

# Late Spontaneous Recanalization of Chronic Middle Cerebral Artery Occlusion

Cheol-Young Lee, MD<sup>1,3</sup>, Chang-Woo Ryu, MD<sup>2</sup>, Jun-Seok Koh, MD<sup>1</sup>, Gook Ki Kim, MD, PhD<sup>1</sup>

Early spontaneous recanalization of the middle cerebral artery in acute ischemic phase artery is not uncommon, whereas the late spontaneous recanalization of chronic occluded artery is a very rare phenomenon and exact incidence and the timing of this event have not been quantified. We present a case in which late spontaneous recanalization of long-lasting middle cerebral artery occlusion occurred in the absence of surgical, endovascular and thrombolytic treatments.

**Key Words** : Arteriosclerosis; Cerebrovascular disease; Angiography

Acute occlusion of middle cerebral artery (MCA) has been associated with high morbidity and mortality, compromising cerebral blood flow to the frontal and temporal lobes and the basal ganglia [1, 2]. However, chronic occlusion of MCA is sometimes identified incidentally by magnetic resonance (MR) angiography in asymptomatic patients or can present with ischemic stroke resulting from hemodynamic compromise. In chronic occlusion with enough collateral circulation, conservative methods including continued heparin and/or anti-platelet therapies can be used. Early spontaneous recanalization after acute embolic occlusion is a common phenomenon in the intracranial circulation. However, most observers agree that late spontaneous

recanalization of the long-standing occlusion of intracranial artery is virtually impossible [1, 3]. To our knowledge, late spontaneous recanalization of occluded artery has been documented only in the extracranial circulation. We report a case in which late spontaneous recanalization of chronic MCA occlusion occurred in the absence of surgical, endovascular and thrombolytic treatments. We found a limited documented report of late spontaneous recanalization of the posterior cerebral circulation [4]. To the best of our knowledge, this is the first published report for late spontaneous recanalization of the completely occluded MCA in chronic state.

## CASE REPORT

A 53-year-old man presented with transient acute onset of left-sided numbness and speech disturbance one week prior to admission. These symptoms lasted for only several minutes and spontaneously resolved. Transient ischemic attack or acute cerebral infarction were possible diagnoses, so MRI and MR angiography were performed, showing an occlusion of his right MCA at the proximal M1 segment without any acute ischemic lesion or infarcted area on MRI, including diffusion-weighted images. The next day, a conventional angiogram was taken and revealed a tapered occlusion at proximal M1 segment of right MCA, collateral pathways through perforating arteries, rich

<sup>1</sup>Department of Neurosurgery & <sup>2</sup>Radiology, Kyung Hee University Hospital at Gangdong, Seoul, Korea

<sup>3</sup>Department of Neurosurgery, Konyang University Hospital, Daejeon, Korea

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**Correspondence to:** Cheol-Young Lee, MD, Department of Neurosurgery, Konyang University Hospital, 685, Gasooon-dong, Seo-gu, Daejeon 302-718, Korea.

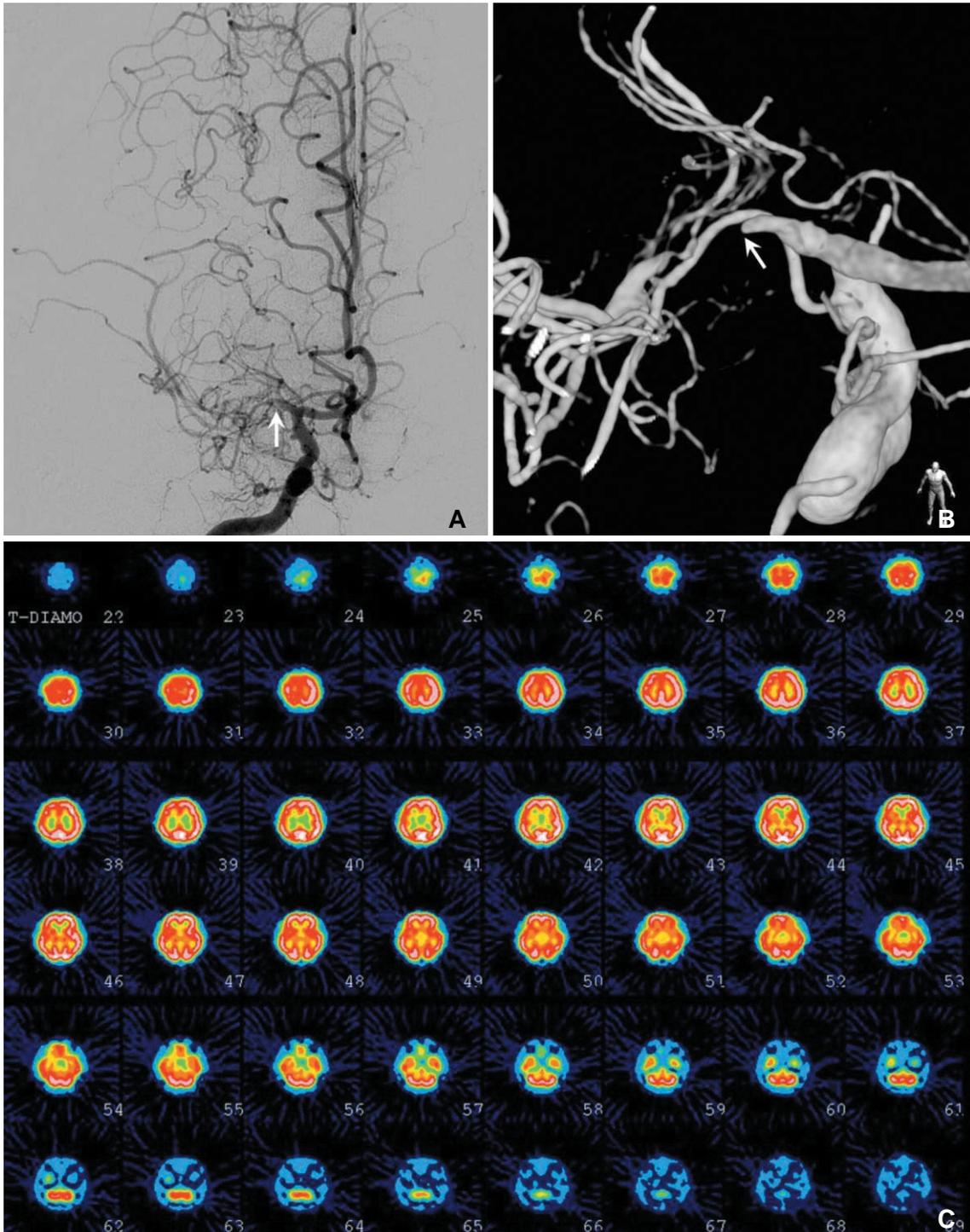
Tel. 82.42.600.9130 Fax. 82.42.600.9500

E-mail: leecy009@hanmail.net

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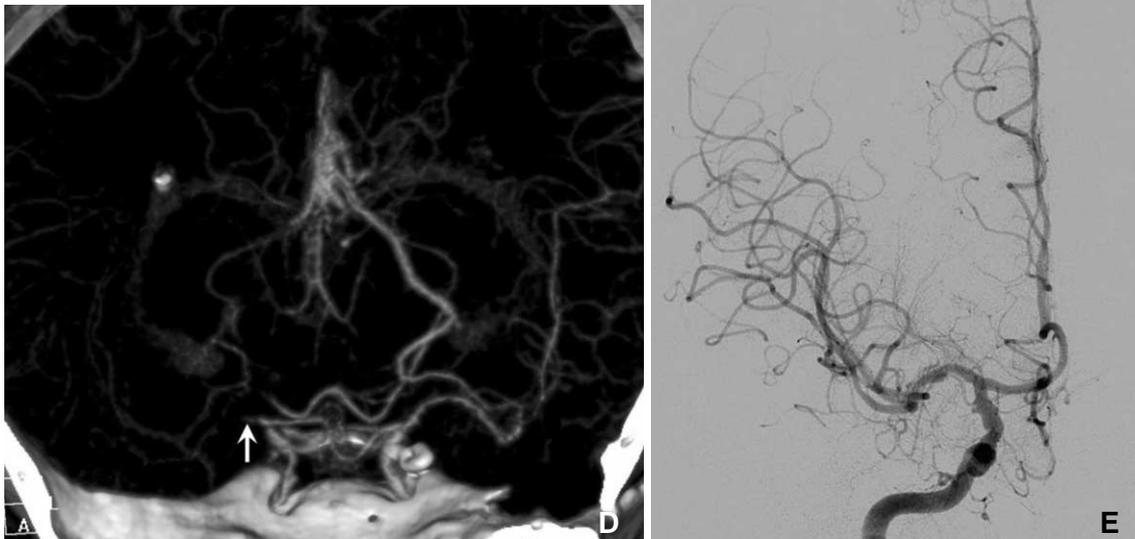
pial collateralization from distal anterior cerebral artery, and a shift of the watershed zone (Fig. 1A, B). These findings were compatible with the long-standing stage of the occlusion of MCA rather than the acute

occlusion. He also has an incidental saccular aneurysm at left posterior communicating artery (P-com) origin. The brain single photon emission computed tomography (SPECT) showed mildly decreased perfusion in



**Fig. 1. A.** An Initial right carotid angiogram shows the occlusion of the proximal trunk of right middle cerebral artery (MCA) (arrow). The pial collaterals from anterior cerebral artery and the shift of the watershed zone are noted. **B.** Three-dimensional rotation angiography clearly reveals a tapered occlusion of proximal segment of M1 (arrow). Distal segment from occlusion and lenticulostriate arteries are filled by contrast media through basal collaterals. **C.** Diamox SPECT shows mild decrease of the reserve capacity at right MCA territory

Continued



**Fig. 1. D.** CT angiography that is performed two months later shows persistent occlusion of right MCA (arrow). **E.** Twenty months later, right carotid angiography shows the recanalization of previously occluded segment of right MCA.

right middle cerebral artery territory and the vascular reserve was also decreased mildly after acetazolamide injection (Fig. 1C). An echocardiogram and myocardial SPECT excluded cardiac embolus as the etiology of the occlusion. Although the patient had no past medical history, untreated hypertension and diabetic mellitus were found during evaluation. In this setting of presumably chronic occlusion, surgical, endovascular and thrombolytic therapies would cause serious complications. Therefore conservative managements were selected for this patient. The patient was initially treated with antiplatelet and circulating drug and carefully observed for two weeks. He had no further neurologic symptoms.

After two months, the left P-com aneurysm was managed by the microsurgical aneurysmal neck clipping and there were no developing or new neurologic symptoms after surgery. Immediate post-surgical computed tomography angiogram (CTA) showed persistent right MCA occlusion and complete aneurysm clipping (Fig. 1D). The medications were continued during the follow-up period. The brain CTA of 21 months after neck clipping developed recanalization of the previously occluded right MCA. A subsequent conventional angiogram confirmed nearly complete patency of the right MCA with focal mild stenosis, normal blood flow through MCA, and the normalization of the shift of watershed zone (Fig. 1E). Our patient experienced late spontaneous recanaliza-

tion and restoration of blood flow by an unknown mechanism within 21 months, without any neurologic deficit.

## DISCUSSION

Spontaneous recanalization of the occluded MCA has been a common finding in acute ischemic stroke. Several studies demonstrated that most recanalization may occur in the acute phase of stroke, within approximately 48 hours after onset [2, 5]. A recent meta-analysis of thrombolytic therapy attempted to quantify spontaneous recanalization in ischemic stroke. Spontaneous recanalization occurred in 21.4% of patients within 24 hours and in 52.7% of patients by a week [6]. Although the analysis of these data had limitations, including the variety of times examined, but the suggestion is that the natural history of cerebral embolus is dissolution and spontaneous recanalization over a period of time. Like these studies, spontaneous recanalization of MCA, in which occlusion is apparent in the acute phase have occasionally occurred in the subacute phase of stroke. But late recanalization in the chronic phase has not been reported to the best of our knowledge.

Unlike recanalization of occluded artery in acute stroke, the spontaneous recanalization of a long-standing occlusion of extracranial artery has been only anecdotally reported [7–9]. The mechanism by which

recanalization of chronically occluded carotid arteries occurs is still little known. Lammie et al. [7], in 1999, suggested the possibility of occlusions resulting from ulcerated plaque thrombosis presenting long-term recanalization by thrombolysis. Colon et al. [8], in 1999, published a series of four cases of spontaneous recanalization of the internal carotid artery, which through imaging examinations and intraoperative finding, proved to be a hypertrophy of vasa vasorum, causing reperfusion of the distal to the occlusion. Another possible mechanism is that, in the case of myointimal hyperplasia or atherosclerotic disease, the neovascularization is induced, which in the long term can allow perfusion distal to vessel occlusion. Persistence of some embryonic vessels can also account for the complete non-occlusion of the whole internal carotid artery segment, allowing action of varied mechanisms for vessel recanalization. Nowadays some groups have been following patients with carotid occlusions, with the aim of better evaluating the natural history of such lesions. Verlato et al. [10], in 2000, published a cohort study including 41 patients with carotid occlusion. The mean follow up periods were 44.5 months. In one case with asymptomatic carotid occlusion of 41 patients, spontaneous recanalization was identified at three years after the diagnosis, remaining without symptoms for the whole period.

The mechanism of late recanalization of chronic occlusions of MCA could be one as remarked above or not. Unlike the extracranial carotid artery, the intradural cerebral artery has a little chance to the hypertrophy of vasa vasorum or the revascularization within a plaque due to the nature of intracranial artery, including the lack of vasa vasorum and the smaller caliber. In our case, we cautiously speculate that the recanalization process could be that the repeated embolic formation and accumulation were progressed to complete obstruction for some period of time in focal thrombotic area. After antiplatelet therapy, the thrombolysis occurred gradually instead of repeated embolic

accumulation. Finally, spontaneous recanalization occurred at this late stage.

The angiographic changes in our patient illustrate that the chronic MCA occlusion were spontaneously recanalized by an unknown mechanism within 21 months, with a good clinical outcome. The incidence, mechanism, and ideal management of this extraordinary finding remain unclear. Through this rare case, further investigation and studies into the underlying mechanism of spontaneous recanalization should be performed.

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