

# Subdural Hematoma without Subarachnoid Hemorrhage Caused by the Rupture of Middle Cerebral Artery Aneurysm

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Pure subdural hematomas caused by a ruptured intracranial aneurysm are extremely rare. We describe the case of a 42-year-old woman who presented with headache without evidence of head trauma. Magnetic resonance angiography and conventional cerebral angiography revealed a ruptured aneurysm at the right middle cerebral artery bifurcation. The patient underwent surgical treatment and had a good outcome without any neurological deficit. The mechanisms and clinical characteristics of this condition are discussed.

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Keywords Cerebral aneurysm, Subdural hematoma, Middle cerebral artery

## INTRODUCTION

In most cases, subdural hematomas (SDHs) are caused by head trauma.<sup>7/33)</sup> Spontaneous SDH is an uncommon condition, found in only 2.6% of acute subdural hematoma (ASDH) cases. Underlying etiologies for spontaneous SDH include ruptured aneurysms with or without concomitant subarachnoid hemorrhage (SAH), arteriovenous malformations, meningiomas, dural metastatic diseases, and hematologic or solid neoplasm.<sup>7)</sup>

Spontaneous pure ASDH without concomitant SAH due to the rupture of a cerebral aneurysm is extremely rare and only 40 cases were reported between 1981 and 2012.<sup>9)23)35)</sup> We report a case of spontaneous ASDH without evidence of SAH caused by a ruptured aneurysm at the middle cerebral artery (MCA) bifurcation, presenting with repeated sentinel headaches.

# CASE REPORT

A 42-year-old woman was admitted to our institution with a history of sudden onset headache without any accompanying neurological deficit. A week prior to admission, the patient experienced two episodes of transient headache, each lasting a few hours. She had noted occasional headaches over the last few years; however, they did not last more than a few minutes and were not severe. She had no definite history of head trauma, hypertension, or coagulopathy.

The initial computed tomography (CT) scan of the brain revealed an ASDH without evidence of any other type of hemorrhage including SAH (Fig. 1), and laboratory studies, including coagulopathy screening, showed no abnormalities. For further evaluation, magnetic resonance angiography (MRA) and subsequent

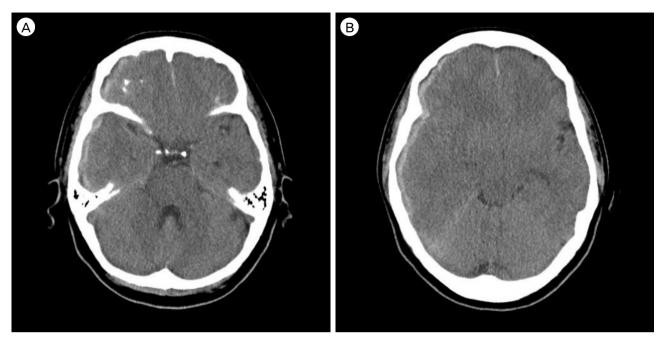


Fig. 1. Initial brain imaging findings of case. Computed tomography scans (A, B) shows no evidence of subarachnoid hemorrhage at the basal cistern and the sylvian fissure and there is only acute subdural hematoma on the right fronto-temporo-parietal area.

conventional cerebral angiography were conducted, revealing a right middle cerebral artery bifurcation aneurysm (Fig. 2).

An aneurysmal neck clipping surgery was performed on the second day of hospitalization. A right pterional approach was used as usual, but when a semi-lunar shaped dural incision was made, only a dark bloody SDH without SAH was noted. There was no discoloration of brain parenchyma; no hematoma was observed in the subarachnoid space. After total removal of the SDH, sylvian fissure dissection was performed and the aneurysm was identified at the middle cerebral artery bifurcation. The aneurysmal dome extended antero-inferiorly and laterally and was tightly adherent to the arachnoid membrane (Fig. 3). Postoperative CT revealed resolution of the ASDH without any complications related to the surgery. The patient's hospitalization was uneventful and a follow-up three-dimensional CT angiography performed 1 year after the operation showed no remnant or recurrent aneurysm (Fig. 4).

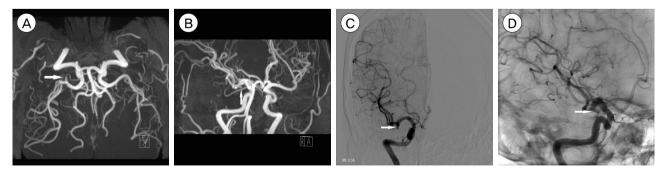


Fig. 2. Magnetic resonance angiography (A, B; white arrows) and conventional cerebral angiography (C, D; white arrows) show the presence of the right middle cerebral artery bifurcation aneurysm extending antero-inferiorly and laterally.

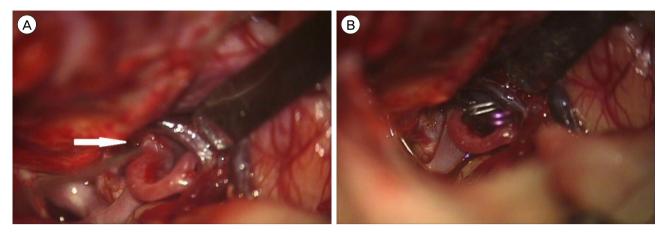


Fig. 3. Intraoperative photographs shows the aneurysmal sac penetrating into subdural space (A; white arrow). Permanent 7 mm straight mini-clip was introduced to the neck portion of the aneurysm successfully (B).

### DISCUSSION

The mechanism of ASDH caused by rupture of an intracranial aneurysm is different from that of ASDH caused by trauma and tearing of cortical and bridging veins.<sup>10)</sup> According to previous studies, various mechanisms have been suggested to explain the occurrence of ASDH without SAH after aneurysm rupture. First, previous repeated minor ruptures of the aneurysm occur, causing small bleeds and the formation of tight adhesions between the aneurysm and the neighboring

arachnoid membrane. Eventually, massive bleeding fills the subdural space directly after a major rupture occurs.<sup>8)16)23)25)</sup> Second, massive high-pressure bleeding causes laceration of the arachnoid membrane with bleeding into the subdural space.<sup>8)14-16)18)</sup> Third, the rupture of a subdural carotid artery aneurysm results in a pure ASDH.<sup>12)15)24)</sup> Fourth, the cavernous sinus wall is eroded by the acute enlargement of an intra-cavernous aneurysm after thrombosis, leading to pure ASDH.<sup>18)35)</sup>

In the present case, intraoperative findings revealed



Fig. 4. Follow-up computed tomography angiography 1-year after the operation. The clip is observed in the source image of the computed tomography angiography (A), and there was no evidence of the remnant aneurysm (A, B).

No. First author, Yea	'ear	Age	Sex	Symptoms and signs	Location of aneurysm	Location of subdural hematoma	Treatment	Outcome
Rengachary and Szymanski,	/manski,	49	Σ	Confusion, dysphagia	MCA	Convexity	Hematoma evacuation and clipping	Good
	-	34	ц	Headache	PromA	Convexity	Hematoma evacuation	Good
3 Williams et al., 1983 <sup>39)</sup>	39)	18	. ц.	Coma	PcomA	Convexity	Hematoma evacuation and clipping	Disabled
	:	55	щ	Headache	PcomA	Tentorium and interhemispheric		Good
	(3 <sup>6)</sup>							
5 O'Leary and Sweeny, 1986 <sup>27</sup>	V, 1986 <sup>21</sup>	28 28	ц :	Coma	MCA	Convexity		Death
	988 <sup>-0</sup>	43	Σı	Coma	PcomA	Tentorium and convexity	evacuation and	Cood
	988-01	8 i	ц	Coma	PcomA	Tentorium and convexity	evacuation and	Disabled
	39-11	ß		Coma	MCA	Convexity	evacuation and	Disabled
_	175	44	ц	Semicoma	PcomA	Convexity	evacuation and	Disabled
10 Watanabe et al., 1991 <sup>30</sup>	1.51	51	Σ	Semicoma	Distal ACA	Interhemispheric and convexity		Death
	3471	27	Σ	Coma	AcomA	Convexity		Death
12 Hatayama et al., 1994	94	55	Σ	Semicoma	Distal ACA	Interhemispheric and convexity		Good
	94"	66	ш	Semicoma	Distal ACA	Interhemispheric, convexity, and	Hematoma evacuation and clipping	Disabled
	<del>,</del> 15)	Ĺ	L			Tentorium		
14 ISTIIUASTILELAU, 1997		+ c		נישיניטישיי	Prom A	Communication convexity	evacuation and	
		Ω L Ω C	L I		PcomA		evacuation and	0005
		-7	<b>т</b> I	Headache	PcomA	Convexity	evacuation and	0005
	2	22	LL 1	Coma	PcomA	Convexity	Hematoma evacuation and clipping	Good
		61	LL.	Headache	MCA	Tentorium and convexity	Clipping	Good
	107	52	ш	Coma	PcomA	Tentorium and convexity	Hematoma evacuation and clipping	Good
20 Ishikawa et al., 2000 <sup>16)</sup>	0 <sup>16)</sup>	62	Σ	Headache, ptosis	PcomA	Tentorium and interhemispheric	Clipping	Good
	2 <sup>14)</sup>	28	щ	Coma	PcomA	Convexity	Hematoma evacuation	Death
		55	ш	Headache, ptosis, semicoma	PcomA	Convexity	Hematoma evacuation and clipping	Good
		56	Σ	Headache, drowsiness	AcomA	Convexity	Hematoma evacuation	Death
	-	28	Σ	Headache	PcomA	Convexity	Hematoma evacuation and clipping	Good
		39	щ	Headache	PcomA	Convexity		Good
	(	46	Σ	Coma	MCA	Convexity	Hematoma evacuation and clipping	Death
		35	ш	Coma	PcomA	Convexity	None	Death
28 Katsuno et al., 2003 <sup>17)</sup>	3 <sup>17]</sup>	63	ш	Headache, nausea, dizziness	Distal ACA	Interhemispheric and convexity	Hematoma evacuation and clipping	Good
	004 <sup>21)</sup>	42	ш		AcomA	Tentorium and convexity	evacuation and	Good
_	19)	62	ш	Headache, semicoma	Bifurcation	Convexity	Hematoma evacuation and coiling	Good
a Triantafullonollulation		ΥĽ	ц	Headache searcea utosis		sind sincerver part termine	Hematoma eviacitation	Comatore
2006 <sup>36)</sup>		3	-			converse and caven load and		
32 Gilad et al., 2007 <sup>8)</sup>		47	Σ	Nausea, vomiting	AcomA	Sella, miaratina to spinal canal	Coiling	Good
	(;	47	ш	Not described	AcomA	Not described	Clipping	Good
	22)	75	Σ	Headache, vomiting	MCA	Anterior fossa	Hematoma evacuation and resection	Not
				n				described
35 Field and Heran, 2010 <sup>5)</sup>	010 <sup>5)</sup>	33	Σ	Headache, Terson syndrome	MCA	Convexity	Not described	Not
								described
36 Weil et al., 2010 <sup>38)</sup>	10	51	LL I	Coma	MCA	Convexity	Hematoma evacuation and coiling	Disabled
	, <sup>m</sup>	47	ц I		PcomA	Convexity	Colling	Good
	12	09	т:	Headache, abducens palsy	MCA	Convexity	Clipping	000J
	ī	40 4 0	Σι		AcomA	lentorium and convexity		, 1000
40 MITKA, 2012		<del>5</del>	ᄂ	Headance, nausea, vomiting Liondocho	PCOMA	Lonvexity Tentorium and romavity	Hematoma evacuation and colling Liemsteins evacuation and clinician	2000
		47	L	הפממכתיב	SIN	ובנווסנומנוו פנומ רחנאבצווא	בענוקוסניוש באשרחשווחנו שנומ הוואאוויוא	כררמ

a tight adhesion between the aneurysmal dome and the arachnoid membrane. The patient reported recurrent episodes of sentinel headache, suggesting that repeated small bleedings led to formation of the tight adhesion. These findings are consistent with the first mechanism described above.

Including the present case, we analyzed 41 cases reported with pure ASDH caused by the rupture of an intracranial aneurysm and have summarized them in Table 1. Of these, 10 were caused by the rupture of an MCA aneurysm, including the present case. In those cases, ASDH was located at the convexity in seven cases, at both the tentorium and convexity in two cases (including the present case), and at the anterior fossa in one case. Six of these 10 cases underwent hematoma evacuation and clipping, as in the present case, and two cases underwent clipping only. In one case, surgery could not be performed, and in another case, treatment was not described. These 10 cases presented with a variety of symptoms and signs and experienced different outcomes. Four cases were comatose at admission, and, in these patients, the final outcomes were poor; two patients died and the other two patients were disabled. The other six cases presented with an alert to confused mental status with headache and/or other minor neurological deficits; in four cases, the final outcomes were reported to be good, and there was no description of outcome in the other two cases.

Among all 41 cases analyzed, 17 patients presented with a semi-comatose to comatose mental status on admission, and only five of these patients (29%) had good outcomes. Six patients died and another six patients were disabled. The remaining 24 cases with a relatively good neurological status had generally good outcomes. Excluding two cases without outcome description, 91% (20/22 patients) had good outcomes, one patient died, and one patient remained comatose. Based on these results, it appears that a favorable neurological status at admission is a predictive factor for a good outcome. These findings are consistent with those of Schuss et al. who studied patients with ASDH and SAH, and concluded that a good neurological status at admission is a predictive factor for a good outcome.<sup>32)</sup>

#### CONCLUSION

The rupture of an intracranial aneurysm may be the cause of a pure ASDH in patients without a history of trauma, but this is often mistakenly ruled out because of the absence of SAH. Subsequently, determining the correct cause and providing the proper treatments in these patients may be delayed.

Therefore, when a patient presents with a pure SDH without a history of trauma or coagulopathy, imaging studies, such as three-dimensional CT angiography or MRA, should be performed to evaluate for vascular lesions to determine the underlying cause and to guide optimal treatment for the patient.

#### Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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