Cerebral Aneurysms

Early Detection is Key to Survival

Due to its devastating mortality rate, physicians should remain on high alert for early signs and symptoms of cerebral aneurysms.

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In this article:

1. What are the risk factors of aneurysm?
2. What are the symptoms?
3. What investigations should be performed?
4. What is the treatment?

Mrs. Webb's case

Mrs. Webb, 72, presented to three different physicians over a 10-day period with severe headache (sudden onset), nausea, vomiting, photophobia, and neck stiffness. On day 10, she was brought comatose to the emergency department. A computed tomography scan showed diffuse subarachnoid hemorrhage (SAH) and a left frontal intracerebral hematoma. Contrast angiography demonstrated an anterior communicating aneurysm.

Due to her severe and persisting deficit, supportive care was withdrawn.

Mrs. Adams' case

Mrs. Adams, 54, developed an altered level of consciousness and right hemiparesis seven days after rupture of an intracranial aneurysm. Cerebral vasospasm was confirmed by contrast angiography. Triple H therapy (hypertension, hypervolemia, and hemodilution) was instituted, partially reversing the hemiparesis.

Over the past 20 years, there have been significant advances in the diagnosis and treatment of intracranial aneurysm. The consequence of subarachnoid hemorrhage (SAH) secondary to aneurysm rupture, however, remains a significant problem, with only one in three patients returning to their former quality of life.[1]

Autopsy data demonstrate that 2% of the population harbour a cerebral aneurysm, and half of these patients present with SAH.[2] A variable incidence of SAH has been reported, ranging from six to 11/100,000/year.[3] The incidence is higher in women. The peak age for SAH is between 55 and 60 (Figure 1). Familial aneurysms usually present at a younger age.[4]

What is the pathophysiology of intracranial aneurysms?

Intracranial aneurysms are acquired lesions that occur at branching points of major intracranial arteries. They are related to hemodynamic degradation of the internal elastic lamina in the wall of cerebral arteries. Hemodynamic stress is increased by anatomical anomalies of the circle of Willis. Eighty-five per cent of aneurysms occur in the anterior (carotid) circulation and 15% occur in the posterior (vertebro-basilar) circulation. A genetic predisposition is increasingly recognized.[5] Less common causes include trauma, infection, radiation, inflammatory conditions, and neoplasia.

What are the risk factors?

An avoidable risk factor associated with intracranial aneurysm development and rupture is cigarette smoking. Other risk factors are listed in Table 1. These risk factors are likely additive, amplifying the injurious effect of hemodynamic stress.

What are the symptoms?

The most common presentation of an intracranial aneurysm is SAH. Patients with SAH describe sudden onset of severe headache ("thunderclap") often associated with nausea, vomiting, photophobia, and neck stiffness (Table 2). In up to 30% of patients, the SAH diagnosis is not established, leading to catastrophic conse-

Mr. Lafleur's case

Mr. Lafleur, 65, improved clinically following a coma-producing SAH. He underwent a craniotomy and microsurgical clipping of his aneurysm the following day. Followup cerebral angiography at day 7 confirmed aneurysm obliteration, and absence of vasospasm. The patient made an unremarkable recovery.

Table 1

Risk factors for aneurysmal SAH

- Smoking
- Family history
- Female
- Polycystic kidney disease
- Collagen vascular disorders
- Hypertension
- Alcohol

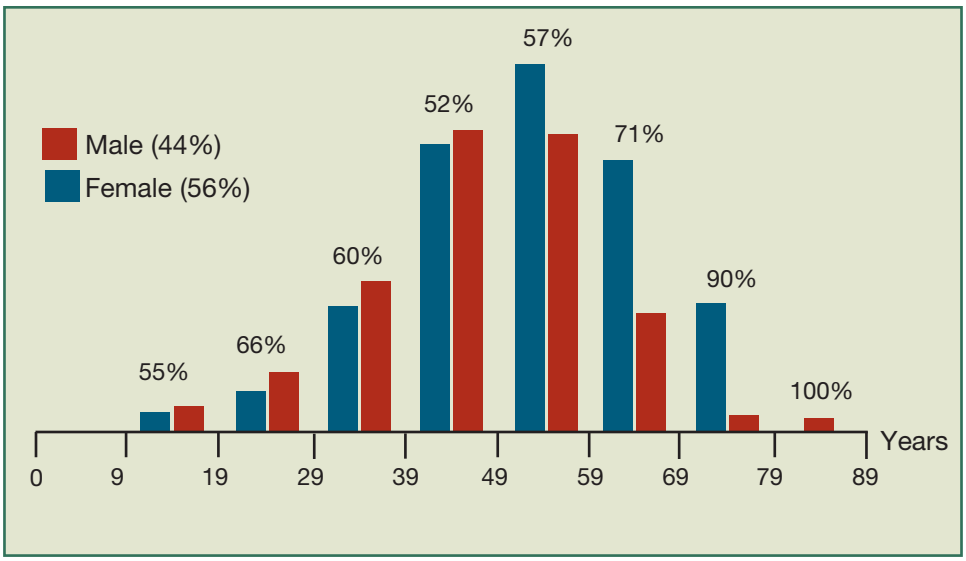

Figure 1. Age distributon for aneurysmal SAH.[9,10]

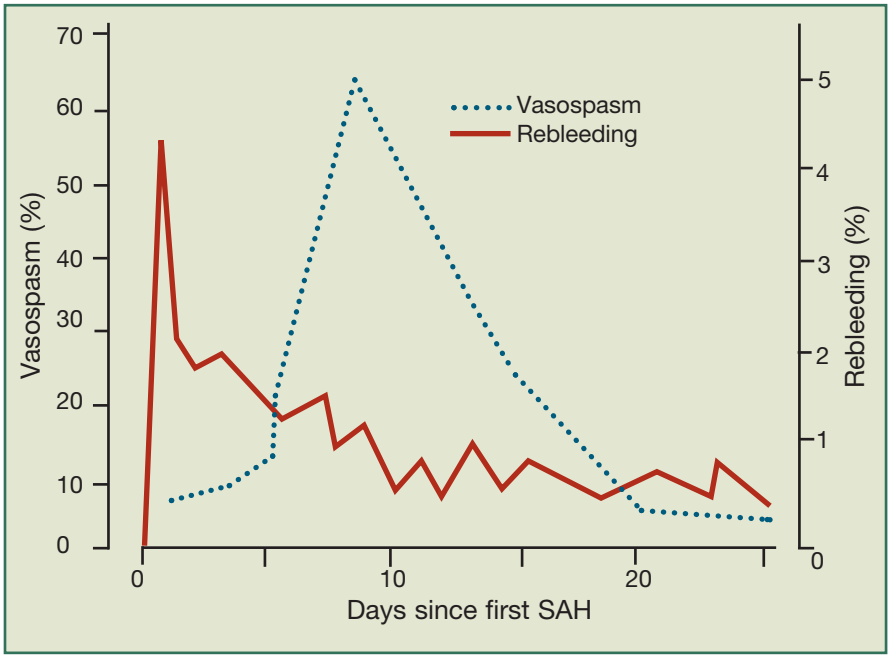

Figure 2. The time course for rebleeding and cerebral vasospasm following aneurysmal SAH.[2]

Table 2
Presenting symptoms of aneurysmal SAH

Symptom	Prevalence
Headache	> 90%
Nausea/vomiting	45%
Photophobia	50%
Neck stiffness	50%
Altered level of consciousness	50%
Focal neurologic deficit	30%

quences. Unfortunately, the literature labels these missed events as "warning leaks" or "sentinel hemorrhages," implying a false benignity.

Extensive SAH is often coma-producing and may be accompanied by intracerebral hematoma or interventricular hemorrhage. Depending on clot location, the patient may display lateralizing neurologic deficits. Expanding (unruptured) aneurysms are unstable and may produce cranial nerve palsies, in particular the non-pupil sparing occulomotor palsy caused by a posterior communicating artery aneurysm. This constitutes a neurosurgical emergency. In rare instances, an aneurysm is the source of an embolic stroke.

What is the course of SAH?

Should patients survive the initial SAH, they are further imperiled by the risk of rebleeding and cerebral vasospasm. The risk of rebleeding is highest immediately following aneurysmal rupture, but decreases over 48 hours to 1% per day for the initial two weeks. At one year, 50% of patients have rebled, with a 43% mortality rate (Figure 2).[2]

Vasoactive breakdown products of subarachnoid blood induce symptomatic vasospasm of the intracranial vessels in 30% of patients following aneurysmal SAH. Onset typically occurs at day 4, peaks between days 7 and 10, and resolves by day 18 following SAH. The consequences of vasospasm include stroke and death.[2]

What investigations should be performed?

Before initiating investigations, the patient's condition must be stabilized. The airway must be assessed and secured, hemodynamic parameters normalized, and symptomatic control of pain, nausea, and vomiting initiated. Secondary to catecholamine storm associated

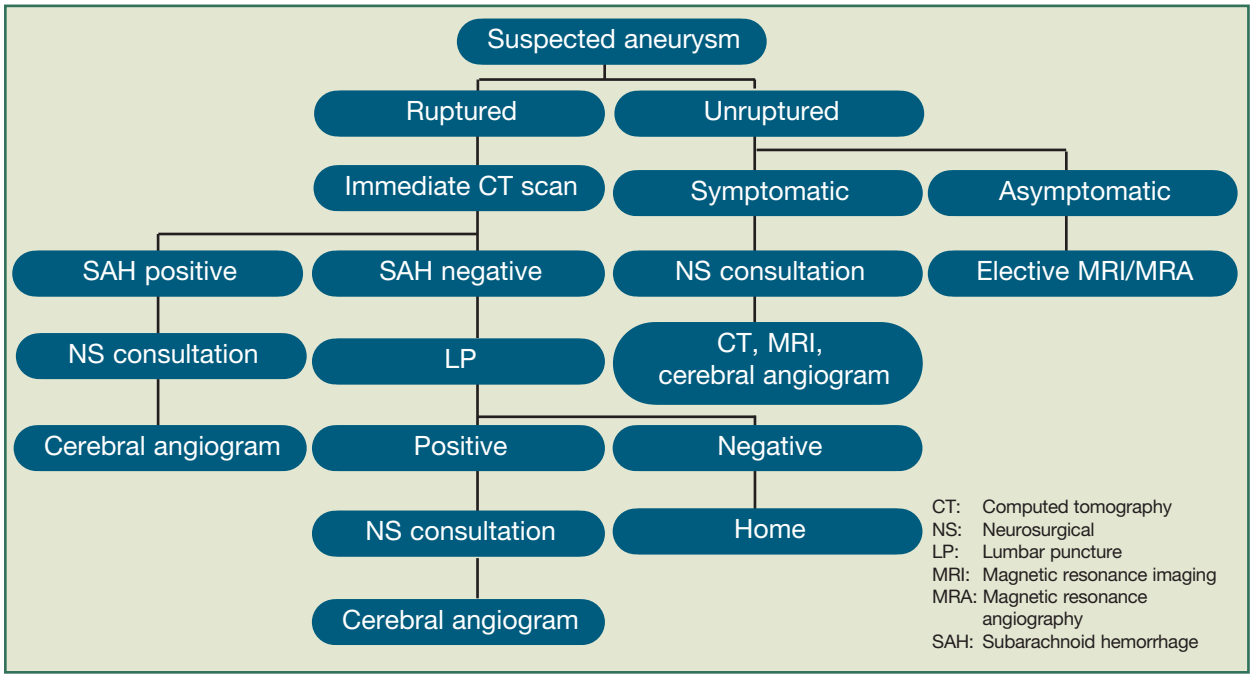

Figure 3. Investigation of suspected cerebral aneurysm and aneurysmal SAH.

with SAH, patients may develop cardiac arrhythmias, cardiac ischemia, and, rarely, pulmonary edema. Figure 3 outlines an algorithm for investigation of suspected intracranial aneurysm.

If the clinical presentation suggests SAH, immediate computed tomography (CT) brain imaging is necessary. If the CT is negative, a lumbar puncture must be performed and cerebrospinal fluid (CSF) expeditiously examined for xanthochromia. Xanthochromia occurs secondary to lysis of red blood cells and may take up to 12 hours

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Take-home message

Symptoms
- The most common symptom of aneurysm is SAH.
- The symptoms of SAH include severe headache, nausea, vomiting, photophobia, and neck stiffness.

Diagnosis
- If SAH is suspected, an immediate CT scan is necessary.
- If the CT is negative, lumbar puncture must be performed and CSF examined.

Treatment
- Treatment options include microsurgery or endovascular intervention.
- Direct surgical repair remains the gold standard of treatment.

to appear. Xanthochromic CSF confirms the diagnosis of SAH. Bloody CSF that does not clear in serial tubes is also indicative of SAH. Both situations require neurosurgical consultation and cerebral angiography. If symptoms of SAH are remote and CSF unremarkable, clinical suspicion of SAH warrants neurosurgical consultation. Given the potential consequences, it is imperative that a diagnosis of SAH not be missed.

Symptomatic, unruptured aneurysms, particularly those presenting with occulomotor palsy (cranial nerve III), require immediate neurosurgical consultation. Asymptomatic, unruptured aneurysms are usually serendipitous findings discovered during investigation of other problems. They are often detected by screening for familial aneurysms using magnetic resonance angiography (MRA). CT angiography is not recommended. Based on current data, we recommend screening individuals with two first-degree relatives with a known intracranial aneurysm.[6]

What is the treatment?

Treatment options include microsurgery or endovascular intervention. Influencing factors include aneurysm size, location, patient age, neurologic and medical condition, and institutional expertise. Direct surgical repair remains the gold standard of treatment. An increasing proportion of patients are now managed using endovascular techniques and this number will continue to increase as technology advances.[7] However, the durability of this treatment remains uncertain. Supportive care may be an appropriate option in patients with severe neurologic deficits. Treatment of asymptomatic aneurysms remains controversial and requires neurosurgical input.[8]

Keep suspicion index high!

Despite improvements in the understanding, diagnosis, and treatment of aneurysms and aneurysmal SAH, the morbidity and mortality from these lesions remain a problem. Ten to 15% of patients die before reaching the hospital.[1] Rebleeding and vasospasm are the major causes of morbidity and mortality among patients surviving the initial hemorrhage. Of the victims of aneurysmal SAH, one-third die or have severe disability, one-third have mild to moderate disability, and one-third return to former activities.[2]

The impact of aneurysmal SAH remains unchanged, despite advances in diagnostic and treatment strategies. It is imperative that physicians maintain a high index of suspicion for this condition and establish the diagnosis as soon as possible. Consideration should be given to referring patients to tertiary centres

with microsurgical and endovascular expertise. Technological advances, such as MRA, have made effective screening possible. Risk factor modification, especially cigarette smoking and blood pressure control, is essential to influence the natural history of this disease.

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