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Teaching Caseimage: Acute unilateral internuclear ophthalmoplegia (INO) rapidly progressing to Exotropic Bilateral INO (EBINO)

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A 69-year-old man with hypertension, diabetes, and chronic atrial fibrillation on apixaban developed acute onset dizziness and diplopia. Initial neurological examination was remarkable for a right-sided internuclear ophthalmoplegia (INO) and a mild left upper motor neuron (UMN) facial palsy. Direct and indirect pupillary light reflexes were normal, visual fields were full to confrontational testing, and there was no ptosis. Initial non-contrast CT and MRI of the head revealed chronic white matter ischemic changes. Twenty four hours later his diplopia worsened, and interval examination revealed an exotropic bilateral INO (EBINO) with slow abducting saccades during fast eye movements [Video 1]. Vertical gaze and convergence were normal, and no up beating nystagmus were appreciated. The left-sided facial palsy was confirmed by the patient to be chronic. The remainder of neurological examination was unremarkable. A repeat MRI of the brain demonstrated an acute ischemic infarct only in the medial dorsal pons, and chronic microvascular changes [Fig. 1].

Wall-eyed bilateral INO has been long described. Hereafter, we will describe the condition with the less pejorative phrase EBINO. Classically the patient with EBINO will have significant exotropia with bilateral limitation of adduction of the eyes with abducting nystagmus. The underlying pathophysiological process occurs from disruption of the medial longitudinal fasciculus (MLF) which allows communication between the abducens nerve nucleus and the contralateral oculomotor nerve nucleus for a coordinated horizontal gaze. EBINO is specifically caused by lesions affecting the bilateral MLF.[1] The vast majority of cases of EBINO are observed in multiple sclerosis by a demyelinating plaque affecting both MLF, and seldom seen in brain stem stroke.[2] A single terminal paramedian tegmental pontine artery occasionally supplies the region of the MLF bilaterally.[3] Commonly in stroke etiologies of EBINO, the onset of presentation is sudden; however, the sequential progression in 24-h from unilateral INO to EBINO is rare.[4] The critical takeaway from this case is to always consider a common condition as the cause of an uncommon syndrome, as demonstrated in the repeat MRI showing a lacunar infarct causing EBINO.[5] Symptom progression in patients with lacunar strokes has been described in the past, commonly evolving within days after onset.[6,7]

Patient consent

Consent to publish this case report has been obtained from the patient(s) in writing.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

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Abbreviations: INO, Internuclear Ophthalmoplegia; EBINO, Exotropic Bilateral INO; MLF, Medial Longitudinal Fasciculus.

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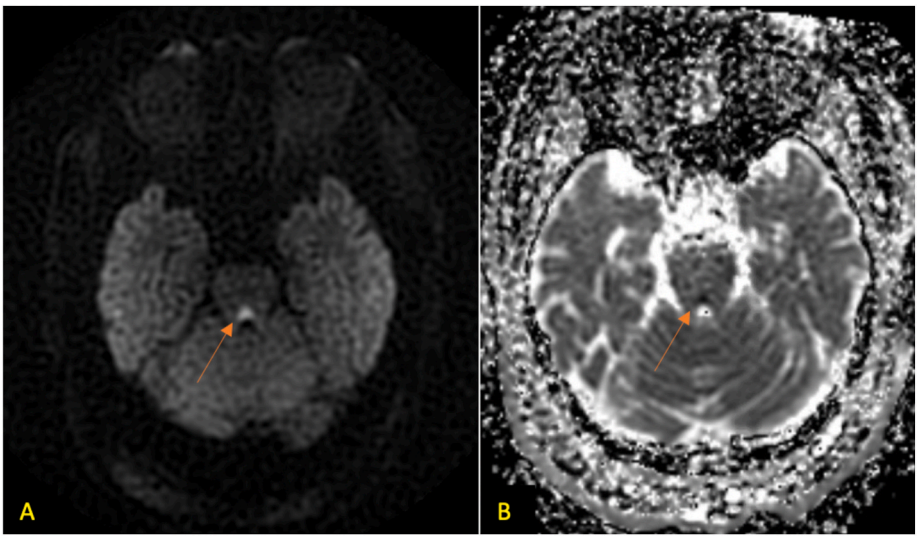

Fig. 1. (A) Brain MRI-DWI (Diffusion Weighted Imaging sequence) twenty-four hours after progression from unilateral INO to EBINO reveals a hyperintense lesion in the midline of the dorsal aspect of the upper pons involving the bilateral medial longitudinal fasciculus regions (arrow). (B) Brain MRI-ADC (Apparent Diffusion Coefficient map) demonstrating a hypointensity in the matching area of restricted diffusion confirming an acute infarction (arrow).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ajoc.2022.101509.

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