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Bulletin
NEXT MEETING

Tuesday February 12, 2019

Dr. Gary Gerber
To Present
How Culture Eats Strategy
For Lunch

6:00 p.m. Buffet
Sponsored By: Cooper Vision
Meeting sponsors:
NovaBay and
St. Peter’s Community Pharmacy
RSVP no later than February 7, 2019
paula@stlouisoptometricsociety.org
314 725-2020

7:00 p.m. Business Meeting

7:30 p.m. Gary Gerber, O.D.
Please join us for this informative and sure to be entertaining meeting. There WILL be some “MENTALISM” (magic), which Dr. Gerber is known for. He is one of Optometry’s “Most in demand” speakers. Please mark your calendar and plan to attend.

RSVP no later than February 7, 2019
paula@stlouisoptometricsociety.org
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COMMITTEES:
2018-2019 Committees:
Membership: Dr. Mary Beth Rhomberg
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Immunology involves the development of an immune response with the fundamental goal being to fend off foreign pathogens. Therefore, one must recognize self from non-self. Mistakes occur and this leads to hypersensitivity or autoimmunity.

**Anterior Segment Disease**

Episcleritis is the common and usually benign inflammation of episclera which lies between conjunctiva and sclera. There are two forms: simple or nodular. Simple is typically sectoral or diffuse. Nodular is more likely associated with systemic disease in about 30% of cases. It is important to note that episcleritis does not progress to scleritis. Although both conditions can be present at the same time, episcleritis does not turn into scleritis. It is not a continuum. The main difference between episcleritis and scleritis is the absence of a violaceous hue which can be appreciated with something as simple as an external light source.

Common sources of episcleritis include dry eye, collagen vascular disease and seronegative spondyloarthopathies. Dry eye is seen often and are treated with artificial tears and lubrication. Other causes include the collagen vascular diseases, such as rheumatoid arthritis, lupus, vasculitides, and Sjogrens, and spondyloarthopathies (HLA-B27 diseases), such as ankylosing spondylitis, IBD, Reiters and psoriatic arthritis.

So do you test these patients with episcleritis for underlying conditions? Typically, if the case is an initial presentation, testing may not be done. However, Dr. Durrani suggests that testing may be pursued, especially with nodular since 30% will have an underlying systemic condition. Testing may include HLA B27, Sacroiliac films, Chest X-Ray, Serum ACE, ESR and possible rheumatology referral. The tests that Dr. Durrani will order on initial nodular episcleritis presentations are an HLA B27, a chest x-ray, RPR (Syphilis) and a quantiferon gold. The other tests can be also used and should be based on the HPI of the patient as to what other tests may be useful.

Treatment for simple episcleritis may include lubrication. FML is also very effective. FML 0.1 or 0.25 % QID 1-2 weeks (low intraocular penetration and risk of IOP elevation)

One can also use oral NSAIDS as adjunct if they are still having pain or irritation (caution: GI symptoms, consider PPI).

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Ibuprofen 800 mg TID (OTC)
Flurbiprofen 100 mg TID
Indomethacin 25 mg TID

Scleritis
Anterior scleritis is far more common symptoms include a painful, red eye. Diffuse anterior/sectoral is most common, but least severe. Other forms include nodular, necrotizing and scleromalacia perforans. Necrotizing presents with extreme pain with scleral loss or damage and possible uveal protrusion and has a high association with systemic disease. Scleromalacia perforans is necrotizing scleritis without inflammation. Pain is typically absent and is usually due to vascular occlusion. Both latter forms are considered more rare but are considered emergencies due to the high risk of perforation. These conditions are best examined with an external light for the characteristic reddish/edematous appearance with violaceous hue. Other ways to discern are to use a proparacaine soaked CTA to see if the vessels move and also to see if the vessels blanch with phenylephrine.

Posterior Scleritis is much less common. A patient may present with pain, decreased vision or pain with eye movement. Symptoms may often go along with optic neuritis. Examination may reveal serous retinal detachment and/or choroidal folds. You may see striae in the perifoveal region. B-scan typically shows characteristic “T-sign” due to thickening of uveal tissue. Rheumatoid arthritis constitutes 20-33% of cases. 50% of patients have identifiable systemic disease. 15% of patients who go on to have systemic disease present with eye findings first! 90% of patients with Wegener’s die within 5 years without treatment due to systemic vasculitis. The most common causes of infectious scleritis are varicella or herpes simplex. Other causes may include post-scleral buckle, trauma, etc.

Scleritis testing should be tailored based on history. Treatment includes oral medications. Drops are contraindicated based on risk of scleral melt or global rupture.

Non-necrotizing:

- NSAIDS (non-necrotizing)
- Flurbiprofen 100 mg po TID, indomethacin 25 mg po TID, Ibuprofen 800 mg TID
- Prednisone- start at 1 mg/kg
- 25% require immunosuppressants (e.g. methotrexate)

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Necrotizing: immediate pulse dosed steroids due to risk of perforation
Scleromalacia perforans: may require grafting with donor sclera or corneoplast
Peripheral Ulcerative Keratitis (PUK) is a subflavor of scleritis and is a very rare, juxtalimbal corneal inflammation with progressive thinning and scarring of the cornea. Symptoms include pain, tearing, photophobia and redness. This is a manifestation of occult systemic disease and is associated with higher morbidity and mortality without treatment. 50% of patients have an associated collagen vascular disease, such as Wegener’s, Sjogren’s, Lupus, Relapsing polychondritis and Polyarteritis Nodosa. These patients must be treated as an eye emergency and should see rheumatology or a corneal specialist. Treatment includes pulse with IV Solumedrol 250 mg QID due to high risk of perforation, immunosuppressants in conjunction with rheumatologist. Corneal specialists will typically use cyanoacrylate glue, conjunctival recession of limbal blood vessels (controversial), and PKP.

Uveitis
Uveitis is inflammation of uveal tissue – iris, ciliary body, choroid. 10% of cases of legal blindness in the US have been attributed to sequelae of uveitis (e.g. glaucoma, cataracts, optic neuritis). Classic symptoms include redness, pain, photophobia, epiphora, floaters, and vision loss. Exceptions include JIA, Fuch’s, chronic uveitis and intermediate/posterior forms. Classification of uveitis can be broken down as follows:

- Course: Acute (isolated episode), Recurrent (subsequent episodes within 3 month period) or Chronic (uveitis lasting more than 3 months)
- Laterality: Unilateral/Bilateral
- Anatomic Location:
  - Anterior
  - Intermediate
  - Posterior
  - Pan (invvoling all locations)
- Granulomatous/Non-granulomatous – overlaps
- Infectious/Noninfectious

There are lots of complications of uveitis and those that are chronic in nature may result in vision-threatening sequelae. These may include macular edema, cataracts, glaucoma, synechiae, band keratopathy, vitreous opacities, vitreous hemorrhage, CNVM and retinal detachment. Anterior uveitis generally has a good prognosis (90% maintain 20/40 or better vision). This condition can result from various etiologies: Inflammatory, infectious, Fuchs, Pediatric and Post-surgical.

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Inflammatory uveitis

- **Clinical features:**
  - HLA B-27 Spondyloarthopathies: acute onset, fibrin, hypopyon
    - Systemic: arthritis (hip/girdle pain), GI/GU/derm symptoms
  - Sarcoïd: cough, SOB, rash

- **Testing:**
  - HLA Haplotyping: HLA B-27, HLA-B5, B51
  - Imaging: sacroiliac films, chest x-ray versus CT
  - Consider serum ACE
  - Consider Rheum consult

**HLA B27 associated uveitis**

About 40% of anterior uveitis is HLA-B27 related and 60-70% will have systemic disease. 90% of cases will be acute, recurrent and may alternate between eyes. 10% of cases will be chronic and these are the patients we worry about because they are likely to have sequelae. 25% of patients with ankylosing spondylitis. These may often be younger patients (30-40 year old men with hip/girdle/back pain). These patients should be referred to a rheumatologist.

Anterior uveitis that is not responsive to steroids may be derived from causes such as infectious, masquerade, or Fuchs.

**Infectious anterior uveitis**

- **Clinical Features:**
  - Syphilitic: +/- pupillary abnormalities (accommodate but don’t react), “great masquerader”
  - TB: Range from anterior chamber inflammation to panuveitis
  - Herpetic: unilateral; iris atrophy, elevated intraocular pressure, pigmented KP

- **Testing:**
  - Syphilitic: RPR with reflex titers (Syphilis IgG or FTA-Abs)
  - Mycobacterium TB: Quantiferon Gold / PPD
  - Herpetic: Anterior chamber paracentesis for HSV/VZV PCR if suspicious
    - HSV titer - look at IgM and IgG. If IgM is positive that can be a sign of acute disease and may want to consider putting them on Valtrex
  - Lyme Ag if suspicious
Fuchs

- Clinical Features:
  - Heterochromia, cataract, KPs (stellate - evenly distributed)
  - Blurred vision and floaters without pain, redness or photophobia
  - AC reaction, mild vitreous reaction; no CME or synechiae
  - Cataract/glaucoma/hyphema
- Testing: none specifically available, baseline workup should be considered to make sure nothing is missed
- Treatment: Generally not responsive to steroids (treat glaucoma or cataracts)
  - Possibly related to toxoplasmosis infection or rubella

Pediatric

JIA is the most common systemic disorder associated with uveitis in childhood (75% of anterior).
- Clinical signs:
  - Female, oligoarticular, young age, + ANA, RF-
  - Chronic, bilateral, nongranulomatous anterior uveitis
  - Often present with no symptoms but may have blurry vision and a mild AC reaction
- Treatment: monitor for any activity and notify specialist
  - Referral to Rheumatologist and uveitis specialist
  - Immunosuppressants - MTX, Cyclosporine
  - TNF-alpha antagonists
  - CAUTION WITH DROPS DUE TO IOP RESPONSE
- Prognosis:
  - 25% of eyes legally blind (glaucoma, cataract)

Post-surgical - first described in 1987
- Clinical Signs:
  - Delayed-onset uveitis
  - Insidious, recurrent inflammation following cataract surgery/intraocular surgery
  - Can occur anytime post-operatively
  - Most commonly occurs within first year post cataract surgery
  - Posterior capsular plaque (40-100%), granulomatous KP
  - Initially responds to steroids

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• Diagnosis/Treatment:
  o Refer to retina specialist in any patient with prolonged, recurrent anterior uveitis occurring post cataract surgery
  o Vitrectomy/capsulectomy/removal of IOL-capsular bag complex with cultures
  o Most commonly P. acnes
  o Injection of intravitreal antibiotics; prolonged topical fluoroquinolones

• Visual outcomes:
  o Relatively good
  o 20/40 or better

Intermediate uveitis can be inflammatory, infectious or due to a neoplastic/masquerade syndrome.

Inflammatory intermediate uveitis
• Isolated inflammation within the vitreous cavity, no AC or retina
  o Snowballs - white blood cell aggregates within the vitreous cavity indicative of active inflammation
• Rule of 25s: 25% require treatment, 25% require no treatment, 50% require intermittent treatment
• Prognosis: 75% of patients maintain 20/40 vision or better after 10 years
• Clinical signs and symptoms: vitreous floaters, decreased vision, may see some white blood cells floating around
• Testing: HLA B27, Chest X-ray, Serum ACE, RPR, Quantiferon Gold, +/- MRI Brain

Infectious intermediate uveitis - less common
• Testing:
  o Lyme antibodies (ELISA, Western Blot for confirmation)
  o RPR/Syphilis IgG or FTA-Abs
  o Quantiferon TB Gold

Neoplastic/Masquerade intermediate uveitis - less common
• Clinical Signs and Symptoms:
  o Age 50+ with new onset
  o 80-90% bilateral disease, although may initially present as unilateral
• Testing:
  o Vitreous biopsy (cytology, flow cytometry, IL10/IL6)
  o Systemic evaluation: MRI Brain with and without contrast, lumbar puncture
• When to treat?
  o Referral to ocular oncology – may require systemic chemotx, radiation, or intravitreal Methotrexate

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Intermediate uveitis
You should have a low threshold to refer in patients:

- 4th to 6th decades of life
- History of cancer
- Neurological signs and symptoms
- “B symptoms” (weight loss, fatigue, night sweats)
- Symptomatic floaters or decreased vision

When to treat? Use orals, not topicals

- Severe vitreous debris
- CME

Posterior uveitis
Rates of vision loss approach 50-60% with this condition.

Inflammatory
- White Dot Syndromes - some look alike, some have a viral prodrome, some get better on their own and some don’t
  - Birdshot chorioretinopathy
  - APMPPPE
  - Serpiginous
  - MEWDS
- Posterior inflammatory Choroiditis/Multifocal choroiditis
- Sarcoidosis
  - 25-50% have uveitis
  - Eye disease is initial manifestation in 20% of cases
  - Classic is granulomatous but can be non-granulomatous
  - Can present with panuveitis
  - Typically bilateral and chronic
  - ~66% anterior
  - Iris nodules (Koeppe)
  - Granulatomous
- VKH
- Behcet’s
- Vasculitis
- Sympathetic ophthalmia

Infectious
- Syphilis
- Toxoplasmosis
- Toxocariasis
- Mycobacterium associated
- Herpes virus associated – ARN, PORN
- Endophthalmitis (endogenous)

RULE OUT INFECTION first:
Check RPR/FTA-Abs/Syphilis IgG, Quantiferon TB Gold

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Endogenous endophthalmitis - must rule this out
Referral to retina specialist ASAP with any posterior findings, especially in absence of any systemic disease

Masquerade
- Lymphoma/Leukemia
- Metastases
- Melanoma/amelanotic melanoma
- Cancer associated retinopathy, Melanoma associated retinopathy

Uveitis therapy/treatment
- Local - base treatment on cellular reaction
  - Topical corticosteroids
    - Anterior uveitis (Not effective for intermediate or posterior uveitis!)
  - Caution:
    - Steroid response (generally develops within 2 weeks)
      - Utilize aqueous suppressants
      - Cataract development/progression (chronic uveitis)
  - Taper appropriately:
    - Half the dose based on how long the patient has been on the steroid drop
    - Recurrent disease may be due to tapering too quickly
  - Prednisolone acetate 1% (brand tends to be better than generic)
    - Mainstay of treatment
    - Q30 min more effective than q1h (consider loading dose) - do not be afraid to start hard with drops in an effort to control inflammation
  - Caution using Durezol: Strong steroid response particularly in children
  - Lotemax – useful for anterior uveitis, less likely to cause IOP increase
    - Good for strong steroid responders
  - Steroid responders may have elevated IOP for a month post-steroid drop treatment
  - Acute recurrent disease (< 3 months):
    - Consider that topical drops were tapered too quickly!
  - Chronic uveitis:
    - May require slow taper to 1 gtt QoD, 3x/week, etc

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Regional treatment
- Retisert – fluocinolone acetonide
- Subtenon’s triamcinolone (40 mg)
- Intravitreal Triesence (4 mg triamcinolone)
- Ozurdex implant
- High rates of glaucoma and cataract

Oral and intravenous treatment
- In eyes with bilateral inflammation not responding to topical treatment over a few weeks, may need to progress to systemic agent
- Intravenous Solumedrol 1 gm/day for 3 days
  Severe cases: concern for vision loss
- Oral Prednisone – quick onset of action
  Less risk of IOP increase and cataract
  Systemic side effects
  Initial dose – 1 mg/kg/day
  Maximum adult oral dose – 60-80 mg/day
  If starting over 40 mg/day:
    Decrease by 10 mg/week every 1-2 weeks
    40-20 mg/day: decrease by 5 mg/day every 1-2 weeks
    20-10 mg/day: decrease by 2.5 mg/day every 1-2 weeks
    10-0 mg/day: decrease by 2.5 mg/day every 1-4 weeks
  Monitor: BP/weight/glucose (advise to have checked by PCP q12 weeks)
  Supplements: Calcium 1500 mg daily and vit D 800 IU daily
  Avoid highest dose for more than 4 weeks to avoid risk of ischemic necrosis of bone

Chronic Uveitis
- 10 mg po prednisone daily generally acceptable for chronic uveitis, although ideally would like this to be lower (~5-7.5 mg by 12 weeks)

IF FLARES ON PO PREDNISONE TAPER:
- Double dose of prednisone for 2-4 weeks then must taper slowly.
  If flares 1 time then consider the fact that you initially tapered too quickly
  OTHERWISEWISE: Refer to rheumatology for immunosuppressant medications

Immunosuppressive drugs for Ocular Inflammation

*******Continued*******
The State Board is Scheduled to meet in Jefferson City February 8, 2019. We hope to have two new Board members by then.
The MOA Legislative Conference will be held January 13th and 14th in Jefferson City. Online registration is closed; however, doctors can register on-site at the conference. The Legislative Conference will be held at the DoubleTree Hilton and Millbottom, as last year. Sunday will consist of MOA Board meetings and Committee meetings, if you are a current committee member. Committee meetings will start at 1:00pm and end at 4:00pm at the Millbottom. On Monday, there will be a one-hour lecture on “Preparation for Future Legislation” presented by Daniel Carey of the AOA. Following will be two hours of continuing education on the MOA Insurance Plan and review presented by Drs. Barrett, Lake, Scullawl, and Herriott. It’s imperative doctors from across the state attend this presentation, as the MOA will not be making additional presentations. Later will be visits to the Capitol and cinnamon roll delivery (less than $5 per package, due to new rules). Attendance is important as we need to establish relationships with newly elected legislators at the Capitol, as well as fostering existing relationships. Monday evening, the Missouri Optometric Foundation is sponsoring the Legislative Reception at the Millbottom.

As a reminder, AOA Optometry’s Meeting will be held here in St. Louis from June 19th-23rd. The MOA will be hosting a Golf Tournament on Tuesday, June 18th and the MOA Leadership Retreat will be held Sunday, June 23rd, at Hilton at the Ballpark – both the MOA Board meeting and Committee meetings will take place. More details to come.

Finally, mark your calendars for the MOA Annual Convention, which will be held October 3rd-6th at Tan-Tar-A Resort in Lake of the Ozarks.
Menicon America is now offering the Miru 1 month disposable silicone hydrogel lens in spherical, toric and multifocal designs. The toric goes up to -1.75 cyl and the multifocal is near center design with a low add power only. B&L received clearance from the FDA for the Ultra multifocal toric. They are expected to be available by mid 2019.
The US Food and Drug Administration (FDA) will allow marketing of EyeBOX (Oculogica), the first noninvasive, baseline-free test to help diagnose concussions. The company plans to market the device for use in children ages 5 and older and adults up to 67 years of age, starting with a pilot launch for select, qualified sites.

EyeBOX uses eye-tracking to provide objective information that helps clinicians assess patients who have a suspected concussion with a simple, 4-minute test that does not require a baseline test.
Associate position available in a growing private practice in St. Peters. The office is ultra-modern and fully equipped with the latest technology to practice full scope optometry. LASIK co-management, specialty contact lenses, ocular pathology, glaucoma, and low vision background is a plus. Hours are flexible, no weekends, 20 to 30 hours week, benefits available. Excellent opportunity for the right candidate. Please contact me for details via email at: eappelmanod@gmail.com

UPCOMING EVENTS

NEXT MEETING
Tuesday February 12, 2019
Dr. Gary Gerber:
To Present
How Culture Eats Strategy for Lunch

Wednesday February 27, 2019
Galanis Cataract and Laser Eye Center
3-Hour CE Drury Inn-Forest Park
See Below for Information and Registration

UMSL College of Optometry
Watch for more information on upcoming CE
Continuing Education Program -
Wednesday February 27, 2019
5.30 p.m – 9.00 p.m
Drury Inn- Forest Park, 2111 Sulphur Ave.,
St Louis, MO  63139
(Highway 44 at Hampton)
● Update on Cataract Surgery and Glaucoma
● Retinal Complications of Cataract Surgery
and their Management
● Medical vs. Cosmetic uses of botulinum toxin ●
Dermal fillers: The good, the bad and the ugly
Moderators: John Galanis, M.D., Alia Durrani,
M.D., John Holds, M.D. Adam Buchanan, M.D.
3.0 hrs CE-
*change of venue with increased seating
available *
There is no cost for the program but
registration is required
Please R.S.V.P. to Diana Moellering, C.O.T.
dmoellering@drgalanis.com.
314-633-8575
Dinner and cocktails  5.30 -6.00 p.m