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Right lower limb apraxia in a patient with left supplementary motor area infarction: intactness of the corticospinal tract confirmed by transcranial magnetic stimulation

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Abstract

We reported a 50-year-old female patient with left supplementary motor area infarction who presented right lower limb apraxia and investigated the possible causes using transcranial magnetic stimulation. The patient was able to walk and climb stairs spontaneously without any assistance at 3 weeks after onset. However, she was unable to intentionally move her right lower limb although she understood what she supposed to do. The motor evoked potential evoked by transcranial magnetic stimulation from the right lower limb was within the normal range, indicating that the corticospinal tract innervating the right lower limb was uninjured. Thus, we thought that her motor dysfunction was not induced by motor weakness, and confirmed her symptoms as apraxia. In addition, these results also suggest that transcranial magnetic stimulation is helpful for diagnosing apraxia.

***Key Words:** nerve regeneration; lower limb apraxia; supplementary motor area; cerebral infarct; transcranial magnetic stimulation; corticospinal tract; stroke; neural regeneration*

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Introduction

Stroke is one of the most common causes of major disability in adults. One of the most serious disabling sequelae after stroke is motor weakness, and over half of stroke patients experience a residual motor deficit (Duncan et al., 1992). Motor weakness is attributed to injury of the neural tracts associated with motor function. However, motor weakness-like symptoms can also be accompanied by apraxia, defined as the inability to perform learned skilled movements despite having no motor deficits (Heilman and Rothi, 2003; Gross and Grossman, 2008).

The corticospinal tract (CST) is the neural tract most closely linked to motor function in the human brain (Ward et al., 2006), and the CST should be preserved in patients with apraxia following stroke. Diagnosis of apraxia can be difficult because it is made by clinicians' observation of movements and is frequently confused with motor weakness. Therefore, confirming preservation of the CST would aid diagnosis of apraxia after stroke.

Transcranial magnetic stimulation (TMS) stimulates the neurons of the CST and provides a unique ability to estimate the amount of CST fibers by measuring motor evoked potential (MEP) amplitude (Rossini et al., 1994). Therefore, TMS could be useful for demonstrating changes in the amount of CST fibers and diagnosing apraxia. Some studies have used TMS for diagnosis of apraxia of the upper limb after cerebral infarction (Hong et al., 2012; Jang, 2013). However, little is known about apraxia of the lower limb following stroke.

In the current study, we describe a patient who showed right lower limb apraxia after cerebral infarction in the left supplementary motor area (SMA), with demonstrated preservation of the CST using TMS.

Case Report

Patient presentation

A 50-year-old, right-handed, woman underwent conservative treatment of the left prefrontal cortex (mainly left SMA) due to left anterior territory artery occlusion at the neurology department of a university hospital. At the onset of her cerebral infarction, the patient presented with complete weakness of the right lower limb (Medical Research Council [MRC] (Medical Research Council, 1976): 0/5) and moderate weakness of the right upper limb (MRC: 3/5) (**Table 1**). One week after onset, the patient was transferred to the rehabilitation department of the same hospital. At that time, mild weakness of her right upper limb (MRC: $4^+/5$) was shown and severe weakness of her right lower limb (MRC: $1–2^+/5$) was still present (**Table 1**). She was able to walk with moderate assistance. Her Mini-Mental State Exam score was

Table 1 Changes in Medical Research Council scores of affected (right) extremities in the patient

	Onset	1 week	3 weeks	3 months
Shoulder abductor	3	4^+	4^+	5
Elbow flexor	*3*	*4*$^+$	4^+	*5*
Finger flexor	3	4^+	4^+	5
Finger extensor	3	4^+	4^+	5
Hip flexor	0	2^+	2^+	4^+
Knee extensor	0	1	2^-	4^+
Ankle dorsiflexor	0	1	1	4^+

Medical Research Council scores are as follows: 0, no contraction; 1, palpable contraction but no visible movement; 2, movement without gravity; 3, movement against gravity; 4, movement against a resistance lower than the resistance overcome by the healthy side; and 5, movement against a resistance equal to the maximum resistance overcome by the healthy side.

28 and she presented with mild clumsiness in the right upper limb (Jepsen-Taylor Hand Function Test: right 89, left 97; Box & Block Test: right 47, left 59), although she could move her left hand smoothly. Somatosensory, ideomotor, and ideational apraxia tests of the upper limbs did not indicate any significant abnormalities.

She underwent comprehensive rehabilitative management, including movement therapy and neuromuscular electrical stimulation of the affected finger extensor and ankle dorsiflexors. Movement therapy focused on improvement in the motor function of the right hemiplegia and was performed five times a week. At 3 weeks after onset, she was able to walk and climb stairs spontaneously without any assistance but with slight limping. Good right hip flexion, right knee extension, and right ankle dorsiflexion were observed while walking and climbing stairs. However, when she was asked to walk fast or slow and to run, she understood what she was supposed to do but could not do so. When we instructed her to move her right feet up and down, she was unable to move her right foot, although she could move her left lower limb correctly. She was also unable to imitate gestures, such as kick a ball or trace a line on the floor with her right lower limb, even though she understood what she supposed to do. In addition, when her motor weakness was evaluated in sitting and supine positions, she could not move her right lower limb against gravity (MRC: 1–2$^+$) (**Table 1**).

Three months after onset, she was able to control her gait speed and run. In addition, she could kick a ball and trace a line on the floor with only slight clumsiness. On motor evaluation in the sitting position, only minimal motor weakness in her right lower limb was observed (MRC: 4$^+$) (**Table 1**) compared with her left lower limb. The function of her right upper limb was normal (MRC: 5).

Transcranial magnetic stimulation

TMS was performed 3 weeks after infarct onset. A Magstim Novametrix 200 magnetic stimulator with a 9-cm mean diameter circular coil (Novametrix Medical Systems Inc., Wallingford, CT, USA) was used for TMS. Cortical stimulation was performed with the coil held tangentially over the vertex. Stimulation of the left hemisphere was provided by a counterclockwise current, and stimulation of the right hemisphere was provided by a clockwise current. MEPs were obtained at 1 week from both abductor pollicis brevis (APB) and tibialis anterior (TA) muscles in a relaxed state. The excitatory threshold (ET) was defined as the minimum stimulus required to elicit an MEP with a peak-to-peak amplitude of 50 or greater in two out of four attempts. Stimulation intensity was set at the ET plus 20% of the maximum stimulator output. MEPs having the shortest latency and average peak-to-peak amplitudes were adopted. The MEPs evoked from the affected upper and lower limbs were within the normal range (right APB: latency = 22.2 ms, amplitude = 4,900 μV, ET = 50%; left APB: latency = 22.2 ms, amplitude = 4,900 μV, ET = 50%; right TA: latency = 30.1 ms, amplitude = 4,100 μV, ET = 60%; left TA: latency = 30.3 ms, amplitude = 4,200 μV, ET = 60%) (**Figure 1**) (Jang et al., 2005; Cacchio et al., 2011).

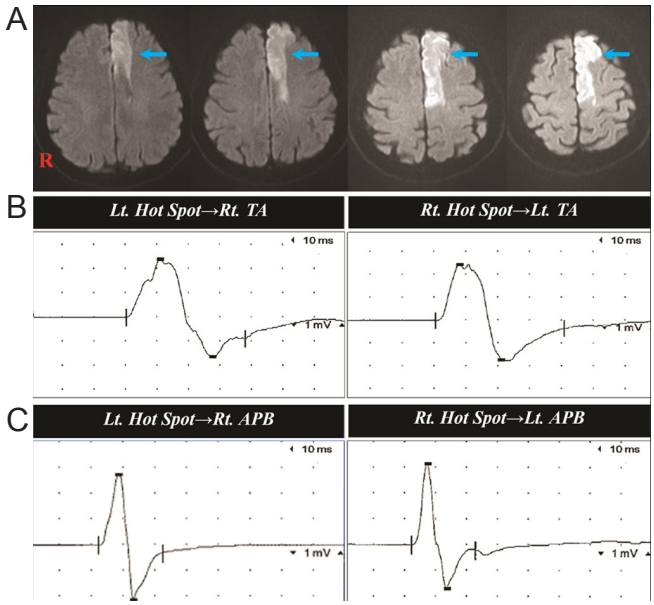

Figure 1 Imaging and transcranial magnetic stimulation results in a patient with cerebral infarction
(A) Diffusion brain magnetic resonance images acquired at onset show an infarction in the left anterior cerebral territory (blue arrows). (B) MEPs were obtained from both the left and right TAs at 1 week after onset by stimulating the hot spot of the contralateral motor cortex. The latency and amplitude of MEP from the left TA were 30.3 ms and 4,200 μV, respectively, and those from the right TA were 30.1 ms and 4,100 μV, respectively. (C) The latency and amplitude of 1-week MEPs obtained from both APBs were 22.2 ms and 4,900 μV. APB: Abductor pollicis brevis; MEP: motor evoked potential; TA: tibialis anterior; Lt: left; Rt: right.

Discussion

Here, we report a patient with right lower limb apraxia following infarction in the left anterior descending coronary artery territory, mainly in the left SMA. Our patient was able to independently walk and climb stairs at 3 weeks after onset, although the patient exhibited slight limping. However, she could not control gait velocity and move her right lower

limb following clinicians' verbal commands, even though she understood what she was asked. She was also unable to imitate gestures with her right lower limb. The MRC of the right lower extremity was between the 1 and 2^+ grade. These symptoms seemed to arise from spatial and temporal errors, rather than a disturbance in the conceptual knowledge of motor performance and sequencing of a series of acts. Therefore, our patient's right leg symptoms were thought to be compatible with those of ideomotor apraxia rather than ideational apraxia. Furthermore, we confirmed that the CST from the affected (left) hemisphere to the right TA muscle showed normal findings in terms of MEP parameters (latency and amplitude) on TMS, which indicates that the CST innervating the affected lower limb was uninjured. Because CST is the most important neural tract in motor function, motor dysfunction manifested in the patient seems not to be induced by motor weakness. We believe that the patient's symptoms, such as a limping gait and low MRC score ($1–2^+$), could be confused with motor weakness due to motor tract injury. Therefore, confirming the intactness of the CST is helpful for ruling out motor weakness and diagnosing apraxia.

SMA participates in internal guidance or planning of movement, and patients with SMA lesions have shown ideomotor apraxia (Watson et al., 1986; Marchetti and Della Sala, 1997). Thus, the symptoms of ideomotor apraxia in our patient's right lower limb seemed to be caused by the cerebral infarction in the left SMA.

Nonetheless, two studies (Kwon and Jang, 2012; Ito et al., 2013) have reported apraxia of the lower limb. However, these two previous studies did not prove the intactness of the CST, and Kwon and Jang (2012) stated that the symptoms of their patient were not completely compatible with apraxia.

In conclusion, the motor dysfunctions of the right lower limb shown in our patient were considered to be symptoms of ideomotor apraxia. We demonstrated that the CST was not injured by cerebral infarction using TMS. The TMS findings consolidated our belief that our patient's lower limb symptoms were due to apraxia, not motor weakness. We believe that our study shows the usefulness of TMS for diagnosing lower limb apraxia. However, this study has some limitations. First, this is a single case study. Second, we ruled out an injury of CST only, and did not evaluate the state of other neural tracts (non-CST, such as corticoreticular pathway) which might be associated with motor weakness of our patient. Therefore, further studies addressing these limitations are needed.

Author contributions: *MCC conceived and designed the study, was responsible for data acquisition, manuscript development and manuscript writing. MHC was in charge of data acquisition, manuscript development and manuscript writing. Both of these two authors approved the final version of this article.*

Conflicts of interest: *None declared.*

References

Cacchio A, Paoloni M, Cimini N, Mangone M, Liris G, Aloisi P, Santilli V, Marrelli A (2011) Reliability of TMS-related measures of tibialis anterior muscle in patients with chronic stroke and healthy subjects. J Neurol Sci 303:90-94.

Duncan PW, Goldstein LB, Matchar D, Divine GW, Feussner J (1992) Measurement of motor recovery after stroke. Outcome assessment and sample size requirements. Stroke 23:1084-1089.

Gross RG, Grossman M (2008) Update on apraxia. Curr Neurol Neurosci Rep 8:490-496.

Heilman KM, Rothi LJG (2003) In: Clinical Neuropsychology (Heilman KM, Valenstein E, eds.), pp215-235. New York: Oxford University Press.

Hong JH, Lee J, Cho YW, Byun WM, Cho HK, Son SM, Jang SH (2012) Limb apraxia in a patient with cerebral infarct: diffusion tensor tractography study. NeuroRehabilitation 30:255-259.

Ito I, Ito K, Shindo N (2013) Left leg apraxia after anterior cerebral artery territory infarction: functional analysis using single-photon emission computed tomography. Eur Neurol 69:252-256.

Jang SH (2013) Motor recovery by improvement of limb-kinetic apraxia in a chronic stroke patient. NeuroRehabilitation 33:195-200.

Jang SH, Cho SH, Kim YH, You SH, Kim SH, Kim O, Yang DS, Son SM (2005) Motor recovery mechanism of diffuse axonal injury: a combined study of transcranial magnetic stimulation and functional MRI. Restor Neurol Neurosci 23:51-56.

Kwon H, Jang SH (2012) Delayed recovery of gait function in a patient with intracerebral hemorrhage. J Rehail Med 44:378-380.

Marchetti C, Della Sala S (1997) On crossed apraxia. Description of a right-handed apraxic patient with right supplementary motor area damage. Cortex 33:341-354.

Medical Research Council (1976) Aids to examination of the peripheral nervous system. London, England, Her Majesty's Stationery Office.

Rossini PM, Barker AT, Berardelli A, Caramia MD, Caruso G, Cracco RQ, Dimitrijević MR, Hallett M, Katayama Y, Lücking CH (1994) Non-invasive electrical and magnetic stimulation of the brain, spinal cord and roots: Basic principles and procedures for routine clinical application. Report of an IFCN committee. Electroencephalogr Clini Neurophysiol 91:79-92.

Ward NS, Newton JM, Swayne OB, Lee L, Thompson AJ, Greenwood RJ, Rothwell JC, Frackowiak RS (2006) Motor system activation after subcortical stroke depends on corticospinal system integrity. Brain 129:809-819.

Watson RT, Fleet WS, Gonzalez-Rothi L, Heilman KM (1986) Apraxia and the supplementary motor area. Arch Neurol 43:787-792.

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