Abstract Title: Diagnosis and Management of Strabismus Post Scleral Buckle Surgery

Abstract: A patient presents with recent onset diplopia following scleral buckle surgery. This case reviews potential causes for acute onset diplopia and explores the use of vision therapy and prisms in restrictive strabismus post surgery.

I. Case History

- Patient demographics: 55 year old white male
- Chief complaint: recent onset diagonal double vision in the distance
- Ocular History:
  - Cataract surgery with IOL implant of the right eye (2007)
  - Pars plana vitrectomy (x 3) of the right eye (2014)
  - Scleral buckle (x 2) of the right eye (2014)
  - Nuclear Sclerotic Cataract of the left eye
  - Horseshoe retinal tear of the left eye (2/2016)
  - Retinal cryopexy of the left eye (2/2016)
  - Pars Plana vitrectomy of the left eye (4/2016)
  - Membrane peel and scleral buckle of the left eye (4/2016)

- Medical History:
  - Hypercholesterolemia
  - Sleep Apnea
  - Fatty Liver Disease
  - Major Depression
  - MTHFR Mutation
  - Morbid Obesity

- Medications:
  - Aleve 220mg
  - Allopurinol 100mg
  - AndroGel 20.25mg/1.25gr
  - Aspirin 81mg
  - Atorvastatin 40mg
  - Colace 100mg
  - DHEA 50mg
  - Neurotonin 100mg
  - Norco 5-325 mg
  - Nystatin 500,00 unit tablet
  - Wellbutrin SR 150mg
  - Xarelto 20mg
  - Pregnenolone
  - Super Energy Herbal Complex
  - Vitamin D
  - Multivitamin

- Other Salient Information:
  - The onset of the double vision was 6 weeks prior to the exam (two months after his most recent scleral buckle surgery).
• The double vision is diagonal in nature and present frequently when looking in the distance (watching TV, driving).
• The patient denies diplopia when he is reading and reports that his symptoms are better when doing near work. He denies diplopia prior and immediately after his most recent scleral buckle surgery.
• Two weeks prior to this visit, he was seen by a pediatric ophthalmologist who recommended prism and/or surgery to correct the double vision. The patient has had poor adaptation to Fresnel prism (intolerable blurred vision and unstable fusion), and is uncomfortable proceeding with surgery. He is seeking a second opinion regarding his treatment options for the double vision. The cause of the new-onset diplopia had not yet been investigated.

II. Pertinent findings

- Refractive Status and Best Corrected Visual Acuity:
  - OD: -5.50-0.50x135  DVA: 20/50
  - OS: -5.75-0.50x180  DVA: 20/15
- EOMs: No observable underactions or overactions. Full and unrestricted OD/OS.
- Pupils are equal, round, and reactive to light. No afferent pupillary defect noted.
- Confrontations: full to finger counting OD/OS
- Ocular Deviation:
  - Cover Test (7/18/2016):
    - 3m: 10pd Intermittent Right Esotropia, 8pd Intermittent Right Hypertropia
    - 40cm: 4pd Intermittent Right Esotropia, 6pd Intermittent Right Hypertropia.
  - Cover Test (10/19/2015):
    - 3m: Orthophoria
    - 40cm: 4 Exophoria
- Sensory Fusion:
  - Worth 4 Dot (7/18/2016):
    - Distance/Near: 4 dots to 5 dots (With red filter over OD, red dots positioned lower than green dots)
    - Interpretation: Intermittent diplopia consistent with right hyper
  - Worth 4 Dot (10/19/2015):
    - Distance/Near: 4 dots
    - Interpretation: Stable Fusion
- Comitancy:
  - Maddox Rod Test (maddox rod over OD):
    - Lateral Right Gaze: Red line lower than penlight by 0.5 units
    - Primary Gaze: Red line lower than penlight by 1 unit
    - Lateral Left Gaze: Red line lower than penlight by 1 unit
    - All other gazes: Almost super-imposed (red line very slightly lower than penlight)
      - Interpretation: Vertical diplopia greatest in primary gaze and left gaze. Data does not correlate with a specific muscle paresis pattern.
  - Parks 3-Step:
    - RIGHT Hyper
    - Vertical diplopia worse on LEFT Gaze
    - Vertical diplopia significantly worse on RIGHT head tilt
      - Interpretation: Right Superior Oblique under action
Correspondence:
- Major Amblyoscope Testing:
  - <D 10BO, 7BD OD
  - <S 10BO, 7BD OD
  - Douse: No movement
    - Interpretation: Normal Correspondence
  - Vertical fusional vergence ranges around <D:
    - Right infravergence: break 4pd/recovery 2pd
  - Horizontal fusional vergence ranges around <D:
    - PFV: 10pd BO (no blur point reported)
    - NFV: 14pd BI (no blur point reported)

- Prolonged upgaze fixation test:
  - After 1 minute of upgaze fixation, no change in palpebral fissure width or amount of ptosis OD/OS.

- Prism Trialing in Free Space:
  - Maddox Rod (red filter over OD): Intermittent fusion with 8BD over OD, but after several seconds, patient reported red line drooping further and further down. Increasing vertical prism to 16BD helped stabilize fusion for several seconds, but vertical diplopia was once again reported.
  - With 3BO OS, 5BD OD: fusion of 20/80 Snellen letter for several seconds, with diplopia (consistent with right hyper) after blinking.
  - Yoked 2BD OD/OS (in attempt to shift eyes in down gaze to reduce diplopia): Intermittent, unstable fusion.

III. Differential diagnosis

- Primary/leading:
  - Mechanical extra-ocular muscle restriction secondary to scleral buckle surgery

- Others:
  - Acquired cranial fourth nerve palsy (ischemic, cerebrovascular accident, mass)
    - Parks 3-Step testing showed a pattern consistent with a right superior oblique palsy. However, a superior oblique palsy is inconsistent with other comitancy data as well as the patient's entering symptomology. Diplopia is encountered greatest in down gaze in a patient with superior oblique palsy; however, this patient complained of double vision in the distance that is not present at near or in down gaze.
  - Decompensated congenital superior oblique palsy
    - In addition to above, the patient did not present with a head tilt and compensating vertical vergence ranges were insignificant, reducing the likelihood of decompensated congenital fourth nerve palsy.
  - Ocular myasthenia
    - Patient denied an increase in frequency of diplopia in the evening versus the morning. Results of upgaze testing demonstrated no change in palpebral fissure width OD/OS, inconsistent with myasthenia gravis.
  - Cavernous sinus syndrome
    - Given the new-onset esotropia in addition to the non-comitant vertical strabismus, it is possible that there could be a lesion at the cavernous sinus affecting multiple cranial nerves (CN IV and CNVI).
  - Skew deviation
Third nerve palsy
- Thyroid eye disease
- Multiple sclerosis
- Trauma
- Orbital tumor
- Neurosyphilis

IV. Diagnosis and discussion

- Persistent diplopia from restrictive strabismus is a potential complication following scleral buckle surgery. Up to 25% of patients experience persistent diplopia lasting more than three to six months after scleral buckle surgery.\(^1\) Vertical and cyclodeviations are encountered with greater frequency than horizontal deviations.\(^8\) Possible causes of diplopia post scleral buckle surgery include distortion of the globe by the buckle, damage to the rectus muscle insertions by traction sutures, and post-operative tissue swelling and scarring. Permanent orbital adhesions, muscle fibrosis, and tissue scarring often bind the rectus muscles down to the underlying sclera.\(^3,6\) The physical presence of the buckle is also a causative factor for development of strabismus after surgery.\(^7\) Other possible causes for persistent diplopia include the decompensation of an existing fusional weakness and myotoxicity from local anesthetic injection.\(^3,8\)

- Although the acute onset diplopia corresponds with the time of this patient’s scleral buckle surgery, it is important to rule out other possible, concerning etiologies. Differential diagnoses for an acute onset non-comitant strabismus include: fourth nerve palsy, third nerve palsy, skew deviation, thyroid eye disease, myasthenia gravis, multiple sclerosis, trauma, orbital tumor, cavernous sinus syndrome, and neurosyphilis.\(^10\) These differential diagnoses are possible, but less likely based on the patient’s case history and clinical presentation. Referral to neuro-ophthalmology ruled out a neurological etiology and excluded these diagnoses, confirming the cause to be extra-ocular muscle restriction secondary to scleral buckle surgery.

V. Treatment, management

- Determination of the cause of the new-onset non-comitant strabismus:
  - The patient was referred to neuro-ophthalmology; neurology ruled out neurological etiology per patient report, confirming the cause to be extra-ocular muscle restriction secondary to scleral buckle surgery.
  - Further testing/work-up to help exclude the above differential diagnoses if indicated
    - CN III or IV palsy, cavernous sinus syndrome, multiple sclerosis: Further neurological work-up or neuroimaging with CT or MRI
    - Myasthenia Gravis: Blood work (Anti-AchR, MuSK), Single fiber electromyography (EMG), Chest X-ray/Chest CT
    - Skew Deviation: assess for presence of head tilt, non-comitant strabismus, brainstem symptoms including vertigo, possible history of stroke or infarction
    - Orbital tumor: MRI of the orbits
    - Thyroid eye disease: CT or MRI to rule out tendon-sparing fusiform enlargement of the extra ocular muscle, thyroid panel
    - Neurosyphilis: Blood work (VDRL, RPR, FTA-ABS, TPPA, EIAs)
• Management of persistent diplopia:
  o Based on the above prism evaluation and history of poor adaptation with Fresnel prism, the patient did not appear to be a good candidate for prism at the time; he showed intermittent fusion, followed by vertical diplopia through a series of different prism magnitude presentations. Prism was not prescribed. A trial of 5-10 vision therapy sessions was recommended instead, in attempt to stabilize his motor fusion to potentially make him a better surgical or prism candidate.
  o After two weeks, the patient followed up with his habitual optometrist where he demonstrated more stable intermittent fusion in office (about 5 minutes at a time) with base out prism OS and base down prism OD. Fresnel prism (4BO OS and 3BD OD) was prescribed and dispensed.
  o The patient voluntarily consulted a second pediatric ophthalmologist who recommended that he have the BO and BD prism ground into glasses, with simultaneous fusional vergence training through his prism correction. The goal of motor fusional vergence training through vision therapy would be to improve the control the deviation and maximize the success of prism correction without the need for surgery.
  o The patient recently reported minimal instances of diplopia (although intolerable blurred vision) through his Fresnel prisms over a trial period of 6 weeks. He has ordered ground-in prisms and will be returning in 1 month to initiate vision therapy at our clinic. Therapy will emphasize peripheral fusion, smooth divergence, and smooth vertical vergences initially at near (where fusion is easily established). As binocularity is improved, vectograms, computer based vergence exercises, and loose prism jumps will be employed to help facilitate fusion in the distance as well.
  o Often times, diplopia after scleral buckle surgery spontaneously resolves within the first three months as the tissue swelling from surgery subsides. With persistent diplopia, however, treatment is necessary. Management options for diplopia include prisms, optometric vision therapy, monocular occlusion, botulinum toxin injection, scleral buckle removal, and strabismus surgery.12

• Relieving/Neutralizing Prism
  • Prisms are often an effective treatment option for persistent diplopia post ocular surgery. While one study demonstrated a 40% success rate in eliminating diplopia using prism alone, another study demonstrated that prism increases the success rate by 50% when combined with scleral buckle removal.1
  • Prism tends to be an effective treatment options for smaller vertical deviations post surgery.11
  • When prisms are used, it is important to prescribe the least amount to minimize negative prismatic effects including distortions, reflections, etc.12,13

• Vision Therapy
  • When there is significant vertical and horizontal strabismus post surgery, a combination of prism and optometric vision therapy can be employed, as spontaneous recovery is less likely with larger deviations.11
  • Optometric vision therapy can be incorporated in order to minimize the final amount of ground-in prism.11
  • If a patient demonstrates decompensated fusional ability after surgery, they may still be symptomatic even with prisms or surgical interventions as they may experience unstable fusion with more visual demands or greater fatigue. Vision therapy to
VI. Conclusion

An acute non-comitant strabismus can be alarming to an eye care provider, as a neurological etiology is a salient cause that immediately comes to mind. This case demonstrates the importance of paying attention to the case history when determining leading differential diagnoses. While it is crucial to rule out neurological etiology in a new onset strabismus, it is important to remember that retinal detachment surgery can result in restrictive strabismus and should be considered as a cause to acute onset diplopia when there is history of recent ocular surgery. When comitancy data is not consistent or does not fit with a specific muscle palsy pattern, keep in mind other etiologies such as muscle restriction post surgery and skew deviation.

It is important to know when prisms and/or optometric vision therapy is indicated for patients with persistent diplopia secondary to scleral buckle surgery. While prism is often an effective treatment modality, some patients may still experience unstable fusion despite prism. In these cases, vision therapy exercises to expand motor fusional ranges can be effective for improving the control the deviation, maximizing the success of prism correction, and thereby reducing diplopia without the need for surgery.

VII. Bibliography


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