

A rare case of severe cognitive impairment which prolonged after first lacunar infarct in right internal capsule

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ABSTRACT

Lacunar infarcts have been defined as small lesions within a diameter of 1.5 centimeters causing mild symptoms. However, we treated a patient showing prolonged, severe disturbance of the cognitive function after a lacunar infarct. The 85-year-old woman was found lying on the floor of her house, and she was transported to our hospital. Computed tomography (CT) revealed a right lacunar infarct, and magnetic resonance imaging (MRI) showed severe periventricular hyperintensity (PVH) of her

brain. Her severe consciousness disorder showed improvement, but severe cognitive impairment including disorientation remained when she was transferred to another hospital. These findings suggested that the reserve capacity of her brain was minimal. The addition of a lacunar infarct to a severe situation like this might induce severe cognitive impairment. **Key words:** lacunar infarct; periventricular hyperintensity; poststroke dementia

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INTRODUCTION

Strokes are divided into three types: cardiogenic, atherothrombotic, and lacunar infarcts. Cardiogenic infarct is caused by emboli from the heart. Atherothrombotic infarct occurs due to occlusion caused by thrombus formation on arterial walls or thrombus separation from arterial walls such as in the internal carotid and middle cerebral arteries. Lacunar infarct occurs due to occlusion caused by a microthrombus or lipohyalinosis in a penetrating artery in the basal ganglia, thalamus, and pons. In addition, the diameter of a lacunar infarct is less than 1.5 cm; therefore, the symptoms are mostly mild [1,2].

However, if lacunar infarcts occur repeatedly or frequently, they may lead to cognitive impairment or dementia [1,2]. Some causes of cognitive impairment or dementia induced by repeated lacunar infarcts have been suggested. One is a diffuse white-matter lesion, and another is subclinical Alzheimer's disease. These may be associated with cognitive impairment or dementia caused by repeated lacunar infarcts [1].

We encountered the rare case of an elderly patient who showed marked deterioration of the cognitive function and prolonged cognitive impairment after her first lacunar infarct at the right internal capsule. We discuss the cause of the symptoms.

Case presentation

The patient was a right-handed, 85-year-old woman. Since she had been living alone without difficulties or problems, and her family considered that her cognitive function was within the normal range. One evening, a visitor found her lying on the floor of her house, and the left corner of her mouth was sagging. Therefore, stroke was suspected and she was transported to the emergency department of our hospital. She was in a semicoma during transportation; that is, she showed the avoidance of painful stimulation by withdrawing her upper limbs, indicating a consciousness level of 100 on the Japan Coma Scale (JCS) [3].

On admission, her blood pressure was 161/86 mmHg, pulse was 71 beats per minute, and oxygen saturation was 99%. Neurological findings on admission showed left hemifacial paralysis and left hemiparesis (Manual Muscle Test [MMT] [4]; left upper and lower limbs: 0 (no contraction of muscle) -1 (only contraction of muscle)). The Japan Coma Scale (JCS) indicated a consciousness level of 30, with her eyes opening on repeated calls with painful stimulation. On routine hematological and blood chemical tests, the white cell count was 8,100 per mm³, platelet count was 230,000 per mm³, hemoglobin was 11.9 g/dL, activated partial-thromboplastin time was 22.2 sec, prothrombin

activity was 106%, sodium was 139 mmol/L, potassium was 4.4 mmol/L, urea nitrogen was 28.6 mg/L, creatinine was 0.95 mg/dL, aspartate aminotransferase was 20 U/L, alanine aminotransferase was 14 U/L, total cholesterol was 208 mg/dL, C-reactive protein was 0.2 mg/L, and electrocardiographic findings were normal. Computed tomography (CT) (Fig. 1) indicated only a single lacunar infarct at the posterior limb of the right internal capsule without brain atrophy.

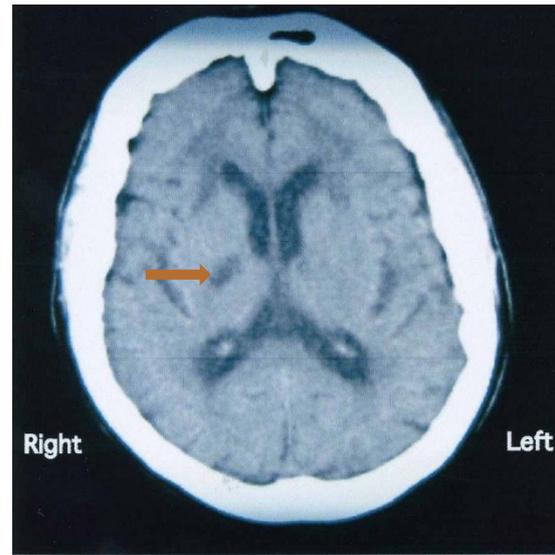


Figure 1. CT of the brain. A CT image obtained on the fourth hospital day showed a right lacunar infarct at the posterior limb of the internal capsule (arrow).

However, N-isopropyl-P-¹²³I-iodoamphetamine single photon emission CT (123I-IMP SPECT) analysis (Fig. 2) showed that cerebral blood flow (CBF) was not reduced in any brain area. Furthermore, there was no difference in CBF between the left and right in any brain area.

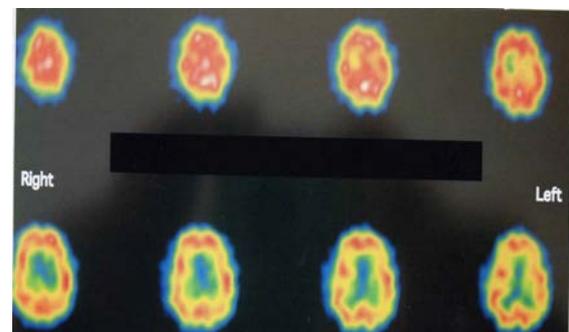


Figure 2. ¹²³I-IMP SPECT scan of the brain. ¹²³I-IMP SPECT images obtained on the seventh hospital day did not show any abnormality.

Magnetic resonance imaging (MRI) (Fig.1C) at 1.5 Tesla indicated periventricular

hyperintensity (PVH) which showed a grade-3 PVH pattern using Fazekas' method [5,6], although MRI did not indicate lacunar infarct because the patient underwent MRI studies directly after the infarct had developed. Magnetic resonance angiography (MRA) showed stenosis of the middle cerebral artery (MCA).

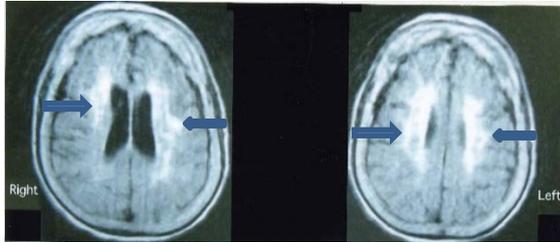


Figure 3. MR flair images. MR flair images showed severe PVH (grade 3 using Fazekas' method), obtained directly after the patient's admission (arrows).

Paralysis continued to worsen after admission. The MMT score of the left upper and lower limbs fell to 0, and thereafter remained at that level. Furthermore, she could not move her right lower limb because of disuse atrophy and severe lumbar pain due to osteoarthritis. Therefore, she could not sit upright on a bed. During the first month, the patient's consciousness ranged from 3 on the JCS (she opened her eyes, but she could not state her name or birthday) to 10 on the JCS (she opened her eyes when called). In addition, the score on the Mini-Mental State Examination (MMSE) [7] continued to be "0". During the fifth month, her consciousness remained at 3 on the JCS, and then she began to respond with a simple greeting and she talked in a simple way to those around her, for example "I am hungry" and "Is it bath time?", but could not respond to any other questions. The MMSE score remained at 0 until discharge. During admission, she often had a fever due to chronic urinary tract infection, but the symptoms had improved at the time of discharge. She was transferred to a rehabilitation hospital six months after admission. The memory of this patient continued to show severe disturbance as the MMSE score was "0" points.

DISCUSSION

This patient could live independently prior to admission, but she was very elderly (85 years old), and head MRI indicated severe PVH. This patient suddenly showed a disturbance of consciousness immediately after developing her first lacunar infarct at the posterior limb of the right internal capsule, and eventually demonstrated severe cognitive disorder. It has been reported that severe cognitive disorder could result from stroke and neural degeneration. However, it has not been reported that the first

lacunar infarct, as in this case, could lead to the sudden onset of severe cognitive disorder.

In this patient, SPECT analysis of her brain did not indicate a decrease in cerebral blood flow and did not show a remote effect of the infarct, although MRA of her brain indicated MCA stenosis.

However, this patient showed severe PVH (grade 3 using Fazekas' method). PVH could be induced by a hypoxic environment in the brain [8]. White matter lesions such as PVH are associated with an increased risk of dementia [9]. Therefore, her brain functions showed subclinical deterioration caused by PVH. This patient showed deterioration of the consciousness level (100 on the JCS) directly after right lacunar infarct. Her conscious level improved, but her cognitive function continued to show severe disorder, and she could not respond to simple questions (where are we? What day is today?), as the data of MMSE showed. In this patient, a lacunar infarct of the right internal capsule induced severe symptoms, but a similar finding has not been reported.

The area where this patient suffered the infarct passed through the ascending and descending fibers composed of the projecting fibers connecting the cerebral cortex with the basal ganglia, brain stem, and cerebellum. Therefore, the reason for the severe paralysis of the patient's left upper and lower limbs is damage caused by the lesion of one of the descending fibers; in other words, the pyramidal tract (corticospinal tract). In addition, because the patient also had consciousness disorder as well as limb paralysis, it is a strong possibility that the lesion had spread to the circumference of the pyramidal tract, that is, the area of the ascending fibers.

However, the pyramidal tract cannot affect the cognitive function, because it is a pathway for motor neurons. Therefore, in this patient, we suggest that the damage of the projecting fibers from the brain stem, ascending fibers, caused deterioration of the consciousness and cognitive function.

In this case, the pathway damaged by the lacunar infarct may be associated with the ascending reticular activating system (ARAS) [10]. ARAS is a complicated network controlling arousal, projecting to the brain cortex and bypassing the thalamus from the upper brain stem or basal forebrain [10].

Impairment of consciousness similar to this case after lacunar infarct in these networks has not been reported. However, this patient was advanced in age (85 years old), and so we are of the opinion that her brain plasticity was very low level. In addition, her brain showed severe PVL on MRI. These findings suggest that there may have been few fibers projecting from the brain stem or basal forebrain to the cortex. Thus, her spare brain capacity may have been very limited. In addition, the patient developed severe symptoms due to this lacunar infarct. It is known that lacunar infarcts

cause mild symptoms, but this case adds new findings regarding such infarcts.

CONCLUSIONS

Based on these inferences, we conclude that the fibers of the internal capsule may provide a clue to the consciousness and cognitive function of the advanced elderly with severe PVH. Following lacunar stroke, the advanced elderly with severe white matter lesions require particular attention.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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