10.4.5. Open Angle Glaucoma (V): Secondary Open-Angle Glaucoma (III)

Intraocular Tumors

- unilateral chronic glaucoma
- pathogenesis
  - open-angle
    - direct tumor invasion of the anterior chamber angle
    - deposition of tumor cells, tumor-filled macrophages, inflammatory cells, and cellular debris within the trabecular meshwork
    - intraocular hemorrhage
  - angle-closure
    - rotation of ciliary body or anterior displacement of the lens–iris interface
    - neovascularization of the angle
    - pupillary block glaucoma caused by posterior synechiae
- tumor types
  - adults
    - uveal melanoma
    - metastatic carcinoma
    - lymphomas
    - leukemia
  - children
    - retinoblastoma
    - juvenile xanthogranuloma
    - medulloepithelioma

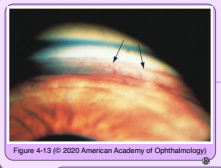
Elevated Episcleral Venous Pressure

- See Table 4-3
- 8–10 mm Hg
- normal episcleral venous pressure
- etiology
  - head trauma
  - most cases are idiopathic
  - ± familial
- clinical presentation
  - unilateral or bilateral
  - chronic red eye without discomfort or allergic symptoms
  -
    Figure 4-14 (© 2020 American Academy of Ophthalmology)
  - tortuous, dilated episcleral veins
  - signs of ocular ischemia or venous stasis
  - blood in the Schlemm canal
  - gonioscopy
  - sudden, severe carotid-cavernous fistulas
    - proptosis
    - other orbital or neurological signs
    - may require neuroradiologic intervention
- treatment
  - prostaglandin analogues
  - aqueous humor suppressants
  - glaucoma filtering surgery
    - complicated by ciliochoroidal effusion or suprachoroidal hemorrhage
  - laser trabeculoplasty is not effective

Inflammatory Glaucoma

- pathogenesis
  - open-angle
    - trabecular meshwork edema
    - trabecular meshwork endothelial cell dysfunction
    - trabecular meshwork blockage by fibrin and inflammatory cells
    - blockage of the Schlemm canal by inflammatory cells
    - prostaglandin-mediated breakdown of the blood–aqueous barrier
    - steroid-induced glaucoma
      - reducing inflammation and improving aqueous production
      - decreasing outflow through the trabecular meshwork
  - angle-closure
    - peripheral anterior synechiae
    - posterior synechiae with iris bombé
- uvelitides commonly associated with open-angle inflammatory glaucoma
  - Fuchs heterochromic iridocyclitis
  - herpes zoster iridocyclitis
  - herpes simplex keratouveitis
  - toxoplasmosis
  - juvenile idiopathic arthritis
  - pars planitis
- miotic agents should be avoided in patients with iritis
  - ± aggravate inflammation
  - ± cause posterior synechiae
- prostaglandin analogues
  - studies have demonstrated their ocular hypotensive efficacy in eyes with anterior uveitis
  - ± exacerbate inflammation in some eyes with uveitis and herpetic keratitis
- inadequately controlled inflammation with elevated IOP is often mistaken for steroid-induced glaucoma
  - presence of KP suggests iritis as the cause of IOP elevation
    - gonioscopy may reveal subtle trabecular meshwork precipitates
  - in the face of active inflammation, elevated IOP should be presumed to be inflammation-related rather than steroid-induced

Fuchs heterochromic iridocyclitis

- epidemiology
  - relatively rare
  - middle-aged men and women
  - rubella virus infection may be the underlying cause
- glaucomatocyclitic crisis (Posner-Schlossman syndrome) may be caused by herpes simplex virus
- clinical presentation
  - chronic
  - unilateral
  - in patients with blue irides, the affected eye may appear hyperchromic
    - iris hypochromia
    - loss of iris pigment
  - low-grade anterior chamber reaction
  - small, stellate, pancorneal KP
  - posterior subcapsular cataracts
  - 
    Figure 4-13 (© 2020 American Academy of Ophthalmology)
  - not associated with fibrous membrane and do not lead to PAS and secondary angle closure
  - multiple fine vessels that cross the trabecular meshwork
    - gonioscopy
  - spontaneously or with trauma, including cataract or glaucoma surgery
    - fragile and may cause an anterior chamber hemorrhage
  - 15%
    - secondary OAG
  - IOP does not correspond to degree of inflammation
- treatment
  - corticosteroids
    - generally not effective
    - could potentially cause a steroid-induced IOP elevation
  - aqueous suppressants
    - OAG may be difficult to control

Glaucomatocyclitic crisis (Posner-Schlossman syndrome)

- epidemiology
  - uncommon
  - middle-aged patients
- etiology
  - unknown
  - infection with herpes simplex virus has been implicated
- open-angle inflammatory glaucoma
  - recurrent bouts of markedly increased IOP and low-grade anterior chamber inflammation
- symptoms
  - unilateral
  - blurred vision
  - mild eye pain
- clinical presentation
  - mild iritis
  - few KP that are small, discrete, and round
    - usually resolve spontaneously within a few weeks
    - may be seen on the trabecular meshwork
      - "trabeculitis"
  - markedly elevated IOP
    - 40–50 mm Hg range
    - in between bouts, the IOP usually returns to normal
      - with increasing numbers of attacks, a chronic secondary glaucoma may develop
  - ± corneal edema
- prevention
  - no evidence that long-term suppressive therapy with topical nonsteroidal anti-inflammatory agents or mild steroids is effective
- differential diagnosis
  - recurrent attacks of acute angle-closure glaucoma
  - cytomegalovirus
    - distinguishing glaucomatocyclitic crisis from cytomegalovirus is important, because specific antiviral therapy for cytomegalovirus is available

Ophthalmology Mind Maps/Arman Mashayekhi, MD