

Case Report

Paraplegia caused by cerebral contusions in the bilateral precentral gyri

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Abstract

Background: Paraplegia is mainly caused by spinal cord disease and rarely occurs due to head trauma. In this report, we describe a case of paraplegia caused by cerebral contusions in the bilateral precentral gyri.

Case Description: A 72-year-old man was admitted to our hospital with mildly impaired consciousness and severe pure motor paralysis in both legs. He was healthy until the morning of the day, but his wife found him injured in front of his house upon returning home. He had a subcutaneous hematoma in his occipital region, and seemed to have slipped by accident. Computed tomography of the brain and magnetic resonance imaging (MRI) of his spinal cord revealed no apparent cause of the paraplegia, although an MRI of his brain clearly revealed cerebral contusions in the bilateral precentral gyri. The cerebral contusion was diagnosed as the cause of pure motor paralysis of lower extremities. He received rehabilitation, and manual muscle testing of his legs revealed improvements. In the subacute phase, the precentral gyrus lesion disappeared on MRI.

Conclusion: We must emphasize that cerebral contusion can be a differential diagnosis for paraplegia. In the acute phase, fluid-attenuated inversion recovery (FLAIR) MRI coronal and sagittal images are useful for identifying precentral gyri contusions. Paraplegia caused by a cerebral contusion may be misdiagnosed as a spinal concussion due to the disappearance of the precentral gyrus lesion on FLAIR MRI in the subacute phase.

Key Words: Bilateral precentral gyri contusions, head injury, paraplegia, pure motor paralysis, spinal concussion

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INTRODUCTION

Paraplegia is most commonly caused by spinal cord conditions including traumatic injuries, degenerative diseases, tumors, and infections.^[10,13] Although uncommon, multiple sclerosis,^[12] meningioma,^[12] and epidural hematoma at the vertex^[6,8] can be among the causes of paraplegia. However, paraplegia is rarely caused by cerebral contusions.^[4] We experienced a case of acute

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pure motor paralysis of both legs caused by cerebral contusions in the bilateral precentral gyri.

CASE PRESENTATION

A 72-year-old Japanese male was transferred to our hospital with mildly impaired consciousness and gait disturbance. He was healthy until the morning of that day, but his wife found him injured in front of his house upon returning home. He had a subcutaneous hematoma in his occipital region, and seemed to have slipped by accident. In our initial evaluation, his Glasgow Coma Scale score was 13 (E3V4M6). He was unable to raise his legs or stand. A neurological examination revealed pure motor paralysis of the legs. There were no sensory disturbances. Manual muscle testing revealed scores of 5/5 for the upper limbs. However, these scores were 0/5 for the bilateral iliopsoas, tibialis anterior and tibialis posterior, 3/5 for both adductors and hamstrings, and 1/5 for both quadriceps femoris. Deep tendon jerks in both the lower and upper limbs were slightly diminished and without laterality. No significantly abnormal laboratory findings, including hypokalemia or an inflammation response, were noted in a general blood exam. Computed tomography (CT) of the brain revealed a subarachnoid hemorrhage, subdural hematoma, and contusions in his basal frontal lobes and superior frontoparietal lobes [Figure 1a and b]. Magnetic resonance imaging (MRI) of his spine revealed cervical spinal canal stenosis and lumbar spondylolisthesis; however, these lesions could not account for the pure motor paralysis of both legs [Figure 2]. Further brain MRI investigations were performed. Axial T2*-weighted MRI revealed contusions in both basal frontal lobes and bilateral parasagittal regions [Figure 1c]. Axial, coronal, and sagittal fluid-attenuated inversion recovery (FLAIR)

MRI images clearly demonstrated white matter lesions in the bilateral precentral gyri [Figures 3a and d]. He exhibited pure motor paralysis of both legs without sensory disturbances, such as paraesthesia, and the lesions of the bilateral precentral gyri accounted for this symptom. We concluded that the cerebral contusions caused the paraplegia because his pure motor paralysis occurred rapidly, and he had a subcutaneous hematoma in his occipital region. He was treated conservatively. The deep tendon reflexes of lower extremities increased during hospitalization. He received rehabilitation, but apathy and executive dysfunction slowed improvements in his gait. At 2 weeks after hospitalization, he was able to raise his knees. Coronal and axial FLAIR MRI image at that time revealed areas of high intensity in bilateral precentral gyri that were broader than those observed on the initial MRI [Figures 3b and e]. As rehabilitation continued, his consciousness improved modestly, and the paraplegia also gradually improved. At 7 weeks after hospitalization, manual muscle testing of his lower limbs revealed improvements to almost 4/5 in both legs. He received gait training and was able to walk approximately 20 m with a walker. At this time, the total size of the lesion on FLAIR MRI was significantly reduced [Figures 3c and f]. He was discharged to a subacute rehabilitation hospital 8 weeks after hospitalization.

DISCUSSION

We reported a case of paraplegia caused by cerebral contusions. Low-density areas were observed around the hemorrhage near the vertex in the initial CT that indicated a cerebral contusion. When a high-density lesion is located around the vertex, it may be difficult to distinguish between traumatic subarachnoid hemorrhage, subdural hematoma, and cerebral contusion on the first examination of the CT images [Figure 1a]. A subsequent MRI clearly revealed cerebral contusions in the bilateral

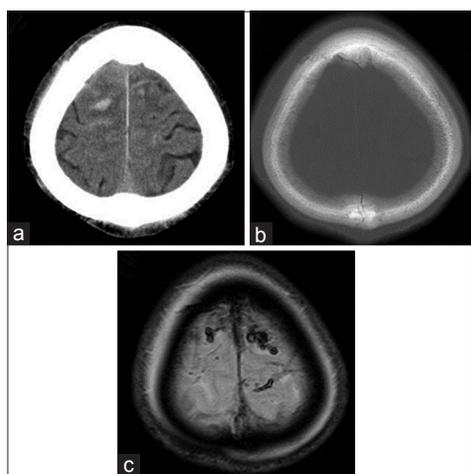


Figure 1: Axial computed tomography image near the vertex on admission showing a hemorrhage and a peripheral low-density area (a). Axial bone window computed tomography image near the vertex revealing no fracture (b). Axial T2*-weighted magnetic resonance image clearly revealing cerebral contusions in the bilateral parasagittal region near the vertex (c)



Figure 2: Sagittal T2-weighted magnetic resonance image of the cervical and lumbar spine

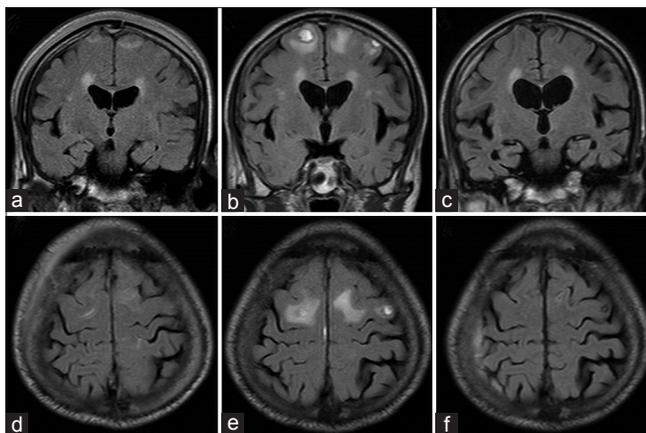


Figure 3: Coronal (a) and axial (d) fluid-attenuated inversion recovery magnetic resonance images acquired on admission showing white matter lesions in the bilateral precentral gyri. These lesions were exacerbated 2 weeks after admission (b, e) and subsequently decreased by 7 weeks (c, f)

parasagittal precentral gyri, which control voluntary movements of the lower extremities. He exhibited acute pure motor paralysis of both legs without sensory disturbances, and the lesions of the bilateral precentral gyri accounted for this symptom. Coronal MRI was particularly useful for identifying the lesion located around the vertex [Figures 3a-c]. Cervical canal stenosis was not accountable for the symptom of this patient because of the absence of sensory and motor dysfunction in his upper extremities. In addition, the deep tendon reflexes of lower extremities increased during hospitalization which indicated that paraplegia was not caused by the lumbar spondylolisthesis. The mechanism of precentral gyrus contusion is thought to be related to the shearing stress caused by rotational acceleration during trauma. Stretching in the sagittal plane along the anterior–posterior axis of the brain could produce a contusion in the superior frontoparietal cortex. Angular and rotational acceleration tend to generate more shear stress than linear acceleration.^[3,5] Superior frontoparietal cortex contusions are rare and account for up to 4% of cortical contusions.^[2,5]

In this patient, the lesion on FLAIR MRI first became exacerbated and then disappeared by 7 weeks [Figure 3]. According to a previous study of traumatic axonal injury,^[9] the total lesion volume on FLAIR MRI at 3 months was significantly reduced compared with that on early MRI, and the lesion volume on early MRI was related to the Glasgow Outcome Scale score at 12 months after injury. Because our patient had a relatively broad lesion on the FLAIR MRI, his gait disturbance was severe.

We know of only one previous case report describing paraplegia^[4] and six cases of monoplegia^[1] caused by cerebral contusion. The former case also exhibited severe paraplegia; however, the patient fully recovered after 2

weeks possibly because the FLAIR MRI lesion was smaller than the lesion in our patient. A previous report reviewed six cases of monoplegia caused by cerebral contusion; five of the cases recovered from the gait disturbances after 1 week to 6 months of rehabilitation.^[1] Based on these past reports of recovery, the patient received vigorous rehabilitation despite the patient's initial severe paraplegia and executive dysfunction, and his gait disturbance improved.

The reasons that most patients recover from paralysis caused by cerebral contusions is not fully understood. There are several international studies regarding the prediction of the outcomes of patients with traumatic brain injuries.^[7] In the acute stage, high-intensity areas on FLAIR MRI indicate diffuse axonal injury, edema, and hemorrhage. In the subacute and chronic stages, the high-intensity areas indicate gliosis and encephalomalacia.^[11] Judging from the fact that the FLAIR lesion of our patient had almost disappeared by 7 weeks after the injury, the axonal injury was not sufficiently severe to cause gliosis. We believe that his paraplegia gradually recovered as the influence of vasogenic edema decreased.

Paraplegia caused by a cerebral contusion may be misdiagnosed as a spinal concussion because lesions of the precentral gyrus on FLAIR MRI disappear in the subacute phase.

CONCLUSION

We conclude that bilateral cerebral contusions of the precentral gyri can cause paraplegia. Coronal MRI in the acute phase is useful in the diagnosis and the prediction of the functional prognosis.

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Conflicts of interest

There are no conflicts of interest.

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