Original Article

Risk factors and consequences of unexpected trapping for ruptured anterior communicating artery aneurysms

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Abstract

Background: While clipping cerebral aneurysms at the neck is optimal, in some cases this is not possible and other strategies are necessary. The purpose of this study was to describe the incidence, risk factors, and outcomes for inability to clip reconstruct ruptured anterior communicating artery (ACoA) aneurysms.

Methods: Of the 70 cases of ruptured ACoA aneurysms between January 2006 and December 2013, our institutional experience revealed four cases of small ACoA aneurysms that had been considered clippable prior to operation but required trapping. When a unilateral A2 segment of anterior cerebral artery (ACA) was compromised by trapping, revascularization was performed by bypass surgery. Clinical presentation, angiographic characteristics, operative approach, intraoperative findings, and treatment outcomes were assessed.

Results: Very small aneurysm under 3 mm was a risk factor for unexpected trapping. The reason for unexpected trapping was laceration of the aneurysmal neck in two cases, and lack of clippaple component due to disintegration of entire aneurysmal wall at the time of rupture in the others. Aneurysms with bilateral A1 were treated with sole trapping through pterional approach in two cases. The other two cases had hypoplastic unilateral A1 segment of ACA and were treated with combination of aneurysm trapping and revascularization of A2 segment of ACA through interhemispheric approach. No patients had new cerebral infarctions of cortical ACA territory from surgery. Cognitive dysfunction was observed in three cases, but all patients became independent at 12-month follow up.

Conclusions: Unexpected trapping was performed when ruptured ACoA aneurysms were unclippable. Trapping with or without bypass can result in reasonable outcomes, with acceptable risk of cognitive dysfunction.

Key Words: Anterior communicating artery aneurysm, cerebral revascularization, cognitive function, subarachnoid hemorrhage, unexpected trapping

INTRODUCTION

Rupture of anterior communicating artery (ACoA) aneurysm is one of the most common causes of aneurysmal subarachnoid hemorrhage (SAH).[1] Currently, most of the aneurysms are treated with neck clipping by craniotomy, or intraaneurysmal coiling by endovascular surgery, both of which preserve ACoA. Giant or fusiform aneurysms of ACoA, which lack defined aneurysmal neck, have been treated with intentional trapping.[10,14] Some aneurysms must be unexpectedly trapped due to intraoperative troubles for various reasons, such as intraoperative aneurysmal neck rupture and lack of clippable component.[14,29]

When ruptured ACoA aneurysms are treated with unexpected trapping, perforators from ACoA must be spared to prevent postoperative cognitive dysfunction.[7,15] In addition, unexpected trapping of ACoA compromises A2 segment of anterior cerebral artery (ACA) in cases of contralateral hypoplastic A1 segment.[14,29] However, reconstruction of A2 segment in the deep operative corridor is challenging, especially under urgent conditions.

Here, we describe four cases of ruptured ACoA aneurysms, which were unexpectedly trapped. Through reviewing these cases, risk factors of unexpected trapping and operative strategy to preserve surrounding vessels are discussed.

METHODS

Patients' characteristics and preoperative workup

Between January 2006 and December 2013, the authors consecutively treated 70 patients with SAH from ruptured ACoA aneurysms. The choice of treatment, clipping or coiling, was determined according to discussions between surgical and endovascular teams. As a result, 32 cases were assigned to undergo surgical clipping. The morphology of the aneurysm such as broad neck or difficult access route was a factor influencing treatment decision away from coiling in a number of these cases. Formal approval of the institutional ethics review board was not required because of the retrospective nature of our analysis and information obtained from routine clinical care of these patients did not contain any unique identifiers linking these patients.

Computed tomography (CT) angiography was routinely obtained on admission. In surgically treated cases, catheter angiography was performed only when CT angiography provided insufficient information for clipping, or when responsible aneurysm was not detected. The size of the aneurysm was measured on CT angiography. Catheter angiography was only used to measure the size when CT angiography failed to detect the aneurysm.

Operative strategy

Both pterional and interhemispheric approaches were used, but surgeons' preference shifted to the interhemispheric approach over time, where it was selected for 3 cases in the first 16 cases, whereas for 10 in the latter 16 cases. The aneurysm was unexpectedly trapped in four patients where neck clipping was impossible. When unilateral A2 segment of ACA was compromised by trapping, revascularization with bypass surgery was added.

Postoperative patient management

If symptomatic hydrocephalus was present soon after SAH, an external ventricular drain was used to decompress the ventricular system. When hydrocephalus persisted, or if it developed late in the clinical course, ventriculoperitoneal shunt was inserted. Normal blood pressure and normal hydration were maintained to prevent symptomatic vasospasm, and oral calcium channel blocker or intravenous fasudil chloride was given if applicable. Rehabilitation of impaired cognitive function commenced by speech therapists as early as 2 days after operation and continued at least for 3 months. Outcome was assessed with the modified Rankin Scale (mRS) score at 12 months.

Statistical analysis

Fisher's exact probability test was used to statistically assess whether very small aneurysms are associated with incidence of unexpected trapping. Commercially available software (SPSS version 20, IBM Corp. Armonk, NY, USA) was used to create 2×2 contingency table and for analysis. Results were considered significant when $P < 0.05$.

RESULTS

Among 32 surgically treated ruptured ACoA aneurysms, unexpected trapping was performed in 4 cases and are summarized in Table 1. All four patients were female and presented with SAH of good clinical grade (World Federation of Neurosurgical Societies [WFNS] grade I-II). The size of the aneurysms ranged from 0.5 to 6 mm, three of which are under 3 mm. Because the mean size of 28 aneurysms treated with neck clipping was 6.3 ± 3.0 (standard deviation) mm (range 4-20 mm), very small aneurysms under 3 mm were significantly associated with higher incidence of unexpected trapping ($P < 0.001$, Fisher's exact probability test).

The reason for unexpected trapping was laceration of the aneurysmal neck in two cases, and lack of clippable component due to disintegration of entire aneurysmal wall at the time of rupture in the others. Bypass surgery

Table 1: Summary of patients who have undergone unexpected trapping for ruptured anterior communicating artery aneurysm*

Case no.	Age, gender	WFNS grade	Aneurysm size (mm)	Presence of A1	Approach	Reason for trapping	Treatment	mRS at 12 months
1	58, F	I	2	Bilateral	Pterional	Neck laceration during procedure	Trapping	0
2	61, F	II	0.5	Bilateral	Pterional	Lack of clippable component	Trapping	1
3	66, F	I	2	Unilateral	Interhemispheric	Lack of clippable component	Trapping+STA-ACA bypass	2
4	58, F (ADPKD)	I	6	Unilateral	Interhemispheric	Neck laceration during procedure	Trapping+A3-A3 bypass	1

*WFNS: World Federation of Neurosurgical Societies, mRS: modified Rankin Scale, STA: superficial temporal artery, ACA: anterior cerebral artery, A3: A3 segment of anterior cerebral artery, ADPKD: autosomal dominant polycystic kidney disease

for revascularization was performed in two cases, where unilateral A2 segment of ACA was compromised by trapping due to aplasia of ipsilateral A1 segment of ACA. Sole trapping without bypass was performed in the other two cases with bilateral equivalent A1 segment of ACA. Under this maximum revascularization strategy, none of the patients experienced ischemic complications of cortical ACA regions, or resultant leg weakness.

Cognitive dysfunction was observed in three patients out of four who underwent unexpected trapping. The cognitive dysfunction was relatively severe soon after operation, but gradually improved in 12 months, representing mRS 1-2. Detailed clinical course of each patient is described below.

Two cases with bilateral A1

Our initial two cases represent similar anatomical feature, operative findings, and clinical course. Case 1 was a 58-year-old female who presented with diffuse SAH of WFNS grade I [Figure 1a]. Her catheter angiography demonstrated 2 mm wide-neck aneurysm at left A1-A2 junction, with presence of bilateral equivalent A1 segment of ACA [Figure 1b]. Direct surgery of the aneurysm was performed by left pterional approach. The aneurysm looked clippable, but proximal aneurysm neck was lacerated during exploration with a dissector [Figure 1c]. Because any further attempts of neck clipping to spare ACoA were unsuccessful, the aneurysm was trapped in the short segment with two aneurysm clips under flow control with temporary clips [Figure 1d]. The largest perforator of ACoA, a hypothalamic artery, originated adjacent to the proximal neck but it was spared outside trapping [Figure 1d]. Her postoperative course was uneventful. Her cognitive and hypophyseal function remained normal. Complete obliteration of the aneurysm was confirmed on postoperative CT angiography [Figure 1e] and no cerebral infarction was observed on postoperative MRI [Figure 1f]. She was discharged home and her mRS was 0 at 12 months.

Case 2 was a 61-year-old female of diffuse SAH with thick hematoma in the interhemispheric fissure [Figure 2a]. Her WFNS grade was II with Glasgow Coma Scale (GCS) E3V5M6. Catheter angiography failed to detect definite
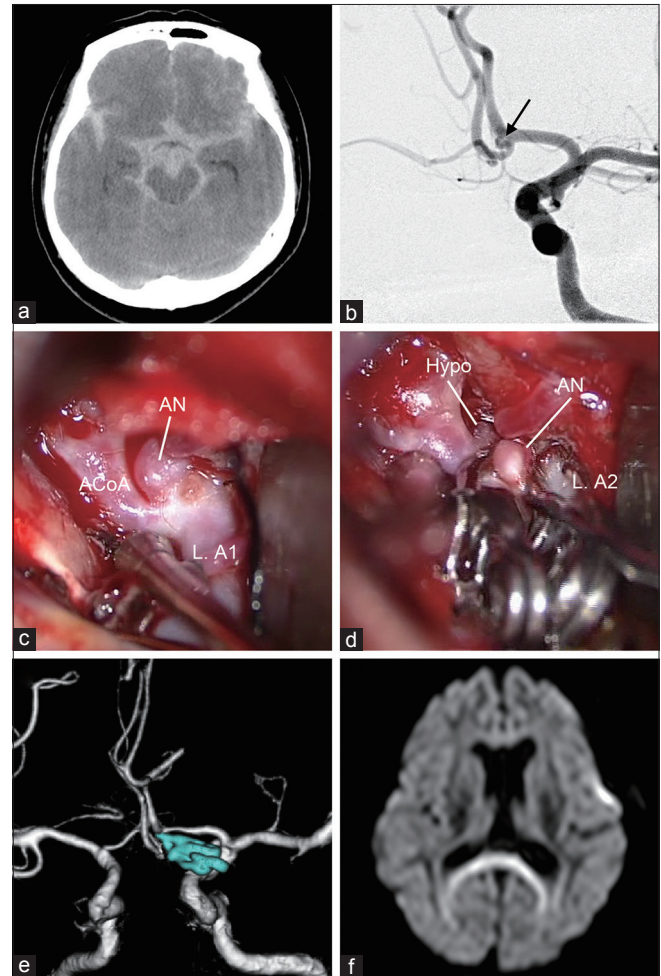

Figure 1: Case 1. Images and intraoperative photographs obtained in a 58-year-old female who suffered SAH of WFNS grade I. (a) Head CT scan obtained on admission demonstrates diffuse SAH. (b) Oblique view of left carotid angiogram with right carotid compression, following negative CT angiogram, reveals a 2 mm saccular aneurysm at the left A1-A2 junction (arrow). (c) Intraoperative photograph of ACoA complex through left pterional approach. A small aneurysm is observed at the left A1-A2 junction. (d) The aneurysm is trapped with two aneurysm clips. Hypothalamic artery is preserved. (e) Postoperative CT angiogram shows the aneurysm is trapped and neither of bilateral A2 segments of ACA is obliterated. (f) Postoperative MRI (diffusion-weighted imaging) demonstrates no ischemic infarction. ACoA= anterior communicating artery, AN= aneurysm, L. A1= left A1 segment of ACA, L. A2= left A2 segment of ACA, Hypo= hypothalamic artery
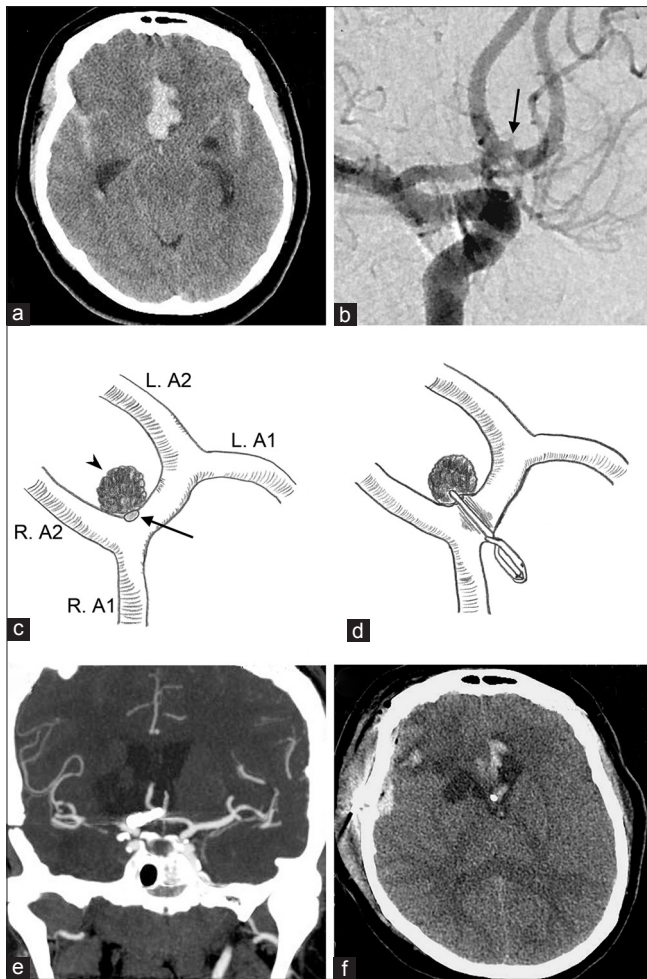
Figure 2: Case 2. Images, and schemes of intraoperative findings obtained in a 61-year-old female who suffered SAH of WFNS grade II. Intraoperative photographs are not available. (a) Head CT scan obtained on admission demonstrates diffuse SAH with a massive clot in the interhemispheric fissure. (b) Oblique view of right carotid angiogram only reveals a minute protuberance at right A1-A2 junction (arrow). (c) Schematic picture of intraoperative findings through right pterional approach. A fragile fibrin cap (arrowhead) covers a small hole (arrow) on right A1-A2 junction. No defined aneurysm wall is observed. (d) An aneurysm clip is applied on the small hole across ACoA. A single clip is wide enough to close the small hole. (e) A coronal image of postoperative CT angiogram shows bilateral A2 segments of ACA are intact and left A1 segment of ACA is present. (f) Postoperative head CT scan reveals cerebral edema around thick subarachnoid clot and ischemic infarction of right caudate head caused by prolonged temporary clipping. However, most of ACA territory is spared. L. A1= left A1 segment of ACA, L. A2= left A2 segment of ACA, R. A1= right A1 segment of ACA, R. A2= right A2 segment of ACA

aneurysms, only to disclose a minute protuberance at right A1-A2 junction [Figure 2b]. Surgical exploration of ACoA was performed through right pterional approach. Exposure of ACoA complex revealed that aneurysm wall was missing and probable rupture point on ACoA was only covered with a mass of fibrin net, suggesting there was no clippable component [Figure 2c]. The fibrin cap was avulsed during exploration, resulting

in massive bleeding from a rent on ACoA. Under flow control, a single permanent clip was applied on the rent across ACoA since contralateral A1 was present on preoperative CT angiography [Figure 2d and e]. Hypothalamic artery was not observed in the operative field. Postoperatively, the patient developed moderate recent memory disturbance and diabetes insipidus. Follow up CT scan revealed ischemic change of right caudate nucleus [Figure 2f], but it was unclear whether her symptoms were caused by intraoperative procedures or initial damage from the SAH. Her memory disturbance improved by rehabilitation and her diabetes insipidus was well controlled by daily spray of desmopressin. She was discharged home, and her mRS was 1 at 12 months with persistent use of desmopression.

Case 3 with unilateral A1

Case 3 presented the most complex clinical course. A 66-year-old female was referred to our hospital for SAH of WFNS grade I (E4V5M6) [Figure 3a]. She underwent endovascular coiling for the aneurysm at left P2 segment of posterior cerebral artery [Figure 3b]. However, on the 7th day after initial SAH, the patient revealed recurrent SAH with severe headache and slight drowsiness. Her catheter angiography demonstrated complete obliteration of left P2 aneurysm and abnormal aneurysm-like dilatation on ACoA [Figure 3c]. Compared with CT angiography obtained at the first SAH [Figure 3c], this aneurysmal dilatation had been growing in 7 days, suggesting that unruptured posterior cerebral artery aneurysm was treated and the ACoA aneurysm ruptured twice. Endovascular treatment of this ACoA aneurysm was not feasible and microsurgical clipping through anterior interhemispheric approach was planned.

Operation commenced with bifrontal craniotomy and the interhemispheric fissure was widely opened from genu of corpus callosum to rectus gyrus. There was no clippable component at the aneurysm neck because entire aneurysmal wall was disintegrated and the virtual neck of the aneurysm was only covered with a fragile fibrin cap [Figure 3d]. Because aneurysmal neck clipping with the goal of ACoA preservation seemed difficult and right A1 segment was aplastic on imaging [Figure 3c], left superficial temporal artery (STA)-right ACA bypass was performed before dissecting the aneurysm in the event that aneurysm trapping was necessary [Figure 3e]. Left STA was selected as a graft because it was larger than right STA. The parietal branch of left STA was harvested as an interposition graft. Both ends of the free graft were anastomosed with the frontal branch of the left STA and callosomarginal artery, in end-to-end and end-to-side fashion, respectively [Figure 3f]. However, dissection of the aneurysm revealed that trapping of the longitudinal rent on ACoA contained a risk of sacrificing blood flow to the hypothalamic artery, the origin of which is adjacent to the aneurysm. Finally to spare the hypothalamic artery,
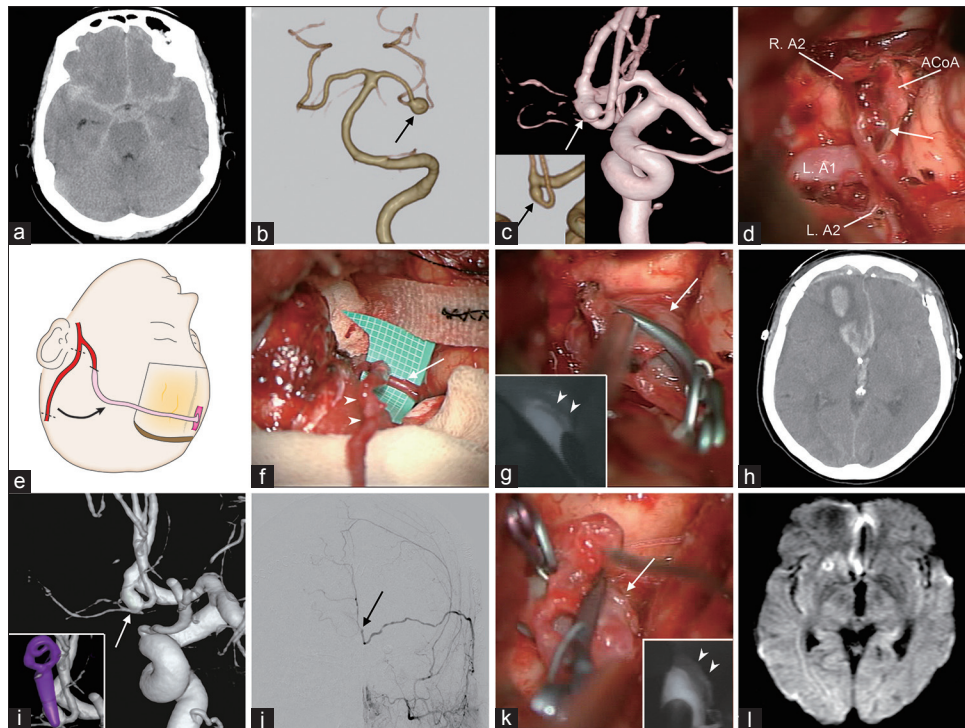
Figure 3: Case 3. Images, intraoperative photographs, and a schematic operation plan of a 66-year-old female who suffered SAH of WFNS grade I. (a)Head CT scan obtained on admission demonstrates diffuse SAH. (b) CT angiogram of left vertebral artery reveals a 5 mm saccular aneurysm at the left P1 segment of posterior cerebral artery *(arrow)*. (c) Three-dimensional rotational left carotid angiogram obtained when SAH recurred on the 7th day after initial SAH. A 2 mm aneurysm-like dilatation *(white arrow)* is observed on ACoA. CT angiogram of bilateral internal carotid artery obtained on admission *(inset)* reveals small dilatation of ACoA *(black arrow)*. Retrospectively compared with the imaging on day 7, the aneurysm-like dilatation is growing. Right A1 segment of ACA is aplastic. (d) Intraoperative photograph of ACoA complex through interhemispheric approach. A fragile fibrin cap *(arrow)* is observed on ACoA and no defined aneurysm wall is observed. Right A2 segment of ACA winds around the fibrin cap. (e) Schematic imaging of left STA-right ACA bypass with interposition graft. The parietal branch of left STA (between dashed lines) is detached and used as the interposition graft between the frontal branch of left STA and right callosomarginal artery. (f)Intraoperative photograph of the bypass. The distal end of the interposition graft *(arrowheads)* is anastomosed with the right callosomarginal artery *(arrow)* in end-to-side fashion. (g)The aneurysm is clipped by slightly incorporating the normal vessel wall of ACoA. Hypothalamic artery is preserved both morphologically under the microscope *(arrow),* and optically under the indocyanine green videoangiography *(arrowheads)*. (h) Head CT scan is obtained 7 days after the second SAH when patient's consciousness suddenly deteriorated. Intraventricular and intracerebral hemorrhage suggests the third rupture of the aneurysm. (i)Three-dimensional rotational catheter angiogram (subtracted image) reveals aneurysm-like dilatation on ACoA *(arrow)*. Inset shows nonsubtracted image of the same angiogram, suggesting displacement of the aneurysm clip. (j) Left external carotid angiogram at the third SAH demonstrates blood flow of right distal ACA territory through patent left STA-right ACA bypass. End-to-side anastomosis between interposition graft and callosomarginal artery is indicated *(arrow)*. (k) The aneurysm is trapped with two aneurysm clips. The proximal blue clip is applied adjacent to the origin of the hypothalamic artery *(arrow)*. Flow signal of the hypothalamic artery under indocyanine green videoangiography is still visible but diminished *(arrowheads)*. (l) Postoperative MRI (diffusion-weighted imaging) shows residual hematoma in the right frontal lobe, but no ischemic infarction. ACoA= anterior communicating artery, L. A1= left A1 segment of ACA, L. A2= left A2 segment of ACA, R. A2= right A2 segment of ACA

an aneurysm clip was placed parallel to the rent on ACoA with slight incorporation of the arterial wall [Figure 3g]. The intraoperative indocyanine green videoangiography confirmed blood flow both in ACoA and hypothalamic artery [Figure 3g]. In summary, right A2 was reconstructed for potential trapping, but, in fact, neck clipping was performed with physiological flow of ACoA reserved.

The patient recovered well, although she was slightly confused due to symptomatic vasospasm. However, on the 7th day after the onset of the second SAH, the patient suddenly became comatose. Head CT scan demonstrated the third ictus of SAH with intracerebral

and intraventricular hemorrhage [Figure 3h]. Catheter angiography revealed irregular bulging of ACoA underneath the aneurysm clip, suggesting clip displacement and rerupture [Figure 3i]. After confirming the left STA-right ACA bypass was still patent on the angiography [Figure 3j], reoperation was performed by the interhemispheric approach and the aneurysm was trapped with two aneurysm clips. The proximal clip was applied obliquely and the clip blade was placed just distal to the origin of the hypothalamic artery. Although blood flow of the hypothalamic artery looked diminished by the Doppler ultrasonography and the indocyanine green videoangiography, the clips were

not replaced to prioritize complete trapping [Figure 3k].

Her recovery of consciousness from comatose was excellent. Postoperative MRI revealed no cerebral infarction related to the surgical procedure [Figure 3l]. She underwent ventriculoperitoneal shunt in the chronic stage. For a few months after operation, she manifested a wide range of cognitive dysfunction as frontal lobe syndromes, including memory disturbance and impaired comprehension, initiation, and motivation. She was able to walk independently, but most of her daily life was dependent in terms of cognitive function. However, after intensive rehabilitation, she was discharged home and was able to look after her own affairs without assistance. Her hypophyseal function was normal through her clinical course. Her mRS at 12 months was 2.

Case 4 with unilateral A1

A 58-year-old female walked into our clinic with intractable dizziness and nausea for 3 days. She was undergoing dialysis for her chronic renal failure caused by familial polycystic kidney disease. She had a history of neck clipping of unruptured left middle cerebral artery aneurysm some 13 years ago at another institution. Her GCS was E4V5M6 so WFNS grade was I. Head CT scan demonstrated small amount of SAH in the interhemispheric fissure [Figure 4a]. CT angiography revealed 6 mm ACoA aneurysm, as well as complete clipping of left middle cerebral artery aneurysm [Figure 4b]. Endovascular surgeons were reluctant from coiling because of wide neck of the aneurysm.

Operation was performed via anterior interhemispheric approach. Dissection of interhemispheric fissure between rectus gyrus and genu of corpus callosum provided sufficient exposure of the ACoA aneurysm. During dissection, left olfactory nerve was missing and A1 segment of left ACA adhered to surrounding brain tissue, suggesting ACoA had been explored in the previous operation. The adhesion was more prominent around the aneurysm, and hypothalamic artery was encased in the aneurysmal wall with glue material [Figure 4c]. Gentle and meticulous dissection was performed, but minor leak of blood occurred from the neck of the aneurysm adjacent to right A2. Any attempts for neck clipping only enlarged laceration of aneurysmal neck. Trapping of the aneurysm including the neck laceration was unavoidable to secure the aneurysm, with simultaneous revascularization of right A2 because right A1 was aplastic on initial CT angiography [Figure 4b]. Temporary hemostasis of lacerated neck was obtained by compression with cottonoid under flow control for a few minutes. Back to A3 segment of ACA, A3-A3 side-to-side anastomosis was performed with bilateral A3 cross-clamping time
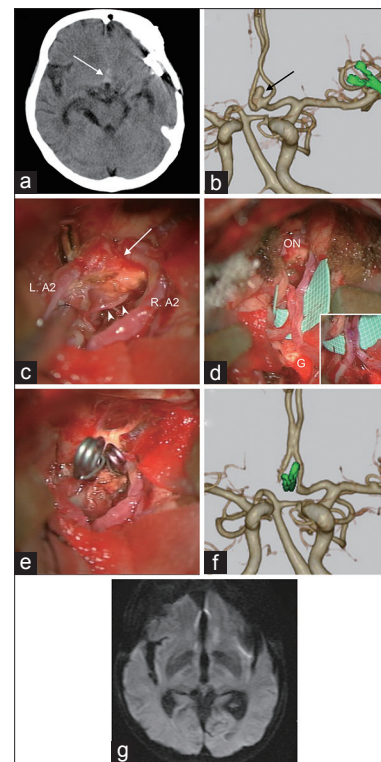

Figure 4: Case 4. Images and intraoperative photographs obtained in a 58-year-old female who suffered SAH of WFNS grade I. (a) Head CT scan obtained on admission demonstrates localized SAH in the interhemispheric fissure *(arrow)*. (b) CT angiogram reveals a 6 mm wide-neck saccular aneurysm on ACoA (arrow). Right A1 segment of ACA is aplastic. (c)Intraoperative photograph of ACoA complex through interhemispheric approach. The aneurysm is explored with a temporary clip applied on left A1 segment of ACA. Hypothalamic artery *(arrowheads)* is encased in the posterior wall of the aneurysm. Minor bleeding occurs at the neck of the aneurysm *(arrow)*. (d) Preparation of A3-A3 side-to-side anastomosis in the widely opened interhemispheric fissure from optic nerve (ON) to genu of the corpus callosum (G). Inset shows the bypass is successful. (e) The aneurysm, together with hypothalamic artery, is trapped with two aneurysm clips. (f) Postoperative CT angiogram shows the aneurysm is completely obliterated and right A2 segment of ACA is supplied through A3-A3 bypass. (g) Postoperative MRI (diffusion-weighted imaging) shows no ischemic infarction. L.A2= left A2 segment of ACA, R.A2= right A2 segment of ACA, ON= optic nerve, G= genu of corpus callosum

37 min [Figure 4d]. The aneurysm, together with hypothalamic artery, was trapped with two aneurysmal clips [Figure 4e]. Complete elimination of the aneurysm and reconstruction of right A2 segment of ACA was confirmed by Doppler ultrasonography, indocyanine green videoangiography, and postoperative CT angiography [Figure 4f].

The patient revealed good postoperative recovery with no apparent ischemic change on follow-up MRI [Figure 4g]. She developed recent memory disturbance and topographical disorientation, which severely affected her social activity. However, after several months at home, her cognitive function recovered well to almost normal. Her postoperative

hypophyseal function was normal. Her mRS at 12 months was 1.

DISCUSSION

In this article, we describe four cases of ruptured ACoA aneurysms that were unexpectedly trapped. Very small aneurysms under 3 mm were significantly associated with incidence of unexpected trapping. Trapping with elective bypass was a reasonable strategy to prevent disastrous rerupture and substantial cerebral infarction, with acceptable long-term outcome of cognitive function.

Very small aneurysms, which are defined as aneurysms under 3 mm, are associated with higher risk of procedure-related rupture when treated by endovascular coiling and generally treated surgically.[18,23] However, direct surgery of very small aneurysms is not necessarily easy for following reasons. First, these aneurysms have thin and fragile wall, carrying risk of narrowing or tearing the parent vessel.[19] Slight narrowing of a big parent vessel such as internal carotid artery rarely causes a problem, but previous studies demonstrated ruptured aneurysms under 3 mm are more often located on ACoA, the average caliber of which is as small as 1.5 mm.[3,15] Second, very small appearance of ruptured aneurysms on preoperative imaging might be a consequence of filling defect of contrast media caused by intraaneurysmal thrombus formation or disintegration of aneurysm wall.[25] Discrepancy between preoperative imaging and intraoperative findings occasionally makes the surgery difficult.[5,8] Thus, very small ACoA aneurysm is a primary risk factor of unexpected trapping.

In our case series of SAH, A2 segment of ACA was reconstructed by bypass surgery when it was compromised by aneurysm trapping. Aplasia of unilateral A1 segment is not uncommon, and this anatomical variant is associated with high incidence of aneurysms in the region of ACoA.[27,28] Thus, potential compromise of A2 segment of ACA contralateral to dominant A1 segment should never be overlooked in cases of ACoA aneurysms with risk factors of unexpected trapping. The need for revascularization of the A2 segment is still controversial. In the case series of giant ACoA aneurysms, therapeutic A1 or A2 segment occlusion was tolerated without bypass even when contralateral A1 is aplastic, because of robust leptomeningeal anastomosis.[6,13] However, this phenomenon might be specific in the giant aneurysm cases where anterograde blood flow has been gradually compromised by compression by aneurysm sac or by reservoir effect of the giant aneurysm, facilitating development of leptomeningeal anastomosis.[16,17] In that sense, unexpected trapping

of small ACoA aneurysm may cause drastic reduction of the blood flow in compromised A2 segment with poor leptomeningeal anastomosis, resulting in cerebral infarction. In other words, validity of the bypass for ACA ischemia might be more emphasized in trapping of small ACoA aneurysms. There are only two cases reported where small ACoA aneurysm was trapped and the unilateral A2 segment was occluded.[14,29] Because A2 segment was reconstructed with bypass in both cases, it is uncertain whether the patient tolerates occlusion of unilateral A2 segment without bypass. However, because early cerebral infarction unrelated to cerebral vasospasm is associated with poor clinical outcome after aneurysmal SAH, surgeons must maximally avoid procedure-related cerebral infarction with possible cerebral revascularization.[11,26] In addition, severity of upcoming vasospasm may not be as devastating when the bypass augments hemodynamics of compromised ACA territory.

We used two types of revascularization: STA-ACA bypass with interposition graft and A3-A3 *in situ* bypass, when unilateral A2 segment was compromised. Each procedure has advantages and drawbacks.[10,14,20] STA-ACA bypass is technically easier because more superficial recipient artery (e.g. callosomarginal artery) could be selected and because most surgeons are familiar with end-to-side anastomosis. In addition, simultaneous cross-clamping of bilateral A3, which impairs blood flow of bilateral frontal lobe, is avoided. On the contrary, A3-A3 bypass is a less tedious procedure of single anastomosis without harvesting long grafts that are associated with low long-term patency rates. Furthermore, physiological anterograde blood flow is preserved through donor arteries that match the caliber of recipient arteries. To apply for acute surgery of ruptured aneurysms, use of A3-A3 bypass is recommended because it is superior hemodynamically to counteract subsequent cerebral vasospasm, although side-to-side anastomosis in a deep corridor is technically challenging.[22]

In this study, we found interhemispheric approach was more advantageous than pterional approach to maximally avoid unexpected trapping. Wider front view of ACoA provided by interhemispheric approach enables finer manipulation and various options for clipping trajectory.[9] Laceration of the blinded neck in case 1 through pterional approach would have been avoided through interhemispheric approach. Even when trapping is inevitable, the hypothalamic artery originating from posterior aspect of ACoA is more easily identified and spared. Most importantly, exposure of A3 segment of ACA or callosomarginal artery for revascularization is only available through interhemispheric approach[9,10,14,15,29] when the unilateral A2 segment is compromised by trapping.

The unsolved problem of unexpected trapping of ACoA is impairment of cognitive function when perforating arteries are involved. Previous studies showed that cognitive dysfunction was more likely observed after rupture and/or repair of ACoA aneurysms, which is referred to as "ACoA syndromes".[2,4,7,24] About three decades ago, Gade *et al.* reported that 23% (11/48) of ruptured ACoA aneurysms were trapped, and 82% (9/11) of the patients treated with trapping presented with cognitive dysfunction, whereas 16% (6/37) with neck clipping. They suggested that postoperative cognitive dysfunction was attributable to sacrifice of perforating arteries from ACoA by trapping.[7] In cases where perforating arteries are close to unclippable aneurysms, any effort to avoid trapping of ACoA is attempted, such as parallel clipping by slightly catching the arterial wall, microsuture of avulsed neck, or clip wrapping.[12,21] However, these techniques are not always feasible, and efficacy to prevent rerupture is unknown. Incomplete clipping of the aneurysm to preserve a perforator may result in recurrence of SAH, as presented in case 3. For complete obliteration, s ome ruptured ACoA aneurysms must still be trapped despite recent development of microsurgery and endovascular coiling. Our case series suggest that long-term outcome of cognitive function is acceptable after trapping even when perforators are involved, as long as blood flow of A2 segment is secured.

Limitations of our study include the small number of patients and its retrospective approach. The high incidence of unexpected trapping might be caused by a referral bias. Coilable aneurysms might be treated in other hospitals and the patients with uncoilable aneurysms might be predominantly referred for clipping to our institute, where direct aneurysm surgery is always available. However, we believe cases described here cover most of possible causes and sequelae of unexpected trapping of ruptured ACoA aneurysms and would like to stress validity of revascularization of ACA territory when unilateral A2 is sacrificed. Future evaluations with a large number of patients are warranted to address precise incidence of unexpected trapping and proper strategy to treat ACoA aneurysms with risk factors of unexpected trapping.

CONCLUSIONS

Unexpected trapping was performed when ruptured ACoA aneurysms were unclippable. Very small aneurysms under 3 mm were significantly associated with incidence of unexpected trapping. Trapping of ACoA aneurysm is acceptable to obtain reasonable results as long as maximum cerebral revascularization is performed.

REFERENCES

1. Agrawal A, Kato Y, Chen L, Karagiozov K, Yoneda M, Imizu S, *et al.* Anterior communicating artery aneurysms: An overview. Minim Invasive Neurosurg 2008;51:131-5.
2. Böttger S, Prosiegel M, Steiger HJ, Yassouridis A. Neurobehavioural disturbances, rehabilitation outcome, and lesion site in patients after rupture or repair of anterior communicating artery aneurysms. J Neurol Neurosurg Psychiatry 1998;65:93-102.
3. Chalouhi N, Penn DL, Tjoumakaris S, Jabbour P, Gonzalez F, Starke RM, *et al.* Treatment of small ruptured intracranial aneurysms: Comparison of surgical and endovascular options. J Am Heart Assoc 2012;1:e002865.
4. Chan A, Ho S, Poon WS. Neuropsychological sequelae of patients treated with microsurgical clipping or endovascular embolization for anterior communicating artery aneurysm. Eur Neurol 2002;47:37-44.
5. Di Lorenzo N, Guidetti G. Anterior communicating aneurysm missed at angiography: Report of two cases treated surgically. Neurosurgery 1988;23:494-9.
6. Drake CG, Peerless SJ, Ferguson GG. Hunterian proximal arterial occlusion for giant aneurysms of the carotid circulation. J Neurosurg 1994;81:656-65.
7. Gade A. Amnesia after operations on aneurysms of the anterior communicating artery. Surg Neurol 1982;18:46-9.
8. Iwanaga H, Wakai S, Ochiai C, Narita J, Inoh S, Nagai M. Ruptured cerebral aneurysms missed by initial angiographic study. Neurosurgery 1990;27:45-51.
9. Ito Z. The microsurgical anterior interhemispheric approach suitably applied to ruptured aneurysms of the anterior communicating artery in the acute stage. Acta Neurochir (Wien) 1982;63:85-99.
10. Kim K, Mizunari T, Mizutani N, Kobayashi S, Takizawa K, Kamiyama H, *et al.* Giant intracranial aneurysm of the anterior communicating artery treated by direct surgery using A3-A3 side-to-side anastomosis and A3-RA graft-STA anastomosis. Acta Neurochir (Wien) 2006;148:353-7.
11. Kumar A, Brown R, Dhar R, Sampson T, Derdeyn CP, Moran CJ, *et al.* Early vs delayed cerebral infacrtion after aneurysm repair after subarachnoid hemorrhage. Neurosurgery 2013;73:617-23.
12. Lanzino G, Spetzler RF. Clip wrapping for partial avulsion of aneurysm neck: Technical note. J Neurosurg 2003;99:931-2.
13. Lownie SP, Drake CG, Peerless SJ, Ferguson GG, Pelz DM. Clinical presentation and management of giant anterior communicating artery region aneurysms. J Neurosurg 2000;92:267-77.
14. Mabuchi S, Kamiyama H, Kobayashi N, Abe H. A3-A3 side-to-side anastomosis in the anterior communicating aneurysms surgery: Report of four cases. Surg Neurol 1995;44:122-7.
15. Marinković S, Milisavljević M, Marinković Z. Branches of the anterior communicating artery. Microsurgical anatomy. Acta Neurochir (Wien) 1990;106:78-85.
16. Maruya J, Nishimaki K, Minakawa T. Hyperperfusion syndrome after neck clipping of a ruptured aneurysm on a dolichoectatic middle cerebral artery. J Stroke Cerebrovasc Dis 2011;20:260-3.
17. Murakami H, Inaba M, Nakamura A, Ushioda T. Ipsilateral hyperperfusion after neck clipping of a giant internal carotid artery aneurysm. Case report. J Neurosurg 2002;97:1233-6.
18. Nguyen TN, Raymond J, Guilbert F, Roy D, Bérubé MD, Mahmoud M, *et al.* Association of endovascular therapy of very small ruptured aneurysms with higher rates of procedure-related rupture. J Neurosurg 2008;108:1088-92.
19. Nussbaum ES, Erickson DL. The fate of intracranial microaneurysms treated with bipolar electrocoagulation and parent vessel reinforcement. Neurosurgery 1999;45:1172-4.
20. Park ES, Ahn JS, Park JC, Kwon DH, Kwun BD, Kim CJ. STA-ACA bypass using the contralateral STA as an interposition graft for the treatment of complex ACA aneurysms: Report of two cases and a review of the literature. Acta Neurochir (Wien) 2012;154:1447-53.
21. Park J. Saccular aneurysm with basal rupture angiographically depicted as an aneurysm with stalk-like narrow neck: Report of 2 cases. J Neurosurg 2011;114:1065-8.

22. Sanai N, Zador Z, Lawton MT. Bypass surgery for complex brain aneurysms: An assessment of intracranial-intracranial bypass. Neurosurgery 2009;65:670-83.
23. Sluzewski M, Bosch JA, van Rooij WJ, Nijssen PC, Wijnalda D. Rupture of intracranial aneurysms during treatment with Guglielmi detachable coils: Incidence, outcome, and risk factors. J Neurosurg 2001;94:238-40.
24. Stenhouse LM, Knight RG, Longmore BE, Bishara SN. Long-term cognitive deficits in patients after surgery on aneurysms of the anterior communicating artery. J Neurol Neurosurg Psychiatry 1991;54:909-14.
25. Suzuki J, Ohara H. Clinicopathological study of cerebral aneurysms. Origin, rupture, repair, and growth. J Neurosurg 1978;48:505-14.
26. Vergouwen MD, Ilodigwe D, Macdonald RL. Cerebral infarction after subarachnoid hemorrhage contributes to poor outcome by vasospasm-dependent and –independent effects. Stroke 2011;42:924-9.
27. Wilson G, Riggs HE, Rupp C. The Pathologic anatomy of ruptured cerebral aneurysms. J Neurosurg 1954;11:128-42.
28. Yaşargil MG, Fox JL, Ray MW. The operative approach to aneurysms of the anterior communicating artery. In: Krayenbühl H, editor. Advances and Technical Standards in Neurosurgery. New York: Springer-Verlag, Wien; 1975. p. 113-70.
29. Yokoh A, Ausman JI, Dujovny M, Diaz FG, Berman SK, Sanders J, *et al.* Anterior cerebral artery reconstruction. Neurosurgery 1986;19:26-35.