Case report

Complete recovery after spontaneous thrombolysis of a middle cerebral artery embolism: An imaging report

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Abstract

A 53-year-old-man demonstrated an embolism of the left middle cerebral artery, a rapid spontaneous thrombolysis and a subsequent recovery of the cerebral perfusion. These events were documented using MR angiography, perfusion-weighted MR imaging and digital subtraction angiography. The patient recovered consciousness and his symptoms of right hemiparesis and aphasia disappeared completely, representing the so-called spectacular shrinking deficit. This case provides evidence that an early spontaneous thrombolysis may recover cerebral perfusion and alleviate the symptoms of the patient.

Keywords: Spontaneous thrombolysis; Spectacular shrinking deficit; Perfusion MRI

1. Introduction

Rapid recovery of profound hemispheric symptoms within a short-term after cerebral embolism is called spectacular shrinking deficit (SSD) \cite{1}. SSD is postulated to result from rapid embolic lysis, fragmentation, and migration along the internal carotid/middle cerebral axis, leading to restored tissue perfusion before irreversible tissue damage has occurred \cite{2}. In this report, imaging of the rapid spontaneous thrombolysis of a middle cerebral artery (MCA) embolism is presented in a patient who showed SSD. The recovery of the abnormal cerebral perfusion detected using the perfusion-weighted MR imaging (PWI) is presented in conjunction with the complete recovery of the symptoms.

2. Case report

A 53-year-old-man presented to our hospital with an acute-onset of right hemiparesis, aphasia, and mild disturbance of consciousness. His symptoms began suddenly, when he stood up for walking at home in the evening. He did not complain of any major headache or neck pain and had no nausea or vomiting. The patient had a history of smoking 40 cigarettes per day and drinking 1.5 L of beer per day for 30 years. He had no history of recreational drug use. The patient had a 10-min episode of transient aphasia 6 months ago. His family history was negative for stroke, neurologic disorders, and vascular risk factors. He was diagnosed with hyperlipidemia 6 years ago. The patient arrived at our hospital from a private hospital at 2 h after the onset of the symptoms. His pulse was 90 beats/min and regular, and his blood pressure was 132/80 mmHg. Body habitus was unremarkable. No cardiac bruits or cardiac murmurs were audible. The patient was drowsy and kept his eyes closed. Once aroused, he could respond to a simple order, but not to a complex order. He demonstrated jargon aphasia. Left–right disorientation and asomatognosia were noted. There was a mild motor paresis on the right side.
Increased deep tendon reflex and Babinski reflex were noted on the right side. The modified Rankin scale was Grade 5. The results of routine blood work were unremarkable, except an increase of anticardiolipin antibody (29 IU/mL) and plasmin and α2-plasmin inhibitor complex (1.7 μg/mL). Holter electrocardiogram and echocardiogram were unremarkable.

CT of the head obtained at 50 min after the onset of symptoms in the private hospital demonstrated a slightly low attenuation in the posterior portion of the left lenticular nucleus, representing an early CT sign of the ischemia. No hemorrhage was observed. A 1.5 T superconducting MR unit (Magnetom Vision, Siemens, Erlangen, Germany) with a standard head coil was used for imaging in our hospital. Single-shot echo-planar spin-echo diffusion-weighted MR imaging (DWI) (echo time/flip angle = 137 ms/90°, b value = 1000 mm²/s) obtained at 2.5 h after the onset of symptoms revealed small areas of abnormal high signal intensity in the posterior portion of the left lenticular nucleus, insula, and cerebral cortex (Fig. 1A). An apparent diffusion coefficient (ADC) map demonstrated a decreased ADC in the posterior portion of the left lenticular nucleus. Conventional MR imaging was unremarkable. MR angiography (repetition time/echo time/flip angle = 32/6.9 ms/20°) did not reveal the left MCA trunk, suggesting that an occlusion existed (Fig. 1B). Single-shot echo-planar gradient-echo PWI (echo time/flip angle = 54 ms/90°) obtained after a bolus injection of gadopentetate dimeglumine of 0.05 mmol/kg of body weight and 20 mL of saline at a rate of 2 mL/s was performed, as previously reported [3]. It revealed a slight decrease in the relative cerebral blood flow (CBF), no apparent change in the relative cerebral blood volume and a prolonged mean transit time (MTT) in the region supplied by the left MCA (Fig. 1C). The lesion-to-contralateral CBF ratio was 0.75. It was recommended to the patient and his family to proceed with cerebral angiography and intraarterial thrombolysis, if technically amenable. The procedure, goals, rationale, treatment alternatives, and risk of angiography and intraarterial thrombolysis were thoroughly discussed with his family. Using a Seldinger technique, a 5.5 F arterial sheath was placed in the right femoral artery and 3000 units of heparin were injected through the sheath as a routine procedure. A 5 F catheter was advanced into the left common carotid artery. Digital subtraction angiography of the right carotid bifurcation and intracranial circulation was obtained using nonionic contrast material at 3 h after the onset of symptoms. Although a stenosis of the left internal carotid artery (ICA) of approximately 50% was observed, no occlusion or stenosis of the left MCA trunk was observed.

![Fig. 1](image_url) (A) Diffusion-weighted MR imaging demonstrates high signal intensity areas in the posterior portion of the left lenticular nucleus and insula. (B) MR angiography does not reveal the left middle cerebral artery (MCA) trunk, which suggests an occlusion. (C) Perfusion-weighted MR imaging demonstrates a prolonged mean transit time (MTT) in the left MCA perfusion territory. The brighter signal represents a prolonged MTT. The MTT of the right MCA perfusion territory and left MCA perfusion territory were 6.6 and 7.4 s, respectively. (D) Left common carotid angiography was performed. No occlusion or stenosis of the left MCA trunk is observed, suggesting spontaneous thrombolysis.
The peripheral branches of the left MCA were slightly sparse, especially in the region of the central artery. These observations supported a diagnosis of a spontaneous thrombolysis and the peripheral migration of the thrombus. The consciousness and the symptoms of the patient recovered rapidly on the angiographic table. Thrombolysis therapy was not performed. Drop infusion of the heparinized saline was continued for 7 days. Subsequently, an aspirin tablet (81 mg) per day was administered.

The MR angiography obtained on the 13th day demonstrated a completely patent left MCA trunk (Fig. 2A). The DWI and fluid-attenuated inversion recovery MR imaging (repetition time/echo time = 9000/110 ms) demonstrated small high signal intensity areas, which represented infarcts, in the posterior portion of the left lenticular nucleus and temporal cortex (Fig. 2B). No bleeding was observed. The lesion-to-contralateral CBF ratio recovered to 0.92. The patient was discharged on the 29th day without any neurologic deficit.

3. Discussion

SSD occurs in 4.8–12% of cerebral embolisms [1,4], and in cardiogenic cerebral embolisms in general [1]. In this report, imaging of a spontaneous thrombolysis of an MCA embolism was presented. Although Darby et al. [5] demonstrated a similar recovery of a PWI abnormality and symptoms after SSD in one patient, the evidence of an MCA occlusion and its recanalization has not been documented. Baird et al. [2] compared the percentage of the cerebral reperfusion territory between patients who developed SSD and those who did not, using single-photon emission computed tomography (SPECT). Patients who developed SSD demonstrated a significantly greater territory of reperfusion (88.6%), when compared with those who did not demonstrate reperfusion (17.6%) during the first 48 h. Shimosegawa et al. [6] reported that a morphological viable brain can be distinguished using SPECT by a lesion-to-contralateral radioactivity ratio of 0.6 within 6 h after the onset of cerebral infarction. In the present case, the lesion-to-contralateral CBF ratio was 0.75 and the region of prolonged MTT almost disappeared completely. Therefore, our results are in good agreement with those of previous studies. The merits of evaluating cerebral perfusion using PWI may be that PWI can be performed during MR imaging and MR angiography, and that PWI can be performed quickly, such as in 1 min [3].

The case presented here demonstrates that the disappearance of abnormal perfusion can be obtained by a rapid spontaneous thrombolysis. Although an absent MCA on MR angiography indicates a poor stroke outcome in the MCA perfusion territory in general [7], a rapid spontaneous
thrombolysis can reverse this outcome. Although successful intraarterial thrombolysis therapy provides a similar tissue reperfusion and recovery of the symptoms [8,9], the possibility of a concomitant spontaneous thrombolysis may exist in some cases. The clinical category was determined to be an atherothrombotic brain infarction in the present case. The mechanism was thought to be an artery-to-artery embolism that originated from an atherosclerotic plaque in the carotid bifurcation. Although SSD occurs in cardiogenic cerebral embolisms in general [1], this case suggests the possibility that some artery-to-artery emboli spontaneously dissolve.

4. Conclusion

A case of a left MCA embolism and its rapid spontaneous lysis was presented. This case provides evidence that an early spontaneous thrombolysis recovers the cerebral perfusion and the symptoms of the patient.

References


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