CASE REPORT

A Case of Inferolateral Oculomotor Fascicular Infarction: A Review of the Clinicoradiological Literature

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Abstract

We report a patient with partial oculomotor paresis due to midbrain infarction. A 69-year-old man noticed diplopia suddenly. Ptosis, and impaired adduction and supraduction were found in the right eye. The pupillary size and light reflexes were normal on both sides. Magnetic resonance imaging disclosed an acute lesion in the right inferolateral oculomotor fascicle. These clinicoradiological findings suggested that the inferolateral fascicular damage could cause palsy of the levator palpebrae, medial rectus, superior rectus and inferior oblique muscles. Physicians should pay more attention to oculomotor fascicular infarction in patients with incomplete oculomotor paresis, and spared pupil sphincter and inferior rectus muscles.

Key words: partial oculomotor paresis, midbrain infarction, oculomotor fascicle, fascicular topography, MRI


Introduction

The oculomotor nerve innervates the levator palpebrae superioris, pupillary sphincter muscle and four extraocular muscles (the superior rectus, inferior rectus, medial rectus and inferior oblique muscles). The oculomotor fascicle connects the oculomotor nucleus and the subarachnoid oculomotor nerve. An anatomical lesion in this region can trigger a variety of abnormal ocular movements and other neurological signs (1, 2). Previous reports of oculomotor fascicular infarction have elucidated the human oculomotor fascicular arrangement innervating the intraocular and extraocular muscles (3-16). Here we report ophthalmologic and radiological findings in a distinct patient with inferolateral oculomotor fascicular infarction and review the clinicoradiological literature of partial fascicular oculomotor paresis in midbrain infarction.

Case Report

A 69-year-old man with prior history of hypertension and current smoking noticed double vision while watching television one evening. On waking up the next morning he was unable to open his right eye and was admitted to our hospital. Physical examination showed blood pressure of 154/90 mm Hg and body mass index of 24.8 kg/m². Visual acuity, visual fields, color perception and ocular fundi were normal in both eyes. Remarkable ptosis and strabismus was noted in the right side (Fig. 1). Upward and medial gaze were impaired in the right eye. Ocular movements were normal in the left eye (Fig. 2). The pupillary size was equal (3.0 mm) and light reflexes were normal on both sides. The neuroophthalmologic examination suggested palsy of the right levator palpebrae superioris, superior rectus, medial rectus and inferior oblique muscles. Other cranial nerves, muscle strength, muscle stretch reflexes, sensation and coordination were normal. The plantar responses were flexor. Routine laboratory studies were within normal ranges. Serological studies of the autoimmune and endocrine system were normal. Chest X-ray, electrocardiogram and carotid ultrasonography were not remarkable. At three days after the onset of diplopia, diffusion weighted imaging and apparent diffusion coefficient map disclosed an acute lesion in the right paramedian midbrain tegmentum (Fig. 3). Eight days later, a T2-hypersintense lesion was present in the inferolateral re-
region of the right oculomotor fascicle (Fig. 4). He was diagnosed with partial fascicular oculomotor paresis due to paramedian midbrain infarction. Treatment with edaravone (60 mg/day for 14 days, iv) and clopidogrel sulfate (75 mg/day, po) was performed immediately after admission. Incomplete oculomotor nerve paresis persisted for 2 months. Afterwards, he was transferred to a local hospital for rehabilitation.

**Discussion**

We described the clinicoradiological findings in a patient with inferolateral oculomotor fascicular infarction. Neuro-ophthalmologic examination showed impairment of the right levator palpebrae, superior rectus, medial rectus and inferior oblique muscles. The pupillary sphincter and inferior rectus muscles were preserved.

Pupil-sparing incomplete oculomotor nerve paresis occurs in several pathognomonic sites and variable disorders, including diabetes mellitus, cerebrovascular diseases, cerebral aneurysms, trauma, inflammatory disease, multiple sclerosis and tumors (1, 2). Recent development of radiological techniques using magnetic resonance imaging (MRI) could lead to the correct diagnosis of oculomotor fascicular infarction. In general, the frequency of midbrain infarction is rare in stroke patients. A previous clinicoradiological study mentioned 22 patients with isolated midbrain infarction (8). The frequency of those patients was 2.3% in 1,015 patients with the first stroke and 8.1% in 281 patients with posterior circulation infarction. The most common sites of midbrain in-
Partial oculomotor fascicular paresis (8). The annual rate of midbrain infarction among our inpatients with cerebral infarction is estimated at approximately 1-2%. The rate of oculomotor fascicular infarction may be less than 1%. Previous reports of partial fascicular oculomotor paresis due to midbrain infarction are listed in Table 1. All cases had pupil-sparing incomplete oculomotor nerve paresis except for one case reported by Murakami et al (7). The oculomotor fascicle is localized in the paramedian ventral midbrain. The fascicular fibers are divided topographically into four regions; the lateral, medial, superior and inferior subnuclear fibers (4, 16). The two-dimensional model of Castro et al (4) proposes that the oculomotor fascicle is somatotopically arranged laterally to medially as follows: the inferior oblique, superior rectus, medial rectus, levator palpebrae superioris, inferior rectus and pupillary fibers. Ksiazek et al (16) postulated a three-dimensional model of the oculomotor fascicles. The rostrocaudal construction indicated that the pupil sphincter fibers run in the most superior site followed by nerve fibers for the inferior rectus, inferior oblique, medial rectus, superior rectus and levator palpebrae superioris. Therefore, oculomotor fascicular fibers innervating the pupillary sphincter and inferior rectus muscles were arranged in the rostral and medial part, and passed through the red nucleus (16). The present patient exhibited the lesion in the caudal and lateral region of oculomotor fascicle on MRI, therefore the pupillary sphincter and inferior rectus muscles were not impaired. Pupil-sparing oculomotor nerve palsy is well known in patients with diabetes mellitus. Diabetic oculomotor mononeuropathy is a similar finding to oculomotor fascicular infarction. However, in addition to normal pupillary reactions, the inferior rectus was preserved in the present and previous patients with oculomotor fascicular infarction (3-5, 7-10, 12, 13, 15). Johkura (15) also emphasized that the neighboring topography of pupillary sphincter and inferior rectus muscles play a crucial key in the discrimination of extramedullary and fascicular oculomotor nerve palsy. Anatomical awareness of the fascicular arrangement is beneficial in lesion diagnosis in ophthalmoparetic patients.

In conclusion, we highlighted the neuro-ophthalmologic profile and MRI findings in a patient with partial fascicular oculomotor paresis due to midbrain infarction. The clinicoradiological hallmarks of the present patient confirmed the concept that inferolateral oculomotor fascicular damage could cause palsy of the levator palpebrae, medial rectus, superior rectus and inferior oblique muscles. Preservation of ocular infraction is a useful examination for the diagnostic differentiation from diabetic oculomotor mononeuropathy. Physicians should consider an oculomotor fascicular lesion in patients with pupil- and inferior rectus musclesparing oculomotor nerve paresis.

The authors state that they have no Conflict of Interest (COI).
Table 1. Main Previous Literature of Partial Fascicular Oculomotor Paresis in Pure Midbrain Infarction

<table>
<thead>
<tr>
<th>Authors (*)</th>
<th>reported years</th>
<th>Age/gender</th>
<th>Impaired ocular muscles</th>
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<tbody>
<tr>
<td>Nadeau et al (3) 1983</td>
<td>62 years/male</td>
<td>LP, MR, SR, IO</td>
<td></td>
</tr>
<tr>
<td>Castro et al (4) 1990</td>
<td>69 years/female</td>
<td>IO</td>
<td></td>
</tr>
<tr>
<td>Hriso et al (5) 1991</td>
<td>75 years/female</td>
<td>LP, SR, IO</td>
<td></td>
</tr>
<tr>
<td>Breen et al (6) 1991</td>
<td>23 years/female</td>
<td>IR, LP, MR, SR, IO</td>
<td></td>
</tr>
<tr>
<td>Murakami et al (7) 1994</td>
<td>62 years/male</td>
<td>PS, LP, MR</td>
<td></td>
</tr>
<tr>
<td>Bogousslavsky et al (8) 1994</td>
<td>67 years/male</td>
<td>MR, SR</td>
<td></td>
</tr>
<tr>
<td>Schwartz et al (9) 1995</td>
<td>34 years/female</td>
<td>LP, MR, SR, IO</td>
<td></td>
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<tr>
<td>Saeki et al (10) 2000</td>
<td>52 years/female</td>
<td>MR, SR</td>
<td></td>
</tr>
<tr>
<td>Champion et al (11) 2002</td>
<td>24 years/female</td>
<td>IR, LP, MR, SR, IO</td>
<td></td>
</tr>
<tr>
<td>Oshiro et al (12) 2003</td>
<td>74 years/male</td>
<td>LP, SR, IO</td>
<td></td>
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<tr>
<td>Rabadi et al (13) 2005</td>
<td>79 years/male</td>
<td>MR</td>
<td></td>
</tr>
<tr>
<td>Lee et al (14) 2006</td>
<td>56 years/female</td>
<td>IR</td>
<td></td>
</tr>
<tr>
<td>Johkura (15) 2009</td>
<td>79 years/male</td>
<td>LP, MR, SR</td>
<td></td>
</tr>
<tr>
<td>Present patient</td>
<td>69 years/male</td>
<td>LP, MR, SR, IO</td>
<td></td>
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</tbody>
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IO: inferior oblique; IR: inferior rectus; LP: levator palpebrae; MR: medial rectus; PS: pupil sphincter; SR: superior rectus.

*Reference number.

References


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