

# CLINICAL FEATURES, TREATMENT, AND PROGNOSIS OF PATIENTS WITH ACUTE SUBDURAL HEMATOMAS PRESENTING IN CRITICAL CONDITION

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**OBJECTIVE:** Spontaneous acute subdural hematoma (aSDH) may be caused by aneurysm rupture. Patients can present in very poor clinical condition with anisocoria or even bilaterally dilated pupils, absent brainstem reflexes, and cardiac insufficiency. For the clinician, the question is how should these patients be treated? Large series on this subject do not exist because aSDH is a rare event. This report focuses on the prognosis and adverse prognostic factors of these patients.

**CLINICAL PRESENTATION:** We present eight cases of aSDH and subarachnoid hemorrhage attributable to aneurysm rupture. All patients were World Federation of Neurosurgical Societies Grade 5. Four presented with anisocoria, three presented with bilaterally fixed and dilated pupils, and one developed anisocoria in the course of treatment.

**TREATMENT:** As a result of prolonged hypoxia before admission, one patient was not treated and died. In one patient, surgical decompression could not be performed in the acute phase as a result of significant comorbidity. All other patients received decompressive surgery, obliteration of the aneurysm, and medical therapy as well as extensive rehabilitation measures. After 6 months, four had no or only minor neurological deficits; one patient was independent despite hemiparesis. Two patients whose surgical decompression had to be delayed as a result of severe cardiac instability recovered poorly, showed severe neurological deficits, and required permanent care. However, none of the patients survived in a persistent vegetative state.

**CONCLUSION:** Within the spectrum of aneurysmatic hemorrhage, patients with aSDH represent a distinct subgroup. Despite a very poor clinical condition on admission, recovery with only minor deficits or even without neurological deficit is possible. Mass effect and herniation induce a poor clinical condition, which is not directly related to the underlying subarachnoid hemorrhage. Hence, clinical grading systems such as the Hunt and Hess scale or World Federation of Neurosurgical Societies grading are not applicable. We suggest that whenever the medical condition allows, rapid surgical decompression should be performed even in patients who present in very poor neurological condition.

**KEY WORDS:** Aneurysm, Aneurysmatic subdural hematoma, Coma, Decerebration, Outcome, Subarachnoid hemorrhage, Subdural

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Sudden and spontaneous onset of neurological deterioration and pupillary disturbance suggests intracranial hemorrhage with significant mass effect. This may be attributable to spontaneous acute subdural hematoma (aSDH) caused by aneurysm rupture. The incidence of aneurysmal aSDH has been reported to be 5 to 22% in autopsy studies,

including patients with small amounts of subdural blood and those who died before reaching medical treatment (8, 13). In computed tomographic (CT)-based clinical studies, the incidence of aneurysmal aSDH has been found to be 2 to 10.3% (16, 22).

Since the 1950s, several authors have discussed the appropriate management of patients

with aneurysmal aSDH. In 1953, Clarke and Walton (6) classified aneurysmal aSDH based on published case reports: 1) patients with rapidly fatal hemorrhage, 2) patients with an insignificant amount of subdural blood, and 3) patients with clinically significant hematoma, which was not rapidly lethal. It was concluded that Group 3 alone was suitable for treatment. Since then, several groups have reported good outcome after rapid surgical decompression, although patients presented in poor neurological condition (17, 18). In general, early decompression of significant mass lesions has shown to be favorable and has been recommended even in larger studies (10, 16, 22). However, in larger series, a significant number of patients did not receive surgical treatment or were not treated at all. However, the reasons for withdrawing therapy in poor-grade patients were not discussed in detail.

Improvements in emergency treatment and resuscitation help a growing number of patients survive the first hours and reach hospitals in very poor neurological condition. The situation is challenging because it requires rapid evaluation of whether angiography should precede surgery, whether surgery should be postponed because of severe comorbidities, or if therapy should be completely withheld to prevent vegetative survival. Large systematic series focusing on the outcome and prognosis of patients with aneurysmal aSDH who present in very poor clinical condition do not exist. Currently, decisions must be based on personal experience and case reports. In this article, we report eight patients with aneurysmal aSDH who presented in a comatose state with pupillary disturbance.

**Illustrative Cases**

We present a series of eight patients with aneurysmal aSDH who were admitted in a comatose state and with pupillary disturbance within a 3-year period. In this period, a total of 115 patients with aneurysmal SAH were admitted to our department. Eight patients, all women, had aSDH visible on CT scans. On admission to our department, all of the reported patients were intubated and mechanically ventilated. Five patients were primarily admitted to our department and three were transferred from external hospitals. The clinical state before intubation was reported by emergency physicians who accompanied the transfer to our department. According to their initial examination, all patients were graded as World Federation of Neurosurgical Societies Grade 5 (Table 1).

Except for one patient who was not specifically treated and died subsequently (Patient 8), all patients were discharged into rehabilitation centers. Neurological outcome was assessed after 6 months and is presented using the Glasgow Outcome Scale (GOS) and the Barthel Index.

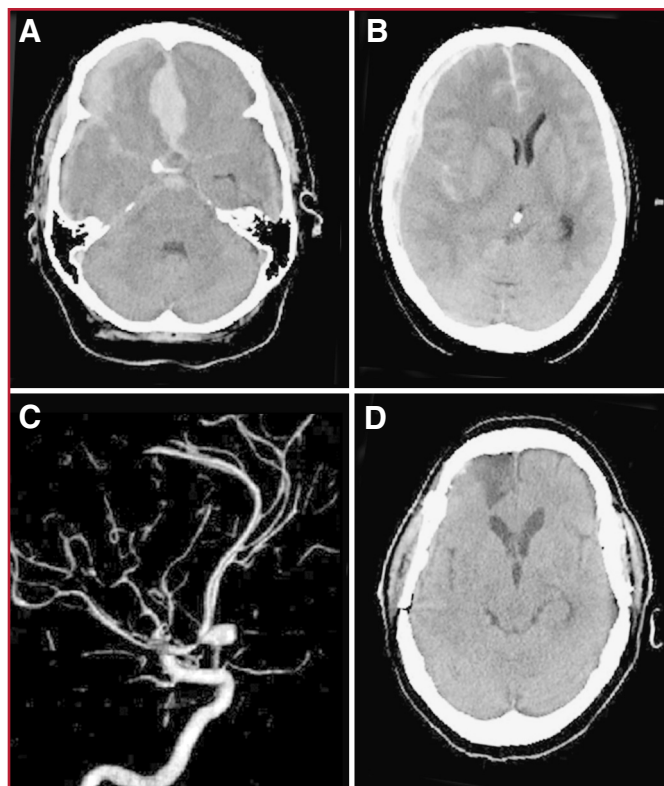
*Patient 1*

A 55-year-old woman was admitted to an external hospital with severe headache. Four hours later, she deteriorated to Glasgow Coma Scale [GCS] 6, was intubated, and transferred to our hospital. On admission, she had no pupillary disturbance. CT scans showed a right frontotemporal and interhemispheric subdural hematoma (Fig. 1). External ventricular drainage was implanted for intracranial pressure monitoring and cerebrospinal fluid drainage. Angiography demonstrated an aneurysm of the anterior communicating artery, which was treated by endovascular coiling. Twenty-four hours later, intracranial pressure increased and anisocoria developed. Immediate craniectomy and evac-

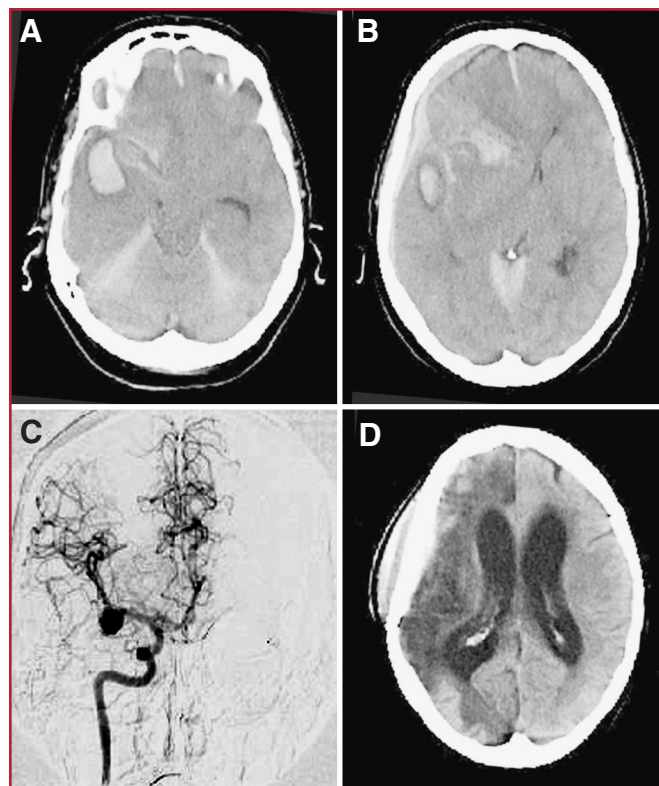
**TABLE 1. Details of clinical presentation, treatment, and outcome<sup>a</sup>**

Patient no.	Age (yr)/sex	State on admission, pupillary state	Location of subdural hematoma	Aneurysm	Treatment of hematoma and aneurysm	Complications, infarction	Outcome
1	55/F	WFNS 5, anisocoria after 24 hours	Right frontal and interhemispheric	Acom	Endovascular coiling; decompression as anisocoria developed	Vasospasm, angioplasty, right frontobasal infarction	GOS 4 Barthel 70
2	56/F	WFNS 5, MI, bilaterally fixed pupils	Right frontotemporal and ICH	MCA	Delayed treatment; decompression and vasospasm, clipping after 24 hr	Cardiac instability, right MCA and partial ACA infarction	GOS 3 Barthel 20
3	55/F	WFNS 5, anisocoria	Right frontotemporal	Pcom	Surgical decompression; endovascular coiling	Vasospasm, right parietal infarction	GOS 4 Barthel 70
4	55/F	WFNS 5, anisocoria	Right frontal and interhemispheric	Acom	Surgical decompression; endovascular coiling	Vasospasm, right frontobasal infarction	GOS 5 Barthel 100
5	43/F	WFNS 5, bilaterally fixed pupils	Left frontotemporal	Pcom	Surgical decompression; endovascular coiling	Vasospasm, angioplasty, partial left MCA infarction, VP shunt	GOS 3 Barthel 70
6	54/F	WFNS 5, MI, resuscitated anisocoria	Right frontotemporal	Acom	Delayed treatment ventriculostomy; endovascular coiling	Cardiac instability, vasospasm, right MCA and partial ACA infarction	GOS 3 Barthel 0
7	42/F	WFNS 5, anisocoria	Right frontotemporal	Pcom	Surgical decompression and clipping	Vasospasm, angioplasty, no infarction	GOS 5 Barthel 100
8	55/F	WFNS 5, hypoxic, bilaterally fixed pupils	Right frontotemporal and ICH	MCA	No therapy as a result of prolonged hypoxia before admission	Deceased	GOS 1

<sup>a</sup> WFNS, World Federation of Neurological Surgeons, clinical classification; Acom, anterior communicating artery; GOS, Glasgow Outcome Scale; MI, myocardial infarction; ICH, intracerebral hemorrhage; MCA, middle cerebral artery; ACA, anterior cerebral artery; Pcom, posterior communicating artery; VP, ventriculoperitoneal.



**FIGURE 1.** **A** and **B**, CT scans showing Patient 1 after deterioration. **C**, three-dimensional angiography showing an aneurysm of the anterior communicating artery. **D**, CT scan obtained 5 months after bleeding showing partial infarction of the right anterior cerebral artery territory.



**FIGURE 2.** **A** and **B**, CT scans showing Patient 2 at the time of admission. **C**, anteroposterior view showing a large middle cerebral artery aneurysm. **D**, CT scan obtained 6 months later showing infarction in the right middle cerebral artery and frontal anterior cerebral artery territory.

uation of the hematoma was performed and anisocoria resolved. Despite angioplasty for treatment of consecutive vasospasm, a small right frontobasal infarction developed. After 6 months, the patient had no formal neurological deficit, was independently mobile for short distances, and was able to communicate freely (GOS 4, Barthel 70).

#### Patient 2

A 56-year-old woman collapsed at home and was found in a deep coma with bilaterally fixed and dilated pupils and insufficient respiration. She was intubated and transported to our hospital. CT scans showed an acute subdural hematoma over the right hemisphere and intracerebral hemorrhage (Fig. 2). Immediate surgery was not feasible because of acute myocardial infarction and persistent cardiocirculatory instability with recurrent phases of bradycardia and hypotension requiring high doses of catecholamines. One hour after admission and repeated infusions of mannitol, the left pupil became reactive to light again. Twenty-four hours after the event, cardiac function was slightly improved; angiography was performed and showed a large aneurysm of the right middle cerebral artery. A craniectomy was performed, the hematoma was evacuated, and the aneurysm was clipped. Pupillary reactivity returned on both sides. In the further course of intensive care treatment, the patient had to be mechanically resuscitated, experienced cerebral vasospasm, and developed infarction of the right middle cerebral artery territory and anterior cerebral artery territory. After 6 months, verbal communication on a simple level was possible, she

had severe left hemiparesis, and she remained dependent on permanent care (GOS 3, Barthel 20).

#### Patient 3

A 55-year-old woman had been complaining about neck pain for several days. During a car trip, she suddenly felt a severe headache and rapidly deteriorated neurologically. She was found comatose with anisocoria and no motor response (GCS 3) and was immediately intubated and transported to our hospital. CT scans showed subarachnoid hemorrhage and a right subdural hematoma. An immediate craniectomy was performed, the hematoma was evacuated, and an external ventricular drainage was placed. The pupillary state normalized after surgery. Angiography and endovascular therapy of an aneurysm of the posterior communicating artery were performed the next day. She experienced vasospasm, which was not accessible to angioplasty, and developed right temporoparietal infarction despite hyperdynamic therapy. After 6 months, the patient returned home with minor left hemiparesis (GOS 4, Barthel 70).

#### Patient 4

A 55-year-old woman reported a severe headache and was admitted to an external hospital in a soporous state. CT scans showed subarachnoid hemorrhage and right frontotemporal and interhemispheric subdural hematoma. Her condition deteriorated further, and she became comatose and developed anisocoria. On arrival at our department, an



immediate craniectomy was performed and an external ventricular drainage was placed. The patient's pupils normalized. On the next day, an aneurysm of the anterior communicating artery was treated by endovascular coiling. After 6 months, she had no residual deficit and had returned to the office (GOS 5, Barthel 100).

*Patient 5*

A 43-year-old woman was found comatose (GCS 3) with anisocoria but spontaneously breathing and was transported to an external hospital. There, she was intubated as a result of progressive respiratory insufficiency. During the transport to our hospital, she developed bilaterally fixed and dilated pupils. CT scans showed subarachnoid hemorrhage and a left subdural hematoma. She underwent an immediate craniectomy and evacuation of the hematoma and pupillary reactivity normalized. Angiography was performed thereafter and an aneurysm of the left posterior communicating artery was treated by endovascular coiling. She experienced vasospasm and developed partial infarction of the left middle cerebral artery territory despite balloon angioplasty on Day 7. Two months later, a ventriculoperitoneal shunt was implanted. After 6 months, the patient returned home with right hemiparesis using a wheelchair for longer distances. She had no speech disorder and was largely independent (GOS 3, Barthel 70).

*Patient 6*

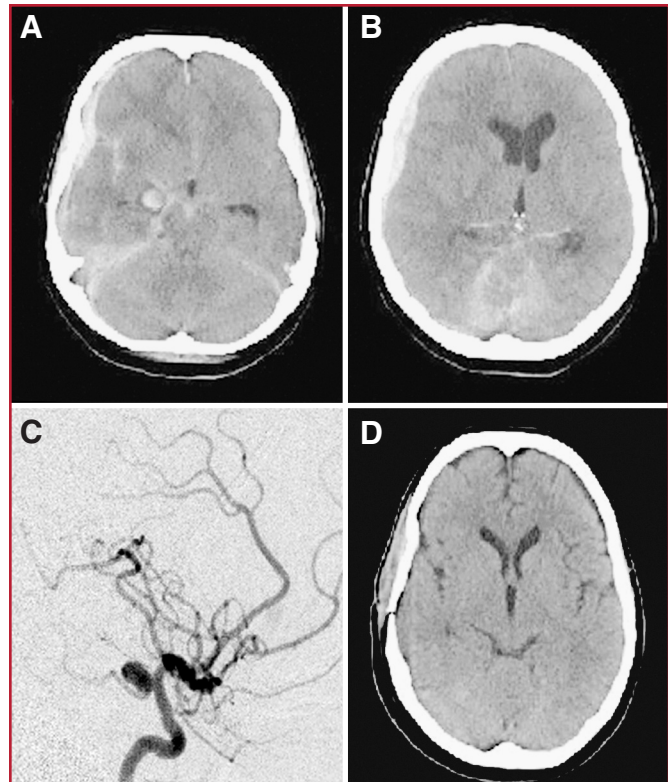
A 54-year-old woman collapsed during work and had to be mechanically resuscitated. She was found comatose with no motor response and anisocoria and was immediately intubated (GCS 3). Admission CT scans showed subarachnoid hemorrhage and a right subdural hematoma. As a result of myocardial infarction and sustained cardiac instability, she received an external ventricular drainage as a primary measure. Pupillary state returned to normal and remained so during the further course of treatment. A follow-up CT scan performed the next day showed a decrease of midline shift. As a result of the normalized pupillary state, decreased mass effect on CT scans, and persistent cardiac instability, surgical decompression was not performed in the acute stage. An aneurysm of the anterior communicating artery was treated by endovascular coiling. She developed bilateral frontobasal infarctions and partial infarction in the right middle cerebral artery territory. Three weeks later, the hematoma was evacuated through a burr hole. A ventriculoperitoneal shunt was implanted 2 months after the insult. After 6 months, she was able to communicate verbally on a simple basis and had no formal paresis. She was transferred home but was not able to walk and remained dependent on permanent care (GOS 3, Barthel 0).

*Patient 7*

A 42-year-old woman collapsed suddenly during gardening work. She was found deeply comatose without motor response and with anisocoria and was immediately intubated. CT scans showed subarachnoid hemorrhage, right subdural hematoma, and acute occlusive hydrocephalus (Fig. 3). She primarily received an external ventricular drainage and pupillary state normalized. Angiography showed five aneurysms. Three aneurysms were clipped, and evacuation of the subdural hematoma was performed. She experienced vasospasm and angioplasty was performed. After 6 months, the patient returned to normal life without neurological deficits (GOS 5, Barthel 100).

*Patient 8*

A 55-year-old woman had experienced nausea/vomiting during the previous evening. In the morning, she was found unconscious and gasping by her husband. Because he was not able to wake her, an ambulance was called. She was found deeply comatose with no motor

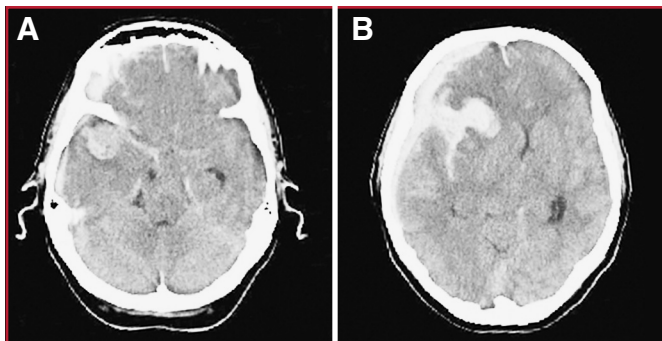


**FIGURE 3.** *A and B*, CT scans showing Patient 7 at the time of admission demonstrating an aneurysm of the right posterior communicating artery, the origin of bleeding. *C*, angiography demonstrating further aneurysms of the right middle cerebral artery, the left middle cerebral artery, the left pericallosal artery, and a basilar tip aneurysm. *D*, CT scan obtained 6 months later showing no signs of infarction.

response and with bilaterally fixed and dilated pupils (GCS 3). She had aspirated and was cyanotic and hypoxic. She was intubated and transported to our hospital. CT scans showed right subdural hematoma, subarachnoid hemorrhage, and intracerebral hematoma most likely originating from an aneurysm of the middle cerebral artery (Fig. 4). Therapy was discontinued because of prolonged hypoxia before admission (7, 20).

**DISCUSSION**

Most previous studies of aneurysmal aSDH report that some of the patients presented in good neurological condition on hospital admission (6, 9, 22). In contrast, all patients presenting in our department with aneurysm rupture and aSDH visible on CT scans were graded World Federation of Neurosurgical Societies Grade 5. The patients were graded according to the statements of emergency physicians who performed the primary evaluation before intubation. If a patient shows signs of acute cardiac or respiratory insufficiency, time may be scarce and primary evaluation may be difficult. However, classification according to the GOS before intubation and pupillary state has been well documented in the reported patients.



**FIGURE 4.** **A** and **B**, CT scans of Patient 8 on admission showing right subdural hematoma, subarachnoid hemorrhage, and intracerebral hematoma. This patient was not treated because of prolonged hypoxia before hospital admission.

Overall mortality in poor-grade subarachnoid hemorrhage (World Federation of Neurosurgical Societies Grade 5) with or without intracranial hematoma has been reported to be as high as 60%. Functional outcome is achieved in only a small portion of patients (4, 21).

The mortality rate after traumatic aSDH has been reported to be as high as 60% in adults (12, 23). The mortality rate of patients with traumatic aSDH presenting with unilaterally or bilaterally unreactive pupils has been reported as 81 and 97%, respectively (11). Again, only a small portion of patients with anisocoria on admission regain functional outcome. With bilaterally fixed and dilated pupils, the chance of good recovery is close to zero (1). Traumatic aSDH usually appear together with primary structural damage to the brain tissue. The latter seems to be the decisive factor determining outcome after traumatic brain injury with aSDH rather than the subdural hematoma (12, 23). In contrast, there is no primary damage of brain tissue in aneurysmal aSDH and those patients seem to have a more favorable prognosis.

After the first publications in the 1950s, several reports have been published showing varying results. Sachs and Bernat (18) and Ryan (17) each published a case of aneurysmal aSDH presenting with signs of brainstem herniation. Both patients underwent immediate evacuation of the hematoma and recovered well. Barton and Pitts (2) reported mainly good recovery after aneurysmal aSDH in 10 out of 11 patients who underwent surgical decompression. In contrast to our patients, most patients in this series presented in good neurological condition. Other series report mixed results ranging from death to recovery without deficit (9, 14) or report mainly unfavorable results with only a few patients regaining functional recovery (15, 22). In those studies, however, a significant number of patients was not treated and died. The reasons or conditions for withdrawing treatment are not discussed. In our series, only one patient was not treated because she had aspirated and was hypoxic for several hours before intubation and admission to our hospital. She subsequently died a few hours after admission.

The aim of surgical treatment in the acute stage was to perform decompressive craniectomy and remove the sub-

dural hematoma. In contrast to the other patients who underwent operation in the acute stage, Patient 7 showed no significant brain swelling after removing the hematoma and the bone flap was reinserted. All other patients received decompressive craniectomy.

If osmotherapy and/or external ventricular drainage were sufficient in reversing signs of herniation as expressed by pupillary disturbance, angiography preceded surgical decompression. If not, we performed immediate decompression without angiography. We did not face any bleeding complications during the decompressive procedures even in those patients whose aneurysms had not been obliterated. If angiography was performed before decompression and the aneurysm was not found suitable for endovascular obliteration, decompression and clipping were performed in one procedure. Generally, we consider decompression the primary target in this acute situation, in particular, if signs of herniation cannot be reversed by primary measures such as osmotherapy, sedation, or external ventricular drainage.

Two patients developed major deficits and had an unfavorable outcome. Both patients sustained acute myocardial infarction and acute heart failure, making an immediate operation unfeasible. It is difficult to distinguish whether poor medical condition or the resulting delay of surgical decompression had a greater negative impact on final outcome. Insufficient cardiac output causes a global decrease of brain perfusion. Extraxial masses, in turn, cause focally reduced brain perfusion (5, 19). The combination of both is likely to be particularly unfavorable, especially because subarachnoid hemorrhage itself is associated with acutely reduced brain perfusion (3). Rapid removal of the mass lesion by surgical decompression is the only procedure able to reverse the acute reduction of brain perfusion in this situation.

Local pressure on the underlying brain and blood vessels may also gate cerebral vasospasm and infarction, as suggested by the fact that all but one patient (Patient 7; Fig. 3) developed infarctions in the area of the subdural hematoma. Patient 1 did not undergo operation immediately because she had no pupillary disturbance at the time of admission. Earlier surgical decompression might have resulted in faster and even more complete recovery in this patient.

We suggest that whenever possible, rapid surgical decompression should be performed in poor-grade patients with aneurysmal aSDH unless significant comorbidities exist and there is doubt that the patient will survive the surgical procedure, e.g., Patients 2 and 6. Considering the poor recovery of the two patients who could not be treated immediately, it may be discussed whether or not patients in an unstable medical condition should receive delayed treatment at all, particularly if cardiocirculatory stabilization requires excessive time.

Five patients in this series reached a favorable outcome, although they also experienced cerebral vasospasm and other complications of prolonged therapy. However, they remained in a stable medical condition throughout the course of therapy and demonstrate that aneurysmal aSDH can be survived with only minor residual deficits, even if patients present in very

poor neurological condition. Midline shift and uncal herniation may mimic a clinical condition that appears to be worse than the accompanying subarachnoid hemorrhage can be held responsible for.

## CONCLUSION

Aneurysmatic subarachnoid hemorrhage complicated by aSDH is a critical condition. Patients may present in exceptionally poor neurological condition with signs of apparent uncal or transtentorial herniation. However, if rapid surgical decompression and maximum medical treatment can be performed, prognosis can be favorable.

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## COMMENTS

The authors have reported a small series of patients presenting with extension of subdural hemorrhage after aneurysmal subarachnoid hemorrhage (SAH). Each of these patients presented in poor condition; however, after 6 months, four had no or only minor neurological deficits. I agree with the authors that aggressive therapy is worthy of consideration in this group of patients. Although these patients may present with severe neurological deficits, urgent evacuation of the subdural and/or intracerebral hemorrhage can be lifesaving and improve the chances for a good neurological recovery.

However, I question the logic of performing endovascular therapy in this series of patients who are already undergoing a surgical procedure. In the first case, endovascular therapy was performed before surgery, thus unnecessarily delaying surgical treatment. Four of the other treated patients underwent a separate endovascular procedure after the surgical procedure. This required the patient to be exposed to two separate therapeutic procedures, each with their attendant risks, rather than treating the hematoma and aneurysm at the same time. With rare exceptions, the only benefit of endovascular therapy is to avoid a surgical procedure. If a surgical procedure needs to be performed for evacuation of a subdural or intracerebral hematoma, I think the aneurysm should be treated during the same procedure, if at all possible.

When patients present with a life-threatening subdural or intracerebral hemorrhage associated with aneurysmal rupture, we make every attempt to avoid unnecessary preoperative diagnostic studies and plan the urgent surgical procedure based on the distribution of blood. Once the offending hematoma has been removed, we typically perform intraoperative angiography to better understand the angioarchitecture of the intracranial circulation so definitive treatment of the aneurysm can be accomplished during the same procedure. Regardless of differences in philosophy, the authors are to be congratulated on the good outcomes in a significant number of these patients.

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In this series, Westermaier et al. present the results of aggressive surgical treatment in seven out of eight patients with an aneurysmal acute subdural hematoma (aSDH). One patient was not treated because of prolonged anoxia before presentation. All patients were scored as WFNS Grade 5 and had pupillary disturbances. Of the seven treated patients, three underwent decompression of the aSDH followed by endovascular coiling, two underwent decompression of the aSDH and aneurysm clipping, one had a ventriculostomy followed by aneurysm coiling, and one underwent coiling followed by delayed surgical decompression. The patient who was not treated died. Two of the remaining seven patients had an excellent outcome (Glasgow Outcome Scale 5), three patients were graded as Glasgow Outcome Scale 4, and two patients were graded as Glasgow Outcome Scale 3. None of the patients survived in a persistent vegetative state.



The conclusion of this case series is that patients with aneurysmal aSDH who present in a WFNS Grade 5 condition with pupillary abnormalities may have a better prognosis with aggressive surgical therapy than Grade 5 SAH patients without an acute subdural hematoma. Thus, in selected poor grade aneurysmal SAH patients with an aSDH, aggressive surgical treatment of the subdural hematoma with either immediate aneurysm clipping or delayed aneurysm coiling should remain a consideration.

**Carl B. Heilman**  
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This report describes a single institution's 3-year experience with aSDH accompanying aneurysm rupture. This is an eight patient case series derived from a total of 115 patients with ruptured aneurysms treated in their department during that time period (5.3%). Subdural hematomas accompanying aneurysm rupture large enough to cause, or contribute significantly to, uncal herniation are not common and are most often associated with middle cerebral artery and posterior communicating artery aneurysms. In this series, however, three patients had ruptured anterior communicating artery aneurysms. The central idea that grew out of this unusual clinical experience is that, although aneurysmal aSDHs are associated with severe rupture and poor neurological condition and prognosis, they should nevertheless be considered for aggressive surgical management when causing uncal herniation and when there is otherwise no vital brain destruction apparent. Their own treatment approach varied according to individual patient features, but one that was interest-

ing and successful in several patients was that of a rapidly performed large craniectomy, subdural hematoma evacuation, insertion of an external ventricular drain, and subsequent cerebral angiography and endovascular occlusion of the aneurysm. Depending on the relative expertise of the operating surgeon in acute aneurysm clipping and the availability of an endovascular team, this particular approach deserves consideration.

**J. Max Findlay**  
Edmonton, Canada

Approximately 80% of our patients with ruptured aneurysms undergo operation within 24 hours and those with hematomas are taken into the operating room immediately. As we treat patients with population responsibility and cannot exclude anyone, we must also care for poor-grade patients. This study is in parallel with our experience, showing the benefits of early surgery, even in poor-grade patients, to save lives. This report also addresses the necessity of neurosurgeons and interventionalists to work together when selecting patients for each modality. Why would one not clip the aneurysm after taking the subdural hematoma out as the cranium is already open and one could do it using the same approach? Therefore, we need dedicated neurovascular centers to treat these patients to guarantee the best possible outcome.

**Reza Dashti**  
**Mika Niemelä**  
**Juha A. Hernesniemi**  
Helsinki, Finland



"Flensing" (1954), woodblock and paper, copied from a 1773 picture scroll, Taiyo Fishing Company. (Courtesy of New Bedford Whaling Museum). From: Matthews, LH: *The Whale*. New York, Simon & Schuster, 1968.