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Acute middle cerebral artery occlusion caused by spontaneous thrombosis of a small internal carotid artery aneurysm: illustrative case

Ryunosuke Yoshihara, MD, Koichiro Shindo, MD, Tatsuya Ogino, MD, and Hirohiko Nakamura, MD, PhD

Department of Neurosurgery, Nakamura Memorial Hospital, Sapporo, Hokkaido, Japan

BACKGROUND Spontaneous thrombosis of a saccular, unruptured, intracranial aneurysm is rare in nongiant aneurysms. Herein, the authors present a case of acute middle cerebral artery occlusion (MCO) caused by spontaneous thrombus of a small internal carotid artery (ICA) aneurysm.

OBSERVATIONS A 68-year-old woman presented with increased somnolence, right-sided hemiplegia, hemispatial neglect, and total aphagia. Left MCO and a small left ICA aneurysm were suspected based on magnetic resonance angiography (MRA). The authors detected early ischemic lesions from diffusion-weighted imaging (DWI). The DWI–Alberta Stroke Program Early Computed Tomography Score was 6. T2*-weighted imaging (T2*WI) showed a thrombus, the so-called susceptibility vessel sign, at the left MCO site. Another suspected thrombus was also found in the ipsilateral ICA aneurysm. The authors treated acute phase MCO with mechanical thrombectomy (MT), after which secondary stroke prophylaxis consisting of warfarin potassium was started. Since follow-up T2*WI showed the thrombus had disappeared from the left ICA aneurysm and the whole aneurysm was clarified by MRA, coil embolization was performed. After coil embolization, there was no ischemic recurrence.

LESSONS Aneurysms are infrequently found proximal to occlusion sites during MT. If the proximal aneurysm is a potential embolic source, treatment of the said aneurysm may prevent stroke.

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KEYWORDS mechanical thrombectomy; spontaneous aneurysmal thrombosis; small cerebral aneurysm; ICA aneurysm; MCA occlusion

Based on several recent landmark randomized trials, mechanical thrombectomy (MT) of acute ischemic stroke has become a standard of care and has received the highest recommendation—class I, level of evidence A—by the American Heart Association/American Stroke Association (AHA/ASA).[1] As a result, the use of MT has dramatically increased over the past several years. During MT, an aneurysm may be detected by chance. Unruptured, unrecognized aneurysms have a 3% incidence rate in the general population, with similar risk factors as ischemic stroke.[2] These aneurysms have the risk of iatrogenic rupture and other potentially catastrophic complications. There were a few reports of complete thrombosis, of bifurcation type unruptured aneurysm, that caused the occlusion of the parent artery resulting in ischemic stroke of its entire vascular territory. We, herein, present a case of acute middle cerebral artery (MCA) occlusion caused by spontaneous thrombosis of a small internal carotid artery (ICA) aneurysm.

Illustrative Case

A 68-year-old female presented with increased somnolence, right-sided hemiplegia, facial droop, hemispatial neglect, and total aphagia: all other vital signs were normal. The initial score per the National Institutes of Health Stroke Scale (NIHSS) score was 32. She had fallen at the time of onset and had a contusion on her head, which was sutured at the emergency room.

She had a medical history of hypertension, hyperlipidemia, and angina pectoris. Nine days before onset, she had undergone percutaneous coronary intervention for angina pectoris. She continued

ABBREVIATIONS AHA = American Heart Association; ASA = American Stroke Association; CFD = computational fluid dynamics; DAPT = dual antiplatelet therapy; DWI = diffusion-weighted imaging; DWI-ASPECTS = Diffusion-Weighted Imaging–Alberta Stroke Program Early Computed Tomography Score; ICA = internal carotid artery; MCA = middle cerebral artery; MCO = middle cerebral artery occlusion; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging; MT = mechanical thrombectomy; NIHSS = National Institutes of Health Stroke Scale; PCom = posterior communicating artery; T2*WI = T2*-weighted imaging.

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dual antiplatelet therapy (DAPT): clopidogrel sulfate 75 mg/d by mouth along with aspirin 100 mg/d by mouth. The postoperative course was uneventful. She did not smoke or drink and had an insignificant family medical history, and electrocardiography showed no evidence of atrial fibrillation.

Left M1 distal occlusion and small left ICA aneurysm were suspected based on MRA (Fig. 1A), performed 69 minutes after the onset of these symptoms. We detected early ischemic lesions with a Diffusion-Weighted Imaging–Alberta Stroke Program Early Computed Tomography Score (DWI-ASPECTS) of 6 (Fig. 1B). T2*-weighted imaging (T2*WI) showed a thrombus, the so-called susceptibility vessel sign, at the left MCA occlusion site (Fig. 1C). A similar lesion at the ipsilateral ICA aneurysm indicated a thrombus (Fig. 1D). Because of the head contusion, she was immediately transferred to the neuroangiography suite for attempted MCA clot removal without intravenous administration of recombinant tissue plasminogen activator. Conventional angiography revealed left M1 distal occlusion and small left ICA-posterior communicating artery (PCom) aneurysm (Fig. 2A). We performed MT of the MCA occlusion. The microcatheter passed through the occlusion site, and the contrast injection showed its location to be adequately distal to the occlusion site. The stent retriever (Solitaire Platinum 4 $\times$ 20 mm, Medtronic) was deployed in the M2 segment to cover the thrombus (Fig. 2B). Recanalization of the MCA was achieved on the first attempt resulting in a thrombolysis in cerebral infarction score of 3 (Fig. 2C). Onset-to-treatment (recanalization) time was 122 minutes. The patient was started on secondary stroke prophylaxis consisting of warfarin potassium 2 mg/d by mouth with DAPT after MT, as our first impression of etiology was cardiogenic embolization. However, no cardiac embolic sources were revealed by transesophageal echocardiography as well as Holter monitoring, and the results of blood examination, including coagulation tests, were normal. Based on the Trial of ORG 10172 in Acute Stroke Treatment classification, we judged the possibility of paradoxical embolism and cardiogenic embolism as an etiology for MCA occlusion to be low.

Follow-up T2*WI showed that the thrombus had disappeared from the left ICA aneurysm (Fig. 3A–C). Concurrent with the T2*WI findings, morphological changes in the aneurysm were observed on MRA. The aneurysm dome appeared as if it had grown and changed to a club-like shape (Fig. 3D–F). It was thought that the change in shape was caused by the disappearance of the thrombus in the aneurysm. This change was completed on the 10th day, after which the shape remained stable.

The NIHSS, after 3 months from the onset, showed improvement, dropping from 32 to 11 since the initial assessment. We planned coil embolization of the ICA aneurysm to prevent rupture of the aneurysm and recurrence of cerebral infarction after recovery rehabilitation for the sequelae of cerebral infarction. The aneurysm should have been treated early because we considered the aneurysm to be the embolic source. However, the treatment was 6 months after MT because the patient and her family wanted a coil embolization after experiencing several months of posthospital life.

Follow-up angiography 6 months after MT showed an ICA-PCom aneurysm with a hypoplastic PCom, measuring 5.80 $\times$ 3.30 mm in the transverse and craniocaudal dimensions with a neck 1.85 mm wide (Fig. 4A). The calculated aspect ratio (aneurysm depth to aneurysm neck width) was 3.14. Computational fluid dynamics (CFD) showed that the wall shear stress was markedly low and that the blood flow was static at the tip of left ICA aneurysm (Fig. 4B). Further, the angiogram revealed no residual thrombosis in the ICA aneurysm. The disappearance of the thrombus was considered to be an effect of warfarin potassium.

Coil embolization of the ICA aneurysm was performed under general anesthesia, leaving a neck remnant because the hypoplastic PCom had branched from the neck of the aneurysm (Fig. 4C).

After coil embolization, we performed follow-up MRI every 6 months. Thirty-six months have passed since coil embolization with no evidence of ischemic recurrence or hemorrhagic events. Aneurysmal regrowth was also not observed.

Discussion

Observations

To the best of our knowledge, this is the first reported case of acute MCA occlusion caused by spontaneous thrombosis of a small ICA aneurysm.

A coincidental aneurysm prevalence rate of 3.7% in stroke patients with large vessel occlusion has been reported by Zibold et al.[3] In their study of 300 patients, most aneurysms were small (average diameter, 3.8 mm).

Although rare, cerebral infarction caused by spontaneous thrombosis of a small unruptured cerebral aneurysm is one of the causes for ischemic stroke. There were some reports of complete thrombosis of bifurcation-type unruptured aneurysm, known as MCA bifurcation aneurysm, causing occlusion of the parent artery resulting in ischemic stroke of its entire vascular territory.[4] However, there have

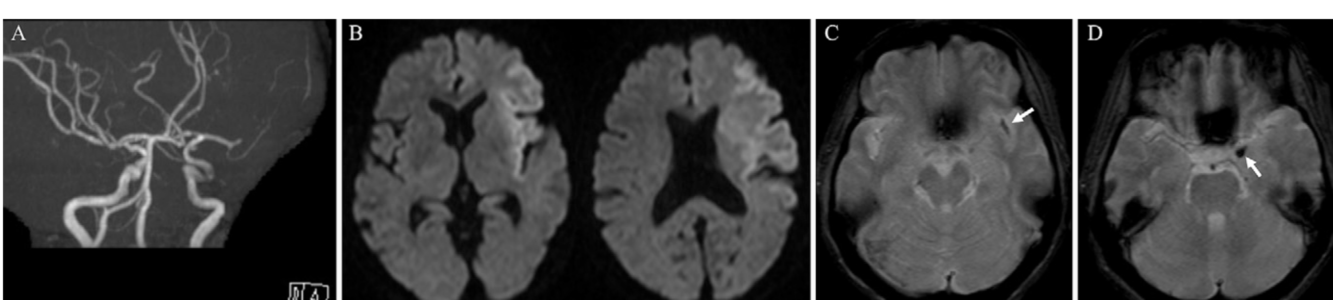

FIG. 1. Imaging findings on admission. **A:** MRA demonstrated left M1 distal occlusion and small left ICA aneurysm. **B:** DWI demonstrated early ischemic lesions; the DWI-ASPECTS was 6. **C:** T2*WI showed the presence of a low intensity signal (*white arrow*), known as the susceptibility vessel sign, in the left MCA occlusion site. **D:** T2*WI showed a similar lesion at the left ICA aneurysm (*white arrow*).

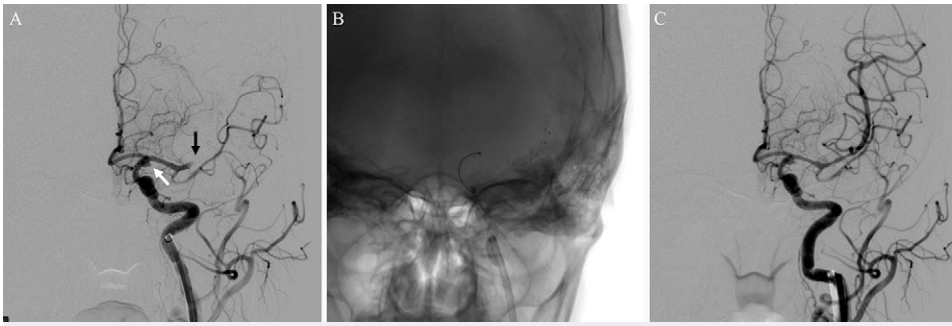

FIG. 2. Intraprocedural angiography findings of MT. **A:** Conventional angiography revealed left M1 distal occlusion (*black arrow*) and small left ICA-PCom aneurysm (*white arrow*). **B:** A stent retriever was deployed from the M2 segment proximal to the M1 segment. **C:** After a first pass with stent retriever, complete recanalization was achieved.

been no reports of a side-wall type aneurysm causing parent artery occlusion. Hence, in our case, it was proposed that a thrombus, formed in the ICA aneurysm, extended to the neck of the aneurysm and caused artery-to-artery embolism.

Calviere et al.[5] reported the association of ischemic stroke with aneurysm thrombosis in some cases and his findings suggest that aneurysm thrombosis is a dynamic process which is associated with a low rate of ischemic recurrence on antiplatelet therapy but may be followed by subarachnoid hemorrhage. Spontaneous thrombosis is probably linked to an intra-aneurysmal blood stasis and low wall share stress at the tip of the aneurysm.[6] Its hemodynamic stress on the aneurysmal wall is considered to cause an increased incidence of endothelial damage leading to aneurysm rupture.[7] Gu et al.[8] analyzed the related factors of intracranial anterior circulation saccular artery thrombosis and its characteristics using high-resolution MRI. Intracranial unruptured aneurysms with aspect ratio values higher than 2.5 indicated the possible formation of thrombosis in the aneurysm. Our case had these intra-aneurysmal conditions for thrombus formation, and the treatment for the aneurysm was helpful not only in preventing the recurrence of cerebral infarction but also the rupture of the cerebral aneurysm.

Finally, we performed MT by a simple technique of a stent retriever alone at that time. However, MT was fortunately accomplished without any problems. It is crucial to be careful not to rupture the aneurysm while performing a thrombectomy with a proximal aneurysm. We now think a combined technique is better. If a stent retriever with a diameter larger than that of the internal carotid artery is selected, the stent may protrude toward the

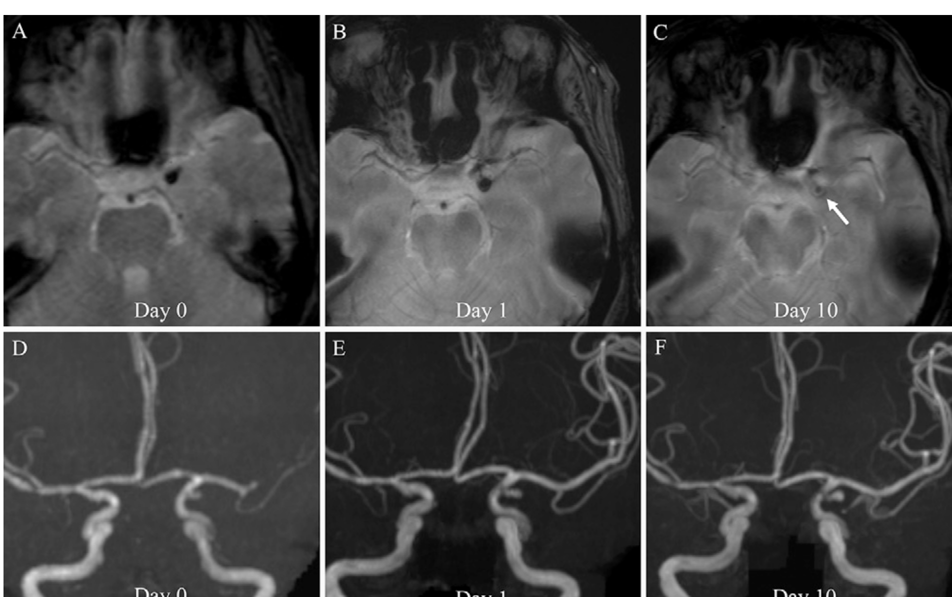

FIG. 3. Temporal changes revealed by T2*WI and MRA images. **A:** T2*WI on admission showed a small low-intensity circle (*white arrow*) adjacent to left ICA. **B and C:** Over time, the low-intensity circle (*white arrow*) shrank and disappeared. **D–F:** In parallel with the T2*WI series, MRA revealed the shape of the left ICA aneurysm.

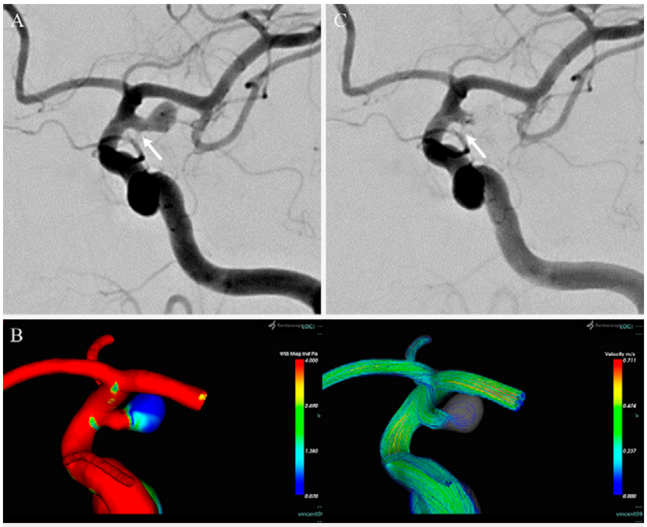

FIG. 4. Intraprocedural angiographic findings of coil embolization. **A:** A left ICA-PCom aneurysm with the calculated aspect ratio 3.14 was detected by angiography. The hypoplastic PCom had branched from the neck of the aneurysm (*white arrow*). **B:** CFD based on preprocedural angiographical data showed that the wall shear stress was markedly low and that the blood flow was static at the tip of the aneurysm. **C:** Coil embolization was completed, leaving an aneurysmal neck remnant to preserve the hypoplastic PCom.

aneurysm and damage the neck of the aneurysm. A better method would be to place the intermediate catheter beyond the neck and pull the stent retriever into the intermediate catheter to prevent unexpected aneurysm rupture.

Lessons

An aneurysm located proximal to the occluded site found during MT may act as a source of embolus.

An aneurysm with a high aspect ratio increases the likelihood of an embolic source and CFD information may help diagnose an intraaneurysmal condition which promotes thrombus formation. These aneurysms have properties similar to that of ruptured aneurysms and may require treatment to prevent rupture. If the proximal aneurysm is considered an embolic source, treatment of the aneurysm may prevent stroke. More research is needed to elucidate the effect of aneurysms on preventing cerebral embolism.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Shindo, Yoshihara, Ogino. Acquisition of data: Shindo, Yoshihara, Ogino. Analysis and interpretation of data: Shindo, Yoshihara, Ogino. Drafting the article: Shindo, Yoshihara, Ogino. Critically revising the article: Shindo, Ogino. Reviewed submitted version of manuscript: Shindo, Yoshihara, Ogino. Approved the final version of the manuscript on behalf of all authors: Shindo. Statistical analysis: Shindo, Yoshihara, Ogino. Administrative/technical/material support: Shindo, Ogino. Study supervision: all authors.

Correspondence

Koichiro Shindo: Nakamura Memorial Hospital, Sapporo, Hokkaido, Japan. k.shindo@med.nmh.or.jp.