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Sellar hematoma as a new potential radiological clue for superior hypophyseal artery aneurysm rupture. A case report

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ABSTRACT

Background. In cases of spontaneous subarachnoid haemorrhage (SAH) with multiple intracranial aneurysms (MIAs) detected on angiography, some radiological clues assist in determining the site of a ruptured aneurysm which is bleeding is quite beneficial for the selection of the best treatment strategy.

Case description. We report a case of a 60 years old patient who presented with spontaneous SAH, sellar hematoma, and three different aneurysms detected in angiography. Although the right Posterior communicating artery (PcomA) aneurysms showed Murphy's teat on angiography intraoperatively, we discovered that the right superior hypophyseal aneurysm (SHA) was the source of the index bleeding. Both aneurysms were clipped successfully.

Conclusion. From the preoperative radiological and intraoperative surgical findings, we propose that sellar hematoma on a non-contrast CT scan is a new potential sign to be correlated with superior hypophyseal artery aneurysm rupture.

INTRODUCTION

Cranial computed tomography (CT) scan without contrast is the most sensitive imaging choice for the diagnosis of aneurysmal subarachnoid hemorrhage (SAH), with a sensitivity of 93% within the first 24 hours of onset [14]. However, the CT scan value for determining the primary source of the hemorrhage (i.e., the site of a ruptured aneurysm) has not been demonstrated with some exceptions. Examples of such exceptions include unilateral pure Sylvian SAH and gyrus rectus hematoma as potential clues for Middle cerebral artery (MCA) and Anterior communicating artery (AcomA) aneurysms, respectively [4], propped by an accuracy ratio up to 78% of all cases with challenging multiple intracranial aneurysms (MIAs) [8]. In cases of spontaneous SAH with MIAs detected on angiography, these clues are paramount to direct the most appropriate management strategy. In literature, we are the first paper demonstrating the presence of sellar hematoma associated with the typical basal SAH on an initial CT scan as a new potential clue for superior hypophyseal artery (SHA) aneurysm rupture in a case of MIAs.

Case scenario

A previously healthy, 60-year-old female was admitted with the complaint of a sudden severe headache, photophobia, and drowsy associated with meningismus, with no weakness. A brain CT scan disclosed acute SAH with no specific clue except for an apparent sellar hematoma (Figure 1 and 2). The cerebral catheter angiography showed three intracranial aneurysms distributed to (right PcomA, SHA, and left PcomA aneurysms) (Figure 3).

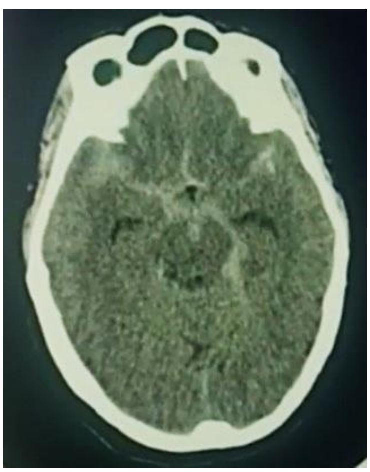

Figure 1. Non-contrast cranial CT scan axial section showed basal subarachnoid hemorrhage with no predominant clue for the location of the ruptured aneurysm.

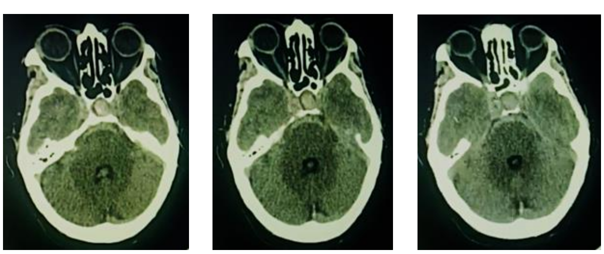

Figure 2. Non-contrast cranial CT scan axial sections (the same CT in figure 1) showing a hematoma in the sella turcica.

According to the angiography, the right PcomA aneurysm was the most probable site of rupture due to having murphy's teat (daughter cyst). Notably, because our country lacked an endovascular facility, the decision was made to manage the case surgically, using the right pterional approach. The patient was in a supine position with the head tilted to the left side, inverted curvilinear skin incision 1 cm posterior to the hairline, and the craniotomy, osteoplastic bone flap, and durotomy were then performed. Following the dissection of the Sylvian fissure, the supraclinoid internal carotid artery (ICA) and the neck of the right PcomA aneurysm were dissected and clipped. Then, intra-operatively, we encountered a huge intraoperative rupture from the right SHA aneurysm after dissection of the clipped right PcomA aneurysm. Eventually, both aneurysms were clipped with no intraoperative or postoperative complications.

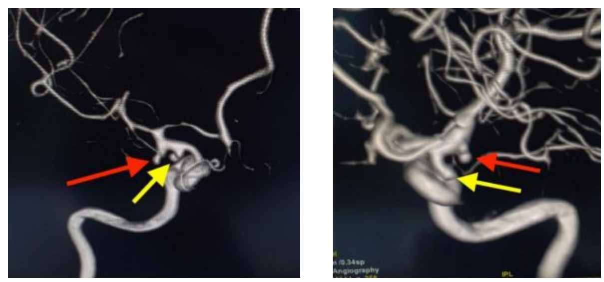

Figure 3. 3D Cerebral catheter angiography. (A and B) Right ICA anterior and left oblique views respectively, showing PcomA (red arrow) and SHA (yellow arrow) aneurysms. The PcomA aneurysm has a daughter cyst.

The postoperative course was uneventful. We decided to follow the left PcomA aneurysm due to its size and configuration (small, shallow, and wide neck). At a 6-moth follow-up, the patient is conscious without any neurological deficit, and the Left PcomA aneurysm is stable.

Discussion

Non-contrast brain CT scan is the most sensitive imaging study in cases with SAH. The CT scan has 100% sensitivity and specificity when a CT is performed within 6 hours of headache onset; sensitivity is about 93% during 24 hours of onset, 80% at three days, and 50% in the first week [1,4]. Ruptured aneurysms constitute the most common etiology for 80% of nontraumatic (spontaneous) SAH, with a 0.4-6% prevalence of intracranial aneurysms in the general population [9,13]. Multiple intracranial aneurysms (MIAs) are evident for 15-33.5% of all patients with aneurysms [5]. Detecting the primary source of aneurysmal SAH is relatively not straightforward and not without difficulty, and this increases the likelihood of re-bleeding in patients with MIAs. An observational study reported mortality rates for an aneurysmal SAH of 10% to 32% within the first day, 27% to 43% during the first week, and 49% to 56% in the first month after onset [9]. Most deaths occur due to re-bleeding from the same aneurysm; if untreated, re-bleeding occurs in 15% of patients on the first day and in 40% of people by one month after SAH [9].

In cases with MIAs, the definite indication of a ruptured aneurysm is the angiographic dye extravasation (smoking gun) sign [10]. However, this is an uncommon sign that denotes a grim prognosis [5]. Other high-resolution angiographic findings include the area of focal spasm, irregularity in aneurysm shape (Murphy's teat), focal mass effect, and shifts in aneurysm shape upon repeat imaging. If none of the above help, the suspected ruptured one is the largest aneurysm [4,10]. While these signs have a large predictive value for bleeding sources, they are relatively rare [10].

CT scanning can provide suggestions about where the rupture is through potential pivotal clues based on the pattern of a SAH that has been proven effective in 78% of all cases with MIAs, especially for MCA and AcomA aneurysms [4,8,12]. The most evident clue is the thickness of the clot located within the subarachnoid space. Occasionally, an apparent focus clot within the SAH area represents the aneurysm responsible for hemorrhage [12]. Another clue is the hemorrhage distribution pattern, including SAH concentrated in the anterior interhemispheric fissure with or without blood in lateral ventricles or within the gyrus rectus suggests AcomA aneurysm rupture [8,12]. While blood mainly concentrated in unilateral Sylvian fissures is compatible with PcomA or MCA aneurysm on that side [12]. Likewise, SAH predominantly within the prepontine or peduncular cistern suggests a basilar apex or superior cerebellar artery (SCA) aneurysms [8]. Also, patterns of blood primarily in the fourth and third ventricle indicate lower posterior fossa source, such as posterior inferior cerebellar artery (PICA) aneurysm or Vertebral artery (VA) dissection [8,12]. Then, blood primarily within the third ventricle suggests a basilar apex aneurysm [8]. These potential clues are of critical importance for the surgeon to prevent re-bleeding by determining which aneurysm needs urgent surgery and to assist in deciding optimal surgical strategies and procedures. It has been lucid that the most prevalent cause of late re-bleeding is the ruptured aneurysm misidentified at the time of initial surgery [11].

In our case, The aneurysm suspected of being ruptured was the right PcomA aneurysm, as it contained a murphy's teat. A cerebral CT scan showed acute SAH with none of those mentioned above CT clues. CT revealed a concentrated blood pattern within the sella turcica (sellar hematoma) (Figure 1 and 2). Patients with SHA aneurysms are vulnerable to developing MIAs, as in our case [7]. These aneurysms are uncommon entities and more likely to be ruptured at a relatively smaller size than other intracranial aneurysms [7]. SHA aneurysm originates from the supraclinoid segment, where SHAs that supply the hypophysis and optic chiasm consistently arise from the ICA near the lateral aspect of the sella.

Mostly SHA aneurysms fall into two classifications that are divided into sellar and supraclinoid variants. The sellar variant is directed medially over the diaphragma sellae, while the paraclinoid variant projects inferomedially. These projections are directed by the size of the supraclinoid ICA segment and the height of the lateral sellar wall [1,8]. The second classification falls within the sellar variant itself, which has two different varieties: suprasellar or parasellar [6]. The rupture of a parasellar aneurysm typically presents with SAH [1]. In our patient, the SHA aneurysm was a parasellar variant lying over the sellar area, which was the source of sellar hematoma in the initial CT scan. The sudden massive intraoperative rupture of the right SHA aneurysm affirmed that the initial ruptured aneurysm was the same aneurysm, which we did not take seriously despite the clear sign of sellar hematoma.

In literature, here, we are the first to report the concentrated blood pattern within the sella turcica (sellar hematoma) in non-contrast brain CT scan, as a new potential radiological clue to be correlated in cases with SHA aneurysm rupture.

To our knowledge, Cranial CT scanning can show sellar hematoma, and SAH from an SHA aneurysm rupture appears as high-density blood concentrated within the sellar area and suprasellar subarachnoid spaces. With or without typical star shape SAH through major cerebral fissures [8,10]. In contrast, pituitary apoplexy (PA) is a rare and potentially severe clinical condition caused by neurologic deterioration as a result of a sudden expansion of a mass within the sella turcica, usually due to an acute ischemic infarction or hemorrhage of the pituitary gland [2,10]. On CT scan, it appears an intrasellar mass with hemorrhagic components, seen in 80% of pituitary apoplexy cases as a patchy or hyperdensity in CT scan within a pituitary lesion, leading to sellar enlargement in up to 94 % of cases [2,3]. So these radiological signs of PA, including heterogeneous pituitary mass, are utterly different from clear sellar hematoma with typical basal SAH, as in our case. So the PA can be excluded from the differential diagnosis list for patients with SHA aneurysms and MIAs.

In summary, the sellar hematoma on the initial CT imaging is a possible milestone sign that hands over a significant indication for SHA aneurysm rupture to answer the question of which aneurysm rupture is responsible for this SAH in cases with MIAs.

Conclusion

In cases of spontaneous subarachnoid hemorrhage with imaging showing multiple intracranial aneurysms, the presence of a radiological clue for the source of bleeding in an initial CT scan is quite beneficial for the selection of the best treatment strategy. Sellar hematoma on a non-contrast CT scan is a new potential sign to be correlated with superior hypophyseal artery aneurysm rupture.

Abbreviations

CT; computed tomography,
SAH; subarachnoid hemorrhage,
MCA; Middle cerebral artery,
AcomA; Anterior communicating artery,
MIAs; multiple intracranial aneurysms,
PcomA; posterior communicating artery,
ICA; internal carotid artery,
SCA; superior cerebellar artery,
PICA; posterior inferior cerebellar artery,
VA; vertebral artery,
PA; pituitary apoplexy.

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