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CLINICAL ARTICLE

Acute subdural hematoma from ruptured cerebral aneurysm

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Abstract
Purpose The combination of ruptured aneurysms with acute subdural hematomas (aSDHs) is a rare presentation. Patients with aSDH associated with aneurysmal bleeding represent a subgroup within the spectrum of aneurysmatic hemorrhage. We summarize the clinical characteristics, diagnostic evaluation, and management of a series of cases presenting with aSDH associated with aneurysmal subarachnoid hemorrhage (SAH).
Methods Medical records and surgical reports of 743 consecutive patients admitted to our institution with SAH from January 1995 to December 2007 were screened to detect cases of associated aSDH. Admission evaluations included the Glasgow Coma Scale (GCS) and the subarachnoid grade of the World Federation of Neurosurgical Societies (WFNS). Radiological assessment included computer tomography (CT) scan, CT angiography (CTA), and digital subtraction angiography (DSA). The presence and volume of SAH, intracerebral hemorrhage (ICH), and aSDH were documented. Outcome was measured in terms of Glasgow Outcome Scale (GOS) and modified Rankin Scale (mRS) at 4–8 months.
Results A total of seven cases (0.9%) presenting with aSDH (mean width: 11.2 mm±4.8 mm, range: 5–20 mm) attributable to SAH were documented. Three of these patients were admitted with a suspicion of trauma. Five patients presented with WFNS grade 5, one patient with WFNS grade 3, and one patient with WFNS grade 1. All patients underwent evacuation of the aSDH. In four patients, surgical obliteration of the aneurysm was achieved in the same procedure. Two patients underwent delayed occlusion of the aneurysm: one by coiling and one by clipping. Three of the seven patients recovered completely from their neurological deficits (GOS 5, mRS 0–1), three recovered with mild disability (GOS 4, mRS 2–3), and one died within 8 h after the decompressive procedure.
Conclusions The incidence of aSDH associated with SAH is low. Most of the patients with aSDH due to a ruptured aneurysm present in exceptionally poor neurological condition. Nevertheless, rapid surgical treatment of the hematoma and aneurysm obliteration can lead to a favorable outcome. Routine CTA should be performed in all patients presenting with an aSDH associated with SAH and no clear history of trauma.

Keywords Subarachnoid hemorrhage · Acute subdural hematoma · Cerebral aneurysm · Clipping · Coiling

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Introduction

Every third patient who suffers a subarachnoid hemorrhage (SAH) due to saccular aneurysm rupture presents with an intracranial hematoma [21]. However, the combination of ruptured aneurysm and spontaneous acute subdural hematoma (aSDH) is rare. A few hundred cases have been described since the first report in 1855 [9]. According to the published clinical and autopsic series, the incidence of aSDH due to aneurysm rupture varies from 0.5% [14, 27] to 8% [21] and 2% [5, 22] to 22% [8], respectively.

The rarity of aneurysmal aSDH makes it difficult to accomplish reliable clinical guidelines. Large series do not exist, and thus treatment decisions are mainly based on personal experience. The majority of patients present in severe clinical condition, and immediate surgical manage-

ment are required. The clinician has to decide which preoperative diagnostic study should precede surgery, whether obliteration of the aneurysm should be performed during hematoma evacuation, and if patients should undergo a separate delayed intervention. Justification for withholding aggressive therapy in poor-grade patients in order to prevent vegetative survival is controversial and there is growing evidence that a significant number of patients can recover with only minor or even without neurological deficits.

We report the incidence, clinical and radiological findings, treatment strategies, and outcome of seven cases presenting with aSDH after ruptured aneurysm within a 12-year period at our institution.

Methods

The authors searched retrospectively for cases of aneurysmal aSDH larger than 5 mm width by reviewing paper and electronic medical records, surgical reports, cerebral digital subtraction angiographies (DSAs), computer tomography (CT) angiographies (CTAs), and ordinary cranial CT scans of 743 nontraumatic SAH patients treated in the intensive care unit at our institution between January 1995 and December 2007. Intracranial aneurysm had been diagnosed by DSA or CTA and coexisting aSDH by CT scan.

The clinical characteristics included gender, age, initial neurological deficits, consciousness level evaluated with the Glasgow Coma Scale (GCS) [25], SAH grade based on the World Federation of Neurological Surgeons (WFNS) [26] scale, history prior to admission, time to surgery, treatments received, and outcome after rehabilitation evaluated with the Glasgow Outcome Scale (GOS) [13] and modified Rankin Score (mRS) [4]. The patients were followed-up at 4–8 months.

CT-scan studies included checking for the presence of SAH or intracerebral hemorrhage (ICH), visualized subarachnoid blood based on the Fisher Grading [6], degree of midline shift, and maximum thickness and localization of the aSDH. Angiographic evaluation included aneurysm localization, number of aneurysms, and mean size of the aneurysm.

Results

During the period from January 1995 to December 2007, 743 patients presenting with aneurysmal SAH were admitted to our institution. Seven (0.9%) patients were identified as having a coexisting aSDH, as recognized by CT scan. Clinical characteristics, hemtoma, and aneurysm configurations are presented in Table 1.

In the seven patients (one man, six women) the median age was 45 years (range 27–68 years). The GCS on admission was 15 in one patient, 13 in one patient, five in one patient, four in two patients, and three in two patients. The clinical SAH grade evaluated with WFNS at the time of admission was grade 5 in five patients, grade 3 in one patient, and grade 2 in one patient. Two patients presented with bilaterally dilated fixed pupils and three with unilateral dilated unreactive/fixed pupils. One patient complained of right occulomotor paresis and one patient exhibited a mild left-sided hemiparesis without any signs of further neurological deterioration.

In two patients with good clinical condition, conventional DSA was performed after initial CT scan. In four patients with poor neurological status, CTA after initial CT scans replaced the DSA. One patient underwent emergency hematoma evacuation with CT scans from referring hospital without any further diagnostics at our institution. All patients demonstrated some degree of SAH on their initial CT scan. Three patients experienced Fisher grade II, two were classified with Fisher grade III, and two showed an intracerebral hematoma (ICH) associated with the SAH (Fisher grade IV). The thickness of the aSDH ranged from 5 to 20 mm (mean 11.2 mm±4.8 mm), and all aSDH were located ipsilateral to the identified aneurysms. The midline shift on CT scans ranged between 4 and 23 mm (mean 11.7 mm±6.9 mm).

Angiographic studies showed maximum ruptured aneurysm size ranging from 2 to 14 mm (mean 8.0 mm± 4.4 mm). One aneurysm was identified at the distal end of the internal carotid artery close to the posterior communicating artery (Pcom), two were located at the junction of the internal carotid artery (ICA) and Pcom, two were found at the distal middle cerebral artery (MCA), and another two were located at the pericallosal artery. In three patients more than one aneurysm was detected.

All seven patients underwent operation for the aSDH within 18 h (range: 1–18, mean: 6.6 h) after initial bleeding, and all but one underwent craniectomies. In one case a rebleeding prior to surgery could be documented. Surgical treatment included hematoma evacuation and surgical occlusion of the aneurysm (four patients), and emergency craniotomy for hematoma evacuation with delayed clipping (one patient) or coiling (one patient). Finally, one patient who developed hemodynamic instability during hematoma evacuation could not undergo surgical occlusion of the aneurysm. This patient died within 8 h after surgery.

With regard to outcome, three patients had a good recovery (GOS 5; mRS 0–1), with no symptoms at all in one (mRS 0) and mild cognitive deficits in two (mRS 1). Three patients recovered with moderate disability (GOS 4; mRS 2–3) manifesting as residual left-sided hemiparesis in two patients (mRS 2) and gait ataxia in one (mRS 3). The

Table 1 Summary (characteristics) of seven cases of aneurysmal acute subdural hematomas (*MLS* midline shift, *f* female, *m* male, *Rt* right, *MCA* middle cerebral artery, *ICA* internal carotid artery, Pcom*Pcom* posterior communicating artery, *EVD* external ventricular drainage)

Case no.	Age/sex	Initial clinical findings[a]	Initial diagnostics	Fisher grade	ICH	Side of aSDH	Size of aSDH	MLS	Location of aneurysm	Size/no. of aneurysms	Surgical management/ time to surgery	Outcome
1	44/f	GCS 3, WFNS 5, bilateral fixed pupils	CT scan	II	No	Rt	15 mm	10 mm	Pericallosa Rt	5 mm/2	Craniectomy, hematoma evacuation and delayed clipping/4 h	Full recovery, mild cognitive deficits GOS 5, mRS 1
2	50/f	GCS 13, WFNS 3, mild left-sided hemiparesis	CT scan, DSA	IV	Yes	Rt	9 mm	23 mm	MCA Rt	11 mm/2	Craniectomy, hematoma evacuation and delayed coiling/12 h	Mild left-sided arm paresis, GOS 4, mRS 2
3	39/m	GCS 4, WFNS 5, bilateral fixed pupils	CT scan, CTA	III	No	Rt	10 mm	14 mm	ICA-Pcom Rt	5 mm/1	EVD, craniectomy, hematoma evacuation and immediate clipping/18 h	Residual left-sided hemiparesis, GOS 4, mRS 2
4	58/f	GCS 5, WFNS 5, dilatation of the right pupil	CT scan, CTA	IV	Yes	Rt	5 mm	4 mm	MCA Rt	14 mm/1	Craniectomy, hematoma evacuation and immediate clipping/3 h	Full recovery, mild cognitive deficits GOS 5, mRS 1
5	45/f	GCS 4, WFNS 5, dilatation of the right pupil	CT scan, CTA	II	No	Rt	20 mm	18 mm	ICA-Pcom Rt	7 mm/2	Craniectomy, hematoma evacuation and immediate clipping/2 h	Gait ataxia, GOS 4, mRS 3
6	68/f	GCS 15, WFNS 1, right oculomotor paresis	CT scan, DSA	II	No	Rt	10 mm	6 mm	Distal ICA–Pcom Rt	2 mm/1	Craniotomy, hematoma evacuation and immediate clipping/6 h	Full recovery, no symptoms at all, GOS 5, mRS 0
7	27/f	GCS 3, WFNS 5, bilateral fixed mydriasis	CT scan, CTA	III	No	Rt	10 mm	7 mm	Pericallosa Rt	12 mm/1	Craniectomy, hematoma evacuation/1 h	Deceased, GOS 1, mRS 6

[a] All patients presented with stable hemodynamic conditions

seventh patient died within 8 h post surgery from uncontrollable raised intracranial pressure (GOS 1; mRS 6).

There was no correlation between outcome (GOS; mRS) and initial GCS, SAH grade on admission, neurological deficits, degree of midline shift, or thickness of the aSDH. Considering the site of the ruptured aneurysms in relation to the location and expansion of the subdural blood collection, we found that all aSDH were formed at the unilateral site of the cerebral aneurysm location.

Illustrative cases

Case 1 A 44-year-old female patient was admitted to a local hospital emergency room with a history of a bicycle accident. She suffered severe headache, was disoriented and drowsy, but did not present with any neurological deficits. Within minutes, the patient became comatose, was intubated, and was transferred to our hospital. On admission, the patient presented with GCS 3 and bilaterally fixed pupils (WFNS 5). The non-contrast-enhanced head CT scan demonstrated a right fronto-temporal aSDH, resulting in a 5-mm midline shift with no associated cerebral contusions or skull fractures and only small traces of subarachnoid blood (Fisher grade II).

An emergency craniectomy and evacuation of the aSDH was performed. The papillary state normalized after surgery. A source of bleeding was not identified. Later on, eyewitnesses reported that the patient collapsed uninfluenced. An angiogram revealed a right pericallosal artery aneurysm and an incidental right carotid-ophthalmic aneurysm. The pericallosal artery aneurysm was clipped and the bone flap reimplanted during the same procedure. Two weeks after the bleed the patient had made a good recovery and was discharged to her local hospital. Two months later, the right carotid-ophthalmic aneurysm was clipped electively. At 8 months' follow-up, the patient had no formal neurological deficit (GOS 5). The patient was able to resume her normal daily activities and previous job (mRS 1).

Case 5 A 45-year-old female who experienced a sudden, severe headache without a history of trauma was first admitted at a referring hospital. During initial CT scan the patient rapidly became drowsy and deteriorated to GCS 3, with subsequent intubation and transfer to our institution. CT scan showed a massive right fronto-temporo-parietal aSDH 20-mm thick, resulting in 18-mm midline shift with only traces of SAH in the right cistern ambiens (Fisher grade II). The patient was immediately referred to our institution. On admission, the patient presented in deep coma with no motor response and fixed right-sided pupil (WFNSV). A suspicious Pcom configuration on initial CTA was verified during urgent craniectomy and hematoma

evacuation. A right-sided Pcom aneurysm was found and clipped in the same operative procedure. The papillary state normalized during decompressive surgery. Following an uneventful hospitalization, the patient was transferred for rehabilitation. Despite extensive neurorehabilitation, the patient recovered with moderate disability, comprising gait ataxia (GOS 4; mRS 3) (Fig. 1).

Case 7 A 27-year-old female was transferred to our hospital with a history of sudden, vigorous postcoital headache and rapid neurological deterioration. The paramedics found the patient in a deep comatose state with bilaterally dilated and fixed pupils (WFNS 5). At the time of admission, the patient's hemodynamic situation was stable. Initial CT showed a 10-mm thick right-sided aSDH and interhemispheric SAH (Fisher grade III). Simultaneous CTA revealed a pericallosal artery aneurysm. The patient's circulation became unstable during hematoma evacuation and she died 8 h after decompressive craniotomy and hematoma evacuation (GOS 1; mRS 6).

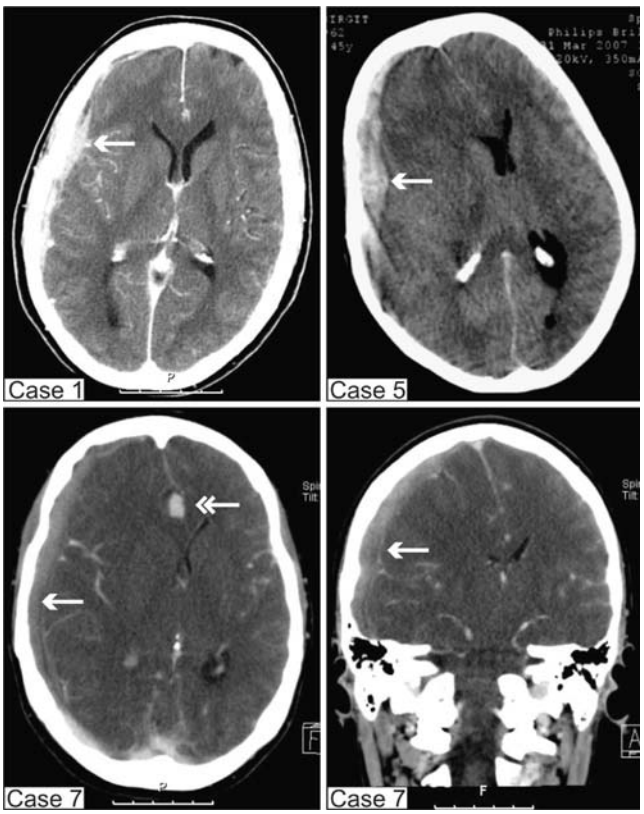

Fig. 1 Acute subdural hematomas (*white arrows*) causing moderate (*Case 1*) to severe (*Case 5*) mass effect with significant midline shift and uncal herniation (*Case 7*). *Left lower panel* (*Case 7*) shows extensive acute subdural hematoma formation from ruptured pericallosal artery aneurysm (*double white arrow*)

Discussion

Head injuries account for nearly 95% of all aSDH [18]. In the majority of cases, the history will distinguish a traumatic from a spontaneous cause. Diagnosis can be complicated in aSDH patients in whom loss of consciousness due to aneurysm rupture results in or mimic an accident (Case 1). CT features such as super-added contusions, skull fractures, punctuate hemorrhage (sheering injuries) in head-injured patients, or hematomas related to common aneurysm sites [20] with extension in the subarachnoid space may help in determining the etiology. Only in rare instances does pure aSDH occur in the absence of SAH [17]. However, specific radiologic features of aneurysmal aSDH are not reported. In our case series, all aSDHs were associated with SAH, and two patients presented with additional ICH. Nevertheless, the diagnosis of aneurysmal aSDH must be considered also in the absence of ICH or SAH when there is no history or sign of trauma.

Localisation of aneurysms associated with aSDH In a review of 56 series containing 148 cases, Fox [7] found that 43% of aneurysms were located in the ICA, 22% at the MCA, 22% at the anterior communicating artery, 4% vertebrobasilar, and 9% in other locations [19]. Strang et al. [24], reporting on 60 cases of ruptured aneurysm, found the location to be at the ICA in 53%, at the MCA in 27%, and at the ACA in 17%. The sites of origin responsible for the aSDH in our series were in line with these published figures, with ICA-Pcom aneurysm in three cases and MCA aneurysms in two cases. Interestingly, the highest predominance of ICA-Pcom origin is found in cases of pure aSDH without associated ICH (58%) [15] or SAH (67%) [17]. Aneurysms arising from the segment of the ICA, where the vessel passes through the subdural space on its way to the circle of Willis (plica petroclinoidae), may cause direct blood flow into the subdural space. Rupture of an aneurysm adhering to either the tentorium or falx and located in the subdural space may also cause pure SDH. Three cases of pure aSDH have been described in which distal ACA aneurysms were involved in adhesion of the aneurysm domes to the falx in the subdural space [10]. In rare cases, rupture of cortical aneurysms results in aSDH without SAH [11, 29]. In one case, a giant aneurysm extended with its fundus into the subdural space [16].

Pathophysiology of aSDH in SAH The occurrence of aSDH due to ruptured aneurysms adherent to the subdural space would in part explain the high predominance of ICA-Pcom aneurysm rupture in cases of pure aSDH [12, 15, 17]. However, aneurysm rupture in combination with aSDH, SAH, or ICH, as noted in all our patients, is not explained

by these mechanisms. The most frequently mentioned hypothesis of aSDH with SAH or ICH is based on the two mechanisms proposed by Clarke and Walton [5]: (1) bleeding directly into the subdural space and (2) bleeding into the subdural space via the subarachnoid space.

In the first situation, with bleeding directly into the subdural space, a slight aneurysm leak creates a local hematoma. Organization of the hematoma involves the arachnoid and pia mater, so that the aneurysm is no longer lying free in the subarachnoid space (adhesions form between the aneurysm fundus and the inner layer of the arachnoid membrane). As the arachnoid becomes a part of the aneurysm wall, it virtually projects into the potential subdural space. Interestingly, three out of seven patients in our series presented with more than one aneurysm on angiography. Considering the evidence that presence of multiple aneurysms represents a risk factor for sentinel bleeding [3], one might speculate that the aSDH of these patients developed/formed according to the above-mentioned mechanism.

In the second situation, with bleeding into the subdural space via the subarachnoid space, the arachnoid, a delicate membrane, can be destroyed at some distant weak point by a high-pressure bleed, and thus blood reaching the subdural space can collect to produce a subdural hematoma. These two pathophysiological sequelae may explain the most frequent situation of aneurysmal aSDH in combination with SAH.

A third mechanism has been proposed to include cases of aSDH formation with ICH and SAH [2]: massive intracranial hemorrhage ruptures through the cortex and lacerates the arachnoid membrane, and blood extravasation occurs into the subdural space. Proposed mechanisms of aSDH with and without SAH or ICH are summarized in Table 2.

Treatment options of aSDH from ruptured aneurysm Given the rarity of the disease, no guidelines have been established. In most reports the patient's clinical features on admission are bad; often patients present in a comatose state, with papillary disturbance (uncal herniation) and unreactive and dilated pupils. Differential diagnosis, as well as treatment modalities, can be complicated by the rapid clinical course and a mixture of symptoms due to the ruptured aneurysm, on the one hand, and the mass effect of the hematoma, on the other hand.

In patients who are neurologically stable, management may proceed in a standard manner with DSA. This procedure offers guidance for decisions on the best aneurysm occlusion strategy and surgical approach. If the aneurysm is suitable for endovascular obliteration, it can be occluded during the same procedure, followed by craniotomy and hematoma evacuation. If the aneurysm is not suitable for endovascular occlusion, decompression and clipping can be performed in addition to hematoma evacuation.

In the management of comatose patients or patients whose level of consciousness is deteriorating rapidly, the choice of initial diagnostics is more demanding. The aSDH may be the major determinant of neurological grade, and prompt hematoma evacuation may be life-saving. The emergency situation forces the neurosurgeon to postpone DSA and to determine the surgical strategy based on CTA findings. If CTA is negative for bleeding sources, intraoperative exploration of the entire anterior circulation was recommended by some authors, since in nearly 90% of cases of aneurysmal aSDH the responsible aneurysm is found in the anterior circulation [7, 24]. Initial endovascular therapy delays surgical treatment and cannot be considered in rapidly deteriorating patients. However, separate endovascular obliteration after a surgical procedure for evacuation of the aSDH remains a good option to avoid an additional operative procedure in a critically ill patient. If feasible, intraoperative angiography (i.e., hybrid operating room) would be the method of choice to verify the angioarchitecture of the intracranial circulation and to guide clipping at the time of hematoma evacuation.

Table 2 Mechanisms of aSDH due to ruptured cerebral aneurysm with or without SAH and/or ICH

aSDH without SAH

I. Rupture of an aneurysm arising from a bleeding point in the plica petroclinoidea
II. Rupture of an aneurysm adhering to the tentorium or falx.
III. Rupture of a cortical artery aneurysm protruding through the arachnoid.
IV. Rupture of an aneurysm extending into the subdural space.
V. Rupture of a cortical aneurysm.

aSDH with SAH or ICH

I. Sentinel hemorrhages cause adhesion between the aneurysm and the arachnoid, creating a path for the blood, and the final rupture occurs into the subdural space.
II. High-pressured stream of blood from the leaking aneurysm may burst through the arachnoid at some distant weak point into the subdural space.
III. Massive intracranial hemorrhage ruptures through the cortex and lacerates the arachnoid membrane, with subsequent jetting of blood into the subdural space.

Despite poor neurological presentation, all but one of the patients in our series reached a satisfactory outcome. In the past, patients suffering from acute subdural hematomas as a result of a ruptured aneurysm were considered unsalvageable [1]. Since then, resuscitation, diagnostic and emergency treatment options have improved [19, 23, 28]. However, if the patient's medical condition does not allow immediate intervention, a good recovery may not be possible even today [28]. Patients with unstable circulation would not have qualified for immediate surgery and would have been at exceptional risk of poor outcome. Therefore, the good results of this series have to be seen in consideration of the fact that all our patients' hemodynamic situation was stable at the time of admission. Weir at al. [27] stated in their 18 cases that patients who died showed greater midline shifts, larger hematomas, and a higher percentage of preoperative herniation. In our series, these parameters were not predictive of survival, nor did they correlate with outcome.

In summary, this study confirms the low incidence of aSDH associated with aneurysmal SAH. Often there is no primary damage of brain tissue, and the subdural blood collection mimics a clinical situation that appears to be worse than can be attributed to the SAH. In this situation, urgent decompressive craniectomy and hematoma evacuation with immediate aneurysm clipping can lead to a favorable outcome. Our results support routine CTA in patients presenting with aSDH and no clear history of trauma

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Comment

In non-comatose patients with aSDHs associated with SAH, it is of utmost importance to get accurate clinical information regarding the symptom onset and co-morbidity, including possible anticoagulation

therapy. Small amounts of blood may be seen relatively often in the subdural space in patients with massive SAH, but large hematomas necessitating removal per se are rare. The history of typical symptoms of SAH together with blood is SA and subdural space with no trauma history naturally indicates aneurysmal origin in most cases, and for anatomical reasons ICA-PCom being the most common in the present series and literature. CTA is replacing DSA rapidly in primary aneurysm diagnostics in many centers as a non-invasive and quick method. As the authors conclude, it should be performed with a low threshold to exclude aneurysmal origin when in doubt, especially in comatose patients presenting with aSDH associated with SAH, possibly with no previous medical history available on admission.

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