Multiple intracranial aneurysms in subarachnoid haemorrhage. Which one has bled? A diagnostic dilemma.

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ABSTRACT

This paper is intended as an illustrative teaching case. It gives a prototype case of a patient with subarachnoid haemorrhage and multiple intracranial aneurysms, where the CT data is non-conclusive as to the source of haemorrhage. The paper then discusses the diagnostic challenges and management pearls, pertaining to such scenarios. The paper concludes with a few “take-home points” that summarise the criteria to be applied in such cases.

CASE DESCRIPTION

A 39-year-old male patient with autosomal dominant polycystic kidney disease (ADPKD) presented to the emergency department with a sudden-onset, severe headache and vomiting followed by an altered level of consciousness (GCS:10-E2M5V3). A computed tomography (CT) scan of the head revealed diffuse subarachnoid hemorrhage (SAH) (the typical star-shape sign), but did not give a clue for the source of bleeding (figure 1.A). Cerebral CT angiography (CTA) revealed two intracranial saccular aneurysms in the anterior communicating artery (Acom) and the right middle cerebral artery (MCA); of 7 mm each with no vasospasm observed (figure 1.B,C). However, it could not be determined from the CTA data which aneurysm has ruptured. Cerebral catheter angiography is not available in our country. Thus, the team assumed that the ruptured aneurysm was located at the Acom, and planned the surgery accordingly. The patient underwent surgery on the day of admission. Intraoperatively, it was evident that the anterior communicating artery (Acom) was indeed the source of bleeding. The aneurysm was clipped and secured, without complications. Early postoperative cranial CT scan showed 2 clips in position with no other significant findings (figure1.D). The patient had an uneventful recovery and he remained well.
A 39-year-old male patient with autosomal dominant polycystic kidney disease (ADPKD) presented to the emergency department with a sudden-onset, severe headache and vomiting followed by an altered level of consciousness (GCS:10-E2M5V3). A: An initial cranial CT scan showing SAH in the basal cisterns (the typical star-shape sign) with no clue to the source of bleeding. B and C: A cerebral CT angiography- 3D reconstruction- revealing multiple intracranial saccular aneurysms in the Acom (B inferior view with an arrow pointing to the Acom aneurysm) and the right MCA (B anterior view with an arrow on the superiorly directed, right MCA aneurysm); both aneurysms are regular, rounded-shaped, have no murphy's teat and, have a size of 7 mm each with no vasospasm noticed. D: An early, post-operative cranial CT scan showing 2 clips in position with no other significant findings.

**DISCUSSION**

The incidence of Multiple intracranial aneurysms (MIAs) amongst all patients with aneurysms is around 15-33.5% [7] [4] [10]. Hypertension has been identified as the risk factor most commonly associated with MIAs [2]. MIAs are more common in women, with a female to male ratio of 5:1. The ratio is even higher (11:1) in patients with three or more aneurysms [10]. Overall, the anterior communicating artery (Acom) is the most common location for intracranial aneurysms (35%) [8]. Common sites for multiple intracranial aneurysms include the posterior communicating artery (PCA) the middle cerebral artery (MCA), the anterior communicating artery, and the ophthalmic artery [10].

In patients with SAH and MIAs, verifying the site of bleeding is not always a straightforward task. Furthermore, misidentification of the true ruptured aneurysm is a major cause of post-operative re-bleeding and mortality. In one series that examined a cohort of 76 patients with SAH and MIAs, the rate of false localization of the ruptured aneurysm was 9%, resulting in rebreeding and mortality at the rates of 5.3% (n=4/76) and 2.6 (n=2/76), respectively [6]. The rate of rupture site misidentification is even higher in patients with a non-definitive bleeding pattern on initial CT scan, quoted at 16.2% [11].

The only absolute evidence for aneurysm rupture is angiographic dye extravasation or the “smoking gun” sign [10]. Other highly accurate angiographic findings include focal spasm, aneurysm shape irregularities (Murphy’s Teat), larger size, focal mass effect and aneurysm shape shifts on repeat imaging [3, 10]. While these signs have a high predictive value of the rupture site, they are fairly rare [10].

Clinical signs are less reliable when it comes to identifying the site of hemorrhage. Reports on their usefulness vary, with a sensitivity range of 7-33%[10] [1]. Sometimes, the CT scan shows a clear hemorrhage pattern that suggests the source of the bleeding [5]. Nevertheless, where there is a diffuse, symmetrical pattern of hemorrhage or localized bleeding along with numerous aneurysms in the vicinity, the CT is unable to delineate the site of rupture [5]. One reliable sign is that might give a clue to the site of rupture is the location of the epicenter of hemorrhage (area with most contraction) on CT or MRI [3].

In our case, none of the above-mentioned radiological signs were present, and hence prediction of the site of the ruptured aneurysm was responsibility of the treating neurovascular team. In this case, the Acom was presumed to be the ruptured site given the frequency of these aneurysms and the fact that they are more prone to rupture than MCA aneurysms. In this case, the surgery was directed only at the ruptured Acom aneurysm. Some surgeons opt for the treatment of all significant aneurysms at the time of surgery to
reduce the risk of late re-bleeding, while others recommend treating the ruptured aneurysm only [12]. In either case, prompt identification and treatment of the ruptured aneurysm remains a priority, as it has significant implications on patient outcomes. In fact, it has been shown that the most common cause of late re-bleeding is the originally ruptured aneurysm which was not identified at the time of initial surgery [9].

It is not yet determined whether MIAs are associated with worse surgical outcomes. However, several risk factors have been investigated for their prognostic significance in patients with SAH and MIAs. One population-based study has shown that for patients > 70 years of age, the prognosis is less favorable for those with SAH and multiple aneurysms as compared to those with SAH and a single aneurysm [7]

In summary, the present case has illustrated the potential challenging nature of SAH in patients with MIAs and emphasized on the importance of adopting a systematic, step-wise progression through preoperative planning, and the critical impact this has on surgical outcomes.

Take-home points:
- Patients with MIAs and SAH present a challenge to the neurovascular surgeon.
- When the bleeding pattern on the initial CT scan is non-definite, meticulous examination of the CTA and/or DSA data becomes very important and should take into consideration the following criteria [3]
  - CT/MRI: Where is the area exhibiting the greatest concentration (Aka. The epicenter of SAH)?
  - CTA/DSA: Where is the area with local vasospasm?
  - Morphology: Is there a “Murphy’s teat” – a daughter cyst- or an area of irregularity- in any of the aneurysms?
  - Size: Which aneurysm is larger?
- If none of the above criteria applies, as in this case, then the judgment of the treating team is critical. The caveat is that, when such a time-sensitive decision is required, it is safer to assume that the Acom is culprit site, as it is the predilect site of intracranial aneurysms, and is , hence, overall the most common site of rupture.

**ABBREVIATIONS**
ADPKD: Autosomal dominant polycystic kidney disease
GCS: Glasgow coma scale
EMV: Eye movement and verbal response
CT: Computed tomography
SAH: Subarachnoid haemorrhage
3D: Three dimensional
Acom: Anterior communicating artery
MCA: Middle cerebral artery
CTA: Computed tomographic angiography
MIAs: Multiple intracranial aneurysms
MCA: Middle cerebral artery
PCA: Posterior communicating artery
MRI: Magnetic resonance imaging
DSA: Digital substraction angiography
Aka: Also known as

**REFERENCES**
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