle central elevation. An OCT verifies the presence of a PED and an IVFA confirms the suspicion of melanoma. Imaging, treatment, and prognosis will be discussed. **(350 character limit)**

I. Case History

   a. Patient Demographics

      i. 68 year old white male

   b. Chief Complaint

      i. A 68 year old white male presents to the eye clinic for his annual dilated exam. He reports no visual changes or ocular complaints.

c. Ocular, Medical History

i. Choroidal nevus OS

1. To date it has been documented as flat and stable compared to historical photos

ii. Medical History

1. Diabetes
2. Hypertension
3. Hyperlipidemia
4. Hypothyroidism
5. GERD
6. CAD
7. Acute myocardial infarction

d. Medications

i. Atenolol, losartan, metformin, potassium chloride, zolpidem tartrate, docusate, clopidogrel bisulfate, simvastatin, HCTZ, levothyroxine, ranitidine, fish oil, cyanocobalamin, cholecalciferol, ASA, MVI

e. Other Salient Information

II. Pertinent Findings

a. Clinical

i. Entering VA sc

1. OD: 20/20-
2. OS: 20/20-

ii. Pupils: PERRL (-)APD

iii. EOM: FROM OU

iv. CF: FTFC OD,OS

v. Ta: 17/19 mmHg

b. Physical

    i. SLE: unremarkable

    ii. Fundus

        1. Photo of nevus

        2. C/D: .35v/h shallow OD; .3v/h OS; pink & distinct (-)NVI OU

        3. Macula: flat & intact (-)CSME OU; ~1.25DD nevus with trace central elevation, drusen, and slightly feathery borders located nasally OS

        4. Vessels: normal caliber OU

        5. Periphery: flat, no holes/tears/breaks/RD (-)NVE 360 OU

c. Laboratory Studies

d. Radiology Studies

e. Others

    i. OCT (5 Line HD Raster)

        1. Centrally located PED

    ii. IVFA

        1. early patchy choroidal filling pattern, evidence of feeder vessels, and central pooling

III. Differential Diagnosis

a. Primary/Leading

    i. Metastatic melanoma

    ii. Hemangioma

    iii. Disciform lesion

b. Others

    i. Angioid streaks

    ii. Choroiditis

iii. Coats Disease

iv. Retinal Detachment

v. Leukemia and Lymphoma

vi. Limited Choroidal Hemorrhage

vii. Lyphoid hyperplasia

viii. Uveal Effusion

ix. Sclerouveitis

IV. Diagnosis and Discussion

a. Elaborate on condition

i. Most common primary intraocular malignancy in adults

ii. Focal accumulation of melanocytes that undergo neoplasia

iii. Typically elevated, dome-shaped subretinal mass ranging from dark brown to totally amelanotic

iv. Rate of malignant transformation over 10-year period estimated at 21 in 100,000

v. Rate of metastasis at 10 years

1. small: 10%

2. medium: 23%

3. large: 52%

vi. Prognostic Factors

1. Cell Type

a. definitions

2. Tumor Size

a. Definitions

b. Mortality rates

b. Expound on unique features

i. To Find Small Ocular Melanomas Using Helpful Hints Daily

1. no factors displayed, 3% risk of growth
2. one factor = 38%
3. 2+ factors = 50%

V. Treatment, Management

a. Observation

i. Fundus photography

ii. B-scan ultrasound

iii. OCT Enhanced Depth Imaging

iv. IVFA/ICG

b. Gamma Knife Radiation Surgery

c. Laser Photocoagulation

d. Transpupillary Thermotherapy

e. Brachytherapy

f. External-Beam, Charged-Particle Radiation Therapy

g. Local Tumor Resection

h. Enucleation

i. Refer to research where appropriate

i. Collaborative Ocular Melanoma Study

1. Medium Tumor Trial

a. Enucleation vs brachytherapy

b. Survival rates

2. Large Tumor Trial

a. Standard enucleation vs enucleation with radiation

b. Survival rates

j. Bibliography, literature review encouraged

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VI. Conclusion

a. Clinical Pearls

i. Fundus photography

1. monitor basal diameter growth and other changes in appearance

2. lighting can affect apparent size of a nevus

ii. Newly detected malignancies should be referred for additional testing to detect for possible metastasis

    1. liver enzymes
    2. carcinoembryonic antigen (CEA)
    3. neuroimaging
    4. chest CT

iii. Rate of malignant transformation over a 10-year period estimated at 21 in 100,000

iv. OCT Enhanced Depth Imaging yields improved resolution of deeper layers—consider OCT in addition to photos for monitoring

v. ICG Angiography is better for the detection of tumor vessels

vi. Recommended follow-up for respective tumor sizes