·Letter to the Editor·

Jerky see-saw nystagmus in internuclear ophthalmoplegia from a lower pontine lesion

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Dear Sir,

I am Dr. Jing-Wen Gong, from the Department of Ophthalmology, Zhejiang Provincial People's Hospital, Hangzhou, China. I write to present a peculiar case report of jerky see-saw nystagmus in internuclear ophthalmoplegia from a lower pontine lesion.

See-saw nystagmus (SSN) first described by Maddox [1], is an eye movement disorder characterized by alternating elevation and intorsion of one eye and simultaneous depression and extorsion of the other eye (Figure 1). Commonly, it is associated with chiasmal and parasellar lesions or invading the brainstem bilaterally at the meso-diencephalic junction.

Here we reported a patient who presented with jerky see-saw nystagmus with internuclear ophthalmoplegia (INO) due to a lower pontine lesion which was adjacent to the bulbus medullae and discussed the possible mechanisms of see-saw nystagmus in patients with INO.

A 33-years-old female presented in June 2010 due to the insidious onset of visual impairment in both eyes for 2 years and sudden-onset diplopia. She had ever visited the local clinic where paralytic strabismus was diagnosed. We advised her to take a brain MRI examination, but for some unaccountable reason she refused and left. Half a year later, she came to our department again and complained for the new symptom that her eyes 'kept dancing'.

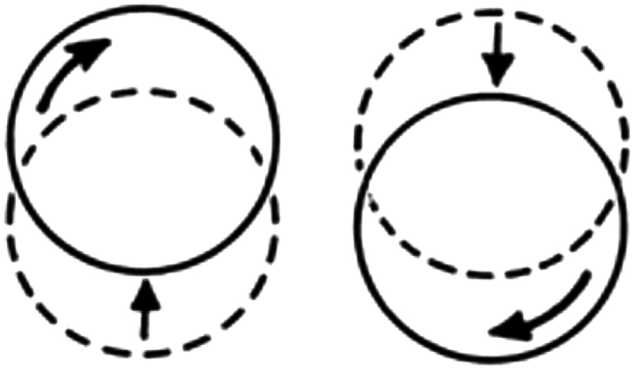

Figure 1 Diagram of combined rotatory and vertical nystagmus, the excursion of these movements has been exaggerated for greater clarity. As one eye rises, the other falls, the rising eye is introverted and the falling eye is extroverted.

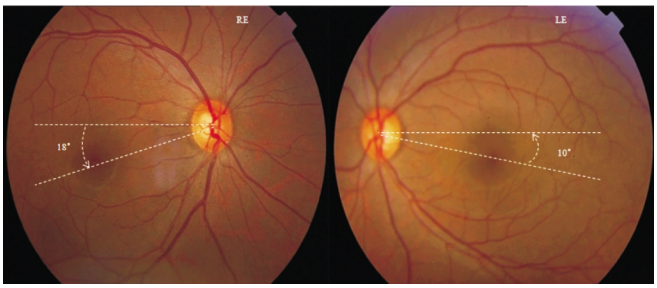

Figure 2 Fundus photographs show extorsion of the right eye.

The patient had a corrected visual acuity of 12/20 OD and 16/20 OS. Intraocular pressure (IOP) were 12mmHg (OD) and 14mmHg (OS). Her face turned right. Eye movement examination revealed combined lesions of the left INO and the right abducens nucleus: adduction paralysis of the left eye and abduction paralysis of the right eye. The convergent eye movement was slightly impaired and unstable. Another obvious clinical abnormality was a primary-position, jerk-waveform, torsional-vertical nystagmus with the following characteristics: 1) The torsional component was conjugate. The fast phases were anticlockwise. 2) The vertical component was disjunctive. The fast phases were up-beating in the intorting right eye, down-beating in the extorting left eye. 3) The nystagmus was present in all gaze directions and unaffected by changes in head position or in vergence angle. Fundus examination demonstrated extorsion of the right eye (Figure 2). It was found in neurological examinations that her Pin sensation was depressed over the right face and

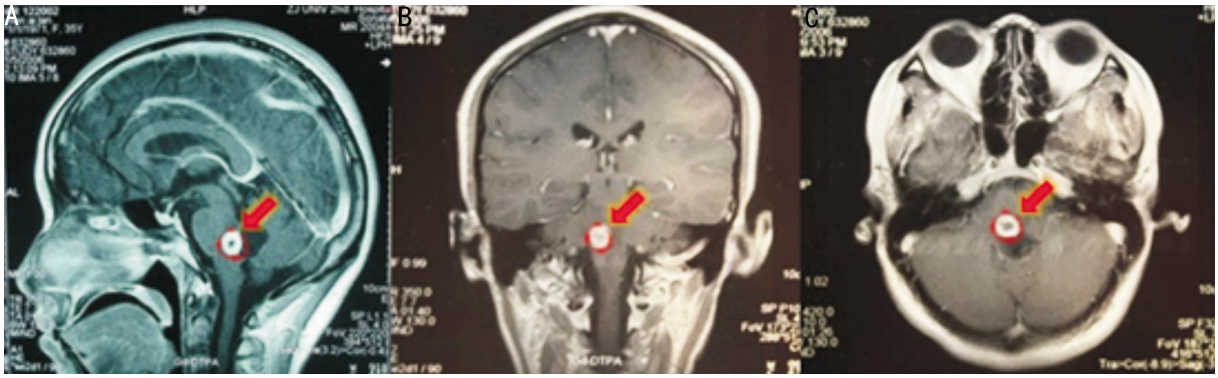

Figure 3 Brain magnetic resonance imaging (MRI) (T1-weighted image enhanced with gadolinium) from this patient. The red arrows indicate the tumor A: Sagittal image indicates at the dorsal brain-stem adjacent to the bulbus medullae there is a large mixed-signal-intensity lesion which compresses truncusencephalicus and the forth ventricle; B: Coronal image shows the lesion locates at the right pontine; C: Axial image shows a mixed signal intensity lesion.

forehead companied with mild right-sided finger-to-nose dysmetria.

Examination of visual field showed temporal hemianopic loss in the right eye. Brain MRI disclosed that a large lesion in the dorsal brain-stem, but the vestibular nuclei and mesencephalon were not invaded. The enhanced axial T1-weighted image showed at the right pontine there was a large lesion of mixed signal intensity (Figure 3).

She was finally diagnosed with a brainstem tumor and then received a removal surgery. The grade-II brainstem astrocytoma was confirmed by postoperative pathology. One month later, her best corrected visual acuity got some recovered to16/20 OD and 20/20 OS, with facial paralysis improved and visual fields returning to normal, but the see-saw nystagmus still existed.

Internuclear Ophthalmoplegia (INO) is characterized by adduction paresis of the ipsilesional eye and dissociated abducting nystagmus of the contralesional eye on attempted gaze to the contralesional side [2]. It is caused by the lesion involving the medial longitudinal fasciculus (MLF) between the abducens and oculomotor nuclei. This patient showed combined lesions of the left INO and the right abducens nucleus. The lesion in the MLF caused by enormous tumor in pons leaded to the adversion paresis of the left eye, and it also affected the abducens nucleus and/or the paramedianpontine reticular formation (PPRF), which produced the conjugate horizontal gaze palsy to the ipsilateral side.

Although see-saw nystagmus has been studied by many researchers, the exact etiology of SSN is still unclear, which may be associated with chiasmal and parasellar lesions such as pituitary adenoma, craniopharyngioma, syringobulbia, multiple sclerosis, severe head trauma, Arnold-Chiari malformations, congenital achiasma, cone-rod dystrophy, septo-optic dysplasia and retinitis pigmentosa [3-8]. Recent evidence suggests that most of the SSN are caused by dysfunction of the rostral brainstem [9]. Our case adds the lesion of lower pontine which was adjacent to the bulbus medullae, as a possible etiology for SSN.

The occurrence of jerk SSN in the INO patient, with the torsional fast phases beating toward the side of the lesion, has been rarely reported. According to the clinical and experimental evidences, it has been established that two midbrain nuclei have a major role in the control of normal torsional and vertical eye movements: the interstitial nucleus of Cajal (INC) acting as a velocity-to-position integrator and the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) acting as a torsional fast-phase generator[10]. Via the MLF, the INC carries excitatory fibers originating in the contralateral vertical semicircular canals, probably from the otoliths, to the ocular motor nuclei [11]. The MLF lesion could cause INC inactivation and induce a contraversive slow phase. We speculate that jerky SSN in our case is caused by the combined disruption of the fibers from contralateral anterior and posterior canals (Figure 4). As we know, the fibers from the contralateral anterior canal may link the superior vestibular nuclei to the superior rectus and inferior oblique subnuclei of the oculomotor nuclei. The fibers from the contralateral posterior canal innervate the inferior rectus in the ipsilateral eye and superior oblique in the contralateral eye. Thus, the combined disruption of the above two pathways would result in a weakness of the tonic innervation to the inferior rectus and inferior oblique in the left eye and the superior rectus and superior oblique in the right eye. Finally it caused the extorsional downbeat nystagmus in the ipsilesional eye accompanied by intorsional upbeat nystagmus in the contralesional eye [12,13].

The exact mechanism of jerky SSN in INO is still unknown. According to the previous reports, INO may accompany various patterns of torsional-vertical nystagmus, depending on the pathways from contralateral vertical semicircular canals involved [13]. Our case expands the causes of jerky SSN and lends further support to the notion that the level of chiasma or midbrain lesions may not be a prerequisite for its development.

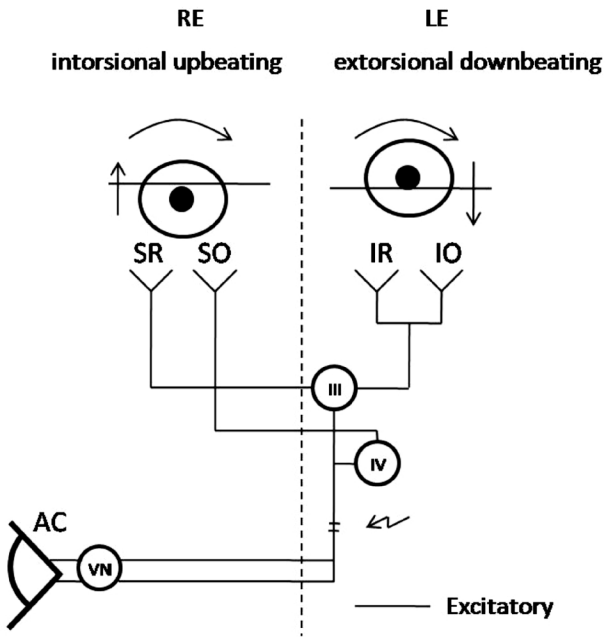

Figure 4 Hypothetical explanation of seesaw nystagmus in pontine lesion, Schematic representation of the three neuron vestibulo-ocular reflex arc between the semicircular canal and the extraocular muscles, showing combined damage to the pathways from the posterior and anterior semicircular canals (angled arrow) would give rise to conjugate ipsiversive torsional nystagmus with opposite vertical components RE: right eye; LE: left eye; SR: superior rectus; IR: inferior rectus; SO: superior oblique; IO: inferior oblique; III: oculomotor nucleus; IV: trochlear nucleus; VN: vestibular nucleus; AC: anterior semicircular canal; PC: posterior semicircular canal.

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