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Clinical Study

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

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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 variable outcomes. We reviewed our experience by focusing on patients in coma 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 these presented with coma. Their prospectively collected clinical and outcome data were reviewed and compared with that 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 one had good recovery. Overall, we observed good long-term outcomes in both SAH patients in coma with aSDH and those without aSDH (38.5% *versus* 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 aSDH 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.

1. 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 Scale score (GCS) of 8 or less die or remain severely disabled 6 months after hemorrhage [1,2]. In addition to clinical predictors, promising neuroimaging examinations such as perfusion CT have been incorporated into the initial SAH diagnostic workup 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 rarely studied. Previous sentinel bleeds may cause the aneurysm dome to adhere to the surrounding arachnoid, which could then be torn by a new hemorrhage which can then drain into the subdural space. A powerful jet of blood from an aneurysm rupture may 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 patients has classically been considered to be poor, leaving the question as to 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

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hypothesized that the neurological outcome of these patients, provided they undergo early aSDH evacuation, does not differ from same-grade SAH patients without aSDH.

2. 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 $\leqslant$ 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 at the emergency department. Maximum aSDH thickness and brain midline shift were measured. Each patient then underwent either an angio-CT scan or catheter cerebral angiography according to their 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 or 5 (good recovery or moderate disability) and poor functional outcome corresponded to a GOS score of 1–3.

Data were analyzed and summarized using the Statistical Package for the Social Sciences (version 20, IBM, Armonk, NY, 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 to be at high risk of statistical error type II and was not undertaken.

3. Results

Of the 19 SAH patients who developed aSDH, 13 (2.9%) presented with coma. In this patient subgroup of four men and nine women, the median age was 51 years (range 40–59). During the first neurological evaluation, the majority of patients (10/13, 76.9%) showed anisocoria and most (9/13, 69.2%) had a GCS score of either 3 or 4 (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 by the aneurysm of the third cranial nerve. In the remaining patients, however, anisocoria could not be explained by any cause other than uncal brain herniation.

The most common aneurysm location was the PCoA, which harbored five aneurysms (38.4%). The remaining cases were evenly distributed between the middle cerebral artery (four aneurysms, 30.7%) and the anterior cerebral artery (four aneurysms, 30.7%). Three patients (23.1%) also had one or more unruptured aneurysms, a frequency that was similar to that of patients without aSDH (19 patients out of 104 [18.2%]).

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

Exclusion of the ruptured aneurysm was performed using microsurgical clipping in nine patients and endovascular coiling in the remaining four patients. Of the four patients who underwent endovascular treatment, two also required surgical evacuation of the aSDH. In the other two 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, two of the 13 patients (15.4%) required a ventriculoperitoneal shunt.

Five patients died during the perioperative period (38.5%). Out of the surviving patients, five (5/8; 62.5%) had a good functional outcome (GOS 4–5) and three remained in a poor neurological condition at 6 to 12 month follow-up. In the cohort of SAH patients without associated aSDH, 104 patients presented with a GCS $\leqslant$ 8. Two patients were lost to follow-up. Of the 102 patients with outcome data, 27 (26.4%) showed good functional status (GOS 4–5) at the most recent follow-up.

4. 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 anterior cerebral artery and middle cerebral artery aneurysms were diagnosed less frequently. aSDH associated with a 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 incidence of multiple aneurysms among our aSDH patients [9,11].

In addition to the specific parent artery, three 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 pull on the aneurysm dome, projecting it into the subdural space. Another hypothesis suggests that a high pressure hemorrhage 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].

4.1. Surgical management

Surgical management of SAH with associated aSDH is challenging due to the critical condition of the patient. In addition, fear of aneurysm rebleed during cranial decompression is a concern during the procedure. At our center, however, only one intraoperative aneurysm rupture was recorded, an event that occurred during dome dissection. Rapid evacuation of the subdural hematoma does not seem to provoke a sufficient degree of transmural pressure change in the aneurysm wall that can 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 $\leqslant$10 mm. Although this thickness would not necessarily require surgical evacuation in a trauma patient [16,17], most of the