1. Patient Profile
   a. 41 year old white male
   b. Complaining of angle closure-type symptoms
      i. Extreme eye pain, nausea and vomiting, blurry vision
   c. Past history of penetrating eye trauma at age 10
      i. Sectoral aniridia, traumatic cataract, angle recession, choroidal rupture
   d. CE/IOL OS in 1999, IOL repositioned 2004
   e. Cosmetic contact lens wearer

2. Clinical Data
   a. Vision 20/20 OD, 20/420 OS (previous BCVA 20/80 OS)
   b. Marked corneal edema
   c. 1+ Anterior chamber cell
   d. IOP 15mmHg OD, 58mmHg OS

3. DDx Acute IOP elevation
   a. Angle closure
   b. Glaucomatocyclitic crisis
   c. Angle recession glaucoma *
   d. Psuedoexfoliative glaucoma
   e. Uveitic glaucoma
   f. Other etiologies

4. Treatment plan
   a. In house topical tx and oral Diamox
      i. Monitor until IOP is lowered
   b. Rx Diamox for short course, Rx topical ocular antihypertensives
   c. Monitor closely – call 24 hours, return 48 hours

5. Angle recession glaucoma
   a. Pathophysiology
   b. Treatment options
      i. Topical vs surgical
      ii. Laser not indicated (NdYAG trabeculopuncture?)
   c. Why does IOP elevate so many years after insult?

6. Clinical Course
   a. IOP decrease OS to 2mmHg 48 hours later
   b. Advised d/c PGA and Diamox
   c. Pt. d/ced all meds
      i. IOP elevation and sx recur
   d. Managing the desperate patient

7. Teaching Points
   a. IOP elevation in angle recession may reach angle closure levels
   b. IOP elevation in angle recession may occur many years after insult
      i. Lifelong glaucoma risk
   c. Glaucoma likelihood linked to amount of angle recession
d. Patients in extreme pain represent a unique management challenge

Robert Wang, OD
Alternative glaucoma treatments

Are black coffee and Irish whiskey viable home treatment options for Glaucoma? Ninety two year old male lost to follow up for several years with advanced glaucoma with a history of poor compliance with glaucoma medication. Patient has long preferred to treat his glaucoma with black coffee and Irish whisky.

I. Case History
   a. Patient demographics
      91 y/o white male
   b. Chief complaint
      Difficulty at distance and near, patient feels that he needs a new refraction.
   c. Ocular, medical history
      Ocular: advance glaucoma both eyes, s/p SLT with poor compliance with gtt.
      Medical: Hypothyroidism, CAD, hyperlipidemia, diabetes mellitus without mention of complication, left inguinal hernia, disturbances of sensation of smell and taste.
   d. Medications
      Levothyroxine
   e. Other salient information
      Patient has a history of poor compliance with glaucoma follow up and glaucoma gtts. Patient has been utilizing a home remedy of black coffee and Irish whiskey to control his IOP.

II. Pertinent findings
   a. Clinical
      Entering VA:
      OD: 20/30
      OS: 20/40
      RRL + APD left
      Goldmann IOP’s 20 OD, 19 OS
      Posterior Segment:
      OD: .95/.95 only nasal tissue remaining
      OS: .99/.99 pallor
   b. Physical
   c. Laboratory studies
      Kinetic HVF
      Right
      Temporal, 0 axis: 10 degrees
      Temporal superior, 45 axis: 0 degrees
      Superior, 90 axis: 0 degrees
      Superior nasal, 135 axis: 0 degrees
Nasal, 180 axis: 0 degrees
Inferior nasal, 210 axis: 20 degrees
Inferior, 270 axis: 0 degrees
Inferior temporal, 315 axis: 0 degrees

Left
Nasal, 0 axis: 0 degrees
Superior nasal, 45 axis: 0 degrees
Superior, 90 axis: 0 degrees
Superior temporal, 0 axis: 0 degrees
Temporal, 180 axis: 0 degrees
Inferior temporal, 210 axis: 10 degrees
Inferior, 270 degrees: 30 degrees
Inferior nasal, 315 degrees: 0 degrees
d. Radiology studies
e. Others

III. Differential diagnosis
a. Primary/leading
   End stage advanced glaucoma left > right.

b. Others
   Retrograde optic atrophy secondary to intracranial mass
   Retrograde optic atrophy secondary to CVA
   Optic nerve head pits

IV. Diagnosis and discussion
a. Elaborate on the condition
   Open angle glaucoma progressive visual field loss related to intraocular pressure that is too high for the patient’s eye to sustain without optic neuropathy. Two main theories exist for the mechanism of glaucoma. The first is a stasis of axoplasmic flow secondary to increased intraocular pressure resulting in poor optic nerve perfusion, while the second is a shearing effect at the lamina cribrosa secondary to increased intraocular pressure. No predictions towards sex exist however there does appear to be a greater susceptibility towards African Americans than Caucasians. Patients tend to be asymptomatic with no visual complaints until very late in the disease.

b. Expound on unique features
   Our patient believed that consumption of large quantities of black coffee combined with Irish whiskey would keep his intraocular pressure under control. Research finds that there is no clear verdict for intraocular pressure as related to caffeine consumption, there does seem to be some supporting evidence for the lowering of intraocular pressure with alcohol consumption; however this lowering effect is related to the overall dehydration effect.

V. Treatment, management
a. Treatment and response to treatment
   Our patient had a history of being repeatedly lost to follow up as several doctors attempted to control his glaucoma with gtt’s. Patient continued to
believe his home remedy was keeping his intraocular pressure under control until his older kinetic visual field was drawn on his current visual field. Confronted with this evidence the patient admitted that his home remedy may have not been working as well as he had hoped. In speaking with the patient the clinician was able to find a common ground in the fact that both of them had Camp Matthews in their history. Our clinician had gone to UCSD (formerly Camp Matthews) for undergrad and our patient had served in the Army and was stationed at Camp Matthews. Our patient was immediately started on Travatan qpm both eyes and a consult was placed to OKC eye for a recommended bilateral trabeculectomy. Our patient underwent a bilateral trabeculectomy with express shunt in both eyes. Following this procedure unfortunately our patient has been lost to follow up.

b. Refer to research where appropriate
c. Bibliography, literature review encouraged

VI. Conclusion
   a. Clinical pearls, take away points if indicated
      Glaucoma itself is not incredibly difficult to treat, the most difficult parts of glaucoma lie in human nature. Is your patient compliant? Our patient had a long history of non compliancy extending at least 7 years within the VA and several years outside of the VA. In speaking with the patient I was able to find some common ground in speaking with him, and the visual evidence of the change in his kinetic visual field was very helpful in convincing the patient to re-start glaucoma gtts.

*Uncontrollable ocular hypertension in an atypical presentation of chronic Herpes Zoster associated anterior uveitis*
American Academy of Optometry
Grand Rounds
Sarah Macliver, OD, FAAO
Faculty and Clinical Lecturer at University of Waterloo School of Optometry

Abstract: A case of atypical recurrent non-granulomatous anterior uveitis OD with iris atrophy, diffuse stellate KPs, fixed pupil, and ocular hypertension is presented. Herpes Zoster is diagnosed. Requires close IOP monitoring and aggressive IOP lowering management.

I. Case History

- Patient demographics: The case report describes a 31-year-old man from Libya with a history of recurrent non-granulomatous anterior uveitis on two separate occasions, both in Libya. Extensive investigation into systemic associations in Libya were unremarkable. He moved to Canada in 2008.
- Initial Presentation: Presented to out ocular disease clinic with a red, photophobic, dull aching, teary OD. There was no associated vision loss or disturbance.
- Ocular History: Two prior episodes of acute non-granulomatous anterior uveitis that resolved successfully over a month each with a course of topical steroid. One episode in 2002, other in 2007.
- Medical History: Fatty liver, brother died of lymphoma when he was 30 yo.
- Medications: None

II. Pertinent Findings

- First Visit (Mar 4, 2013)
  - BCVA: 6/6 OD 6/6 OS
  - Pupils: PERRL (-) RAPD
  - EOMs: Unrestricted (-) pain, diplopia
  - Intraocular pressure: 11 mmHg OD; 14 mmHg OS
  - Anterior Segment:
    - External adnexa, lids/lashes, within normal limits.
    - Conjunctiva: 3+ circumlimbal hyperemia OD, quiet OS
    - Cornea: (+) keratic precipitates (extending centrally, more concentrated inferiorly) clear and quiet OS
    - Iris: Flat and intact OU
    - Anterior chamber: 3+ cells and flare OD, Deep and quiet OS
  - Posterior Segment: Lens: cl OU, Vitreous: cl OU (-) cells, Optic nerve, macula, vessels, periphery within normal limits OU
  - No imaging indicated
  - Laboratory studies done because of recurrent nature of uveitis:
    - ESR 2
    - CRP <1
    - ANA negative
    - HLA-B27 negative
• Chest x-ray: negative
• Tb – negative
• Patient was started on Maxidex q1h, Maxidex ung qhs and homatropine 5% tid. Uveitis started to clear as normal.

One month later (April 4):
• Present with symptoms of increased discomfort and noticing a “floater” OD. Visual acuity unchanged.
• Anterior segment: 1+ circumlimbal injection, diffuse fine pigmented KPs greatest superiorly and centrally, patchy sectoral iris atrophy, fixed and mid-dilated pupil, 4+ cells (pigment = WBCs with fibroid appearance), diffuse pigment on lens surface.
• Posterior segment: Vitreous and retina clear. Unremarkable OU
• IOPs 17 mmHg OD and OS
• Gonioscopy: Open, 4+ PTM pigmentation
• Imaging: Anterior segment OCT no concave iris formation or evidence of iridolenticular contact
• Referral to Uveitis Specialist made

III. Differential Diagnosis:
• Viral associated Uveitis (HSV/HZV/CMV)
• Fuch’s heterochromic iridocyclitis
• Pigment dispersion syndrome
• Ocular Tumour

IV. Diagnosis:
• Diagnosed as Herpes Zoster non-granulomatous anterior uveitis.
  • Clinical diagnosis based on presence of: diffuse iris atrophy, fine stellate diffuse KPs, and pigment liberation
  • There was no keratitis or posterior involvement
  • No medical history of past Herpes Zoster, Varicella, or Shingles infection

Discussion and unique characteristics
• The patient initially presented with a very typical recurrent non-granulomatous anterior uveitis (lowered IOP, inferior KPs, 3+ cells and flare, and no posterior synechiae)
• After a month, the condition changed and patchy iris atrophy, fine stellate KPs, intense pigment liberation and a fixed mid-dilated pupil developed. A couple weeks later, ocular hypertension developed. All signs typical of HZ viral uveitis
• HZV typically presents in the eye as an infection involving the ophthalmic branch of the trigeminal nerve
• Herpes viral uveitis usually presents with kerato-uveitis; however, uveitis without corneal involvement is more common in HZV than HSV associated uveitis
• Less commonly, Herpes Zoster uveitis can present in isolation with a very characteristic presentation of diffuse KPs, iris atrophy, and ocular hypertension as with our patient
• HZV associated uveitis tends to have a chronic course and IOP rise is seen in 50-90% of these patients
• Secondary glaucoma is the most common complication in herpetic uveitis and it has been estimated to occur in about 50% of HZV individuals
• It can be **blinding** if not controlled adequately
• Typically, IOP can be controlled by controlling the inflammation with potent steroids
  • Not possible to control medically in about 25-30% of cases
  • If active inflammation present – ALT/SLT avoided
  • IOP rise can be rapid and poorly responsive to medication
• IOP control needs to be aggressive and immediate otherwise can secondary glaucoma can quickly progress and become blinding
• Along with management of glaucoma, long term antiviral prophylaxis is recommended, although this is controversial

V. **Treatment and Management**
• Initial treatment of Maxidex q1h ophthalmic solution, Maxidex qhs ung, homatropine 5% bid OD,
• After diagnosis of Herpes Zoster (s/p development of fine KPs, iris atrophy) associated uveitis, 800 mg 5x/day initiated po
• Maxidex ophthalmic solution continued at 10x/day OD
• Pigment liberation and uveitis ongoing after 9 months, although fine KPs had resolved and progression of iris atrophy had subsided
• IOP rise noted after 2 months of therapy and deemed to be multifactorial due to steroid response, hypertensive uveitis, pigment dispersion in angle
• Summary of IOP management (below in chart)
• IOP began to rise after 2 months into Maxidex 1 gt q1h
• Key Points:
  • Multifactorial cause of IOP rise: steroid response since high frequency dosing of Maxidex > 2 months
  • Inflammation clearly played a role
  • IOP would rise when steroid was tapered
  • Increasing steroid caused lowering
    • Eventually medical management (steroid and IOP lowering medication) could no longer control IOP
    • Neptazene 500 mg tid was initiated, when unsuccessful Diamox 250 mg qid po was given
    • Paracentesis was done by glaucoma surgeon during IOP spike but lowering result only lasted 12 hours
    • Emergent surgery was done to improve IOP
    • Filtration surgery done on Oct. 31
    • Emergent ex-PRESS mini-shunt with MMC
    • Difficulty controlling IOP following eXpress mini shunt because of increase in inflammation
    • Inflammation subsided and IOPs stabilized at 12 mmHg 10 days later
Maxidex QID was continued due to continuation of mild AC reaction

<table>
<thead>
<tr>
<th>Date</th>
<th>IOP (OD,OS)</th>
<th>Dose of Maxidex drops</th>
<th>IOP lowering Meds (+ Valtrex 500 mg bid)</th>
<th>Notes:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apr 30</td>
<td>17,18</td>
<td>q1hr</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>May 6</td>
<td>20,23</td>
<td>10x/day</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>May 13</td>
<td>22,20</td>
<td>10x/day</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>May 21</td>
<td>28,18</td>
<td>10x/day</td>
<td>Timoptic-XE qam</td>
<td></td>
</tr>
<tr>
<td>May 27</td>
<td>22,20</td>
<td>10x/day</td>
<td>Timoptic-XE qam</td>
<td></td>
</tr>
<tr>
<td>June 3</td>
<td>28,19</td>
<td>10x/day</td>
<td>Timoptic-XE bid</td>
<td></td>
</tr>
<tr>
<td>June 6</td>
<td>29,19</td>
<td>10x/day</td>
<td>Cosopt bid started June 6 increase in inflammation noted</td>
<td></td>
</tr>
<tr>
<td>June 8</td>
<td>19,18</td>
<td>10x/day</td>
<td>Cosopt bid</td>
<td>Stable inflammation</td>
</tr>
<tr>
<td>June 15</td>
<td>22,18</td>
<td>10x/day</td>
<td>Cosopt bid</td>
<td></td>
</tr>
<tr>
<td>June 22</td>
<td>37,21</td>
<td>10x/day</td>
<td><strong>Alphagan</strong> bid, Cosopt bid</td>
<td>DEFINITE increase in inflame. (pain, redness, increase cells)</td>
</tr>
<tr>
<td>June 24</td>
<td>17,18</td>
<td>10x/day</td>
<td>Alphagan bid, Cosopt bid</td>
<td>Improvement in symptoms</td>
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<tr>
<td>July 15</td>
<td>23,15</td>
<td>8x/day</td>
<td>Alphagan tid, Cosopt bid</td>
<td>Occasional discomfort in morning</td>
</tr>
<tr>
<td>July 17</td>
<td>26,20</td>
<td>12x/day</td>
<td>Alphagan tid, Cosopt bid</td>
<td>Increase in discomfort</td>
</tr>
<tr>
<td>July 18</td>
<td>23, 19</td>
<td>Q1h</td>
<td>Alphagan tid, Cosopt bid</td>
<td>Improvement</td>
</tr>
<tr>
<td>Aug 2</td>
<td>28, 17</td>
<td>12x/day</td>
<td>Alphagan tid, Cosopt bid</td>
<td>Attempting to reach OMD</td>
</tr>
<tr>
<td>Aug 9</td>
<td>24, 15</td>
<td>10x/day</td>
<td>Alphagan tid, Cosopt bid</td>
<td>Improvement noted</td>
</tr>
<tr>
<td>Aug 13</td>
<td>22, 16</td>
<td>10x/day</td>
<td>Alphagan tid, Cosopt bid</td>
<td></td>
</tr>
<tr>
<td>Aug 26</td>
<td>25, 16</td>
<td>8x/day</td>
<td>Alphagan tid, Cosopt bid</td>
<td>Appt with OMD 2 months!</td>
</tr>
<tr>
<td>Sept. 3</td>
<td>22, 16</td>
<td>6x/day</td>
<td>Alphagan tid, Cosopt bid</td>
<td></td>
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<tr>
<td>Sept. 11</td>
<td>30, 17</td>
<td>6x/day</td>
<td>Alphagan tid, Cosopt bid</td>
<td>Patient reports not taking cosopt day prior</td>
</tr>
<tr>
<td>Sept. 20 (OMD)</td>
<td>38, 13</td>
<td>4x.day</td>
<td>Alphagan tid, Cosopt bid Neptazane 50mg tid</td>
<td>OMD: inflam + steroid response</td>
</tr>
<tr>
<td>Sept. 23</td>
<td>26, 13</td>
<td>QID</td>
<td>Alphagan tid, Cosopt bid Neptazane 50mg tid</td>
<td>Improvement in discomfort</td>
</tr>
<tr>
<td>Sept. 30</td>
<td>34, 14</td>
<td>QID</td>
<td>Alphagan tid, Cosopt bid Neptazane 50mg tid</td>
<td>Report to OMD</td>
</tr>
<tr>
<td>Oct. 18 (OMD)</td>
<td>44, 15</td>
<td>BID</td>
<td>Alphagan tid, Cosopt bid Neptazane 50mg tid</td>
<td>Referral to Glc specialist</td>
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<tr>
<td>Oct. 21 (OMD/#2)</td>
<td>47</td>
<td>BID</td>
<td>Paracentesis in am Alphagan tid, Cosopt bid Neptazane 50mg tid</td>
<td>Pm: IOP = 10 mmHg</td>
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<tr>
<td>Oct. 21</td>
<td>53</td>
<td>BID</td>
<td>Increase Diamox, meds q2h</td>
<td>IOP decreased to 36mmHg</td>
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<tr>
<td>Oct. 23</td>
<td>36</td>
<td>BID</td>
<td>Discussed filtration surgery</td>
<td></td>
</tr>
</tbody>
</table>

VI. Conclusions and Take Away Points
- Infectious viral anterior uveitis is more prevalent in the younger demographic than we think and should be considered in a presentation of iris atrophy, diffuse stellate punctate KPs and ocular hypertension
- It is important to make an accurate diagnosis because of the risk of uncontrollable ocular hypertension and progression to glaucoma
- Surgical intervention is often to necessary to achieve adequate IOP control

REFERENCES: