Original Article

Evaluation of Early Recurrence after Coil Embolization for Ruptured Anterior Communicating Artery Aneurysms

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Objective: We retrospectively examined the risk factors for early recurrence in patients with ruptured anterior communicating artery (AcomA) aneurysms who underwent coil embolization.

Methods: Forty-four patients with ruptured AcomA aneurysms who underwent coil embolization between January 2012 and June 2021 were included. Patient backgrounds, anatomical features, intraoperative anticoagulation, and radiological findings before and after treatment were reviewed retrospectively. Univariate analysis was performed separately for each item investigated in the early recurrence (ER) and non-early recurrence (NER) groups. Additionally, the relationship between changes in embolic status (Raymond-Roy classification [RRC]) from immediately after surgery to 2 weeks later and severity of disease was investigated.

Results: Re-treatment was performed in a total of 8 (18.2%) cases. Two cases were detected and treated in the chronic phase with no re-rupture. In the ER group, 6 (13.6%) cases had RRC class 3 filling without evidence of coil compaction on digital subtraction angiography performed 2 weeks after the initial embolization, and were re-treated. The mean intraoperative activated clotting time (ACT; $p = 0.0226$; NER median 189.5 s, ER median 149 s), contralateral A1 diameter ($p = 0.0264$; NER median 0.85 mm, ER median 0.26 mm), and volume embolization rate (VER; $p = 0.02$, NER median 35.57%, ER median 20.86%) were significantly lower in the ER group. The more severe the Hunt and Hess grade, the worse the embolic condition (RRC) tended to be after 2 weeks ($p = 0.0339$).

Conclusion: In this study, factors such as low intraoperative ACT, low VER, contralateral A1 hypoplasia, and condition severity may be associated with early recurrence after acute coil embolization for ruptured AcomA aneurysms.

Keywords ▶ ruptured anterior communicating artery aneurysm, coil embolization, recanalization, intraoperative anticoagulation, early recurrence

Introduction

The efficacy of coil embolization in the acute treatment of subarachnoid hemorrhage caused by a ruptured cerebral aneurysm is well established in large trials, including those comparing it with neck clipping, one of the main treatment modalities for preventing acute re-rupture of ruptured cerebral aneurysms.[1,2] While coil embolization offers the advantage of being relatively minimally invasive with good functional prognosis,[2,3] postoperative issues such as recurrence and the need for re-treatment remain, with a particular concern being the risk of early recurrence and rebleeding.[1,4,5] Anterior communicating artery (AcomA) aneurysms are among the most frequently encountered lesions in clinical practice and are a significant source of subarachnoid hemorrhage. They have been identified as a risk factor for early rebleeding after embolization.[6–9] Over the past decade, coil embolization has been the first-line treatment for acute re-rupture prevention of subarachnoid hemorrhage caused by ruptured AcomA aneurysms at our hospital. In this study, we retrospectively evaluated the factors leading to early recurrence in patients with ruptured

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AcomA aneurysms who underwent coil embolization as an acute treatment.

Materials and Methods

Patient and aneurysm characteristics

Forty-four patients with ruptured AcomA aneurysms who underwent coil embolization between January 2012 and June 2021 were included in the study. Patient backgrounds, anatomical features, clinical courses, and imaging findings before and after treatment were reviewed retrospectively.

The observed variables included age, sex, the severity of disease (Hunt and Hess [H&H]), the maximum diameter of the aneurysm, neck diameter, depth-to-neck ratio, ipsilateral and contralateral A1 diameters (iA1 and cA1), and their ratios (the diameters of the initial, middle, and terminal points of the bilateral A1 segments were measured in the anteroposterior view on initial 3D computed tomography (CT) angiography; the diameter of the A1 segment was calculated as an average of 3 measurements) (**Fig. 1**). Ipsilateral A1 indicates the dominant side, which coincides with the approach side. When the diameters of the left and right A1s were equal, the side that was easier to approach was defined as the ipsilateral A1, based on its relationship to the direction of aneurysmal growth. Data on other variables including, volume embolization rate (%VER), intraoperative activated coagulation time (ACT) (intraoperative mean of activated coagulation time [ACTm]), evaluation of embolic status at the end of procedure and 2 week after the procedure (Raymond–Roy classification [RRC]),[10] perioperative complications (intraoperative rupture, ischemic complications), and outcome (modified Rankin Scale [mRS]: before onset, after 3 months) were collected.

This survey was conducted with the approval of the Ethics Committee of Omuta City Hospital (approval no. 2208).

Procedures and perioperative management

Initial treatment

All embolization procedures were performed under general anesthesia using a biplane digital subtraction angiography (DSA) device. Postoperatively, patients remained under general anesthesia until the following day.

The working angle was determined from the preoperative DSA and 3-dimensional (3D) DSA images, and embolization was performed using either simple or adjunctive techniques (double-catheter technique, balloon-assisted technique). For each additional coil, DSA was performed to confirm embolic status. The procedure was terminated when the microcatheter kicked back and became difficult to reinsert or when no more coils could be added. Intraoperative antithrombotic therapy consisted of continuous heparin-saline (5000 IU/L saline mixture) from the A line, with no heparin bolus administration. Antiplatelet agents (ozagrel sodium 80 mg and cilostazol 200 mg) were administered for 2 weeks postoperatively. Intraoperative ACT measurements were taken immediately after puncture, shortly before insertion of the first coil, every 30 min thereafter, and at the end of the procedure.

Evaluation and follow-up of postoperative aneurysms

Postoperative early recurrence was assessed by DSA at 2 weeks and subsequently monitored using non-contrast magnetic resonance angiography (MRA; 1.5T magnetic strength) and plain radiography (to evaluate coil mass shape). MR imaging (MRI) and plain radiography were performed at 1 week, 1 month, 3 months, 6 months, and 1 year postoperatively and annually thereafter. DSA in the chronic phase was performed when recurrence was suspected by MRI and plain radiography. All cases with RRC 3 on DSA at 2 weeks were considered early recurrence, including those with no change in embolic status. Early recurrence of RRC class 3 detected 2 weeks after the initial treatment was re-treated within 2 weeks, in all but 1 case. The indication for early re-treatment was based on RRC class 3 recurrence findings on DSA.

Statistical analyses

The relationship between early recurrence and each variable was analyzed using univariate analysis. Fisher's exact test was applied for categorical variables, and Wilcoxon rank-sum test was used for continuous variables. There was a change in embolic status (RRC) immediately after surgery, and, 2 weeks later, Cochran–Armitage trend test was used to determine if there was a relationship with severity of disease.

A p-value of <0.05 was considered statistically significant. Statistical analyses were performed using JMP Pro software (version 15; SAS Institute, Cary, NC, USA).

Results

There were 44 patients with ruptured AcomA aneurysms who underwent coil embolization in acute phase during

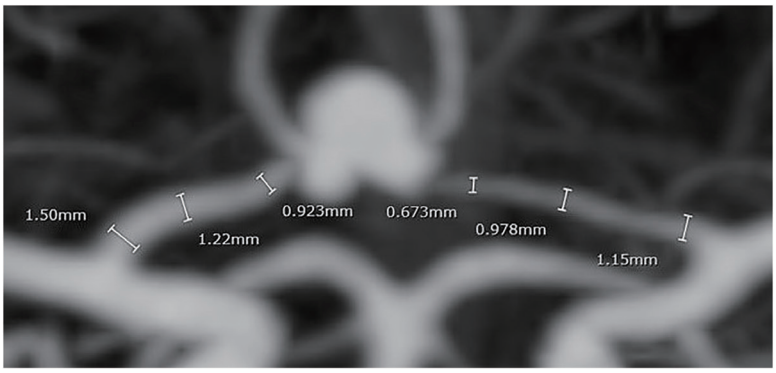

Fig. 1 Calculation of A1 diameter. The diameters of ipsilateral (iA1) and contralateral (cA1) segments and their ratios were measured at the initial, middle, and terminal points of the bilateral A1 segments in the anteroposterior view on the initial 3D CT angiograms. The A1 segment diameter was calculated as the average of 3 measurements.

Table 1 Combined outcomes for all variables

Number (n)		44
Age (median + whole range) years		62.5 (39–92)
Sex (n)		Men: 19; Women: 25
H&H grade (n)		GI: 1; GII: 18; GIII: 16; GIV: 1; GV: 8
Fisher group (n)		Group 2: 5; Group 3: 39
*Dome, mm (IQR)		4.83 (3.78–6.90)
*Neck, mm (IQR)		2.58 (2.05–3.37)
*Depth/Neck ratio, (IQR)		1.63 (1.32–2.25)
*iA1, mm (IQR)		1.53 (1.21–2.01)
*cA1, mm (IQR)		0.80 (0.59–1.08)
*cA1/iA1 ratio, (IQR)		0.51 (0.32–0.80)
*%VER, (IQR)		32.37 (23.58–40.28)
*ACTm, sec (IQR)		186.3 (155–227.70)
R-R-Classification, n	Immediate 44	C1: 32; C2: 8; C3: 4
	2 weeks after **42	C1: 26; C2: 10; C3: 6
Complications, n (%)	Intraoperative rupture	4 (9.1)
	Ischemic complication	Overall 6 (13.6); Permanent 2 (4.4)
mRS, n (%)	Before onset	0–2: 44 (100); 3–5: 0 (0)
	3 months after onset	0–2: 31 (70.5); 3–6: 9 (20.5); Unknown: 4 (9)
Early recurrence, n (%)		6 (13.6)
All recurrence, n (%)		8 (18.2)
All re-ruptures after coil embolization		1 (2.3)

*Continuous variables are expressed as median and interquartile range (IQR).
**Two patients died, 1 from severe vasospasm and 1 from cerebral herniation due to uncontrolled intracranial hypertension.
ACTm, mean activated coagulation time; cA1, contralateral A1 segment; H&H, Hunt and Hess; iA1, ipsilateral A1 segment; IQR, interquartile range; mRS, modified Rankin Scale; R-R-Classification, Raymond-Roy classification; VER, volume embolization ratio

the study period. Median age was 62.5 years, 56.8% were women, H&H grade ≥IV (20.5%), and Fisher group 3 (88.6%). Aneurysms were characterized by a median maximum dome diameter of 4.83 mm (interquartile range [IQR] 3.78–6.90), a median neck diameter of 2.58 mm (IQR 2.05–3.37), a median depth/neck ratio of 1.63 (IQR 1.32–2.25) (**Table 1**).

The early recurrence (ER) group and non-early recurrence (NER) group were separated for each study item (**Table 2**). The mean intraoperative activated clotting time

Table 2 Univariate analysis for ER and NER groups

Variables		NER group (n = 38)	ER group (n = 6)	*p*-value
Age, years		65 (56.75–79)	59 (53–65.75)	0.2341
Dome (mm)		4.83 (3.55–6.96)	5.41 (4.17–7.00)	0.6465
Neck (mm)		2.45 (2.04–3.32)	3 (2.58–4.06)	0.293
Depth/Neck ratio		1.68 (1.37–2.26)	1.15 (0.92–2.26)	0.0909
iA1 (mm)		1.57 (1.21–2.07)	1.47 (1.17–1.61)	0.395
cA1 (mm)		0.85 (0.67–1.11)	0.26 (0–0.75)	0.0264
cA1/iA1 ratio		0.52 (0.38–0.80)	0.17 (0–0.65)	0.1478
%VER (%)		35.57 (24.55–41.28)	20.86 (18.13–28.26)	0.02
ACTm (sec)		189.5 (158.25–230.50)	149 (145.76–186.3)	0.0226
Sex, women		W: 21 (55.3)	W: 4 (66.7)	0.6843
H&H grade ≥IV		6 (15.8)	3 (50)	0.0891
Fisher group		Group 2: 5 (5.3) Group 3: 33 (86.8)	Group 2: 0 (0) Group 3: 6 (100)	1
Intraoperative complications	Rupture	4 (10.5)	0 (0)	1
	Ischemia	5 (13.2)	1 (16.7)	1

ACTm, %VER, and cA1 values were significantly lower in the ER group than in the NER group.
Continuous variables are expressed as median and interquartile range (IQR), and categorical variables are expressed as number (%).
A *p*-value <0.05 for continuous variables and a 2-sided *p*-value <0.05 for categorical variables were considered significant.
ACTm, intraoperative mean activated coagulation time; cA1, contralateral A1 segment; ER, early recurrence; H&H, Hunt and Hess; iA1, ipsilateral A1 segment; NER, non-early recurrence; VER, volume embolization ratio

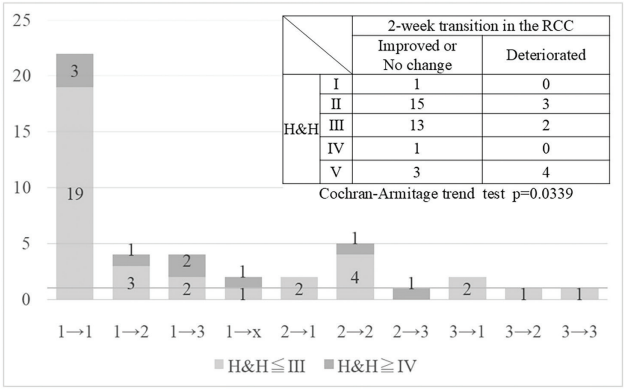

Fig. 2 Changes in RRC from immediately after surgery to 2 weeks later. Of the 40 patients with RRC1 or 2 immediately after surgery, 5 deteriorated to RRC3 after 2 weeks. Four patients had RRC3 immediately postoperatively, 3 of which improved to RRC1 or 2 after 2 weeks. There was a significant trend between H&H severity and change in RRC (p = 0.0339 Cochran–Armitage trend test). The vertical axis of the graph shows the number of cases, and the horizontal axis shows the RRC divided by a 2-week transition. In the axis labels, numbers → numbers indicate the change from RRC immediately after surgery to RRC 2 weeks later. The numbers in the graph bars indicate the number of cases. The table in the graph shows the relationship between severity and change in RRC. 1→X: cases in which DSA could not be performed 2 weeks after surgery. H&H, Hunt and Hess grade; RRC, Raymond-Roy classification

(p = 0.0226; NER median 189.5 s [IQR 158.25–230.5], ER median 149 s [IQR 145.76–186.3]), contralateral A1 diameter (p = 0.0264; NER median 0.85 mm [IQR 0.67–1.11], ER median 0.26 mm [IQR 0–0.75]), and volume embolization rate (p = 0.02, NER median 35.57% [IQR 24.55–41.28], ER median 20.86% [IQR 18.13–28.26]) were significantly lower in the ER group. Although not significantly different, severe cases of H&H grade ≥IV (p = 0.0891, NER 15.8%, ER 50%) and aneurysms with low depth/neck ratios (p = 0.0909, NER median 1.68 [IQR 1.37–2.26], ER median 1.15 [IQR 0.92–2.26]) also tended to recur earlier. In addition, in the Cochran–Armitage trend test, the more severe the H&H grade, the worse the embolic condition (RRC) tended to be after 2 weeks (p = 0.0339, **Fig. 2**).

The initial postoperative RRC was class 1–2 in 40 cases (90.9%) and class 3 in 4 cases (9.1%). DSA at 2 weeks

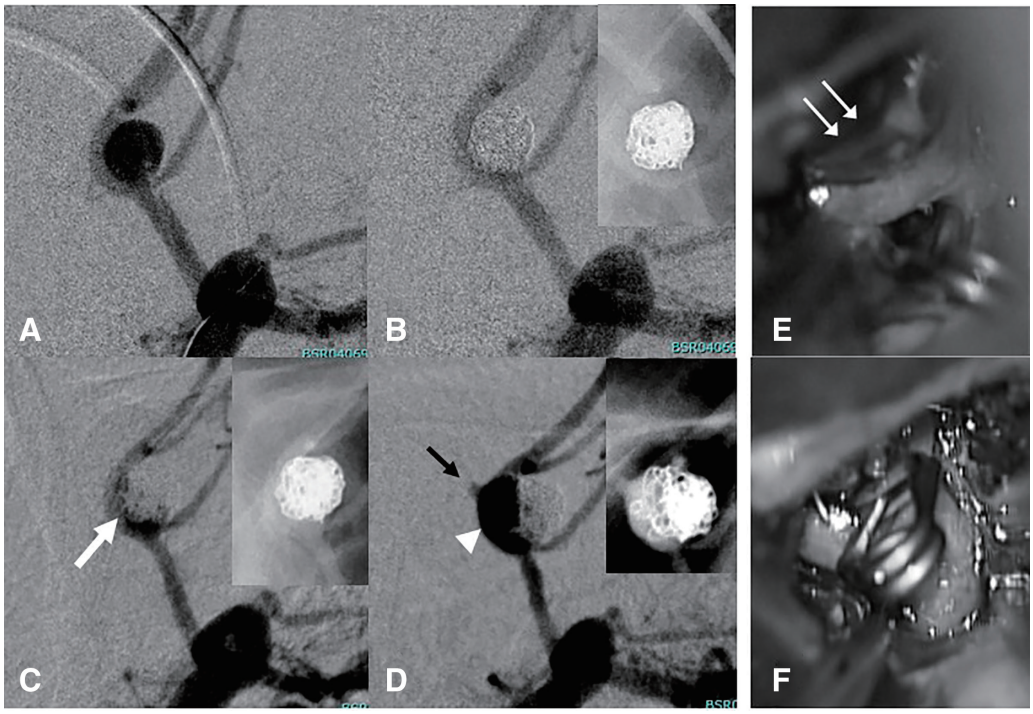

Fig. 3 A case (Case 1) of rebleeding after successful coil embolization. (**A**–**D**) Anterior working view with left internal carotid angiography. (**E**, **F**) Intraoperative findings during neck clipping with right frontotemporal craniotomy. (**A**) Before coil embolization. (**B**) Immediately after initial coil embolization. (**C**) On day 14, no change in the shape of the coil mass, but a slight influx of the contrast agent was observed (white arrow). (**D**) On day 43, the aneurysm re-ruptured and regrowth with coil compaction (white arrowhead). A new bleb is observed in the recurrent area (black arrow). (**E**) The coil mass was observed through the regrown part (double white arrow). (**F**) Temporary clip was applied to the left A1, the coil mass was removed, and neck clipping was performed. Thereafter, the surrounding area was dissected, but the new bleb could not be clearly seen because the wall of the aneurysm was already greatly deformed.

was performed in 42 patients (95.5%), with RRC class 3 observed in 6 patients (13.6%). Five of the patients with RRC class 1 or 2 after initial surgery showed deterioration to class 3. One case with RRC 3 at the time of initial treatment and expected improvement of embolic state due to thrombosis but with no improvement after 2 weeks of DSA was also considered early recurrence. Three cases (50%) of the RRC class 3 at 2 weeks were severe cases with H&H grade ≥IV. On the other hand, 3 cases improved from RRC class 3 to RRC class 1 or 2 at 2 weeks after the initial surgery, all of which were mild cases with H&H grade ≤ III. Re-treatment was performed in 8 (18.2%) cases. Two patients were detected and treated in the chronic phase without any re-rupture. In these chronic re-treatment cases, 1 patient underwent additional coil embolization for simple coil compaction and stent-assisted coil embolization in 1 case of aneurysm re-enlargement.

In 6 patients, RRC 3 early recurrence was confirmed by DSA performed 2 weeks after the initial treatment, leading to re-treatment. In 1 of these 6 cases, a part of the coil loop had deviated into the parent artery at the 2-week DSA, and had deteriorated to RRC class 3 body filling (BF). Consequently, additional embolization was performed using a simple technique. The other 5 patients had RRC class 3 BF without coil compaction or aneurysmal re-enlargement on DSA performed 2 weeks after the initial embolization. One of these 5 cases was followed up conservatively, because the patient exhibited impaired consciousness and decreased ADL due to concomitant normal pressure hydrocephalus (NPH), and treatment for NPH was prioritized over re-treatment of the aneurysm. Clinical symptoms tended to improve after ventriculoperitoneal shunt, but re-rupture occurred 43 days after the initial treatment. At the time of re-rupture, DSA showed coil compaction, regrowth of the aneurysm, emergence of a new bleb, and we performed neck clipping as re-treatment. (Case 1, **Fig. 3**). In the remaining 4 patients, re-embolization was performed within 2 weeks after recurrence was detected, simply by adding coils without the use of stents.

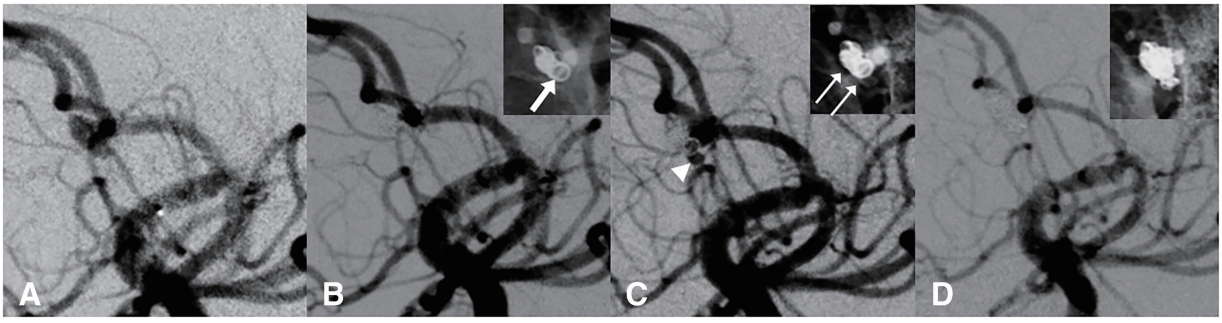

Fig. 4 A case (Case 2) of early recurrence without coil compaction after coil embolization. Anterior working view with left internal carotid angiography. (**A**) Before coil embolization. (**B**) Immediately after the initial coil embolization. DSA showed complete obliteration, but a rough coil compartment in the lower part of the aneurysm (white arrow) was thrombosed and could not be coiled any further. (**C**) On day 14, the lower portion of the aneurysm, which had a rough coil compartment at the time of initial embolization, was recanalized without coil compaction (white double arrow). In addition, a new bleb was found below the aneurysm, which was not seen in the initial DSA (white arrowhead). (**D**) On day 17, second coil embolization was performed with the balloon neck remodeling technique.

Discussion

The frequency of recurrence requiring re-treatment after coil embolization at all sites of ruptured cerebral aneurysms has been reported to range from 10.3% to 18%. Studies focusing on early rebleeding after coil embolization of ruptured cerebral aneurysms have reported rates ranging from 1.0% to 3.8%.[6–9,11,12] Kim et al.[9] reported rates as high as 4.9% for AcomA aneurysms. The reported risks of early rebleeding include relatively small diameter (<4–6 mm), incomplete occlusion, low VER, adjacent hematoma, presence of bleb, AcomA location, contralateral A1 hypoplasia, increasing Fisher grade, fibrinolytic therapy for intraoperative thrombotic complications, and postoperative anticoagulation.[6–9,12] Early rebleeding is most common within 3 days of coil embolization,[6,7,9,12] and there have been reports of recanalization without coil compaction or regrowth.[9,12] The cause of early rebleeding is thought to be the dissolution of a thrombus that forms within the coil mass due to inadequate embolization, leading to recanalization.[9,12] Although we did not observe very early rebleeding within 3 days after embolization, our early recurrent cases share several characteristics similar to those reported in the literature, including low VER, and contralateral A1 hypoplasia and recanalization without coil compaction or regrowth. Also, the more severe the H&H grade, the worse the embolic condition (RRC) tended to be after 2 weeks.

In recent years, reports have demonstrated not only the benefits but also the safety of heparin use for ischemic complications during coil embolization of acute ruptured cerebral aneurysms.[13] There seems to be a consensus that aggressive anticoagulation during embolization is essential, even in the acute phase of rupture. However, some reports indicate that postoperative anticoagulation is a risk factor for early rebleeding.[7,9,12] Kim et al.[9] suggested that antithrombotic therapy should be minimized in the postoperative period to promote thrombosis within aneurysms after coil embolization. During the study period, at our institution, intraoperative anticoagulation for embolization of acute ruptured cerebral aneurysms was limited to continuous administration from the A line because of concerns about the possibility of intraoperative rupture and the difficulty of hemostasis in such cases. However, in all 4 cases of intraoperative rupture in this study, hemostasis was achieved by prompt heparin antagonism and additional coil insertion. There were no cases where intraoperative rupture appeared to have affected the prognosis. Of course antithrombotic therapy is important for managing the risk of perioperative ischemic complications. Moreover, as discussed below, we speculate that thrombus formation in the aneurysm too early in the operation may result in low VER, and we believe that intraoperative anticoagulation is important to prevent this phenomenon.

Uneven coil distribution and low VER within an aneurysm can result in residual intra-arterial blood flow after coil embolization and re-dissolution of the thrombus once it forms in the coil mass.[9,11] In Case 2 (**Fig. 4**), the point of rupture—the bleb—would have developed thrombotic occlusion immediately after the rupture. An inadequate coil compartment was formed in the area adjacent to the bleb, and thrombosis progressed without adequate coil insertion, leading to temporary complete occlusion on DSA. When recurrence was confirmed, the thrombus in this compartment and the adjacent bleb had dissolved and could be visualized with a contrast medium. We should not

ignore the fact that 5 of the patients who were RRC class 1 after the initial surgery deteriorated to RRC class 3 just 2 weeks later (**Fig. 2**). When embolization is completed with low VER or compartment formation, there is a risk of early recanalization, even if complete obliteration is achieved on DSA. Especially in cases of inadequate intraoperative anticoagulation, intra-aneurysmal thrombus formation may occur before the coils are fully filled, potentially leading to aneurysm occlusion. The possibility that the thrombus may dissolve postoperatively, allowing early recanalization of the aneurysm, should also be considered. In the embolization of ruptured cerebral aneurysms, adequate "intraoperative" anticoagulation and minimal "postoperative" anticoagulation are crucial. Since the observation period of the present study, 2000–5000 units of heparin have been administered bolus after insertion of the sheath at the time of initial embolization to promptly prolong ACT to at least twice the previous value, or 220–300 s in actual measured values.

Of course, we should also consider the possibility that the preoperative DSA failed to delineate the entire aneurysm due to thrombus formation already present in the aneurysm. It has been reported that patients with subarachnoid hemorrhage have increased systemic coagulability, which is more likely to more severe cases.[14] It is also possible that in severe cases, the contrast medium may have an inferior ability to delineate aneurysms due to increased intracranial pressure compared to mild cases. In our case of early recurrence, 3 out of 6 cases were severe cases with H&H grade ≥IV. In addition, severe cases tended to have worse embolic status just after 2 weeks compared to mild cases.

Studies with large sample sizes and meta-analyses have shown that the larger the aneurysm, the higher the risk of recurrence after the entire postoperative period of coil embolization.[15,16] In our chronic recurrence cases, 1 large size case was due to simple coil compaction, and the other small size and wide neck case was due to aneurysm coil compaction and regrowth. On the other hand, early recurrence cases were characterized by neither coil compaction nor aneurysmal regrowth. As for the difference in mechanism between early recurrence and chronic recurrence, we think that early recurrence may be due to dissolution of the thrombus within the aneurysm, while chronic recurrence may be due to coil compaction or regrowth of the aneurysm, which causes reopening.

Our study focused on early recurrence 2 weeks after coil embolization of ruptured AcomA aneurysms, but because the aneurysms were small and few in number, there was no statistically significant relationship between several anatomic characteristics, including size, and early recurrence.

However, contralateral A1 hypoplasia has been reported as a risk for early recurrence after embolization of ruptured AcomA aneurysms, which was a common anatomic feature in this study.

The coexistence rate of contralateral A1 hypoplasia and aplasia in AcomA aneurysms has been reported to be very high (36%–61.1%).[17–19] Contralateral A1 hypoplasia and aplasia are associated with aneurysm formation and enlargement,[20] rupture,[21] and recurrence due to coil compaction after coil embolization.[22] Many of these events are associated with increased hemodynamic and wall stress due to contralateral A1 hypoplasia. From our results, we infer that hemodynamic stress on aneurysms caused by contralateral A1 hypoplasia not only affects coil compaction and aneurysm regrowth, which can occur at any time after coil embolization, but also affects the early recurrence characterized by the absence of coil compaction or aneurysmal regrowth.

Dorfer et al.[23] reported that coil compaction and aneurysm re-enlargement necessitate re-treatment in patients with significant intra-aneurysmal blood flow or the appearance of new sacs. Modified Raymond-Roy Classification (mRRC) 3b is described as residual aneurysm with contrast along the aneurysm wall, which is unstable and has a high risk of recurrence.[24] Funakoshi et al.[25] reported that the risk of rebleeding is high in cases of aneurysmal re-enlargement, the appearance of new sacs, and intra-aneurysmal blood flow classified as grade 3b in the mRRC.

All of the 6 early recurrences we experienced were RRC class 3 at just 2 weeks postoperatively, and 5 of them corresponded to the-later reported mRRC class 3b. One of the 5 patients who did not receive early re-treatment had rebleeding, but the others who had early recurrence were treated promptly and did not experience rebleeding. Early re-treatment of patients with early recurrence corresponding to mRRC class 3b is considered appropriate.

Limitations

There are several limitations to this study: retrospective design, small size of most of the aneurysms, and small sample size. Therefore, detailed statistical analysis of the risk factors was not possible. Although there was just 1 primary surgeon, it is possible that there were changes in procedural preferences and advancements in technology

during the long 10-year study period. Selection bias and protocol deviations are also expected. Another limitation of this study is that the total intraoperative heparin dose could not be calculated because the intraoperative heparin administration rate from A line was not constant, and was done manually, and the time required for the surgery was not taken into account.

Conclusion

In this study, factors such as low intraoperative ACT, low VER, contralateral A1 hypoplasia, and severe cases may be associated with early recurrence after acute coil embolization for ruptured AcomA aneurysms. Whether adequate intraoperative anticoagulation prevents low VER and contributes to the prevention of early recurrence needs to be further investigated under a rigorous protocol with a larger number of patients.

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Disclosure Statement

The authors declare that they have no conflicts of interest.

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