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Clinical presentation of cerebral aneurysms

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ABSTRACT

Presentation of a cerebral aneurysm can be incidental, discovered at imaging obtained for unrelated causes, can occur in the occasion of imaging obtained for symptoms possibly or likely related to the presence of an unruptured aneurysm, or can occur with signs and symptoms at the time of aneurismal rupture.

Most unruptured intracranial aneurysms are thought to be asymptomatic, or present with vague or non-specific symptoms like headache or dizziness. Isolated oculomotor nerve palsies, however, may typically indicate the presence of a posterior circulation aneurysm.

Ruptured intracranial aneurysms are by far the most common cause of non-traumatic subarachnoid hemorrhage and represent a neurological emergency with potentially devastating consequences. Subarachnoid hemorrhage may be easily suspected in the presence of sudden and severe headache, vomiting, meningism signs, and/or altered mental status. However, failure to recognize milder and more ambiguous clinical pictures may result in a delayed or missed diagnosis.

In this paper we will describe the clinical spectrum of unruptured and ruptured intracranial aneurysms by discussing both typical and uncommon clinical features emerging from the literature review. We will additionally provide the reader with descriptions of the underlying pathophysiologic mechanisms, and main diagnostic pitfalls.

1. Introduction

Presentation of a cerebral aneurysm can be incidental, discovered at imaging obtained for unrelated causes, can occur in the occasion of imaging obtained for symptoms possibly or likely related to the presence of an unruptured aneurysm, or can occur with signs and symptoms at the time of aneurismal rupture.

The prevalence of unruptured intracranial aneurysms (UIAs) is estimated by neuroimaging and post-mortem studies in 3.6–6% of the general population [1,2]. UIAs are more frequent in women (about 66–70%) and in older patients, and are very rare before the age of 20. Most of them (up to 91% [3]) are thought to be asymptomatic or present with non-specific symptoms and are discovered incidentally at imaging studies performed for various reasons [3]. The majority of intracranial aneurysms will never rupture. In a recent Japanese study encompassing 5720 patients who were followed for 20 years, the average annual rate of rupture was as low as 0.5% for aneurysms smaller than 7 mm, and reached 33.4% only among those larger than 25 mm (overall rate 0.95%). Several factors have been found to be associated with an increased risk of rupture: these include both anatomical features (i.e. aneurysm size, specific location, presence of a sac bleb), and demographic characteristics and medical conditions (female sex, age over 60, hypertension, and smoking) [3,9]. On the other hand, the existence of triggering factors for rupture during day-life circumstances such as sexual intercourse, physical exercise, strong coffee drinking and anger [10], and/or physiologic events such as defecation and micturition [11,12] (which may determine a transient increase in the intracranial blood pressure due to Valsalva maneuver), is a matter of debate [13]. However, although situations that increase the intracranial blood pressure pose a theoretical risk for aneurysmal rupture, common day-life circumstances which precede aneurysmal SAHs (i.e. chatting/watching television/staying home in 13.8% of cases) are not strenuous activities [11].

Ruptured intracranial aneurysms (RIAs), on the other hand, are by far the most common cause of SAH (85%) [4] with half of patients being younger than 55 [5], and represent a neurological emergency with potentially devastating consequences. In recent study cohorts, case-fatality rates within the first months, although declined in the last decades, are still reported to be as high as 35–39% [3,6,7], and sudden death is reported to occur in about 10% [8].

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Box 1: RIAs presenting symptoms

Typical

- Thunderclap headache (sudden, severe with maximum intensity peak within seconds/minutes)
- Nausea/vomiting – nuchal rigidity – photophobia
- Transient or persistent or delayed altered mental status/loss of consciousness
- Other signs of increased ICP

Less frequent

- Mild intensity headache
- Seizures
- Focal neurological symptoms (dysarthria, lateralized sensory/motor symptoms)
- EKG abnormalities mimicking myocardial infarction
- Agitation, confusion, obtundation mimicking psychiatric disorders

We will describe the clinical spectrum of unruptured and ruptured intracranial aneurysms by discussing both typical and uncommon clinical features emerging from the literature review. We will additionally provide the reader with descriptions of the underlying pathophysiologic mechanisms, and main diagnostic pitfalls.

2. Clinical presentation of unruptured intracranial aneurysms

According to the investigators from the International Study of Unruptured Intracranial Aneurysms (ISUIA), symptoms "unrelated to rupture" seldom occur in patients with intracranial aneurysms measuring 7 mm or less, and are also uncommon in those between 8 and 20 mm [14]. Nonetheless, several clinical scenario have been linked to variable extent to UIAs (Table 1).

Patients' complaints or conditions leading to imaging and detection of UIAs are available from large prospective studies that evaluated their natural history and treatment outcomes. According to ISUIA investigators, the most common medical condition that leads to the diagnosis of UIAs is headache (36%), followed by ischemic cerebrovascular events (17.6%), and cranial nerves (CN) deficits (15.4%) [4].

2.1. Headache

In two large retrospective cohorts, headache without any specific clinical feature was the most common condition that led to imaging in about 37% [1,3,4]. Moreover, the International Classification of Headache Disorders (ICHD) [15] reported a prevalence of non-specific chronic headache in 18% of patients with UIAs. On the other hand, it is well known that headache without specific features and/or other neurological clues is a very common condition that may be present as a primary syndrome or may be secondary to a number of neurological and systemic diseases, accounting for up to 4% of physicians consultations [16]. Interestingly, however, in a group of patients with UIAs and chronic headache of various degrees of severity, a marked improvement of their symptoms was reported after treatment of the aneurysm in 65.3% of cases [17]: these findings would corroborate the assumption of a direct relationship between UIAs and chronic headache.

Several case-reports [18–22] suggest a specific relationship between UIAs and episodes of sudden, intense headache with peak intensity at onset and resolution within the following 72 h, the so-called "Thunderclap headache" (TH), which is often also described by patients as the "worst headache" in their life. While TH is reported to precede subarachnoid hemorrhage (SAH) with a frequency varying from 10% to 50% [23,24] and is therefore also referred as a "sentinel headache" [23–25], it has also been reported by patients with UIAs with no evidence of SAH at CT and/or lumbar puncture. Several pathophysiologic mechanisms that may explain occurrence of acute headache in absence of macroscopic bleeding have been hypothesized [18–21], and include sudden expansion or dissection of the aneurysmal sac, formation of a daughter sac ("bleb"), acute aneurysmal thrombosis and/or inflammation, and occult micro-hemorrhage. However, numerous possible etiologies of TH, other than UIAs and SAH, are known, encompassing a variety of entities, such as: primary benign TH, cervical artery dissection, cerebral venous sinus thrombosis, stroke, intracranial hemorrhage, reversible cerebral vasoconstriction syndrome, reversible posterior leukoencephalopathy and use of serotonin-enhancing drugs (for a review, see [26]). The specificity of the symptom TH is therefore low, and ultimately makes it unclear whether aneurysms identified at imaging in occasion of a TH are truly symptomatic or incidental [27,28].

2.2. Ischemic cerebrovascular events

In patients with UIAs who subsequently suffered stroke or TIA episodes, a few studies attempted to specifically attribute those cerebrovascular ischemic events to emboli departing from aneurysms of about 3–6%, rather than to other possible causes of thromboembolism (for a review, see [29]). Moreover, those patients who subsequently received treatment for their aneurysms were reported to have a lower recurrence of cerebrovascular attacks [29]. In these cases, however, patients' neurological symptoms were referred to embolization from UIAs only on the basis of the location of the ischemic lesion in a vascular distribution distal to the aneurysm, of the exclusion of other possible extracranial sources of embolisms (i.e. cardioembolic), and by relying on the primary neurologist or neurosurgeon opinion. Despite the uncontrolled nature of these studies, it seems reasonable, however, that in at least some of those patients with UIAs who present with cerebrovascular ischemic episodes, embolization from the aneurysmal sac may have occurred.

2.3. Cranial nerves deficits

Isolated oculomotor nerve palsies are classically associated with posterior circulation UIAs, which represent the second most common cause in non-traumatic adult patients after diabetic microvasculopathy [30,31]. Mass effect from posterior communicating artery (PCoA) aneurysms (especially larger ones) is indeed a well known cause of CN III compression in the subarachnoid space and may represent a neurological emergency. After leaving the interpeduncolar cistern, the III CN courses forward and laterally between the posterior cerebral artery above and the superior cerebellar artery (SCA) below to run briefly alongside the PCoA. At this level, the parasympathetic pupillary fibers are located dorsally and peripherally: therefore, a midriatic pupil is common and may

Table 1
Most common clinical conditions that led to imaging and detection of UIAs in large cohorts.

Headache/headache or dizziness	37%[a]; 36%[c]; 18%[d]/47.4%[b]
Ischemic cerebrovascular disease	17.6%[c]; 22.6%[a]
Cranial nerves deficits	15.4%[c]; 2.2[a]

[a] Jeon et al. [1].
[b] Morita et al. [3].
[c] International Study of Unruptured Intracranial Aneurysms Investigators [4].
[d] The International Classification of Headache Disorders [15].