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Aneurysmal subarachnoid hemorrhage and acute subdural hematoma, neurosurgical and endovascular rescue. Case report.

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Aneurysmal subarachnoid hemorrhage and acute subdural hematoma, neurosurgical and endovascular rescue. Case report.

Abstract

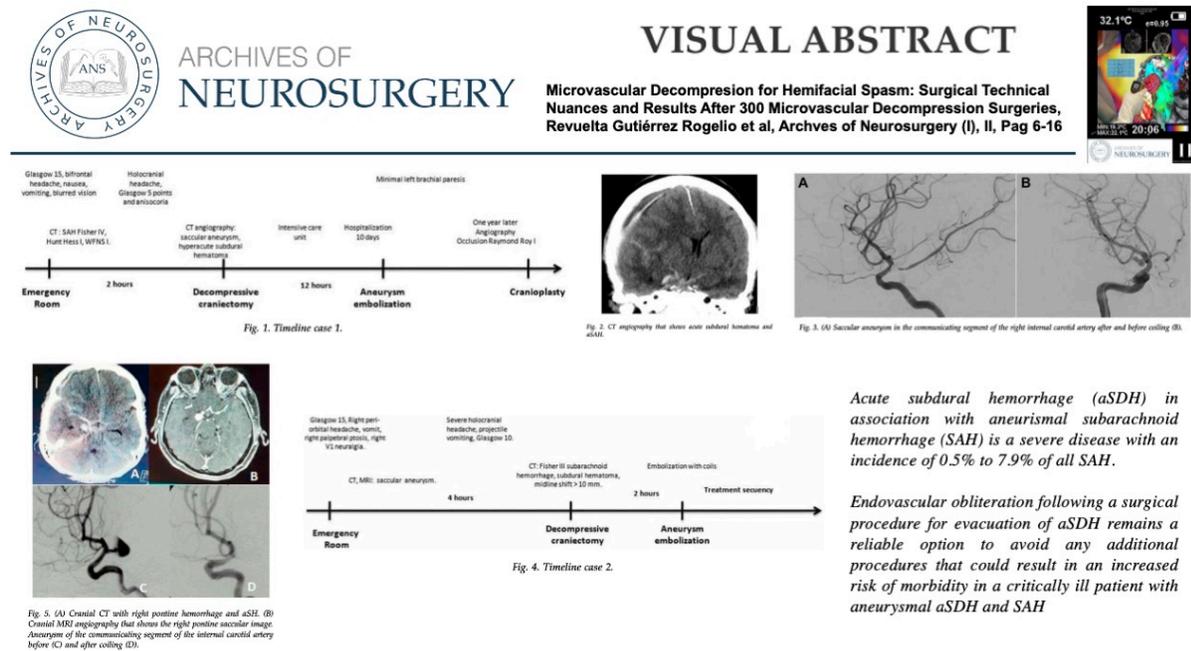
Introduction: Acute subdural hemorrhage (aSDH) in association with aneurysmal subarachnoid hemorrhage (SAH) is a severe disease with an incidence of 0.5% to 7.9% of all SAH. Due to the rarity of aneurysmal aSDH, it remains difficult to define a comprehensive management protocol. In this case review, following the CARE guidelines, we show the hybrid management of this pathology to know the importance of using different types of neurosurgical treatments in case of two severe diseases in critical patients.

We present two patients who developed sudden onset cephalgia and neurological impairment secondary to aneurysmal subarachnoid hemorrhage associated with acute subdural hematoma. These cases required emergency decompressive craniectomy and aneurysm embolization with coils that have a favorable outcome.

In patients with massive and rapidly fatal subdural hemorrhage, emergency craniotomy with hematoma evacuation and immediate brain decompression before definitive aneurysm surgery has shown to be a good treatment option with excellent survival outcomes.

Conclusion: Endovascular obliteration following a surgical procedure for evacuation of aSDH remains a reliable option to avoid any additional procedures that could result in an increased risk of morbidity in a critically ill patient with aneurysmal aSDH and SAH.

Visual Abstract



Acute subdural hemorrhage (aSDH) in association with aneurysmal subarachnoid hemorrhage (SAH) is a severe disease with an incidence of 0.5% to 7.9% of all SAH.

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Keywords

subarachnoid hemorrhage, subdural hematoma, endovascular rescue, case report.

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Aneurysmal Subarachnoid Hemorrhage and Acute Subdural Hematoma, Neurosurgical and Endovascular Rescue. Case Report

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Abstract

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1. Introduction

Acute subdural hemorrhage (aSDH) associated with aneurysmal subarachnoid hemorrhage (SAH) is a severe disease involving 0.5%–7.9% of all SAH case [1]. Due to the rarity of aneurysmal aSDH, it remains challenging to define a comprehensive management protocol. Patients with a Glasgow Coma Scale score below eleven points at admission or a rapidly deteriorating level of consciousness, urgent surgical decompression, and immediate aneurysm obliteration usually result in a favorable outcome. Nevertheless, endovascular obliteration after the evacuation of an aSDH remains a reliable option for those cases where

aneurysm clipping was not feasible in the initial procedure [1]. With the present cases, we aim to demonstrate the hybrid management for this pathology.

2. Case report 1 (Fig. 1)

A 50 years old female with a history of recent headaches presented with sudden onset of severe bifrontal headache associated with postural change, nausea, and vomiting; at the arrival to the emergency service, the main symptoms were general weakness sensation and diaphoresis, and blurred vision.

Physical examination revealed a Glasgow Coma Scale score of 15 points; the neurological exam was normal except for decreased venous pulses in the

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ocular fundus, generalized hyperreflexia, and a slightly stiff neck. The computer tomography scan (CT-scan) showed subarachnoid hemorrhage classified as Fisher IV, Hunt Hess I, and World federation Neurosurgeons Society (WFNS) I. Sixty minutes later, a new episode of holocranial headache followed by a decrease in Glasgow Coma Scale score to 5 points and anisocoria with the need for orotracheal intubation. CT angiography found a saccular aneurysm in the communicating segment of the right internal carotid artery and a 15 mm thickness hyperacute right frontotemporal subdural hematoma, right uncus herniation, and 12 mm midline shift (Fig. 2). We performed an emergency decompressive craniectomy to evacuate the hematoma 2 h since the beginning of symptoms; clipping of the aneurysm was not possible due to hemodynamic instability and severe cerebral edema. After 12 hours in the intensive care unit, we performed a cerebral angiography and aneurysm embolization with coils (Fig. 3). Finally, the patient was stable and showed improvement and left the hospital after ten days with minimal left brachial paresis. One year later, the follow-up angiography showed total aneurysm occlusion (Raymond Roy Class I). Lastly, we performed a cranioplasty twelve months after the initial decompressive craniectomy.

Abbreviations	
SDH	Subdural hemorrhage
aSDH	acute subdural hemorrhage
CT	computer tomography
WFNS	World federation neurosurgeons society
ACA	Anterior cerebral artery
ACoA	Anterior communicating artery
ICA	internal carotid artery
MCA	Middle cerebral artery
MRI	magnetic resonance image.

with a Glasgow Coma Scale score of ten and mydriasis. In addition, a new CT scan showed Fisher III subarachnoid hemorrhage and right frontotemporal subdural hematoma with midline shift >10 mm. Therefore, we performed a decompressive craniectomy in the following 4 h to evacuate the hematoma. However, it was impossible to access the aneurysm because of the severe brain edema, and we established treatment with endovascular therapy for aneurysm embolization in a second stage (Fig. 5).

3. Case report 2 (Fig. 4)

A 47-year-old female with a history of hypertension and headaches for over three years had a sudden onset of severe headache in the right periorbita associated with vomiting. Upon arrival in the emergency room, she demonstrated a score of 15 points on the Glasgow Coma Scale, right palpebral ptosis, and right V1 neuralgia. CT scan showed a right pontine saccular image compared to magnetic resonance image (MRI) angiography, where it appeared to be an aneurysm of the communicating segment of the internal carotid artery. Clinical features progressed to severe holocranial headache, projectile vomiting, and neurological deterioration

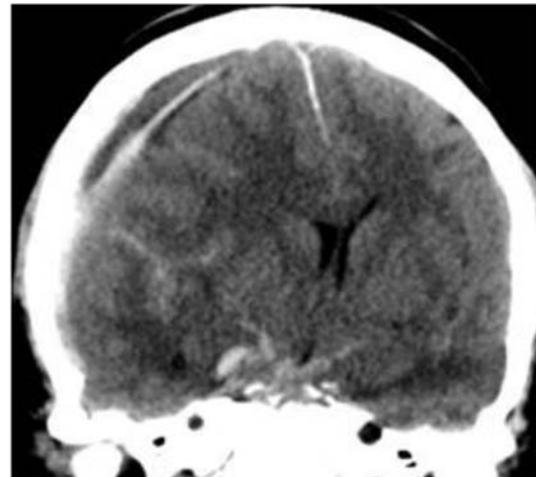


Fig. 2. CT angiography that shows acute subdural hematoma and aSAH.

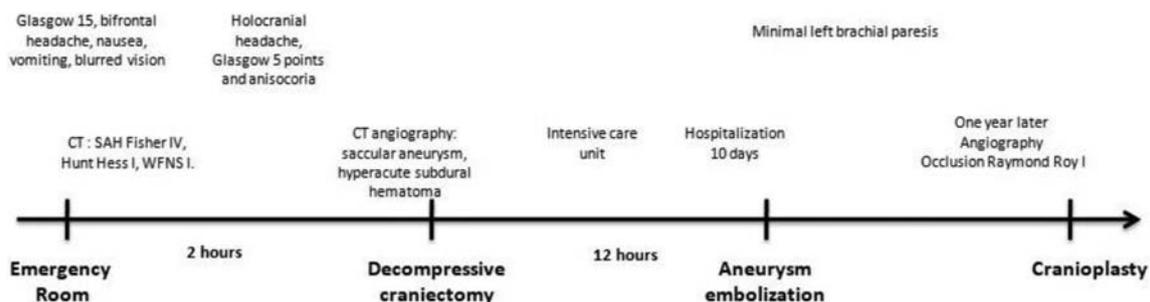


Fig. 1. Timeline case 1.

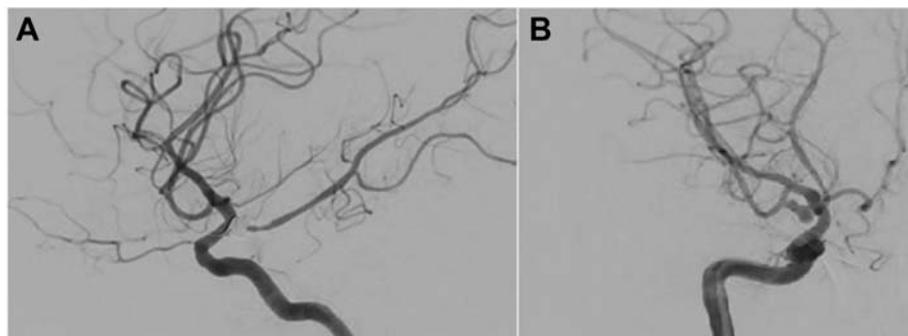


Fig. 3. (A) Saccular aneurysm in the communicating segment of the right internal carotid artery after and before coiling (B).

4. Discussion

Acute SDH resulting from the rupture of an aneurysm was first reported in 1855 described as a severe disease that occurs in 0.5%–7.9% of all subarachnoid hemorrhages [1]. A third of the patients who suffer a subarachnoid hemorrhage due to a saccular aneurysm rupture presents an intracranial hematoma [1]. However, the combination of a ruptured aneurysm and the spontaneous acute subdural hematoma is undoubtedly rare [1].

Since the first report in 1934, many cases of spontaneous aSDH have been reported [1]. The leading causes included aneurysmal bleeding, perisylvian cortical arteries rupture, tumors, and neoplastic diseases [1].

There exist four mechanisms by which blood can reach the subdural space from a ruptured aneurysm [2]. 1) Successive small hemorrhages allow the development of adhesions toward the subdural space where the final rupture occurs [2]. 2) Abrupt rupture of the arachnoid membrane by the rapid increase in pressure from the ruptured aneurysm [2]. 3) A massive hemorrhage ruptures the cortex and lacerates the arachnoid membrane [2]. 4) An aneurysm in the part of the carotid within the subdural space ruptures and directly causes aSDH [2].

Clarke and Walton classified patients into three groups based on the size of the subdural clot and the clinical course: Group I is defined by massive and rapidly fatal intracranial hemorrhage, Group II comprises an insignificant quantity of subdural blood and is not fatal, and Group III involving significant subdural hematoma, which is not rapidly fatal [2].

In those cases where the origin of the aSDH is indeterminate but occurs in association with subarachnoid hemorrhage, intracranial hypertension, intracortical clot, xanthochromic, or bloody ventricular fluid; a cerebral angiography is mandatory [3].

An aneurysm at any site can cause aSDH upon rupture [3]; nevertheless, those occurring from the rupture of the distal Anterior cerebral artery (ACA)

or Anterior communicating artery (AcomA) aneurysms are rare, while those in the internal carotid artery (ICA) and middle cerebral artery (MCA) aneurysms are common [3]. Reynolds and Shaw found that the anterior communicating artery aneurysms were the most common origin for subdural hematomas [3]. It deserves to mention that vertebrobasilar aneurysms are separated by a thick barrier called the Lilliequist membrane, which might hinder the development of acute SDH [3].

Risk factors to be considered for increased risk of co-occurrence of aSDH with aneurysmal subarachnoid hemorrhage are older age, aneurysm at the posterior communicating artery, sentinel headache, and intracranial hemorrhage.

It is suggested that the presence of an aneurysmal aSDH depends on the aneurysm anatomy and perianeurysmal environment [4]. For example, interaction with adjacent structures may occur; when an aneurysm protrudes into the basal cisterns and interacts mainly with the basal arachnoid membrane, aneurysm rupture might result in aneurysm rupture of the arachnoid membrane with a subsequent aSDH [4].

Due to the rarity of aneurysmal aSDH, it remains challenging to define a comprehensive management protocol [5]. However, patients with a poor neurological exam at admission and rapidly deteriorating levels of consciousness, urgent surgical decompression, and immediate aneurysm obliteration result in a favorable outcome [5].

Patients presenting with both SAH and aSDH have poor admission grades and prognoses [6]. The literature reports a 5% incidence of massive subdural hematoma [6]. These patients with signs of cerebral herniation underwent CT angiography and surgical clipping with the simultaneous evacuation of the space-occupying hematoma [6]. A decompressive craniectomy was added for those with brain swelling, followed by cranioplasty more than two months after in survivors [6].

The angiography must be performed before the operation if possible. Angiography must be

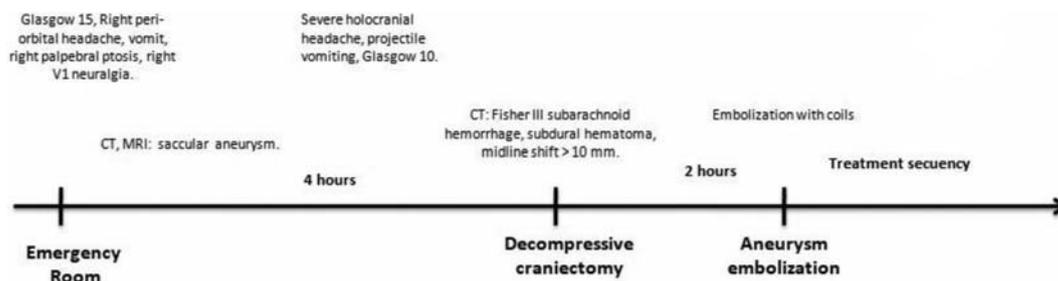


Fig. 4. Timeline case 2.

performed before the operation whenever possible, considering endovascular embolization in the same stage. However, diagnostic-therapeutic angiography can be delayed to the most immediately possible postoperative stage in those cases requiring urgent surgery for hematoma evacuation because of life-threatening neurological compromise [6].

Special consideration for coil embolization should be given when surgery is impossible or risky. For example, a poor Hunt-Hess grade or evidence of significant brain swelling without a mass lesion may increase the risk of surgical retraction but influences lesser the difficulty of coil embolization treatment [7]. According to the American Heart Association, the adverse outcomes, defined as discharge to a nursing home or rehabilitation center, is less frequent in those treated with endovascular therapy (10% versus 25% with surgery), as is the risk of in-hospital death (0.5% versus 3.5%) [7].

Despite a deplorable clinical condition on admission, recovery with the only minor deficit is possible, as Westermaier et al. [8] showed in five of eight patients treated with endovascular coiling, obtaining a Glasgow Outcome Score between 3 and 5 points.

Mydriasis and Aneurysmal aSDH are described among the factors predicting poor prognosis, compared to those who suffered aneurysmal aSDH without SAH [6].

The CT appearance of a subdural hematoma secondary to a ruptured intracranial aneurysm may give no clue to the true origin, but CT angiography has an acceptable sensitivity. The reasons are obvious: speed of diagnosis, the safety of the procedure, and high diagnostic accuracy for acute intracranial hematomas and aneurysm identified [6].

5. Conclusion

Endovascular obliteration after a surgical procedure for evacuation of aSDH remains an excellent option to avoid an additional morbidity procedure in a critically patient who presents aneurysmal aSDH and SAH.

Authorship

- José Omar Santellán Hernández-the conception and design of the study, or acquisition of data, or analysis and interpretation of data.
- José Ramón Aguilar Calderón-drafting the article or revising it critically for important intellectual content.
- Abraham Ibarra de la Torre-the conception and design of the study, or acquisition of data, or analysis and interpretation of data.
- Ulises García Gonzalez-final approval of the version to be submitted.

Publication Comment:

In this article, the authors presented two cases of aneurysmal rupture associated with an acute subdural hematoma. Each pathology represents an

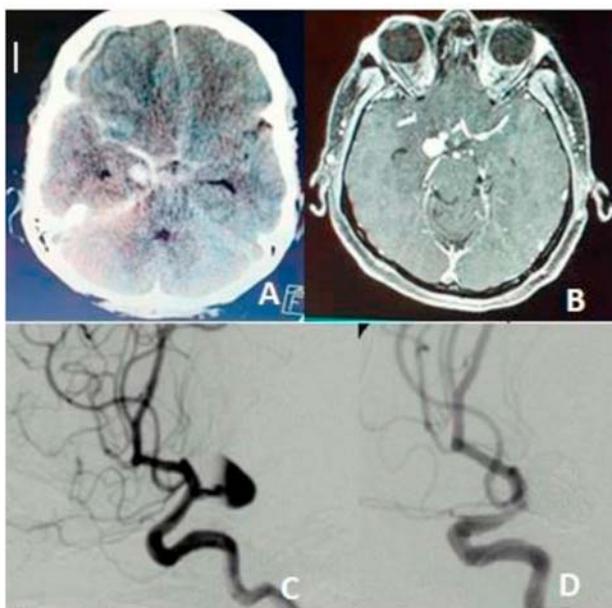


Fig. 5. (A) Cranial CT with right pontine hemorrhage and aSDH. (B) Cranial MRI angiography that shows the right pontine saccular image. Aneurysm of the communicating segment of the internal carotid artery before (C) and after coiling (D).

urgency, given its associated high morbidity and mortality, and even more combined.

There are several treatment alternatives to treat both pathologies:

Craniotomy or craniectomy with subdural hematoma evacuation and aneurysm exclusion by clipping in the same surgical event.

Craniotomy or craniectomy with the hematoma evacuation excluding the aneurysm by endovascular technique in two events.

The authors present two adequately resolved cases that were treated by craniectomy and endovascular therapy with excellent results. They justified its management due to the significant cerebral edema that makes the microsurgical approach difficult in these cases. When the cerebral edema is not so severe, and the hemodynamic conditions of the patient allow it, it is possible, in addition to draining the hematoma, to resolve the aneurysm by microsurgery, both in the same surgical time. However, such management is sometimes not possible if cerebral edema prevents access to the aneurysm or if the patient's neurological and hemodynamic conditions do not allow it. Both alternatives are suitable.

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Informed consent

Patients have informed consent of all diagnostic and therapeutic procedures according to the policies of Hospital Central Sur de Alta Especialidad.

Conflicts of interest

The authors declare no conflict of interest.

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