Resident’s Day Submission
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Abstract: A 27 year old Caucasian male presents for evaluation of diplopia and dizziness. This case discusses a patient with sixth nerve palsy secondary to traumatic brain injury. The patient is being treated with occlusion therapy.

I. Case History

• 27 year old male presented for a neuro-optometric examination.
• Chief Complaint: horizontal double vision and dizziness.
• Medical History: Two months prior to the examination, the patient suffered a traumatic brain injury secondary to a 45 feet fall at work. Upon admission to the hospital, the patient had a Glasgow coma score (GCS) of 3. The patient was hypotensive, tachycardic, and febrile upon arrival to the hospital. A computed tomography (CT) scan revealed a right temporal epidural hematoma (EDH) along the right convexity, a right temporal lobe contusion and subarachnoid hemorrhage, a hemorrhage along the left posterior lateral midbrain, a longitudinally oriented left temporal bone fracture extending into the carotid canal, a small amount of pneumocephalus adjacent to the right temporal lobe representing a right temporal bone fracture, and facet widening of the C4-5, C5-6, and C6-7 cervical spine. The patient had multiple facial fractures and a left corneal abrasion for which he was evaluated by ophthalmology and was given erythromycin ointment. The patient also suffered from a myocardial contusion complicated by rhabdomyolysis.
• Medications:
  • chloral hydrate 500mg PO bedtime
  • docusate 100mg PO Q12H
  • enoxaparin 30mg SUB-Q Q12H
  • ferrous sulfate 300 mg PO TID
  • polyethylene glycol 3350 17 gm PO Q-M-W-F
  • propranolol 20 mg PO Q6H
  • acetaminophen-hydrocodone 15 ml PEG Q8H
  • erythromycin ophthalmic 1 appl left eye bedtime
  • ocular lubricant 1 drop OU QID
• Ocular History: laceration of the left eye which was surgically repaired at age 16.
• Social History: The patient worked in a chemical plant, smoked a pack a day, and had a history of significant substance abuse including alcohol, cocaine, and methamphetamine usage.
II. Pertinent findings

Upon eye examination, the entering acuities were of 10/10-2 OD and 10/15-3 OS in the distance and 20/30+ OD and 20/20-2 OS at near. Confrontation visual fields were full in each eye. Hirschberg and Krimsky revealed a 35 prism diopter constant left esotropia. Extraocular motilities (EOM) showed an abduction deficit (-5 underaction) in the left eye and full range of motion of the right eye. Pupils testing revealed 5 mm pupil in light and 6.5mm in dim of the OD and 3.5mm in light and 4.5mm in the dim of the OS with no afferent pupillary defect. Slit lamp examination was unremarkable except for a linear corneal scar and underlying transillumination defect (TID) of the iris at the 3 o’clock position in the OS. Intra-ocular pressures using tonopen was 13 mmHg OD and 8 mmHg OS. Internal ocular health was unremarkable.

III. Differential diagnosis

- Traumatic cranial nerve (CN) sixth palsy
- Restrictive esotropia due to orbital trauma
- Myasthenia gravis

IV. Diagnosis and discussion

- Diagnosis:
  - Left CN VI palsy secondary to traumatic brain injury
  - Constant left esotropia with diplopia secondary to left CN VI palsy
  - Anisocoria OD > OS
  - Corneal scar with corresponding iris TID, likely from past ocular injury

Sixth nerve palsy is the most prevalent extraocular muscle palsy; with an incidence of 11.3 in 100,000 \(^4\). Patients present with horizontal diplopia greater in the distance than near and an abduction deficit of the ipsilateral eye \(^1,2,3\). The diplopia is further exaggerated when the patient looks in the gaze of the affected lateral rectus muscle \(^2\).

In most cases sixth nerve palsies have an undetermined etiology (~26%). Other etiologies in the adult population include hypertension (~19%), coexistent hypertension and diabetes (~12%), trauma (~12%), multiple sclerosis (~7%), neoplasm (~5%), diabetes alone (~4%), cerebrovascular accident (4%), post-neurosurgery (3%), aneurysm (~2%) and other such as upper respiratory tract infection, congenital, and neurosarcoid (~8%) \(^4\).

It is important to note the anatomy of the abducens nerve as a lesion may occur anywhere along the course of the nerve. The abducens nucleus lies on the floor of the fourth ventricle in the dorsal pons lateral to the medial longitudinal fasciculus (MLF) where the facial nerve loops over it. The abducens nerve projects fibers to the ipsilateral lateral rectus muscle and to the contralateral medial rectus subnucleus via the MLF. The nerve emerges from the caudal pons and courses through the subarachnoid space superiorly to the level of the petroclinoid ligament. The nerve then enters the Dorello’s canal and enters into the cavernous sinus. Finally, the nerve...
enters the orbit through the superior orbital fissure and innervates the lateral rectus muscle\textsuperscript{6,7}. The abducens nucleus, subarachnoid space, petrous apex, and orbit are the common areas susceptible to trauma\textsuperscript{6}. Basilar skull fractures may also cause traumatic sixth nerve palsy\textsuperscript{2}.

V. Treatment, management

Sixth nerve palsy can be classified into three sections: traumatic, neurologically isolated, and non-neurologically isolated\textsuperscript{4,8}.

- **Traumatic:** Patients are treated conservatively with occlusion patch or temporary prism. Almost all partial traumatic sixth nerve palsies and 1/3 of complete traumatic sixth nerve palsies recover after 3 to 6 months.

- **Neurologically isolated:** Focused evaluation and management of underlying condition is needed in these patients. Patients are conservatively managed and closely followed until resolution or if new neurological signs or symptoms present. If new neurological signs and symptoms occur, a full comprehensive neurological evaluation including neuro-imaging is indicated.

- **Non-neurologically isolated:** These patients require a full comprehensive neurological evaluation with neuro-imaging.

Ocular examination includes visual acuities, extraocular muscle testing, forced duction testing, corneal sensitivity testing with cotton wisp, orbicularis strength testing, and biomicroscopy and funduscopic evaluation. Neuro-imaging is indicated when patients are younger than 45 years, other neurologic signs or symptoms are associated, vasculopathic disease is not present, papilledema is present, bilateral sixth nerve palsy is present, if head trauma is suspected, or if palsy does not resolve in 3 to 6 months. If giant cell arteritis is suspected immediate ESR, CRP and platelet panel must be ordered\textsuperscript{2,3}.

Treatment for sixth nerve palsy includes occlusion therapy, prism, botulinum toxin (Botox), Botox combined with strabismus surgery, and strabismus surgery alone. Approximately fifty percent of acquired sixth nerve palsies spontaneously recover after 3 months of onset\textsuperscript{8}. It is for this reason an occlusion patch is used during the acute phase to eliminate diplopia. An occlusion foil may also be applied to the spectacles as an alternative. Prism may be used temporarily during the acute phase if the deviation is small enough to help the patient regain fusion. A prism can also be used permanently in patients with chronic stable sixth nerve palsy. A Botox injection is only considered after 3 months of onset if no significant improvement is seen. Surgical treatment is not considered until after 6 months of onset and only if the deviation remains stable\textsuperscript{1}. Recent studies have shown that initial Botox treatment alone and a single strabismus surgery have a low success rate; however, there was a 75\% success rate for chronic sixth nerve palsy with additional strabismus surgery, longer follow up, and use of a prism\textsuperscript{9,10}. A follow up examination should be done every 6 weeks until the palsy has resolved\textsuperscript{3}. Neuro-
imaging should be reconsidered if abduction deficit increases or new neurologic signs or symptoms develop.

Since our patient had traumatic sixth nerve palsy, our patient was conservatively managed. Our patient was advised to use a patch to occlude one of his eyes to prevent diplopia. We discussed with the patient and family the healing time for traumatic sixth nerve palsy and the need to monitor for improvement or stability every two months.

VI. Conclusion

An optometrist has the ability to successfully manage a patient presenting with sixth nerve palsy. By performing the appropriate tests, an optometrist should be able to diagnose sixth nerve palsy and recommend a course of action that will yield the greatest probability of success. As with most diagnoses, it is crucial for the optometrist to stress the importance of follow-up examinations. Finally, we need to successfully work with other healthcare professionals, like the neurologist and the primary care physician, to manage the underlying conditions that caused the ailment.

Bibliography: