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Simultaneous Endovascular Treatment of Ruptured Cerebral Aneurysms and Vasospasm

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Objective: The management of patients with ruptured cerebral aneurysms and severe vasospasm is subject to considerable controversy. We intended to describe herein an endovascular technique for the simultaneous treatment of aneurysms and vasospasm.

Materials and Methods: A series of 11 patients undergoing simultaneous endovascular treatment of ruptured aneurysms and vasospasm were reviewed. After placement of a guiding catheter within the proximal internal carotid artery for coil embolization, an infusion line of nimodipine was wired to one hub, and of a microcatheter was advanced through another hub (to select and deliver detachable coils). Nimodipine was then infused continuously during the coil embolization.

Results: This technique was applied to 11 ruptured aneurysms accompanied by vasospasm (anterior communicating artery, 6 patients; internal carotid artery, 2 patients; posterior communicating and middle cerebral arteries, 1 patient each). Aneurysmal occlusion by coils and nimodipine-induced angioplasty were simultaneously achieved, resulting in excellent outcomes for all patients, and there were no procedure-related complications. Eight patients required repeated nimodipine infusions.

Conclusion: Our small series of patients suggests that the simultaneous endovascular management of ruptured cerebral aneurysms and vasospasm is a viable approach in patients presenting with subarachnoid hemorrhage and severe vasospasm.

Index terms: *Aneurysm; Vasospasm; Coil embolization; Angioplasty*

INTRODUCTION

The optimal treatment of patients presenting with subarachnoid hemorrhage (SAH) from ruptured cerebral aneurysms is expeditious aneurysmal obliteration. Once the aneurysms are secured, however, patients may be subject to marked cerebral vasospasm, facing new peril from decreased cerebral perfusion (1-4). For various reasons, patient evaluation may not take place until several days after SAH occurs (1), in which case, the initial diagnostic angiography may reveal severe vasospasm, either symptomatic or asymptomatic, in addition to the aneurysmal rupture.

A number of applicable treatment strategies have been suggested as follows: 1) conservative management; 2) watchful waiting, allowing vasospasm to resolve; 3) microsurgical clip application or coil embolization, with immediate endovascular treatment of vasospasm thereafter

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(chemical or balloon angioplasty); and 4) the reverse, endovascular treatment of vasospasm, with immediate coil embolization or surgical clipping thereafter (1, 5, 6). In the event of severe vasospasm, management of such patients is still controversial. Herein, a novel endovascular technique is introduced to simultaneously address both aneurysmal rupture and vasospasm.

MATERIALS AND METHODS

Patient Population

A total of 2328 saccular aneurysms in 2027 patients were treated by endovascular coil embolization at our institution between January 2008 and July 2013. Among the 410 patients who with ruptured aneurysms, 14 patients displayed moderate-to-severe vasospasms on their initial cerebral angiographies and subsequently received endovascular treatment of both the aneurysm and the vasospasm in one session. In three patients, the endovascular procedures were performed sequentially (i.e., intra-arterial nimodipine infusion immediately after coil embolization), thus excluding them from our study. The other 11 patients, undergoing simultaneous endovascular management of both aneurysmal rupture and vasospasm, served as the study cohort (females, 6; males, 5; mean age, 49.8 ± 11.1 years). Patients' clinical conditions were assessed by the Hunt and Hess grade at the time of their presentation to our institution. Clinical and radiographic features of these patients are shown in Table 1.

Informed consent was obtained from all patients after in-depth consultation, delineating the risks, benefits, and alternatives (including aneurysm clipping), as part of multidisciplinary neurosurgical and neurointerventional decision-making. This study was conducted with the approval of the Institutional Review Board of the Seoul National University Hospital.

Therapeutic Strategy

After placement of a guiding catheter in the proximal internal carotid artery (ICA) for coil embolization, an infusion line of nimodipine was wired to a hub of the guiding catheter. A microcatheter to deliver the coils was then advanced into the guiding catheter through a second hub. Nimodipine was infused continuously through the guiding catheter during the coil embolization procedure, as illustrated in Figure 1.

For vasospasms so severe that advancement of the microcatheter for coil delivery was precluded, nimodipine at a minimal dosage (1 mg) was infused preliminarily. Once the lumen reached the minimal caliber for microcatheter passage, coil embolization was performed under continuous intra-arterial infusion of nimodipine.

The infusion dose was determined by the severity of the vasospasm and the degree of response to the nimodipine infusion. Generally, 50 mL (10 mg) of nimodipine diluted in 150 mL of a normal saline solution was infused at a rate of 2 mL/min. After infusing 3 mg, control angiography was performed to determine the need for additional nimodipine. Blood pressure was continuously monitored through a femoral arterial line during nimodipine infusion, maintaining systolic pressure at 110–130 mm Hg.

Endovascular Procedure

All procedures were performed under general anesthesia. Aneurysmal configuration and arterial architecture were evaluated using the Integris Biplane System V (Philips Healthcare, Best, the Netherlands), incorporating high-resolution three-dimensional rotational angiography. Because all patients presented with SAH, antiplatelet medication was not administered prior to the procedure. However, a bolus (3000 IU) of heparin was given at the start of the procedure, and most patients received booster doses of heparin (infused at 1000 IU/hour) in conjunction with the monitoring of activated clotting time. After the procedure, maintenance antiplatelet medication was not routinely prescribed, unless coil protrusion or procedural thromboembolism occurred.

Two experienced neurointerventionists independently reviewed the immediate angiographic results using the 3-point Raymond scale to classify coil embolization as complete occlusion (no residual filling of aneurysm with contrast medium), residual neck (limited residual contrast filling at aneurysmal base), or residual aneurysm (any contrast filling of aneurysmal sac) (7).

By initial angiogram, vasospasm was designated as focal or diffuse, according to distribution of affected arteries, and graded as a mild (< 33%), moderate (34–66%), or severe (> 67%) reduction in normalized arterial diameter, reflecting the degree of vasospasm (2). Any improvement in vasospasm after nimodipine infusion was gauged by the diameter ratio (pre- and post-infusion) of the narrowest segment.

Table 1. Summary of Patients' Data

No	Sex	Age	Size (mm)	D/N Ratio	H-H Grade	Location	Interval*	Initial Spasm	NDP Dose	Response of NDP†	Coiling Technique	Occlusion Grade	Procedural Complication	Additional NDP Infusion Day	Follow-Up Result	GOS
1	51	F	2.9	1.0	II	ICA	5	Focal, moderate, aSx	3 mg	50%	Single MC	RN	None	8 days	Residual neck at 36 month	5
2	37	M	8.1	2.5	II	AcomA	3	Focal, moderate, aSx	3 mg	48%	Single MC	RN	None	5 days	Complete occlusion at 60 month	5
3	46	F	4.8	3.0	IV	ICA	7	Diffuse, moderate, aSx	3 mg	38%	Single MC	RN	None	None	Complete occlusion at 36 month	3
4	31	M	5.1	2.0	IV	AcomA	2	Diffuse, moderate, aSx	3 mg	40%	Single MC	RN	None	5 days	Complete occlusion at 36 month	4
5	40	M	6.0	2.0	III	AcomA	9	Diffuse, severe, Sx	5 mg	82%	Single MC	RN	None	7 days	No follow-up	5
6	72	F	10.2	1.3	II	AcomA	8	Focal, moderate, aSx	3 mg	71%	Single MC	RN	aSx thrombus	5 days	Complete occlusion at 24 month	5
7	53	F	8.4	3.0	III	ACA	9	Diffuse, severe, Sx	3 mg	31%	Single MC	RN	None	2 days	Complete occlusion at 12 month	5
8	54	M	6.2	2.8	II	AcomA	13	Diffuse, moderate, aSx	5 mg	36%	Single MC	RN	None	None	Complete occlusion at 12 month	5
9	56	F	5.5	2.2	II	PcomA	7	Diffuse, moderate, aSx	3 mg	100%	Single MC	CO	None	3 days	Complete occlusion at 36 month	5
10	56	M	10.4	2.6	II	AcomA	14	Diffuse, moderate, aSx	5 mg	52%	Double MC	CO	None	None	Complete occlusion at 15 month	5
11	52	F	5.5	1.6	III	MCAB	10	Diffuse, severe, aSx	5 mg	68%	Single MC	RN	None	3 days	Complete occlusion at 24 month	5

Note.— *Interval means duration between symptom onset and coil embolization, †Response of nimodipine means degree of vosodilation (based on spastic arterial diameter) after nimodipine infusion. ACA = anterior cerebral artery, AcomA = anterior communicating artery, An = aneurysm, aSx = asymptomatic, CO = complete occlusion, D/N = depth to neck, F = female, GOS = Glasgow outcome scale, H-H = Hunt and Hess, ICA = internal carotid artery, M = male, MC = microcatheter, MCAB = middle cerebral artery bifurcation, NDP = nimodipine, PcomA = posterior communicating artery, RN = residual neck, Sx = symptomatic

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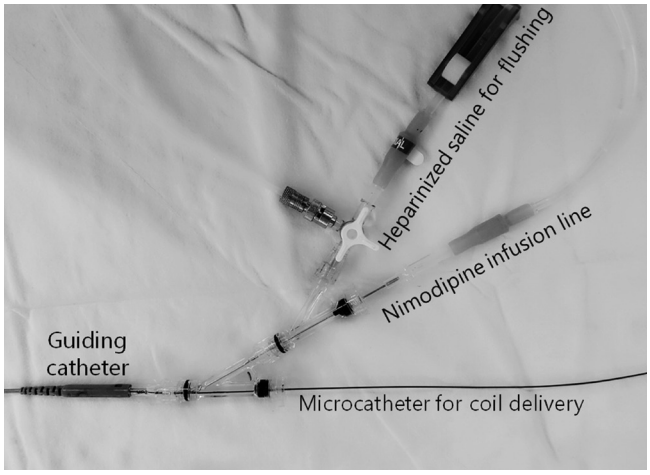

Fig. 1. Guiding catheter hub system for simultaneous management of ruptured aneurysm and vasospasm.

Clinical and Radiological Follow-Up

In all aneurysms, standard plain radiography was recommended at 1 and 3 months post-embolization. Magnetic resonance angiography (MRA) and/or plain radiography was recommended 6, 12, 24, and 36 months after coil embolization. Conventional angiography was advised if aneurysmal recanalization was suspected by noninvasive means, such as MRA or plain radiography, so that further treatment could be rendered as needed.

The Glasgow outcome scale (GOS) was engaged to assess clinical outcomes. Anatomic follow-up results were also categorized using Raymond scale: complete occlusion, neck remnant, or residual sac. Repeat embolization was recommended for patients showing residual sac, considered to be a major recanalization.

RESULTS

Simultaneous treatment was conducted in 11 ruptured aneurysms with vasospasm (Hunt and Hess grades II [6 patients], III [3 patients], and IV [2 patients]). Among them, two patients (Patients 5 and 7) had focal neurologic deficits (hemiparesis Gr III and IV, respectively) at the time of their presentation to our institution. The interval between symptom onset and coiling varied from 2–14 days (median, 7.9 ± 3.7 days). Most of the patients (n = 8) were seen at our institution after Day 5 following rupture, although one patient arrived the day of rupture, showing multiple aneurysms of a larger basilar tip aneurysm by angiography, as well as a tiny aneurysm of ICA bifurcation. The former was considered the source of hemorrhage and was treated the same day. After 7 days, however, vasospasm was evident by transcranial Doppler, and angiography disclosed the expansion of the aneurysm at the ICA bifurcation, confirming it as the actual site of rupture (Patient 3). The anterior communicating artery (AcomA) was the most common site involved (n = 6), followed by the ICA (n = 2), posterior communicating artery (PcomA) (n = 1), middle cerebral artery (MCA) (n = 1), and anterior cerebral artery (ACA) (n = 1) (Table 1). Initial degrees of vasospasm were moderate in eight patients and severe in three. Nimodipine (1 mg) was infused before coil embolization in two patients with severe vasospasm (Patients 5 and 7) to facilitate microcatheter passage, followed by additional continuous nimodipine (2 mg) infusion during the coil embolization. In the other nine patients, nimodipine infusion and coiling took place simultaneously. A single microcatheter sufficed in 10 patients, with one requiring double microcatheter use. Complete aneurysmal occlusion was achieved in two instances, the remaining nine patients retaining neck remnants. Degrees of vasodilatation with nimodipine infusion ranged from 31–100%. In two instances, asymptomatic thrombi developed during the procedure, both attributable to minimal coil protrusion and were resolved with tirofiban infusion. After coiling, additional intra-arterial nimodipine infusion was performed in 8 patients over 2–8 days. At the time of discharge, the GOS in nine patients was 5 (good recovery). The other two sustained permanent neurologic sequelae (GOS 3 and 4) due to higher grade SAH at presentation. After a mean follow-up period of 29.1 ± 14.8 months, 10 patients were evaluated by MRA and/or conventional angiography. None of them showed major recanalization. Only one experienced minor recanalization and complete aneurysmal occlusion was achieved in the rest.

Illustrative Cases

Patient 9

A 56-year-old female with a severe headache arrived at the emergency room of our facility with SAH (confirmed by CT). Conventional angiography revealed a PcomA aneurysm (with a bleb), as well as diffuse vasospasms of the MCA and ACA. A 6-Fr guiding catheter was placed in the cervical segment of the left ICA. After wiring an infusion line to a hub of guiding catheter, nimodipine was given continuously at a rate of 3 mg/30 minutes. A single microcatheter for coil delivery was also inserted into the aneurysmal sac and coil embolization was performed. As

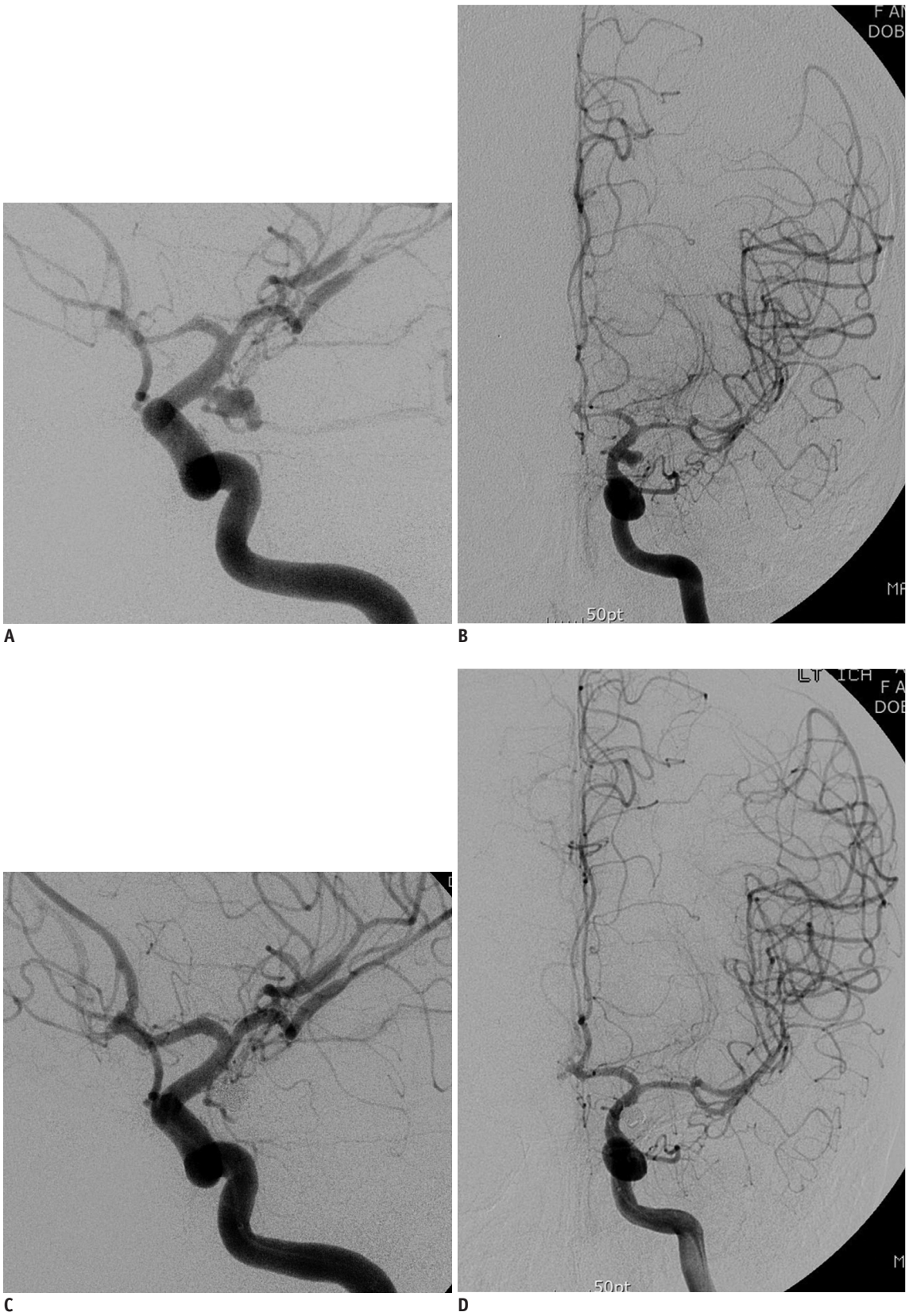

Fig. 2. Ruptured posterior communicating artery (PcomA) aneurysm.
Conventional angiography of ruptured PcomA aneurysm **(A)**, diffuse vasospasm **(B)**, completion angiography after simultaneous treatment (aneurysm coiling and 3-mg nimodipine infusion), showing near-total occlusion of aneurysm by coils **(C)**, and improvement in vasospasm **(D)**.

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coiling neared the finish, 3 mg of nimodipine was infused. Completion angiography indicated that the aneurysm was occluded, with a small neck remnant, and the vasospasm improved (Fig. 2). This patient required additional intra-arterial nimodipine infusion during the next 3 days but was discharged without complications.

Patient 5

A 40-year-old male was admitted for endovascular treatment of AcomA aneurysms, presenting with SAH. His arrival at the emergency room was 9 days after the onset of symptoms. The aneurysm had a sizeable daughter sac and vasospasm was severe, involving both the ACA and the MCA. Spasm of the ACA (in the A1 segment) proximal to the aneurysm prevented the passage of a microcatheter for coil delivery, necessitating a 1-mg infusion of nimodipine in order to proceed. An additional 4 mg of nimodipine was infused thereafter during coil embolization. Completion angiography indicated successful aneurysmal occlusion, and the vasospasm improved dramatically. This patient likewise required additional intra-arterial nimodipine infusion during the next 7 days but was discharged without complications (Fig. 3).

DISCUSSION

Cerebral vasospasm is a major source of morbidity and mortality from SAH due to aneurysms (2, 8, 9). Angiographic evidence of vasospasm may be found in up to 70% of patients, typically 5–14 days after occurrence of SAH (8-11). In general, preventive and therapeutic management of vasospasm is performed after aneurysmal

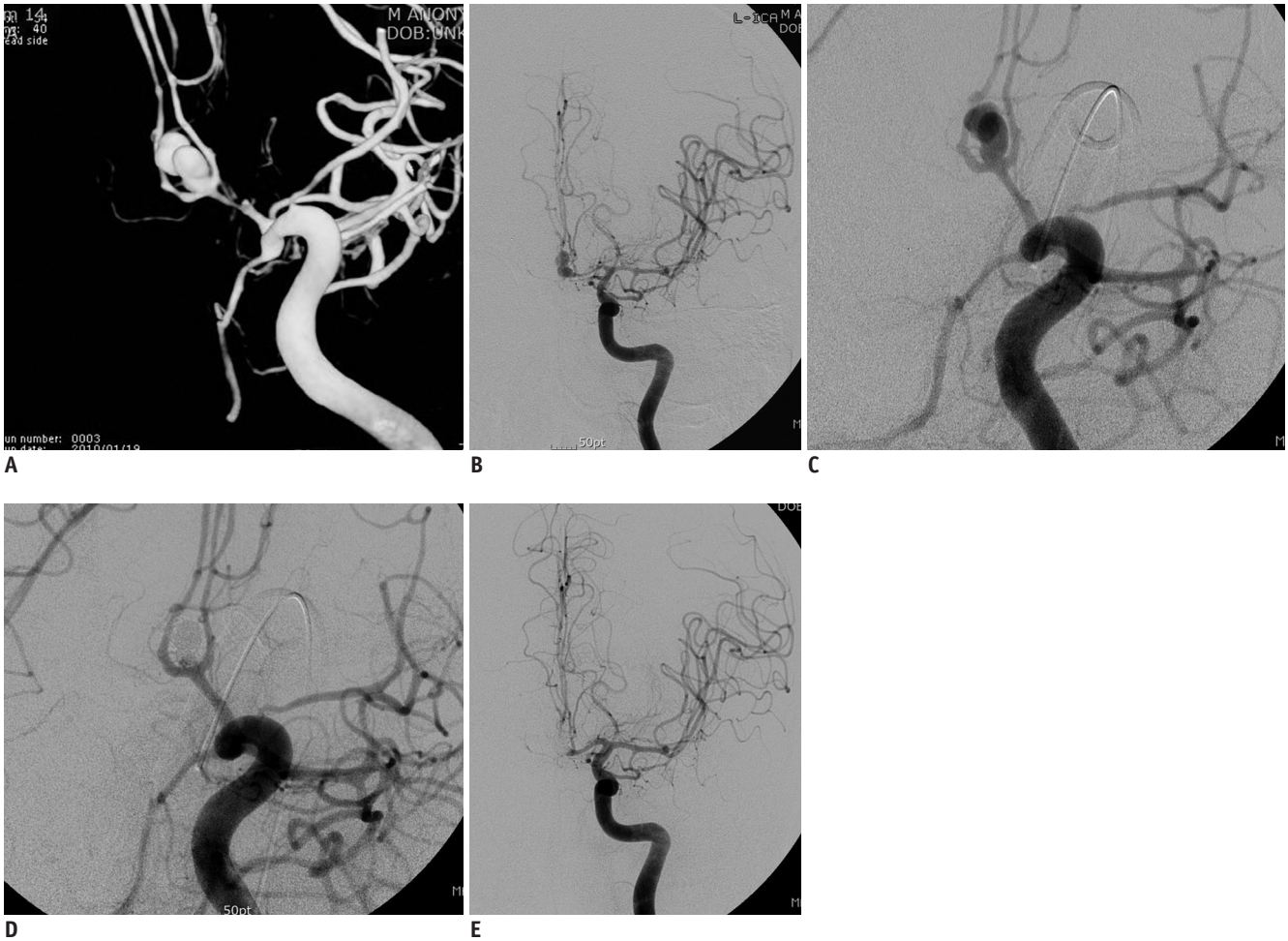

Fig. 3. Ruptured anterior communicating artery (AcomA) aneurysm.
Cerebral angiography of ruptured AcomA aneurysm **(A)**, severe vasospasm, precluding passage of microcatheter for delivery of coil **(B)**, follow-up angiography after intra-arterial infusion of nimodipine (1 mg), showing slightly dilated proximal anterior cerebral artery (in A1 segment) **(C)**, final angiography after simultaneous treatment (aneurysm coiling and 3-mg nimodipine infusion), showing occluded aneurysm **(D)**, and improvement in vasospasm **(E)**.

obliteration by clip or coil. Aggressive management of the vasospasm, such as triple-H therapy (i.e., hypertension, hypervolemia, hemodilution) and angioplasty, should only be initiated after aneurysmal occlusion. However, some patients may develop acute vasospasm within 3 days after SAH, and some may only seek medical attention after more than 5 days have elapsed. In such cases, particularly with severe and symptomatic vasospasm, treatment of the ruptured aneurysm and vasospasm should be implemented simultaneously. Surgical management otherwise may be technically challenging, and the vasospasm may be exacerbated (1).

In this study, we detailed our experience in simultaneously treating the uncontained aneurysms and severe vasospasm of 11 patients. This approach was safe and effective, at least in our small series. These patients were subjected to aneurysmal coiling and chemical angioplasty in one session, entailing less time and less risk of procedural complications (i.e., thromboembolism) than consecutive procedures. In addition, an additional microcatheter insertion expressly for nimodipine infusion was not required.

In two patients, vasospasm occurred proximal to the aneurysms, precluding microcatheter passage. Excessive proximal arterial dilatation by nimodipine in an unprotected setting increases the risk of rupture for unsecured aneurysms. Therefore, minimal doses of nimodipine (1 mg) were initially infused to gain passage for the microcatheter. Coil embolization and chemical angioplasty were then simultaneously performed. This is our recommended approach in instances where the vasospasm proximal to the aneurysm is severe.

In the past, severe vasospasm was considered a relative contraindication for surgical clipping of ruptured aneurysms. This concept delayed surgery and therefore was questioned, given the high risk of re-bleeding. In patients with unsecured aneurysms and symptomatic vasospasm, endovascular treatment may offer the following advantages, compared with open surgery: 1) less invasiveness and manipulation, reducing brain ischemia due to retraction in the vasospastic territory, 2) shorter procedural times to reduce related complications and limit general anesthesia, 3) earlier aggressive management of severe vasospasm, potentially improving clinical outcomes, and 4) less risk of exacerbating arterial narrowing through manipulation (1, 12-14).

Managing vasospasm prior to aneurysmal obliteration may increase the risk of re-bleeding. Chemical or balloon angioplasty may instead be performed immediately after surgical clipping or endovascular coiling, particularly if the aneurysmal configuration is not conducive to coil embolization. However, simultaneous treatment by our technique is superior to consecutive management. Murayama et al. (1) reported the consecutive treatment of ruptured aneurysms and symptomatic vasospasm, both done in one session, but with a time lag. If the vasospasm was not severe, they achieved aneurysmal occlusion using coils prior to angioplasty. Otherwise, angioplasty was performed before aneurysmal occlusion. Our approach is novel, although both treatments are performed in one session. Brisman et al. (5) also reported two cases of intentional partial coil occlusion and delayed clipping of wide-necked MCA aneurysms in patients presenting with severe vasospasm.

Transluminal balloon angioplasty and intra-arterial infusion of pharmacologic vasodilators are now considered the standard of care for medically refractory vasospasm (3, 12). Balloon angioplasty is highly effective in relieving the focal vasospasm of proximal major vascular segments at the circle of Willis, whereas intra-arterial vasodilators are capable of easing distal and diffuse vasospasm (2, 3, 15, 16). Although more invasive, the former offers a more prolonged vasodilatory effect in instances where arterial wall rupture and dissection are possible. Use of intra-arterial vasodilators is less invasive and safer, but repeated treatment is often necessary (2, 17, 18). In patients with unsecured ruptured aneurysms and severe vasospasm, balloon angioplasty without protection carries greater risk than intra-arterial vasodilator infusion, owing to an abrupt increase in blood flow to the unsecured aneurysm. With protection, however, balloon angioplasty is a reasonable treatment alternative, despite the added time involved.

Some critics may claim that asymptomatic moderate or severe vasospasm does not require intraarterial nimodipine infusion. However, in the setting of vasospasm with decreased cerebral blood flow, devices (such as a guiding system and microcatheter) placed within the ICA during the coil embolization can pose a risk for further reduction of cerebral blood flow. Furthermore, given that the vasospasm can get aggravated by navigating spasmatic arteries with the microcatheter and microguidewire during coil embolization, the primary goal of our simultaneous management was to help avoid the aggravation of cerebral ischemia in the process of the coil embolization. Moreover, the transcranial Doppler ultrasonography value immediately

after the simultaneous management served as the reference value to determine the need for additional treatment for vasospasm in the next few days. In our series, the majority of our patients (8 of 11, 72.7%) required the use of additional intra-arterial nimodipine infusion for better outcomes.

Our investigation is limited in that was a retrospective study with a small population. In addition, because all patients in this series had vasospasms associated with unsecured ruptured aneurysms at the time of presentation to our institution, it was uncertain whether the symptoms were caused by SAH, vasospasm, or a combination of both. Nevertheless, in terms of prevention (or treatment) for the aggravation of vasospasm-induced ischemia during the coil embolization, we do believe that this simultaneous management constitutes a good alternative in the disadvantageous condition of unsecured ruptured aneurysms combined with severe vasospasm. Further study with a larger study population is warranted to ascertain the efficacy and safety of this approach.

In conclusion, our small series suggests that simultaneous endovascular management of ruptured aneurysms with severe vasospasm is a safe and effective therapeutic option.

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