Acute Aneurismal Bilateral Subdural Haematoma without Subarachnoid Haemorrhage: A Case Report and Review of the Literature

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Spontaneous pure acute bilateral subdural haematoma (ASDH) without intraparenchymal or subarachnoid haemorrhage caused by a ruptured cerebral aneurysm is extremely rare. It can follow rupture of different aneurysms specially located in anterior incisural space; the most frequently encountered location is the PcoA aneurysms as demonstrated in the present case. We present a case report of a PcoA aneurysm presenting as pure bilateral ASDH. A high level of suspicion for bleeding of arterial origin should be maintained in all cases of acute subdural haematoma without history of trauma. The neurological status on admission dictates the appropriate timing and methodology of the neuroradiological investigations.

1. Introduction

In the majority of cases, acute subdural haematomas (ASDHs) are related to head trauma and are typically caused by disruption of superficial cerebral or cortical bridging veins. Spontaneous ASDHs are uncommonly encountered. Arteriovenous malformations [1], cocaine abuse [1], and many other causes have been proposed for this pathology [2]. Hemorrhage of aneurysms often presents as subarachnoid haemorrhage (SAH) and intracerebral haemorrhage (ICH); the ASDH is rare. ASDHs constitute neurosurgical emergencies and immediate treatment must be conducted before neurological deficits become irreversible.

We report a case of a spontaneous bilateral pure ASDH due to rupture of an aneurysm of the left posterior communicating aneurysm (PcoA) with reviewing of literature.

2. Case Report

A 51-year-old controlled diabetic and hypertensive man presented with a history of sudden onset of headache followed by reduction of the level of consciousness (GSC = 7) and right anisocoria, which could be old or just “false, localizing sign.” There was no history of trauma or previous neurological disease. CT scan revealed a bilateral high density subdural haematoma at the brain convexity without significant mass effect, SAH, or ICH (intracerebral hemorrhage); MRI confirmed the CT diagnosis (Figure 1). On admission, patient was somnolent and disoriented. There was no evidence of head injury and general physical examination was unremarkable. On admission, haemoglobin was 9 g/dL; other laboratory data including a coagulopathy screen and CSF analysis were normal.

Owing to rapid clinical deterioration, he was intubated and taken to the operating room. Emergency bilateral frontotemporal craniotomy was performed and haematoma evacuated. There were neither signs of damaged underlying cortex nor typical signs of SAH on the surface. A bleeding cortical artery or other abnormalities could not be identified. On the first postoperative day, he recovered consciousness and was able to follow commands and to move all extremities. Because of the spontaneous course of the ASDH, he underwent...
cerebral angiography. This revealed a left saccular PCoA aneurysm (4.5 × 7.5 mm) (Figures 2(a) and 2(b)). AP-view angiography showed an irregular shape of the aneurysm with additional small outpouching indicating possible point of rupture at the inflow zone (Figures 2(a) and 2(b)). The patient underwent successful coiling of the aneurysm (Figure 2(c)). His following hospital stay was uneventful, and he was discharged on the 11th postoperative day without neurological deficits, being able to return to his normal life.

3. Discussion

ASDHs develop spontaneously in patients without history of trauma or coagulopathy and an aneurysm rupture is responsible for the majority of such cases with incidence of about 0.5% to 7.9% [3–6].

Several mechanisms have been proposed to explain the occurrence of ASDH after aneurysm rupture. Firstly, successive minor sentinel haemorrhages may fix an aneurysm to local arachnoid adhesions (Figure 3) resulting in bleeding directly into the subdural space when an arachnoid tear occurs after aneurysmal rupture or simply through a weak point at the arachnoid membrane without previous sentinel bleeding [6]. A second mechanism may be due to a haemorrhage under high pressure, leading to pia-arachnoid rupture and extravasation of blood into the subdural space, where in this scenario the subdural hematoma may develop secondary to the compensatory decompression of an intracerebral
Figure 3: Diagram illustrating the different stages that possibly make an aneurysm bleed in subdural space.

Biesbroek et al. reported retrospectively on 1757 ruptured aneurysms where 63 cases had an ASDH (as a presenting manifestation). Increasing age, sentinel headache, ICH, and aneurysms at the PCoA were independent risk factors for ASDH. Patients with a basilar or vertebral aneurysm have a low risk for ASDH [4]. The incidence of pure ASDH, without associated ICH or SAH, due to a ruptured aneurysm is extremely rare. The reported cases in literature are summarized in Table 1. The most frequent site of aneurysm causing pure ASDH was at the origin of the PCoA from the internal carotid artery (IC-PC) (60% of the cases), followed by the distal anterior cerebral artery (ACA) (16%) and middle cerebral artery (12%).

In the present case, the bilateral ASDH due to a PCoA aneurysm presented as pure bilateral ASDH. Anatomically, the anterior incisural space, which is located anterior to brainstem, contains the posterior communicating artery (PCoA), anterior choroidal artery, and basilar bifurcation; additionally, it contains the supracallosal portion of the internal carotid artery [4]. This space opens laterally into the part of the Sylvian fissure situated below the anterior perforated substance [4]. This explains the occurrence of a subdural haematoma following aneurysm rupture arising from arteries located in this space like PCoA aneurysm as in the present case, where blood finds its way through the abovementioned pathway to the subdural spaces.

Pure ASDH following rupture of intracranial aneurysm carried a poor prognosis in 34.3% (12 of 357; 14.3% disabled and 22.8% died due to bleeding) of the reported cases in literature. The 22.8% mortality rate in this group of patients is slightly higher than the mortality rate of simple traumatic subdural haematomas (reported to be 20%) [4]. The simple traumatic ASDHs are distinguished from the complicated traumatic subdural haematomas by the absence of parenchymal damage [4]. This assumed that the difference in mortality rate may be due to the initial elevated intracranial pressure caused by the subdural haematoma or by rebleeding of the aneurysm before its occlusion. Therefore, adequate diagnostic investigations and respective prompt treatment are essential for a better outcome. If the patient presents with a stable neurological condition, angiography should be performed prior to surgery to dictate the best strategy. In the presence of a definite bleeding source, emergency surgery should be adopted to evacuate the haematoma and operate on the bleeding source.

If the angiography does not demonstrate the source of bleeding, the patient can be managed conservatively or surgically according to the subsequent evolution of the neurological status. In cases of patients presenting with rapid neurological deterioration, immediate decompression...
Table 1: Cases of pure subdural haematoma (without subarachnoid haemorrhage and without intraparenchymal haematoma) caused by rupture of intracranial aneurysm [5, 6].

| Case | Author | Age (years) | Sex | Symptoms/signs | Location of aneurysm | Location of subdural haematoma | Treatment | Outcome |
|------|--------|-------------|-----|----------------|----------------------|--------------------------------|-----------|---------|
| 1    | Rengachary et al. (1981) [7] | 49 | M | Confusion and dysphasia | Sylvian branch of MCA | Convexity | Haematoma evacuation and clipping | Good |
| 2    | Eggers et al. (1982) [8] | 34 | F | Headache | IC-PC | Convexity | Haematoma evacuation and clipping | Good |
| 3    | Williams et al. (1983) [9] | 18 | F | Coma | IC-PC | Convexity | Haematoma evacuation and clipping | Disabled |
| 4    | Friedman et al. (1983) [10] | 55 | F | Headache | IC-PC | Tentorium and interhemispheric | Clipping | Good |
| 5    | O'Leary et al. (1986) [11] | 28 | F | Coma | MCA | Convexity | None | Dead |
| 6    | Kondziolka et al. (1988) [12] | 43 | M | Coma | IC-PC | Tentorium and convexity | Haematoma evacuation and clipping | Good |
| 7    | Kondziolka et al. (1988) [12] | 38 | F | Coma | IC-PC | Tentorium and convexity | Haematoma evacuation and clipping | Disabled |
| 8    | Shinmura et al. (1989) [13] | 44 | F | Coma | MCA | Convexity | Haematoma evacuation and clipping | Disabled |
| 9    | Onda et al. (1989) [14] | 51 | F | Semicoma | IC-PC | Convexity | Haematoma evacuation and clipping | Disabled |
| 10   | Watanabe et al. (1991) [15] | 27 | M | Semicoma | Distal ACA | Interhemispheric and convexity | Haematoma evacuation and clipping | Dead |
| 11   | Ragland et al. (1993) [16] | 55 | M | Coma | AcomA | Convexity | Haematoma evacuation | Dead |
| 12   | Hatayama et al. (1994) [17] | 55 | M | Semicoma | Distal ACA | Interhemispheric and convexity | Haematoma evacuation and clipping | Good |
| 13   | Hatayama et al. (1994) [17] | 66 | F | Semicoma | Distal ACA | Interhemispheric, convexity, and tentorium | Haematoma evacuation and clipping | Disabled |
| 14   | Ishibashi et al. (1997) [18] | 54 | F | Headache | IC | Tentorium and convexity | Haematoma evacuation and clipping | Good |
| 15   | Satoh et al. (1999) [19] | 58 | F | Semicoma | IC | Convexity | Haematoma evacuation and clipping | Good |
| 16   | Satoh et al. (1999) [19] | 25 | F | Headache | IC | Convexity | Haematoma evacuation and clipping | Good |
| 17   | Satoh et al. (1999) [19] | 22 | F | Coma | IC | Convexity | Haematoma evacuation and clipping | Good |
| 18   | Nonaka et al. (2000) [20] | 52 | F | Coma | IC | Tentorium and convexity | Haematoma evacuation and clipping | Good |
| 19   | Ishikawa et al. (2000) [21] | 62 | M | Headache and ptosis | IC | Tentorium and interhemispheric | Clipping | Good |
| 20   | Inamasu et al. (2002) [22] | 28 | F | Coma | IC | Convexity | Haematoma evacuation | Dead |
| 21   | Araki et al. (2002) [23] | 55 | F | Headache, ptosis, and semicoma | IC | Convexity | Haematoma evacuation and clipping | Good |
| 22   | Blake et al. (2003) [24] | 35 | F | Coma | IC | Convexity | Non | Dead |
| 23   | Katsuno et al. (2003) [25] | 62 | F | Headache, nausea, and dizziness | Distal ACA | Interhemispheric and convexity | Haematoma evacuation and clipping | Good |
| 24   | Shenoy et al. (2003) [26] | 45 | F | Headache and blurring of vision | MCA | Convexity | Haematoma evacuation and clipping | Good |
| 25   | Shenoy et al. (2003) [26] | F | | Semicoma and hemiparesis | IC-PC | Convexity | Haematoma evacuation and clipping | Good |
surgery should be performed before performing angiography. In the absence of intraoperative identification of a cortical arterial rupture or other source of bleeding, complementary postoperative arteriography is required, to rule out sources which could not be detected during surgical evacuation.

In summary, a high level of suspicion for bleeding of arterial origin should be maintained in all cases of ASDH without history of trauma which may mandate vascular assessment as routine.

**Conflict of Interests**

The authors declare that there is no conflict of interests regarding the publication of this paper.

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