Bilateral Globus Pallidus and Midbrain Infarcts in a Patient with Polysubstance Use Disorder After Cardiac Arrest

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Introduction

Internuclear ophthalmoplegia (INO) is an ocular movement disorder presenting as inability to perform conjugate lateral gaze and ophthalmoplegia due to damage to the medial longitudinal fasciculus (MLF).[1] MLF can be damaged by any ischemic, demyelinating, neoplastic, or inflammatory lesion in pons or midbrain. Previous case reports have described bilateral globus pallidi lesions in patients with substance use disorder, and after cardiac arrest.[2] CN III, responsible for pupillary constriction and most eye movements, exits the brainstem at base of midbrain.[3] Convergence insufficiency (CI) occurs when patients cannot converge eyes and maintain binocular fusion while focusing on near target.[4] Exodeviations are present in 1% of the population,[5] and CI is present in 11-19% of children with an exodeviation.[6]

Case Presentation

- Patient was a 21-year-old male with history of Marcus Gunn Jaw Winking Syndrome, physiologic anisocoria, complaining of binocular diplopia
- Further medical history of alcohol use disorder, depression, seizure, polysubstance abuse (marijuana, cocaine, opioids)
- Admitted to ICU after found down for 18 hours at home, intubated in ED for low Glasgow Coma Score, suffered cardiac arrest for two minutes due to hyperkalemia and rhabdomyolysis
- Urine toxicology positive for fentanyl, opiates
- Ophthalmology consulted for new-onset binocular diplopia and transient anisocoria OD>OS
- Ophthalmic exam revealed exotropia with limited upgaze of both eyes and absence of convergence, ptosis OD (MRD1 2mm) (**Figure 1**), juxtapapillary intraretinal heme OD, tortuous retinal vessels OU
- Sustained nystagmus in left lateral gaze, vestibulo-ocular reflex intact, no light-near dissociation, transient anisocoria by witnesses
- MRI brain showed enhancing lesions in bilateral globus pallidus (**Figure 2a**) and midbrain (**Figure 2b**) with questionable abnormal T2 signal with loss of CSF along right CN III

Testing

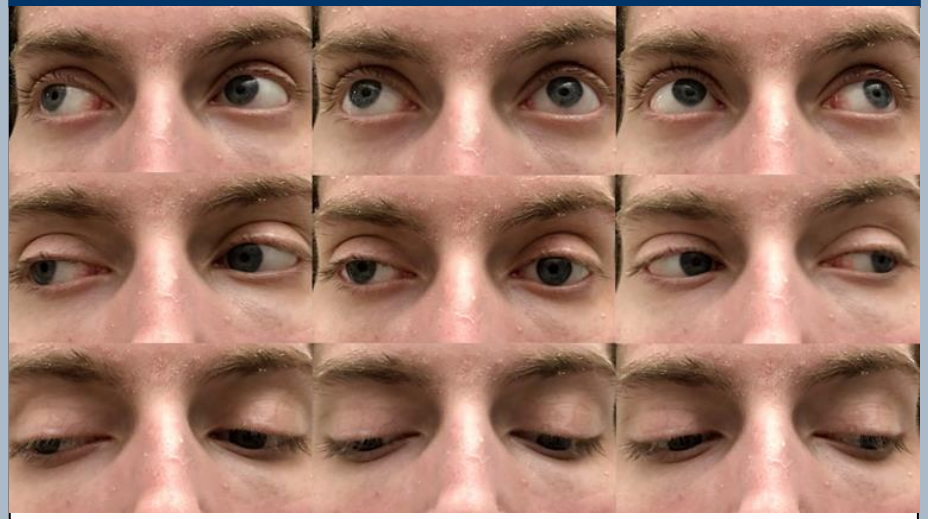
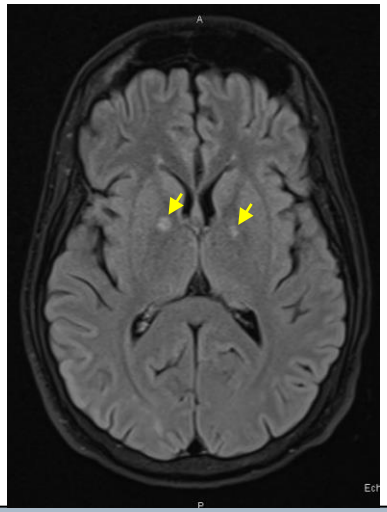


Figure 1: Ocular motility photo (above). Exotropia and mild right-sided ptosis (MRD1: 2mm). Anisocoria no longer present, while convergence insufficiency persisted.



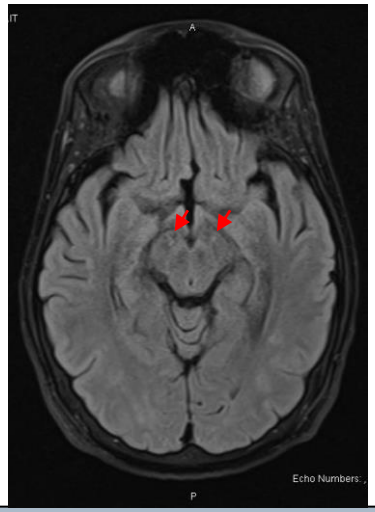


Figure 2a, 2b: T2-Weighted MRI, transverse. Hyperintense lesions in bilateral globus pallidi (yellow arrows) and midbrain (red), where CN III fascicles become subarachnoid.

Case Presentation (continued)

- Initial concern for vertical supranuclear gaze palsy vs. Parinaud's dorsal midbrain syndrome
- Patient advised to patch either eye, neurology consulted
- Subsequent inpatient follow-up revealed improved upgaze restriction, and symmetric, mildly restricted adduction OU without contralateral nystagmus
- 30 PD constant exotropia in primary, up, downgaze at near with Hirschberg
- 20-25 PD constant exotropia in primary gaze at distance with Hirschberg
- Patient remained unable to converge
- Outpatient follow-up one month later revealed resolution of diplopia post-patching, full extraocular movements, small exotropia on left gaze, and no intraretinal heme
- Likely diagnosis: convergence insufficiency with possible internuclear ophthalmoplegia vs. CN III neuropathy

Conclusion

- Cardiac arrest and toxin exposure may result in neurological ischemia, impacting the globus pallidus and brainstem
- Ischemic changes may impact cranial nerves in the acute setting, causing symptoms like binocular diplopia and transient anisocoria

Reference

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