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Middle cerebral artery fusiform aneurysm presented with stroke and delayed subarachnoid hemorrhage trapping, thrombectomy, and bypass

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Abstract

Background: Ischemic stroke is a well-described but less frequent consequence of ruptured or unruptured intracranial aneurysms. To date, the optimal form of treatment for patients with a thrombosed cerebral aneurysm has not yet been well-defined.

Case Description: Here, we report a case of a 68-year-old female patient presenting with cerebral stroke. Five days poststroke multislice computed tomography (MSCT) and MSCT angiography were performed for the evaluation of clinical deterioration, showing a left M2 middle cerebral artery (MCA) bifurcation aneurysm and subarachnoid hemorrhage. Having in mind the high mortality and morbidity rates after a re-rupture, as well as the digital subtraction angiography features of the aneurysm, urgent surgery was performed consisting of aneurysm trapping and superficial temporal artery (STA) to M3 MCA segment end-to-side anastomosis. The surgery and early postoperative period proceeded uneventfully and the patient gradually recovered from the previously diagnosed expressive dysphasia and cranial and extremity motor deficit.

Conclusion: Our case describes a complex aneurysm treatment that consisted of aneurysm trapping, thrombus removal and an STA-M3 MCA branch bypass creation for the protection of the patent M3 insular MCA branch and prevention of further ischemia. This procedure rewarded us with an excellent clinical result.

Key Words: Cerebral ischemia, intracranial aneurysm, stroke

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INTRODUCTION

Ischemic stroke is a well-described but less frequent consequence of ruptured or unruptured intracranial aneurysms. Thrombus formation linked to transient ischemic attacks (TIAs) and ischemic stroke has been reported in giant and fusiform as well as in small aneurysms. The reported prevalence rates of cerebral ischemia directly associated with intracranial aneurysm vary from 0.6% to 11%.[16,20]

We report a case of a 68-year-old female patient diagnosed with a cerebral stroke. Five days poststroke native head multislice computed tomography (MSCT) and MSCT angiography (MSCTA) were performed for

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the evaluation of clinical deterioration. Studies revealed a left M2 middle cerebral artery (MCA), bifurcation aneurysm, and subarachnoid hemorrhage (SAH). Surgical treatment consisted of aneurysm trapping and superficial temporal artery (STA) to M3 MCA segment end-to-side anastomosis.

CASE REPORT

Preoperative course

This 68-year-old female patient presented with right-sided supranuclear facial palsy and right-sided extremity palsy. The symptoms were of sudden onset with no accompanying headaches or vomiting, or a history of recent trauma. The initial head MSCT showed no obvious signs of cerebral ischemia [Figure 1]. The patient was admitted to the Department of Neurology for symptomatic treatment of an ischemic stroke. Percutaneous transarterial thrombolysis was not performed because the time interval from the onset of the symptoms and hospitalization exceed 4.5 h, in addition to the fact that there were no signs of cerebral ischemia on the MSCT scan.

The patient's medical history revealed chronic diseases which included arterial hypertension, diabetes mellitus, and chronic gastritis, as well as surgical and oncological treatment for an invasive intraductal breast carcinoma.

Four days after an uneventful stay at the Department of Neurology, the patient experienced a severe headache followed by vomiting and dysphasia, as well as deterioration of consciousness, thus becoming somnolent. Urgent MSCT revealed a demarcated ischemic area in the left frontal opercular region and an SAH in the left sylvian fissure, as well as the presence of blood in the IV ventricle. MSCTA and digital subtraction angiography (DSA) revealed an aneurysm on the left second M2 MCA branch bifurcation, 5 mm from the

Figure 1: Initial native multislice computed tomography showing no obvious signs of cerebral ischemia

primary MCA bifurcation. The absence of blood flow was noticed in the "smaller" frontal opercular M3 segment, implying thrombosis. Filiform blood flow was noticed in the "larger" insular M3 segment, resembling partial vessel thrombosis. The aneurysm seemed to be fusiform, just at the second bifurcation, with full thrombosis of the frontal opercular branch. There were no radiological signs of vasospasm. By comparing the DSA and MSCT scans, it could be concluded that the fully thrombosed frontal M3 branch was the cause of the cerebral ischemia and that the second opercular M3 branch was in peril due to partial thrombosis [Figure 2].

The left P1 segment was hypoplastic, and the posterior cerebral arteries on both sides were dominantly supplied by the posterior communicating artery . The right vertebral artery showed to be hypoplastic. There were no signs of the presence of other saccular aneurysms or arteriovenous malformations.

As the shown aneurysm was favorable for surgical management, urgent surgery was indicated.

Operation

Surgery objectives

As DSA revealed a fusiform-shaped aneurysm at the left M2 MCA bifurcation on the superior M2 branch with signs of postaneurysm thrombosis, the plan was to prepare the STA for a possible bypass.

Left osteoplastic frontotemporal craniotomy with the preparation of the frontal STA branch was performed under general anesthesia and in the supine position,

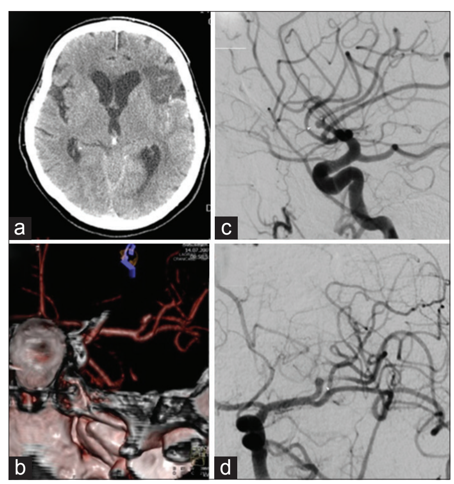

Figure 2: (a) Native multislice computed tomography showing left-side fronto-opercular cerebral ischemia and subarachnoid hemorrhage. (b) Multislice computed tomography angiography showing an aneurysm on the left M2 middle cerebral artery branch 5 mm from the M1 middle cerebral artery branch bifurcation. Digital subtraction angiography, lateral (c) and anteroposterior (d) view showing an M2 branch bifurcation aneurysm, absence of blood flow in the "smaller" frontal opercular M3 segment implying thrombosis and filiform blood flow in the "larger" insular M3 segment resembling partial vessel thrombosis

with the head rotated to the right and positioned in a Mayfield holder. After the removal of the bone, the dura was opened and suspended downward. The left sylvian fissure was widely opened and a fusiform aneurysm at the superior M2 bifurcation, including two M3 branches 5 mm from the M1 MCA segment bifurcation was intraoperatively verified. The flow in both M3 branches was checked by micro-Doppler, revealing the absence of flow and verifying a thrombosed frontal opercular M3 vessel irrigating the already ischemic part of the frontal operculum. The larger insular M3 MCA branch showed no flow on Doppler analysis, as compared to the visible filiform flow shown on the previous DSA. Bearing in mind the clearly ischemic portion of the frontal operculum irrigated by the smaller upper M3 branch, as shown by DSA and micro-Doppler, as well as the fact that the whole circumference of the vessel wall was evidently changed into a fusiform aneurysm, the permanent solution was complete M2 occlusion by placing a clip proximally to the aneurysm [Figure 3].

The postaneurysm part of the lower larger M3 MCA branch was opened with a small linear incision confirming the thrombus within it. After the thrombus evacuation and verified backflow, we decided to protect this vessel by creating an STA-MCA bypass [Figure 4].

As a preparation for the bypass procedure, the patient was given a loading dose of 300 mg of clopidogrel and 300 mg of aspirin. The STA was prepared with a fish-mouth opening and sutured with 10-0 prolene to the recipient vessel by end-to-side anastomosis [Figure 5].

After the bypass procedure, back-bleeding from the aneurysm occurred. This bleeding was successfully stopped by placing another clip distal to the aneurysm and in front of the bypass on the lower larger M3 MCA branch, thus creating a definite aneurysm trapping [Figure 6].

A second bypass or vessel occlusion by clipping was not performed on the upper smaller branch that irrigated the already ischemic part of the frontal operculum.

A micro Doppler was used to check the bypass patency, and upon patency confirmation, the wound was closed in the usual manner. A notch was created on the bone flap to protect the STA vessel.

Postoperative course

On the second postoperative day, DSA showed a patent bypass [Figure 7]. The ischemic region on the MSCT scan remained unchanged. The patency of the bypass was regularly checked by micro-Doppler. The patient gradually recovered from the previously diagnosed expressive dysphasia and cranial and extremity motor deficit.

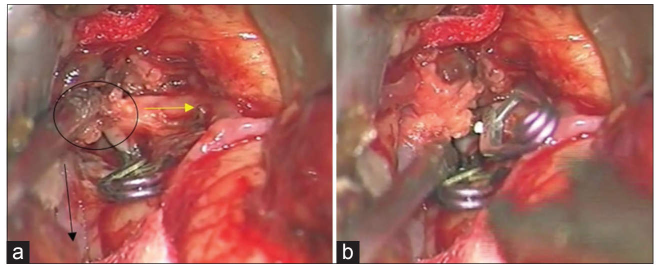

Figure 3: (a) Intraoperative picture showing fusiform aneurysm on the M2 middle cerebral artery branch, encircled. Smaller upper M3 branch is marked with a yellow arrow and lower larger M3 middle cerebral artery branch is marked with a black arrow. A temporary clip has been placed on the M2 while aneurysm preparation is performed. (b) Intraoperative picture showing definite clip placement on the M2 middle cerebral artery branch for definite occlusion

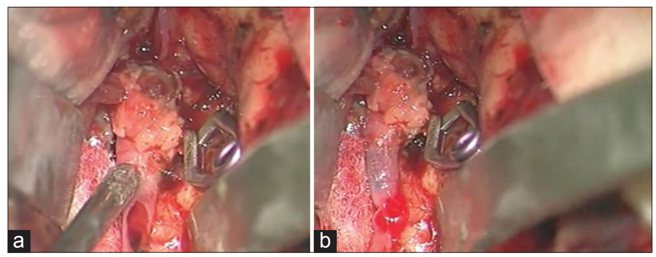

Figure 4: Intraoperative picture showing: (a) Thrombus removal from the "larger" insular M3 middle cerebral artery branch after a small linear vessel incision, (b) backflow verification after thrombus removal

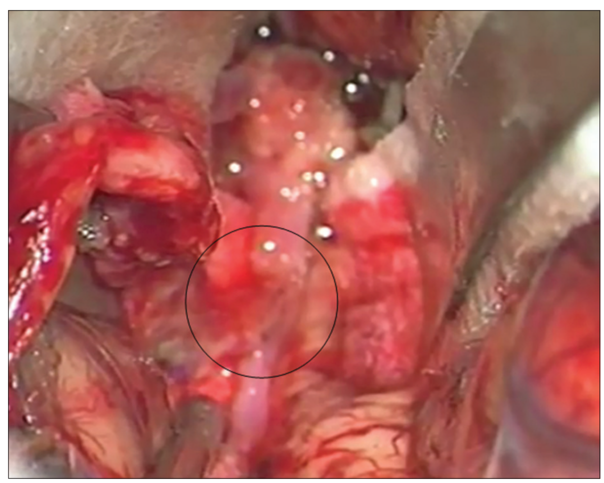

Figure 5: Intraoperative picture showing superficial temporal artery to M3 middle cerebral artery branch end-to-side anastomosis, encircled

DISCUSSION

As shown in previous studies, TIAs and cerebral infarctions are associated with intracranial aneurysms. It is clear that an aneurysm sac can act as a source of distal embolization. Reported prevalence rates of cerebral ischemia directly linked to intracranial aneurysm vary

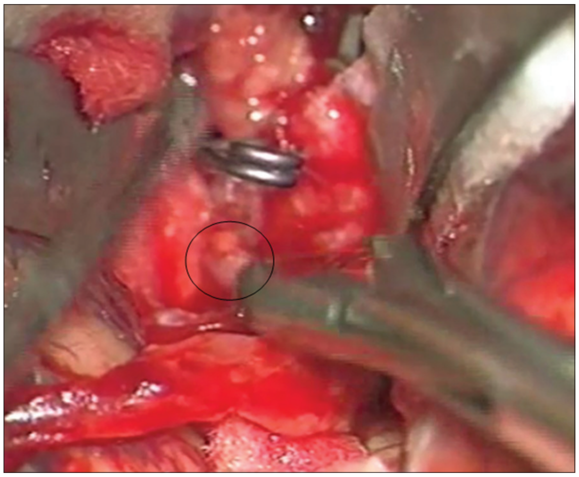

Figure 6: Intraoperative picture showing the final clip placed right before the anastomosis, encircled, thus creating aneurysm trapping

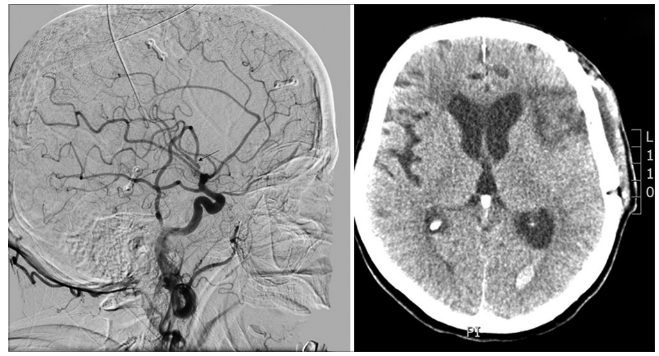

Figure 7: Postoperative digital subtraction angiography and multislice computed tomography on the day of discharge, anastomosis is marked by arrow

from 0.6% to 11%.[16,20] Thrombosis of unruptured aneurysms has been associated with the calcified atherosclerotic walls of giant saccular and fusiform aneurysms together with the combined action of turbulent and "stagnant-zone" flow of blood.[3,19] Clinical studies showed that smaller aneurysms are also linked to TIAs and ischemic strokes.[1,6,7,9] Thromboembolism from small clot-containing aneurysms has been reported to occur in 5–59% of patients.[24,25] However, TIAs or strokes from small aneurysms are not frequently reported.[7,10,17,21]

Nagashima *et al*. observed that surgical treatment of aneurysms in the presence of cerebral infarctions represented high-risk procedures, and because of the high morbidity and mortality rates in such cases they recommended a conservative approach for most of these patients.[15] Later, clinical studies have implied that surgery could provide better results by lowering the recurrent rate of cerebral ischemia though this benefit is obscured by postoperative complications.[16,20] Furthermore, as it has been reported, an intra-aneurysmal clot does not protect against aneurysm rupture,[25] and TIAs and ischemic strokes may precede SAH.[7] Thus, nonoperative treatment of unruptured aneurysms with TIA or stroke is questionable.[2,3,7,9,18] To date, the optimal form of treatment for patients with a thrombosed cerebral aneurysm has not yet been well-defined. In our case, the ischemic event followed by SAH revealed the presence of a previously unruptured aneurysm, and having in mind the high mortality and morbidity rates after re-rupture, as well as the DSA features of the aneurysm, urgent surgery was vitally indicated.

Aneurysm thromboses after SAH have been infrequently reported with an incidence of 1–2%.[4,5,8,11,14,22,23] In our case, it is possible that the thrombus formation in the lower larger insular M3 MCA branch was facilitated by the events after the SAH, but it is impossible for the thrombus formation to be exclusively linked to the aneurysm rupture, in view of the complete thrombosis in the adjacent fronto-opercular M3 MCA branch.

Today, in the advanced endovascular era, the need for intracranial bypass surgery has dramatically decreased, and it is only recommended for selected cases where endovascular intervention is not feasible. According to Kalani *et al*., the most frequent indications for intracranial bypass surgery in the modern era are complex aneurysms, moyamoya disease, and vascular occlusive disease.[13] In the previously mentioned study, complex aneurysms represent the largest indication group for bypass surgery with a favorable outcome. Bypass surgery for vascular occlusive disease represents a small indication group recommended for patients who remain refractory to maximal medical management.[12,13] Our case describes a complex aneurysm treatment that consisted of aneurysm trapping, thrombus removal, and an STA-M3 MCA branch bypass creation for the protection of the patent M3 insular MCA branch and prevention of further ischemia. This procedure rewarded us with an excellent clinical result.

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Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Antunes JL, Correll JW. Cerebral emboli from intracranial aneurysms. Surg Neurol 1976;6:7-10.
2. Atkinson JL, Lane JI, Colbassani HJ, Llewellyn DM. Spontaneous thrombosis of posterior cerebral artery aneurysm with angiographic reappearance. Case report. J Neurosurg 1993;79:434-7.
3. Brownlee RD, Tranmer BI, Sevick RJ, Karmy G, Curry BJ. Spontaneous thrombosis of an unruptured anterior communicating artery aneurysm. An unusual cause of ischemic stroke. Stroke 1995;26:1945-9.
4. Crompton MR. The pathogenesis of cerebral infarction following the rupture of cerebral berry aneurysms. Brain 1964;87:491-510.
5. Edner G, Forster DM, Steiner L, Bergvall U. Spontaneous healing of intracranial aneurysms after subarachnoid hemorrhage. Case report. J Neurosurg 1978;48:450-4.

6. Eller TW. MRI demonstration of clot in a small unruptured aneurysm causing stroke. Case report. J Neurosurg 1986;65:411-2.
7. Fisher M, Davidson RI, Marcus EM. Transient focal cerebral ischemia as a presenting manifestation of unruptured cerebral aneurysms. Ann Neurol 1980;8:367-72.
8. Fodstad H, Liliequist B. Spontaneous thrombosis of ruptured intracranial aneurysms during treatment with tranexamic acid (AMCA). Report of three cases. Acta Neurochir (Wien) 1979;49:129-44.
9. Friedman JA, Piepgras DG, Pichelmann MA, Hansen KK, Brown RD Jr., Wiebers DO. Small cerebral aneurysms presenting with symptoms other than rupture. Neurology 2001;57:1212-6.
10. Fukuoka S, Suematsu K, Nakamura J, Matsuzaki T, Satoh S, Hashimoto I. Transient ischemic attacks caused by unruptured intracranial aneurysm. Surg Neurol 1982;17:464-7.
11. Hamilton MG, Dold ON. Spontaneous disappearance of an intracranial aneurysm after subarachnoid hemorrhage. Can J Neurol Sci 1992;19:389-91.
12. Kalani MY, Ramey W, Albuquerque FC, McDougall CG, Nakaji P, Zabramski JM, *et al.* Revascularization and aneurysm surgery: Techniques, indications, and outcomes in the endovascular era. Neurosurgery 2014;74:482-97.
13. Kalani MY, Rangel-Castilla L, Ramey W, Nakaji P, Albuquerque FC, McDougall CG, *et al.* Indications and results of direct cerebral revascularization in the modern era. World Neurosurg 2015;83:345-50.
14. Lin JP. Thrombosis of aneurysm of anterior communicating artery. Case report. Acta Radiol Diagn (Stockh) 1969;8:74-80.
15. Nagashima M, Nemoto M, Hadeishi H, Suzuki A, Yasui N. Unruptured aneurysms associated with ischaemic cerebrovascular diseases. Surgical indication. Acta Neurochir (Wien) 1993;124:71-8.
16. Nanda A, Vannemreddy PS. Cerebral ischemia as a presenting feature of intracranial aneurysms: A negative prognostic indicator in the management of aneurysms. Neurosurgery 2006;58:831-7.
17. Ohno K, Suzuki R, Masaoka H, Matsushima Y, Monma S, Inaba Y. Unruptured aneurysms in patients with transient ischemic attack or reversible ischemic neurological deficit. Report of eight cases. Clin Neurol Neurosurg 1989;91:229-33.
18. Okawara SH. Warning signs prior to rupture of an intracranial aneurysm. J Neurosurg 1973;38:575-80.
19. Parenti G, Fiori L, Marconi F. Intracranical aneurysm and cerebral embolism. Eur Neurol 1992;32:212-5.
20. Qureshi AI, Mohammad Y, Yahia AM, Luft AR, Sharma M, Tamargo RJ, *et al.* Ischemic events associated with unruptured intracranial aneurysms: Multicenter clinical study and review of the literature. Neurosurgery 2000;46:282-9.
21. Sakaki T, Kinugawa K, Tanigake T, Miyamoto S, Kyoi K, Utsumi S. Embolism from intracranial aneurysms. J Neurosurg 1980;53:300-4.
22. Shah S, Murthy SB, Hannawi Y, Rao CP. The utility of cone beam volume CT in the evaluation of thrombosed intracranial aneurysms in subarachnoid hemorrhage. J Neurointerv Surg 2013;5:e51.
23. Spallone A, Peresedov VV, Kandel EI. Spontaneous cure of ruptured intracranial arterial aneurysms. Surg Neurol 1981;16:367-70.
24. Sutherland GR, King ME, Peerless SJ, Vezina WC, Brown GW, Chamberlain MJ. Platelet interaction within giant intracranial aneurysms. J Neurosurg 1982;56:53-61.
25. Whittle IR, Dorsch NW, Besser M. Spontaneous thrombosis in giant intracranial aneurysms. J Neurol Neurosurg Psychiatry 1982;45:1040-7.