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Case Report

다발성 경화증에서 교대성편마비의 해부학적 소견

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정혜영 · 정은주 · 김응규 · 배종석

Anatomical Findings of Hemiplegia Cruciata in Multiple Sclerosis

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Hemiplegia cruciata (HC) manifests as paralysis of the ipsilateral arm and contralateral leg. Herein, we report a 64-year-old man with weakness of the right leg and of the left arm after multiple sclerosis (MS). His brain and spine magnetic resonance imaging show a lower medulla lesion, which is extended to posterior part of C1 spine through cervicomedullary junction. HC usually results from stroke or trauma, but it is rare as presenting symptom of MS. (Korean J Clin Neurophysiol 2014;16:39-41)

Key Words: Crossed hemiplegia, Multiple sclerosis, Pyramidal decussation

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The syndrome of hemiplegia cruciata is defined as paralysis of an ipsilateral arm and the contralateral leg.[1] The neuroanatomical explanation for this syndrome involves the complex somatotopic and anatomical segregation of the corticospinal tracts in the decussation at the lower medulla oblongata or cervicomedullary junction.[1] At this level, the ventromedially located arm fibers decussate rostral to the leg fibers, and a lesion at this specific point during the course through the brainstem and the spinal cord could cause hemiplegia cruciata regardless of the etiology.[2,3]

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In this paper, we present a patient with multiple sclerosis (MS) who developed hemiplegia cruciata caused by junctional lesions between the cervical cord and the lower medulla. A symptomatic correlation with MRI findings is also discussed.

Case Report

A 64-year-old man visited the emergency department due to weakness in his right leg. A week before admission, he had suddenly developed weakness and a tingling sensation in his left arm. He had previously been diagnosed as optic neuritis one year before, having presented with a right visual disturbance. He had been treated with steroid and the symptom had been relieved. At this time of presentation, he did not complain of visual disturbances. He also had been treated for hypertension and gout, and family history was unremarkable.

On neurological examination, he showed a motor weak-

ness in the left arm and right leg. His motor power according to the Medical Research Council muscle power grading system could be roughly graded as 4 in affected extremities, which was slightly worse on the distal part of the left arm and the proximal part of the right leg. Although deep tendon reflexes were hyperactive in all extremities, Babinski sign was not evoked. Other neurological findings including sensory function was normal

Cerebrospinal fluid findings revealed a normal range of leukocytes and glucose, a slightly increased protein level, and a negative oligoclonal IgG band. Serology was non-reactive to VDRL (venereal disease research laboratory), ANA (anti-nuclear antibody), ANCA (anti-neutrophil cytoplasmic antibody), lupus anticoagulant, antiphospholipid antibodies (IgG and IgM) and dsDNA antibodies. Anti-Ro and anti-La antibodies, aquaporin-4 antibody, and neuromyelitis optica (NMO) IgG antibody were all negative. The IgG index was 0.0036.

Brain MRI showed a high signal intensity (HSI) lesion in the lower medulla on T2 weighted image, which showed a gadolinium enhancement on T1 weighted image (Fig. 1A and B).

Cervical spine MRI also revealed a HSI lesion with gadolinium enhancement at the cervicomedullary junction, extending to posterior part of upper cervical cord at the level of 1st cervical vertebra (Fig. 1C-E). Visual evoked potentials revealed bilaterally prolonged P100 latencies. After treatment with a course of high dose intravenous methylprednisolone for 5 days, his symptoms slowly improved. In addition, 250 μg of interferon-1b (Betaferon®) was subcutaneously administered every other day in order to prevent further attacks of MS.

The patient has given his informed consent, and this statement of his case has been approved by the institute's committee on human research.

DISCUSSION

Although hemiplegia cruciata was first identified in the early 20th century,[1] its clinical importance had been gradually neglected. Anatomically, the corticospinal decussation extends longitudinally from the cervicomedullary junction to the C-2 level.[2] More specifically, the motor tract of the

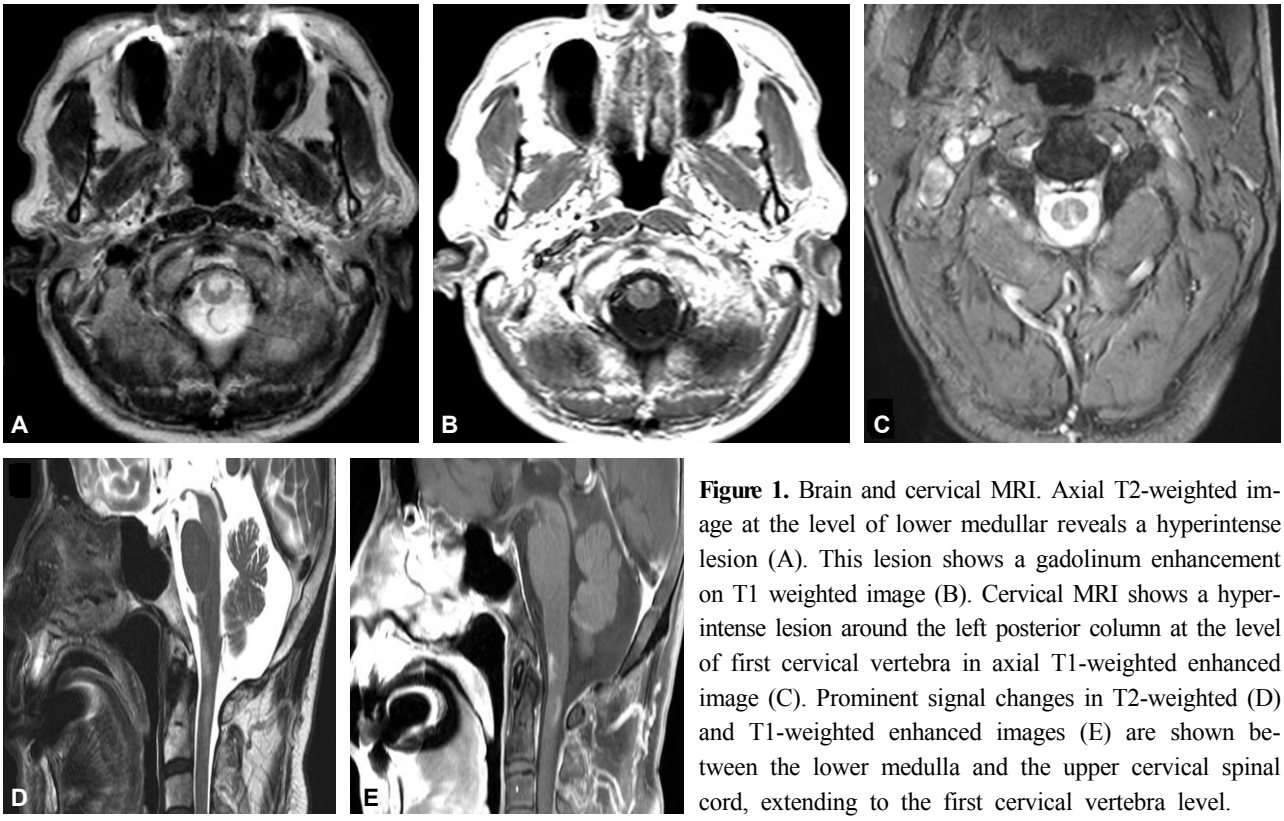

Figure 1. Brain and cervical MRI. Axial T2-weighted image at the level of lower medullar reveals a hyperintense lesion (A). This lesion shows a gadolinum enhancement on T1 weighted image (B). Cervical MRI shows a hyperintense lesion around the left posterior column at the level of first cervical vertebra in axial T1-weighted enhanced image (C). Prominent signal changes in T2-weighted (D) and T1-weighted enhanced images (E) are shown between the lower medulla and the upper cervical spinal cord, extending to the first cervical vertebra level.

upper extremities crosses rostrally in the cervicomedullary junction, whereas tract of lower extremities crosses caudally between the C-1 and C-2 region.[2,4] Therefore, the topographic anatomy of the regions could account for various neurologic presentations, depending on the intricate details of neural pathways affected by the lesion on the descending corticospinal tracts. The hemiplegia cruciata can be commonly caused by lesions at the lateral aspect of the pyramidal decussation, which longitudinally extends from the rostral to the caudal of the pyramidal fibers between the lower medullocervical junction and C1.

Hemiplegia cruciata can be caused by uncommon neurological complications, such as traction or direct compression, tumor, ischemia or stroke, aberrant changes in cerebrospinal fluid dynamics, and complications of surgery.[5-7]

Because this patient had two separate clinical attacks, including a reasonable historical evidence of a previous attack of optic neuritis, and an objective MRI evidence of the lesions, a diagnosis of MS could be made according to the McDonald criteria.[8] The brain and spinal MRIs of this patient showed lesions at the cervicomedullary junction and upper cervical cord at the level of 1st cervical vertebra, which is more predominant toward the left side. In sagittal images on the cervical MRI, the lesion at the cervicomedullary junction is located anterior relative to the lesion at the upper cervical cord. Therefore, the left sided lesion at cervicomedullary junction would have affected crossed pyramidal tract to left arm and also uncrossed pyramidal tract to right leg, producing a hemiplegia cruciata observed in this patient. In addition, the left posterior cord lesion at the level of 1st cervical vertebra is most likely related to a tingling sensation in the left arm.

Although mechanical injuries or complications secondary to the surgeries at the cervicomedullary junction is frequently implicated as a cause of hemiplegia cruciata, non-mechanical etiologies such as metabolic disorder might be responsible as well.[9,10]

Because a correct interpretation of symptoms and signs is essential for the diagnosis of neurological disorders, physicians should perform comprehensive neurological examinations with an exact knowledge about the neuroanatomy.

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