Title: Determining the Etiology of an Acute Pars Planitis

After diagnosing pars planitis, it is imperative to explore the etiology before proceeding with a treatment plan. In this case, we explore the potential pitfalls of determining causation in order to provide safe-effective care.

I. Case History
   a. 65 year old African American male
   b. CC: Acute onset of redness, floaters, and pain O.D.
       Latanoprost OU qhs x 3 days prior to onset of symptoms
   c. Ocular history:
       Choroidal nevus O.S.
       Glaucoma O.U.
       Cataracts OU
       Hypertension without ocular manifestations OU
   d. Medical history:
       HTN
       Hypercholesterolemia
       Cervical spondylosis with myelopathy
       Headaches
       Gout
       Diabetes mellitus
   e. Medications
       Metoprolol Tartrate, Lisinopril, Cholecalciferol, Aspirin, Amlodipine, Colchicine
       Loratadine, Atorvastatin, Sildenafil

II. Clinical
   a. Physical
      Vision with correction
      1. O.D. 20/60- PH NI
      2. O.S. 20/25- PH 20/20
      3. Corrected to 20/20 OD one month prior
      Slit Lamp: Anterior Chamber:O.D. 4+ cells; fine KPs inferiorly
      Intraocular Pressures (Goldmann)
         O.D. 24
         O.S. 13
      Dilated Retinal Evaluation
         hazy view into retina O.D.: approx 20/50
         CDR 0.3 O.D. flat pink and discrete
         0.6 O.S. flat pink and discrete
         Subtle sheathing of the venules O.D.
      Periphery OD:
         (+) numerous round snow balls mid-peripheral
         (+) snowbanking: temporally extending from 11:00 counterclockwise to 2:00.
         (+) small dot hemorrhage @ 9:00
      Vitreous: (+) vitreous opacities/cells OD Gr II+
      OCT: No Macular edema
         Prominent retinal vessels: dramatic increase in retinal vessel size consistent with posterior pole vasculitis OD
         Possible Optic neuropathy; less cupping on OCT than previous
      Serial OCT ONH O.D. shows previous large cupping 0.6
      Fundus photography:
multiple snowball lesions OD
classic pars planitis, snowbanking, >temporally
subtle vascular sheathing
Asymmetrical cupping OS> OD
b. Laboratory/Radiology studies
   1. Chest X-Ray for tuberculosis: normal
   2. MRI without contrast for multiple sclerosis:
      nonspecific mild white matter disease
   4. Sedimentation Rate: 6/Normal
   5. Anti-nuclear Antibody: POSITIVE
Pending Labwork:
   1. ANCA/Vasculitides
   2. HLA DR+DQ Ag
   3. Green Li/Hep BLD/Plasma
   4. Lyme Screen
   5. Toxoplasmosis Panel
   6. ACE
   7. HLA B27 Ag
   8. Quatiferon- TB Gold
Pending vitreous/anterior chamber tap
   1. Polymerase Chain Reaction (PCR for HSV, CMV, toxoplasmosis)
III. Differential diagnosis for causation of intermediate uveitis/pars planitis
   a. Acute retinal necrosis (ARN)
      Treatment should be quick and aggressive with po antivirals
   b. Sarcoidosis associated pars planitis
   c. Multiple Sclerosis associated pars planitis
   d. Idiopathic: majority of pars planitis cases have an undetermined etiology
IV. Diagnosis and discussion
   Pars Planitis refers to a specific presentation of intermediate uveitis that by definition
   includes inflammatory exudates located at the pars plana and may cause cause
   posterior vasculitis. Although it is often described as an isolated and idiopathic finding
   (50-70%), it has also been associated with multiple sclerosis, sarcoidosis, Lyme
   disease, and a variety of inflammatory conditions. It is imperative to determine the
   cause of the pars planitis through clinical examination, serological and radiological
   testing in order to effectively diagnose, treat, and manage any associated conditions.
   It is estimated that 20% of patients with pars planitis will lose vision due to macular
   edema and inflammation. Topical therapies alone are not sufficient to effectively
   treat pars planitis. The standard of care is regional or peri-orbital steroid injection
   with the addition of po NSAID and or systemic immunosuppressive chemotherapy if
   needed to decrease the inflammation to prevent permanent vision loss. Aggressive
   treatment has been suggested even in mild to asymptomatic cases. Unfortunately the
   regional orbital treatment with steroids may exacerbate Acute Retinal Necrosis due to
   HSV and other infectious mimickers of pars planitis. Not treating the herpetic eye
   condition with appropriate antiviral therapies in a timely manner could allow for rapid
   progression and loss of vision. While awaiting the results of PCR testing of a/c and
   vitreous, a systemic antiviral treatment may be initiated prior to the regional treatment
   of steroids for pars planitis, as in this case.
V. Treatment, management
   Discontinue Latanoprost
   Start Pred Forte q 15 min for the first hour O.D., then Qhr O.D.
      To reduce inflammation with anterior uveitic findings
      Topical therapy alone is not effective for direct treatment of the pars
      planitis
   Start Cyclogyl BID O.D.
   Obtain serological and radiological testing
Obtain PCR testing of the a/c aqueous humor
Start Valtrex 1000 mg TID
If Quatiferon- TB Gold and PCR is negative start the following
   a. Prednisone 60 mg po until treatment option b can be given
   b. Regional steroid injection if serological, radiological and PCR is consistent with pars planitis.
   c. Consider pars plana vitrectomy if recalcitrant and/or vision is threatened

VI. Conclusion
Topical therapies alone are not sufficient to effectively treat pars planitis. The standard of care is regional or peri-orbital steroid injection(s) in addition to treating any of the associated conditions if present. Aggressive treatment is required to prevent the associated loss of vision in 20% of cases of pars planitis. Other conditions that mimic pars planitis challenge the clinician because the initiation of the incorrect treatment modality may have serious and devastating consequences. It is imperative for the clinician to fully evaluate and differentiate pars planitis with the use of clinical, serological and radiological testing in order to effectively diagnose, treat, and manage the condition before it is able to precipitate the loss of vision.

VII. Clinical pearls
1. Topical steroids is not the standard of care for Pars Planitis.
2. Do not hesitate to order lab work or further tests to determine etiology.
3. If definitive differential diagnosis cannot be made quickly, assess if the treatment exacerbates other mimickers and act accordingly.
4. OCT is valuable not only to evaluate macular edema but also may assist in establishing the diagnosis of vasculitis with an significant increase in the retinal vessels size.

VIII Bibliography: