Case Report

Flow alteration therapy for impending rupture of intracranial giant aneurysm after flow diverter placement

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ABSTRACT

Background: Flow diverter (FD) placement is generally effective for intractable internal carotid artery (ICA) aneurysms. However, salvage treatment for the aneurysm enlarging even after FD placement remains to be elucidated. Additional overlapping FD placement is considered the first-line treatment for residual or recurrent aneurysms. However, it is unclear whether overlapping FD is also effective for enlarging giant aneurysms that are considered impending rupture status. Although parent artery occlusion is a promising option, treatment strategy must be optimized, especially when a critical perforating artery is involved.

Case Description: A 74-year-old woman experienced rapid symptomatic growth of her giant supraclinoid ICA aneurysm 10 months after FD placement. We assumed that reinforcement of flow diverting effect alone would be less effective for this extremely intractable aneurysm with more aggressive clinical feature so that surgical bailout by parent artery occlusion was planned. Complete ICA obliteration underneath the aneurysm was unavailable due to the presence of anterior choroidal artery. Thus, we took a flow alteration strategy, where we created minimal retrograde flow through the parent artery by a combination of an extracranial-intracranial bypass and targeted endovascular proximal parent artery obliteration, resulting in prevention of aneurysmal rupture and further growth.

Conclusion: Impending rupture of the intracranial giant aneurysm after FD placement may be controllable with a tailor-made parent artery occlusion strategy even when a critical perforating artery is involved.

Keywords: Extracranial-intracranial bypass, Flow alteration therapy, Flow diverter, Giant intracranial aneurysm, Parent artery occlusion

INTRODUCTION

Recent studies have shown favorable clinical results of flow diverter (FD) placement to treat intractable intracranial aneurysms.[2,3,14] However, potential complications of FD placement include delayed aneurysmal rupture, which is disastrous and fatal in most cases.[11] Additional treatment strategies for impending rupture of the unruptured intracranial giant aneurysm after FD placement remain to be elucidated. In this article, we treated a patient with giant supraclinoid internal carotid artery (ICA) aneurysm enlarging after FD placement. Flow alteration strategy by

combination of extracranial-intracranial bypass and targeted endovascular parent artery occlusion successfully prevented rupture and further growth while securing the anterior choroidal artery (AchA) involved in the aneurysmal segment.

CASE PRESENTATION

A 74-year-old woman presented with intermittent headache and screening magnetic resonance imaging (MRI) showed a suprasellar mass lesion with a flow void, which was confirmed with cerebral angiography as a 32 mm giant aneurysm at the supraclinoid segment of the right ICA [Figures 1a and b]. The AchA and the posterior communicating artery (PCoA) arose from the same segment as the aneurysm across the ICA [Figure 1c]. The patient was treated with FD placement, where Pipeline Flex (4.75 mm × 25 mm, Medtronic, Minneapolis, USA) was placed from the M1 portion of the middle cerebral artery to the ophthalmic segment of the ICA with rough aneurysmal packing with detachable coils, which significantly reduced intra-aneurysmal blood flow [Figures 1d and e].

At the 6-month follow-up, the patient was neurologically intact and the aneurysm was stable in size. However, she presented with gait disturbance, cognitive dysfunction, and symptomatic seizure, 10 months after FD placement. Repeat MRI showed aneurysmal enlargement to 38 mm with remarkable perianeurysmal edema, together with hydrocephalus caused by obstruction of bilateral foramen of Monro [Figures 1f and g]. Cerebral angiography showed residual blood flow at the aneurysmal neck with coil mass deformation [Figure 1h]. Because delayed growth and symptomatic change of the aneurysm were considered impending rupture, we planned parent artery occlusion strategy. Given complete ICA obliteration underneath the aneurysm was unavailable due to presence of AchA, flow alteration therapy using a high-flow extracranial-intracranial bypass and targeted endovascular proximal parent artery obliteration performed.

Operation was performed in the hybrid operating room where both surgery and flat-panel detector angiography (Siemens, Berlin, Germany) are available.[8] Schematic imaging of our treatment strategy is shown in Figure 2a. Under general anesthesia, a 4 French (Fr) 90 cm guiding sheath was inserted into the right femoral artery. The external carotid artery-radial artery interposition graft-M2 portion of
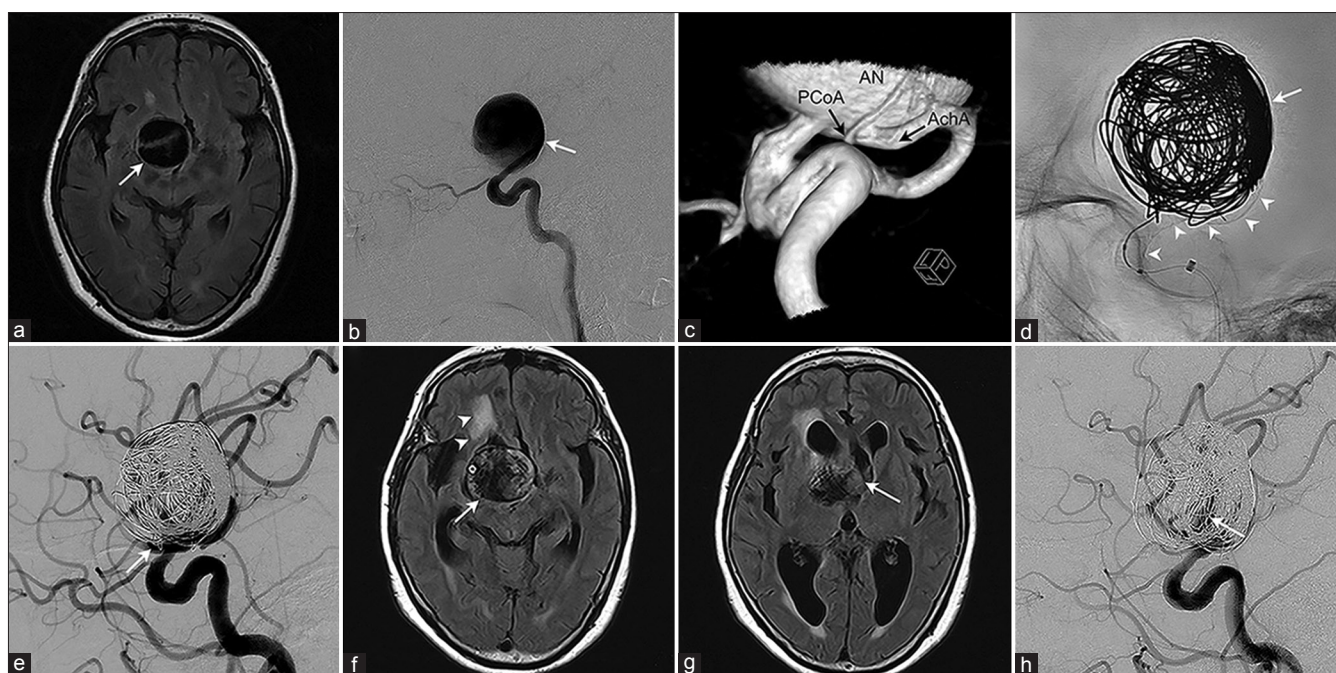

Figure 1: Radiological imaging of a 74-year-old woman who presented with the right intracranial giant internal carotid artery aneurysm. (a) Head magnetic resonance (MR) imaging shows a 32 mm round mass with a flow void compressing right hypothalamus (arrow). (b) Lateral view of the right internal carotid angiogram shows a giant aneurysm at the supraclinoid portion of the internal carotid artery (arrow). (c) A posterior-lateral-inferior view of reconstructed three-dimensional right internal carotid angiogram shows orifices of the posterior communicating artery and anterior choroidal artery across the wide aneurysmal neck. (d) Intraoperative working angle fluoroscopy shows deployed flow diverter (arrowheads) with intra-aneurysmal detachable coils (arrow) being packed. (e) Postprocedural common carotid angiogram shows remarkable flow reduction of the aneurysm with a small amount of neck remnant (arrow). (f) Head MR imaging 10 months after the flow diverter placement shows aneurysm enlargement to 38 mm (arrow) with perifocal edema (arrowheads). (g) Head MR imaging at the level of the anterior horn of the lateral ventricle shows hydrocephalus caused by obstruction of bilateral foramen of Monro (arrow). (h) The right internal carotid angiogram at aneurysmal enlargement shows residual intra-aneurysmal flow (arrow) with deformed coils.
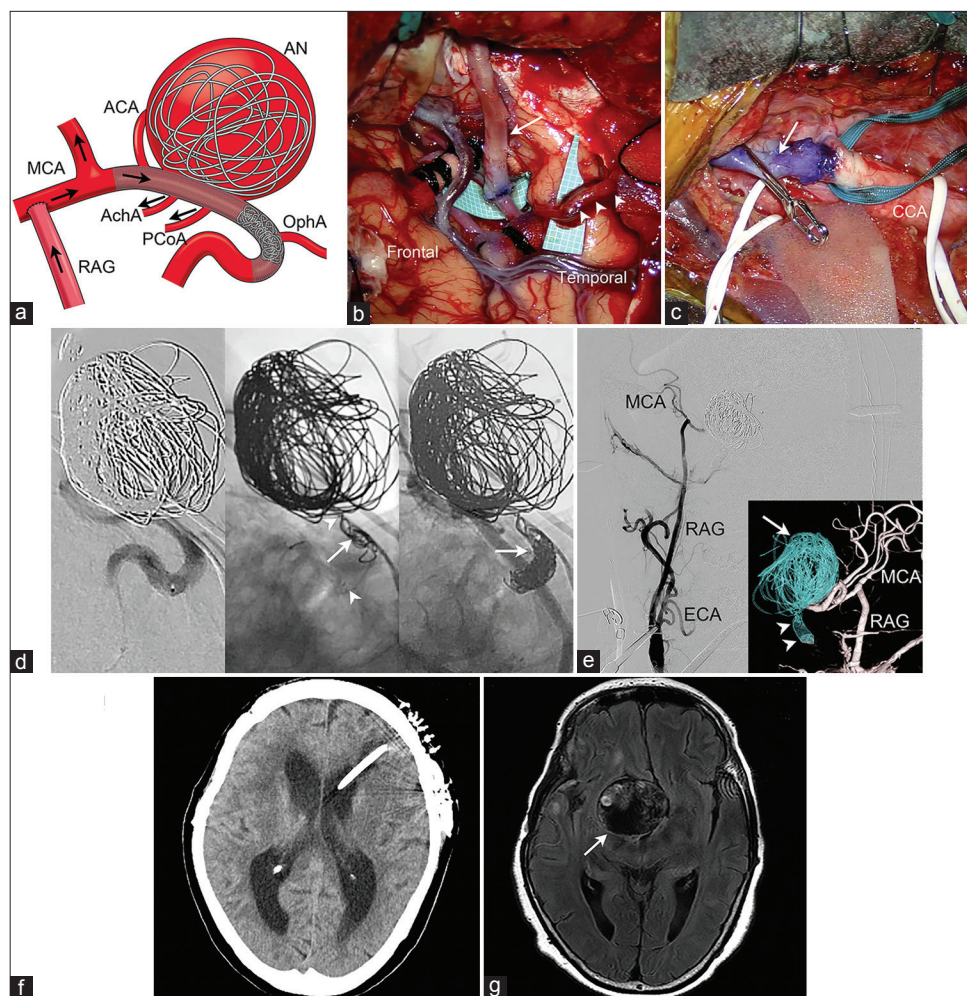
Figure 2: Schematic imaging, operative findings, and postoperative course, of the flow alteration treatment in the hybrid operating room. (a) Schematic imaging of the treatment strategy. A high-flow external carotid artery-radial artery graft-middle cerebral artery bypass is made, and the internal carotid artery is obliterated with coils just proximal to the posterior communicating artery. Black arrows show direction of the blood flow after the procedure. (b) Intraoperative photograph of the extracranial-intracranial bypass, cranial part. The free radial artery graft (arrow) and superficial temporal artery (arrowheads) are anastomosed with M2 and M4 portions of the middle cerebral artery, respectively. (c) Intraoperative photograph of the bypass, cervical part. The proximal end of the radial artery graft (arrow) is anastomosed with the external carotid artery. (d) Endovascular procedure. (Left) The course of the right internal carotid artery is shown through contrast medium injection from the microcatheter. (Center) Fluoroscopic imaging shows that an open-cell stent Neuroform Atlas has been deployed between markers at the both ends (arrowheads) and the first coil (arrow) is readily stabilized, being anchored by stent strut. (Right) Fluoroscopy shows that the ophthalmic segment of the right internal carotid artery is completely obliterated with coils (arrow). (e) Conventional anteroposterior and reconstructed posteroanterior view (inset) of the right common carotid angiogram show disappearance of intra-aneurysmal flow (arrow) because of parent artery occlusion with coils (arrowheads), as well as patency of the high-flow bypass. (f) Postoperative head CT scan shows that the patient's hydrocephalus improves due to the left ventriculoperitoneal shunt placement. (g) Head magnetic resonance imaging 12 months after the flow alteration therapy shows that the aneurysm is stable (arrow) with reduced perifocal edema. ACA: Anterior cerebral artery, AChA: Anterior choroidal artery, AN: Aneurysm, ECA: External carotid artery, MCA: Middle cerebral artery, OphA: Ophthalmic artery, PCoA: Posterior communicating artery, RAG: Radial artery graft.

middle cerebral artery (EC-RA-M2) high-flow bypass was performed, as previously described [Figures 2b and c].[5,12] Immediately after high-flow bypass completion, a tourniquet was placed at the cervical ICA to control anterograde blood flow and squeezed as appropriate. Endovascular parent artery occlusion was performed under retrograde blood flow provided by the high-flow bypass and proximal ICA clamping. First, a 4 mm × 21 mm Neuroform Atlas open-cell stent (Stryker, Fremont, USA) was deployed at the ophthalmic segment of the ICA so that the distal end of the stent was placed just proximal to the aneurysmal neck. Then, the microcatheter was advanced into the deployed stent and the ophthalmic segment of the ICA was tightly packed with 6 Target detachable coils (Stryker) without compromising the PCoA orifice [Figure 2d]. Initial coils were readily stabilized in the stent presumably because the stent strut worked as a scaffold and anchored the coils, contributing to shorter procedure time and prevention of distal coil migration. Postembolization common carotid angiography revealed complete proximal ICA occlusion, lack of contrast medium inflow into the aneurysm, and a patent high-flow bypass [Figure 2e].

The patient's postoperative course was uneventful. Postoperative MRI showed no ischemic complications. She underwent ventriculoperitoneal shunt with endoscopic septostomy 3 weeks after the aneurysm treatment [Figure 2f]. Her gait and memory abnormalities improved, suggesting resolution of the hydrocephalus, although spatial neglect and inattention partially remained as mass effects. She was discharged home after a 3-month cognitive rehabilitation training. She did not experience aneurysmal rupture for a year, and follow-up MRI at 6 months and 1 year [Figure 2g] revealed a stable aneurysmal size, with reduced perianeurysmal edema.

DISCUSSION

Recently, FD placement for intractable intracranial aneurysms has been developed. FD treatment reserves anterograde flow of the parent artery and maintains blood flow of the adjacent perforating arteries through the stent strut, while reducing aneurysmal inflow and causing progressive aneurysmal thrombosis.[2,3,14] However, delayed aneurysmal rupture occurs in approximately 2% of FD placement procedures.[3,11] Because aneurysmal rupture after FD placement is generally disastrous and associated with high mortality rate, detection of impending rupture and timely treatment is mandatory.[11]

Our patient experienced symptomatic aneurysmal growth between 6 and 10 months after FD placement. Because symptomatic growth of giant intracranial aneurysms has a high risk of rupture and has been considered as impending rupture status, immediate treatment to exclude the aneurysm from the normal blood circulation has been proposed.[16,17]

This is also the case with aneurysms after FD placement, where extensive intra-aneurysmal thrombogenesis as a part of healing process may even cause thrombus-induced wall degradation depending on hemodynamic conditions, causing aneurysmal rupture.[11] Both reconstructive and deconstructive techniques are available for giant aneurysms even after failed FD treatment.[4] Addition of overlapping FD placement for reconstruction has been used to treat recurrent or recanalized aneurysms after FD placement.[6] However, because complete aneurysm occlusion by increasing flow-diverting effects takes months, risks of delayed rupture remain. Furthermore, the hemodynamic pattern that caused aneurysmal growth and thrombus-induced wall degradation would not drastically change by increasing flow-diverting effect alone. On the other hand, parent artery occlusion as deconstructive technique not only reduces intra-aneurysmal blood flow but also immediately mitigates hemodynamic stress at the inflow zone by creating reverse flow, which may have prevented aneurysmal rupture and further growth.

Parent artery occlusion strategy in the present case should be optimized according to anatomical features of the aneurysm. After FD placement, the presence of dense mesh stent in the ICA made clip ligation impossible. In addition, the orifice of AchA was located at the same segment as the aneurysm across the ICA. Complete occlusion of the ICA underneath the entire aneurysmal neck (i.e., internal trapping) was unavailable; thus, proximal occlusion with coils was performed. With this, we sought to reverse and minimize blood flow of the ICA while avoiding ischemic complications.[7,13] Although being unintentionally combined, reverse, and minimal flow created by flow alteration may have synergized with the flow diversion effect by the preexisting FD to suppress aneurysmal growth and rupture. We selected the supraclinoid ICA just proximal to the PCoA as the obliteration site. Thus, we were able to minimize the retrograde ICA blood flow by choosing the tiny PCoA as an outflow conduit of the flow alteration strategy, rather than choosing the larger ophthalmic artery.[5,7] PCoA preservation also contributed to avoiding blind alley formation for the retrograde flow at the distal end of the coil mass, which may cause delayed AchA thrombosis.[9,10] Even though sufficient blood flow is provided through the extracranial-intracranial bypass, endovascular supraclinoid ICA obliteration is associated with ischemic complications, presumably because of distal coil migration to the AchA and PCoA.[13] Together with proximal flow control by a tourniquet, the open-cell stent placed just proximal to the aneurysmal neck successfully prevented ischemic complications by anchoring the coils in place and preventing distal coil migration in the slippery FD with high metal coverage rate [Figure 3].

To the best of our knowledge, this is the first report describing successful tailor-made flow alteration for impending rupture of the giant supraclinoid intracranial
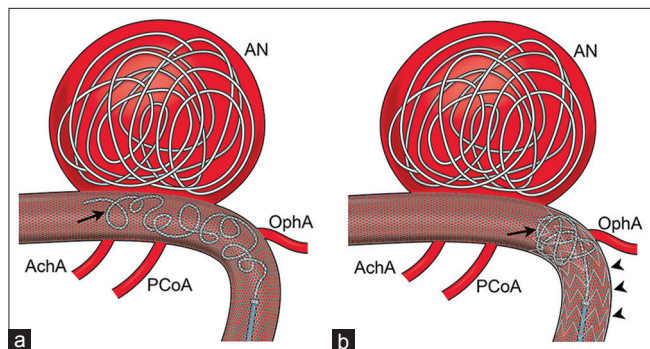
Figure 3: (a) Schematic imaging of coil behavior when the additional Neuroform Atlas stent is absent. The distal coil end (arrow) is unstable and protrudes toward the anterior choroidal artery and the posterior communicating artery in the cylinder-like slippery flow diverter. (b) Schematic imaging of coil behavior when the additional Neuroform Atlas stent (arrowheads) is present. The open-cell struts of Neuroform Atlas works as a scaffold and prevent coils (arrow) from protruding distally.

aneurysm after FD placement. Although we believe that flow alteration strategy is justified to obtain maximal effect to control intractable aneurysm presenting aggressive feature even after FD placement, little is known about hemodynamic compromise of AchA that has already been covered by FD and subsequently exposed to decreased ICA flow by flow alteration. In our case, outlet flow through the tiny PCoA may be high enough to prevent blind array formation and delayed thrombosis of the AchA. Another possibility is that AchA is intrinsically tolerable for FD coverage as described in the previous literature.[15] A previous case report of successful open surgical bailout for failed FD, where ischemia of AchA that had already been covered with multiple FDs did not occur by rescue parent artery occlusion of the ICA with low-flow bypass surgery, encouraged us to take our tailor-made flow alteration strategy for the intractable giant aneurysm.[1] Further investigations would be warranted to establish safety and efficacy of parent artery occlusion strategy by accumulating rescue therapy for unsuccessful FD treatment.

CONCLUSION

In this article, we described a successful flow alteration therapy for a giant supraclinoid ICA aneurysm enlarging after FD placement. Impending rupture of the FD-treated aneurysms may be safely controlled with tailor-made strategy using extracranial-intracranial bypass and targeted endovascular parent artery occlusion.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Conflicts of interest

There are no conflicts of interest.

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