CASE REPORT

Midpontine Tegmentum Infarction with “One-and-a-Half Syndrome” Demonstrated by Magnetic Resonance Imaging

Kouichi Ohta, Fumio Gotoh, Yasuo Fukuuchi, Norio Tanahashi and Tamotsu Shinohara

Department of Neurology, School of Medicine, Keio University, Tokyo, Japan

(Received for publication on June 14, 1994)

Abstract. Magnetic resonance imaging (MRI) revealed a small midpontine tegmentum infarction in a patient with Fisher's one-and-a-half syndrome. The lesion was extremely restricted to the unilateral paramedian area of the midpontine tegmentum involving both the paramedian pontine reticular formation and medial longitudinal fasciculus. The typical form of this syndrome, that is, a combination of lateral gaze palsy and unilateral internuclear ophthalmoplegia, can be caused by a midpontine lesion. (Keio J Med 43 (3): 164–166, September 1994)

Key words: cerebral infarction, paramedian pontine reticular formation, medial longitudinal fasciculus, ophthalmoplegia

Introduction

Fisher's “one-and-a-half syndrome” is a well-defined neuro-ophthalmological disorder which comprises horizontal gaze palsy in one direction and internuclear ophthalmoplegia in the opposite direction.¹ It has been described in brainstem infarction, multiple sclerosis, pontine hemorrhage, pontine tumors, and other conditions.² The theoretical explanation of this syndrome has been previously based on neuropathological findings.^{3,4} In the present case report, we demonstrate the precise location of a responsible lesion visualized by magnetic resonance imaging (MRI) and discuss the neuroanatomical basis of this syndrome.

Case Report

A 67-year-old man with diabetes mellitus, hypertension and chronic renal failure under hemodialysis suffered sudden onset of diplopia and weakness of the right limbs. On admission to our hospital, he had horizontal gaze palsy to the left (Fig 1a). On attempted horizontal gaze to the right, paresis of adduction of the left eye was revealed, and the right abducting eye exhibited

monocular horizontal jerk nystagmus (Fig 1b). The oculoccephalic responses on head rotation were preserved. Convergence (Fig 1c) and vertical gaze were intact. Pupillary function was normal. Other cranial nerves failed to exhibit any abnormal findings. The patient had mild hemiparesis and cerebellar dysmetria in the right limbs.

A brain CT scan demonstrated lacunar infarcts in the left upper basis pontis and bilateral basal ganglia. MRI demonstrated a small lesion restricted to the left tegmentum of the midpons in addition to the lesions demonstrated by CT. The MRI lesion revealed a high intensity in the T₂-weighted image and a low to iso-intensity in the T₁-weighted image (Fig 2) and was diagnosed as an infarction. No lesion was detected in the lower pons.

The right hemiparesis and left horizontal gaze palsy subsided in the third week, although internuclear ophthalmoplegia remained.

Discussion

The present patient manifested typical one-and-a-half syndrome due to left midpontine tegmentum infarction.

太田晃一, 後藤文男, 福内靖男, 棚橋紀夫, 篠原 保

Reprint requests to: Dr Kouichi Ohta, Department of Neurology, School of Medicine, Keio University, 35 Shinanomachi, Shinjuku-ku, Tokyo 160, Japan

Fig 1 The patient's eye movements. a: Left lateral gaze. b: Right lateral gaze. The arrow indicates monocular horizontal jerk nystagmus. c: Convergence.

Fig 2 MRI axial images (1.5 Tesla, spin-echo sequence) demonstrating a small lesion restricted to the left medial tegmentum of the midpons. a: The lesion shows a high intensity in the T₂-weighted image (TR 2500 ms, TE 90 ms). b: A low to iso-intensity in the T₁-weighted image (TR 440 ms, TE 15 ms).

Right ataxic hemiparesis was attributable to the infarction of the left upper basis pontis which represents the classical focus of this type of lacunar stroke.⁵

Recently, a few MRI studies have confirmed brainstem infarction as the responsible lesion for one-and-a-half syndrome. Hommel *et al* reported two cases with the syndrome caused by brainstem infarction diagnosed by MRI. One case had a large lesion in the upper two-thirds of the ventral pons spreading to the paramedian tegmentum of the lower pons,⁶ and the other case had a lesion in the ventral midpons.⁷ A case reported by Dumas *et al* demonstrated a small MRI lesion in the paramedian tegmentum of the lower pons.⁸ As compared

to these past reports, the area of infarction on MRI in the present case was more restricted to the unilateral, paramedian, and dorsal area of the pontine tegmentum, and was localized at a more rostral level. This small portion includes both the paramedian pontine reticular formation (PPRF) and the medial longitudinal fasciculus (MLF).

Fisher emphasized a caudal location for the lesion in his original article on one-and-a-half syndrome so as to demonstrate caudal crossing of the MLF fibers at the level of the vestibular nucleus.¹ He also noted that the ocular response on head rotation varied from case to case. Although he ascribed this to the integrity of the vestibular-conjugate gaze center connexions, it is possible that the abducens nucleus was involved in some of his cases. Later authors have suggested that impairment of lateral conjugate eye movements in one direction in the one-and-a-half syndrome can be produced by damage to the abducens nucleus, or both the PPRF and the ipsilateral abducens nucleus, or the abducens nerve and the contralateral MLF, as well as isolated damage to the PPRF.^{3,4} Nevertheless, as Fisher originally postulated, the conjugate palsy occurring in the syndrome should constitute a supranuclear gaze palsy due to lesions of the "conjugate lateral gaze center". Thus, the PPRF is of primary importance as the pontine supranuclear lateral gaze center, and a PPRF lesion simply combined with an ipsilateral MLF lesion can produce the typical one-and-a-half syndrome from Fisher's original standpoint. Judging from the results of necropsy studies, such a lesion is more likely to appear in the midpons and upper pons.⁴ This reflects the anatomical fact that the PPRF in the lower pons is contiguous with the abducens nucleus and that its lesions hardly spare the abducens nucleus. As in the present case, a preserved oculocephalic response suggests that the causative lesion is rostral in the PPRF and that the abducens nucleus is spared. This is in a good accordance with the present MRI finding that the level of the responsible lesion was situated not in the lower pons but in the midpons.

References

1. Fisher CM: Some neuro-ophthalmological observations. *J Neurol Neurosurg Psychiatry* 1967, 30: 383–392
2. Wall M, Wray SH: The one-and-a-half syndrome — a unilateral disorder of the pontine tegmentum: a study of 20 cases and review of the literature. *Neurology* 1983, 33: 971–980.
3. Pierrot-Deseilligny C, Chain F, Serdaru M, Gray F, Lhermitte F: The 'one-and-a-half' syndrome: electro-oculographic analyses of five cases with deductions about the physiological mechanisms of lateral gaze. *Brain* 1981, 104: 665–699
4. Bogousslavsky J, Miklossy J, Regli F, Deruaz JP, Despland PA: One-and-a-half syndrome in ischaemic locked-in state: a clinicopathological study. *J Neurol Neurosurg Psychiatry* 1984, 47: 927–935

5. Fisher CM: Ataxic hemiparesis. A pathologic study. *Arch Neurol* 1978, 35: 126–128
6. Hommel M, Gato JM, Pollack P, Borgel F, Reeves AG, Crouzet G, Lebas JF, Perret J: Magnetic resonance imaging and the “one-and-a-half” syndrome: a case report. *J Clin Neuro Ophthalmol* 1987, 7: 161–164
7. Hommel M, Besson G, Tarel V, Gaio JM, Pollack P, Borgel F, Lebas JF, Perret J: MRI in horizontal oculomotor disorders due to brainstem infarcts. *Rev Neurol (Paris)* 1988, 144: 18–24 (in French)
8. Dumas G, Charachon R, Hommel M, Perret J, Lebas JF: Oculography and magnetic resonance imaging in brainstem infarction. *Ann Otolaryngol Chir Cervicofac* 1988, 105: 47–57 (in French)