Title: Known vasculopathy with history of NAION presents with PION in setting of coronary artery bypass graft surgery complicated by sepsis and hypotension

Abstract: Unique case report of patient with unilateral posterior ischemic optic neuropathy in the setting of coronary artery bypass graft surgery with previous history of non-arteritic anterior ischemic optic neuropathy in the fellow eye

I. Case History:
- 56 year-old white male (on initial presentation of symptoms 2/21/08, currently 64)
- 59 years old on presentation of PION 12/2010

Chief complaint:
2/21/08: Blurry vision in the left eye that started yesterday at 1pm lasting 15-20 mins and headache around left temple. Complaining of inferior black/gray vision and when looking at examiner’s nose, sees black space where chin should be. Reports upon closing left eye he sees a bright cloud.

12/22/10: Patient reports he suddenly noticed loss of vision in right eye 2 nights ago. When covering left eye, he reports right eye vision is blurry and white. Nurse reported he was unable to count fingers with or without glasses at 6am next morning. Patient stating his vision loss may have first presented upon awakening from anesthesia but he did notice it until 2 weeks later. Denies headache, jaw claudication, scalp pain, fever, malaise. Note he was unable to talk from 11/15/10 until 12/9/10 secondary to severe state and sedation following surgery on 11/15/10.

Ocular history:
- History of right-sided bell’s palsy in 2006
- Small, crowded nerves

Medical history
- History of numbness in arms and legs attributed to cervical stenosis
- History of migraines with visual aura, cervical stenosis, hyperlipidemia, hypertension, obesity, Viagra use, alcohol dependency (>11 beers/day frequently), GERD, PTSD
- Diagnosed with sleep apnea prior to 2000
- In 2010:
  - Patient was admitted to Maine Medical Center after he was having chest pain while hunting. Found to have 90% left main obstruction and 60% coronary artery occlusion. Hospitalized in Maine from 11/10/2010 until 12/1/2010 where he underwent coronary artery bypass graft surgery on 11/15/10. He was subsequently transferred to VA Boston Healthcare after persistent failure to wean off ventilator, agitation, fever/hypotension requiring pressors and pericardial effusion

Medications
- Carisoprodol 350mg 1 tab TID to relax muscles
- Citalopram hydrobromide 40mg 1½ tab every morning
- Gabapentin 300mg 1 tab TID to prevent seizures/pain
- Lisinopril 20mg tab daily
- Mometasone furoate 50mcg 120D nasal spray 2 puffs twice/day
- Omeprazole 20mg daily
- Oxycodone 5mg/acetaminophen 325mg as needed for pain
- Vardenafil HCL 20mg tab one hour before activity

II. Pertinent findings

Clinical findings:

NAION initial presentation:
- BCVA 20/20 OD/OS
- Confrontation fields full OD and facial field missing inferonasal and inferior OS, also inferonasal defect on finger counting OS
- Inferior altitudinal defect OS with 24-2 VF **VF available**
- color desaturation OS: 100% OD, 80% OS
- IOP 15mmHg OU
- no APD
- tenderness when left temple artery is palpated, tingling cheek, intermittent pain left temple
- Fundus findings- “disc at risk” OU small and congested cup
  o OD: vit cl, disc sharp margins slight elevation nasal wnl, macula flat, periphery clear
  o OS: vit cl, disc with blurred superior margins of the disc and edema, sharp margins superiorly, greyish cotton wool spot temporal to disc margin in papillomacular bundle, macula WNL **fundus photos available**

PION initial eye examination 12/22/10
- HM vision , first eye exam following cardiac surgery in the setting of CABG and hypotension requiring pressors/ICU
- APD OD noted in nursing notes during hospital stay beginning on 12/1/10
- 12/22/10:
  o BCVA HM PH NI OD, PH 20/25 OS
  o Red desat OD half as red as OS and darker
  o Pupils +APD OD
  o CVF nasal field cut OD, full OS
  o OD c/d 0.2, sharp margins, mild palor, nasal fullness
  o OS c/d 0.2, sharp margins, pal superficially along inf arcade (gliosis vs. resolving CWS) **fundus photos available**

Clinical exam 8/23/16
- OCT of posterior pole showed significant thinning across entire posterior pole inf>sup OD, very thin superior OS
- OCT RNFL showed significant thinning 360 OD and global thinning OS (thinnest SN and ST)
- Fundus findings
  o c/d 0.2, diffuse pallor OD
Laboratory studies:

Physical
- Left temporal artery biopsy 2/2/08 negative
- Right temporal artery biopsy 7/7/08 negative

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- CBC normal
- CT scan negative
- MRI: no acute territorial infarcts are present within the brain on diffusion images

III. Differential diagnosis

NAION 2008
- Primary/leading: Non-arteritic anterior ischemic optic neuropathy v non-arteritic; likely non-arteritic secondary to typical NAION presentation
- Superior disc is most vulnerable in nonarteritic anterior ischemic optic neuropathy and left optic nerve sectorally (superiorly)swollen on presentation
- Left eye’s visual deficit is classic for nonarteritic variant-preservation of acuity with largely inferior altitudinal defect
- Patient had two negative temporal artery biopsies
- Patient’s age at onset was 56
  - In large scale 27-year planned study of 145 biopsy-confirmed GCA patients, the youngest patient was 56 yo (hayreh, ophthalmologica 2003)
- Others considered- Arteritic anterior ischemic optic neuropathy

PION 2010- Giant cell arteritis v. surgical PION
- GCA highly unlikely given absence of other clinical symptoms and young age. More likely PION in setting of decreased vision first noted after surgery/ICU sedation, pale nerve, no swelling of nerve. This may have occurred either during CABG or during subsequent sepsis/hypotension
- Two negative temporal artery biopsies in past
- Vision significantly reduced to HM, classic for surgical PION
- ESR (74) likely elevated secondary to recent surgery, not GCA
IV. Diagnosis and discussion

Summary

- Patient experienced sequential ischemic optic neuropathies—the first NAION in left eye in 2008, second likely shock induced PION in right eye following surgery

Statistics and other information about condition:

- Sildenafil has theorized association with NAION, especially in settings of small c/d ratio (Pomeranz 2002), ?viagra use in this case, use just prior to onset not documented
- Non-arteritic anterior ischemic optic neuropathy is described as hypoperfusion of short posterior ciliary arteries that supply optic nerve head, risk of NA-ION in fellow eye is 25%, hypotension and anemia maybe be predisposing factors
- Prognosis for vision recovery is poor in PION, no proven effective treatment
- Risk factors for surgical PION: pediatric or elderly, male sex, obesity, anemia, hypotension or hypertension, perioperative blood loss, prolonged surgical time, prone positioning during surgery (Su 2016)
- Incidence of visual disturbances after spinal surgery is reportedly between 0.028 to 0.2%. Most prevalent visual disturbance is posterior ischemic optic neuropathy. Incidence of visual disturbances after cardiac surgery is 0.0009 to 25.6%. Most prevalent visual disturbance is anterior ischemic optic neuropathy. Risk factors include age, diabetes, long cardiopulmonary bypass time, and anemia (Kawaguchi 2009).
- Retrospective study of 42 patients with PION included 28 patients with etiology non-arteritic, 12 with arteritic, and 3 surgical. Visual acuity varied between 20/20 and no light perception and all 4 surgical PIONs were less than count fingers. Initially optic disc and fundus photos were within normal, developed palor in 6-8 weeks (Hayreh 2004)
- Cardiac and spinal fusion surgery have highest rates of perioperative vision loss—estimated 8.64/10,000 in cardiac and 3.09/10,000 in spinal fusion (Shen 2009).
- In a series of 27,915 patients who underwent cardiopulmonary bypass surgery, Nuttal et al found 0.018% patients with PION. Bilateral in 60.9% and 65.5% with one or more vascular risks. Mean hemoglobin was 9.5g/dl and ranged from 5.5h/dl to 14.2 g/dl (Buono 2005) Normal hemoglobin range for men is 14-18.
- The majority of PION patients without a history of clinically severe vascular disease, eight (89%) of nine, experienced a minimum postoperative hemoglobin value of <8.5 g/dL (Nuttall 2001).
- Occasionally ESR can be elevated after a variety of surgical procedures which may raise suspicion of giant cell arteritis. In the absence of other clinical signs or symptoms, diagnosis of giant cell arteritis should not be made on basis of elevated ESR or C-reactive protein alone (Buono 2005)

V. Treatment, management

NAION

- Patient placed on 60mg oral prednisone upon presentation and scheduled temporal artery biopsy and MRI for next day. Admitted to hospital and also started on brimonidine BID OS secondary to presumed neuroprotective effects
- Stopped prednisone 8 days later when arteritic anterior ischemic optic neuropathy was ruled out
- Patient instructions:
o avoid systemic hypotension including dosing BP meds in AM rather than PM
o avoid nighttime binge eating to prevent shunting of blood to GI tract in setting of nocturnal drop in BP, which could exacerbate poor perfusion of optic nerves
o avoid viagra and viagra-like products
o pt already underwent ENT surgery to treat sleep apnea

- although, controversial, suggested aspirin
- consider prophylactic IOP lowering in future with alphagan
- polycarbonate protective eyewear

PION

- prednisone 80mg PO daily, draw CRP and if elevated consider temporal artery bx and continue steroids. If normal, taper off prednisone
- polycarbonate protective eyewear
- no proven effective treatments other than preventing future occurrences

VI. Conclusion:

- should patients deemed “at risk” take prophylactic IOP lowering medication prior to surgery?
- are patients with history of NAION at more risk for PION during surgery?
- What is mechanism or reason for patients with PION to have bilateral v. unilateral presentation? Is there a pattern?
- Is having NAION protective against recurrence in same eye or in this case protective against PION? Considering patient had NAION in left eye and 2008 and unilateral PION in fellow eye 2 years later
- Consider pt history of ethanol abuse x many years

References:


