Subdural Hematoma Due to Ruptured Intracranial Aneurysm

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Abstract

Subdural hematoma (SDH) was observed in 15 of 484 cases of aneurysmal subarachnoid hemorrhage (SAH). There were four males and 11 females, with ages ranging from 39 to 75 years. The clinical grades (Hunt and Hess) on admission were II in three cases, III in two, IV in four, and V in six. The ruptured aneurysms were located in the middle cerebral artery (MCA) in six cases, anterior communicating artery in three, internal carotid artery in two, and distal anterior cerebral artery (ACA) in two, with two cases unconfirmed. A high proportion of aneurysms occurred in the MCA and distal ACA. Aneurysmal neck clipping and removal of SDH were performed in the acute stage of seven cases, without intraoperative rerupture. The outcomes 1 year after SAH of the seven patients undergoing surgery were good recovery in five, but in two, vegetative state due to preoperative rerupture or medical complications. All eight patients without surgical intervention died. A good prognosis for patients with ruptured intracranial aneurysms accompanied by SDH can be expected with direct surgical intervention in the acute stage, even if the clinical grade on admission is poor.

Key words: intracranial aneurysm, subarachnoid hemorrhage, subdural hematoma

Introduction

Subarachnoid hemorrhage (SAH) almost invariably develops following the rupture of a cerebral aneurysm, and is often complicated by intracerebral hematoma (ICH) and intraventricular hematoma (IVH). But only extremely rarely does subdural hematoma (SDH) occur. The clinical incidence of SDH complications is reported to be 0.5–7.9%.^{2,3,12)} In the present study, the clinical grade on admission, location and size of the ruptured aneurysm, and outcome were examined in cases of SDH.

Materials and Methods

Between January of 1979 and December of 1988, a total of 484 cases of SAH due to ruptured intracranial aneurysms were admitted to the Department of Neurosurgery, Shimane Prefectural Central

Hospital. In 15 cases, SDH was demonstrated by computed tomography (CT). The clinical grade on admission, site, size, and presence or absence of bleb of ruptured aneurysms, and the outcome of the 15 cases with SDH and 469 cases without SDH were compared. The clinical grade on admission was assessed by the Hunt and Hess scale.⁶⁾ The outcome was evaluated using the Glasgow Outcome Scale⁷⁾ 1 year after SAH.

Results

CT scanning was conducted on 452 of the 484 cases within 14 days of the aneurysmal rupture. The CT findings are summarized in Table 1. SDH was observed in 15 cases, and their clinical features are summarized in Table 2.

Of the 469 cases without SDH, the clinical grades on admission were I in 53 (11%), II in 177 (38%), III

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Table 1 CT findings in 452 cases with aneurysmal SAH*

CT findings	No. of cases
SAH only	307
ICH	109
IVH	86
SDH	15

*No CT scan was performed in the acute stage of 32 cases.

in 110 (23%), IV in 56 (12%), and V in 73 (16%). Among the 15 cases with SDH, the clinical grades were II in three (20%), III in two (13%), IV in four (27%), and V in six (40%), with the majority of cases belonging to the poorer grades ($p < 0.05$). Eighty-six (18%) of 469 non-SDH patients were paretic on admission. The frequency of paresis was higher among the SDH patients, in six (40%) of 15 cases ($p < 0.05$).

The initial CT scans of the 15 SDH cases showed SDH + SAH in four, SDH + SAH + ICH in six, SDH + SAH + ICH + IVH in four, and SDH only in one. The SDH was observed after initial rupture in 10 cases and after rerupture in five.

The location of ruptured aneurysms could be confirmed in 427 of 469 non-SDH cases. The sites were the anterior communicating artery (AcomA) in 164

Table 3 Maximum diameter of ruptured aneurysms*

Diameter (mm)	With SDH	Without SDH
< 5	5 (38%)	116 (27%)
< 10	3 (23%)	216 (51%)
< 20	1 (8%)	79 (19%)
< 25	1 (8%)	1 (0.2%)
≥ 25	3 (23%)	15 (4%)
Total	13	427

*Ruptured aneurysms were not measured in two cases with SDH and in 42 without SDH.

cases (38%), internal carotid artery (ICA) in 117 (27%), middle cerebral artery (MCA) in 101 (24%), distal anterior cerebral artery (ACA) in 26 (6%), and vertebrobasilar artery in 19 (4%). The ruptured aneurysms could be confirmed in 13 of 15 SDH cases, being the AcomA in three (23%), ICA in two (15%), MCA in six (46%), and distal ACA in two (15%). Therefore, a high proportion of aneurysms occur in the MCA and distal ACA. The presence or absence of the bleb of aneurysm showed no definite trend. Larger or giant aneurysms were relatively frequent in the 13 cases with SDH ($p < 0.01$) (Table 3).

Neck clipping and removal of SDH were performed during the acute stage in seven of the 15 SDH patients. No ruptures occurred during the surgical pro-

Table 2 Clinical summary of 15 patients with SDH due to ruptured intracranial aneurysm

Case No.	Age/ Sex	Grade*	Paresis	Other CT findings			Site	Aneurysm		Direct operation	Outcome**
				SAH	ICH	IVH		Bleb	Diameter (mm)		
1	67/F	IV	+	+	-	-	MCA	+	30	+	VS
2	50/F	III	-	+	+	+	ICA	-	28	-	D
3	67/F	V	+	+	+	-	MCA	-	4	-	D
4	52/F	V	-	+	+	+	ND	-	-	-	D
5	69/F	II	-	+	+	+	AcomA	+	27	-	D
6	63/F	II	-	+	-	-	MCA	-	4	+	GR
7	73/M	IV	+	+	-	-	AcomA	+	7	-	D
8	75/F	II	-	+	-	-	MCA	+	9	+	VS
9	64/M	V	-	+	-	-	ND	-	-	-	D
10	72/F	IV	+	+	+	-	distal ACA	-	4	+	GR
11	70/M	IV	+	+	+	-	MCA	+	6	+	GR
12	72/F	V	-	+	+	-	ICA	-	4	-	D
13	59/M	V	-	+	+	+	MCA	+	22	-	D
14	39/F	V	+	+	+	-	distal ACA	-	3	+	GR
15	71/F	III	-	+	+	-	AcomA	+	11	+	GR

*Hunt and Hess grade⁶⁾ without modification on admission. **Glasgow Outcome Scale⁷⁾ 1 year after initial SAH. GR: good recovery, MD: moderate disability, SD: severe disability, VS: vegetative state, D: died. #Absent on initial CT. ND: not detected.

Table 4 Outcome 1 year after aneurysmal rupture*

Outcome**	With SDH		Without SDH	
	Surgery (+)	(-)	(+)	(-)
GR	5	0	206	27
MD	0	0	31	0
SD	0	0	29	3
VS	2	0	9	3
D	0	8	38	113
Total	7	8	313	146

*Ten cases without SDH did not survive 1 year after aneurysmal rupture. **Glasgow Outcome Scale.⁷⁾

cedure. The outcomes of five patients were good, but one (Case 1) developed decubitus and pneumonia and another (Case 8) became vegetative in association with preoperative rerupture and cardiac failure. However, none of the eight patients without surgery have survived. Two patients were inoperable because of rerupture or severe spasm on admission. Five of six patients in grade V on admission were inoperable, and in one of four patients in grade IV on admission, clinical grade became aggravated to grade V and the operation was impossible. Only one patient in grade V received surgical intervention and survived. The outcomes of the 459 non-SDH patients surviving more than 1 year were good recovery in 233 (51%), moderate disability in 31 (7%), severe disability in 32 (7%), vegetative state in 12 (3%), and death in 151 (33%) (Table 4).

Case Reports

Case 8: A 75-year-old female was admitted on August 20, 1985, because of severe headache. Her consciousness was almost clear. The initial CT scans on admission showed only SDH and not SAH, suggesting chronic SDH (Fig. 1 upper). Burr-hole irrigation was therefore performed, but fresh hematoma was observed under the dura mater. She became unconscious while returning from the operating room. Repeat CT scans revealed SAH with SDH (Fig. 1 lower). Right carotid angiograms revealed an MCA aneurysm. Complications of pulmonary edema and cardiac failure appeared at the same time. Aneurysmal neck clipping and SDH removal were performed on the 2nd day. One year later, she continued to be vegetative.

Case 14: A 39-year-old female was admitted on April 17, 1988, because of a sudden loss of consciousness. On admission, she was in a deep coma with decerebrate posturing, absent pupillary light reflex

Fig. 1 Case 8. upper: CT scans on admission, showing SDH but not SAH. lower: CT scans after aggravation, demonstrating SAH with SDH.

Fig. 2 Case 14. CT scans on admission, showing SAH, ICH, and SDH with the midline shift.

on the right, and anisocoria. CT scans revealed SAH, ICH, and SDH with a midline shift (Fig. 2). Right carotid angiograms revealed a distal ACA aneurysm. Aneurysmal neck clipping and removal of the SDH were performed the same day. One year after the onset, she was in good health.

Discussion

Clarke and Walton⁴⁾ classified SDH due to ruptured intracranial aneurysms into two groups based on the path of the blood from the ruptured aneurysm into the subdural space: by bleeding directly into the subdural space and bleeding into the subdural space *via* the subarachnoid space. Barton and Tudor²⁾ described four mechanisms by which blood from a leaking aneurysm can reach the subdural space. 1) Successive small hemorrhages allow adhesions to develop, and the final rupture occurs into the subdural space. 2) The arachnoid membrane is ruptured by the rapid accumulation of blood under pressure from the leaking aneurysm. 3) A massive hemorrhage ruptures the cortex and lacerates the arachnoid membrane. 4) An aneurysm in the part of the carotid within the subdural space ruptures and directly causes SDH.

The confirmation of the presence or absence of successive small hemorrhages as in 1) is difficult. However, warning signs such as headache may reflect their presence.²⁾ In the present study, three of 15 cases had such episodes. Mechanism 2) is said to be the commonest, but is difficult to differentiate completely from 1). As complications such as ICH are common in SDH cases, 3) is quite plausible. In the present study, associated SDH and ICH complications were very frequent, in 10 of 15 cases. In Cases 10 and 14, an ICH within the frontal lobe caused by the rupture of a distal ACA aneurysm penetrated the interhemispheric fissure and entered the subdural space. In Case 11, the ICH due to rupture of a MCA aneurysm was extremely large, flowing into the Sylvian cistern and further extending to the subdural space. However, the AcomA aneurysm in Case 15 was confined to the frontal lobe, and no associated SDH occurred. The SAH itself was severe in only four cases (Cases 2, 3, 7, and 12), moderate in five, slight in five, and absent in one. SDH does not therefore necessarily occur with massive hemorrhage into the subarachnoid space. Mechanism 4) has yet to be confirmed, and is considered unlikely by some authors.⁸⁾

An aneurysm at any site can cause a SDH upon rupture.¹⁰⁾ The rupture of distal ACA or AcomA aneurysms is rare, and that of ICA and MCA aneurysms common.^{1,5,12)} In the present study, the ruptures of MCA and distal ACA aneurysms were relatively frequent, observed in six cases and two, respectively.

SDH but not SAH was observed on admission of Case 8 with a MCA aneurysm, resulting in a wrong diagnosis. The delay in controlling the systemic

blood pressure caused rerupture of the aneurysm, which aggravated the clinical grade to IV. As frequently emphasized in the past, this is a case demonstrating clearly the grave probability that SDH has developed from a ruptured intracranial aneurysm.^{1,8,9)}

Surgical removal of SDH in the acute stage involves the risk of rupture due to rapid decompression. However, some recent reports have suggested that aneurysmal neck clipping and SDH removal by extensive craniotomy can be conducted simultaneously.¹⁾ In all our cases, surgery in the acute stage caused no ruptures.

Previous authors have pointed out that in the majority of SDH cases due to ruptured aneurysm the clinical grades are severe.^{8,11)} In six of the 15 present cases, the clinical grade was V. Case 4 was dead on arrival, and Case 9 was in the non-filling state during cerebral angiography. Cases 3, 12, and 13 died on days 0, 1, and 2, respectively. However, surgery conducted on Case 14 (grade V) during the acute stage resulted in good recovery. Extremely favorable results have been observed in SDH cases with poor clinical grades on admission. The SAH was minimal in most cases, and the common causes of the poor clinical grade were increased intracranial pressure due to SDH and the midline shift. Even if the clinical grade is poor, in cases of mild SAH the delayed ischemic neurological deficiency due to cerebral vasospasm may be small.¹⁾ As SDH is a hematoma caused by blood flow from the subarachnoid or intracranial space, the brain damage is smaller than that caused by trauma. Sachs and Bernat¹¹⁾ have pointed out the necessity for prompt diagnosis using CT scans and mannitol treatment. In addition, positive steps should be taken to treat patients with ruptured aneurysms accompanied by SDH, even if the clinical grade on admission is poor.

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