Jessica Steen OD
Justin Schweitzer OD

Case files: the glaucoma chronicles

This course will discuss a broad range of glaucoma cases and glaucoma masqueraders from two unique referral-based settings. Tools for diagnosis, risk assessment, and management of disease will be highlighted across the spectrum of glaucoma severity.

Glaucoma (2 hours)
Learning objectives: At the conclusion of this course, attendees will be able to:

1) Evaluate management strategies for individuals with open angle glaucoma and angle closure spectrum disease
2) Examine alternative medication and treatment options for intraocular pressure lowering
3) Apply medical evidence through recent notable publications into clinical care

I. Case: Normal Tension Glaucoma (Schweitzer)
   a. Landmark Studies
   b. Other factors contributing to progression
      i. Nocturnal blood pressure
      ii. Diurnal IOP rise
      iii. Hypotension
      iv. Ocular perfusion pressure
      v. Sleep apnea
      vi. CSF Pressure
   c. Treatment considerations
      i. Medication decisions and choices
      ii. New therapies in NTG
         1. O-PAP

II. Retinal pathology or glaucoma? (Steen)
   a. 64 year old male referred for evaluation of suspicion of glaucoma due to screening field abnormality
   b. Peak IOP 15mmHg OD, 14mmHg OS
   c. CCT 540 microns OD and OS
   d. Visual field defect
   e. RNFL defect with corresponding GCIPL loss
   f. What else can cause RNFL defect—and therefore visual field defect?
      i. Retinal ischemia
      ii. Nonarteritic ischemic optic neuropathy
      iii. Optic disc drusen
   g. Glaucoma is a chronic, progressive optic neuropathy
   h. Is there change over time?
   i. Take the time that is needed to establish a diagnosis

III. Case: Patient Intolerance and Dry Eye with Glaucoma (Schweitzer)
   a. Impact of Multiple Glaucoma Medications on Dry Eye Disease

i. Fechtner RD et al. Cornea 2010
    1. Incidence of DED among glaucoma patients
        a. 1 bottle = 11%
        b. 2 bottles = 39%
        c. 3 bottles = 40%
ii. Erb C et al, 2008
    1. Incidence of DED among 19,665 glaucoma patients
        a. 1 bottle = 51%
        b. 2 bottles = 55%
        c. 3 bottles = 60%

b. Effects on Meibomian Glands
c. Treatment Challenges
    i. Visual field progression in patients with no side effects vs with side effects
d. Treatment Considerations
    i. Preservative-Free Solutions
        1. PF-Latanoprost
        2. BAK-Free Latanoprost
    ii. SLT
        1. LiGHT 6 year trial
        2. Low-Energy SLT (COAST Trial)
    iii. Glaucoma Drug Delivery
        1. Bimatoprost SR
        2. Travoprost intraocular implant

IV. 63 year old male with a history of "narrow angles" and bilateral LPI (Steen)
a. "About 25 years ago" (1999-at the age of 38)
b. Latanoprost QHS OU with reported peak untreated IOP of high 20s
c. Hypertension and anxiety
    i. Lisinopril and clonazepam
        1. Impact of clonazepam?
            a. No events of significant blurred vision, haloes around lights, significant nausea or headache
d. BCVA 20/20 OD and OS
    i. Refractive error approximately +2.00 OD and OS
e. Patent LPI 1:00 OD and OS
    i. Role of LPI in prevention of primary angle closure
        1. ZAP (Zhongshan Angle-Closure Prevention Study)
        2. 14 year outcome of angle closure prevention in ZAP study
f. Moderately deep central anterior chamber and quiet anterior chamber
    i. Gonioscopy: open to anterior trabecular meshwork 360 OD; 270 degrees OD
    ii. Convex iris approach, no PAS, AR, NVA
        1. 1+ PTM pigment with compression
    iii. Anterior chamber OCT

1. Most effective to determine whether the angle is open or closed-not a replacement for gonioscopy

iv. Do we feel comfortable dilating this patient?

g. EAGLE study
   i. Does this patient meet EAGLE inclusion criteria?

h. What about the clonazepam?

i. Emergency call from primary care optometrist—IOP is 32mmHg OD and OS at comprehensive eye examination
   i. What is the mechanism for elevated intraocular pressure
   ii. Not related to pupil block

j. Now what?
   i. Lower the pressure-is this an acute emergency?
      1. Medical therapy is not disease modifying
   ii. Arrange for cataract surgery
      1. How soon?

V. Case: Advancing Glaucoma in a Pseudophakic Patient on Multiple Medications (Schweitzer)

a. MIGS Considerations and Options
   i. Stenting Procedures
      1. Trabecular microbypass stents
      2. Schlemm canal microstents
   ii. Goniotomies
   iii. Ab-interno trabeculotomy plus canaloplasty
   iv. Subconjunctival devices
   v. Future MIGS
   vi. Post-operative Considerations with MIGS
      1. Stopping glaucoma medications
      2. IOP Spikes
      3. Hyphema
      4. Hypotony
      5. Establish New Baselines

VI. Streamlining medical therapy (Steen)

a. 51 year old male with diagnosed pigmentary glaucoma presents for a second opinion on therapy
   i. He is cautious about pursuit of SLT but wishes to reduce medical therapy
   ii. Currently taking:
      1. Latanoprost QHS OU, dorzolamide-timolol BID OU, brimonidine BID OS
         a. IOP 17mmHg OD, 21mmHg
   iii. What other options exist?
   iv. Netarsudil
      1. Rho kinase inhibitor/norepinephrine transport inhibitor
         a. Primarily acts to increase trabeculae meshwork outflow

2. Available alone (netarsudil 0.02%-Rhopressa) and in combination with latanoprost 0.005% (Rocklatan)
3. Dosage-once daily in the evening
4. Most common adverse events
    a. Hyperemia, corneal verticillata

v. MORE phase 4 trial
1. Multicenter, prospective, open-label study
    a. No comparator, treated IOP =>20mmHg
2. Three groups:
    a. Latanoprost alone
    b. Latanoprost +1 agent
    c. Latanoprost +2 agents
3. All groups switched to netarsudil/latanoprost alone
4. Expected outcome?
    a. How is this possible?

b. Leaving South Florida in 2.5 weeks for the summer—is this enough time to assess medication efficacy and tolerability?
    i. 16 days of treatment: 15mmHg OD, 21mmHg OS