Tiny magnetic resonance imaging black spot - Presenting an interesting case of internuclear ophthalmoplegia with radioanatomical correlation

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ABSTRACT

Horizontal gaze palsy involves three different entities of similar imaging findings with subtle differences and a prominent difference in the clinical findings. A thorough knowledge of the anatomy of the pathway of horizontal gaze pathways is essential before coming to the correct diagnosis. Here, we report the case of a 42-year-old male patient presented with a history of impaired vision for 1 day. Through this case report, we will revisit these three entities with a brief discussion on the pathways involved in horizontal gaze palsies.

Key words: *Horizontal gaze palsy, Internuclear ophthalmoplegia, Susceptibility-weighted imaging*

Internuclear ophthalmoplegia (INO) is a type of horizontal gaze palsy, the others being lateral gaze palsy and one-and-half syndrome [1]. Horizontal eye movements are a result of the synchronous activity of various nuclei and pathways in the brainstem. These include the abducent, oculomotor nuclei, medial longitudinal fasciculus (MLF), and paramedian pontine reticular formation (PPRF). The pons is the main site for the control and synthesis of these movements. Any insult in the pons can result in various types of horizontal gaze palsy [2,3]. Magnetic resonance imaging (MRI) helps to depict accurately the location and etiology of the lesion. This information combined with clinical history and examination can help us to come to the diagnosis regarding the type of palsy [4].

CASE REPORT

A 42-year-old male patient presented to the department with a history of impaired vision for 1 day. There was no associated comorbidity. No history of difficulty in walking, headache, loss of consciousness, or seizure was there. He was diagnosed as hypertensive 2 years back on irregular treatment. There were no similar episodes in the past.

On clinical examination, there was no neurological deficit. The general physical examination was normal. Higher mental functions were normal. The patient complained of blurring of vision; however, visual acuity was within normal limits. While examining conjugate gaze toward the right, the right lateral rectus was normal with the left medial rectus palsy. The abducting right eye had nystagmus. The adducting left eye was impaired. On examining conjugate gaze toward left, both the eyes moved normally. Convergence was normal. There was no vertical nystagmus noted. Rest of the neurological examination was within the normal limits.

MRI was performed (1.5 T MRI scanner Avanto, Siemens, Erlangen, Germany, with a head coil) which revealed T2-/fluid-attenuated inversion recovery hyperintense area in the dorsal pons on the left side in the paramedian position showing diffusion restriction and focal blooming on susceptibility-weighted imaging (SWI) and no enhancement (Fig. 1). Computed tomography was also performed which did not reveal any hyperdense focus in the pons to suggest hemorrhage or calcification. Therefore, a diagnosis of ischemic etiology with microhemorrhages involving the region of MLF on the left side of pons was implicated. The tiny focal blooming on SWI was the striking feature of imaging in this case.

DISCUSSION

Horizontal eye movements are controlled by the muscles lateral rectus and medial rectus, which are, in turn, controlled by the abducent and oculomotor nerves, respectively. These are, in turn, interconnected by the MLF. The MLF is a white matter tract, which interconnects oculomotor, the trochlear, the abducens, and the vestibular nuclei, and is located in the posterior part of the pons just anterior to the fourth ventricle [1]. The PPRF is involved in the conjugate horizontal eye movements. The PPRF which is located near the abducens nucleus in the pontine tegmentum receives impulses from the frontal and parietal eye field through the cortex and transmits impulses to the ipsilateral abducens nucleus [5]. All these structures work in tandem to control the conjugate horizontal gaze (Fig. 2) [1]. The line diagram in Fig. 2 [1] describes this in detail. Line diagram shows signal (solid straight arrow) from one of the PPRFs (green ovals) activating the ipsilateral abducens

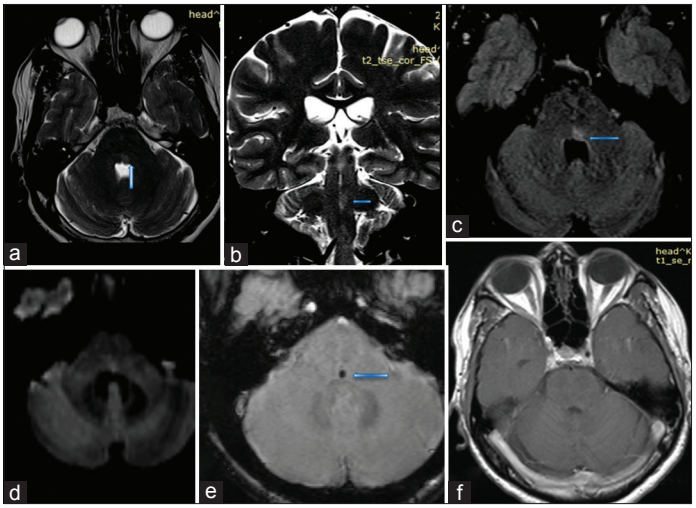

Figure 1: (a) Axial T2 image shows hyperintense area in the left dorsal pons. (b) Coronal T2 image shows hyperintense area in the left aspect of Pons (arrow) (c) Axial fluid-attenuated inversion recovery hyperintense area in the left dorsal pons. (d) Axial diffusion-weighted image shows diffusion restriction. (e) Axial susceptibility-weighted imaging shows focal area of blooming. (f) Axial T1 post-contrast shows no area of enhancement

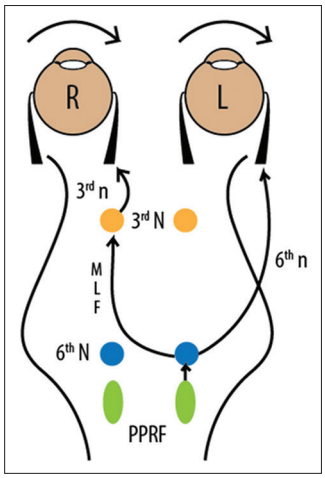

Figure 2: Brainstem pathways of horizontal eye movement. PPRFs (green ovals) activating the ipsilateral 6th n (blue circles) as a horizontal gaze center, ipsilateral 6th n, and contracts the ipsilateral lateral rectus muscle. The contralateral 3rd n (orange circles) is simultaneously activated causing contraction of the contralateral medial rectus

nucleus (6th n, blue circles) as a horizontal gaze center. The signal is transmitted to the ipsilateral abducens nerve (6th n, cranial nerve VI) and contracts the ipsilateral lateral rectus muscle. The contralateral oculomotor nucleus (3rd n, orange circles) is simultaneously activated through the MLF (curved arrow) from the abducens nucleus and transmits the signal to the contralateral oculomotor nerve (3rd n, cranial nerve III), which results in conjugate horizontal eye movement because of the contraction of the contralateral medial rectus muscle.

The three main disorders involving the brainstem, in turn, affecting the horizontal conjugate gaze palsy are lateral gaze palsy, INO, and one-and-a-half syndrome.

INO is caused by a lesion in the MLF [6]. The result of which is impairment of adduction of the ipsilateral eye and

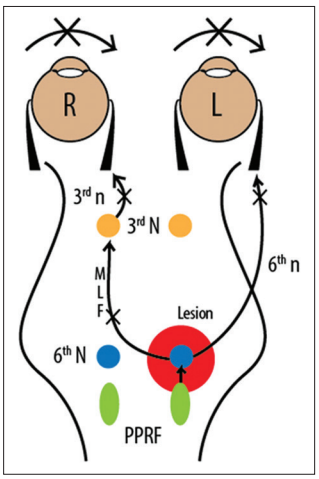

Figure 3: Diagram shows the mechanism for lateral gaze palsy: A pathologic lesion (large dark red circle) involves one of the paramedian pontine reticular formations (green ovals) or one of the 6th n=interruptions of signal transmission or eye movement that is a result of the lesion

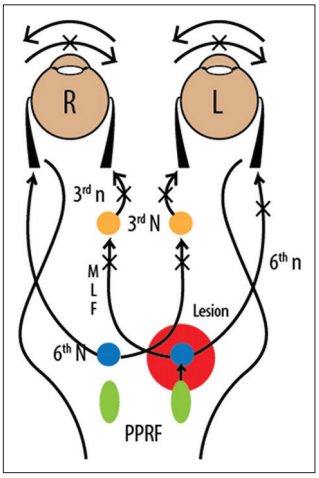

Figure 4: Diagram shows the mechanism for one-and-a-half syndrome: A pathologic lesion (dark red oval) in the dorsal pontine tegmentum involves both the ipsilateral paramedian pontine reticular formation (or the abducens nucleus) and the ipsilateral medial longitudinal fasciculus. ×=interruptions of signal transmission or eye movement that is a result of the lesion

the preservation of abduction of the contralateral eye when the patient tries to gaze in the contralateral direction. Even though the most common etiology is a pontine infarction, other causes such as demyelination, hemorrhage can be encountered [7]. Since the MLF is interrupted, the signal from the contralateral abducent nerve is not relayed to the ipsilateral oculomotor nerve, and hence, ipsilateral medial rectus palsy occurs when the patient looks in the contralateral direction [1].

Lateral gaze palsy occurs when there is a lesion similar in location to that encountered in INO, except that the size is much larger that it involves the MLF and also the ipsilateral abducens nerve (Fig. 3) [1]. Hence, when the patient tries to look in the ipsilateral direction, there is paresis of ipsilateral lateral rectus and contralateral medial rectus. These are also caused by pontine lesions which are bigger such as pontine infarctions, brainstem glioma, and pontine metastases [1,7].

One-and-a-half syndrome (Fig. 4) is a combination of the above two such that the patient has lateral gaze palsy in one direction and INO in the other direction with a contralateral gaze [1]. Therefore, abduction of the contralateral eye is the only preserved horizontal eye movement [8]. These occur when there is a lesion in the dorsal pontine tegmentum which affects the ipsilateral PPRF and/or abducent nucleus with the ipsilateral MLF [9]. The most common causes include infarctions and demyelinating disease including multiple sclerosis, hemorrhage, aneurysm or vascular malformation, neoplasm, and metastasis.

CONCLUSION

Familiarity with the brainstem pathways controlling conjugate horizontal eye movement helps to diagnose the small pontine lesion and the accurate localizing of the lesion, particularly when there is a very tiny infarct and when it is not visualized in initial studies and we may need to tailor the MRI sequences for its accurate visualization. Since the lesions involving these lesions are located in similar locations prior clinical history, and examination is of utmost importance in giving the diagnosis with confidence.

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