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RIGHT SUPERIOR OBLIQUE Palsy OF TRAUMATIC ETIOLOGY

ABSTRACT

A patient presents with a right superior oblique palsy of traumatic etiology following a dirt bike accident. Neuroimaging does not show any abnormalities. In such a case, careful management and observation of the patient is crucial to remediate any disabling diplopia.

CASE HISTORY

-Patient demographics

53 year old, white male

-Chief complaint

Patient complains of worsening double vision since suffering from a dirt bike accident a couple of weeks prior. He reports that it is constant and that things appear diagonal to one another. When he closes either eye, the double vision goes away. He reports that it is worse in right head tilt and right head turn (left gaze). It is noticed at both distance and near. He gets relief by closing one eye or tilting his head to the left with his chin down.

-Ocular history

Previous eye examinations were remarkable for vitreo-retinal tufts in the periphery of the left eye, mild senile cataracts in both eyes, and minimal refractive error including presbyopia.

-Medical history

The patient’s medical history is remarkable for coronary artery disease, old myocardial infarction, and depressive disorder.

-Medications

He is on a number of medications for depression including Depakote, Lamictal, Celexa, and Trazadone. He takes Simvastatin for cholesterol and Metoprolol for blood pressure. He is also on Plavix and ASA 325/day for clot prevention.

-Other salient information (Summary of events and VA visits)

The patient was first seen in urgent care at VA Brockton on 06/29/10. He was in a dirt bike accident on 06/25/10. He was not wearing a helmet and fell on his face and his chest hit a stump. Per patient, he was
taken to Jordan Hospital then Boston Medical Center, where he spent the night and a CT scan was performed. The patient reported that he suffered rib fractures, right clavicle fracture, and a concussion. He complained of double vision, diffuse generalized headache, lightheadedness, and unsteady balance that had persisted but not worsened since his accident. He was told his double vision would resolve in a couple of weeks. When it did not resolve, he returned on 07/20/10 to urgent care, which referred him to the eye clinic. He was first evaluated by optometry at VA Brockton on 07/20/10, at which time he reported that his double vision had been present since a couple of days after his accident and had been getting worse over time. He was diagnosed with a right superior oblique palsy. He deferred treatment at that point and elected to close one eye or tilt his head to the left for relief. After that visit to the eye clinic, he was referred to neurology, where he was seen on 08/12/10, for further work-up. At that visit, there was no evidence of additional CN palsies or other focal neurological deficits. Neurology ordered a repeat CT scan. He returned on 08/17/10 to the eye clinic and his double vision was stable; however, he elected for glasses with prism to resolve his diplopic symptoms.

**PERTINENT FINDINGS**

-**Clinical**
  
  -Visual acuity, without correction, 20/25 OD, OS
  -Pupils: PERRL (-)-APD
  -EOMS: full, extensive, smooth, and accurate OD and OS, no restrictions
  -Confrontational fields: FTCF OD and OS
  -Cover test: constant right hyper
  -Parks 3 Step:
    -Primary gaze: 5 pd BD OD
    -Left head tilt: 2 pd BD OD
    -Right head tilt: 9 pd BD OD
    -Left gaze: 12 pd BD OD
    -Right gaze: ~1 pd BD OD

  **Double vision worse in right head tilt and left gaze.**

  -With double maddox rod test, measured <10 degrees extorsion OD.
  -Anterior segment unremarkable.
  -Posterior segment unremarkable.
  -Vitreo-retinal tufts noted, and stable to past, in the periphery inferiorly and superiorly temporally of the left eye.

-**Physical**

There was no evidence of facial bruising, trauma to either globe, no suggestion of orbital process right/left, and no papilledema right/left to argue for increased intracranial pressure in the setting of head trauma.

-**Radiology studies**

A head CT was apparently performed at Boston Medical Center, but the report was unavailable. Per patient, he stated that he was told the scan was normal. A repeat CT was performed at VA West Roxbury seven weeks after the incident and did not show any abnormalities. Patient has hardware in his left arm, which precluded MRI.
DIFFERENTIAL DIAGNOSIS

-Primary/leading

  Given the temporal relationship of the diplopia to his head injury, this is almost certainly a right superior oblique palsy of traumatic etiology.

-Others

An **isolated fourth nerve palsy** could also be caused by the following:

  a. Decompensation of a congenital trochlear nerve palsy

  "The presence of head tilt on old photographs and large vertical fusion amplitudes supports decompensation of a congenital trochlear nerve palsy (Jacobson 266)."

"The fourth cranial nerve nucleus is located in the dorsal mesencephalon. From here, the nerve fibers then decussate and exit the brain stem dorsally into the subarachnoid space. The nerve then courses around the brain to enter the cavernous sinus, superior orbital fissure, orbit, and innervate the superior oblique muscle." (Handbook of Ocular Disease Management website)

  b. In the brainstem, causes can include hemorrhage, infarction, trauma, hydrocephalus and demyelinization (may see a Horner's).

  c. In subarachnoid space, causes can include tumors, aneurysms, trauma and ischemic vasculopathy (diabetes, hypertension) (Mollan 644).

  d. In cavernous sinus, causes can include herpes zoster, inflammation of the cavernous sinus or posterior orbit, meningioma, metastatic disease, pituitary adenoma, and carotid cavernous fistula (may have co-involvement of CN3, 6, V1 and V2).


**Rare: Tumor, hydrocephalus, aneurysm, GCA (Kanski 820)**

**Binocular vertical diplopia** could also be produced by the following (The Wills Eye Manual, p. 236):

  a. Myasthenia gravis – ptosis, trouble breathing or swallowing, worsen with fatigue
  b. Thyroid-related orbitopathy – proptosis, eyelid lag, positive forced-duction test
  c. Orbital inflammatory pseudotumor – pain, proptosis
  d. Orbital fracture – history of trauma, positive forced-duction test
  e. Skew deviation – three step does not isolate a particular muscle
  f. Incomplete third nerve palsy – inability to look down and out, droopy lid
  g. Brown syndrome – limitation of elevation in adduction due to restriction of superior oblique tendon, congenital or acquired
  h. Giant Cell Arteritis - >50 years old, systemic symptoms, nonspecific motility defects due to extraocular muscle ischemia or neural ischemia
**DIAGNOSIS**

The superior oblique muscle originates from the orbital apex. The primary action of the superior oblique muscle is intorsion, the secondary action is depression (primarily in the adducted position) and the tertiary action is abduction. It is the only extraocular muscle innervated by the fourth cranial nerve. The trochlear nerve has unique features. It is the only cranial nerve that exits from the dorsal aspect of the brain. It is also a crossed cranial nerve; therefore, it innervates the contralateral superior oblique muscle. Moreover, it is a very long and thin nerve, which makes it susceptible to trauma.

"Acute onset of vertical diplopia in the absence of ptosis, combined with a characteristic head posture, strongly suggests fourth nerve disease." (Kanski 820).


- Binocular diplopia – vertical or oblique

Signs of a right superior oblique palsy (Kanski 821).

- Right hypertropia in the primary position of gaze due to weakness of the right superior oblique.
- Right limitation in depression in adduction due to superior oblique weakness.
- Excyclotorsion.
- The right hypertropia increases on left gaze due to right inferior oblique over-action and right head tilt.
- A head tilt toward the left shoulder to eliminate double vision.

**DISCUSSION**

In this case, impact of the midbrain/CNIV against the right tentorium is the most likely mechanism of such an injury. "After head impact, the brainstem moves backwards, so that the tentorium collides with either the dorsal midbrain or the fourth cranial nerve exit zone in the anterior medullary velum (Sudhakar 62)." In many cases, neuroimaging fails to show a lesion; however, a midbrain hemorrhage is occasionally noted (Sudhakar 50). CT may not be sufficient to identify such a hemorrhage, but MRI is precluded in this case. Additionally, given that this patient's CT scan was performed seven weeks following the head injury, an earlier hemorrhage may have resolved. Moreover, the orbit scan was expected to have low yield since the patient did not show any signs of facial bruising five days post-incident with reported negative initial head CT (no orbital fracture) and lack of orbital signs on examination.

**TREATMENT AND MANAGEMENT**

-Treatment and response to treatment

The patient was offered the option of monocular occlusion and/or prism to eliminate his diplopic symptoms. He initially elected to tilt his head (chin down, head tilt to left) or to close one eye to limit symptomology. This abnormal head posture helps to avoid diplopia. Per Kanski, "to intort the eye (alleviate excyclotorsion) there is contralateral head tilt and to alleviate the inability to depress the eye in adduction, the face is turned to the left and the chin is depressed."
However, three weeks later, he chose to use glasses with prism to eliminate his double vision. Glasses with 2.5pd BD OD and 2.5pd BU OS were ordered. He reported single vision with these glasses.

Since the CT scan was negative, it was decided to follow the patient periodically, quantifying the deviation to determine if it is resolving and attempt to remediate any disabling diplopic symptoms.

Patient will be seen again in mid-September.

Some options for management of persistent binocular diplopia (The Wills Eye Manual):

1. Head tilt/eye closure alone
2. Prism (Fresnel temporary prism or ground-in prism)
3. Monocular occlusion (patch, clip-on occlude, occlusive foil on spectacle lens, opaque contact lens)
4. Extraocular muscle re-alignment if no resolution in 6 months to 1 year.

"Defer surgery at least 6 months after the onset of palsy for the deviation to stabilize and because many palsies resolve spontaneously (237)."

CONCLUSION

-Clinical pearls, take away points if indicated

Cases of vertical diplopia should be considered a fourth nerve palsy until proven otherwise. The priority in patient care is patient education and minimizing disabling diplopia symptoms. Patients who have traumatic fourth nerve palsy should be observed for six months prior to surgical intervention because of the possibility of spontaneous resolution. Some traumatic palsies may recover as late as one year after injury. The literature shows that neuroimaging is not vital at the onset of diagnosis. However, if the palsy does not resolve in three months or if additional neurologic symptoms are present, imaging studies are indicated. In the setting of a cranial palsy following minimal head trauma, other etiologies should be sought such as underlying structural abnormalities. "In children, nearly all cases of isolated fourth nerve palsy are either congenital or traumatic in nature. In adults, approximately 40 percent of all isolated fourth nerve palsies are traumatic, 30 percent are idiopathic, 20 percent are due to vascular infarct, and only 10 percent are due to tumor or aneurysm." (Handbook of Ocular Disease Management website)
**I have signed consent from the patient to use these photos for academic purposes.**

A. Photos of patient – approximately a month after his accident

**PRIMARY GAZE**

* RIGHT HEAD TILT  
* LEFT HEAD TILT

RIGHT GAZE  
*LEFT GAZE
LITERATURE REVIEW/BIBLIOGRAPHY


Informative websites:

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