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Internal Trapping of a Growing Ruptured Dissecting Aneurysm of the A1 Segment: A Case Report and Literature Review

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Abstract

A 47-year-old man presented with sudden-onset headache and Fisher group 3 subarachnoid hemorrhage. The World Federation of Neurological Surgeons grade was II. Digital subtraction angiography (DSA) only showed a vessel wall irregularity in the A1 segment of the right anterior cerebral artery (ACA), but an obvious bleeding source was not detected. Repeat angiography showed a tiny aneurysmal dilatation in the A1 segment with an intimal flap. The aneurysm enlarged on subsequent angiograms. Dissecting aneurysm was diagnosed, and the patient underwent internal trapping of the A1 segment to prevent rerupture. Postoperative DSA showed complete obliteration of the dissected segment. Magnetic resonance imaging showed a clinically silent cerebral infarction in the territory of the A1 segment perforators. Parent vessel occlusion for a dissected A1 segment can be effective, provided that sufficient collateral blood flow from the contralateral ACA is observed. We recommend endovascular trapping in this setting and hope that fellow clinicians select this approach for this rare pathology.

Keywords: anterior cerebral artery dissection, internal trapping, subarachnoid hemorrhage

Introduction

Intracranial artery dissection may result in subarachnoid hemorrhage (SAH), intracerebral hemorrhage, or cerebral infarction. Compared with dissection of other intracranial arteries, dissection of the anterior cerebral artery (ACA) is rare, especially in the proximal segment. We report our experience with a patient who had a ruptured dissecting aneurysm of the A1 segment of the right ACA.

Case Report

A 47-year-old man presented with sudden-onset headache, vomiting, and impaired consciousness. Computed tomography of the head showed Fisher group 3 subarachnoid hemorrhage (Fig. 1A). The World Federation of Neurological Surgeons grade was II. Digital subtraction angiography (DSA) and three-dimensional rotational angiography showed a vessel wall irregularity in the A1 segment of the right ACA, but an obvious bleeding source was not detected (Fig. 1B, C). Repeat angiography on day 5 showed a 1.6 × 1.5 × 1.5 mm aneurysmal dilatation and an intimal flap in the A1 segment of the right ACA (Fig. 2A, D, G). A dissecting aneurysm of the right A1 segment was diagnosed. The patient was treated conservatively based on our expectations of spontaneous aneurysm remodeling. However, on day 23, angiography showed an increase in aneurysm size (2.2 × 2.1 × 2.0 mm) despite the improvement in vessel wall irregularity (Fig. 2B, E). Fourteen days later, angiography showed even more aneurysmal growth (Fig. 2C, F, H). The increased risk of rerupture was considered.

The Matas test was performed, which showed sufficient collateral blood flow from the contralateral ACA to the ipsilateral ACA territory (Fig. 3A). We performed trapping of the A1 segment, prioritizing rupture prevention over the risk of cerebral infarction due to the occlusion of the perforating arteries of the A1 segment.

First, a 7 Fr guiding catheter was sent to the right internal carotid artery, and a 4.2 Fr distal access catheter was

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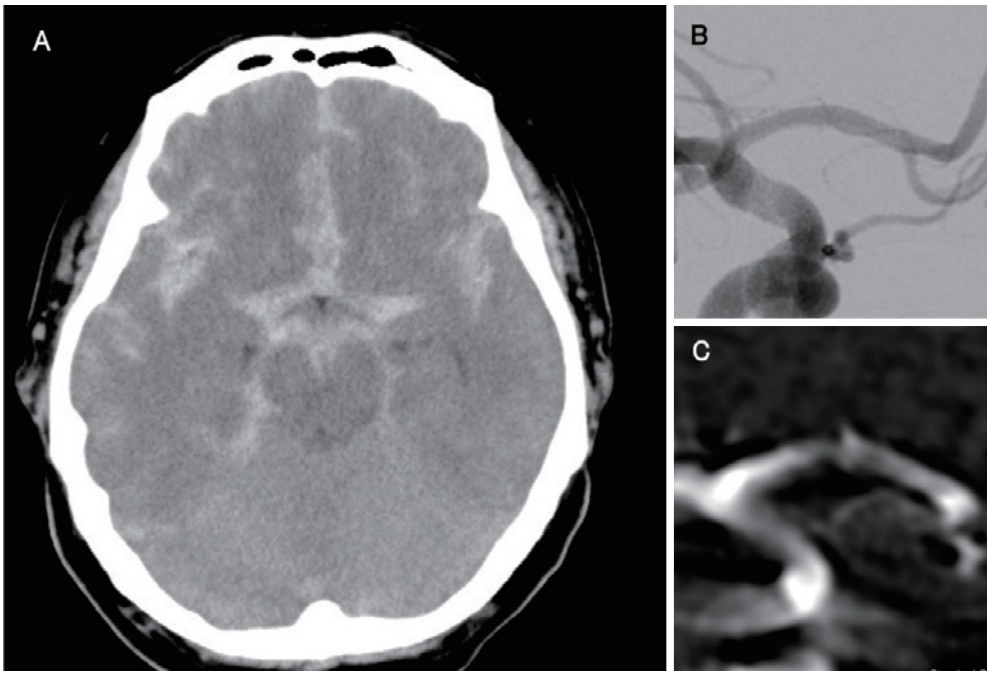

Fig. 1 Computed tomography of the head showed diffuse subarachnoid hemorrhage (A). Digital subtraction angiography (B) and three-dimensional rotational angiography (C) showed a wall irregularity in the A1 segment of the right anterior cerebral artery.

guided to the carotid siphon. After the navigation of a microcatheter into the distal A1 segment, seven detachable coils were deployed to obliterate the dissected A1 segment (Fig. 3B, C). Postoperative bilateral internal carotid angiography showed complete obliteration of the dissected segment (Fig. 3D). Magnetic resonance imaging showed cerebral infarction in the territory of the perforating arteries of the A1 segment (Fig. 3E). However, the patient did not exhibit new neurological deficits nor did he develop vasospasm or hydrocephalus during his hospitalization. On day 51, he was transferred to another hospital for rehabilitation because of mild cognitive deficits. His modified Rankin scale score was 1.

Discussion

Dissection of the ACA is rare, particularly in the proximal segment. Mizutani reported that only 5.3% of intracranial artery dissections involve the ACA.[1] The etiology of dissection remains unknown. A summary of 34 patients with A1 dissection from 25 studies is shown in Table 1.[2-25] Twenty-nine presented with SAH and five with cerebral infarction. Interestingly, most cases were from Japan. Debette et al. also reported that most reports of patients with intracranial artery dissection were from Asia.[26] It is unclear whether this is attributable to publication bias, differences in disease prevalence among ethnic groups, or both.

SAH group

Among the 29 patients who presented with SAH, direct surgery or endovascular treatment was performed in 26. Time from onset to treatment was reported in 17 patients: 11 underwent treatment within 1 week; 4, within 1 month; and 2, after 1 month. Among the three patients who received conservative care, the outcome was favorable in two (good recovery and moderate disability, respectively) and death in one.

Timing of dissection diagnosis was reported in 26 of the 29 SAH patients. Dissection was not identified at the initial examination in 5 of the 26 (19.2%). Dissection findings were identified on repeat DSA within 1 week in three of these five. In our patient, initial DSA showed only a vessel wall irregularity in the A1 segment. Dissection was diagnosed on repeat angiography performed on day 5 based on the presence of a small aneurysm and an intimal flap.

Mizutani et al. reported that the healing process for cerebral dissecting aneurysms presenting with SAH begins with neointimal proliferation at 1 week and may continue even after 1 month, depending on the extent of the wall injury.[1] In our patient, we initially selected conservative treatment and anticipated eventual aneurysm dissipation after vascular remodeling occurred. However, because the aneurysm continued to grow, internal trapping was performed.

Mitsuhara et al. reported that A1 segment dissection with SAH is associated with poor prognosis; only 6 of 21 patients in their series (28.6%) experienced good recov-

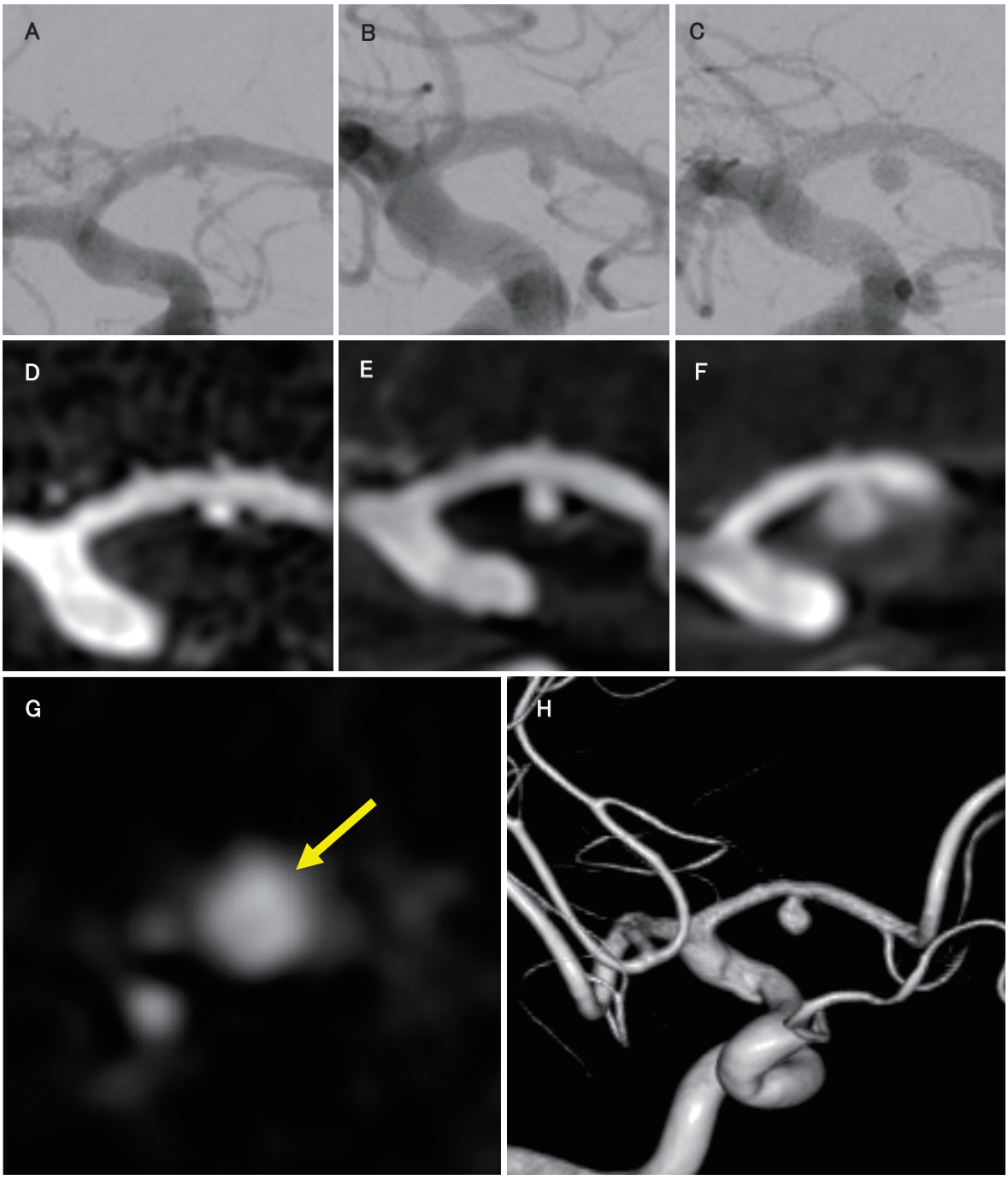

Fig. 2 Digital subtraction angiography (right internal carotid artery injection) on day 5 (A), day 23 (B), and day 37 (C). Three-dimensional rotational angiography (maximum intensity projection sagittal view) on day 5 (D), day 23 (E), and day 37 (F). Both imaging modalities demonstrated vessel wall abnormalities and aneurysmal enlargement over time. An intimal flap (arrow) was visible on three-dimensional rotational angiography (maximum intensity projection coronal view) on day 5 (G). Three-dimensional rotational angiography on day 37 clearly showed enlargement of the aneurysm (3.5 × 3.5 × 3.5 mm) and a vessel wall irregularity in the A1 segment (H).

ery.[23] By contrast, 14 of the 29 SAH patients in our review (48.3%) experienced good recovery, and all 4 who underwent endovascular treatment experienced a good outcome. Given that we included endovascular treatment cases, the outcomes in our review may be better than those in the previous one. Endovascular treatment is less invasive than surgery, and it enables consideration of collateral blood vessels from the contralateral side.

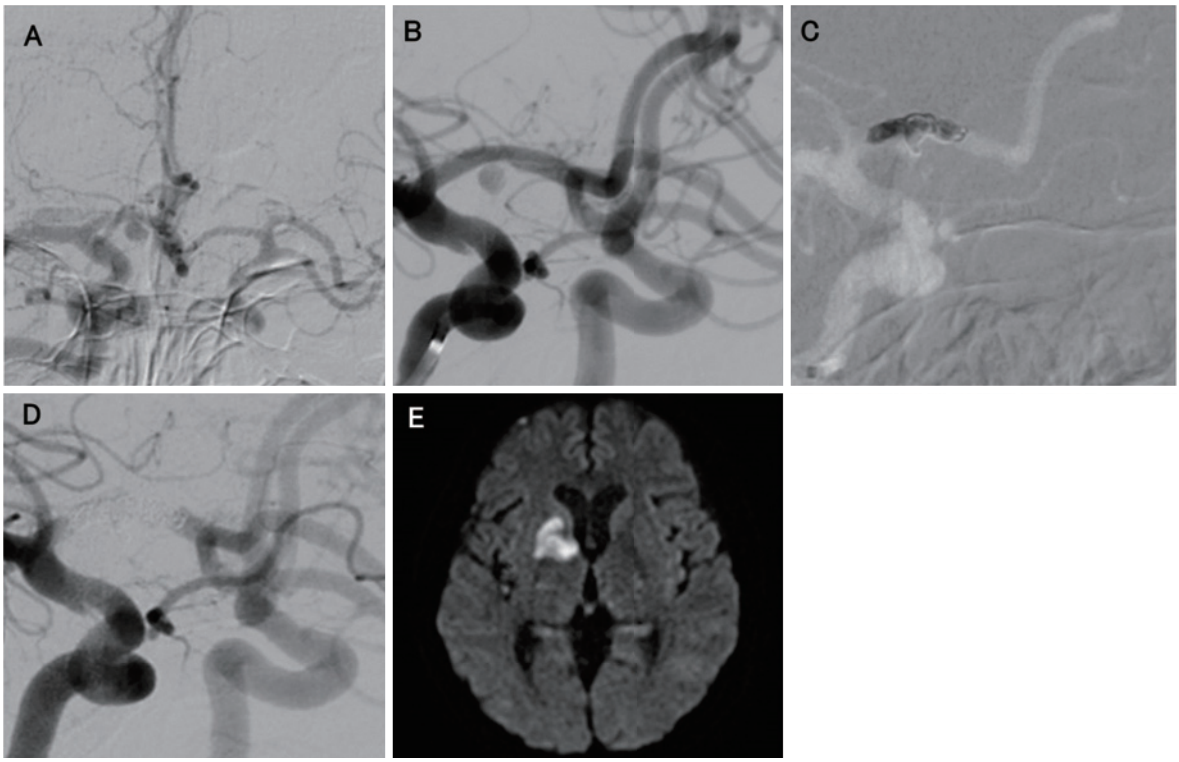

Fig. 3 The Matas test showed sufficient collateral blood flow from the contralateral anterior cerebral artery (A). Bilateral internal carotid artery (ICA) angiography (B). No intraoperative aneurysmal rupture was observed during the internal trapping of the A1 segment (C). Bilateral ICA angiography after trapping showed complete obliteration of the dissected segment (D). Magnetic resonance imaging after trapping showed cerebral infarction in the territory of the A1 segment perforators (E).

Infarction group

All five patients who presented with cerebral infarction in our review initially received conservative therapy, but two ultimately required surgical intervention. The first experienced SAH after 1 month and underwent surgical clipping and coating; the other developed a saccular-like dissecting aneurysm after 2 years and underwent surgical trapping. All five cases who presented with cerebral infarction experienced good recovery.

Therapeutic choice

Deconstructive surgery (surgical trapping with or without bypass and internal trapping) was performed in 15 of the 28 patients who underwent surgical treatment (53.6%). Of these, seven (46.7%) experienced good recovery. When considering deconstructive surgery, it is important to evaluate for the patency of the anterior communicating artery and the presence of contralateral A1 segment aneurysms. Kashimura et al. performed bypass surgery in addition to trapping in a patient with a fusiform aneurysm of the proximal A1 segment because of insufficient collateral blood flow from the contralateral ACA.[17] In our patient, the Matas test showed sufficient collateral blood flow from the contralateral ACA territory. Thus, we performed internal trapping without bypass.

Postoperative magnetic resonance imaging in our patient showed a cerebral infarction in the territory of the A1 segment perforators that was clinically silent. Perlmutter et al. reported that this segment has an average of eight basal perforators (range, 2-15) other than the Heubner artery.[27] In our review, all four patients with postoperative infarction experienced good recovery.

Reconstructive surgery (clipping, coating, wrapping, and endovascular treatment with a stent or flow diverter) was performed in 13 of the 28 patients who underwent surgery (46.4%). These patients had insufficient collateral blood flow from the contralateral ACA. Eight of them (61.5%) experienced good recovery.

Although dissection of the A1 segment of the ACA is rare and its etiology remains unknown, endovascular therapy may be an effective treatment option.

Table 1 Thirty-four patients with dissection of the A1 segment of the anterior cerebral artery

No.	Author/year	Age	On-set	Grade	Initial examination findings	Time to detection of dissection	DSA findings	Treatment	Time to treatment	Postoperative ischemic event	Re-rupture	Out-come
1	Shigemori et al./1987[2]	54	SAH	WFNS grade I	N	day 19	Fusiform dilatation	DS (Clipping and coating)	N/A	N/A	N	MD
2	Matsumoto et al./1997[3]	58	SAH	WFNS grade III	Y	-	Stenosis with dilatation	DS (Trapping)	day 4	Vasospasm	N	MD
3	Honda et al./1997[4]	48	CI→ SAH	-	Y	-	String and pearl sign	DS (Clipping and coating)	1 month	N	N	GR
4	Sato et al./1998[5]	59	SAH	WFNS grade V	Y	-	Fusiform dilatation	DS (Clipping)	day 2	N/A	N	MD
5	Hashimoto et al./1999[6]	61	SAH	N/A	Y	-	Stenosis with dilatation	DS (Clipping and coating)	N/A	N	N	GR
6	Tanikawa et al./1999[7]	64	SAH	N/A	N/A	-	N/A	DS (Clipping and coating)	N/A	N/A	N/A	GR
7	Hirao et al./2001[8]	58	SAH	WFNS grade III	Y	-	String and pearl sign	DS (Trapping)	N/A	N	N	GR
8		39	CI	-	Y	-	Fusiform aneurysm	Conservative	-	-	-	GR
9	Nomura et al./2001[9]	5	SAH	WFNS grade III	Y	-	Obstruction	DS (Trapping)	day 1	N	N	MD
10	Hatayama et al./2001[10]	50	SAH	WFNS grade V	Y	-	Stenosis with dilatation	DS (Trapping)	day 38	N/A	N/A	MD
11	Ueno et al./2001[11]	62	SAH	WFNS grade IV	Y	-	Fusiform dilatation	DS (Trapping)	day 2	N	N	MD
12	Mori et al./2002[12]	48	SAH	N/A	Y	-	Double lumen	DS (Wrapping)	day 2	N	N	MD
13		43	SAH	WFNS grade I	Y	-	Fusiform dilatation	DS (Clipping and coating)	day 2	N	N	GR
14	Ohkuma et al./2003[13]	39	SAH	N/A	N/A	N/A	Stenosis with dilatation	DS (Clipping and coating)	N/A	*	*	VS
15		66	SAH	N/A	N/A	N/A	Stenosis with dilatation	DS (Trapping)	N/A	*	*	VS
16		66	SAH	N/A	N/A	N/A	Stenosis with dilatation	DS (Trapping)	N/A	N/A	N/A	MD
17		43	CI	-	N/A	N/A	Stenosis	Conservative	-	-	-	GR
18		67	CI	-	N/A	N/A	Stenosis with dilatation	Conservative	-	-	-	GR
19	Leach et al./2004[14]	39	SAH	N/A	N	day 2	Focal dilatation	DS (Trapping)	day 2	Infarction (caudate nucleus)	N	GR
20	Iwashita et al./2005[15]	53	CI	-	Y	-	Intimal flap→ Saccular-like aneurysm	DS (Trapping)	2 years	N	N	GR

Table 1 Thirty-four patients with dissection of the A1 segment of the anterior cerebral artery (continued)

No.	Author/year	Age	On-set	Grade	Initial examination findings	Time to detection of dissection	DSA findings	Treatment	Time to treatment	Postoperative ischemic event	Re-rupture	Out-come
21	Thines et al./2006[16]	65	SAH	N/A	Y	-	Fusiform dilatation	Conservative	-	-	N	GR
22		41	SAH	N/A	Y	-	Fusiform dilatation	DS (Wrapping)	N/A	N	Y (2 weeks after surgery)	MD
23	Kashimura et al./2006[17]	65	SAH	N/A	Y	-	Small bulge	DS (Trapping+bypass)	day 10	N	N	GR
24	Lv et al./2009[18]	43	SAH	WFNS grade I	Y	-	Small bulge	EVT (Stenting → Internal trapping)	1 month→ 4 months	N/A	N	GR
25	Nakajima et al./2009[19]	64	SAH	WFNS grade IV	Y	-	Fusiform dilatation	DS (Trapping)	day 1	N/A	N	MD
26	Satoh et al./2011[20]	51	SAH	WFNS grade I	Y	-	Dilatation	DS (Trapping)	day 1	Infarction (caudate nucleus)	N	GR
27	Divitiis et al./2015[21]	20	SAH	Hunt and Hess 2	Y	-	Aneurysm with false lumen	DS (Clipping)	day 1	Infarction (genu of internal capsule)	N	GR
28	Fukuma et al./2015[22]	47	SAH	N/A	Y	-	Stenosis without dilata-tion	Conservative	-	-	N	MD
29	Mitsuhara et al./2018[23]	65	SAH	N/A	Y	-	Fusiform dilatation	DS (Trapping)	day 1	N	N	MD
30		53	SAH	N/A	N	day 2	Slight dilatation→ Aneurysmal bulge	Conservative	-	N	Y	D
31		35	SAH	N/A	N	day 18	Aneurysmal bulge	DS (Clipping)	day 24	N	N	GR
32	Otaki et al./2021[24]	67	SAH	WFNS grade IV	Y	-	Fusiform dilatation	EVT (Stenting→Coiling)	day 8→ day 53	N	N	GR
33	Giogianni et al./2022[25]	60	SAH	WFNS grade II	Y	-	Small aneurysm	EVT (Flow diverter)	N/A	N	N	GR
34	Present case	47	SAH	WFNS grade II	N	day 5	Aneurysm with intimal flap	EVT (Internal trapping)	day 40	Infarction (caudate nucleus)	N	GR

SAH, subarachnoid hemorrhage; CI, cerebral infarction; WFNS, World Federation of Neurological Surgeons; Y, yes; N, no; N/A, not available; DSA, digital subtraction angiography; DS, direct surgery; EVT, endovascular treatment; GD, good recovery; MD, moderate disability; VS, vegetative survival; D, death

*Progressed to a vegetative state because of recurrent SAH or vasospasm

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Informed Consent

Informed consent for publication was obtained from the patient.

Conflicts of Interest Disclosure

All authors declare that there is no conflict of interest.

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