OLGU SUNUMU **CASE REPORT**

TWO CASES OF VERTICAL GAZE PALSY: ONE WITH AND THE OTHER WITHOUT MIDBRAIN INVOLVEMENT

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ABSTRACT

Vertical gaze palsies are known to be resulted from lesions of the rostral interstitial medial longitudinal fasciculus, nucleus of Darkschewitsch, interstitial nucleus of Cajal and posterior commisure, all of which are located in the mesencephalic reticular formation. However, in some cases with acute vertical gaze palsy MRI revealed thalamic infarction without midbrain involvement indicating that thalamus also is involved in the control of vertical gaze. In this report two cases with vertical gaze palsy, one with and the other without associated midbrain involvement were presented and discussed with their clinical and MRI findings.

Key words: Vertical gaze palsy, thalamic infarction, MRI

BEYİN SAPI TUTULUŞU BULUNAN VE BULUNMAYAN İKİ VERTİKAL BAKIŞ PAREZİSİ OLGUSU

ÖZET

Rostral interstisyal medial longitüdinal fasikül, Darkschewitsch nükleusu, Cajal'ın interstisyal nükleusu ve posterior komisura mezansefalik retiküler formasyon içerisinde bulunan ve lezyonları vertikal bakış parezisine yol açan oluşumlardır. Oysa ki, vertikal bakış parezisi bulunan bazı olgularda MR ile mezansefalonda lezyon olmaksızın talamusta infarkt bulunduğunun gösterilmesi vertikal bakışın kontrolünde talamusun da rol aldığını düşündürmektedir. Burada, birisinde vertikal bakış parezisine mezansefalik bir infarktın eşlik edip diğerinde etmediği iki talamik infarktlı hasta sunulmuş, klinik ve MR bulguları ile birlikte tartışılmıştır.

Anahtar Sözcükler: Vertikal bakış parezisi, talamik infarkt, MR

INTRODUCTION

Isolated thalamic infarction is not frequently reported and usually observed in older patients. In recent years, studying these patients especially with neuroimaging has generated considerable interest with regard to their clinical characteristics and nuclear involvement. Ocular motor findings are reported to be amongst frequent and striking findings of paramedian thalamic infarctions including vertical gaze palsy (1).

CASE PRESENTATION

Case 1:

A 63 year-old right handed man with hypertension was admitted to the emergency department of our hospital for sudden loss of cosciousness followed by a single convulsion that lasted less than a minute. His blood pressure was recorded as 250/120 mmHg on his chart, so the convulsion was attributed to an accelerated hypertensive attack. His past medical history was remarkable for hyperlipidemia and an uncontrolled hypertension together with heavy cigarette smoking for at least five years. Two months ago he had had an episode of transient amnesia, blurred vision, unsteadiness and loss of consciousness for approximately half an hour. After having transferred to the neurology department he was conscious with normal speech and cognition. His neurologic examination disclosed upward and downward gaze palsy on voluntary and smooth pursuit movements, he had a mild truncal ataxia and a right upper hemifield visual loss that was attributed to an ischemic optic neuropathy. He had normal visual acuity and fundi. Pupils were normally reactive to light but convergence was abolished. Horizontal saccadic and pursuit eye movements were normal. Vertical movements of both eyes were restricted with approximately 10 degrees upward and 20 degrees downward. On oculocephalic maneuvers the amplitude of vertical gaze was normal. Routine serological tests were unremarkable except for a moderate hyperlipidemia. His EEG was normal. Cranial magnetic resonance imaging (MRI) obtained on the first day of symptoms showed acute infarctions located to bilateral cerebellar and left thalamic area (Fig 1a). There was no evidence of midbrain

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involvement (Fig 1b). MR angiography demonstrated patent vertebral and basilar arteries. He was discharged two weeks later with no improvement of his neurological findings. Eight months after the onset the patient was able to look downward but upward gaze was still restricted. His repeated MRI showed the presence of aforementioned lesions without any midbrain involvement.

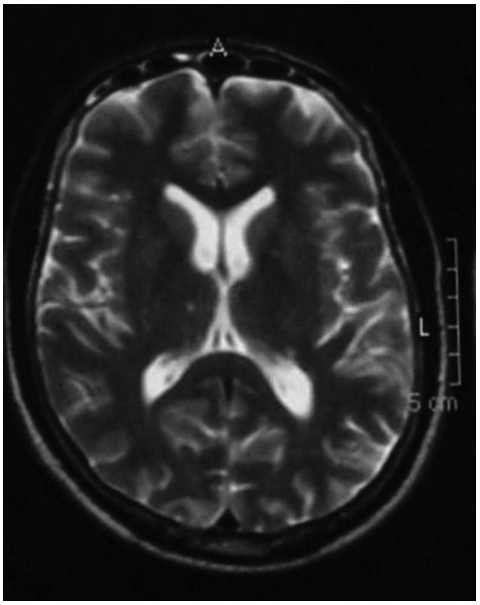

Fig 1a- Conventional MRI of the Case 1. Infarctions in both cerebellar hemispheres, in the right caudate nucleus and left thalamus

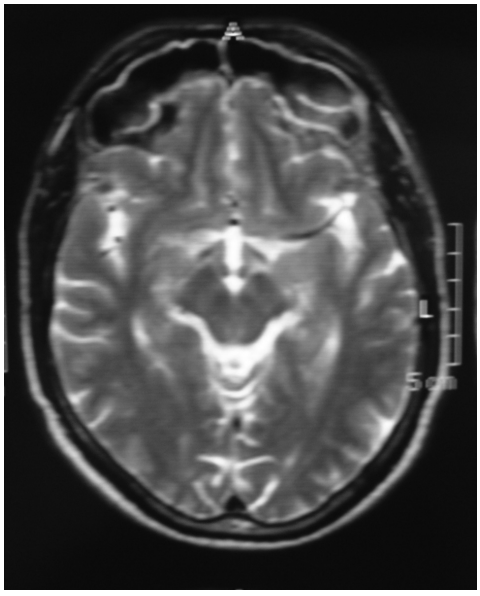

Fig 1b- No infarction was noted in the mesencephalon.

Case 2:

A 73 year old right handed woman presented with sudden onset of left sided hypesthesia and inability to look upward. Her past medical history was remarkable for diabetes and hypercholesterolemia. On examination she had a mild left hemiparesis with lower facial paresis and complete left sided sensory loss including her face.

Vertical saccadic and pursuit eye movements were restricted to 5 degrees upward and 10 degrees downward. On oculocephalic maneuvers, the amplitude of vertical gaze was normal. Optokinetic nystagmus test revealed upward and downward hypometric saccades and saccadic smooth pursuit eye movements. Pupils were isocoric and reactive to light but convergence was incompletely performed. Diffusion MRI obtained on the first day of symptoms revealed acute infarction located to the left medial thalamus and right mesencephalon (Fig 2a,b). MR angiography demonstrated loss of flow in the A1 segment of the anterior cerebral artery without evidence of vertebral or basilar artery stenosis. On the third day of admission her mild hemiparesis was resolved followed by the complete resolution of the left sided hemihipoesthesia at the and of the first week. When she was discharged at the end of the third week, her vertical gaze palsies continued. At the follow up control six months later, no improvement in her neuroophtalmological condition was observed.

DISCUSSION:

In these two patients, an acute conjugate vertical gaze palsy was the prominent clinical presentation. The first patient exhibited a unilateral lesion in the paramedian thalamic territory. No involvement in the midbrain was noted on MRI and other ischemic lesions (e.i. in the cerebellum) were not in the areas known to be involved in the control of vertical gaze. The patient carried important risk factors for small artery disease such as longstanding and uncontrolled hypertension, hypercholesterolemia and heavy cigarette smoking. In the second patient MRI revealed two separate lesions, one in the paramedian thalamus and the other in the midbrain. Small artery disease was presumed as the potential cause in these two patients because of the smaller than 15 mm infarctions and involvement of deep perforators on MRI.

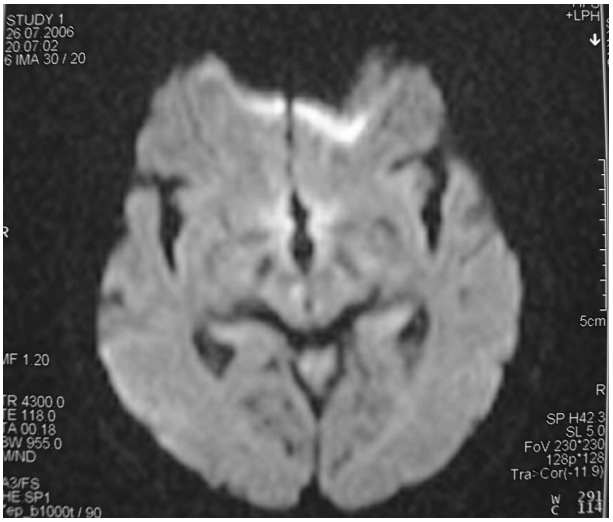

Fig 2a,b- Diffusion weighted MRI of the Case 2 showed diffusion restriction in the left medial thalamus (a) and in the right mesencephalon (b).

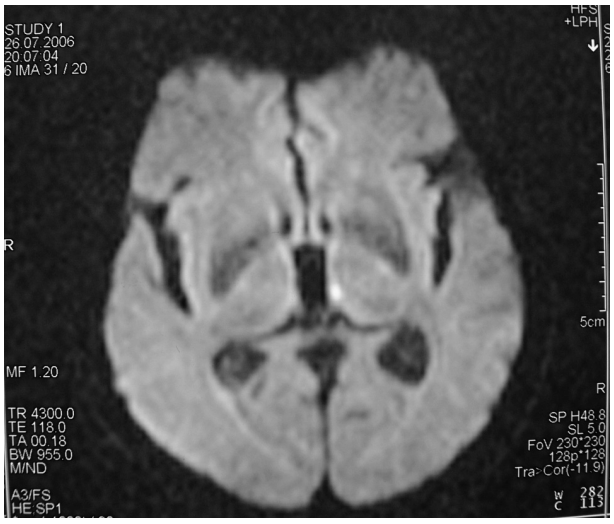

Fig 2a

The neural structures known to be involved in the control of vertical gaze are located in the mesencephalic reticular formation. These include the nucleus of Darkschewitch, the interstitial nucleus of Cajal, and the posterior commisure (2). At the midbrain level, a lesion may give rise to conjugate vertical gaze palsy, combined upward and downward gaze palsy, dyconjugate vertical disturbances and vertical one and a half syndrome. Hommel et al (1991) reported 11 cases of conjugate and dysconjugate vertical gaze palsies caused by a unilateral midbrain stroke (3).

Vertical gaze palsies may also result from paramedian thalamic infarctions (1,2). In contrast to the supranuclear pathways for horizontal gaze, those involved in the control of vertical gaze are not well understood. In this regard, cases with isolated paramedian thalamic infarct may be useful for understanding the pathways involved in supranuclear control of vertical gaze.

In five patients with vertical gaze palsy Gentilini et al. (1987) demonstrated isolated paramedian thalamic infarction on the CT of five patients with vertical gaze palsy. Three of the patients had no midbrain involvement while two of them had additional damage to the MLF (4). Tatemichi et al. (1992) prospectively examined 11 patients with MRI documented infarction in the paramedian thalamopeduncular region. There were 7 patients with vertical gaze palsy in their registry and 4 of these showed infarction in the posterior commisure. In 3 of these 4 patients an infarction in the thalamus was associated. In this study the vertical gaze palsy was attributed to the mesencephalic infarctions (5). Swanson and Schmidley (1985) reported the first case of vertical gaze paresis whose MRI showed bilateral thalamic infarcts. In this patient there was no apparent lesion in the midbrain although the 5 mm slices did not visualize the level of the red nucleus (6). In another MRI study Clark et al demonstrated thalamic infarctions without associated lesion in the midbrain in three patients with vertical gaze palsies. In two of these patients the thalamic lesion was unilateral, and the other had bilateral thalamic lesions. The authors also mentioned that lack of any lesion in the midbrain might have resulted from that the lesion was too small to be detected by the thickness of MRI slices (2). In a series of patients with thalamic infarction, Kumral et al (2000) reported 7 patients with upward gaze palsy. Three of these patients had associated downward gaze palsy and all of them had bilateral paramedian thalamic infarcts without midbrain involvement (1).

Vertical gaze palsy may be resulted from bilateral lesions of the riMLF (dorsomedial to the anterior pole of the red nucleus) or unilateral lesions of the posterior commisure (7,8). The vascular supply to these areas is derived from the same vessels as those supplying the medial thalamus, i.e.,the thalamic paramedian arteries (9). Related to their vascular supply, midbrain and thalamic infarctions frequently coexist. Paramedian thalamic and paramedian peduncular arteries may arise from a single vessel near the basilar artery bifurcation and this may result in infarction of

both midbrain and thalamic regions on the same side. When paramedian peduncular arteries arise separately from the paramedian thalamic vessels, isolated midbrain or isolated median thalamic infarcts can occur (2). In our second patient the coexisting mesencephalic lesion is contralateral to the thalamic lesion suggesting that one of these vessels were involved separately on either side.

In a case of unilateral thalamic infarction with vertical gaze palsy, high resolution MR revealed involvement of the rostral mesencephalon as well. The authors suggested that in previous patients with vertical gaze palsy attributed to a unilateral thalamic infarction a coexisting mesencephalic involvement may have been missed because of inappropriate imaging techniques (10). In our first case too small a lesion might have escaped from the thickness of MRI slices. However, it has been shown in the monkey that projections from the frontal and supplementary eye fields traverse the medial thalamus and reciprocal inputs from thalamus project to these areas (2). So it is possible that the unilateral thalamic lesion in our first patient might have produced the vertical gaze palsy by interrupting supranuclear inputs. This issue can reliably be confirmed only by further necropsy studies.

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