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Cerebral Aneurysms

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1. Introduction

Intracranial aneurysms (IAs) occur in 0.2%-9% of adults (18, 35, 54). This wide range probably reflects methodological differences between studies: prospective or retrospective designs, diagnostic tools (angiography or autopsy) and study populations. IAs are present with subarachnoid haemorrhage (SAH) in the majority of cases. Aneurismal subarachnoid haemorrhage is a sudden and often catastrophic event with high mortality and morbidity rates: 15% die before hospitalisation and 36% die within 48 hours of its onset, 43% in the first week and 57% within six months (3). Furthermore, half of the survivors manifest physical or psychosocial deficits one year after SAH (22).

In the USA, it is estimated that SAH accounts for over 25% of all stroke-related years of potential life lost before the age of 65 (37). In Japan, SAH inflicts as many people as automobile accidents (AMA), about 13,000 per year (60). Recent improvements in the management of patients with aneurismal SAH have slightly reduced fatality rates over the last three decades (28). However, the severity of the initial bleeding and the clinical condition of the patients following SAH remain the main predictors for their outcome. Therefore, we believe that the treatment of unruptured aneurysms and the prevention of their formation and progression represent the best practical measures to avoid the grim outcomes of SAH. This requires a good understanding of the pathogenesis of IAs and the role of the various risk factors in their formation and progression.

2. Definition of cerebral aneurysm

The word aneurysm comes from the Latin word *aneurysma,* which means dilatation. Aneurysm is a persistent localised dilatation of the vessel wall, usually an artery. Saccular aneurysms account for the vast majority (98%) of all intracranial aneurysms, and the word aneurysm in this work refers to this form. Other types of aneurysms are fusiform, dissecting, infectious (mycotic) and traumatic aneurysms.

Saccular aneurysms consist of three main regions: the neck, the sac and the dome. The neck is that part of an aneurysm where it joins the parent artery, while the sac represents the cavity of the aneurysm and the dome relates to the convex wall facing the neck of the aneurysm. The size of an aneurysm is usually described as the maximum distance between the neck and the dome, and more detailed description of an aneurysm's size requires the

measurement of its two dimensions: the maximum distance between the neck and the dome and the maximum distance between the sides of the aneurysm (figure 1). Saccular aneurysms are specific to the intracranial arteries because their walls lack an external elastic lamina and contain very thin adventitia factors that may predispose to the formation of aneurysms. An additional feature is that they lie unsupported in the subarachnoid space.



Fig. 1. 3D DSA image of a left ICA showing a left ICA saccular aneurysm, measuring 22.7 mm (1) X 13.5 mm (2) and projecting medially

3. Historical background

The first description of saccular cerebral aneurysm in the medical literature was made in the eighteenth century (1761) by Morgagni (58) and Biumi (2), who first described the dilatation of the cerebral arteries and showed that their rupture might lead to SAH. Morgagni and Biumi's observations were not further evaluated until 1859, when Sir William Gull (21) offered recognition of the pathological nature of the lesion by his often quoted statement: "whenever young persons die with apoplexy, and after death a large effusion of blood is found, especially if the effusion be over the surface of the brain, in the meshes of the pia matter, the presence of aneurysm is probable."

In 1927, Egas Moniz (57) introduced cerebral angiography to the medical community and the clinician finally developed a method to diagnose CA. In 1933, Dott (10) presented a series of 8 patients who had undergone angiography with a diagnosis of subarachnoid haemorrhage, describing the location of their aneurysms and reporting his operative results. This was followed by many reports describing different methods for the management of CA, among which was that of Dandy (9) who described a series of 108 patients, 30 of them having had an intracranial procedure while others had carotid ligation. The technique of angiography is considered to be the key for all research in the field of cerebral aneurysm.

4. Epidemiology of cerebral aneurysms

Intracranial aneurysms are reported to be present in about 5% (range: 0.2%-9.0%) of the general population (18, 35, 54). The prevalence figures are clearly related with the method employed and the region and population studied. The true prevalence of intracranial aneurysms remains unknown, since most aneurysms remain undiagnosed until they rupture or produce neurological deficits. In one series of 72 consecutive patients undergoing coronary angiography, incidental intracranial aneurysms were found in 5 cases (6.9%) (36); another series reported a 2.8% prevalence of asymptomatic incidental aneurysms in 4518 patients undergoing magnetic resonance angiography (31). Reviewing 3684 cerebral arteriograms, Winn et al. reported a 0.65% prevalence of asymptomatic IAs (100).

A high incidence of IA has been reported in Japan and Finland (14, 36); on the other hand, a low incidence was reported in India, Iran, the Middle East and many parts of Africa (39, 61, 62), and it is difficult to determine whether this regional variation is genuine or related to the difference in diagnostic workup among these countries.

Aneurysms are typically diagnosed in people aged 40-60 years, reaching a peak in people aged 55-60 years (23, 60). Intracranial aneurysms are uncommon in children, accounting for fewer than 2% of all cases. When aneurysms occur in the paediatric age group, they are more often post-traumatic or mycotic rather than degenerative, and they have a slight male predilection. Aneurysms in children are also larger than those found in adults (55, 67).

Cerebral aneurysms affect women more frequently than men. In an autopsy study from Japan, the prevalence of aneurysms for women was 2.4 times higher than that for men (35). Among men, the prevalence of aneurysms remained unchanged across the range of age groups. In contrast, there were 2 peaks in the prevalence of aneurysms for women falling in the 40-49 and 60-69-year age groups (35). A cerebral angiogram based on a retrospective study showed that 67% of the patients harbouring asymptomatic aneurysms were women (100).

5. Location and nomenclature

Aneurysms commonly arise at the branching site of major arteries. It also occurs at a turn or curve of the artery, and points in the direction that the blood would have gone if the curve at the aneurysm site were not present (71). Aneurysm is usually referred to by the name of the parent artery from which they originate. Cerebral aneurysms occur more frequently in arteries of the anterior circulation. In large forensic clinical and autopsy series, the locations of intracranial aneurysms are the internal carotid artery (ICA) (24%-41%), the anterior cerebral artery (ACA) (30%-39%), the middle cerebral artery (MCA) (20%-33%) and the vertebrobasilar arteries (VBA) (4%-12%) (95) (Figure 2).

6. Presentation of unruptured cerebral aneurysms

Due to the recent spread of non-invasive diagnostic modalities (CTA and MRA), the majority of unruptured IAs are diagnosed incidentally. Here are some specific syndromes associated with particular aneurismal locations.

- *Anterior communicating artery:* Usually, ACoA aneurysms are silent until they rupture. Suprachiasmatic pressure may cause altitudinal visual field deficits, aboulia or akinetic mutism, amnestic syndromes and hypothalamic dysfunction.

- *Anterior cerebral artery:* Most are asymptomatic until they rupture, although frontal lobe syndromes, anosmia and motor deficits may be noted.
- *Middle cerebral artery:* This typically affects the first or second division in the sylvan fissure. Aphasia, hemiparesis, hemisensory loss, anosognosia and visual field defects may be noted.
- *Posterior communicating artery:* These are directed laterally, posteriorly and inferiorly. Pupillary dilatation, ophthalmoplegia, ptosis, mydriasis and hemiparesis may result.
- *Internal carotid artery:* Supraclinoid aneurysms may cause ophthalmoplegia due to the compression of the cranial nerve (CN) III or variable visual defects and optic atrophy due to the compression of the optic nerve. Chiasmal compression may produce bilateral temporal hemianopsia. Hypopituitarism or anosmia may be seen with giant aneurysms. Cavernous-carotid aneurysms exert mass effects within the cavernous sinus, producing ophthalmoplegia and facial sensory loss.
- *Basilar artery:* The clinical findings are usually those associated with SAH, although bitemporal hemianopsia or an oculomotor palsy may occur. Dolichoectatic aneurysms may cause bulbar dysfunction, respiratory difficulties and neurogenic pulmonary oedema.
- *Vertebral artery or posterior inferior cerebellar artery:* Aneurysms at these arterial segments typically result in ataxia, bulbar dysfunction or spinal involvement.

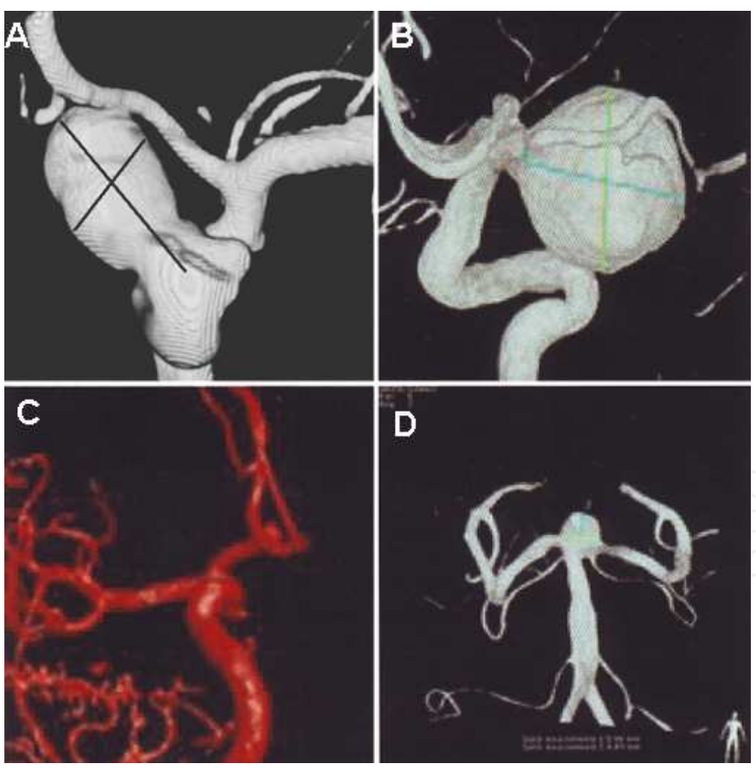


Fig. 2. 3D DSA images demonstrating common sites of IAs. A: Right ICA, demonstrating right giant ICA saccular aneurysm, measuring 22 mm X 11.8 mm (A). B: Right MCA trifurcation aneurysm (26X19 mm). C: Anterior communicating artery small aneurysm. D: 3D DSA image of the vertebrobasilar arteries showing basilar top aneurysm measuring 24X16

7. The natural course of incidental aneurysms

The increased sensitivity of neuroimaging techniques has enabled the more frequent diagnosis of unruptured aneurysms. Because the most devastating complication of an unruptured aneurysm is subarachnoid haemorrhage, it has been considered desirable to treat these aneurysms before they rupture. However, the optimal treatment strategy for patients with unruptured aneurysms remains controversial. The management decision requires knowledge of the natural history of the patient and an accurate assessment of the risks related to various treatment options.

No consensus exists regarding the natural course of unruptured aneurysms. Unruptured aneurysms may remain asymptomatic and static; they may grow and or rupture. On the other hand, there are some reports describing the spontaneous regression of aneurysms (82, 97).

A systematic review of the literature on the risk of the rupture of aneurysms identified nine studies with a total of 3907 patient years of follow-up (72). During follow-up, 75 of 495 (15.2%) patients suffered an SAH, giving an annual rupture rate of 1.9%. Aneurysms were significantly more likely to rupture in women than in men (RR 2.1, 95% CI 1.1-3.9) and the risk of rupture increased with age, e.g., in the group of patients aged 60-79 years, the RR of rupture was 1.7 (95% CI 0.7-4.0) compared with those aged 40-59 years. The review also showed higher RR in symptomatic aneurysms when compared to asymptomatic aneurysms (6.5% versus 0.8% respectively) (72).

The initial size of the intact aneurysm and the subsequent rupture rate is a complex issue. In Juvela's study (38), there was no disparity in the size of the aneurysm on digital subtraction angiography (DSA) at the start of follow-up between patients who later had a SAH and those who did not (median 4 mm, range 2-25 mm in those with later SAH versus median 4 mm, range 2-26 mm in those without). Of the aneurysms which later ruptured, 67% were <6 mm in diameter.

In a study by Yasui et al., (103) 234 patients with and without SAH were evaluated during a period of 6.25 years. Thirty-four patients (14.5%) bled, with an average annual rupture rate of 2.3%. In a separate study, these authors evaluated aneurysm size in 25 patients with the rupture of a previously unruptured aneurysm (102).Twenty-two of the newly ruptured aneurysms were <9 mm in diameter at initial diagnosis, and 16 were <5 mm in diameter. The authors concluded that even the smallest UIAs require "radical treatment or careful follow-up."

The largest ever study to follow-up unruptured aneurysms is the ISUIA, with 2621 patients (33, 34). This studied two groups of patients retrospectively: (i) patients with asymptomatic aneurysms with no prior SAH, and (ii) those with multiple aneurysms who had previously sustained an aneurismal SAH. The investigators also studied prospectively the risks of the treatment of asymptomatic unruptured aneurysms (34). The results of the ISUIA indicate a tiny rupture risk of 0.05% per annum for small aneurysms (<I 0 mm diameter) in patients who have not had an SAH previously, and 0.5% per annum for large aneurysms and for all aneurysms in patients who had previously sustained SAH from another aneurysm. Of the 1449 included patients with 1937 unruptured saccular aneurysms <12 mm diameter, 32 patients had confirmed aneurysm rupture during follow-up; the mean duration of follow-

up was 8.3 years (12023 patient years in total). In the cohort that had previously not had an SAH, only one of 12 aneurismal ruptures occurred in an aneurysm <10 mm in diameter, compared with 17 of 20 patients in the cohort who had previously had an SAH. This study also found that the only significant predictors of rupture were the size and location of the aneurysm: aneurysms >10 mm diameter had an RR of rupture of 11.6; for posterior circulation aneurysms, the RR was 13.8 and 13.6 for basilar tip and vertebrobasilar locations, respectively, and 8.0 for posterior communicating artery aneurysms.

The discrepancy in aneurismal rupture rates between the systematic reviews (38, 72, 102, 103) and the ISUIA (33, 34) requires explanation. The majority of ISUIA patients were identified retrospectively from hospital records (1981 onwards, with the identification process commencing in 1992) and only survivors with persistently asymptomatic aneurysms - in whom a complete set of angiograms could be traced - were eligible for inclusion. These patients might not be entirely representative of the natural history of all aneurysms: e.g., subjects who had suffered a fatal episode of SAH, or where an asymptomatic aneurysm had been treated since 1981, or else who had incomplete angiograms, could not be included in the ISUIA. The prospective analysis and follow-up of patients with asymptomatic aneurysms provides less biased data.

Although many authors tried to define a critical size for saccular aneurysm rupture, we believe that a critical size below which SAH does not occur does not appear to exist. Other risk factors for IA rupture include age, sex, hypertension, and multiple aneurysms and previous SAH. Identifying the mechanism of aneurysm formation, growth and rupture will explain the role of the various risk factors - including aneurismal size - in the rupture of IAs.

8. Treatment of unruptured IAs

Aneurysms may be treated by surgical clipping (or wrapping) or by interventional neuroradiology. Surgical treatment, having been in use routinely for >40 years, has fairly clearly defined the risks and morbidity.

A systematic review of surgical treatment for unruptured aneurysms was performed by Raaymakers and colleagues, who identified 61 studies including 2460 patients and at least 2568 aneurysms published between 1966 and June 1996 (69).

Only studies in which at least 90% of patients were treated by clipping (as opposed to wrapping or other surgical techniques) were included. Unfortunately, only eight of the studies were prospective, the rest being retrospective and - in virtually all studies - the neurosurgeon performing the operation was also the observer of the outcome. The median follow-up was only 24 weeks (range 2-234 weeks) in the 21 studies which reported the time of outcome assessment. The overall permanent morbidity occurred in 10.9% (95% CI 9.6%-12.2%) of patients and mortality was 2.6% (95% CI 2.0%-3.3%). The lowest morbidity and mortality was found with small anterior circulation aneurysms (mortality 0.8%, morbidity 1.9%), and the worst with large posterior fossa aneurysms (mortality 9.6%, morbidity 37.9%).

The prospective arm of the ISUIA also addressed the issue of the risks of surgical intervention in unruptured aneurysms (34). This enrolled 1172 patients (211 of whom had a history of previous SAH) and 996 underwent surgery. The surgery-related mortality at 1 year was 3.8% (95% CI) in patients with no prior SAH and 2% in patients who had

previously suffered an SAH from a different aneurysm which had already been treated. The morbidity was 12.0% and 12.1%, respectively. The mortality figures at 1 month in the ISUIA study were similar to those in the systematic review, at a median of 24 weeks, 2.3% versus 2.6%, respectively. Age was the only independent predictor of outcome in the ISUIA study: the RR of surgery-related morbidity and mortality at 1 year was X5 in the group >64 years of age compared with patients <45 years of age.

The effectiveness and risks of aneurysm coiling are less certain because the technique is newer and still developing. The USA Multicenter Study Group identified a 1% mortality and a 4% morbidity for unruptured aneurysm treatment, with 78% of aneurysms being completely occluded. The rupture rate of partially coiled aneurysms was 0.5% per annum from the limited follow-up data available (91). There is some evidence that even partial treatment by GDC confers benefits in the early post-rupture period; post-GDC treatment haemorrhage occurred in only nine of 403 patients, although the length of follow-up was very limited in many patients (91).

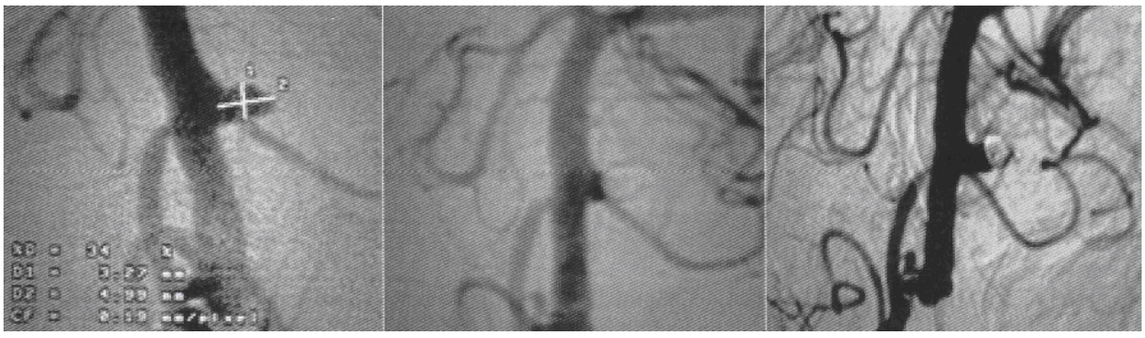


Fig. 3. Demonstrating the enlargement of BA AICA aneurysm following coiling with GDC. A: Initial Left VADSA; B: DSA following coiling; C: DSA six months after coiling showing coil impaction and enlargement of the aneurismal cavity.

In the case of an unruptured aneurysm, should one decide treatment was necessary then the long-term results of coiling are particularly relevant because coiling could provide a less invasive alternative to surgery. It is worth noting that the published rupture rate of partially coiled aneurysms is the same as that reported from the ISUIA study for untreated unruptured aneurysms >10 mm diameter or for any unruptured aneurysm in a patient with a previous SAH. The regrowth rate of partially coiled aneurysms is still being defined, and so there are considerable uncertainties about the long- and short-term effectiveness of coiling.

Apart from direct treatment of the aneurysm, it is likely that there are other ways of reducing the risk of rupture for incidentally discovered aneurisms, which could - collectively - have a useful effect. The cessation of smoking, careful control of blood pressure and the avoidance of risk factors for atherosclerosis (careful diet, regular exercise, etc.), while unproven, may help reduce both the risk of formation of aneurysms and the risk of rupture, as well as improving general health.

8.1 Management considerations and recommendations

Aneurismal SAH is a devastating condition for which prevention has been advocated as the most effective strategy aimed at lowering mortality rates. However, all current treatments

carry risks, and recommendations for treatment versus observation are often difficult and controversial. Treatment complications generally occur at around the time of the procedure, but they could potentially improve during the patient's remaining lifetime. In contrast, the risk of rupture of an untreated aneurysm is cumulative but may provide a period of unimpaired life. Non-lethal complications in both settings can potentially improve over time.

Deliberations must take into account important characteristics of the aneurysm and the patient in whom it exists. Of the former, particular consideration must be given to aneurysm size, form and location and its symptomatic versus incidental status. As a general rule, exclusively extradural, intracavernous (internal carotid artery) aneurysms, even if symptomatic with pain or ophthalmoparesis, do not carry a major risk for intracranial haemorrhage, and thus management decisions are primarily aimed at symptom relief rather than haemorrhage prevention.

Among patient factors, the patient's age, general medical condition, previous history of SAH and family history of aneurismal SAH are prime considerations in the treatment analysis. Symptoms due to UIAs should be discriminated relative to those developing rapidly and related to smaller aneurysms, presumably due to acute aneurismal expansion. Generally, symptomatic aneurysms are larger, occasionally giant in size and sometimes partially thrombosed, producing subacute symptoms due to adjacent cranial nerve or brain compression. Such lesions carry a major risk for both progressive neurological deficit and aneurysm rupture.

The existing body of knowledge supports the following recommendations (options) regarding the treatment of UIAs:

1. The treatment of small incidental intracavernous ICA aneurysms is not generally indicated. For large symptomatic intracavernous aneurysms, treatment decisions should be individualised on the basis of the patient's age, the severity and progression of symptoms and treatment alternatives. The higher risk of treatment and shorter life expectancy in older individuals must be considered in all patients, and it favours observation in older patients with asymptomatic aneurysms.
2. Symptomatic intradural aneurysms of all sizes should be considered for treatment, with relative urgency for the treatment of acutely symptomatic aneurysms. Symptomatic large or giant aneurysms carry higher surgical risks that require a careful analysis of individualised patient and aneurismal risks and surgeon and centre expertise.
3. Coexisting or remaining aneurysms of all sizes in patients with SAH due to another treated aneurysm carry a higher risk for future haemorrhage than do similar sized aneurysms without a prior SAH history, and they warrant consideration for treatment.
4. If a decision is made for observation, re-evaluation on a periodic basis with CT/MRA or selective contrast angiography should be considered so as to check for changes in aneurismal size and shape.
5. In consideration of the apparent low risk of haemorrhage from incidental small (<10 mm) aneurysms in patients without previous SAH, treatment rather than observation cannot generally be advocated. However, special consideration for treatment should be given to young patients in this group. Likewise, small aneurysms approaching the 10 mm diameter size, those with daughter sac formation and other unique hemodynamic

features, and patients with a positive family history for aneurysms or aneurismal SAH deserve special consideration for treatment.

6. Asymptomatic aneurysms of ->10 mm (ISUIA) or >7mm (ISUIA-II) in diameter warrant strong consideration for treatment, taking into account the patient's age, existing medical and neurological conditions and the relative risks of treatment.

These recommendations are based on our limited knowledge concerning the natural history of unruptured aneurysm. In our clinical practice, we recommend - whenever possible - the active management of these lesions

9. Subarachnoid haemorrhage (SAH)

Primary SAH is defined as a bleeding which takes place primarily in the intracranial subarachnoid space and is not secondary to some other intracranial haemorrhage (66). In a recent systematic review of the literature, the worldwide overall incidence of SAH was 10.5 per 100,000 person years (85). However, the incidence in Japan was 21.0 per 100,000 person years and in Finland it was 22.0 per 100,000 person years (63). The overall incidence of aneurismal SAH has remained constant during recent decades (50), but increases almost linearly with increasing age (16).

9.1 The aetiology of SAH

In more than 80% of cases, the cause of primary SAH is the rupture of an intracranial aneurysm (77, 85). SAHs of unknown origin represent 9%-15% of cases (75). The source of the bleeding of unknown origin can be the rupture of a small perforating artery or a micro-arteriovenous malformation (AVM) which is not identifiable in diagnostic imaging (75). Intracranial artery dissections, cerebral AVMs, dural AVMs, trauma, bleeding disorders, substance abuse, a spinal origin of the haemorrhage and other rare conditions account for primary SAH in less than 5% of cases (85).

9.2 Clinical presentation

This is characterised by the acute onset of severe headache, which patients often describe as "the worst headache of my life." The sudden elevation of intracranial pressure associated with aneurismal rupture may lead to a precipitous decline in cerebral perfusion pressure, causing syncope (50% of cases), confusion or mild impairment in alertness. Other symptoms include:

- Seizures: Focal or generalised seizures are present in 25% of aneurismal SAH cases, with most events occurring within 24 hours of onset.
- Manifestations of meningeal irritation: Neck pain or stiffness, photophobia, sonophobia or other hyperesthesia may be noted with SAH.
- Autonomic disturbances: The subarachnoid accumulation of the products of blood degradation may elicit fever, nausea or vomiting, sweating, chills and cardiac arrhythmias.
- Focal neurological complaints: Haemorrhage or ischemia may manifest with focal deficits, including weakness, hemisensory loss, language disturbances, neglect, memory loss or olfactory disturbances. Focal symptoms are more common with giant aneurysms.

- Respiratory dysfunction or cardiovascular instability: These are ominous signs of brainstem compression.
- SAH can be associated with myocardial stunning (transient), sometimes with the features of tako-tsubo syndrome (transient left ventricular apical ballooning syndrome), and with lethal ventricular arrhythmia, all probably caused by SAH-induced catecholamine surge.

The physical examination of patients with aneurismal SAH may reveal nuchal rigidity, a decreased level of consciousness, subhyaloid haemorrhages, pupillary abnormalities (i.e., typically dilated), ophthalmoplegia, cranial neuropathies and other focal deficits. Giant aneurysms may cause mass effects or distal thromboembolism with prominent focal deficits, optic atrophy or other cranial neuropathies as well as brainstem compression.

9.3 The diagnosis of SAH

CT and MRI

If SAH is suspected, CT scanning is the first line in investigation because of the characteristically hyperdense appearance of extravasated blood in the basal cisterns. The pattern of haemorrhage often suggests the location of any underlying aneurysm (86) (Figure 4). CT studies performed within 12 h after the haemorrhage were found to be negative in 2% of patients with SAH (84).

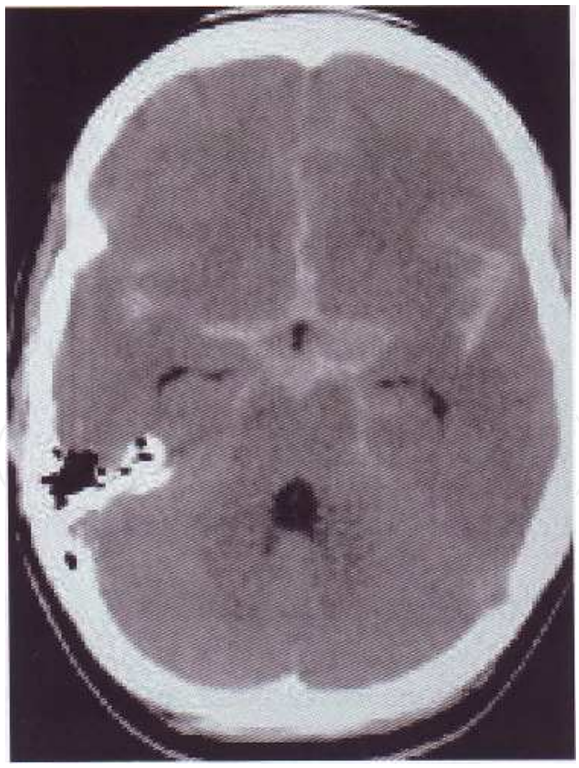


Fig. 4. CT scan of a 47-year-old woman presented with headache and vomiting; her CT scan in the emergency department revealed subarachnoid haemorrhage involving the sylvan and interhemispheric fissure, the interpeduncular and the ambient cistern.

MRI with FLAIR (fluid attenuated inversion recovery) techniques demonstrates SAH in the acute phase as reliably as CT (59), but MRI is impracticable because the facilities are less readily available than CT scanners, and restless patients cannot be studied unless anaesthesia is given. In the subacute cases, however, MRI is increasingly superior to CT in detecting SAH (59).

Lumbar puncture

Lumbar puncture is still an indispensable step in the exclusion of SAH in patients with a convincing history and negative brain imaging. Lumbar puncture should not be carried out rashly or without some background knowledge. The first rule is that at least 6 h and preferably 12 h should have elapsed between the onset of headache and the spinal tap. The delay is essential because if there are red cells in the CSF, sufficient lysis will have taken place during that time for bilirubin and oxyhaemoglobin to have formed (90). The pigments give the CSF a yellow tinge after centrifugation (xanthochromia) - a critical feature in the distinction of SAH from a traumatic tap - and are invariably detectable until at least 2 weeks later. The 'three tube test' (a decrease in red cells in consecutive tubes) is notoriously unreliable, and a false-positive diagnosis of SAH can be almost as invalidating as a missed one. Spinning down the blood-stained CSF should be done immediately, otherwise oxyhaemoglobin will form *in vitro.* If the supernatant appears crystal-clear, the specimen should be stored in darkness until the absence of blood pigments is confirmed by spectrophotometry.

9.4 Diagnosis of intracranial aneurysm

The gold standard for detecting aneurysms is conventional angiography, but this is not an innocuous procedure. A systematic review of three prospective studies in which patients with SAH were distinguished from other indications for catheter angiography found a complication rate (transient or permanent) of 1.8% (6).

Other imaging modalities are MR angiography (MRA) and CT angiography (CTA), which are non-invasive. A recent review of studies comparing MRA and intra-arterial angiography in patients with recent SAH, under blinded-reader conditions, showed a sensitivity in the range of 69%-100% for detecting at least one aneurysm per patient and 70%-97% for the detection of all aneurysms, with specificity in the range 75%-100% (93). The sensitivity of CTA (compared with catheter angiography) is 85-98%, which is in the same range as that of MRA (1). Hashimoto et al, reported the ability of CTA to detect aneurysms that were missed by conventional angiography (25).

9.5 The grading of SAH

The Fisher (15) grading method (Table 1) is dependent on findings by CT-scan and has been found to be useful in predicting vasospasm after SAH; patients with a thick layer of blood in the subarachnoid space were found to have a higher incidence of cerebral vasospasm (46).

The most common system for grading the clinical condition after SAH is the Hunt and Hess (H&H) (32) scale (Table 2). In the original classification according to Hunt&Hess, the patient grade was increased by one level in the presence of serious underlying medical disorders. On this scale, a higher grade at presentation correlates with increasingly poor clinical outcomes.

Fisher Grade	Blood on CT	No of Pts	Angiographic vasospasm	Clinical vasospasm
1	none	11	4	0
2	Diffuse or vertical layer >1 mm thick	7	3	0
3	Localised clot or vertical layer >1 mm thick	24	24	23
4	Intracerebral or intraventricular blood	5	2	0

Table 1. The Fisher CT grade

H&H Grade	Description
1	Asymptomatic or mild H/A and slight nuchal rigidity
2	Moderate to severe H/A, nuchal rigidity, no deficit except Cr. N. palsy (e.g., III, VI)
3	Mild focal deficit, lethargy or confusion
4	Stupor, moderate to severe hemiparesis, early decerebrate rigidity
5	Deep coma, decerebrate rigidity, moribund appearance
Add one grade for serious systemic disease (D.M, HTN, Atherosclerosis, COPD)	

Table 2. Hunt and Hess classification of SAH

In 1987, the World Federation of Neurological Surgeons (WFNS) (70) proposed a new grading system (Table 3) in which two factors have been assigned to differentiate grades: the level of consciousness - classified with the Glasgow coma scale (GCS) - and focal neurological deficits. The timing of the grading is important because the patient's worst clinical grade is the best predictor of the outcome.

WFNS grade	Glasgow Coma Scale Score	Major focal deficit*
0 (intact aneurysm)	-	-
1	15	absent
2	13-14	absent
3	13-14	present
4	7-12	present or absent
5	3-6	present or absent

*Aphasia and/or hemiparesis or hemiplegia

Table 3. World Federation of Neurologic Surgeons (WFNS) SAH grade

9.6 The outcome of ruptured intracranial aneurysms

Case fatality ranged between 32% and 67% in a review of population-based studies from 1960 onward. The weighted average was 51%. Of patients who survive the haemorrhage, approximately one-third remain dependent (28). Recovery to an independent state does not

necessarily mean that the outcome is good. In a study of quality of life in patients after SAH, only 9 of 48 (19%; 95% CI 9%-33%) patients who were independent 4 months after the haemorrhage had no significant reduction in quality of life (29). All in all, only a small minority of all patients with SAH have a truly good outcome. The relatively young age at which SAH occurs and its poor outcome together explain why the loss of years of potential life before the age of 65 from SAH is comparable to that of ischaemic stroke (37).

Factors that influence prognosis

The predictors for mortality include the patient's decreased level of consciousness, their increased age, the thickness of the subarachnoid haemorrhage clot on computerised tomography, elevated blood pressure, pre-existing medical illnesses and basilar aneurysms (42, 79). A decreased level of consciousness, with the initial haemorrhage or after early rebleeding, may be caused by intracerebral haematoma, subdural haematoma or hydrocephalus. Only by exclusion should it be assumed that the cause is global brain damage as a result of high pressure and subsequent ischaemia.

Rebleeding

In the first few hours after admission for the initial haemorrhage, up to 15% of patients have a sudden episode of clinical deterioration that suggests rebleeding (17). As such, sudden episodes often occur before the first CT scan, or even before admission to hospital - a firm diagnosis is difficult and the true frequency of rebleeding on the first day is invariably underestimated.

In the prospective Cooperative Aneurysm Study (44), the risk of rebleeding with conservative therapy was highest (4%) on the first day after SAH and then remained constant at a rate of 1%-2% per day during the subsequent two weeks. In his earlier series, Pakarinen (66) reported a cumulative frequency of rebleeding of 7%, 16%, 23% and 33% at week 1, week 2, week 3 and week 4 respectively. The mortality at first recurrence was 63.6%, and at second recurrence it had risen to 86%.

Intracerebral haematoma

Intraparenchymal haematomas occur in up to 30% of patients with ruptured aneurysms (87). Not surprisingly, the average outcome is worse than in patients with purely subarachnoid blood (26). When a large haematoma is the most likely cause of the poor condition on admission, the immediate evacuation of the haematoma should be seriously considered (with simultaneous clipping of the aneurysm if it can be identified), often with the aneurysm having been demonstrated only by MR angiography or CT angiography.

Acute subdural haematoma

An acute subdural haematoma, which is usually associated with recurrent aneurismal rupture, but can also occur with the initial haemorrhage, may be life threatening; in these cases, immediate evacuation is also called for (65).

Acute hydrocephalus

Gradual obtundation within 24 h of haemorrhage, sometimes accompanied by slow pupillary responses to light and downward deviation of the eyes, is fairly characteristic of

acute hydrocephalus (73). If the diagnosis is confirmed by CT, this can be a reason for early ventricular drainage, although some patients improve spontaneously in the first 24 h.

Global cerebral ischaemia

The most likely explanation is the elevation of the pressure in the cerebrospinal fluid spaces to the level of that in the arteries at the time of haemorrhage, resulting in a prolonged period of global cerebral ischemia. Such an immediate and potentially lethal arrest of the circulation to the brain is indeed suggested by autopsy evidence and by the recording of intracranial pressure or transcranial Doppler sonography at the time of recurrent aneurismal haemorrhage (19, 78). This is quite distinct from delayed ischaemia, which is either focal or multifocal (see below).

Cerebral vasospasm

The most feared complication of SAH is a form of delayed-onset cerebral arterial narrowing, known as VSP (13). Angiographic VSP is detected in 50%-70% of patients in the first two weeks after SAH (94). In about one-half of cases, angiographic VSP manifests itself by the occurrence of a delayed ischemic neurological deficit, which may resolve or progress to permanent cerebral infarction (53). The development of delayed ischemic deficit (DID) is still considered to be the major cause of morbidity and mortality in those patients who survive long enough to reach the neurosurgical unit. In a recent large multicentre study, DID killed 7% of patients with aneurismal SAH and left another 7% with severe permanent neurological deficits (42).

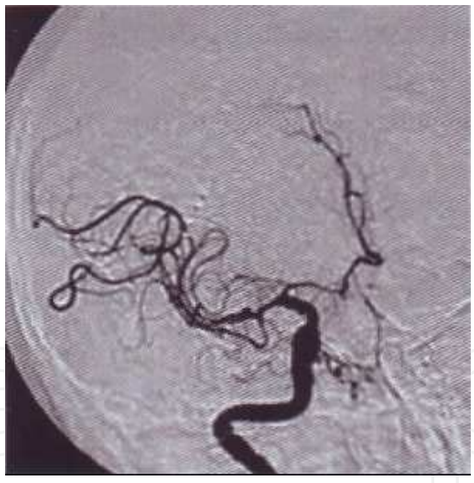


Fig. 5. Left carotid DSA performed postoperatively and demonstrating cerebral vasospasm affecting both ACA and MCA.

9.7 Treatment guidelines for ruptured IA

9.7.1 The prevention of rebleeding

9.7.1.1 Antifibrinolytic drugs

Medical treatment for preventing rebleeding has not yet been successful; treatment with antifibrinolytic agents does reduce the rebleed rate, but fails to improve overall outcome.

A systematic review of antifibrinolytic agents included eight trials published before 2000 that met predefined inclusion criteria and totalled 937 patients (76). By far the largest study was a Dutch-Scottish trial (89). In this meta-analysis, antifibrinolytic treatment did not provide any evidence of benefit on outcome. The risk of rebleeding was significantly reduced by antifibrinolytic therapy, but this was offset by a similar increase of the risk of secondary cerebral ischaemia.

9.7.1.2 Operative clipping of the aneurysm

The results of operative treatment were poor until advances in microsurgical techniques and neuroanaesthesia in the late 1960s allowed neurosurgeons to successfully treat the majority of intracranial aneurysms. The goal for surgical treatment of intracranial aneurysms is to eliminate the aneurysm from the circulation while preserving blood flow through the parent artery and branch vessels. This treatment is best accomplished by direct clip placement across the aneurysm neck.

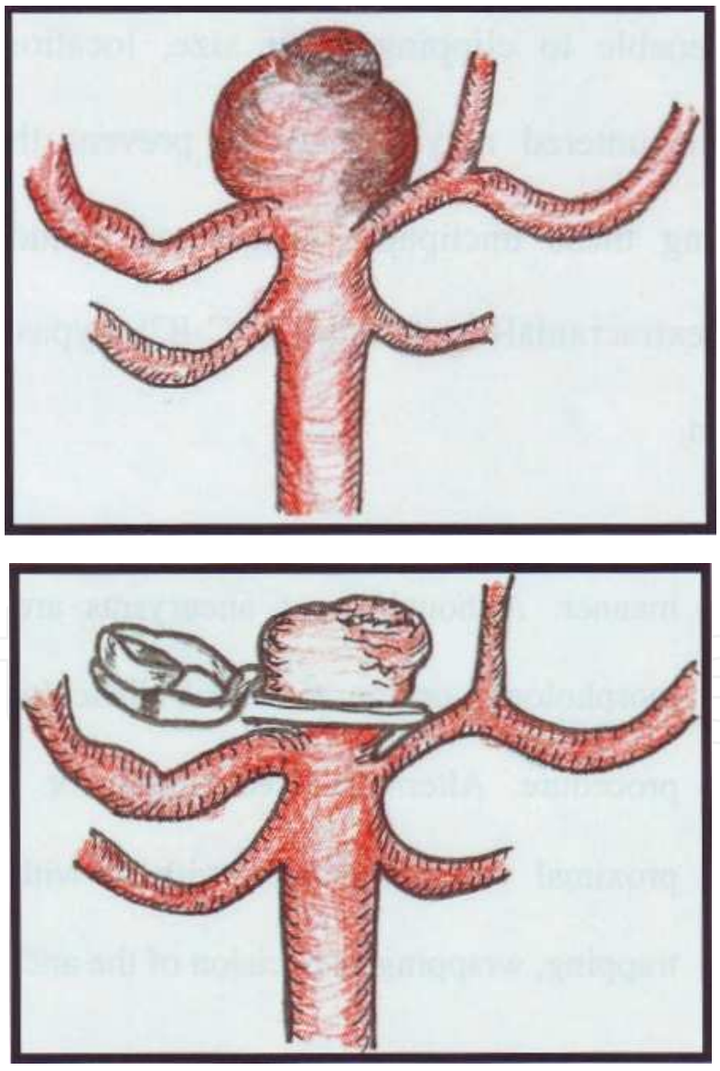


Fig. 6. Illustration of the surgical clipping of a basilar tip aneurysm.

9.7.1.2.1 Technical aspects of the surgical repair of ruptured aneurysms

An operative approach must take into account the location of the aneurysm, and it should allow minimal brain retraction. According to Yasargil (101), the most useful approaches are: 1) pterional craniotomy for aneurysms of the anterior circulation and upper basilar artery; 2) paramedian frontal craniotomy for pericallosal artery aneurysms; 3) lateral suboccipital craniotomy for aneurysms of the vertebral circulation below the origin of the superior cerebellar arteries. The clips for the temporary occlusion of the parent artery have a low closing force so as not to cause permanent damage to the vessel wall. To achieve permanent clipping, there are multiple choices of clips of different sizes, shapes and closing forces. The modern clips are MRI compatible.

Using gentle brain retraction, the arachnoid cisterns are entered with sharp dissection and finally the aneurysm and the adjacent vessels are exposed with a meticulous dissection. Prior to the application of the clip, the aneurysm neck must be free of adhesions to the surrounding arteries and neural structures. In narrow-necked aneurysms, the clip can be placed across the aneurysm neck. However, in complex aneurysms several clips may be needed to appropriately occlude the aneurysm in a stepwise manner. Although most aneurysms are amenable to clipping, their size, location, morphology and the technical difficulties encountered may sometimes prevent the procedure. Alternative techniques for treating these unclippable aneurysms include proximal vessel occlusion with or without extracranial intracranial (EC IC) bypass, trapping, wrapping and excision of the aneurysm.

9.7.1.2.2 The timing of the surgical treatment of ruptured aneurysms

The optimal timing for the surgical treatment of acutely ruptured aneurysms has been under constant investigation and is the subject of major controversy. In the 1960s, operative treatment was still generally delayed for 3 to 4 weeks following SAH so that the brain could recover from the acute effects of SAH. However, mortality and morbidity during the waiting period was high because of the occurrence of VSP and rebleeding (43). An operation during an early phase (within 72 hours following SAH) or even during the acute phase (within 24 hours following SAH) was considered justifiable in order to prevent early rebleeding and allow for the aggressive treatment of VSP, and thus improve the outcome for the patients.

In a randomised trial of the timing of the operation performed, 216 patients were allocated to operation within 3 days, after 7 days or else in the intervening period (64). The outcome tended to be better with the early operations rather than after intermediate or late operations, but the difference was not statistically significant. The same result - i.e., no difference in outcome after early or late operations - has emerged from various observational studies: a multi-centre study from North America (43) and a single institution review in Cambridge, UK (96). The US study found the worst outcome among patients operated on between day 7 and day 10 after the initial haemorrhage. This disadvantageous period for performing the operation in the second week after SAH coincides with the peak time of cerebral ischaemia and cerebral vasospasm, both phenomena being most common between days 4-12.

Although the data available is not consistent, early surgery for patients in a good preoperative clinical grade (Hunt&Hess Gr I-III) has gradually been accepted as a treatment policy by many institutions. Delayed surgery for patients in a poor preoperative clinical

grade, however, may be advisable unless immediate surgical intervention is required because of large haematoma or severe hydrocephalus.

9.7.1.3 Endovascular treatment of ruptured intracranial aneurysms

Until a few years ago, endovascular treatment was restricted to patients in whom the aneurysm was unsuitable for clipping because of the size or location of the aneurysm, or in whom surgical clipping was contraindicated because of the general medical condition of the patient. Since the introduction of controlled detachable coils for the endosaccular packing of aneurysms (20), endovascular embolisation is increasingly used. In some institutes, endovascular embolisation is even proposed as the initial method of treatment (7).

The Guglielmi Detachable Coil system

In 1991, Guglielmi et al. (20) introduced an electrically detachable coil system (GDC) that permitted the readjustment of the coil position before its final detachment. The GDC system consists of a platinum coil attached to a stainless steel delivery wire (pusher). The pusher is coated for electrical isolation with the exception of the most distal part: the detachment zone. A guiding catheter is used in advancing to the ICA or the vertebral artery. A microcatheter with 2-tip-markers and a guidewire are used for hyperselective catheterisation of the aneurismal sac. The distal end of the microcatheter is shaped with steam to tailor the vascular geometry before catheterisation. Digital road mapping may be used so as not to touch the aneurismal wall with the guidewire or catheter. A continuous pressurised flush of heparinised saline is maintained in both the guiding catheter and the microcatheter. The aneurysm sac is then filled with coils of selected shapes and sizes. The system allows for the removal of the coil as well as the repositioning of the mesh to an optimal position. When the coil is seen to be in a suitable position inside the aneurysm, a positive direct electric current is applied to the proximal end of the stainless steel guidewire. The current provokes thrombus formation in the aneurismal cavity and dissolves the uninsulated stainless steel coil closest to the platinum coil, resulting in the detachment of the coil.

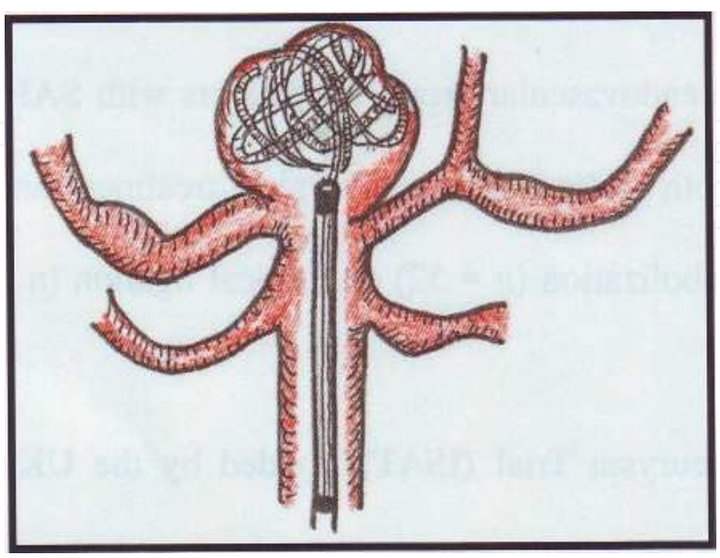


Fig. 7. Of c: Illustration of the oiling procedure with coils filling the aneurismal cavity.

Numerous observational studies have published complication rates, occlusion rates and short-term follow-up results. These have been summarised - up to March 1997 - in a systematic review of 48 eligible studies of 1383 patients (4). Permanent complications in the procedure occurred in 3.7% of 1256 patients in whom this was recorded (95% CI 2.7%-4.9%). A >90% occlusion of the aneurysm was achieved in almost 90% of patients. The most frequent complication was procedure-related ischaemia, even if patients were treated with heparin. The second most frequent complication was aneurysm perforation, which occurred in 2% of patients. Most of the aneurysms treated with controlled detachable coils were located at the basilar artery, followed by the carotid and anterior communicating arteries. Pericallosal arteries are difficult to reach and another problematic site is the trifurcation of the middle cerebral artery because one or more of the branches often originate from the aneurysm.

9.7.1.4 Clipping versus coiling

Indirect comparisons between endovascular and surgical treatment are inappropriate, if only because there are so many differences in study design and among the patients and aneurysms. Moreover, the rerupture of aneurysms may occur even months after apparently successful coiling (52), and the long-term rates of rebleeding after endovascular coiling still need to be established. A first report from a single centre in which >300 patients were followed-up after aneurysm embolisation for a median period of almost 2 years showed rebleeding rates of 0.8% in the first year, 0.6% in the second year and 2.4% in the third year, with no rebleeding in subsequent years (5).

Similarly surgical treatment is not always definitive: in a retrospective review of postoperative angiograms in a series of 66 patients with ruptured aneurysms and 12 additional aneurysms, all treated by surgical clipping, 8% of patients showed aneurysms with a residual lumen or aneurysms that were previously undetected (51). Controlled trials are urgently needed in patients with aneurysms for which it is uncertain whether surgical clipping or endovascular coiling should be the preferred treatment. The first such study, although a small one (109 patients), found no difference in outcome at 3 months between the surgical group and the endovascular group for patients with SAH. All of the patients were suitable candidates for both endovascular and surgical treatment and were randomly assigned to undergo coil embolisation *(n* = 52) or surgical ligation *(n* = 57) (88).

The International Subarachnoid Aneurysm Trial (ISAT), funded by the UK's Medical Research Council (56), is the first large multi-centre randomised study in the world to compare the two methods. The ISAT trial enrolled 2143 patients with ruptured intracranial aneurysms in 43 neurosurgical centres in Europe, North America and Australia, and randomly assigned them to neurosurgical clipping (*n* = 1070) or GDC coils (*n* = 1073). Because interim analysis demonstrated a striking difference between these groups in favour of coiling, the trial was stopped short of its planned goal of 2500 patients. At 1 year follow-up, the risk of death or significant disability (Rankin score 3-6) was 30.6% in the neurosurgical group compared to 23.7% in the endovascular group. There was a 6.9% absolute and 22.6% relative risk reduction in favour of coiling. Many patients were screened but excluded from the study. Of the 9559 patients initially assessed, only 20% were actually enrolled. In the remainder, a decision was made that clipping or coiling was clearly more favourable, and thus the patient could not be subjected to a random treatment choice. Considerable bias could have been introduced into the study during this screening process.

The ISAT patients are not completely representative of the SAH population at large. 88% were of good clinical status (Hunt and Hess grade I or II), 93% of aneurysms were 10 mm or smaller in diameter and 97% were in the anterior circulation. The generalisability of the ISAT data is therefore limited. There was a significant occurrence of post-treatment rebleeding. This was found in 2.4% (26/1048) of endovascular cases compared with 1.0% (10/994) of clipped patients. While these results clearly favour neurosurgery, such an incidence of incompletely treated aneurysms is unusual for either therapy in experienced hands. In conclusion, the ISAT tells us that in patients with a ruptured intracranial aneurysm - for which endovascular coiling and neurosurgical clipping are therapeutic options - the outcome in terms of survival free from disability at 1 year is significantly better with endovascular coiling. Long-term follow-up is very important since rebleeding rates were found to be significantly higher in patients undergoing endovascular coiling.

9.7.2 The prevention of vasospasm

Angiographic VSP is detected in 50%-70% of patients within the first two weeks after SAH (94). In about one-half of cases, angiographic VSP manifests itself by the occurrence of a delayed ischemic neurological deficit (53). Delayed cerebral ischaemia (DID) is still considered to be the major cause of morbidity and mortality in those patients who survive long enough to reach the neurosurgical unit. Despite many years of intensive research, the pathogenesis of secondary cerebral ischaemia following SAH has not been elucidated. It is a generally held belief that after haemorrhage a thus far unidentified factor is released in the subarachnoid space, which induces vasoconstriction and thereby secondary ischaemia. Also, an often quoted study from Boston (of 41 patients in total) postulates a close relationship between the location of subarachnoid blood and the 'thickness' of the clot on the one hand, and the occurrence of vasospasm and delayed cerebral ischaemia on the other (46). Several observations argue against this popular notion. Firstly, the presence of subarachnoid blood, though a powerful predictor of delayed cerebral ischaemia, is not in itself a sufficient factor for the development of secondary ischaemia - secondary ischaemia does not occur in patients with a perimesencephalic (non-aneurismal) SAH (74) and it is rare in patients with SAH secondary to intracerebral haematoma or a ruptured arteriovenous malformation. Secondly, in a larger series of patients than in the Boston study, the site of delayed cerebral ischaemia did not correspond with the distribution or even with the side of subarachnoid blood (30). Thirdly, many patients with vasospasm never develop secondary ischaemia. These observations collectively suggest not only the presence of subarachnoid blood *per se,* but also that its combination with other factors determines whether and where secondary ischaemia will develop.

Classically, the clinical symptoms of VSP have consisted of the impairment of consciousness, confusion, disorientation and worsening neurological deficits such as dysphasia and hemiplegia. The clinical diagnosis of VSP is traditionally based on the time of the onset of the deficits, the rate of the development of the deficits (hours), the nature of the deficits and the exclusion of other factors that may cause the gradual deterioration of the patient or focal neurological signs. The diagnosis is therefore not always definitive. It is especially difficult to differentiate symptoms of VSP from other causes that worsen the clinical state in patients with impaired consciousness.

Despite this lack of pathophysiological insight, some progress has been made in the prevention of secondary ischaemia after aneurismal SAH through changes in general medical care (notably, increased fluid intake and the avoidance of antihypertensive drugs) as well as through specific drug treatment. Transcranial Doppler sonography may suggest impending cerebral ischaemia by means of the increased blood flow velocity from arterial narrowing in the middle cerebral artery or in the posterior circulation; however, narrowing in distal branches often escapes detection. Only velocities >120 cm/s or >200 cm/s are reasonably accurate in excluding or predicting delayed ischaemia, but almost 60% of patients are in the intermediate range (92).

Early surgery

Early surgery was introduced after it was considered that the early clipping of the aneurysm not only prevented rebleeding but also allowed for the surgical removal of spasmogenic clot from the basal cisterns (80). The aim of intrathecal fibrinolytic treatment after SAH is to promote the rapid dissolution of blood clot prior to haemolysis and the release of spasmogenic intermediates.

The management of blood pressure

The management of hypertension is a difficult issue in patients with SAH, especially if their blood pressure rises above 200/110 mmHg. Following intracranial haemorrhage, the range between the upper and lower limits of the autoregulation of cerebral blood flow becomes narrower, which makes the perfusion of the brain more dependent on arterial blood pressure. Consequently, the aggressive treatment of surges of blood pressure entails a definite risk of ischaemia in areas with a loss of autoregulation. The rational approach would, therefore, be to advise against treating hypertension following aneurismal rupture. An observational study from the 1980s showed that the rate of rebleeding was lower but that the rate of cerebral infarction was higher than in untreated patients (99). A further observational study suggested that the combined strategy of avoiding antihypertensive medication and increasing fluid intake may decrease the risk of cerebral infarction (24).

Fluid balance and electrolytes

Fluid management in SAH is important for preventing a reduction in plasma volume, which may contribute to the development of cerebral ischaemia. In approximately one-third of patients, plasma volume drops by >10% within the preoperative period, which is significantly associated with a negative sodium balance. Moreover, fluid restriction in patients with hyponatraemia is associated with an increased risk of cerebral ischaemia. Fluid restriction was applied in the past because hyponatraemia was erroneously attributed to water retention via the inappropriate secretion of antidiuretic hormones.

Despite the incomplete evidence, it seems reasonable to prevent hypovolaemia. We favour giving 2.5-3.5L/day of normal saline, unless contraindicated by signs of impending cardiac failure

Calcium antagonists

Initially, the rationale for the use of calcium antagonists in the prevention or treatment of secondary ischaemia was based on the assumption that these drugs reduce the frequency of vasospasm by counteracting the influx of calcium in the vascular smooth muscle cell.

Clinical trials have been undertaken with three types of calcium antagonists: nimodipine, nicardipine and AT877 (of which nimodipine is the most extensively studied and used). A recent systematic review of all randomised controlled trials on calcium antagonists in patients with SAH showed a significant reduction in the frequency of poor outcomes, which resulted from a reduction in the frequency of secondary ischaemia (12). When analysed separately, the nimodipine trials showed a significant reduction in the frequency of poor outcomes, but the nicardipine and AT877 trials did not. While, nicardipine and AT877 significantly reduce the frequency of vasospasm, the nimodipine trials showed only a trend towards the reduction of vasospasm, despite the fact that a larger number of patients were included. In brief, the administration of nimodipine improves outcomes in patients with SAH, but it is uncertain whether nimodipine acts through neuroprotection or through reducing the frequency of vasospasm, or both. Nicardipine and AT877 definitely reduce the frequency of vasospasm, but the effect on the overall outcome remains unproven, which again underlines the weak relation between vasospasm and outcomes.

Since the systemic administration of vasoactive drugs has been associated with significant side effects and insufficient efficacy, the intrathecal administration of nicardipine prolonged-release implants (NPRI) has been developed. At the time of the surgical clipping of the ruptured aneurysm, NPRIs are positioned next to the large cerebral arteries. Several clinical protocols revealed that NPRIs dramatically reduce the incidence and severity of angiographic vasospasm, which was matched by a reduction in cerebral infarction and delayed ischaemic neurologic deficit. On average, the incidence of angiographic vasospasm decreased from approximately 70% to less than 10%. Its efficacy seemed to be dose-dependent and reduced for peripheral vasospasm, and large controlled trials are needed to further confirm these results.

9.7.3 The treatment of delayed cerebral ischaemia

Treatment with hypervolaemia, haemodilution and induced hypertension - the so-called ‘triple H therapy’ - has become widely used, although evidence from clinical trials is still lacking. In a series of patients with progressive neurological deterioration and angiographically confirmed vasospasm, the deficits could be permanently reversed in 43 of 58 cases (41). In 16 patients who had responded to this treatment, the neurological deficits recurred when the blood pressure transiently dropped but again resolved when the pressure increased. The most plausible explanation for these phenomena is a defect of cerebral autoregulation that makes the perfusion of the brain passively dependent on systemic blood pressure. The risks of deliberately increasing the arterial pressure and plasma volume include the rebleeding of an unclipped aneurysm, increased cerebral oedema or haemorrhagic transformation in areas of infarction, myocardial infarction and congestive heart failure.

Recent developments in endovascular treatments have enabled the direct dilatation of constricted cerebral arteries by transluminal angioplasty (48). Since angioplasty is not effective in dilating the distal arteries, superselective intra-arterial papaverine administration was proposed as an alternative or adjunctive method of treatment (40). Subsequently, the use of intra-arterial papaverine has been found to not provide additional benefits when compared with the medical treatment of vasospasm alone (68). Studies comparing the effects of transluminal angioplasty with intra-arterial papaverine have found angioplasty to be superior in reversing DID and in improving CBF (11, 14).

9.8 Other complications of subarachnoid haemorrhage

Hydrocephalus

Acute hydrocephalus is frequently associated with SAH. In the International Cooperative Study on the Timing of Aneurysm Surgery (43), early hydrocephalus was initially noted in 15% of the overall population (3521 patients). During the course of treatment, 18% of the patients received ventricular CSF drainage, 8% had lumbar CSF drainage and 8% received a permanent shunt device. In the surgical series of 835 consecutive patients with aneurismal SAH, Tapaninaho et al. (81) reported a frequency of 35% of early hydrocephalus. 10% of patients finally developed a shunt-dependent hydrocephalus. Severe bleeding into the CSF cisterns or intraventricularly was the basic prognostic factor in the development of chronic hydrocephalus. Shunt-dependent hydrocephalus had a clear adverse effect on outcomes.

Seizures

There was frequency of 4.5% in the occurrence of seizures during the primary hospital course in the International Cooperative Study on the Timing of Aneurysm Surgery (43). In the series of Hernesniemi et al. (27), epilepsy was seen in approximately 14% of patients with acute aneurismal SAH who were treated surgically and who were still alive two months after treatment. Keranen et al. (45) reported an overall frequency of 14% of late epilepsy in surgically treated patients with supratentorial aneurysms; 2.5% in patients with preoperative Hunt&Hess grade I and 33% in patients with Hunt&Hess grade III-V. Ukkola et al. (83) reported a lower frequency of 8% of secondary epilepsy in their series of 183 consecutive patients operated on for ruptured aneurysms. They noted that the development of secondary epilepsy was associated with MCA aneurysms, temporary clipping during surgery, the wrapping technique used to treat the aneurysm and postoperative angiographic VSP.

10. Screening for occult IA

Screening for occult IA is a complex issue, it being of arguable effectiveness and very expensive. Unless a screening test is very highly sensitive and specific, inexpensive, easy to administer and can be delivered in practice to the appropriate population successfully, it is unlikely to produce worthwhile results and is more likely to increase health care costs and stress among the population and healthcare staff alike. Furthermore, unless one can differentiate between diseases that are likely to remain sub-clinical and those that are likely to cause significant symptoms, the treatment of disease following a screening program may have less of an impact than expected on cumulative mortality rates. In the case of intracranial aneurysms, because we cannot yet tell when aneurysms are going to rupture or form *de novo*, it would be difficult to know which to treat, which to leave alone and how often to screen, etc. The stress of being screened is difficult to quantify and probably depends in part upon the seriousness (in the minds of the screened population) of the disease being sought.

As with any screening exercise, multiple factors need to be considered, such as raising anxieties in the patient or the patient's family, confidentiality issues, 'the right not to know', the problems raised by false-positive and false-negative diagnoses, what age to start investigating patients and how often to repeat the investigations, etc. For intracranial aneurysms, many of these factors remain uncertain. There may be financial costs for the

individuals who are screened. If conservative management is advised, knowledge of the presence of an aneurysm may be worrying to the individual concerned (and to his/her family and employer). Bearing all of these factors in mind, ignorance (of the presence or absence of an aneurysm) may actually be the best course of action for an individual at present.

Several groups have recommended screening for intracranial aneurysms in high-risk groups, namely ADPKD patients and those with a strong family history of aneurismal SAH (47, 49, 98). The efficacy of screening for aneurysms depends crucially on certain parameters relating to the natural history of aneurysms, particularly the prevalence and the annual risk of rupture. The analysis of rupture risk is complicated further by the pattern of aneurysm rupture - some aneurysms appear to develop and rupture rapidly whilst others stabilise (38). Screening will tend to detect the low-risk stable type rather than the high-risk aneurysms. The other critical consideration is the safety and effectiveness of treating a silent aneurysm in a healthy patient. A recent study found that the screening of individuals with a family history of L2 first-degree relatives with intracranial aneurysm with magnetic resonance angiography (MRA), followed by digital subtraction angiography (DSA) and surgery, is not an effective way of reducing morbidity and mortality from ruptured intracranial aneurysm (8).

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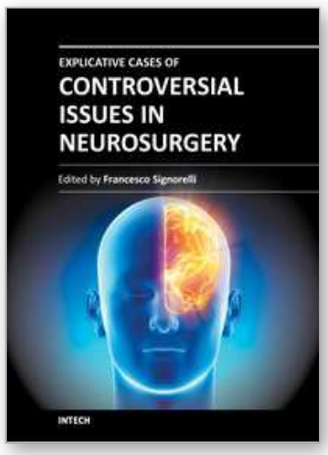
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Explicative Cases of Controversial Issues in Neurosurgery
Edited by Dr. Francesco Signorelli

ISBN 978-953-51-0623-4
Hard cover, 534 pages
Publisher InTech
Published online 23, May, 2012
Published in print edition May, 2012

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How to reference
In order to correctly reference this scholarly work, feel free to copy and paste the following:

Mohammad Jamous, Mohammad Barbarawi and Hytham El Oqaili (2012). Cerebral Aneurysms, Explicative Cases of Controversial Issues in Neurosurgery, Dr. Francesco Signorelli (Ed.), ISBN: 978-953-51-0623-4, InTech, Available from: http://www.intechopen.com/books/explicative-cases-of-controversial-issues-in-neurosurgery/cerebral-aneurysms-and-subarachinoid-hemorrhage

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