• Letter to the Editor •

Horizontal gaze palsy with abducens nerve palsy and skew deviation

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Dear Editor,

Dorsal pontine lesions may cause a variety of complex neuro-ophthalmic deficits, including horizontal gaze palsy (HGP), internuclear ophthalmoplegia, one-and-a-half syndrome, abducens nerve palsy, skew deviation, or any combination of these. Here we present a rare case of an adult patient who developed multiple complicated clinical manifestations after surgical removal of a pontine cavernous hemangioma (PCH). Our case highlights a single pontine lesion may involve complicated neural pathways and result in complicated symptoms and signs, in which abducens nerve palsy or skew deviation is easily missed when combined with HGP. A careful ocular examination revealed valuable neuroanatomical localizing clues: accompanying large esotropia indicating abducens nerve palsy, and conjugate ocular torsion and torsional jerk nystagmus indicating skew deviation.

CASE PRESENTATION

Ethical Approval All procedures performed in studies involving the patient were in accordance with the ethical standards of the institutional and national research regulation and with Declaration of Helsinki. The patient agreed for data to be collected for the purpose of this study, and the informed consent has been obtained.

A 23-year-old female presented to our strabismus clinic with a 5-year history of left face turn and inability to look left without diplopia after surgical removal of a PCH. The initial symptoms of primary PCH were only mild paroxysmal vertigo and headache with numbness in the right limbs. At age of 18y, she underwent presurgical neurological evaluation at an outside hospital. At that time, only slight loss of sensation in the right limb was detected, and the muscle strength of limbs were normal. Brain magnetic resonance imaging (MRI) scan identified a left pontine lesion (8×5 mm^2) indicating PCH. A series of surgical complications occurred after the resection of PCH at the outside hospital, including significant visual loss, ocular motility disorder, left facial paralysis and gait instability. She underwent comprehensive ophthalmic evaluation at our institution. The best corrected visual acuity (BCVA) was 20/60 in the right eye and counting fingers at 60 cm in the left eye. The pupil light reflex was normal in both eyes. Ocular motility examination revealed leftward HGP (Figure 1A) and torsional jerk nystagmus characterized by incyclo-oscillation in the right eye and excyclo-oscillation in the left eye was observed (online supplementary Video 1). Nine-gaze ocular movements examination revealed leftward HGP with extremely large esotropia in primary gaze. Extremely large esotropia was detected in the left eye in primary gaze, which was measured as 123 (50+45) prism diopters (PD) by Krimsky test. No dissociated vertical deviation was detected, and Bielschowsky head tilt tests were negative in both sides. Binocular stereopsis, whether measured with a synoptophore or a stereoacuity test, was absent. Doll's eye movement test showed complete abduction deficit of the left eye, while convergence and vertical movements were relatively intact in both eyes. Abnormal head position (AHP) of left face turn (Figure 1B) could be corrected by placing a base-in 25 PD prism in front of the right eye. Fundus photography revealed severe intorsion of the right eye and extorsion of the left eye (Figure 1C). Bell's phenomenon was intact in both eyes, but the hypophasis and paralytic ectropion of left lower eyelid and inability to close left eye were detected (Figure 1D). The patient's left face presented loss of eyebrow and forehead movement, loss of nasolabial

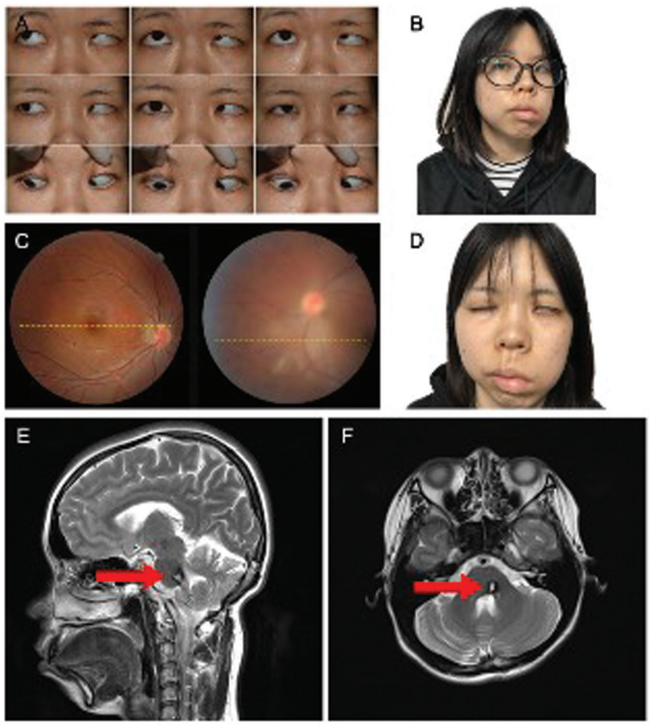

Figure 1 Clinical features of the present case Ocular motility examination in 9 gaze directions (A) revealed leftward HGP with extremely large esotropia of the left eye in primary gaze. Left face turn (B) was 16° measured by orthopedic goniometer, and the AHP could be corrected by placing a 25 PD base-in prism in front of the right eye. Fundus photography revealed objective conjugate ocular counter-roll (C) with the top poles rotated toward the left ear. The yellow dotted lines represented horizontal lines across the fovea. Bell's phenomenon was intact in both eyes, but hypophasis of left lower eyelid was detected (D). Brain MRI scan of a sagittal plane (E) and a transverse plane (F) revealed a local abnormal lesion (about 9×7×11 mm^3 in size, marked with red arrows) in the tegmental area of the left pons which suggesting chronic change after surgical removal of PCH. PD: Prism diopters; HGP: Horizontal gaze palsy; AHP: Abnormal head position; PCH: Pontine cavernous hemangioma; MRI: Magnetic resonance imaging.

folds, and lower lip droops (Figure 1D). The patient had gait instability involved right limbs. Repeat brain MRI showed a chronic lesion in the tegmental area of the left pons (Figure 1E and 1F).

DISCUSSION

HGP implies the inability to move both eyes into ipsilateral gaze when the patient tries to gaze in the affected direction[1]. The frontal eye field generates the excitation signal for horizontal eye movements, which then goes through the midbrain to the paramedian pontine reticular formation (PPRF). The PPRF is located ventrally of the abducens nucleus and transmits an excitation signal to the ipsilateral abducens nucleus. The abducens nucleus is the recipient of this signal, which in turn stimulates the lateral rectus of the ipsilateral

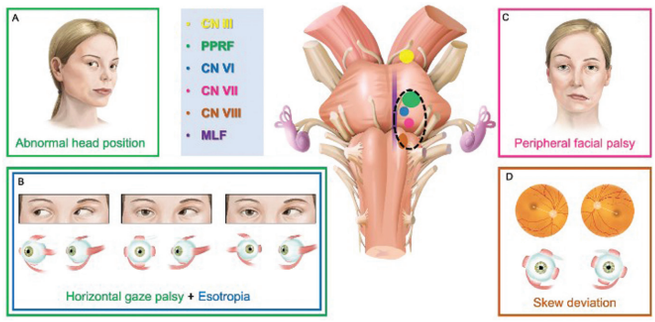

Figure 2 Schematic diagram of neural pathways involved in the present case based on the clinical findings Abnormal head position of left face turn (A) and left horizontal gaze palsy (B) indicated that left PPRF (green) is involved, and accompanying large esotropia in the left eye (B) indicated damage to the left abducens nerve (blue). Left peripheral facial palsy (C) was caused by damage to the left facial nerve (pink). Skew deviations of right intorsion and left extorsion (D) were caused by damage to left side prenuclear vestibular input to ocular motor nuclei (brown and purple). Dotted line (black) represented the area of the brain stem lesion in the present case. This schematic diagram was created with the assistance of the Photoshop software (Adobe, USA) and Cinema 4D software (Maxon Computer GmbH, Germany).

eye. Thereafter, the signal is projected upward through the contralateral medial longitudinal fasciculus (MLF), which is located dorsally to the PPRF, with the objective of transmitting signals to the oculomotor nucleus and medial rectus of the contralateral eye. This process facilitates conjugate eye movement by contracting both the lateral rectus of the ipsilateral eye and the medial rectus of the contralateral eye simultaneously. Disruption of the PPRF or abducens nucleus results in lesion-sided HGP.

The most common cause of HGP is pontine infarction, followed by pontine space-occupying lesions, such as brainstem glioma or pontine metastasis. PCH presenting as HGP is extremely rare reported[2], however, when located on the dorsal midline of the pons, it may involve the complicated neural pathways, including abducens nerve, facial nerve, vestibulo-cochlear nerve, PPRF, and MLF. Therefore, dorsal pontine lesions may cause a variety of complex neural deficits, including HGP, internuclear ophthalmoplegia (INO), one-and-a-half syndrome, abducens nerve palsy, facial nerve palsy, skew deviation, or any combination of these[2-5]. In the present case, a surgical removal of PCH induced multiple complicated disturbances due to relevant lesions involvement (Figure 2).

Among the complex clinical manifestations of this case, some symptoms or signs are easy to be missed if not carefully observed. Typical HGP is concomitant and rarely reported in association with esotropia[5-6]. Unilateral lesion in the PPRF may result in an ipsilateral conjugate HGP, and extremely large ipsilateral esotropia in primary gaze may indicated

Table 1 Clinical manifestations and possibly related lesions in this case

Anatomic locations involved	Corresponding manifestations
Left PPRF	Leftward horizontal gaze palsy
Left CN VI	Esotropia and abduction deficit in the left eye
Left CN VII	Left peripheral facial palsy
Left CN VIII (Posterior semicircular canal pathway)	Excyclo-rotation in the left eye and incyclo-rotation in the right eye (skew deviation); spontaneous counter-clockwise nystagmus

PPRF: paramedian pontine reticular formation; CN: Cranial nerve.

involvement of ipsilateral abducens nucleus in the present case. The abduction palsy accompanied by HGP is difficult to be confirmed, and the esotropia in the setting of a HGP could be easily overlooked. In the present case, Doll's eye movement is another evidence in favor of abducens nerve palsy. The term one-and-a-half syndrome, which was coined by Fisher[7] in 1967, is a clinical disorder results from lesions of the abducens nucleus or PPRF and the adjacent MLF. This produces a combination of an ipsilateral HGP and INO. Our case had a leftward HGP without an INO, and the absence of an INO indicates sparing of MLF fibers.

In addition, our case presented conjugate ocular counter-roll which indicates skew deviation. Skew deviation is a vertical misalignment of the eyes caused by damage to prenuclear vestibular input to ocular motor nuclei[8], that is part of an abnormal ocular righting reflex called the "ocular tilt reaction", which includes ocular torsion and head tilt[9]. The ocular tilt reaction indicates either a unilateral peripheral deficit of otolithic input, or a lesion in the graviceptive brainstem pathways crossing the midline at the pontine level, from the vestibular nucleus to the interstitial nucleus of Cajal in the rostral midbrain. Along this pathway, the impulse will be sent to the oculomotor nucleus, trochlear nucleus, and abducens nucleus to coordinate the extraocular muscle movement and eye position. Since these pathways are difficult to damage individually, the lesions in each part of the vestibular-ocular pathway may coexist with other nerve injury symptoms. Each posterior semicircular canal supplies excitatory input to the ipsilateral superior oblique muscle and the contralateral inferior rectus muscle. So, the lesion in the vestibular pathway which involving the left posterior semicircular canal in the present case caused dysfunction of left superior oblique and right inferior rectus, and further result in abnormal conjugate torsions in fundus. Skew deviation may be associated with a torsional jerk nystagmus, and the upper poles of the eyes tend to beat toward the side of a midbrain lesion[10].

Besides the complicated ocular movement disturbances, the present patient also had left facial palsy and motor disturbance of right limbs. Because of the close proximity of the seventh nerve fascicles to the sixth nerve nucleus and the PPRF, a single lesion may affect these and adjacent structures, leading to several variations (Table 1).

The AHP in patient with simple conjugate HGP is not compensating for diplopia or expanding binocular visual field, but rather that both eyes are unable to move towards ipsilateral gaze. In our case, the AHP of left face turn could be corrected by placing a base-in prism in front of the right eye, indicating that the AHP is due to adduction deficit of the right dominant eye with better visual acuity.

Pontine lesions have the potential to concurrently impact the vestibular nucleus, abducens nerve nucleus, and MLF, resulting in complex eye movement disorders, including skew deviation and abducens nerve palsy. Although such occurrences are rare, they are neuroanatomically plausible, particularly in cases of extensive pontine lesions. In clinical practice, a thorough examination of strabismus (including ocular motility, fundus torsion, nystagmus, head position, and binocular function) is essential for accurate diagnosis and treatment guidance. In this case, we have described a patient after removal of PCH presenting with complicated clinical manifestations that provided neuroanatomical localizing clues: HGP with esotropia (PPRF and abducens nerve), skew deviation with torsional jerk nystagmus (vestibular nerve and MLF), and peripheral facial palsy (facial nerve). AHP caused by HGP is not compensating for diplopia or expanding binocular visual field, but because both eyes are unable to move towards the side of the palsy. However, when HGP is combined with abducens nerve palsy or skew deviation, AHP develops to maintain binocularity, expand the binocular visual field, and dampens the nystagmus. In patients with HGP, accompanying ocular disorders such as large esotropia or objective torsion are essential to avoid missed diagnosis. If large esotropia is present despite the compensatory head position, consider accompanying abducens nerve palsy outside of HGP. Fundus photography is strongly recommended to evaluate the objective ocular torsion. Conjugated ocular rotations accompanied by torsional oscillations may be indicative of a skew deviation caused by vestibular pathway abnormalities. To our knowledge, this is the first reported case of HGP with abducens nerve palsy and skew deviation.

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Conflicts of Interest: Shen T, None; **Yu XY,** None; **Yan JH,** None.

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