Isolated Shoulder Palsy due to Infarction of the Cortical Branch of the Middle Cerebral Artery

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Abstract

A 71-year-old man with hyperlipidemia abruptly developed left-sided isolated shoulder palsy. Cranial magnetic resonance imaging demonstrated infarction of the cortical branch of the right middle cerebral artery (MCA). In the primary motor cortex, there is broad somatotopic representation of various body parts in a particular arrangement, and the area corresponding to the shoulder is very small. Consequently, there have been only 3 reported cases of isolated shoulder palsy due to cerebral infarction, and its vascular supply remains uncertain. The present case indicates that the corresponding area to the shoulder receives its blood from the cortical branch of the MCA.

Key words: homunculus, magnetic resonance imaging (MRI), precentral gyrus, primary motor cortex, pure motor monoparesis, three-dimensional computed tomography angiography (3D-CTA)

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Introduction

Isolated shoulder palsy (ISP) is defined as unilateral shoulder motor weakness without other neurological deficit which is caused by pyramidal tract disorder (1-3). In 1937, based on electrical stimulation of the brain surface during surgery, Penfield and Boldrey (4) reported that there was a broad somatotopic representation of the different body parts in a particular arrangement in the primary motor cortex. Because the area corresponding to the shoulder is very small, to date there have been only 3 reported cases of ISP due to a cortical infarction, and its vascular supply remains controversial (1-3). Here, we describe an additional case, which proves that the corresponding area to the shoulder receives its blood from the cortical branch of the middle cerebral artery (MCA).

Case Report

A 75-year-old Japanese man with hyperlipidemia and smoking habit suddenly presented with difficulty in lifting his left arm, despite the lack of shoulder pain in March 2012. Therefore, the patient was admitted to our Neurology ward. His consciousness was alert. Blood pressure was 124/78 mmHg. The heart rate was 64/min. General examination demonstrated no abnormalities. Cranial nerve impairment was not detected. The patient was right-handed. Hand grasp in power was 28 kg in the right and 23 kg in the left. In the upper extremities, left-sided Barré sign was positive. In the lower extremities, neither Barré sign nor Mingazzini sign was positive on the left side. Manual muscle testing on the left upper limb demonstrated that pectoralis major, deltoitd, supraspinatus, infraspinatus, rhomboideus, serratus anterior, and latissimus dorsi were at grade 3, despite trapzeius, diceps brachii, triceps brachialis, brachioradialis, extensor carpi radialis longus, extensor carpi ulnaris, flexor carpi radialis, and flexor carpi ulnaris at grade 5. Bend, extension, and wiggle were normal in both sides of the fingers. Muscle atrophy was normal in the extremities. Muscle atrophy and fasciculation were not observed. Deep tendon reflexes were all normal and pathological reflexes were not detected in the extremities. Superficial sensation (touch sensation, pain sensation, temperature sensation, and topesthesia), deep sensa-
tion (joint sensation and vibratory sense), and combined sensation (two-point discrimination, graphesthesia, stereognosis, and double simultaneous stimulation) were all normal. Dysmetria, decomposition of movement, and intention tremor were not observed in finger-nose test and nose-finger-nose test. Hyperpronation test and hand pronation supination test were all normal. Truncal ataxia was not observed. In the gait test, arm swing was slow on the left side. There were no other neurologic abnormalities. As a result, there was moderate weakness restricted to the left shoulder with normal joint flexibility, despite normal muscle strength in the elbow, wrist and fingers. Therefore, the patient was diagnosed as having left-sided ISP. Complete blood cell count and blood chemistry were within normal ranges. Chest roentgenogram, electrocardiogram and echocardiogram findings were normal. Carotid ultrasonogram demonstrated occlusion of the right internal carotid artery. Shoulder roentgenogram demonstrated no abnormalities. Cranial magnetic resonance (MR) imaging demonstrated infarct lesion in the territory of the cortical branch of the middle cerebral artery on the right side (arrow). E: The internal carotid artery was not revealed on the right side.

Discussion

Penfield and Boldrey (4) described that the motor shoulder area might be located more medially than the motor hand area in the precentral gyrus. In addition, Celebisoy et al. (5) noted that the hand area in the cerebral motor cortex is located in the middle to lower portion of the anterior wall of the central sulcus. However, in the cerebral motor cortex, detailed vascular supply remains uncertain. Kim (6) speculated that the medial portion of the precentral knob representing topographically ulnar-sided fingers might correspond to the borderzone area between the anterior cerebral artery and MCA, whereas the lateral portion representing radial-sided fingers might be supplied by distal MCA branches.

In 2003, Komatsu et al. (1) described the first case of ISP. Diffusion-weighted and T2-weighted cranial MR imaging demonstrated a small infarct lesion in the precentral gyrus on the contralateral side to shoulder palsy (1). Electrocardiogram did not demonstrate a source of embolus (1). Borderzone infarction was suspected as its pathogenesis, because of severe stenosis of the internal carotid artery based on MR angiography finding (1). However, no photograph of MR angiography was shown (1). In addition, because cerebral
angiography, 3D-CTA, and carotid ultrasonogram findings were not mentioned (1), whether the collateral circulation was present or not was unknown. In 2006, Nah et al. (2) reported the second case of ISP. Cranial MR imaging demonstrated a small infarct lesion in the right precentral gyrus (2). Cranial MR angiography demonstrated no steno-occlusive lesions in the intracranial and extracranial arteries (2). Electrocardiogram demonstrated atrial fibrillation (2). Echocardiogram demonstrated vigorous spontaneous echogenic contrast with sludge in the left atrial appendage (2). However, detailed etiology of infarction was not mentioned, though intravenous heparin therapy was performed (2). In 2007, Uncini et al. (7) reported a case of unilateral shoulder palsy due to a localized cortical infarction. An analysis of functional MR imaging confirmed the location of the corresponding area to the shoulder in the precentral gyrus (7), however, muscle strength weakness of the biceps and triceps were also observed (7). Pathogenesis of infarction was not mentioned (7). In 2011, we noted a third case of ISP. Diffusion-weighted cranial MR imaging demonstrated a cortical infarct lesion in the 2 serial axial images (3). Electrocardiogram, echocardiogram, carotid ultrasonography, and cranial MR angiography demonstrated normal findings (3). Marked blood pressure fluctuations were not observed. Consequently, atherothrombosis was suspected. Thereafter, by administration of intravenous anti-platelet agent, the patient became asymptomatic within 10 days (3).

In these reported 4 cases of unilateral shoulder palsy (1-3, 5), infarct lesion was very small and localized in the precentral gyrus. By contrast, in the present patient, cranial MR imaging demonstrated an infarct lesion in the territory of the cortical branch of the MCA. We considered that this branch might be the central artery. On the 3D-CTA study, despite severe stenosis of the proximal portion of the right ICA, the right MCA was revealed. Consequently, we believed that the cortical branch infarct lesion might be caused by atherothrombosis.

In conclusion, we emphasize that this is the fourth reported case of ISP, which indicates that the cortical branch of the MCA may supply blood for the area corresponding to the shoulder in the precentral gyrus.

The authors state that they have no Conflict of Interest (COI).

References