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TEK TARAFLI MEZENSEFALON İNFARKTINA BAĞLI VERTİKAL BAKIŞ PARALİZİSİ

VERTICAL GAZE PALSY DUE TO UNILATERAL MIDBRAIN INFARCTION

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ABSTRACT
We report a 52 years old male patient who developed acute onset diplopia and vertigo. Neurological examination revealed combined upward and donward gaze palsy and his MRI scan displayed a small unilateral lesion in the right midbrain. The lesion is presumed to have interrupted the pathways involved in vertical gaze palsy just before decussation producing a functionally bilateral lesion. In this case, we would like to question whether there is a right sided dominance in the pathways controlling the vertical gaze.
Key words: Vertical gaze palsy, nucleus of Cajal, midbrain

ÖZET
Bu olgu sunumunda ani başlangıçlı diplopi ve vertigo yakınmaları olan 52 yaşında bir erkek hasta bildirilmiştir. Nörolojik muayenesinde yukarı ve aşağı bakış paralizisi saptanan hastanın kranial MR görüntülemesinde, sağ mezensefalonda küçük, tek taraflı bir lezyon izlendi. Bu lezyonun, vertikal bakışı ilgilendiren yolakları, çaprazlaşmadan hemen önce engelleyerek fonksiyonel olarak bilateral klinik bulgulara yol açtığı düşünüldü ve vertikal bakışı düzenleyen yolaklarda sağ taraf baskınlığının olup olmadığı tartışıldı.
Anahtar kelimeler: Vertikal bakış paralizisi, Cajal nükleus, mezensefalon

INTRODUCTION

The mesencephalon is a premotor structure responsible for vertical and torsional eye movements. Two main anatomical structures are involved in the process of these eye movements: the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) and the interstitial nucleus of Cajal (INC). Both structures project directly to the oculomotor and trochlear nuclei. In general, the riMLF is considered to be the immediate premotor structure for vertical and torsional saccade generation (7) and INC the neural integrator for vertical and torsional eye movements (6,5).
Our report of a patient with bilateral vertical gaze palsy involving midbrain infarct limited to the riMLF and INC on the right side may provide further insight in the understanding of the control of vertical gaze.

CASE

A 52 years old man presented with acute onset diplopia, dizziness, vertigo and imbalance. The neurologic examination revealed bilateral vertical gaze palsy affecting both downward and upward directions (Figure 1,2,3,4). The horizontal eye movements and visual acuity were normal. There was no vergence or pupilar abnormality. Convergence-retraction nystagmus and eye lid abnormalities were absent. Caloric irrigation showed normal responses. The vertical oculocephalic maneuvre elicited a full upward and downward response. Otherwise there were no abnormal neurologic signs.

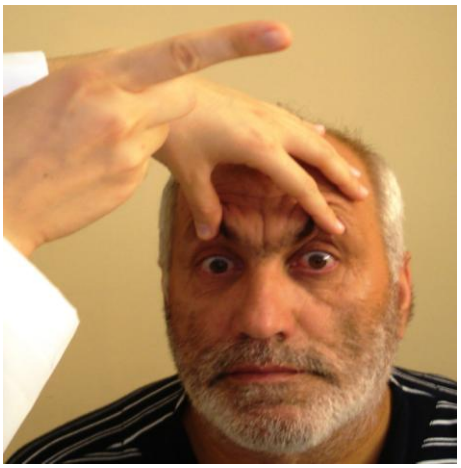

Figure1. Upward gaze of the patient

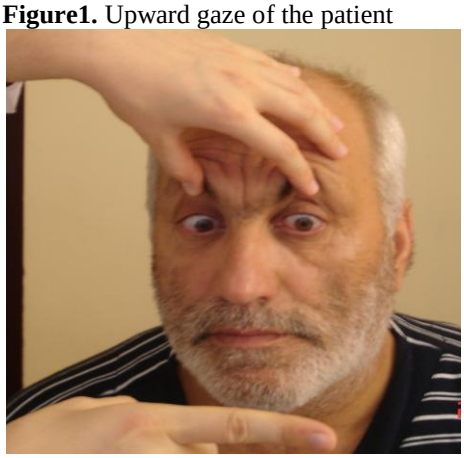

Figure 2. Downward gaze of the patient

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Figure 3. Gaze to the left

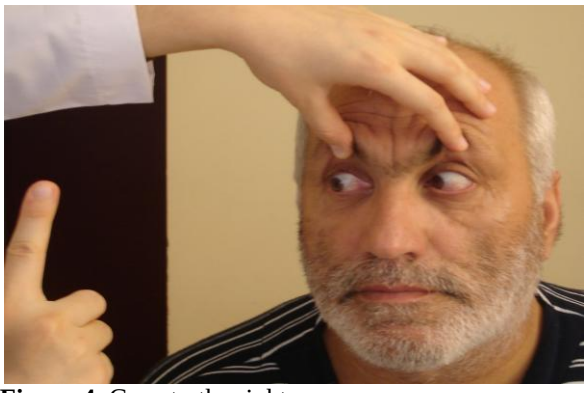

Figure 4. Gaze to the right

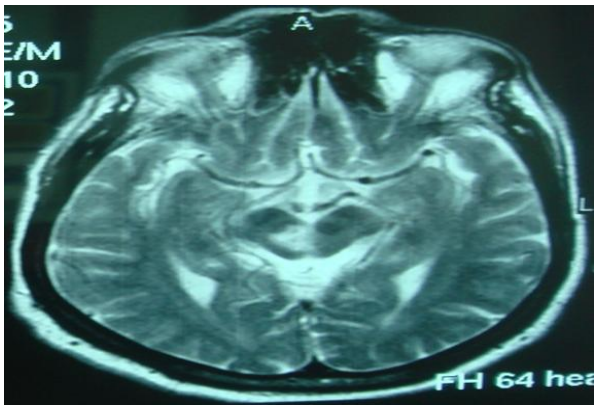

Figure 5. On MRI; T2 image shows unilateral lesion in the right midbrain

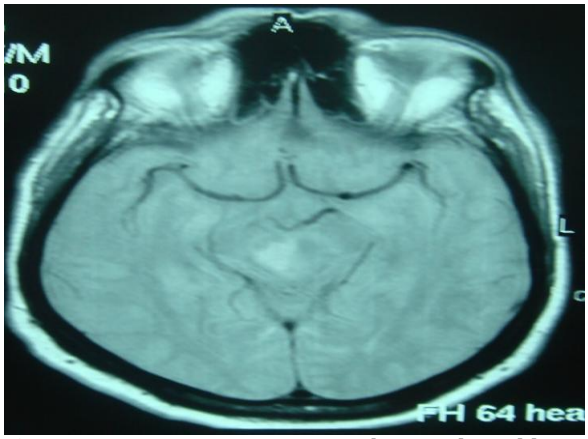

Figure 6. On MRI; proton image show unilateral lesion in the right midbrain

Brain MRI scan showed a small unilateral ischemic lesion in the right midbrain on T2 and proton images (Figure 5,6). The lesion clearly involved the right riMLF, the rostral pole of the interstitial nucleus of Cajal. It spared the posterior commisure and the oculomotor nucleus. Because of its sudden onset, the lesion was suspected to be an ischemic unilateral infarction of the paramedian thalamopeduncular deep penetrating midbrain vessels, despite the fact that vascular diagnostic work up (Doppler and duplex ultrasound, echocardiography) was normal.

The patient was treated with aspirin. The patient's vertical gaze palsy improved vaguely in two months after the onset of symptoms.

DISCUSSION

This patient had an ischemic lesion on the right side of upper mesencephalon causing bilateral vertical gaze palsy through riMLF and INC involvement. In general, bilateral lesions are required to produce clinically apparent deficits of vertical eye movements.

Patients with discrete, bilateral infarction involving the riMLF show deficits of either downward or both upward and downward gazes (4). Our report of a patient with bilateral vertical gaze palsy involving midbrain infarct limited to the riMLF and INC on the right side may provide further insight in the understanding of the control of vertical gaze.

Although unilateral midbrain lesion would not be expected to affect bilateral vertical gaze palsy, such cases have been reported.

Collier et al. described a patient with up and downward gaze palsy with a left paramedian thalamomesencephalic infarct (5). Unilateral experimental lesions of the riMLF cause a mild defect in vertical saccades, because each nucleus contains burst neurons for upward and downward movements; vertical saccadic deficits with unilateral lesions of the riMLF in humans are rare and probably reflect involvement of the commisural pathways (8).

Bougouslavsky et al. have suggested that selective unilateral (right) riMLF involvement may have disrupted bilateral upgaze excitatory and inhibitory inputs and unilateral downgaze excitatory inputs. In this case, they noticed that vertical oculocephalic response (VOR) was not affected therefore, they also suggested that riMLF did not interfere with this reflex (3).

The interstitial nucleus of Cajal is important for holding the eye in eccentric gaze after a vertical saccade and coordinating eye-head movements in roll.

Bilateral INC lesions limit the range of vertical gaze. Unilateral experimental leisons of INC are reported to impair gaze holding function in the vertical plane (7).

A patient whose MRI revealed unilateral right midbrain infarction involving riMLF, INC and spared posterior commisure has also been described . In this case it has been suggested that the lesion has interrupted the pathways involved in vertical gaze just before they decussate, inducing an anatomically unilateral but functionally bilateral lesion (1). This case resembles our case where paralysis of vertical saccades may be explained by complete loss of burst cells in the right riMLF and interruption of crossing fibers from the left riMLF as they traverse the right midbrain tegmentum.

Beside our case, there are only 3 other cases of unilateral

midbrain infarction involving riMLF and INC but sparing PC. Including our case, in all of these cases there was right sided brainstem involvement therefore we also thought that there may be a right sided dominance in these pathways.

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