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Bilateral Vertical Gaze Palsy after Cerebral Digital Subtraction Angiography Due to Unilateral Midbrain Infarction

Dear Editor,

Cerebral digital subtraction angiography (DSA) is an invasive but useful technique for evaluating and treating many cerebrovascular diseases. Local and systemic complications related to DSA have been reported extensively in the literature [1,2]. Ocular complications of DSA include visual field defects, transient cortical blindness, and central retinal artery occlusion. This is the first report to present bilateral vertical gaze palsy after cerebral DSA due to unilateral midbrain infarction.

A 47-year-male patient presented to the ophthalmic department for vertical diplopia since the previous day. He was admitted to the neurosurgical department, where he underwent cerebral DSA. The procedure was conducted to diagnose an unruptured anterior communicating artery aneurysm. In the evening following the procedure, the patient began to complain of ocular symptoms. He had no history of any previous systemic disease, and there were no motor deficits or loss of consciousness. Visual acuity was 20 / 20 in both eyes. The pupils showed normal response to light and near stimulation. No saccades or smooth pursuit eye movements were present on attempted upward and downward gaze in both eyes (Fig. 1A). The Bell phenomenon was preserved. Fundus examination showed incyclotorsion of the right eye and excyclotorsion of the left eye (Fig. 1B, 1C). Diffuse-weighted magnetic resonance imaging (MRI) demonstrated acute infarction of the right upper midbrain in the region containing the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) and the interstitial nucleus of Cajal (INC); however, the posterior commissure (PC) was not affected (Fig. 1D). The patient was managed with antiplatelet treatment. One month later, he showed improved ocular alignment without diplopia (Fig. 1E).

The development of neurological complications after cerebral DSA is relatively infrequent: however, it can lead to permanent disability [1,2]. Previous studies have revealed that the risk of neurological complications was significantly higher in older patients, and those with cardiovascular disease and other underlying health conditions [1,2]. Kaufmann et al. [1] showed that hemiparesis was the most common neurological complication, and aphasia the second most common. However, this case demonstrated only vertical diplopia without other neurological symptoms. Moreover, the patient was relatively young with no other underlying systemic disease. Therefore, the occurrence of neurological complications after cerebral DSA is not entirely predictable, and any atypical ocular symptoms after cerebral DSA should not be overlooked.

Interestingly, in this case, bilateral vertical gaze palsy was caused by unilateral midbrain infarction. Unilateral lesions of the midbrain can cause a variety of vertical motility disorders; however, bilateral vertical gaze palsy due to unilateral midbrain infarction has rarely been reported. The exact mechanism is not yet clearly understood. The midbrain contains important neural structures for controlling vertical gaze, such as the riMLF, INC, and PC [3,4]. The riMLF contains the burst neurons that provide supranuclear input for the generation of vertical and torsional saccades [3]. The right and left riMLF are connected via the PC dorsally, and perhaps also by a commissure that lies ventral to the aqueduct. Upward gaze requires input from both riMLF; a unilateral lesion can produce a bilateral upward gaze deficit. The INC is considered the neural integrator for vertical and torsional gaze [4]. A lesion restricted to the INC may produce two distinct deficits: an ocular tilt reaction (ipsilateral hypertropia, extorsion of the contralateral eye and intorsion of the ipsilateral eye, and

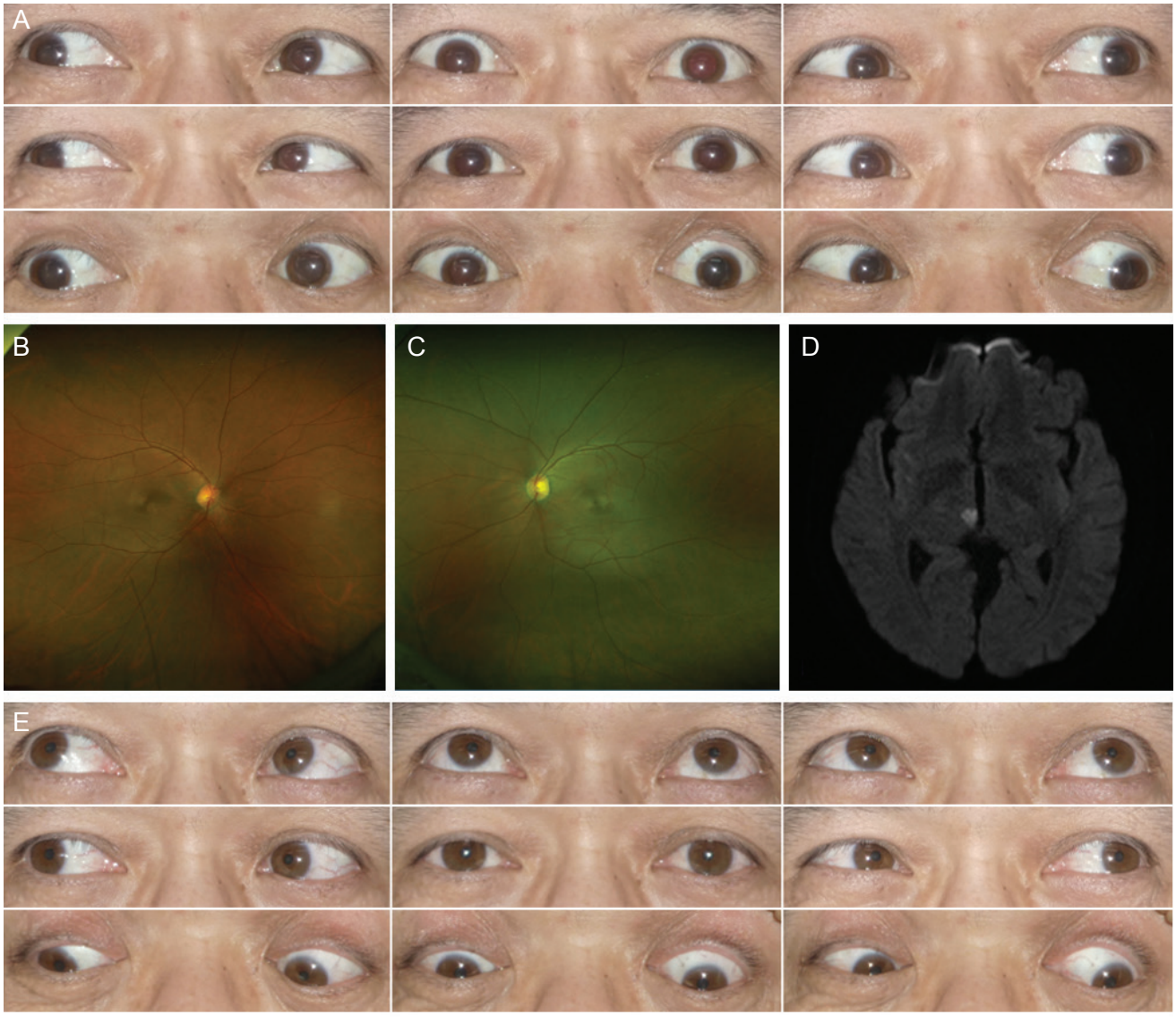

Fig. 1. Images of the patient in nine diagnostic positions of gaze, demonstrating bilateral vertical gaze palsy. (A) The pupils are dilated in both eyes because these photographs were taken after the dilated fundus examination. (B,C) Fundus photographs showed incyclotorsion of the right eye and excyclotorsion of the left eye. (D) Diffuse-weighted magnetic resonance imaging shows high signal in the right upper midbrain. (E) Images of the patient in nine diagnostic positions of gaze, demonstrating normal upward and downward ocular movement in the both eyes one month later.

controlateral head tilt) and defects in vertical pursuit and vertical/ torsional gaze holding. In the case of Pothalil and Gille [5], unilateral rostral midbrain infarction involving the left riMLF and INC but sparing the PC as observed on MRI, induced conjugate downward and upward gaze palsy. They postulated that slowing of downward and upward saccades is probably not only related to a loss of burst neurons in the left riMLF, but also related to additional damage of the crossing fibers from the opposite riMLF, via the ventral commissure, and of afferent fibers originating from the nucleus of the PC after their decussation in the PC. The impairment in downward pursuit reflected a left INC involvement. This hypothesis is consistent with the MRI and ocular findings in our case.

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Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Congenital Bilateral Inferior Rectus Muscle Absence with A-type Exotropia

Dear Editor,

Bilateral congenital absence of the inferior rectus muscle (IR) is very rare, especially without craniofacial deformities or other extraocular muscle (EOM) abnormalities. To our knowledge, this is the first report of bilateral IR absence with severe A-type exotropia without other EOM abnormalities.

A 2-year-old girl presented with exotropia and left hypertropia (LHT) since birth. She was born at full term and had no perinatal disease, family history, or facial deformity. She had left head tilt, 30 prism diopters (PD) of hypertropia, and 30 PD of exotropia in her left eye, with the angle of exodeviation aggravated on downward gaze (A-type exotropia). She also showed 30 PD of right hypertropia on right gaze and 35 PD of LHT on left gaze. Only right hypertropia was present on left head tilt, while LHT was presented and aggravated on right head tilt. Extreme limitation of downward gaze that could not cross the horizontal midline in the left eye and -5 limitation of inferotemporal movement in both eyes were found [1] (Fig. 1A). Slit lamp examination was normal, and fundus fixation photographs showed incyclotorsion of both eyes (Fig. 1B). Orbital magnetic resonance imaging indicated bilateral absence of IR (Fig. 1C).

At age 3, she underwent bilateral lateral rectus (LR) recession 7 mm for exotropia and left superior rectus (SR) recession 7.5 mm for LHT. A forced duction test showed limitation of downward gaze due to left SR contracture. Intraoperative examination showed bilateral absence of the IR, with no other EOM abnormalities (Fig. 1D). The patient showed orthotropia in primary and upward gaze and improvement in exotropia in downward gaze and in limitation of downward movement at 1 year after the surgery (Fig. 1E). The typical manifestation of the IR absence is incomitant hypertropia in the affected eye, which becomes worse with abduction, infraduction, or head tilt to the side opposite the affected eye. Vertical deviation with unilateral IR absence was corrected by recession and anterior transposition of the inferior oblique muscle. However, recession and anterior transposition of the inferior oblique muscle may aggravate incyclotorsion, a condition frequently present in IR absence [2]. Successful surgical outcomes have also been reported in congenital left IR absence with 65 PD LHT and 48 PD exotropia [3]. Transposition of the left medial rectus (MR) and LR to the original IR insertion site and 3-mm resection of the MR resulted in vertical strabismus below 10 PD, but remaining exotropia of 22 to 25 PD. The latter was corrected by a second operation, right MR resection and LR recession. Another report described a pa-