Acute Vertical Ophthalmoplegia Secondary to Unilateral Thalamic Stroke

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Patient & Chief Complaint

• 64 year old Caucasian male

• Presents to ER with sudden onset double vision, mild nausea, and a gait disturbance

• The double vision is described as constant, but resolves if either eye is closed

• The two images were described as separated diagonally
Physical Report from ER

• The patient exhibited disequilibrium while standing and displayed mild left sided gait disturbance while walking

• Otherwise, he was alert and had no other signs or symptoms of neurological deficits

• Blood pressure: 169/102

• Pulse: 108
Initial Studies

• CT Scan (Head & Neck):
  • Unremarkable

• Labs:
  • Glucose: 125 mg/DL (high)
  • Triglycerides: 159 mg/DL (high)
  • All other lab work was unremarkable
Medical History

- Hypertension – *white coat syndrome* (untreated)
- Impaired fasting glucose
- Obesity
- Possible TIA with left lower arm weakness in 2011. A full stroke work-up was performed at the time and all testing came back normal
Medications

• Aspirin, 325 mg QD
• Ibuprofen BID PO PRN
• Tadalafil 10mg PO PRN
• Multivitamin PO QD
Ocular History

• H/O retinal tear OS, s/p laser repair
• Nuclear Sclerotic Cataracts OU
• Refractive Error with presbyopia OU
Exam 1 – Day 1

• VA (cc): 20/20 OD and OS

• Pupils: PERRLA (-) APD

• EOMS:
  • Complete conjugate restriction of elevation and depression of both eyes in primary, right and left gazes
  • Horizontal gazes were intact
  • No gaze evoked nystagmus
Exam 1 (cont.) – Day 1

• CT (cc): 4^ Right Hypertropia with a 5^ Esophoria @ D & N

• Park’s 3 Step/Red Lens Testing: unable to isolate single cranial nerve palsy

• All other cranial nerve testing was normal

• Ocular health via slit lamp examination and undilated funduscopy was unremarkable
Differential Diagnosis

• Primary/leading:
  • Vascular: stroke or hemorrhage, midbrain or thalamus [1]

• Other differentials:
  • Compressive Mass – eg. tumor
  • Progressive Supranuclear Palsy (PSP)
  • Hydrocephalus – eg. cerebral aqueduct stenosis
  • Parinaud’s Dorsal Midbrain Syndrome
Treatment Plan – Exam 1

• Eye patch

• (Via the ER physician) patient was started on Clopidogrel Bisulfate and Atorvastatin

• The 325mg daily Aspirin was discontinued
Radiology Studies – Day 3

• MRI (Orbits):
  • Unremarkable

• MRI/MRA (Brain):
  • No mass, mass effect, or acute hemorrhage
  • 3-mm of increased signal in the inferomedial right thalamus, suggesting a subacute right thalamic lacunar infarct
  • The brainstem and cerebellum sections showed no signs of infarct
Incidence

Acute ischemic strokes of the posterior circulation

11-14% = thalamic infarction

16% = paramedian infarcts

89% = inferomedial

leading neurological findings:

87% = drowsines

39% = vertical gaze palsy \cite{11,12}
Exam 2 – Day 5

- CC: constant double vision, but improving, especially superior gaze

- EOMS:
  - Improved upgaze with greater elevation OD than OS, with upgaze evoked cyclonystagmus OU
  - Downgaze still partially restricted
  - Horizontal gaze remained intact
Exam 2 (cont.) – Day 5

• Doll’s Head (oculocephalic maneuver):
  • Vertical & Horizontal Vestibulo-ocular reflex (VOR) intact

• Optokinetic Drum:
  • Horizontal saccades and smooth pursuits preserved
  • Vertical saccades and smooth pursuit response absent

• Visual Field 30-2: reliable w/o defects OU
Treatment Plan – Exam 2

- Dispensed 4^BD Fresnel prism OD after the patient reported subjective improvement

- At this exam, the patient was educated that his double vision may continue to improve [2]

- Referral to neurology for consultation

- Referred to internal medicine to control vascular risk factors

- Return to clinic 4-6 weeks to reassess binocular vision posture & cranial nerve testing
Exam 3 – 7 weeks

- **CC:** Complete resolution of diplopia
- **EOMS w/ Red Lens:** SAFE
  - Cyclonystagmus resolved
- No detectable restriction of vertical gazes
- **Treatment Plan:**
  - Ground in prism not indicated
  - Continue as scheduled with neurology and primary care
Neurology of Vertical Gaze

• Pursuits = EOMS & VOR
• Saccades = Saccade Testing & OKN Drum
Control of Saccades

Frontal Eye Fields

Superior Colliculus

Rostral Interstitial Nucleus of MLF

Interstital Nucleus of Cajal (INC)

CN III & CN IV nucleus [1]
Control of Pursuits

*not well understood*

Visual Cortex/Parieto-Occipital Junction

\[\downarrow\]

Pontine Nuclei/Cerebellum

\[\downarrow\]

MLF

\[\downarrow\]

Rostral Interstitial Nucleus of MLF

\[\downarrow\]

Interstitial Nucleus of Cajal (INC)

\[\downarrow\]

CN III & CN IV nucleus [1]
The Underlying Neurology

- Selective (either upward or downward) vertical ophthalmoplegia is most often the result of bilateral dorsal or tegmental midbrain lesions [6]
The Underlying Neurology

- Although it has been reported in some cases, it is rare to have bilateral ophthalmoplegia from a unilateral thalamic infarct without midbrain involvement, especially involving both upward and downward gaze [3-5]
Schematic section through thalamus
(at level of broken line shown in figure at right)

**Thalamic nuclei**
- CM: Centromedian
- LD: Lateral dorsal
- LP: Lateral posterior
- M: Medial group
- MD: Medial dorsal
- VA: Ventral anterior
- VI: Ventral intermedial
- VL: Ventral lateral
- VP: Ventral posterior (ventrodorsal)
- VPL: Ventral posterolateral
- VPM: Ventral posteromedial

**Schematic representation of thalamus**
(external medullary lamina and reticular nuclei removed)
Theories on Thalamic Involvement

• Supranuclear afferent oculomotor fibers decussate and/or transverse the mediodorsal nucleus of the thalamus and then project to the frontal eye fields, which are responsible for initiating voluntary vertical gazes [7]

• Interruption to either of these pathways can lead to vertical gaze palsies [7-9]
Oculocephalic (Doll’s Head) Maneuver

- Typically indicated in an awake or comatose patient who has limited or absent voluntary eye movements [1]
What makes a VOR intact?

• Compare the reflex eye movements with VOR head tilt to the voluntary eye movements with EOM testing

• When the reflex movements are greater, the lesion must lie rostral to the midbrain, and is thus supranuclear [1]
Does this correlate with our patient?

Vertical ophthalmoplegia ✅

Intact vertical VOR ✅

= Supranuclear lesion

• The most common cause of acute supranuclear vertical ophthalmoplegia is a thalamic lesion, most commonly a medial thalamic infarction [1]
rostral interstitial MLF (riMLF)

- Located at the junction of the midbrain and thalamus
- Functions as the premotor nucleus, and is responsible for initiation of voluntary & involuntary vertical saccades
- Bilateral (or unilateral) damage typically abolishes all vertical and torsional saccadic movements [10]
riMLF & Saccade Testing

• In an attentive patient with vision 20/200 or better, a robust and symmetric nystagmus should appear in all positions of gaze

• Our patient displayed absent vertical saccades with the OKN drum

• This finding suggests either some direct damage to the riMLF or damage to its projections into the thalamus
Take Away Points

• The role of the thalamus in vertical eye movements

• VOR Testing and the Optokinetic Drum

• The importance of neuroimaging in ruling out emergent pathology and localizing CNS lesions

• Fresnel Prism

Image Sources (in order of appearance):
- https://www.bernell.com/product/A/Press_On
- https://www.bernell.com/category/779