

Case Report

Cerebral Infarction Presenting with Unilateral Isolated Foot Drop

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Weakness of the dorsiflexor muscles of the ankle or toe, referred to as foot drop, is a relatively common presentation. In most cases, foot drop is caused by a lower motor neuron disease such as peroneal peripheral neuropathy, L4–5 radiculopathic sciatic neuropathy, or polyneuropathy. Although upper motor neuron lesions can present as foot drop, the incidence is very rare. Here, we report an extremely rare case in which foot drop was the only presenting symptom of cerebral infarction.

Key Words : Foot drop · Cerebral infarction · Upper motor neuron.

INTRODUCTION

Foot drop is a symptom which presents an inability or weakness in the dorsiflexor muscles of the ankle and the toe. It is mainly caused by neurological lesions of the lower motor neuron system, but upper motor neuron injury may also cause the symptom. As above, the most common lower motor neuron (LMN) pathology for foot drop is peroneal neuropathy, which often results from the compression at the fibular head. For other LMN pathologies, there are L4–5 radiculopathy, lumbar plexopathies, partial sciatic neuropathy and polyneuropathy^{1,4-6,9)}.

Less commonly, an upper motor neuron (UMN) lesion can cause foot drop, which involves the interhemispheric motor cortex, corona radiata, internal capsule, cerebral peduncle, medulla, or spinal pyramidal tract⁹⁾. Isolated foot drop is very rarely caused by an upper motor neuron lesion^{3-6,8,9)}.

In this report, we present an extremely rare case of an isolated foot drop caused by focal cortical infarction of the motor cortex.

CASE REPORT

A 74-year-old male visited the outpatient department presenting a one-week history of right-side foot drop. The patient had no history of hypertension, diabetes, hypothyroidism, arthritis, cancer, hepatitis or stroke. He denied any recent traumatic accidents

to the right leg. The complete physical evaluation was conducted, showing no abnormalities such as visual changes, fever, chills, dizziness, headache, or bowel or bladder problems. In the detailed neurological examination, he was well oriented and exhibited no signs of cranial nerve function disorders such as dysarthria, dysphagia or facial palsy. Distal pulse and sensation were intact in all extremities including the dorsal aspect of the right foot. Passive range of motion was in a normal range in every peripheral joint. The patient was also negative for the straight leg raise test and femoral nerve stretch test. No pathological reflexes were found bilaterally such as Babinski and ankle clonus, with normal deep tendon reflexes. Individual muscle strength tests were normal for all extremities except for the right tibialis anterior and the extensor digitorum. Muscle tone grades in the right tibialis anterior and extensor digitorum were 0/5 when measured by Modified Ashworth Scale.

Electromyogram (EMG) and magnetic resonance imaging (MRI) were performed. EMG showed no remarkable findings and lumbar spinal MRI revealed a diffuse bulging disc at the L4–5 disc space level. However, there was no evidence of severe nerve root compression that was susceptible to current condition. Since the lumbar spinal lesions were ruled out for the patient's symptom, a brain MRI was performed to investigate any potential central lesions. Diffusion-weighted imaging (DWI) showed focal high signal intensity on the left upper motor cortex

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(Fig. 1A). An afferent diffusion coefficient (ADC) map revealed low signal intensity on the same area (Fig. 1B). Subsequently, the patient was diagnosed with subacute cerebral infarction. After one week of treatment with an anti-platelet agent and rehabilitation therapy, the patient's muscle power showed improvement in his right tibialis anterior and extensor digitorum, from 0/5 to 2/5.

DISCUSSION

Foot drop is defined as the weakness of an ankle or one of the toe's dorsiflexors; the tibialis anterior, extensor digitorum longus, or extensor hallucis longus^{1-6,8,9}. It is usually caused by LMN pathology that results from disruption of the deep peroneal nerve. Since the nerve is in contact with the 4th and 5th lumbar spinal nerve, peroneal neuropathy and L4-5 radiculopathy are the most common causes of foot drop^{4,9}. Additionally, UMN pa-

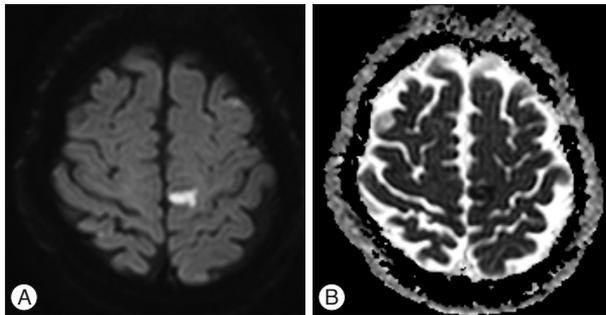


Fig. 1. Brain MRI DWI reveals focal high-signal intensity in the uppermost convexity of the precentral gyrus (A), and low-signal intensity in the same area on ADC map (B).

thologies can also cause foot drop. Since the motor components of the 4th and 5th spinal nerves are controlled by the primary motor cortex via the corona radiata, the internal capsule, the basal ganglia and the ventral gray matter of the spinal cord, any lesion along this neuronal pathway can be the UMN pathology of foot drop. However, since these UMN fibers are condensed and closed with many other functional neuronal fibers, isolated foot drop due to an UMN pathology is very rare^{3,6,8}.

For the diagnosis of foot drop, detailed neurological examination and good anamnesis are essential. Sequential EMG and MRI findings were found to be correlated in 92% of foot-drop patients⁶. We performed an EMG study and spinal MRI to investigate a probable lesion such as peripheral neuropathy or L4-5 radiculopathy. But, none of the results were specific enough to explain the symptom. Although possible UMN lesions were not clearly indicated in previous examinations, showing no signs of abnormal reflexes of the Babinski reflex or ankle clonus, we performed brain MRI in the interest of being thorough; the MRI revealed a diagnosis of subacute cerebral infarction which caused the foot drop.

To our knowledge 19 cases of foot drop due to a central nervous system (CNS) lesion have been reported (Table 1). In these reports, various CNS etiologies were illustrated; brain tumors, metastases, contusion, infarction, abscess, multiple sclerosis, neurocysticercosis. However, the majority of pathologies were manifested in the parasagittal area^{1,2,4-6,8,9}. Direct parasagittal damage or local mass effect on the parasagittal region can cause the foot drop, since the medial homunculus of the primary motor cortex at the mesial surface, adjacent to the supplementary motor area

Table 1. Summary of reported foot drop cases due to CNS lesion

Author (year)	Age/sex	Pathology	Accompanying symptoms or UMN signs	Site of lesion
Eskandary et al. (1995)	18/F	Suspicious of Tumor	Babinski, ankle clonus	Parasagittal region
	33/M	Meningioma	Hemiparesis	Parasagittal region
	14/F	Abscess	Fever, headache, vomiting, papilledema	Parasagittal region
	55/M	Astrocytoma	Hemiparesis, ankle clonus	Parasagittal region
	28/F	Multiple sclerosis	Babinski, hyperreflexia	Periventricular white mater*
	10/M	Contusion	Hemiparesis, Babinski, hyperreflexia	Parasagittal region
Baysefer et al. (1998)	57/M	Anaplastic astrocytoma	Babinski, hyperreflexia	Parasagittal region
	25/F	Meningioma	Papilledema, hyperreflexia	Parasagittal region
	19/F	Astrocytoma	Babinski, hyperreflexia	Parasagittal region
Kohno et al. (1999)	42/F	Cerebral infarction	Hyperreflexia	Parasagittal region
Atac et al. (2004)	21/M	Contusion	None	Parasagittal region
Chatterjee et al. (2004)	60/M	Metastatic adenocarcinoma	Babinski, hyperreflexia	Parasagittal region
Djekidel et al. (2006)	68/M	Metastatic adenocarcinoma	None	Parasagittal region
Westhout et al. (2006)	69/M	Tumor	Monoparesis	Parasagittal region
Ku et al. (2007)	68/M	Cerebral infarction	None	Parasagittal region
Oktem et al. (2010)	74/F	Contusion	None	Parasagittal region
Hiraga et al. (2010)	70/M	Subcortical hemorrhage	None	Frontal subcortex*
Sahu et al. (2012)	14/M	Neurocysticercosis	None	Parasagittal region
Goia et al. (2013)	12/M	Teratoid/rhabdoid tumor	Babinski, ankle clonus	Parasagittal region
Present case	74/M	Cerebral infarction	None	Parasagittal region

*Subcortical white mater lesion within corona radiata. CNS : central nervous system, UMN : upper motor neuron

(SMA), or at the top of the lateral surface of the precentralgyrus are involved in ankle and toe movement^{5,9}). Usually in the motor system, SMA is responsible for motor functions such as planning and initiation. Therefore, the lesions involving the SMA may cause astasia or apraxia rather than weakness. But, rarely it can cause motor deficits because the SMA proper transfers corticospinal efferences and is connected with the primary motor cortex⁷). There are only two previous reports on isolated foot drop caused by lesions other than parasagittal lesions^{1,3}). In one of those cases, the lesion was within the corona radiata, which conveys neuronal impulses from the cerebral cortex of the primary somatotopic motor area to toe and ankle movement. According to these previous reports, lesions of the cortical surface are more likely to cause foot drop than deeper lesions. Because the descending motor axons are compacted into a small area by deeper lesions, foot drop due to the subcortical lesions are rarely reported³).

Patients with cerebral pathologies of foot drop usually have had UMN symptoms, such as Babinski reflex, hyperreflexia, and ankle clonus¹). However, CNS pathologies can be found in some patients without any UMN symptoms, as seen in our case^{4,6,8}). In such cases, diagnosis is delayed due to its atypical presentation which resembles a peripheral type of foot drop.

To our knowledge, this is the third report of unilateral isolated foot drop from a cerebral infarction. Brain MRI showed ischemic damage in the somatotopic ankle and toe motor homunculus.

CONCLUSION

We report a case of unilateral isolated foot drop caused by ce-

rebral infarction. In the initial examination of said patient, it could be easily misidentified as a peripheral pathology or misdiagnosed as other irrelevant conditions. To avoid such mistakes, CNS evaluation should be considered if the lumbar MRI and EMG results are negative for the symptom. Even if the incidence is extremely rare, central isolated foot drop present without any UMN symptoms.

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