Glaucoma Clinical Conundrums
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Abstract: During the course of caring for patients with glaucoma, clinicians will undoubtedly encounter clinical issues that are challenging and controversial. This lecture will examine several clinical conundrums such as to what to do when a patient develops a disc hemorrhage, what to do when patients can’t or won’t use medications, and whether or not the patient has glaucoma or some other disease. This course will discuss these and other clinical conundrums and, combined with evidence based medicine, suggest options and approaches for each situation.

Conundrum: Is this really glaucoma?
- Optic nerve anomalies: ONH hypoplasia, coloboma, buried drusen, optic pits, tilted disc, obliquely inserted disc, etc. These are misdiagnoses.
- Neurological diseases: optic atrophy, chiasmal syndromes, compressive lesions of the anterior visual tract. These are misdiagnoses.
  - Arteritic anterior ischemic optic neuropathy does have enlargement and deepening of the optic cup, but has other features including devastating sudden vision loss and disc pallor that are not found in glaucoma

Not All –Omas are Glaucoma:
“-Omas”
- Pituitary adenoma
- Craniopharyngioma
- Meningioma
- Glioma
- Metastatic carcinoma
- “Ischemioma”
- Anterior ischemic optic neuropathy (AION)
  - Both arteritic and non-arteritic
- Congenitaloma
- Coincidentaloma
- Prolonged compression results in optic atrophy, which may present with pallor and/or cupping of the nerve head.
- Optociliary collateral vessels may be noted at the disc margin.
- There will frequently be increased progressive cupping of the optic nerve head somewhat similar to that seen in glaucoma.
  - The main differentiating factor from glaucomatous optic atrophy is the pallor of the remaining neuroretinal rim in compressive neuropathy.
- Additionally, there is more significant neuroretinal rim compromise in the form of notching that occurs in glaucoma but not in compressive lesions where cup increase is more symmetrical with pallor.
- Associated field defects include central scotomas, arcuate or altitudinal defects, paracentral scotomas, field constriction, and defects respecting the vertical hemianopic line.

**Conundrum:** Help, my patient can’t/won’t afford/use medications. What do I do?
- Resources for patient assistance
  - RXHOPE
  - Drug company patient assistance programs
- Generic medications
  - Issues, safety, effectiveness of generics for glaucoma
  - Laser Trabeculoplasty: SLT and Efficacy
  - SLT & ALT are equivalent in their capacity to decrease the IOP in glaucoma patients
  - Produces different effects at the treatment site
  - ALT induces mechanical alterations, due to collateral thermal effects
  - SLT induces no apparent tissue alterations, due to the lack of such thermal effects
  - It is believed that SLT can be performed more than once, but this is unproven. Most will only repeat one time.
  - Lack of coagulative necrosis produced by SLT is related to the nanosecond duration of each pulse and the selective targeting of melanin chromophores
  - Application of SLT obliterated macrophages, leaving the non-pigmented lining TM cells intact
  - An acceptable if ephemeral initial therapy option
- Trabeculectomy vs. Minimally Invasive Glaucoma Surgery (MIGS):
  - Appear to have improved safety profile over trabeculectomy, but reduced efficacy
  - Procedures:
    - Canaloplasty
    - Trabectome
    - Glaukos iStent
    - ECP

**Conundrum:** When is surgery wrong for the patient?
- When the risk of surgery is greater than its expected benefit.
- When it is more dangerous to undergo a surgical procedure than to continue on the same medical treatment.
- When you would not recommend the same intervention to your family members
  - Establishing the treatment course
    - Is the disc or field status stable or worse?
    - If progression has occurred, over what time period?
    - What is the rate of change?
    - What is the risk of visual disability in the patient’s lifetime?
    - Is the patient aware of either decreased central visual acuity or peripheral visual field loss?
      - Classic question: Is it the cataract or the glaucoma or the age related macular degeneration?
When is filtering surgery wrong?

- The blind painful eye
  - Treatment is aimed to achieve a comfortable eye (neovascular glaucoma)
  - Time honored concept – no invasive surgery in a blind eye
    - Alcohol/ chlorpromazine injection, cyclodestruction
  - Rare reports of sympathetic ophthalmia after cyclodestructive procedures (cyclophotocoagulation)
  - Meds: IOP reduction, steroids, atropine
  - If pain cannot be controlled – enucleation or evisceration are excellent options

Surgical risks

- Intraoperative suprachoroidal hemorrhage (“explosive hemorrhage”)
  - Risks – elderly, hypertensive, prior vitrectomy, aphakia, very high preoperative IOP
  - About 1:1500 to 2000 overall
- Postoperative endophthalmitis
  - About 1:1500 to 1:2000 eyes in USA
- Ptosis
  - Uncommon, probably less than 2%
- Trabeculectomy
  - Immediate postoperative period
    - Hypotony – flat anterior chamber, acute cataract, angle closure, choroidal effusion
    - “Wipe out” or “snuff out” syndrome – acute loss of central acuity without obvious intraoperative complication
    - Decreased visual acuity - Patient only knows that they see much worse after surgery
- Glaucoma drainage implant surgery
  - Muscle imbalance – noncommitant diplopia
- Late postoperative period
  - Posterior synechiae formation – poor dilation
  - Cataract formation
  - Bleb scarring and return of high IOP
- Very late postoperative period
  - Endophthalmitis and blebitis
    - Remember “RSVP”
      - R – Redness
      - S – Sensitivity to light
      - V – Vision Change
      - P – Pain

Conundrum: Help! My patient has a disc hemorrhage and the pressure is 12 mm. Now what?

- Disc hemorrhages
  - Patients with normal tension glaucoma, primary open angle glaucoma, ocular hypertension
    - Anemia, posterior vitreous detachment, vascular occlusion can cause hemorrhages of the disc that are mistaken for glaucomatous disc hemorrhages
Ischemic or mechanical
Probable infarction of the blood supply to the ONH
Inferior, inferior temporal, superior, superior temporal regions of the disc most susceptible and account for virtually all true disc hemorrhages
  - Hemorrhages at other areas of the disc (nasal and temporal) tend to not be associated with glaucoma
Typically occurs where notches occur
Resides in the retinal nerve fiber layer
  - Not in the cup!
Small and contiguous with the neuroretinal rim
Can be recurrent and, if it recurs, it typically is in the same place on the disc each time
Precedes notching, NFL defect, field loss. Perhaps the earliest change in glaucoma (if it happens)
More common in patients with large IOP variations
Meaning is unclear – possibly indicates poor control of IOP?
  - EMGT study showed that lowering IOP cannot prevent disc hemorrhages from occurring
Disc hemorrhages do not constitute a diagnosis of glaucoma nor a progression or conversion to glaucoma or an endpoint for any major glaucoma studies

Conundrum: The diagnostic imaging doesn’t agree with my diagnosis? Now what?
- Issue of ‘red disease’
- Understanding imaging interpretation and role in glaucoma diagnosis