Emergent surgical embolectomy for middle cerebral artery occlusion due to carotid plaque rupture followed by elective carotid endarterectomy

Case report

Satoshi Kiyofuji, M.D., Tomohiro Inoue, M.D., Hirotaka Hasegawa, M.D., Akira Tamura, M.D., Ph.D., and Isamu Saito, M.D., Ph.D.

Department of Neurosurgery, Fuji Brain Institute and Hospital, Fujinomiya City, Shizuoka, Japan

Embolic intracranial large artery occlusion with severe neurological deficit is associated with an extremely poor prognosis. The safest and most effective treatment strategy has not yet been determined when such emboli are associated with unstable proximal carotid plaque. The authors performed emergent surgical embolectomy for left middle cerebral artery (MCA) occlusion, and the patient experienced marked neurological recovery without focal deficit and regained premorbid activity. Postoperative investigation revealed “vulnerable plaque” of the left internal carotid artery without apparent evidence of cardiac embolism, such as would be seen with atrial fibrillation. Specimens from subsequent elective carotid endarterectomy (CEA) showed ruptured vulnerable plaque that was histologically consistent as a source of the intracranial embolic specimen. Surgical embolectomy for MCA occlusion due to carotid plaque rupture followed by CEA could be a safer and more effective alternative to endovascular treatment from the standpoint of obviating the risk of secondary embolism that could otherwise occur as a result of the manipulation of devices through an extremely unstable portion of plaque. Further, this strategy is associated with a high probability of complete recanalization with direct removal of hard and large, though fragile, emboli.

(http://thejns.org/doi/abs/10.3171/2014.4.JNS132441)

Key Words • acute ischemic stroke • intracranial embolism • carotid endarterectomy • embolectomy • internal carotid artery stenosis • vascular disorders

Although traditional direct surgical embolectomy through a craniotomy for acute ischemic stroke has been performed since the 1950s, intravenous administration of tissue plasminogen activator and endovascular methods have become recognized as faster and are now the gold-standard treatment for embolism. Most recently, endovascular treatment for acute intracranial large artery occlusion has benefitted from the introduction of new devices, such as the MERCI retriever, the Penumbra system, and the Solitaire stent. Recent endovascular embolectomy devices have been used to accomplish up to 80%–90% recanalization rates (Thrombolysis in Myocardial Infarction [TIMI] Grades 2–3), but the complete recanalization rate (TIMI Grade 3) achieved with such devices is not satisfactory, especially when the clot burden is high. By contrast, a recent report of surgical embolectomy demonstrated a high complete recanalization rate and an acceptable safety profile, especially when using MRI to select appropriate patients. Among cases of devastating acute intracranial large artery occlusion, those arising from intracranial emboli associated with carotid plaque rupture are rare. Given the possible high embolic clot burden associated with ruptured plaque and secondary engrafted thrombus, the treatment strategy for this situation remains controversial.
Furthermore, because of proximal, extremely unstable plaque, it can be quite difficult to deploy the endovascular device without generating additional emboli.

In the present report we describe a unique case of emergent direct surgical embolectomy through a craniotomy for occlusion caused by ruptured carotid plaque, followed by elective carotid endarterectomy (CEA) to prevent further stroke associated with the extremely unstable plaque.

Case Report

History and Examination. A 78-year-old woman with a history of hypertension suddenly collapsed at home and was transported by ambulance to our hospital. She was taking antihypertensive drugs only and had never been treated with any antiplatelet or anticoagulation drugs. Upon arrival, she showed right hemiplegia, global aphasia, and conjugate deviation of her eyes to the left. Her National Institutes of Health Stroke Scale (NIHSS) score was 17.

Initial CT showed abnormal high-density signal in the bifurcation of the left middle cerebral artery (MCA), suggestive of some calcified embolus, without any signs of early ischemia (Fig. 1A and B). She was taken to the MRI suite where MR angiography (MRA) demonstrated an occluded M1 on the left, while diffusion-weighted imaging (DWI) showed only minimal ischemia with spotty high-signal-intensity lesions in the left insular cortex, frontal lobe, and temporal lobe (Fig. 1C–F). Since there was a mismatch between the expected infarction area on MRA and actual lesions on DWI, as well as a mismatch between severe neurological symptoms and minimal lesions on DWI, emergent recanalization treatment was indicated. Considering the potentially highly calcified appearance of embolic material on CT that could be refractory to the pharmacological effect of intravenous tissue plasminogen activator, in addition to the fact that endovascular service is not available in emergent settings at our institution, we decided, based on our experience with high complete recanalization rates in such procedures, to proceed with emergent surgical embolectomy. After a thorough discussion with her family, we started the operation 1.5 hours after the patient’s arrival at our hospital.

First Operation. The procedure was conducted as described in detail elsewhere. In short, through a left frontotemporal craniotomy, the left MCA bifurcation was exposed. A whitish thrombus was seen from outside the MCA bifurcation (Fig. 2A). A transverse incision was made in the bifurcation, and the apparently partially calcified, hard though fragile embolus was completely removed (Fig. 2B). The incision was sutured with 9-0 nylon (Fig. 2C). Complete recanalization (TIMI Grade 3) was confirmed intraoperatively with an ultrasound Doppler flow meter and immediately postoperatively with an MRA study (Fig. 3). The recanalization time was 42 min-

![Fig. 1. Images obtained at patient’s arrival, before the embolectomy. Head CT (A) shows an abnormal high-density lesion (arrow) in the left MCA. The Hounsfield unit count of the emboli (arrow) is 164.9, while those of the sphenoid ridge (bone) and contralateral MCA are 403.2 and 26.4, respectively (B). The left MCA is occluded at M1 (C, arrow), and diffusion-weighted images (D–F) demonstrate high-intensity lesions in the insula, frontal lobe, and temporal lobe.](image-url)
Surgical embolectomy followed by carotid endarterectomy

utes from the start of surgery, 2 hours 32 minutes since
the arrival of the patient, and approximately 3 hours 30
minutes since symptom onset.

First Postoperative Course. Postoperatively, the pa-
tient showed marked neurological recovery without addi-
tional lesions on DWI. She had full restoration of motor
function, resulting in an improvement in her NIHSS score
from 17 to 5 at 3 weeks postoperatively. Although preop-
erative and intraoperative electrocardiogram (ECG) did
not show atrial fibrillation, a chest radiograph at admission
revealed left ventricular hypertrophy. Therefore, we evalu-
ated for possible cardiac embolic causes. Holter ECG and
chest ultrasonography by a cardiologist were essentially
negative, and thus there was no persuasive evidence of a
cardiogenic event. Meanwhile, neck MRI showed only
moderate carotid stenosis with a slightly high-intensity le-
sion in the left carotid plaque on magnetization-prepared
rapid acquisition of gradient echo (MPRAGE) imaging,
suggesting possible unstable plaque deposition (Fig. 4A–
D).20 We proceeded with digital subtraction angiography
(DSA), which demonstrated intermediate stenosis and an
irregularly surfaced left internal carotid artery (ICA) with
ulceration consistent with ruptured plaque (Fig. 4E). Given
these findings, we suspected that the left ICA plaque was
the cause of the M1 embolus. At that time, since the patient
had regained her premorbid level of activities of daily liv-
ing, which included ambulating and eating as usual, and
after a thorough discussion with her and her family, we
decided to proceed with elective carotid endarterectomy
(CEA) to remove the embolic origin.

Second Operation. The CEA was performed 3 weeks
after the surgical embolectomy. Video 1 features actual
procedures of the surgical embolectomy and subsequent
CEA.

Video 1. Clip demonstrates actual procedures of the surgical
embolectomy and subsequent CEA. Copyright Satoshi Kiyofuji.
Published with permission. Click here to view with Media
Player. Click here to view with Quicktime.

The plaque of the carotid artery is shown in Fig. 5.
There were significantly calcified lesions in the plaque,
which resembled the embolus in her left MCA bifurca-
tion. Pathological investigation showed similarities be-
tween the MCA embolus and the carotid plaque compo-
nent and revealed that the two specimens were almost
identical pathologically.

Second Postoperative Course. Postoperative DWI
did not show any additional ischemic lesion, and MRA
demonstrated a smooth and widely patent left carotid
artery. Antiplatelet therapy was started postoperatively
instead of anticoagulation therapy, and rehabilitation
enabled the patient to live independently. At 6 months
postoperatively, her modified Rankin Scale score was 1
without focal neurological deficit.

Discussion

Sudden and extremely severe clinical manifestations
in patients with ischemic stroke can suggest cardiac emb-
olism associated with atrial fibrillation. About 30% of
strokes are associated with atrial fibrillation, and such
strokes tend to be devastating.3 Thus, in the featured pa-
tient, we initially assumed that the removed embolus was
of cardiac origin given the sudden onset of her symptoms,
severe neurological deficits, and concomitant cardiomeg-

Fig. 2. Intraoperative views of the embolectomy. A white thrombus (A, arrow, near center of image) appeared in the MCA
bifurcation and was resected (B). Arteriotomy at the bifurcation was sutured after resection of the thrombus (C).

Fig. 3. Magnetic resonance angiogram obtained after the embolec-
tomy. Full recovery of the blood flow of the left MCA was achieved.
Therefore, anticoagulation therapy with heparin followed by warfarinization was started in response to presumed paroxysmal atrial fibrillation. However, a negative postoperative cardiac evaluation directed our attention to the cervical portion of the carotid artery as a possible embolic source, despite the fact that cervical MRA showed only low-grade carotid stenosis. Finally, imaging of the plaque using MRI in conjunction with conventional DSA revealed left low-grade carotid stenosis with “vulnerable plaque” as the cause of the embolus. It is quite rare to find that the embolus removed in a surgical embolectomy resembles the ruptured irregular surface and/or contents of the carotid plaque in CEA. Both specimens were hard, yellow, and highly calcified. The pathology report confirmed that the embolus was identical to the ICA plaque in terms of histological features, that is, a highly calcified lesion in the background of some cholesterol crystals. A recent report demonstrated a high rate of recurrent microembolic events, even in low-grade carotid stenosis, once a patient became symptomatic. Indeed, the contents of the plaque were judged as unstable, according to MRI.20 Thus, we need to consider the possibility that even low-grade carotid stenosis can cause large intracranial emboli, especially when emboli of cardiac origin are excluded.

Although there are some case reports on the endovascular removal of distal emboli associated with carotid artery stenting (CAS), direct microsurgical embolectomy of emboli originating from an ICA plaque rupture is quite rare.1,15 Given a potential high clot burden of emboli released from ruptured carotid plaque and the hard but fragile nature of the mixture of calcified plaque with secondary engrafted fibrin clot, it might be difficult to...
achieve appropriate recanalization by pharmacological thrombolysis or endovascular methods. In fact, one study demonstrated that emboli composed of platelet, atheroma, and cellular debris are more resistant than erythrocyte-rich emboli to any revascularization treatment whether it consists of a fibrinolytic agent, mechanical endovascular embolectomy, or a combination of the two. The unusually high Hounsfield unit count and accompanying calcified appearance of the emboli were quite unlikely to be those of simple erythrocyte-rich emboli. By contrast, the high complete recanalization rate and the acceptable safety profile of surgical embolectomy through a craniotomy, regardless of the embolic clot burden, have recently been described. The safety of the access to an embolic lesion should also be considered. In cases of intracranial major artery embolism coinciding with extremely unstable carotid plaque and possible rupture, endovascular removal of the emboli could involve high risk. If a catheter or guidewire passes through the plaque portion, a thrombus attached to the vulnerable plaque could migrate, causing further distal intracranial embolism. Carotid artery stenting could be tried before removal of the intracranial embolus, but CAS itself is associated with a high risk of distal embolism, especially when the plaque is fragile with ulceration or hemorrhage. Surgical embolectomy through a craniotomy could be performed to avoid the risk of irritating the vulnerable carotid plaque, while also minimizing the risk of distal emboli during embolectomy, since temporary clips are applied in distal arteries before the removal of emboli. Furthermore, in the present case, we preclamped the external carotid artery and common carotid artery to prevent microemboli during the dissection phase of the CEA, which effectively prevented even asymptomatic ischemia, as confirmed on postoperative DWI. Thus, in cases of intracranial emboli associated with the deposition of unstable carotid plaque, direct surgical embolectomy followed by CEA could have advantages over endovascular treatment from the standpoint of preventing further ischemic events during intervention. The issue of time to reperfusion should be addressed. In theory, surgical embolectomy may take longer than endovascular methods. This is because endovascular methods can be used to achieve recanalization essentially within the same procedure and immediately following diagnostic DSA, whereas surgical embolectomy requires transfer of the patient from the radiology suite to the operating room. To minimize time loss, we made an effort to transport patients directly from the MRI suite to the operating room. Standard frontotemporal craniotomy with minimal hemostasis required about 20 minutes, and in the present case we were able to accomplish recanalization in 42 minutes since the start of surgery and in 2 hours 32 minutes since the patient arrived at our hospital, which, we believe, would compare favorably with the time to recanalization associated with the endovascular method.

Disclosure
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 to the study and manuscript preparation include the following. Conception and design: Inoue. Acquisition of data: Kiyofuji. Drafting the article: Kiyofuji. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Kiyofuji. Administrative/technical/material support: Hasegawa, Tamura, Saito.

References

Supplemental online information:

Address correspondence to: Satoshi Kiyofuji, M.D., Department of Neurosurgery, Fuji Brain Institute and Hospital, 270-12 Sugita, Fujinomiya City, Shizuoka 418-0021, Japan. email: kiyofuji-tky@umin.ac.jp.