A Case of Traumatic Medial Longitudinal Fasciculus Syndrome Whose Causal Lesion Was Detected by Thin-Section MRI with Susceptibility-Weighted Imaging

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Keywords
Medial longitudinal fasciculus syndrome · Trauma · Magnetic resonance imaging · Susceptibility-weighted imaging

Abstract
The aim of the study was to report a case of traumatic medial longitudinal fasciculus (MLF) syndrome diagnosed with brain magnetic resonance imaging (MRI) after a head injury. A 71-year-old male complained of diplopia after he was bruised the back of his head when he was hit by a bicycle and fell down. He showed failure of adduction in the right eye, and mild nystagmus was found in the left eye when looking to the left. Convergence was intact. A low-intensity area was found at the middle right site in the lower part of the midbrain using thin-section MRI with susceptibility-weighted imaging (SWI), which suggested a hemorrhage. From the present history, characteristic abnormality of eye movement, and MRI imaging, he was diagnosed with traumatic MLF syndrome. His symptom was resolved, and the eye movement was improved in 2 weeks. A hemorrhage that occurs in the brainstem may be a cause of traumatic MLF syndrome which could be detected by thin-slice MRI with SWI.

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Introduction

Medial longitudinal fasciculus (MLF) syndrome is a manifestation of internuclear ophthalmoplegia (INO) which exhibits a characteristic eye movement abnormality due to an impairment of MLF located in the paramedian area of the midbrain and pons [1]. MLF syndrome is known to be caused by vascular infarction, inflammation, multiple sclerosis, or tumor, but traumatic MLF syndrome is relatively rare [2]. Here, we report a case of traumatic MLF syndrome which was able to be diagnosed with brain magnetic resonance imaging (MRI) after a head injury.

Case Report

A 71-year-old male complained of diplopia. He was hit by a bicycle and fell down, and then, he has bruised the back of his head in the early morning. He visited a neurosurgery hospital and underwent a head CT which revealed no hemorrhage or bone fracture. He felt diplopia when looking to the left immediately after the trauma; hence, he was referred to the Department of Ophthalmology, Osaka City University Hospital, the next day. He had systemic hypertension which was well controlled with oral medications. He had no particular family history, habits, or allergy.

His best-corrected visual acuity was 1.2 in the right eye and 1.0 in the left eye with the Landolt C chart. The intraocular pressure was 16 mm Hg in either eye. The anterior segment, optic media, and fundus findings were normal except for a mild cataract in both eyes. In the examination of eye movement, he showed a failure of adduction in the right eye, and horizontal jerk nystagmus was found in the left eye when looking to the left. Convergence was intact. The result of the 9-way eye position and HESS screen test is shown in Figure 1.

From the above, he was suspected of MLF syndrome. To determine the causal lesion, a thin-slice sagittal MRI of the midbrain and pons was performed. A low-intensity area was found at the middle right site in the lower part of the midbrain using susceptibility-weighted imaging (SWI) minimum intensity projection algorithm, while the lesion was not clearly detectable in diffusion-weighted imaging (DWI) (Fig. 2). According to the previous literature [3, 4], the lesion was considered a microhemorrhage. From the present history, results of the examination of eye movement, and MRI imaging, he was diagnosed with traumatic MLF syndrome.

Clinical Course

He was referred to the Department of Neurology on the same day for systemic examination, and no other neurological abnormality was found. Therefore, he was prescribed 1,500 mg/day of oral vitamin B12 and observed for 2 weeks. On the second visit, his symptom (diplopia) was resolved and his eye movement was improved (Fig. 3). A month after the head injury, his eye movement was fully recovered and oral medication was terminated.

Discussions

A phenotype of INO caused by an impairment of MLF is called MLF syndrome. MLF is a paired nerve bundle running in a craniocaudal direction near the midline within the tegmentum of the midbrain and dorsal pons [5]. MLF connects the oculomotor nucleus and abducens nucleus and plays an important role in horizontal eye movement [6, 7]. MLF
syndrome was first described by Bielschowsky in 1902, which may be caused by isolated lesions in MLF and characterized by the next trials: (1) adduction disturbance of the affected eye in conjugate deviation, (2) monocular nystagmus in abduction of the contralateral eye.

**Fig. 1.** 9-way eye position. 

- **a** A failure of adduction in the right eye is found when looking to the left. Convergence was intact.
- **b** Hess screen test revealed a failure of adduction in the right eye.

**Fig. 2.** Images of MRI. 

- **a** SWI and (b) DWI. A low-intensity area is found at the right MLF region (middle right site in the lower part of the midbrain) in SWI (red arrow), but the lesion is not clearly detected in DWI.
(dissociated nystagmus), (3) convergence is intact [1, 2]. Dissociated nystagmus is usually resolved earlier than adduction disturbance and becomes undetectable after the acute phase; hence, some cases were diagnosed as MLF syndrome only with monocular adduction disturbance [5]. In addition, the present case showed mild exotropia at the first presentation which could fit into Wall-Eyed Monocular Inter-Nuclear Ophthalmoplegia, a rare variant of INO [8, 9]. A previous report described that the most frequent cause of MLF syndrome was brain infarction (38%) and the next was multiple sclerosis (34%). Other causes were a tumor, side effects of anti-depressant, Wernicke’s encephalopathy, metabolic diseases like hepatic encephalopathy, and the rate of head injury was 3.9% [10].

The mechanism of traumatic MLF syndrome is considered to be a primary injury or a secondary injury of the brainstem. A primary injury may occur by a direct reach of shear force to the dorsal brainstem since the ventral brainstem is fixed with perforating branches of the basilar artery [11, 12]. A secondary injury may be caused by vascular damage due to neurovascular friction between perforating branches of the basilar artery and brainstem associated with traumatic force [11, 12]. The present case showed a lesion suspected of microhemorrhage in MRI image and early recovery with observation, which suggested the secondary injury of the brainstem.

In past, MLF syndrome was mostly diagnosed only with clinical symptoms, but recent advances in imaging techniques enable us to find causal lesions in many cases. However, in mild cases of MLF syndrome secondary to cerebrovascular disorder without other neural symptoms, it is often difficult to detect the causal lesions with MRI since the lesions are thought to be very small [5].

Thin-section (2.2 mm slicing, gapless) MRI was reported to detect causal lesions in 96.7% of acute cerebral infarction cases which was significantly better than conventional section (90.3% with 5–8 mm slicing, 1 mm gap) in DWI [13, 14]. Therefore, thin-section MRI might increase the accuracy of diagnosis of traumatic MLF syndrome if a small lesion is suspected.
Previous literature mentioned that most traumatic MLF syndrome was resolved with time from a few days to a few months [10–12, 15]. The present case showed a remarkable improvement of the symptoms over 2 weeks probably because the damage of trauma was relatively mild since the area of hemorrhage at MLF was small and no other neurological disorder or fracture was found. There is no consensus regarding the treatment for traumatic MLF syndrome. Dehydrating agents, barbiturates, and steroids may be used if it is accompanied by cerebral contusion, but mild cases are just observed with/without vitamin intake like our case [10–12, 15].

In conclusion, we have experienced a case of traumatic MLF syndrome without other neurological problems which were diagnosed with thin-slice MRI and recovered over 2 weeks of observation. Ophthalmologists should be aware of this clinical entity when being consulted for a case of head injury.

**Statement of Ethics**

This study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the subject for the publication of case reports and accompanying images. Ethical approval is not required for this study in accordance with local or national guidelines.

**Conflict of Interest Statement**

The authors have no conflicts of interest to declare.

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**Author Contributions**

Yosuke Ueno collected the data and drafted the manuscript. Shigeru Honda interpreted the data and critically reviewed the manuscript. Both authors read and approved the final manuscript.

**Data Availability Statement**

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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