

Isolated Distal Leg Weakness due to a Small Cerebral Infarction Masquerading as a Spinal Lesion

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Acute stroke with isolated monoparesis manifesting as distal weakness of a single lower extremity has rarely been described. We report two patients with small cortical infarction who had distal weakness of a single lower extremity. In both cases, diffusion-weighted image (DWI) was used to detect small lesions in the contralateral cortex. These cases illustrate that small cortical infarction can cause isolated monoparesis limited to distal part of the leg and it may be misdiagnosed as spinal lesions, especially when lower back pain and transient sensory symptoms are accompanied. In case of the abrupt onset of weakness limited to one lower limb, the possibility of stroke should be considered and careful attention to identify cortical lesions using magnetic resonance imaging, especially DWI is required.

KEY WORDS : Pure motor monoparesis · Cerebral infarction.

Introduction

Pure motor monoparesis (PMM) is a rare condition characterized by isolated motor deficit confined to one limb without sensory deficit and may be caused a space-occupying lesion in the contralateral superficial frontoparietal cortex^{4,6-9,12}. Reported etiologies of PMM include brain tumor, brain abscess, ischemic stroke, and intracerebral hemorrhage^{6,7,11}. Among them, PMM is seen in 2-22% of patients with pure motor stroke, accounting for 3-14% of ischemic strokes^{1,4}. It has been reported that stroke in the majority of patients with PMM is ischemic, and hemorrhagic stroke is found in only 3-24%^{8,9}. The paucity of clinical signs may make it difficult determine its origin, being peripheral or central. We report two patients with PMM presenting with a pure motor deficit confined to lower extremity presumably due to cortical infarction demonstrated by diffusion-weighted image (DWI).

Case Report

Case 1

A 48-year-old right-handed woman with history of hypertension presented with a sudden onset of weakness of her

right leg. The patient had fallen two to three times a day while going for her daily walk for 5 days. Five days after the onset, the patient was admitted to the our institution. On admission, the patient was alert and oriented. The patient complained of low back pain and a tingling sensation of the right leg. Blood pressure was 140/95mmHg and pulse rate was 70 per minute. Neurological examination showed weakness of the right leg involving anterior tibial (foot dorsiflexors) and extensor hallucis muscle. The dorsiflexion power of the right foot was graded 4/5 by manual muscle testing. The right extensor hallucis muscle was more severely affected and the dorsiflexion power of the right big toe was graded 3/5 by manual muscle testing. Deep tendon reflexes were weak in the right lower limb. Babinski sign was negative bilaterally. Although the patient complained of a tingling sensation of the right leg, there were no sensory deficits. The rest of the neurological examination was unremarkable. Routine laboratory tests revealed no abnormalities and electrocardiography was normal. The patient had no history of diabetes mellitus and cardiac disease. At first, these clinical findings were thought be caused by lumbar spinal lesion such as herniated disc because of lower back pain, tingling sensation of the right leg, distal leg weakness, and decreased deep tendon reflexes. How-

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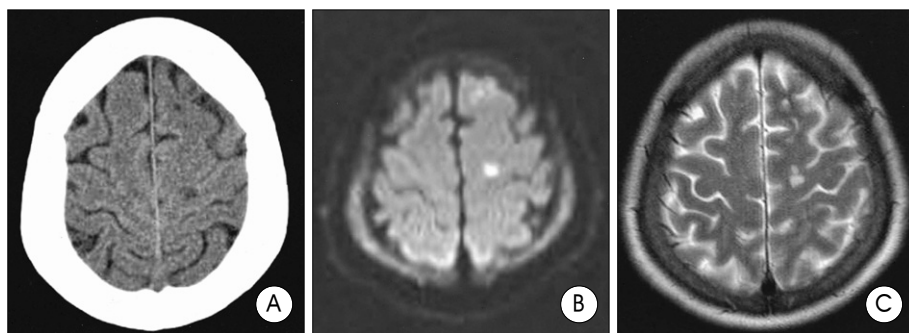


Fig. 1. A brain computed tomography performed six days after onset revealing a low-density lesion in the left frontoparietal region (A). Diffusion-weighted image performed six days after onset showing a small acute infarct with high signal intensity in the left precentral gyrus (B). T2-weighted image demonstrating high intensity in the same area (C).

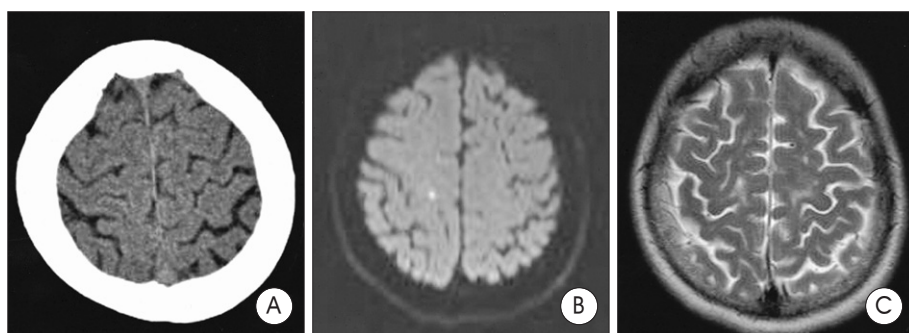


Fig. 2. A brain computed tomography performed one day after onset showing no lesions in the cerebral hemisphere (A). Diffusion-weighted image performed one day after onset revealing a small acute infarct with high signal intensity in the right precentral gyrus (B). T2-weighted image demonstrating high intensity in the same area (C).

ever, magnetic resonance image (MRI) of the lumbar and thoracic spine revealed only mild protrusion of the L5-S1 disc and electromyography on the right leg was normal. On the next day of admission, we performed computed tomography (CT) of the head was taken to rule out the possibility of cerebral lesion. Brain CT revealed a low-density lesion in the left frontoparietal region, suggesting infarction (Fig. 1A). Six days after the onset, DWI showed an acute infarct with reduction of diffusion in the left precentral cortex (Fig. 1B) and T2-weighted MRI showed increased signal intensity (Fig. 1C). Magnetic resonance angiography (MRA) showed no vascular abnormalities. A left carotid angiogram was also normal. On the sixth day after ictus, the patient was diagnosed as cerebral infarction. Therefore, the patient was only prescribed aspirin 100mg and clopidogrel 75mg per day without heparinization. Fourteen days after ictus, the strength in the right distal leg began to improve, and on the 26th day of admission, the strength of the right extensor hallucis became 4/5 and the patient was discharged.

Case 2

A 49-year-old right-handed woman with history of hypertension presented with difficulty walking because she was

unable to effectively dorsiflex her left ankle during the swing phase of walking one day prior to admission. The patient had suffered from back pain for 5 years. On admission, the patient was alert and oriented. She had no history of diabetes mellitus and cardiac disease. Blood pressure was 130/70mmHg and pulse rate was 82 per minute. Neurological examination showed the weakness of the tibialis anterior muscle, gastrocnemius muscle, and extensor and flexor muscles of the toes on the left side (Gr. IV/V). The remaining muscles of left leg retained normal strength. The patient complained of a tingling sensation of the left leg. However, sensation was normal in all modalities including pain, proprioception, vibration, and two point discrimination. Deep tendon reflexes were normal. Babinski sign was negative bilaterally. Initially, because of these findings, lumbar spinal lesion was suspected. In routine laboratory

examinations, blood glucose, and serum total cholesterol were all normal. There were no abnormalities in the MRI of lumbar spine. Electromyography on the left leg was normal. Brain CT performed on the next day after ictus revealed no lesion (Fig. 2A), but DWI showed a small acute infarct on the right motor cortex (Fig. 2B) and T2-weighted MRI showed slightly increased signal intensity (Fig. 2C). MRA, however, revealed no vascular abnormalities. This small cortical infarction was finally thought to be responsible for neurologic findings. The patient was started on intravenous heparin (heparin 20000 U mixed with normal saline 1000ml : infusion rate 40ml/hr). On the fifth day of admission, systemic heparinization was discontinued and the patient was prescribed clopidogrel 75mg per day. After rehabilitation, the left leg weakness gradually improved and the patient was discharged on the eighth day of admission.

Discussion

A common presentation in a neurosurgical clinic is loss of muscle strength. When evaluating weakness, important considerations are the temporal profile (acute, subacute, chronic), distribution of weakness (proximal, distal, symmetric,

asymmetric), progression of the weakness, and associated symptoms such as pain and sensory changes.

Among several types of weakness, our patients presented with abrupt onset of weakness of ankle and toe dorsiflexion without sensory deficit. This sign may be associated with a variety of conditions such as dorsiflexor injuries (rupture of the tibialis anterior tendon, compartment syndrome), peripheral nerve lesions (lumbosacral plexopathy, lumbar radiculopathy, mononeuropathies of the deep peroneal, common peroneal, or sciatic nerves), upper motor neuron lesions (motor neuron disease such as amyotrophic lateral sclerosis, spinal cord lesions, cortical cerebral lesions), myopathy, drug toxicities or diabetes. These lesions can be differentiated by through clinical, radiological and electrodiagnostic examinations. Hematologic evaluation can be done to screen a metabolic cause, such as diabetes or presence of a toxin. MRI or magnetic resonance neurography is useful in visualizing areas surrounding damaged nerves. An electromyography may be useful in distinguishing between the different types of nerve damage that can be responsible for isolated distal leg weakness. Upper motor neuron lesions can be differentiated from peripheral nerve lesions on examination by eliciting hyperactive deep tendon reflexes and a positive Babinski sign. The patient's muscle tone and the presence of muscle atrophy can also suggest etiology.

In the present cases, associated symptoms were lower back pain and tingling sensation of the one lower limb. Neurological examination showed no increase of deep tendon reflex, no pathological reflexes, and sensation was normal in all modalities. Therefore, spinal lesions or other peripheral lesions were considered initially. However, contralateral small infarctions in the precentral cortex was demonstrated by using DWI, and lesions corresponded well with the site of the contralateral lower limb in the primary somatotopic motor area. Hence, these lesions were considered to be responsible for the neurologic symptom and signs of these patients.

Isolated monoparesis limited to distal leg is not a common feature in ischemic infarcts like present cases. Theoretically, any lesions suitably placed along the course of the corticospinal tract can provoke PMM. Although previous reports have suggested that the lesions most likely to produce PMM are those in the cortical or near-cortical area because somatotopic motor representation is most widely separated at this level, recent reports showed that only 48% of patients with monoparesis had a cortical lesion and the lesions in the subcortical area and brainstem were present in 31% and 8%, respectively^{9,12}. The clinical presentations were arm monoparesis in 63%, facial weakness in 22%, and leg monoparesis in 15%⁸. These reports concluded that leg deficits were most frequently associated with small cortical lesions in the anterior cerebral artery territory, arm motor paresis with middle cerebral artery lesions,

and facial deficit with subcortical lesions^{2,8,9}. In detail, the primary somatotopic motor area of toe and ankle movement was reported to be located within the mesial surface and the top of the lateral surface of the precentral gyrus^{3,6,10,11}.

Abrupt onset of neurological deficits is usually suggestive of a stroke. However, a non-sudden onset may be seen in 50% of pure motor stroke¹. It has been also reported that PMM may be associated with temporary sensory deficit. It may occur among 14-42% of patients with PMM due to extension of the cerebral edema along the central sulcus to postcentral gyrus. Furthermore, small cortical lesions can be missed by carelessly performed scan and one can easily overlook such a small lesion. Brain CT may show no lesions in about 40% of the patients with monoparesis following stroke⁹. This is especially true in the case of PMM confined to lower limb because the lesion may be located in the top of the frontal lobe cortex, an area that can be easily missed by routine scans⁴. Therefore, the patients with PMM may be misdiagnosed as spinal lesions, when the patients present with a slow onset of isolated monoparesis associated with temporary sensory deficit^{5,7}.

Conclusion

From our experiences, it is strongly recommended to consider the possibility of stroke in examining every patient manifesting PMM, especially when the onset is acute, and to search for small cortical infarctions using DWI. Although the favorable outcome of patients with PMM following stroke has been shown, prompt evaluation and management are necessary to prevent further progression of stroke and to provide better outcome.

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