

Case report

Selective vertical saccadic palsy from unilateral medial thalamic infarction: clinical, neurophysiologic and MRI correlates

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Background – Impairment of vertical gaze has been attributed to lesions involving the neural structures at the mesodiencephalic level. **Objective** – Eye movements were studied in a patient with a unilateral paramedian thalamic infarction documented by MRI. **Case description** – A 63-year-old man presented 3 days after sudden onset vertical diplopia and hypersomnia. Eye movements were studied with electro-oculography and revealed impairment of vertical saccades with sparing of the vertical vestibulo-ocular reflex, vertical pursuit, Bell's phenomenon and vertical optokinetic nystagmus. MRI scan revealed a circular zone of altered signal intensity, suggesting infarction, in the paramedian ventral part of the right thalamus. **Conclusions** – This case demonstrates that a unilateral lesion mainly affecting the dorsomedial nucleus of the thalamus can result in selective impairment of vertical saccades and suggests that the corticofugal fibers mediating vertical saccades traverse in the medial thalamus en route to the rostral midbrain.

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Abnormalities of vertical gaze in addition to hypersomnia and amnesia are the cardinal features of paramedian thalamic infarction (1–4). In these reports, the vertical gaze palsy was, however, almost invariably due to coexisting lesions at the mesodiencephalic junction, in which the neural structures for vertical gaze (rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF), the interstitial nucleus of Cajal, the nucleus of Darkschewitsch, and the posterior commissure and its nuclei) are located (5, 6).

Sixteen cases of vertical gaze palsy from pure medial thalamic infarction have been reported (1, 7–15). However, none of these patients presented with selective impairment of vertical saccades due to unilateral paramedian infarction.

Eye movements were studied in a patient with a discrete unilateral infarction in the paramedian part of the right thalamus. Correlations of defective vertical saccades with magnetic resonance imaging

(MRI) findings indicate the role of the thalamus in the mediation of vertical saccades in humans. To our knowledge, this pattern of abnormal eye movements has not been reported in association with a unilateral infarction at this location.

Case report

A 63-year-old man presented 3 days after an acute onset vertical diplopia and hypersomnia. There was no history of cerebral infarctions or other neurological diseases and the patient had no risk factors for stroke. Neurological examination on admission showed an alert patient with normal speech ability and cognitive functions. In particular, there was no anterograde or retrograde amnesia, confabulations or perseverations.

Neuro-ophthalmologic examination revealed normal visual acuity, fields and fundi. The pupils were equal, and normally reactive to light and near

stimuli. There was no ptosis. There was no eye deviation in primary position. Vertical voluntary saccades were reduced above and below the midposition, with sparing of the vertical vestibulo-ocular reflex (VOR) and vertical pursuit (Fig. 1). Bell's phenomenon, vertical optokinetic nystagmus (OKN) and all types of horizontal eye movements were preserved.

The other cranial nerves were unaffected. There were no sensory and motor deficits, all tendon reflexes were normal and plantar responses were flexor.

Blood pressure was 160/80 mmHg. Cardio-pulmonary examination was normal. There were no cervical bruits. The remainder of the general examination was also normal.

All serum laboratory values were within the normal range. Digital subtraction angiography of carotid and vertebral arteries was normal. MRI scans were obtained 4 days after the onset of symptoms. MRI images (5.00 mm thick slices in the axial and coronal planes and 3.00 mm slices in the sagittal plane) revealed a circular zone of altered signal intensity, suggesting infarction, in the right paramedian ventral part of the thalamus (Fig. 2). There were no supratentorial, cerebellar or other brainstem lesions. The mesodiencephalic region was well visualized in all dimensions and appeared to be unaffected.

Two months after the onset of the vertical diplopia the patient recovered completely, except for slightly restricted downward saccades.

Method

Oculographic study

On the 4th day after the onset of the symptoms, eye movements were recorded, and assessed qualitatively, while the patient was cooperative and attentive. He was not taking any other medication than acetylsalicylic acid and dipyridamole. Direct current electro-oculography was used for each eye separately, in darkness, with the head immobilized (16). The following movements were studied: (a) horizontal and vertical visually guided saccades, performed by instructing the patient to look as fast as possible at a suddenly appearing 10° or 20° lateral or vertical target; (b) horizontal and vertical smooth pursuit induced by instructing the patient to follow a target moving sinusoidally with an amplitude of ± 20 at 25 or 40°·s⁻¹. The saccades and smooth pursuit were elicited in a dimmed room. Horizontal (or vertical) OKN was elicited with a whole-field stimulus consisting of vertical (or horizontal) black and white stripes moving horizontally (or vertically) at 10, 20 and 30°·s⁻¹ across a screen

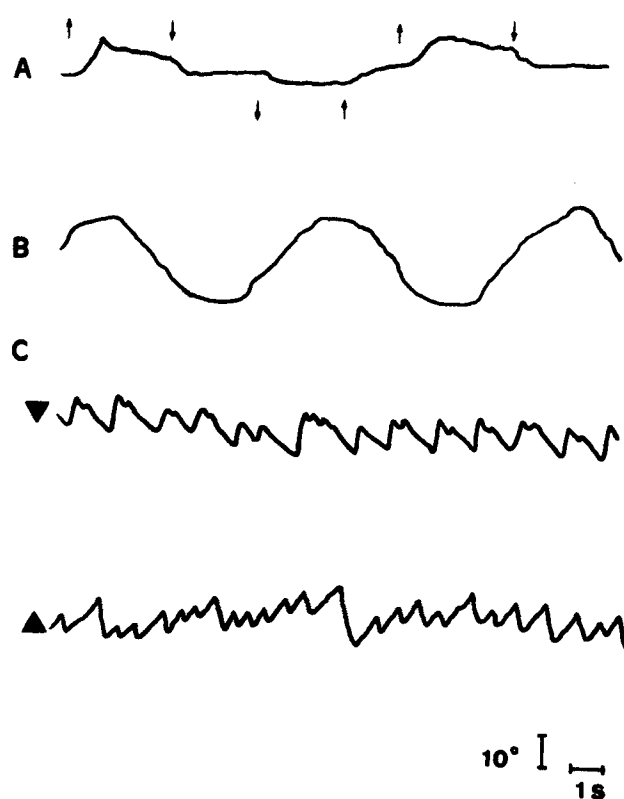
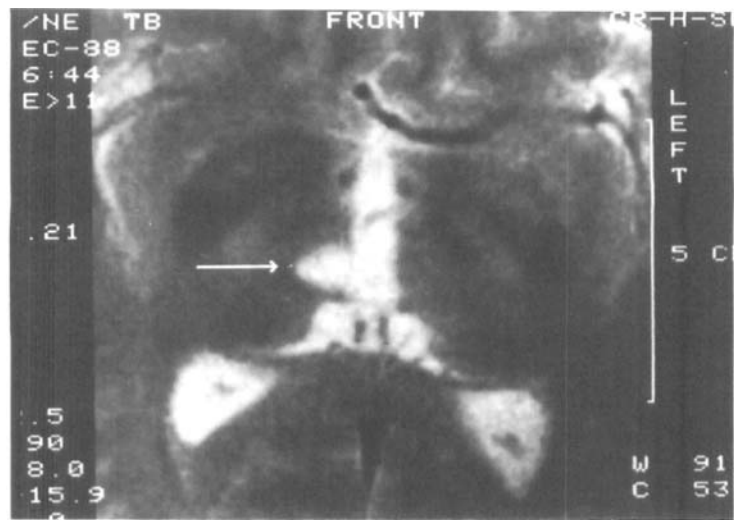


Fig. 1. Vertical eye movement recordings from the right eye. (A) Vertical visually guided saccades. The upward deflection indicates the upward movement of the target and the downward deflection the downward movement of the target. A 20° upward saccade is slow and hypometric. A backdrift towards the primary position was occasionally observed. The downward saccade back to the midposition is very slow. A 20° downward saccade was also very slow and hypometric. (B) Vertical smooth pursuit was normal. (C) Optokinetic nystagmus (▼: downward, ▲: upward) was normal in both directions.

covering 60° of the visual fields. Horizontal VOR were studied in complete darkness on a vestibular chair rotating at 0.40 Hz with an amplitude of 20° on each side of the midline. Vertical vestibular eye movements were induced by active vertical head shaking with the head erected (active vestibulo-ocular and otolith-ocular reflexes). Bell's phenomenon was tested by manually resisting the patient's attempts at forced eye closure.

Discussion

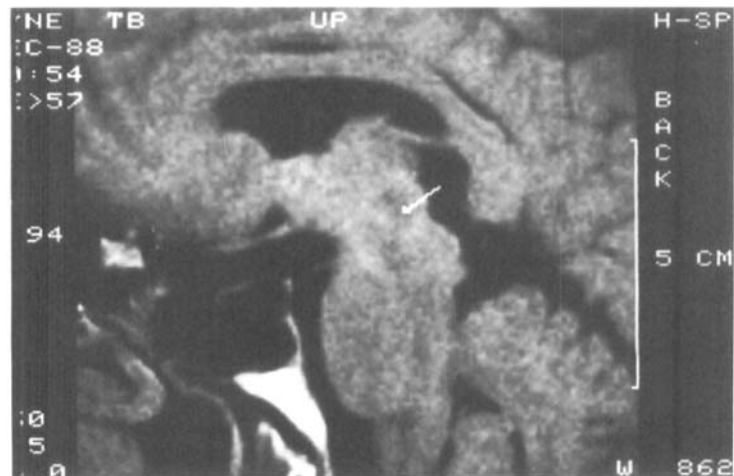
This patient presented with loss of vertical saccades with preserved vertical pursuit and VOR from a right paramedian thalamic infarction. There was no clinical evidence of midbrain involvement, particularly of the posterior commissure and/or riMLF, which would typically result in loss of both saccadic and pursuit vertical eye movements (17). Furthermore, MRI did not reveal any ischemic lesions at the mesodiencephalic level, midbrain or lower



(A)



(B)



(C)

Fig. 2. (A) Magnification of T₂-weighted axial brain MRI scan at the level of the pulvinar, dorsomedial, ventral posteromedial and ventral posterolateral nucleus of the thalamus. The lesion is compatible with an infarction (arrow) and affects mainly the right dorsomedial nucleus. (B) Magnification of T₁-weighted coronal brain MRI image showing the lesion (arrow) affecting mainly the right dorsomedial and centromedian nuclei without extension to the rostral midbrain. (C) Parasagittal T₁-weighted MRI image of the brainstem depicting the localization of the lesion (arrow) in the thalamus.

brainstem. Topographically, the infarct was localized with the use of corresponding anatomical sections as described by Tatemichi et al. (4) and by MRI neuroanatomy (18). According to these methods, a section through the pulvinar, dorsomedial, ventral posteromedial and ventral posterolateral nuclei of the thalamus corresponded to the level of the infarction, with the core of the ischemic lesion affecting the right dorsomedial nucleus of the thalamus.

Vertical gaze palsy has previously been reported in infarctions limited to the paramedian thalamus (1, 7–15). In only 6 of these patients vertical gaze palsy was characterized by selective impairment of vertical saccades (1, 9–13) and, unlike in our patient, the lesions were all bilateral. However, all of these cases except for 1 (12), were documented by CT scan, making precise anatomoclinical correlations difficult. The patient reported by Swanson & Schmidley (12) presented with selective vertical saccadic palsy and paramedian infarction on MRI. However, the lesion was also bilateral and the rostral midbrain was not visualized properly due to the limited quality of the images.

The cerebral cortical commands for saccades are delivered to the brainstem via two parallel corticofugal projections (19). An anterior pathway projects from the human homologue of the frontal eye fields directly, and indirectly to the riMLF (19). This pathway primarily generates intentional saccades. The posterior pathway is responsible for reflexive saccades and projects from the parietal cortex to the superior colliculus and subsequently to the riMLF (19). In our patient, impairment of vertical saccades was therefore attributed to the thalamic lesion *per se* by interruption of the corticofugal fibers as they traverse the medial thalamus *en passage* to the rostral midbrain structures subserving vertical saccades. The precise arrangements of these converging pathways in the thalamus remain, however, uncertain. In accordance with the prevailing concept, that supranuclear vertical gaze palsy spares the VOR, our patient had preserved VOR in all directions (20). These data also indicate that, at least at the thalamic level, the pathways for vertical smooth pursuit project separately from the pathways for vertical saccades. Moreover, it seems that unilateral disruption of corticofugal projections mediating vertical saccades can result in vertical saccadic palsy.

In addition to vertical gaze palsies, unilateral paramedian infarction can also cause hypersomnolence, amnesia and “frontal syndrome” (13). The anatomical structures involved in hypersomnia are the midline thalamic nuclei in particular the medial anterior part of the centromedian nucleus and inferior region of the dorsomedial nucleus, which form part of the rostral extension of the midbrain

reticular activating system (13). The incomplete expression of the syndrome in our patient might probably be related to the discreteness of the thalamic lesion.

The medial part of the thalamus is supplied by perforating branches that arise from the arterial segment between the bifurcation of the top of the basilar artery (basilar communicating artery) and the posterior communicating and posterior cerebral arteries (2). The paramedian thalamic arteries arise from the basilar communicating artery. Occasionally, both paramedian thalamic arteries arise from a single common stem originating from one or the other basilar communicating artery. This variation in vascular anatomy of the region probably accounts for the variable clinical presentation (2). In addition, sparing of the midbrain is probably explained by the fact that the superior and inferior paramedian mesencephalic arteries arise separately from each other from the basilar communicating arteries.

In conclusion, more sophisticated methods than the one used for investigation of this patient's vertical eye movements might have revealed additional subtle abnormalities. However, this would not substantially jeopardize our conclusion that a unilateral lesion mainly affecting the dorsomedial nucleus of the thalamus can result in selective impairment of vertical saccades.

References

1. GUBERMAN A, STUSS D. The syndrome of bilateral paramedian thalamic infarction. *Neurology* 1983; 33: 540–6.
2. MEISSNER I, SAPIR S, KOKMEN E, STEIN SD. The paramedian diencephalic syndrome: a dynamic phenomenon. *Stroke* 1987; 18: 380–5.
3. BOGOUSLAVSKY J, MIKLOSSY J, DERUZZA JP, REGLI F, ASSAL G. Unilateral left paramedian infarction of the thalamus and midbrain: a clinico-pathological study. *J Neurol Neurosurg Psychiatry* 1986; 49: 686–94.
4. TATEMACHI T, STEINKE W, DUNCAN C. et al. Paramedian thalamo-peduncular infarction: clinical syndromes and magnetic resonance imaging. *Ann Neurol* 1992; 32: 162–71.
5. PIERROT-DESEILLIGNY C, CHAIN F, GRAY F, SERDARU M, ESCOUROLLE R, LHERMITTE F. Parinaud's syndrome: electro-oculographic and anatomical analyses of six vascular cases with deductions about vertical gaze organization in the premotor structures. *Brain* 1982; 105: 667–96.
6. DELEU D, BUISSERET T, EBINGER G. Vertical one-and-a-half syndrome. Supranuclear downgaze paralysis with monocular elevation palsy. *Arch Neurol* 1989; 46: 1361–63.
7. SCHUSTER P. Beiträge zur Pathologie des Thalamus opticus. I. Mitteilung: Kasuistik. Gefäßgebiet des A. Thalamo-geniculata, der A. Thalamo-perforata, der Tubero-thalamica und der Lenticulo-optica. *Arch Psychiat Nervenkr* 1936; 105: 358–432.
8. CASTAIGNE P, BUGE A, CAMBIER J, ESCOUROLLE R, BRUNET P, DEGOS JD. Démence thalamique d'origine vasculaire par ramollissement bilatérale, limité au territoire du pédicule rétro-mamillaire. A propos de deux observations anatomocliniques. *Rev Neurol (Paris)* 1966; 114: 89–107.

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9. MILLS RP, SWANSON PD. Vertical oculomotor apraxia and memory loss. *Ann Neurol* 1978; 4: 149–53.
10. SCHOTT B, MAUGUIÈRE F, LAURENT B, SERCLERAT O, FISHER C. L'amnésie thalamique. *Rev Neurol (Paris)* 1980; 136: 117–30.
11. PETIT H, ROUSSEAU M, CLARISSE J, DELAFOSSE A. Troubles oculocéphalomoteur et infarctus thalamo-sous-thalamique bilatéral. *Rev Neurol (Paris)* 1981; 137: 709–22.
12. SWANSON R, SCHMIDLEY J. Amnesic syndrome and vertical gaze palsy: early detection of bilateral thalamic infarction by CT and NMR. *Stroke* 1985; 16: 823–7.
13. GENTILINI M, DE RENZI E, CRISI G. Bilateral paramedian thalamic artery infarcts: report of eight cases. *J Neurol Neurosurg Psychiatry* 1987; 50: 900–9.
14. BASSETTI C, MATHIS J, GUGGER M, LÖVBLAD KO, HESS CW. Hypersomnia following paramedian thalamic stroke: a report of 12 patients. *Ann Neurol* 1996; 27: 567–8.
15. CLARK JM, ALBERS GW. Vertical gaze palsies from medial thalamic infarctions without midbrain involvement. *Stroke* 1995; 26: 1467–70.
16. DELEU D, MICHOTTE A, EBINGER G. Impairment of smooth pursuit in pontine lesions: functional topography based on MRI and neuropathologic findings. *Acta Neurol Belg* 1997; 97: 28–35.
17. RANALLI PJ, SHARPE JA, FLETCHER WA. Palsy of upward and downward saccadic, pursuit, and vestibular movements with unilateral midbrain lesion: Pathophysiologic correlations. *Neurology* 1988; 38: 114–22.
18. JACKSON GD, DUNCAN JS. MRI neuroanatomy. A new angle on the brain. Edinburgh: Churchill Livingstone, 1996.
19. STANTON GB, GOLDBERG ME, BRUCE CJ. Frontal eye fields efferents in the Macaque monkey. I. Subcortical pathways and topography of striatal and thalamic fields. *J Comp Neurol* 1988; 271: 473–92.
20. BALOH RW, FURMAN JM, YEE RD. Dorsal midbrain syndrome: clinical and oculographic findings. *Neurology* 1985; 35: 54–60.