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Prognosis of patients in coma after acute subdural hematoma due to ruptured intracranial aneurysm

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DISCLOSURE / CONFLICT OF INTEREST

We, as the authors, state that to the best of our knowledge, no aspect of our personal or professional circumstances, either currently or in the past 12 months, places any of us in the position of being in conflict of interest with any entity that may potentially benefit from or be harmed by the publication of the results of our study.

ABSTRACT

Acute subdural hematomas (aSDH) secondary to intracranial aneurysm rupture are rare. Most patients present with coma and their functional prognosis has been classically considered to be very poor. Previous studies mixed good-grade and poor-grade patients and reported uneven outcomes. We reviewed our experience by focusing on coma patients only and hypothesized that aSDH might worsen initial mortality but not long-term functional outcome. Between 2005 and 2013, 440 subarachnoid hemorrhage (SAH) patients were admitted to our center. Nineteen (4.3%) were found to have an associated aSDH and 13 (2.9%) of them presented with coma. Their prospectively collected clinical and outcome data were reviewed and compared with those of 104 SAH patients without aSDH who presented with coma during the same period. Median aSDH thickness was 10 mm. Four patients presented with an associated *"aneurysmal cortical laceration"* and only 1 had good recovery. Overall, we observed good long-term outcomes in both SAH patients in coma presenting aSDH and those without aSDH (38.5% vs. 26.4%). Associated aSDH does not appear to indicate a poorer long-term functional prognosis in SAH patients presenting with coma. Anisocoria and brain herniation are observed in patients with subdural hematoma thicknesses that are smaller than those observed in trauma patients. Despite a high initial mortality, early surgery to remove the aSDH results in a good outcome in over 60% of survivors. Aneurysmal cortical laceration appears to be an independent entity which shows a poorer prognosis than other types of aneurysmal aSDH.

Keywords: Acute Subdural Hematoma; Ruptured Intracranial Aneurysm; Subarachnoid Hemorrhage; Coma; Outcome; Prognosis.

INTRODUCTION

Despite advances in the management and treatment options for subarachnoid hemorrhage (SAH), initial clinical presentation and level of consciousness remain the strongest predictors of neurological outcome. According to previous studies, 50% of patients who initially present with a Glasgow coma score (GCS) of 8 or lower die or remain severely disabled 6 months after hemorrhage [1,2]. In addition to clinical predictors, promising neuroimaging exams such as perfusion computed tomography (CT) have been incorporated into the initial SAH diagnostic workout in recent years in an attempt to better identify patients with poor functional outcome [3,4]. Regardless of the predictors used, the certainty of poor long-term outcome may help the physician to select the most appropriate treatment strategy, which may include, in certain patients, watchful waiting and excluding the aneurysm in a deferred fashion [5].

Acute subdural hematoma (aSDH) related to intracranial aneurysm rupture occurs in 2% to 10% of SAH patients [6]. Approximately 50% of SAH patients with associated aSDH initially present with coma. The causes leading to aSDH in SAH patients are diverse and have been scarcely studied. Previous sentinel bleeds may cause the aneurysm dome to adhere to the surrounding arachnoid, which could then be torn by a new bleed and easily drain into the subdural space. A powerful jet of blood from an aneurysm rupture could cause a direct intraparenchymal lesion opening into the subdural space and thus produce an aSDH. Certain aneurysm locations may be more prone to aSDH due to their specific anatomy. Despite the lack of studies involving large aSDH patient series, the prognosis of these cases has classically been considered to be poor, leaving doctors to wonder whether treatment of these comatose patients might reduce mortality in exchange for an increased number of severely disabled survivors.

This report describes our experience in the management of patients with SAH and aSDH. Unlike previous studies, our review focuses on patients who initially presented with coma. We hypothesized that the neurological outcome of these patients, provided they undergo early aSDH evacuation, does not differ from same-grade SAH patients without aSDH.

CLINICAL MATERIALS AND METHODS

All SAH patients treated in our center were registered in an internal database and their outcomes were prospectively followed up. In the period between January 2005 and September 2013, 440 patients with aneurysmal SAH were registered in the database. Nineteen patients (4.3%), 13 of whom presented with coma (2.9%) (GCS≤8), had concomitant aSDH. We evaluated their demographic features, neurological status at admission, diagnostic imaging, clinical management, and neurological outcome. We also evaluated the neurological outcomes of same-grade SAH patients (104 in our registry) who did not present with concomitant aSDH.

As part of routine examination, every patient with suspected SAH underwent a CT scan on arrival to the emergency department. Maximum aSDH thickness and brain midline shift were measured. Each patient then underwent either an angio-CT scan or a complete cerebral angiography according to his or her specific clinical situation.

All aSDH patients underwent follow-up and their 6-to-12-month outcome was evaluated. Functional status was assessed using the Glasgow outcome scale (GOS). Good functional outcome corresponded to a GOS score of 4 to 5 (good recovery or moderate disability) and bad functional outcome corresponded to a GOS score of 1 to 3.

Data were analyzed and summarized using the SPSS for Mac program (Version 20, SPSS, Inc., New York, USA). Characteristics of patients included in the study were evaluated using

only descriptive statistics. Due to the small number of aSDH patients available, comparison analysis was deemed high risk of statistical error type II and was disregarded.

RESULTS

Of the 19 SAH patients who developed a subdural hematoma, 13 (2.9 %) presented with coma. In this patient subgroup of 4 men and 9 women, the median age was 51 years (min: 40, max: 59). During the first neurological evaluation, the majority of patients (10/13, 76.9%) showed anisocoria and most (9/13, 69.2 %) scored either 3 or 4 GCS points **(Table 1)**. Four of the patients presenting with anisocoria had an aneurysm located in the posterior communicating artery (PCoA), possibly indicating that this sign was due to direct compression of the aneurysm over the third cranial nerve. In the remaining cases, however, anisocoria could not be explained by any cause other than uncal brain herniation.

The most common aneurysm location was the PCoA, which harbored 5 aneurysms (38.4 %). The remaining cases were evenly distributed between the middle cerebral artery (MCA) (4 aneurysms, 30.7 %) and the anterior cerebral artery (ACA) (4 aneurysms, 30.7 %). Three patients (3/13, 23.1%) presented with 1 or more unruptured aneurysms, a frequency that was similar to that of patients without aSDH [19 cases out of 104 (18.2%)].

All patients presented SAH associated with aSDH with the exception of 1 patient, who presented with a pure aSDH (without SAH). In 4 cases (30.7%), intraparenchymal hematoma (IPH) associated with aSDH was observed. Maximum subdural hematoma thickness on the initial CT ranged from 3 mm to 15 mm, with a median value of 10 mm. The median observed midline shift was 8 mm (min:2, max:17 mm).

Exclusion of the ruptured aneurysm was performed using microsurgical clipping in 9 patients and endovascular coiling in the remaining 4 patients. Of the 4 patients who underwent endovascular treatment, 2 also required surgical evacuation of the aSDH. In the other 2 endovascular cases, subdural hematoma thickness and midline shift were both less than or equal to 3 mm. Eight patients required primary decompressive craniotomy due to brain swelling. After the acute phase, 2 of the 13 patients (15.4%) required a ventriculoperitoneal shunt.

Five patients died during the perioperative period (38.5 %). Out of the surviving patients, 5 (5/8; 62.5%) had a good functional outcome (GOS 4-5) and 3 remained in poor neurological condition at the 6-to-12 month follow-up. In the cohort of SAH patients without associated subdural hematoma, 104 patients presented with a GCS less than or equal to 8. During follow-up, 2 patients were lost. Of the 102 patients with outcome data, 27 (26.4%) showed good functional status (GOS 4-5) at the most recent follow-up.

DISCUSSION

Patients with concomitant aSDH represent 2% to 10% of SAH patients [6,7]. Most of these cases are associated with PCoA aneurysm rupture. Due to the cisternal route followed by the internal carotid artery before entering the circle of Willis, PCoA aneurysms most often project their dome into the subdural space and thus facilitate the formation of subdural hematomas [8,9], which in some exceptional cases may even result in a pure aSDH [10]. In our series, most aSDH cases (38.4%) were related to PCoA aneurysms, while ACA and MCA aneurysms were diagnosed less frequently. aSDH associated with posterior circulation aneurysm is extremely uncommon. In the aSDH patients in the present study, all aneurysms were located

in the anterior circulation. One finding consistent with previous studies was that we did not find a higher percentage of multiple aneurysm cases among our aSDH patients [9,11].

In addition to the specific parent artery, 3 causes are classically described to explain aSDH following an aneurysmal rupture. One theory states that previous bleeding would create adhesions between the arachnoid and the pia mater and end up pulling on the aneurysm dome and projecting it into the subdural space. Another hypothesis suggests that a high pressure bleed either tears the arachnoid or even breaks through the surrounding parenchyma and lacerates the cortical surface, causing drainage into the subdural space [9,12]. Finally, giant aneurysms that surface through the fissures to the subdural space have also been reported as an uncommon cause of aSDH[13, 14].

Surgical Management

Surgical management of SAH with associated aSDH is challenging due to the patient's critical condition. Moreover, fear of aneurysm rebleed during cranial decompression usually permeates the procedure. At our center, however, only 1 intraoperative aneurysm rupture has been recorded, an event that occurred during dome dissection. Rapid evacuation of the subdural hematoma does not seem to provoke any degree of transmural pressure change in the aneurysm wall that could trigger intraoperative rebleeding. This argument, together with the need for rapid subdural hematoma evacuation and the fact that most aneurysms are located in the anterior circulation, supports surgical treatment as the first management option for these patients. Furthermore, early surgery would also allow primary decompressive craniotomy to be performed for easier postsurgical management of intraoperative brain edema [15].

Surgical treatment should also be the first management option in patients whose subdural hematoma is not large. In 10 of the 13 patients in our study, maximum thickness of the

subdural hematoma was less than or equal to 10 mm. Although this thickness would not necessarily require surgical evacuation in a traumatic patient [16,17], most of the aSDH patients had signs of raised intracranial pressure and anisocoria. Blood volume also occupying the arachnoidal cisterns and brain edema following the initial aneurysm burst can result in raised intracranial pressure and anisocoria with hematoma thicknesses that are less than or equal to 10 mm.

Functional Prognosis and Mortality

Most aSDH patients present with coma, indicating an insidious prognosis. However, long-term functional outcome for these patients is controversial and there are discrepancies among different authors. In many comatose patients, brain shift and mass effect produced by aSDH are thought to be the cause of initially poor neurological status. Therefore, early aSDH evacuation in these patients could result in a functional outcome that would be better than initially estimated. On the other hand, some patients may present with coma due to a more aggressive aneurysm rupture, a factor that may be key in determining prognosis. In these cases, early evacuation of the subdural hematoma might not improve neurological outcome.

In patients with a more aggressive aneurysm rupture, a blood jet from the aneurysm could cause intraparenchymal hematoma (IPH) and lacerate the cortex on its way to the subdural space. Out of the 4 patients with aneurysmal cortical laceration (aSDH with IPH) in our series, only 1 with small-volume IPH (8 cc) had good functional outcome. *"Aneurysmal cortical laceration"* (aSDH with IPH) as an entity has not been previously reported in the literature and should be considered individually because it may have a similarly poor prognosis to the "burst lobe" observed in trauma patients[17-20].

Aneurysmal subdural hematomas without SAH (pure aSDH) are rare entities that have been scarcely reported. They usually present a better functional outcome than in other subdural hematomas. In fact, the single patient with a pure aSDH (without associated SAH) at our center achieved a full recovery, scoring a GOS 5 at the last available follow-up. A minor transitory brain injury appears to indicate a better prognosis when compared with other subdural hematomas [10].

Previous studies comparing the prognosis of patients with SAH associated with aSDH and those with SAH alone are usually based on small case series and show heterogeneous results [9,14,21-27]. Most of these series report the results of patients with good initial status together with the results of those with poor and extremely poor initial status, which may account for the aforementioned heterogeneity [28]. An additional aspect to consider is the timing in assessing patient functional outcome. The 3-month cut-off used by many studies could easily run short in determining the final functional status of the patient [29]. The most extensive literature review conducted by Schuss et al. [30] included 10 studies with 111 patients, 83 of whom had a poor initial neurological status and only 19 (23 %) of whom achieved a good outcome.

Our report focuses on aSDH patients who presented with a poor neurological condition that could have prompted a “limitation of the therapeutic effort”, or even care withdrawal. In this context, Westermaier et al. outlined a policy of aggressive initial treatment that yielded good functional outcome in 5 of their 7 comatose patients. Care was withdrawn only in 1 of the reported patients, who later died [26]. Our results are less encouraging: good functional outcome was observed in 5 out of 8 (62.5 %) patients who survived the acute phase. Unlike the aforementioned study, we observed a high perioperative mortality rate (5 patients; 38.5%).

If we take the high mortality rate in the acute phase (38.5 %) out of the equation, our results suggest that early treatment of comatose patients with evacuation of the subdural hematoma and decompressive craniotomy yields good functional outcome in more than half of the initial survivors. These results are similar to those of patients with an initially poor neurological status due to SAH alone, both in our series and in those described by other authors[1,2]. Therefore, despite the high mortality rate of comatose aSDH patients in the acute phase, the prognosis of survivors would not be worse than patients with SAH alone who also present with coma.

Study Limitations and Future Research

The association of SAH with aSDH is uncommon and thus studies on this topic are necessarily based on a small number of cases. Consistently, the number of patients in our report was not large enough to draw solid conclusions and we could only suggest likely scenarios that warrant further investigation. Nonetheless, all the data from small series may enable a future broader analysis that could definitively advance the knowledge in this topic.

Patients with aneurysmal aSDH and a component of IPH ("*aneurysmal cortical laceration*") appear to have the poorest clinical outcome. Cortical laceration suggests a more aggressive jet of blood that would not only be able to break the arachnoid but also make its way through the cortex. This entity should be considered separately because the origin of the subdural hematoma and the therapeutic strategies required are different, and prognosis is generally poor.

CONCLUSION

Patients in coma due to aSDH do not appear to have a worse prognosis than comatose patients with SAH alone. Early surgical treatment, including evacuation of the hematoma and primary cranial decompression (if required), may improve the odds of good functional outcome for survivors despite a significant mortality rate in the acute phase. Unlike trauma patients, patients with subdural hematomas with thicknesses that are lower than expected can present with signs of brain herniation and anisocoria. Factors other than hematoma thickness, such as the volume of blood in the cisterns, possible cortical laceration, or initial brain edema, may play a role in the neurological deterioration observed in these patients.

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Table

Table 1: Summary of patients with aSDH.

	Age and Gender	GCS at Arrival	Anisocoria	Aneurysm location	Midline Shift	Hematoma Thickness	Fisher Grade	IPH	aSDH Evacuation	Aneurysm Treatment	IOR	DC	GOS
Patient													
1	57 F	3	Yes	PCoA	12 mm	10 mm	4	No	Yes	Clip	No	Yes	3
2	48 F	3	Yes	PCoA	7 mm	10 mm	4	Yes (8cc)	Yes	Clip	No	Yes	4
3	52 M	3	Yes	ACA	14 mm	6 mm	4	Yes (45cc)	Yes	Clip	No	No	2
4	45 F	3	Yes	ACM	6 mm	10 mm	3	Yes	Yes	Clip	No	Yes	1
5	50 F	3	Yes	PCoA	9 mm	6 mm	4	No	Yes	Clip	No	No	1
6	55 M	3	No	ACA	3 mm	3 mm	4	Yes (22cc)	Yes	Clip	No	Yes	1
7	49 M	3	Yes	ACA	2 mm	3 mm	3	No	No	Coil	No	No	1
8	55 F	4	Yes	ACA	11 mm	12 mm	4	Yes	Yes	Coil	No	Yes	4
9	51 F	4	Yes	ACM	15 mm	12 mm	4	No	Yes	Clip	Yes	Yes	1
10	45 M	7	No	ACA	3 mm	3 mm	4	No	No	Coil	No	No	4
11	55 F	7	Yes	ACM	17 mm	15 mm	4	Yes (26cc)	Yes	Clip	No	Yes	2
12	40 F	7	Yes	PCoA	8 mm	10 mm	1	No	Yes	Coil	No	Yes	5
13	59 F	8	No	PCoA	6 mm	10 mm	4	No	Yes	Clip	No	No	4
14	53 M	10	Yes	PCoA	12 mm	10 mm	2	No	Yes	Coil	No	Yes	4
15	84 F	10	Yes	PCoA	10 mm	8 mm	4	No	Yes	Clip	No	No	3
16	78 F	12	No	PCoA	2 mm	3 mm	3	No	No	Coil	No	No	5
17	60 M	13	No	PCoA	8 mm	7 mm	4	Yes (50cc)	Yes	Clip	No	No	4
18	70 M	14	No	ACA	3 mm	2 mm	4	No	Yes	Clip	No	No	4
19	55 F	14	No	ACM	15 mm	13 mm	3	No	Yes	Clip	Yes	No	4

2.2. Malformaciones arteriovenosas cerebrales

La resección de las MAVs se basa en la disección cuidadosa del espacio subaracnoideo, la delimitación parenquimatosa de los márgenes y la oclusión de las arterias nutricias. Durante el procedimiento quirúrgico, se producen leves sangrados procedentes de la MAV al disecar los vasos que afluyen a la misma. Habitualmente, estos sangrados son controlados por el cirujano y forman parte de la normal disección de la lesión. Menos frecuente es la rotura intraoperatoria de las MAVs, se da de forma poco habitual en un 5% de las ocasiones(83) y lleva al cirujano a una situación de estrés en la que se precipita la finalización de la cirugía.

La rotura intraoperatoria se define como una situación de sangrado masivo e incontrolable que nos obliga a la exéresis de la MAV de forma urgente. La respuesta quirúrgica a una rotura intraoperatoria es distinta a la de una aneurisma cerebral. En el caso de los aneurismas, existe una secuencia que consiste en taponar, succionar, conseguir un control proximal y/o distal con clips temporales y finalmente clipar el cuello aneurismático. En el caso de las MAVs, la rotura puede ser dramática debido a que el sangrado habitualmente procede de distintas zonas y no se tiene la posibilidad de un control proximal y distal como pasa con los aneurismas. En el tercer trabajo presentado nos centramos en las causas que se relacionan con una rotura intraoperatoria, las distintas técnicas a seguir para evitar esta situación extrema y su correlación con el pronostico funcional.

La localización de la MAVs también parece ser un indicador de evolución clínica del paciente. Determinadas localizaciones tienen una mayor asociación con el sangrado y con una mala evolución clínica. En nuestro último articulo nos centramos en un tipo particular de MAVs que son las de fosa posterior. Estas lesiones poco habituales se caracterizan por presentar una alto porcentaje de sangrado(80%) con una alta tasa de mortalidad inicial(84,85). Estudiamos el

pronóstico funcional de estos pacientes así como las características de estas lesiones.