An Alternative Approach to Corneal and Iris Defects: Corneal Tattooing

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Course Description:

This course presents keratopigmentation (KPT), also called corneal tattooing, as another treatment option in regards to several cornea and iris defects. Indications, contraindications, and post-operative care as well as a case study are presented.

Course Learning Objectives:

- To demonstrate the history behind KPT.
- To know when to refer for corneal tattooing and when not to.
- To review post-operative side effects of corneal tattooing.
- To provide other treatment modalities besides keratopigmentation.

Outline:

1. History of corneal tattooing
2. How is it done?
   a. Superficial corneal staining/Micropuncture
   b. Lamellar intrastromal cannulas
   c. Femtosecond laser
3. What pigment is used?
   a. Historically
   b. Now
4. Other treatment and management options besides corneal tattooing
   a. Iris print contact lens
   b. Iris suturing
   c. Scleral shell
   d. Penetrating keratoplasty
   e. Enucleation
5. Indications for KPT
   a. Both cosmetic and therapeutic
   b. Iris irregularities
      1. Aniridia
      2. Colobomas
      3. Polycoria
4. Fixed mydriasis
5. Surgical
   a. Laser peripheral iridotomy
   b. Cataract surgery
   c. Corneal Irregularities
      i. Corneal scars
      ii. Leukoma (opaque corneas)
6. Contraindications for corneal tattooing
7. Symptoms of these patients
8. Side effects of corneal tattooing
9. Post-operative care
   a. Globe penetration
   b. Surgically induced epithelial defect
   c. Recurrent corneal erosions
   d. Duration and color of stain
10. Case Study
    a. 49 year old Hispanic male complains of glare with vertical ghosting OD. His ocular and medical history include: congenital iris coloboma, iris repair with cataract extraction PCIOI OU, vitrectomy OU, YAG capsulotomy OU, and corneal tattooing OD. He is interested in other options for his glare. Combined therapy options for the patient are discussed.
Course Description:
This course presents a patient case for which visual impairment led to incidental discovery of multiple combined pathologies. In following the clinical course of a lesser-known corneal pathology, the course provides a review of corneal transplantation as well as basic microbiology and immunology as they relate to corneal amyloidosis and plasma cell dyscrasia.

Course Learning Objectives:
- To recognize the clinical appearance of climatic droplet keratopathy
- To identify who may benefit from surgical intervention
- To grossly understand how to interpret pathology slides
- To know when to refer for additional laboratory testing
- To appreciate the importance of inter-professionalism

Outline:

1. Initial presentation
   a. Patient demographics
   b. Chief complaint
   c. Exam findings
   d. Differential diagnoses

2. Corneal transplantation
   a. Characteristics of the cornea which make transplantation possible
   b. Graft failure
      i. Definition
      ii. Pathophysiology
      iii. Causes
   c. Characteristics of high risk corneas
   d. Types of surgical approaches
   e. Reducing the risk of rejection
      i. Pre-, intra-, and post-operative precautions

3. Central corneal ulcer
   a. Clinical appearance
   b. Corneal culture
      i. Characteristics of Bacillus cereus
4. Climatic droplet keratopathy
   a. Definition
   b. At risk populations
   c. Clinical appearance
   d. Classification
   e. Diagnosis, treatment, and management

5. Corneal amyloidosis
   a. Biopsy and cytology slides
   b. Brief review of microbiology/immunology
   c. Significance of climatic droplet keratopathy presenting with amyloidosis
   d. Focal versus systemic amyloidosis

6. Plasma cell dyscrasia
   a. Definition
   b. Pathophysiology
   c. Ophthalmic manifestations
   d. Hematology/oncology workup

7. Takeaways
   a. Summary of patient case
   b. Importance of inter-professionalism

8. Check understanding questions
   a. Which of the following does NOT describe the demographic of patients with climatic droplet keratopathy?
      i. Middle-aged males
      ii. Living in metropolitan areas
      iii. With constant wind
      iv. And high levels of ultraviolet radiation
   b. Which TWO accurately describe the clinical appearance of climatic droplet keratopathy?
      i. Haziness and opalescence of anterior cornea
      ii. Haziness and opalescence of posterior cornea
      iii. Clear to yellow gold spherules in the subepithelium, Bowman layer, or superficial stroma
      iv. Clear to yellow gold spherules in deep stroma, Descemet layer, or endothelium
   c. How might plasma cell dyscrasia present in the eye?
      i. Corneal crystalline deposits
      ii. Subconjunctival hemorrhage
      iii. Ciliary body cyst
      iv. All of the above
   d. True or false: focal amyloidosis in the eye is commonly associated with systemic amyloidosis.
      i. True
      ii. False
AN UNCOMMON CAUSE OF CN VI PALSY
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Course Description:
This course discusses a CN VI Palsy caused by Spontaneous Intracranial Hypotension. It also presents helpful tips for diagnosing and treating CN VI palsies as well as distinguishing between potentially life-threatening causes and other, more common causes.

Course Learning Objectives:
- To review important exam elements in the diagnosis of CN VI palsy
- To discuss when further diagnostic testing (MRI/CT/bloodwork) is indicated
- To discuss potential causes of CN VI palsy
- To review the clinically relevant treatment and management of CN VI palsy, including prism and patching

Outline:
I. Case Presentation of patient with CN VI palsy cause by Spontaneous Intracranial Hypotension
   A. Patient’s presenting complaints and symptoms
   B. Other relevant exam findings
   C. Results of further testing (MRI, lumbar puncture, etc.)
   D. Treatment and management of our patient
II. Discussion on CN VI palsies
   A. Differential Diagnosis
   B. Potential causes of CN VI Palsy
      a. Vasculopathic
      b. Trauma
      c. Stroke
      d. GCA
      e. Idiopathic
      f. Other less common causes
   C. Specific discussion on Spontaneous Intracranial Hypotension
      a. What is it?
      b. What testing confirms this diagnosis?
      c. How rare is this?
   D. When is further testing indicated?
      a. Imaging such as MRI/CT
      b. Bloodwork such as ESR/DRP
      c. Testing for infectious causes such as syphilis/Lyme disease
   E. Basics of treating nerve palsies
      a. Prism vs patching vs other options
      b. Expected course and patient education
Anterior Scleritis

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Course Description
This course will discuss a case of recurrent anterior scleritis and it will include the general overview of scleritis and the treatment/management of the condition.

Course Learning Objectives

- Risk factors and etiology of scleritis
- Clinic presentation of anterior scleritis
- Complications of scleritis and managing scleritis
- Diagnosing scleritis
- Treatment options for scleritis

Outline

1. Case presentation
   a. 42 YO F-throbbing OS pain
      i. POHX scleritis OD, OS, PHMX-RA treated with Remicade & low dose prednisone
      ii. Slit lamp exam
         iii. Plan-Rx’ed prednisolone acetate OS, continue prednisone PO, RTC 1wk for f/u
   b. Follow up visits
2. Cortisol
   a. Increases: BP, blood sugar $\rightarrow$ insulin
   b. Decreases: inflammation, fertility, immune system
3. Cushing’s syndrome
   a. “Moon face”
   b. Osteoporosis
   c. Fat deposition
      i. Abdominal
      ii. Back of neck=”Buffalo hump”
   d. Hyperglycemia
   e. High susceptibility to infections
   f. Lab testing
      i. Cortisol random
      ii. 24hr urine testing
      iii. Dexamethasone testing (LDDST)
4. Scleritis
   a. Women 30-50 YO
   b. 40%-50% associated with connective tissue/autoimmune disease
      i. RA, lupus, polyarteritis nodosa, Wegener granulomatosis, polychondritis.
c. 4%-10% infectious
   i. Herpes zoster=most common (8% of cases)
d. 50% idiopathic
e. Anterior segment= most common
   i. 4 types
      1. Diffuse
      2. Nodular
      3. Necrotizing
         a. With inflammation
         b. Without inflammation

5. Signs and symptoms
   a. Pain-deep boring ache
   b. Photophobia
   c. Inflammation
   d. Focal/diffuse
   e. Raised nodule, avascular area
   f. Posterior scleritis is less common, less likely to cause red eye, more likely to cause blurred/decreased vision.
   g. Perforation of the globe

6. Complications
   a. 14% lose significant VA within 1 yr, 30% within 3 yr.
   b. Necrotizing scleritis & underlying systemic vasculitis mortality rate: 50% in 10 yrs (MI).
   c. Perforation
   d. Permanent scarring
   e. Debilitating chronic pain
   f. Cataract/glaucoma from corticosteroid use.

7. Diagnosing
   a. Slit lamp exam
   b. Testings-anything causing inflammation
      i. ANA, HLA-B27, sed rate, C-reactive protein, CBC, chest x-ray/CT, syphilis, ANCA
   c. Infectious scleritis=smears or rarely biopsies
   d. CT/ultrasonography may be needed for posterior scleritis
      i. T-sign with B-scan

8. Treatment
   a. Initial therapy=systemic corticosteroids
      i. If inflammation returns,-longer dose, may consider pulsed IV corticosteroid
      ii. NSAID for mild cases. (Diffuse & nodular)
      iii. Dosages vary
      iv. Chronic use can cause Cushing’s syndrome
   b. Systemic immunosuppressants
   c. 67% required high-dose glucocorticoids or combo of high-dose glucocorticoids & immunosuppressive
   d. Scleral grafts for threatened perforation.
Dacryocystitis- Multiple Forms of Clinical Presentation, Multiple Methods of Treatment

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Course Description:
A case series of acquired dacryocystitis in adults followed by a discussion of etiology, prevalence, likely pathogens, and therapeutic and incisional treatment options.

Course Learning Objectives:
At the conclusion of this lecture, attendees will be able to:
- Identify common causes and pathogens of dacryocystitis
- Identify multiple treatment options for dacryocystitis
- Feel comfortable managing different presentations of dacryocystitis to completion

Outline:

Case One
  a. Demographics
  b. Entrance exam
  c. Deferential diagnosis
  d. Exam findings
  e. Diagnosis and treatment
  f. Follow ups

Case Two
  a. Demographics
  b. Entrance exam
  c. Deferential diagnosis
  d. Exam findings
  e. Diagnosis and treatment
  f. Follow ups

Dacryocystitis
  a. Nasolacrimal system anatomy
  b. Differentials
  c. Etiology
  d. Incidence/Prevalence
  e. Treatment
  f. Follow ups

Conclusions/Take home points
Title:
Treatment, Management, and Consideration of Bilateral Uveitis in a Previous I.V. Drug User

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Course Description:
The purpose of this course is to act as a refresher for the diagnosis and treatment of uveitis, ordering blood work, as well as special considerations for a patient with a history of I.V. drug abuse.

Learning Objectives:
- To review clinical decision making and differentials in regard to uveitis
- To review diagnostic testing and bloodwork
- To review potential complications with a complex uveitis

Outline:
1. Case Introductions
   a. CC and history
   b. Entrance testing
2. Uveitis Review
   a. Unilateral vs. Bilateral
   b. Granulomatous vs. Non-granulomatous
   c. Anterior vs. Posterior
   d. HLA-B27
3. Case Differentials before testing
4. Bloodwork Review
   a. Baseline blood work
   b. Special considerations with I.V. drug use
5. Treatment review
   a. Medications and dosage
   b. Follow-up
   c. Initial treatment of example case
6. Follow-up #1
   a. Testing
   b. Bloodwork Results
   c. Case considerations
7. Differentials after testing
   a. Follow-up
8. Follow-up #2
9. Retinal Consult #1
   a. Testing
   b. Plan
10. Cornea Consult
    a. Testing
    b. Uveitis cataract discussion
    c. Anterior synechiolysis
11. Retinal Consult #2
    a. Testing
    b. Vitreous tap discussion
    c. Vitrectomy and membrane peel discussion
12. Post-op #1
    a. Surgery discussion
    b. Testing
    c. Endophthalmitis discussion
    d. Tractional retinal detachment discussion
13. Post-op #2
    a. Testing
    b. Cataract surgery considerations
14. Conclusion
    a. Take home points
Why Wait? Treating Central Serous Chorioretinopathy with Eplerenone

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Description
This presentation introduces central serous chorioretinopathy and addresses treatment of the condition with eplerenone.

Learning Objectives
- The proposed pathophysiology of central serous chorioretinopathy.
- The use of imaging to in diagnosing and managing central serous chorioretinopathy.
- The mechanism of action of eplerenone.
- The treatment of central serous chorioretinopathy with eplerenone

Outline
I. Case studies
   a. Patient history
      i. Chief complaint
      ii. Demographics
      iii. Health history
   b. Pertinent exam findings
      i. Visual acuity
      ii. Posterior segment
   c. Imaging
      i. Fundus photography
      ii. Fundus autofluorescence
      iii. OCT
II. Central serous chorioretinopathy
   a. Description/definition
   b. Epidemiology
   c. Risk factors
   d. Symptoms
   e. Signs
   f. Pathophysiology
   g. Diagnosis
   h. Treatment options
III. Eplerenone  
   a. Mechanism of action  
   b. Correlation to central serous chorioretinopathy  
   c. Dose  
   d. Side effects  

IV. Consider Treatment and Follow Up of Cases you Presented Here  
   a. Treatment  
   b. Follow-up  
      i. 1-week follow-up  
      ii. 1-month follow-up  
      iii. Additional follow-up  

V. Summary Conclusion
CHOROIDAL NEOVASCULAR MEMBRANE SECONDARY TO CHRONIC CENTRAL SEROUS RETINOPATHY

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Course Description:
This course goes over the pathophysiology behind these conditions, how they can be related to one another as well as current technology, follow up care and treatment.

Course Learning Objectives:
• To go over the conditions in detail to get a deeper understanding of the underlying process
• To go over the different technological advances that have helped with diagnoses
• To discuss different theories/mechanisms
• To go over the different treatment options/follow up care

Outline:
 Lets start by going over the process of both conditions, what is a choroidal neovascular membrane?
  - New blood vessels growing from beneath the retina (choroid) that eventually break through into the retina that may cause varying amounts of visual disturbances
  - Can happen from a lot of disease processes such as chronic central serous retinopathy, AMD, histoplasmosis, trauma, pachychoroid conditions, etc.

 Next we will go over what is central serous retinopathy?
  - Fluid buildup separating the neurosensory retina from the RPE/choroid
  - Associated with multiple etologies such as stress, type A personality, corticosteroid use, hypertension, sleep disturbances, bacteria

 Technology
  - Optical Coherence Tomography: uses light waves to take cross sectional scans of the posterior segment of the eye. Enhanced depth imaging (EDI) can also help giving a more detailed view of the choroid because this structure can be effected in many pathologies including CSR and CNVM. Central Serous appears as a blister-like swelling between the photoreceptor and RPE layers. Choroidal Neovascular Membrane appears as fluid coming from the choroid spilling into overlying layers of the retina with irregularities to the outer retina layers.
  - OCT Angiography: uses the normal movement of red blood cells in the retinal capillaries to generate flow imaging. Helpful for diagnosis CNVM and easier/less invasive then fluorescein angiography. Can also use en face OCT a to get a more detailed view of the choroid
  - Fundus Autofluorescence: uses blue-light excitation to visualize melanin/lipofuscin in the retina. Helps to identify/locate outer retina/RPE abnormalities based off their reflectance.
  - Fluroscein Angiography: more invasive method that uses Fluorescein but is helpful for detecting areas of leakage throughout the retina.
  - Indocyanine Green Angiography: more invasive method that uses IV Indocyanine Green. Can be performed in addition to FA and helps highlight changes throughout the choroid.
Treatment
- Central Serous Retinopathy:
  - Usually self limiting (3-4 mos)
  - Topical NSAIDs, photodynamic therapy, focal laser, steroidal anti-mineral corticoids, Anti-VEGF

- Choroidal Neovascular Membranes:
  - Anti-VEGF, thermal laser, PDT

Case Study
57 year old Native American female presents to the office with complaints of grey shade to vision OD x 2 days
Visual Acuity 20/30 OD, 20/25 OS
All chair skills normal
Refraction improves vision 20/25 OD, 20/20 OS
Anterior Segment unremarkable except mild nuclear sclerosis
IOP 14/14
Posterior Segment
OD: Central Serous Retinopathy
OS: Unremarkable

Assessment:
Central Serous Retinopathy OD

Plan:
Monitor x 1 month.

Pt returns 1 month later, resolving
Plan:
RTC for CEE/DFE/Mac OCT

Pt returns 2 months later, resolving

Plan:
RTC 1 month for undilated Mac OCT (due to irregular outer retina abnormalities)

Pt returns 1 month later with CNVM, with CSR mostly fully resolved
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**Diagnosis and Management of Vertical Misalignment**

A case series in the optometric management of various vertical ocular deviations will be presented. This lecture will review identification of patients to be tested for vertical misalignment, as well as diagnosis and treatment of specific vertical deviations, including indications for non-optometric work-up.

**Course Objectives:**
- To recognize which patients should be tested for vertical misalignment
- To review clinical tests for diagnosis of vertical misalignment
- To review etiologies of vertical misalignment
- To understand non-optometric work-up, and when to employ it, for patients with vertical deviations
- To review treatment options for patients with vertical deviations

**Outline:**

A. Common symptoms of patients with vertical deviation:
   - Eye strain
   - Fatigue
   - Diplopia
   - Skipping lines

B. Clinical testing review
   - Cover test
   - Maddox Rod
   - Wesson Card

C. Etiologies
   - CN IV palsy
   - Skew Deviation
   - Phoria

D. Further work-up?
   - Acute onset
   - Neurological symptoms
   - Vergence ranges

E. Case Studies
   - 29 yo female, new patient, chief complaint of diplopia and headaches
     i. Onset: 3-4 months ago, when started new office job
     ii. Negligible refractive error, unremarkable ocular health
     iii. Cover test:
        1. Distance: orthophoria with 2pd L hyperphoria
2. Near: 8-10pd exophoria with 2pd L hyperphoria
   iv. CISS: 27
   v. NPC: 1st attempt: 5cm/8cm; 3rd attempt: 8cm/10cm
   vi. Convergence Ranges: Distance: 4/8/4; Near: 8/12/8
   vii. Maddox Rod: 0.5pd L hyper, bigger in R gaze and with L tilt; no vertical
deviation in supine position
   viii. Double Maddox Rod (red over OS): white line straight; red line diagonal
(down left to up right); Interpretation: OS incyclotorsion
   ix. Wesson Card: 4pd BI; no vertical
   x. Diagnosis: Skew Deviation, Convergence Insufficiency (CI)
   xi. Treatment: 4pd BI prism, split, for computer use. Systemic work-up?
   xii. 4 week follow-up: subjective complaints had decreased significantly, and
her CISS score was 9

b. 9yo female, new patient
   i. *exact clinical data being acquired from site of rotation in fall 2019*
   ii. Congenital CN IV palsy

c. 43yo male *subject to change*
   i. Presented in for diplopia s/p TBI in 2013
   ii. h/o LET, s/p corrective surgery at age 12
   iii. Cover Test:
       1. Distance: 18-20pd CLXT with 6pd L hypertropia
       2. Near: 16-18pd CLXT with 6pd L hypertropia
   iv. Treatment: vision therapy, since patient is averse to further surgery
   v. During VT: much improvement in horizontal fusional vergence with 5pd
BD OS
       1. Discussion: correction of vertical often enough to overcome
horizonal
       2. Can be applied to patients with smaller angle horizontal
strabismus
Course Description:
This presentation follows the optometric management course of two adult patients who presented with an abducens nerve palsy. Each patient experienced different symptoms and recovery courses before their final resolution of the abducens nerve palsy. This presentation will address the management of the variable nature of this condition and the questions encountered throughout the recovery process.

Course Objectives:
1. Understand how to identify an abducens nerve palsy.
2. Be confident in ordering the appropriate neurological and blood workup for abducens nerve palsies.
3. How to fit a Fresnel prism lens and when to pursue other treatment options.
4. Learn various presentations throughout the course of abducens nerve palsies.
5. Manage patient symptoms and expectations throughout the healing process.

Outline:
1. Neuroanatomy of abducens nerve pathway
   a. Diseases that commonly affect abducens pathway
      i. Prevalence and incidence
      ii. Typical prognosis and progression of abducens nerve palsies
   b. Other neurological presentations that may be associated with abducens nerve palsies
2. Presenting signs and symptoms of an abducens nerve palsy
   a. Additional history questions to ask
   b. Procedures to be done before dilation
   c. Considerations post-mydrasis
3. Ordering neuroimaging and blood work
   a. MRI vs MRA vs CT
   b. Lab work
   c. When to order and with what urgency
4. Fitting the Fresnel prism lenses
   a. Free-space fusion testing
   b. Selecting the eye to place the prism
   c. Application of the Fresnel prism
   d. Patient education of care for prism
   e. Follow-up schedule
   f. When to consider options beyond Fresnel prism (surgery, ground-in prism)
5. Case reports of two patients with well-controlled diabetes who presented with an abducens nerve palsy that was concluded to be idiopathic after full neurological workups
   a. 34-year-old Filipino woman
      i. Initial visit: 6\(^\text{A}\) constant alternating esotropia
      ii. Three weeks later: 35\(^\text{A}\) constant left esotropia (CLET)
      iii. Neurological symptoms throughout course
      iv. Ocular motility exercises to prevent contracture
      v. Gradual resolution within four months
   b. 44-year-old Caucasian male
      i. Initial visit (two months after onset): 25\(^\text{A}\) CLET
      ii. Three weeks later: comitant 20\(^\text{A}\) CLET
      iii. Sudden resolution within four months
      iv. Possible non-diabetic etiology of palsy

6. Conclusions
   a. The magnitude of an esotropia may increase before it decreases.
   b. A comitant deviation may still be healing.
   c. Each abducens nerve palsy heals on a different course.
   d. Fresnel prism is easy to use and is effective.
Five o’clock Shock: End of Day Ocular Emergencies and Urgencies

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Course Description:
This course will discuss a variety of ocular emergencies and urgencies that may walk through your door before closing time. Our cases will cover diagnostic considerations, management recommendations, pertinent timelines of when to refer to optimize prognosis. We will also review clinical pearls for managing complex cases while racing against the clock.

Course Learning Objectives:
• To identify various ocular emergencies and how to properly manage them
• To recognize when lab testing and/or imaging is warranted
• To understand when a referral is indicated, who to refer to, and referral timeline versus in-office management
• To understand current treatment modalities for various ocular conditions in order to ensure the best prognosis

Outline:
I. Introduction
II. Case 1 – Endophthalmitis after cataract surgery
   a. 62-year-old Native American male presents as walk-in for right eye pain and sudden decrease of vision 3 days post-op PCIOL repositioning surgery
      i. BCVA: OD 20/HM, OS 20/20
      ii. Ocular Health History:
         1. CE PCIOL OU
         2. Dislocated IOL s/p IOL repositioning OD x 2
         3. Retinal Tear s/p cryo/laser OS
      iii. Medical History:
          1. Type 2 diabetes, hyperlipidemia, kidney transplant recipient, CVA, syphilis
   b. Anterior segment photos and posterior segment findings
   c. Clinical presentation
      i. Signs/symptoms
      ii. Pertinent clinical tests and observations during gross clinical assessment
   d. Differentials: TASS vs. Endophthalmitis
   e. Pathology and risk factors
      i. Common culprits
      ii. Prophylaxis antibiotics – intracameral vs. topical
   f. General management/treatment
      i. Clinical exam
      ii. Referral – who and when
      iii. Treatment options, sequencing, and timeline
         1. Vitreous Tap
2. Intravitreal injections
3. Vitrectomy
4. End stage management
   iv. Prognosis

g. Case summary and clinical pearls

III. **Case 2 – Open Globe Injury**
a. 30-year-old Native American male, recently discharged from the hospital after being stabbed in the right eye, presents as walk-in for ocular evaluation
   i. VA: OD LP, OS 20/40
   ii. Ocular history: unremarkable prior to open globe injury
   iii. Medical history: unremarkable

b. Anterior segment photos and SLE findings
c. Open globe injury (OGI) overview
   i. Terminology and classification
   ii. Ocular trauma score
   iii. Signs/symptoms
d. Complications of OGI/laceration
   i. Traumatic cataract, traumatic endophthalmitis, retinal detachment, proliferative vitreoretinopathy, wound leak, and etc.
e. General management/treatment
   i. Work up: TA, Seidel test, B-scan
   ii. Protective eye shield
   iii. Patient education
   iv. Oral and topical treatment
   v. STAT referral for surgical repair
      1. Who to refer to and when
   vi. Surgical Repair – sutures, vitrectomy, RD repair, lensectomy, etc.
f. Prognosis
g. Case summary and clinical pearls

IV. **Case 3 – Third Nerve Palsy**
a. 75-year-old Native American female presents as walk-in for evaluation of double vision
   i. BCVA: OD:20/20-, OS: 20/20-
   ii. Ocular history: mild NPDR OU, mild anisocoria OD>OS, low tension glaucoma, multiple CN VI palsy (vasculopathic, resolved), PCIOL OU, Bells Palsy
   iii. Medical history: Type 2 diabetes, hypertension, hyperlipidemia, herpes zoster
   iv. Initial clinical presentation and subsequent visits
b. Diplopia and CN III palsy overview
   i. Case history
   ii. Signs and symptoms
      1. Complete vs. incomplete
      2. Pupil involving vs. pupil sparing
   iii. Anatomy of CN III
c. Etiology – microvascular, aneurysm, traumatic, etc.
d. Differentials
   i. Motility restriction
   ii. Ptosis
   iii. Anisocoria
e. Aberrant regeneration
f. General management and treatment
   i. Work up
   ii. Lab testing and imaging
   iii. Referrals – who and when
   iv. Treatment
      1. Underlying etiology
      2. Diplopia
   v. Follow-up timeline
   vi. Prognosis

g. Case summary and clinical pearls

V. Case 4 – Bilateral Disc Edema
   a. 20-year-old Native American female presents as walk-in for headache evaluation
      i. BCVA: OD 20/20, OS 20/20
      ii. Ocular history: unremarkable
      iii. Medical history:
          1. Pulmonary Embolism
          2. Seizure Disorder
          3. Antiphospholipid antibody syndrome
          4. Pregnancy-preterm, C section
   b. Clinical tests to aid in diagnosis (initial and following visits)
      i. Fundus photos
      ii. HVF 24-2
      iii. OCT RNFL/Line
      iv. B scan
      v. FAF
   c. Differentials for bilateral disc edema
      i. Signs and symptoms
   d. Pseudopapilledema vs Papilledema
   e. Differentials for this patient
      i. IIH
         1. Definition
         2. Risk Factors
         3. Characteristics
         4. Referral to neurology
         5. Imaging
         6. Lumbar puncture
            a. Normal value vs abnormal value
      ii. Venous Sinus Thrombosis
         1. Risk Factors
         2. Neuro associated symptoms
         3. Referral
         4. Imaging
         5. Anatomy
   f. Classifications of Papilledema
   g. General management and treatment
      i. Referral timeline and to who
      ii. Testing ordered
         1. MRI/MRV
h. Case summary and clinical pearls
   i. Headache Questionnaire

VI. Case 5 – Hypotony Maculopathy
   a. 21-year-old Native American female presents as walk-in for sudden blurry vision in the right eye after blunt trauma 1 week prior
      i. BCVA: OD 20/400 PH 20/40, OS 20/40 PH 20/25
      ii. Ocular History
          1. Lattice degeneration OU
          2. Retinal Hole OD
      iii. Medical History: unremarkable
   b. Clinical Tests to aid in diagnosis
      i. IOP
      ii. Gonio
      iii. OCT macula/RNFL
      iv. B scan
   c. Definition of hypotony
   d. Causes
      i. Over filtration after glaucoma surgery
      ii. Blunt Trauma/penetrating/perforation/open globe
      iii. Cyclodialysis cleft
   e. Prognosis
   f. Treatment for traumatic hypotony maculopathy
      i. Localize the cause
      ii. Appropriate referrals
      iii. Treatment options
         1. Clefts less than 4 clock hours
            a. Topical med
            b. Laser
         2. Clefts more than 4 clock hours
            a. Cryopexy
            b. Scleral flap
      iv. Follow up
      v. Management of IOP spike after cleft repair/resolution
   g. Case summary and clinical pearls

VII. Conclusion
VIII. Questions
Sickle Cell Retinopathy with Secondary Tractional Retinal Detachment

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Course Description:
This course presents an overview and management options for patients with sickle cell retinopathy and will discuss a case regarding a patient with advanced sickle cell retinopathy.

Course Learning Objectives
- to review background information about sickle cell disease: etiology, clinical implications, treatment options, etc.
- to review epidemiology of sickle cell retinopathy based on genetic risk factors
- to explore the pathophysiology of the ocular involvement of sickle cell
- to identify and stage characteristics of sickle cell retinopathy using Goldberg’s classification
- to review management, treatment and prognosis for sickle cell retinopathy regarding optometrists, ophthalmologist and primary care physicians
- to review new ocular options for sickle cell retinopathy

Outline
I. Background
- Background information
  o Caused by point mutation in hemoglobin-β resulting in abnormal hemoglobin-S
    ▪ Point mutation results in the replacement of position 6 glutamic acid with valine.
  o Effect of hemoglobin on red blood cell
    ▪ Erythocyte rigidity, hemolysis, and vaso-occlusion
  o Clinical implications
    ▪ Anemia, vasculopathy, sickle cell crisis
    ▪ Stroke, acute chest syndrome, pulmonary hypertension, etc.
  o Average life expectancy: 40-50 yrs
  o Treatment options
    ▪ Hydroxyurea, blood transfusions, L-glutamine, hematopoietic stem cell transplant

II. Demographics
- Highest in those of African decent
- American Statistics
  o Prevalence of Sickle Cell Trait: 8-10%
  o Prevalence of Sickle Cell Disease: 0.2-0.4%
  o Prevalence of Proliferative Sickle Cell Retinopathy:
    ▪ Sickle Cell Anemia(HbSS): 12.0-54.6%
III. Pathophysiology of ocular involvement
- The mechanism of retinopathy
- Inflammatory cascade leading to retinal ischemia and vascular hypoperfusion
- Overall pathophysiology of sickle cell retinopathy.

IV. Goldberg’s classification of retinopathy and unique features
- Non-proliferative Sickle Cell Retinopathy:
  o Stage I: Peripheral arterial occlusion
  o Stage II: Peripheral arteriovenous anastomoses (hairpin loop)
- Proliferative Sickle Cell Retinopathy:
  o Stage III: Sea fan neovascularization, proliferative
  o Stage IV: Vitreous hemorrhage
  o Stage V: Tractional retinal detachment
- Other Unique Signs
  o Salmon patch retinal hemorrhages
  o Iridescent spots
  o Black sunburst- pigmented lesions with speculated edges
  o Angoid streaks

V. Treatment, Management and Prognosis
- Treatment
  o Retinal detachment treatment: vitrectomy, pneumatic retinopexy, laser photocoagulation, cryotherapy, scleral buckle
  o Neovascularization: Pan-retinal photocoagulation, intravitreal anti-VEGF
  o New systemic options: stem cell transplantation and gene therapy
- Management
  o Primary Care
    ▪ Run blood panels
    ▪ Manage blood pressure
  o Optometry
    ▪ No retinopathy: annual dilated eye exam
    ▪ Retinopathy: Three to six months for dilated exam
    ▪ Referral to Retina specialist for treatment based on degree of retinopathy
- Prognosis
  o Visual prognosis dependent on level of retinopathy and intervention
  o No systemic cure

VI. Case Study
- Patient Profile
  o 36 year old African-American male
- Chief Complaint
  o Flashes of light OS that started two weeks ago accompanied by floaters and veil in inferior temporal visual field
- Ocular History
  o Intermediate/posterior uveitis OU
  o Sickle cell retinopathy OS>OD
- Attempted pan-retinal photocoagulation OS but was unable to tolerate and was lost to follow up

- **Medical History**
  - (+)Sickle cell trait
  - Essential hypertension

- **Medications/Allergies**
  - None

- **Clinical Findings:**
  - **Visual Acuities**
    - OD: 20/25 PH 20/20
    - OS: 20/50 PHNI
  - **Confrontation Fields**
    - OD: Full to finger counting in all four quadrants
    - OS: Inferior nasal visual field miss
  - **Optic Nerve Head**
    - OD: 0.2 round
    - OS: 0.2 round with fibrovascular tissue extending into vitreous
  - **Macula/Posterior Pole**
    - OD: Normal
    - OS: Tractional retinal detachment superior temporally sparing the fovea
  - **Peripheral Fundus:**
    - OD: (-)holes/tears, (+)fibrovascular tissue temporally
    - OS: (-)holes/tears, (+)sea fan neovascularization superiorly
  - **Auxiliary Testing**
    - Spectral Domain-Macula Optical Coherence Tomography
      - OD: normal foveal contour/macular profile
      - OS: disrupted foveal contour with retinal detachment sup-temp to fovea, macula ON
    - Fundus Photography
  - **Retina Management:**
    - Treat active neovascularization with intravitreal Avastin
    - Proceed with retinal detachment repair
    - Lower blood pressure

- **Laboratory Findings**
  - (+)HbSC

- **Differential diagnoses**
  - **Primary:**
    - Sickle cell retinopathy with secondary tractional retinal detachment
      - (+)Sickle cell trait
  - **Others:**
    - Rhegmatogenous retinal detachment secondary to retinal break
      - History of posterior uveitis
    - Hypertensive retinopathy with neovascularization and secondary tractional retinal detachment
      - Blood Pressure: 162/93

- **Management**
- Stat referral to retinal specialist
  - Retinal Specialist
    - Visit 1
      - Active retinal neovascularization (NVE)- treated with avastin injection
      - Tractional retinal detachment encroaching superior temporal fovea (macula on)- repair is contraindicated due to active NVE
      - Return for pars plana vitrectomy, internal limiting membrane peel and panretinal photocoagulation laser and retinotomy with possible oil tampanode in 1 month
    - Visit 2
      - Dorman retinal neovascularization (NVE)
      - Tractional retinal detachment encroaching superior temporal fovea (macula on)- repair is indicated however patient declined surgical intervention. Monitor in 3 months
Case Report: Neovascular Glaucoma

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Course Description:
This course presents a case report on a patient with primary open angle glaucoma OU who developed neovascular glaucoma OS. This lecture will review the condition of neovascular glaucoma and provide a full presentation on patient’s diagnosis, treatment, and management of ocular condition before and after referral to a glaucoma specialist.

Course Learning Objectives:
• To provide an overview of neovascular glaucoma
• To review the etiology, pathophysiology, diagnostic criteria, management, and treatment of the ocular condition
• To provide a better understanding of the course of neovascular glaucoma with a case report on neovascular glaucoma from diagnosis to treatment and management by glaucoma specialist

Outline:
Background
I. Introduction on neovascular glaucoma
   a. Neovascular glaucoma is a sequel of ocular ischemic conditions that result in release of vascular endothelial growth factors, leading to neovascularization of iris and angle
   b. Sequel of ocular ischemic conditions:
      i. Retinal vein occlusion
      ii. Proliferative diabetic retinopathy
      iii. Carotid artery occlusion
      iv. Central retinal artery occlusion
      v. Intraocular tumor
      vi. Chronic retinal detachment
      vii. Chronic/severe ocular inflammation
      viii. Ocular ischemic syndrome
      ix. Anterior segment ischemia
      x. Sickle cell retinopathy
      xi. Retinopathy of prematurity
   c. Epidemiology
      i. Prevalence, incidence
      ii. Age
   d. Pathophysiology
      i. Retinal ischemia (97%)
      ii. Inflammation without retinal ischemia (3%)
   e. Stages of neovascular glaucoma
      i. Early stage (rubeosis iridis)
ii. Secondary open angle glaucoma  
iii. Advanced stage 
f. Signs/symptoms  
   i. Reduced visual acuity  
   ii. Eye pain  
   iii. Very high IOP (> ~60 mmHg)  
   iv. Corneal edema  
   v. Aqueous flare  
   vi. Severe rubeosis iridis  
   vii. Distortion of pupil  
   viii. Synechial angle closure  
g. Risk factors  
h. Diagnostic criteria  
i. Treatment methods  
   i. Panretinal photocoagulation (PRP)  
   ii. Endophotocoagulation  
   iii. Panretinal cryotherapy  
   iv. Goniophotocoagulation  
   v. Anti-VEGF agents via intravitreal injection  
   vi. IOP lowering topical agents  
   vii. Trabeculectomy  
   viii. Antifibrotic agents  
   ix. Valve implant surgery  
   x. Cyclodestructive procedures  
      1. Micropulse transscleral cyclophotocoagulation  

Case Report  
II. 90 year old male with neovascular glaucoma OS secondary to central retinal vein occlusion of left eye.  
III. Case History  
   a. Patient demographics: 90 year old male  
   b. Ocular history:  
      i. Primary open angle glaucoma (dx: ~2000)  
         1. s/p SLT OS x 2009  
         2. Current ocular medications:  
            a. Simbrinza tid OU  
            b. Xalatan qhs OU  
      ii. DM Type 2 with moderate NPDR OU  
      iii. s/p CE/PCIOL OU  
      iv. ERM OS  
      v. Peripheral hemorrhages OU  
   c. Medical history:  
      i. Chronic vascular insufficiency of intestine  
      ii. Gastritis  
      iii. Non-toxic multinodular goiter  
      iv. Thyroid nodule
v. History of prostate seed brachytherapy
vi. Diabetic neuropathy
vii. Back pain
viii. Congestive heart failure
ix. Obstructive sleep apnea syndrome
x. Coronary artery disease
xi. Hypertension
xii. Hyperlipidemia
d. Medications:
   i. Bisacodyl 5 mg tab
   ii. Lancet, softclix
   iii. Lidocaine 5% ointment
   iv. Simvastatin 10 mg tab
   v. Losartan 25 mg tab
   vi. Insulin, detemir 100 unit/ml flextouch 3 ml
   vii. Prednisolone acetate 1% ophthalmic suspension
   viii. Lactobacillus acidophilus cap/tab
   ix. Netarsudil 0.02% ophthalmic solution
   x. Potassium chloride 10meq sa tab
   xi. Furosemide 40 mg tab
   xii. Aspirin 81 mg tab
e. Allergies
   i. Hydrocodone
   ii. Insulin (novolog flexpen)
   iii. Amoxicillin
   iv. Acarbose
   v. Lisinopril

IV. Visit 1: 07/21/2019
a. Chief complaint:
   i. Left sided headache x 10 days
   ii. Pain 9/10
   iii. Blurry vision OS
b. Clinical findings:
   i. Corrected visual acuity: OD: 20/30 (ph 20/25-3); OS: 20/150 (ph 20/80-)
   ii. Pupils equal round and reactive to light; (+)APD OS
   iii. Confrontations: FTFC OD, OS
   iv. IOP #1: OD 24 mmHg; OS 54 mmHg
   v. IOP after in office: Paracentesis OS, Apraclonidine OU, Brinzolamide OU, Diamox 500 mg po: 22 mmHg OS
   vi. Gonioscopy:
      1. OD open and normal
      2. OS open to TM, (+)NVA
   vii. Dilated fundus exam:
      1. OD: 0.6r c/d
2. OS: 0.6r c/d; edema superior; dot-blot hemes and flame hemes inferior and superior

c. Diagnosis:
   i. Neovascular glaucoma OS secondary to retinal vein occlusion OS

d. Treatment:
   i. Start:
      1. Xalatan qhs OU
      2. Simbrinza tid OU
      3. Diamox 500 mg po
   ii. Referred to glaucoma specialist and retinal specialist

V. Visit 2: 07/29/2019
   a. Chief complaint:
      i. Unbearable pain OS
      ii. Patient seen with glaucoma specialist and surgery scheduled for 08/08/2019 per patient
   b. Current ocular medications (per patient report):
      i. Xalatan qhs OU
      ii. Pred forte q6hr OS
   c. Clinical findings:
      i. Corrected visual acuity: OD: 20/30+2; OS: 20/400 (phni)
      ii. Pupils equal round and reactive to light; (+)APD OS
      iii. Confrontations: FTFC OD, OS
      iv. IOP: OD 18 mmHg; OS 62 mmHg
   d. Plan:
      i. Restart simbrinza tid OU
      ii. Continue xalatan qhs OU, diamox 500 mg po
      iii. Follow up appointment with glaucoma specialist scheduled on 08/08/2019

VI. Visit with glaucoma specialist: 08/06/2019: post-op #4day
   a. Micropulse OS x 08/02/2019
   b. Current ocular medications:
      i. Simbrinza tid OS
      ii. Xalatan qhs OS
      iii. Pred forte qid OS
   c. Clinical findings:
      i. Corrected visual acuity: OD: 20/30-2; OS: CF @5 ft (phni)
      ii. Pupils equal round and reactive to light; (+)APD OS
      iii. IOP: OD 23/24 mmHg; OS 28/29 mmHg
      iv. Iris: OS (+)NVI
      v. Anterior chamber: OS light 1+ cell
   d. Plan: RTC on 09/06/2019

VII. Visit with glaucoma specialist: 09/06/2019 – post-op #3week
   a. Current ocular medications:
      i. Simbrinza tid OS
      ii. Xalatan qhs OS
      iii. Pred forte qid OS
b. Clinical findings:
   i. Corrected visual acuity: OD: 20/30; OS: CF @3 ft (20/400)
   ii. Pupils equal round and reactive to light; (+)APD OS
   iii. Confrontations: FTFC OD, OS
   iv. IOP: OD 21/22 mmHg; OS 31/32mmHg
   v. Iris: OS (+)NVI
   vi. Anterior chamber: OS deep and quiet

c. Plan: RTC in 3 weeks

VIII. Discussion
   a. Referral time period
   b. Micropulse transscleral cyclophotocoagulation method vs other treatment procedures

IX. Conclusion

X. References
Course Description:
This course presents a template for the primary care practitioner on how to manage chorioretinitis secondary to acquired syphilis. Syphilitic chorioretinitis is difficult to distinguish from Acute Retinal Necrosis (ARN). Timely treatment of possibly coinciding infections is imperative to preventing severe vision loss. Syphilis, ARN, appropriate treatments, and laboratory studies are discussed.

Course Learning Objectives:
- To review the etiology and ocular complications of Syphilis
- To recognize the clinical presentation of ARN
- To know the initial management of chorioretinitis
- To review appropriate laboratory procedures to order

Outline

I. Background information of Syphilis
   A. Etiology
      1. Sexually transmitted disease caused by the spirochete, *Treponema pallidum*
   B. Epidemiology
      1. 30,644 new cases of syphilis in 2017
      2. 72.7% rate increase compared with 2013
   C. Non-ocular manifestations
      1. Primary: painless chancre at inoculating site
      2. Secondary: rash of palms and soles, fever & malaise
      3. Tertiary: neurological & cardiovascular manifestations
      4. Latent: clinically undetectable
   D. Ocular manifestations
      1. Uveitis (anterior, posterior, pan), retinitis, papillitis, scleritis, vitritis
      2. Syphilitic uveitis highest among men aged 37-58
      3. HIV coinfection and immunocompetence risk
   E. Ocular complications
      1. Retinal detachment, retinal atrophy, optic atrophy, CME, macular holes, ERM
   F. Standard of care:
      1. IV benzyl penicillin at 12-24 million units per day (3-4 million units q4h) for 10-21 days
   G. Laboratory procedures:
      1. No gold standard--definitive diagnosis requires serologic confirmation (TT & NTT) of infection with a reactive CSF and VDRL
         a) Treponemal-specific Testing (TT): TPPA/FTA-TPA/ELISA/EIA
         b) Non-Treponemal Testing (NTT): RPR/VDRL

II. Case Study
   A. Chief complaint: 57-year-old Caucasian male presents with blurred, cloudy vision OD>OS associated mild photophobia and pressure OD, with gradual onset over 3-4 months.
   B. Ocular history: cataracts, blepharitis
   C. Medical history: pre-diabetes, hyperlipidemia, myalgia
   D. Medications: celecoxib, methocarbamol
   E. Allergies: terramycin
   F. Pertinent Findings:
      1. BCVA: OD 20/100 PHNI, OS 20/20-2
         a) Dim reflex on retinoscopy OD
b) Last refraction and BCVA:
   (1) OD: -2.50 -3.00 x 175   20/20-2
   (2) OS: -2.00 -4.25 x 180   20/20-

2. Entrance Testing: Unremarkable
   a) Pupils: PERRL, (-) APD
   b) EOMs: Full, no restrictions OD, OS
   c) Confrontations: FTFC OD, OS

3. Anterior Segment:
   a) Cornea: 2+ granulomatous KPs
   b) Anterior chamber: 3-4+ cell OD, 1+ cell OS
   c) Lens: 1+ nuclear sclerosis OU
   d) Intraocular pressure: 20mmHg OD, 14mmHg OD

4. Posterior Segment:
   a) Vitreous: 2-3+ vitritis OD
   b) CD ratio: 0.50 H/V OD, 0.45 H/V OS
   c) Optic disc: indistinct ONH margins OD
   d) Peripheral fundus: Large area (~6DD) of retinitis superior mid-periphery surrounded by 2 hemes OD (Insert figure)

5. RNFL OCT: (Insert figure) TSNIT curve is elevated, correlating with ONH edema
   a) OD: significant elevation superior/nasal/inferior
   b) OS: mild elevation superior/has

6. OCT Macula: (Insert figure)
   a) OD: Normal foveal contour w/ retinal thickening, no intraretinal edema
   b) OS: Unremarkable

7. B-Scan: (Insert figure)
   a) OD: Vitreous opacities, no retinal detachment

III. Main differentials of retinitis
    A. Syphilis
    B. ARN
      1. Etiology
         a) Herpes
         b) CMV
         c) Epstein-Barr
      2. Epidemiology
         a) Elderly as VZV immunity decrease
         b) Immunocompetent patients suppressed by via corticosteroids or chemotherapeutics
         c) AIDS and immunocompromised patients
      3. Pathophysiology
         a) Viral particles induce inflammation that damages retinal cells
         b) Arterioles vaso-occlude
         c) Contractile membranes form in vitreous
         d) Multiple breaks occur at the margin of necrotic/inflamed retina
         e) Retinal detachment in up to 75% of eyes with ARN within 3 months
      4. ARN Clinical characteristics:
         a) 1 or more Foci of retinal necrosis with discrete borders in periphery
         b) Rapid progression in absence of antiviral therapy
         c) Circumferential spread
         d) Occlusive vasculopathy
         e) Prominent inflammatory reaction in vitreous and anterior chamber
      5. Ocular manifestations
         a) Anterior uveitis (granulomatous and non-granulomatous)
         b) Vitritis
         c) Optic disc edema
         d) Occlusive vasculitis
         e) Necrotizing retinitis
         f) Scleritis
      6. Ocular complications
         a) Rapid progressive vision loss from RD’s, vaso-occlusion, or optic neuropathy
            (1) Retinal detachment (50-75%)
            (2) Retinal atrophy
7. Initial treatment
   a) VZV, HSV-1, HSV-2
      (1) Oral valacyclovir (1 gram TID) is 1st choice
         (a) Increased bioavailability (54%) compared to acyclovir 800 mg 5x/day(20%)
         (b) Decreased medical cost
         (c) Less dosage (some ophthalmologists prescribe BID)
         (d) Regression of retinitis seen in 4 days to 2 weeks with no contralateral eye involvement or RD
      (2) Oral famcyclovir 500 mg TID
   b) CMV
      c) IV ganciclovir or valgancyclovir
         (1) Consider Renal function and bone marrow suppression
      d) If traditional antivirals fail:
         (1) IV Foscarnet
      e) If aggressively progressing, intravitreal injection provides direct and immediate therapy to area of infection:
         (1) Intravitreal foscarnet or ganciclovir

C. Progressive Outer Retinal Necrosis (PORN)

IV. Treatment and Management of Case
   A. For anterior inflammation:
      1. Pred acetate 1% q1h OD
      2. Cyclopentolate qid OD
   B. Laboratory and Radiologic studies:
      1. Results:
         a) ANA, HLA-B27, RF, Bartonella, CMV, Lyme, TB, Toxoplasma, HIV: Negative
         b) Chest X-ray: Unremarkable
         c) ESR and CRP: Normal
         d) RPR Titer, Syphilis Antibody EIA: Reactive
         e) HSV Type II Abs, Varicella Zoster Abs: Reactive
   C. Referral to Retina:
      1. Valtrex 1 gram TID
         a) In presence of vitritis, initial treatment for necrotizing retinitis is antiviral therapy until a definitive diagnosis is determined
         b) VZV is the most common causative agent of intraocular ocular inflammation (retinitis)
         c) Labs later revealed positive Herpes titers, but also elevated VDRL and reactive EIA, confirming diagnosis of Syphilis
   D. Referral to Infectious Disease:
      1. IV penicillin G 18-24 million units/day x 14 days
   E. Response to treatment:
      1. Complete resolution
      2. Maintenance dose of Valtrex 1 g QD

II. Extra case of ocular syphilis, with multifocal retinitis, mistreated as ARN with antiviral therapy and subsequent steroids. (If time permits)

III. V. Conclusion
   A. Because retinitis secondary to syphilis can masquerade as a more severe necrotizing retinitis related to Herpes, initiating both antibacterial and oral antivirals early is paramount to preserving vision
Atypical case of Acute Anterior Uveitis with Hyphema secondary to Herpes Zoster sine Herpete

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Course Description:

This course introduces a rare presentation of Herpes Zoster sine Herpete (ZSH) in an elderly veteran who presented with a red, painful eye. Herpes Zoster sine Herpete is a variant of Herpes Zoster Ophthalmicus (HZO). This course will discuss ZSH, while comparing it to the more common HZO.

Learning Objectives:

- To introduce Herpes Zoster sine Herpete and its unique features.
- To review Herpes Zoster Ophthalmicus.
- Compare and contrast pathophysiology and features between ZSH and HZO.
- Review treatment for HZO presentations.
- Introduce treatment regimen for Patient D, while discussing treatment regimens of other patients.
- Discuss concern of post-herpetic neuralgia in elderly patients.

Outline:

- Case History of Patient D
  - Chief complaint: red, painful right eye, blurry vision, light sensitivity, tearing
  - Ocular history
    - Macular scar in right eye 2’ ocular histoplasmosis
  - Medical history, medications, allergies
    - Prostate cancer
    - Chemotherapy medication: Jevtana
- Pertinent Examination Findings and ancillary testing
  - Visual Acuities
  - Entrance Testing (Pupils)
  - Tonometry
  - Anterior Segment findings
  - Gonioscopy
  - Fundus photography
  - Posterior Segment findings
• Differential Diagnoses
  o HZO uveitis
  o HSV keratouveitis
  o Ocular Ischemic Syndrome
  o Chronic Uveitis
  o Bleeding secondary to chemotherapy medication

• Diagnostic testing and results
  o CBC, CMV, BUN, creatine, HLA-B27, toxo, quantiferon, RPR, FTA-ABS, Lyme, lysozyme, ACE, Herpes simplex virus (HSV) 1&2, VZV
  o Carotid doppler

• Pathology, unique features
  o Comparison to classic HZO presentations
  o Commonality in pathology: reactivation of VZV in the ophthalmic division of trigeminal nerve
    ▪ ZSH: resultant immune reaction affecting iris vessels, iris nerves, and corneal nerves
      • Angiitis and occlusion of iris vessels
      • Infiltration of lymphocytes and antibodies
      • Necrosis of iris and cornea nerves
  o Pathognomonic features of HZO: corneal pseudodendrites, dermatomal forehead lesions, Hutchingson's sign
  o Unique features of ZSH: Acute anterior uveitis with hyphema not secondary to trauma or rubeosis iridis, no dermatomal forehead lesions, no pseudodendrites

• Incidence
  o Incidence and Prevalence of HZO
  o Incidence and Prevalence of ZSH (limited reported cases)

• Risk Factors
  o prior VZV exposure, elderly, immunocompromised, immunosuppressive drugs, cancer
  o comparison to risk factors of HZO

• Symptoms, Signs
  o Review of classic HZO presentations
  o Circumlimbal conjunctival hyperemia, light sensitivity, pain, blurry vision
  o Anterior chamber reaction, corneal edema, hyphema

• Diagnostic criteria
  o Comparison to classic HZO presentations
  o elevated anti-VZV IgG serum levels
  o sectoral ciliary atrophy on iris fluorescein angiography
  o polymerase chain reaction of aqueous humor

• Treatment
  o HZO treatment:
    ▪ Review of oral antiviral therapy
    ▪ Heavy lubrication with preservative free artificial tears
• Prophylactic antibiotic drops or ointment
• Steroid treatment
• Ocular hypotensives
  o Patient D’s treatment regimen and response to treatment
    • Medication:
      • Topical: prednisolone acetate initially q1h then q2h until resolution, atropine bid OD, timolol 0.5% bid OD
      • Oral Valtrex (valacyclovir) 500 tid po
    • Referrals
      • Retina specialist to rule out other etiologies
    • Follow up schedule
      • Daily follow up initially
      • Weekly dilated examinations until resolution of uveitis and hyphema
    • Expectations of treatment:
      • Resolution of uveitis
      • Resolution of hyphema
  • Potential Complications
    o Reactivation of VZV as ZSH or HZO
    o Corneal scarring
    o Secondary glaucoma
      • Angle closure glaucoma
      • Uveitic glaucoma
    o Cystoid macular edema
    o Secondary cataracts
    o Posterior synechiae
    o Post-herpetic neuralgia concern in the elderly
  • Clinical Pearls
    o Recognize unique presentation of ZSH
    o Initiate treatment earlier, avoiding visually threatening ocular sequelae
  • Professional Acknowledgements
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    o Matthew Pezda, MD
  • References
Cranial Nerve Six Palsy Secondary to Petrous Apex Mass

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Course Description:
This course discusses primary treatment and management for non-vascular cranial nerve six palsy secondary to petrous apex mass.

Course Learning Objectives:
- Review anatomy of cranial nerve six pathway
- Different etiologies of cranial nerve palsies
- Work up, management, treatment of cranial nerve palsies
- Discuss pathophysiology and treatment of Chondrosarcomas

Outline
1. Cranial Nerve Palsies
   a. Different etiologies: Idiopathic, Ischemic, Trauma, Neoplasms
2. Work up for cranial nerve palsies
   a. Blood pressure, Blood work, MRI
3. Cranial Nerve Six Pathway
4. Different Cranial Nerve Six Abnormalities
   a. Chondrosarcomas, Gradnigo’s, Nasopharyngea Carcinoma, Cerebellopontine Angle Tumor
   b. Age Considerations
5. Plasmacytoma secondary to multiple myeloma
   a. Pathophysiology
   b. Complications that it can cause
   c. Management
   d. Treatment- surgical options
6. Referrals
   a. Medical internist, radiologist, neurosurgery

Case Study
68-year-old Caucasian male presents with acute binocular diplopia, onset 4 days ago. Horizontal diplopia with greater significance in right gaze and at distance. Denies any injury or trauma. Patient reports for the past 3-4 days his sugar has been in the low 100’s but recently has been as high as 300s.

Ocular History: Mild Non-proliferative Diabetic Retinopathy OU; Cataracts OU
Medical History: diabetes, hypertension, hyperlipidemia
Medications: metformin, insulin, apixaban, aspirin, digoxin, furosemide, losartan, metoprolol, pravastatin, tamulosin
Allergies: none

Pertinent Findings:

**Acuities**: CC: OD 20/30, OS 20/30

**Entrance testing**: Pupils- unremarkable OU

**Extraocular muscles**: constant diplopia in right gaze and primary gaze (at distance)

**Cover Test**: Distance (cc): 8pd AET  
Near (cc): 6pd XP

**Anterior segment**:
- Lens: Nuclear and cortical cataracts OU
- Intraocular pressure: 14mmHg OD, OS

**Posterior Segment**:
- Cup/disc (CD) ratio: OD: 0.5H/V, OS: 0.45H/V
- Posterior Pole: scattered dot blot hemes

**Subsequent Visits** (1 and 2 months after onset): Stable, non-resolving CN VI palsy even with better glucose control

**Laboratory Testing**: CBC, Fasting blood sugar, ANA, VDRL, FTA-ABS, ESR, CRP, Syphilis EIA, RF, EBV, ACE, Lyme. (All unremarkable)

Radiological Studies:
MRI with and without gadolinium based contrast shows two osteolytic skull based lesions within the petrous apex (Figure 1). One centered within the right petrous apex (Figure 2) and the other within the left lower bony clivus (Figure 3). Findings are concerning for dural involvement within the anterior aspect of the right internal auditory canal and possibly the right middle cranial fossa.
A Rare Case of Papilledema Associated with Arnold Chiari Malformation

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Course Description:
This course discusses a rare presentation of papilledema secondary to Arnold Chiari Malformation while also highlighting Chiari malformation as a whole, the effects of prolonged papilledema, and surgical and pharmacological treatment options for these patients.

Course Learning Objectives:
- To understand Arnold Chiari Malformation and its ophthalmic manifestations
- To review papilledema and its effect on vision if left untreated
- To discuss appropriate work-up for patients with both papilledema and papilledema with Arnold Chiari Malformation
- To review surgical and pharmacological management options for Chiari Malformation patients that present with papilledema

Outline

I. Case history

- 58-year-old African-American female
- Chief complaint: current glasses do not work well
- Ocular history: pseudotumor cerebri with history of Diamox use; corneal ulcer OU
- Medical history: Chiari type 1 malformation
- Medications: Diamox (self-discontinued), topiramate
- Allergies: none

II. Pertinent findings

- Clinical
  - Best corrected visual acuity: OD 20/20-, OS 20/25
  - Entrance testing: unremarkable OU
  - Anterior segment
    - Cornea: scar OD
    - Lens: trace nuclear and cortical cataract OU
  - Intraocular pressure: 16 mmHg OD, 16 mmHg OS
  - Significant posterior segment findings
    - Initial exam
      - CD ratio: small due to swelling of ONH OU
      - Optic disc: blurred margins OD, superior blurred margins OS
• Four month follow up
  • CD ratio: 0.10 round OD, 0.10 round OS
  • Optic disc: edema OU
• Nine month follow up
  • CD ratio: 0.15 round OD, 0.15 round OS
  • Optic disc: distinct margins OU
• Auxiliary testing
  • Humphrey Visual Field (24-2)
    • Initial exam:
      • OD: moderate inferior nasal step defect – progressed from previous fields in records
      • OS: mild inferior nasal step defect – stable to previous fields in records
    • Four month follow up:
      • OD: inferior arcuate with scattered shallow superior defects
      • OS: early nasal step with scattered defects
    • Nine month follow up:
      • OD: inferior nasal defect
      • OS: shallow inferior nasal defect
• Retinal Nerve Fiber Layer OCT
  • Initial exam
    • OD: thinning superior temporal and nasal (with corresponding visual field loss), borderline thinning temporal
    • OS: thinning superior temporal (with corresponding visual field loss), borderline thinning temporal
• Radiology imaging
  • Magnetic resonance imaging (MRI) of brain without contrast: Chiari I malformation, characteristics of elevated intracranial pressure
  • Magnetic Resonance Venography (MRV) of brain with and without contrast: no concerning enhancement or mass, no sign of venous sinus thrombosis
• Laboratory studies
  • Spinal tap with opening pressure: elevated at 30 cm H₂O
  • Neurology consult: concern for progressive visual field defect OD and persistent papilledema suggesting intracranial pressure not adequately controlled

III. Differential diagnoses
• Primary: Papilledema OU
• Other: Pseudopapilledema, idiopathic intracranial hypertension, malignant hypertensive retinopathy, ischemic optic neuropathy (GCA), Leber’s hereditary optic neuropathy, diabetic papillopathy, thyroid-related optic neuropathy, uveitis

IV. Diagnosis and discussion
• Diagnosis: papilledema associated with Arnold Chiari malformation
• Discussion
Arnold Chiari malformation

Definition
- Type 1 is a structural defect in which the cerebellar tonsils and inferior cerebellar lobules of the brain are displaced downward through the foramen magnum of the skull and into the upper cervical spinal canal
- Generally congenital and usually presents in adulthood
- Results in compression of the cerebellum and brain stem, as well as obstruction of flow of cerebral spinal fluid, which in turn can cause elevated intracranial pressure

Prevalence
- 1 in 1000 people (0.1% of the population)

Signs/Symptoms
- Most commonly, headache and/or neck pain that worsen with coughing, sneezing, or straining
- CN VI palsy, convergence/divergence abnormalities, visual disturbance, hydrocephalus, syringomyelia, other symptoms related to compression of brainstem/dysfunction of cerebellum
- Rarely, papilledema
- Up to 30% are asymptomatic

Diagnostic Criteria
- MRI or X-ray, cerebellar tonsils that extend below the foramen magnum by ~5mm (some variation in this distance)

Papilledema

Definition
- Swelling of the optic nerves due to elevated intracranial pressure
- Usually bilateral and symmetric between the two eyes, but can be unilateral or asymmetric
- Most concerning complication of papilledema is vision loss caused by axoplasmic flow disruption and intraneuronal ischemia

Symptoms
- Transient vision loss, headache, double vision, nausea, vomiting, decreased visual acuity, visual field loss

Diagnostic Criteria
- Swollen, hyperemic optic disc with blurred disc margin; obscured blood vessels; papillary/peripapillary retinal hemorrhages, loss of spontaneous venous pulsation, dilated/tortuous retinal veins
- Chronic: gray disc, possible shunt vessels, loss of color vision, central visual acuity, and visual field

Visual Field Loss
- Initially: blind spot is enlarged
- Chronic: nerve fiber layer visual field loss (often inferonasal) – central vision usually spared until vision loss has progressed significantly
V. Treatment and Management

- Primary treatment option for patients with Chiari Malformation:
  - Surgical Intervention: suboccipital decompression via suboccipital craniectomy, upper cervical laminectomy and duraplasty, foramen magnum decompression
  - Was not recommended for this patient by ophthalmology
- Additional treatment options for elevated intracranial pressure:
  - Weight loss
    - 5-10% of total body weight
  - Acetazolamide: typically begin with 500mg bid, gradually increased to 2-4g total per day as indicated and tolerated
  - Topiramate: primarily used to treat headaches, but also effective for intracranial hypertension; typically begin with 25-50mg qday, gradually increased to 100-150mg qday
    - Most successful for this patient (improved headache symptoms, optic nerve head appearance, and visual field defect)
  - Alternative surgical treatment options: neurosurgical shunt/venous sinus stenting, optic nerve sheath decompression
- Management: neurology, optometry

VI. Conclusion

- Papilledema requires careful work up to determine etiology and subsequent treatment options. Chiari malformation can result in chronic intracranial hypertension and chronic disc swelling, and if not managed properly, can result in vision loss.
- Although Diamox is often the main pharmaceutical treatment for elevated intracranial pressure, topiramate is an additional option.
- With proper diagnosis and treatment, papilledema can resolve, resulting in improvement of visual field.