Abstract:

Patient presents with symptoms consistent with orbital cellulitis but imaging results suggest otherwise. The source, an abscess, located anterior to the orbital septum, is consistent with preseptal cellulitis. The abscess etiology suggests orbital cellulitis.

I. Case History

- 69 year old African American male veteran
- CC: New onset “swollen, painful, red” left eye that is “running like a leaky faucet” x 1 day; pain 8/10 left eye
- Ocular Hx: Low suspicion glaucoma suspect due to increased C/D ratio, history of Hollenhorst plaque left eye, posterior vitreous detachment right eye, vitreal syneresis both eyes
- Medical History: Depression, alcohol dependence, hypertension, chronic lower back pain, erectile dysfunction
- Medications: Sildenafil, Tramadol, Hydrochlorothiazide, Levetiracetam, Cyclobenzaprine
- No medical or environmental allergies, no family ocular history

II. Pertinent findings

- Clinical Findings:
  - Visual acuities: right eye 20/50 with pinhole acuity 20/20, left eye was 20/80, pinhole no improvement
  - Pupils direct and consensual without APD; severe pain with significantly decreased motility in all fields of gaze, left eye. Motility within normal limits right eye; confrontation visual fields full both eyes
  - Left eye with severe diffuse, periorbital swelling upper and lower lids with complete lid closure; erythema and tenderness upon palpation, firmness to tissue noted superior nasally
  - Bulbar conjunctiva with 4+ diffuse chemosis and 2+ diffuse injection; anterior chamber, iris and cornea within normal limits; continuous serous discharge from left eye
  - Intraocular pressures right eye 20, left eye 15; slit lamp examination within normal limit right eye
• Lens within normal limits right and left eyes, cup to disc ratio right eye 0.65/0.65, left eye 0.7/0.7; optic nerve head, macula, vitreous, vessels and peripheral all within normal limits
• B-scan performed of the left eye within normal limits
• Physical: Temperature: 98.7 degrees F at 3:24pm, pulse 69, strong and steady
• Laboratory studies: CBC c diff within normal limits; conjunctival culture
• Radiology studies: MRI ordered with and without contrast, thin cuts through the orbits with fat suppression; MRI report: bi-lobed nodular mass situated superior to the left globe medial to the lacrimal gland, abnormal enhancement of the soft tissue surrounding the lesion

III. Differential diagnosis

• Orbital Cellulitis vs Preseptal cellulitis
• Orbital pseudotumor

IV. Diagnosis and discussion

• Diagnosis consistent with severe left sided preseptal cellulitis vs orbital cellulitis with pre-septal abscesses on MRI
• Typically the differentiation between orbital cellulitis and preseptal cellulitis is clinically apparent, especially when imaging has taken place. This patient presented with symptoms very consistent with orbital cellulitis including severe pain, especially on attempted motility and severe periorbital swelling. What did not correlate was the absence of fever, the presence of two pre-septal abscesses noted with MRI and the soft tissue swelling all anterior to the orbital septum. Even after imaging this patient the diagnosis was not definitive.

V. Treatment, management

• Treatment and response to treatment
  • Pt admitted to the ward and given IV vancomycin (1250 mg q12h) and zosyn (3.375 gm in dextrose 5%, 50 ml infused over 240 min, q8h)
• One day follow up, improvement noted in periorbital swelling with opening of palpebral aperture. Copious yellow discharge noted with increased drainage of abscess with partial lid eversion; significant decrease in pain; all other exam findings stable
• Given drainage of lesion, initiated Vigamox 6 x day left eye
• CT of orbits and sinuses both with and without contrast ordered to rule out sinusitis
• Daily follow up examinations with continued improvement in pain and clinical signs and symptoms; IV antibiotics discontinued once culture results obtained and switched to oral antibiotic. Prescribed Zosyn, 14 day course given gram negative rod culture of Serratia Marcescens only resistant to Ampicillin/Sulbactam
• Patient was discharged from the hospital and instructed to follow up 5 days after discharge
• Referral made to Ear Nose and Throat specialist for sinus evaluation given that sinuses were the most likely source for the abscess

• Bibliography, literature review:
  • “Risk factor of preseptal and orbital cellulitis” Babar, TF et al; Department of Ophthalmology, Khyber Institute of Ophthalmic Medical Services, Journal of the College of Physicians and Surgeons; Pakista, 2009
  • “Guidelines for management of periorbital cellulitis/abscess”, L. Howe et al, Clinical Otorlaryngology & Allied Sciences, Volume 29, Issue 6, Pages 725-728, December 2004

VI. Conclusion

• Preseptal Cellulitis and Orbital Cellulitis have differentiating signs and symptoms. Given the potential life threatening consequences, accurate diagnosis and treatment are necessary.

• This case is unique as the patient presents with symptoms consistent with an orbital cellulitis but imaging results suggest the source of infection as an abscess located anterior to the orbital septum with no infection/inflammation noted posteriorly. This would be more consistent with a preseptal cellulitis. The nature of the abscess also
suggests an etiology linked to the sinus cavity, but this also would imply the diagnosis of orbital cellulitis. Given the ambiguity of this presentation and abscess it was imperative to treat as an orbital cellulitis although imaging suggested otherwise.
Cordes Outline

I. Case History

- 54 y.o. Navajo Male
- Kidney Dialysis Center calls with a patient complaining of a red/painful eye
- “Sure- send them over”
- CC: Right Eye Red and Painful for last 1-2 weeks. A circle constricted and took away vision in his right eye 2 weeks ago. Did not come in because he thought he would not need/get an appointment Out of glaucoma medication drops for one week (missed appointments) needs refills.

II. Pertinent findings

- **Ocular History**
  - PRP OS
  - PC IOL  OS Surgery March 21, 2007
  - Glaucoma
  - PDR and NPDR OU

- **Medical History**
  - Diabetic (uncontrolled)
  - End Stage Renal Failure
  - Hyperphosphatemia
  - Chronic Pain (Back, Leg, etc)
  - Chronic Bacterial Infections

- **Medications**
- **OCULAR (upon presentation)**
  - Alphaghan 1 gtt TID OU

- **SYSTEMIC**
  - Moxifloxacin Tablets QD
    - Sevelamer 800 mg TID
    - Tylenol PRN

- VA: OD: NLP
- OS: Counting Fingers at 5 feet

- FROM OD, OS
Pupils: Pupil OS- Round reactive to light with a 2+ response  
OD- No response- the pupil is not visible  
IOP OD: 34 OS: 17 (GAT) (NCT 48,34/16)  
SLE  
OD: 3+ Hyphema (3mm)  
OD: Anterior Chamber completely gelatinous  
OD: Iris and other posterior structures not visible  
OS: Anterior Segment - Unremarkable (Ping, PCIOl)  
Dilated Fundus Exam OS  
Extensive PRP scars 360*  
Superior Macular Heme (1/4 DD)  
Raised grey-green mass/mound temporal macula

III. Differential diagnosis

► Retinal Detachment  
► Vitreous Floaters  
► Retinal Hole  
► Retinal Tear

IV. Diagnosis and discussion

► Central Retinal Vein Occlusion  
► Endophthalmitis  
► Giant Retinal Tear/Detachment  
► Pan Uveitis  
► Trauma  
► Neovascularization of the Iris/PDR

V. Treatment, management

► REFERRAL TO RETINAL SPECIALIST (much easier than it sounds)  
► Atropine 1 gtt OD in office, Homatropine 1 gtt TID OD, Alphagan TID OD, Pred Forte 1% 1 gtt every hour  
► Bed rest for the weekend with limited activity  
► Return to clinic on Monday for follow-up, before going to Retinal Specialist Tuesday- patient had dialysis on Monday  
► Next week follow up- still no specialist consultation
Summary:

Hyphema in a Brittle Diabetic

Non-traumatic/surgical hyphemas from neovascularization of the iris are not common. They present a problem not only because of the rise of intraocular pressure but also because of the reduced clearance time of red blood cells from the anterior chamber of proliferate diabetics. In this case study, the patient presented with a history of unilateral idiopathic hyphema for a “few days” and vision loss for two weeks which “disappeared like a circle”. Due to difficult circumstances, (i.e. the patient could not afford gas) the patient was not able to access ophthalmologic care in a prompt fashion.

As result the neo vascular glaucoma progressed in his right eye (hyphema eye) despite the improvement of the inflammation and hyphema. Also, the non-hyphema eye developed neovascularization of the iris and vision loss. As the optometrist of record, management was carried out to the highest level of optometric care. Despite the challenges this patient is still under combined optometric and ophthalmologic care and his hyphema and proliferative diabetic retinopathy still exist.

This case will outline the care and management of the inflammation, vascular growth and rise of intraocular pressure from hyphemas in diabetics from an optometric standpoint. It will also emphasize the importance of identifying and treating high risk patients. Emphasis will also be placed on demonstrating the importance of managing a patient to the highest level of your abilities despite what circumstances and challenges might stand in the way.
An 89 year old white male reports diplopia, brow-ache, ptosis, and numb left superior lip. Prior history includes bilateral, sequential cranial nerve VI palsies and left vocal cord paresis. Previous work up has been negative.

I. Case history
   A. Patient demographics: 89 year old white male
   B. Chief complaint: On 3-26-2012 patient reports 1 day onset of new diplopia, brow-ache, ptosis, and numb left superior lip
   C. Ocular history:
      1. 5-2010: Left cranial nerve VI palsy-resolved
      2. 7-2010: Right cranial nerve VI palsy-treated with 16^ base out prism
   D. Medical history
      1. 1993: Bladder cancer in remission since 1997
      2. 1999: Hypertension
      3. 12-2009: Left vocal cord paresis—hoarse voice
      4. 7-2010: numbness of left lateral thigh
      5. 2-2012: Thrombocytosis
      6. Hyperlipidemia
      7. NO Diabetes Mellitus
   E. Medications
      1. Hydrochlorothiazide
      2. Isosorbide dinitrate
      3. Lisinopril
      4. Simvastatin
   F. Other salient information
      1. Patient had undergone extensive work up at VA and/or Dartmouth Hitchcock Medical Center in 2010
         a. MRI 5-17-2010
         b. MRI 8-16-2010
c. CT thorax 5-14-2010
d. CT abdomen
e. Ultrasound throat
f. Lumbar puncture
g. Thyroid nodule biopsy
h. ACE
i. Acetylcholine receptor antibodies
j. Lyme titers
k. ANCA
l. Anti-MuSK antibodies
m. Thiamine levels

2. All findings were normal.
3. The presumed etiology for the bilateral, sequential cranial nerve VI palsies and vocal cord palsy was ischemia from microvascular disease

II. Pertinent findings

A. Clinical
1. Vision
   a. OD 20/60+
   b. OS 20/30
2. Pupils: equal, round and reactive to light with no afferent defect
3. OD ptosis
4. OD 6 prism diopter hypotropia
5. Confrontations: possible Right field constriction OU
6. Dilated fundus exam unremarkable except secondary fibrosis of PCIOL OS
7. Exophthalmometry=15mm OU
8. Orbicularis testing found normal and equal strength OD, OS
9. No increase in ptosis with extended upgaze
10. Red cap test found no desaturation in central or peripheral vision either eye

B. Physical
1. Cranial nerve testing
   a. Cranial nerve III incomplete palsy OD-pupil spared
   b. Cranial nerve VI palsy
i. OD still present
ii. OS resolved
   c. Cranial nerve V-2 palsy left side
d. All other cranial nerves tested normal
2. Temporal artery palpation-normal with no tenderness
3. Carotid auscultation normal right and left sides-no bruits
4. Ocular auscultation normal OU-no bruits
C. Selected Laboratory studies
   1. ESR=0
   2. C reactive protein=0.5
   3. RBC, Hemoglobin, Hematocrit and Platelets all elevated
   4. Glucose=96
   5. PT, INR, PTT all normal
   6. TSH normal
   7. ANA negative
   8. JAK2 Positive mutation 3-28-12
   9. Serum erythropoietin abnormally low 3-29-12
D. Radiology studies-MRI brain and orbits with/without contrast 3-26-12

1. “Mild cerebral atrophy with mild chronic microangiopathic changes. Old lacunar infarcts noted in the right basal ganglia region. No suspicious mass identified. No acute infarct, hemorrhage or intracranial mass effect identified.
2. Superior ophthalmic veins appear significantly enlarged, left greater than right. Enlargement is greater than 2.5 mm bilaterally. Findings can be associated with increased intracranial pressure. Findings can also be seen with Grave's orbitopathy, orbital pseudotumor and parasellar meningioma, which are not present. No cavernous sinus abnormality demonstrated to suggest cavernous sinus thrombosis or carotid cavernous fistula.”

III. Differential diagnosis

A. Primary/leading
   1. Cavernous sinus syndrome
B. Others
   1. Myasthenia Gravis
2. Chronic Progressive External Ophthalmoplegia
3. Orbital lesions
   a. Tumor
   b. Pseudotumor
   c. Thyroid disease
4. Brainstem disease
5. Guillain-Barre syndrome-Miller-Fisher variant
6. Ischemia from microvascular disease
   a. Hypertension
   b. Hyperlipidemia
7. Sarcoidosis
8. Vasculitic neuropathy
9. Wernicke’s encephalopathy-Thiamine deficiency
10. Lyme disease
11. Increased intracranial pressure
12. Viral cranial neuropathy
13. Metastatic disease
   a. Carcinomatous meningitis
   b. Skull base tumors
14. Polycythemia Vera

IV. Diagnosis and discussion

A. Final diagnosis=Polycythemia Vera
   1. Rare, chronic myeloproliferative disease
   2. Abnormal increase in number of blood cells
      i. Primarily red blood cells
      ii. Elevated platelets possible
      iii. Elevated white blood cells possible

B. Elaborate on the condition-Polycythemia Vera
   1. Signs and Symptoms
      a. Dyspnea
      b. Dizziness
      c. Headaches
      d. Excessive bleeding
e. Splenomegaly
f. Fatigue

2. Epidemiology
   a. Incidence 1.9 / 100,000 per year in Olmstead county, MN from 1935-1989 (UpToDate)

3. Diagnostic Criteria by World Health Organization (see chart on UpToDate)
   a. Major Criteria
      i. Increased red cell volume
      ii. Presence of JAK2 617 v>f mutation
   b. Minor Criteria
      i. Bone marrow biopsy abnormalities
      ii. Serum erythropoietin low
      iii. Endogenous erythroid colony formation in vitro
   c. Diagnosis confirmed by presence of both major criteria and 1 minor criterion or first major criterion with 2 minor criteria

4. Complications
   a. TIA or CVA
   b. Congestive heart failure
   c. Hypertension
   d. Thrombosis
   e. Gout
   f. Acute Myelogenous Leukemia
   g. Untreated survival rate is 6-18 months from diagnosis
   h. Treated survival rate >10 years

C. Expound on unique features
   1. Common ocular findings with polycythemia vera
      i. TIA
      ii. Amaurosis fugax
      iii. Retinal hemorrhages
      iv. Retinal vascular occlusions
   2. Neuro-ophthalmologic complications of polycythemia vera are rare
      i. Literature review found 7 cases of ophthalmoplegia from polycythemia vera
      ii. Only 1 case in the literature reports a Cranial Nerve III palsy as the initial manifestation of polycythemia vera (17. Park)
V. Treatment and management

A. Phlebotomy

1. Bloodletting

2. Avoid Fe supplements

B. Chemotherapy-hydroxyurea

C. Oral aspirin

VI. Conclusion-clinical pearls

A. Polycythemia vera is a rare disease of the bone marrow that leads to proliferation of blood cells, especially the red blood cells

B. Untreated patients may experience heart failure, CVA, thrombosis and acute myelogenous leukemia

C. Ocular complications typically include amaurosis fugax, retinal vascular occlusions, and retinal hemorrhages

D. Ophthalmoplegia is a rare complication of polycythemia vera

E. For any patient with multiple cranial nerve palsies a comprehensive work up should be performed to determine the diagnosis and to prevent morbidity or mortality

References


