Pseudoperipheral Palsy due to Infarction in the Internal Capsule

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Abstract

Pseudoperipheral palsy can be caused by cerebral cortical infarctions; however, it is rarely caused by lacunar infarctions, including those in the posterior limb of the internal capsule. Meanwhile, the somatotopic localization of the corticospinal tract in the posterior limb of the internal capsule remains unknown. We herein report the case of an 81-year-old Japanese woman who presented with a left hand drop. Brain magnetic resonance imaging revealed an acute infarction as the causative lesion at the inferior level of the anteromedial portion of the posterior limb of the right internal capsule. This case report indicates the topography of hand fibers in the internal capsule.

Key words: cerebral infarction, drop hand, internal capsule, isolated hand palsy, pseudoperipheral palsy


Introduction

Pseudoperipheral palsy, also known as isolated hand palsy, is characterized by predominant weakness of the hand associated with cerebral infarction. Lhermitte originally reported pseudoperipheral palsy in 1909. In that report, patients with sensorimotor deficits in the fingers caused by central nervous lesions were described (1). Recent studies have revealed that pseudoperipheral palsy can result from acute infarctions of the precentral knob, the parietal lobe in the central sulcus region, the white matter of the angular gyrus or the vascular border zone (2, 3). Rankin et al. also reported a patient with pseudoperipheral median nerve palsy due to an acute stroke in the corona radiata (4). To the best of our knowledge, pseudoperipheral palsy caused by a lacunar infarction including the posterior limb of the internal capsule (PLIC) has not been previously reported. The topography of the corticospinal tract in the PLIC remains obscure. We herein describe a case of pseudoperipheral palsy caused by an infarction in the PLIC. Our case report may help to enhance understanding of the somatotopic localization of the corticospinal tract in the PLIC.

Case Report

An 81-year-old Japanese woman with hypertension and hypercholesterolemia presented with sudden-onset left hemiparesis. A few hours later, she was referred to our hospital where she was admitted. Her left hemiparesis improved to left hand drop only on admission. Her blood pressure was 192/86 mm Hg, and her pulse was regular. Neurological examinations revealed mild muscular weakness in her left hand (Fig. 1). According to the Medical Research Council scale, the patient’s muscle strength was as follows: grade 1 in the left wrist extensor, grade 4 in the left wrist flexor, grade 3 in the left fingers extensor and grade 4 in the left fingers flexor. Abduction and adduction of the left fingers were also moderately impaired. No muscular weakness or ataxia were observed in other extremities. The examination of the cranial nerves was normal. The tendon reflexes were normal, and the bilateral cutaneous plantar response was flexor. Superficial and deep sensations were also normal. In addition, the patient exhibited a slightly reduced activity and daytime somnolence.

Brain magnetic resonance imaging (MRI) with a 1.5-Tesla system (GE, Munich, Germany) performed on day 1 de-
tected an acute infarction in the right PLIC and part of the thalamus (Fig. 2A, B). There were no lesions in the cerebral cortices. Magnetic resonance angiography showed no evidence of thrombus formation. A tentative diagnosis of pseudoperipheral palsy due to lacunar infarction was made. The laboratory results were normal. An electrocardiogram detected a normal sinus rhythm. Echocardiography was normal, and no thrombi were visible in the left atrium. The intravenous administration of sodium ozagrel (160 mg/day) and edaravone (60 mg/day) was initiated because thrombolysis is not usually performed if a rapid neurological improvement is observed. The left hand weakness gradually improved; however, the left hand drop remained on day 19. Follow-up brain MRI performed on day 19 detected a slightly decreased lesion signal on fluid-attenuated inversion recovery images (Fig. 2C, D). The lesion location was limited to the inferior level of the anteromedial portion of the right PLIC. Seven weeks after onset, the patient was able to extend her left hand to a horizontal line. A nerve conduction study of the upper extremities detected no evidence of left radial nerve palsy. Therefore, the diagnosis of pseudoperipheral palsy due to lacunar infarction in the PLIC was confirmed. The left hand weakness ultimately recovered; however, the slightly reduced activity remained.

![Figure 1](image1.png)

**Figure 1.** The patient exhibited left hand drop on admission.

![Figure 2](image2.png)

**Figure 2.** Brain axial magnetic resonance imaging with diffusion-weighted imaging conducted a few hours after onset detected a lesion in the posterior limb of the right internal capsule and part of the right thalamus (A, B). On follow-up brain magnetic resonance imaging, fluid-attenuated inversion recovery images revealed a limited lesion at the inferior level in the anteromedial portion of the posterior limb of the internal capsule (C, D).
Discussion

We herein reported a case of pseudoperipheral palsy caused by a lacunar infarction in the PLIC. Initially, the patient presented with left hemiparesis, then exhibited left hand drop only a few hours after onset. The patient’s clinical course suggests that the initial diffuse ischemia in the right PLIC improved and was limited to the PLIC-distributing cortical hand areas. The characteristics of the patient’s final lesion indicate the topography of the hand fibers in the PLIC.

The symptoms of pseudoperipheral palsy resemble those of radial, ulnar and/or median nerve palsy, as the neurological deficit is restricted to a specific group of fingers, with or without wrist symptoms. It is known that pure motor hemiparesis is restricted to a specific group of fingers, with or without radial, ulnar and/or median nerve palsy, as the neurologists in the PLIC.

The symptoms of pseudoperipheral palsy can occur clinically in the PLIC. To clarify the more precise topography of the hand fibers in the PLIC, further investigations are needed.

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

References


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