Neuro-ophthalmology in the critically ill patient

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• I have no financial disclosures
Normal Exam

• Pupils
  – Isocoric
  – Reactivity
  – Shape

• Motility
  – Spontaneous
  – Passive

• Corneal reflex
• Oculocephalic reflex
  – Frontal eye fields
• Pupil wiring
• Corneal wiring
Esodevation
Esodeviation

• Differential diagnosis
  – B/L CN 6 palsy
    • Traumatic
    • Increased ICP
  – Thalamic esodeviation
    • Thalamic infarct/hemorrhage
Pseudoabducent palsy:
When a VI nerve palsy is not a VI nerve palsy

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Ophthalmoplegia
Teaching Video NeuroImage:
Near complete ophthalmoplegia in GQ1b antibody-positive Miller Fisher
Video and MRI correlation
Fulminant acute inflammatory demyelinating polyradiculoneuropathy mimicking clinical brainstem death

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Background: The premise for diagnosing brainstem death is the demonstration of irrevocable absence of function of the brainstem, despite the artificial maintenance of ventilation and circulation. Prerequisites include identifying the proximate cause of brain injury and excluding reversible causes of coma (e.g. metabolic, toxic). Fulminant acute inflammatory demyelinating polyradiculoneuropathy (AIDP) variants may result in a clinical state of quadriplegia, respiratory failure and abolition of brainstem reflexes. In such cases, central nervous system (CNS) disorder may be postulated and a diagnosis of brainstem death may erroneously be made.

Case: We report a young, previously healthy female patient that presented with a rapidly progressive course of neurological deterioration, lapsing into a clinical state resembling brainstem death; patient was apparently comatose, unresponsive, with complete ophthalmoplegia, absent brainstem reflexes and flaccid areflexic quadriplegia. Laboratory studies revealed a previously unrecognized systemic lupus erythematosus (SLE). The lack of an unequivocal cause of brainstem death prompted yet further investigation, which resulted in the electrophysiological diagnosis of fulminant peripheral polynueopathy.

Following steroid, plasma exchange and eventually Rituximab therapy, complete recovery was achieved.

Conclusions: Severe neuropathy with complete de-efferentation, can simulate “brain death”. Determination of brain death, albeit clinically evident, should not be done where a cause is not clearly determined. Thence, investigations (radiological, electrophysiological and functional) and careful history taking, where clinical evidence of CNS function is unobtainable, are of paramount importance.
Ophthalmoplegia

• Wernicke’s
Versive eye movements

• Epileptic
• Deficit
  – Frontal eye fields
  – Wrong way eyes
• Hemianopia or neglect
Pupils

• Symmetric
Anisocoria
Pourfour du Petit Syndrome in a Patient with Thyroid Carcinoma

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Horner’s
CN3 palsy
Anisocoria

- Sympathetic
  - Horners
  - Pourfour du petit
- Parasympathetic
  - CN3
- Mechanical
PUPIL

• RAPD
  – Corneal edema
  – Vitreous heme
  – Macula/retina
  – Optic nerve
    • PION p lumbar surgery
  – Hemanopia
Nystagmus

- Epileptic
Nystagmus

• Central patterns
  – Direction changing
HINTS to Diagnose Stroke in the Acute Vestibular Syndrome

Three-Step Bedside Oculomotor Examination More Sensitive Than Early MRI Diffusion-Weighted Imaging

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Background and Purpose—Acute vestibular syndrome (AVS) is often due to vestibular neuritis but can result from vertebrobasilar strokes. Misdiagnosis of posterior fossa infarcts in emergency care settings is frequent. Bedside oculomotor findings may reliably identify stroke in AVS, but prospective studies have been lacking.

Methods—The authors conducted a prospective, cross-sectional study at an academic hospital. Consecutive patients with AVS (vertigo, nystagmus, nausea/vomiting, head-motion intolerance, unsteady gait) with ≥1 stroke risk factor underwent structured examination, including horizontal head impulse test of vestibulo-ocular reflex function, observation of nystagmus in different gaze positions, and prism cross-cover test of ocular alignment. All underwent neuroimaging and admission (generally <72 hours after symptom onset). Strokes were diagnosed by MRI or CT. Peripheral lesions were diagnosed by normal MRI and clinical follow-up.

Results—One hundred one high-risk patients with AVS included 25 peripheral and 76 central lesions (69 ischemic strokes, 4 hemorrhages, 3 other). The presence of normal horizontal head impulse test, direction-changing nystagmus in eccentric gaze, or skew deviation (vertical ocular misalignment) was 100% sensitive and 96% specific for stroke. Skew was present in 17% and associated with brainstem lesions (4% peripheral, 4% pure cerebellar, 30% brainstem involvement; \( \chi^2, P=0.003 \)). Skew correctly predicted lateral pontine stroke in 2 of 3 cases in which an abnormal horizontal head impulse test erroneously suggested peripheral localization. Initial MRI diffusion-weighted imaging was falsely negative in 12% (all <48 hours after symptom onset).

Conclusions—Skew predicts brainstem involvement in AVS and can identify stroke when an abnormal horizontal head impulse test falsely suggests a peripheral lesion. A 3-step bedside oculomotor examination (HINTS: Head-Impulse—Nystagmus—Test-of-Skew) appears more sensitive for stroke than early MRI in AVS. (Stroke. 2009;40:3504-3510.)

Key Words: cerebrovascular accident ■ diagnosis ■ neurologic examination ■ sensitivity and specificity ■ vertigo
Eyelids

• Ptosis
  – MG, AIDP
  – CN3, Horner’s
  – Brainstem (infarct)
NMDA receptor encephalitis
Eye movement abnormalities

- Oculogyric crisis
- Opsoclonus
- Ocular dipping
Questions