Ipsilesional Torsional Nystagmus in Midbrain Infarction: A Rare Entity of Localizing Value

Sir,

The midbrain is home to the control of vertical and torsional gaze. The rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) contains excitatory burst neurons (EBN) that generate vertical and ipsiversive saccades.[1]

A 50-year-old male presented to our neurology clinic with a sudden onset of rotatory vertigo and vertical diplopia 3 days prior to presentation. He was a hypertensive on irregular treatment. On examination, he was alert and well-oriented. Higher mental functions were normal. Oculomotor abnormalities were noted in the form of right eye hypertropia and left eye hypotropia. He had torsional nystagmus (TN) at midposition combined with a vertical component. The quick phase of torsional nystagmus showed rotation of the upper poles to the right (that is, excyclotorsion of the right eye and incyclotorsion of the left eye) in a clockwise direction for the patient [Video 1]. Vertical nystagmus component in gaze straight ahead was upward [Figure 1]. He showed combined up- and downward saccade slowing. There was vertical gaze paresis (down > up). Head tilt was to the left. There was a normal pupillary reaction to light. There was neither convergence-retraction nystagmus nor an eyelid retraction. There was mild swaying to the right on tandem gait and the left plantar was extensor. Other neurological and systemic examinations were unremarkable. His blood pressure was 146/88 mmHg. Complete hemogram, renal, hepatic, and thyroid functions were normal. Magnetic resonance imaging (MRI) of the brain showed an acute infarct in the right caudal midbrain involving the cerebral peduncle and the tegmental regions [Figure 2a and b]. He was treated with antiplatelets and statins. The ophthalmologic symptoms resolved completely over the subsequent 2 weeks although he had mild persistent gait swaying.

Whereas riMLF on each side sends axons of EBN to yoke muscles on both sides responsible for vertical saccades, the control of torsion is unilateral and ipsiversive.[2] Hence, the right riMLF will cause torsion to the right, or clockwise from the subject's point of view (that is, right eye extorsion and left eye intorsion). The intersitial nucleus of Cajal (iC) projects to the oculomotor neurons through ipsilateral connections as well as via the posterior commissure and is responsible for vertical and torsional gaze-holding.[3] Bilateral iC lesions will lead to vertical gaze-evoked nystagmus, impaired vertical and torsional vestibulo-ocular reflex as well as vertical saccade restriction with normal speed within the limited range. Unilateral iC lesions produce ipsiversive torsional nystagmus and contraversive ocular tilt reaction (OTR).[4]

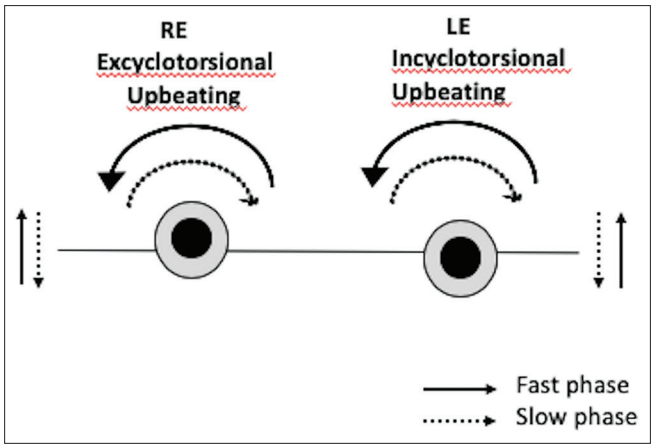

Figure 1: Diagrammatic representation of nystagmus observed in the patient in right eye (RE) and left eye (LE)

Hence, the directionality of the TN helps to distinguish riMLF lesions from iC lesions. Torsional nystagmus is defined as ipsilesional if the rotation occurs in the direction of the lesion, as in our patient. As such, torsional nystagmus is uncommon in midbrain lesions and may occur in either direction (ipsi- or contralesional) as previously reported.[5] Clinically, isolated lesions of the riMLF and iC may be distinguished by several features. Unilateral riMLF lesions lead to contralesional TN without vertical gaze-evoked nystagmus, vertical saccade slowing, and a loss of the quick phase of ipsilesional vestibulo-ocular reflex. Unilateral iC lesions lead to ipsilesional TN, vertical gaze-evoked nystagmus but no slowing of the vertical saccades. The localizing value of nystagmus in midbrain lesions has increased with reports that riMLF versus iC may be localized using TN and torsional saccadic assessment. In a series of 11 patients reporting TN in midbrain lesions, eight patients were found to have contralesional TN and only three to have ipsilesional TN.[5] The clinical findings that our patient exhibited were a combination predominantly of riMLF and iC lesions, similar to case 11 in the abovementioned case series.[5] In this series, oculomotor deficits were found to persist for prolonged periods of time, that extended even up to one year. However, in our case, the resolution of the oculomotor abnormalities occurred over the succeeding 10–14 days [Video 2].

The presence of torsional nystagmus in association with other ocular abnormalities helps provide localizing pointers in midbrain lesions.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

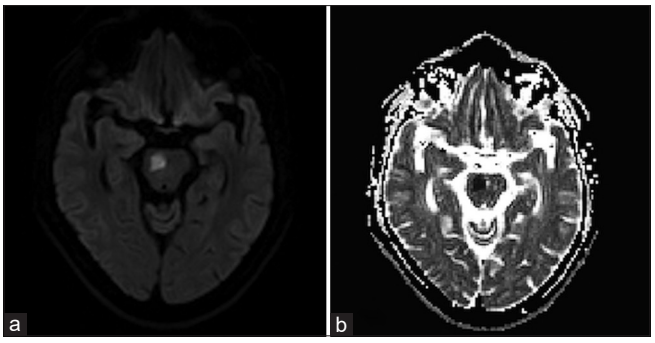

Figure 2: MRI brain showing an acute infarct involving the ventral aspect of the right caudal midbrain on diffusion-weighted imaging (a) and apparent diffusion coefficient (b) sequences

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Submitted: 05-Dec-2019 **Revised:** 12-Dec-2019 **Accepted:** 15-Dec-2019
Published: 09-May-2020

Videos available on: www.annalsofian.org

DOI: 10.4103/aian.AIAN_646_19