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Management of aneurysmal subarachnoid haemorrhage

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Summary

Aneurysmal subarachnoid haemorrhage (aSAH) occurs as a result of rupture of an intracranial aneurysm and affects a younger population compared with ischaemic stroke or intracerebral haemorrhage. Although it makes up only about 5% of all cerebrovascular events, it accounts for over a quarter of the productive life-years lost to stroke. Its surgical and medical treatment represents a multidisciplinary effort. We herein provide an overview of current management options for aSAH.

Key words: *subarachnoid haemorrhage; intracranial aneurysm; cerebral aneurysm; Switzerland*

Introduction

Subarachnoid haemorrhage due to rupture of an intracranial aneurysm (aSAH) has an incidence rate ranging from 5 to 20/100,000 person years [1]. Although aneurysm rupture only represents 5% of all cerebrovascular accidents, aSAH accounts for a quarter of the productive life-years lost to stroke [2]. Hospital stays are long (on average 22 days in patients over 65 years [3]) and the management requires an interdisciplinary team effort including specialised neurointensive care units. In Germany alone, close to half a billion Euro is spent every year on the treatment of aSAH [4]. Despite advances in treatment, there is significant room for improvement since quality of life remains reduced in over 90% of patients 1 year after SAH [5].

Favourable outcome is not only limited by the bleed itself, including the associated early brain injury, but also by conditions such as hydrocephalus, delayed ischaemic neurological deficits and extracranial disease [6]. We herein provide an overview of current management options for aSAH.

Diagnosis and workup at admission

The clinical suspicion of aSAH is based on a history of sudden severe headaches (thunderclap headache), often described as the “worst headache I ever had”. Associated findings include loss of consciousness, neck pain, nausea or vomiting and photophobia. Loss of consciousness, neurological deficits and a decreased level of consciousness are important hallmarks of severe SAH. It is estimated that at least one in eight patients die before reaching hospital [7]. About one in five patients with aSAH recall atypical severe headache in the days before the bleed [8], which is termed sentinel headache and should raise the clinical suspicion for aSAH. Moreover, the presence of a previously diagnosed intracranial aneurysm or a family history of aneurysms or SAH should prompt further investigation. In responsive patients, other factors associated with a higher risk of intracranial aneurysms can be assessed. Such factors include aortic coarctation, polycystic kidney disease, high blood pressure or fibromuscular dysplasia. Genetic disorders associated with an increased risk are Ehlers-Danlos syndrome, Marfan syndrome and other connective tissue diseases.

Computed tomography (CT) of the cranium provides 98% sensitivity to detect SAH within 12 hours of haemorrhage [9]. After the third day, the diagnostic certainty of native head CT decreases to 73% [10]. We recommend that CT-angiography scans (CTA) be routinely performed in order to also rule out the presence of an intracranial aneurysm in the same diagnostic procedure. Magnetic resonance imaging (MRI) reveals subarachnoid blood even after several days in gradient echo sequences (94%–100% sensitivity) and fluid attenuated inversion recovery sequences (81–87% sensitivity) [11], but is unfortunately less available in many emergency departments. In cases where CT imaging reveals no hyperdensity suggestive of haemorrhage and no intracranial aneurysm and MRI is not immediately available, lumbar puncture should be performed. A lumbar puncture is considered to be most sensitive for subarachnoid blood approximately 12 hours after SAH. Cell counts below 10/mm^3 can be considered negative. The presence of xanthochromia as a result of haemolysis and release of bilirubin takes 2 hours to develop, but is a dependable indicator for SAH. The absence of xanthochromia should serve to exclude the diagnosis of SAH only if the lumbar tap was performed at least 12 hours after the suspected haemorrhage [12]. Cerebrospinal fluid (CSF) ferritin levels above the upper limit of 12 ng/ml and a suspicious clinical examination should point to a possible SAH. Additional paraclinical examinations should be taken into account when obtaining CSF within 24 hours of the bleed, since early false negatives have been reported with low ferritin levels [13]. Ferritin rises during the first 1 to 2 weeks after SAH, making it a useful adjunct to diagnosis [14].

Once a diagnosis of spontaneous SAH is ascertained, referral to a neurovascular centre with expertise in treatment of SAH is mandatory.

Blood pressure and heart rate, as well as an electrocardiogram (ECG), are components of most emergency care admission protocols. In the case of abnormal findings or associated cardiopulmonary symptoms, a chest X-ray and cardiac enzymes should be added to the admission panel. Blood pressure is essential for cerebral perfusion, but an unsecured aneurysm is most likely to bleed in the first 48 hours. Therefore, until the ruptured aneurysm is treated, systolic blood pressure can be carefully titrated below 160 mm Hg [7]. Possible agents that have been reported to achieve blood pressure control in patients with SAH in prospective [15] and retrospective [16] series include labetalol, nicardipine and nitroprusside, among others.

Grading scales for aneurysmal subarachnoid haemorrhage

Many grading scales have been devised to categorise patients into groups with diagnostic or prognostic significance. A set of the most commonly used and known scales are recommended by the authors to provide basic and universally readable information on the severity of aSAH and on the patient's status during and after their hospital stay (table 1).

Patient care and monitoring

Basic monitoring

Patients are commonly admitted to an intensive or high-dependency care unit. Depending on the state of consciousness and cardiopulmonary stability, airway control including pulse oxymetry and cardiac surveillance including ECG are initiated. Venous and arterial access facilitates continuous blood pressure control. A urinary catheter can be placed to control fluid levels.

Bed rest

Bed rest is advised until the source of haemorrhage has been secured in order to reduce the risk of rebleeding. In addition, mobilisation has been described to be associated with neurological worsening in the period of vasospasm. It is argued that, with impaired cerebral autoregulation, orthostasis or brusque changes in position may lead to abrupt changes in cerebral blood flow. A retrospective series argued in favour of early mobilisation in a cohort of 25 patients. However, before initiating mobilisation, the investigators ensured the absence of cerebral vasospasm, arterial

Table 1: Grading systems commonly used for patients with aneurysmal subarachnoid haemorrhage

Scale	Description
Fisher Grade (Imaging scale, correlates with risk of vasospasm – highest risk: grade III)	Nominal scale describing four possible distributions of blood in the subarachnoid space on admission CT imaging

Grade	Haemorrhage
I	No visible hematoma
II	Subarachnoid haemorrhage of <1 mm thickness
III	Subarachnoid haemorrhage of >1 mm thickness
IV	Presence of intraventricular or intraparenchymal haematoma

WFNS Score Clinical score – correlates with outcome	A five-point ordinal scale based on a combination of Glasgow Coma Scale and presence of a neurological deficit

Grade	Glasgow Coma Scale	Motor deficit
I	15	–
II	14–13	–
III	14–13	+
IV	12–7	+/–
V	6–3	+/–

Hunt and Hess Score Clinical score – correlates with outcome	Five-point ordinal scale describing the clinical state of a patient considering consciousness and neurological deficit

Score	Motor deficit
1	Asymptomatic, mild headache, slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
3	Drowsiness/confusion, mild focal neurological deficit
4	Stupor, moderate-severe hemiparesis
5	Coma, decerebrate posturing

Modified Rankin scale Grading scale for outcome assessment	A seven-point ordinal scale describing different states of disability from dead to free of symptoms

Grade	Symptoms
0	No symptoms
1	No significant disability. Able to carry out all usual activities despite some symptoms
2	Slight disability without assistance in everyday living, but unable to carry out all previous activities
3	Moderate disability requiring some help. Able to walk without assistance
4	Moderate severe disability without ability to attend own bodily needs
5	Severe disability requiring constant care
6	Dead

CT = computed tomography; WFNS = World Federation of Neurologic surgeons

hypotension or hypertension, elevated intracranial pressure (ICP), coma, hyperventilation and bradycardia [17].

Anticoagulation

Prophylactic anticoagulation is commonly used in hospitalised patients to prevent systemic thrombembolic complications. One prospective randomised study found a beneficial effect of a prophylactic dose of fractionated heparin on the occurrence of vasospasm, ischaemia and chronic hydrocephalus [18], whereas another with similar study parameters failed to show any beneficial effect [19, 20]. We routinely administer prophylactic doses of fractionated heparin to patients once the aneurysm is secured. We also use compression stockings in the setting of SAH, although the evidence supporting their use in stroke patients is controversial [21].

Electrolyte disorders

Several electrolyte disorders can occur with aSAH, including hypernatraemia, hyponatraemia and hypomagnesaemia. Hyponatraemia can occur in up to 20% of patients with aSAH and is attributed to antidiuretic-hormone-related fluid retention or to iatrogenic haemodilution. In addition, or alternatively, a hypovolaemic state associated with hyponatraemia can be caused by atrial natriuretic peptide release, which is termed cerebral salt wasting syndrome [22]. Hyponatraemia is frequently associated with high-grade aSAH and anterior circulation aneurysms, is known to contribute to poor outcome [23] and should therefore warrant treatment.

Glucose management

A recent microdialysis study revealed that high systemic blood glucose levels may be associated with poor neurological grade and low local cerebral glucose levels [24]. Since plasma glucose may be higher than local brain glucose, overly aggressive insulin therapy may induce a cerebral glycopenic metabolic crisis. Therefore, the benefit of tight glycaemic control remains uncertain.

Blood pressure

From admission to treatment of the aneurysm, it may appear reasonable to limit systolic blood pressure to 160 mm Hg to avoid rerupture [25]. Once the aneurysm is secured, hypertension can be tolerated or induced in view of the risk of impaired cerebral autoregulation and delayed vasospasm [26].

Seizures

Epileptic seizures or seizure-like episodes may accompany SAH, particularly in the early period, and may require antiepileptic treatment. Prophylactic seizure-treatment is not recommended [27].

Aneurysm treatment

Rebleeding is the most dangerous complication of aSAH and occurs mostly within hours or days of the initial bleed. Therefore, it is mandatory to perform aneurysm treatment early to prevent rerupture [7].

The main treatment options for aneurysm occlusion remain surgical clipping and endovascular therapy. The seminal International Subarachnoid Aneurysm Trial (ISAT) [28] compared clipping with coiling in a randomised design. The investigators found that surgery resulted in a higher rate of complications with a 30.6% rate of death and disability compared with 23.7% in the coiling group. In contrast, rebleeding rates [29] and aneurysm recurrence rates were higher in the endovascular group [30]. Another randomised trial compared clipping with coiling: the Barrow's Ruptured Aneurysm Trial (BRAT) found that endovascular therapy had 24% unfavourable outcomes (modified Rankin >2) at 1 year, compared with 34% in the surgical group ($p = 0.03$). In the recently published 3-year analysis of the BRAT [31], superiority of coiling failed to reach statistical significance, comparable to the latest 5-year analysis of the ISAT trial [30]. No superiority of the endovascular technique was observed in a smaller previous trial [32]. We recommend that decision making be based on interdisciplinary case-by-case discussions considering aneurysm configuration, location, the patient's condition and resources available for treatment (table 2).

Surgical aneurysm treatment

As a result of the higher rate of recanalisation with endovascular procedures as compared with surgery, surgical treatment can be favoured in young patients with surgically accessible lesions. Good surgical results with better occlusion rates than endovascular techniques can be achieved for aneurysms of the middle cerebral artery [33]. Moreover, presence of significant intraparenchymal haemorrhage (e.g., >50 cm^3), a space-occupying acute subdural haematoma [34] or brain herniation are all arguments for surgery. In emergency cases, hemicraniectomy is an increasingly accepted 2nd tier option for elevated ICP after SAH and can be performed in addition to aneurysm occlusion. The precise configuration of an aneurysm is also of importance for the decision-making process. Even if CTA indicates a surgically treatable lesion, we believe that digital subtraction angiography (DSA) is useful, especially if a hybrid operating room is available. The short time required is usually more than compensated for by the enhanced anatomical detail shown by DSA, which may help characterise the aneurysm(s) and improve decision making. Recent development of hybrid operating suites with intraoperative and postoperative tomography, DSA and flat panel imaging have eliminated the need for time-consuming patient trans-

Table 2: Arguments in favour of surgical or endovascular treatment.

Surgical treatment	Endovascular treatment
Age <40 years	Age >70 years
Middle cerebral artery bifurcation aneurysm	Posterior circulation aneurysms
Large aneurysm neck	Poor clinical grade at admission
Intracerebral or subdural haematoma requiring evacuation	Comorbidities
	Delayed aneurysm treatment during vasospasm period

fers and expanded the possibilities of multimodal management of acute SAH [35, 36].
Since the publication of ISAT, surgical safety has been further improved through increased implementation of neuromonitoring [37], intraoperative video angiography [38] and better bypass techniques [39].

Endovascular aneurysm treatment
Several new coiling devices [40] and stents [41] have greatly enhanced options for endovascular treatment of ruptured intracranial aneurysms and dissections. Despite the high rate of recanalisation, coil occlusion is an important treatment modality for aneurysms with a favourable dome to neck ratio. Owing to the lower complication rate compared with surgery, coiling can be recommended in cases where both treatment modalities are equally feasible. Endovascular treatment of basilar tip aneurysms has a lower complication rate than surgical occlusion and is, in most cases, favoured for lesions of the posterior circulation [42]. Moreover, endovascular techniques are believed to be safer than surgery if aneurysm treatment is performed in the vasospasm period, or for multiple aneurysms. Patients with severe comorbidities or advanced age have additional risk factors for surgery. In these vulnerable patient populations, endovascular treatment may be preferred.

Combined treatment
The controversial "clip versus coil" technique is increasingly being replaced by tailored approaches where "clip and coil" are applied to achieve optimal results at the lowest risk [43]. Suction-decompression techniques have been refined, for example by intraoperative temporary balloon occlusion of a large vessel during aneurysm surgery, with neurosurgeons and interventionalists assisting each other in real time [44].

No aneurysm treatment
In rare cases, patients whose recovery seems unlikely due to catastrophic admission status and imaging, or who are not stable enough (cardiopulmonary or other complications) to undergo treatment, the treatment team may choose not to perform aneurysm occlusion. Such a decision requires a team-based approach with a consensus that the treatment risks outweigh potential benefits. The decision not to treat should not be based on admission grade alone, as survival with mild or moderate disability can reach 50% even in poor grade patients [45].

Management of hydrocephalus

About 15% of patients develop acute hydrocephalus after aSAH [46]. The standard treatment for acute hydrocephalus is CSF drainage by an external ventricular drain (EVD) or lumbar drainage. Patients who are admitted with a Glasgow Coma Score of 9 or lower require continuous intracranial pressure monitoring which can be obtained via an EVD or an intraparenchymal pressure probe. In the case of intraventricular haematoma, thrombolysis can be applied through an EVD in order to dissolve an intraventricular clot, improving CSF dynamics. The 56% relative risk reduction for the development of delayed cerebral vasospasm found in a prospective randomised trial remains an argument in favour of aggressive treatment of ventricle haematoma [47]. Patients require permanent CSF diversion if clinical deterioration or ventriculomegaly is evident after discontinuation of external CSF diversion. A randomised study argued in favour of aggressive weaning from EVD as soon as patients emerge from vasospasm [48]. Definitive surgical CSF diversion, mostly by ventriculoperitoneal shunt, is the method of choice in malresorptive hydrocephalus after aSAH and facilitates early recovery.

Diagnosis, prevention and management of delayed cerebral ischaemia

Delayed ischaemic neurological deficits (DINDs) occur in a significant proportion of patients after aSAH, and CT-proven infarcts occur in 40% [49]. It is suspected that the number of patients suffering cerebral ischaemia after aSAH has been underestimated because a lack of obvious neurological deficits such as motor weakness or aphasia was graded as a good outcome. The prevention, detection and treatment of delayed ischaemia remain unsolved problems: a number of diagnostic bedside methods for continuous monitoring of cerebral blood flow, oxygenation and metabolism are available and are mentioned below [50, 51].

Microdialysis
Microdialysis is a bedside technique employed to monitor cerebral metabolism. A probe is inserted into the brain tissue at risk of ischaemia. Repeated samples for measurement of cerebral metabolites such as glucose, pyruvate, lactate and others are collected. The concentrations of these markers reflect metabolic events in brain tissue with high temporal resolution. An increase in lactate or glutamate or a severe decline in glucose may indicate ischaemia. Another standard measure of metabolism is the ratio of lactate to pyruvate. Glucose utilisation leads to a partial conversion to lactate via pyruvate, depending on the energy level of a cell. An increase of the lactate/pyruvate ratio can reflect low energy levels in the catchment area, which may be due to insufficient perfusion pressure, oxygen or glucose [52]. In patients with SAH, these changes precede the onset of ischaemic deficits and, therefore, provide a helpful tool in the monitoring of these patients. Moreover, increased lactate/pyruvate ratio and glutamate levels are independent predictors of poor outcome 12 months after SAH [53]. The major drawback of the method is that a single probe monitors an area measuring only a few millimetres and provides relevant information only if the probe is located in the region at risk for ischaemia.

Temperature
Fever from any cause occurring in the acute phase of aSAH is known to negatively affect outcome [54]. Furthermore, a retrospective study suggested that active fever avoidance is associated with improved outcome [55]. Since brain temperature may differ significantly from systemic temperature [56], recent efforts have been directed at using sensors, for example in EVD probes, [57] to guide temperature management. A brief overview of temperature monitoring

as part of general neuromonitoring has been provided previously by Springborg et al. [58].

Autoregulation

Experimental and clinical [59, 60] evidence suggests that cerebral vasculature shows a decreased ability to react to altered pressure and carbon dioxide after subarachnoid haemorrhage. Currently, several methods of autoregulation testing exist. Some methods analyse the response of intracranial pressure or blood flow to spontaneous blood pressure fluctuations, and include pressure reactivity indices [61], Doppler reactivity and infrared spectroscopy reactivity [60]. Other methods employ carbon dioxide challenge to actively assess vasoreactivity [62]. Several studies have linked autoregulation impairment with delayed cerebral ischaemia [62, 63].

Intracranial pressure

Intracranial pressure (ICP) monitoring can be performed using an EVD or an intraparenchymal pressure probe and is a prerequisite for all neurointensive care patients with impaired consciousness. EVDs can be kept open or closed depending on the need to release CSF. Monitoring equipment provides mean ICP values which are subtracted from mean arterial blood pressure value to yield cerebral perfusion pressure. The arbitrary rule to keep cerebral perfusion pressure above 60 mm Hg and ICP below 20 mm Hg has been refined in many aspects. Pressure indices calculated online from ICP and blood pressure curves allow for continuous assessment of optimal perfusion pressure [61] with ICP and of cerebral perfusion pressure as treatment targets [64].

Brain tissue oxygen tension

Brain tissue oxygen tension is measured with intraparenchymal devices that are inserted in an analogous manner to intracranial pressure or microdialysis probes. It is strongly correlated with the inspiratory fraction of oxygen and arterial blood pressure. Low brain oxygen tension may indicate that tissue is at risk of ischaemia and, if prolonged, is associated with cerebral infarcts and poor outcome [65, 66].

Cerebral blood flow

One goal of neurointensive care treatment after SAH is the maintenance of regional or global cerebral blood flow (CBF). Intermittent information on global CBF can be obtained using imaging such as perfusion CT or MRI which provide blood flow maps relative to a reference region, i.e., in a presumably healthy hemisphere. Some methods such as xenon-enhanced CT or single-photon emission computed tomography (SPECT), which provide absolute values but are less available, provide only a snapshot and require administration of radionuclides. Continuous monitoring can be carried out by thermodiffusion probes [51], but monitoring is invasive, is limited to a small area, and technical difficulties remain [67]. Combined probes which employ near infrared spectroscopy for regional CBF measurements have shown promising results in preliminary studies [68].

The main drawback of most methods is the inability to monitor CBF directly, and that small sample volumes account for only regional measurements with a potential risk of missing cerebral ischaemia in other vascular territories. Probes combining several parameters such as ICP and CBF are currently under investigation [68]. Recent insights into alterations of CBF in response to waves of cortical depolarisation as precursors of ischaemia may improve the early detection of delayed ischaemic events. However, the development of practicable and widely available monitoring equipment is still in the early stages [69]. Future approaches using advanced bioinformatics may be required for applying multiparameter monitoring to clinical care [70].

The fact that delayed ischaemia after aSAH occurs several days after the bleed in patients who are already in the hospital makes this complication potentially preventable. Most early efforts in preventing these deficits were aimed at treating angiographic cerebral vasospasm, which occurs between days 4 and 14 of SAH. In the most comprehensive and recent randomised controlled trial assessing efficacy of clazosentan, an endothelin receptor antagonist, reduction of angiographic vasospasm did not translate to an improvement in mortality or functional outcome [71]. The calcium-channel blocker nimodipine has a modest impact on delayed cerebral ischaemia and is the only pharmaceutical treatment approved for the treatment of delayed cerebral ischaemia and vasospasm [72]. However, its side effects, particularly systemic hypotension, significantly limit its use in this particular patient population where therapeutic hypertension is mandatory.

Statins

Since statins improve availability of nitric oxide and are known to attenuate endothelial inflammation in the experimental setting, it was hypothesised that delayed cerebral ischaemia may be prevented by the use of statins. A meta-analysis reviewing four randomised trials found that the use of statins led to no measureable benefit [73], although another analysis suggested potential reductions in the incidence of delayed cerebral ischaemia [74]. In any case, patient numbers were small (n = 190) in comparison with trials showing benefits of statins in populations with heart disease [75]. A larger trial is in progress to reassess the potential of statins in aSAH.

Magnesium

Hypomagnesaemia occurs in about one-third of patients after aSAH and was found to be associated with delayed cerebral ischaemia [76]. Experimental evidence of the vasodilatory, anti-inflammatory and antioxidative actions of magnesium led to the hypothesis that it may be a beneficial agent in preventing delayed ischaemia and improving outcome after aSAH. Six phase II and one phase III trial have been published so far [77]. The IMASH study (intravenous magnesium sulphate for aneurysmal subarachnoid haemorrhage) showed no difference in outcome between treatment and placebo groups [78]. Moreover, magnesium is associated with several side effects including hypotension which, in one study, led to termination of infusion in half of the recruited patients [79]. Therefore, there is no high-quality evidence supporting the routine use of magnesium in patients with aSAH.

Local delivery of vasodilators such as nicardipine pellets was found to be of benefit in patients undergoing surgery for aSAH [80]. Although interventional balloon dilation or injection of vasodilatory substances such as calcium-channel blockers or papaverine for symptomatic vasospasm are established procedures, their timing and risks are debatable [81]. Prophylactic angioplasty has not improved outcome and is therefore not recommended [82], whereas balloon angioplasty or chemical intra-arterial delivery of vasodilators are well accepted to prevent ischaemia in cases of impending neurological deterioration.

Extracranial complications

SAH can be associated with severe systemic complications such as neurogenic stress cardiomyopathy or neurogenic pulmonary oedema [83]. Cardiac enzyme elevations or ECG abnormalities were found in eight out of 39 (21%) prospectively studied patients with aSAH [84]. A recent meta-analysis found an association of cardiac complications of SAH with the risk of death, delayed cerebral ischaemia or poor outcome [85]. A retrospective review of 305 consecutive patients revealed that pulmonary complications occur in about 22% of patients with aSAH and include pneumonia and pulmonary oedema [86]. Cardiac and pulmonary complications therefore remain an important limiting factor of patient outcome after aSAH.

Outcome measures

Generally, aSAH affects people of working age. Even slight neurological disturbances can seriously impact their ability to return to their previous occupation. Some authors have argued that the presumed lack of efficacy for drugs reversing delayed cerebral vasospasm may be due to the absence of adequate outcome measures. Current ones such as Glasgow outcome or modified Rankin scales are important because of their ease of use. However, a thorough neurological testing battery, as well as quality-of-life parameters, offer more accurate reflections of clinical status [87].

Follow-up

At discharge and at clinical follow-up the patient is evaluated clinically as well as radiologically. In addition to a neurological exam to assess the evolution of possible neurological sequelae of aSAH, the patient is counselled on factors influencing aneurysm development and rupture. Known modifiable risk factors include arterial hypertension and drug abuse including alcohol, tobacco and cocaine [7].

Radiological follow-up ensures that no recurrence or *de-novo* aneurysm occurs [88]. Current contrast enhanced CT with angiographic reconstruction and magnetic resonance angiography sequences offer sufficient image quality for satisfactory follow-up in most cases [89]. Since CT-angiography may suffer from significant beam-hardening artifacts in selected patients with coiling and clipping, MRI angiography may be the preferred option in these cases [90]. Although there are no universal guidelines for follow-up, suggested empirical intervals for imaging are 6 months, 1 year, 2 years and 5 years after aSAH, as performed within the @neurist network [91]. In case of equivocal findings in noninvasive examinations, or in patients with incompletely obliterated aneurysms for any reason, cerebral arteriography should be performed during follow-up. Moreover, follow-up with angiography should be discussed for patients treated with endovascular stents as treatment success rates and occlusion rates are not yet predictable.

Conclusion

Recent experience and several high-quality studies have improved our ability to monitor and manage this complex neurovascular affection. Future studies should focus on optimised medical management, validation of monitoring tools for cerebral blood flow, prevention of delayed cerebral ischaemia and implementation of more detailed outcome measures.

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