Medial Longitudinal Fasciculus Syndrome Associated with a Subdural Hygroma and an Arachnoid Cyst in the Middle Cranial Fossa

Yoshiaki Minamori, Mayumi Yamamoto, Atsuko Tanaka, Kazuhiro Kanai, Hiroshi Uenishi*, Masao Tanaka*, Kouki Hayashi*, Kyouji Tsuji** and Masayuki Wakahara**

A 60-year-old man complaining of diplopia and vertigo showed bilateral medial longitudinal fasciculus (MLF) syndrome. The CT scan revealed a space-occupying lesion with watery fluid in the left cranial fossa, which was divided into two parts by a thin septum. Surgical trepanation was performed followed by 4 weeks of prednisolone therapy. He was completely cured 5 months later. The plausible causes of MLF syndrome relevant to preexisting space-occupying lesions are discussed.

Key words: computed tomography, cerebrocranial disproportion

Introduction

Medial longitudinal fasciculus (MLF) syndrome is often called internuclear ophthalmoplegia (1), a well known clinical syndrome characterized by impaired adduction ipsilaterally on lateral gaze, nystagmus in the abducting eye, but almost intact adduction on convergence. MLF syndrome is of diagnostic value in determining the site of the lesion. Multiple sclerosis, trauma, neoplasms, cerebrovascular disease, encephalitis, and diabetes mellitus have been reported as causes of this syndrome (2, 3).

An arachnoid cyst is defined by the cavities filled with fluid within arachnoid membrane (4) or between the arachnoid and pia mater (5). The advent of CT scan has increased frequency of diagnosis of the disease, most commonly seen in the middle cranial fossa (6, 7). Recently the reports of a combined state of a subdural hematoma (hygroma) and an arachnoid cyst in the middle cranial fossa have increased in number. The association of the two has been considered a clinical entity (8, 9) in that a subdural hematoma or hygroma may be caused by an arachnoid cyst. Here we describe a rare case in which MLF syndrome is associated with a subdural hygroma as well as an arachnoid cyst in the middle cranial fossa.

Case Report

A 60-year-old Japanese man was admitted to our hospital on February 24, 1989, complaining of a sudden onset of numbness of the lip and double vision on lateral gaze one hour prior to admission. Subjectively the patient complained of a floating and falling sensation, and nausea, but had no complaints of headache. Prior to presentation he had vomited a few times.

Past medical history included left ureteral lithiasis 3 times before 1983, and sigmoidectomy for colon cancer in March 1986. In August 1986, the patient tested positive for urinary sugar at a routine medical check-up. He showed a diabetic pattern with 75g oral glucose tolerance testing (fasting glucose was 100mg/dl and 212 mg/dl at 2 hour). He was treated with dietetic therapy.

On physical examination the patient was 160 cm tall, weighed 62 kg, and had an axillary temperature of 35.3°C. Blood pressure was 180/90 mmHg and radial pulse 68/min and regular. Cardiovascular and abdominal examinations were negative. He was well oriented and cooperative. His neck was supple. Pupils were equal and reactive, but on bilateral movement adduction was limited, less prominently on the left side. Jerky nystagmus was seen in both eyes; downward with upper gaze, horizontally with abduction. Convergence was preserved. Speech was eloquent. Cranial nerves other
than those mentioned above were normal. Tendon reflexes were normal and there was no laterality. No pathological reflexes were present. Mild dysdiadochokinesis was present on his left hand. The left index finger was slightly hypermetric in the finger-to-nose test. The patient showed wide-base and staggering gait. Romberg's sign was negative. Senses of touch and pain were intact. Muscle powers were preserved and no limb palsies were present. The urine was normal. The serum electrolytes and liver functions were normal. The CT scan, performed soon after admission, revealed a low density area of cerebrospinal fluid (CSF) in the left middle cranial fossa which was divided into two parts by a thin septum. The left temporal lobe was atrophic and its tip was absent, but midline shift was not seen. The left posterior horn of the lateral ventricle was shifted posteriorly and slightly caudally (Fig. 1). There was outward bulging and localized thinning of the left temporal bone.

Surgical trepanation into the left cranial fossa was performed on the day of admission to decompress the possibly elevated intracranial pressure. We aspirated 60ml of clear subdural fluid. Following aspiration a white membrane emerged. Following incision, clear fluid was drained from the interior. This fluid specimen contained no red blood cells but 5 white blood cells per cubic millimeter and the N/L ratio was 7/9; glucose level was 142mg/dl and total protein was 182mg/dl.

On the first postoperative day, the patient felt well without complaint of nausea and vertigo, however diplopia persisted. He also noticed that the sense of taste was disturbed, especially on the left side. Prednisolone, 40mg daily, was begun two days after operation. Two weeks postoperatively, his sense of taste had recovered and the disturbance of adduction of his left eye had begun to ameliorate. He was able to adduct his left eye and diplopia disappeared on frontal gaze 4 weeks after operation, and prednisolone was discontinued. Six weeks after admission, adduction in the left side had completely recovered, but MLF syndrome on the right side still remained (Fig. 2). At that point magnetic resonance imaging (MRI) was undertaken (Fig. 3) which showed

Fig. 1. Brain CT scan obtained after intravenous administration of contrast material, showing a subdural hygroma associated with an arachnoid cyst in the left cranial fossa, taken just after admission. They are separated by a thin septum. No lesion could be detected in the midbrain.
an arachnoid cyst in the left cranial fossa separated from a subdural hygroma by a thin septum. It also showed the left atrophic temporal lobe as previously demonstrated by CT scan preoperatively. Midbrain and pons appeared to be normal. The adduction of the right eye had recovered 5 months after operation and was accompanied by disappearance of the double vision. His arachnoid cyst and subdural hygroma did not change in size on repeat CT scan 3 and 12 months later.

Neither oligoclonal Ig-G band nor myelin basic protein in the CSF was found on the repeated examination. Barium enema and echography showed no evidence of recurrence of colorectal cancer. Insulin therapy was required during the administration of prednisolone. He was discharged on December 5, 1989. He has been well and there was no sign of recurrence of MLF syndrome.

Discussion

Disturbance of taste and hypesthesia of the lip of this

Fig. 2. At 6 weeks after surgical trepanation, disturbed adduction of the right eye (upper photo), full conjugate gaze to the right (middle), and preserved convergency (lower) were characteristics of the right MLF syndrome. The left MLF syndrome had already been cured. We missed taking photos with both upward and downward gaze.

Fig. 3. Magnetic resonance imaging (MRI) of the brain stem of the patient. Horizontal sections of T2-weighted image (spin echo method; recovery time 2,300 ms, echo time 90 ms, upper photos) and sagittal (lower right) and coronal (lower left) sections with T1-weighted image (recovery time 600 ms, echo time 22 ms), taken 6 weeks after operation, did not demonstrate lesions responsible for MLF syndrome of this patient. A subdural hygroma associated with an arachnoid cyst is still present in the left middle cranial fossa.
patient in addition to typical bilateral MLF syndrome suggested that pontine MLF and a portion of or the area adjacent to the facial nerve nucleus might have been involved. Neither CT scan nor MRI could demonstrate the brain stem lesion responsible for MLF syndrome in this case. The lesion might have been detected if repeat MRI examinations with thinner slices were performed. It is unlikely that the lesion could have been absorbed when the patient underwent MRI 6 weeks after the onset of the disease, as infarcted lesions in the brain stem have been reported to persist for at least several years by MRI (10).

The arachnoid cyst in the middle cranial fossa with atrophy of the temporal lobe and bulging and thinning of the localized temporal bone of this patient are thought to have been present in his childhood (5). Recently, reports of combined arachnoid cyst and subdural hematoma or hygroma in the middle cranial fossa have gradually increased in number, and this combination has been emphasized as a clinical entity (8, 9, 11). Thirty-three patients with arachnoid cysts in the middle cranial fossa associated with subdural hematomas/hygromas have been reported in the Japanese literature up to 1989. There has been only one reported case in Japan of a subdural hygroma associated with a middle cranial fossa arachnoid cyst (12), although several are reported in the Western literature (8).

Multiple sclerosis in young adults and brain infarction in older adults are major causes of both bilateral and unilateral MLF syndrome (2, 3, 13). Brain stem neoplasm, head injury, diabetes mellitus and encephalitis have been reported as causes of the syndrome, but rarely are these bilateral (2, 3, 13).

It seems plausible that arteriosclerosis of the relevant vessels supplying the brain stem lesion might have been a cause of MLF syndrome in this patient because he was a diabetic and 60 years old. All twelve diabetic patients with associated MLF syndrome reported in Japan before 1988 (14) were of long-standing illness with severe diabetic complications. This patient was a mild diabetic without any complications. It is unclear whether diabetes was responsible for the MLF syndrome in this patient.

Patients with MLF syndrome associated with an arachnoid cyst have never been reported, although supratentorial arachnoid cyst sometimes involved the brain stem by direct compression (15) or without referable reason (16–18).

There have been 4 cases of MLF syndrome associated with a subdural hematoma, to our knowledge; Cogan (2) reported one case with bilateral MLF syndrome and left hemiplegia; Devereaux et al (19) reported 2 cases and Tanaka et al (20) one case. The latter two authors mentioned ischemic accident following transtentorial herniation by a subdural hematoma as the presumed cause of MLF syndrome in view of both clinical manifestations and angiography. However, transtentorial herniation did not appear to be the case in this patient in view of both clinical findings and the result of brain CT scan taken just after onset. It might be possible to assume that the transient rise of the intracranial pressure by newly generated hygroma might have resulted in the distortion of the brain stem, followed by MLF syndrome. The arachnoid cyst in the middle cranial fossa, however, might have compensated for the volume expansion of the hygroma. In fact, patients with a subdural hematoma associated with an arachnoid cyst often have a less evident midline shift by CT scan as compared with the severity of the symptoms and signs (18, 21).

Kusuno et al (12) infers that cranioencephalic disproportion with an arachnoid cyst or with brain atrophy are conditions in which minor head injury tends to tear arachnoid membrane, and chronic subdural hematoma ensues. It is questionable whether this could apply to the brain stem, or not.

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References
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