9.10.3. Complications of Uveitis (III)

Retinal and Choroidal Neovascularization

- retinal neovascularization
  - most often associated with
    - pars planitis
    - sarcoid panuveitis
    - retinal vasculitis of various causes, including Eales disease
  - pathogenesis
    - chronic inflammation
    - capillary nonperfusion
  - treatment
    - reduction of inflammation
    - scatter laser photocoagulation
      - if ischemia is angiographically extensive
      - uveitic retinal neovascularization does not always require panretinal photocoagulation
    - VEGF inhibitor
      - adjunct to IMT and scatter laser photocoagulation
- choroidal neovascularization (CNV)
  - pathogenesis
    - disruption of the Bruch membrane
    - inflammatory cytokines
    - 10% of patients with VKH syndrome
  - treatment
    - reduction of inflammation
      - control of inflammation may reduce the risk of recurrence of CNV
    - anatomical ablation of the CNV
      - focal laser photocoagulation of peripapillary, extrafoveal, and juxtafoveal CNV
    - VEGF inhibitors
      - for subfoveal CNV
    - photodynamic therapy with verteporfin

Rhegmatogenous Retinal Detachment

- 3%
- uveitis itself may be an independent risk factor for the condition
  - panuveitis
  - infectious uveitis
  - pars planitis
  - posterior uveitis
  - are the entities most frequently associated with RRD
- uveitis is often still active in eyes that present with RRD
- repair is often complicated by
  - 30% proliferative vitreoretinopathy (PVR) at presentation
  - vitreous membranes
  - poor visualization
- acute retinal necrosis/CMV retinitis
  - prophylactic laser treatment
    - the rate of detachment is still high
  - difficult to repair because of multiple large posterior retinal breaks
  - pars plana vitrectomy and endolaser treatment with internal silicone oil tamponade
- scleral buckling
  - retinal detachment associated with pars planitis
  - when PVR is present, combined scleral buckling and pars plana vitrectomy is often required

Vitreous Opacification and Vitritis

- vitrectomy may improve visual acuity
  - 68%
  - standard small (25- or 23-gauge) 3-port pars plana vitrectomy

Cystoid Macular Edema

- pathogenesis
  - active intraocular inflammation
    - mediated by the proinflammatory cytokines
      - VEGF
      - interleukin-6
      - cause retinal vascular leakage & retinal pigment endothelium dysfunction
    - severity of CME does not necessarily correspond to the level of inflammatory activity
    - often slow to respond and clear and frequently remains even after visible, active inflammation has resolved
    - smoking appears to be associated with a greater prevalence of CME
  - mechanical vitreomacular traction
- monitored by serial spectral-domain OCT and fluorescein angiography
- treatment
  - control of intraocular inflammation
    - if CME persists despite control of inflammation, therapy directed specifically toward the CME is required
  - periocular injections of corticosteroid
    - superotemporal posterior sub-Tenon injection
      - delivers juxtascleral corticosteroid closest to the macula
    - 20–40 mg of triamcinolone acetonide
    - may be repeated monthly
  - Intravitreal triamcinolone
    - 2–4 mg of intravitreal preservative-free triamcinolone
    - highly effective in reducing CME
    - eliminated more quickly from the vitreous cavity of vitrectomized eyes
    - maximum visual improvement and reduction of CME occurs within 4 weeks
    - eyes with a longer duration of uveitic CME and worse vision on presentation tend to show the least amount of visual improvement
    - corticosteroid-induced elevation of IOP
      - up to 40% of patients
      - especially <40 years
  - implants
    - fluocinolone acetonide implant
      - intravitreal sustained-release drug-delivery system for dexamethasone (700 μg)
        - risk of ocular hypertension is lower compared to fluocinolone implant
  - anti-VEGF therapies
    - short duration action
    - repeat injections are required
  - intravitreal methotrexate (400 μg/0.1 mL)
    - effective in reducing uveitic CME in a limited number of patients
  - topical NSAIDs
    - beneficial in treating pseudophakic CME
    - effectiveness in the treatment of uveitic CME has not been well established
  - oral acetazolamide
    - 500 mg once or twice daily
    - effective in reducing uveitic CME
      - particularly in patients whose inflammation is well controlled
  - systemic interferon therapy
    - effective in resolving recalcitrant CME
    - complete control in 62.5% of patients
    - causes flulike symptoms
  - surgical therapy
    - controversial
    - pars plana vitrectomy in the presence of hyaloidal traction on the macula
    - in the absence of vitreomacular traction the efficacy of pars plana vitrectomy in treating CME is not well understood

Ophthalmology Mind Maps/Arman Mashayekhi, MD