Chapter

125

Aneurysmal Subarachnoid Hemorrhage

PRATAP SANCHETEE, NC BORAH, SURESH SANCHETEE

INTRODUCTION

Presence of blood in subarachnoid space, termed as subarachnoid hemorrhage (SAH), is always a pathological process. The term *'aneurisma'* a Greek word, means 'widening'. Trauma is the most frequent cause of SAH. Nontraumatic SAH accounts for 5 to 10% of all strokes and 5% of all stroke related deaths. The most common etiology of non-traumatic SAH is rupture of a berry aneurysm (80%), followed by rupture of arteriovenous malformation (AVM) (Table 1). Despite advances in diagnostic tools, critical care, and microneurosurgery, rupture of intracranial aneurysm (IA) continues to be a devastating disease. In developing countries, with lack of resources and good public health organization, problems in management are even more severe. Prognosis in patients with SAH depends upon primary hemorrhage and a number of secondary events of which rebleed and vasospasm are most important[1,2]. Many of the secondary insults, if recognized and treated early, are preventable. Thus physician working in mainstream should be able to suspect, diagnose and refer them appropriately. Early clipping or closure of aneurysm has been shown to improve outcome. It must be kept in mind that clinical monitoring is often inadequate in the setting of coma, sedation and neuromuscular paralysis. Neurointensive care of these patients aims at protocol based monitoring and treatment to have a better outcome.

Table 1: Common causes of subarachnoid hemorrhage

- Saccular aneurysm: 66-90% of all aneurysms
- Cerebral arteriovenous malformation (AVM)
- Mycotic aneurysm (bacterial or fungal) - 2.5%
- Angioma
- Primary neoplasm
- Metastatic tumors: Atrial myxoma, choriocarcinoma, undifferentiated carcinoma
- Cortical venous thrombosis
- Blood vessel disorders – Systemic lupus erythematosus (SLE), Moyamoya disease, and granulomatous angiitis
- Systemic bleeding disorders
- Secondary to dissection of blood from an intraparenchymal hematoma
- Congenital and Familial aneurysms - 1.7%
- Aneurysms association with systemic diseases e.g. Fibromuscular dysplasia, Ehlers-Danlos syndrome type IV, Marfan syndrome, Rendu-Osler-Weber syndrome, pseudoxanthoma elasticum, Klippel-Trenaunay-Weber syndrome, coarctation of the aorta, and autosomal dominant polycystic kidney disease

In this review, we have described clinical presentation, merits of various diagnostic and monitoring methods, medical management and other therapeutic options available in the management of intracranial aneurysms.

EPIDEMIOLOGY

Based on angiographic and autopsy studies, incidence of asymptomatic intracranial aneurysms in general population ranges between 0.5% and 5%[3,4]. Annual incidence of rupture in patients with known aneurysm is 1.4% to 2.3%. Rupture risk is related to size and site of IA[5]. Worldwide incidence of SAH is about 6-10 per 100,000 of the population each year, varying according to ethnic and geographic characteristics[6]. Exact data for our country is not available. If left untreated, SAH due to rupture of IA carries a mortality of 45% (32-67%) and 25-33% of survivors will have a substantial morbidity[2,7]. The mean age at presentation

Table 2: Locations of intracranial aneurysms

Location	*Single aneurysm*	*Multiple aneurysms*	*Giant aneurysm*
Internal carotid artery	40%	43%	54%
Anterior cerebral artery complex	32%	21%	10%
Middle cerebral artery	18%	27%	9%
Basilar artery	7%	5%	16%
Vertebral artery	3%	1%	7%

is of 55 years and risk for women is 1.6 times than that of men[8]. According to several epidemiologic studies, 7 to 20% of patients with aneurysmal SAH have a first- or second-degree relative with IA.

Approximately 80 to 85 percent of IA are located in the anterior circulation (Table 2). Common locations of IA are junction of the internal carotid artery and the posterior communicating artery, anterior communicating-artery complex, trifurcation of the middle cerebral artery, junction of vertebral artery and posterior inferior cerebellar artery and bifurcation of the basilar artery. Multiple intracranial aneurysms, usually two or three in number, are found in 20 to 30% of patients.

PATHOPHYSIOLOGY

Our understanding of the causation, growth and reasons for rupture of IA is quite inadequate. Intracranial arteries are susceptible to aneurysm formation as they lack external elastic lamina and adventitia is very thin. Tunica media is either very thin or absent in the saccular, or berry aneurysm. IA can be either congenital or acquired. Commonest morphological type of IA is saccular or berry aneurysm. Other morphological types are fusiform and dissecting aneurysms. Macroscopically, many intracranial aneurysms, especially those that rupture, have an irregular appearance, with one or more daughter sacs and variable wall thickness. The point of rupture is generally in the dome of the aneurysm. Common risk factors associated with development and rupture of IA have been summarized in Table 3. Most of IA rupture in subarachnoid space. However, it can occur at intraventricular, intracerebral, and subdural locations also.

Cerebral vasospasm: Pathophysiology and treatment of cerebral vasospasm consequent to SAH continues to be a major challenge to the clinicians. It occurs in about 40 to 70% of patients and if left untreated, 20% of patients will develop significant global or focal neurological deficit. In general, it occurs between 3 and 15 days after the SAH. It is most likely an inflammatory

Table 3: Risk factors associated with development and rupture of IA

- Age (maximum at 50-59 years), the incidence of hemorrhage increases with age until eighth decade of life
- Gender: F>M in a ratio of 1.6:1.0
- Family history of stroke
- Hypertension including pregnancy-induced hypertension
- Atherosclerosis
- Smoking (more relevant in women)
- A moderate-to-high level of alcohol consumption is an independent risk factor
- Fatty metamorphosis of the liver
- Long-term analgesic use
- Cocaine abuse
- Oral contraceptives
- Size of aneurysm (< 5 mm = 2.5%, 6-10 mm = 41%, 11-15 mm = 87%)
- Location (proximal more likely and intracavernous are least likely to bleed)
- Multiple aneurysms
- Vascular asymmetry in the circle of Willis

reaction in the blood-vessel wall. Extravasated blood and its products in CSF are responsible for development of this dreaded complication. Many spasmogens e.g. oxyhemoglobin, histamine, eicosanoids, endothelin, nitrous oxide and 2-hydroxy-3-methylglutarylcoenzyme have been implicated in the development of vasospasm.

CLINICAL FEATURES

It is important to realize that more than 50% of the patients are misdiagnosed at first visit to their physician. The common incorrect diagnoses are migraine and tension-type headache (Table 4). Failure to obtain accurate history, absence of neurological deficit at admission and incorrect interpretation of imaging study are common reasons for misdiagnosis. Intracranial aneurysms can present in one of the following ways:

1. Asymptomatic: Detected either on investigation of unrelated conditions and while screening for high-risk cases or as co-existence with a ruptured aneurysm (4%). With use of CT and MRI, many more asymptomatic cases will be detected.
2. SAH (89%).
3. Symptoms other than that of SAH (7-20%), e.g. pressure (large or giant IA) on cranial nerves and brain structures, thrombi that embolize distally, and non-specific headache[9].

Unruptured IA are mostly asymptomatic or cause non-specific local pressure symptoms. Most aneurysmal

Table 4: Common mimics and incorrect diagnosis assigned to patients with SAH

- No diagnosis made, or headache of unknown etiology
- Primary headache disorders: migraine, tension, and cluster headaches
- Meningitis and encephalitis
- Acute strokes: hemorrhagic or ischemia
- Hypertensive emergencies
- Pseudotumor cerebri
- Cerebral venous and dural sinus thrombosis
- Pituitary apoplexy
- Intracranial tumor and abscess
- Cervico-cranial artery dissections
- Temporal arthritis
- Systemic infection: flu, gastroenteritis, viral syndrome
- Acute narrow angle closure glaucoma
- Parameningeal infections including sinus-related headache
- Neck problems: cervical disc disease or arthritis
- Psychiatric diagnoses, including malingering and alcohol intoxication
- Trauma related
- Carbon monoxide poisoning

SAH occurs at the time of severe exertion or stress. Prodromal or warning headache from minor blood leakage, referred to as sentinel headache, is present 30-50% of cases but frequently go undetected[10,11]. Classical presentation of SAH is that of a sudden onset of severe headache, not experienced by the patient earlier. It is often referred to as *'first and worst'*. Commonly, it is accompanied with nausea (77%), vomiting, neck pain (35%), photophobia, and visual blurring. Loss of consciousness at onset is evident in about half of the patients. Approximately 10-25% of patients have seizures at onset.

Onset of SAH is often accompanied by sudden increase in blood pressure, which later becomes labile in presence of increased intracranial pressure. Fever is common after third or fourth day of ictus and is independent of associated infection. Signs of meningeal irritation e.g. neck stiffness, low back pain, bilateral leg pain are seen in upto 80% cases. Neck stiffness is caused by the breakdown of blood products within the **subarachnoid** space, and develops several hours after the hemorrhage. It is absent in patients who are in deep coma. Other signs on physical examination include diminished level of consciousness and localizing neurological signs e.g. monocular vision loss (ophthalmic artery aneurysm compressing the ipsilateral optic nerve), third-nerve palsy (posterior communicating artery aneurysm), sixth-nerve palsy (increased intracranial pressure), bilateral lower-extremity weakness or abulia (anterior communicating aneurysm), and combination of hemiparesis and aphasia or visuospatial neglect (middle cerebral-artery aneurysm). Pre-retinal hemorrhage (Terson's syndrome) is seen in upto 5% cases and suggests sudden increase in intracranial pressure. It must be differentiated from commonly seen retinal and subhyaloid hemorrhages. Important neurological complications associated with SAH are vasospasm (46%), hydrocephalus (20%), and rebleeding (7%).

DIAGNOSIS

Computed Tomography (CT)

A non-contrast CT scanning is the first and most important diagnostic study in evaluation for patients with SAH. It is fast, safe, inexpensive, noninvasive, and widely available. In large number of patients, it obviates the need for lumbar puncture (LP). It also helps in differentiating it from other intracranial lesions as mentioned in Table 4. Extravasated blood is identified as high-attenuated areas. It is desirable to have thin cuts through the base of the brain. CT findings are also graded and are useful in prognostication (Table 5). In case of multiple aneurysms, it may be possible to identify the ones that have bled. Sensitivity of CT scan in detecting SAH depends upon amount of blood leaked and duration of illness at the time of study i.e. 93-98% at 12 hours, 75-80% at day three, 70% at day five, 50 percent at day seven and nearly 30 percent at day fourteen.

Magnetic Resonance Imaging (MRI)

The main limitation of MRI is longer acquisition time that is unacceptable in patients with an acute neurological presentation. During acute stage, it is less sensitive than CT study. Modern MRI scanners using

Table 5: Fisher scale for grading SAH on CT scan

Grade	*CT scan*
1	No blood visualized
2	A diffuse deposition or thin layer with all vertical layers of blood (interhemispheric fissure, insular cistern, ambient cistern) less than 1 mm thick
3	Localized clots and / or vertical layers of blood 1 mm or greater in thickness
4	Diffuse or no subarachnoid blood, but with intracerebral or intraventricular clots

gradient echo or FLAIR sequences are as sensitive as CT scan during acute stage[12]. However, the main utility of MRI is during subacute or chronic phase of illness when CT findings may have reverted to normal. Diffusion weighted imaging is valuable in diagnosis of ischemia secondary to vasospasm.

Cerebrospinal Fluid (CSF) Study

Blood from ruptured aneurysm enters the CSF. In next two to four hours, it is hemolysed to produce oxyhemoglobin. It is relevant to appreciate that this process occurs both *in vivo* and *in vitro*. Oxyhemoglobin is then converted to bilirubin by the enzyme hemoxygenase in next 9-15 hours[13]. Thus SAH is characterized by presence of RBCs and xanthochromia in CSF. In the past, CSF study was almost a must in evaluation of suspected SAH. As per current practice, a CT scan is obtained in all patients and LP is done if CT scan findings are negative or equivocal.

Traumatic tap is one of the dilemmas that clinicians face on all occasions. It is a common practice to collect CSF in three to four consecutive tubes and RBC count is done in first and last tube. Diminishing RBC count is a good method for demonstrating traumatic nature of CSF. Crenation of RBCs, and presence of erythrophages lacks sensitivity. Xanthochromia takes few hours to develop and persists for two weeks in all patients with SAH. It must be appreciated that visual inspection alone is not sufficient to evaluate xanthochromia. Spectrophotometer can detect both oxyhemoglobin and bilirubin in CSF and is an excellent test for diagnosing SAH in CT negative patients[14].

Imaging of Aneurysm

Accurate angiographic evaluation of IA is of great importance in planning the management in patients with SAH. Because of its high resolution, digital subtraction angiography (DSA) is the gold standard in imaging cerebral aneurysms. It has a high sensitivity and specificity with false negative results in the range of 5% to 10%. However, it is invasive, expensive, and time-consuming procedure, not available at most of the district centers. Complication rate is upto 1%.

Helical or spiral CT angiography (CTA) is a cheaper noninvasive and faster technique with three-dimensional images. It can be performed along with routine CT study. CTA has a sensitivity of 67 to 100% and specificity of 50 to 100%[15]. Disadvantages include low spatial resolution and poor demonstration of the small aneurysms (< 3-5 mm), internal carotid aneurysms, and posterior circulation aneurysms. Three-dimensional CTA and CT dynamic studies are emerging techniques in this field. CTA must be employed with caution in patients with impaired renal function as a large bolus of contrast material is administered.

MR angiography (MRA) is another diagnostic study that carries no risk. It is one of the best investigations for demonstration of a thrombus in the aneurysmal sac. It can detect lesions as small as 2-3 mm in diameter. However, MRA cannot be performed in patients who have been clipped for earlier SAH. In addition, spatial resolution of MRA is slightly inferior to CTA.

Monitoring in Patients with SAH

Diagnosis of cerebral vasospasm and other complications are far from satisfactory. Unexplained alteration in sensorium and/or appearance of fresh neurological signs and symptoms raises the possibility of vasospasm, hydrocephalus, hyponatremia, subclinical seizures and other systemic complications.

Clinical Monitoring

Clinical grading scales with Hunt and Hess Scale and the World Federation of Neurological Surgeons Scale[16,17] are commonly used to describe neurological condition at admission and considered to be good prognostic predictors (Table 6). The latter is preferable as it uses combination of Glasgow Coma Scale and the presence of focal neurological signs.

Intracranial Pressure (ICP) Monitoring

The main determinant of cerebral blood flow (CBF) is cerebral perfusion pressure (CPP), which in turn is the difference between mean arterial pressure (MAP) and ICP. Aim of ICP monitoring is to maintain adequate CPP. Common causes of raised ICP in SAH are sudden spurt of blood in subarachnoid space, reaction of brain and meninges to extravasated blood, hydrocephalus, diminished compliance of brain and cerebral edema. In critically ill patients, routine procedures such as turning or suctioning can produce deleterious elevations of ICP. ICP monitoring, pressure wave analysis and therapeutic drainage of CSF are possible with indwelling intraventricular catheters.

Transcranial Doppler Ultrasound (TCD)

Through the cranial window major cerebral arteries around circle of Willis are mapped with a low-frequency (2 MHz) pulsed wave probe. It detects elevated flow

Table 6: Clinical grading scales for aneurysmal SAH

Grade	*Hunt and Hess*[16]	*WFNS*[17]
1	Asymptomatic or minimal headache and slight nuchal rigidity	GCS 15, no motor deficit
2	Moderate-to-severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy	GCS 13 to 14, no motor deficit
3	Drowsiness, confusion or mild focal deficit	GCS 13 to 14 with motor deficit
4	Stupor, moderate-to-severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances	GCS 7 to 12, with or without motor deficit
5	Deep coma, decerebrate rigidity, moribund appearance	GCS 3 to 6, with or without motor deficit

velocities in the basal cerebral arteries suggesting vasospasm. TCD is a bedside non-invasive and cheaper procedure that allows serial measurements to identify patients at risk. However, its main limitation is that it is operator dependent.

Continuous Electroencephalography (EEG)

It has a limited role in monitoring patients with SAH. Finer details provided by EEG monitoring are often blurred because of artifacts during recording. Its main utility is in detection of non-convulsive seizures.

Biochemical Markers

Several serum (S-100, neuron-specific enolase, angiogenic factors, intercellular adhesion molecule) and CSF (S-100, neuron-specific enolase, lipid peroxides, cytokines, angiogenic factors, fibrin and fibrinogen degrading products, CSF endothelin-1, nitric oxide metabolites) markers have been evaluated but final verdict is awaited.

TREATMENT

SAH is an emergency and all patients must be stabilised first with maintenance of airway and cardiovascular function. Patients are then transferred to a Neuro ICU and an urgent neurosurgical consultation is obtained. Goals of management at this stage are prevention and treatment of rebleeding and vasospasm, surgical clipping or endovascular occlusion of aneurysm and management of other medical and neurological complications. An outline of treatment has been given in Table 7 and few important points will be discussed.

General Principles

Elevated BP must be normalised with IV agents such as labetalol, esmolol or nitroprusside. Once aneurysm has been clipped, elevation of BP can be permitted as a part of triple 'H' therapy. Seizures occur in approximately 30% of the patients. In view of the devastating effect of a seizure, prophylactic anticonvulsant must be prescribed for few weeks in all cases. Common medical complications that need careful observation are pulmonary edema in 23% (either cardiogenic or neurogenic), and electrolyte disturbances in 28% of patients[18]. Cardiac abnormalities are common after SAH and include ECG changes, cardiac arrhythmias, elevations of cardiac enzymes, and left ventricular dysfunction. Most of these cardiac abnormalities are temporary.

Vasospasm

Patients with SAH are monitored closely with repeated clinical evaluation, CT examination and TCD for development of vasospasm. Two principal approaches to treat vasospasm are hemodynamic augmentation (triple 'H' therapy i.e. hypervolemia, hemodilution and induced hypertension) and endovascular reversal. Central venous catheter monitoring is used to maintain CVP around 8-12 mmHg. Fluids used for hypervolemia are 5% albumin and isotonic crystalloids. Mean arterial pressure is maintained around 110 mmHg with the help of vasopressor drugs. Nimodipine is routinely used as prophylaxis for vasospasm and delayed ischemic damage. It is administered orally in a dose of 60 mg every 4 hourly for 21 days. Intravenous nimodipine is given in a dose 1 mg/hour once vasospasm sets in. Attempts have been made to reverse vasospasm with external ventricular drainage and aggressive irrigation of the basal cisterns. Transluminal angioplasty and endovascular or intra-arterial infusion of vasodilator substances (papaverine) or of calcium channel blockers (verapamil, nimodipine or nicardipine) has been tried. Recently, cisternal washing, free radical scavengers, nitric oxide donors, hypothermia, statins and estrogen have been used to prevent vasospasm with encouraging results[19].

Hydrocephalus

Hydrocephalus develops in about 15-20% of patients who have an aneurysmal SAH. Diagnosis is confirmed

Table 7: Management of subarachnoid hemorrhage

Lifesaving measures	• Monitor closely in neurology intensive care • Treatment for coma or decreased mental status • Cardiac and respiratory stabilization
General and symptomatic	• Strict bed rest and avoidance of straining • Calm environment with least distractions • Stool softeners to prevent straining during bowel movements • Nutrition • Abnormalities of coagulation must be identified and treated. • Analgesic to relieve pain and to reduce ICP Acetaminophen, oxycodone, fentanyl, morphine, etc. • Antiemetics for nausea or vomiting • Sedatives: short-acting agents to be used, e.g. benzodiazepines • GI prophylaxis with H_2 receptor antagonist • Deep venous system prophylaxis with physiotherapy, elastic stockings, heparin • Maintain blood glucose: Maintain level at 80-120 mg/dl, if requires use insulin • Maintain body temperature • Fluid balance: Adequate hydration must be maintained.
Specific	• Targeted at aneurysm • Neuroprotection
Complications	• Raised intracranial pressure: Mannitol, diuretics • Rebleed • Hydrocephalus • Vasospasm • Seizures: Phenytoin and other drugs to prevent seizures • Hyponatremia to be monitored closely • Cardiorespiratory compromise
Long-term	• Rehabilitation • Treatment of depression and cognitive impairment

on CT scan. Symptomatic cases are treated surgically with ventriculostomy or CSF shunting[20].

Rebleeding

Antifibrinolytic agents competitively inhibit plasminogen activation and have been reported to reduce the incidence of rebleeding. However, their use is associated with increase risk of cerebral and systemic thrombosis. Epsilon aminocaproic acid (EACA) is given as 5 gm IV bolus followed by infusion at the rate of 1.5 g per hour. It can be used as a short-term measure before definite treatment of aneurysm.

Surgical Treatment

Whether a given aneurysm should be observed, treated surgically, or managed endovascular, remains controversial. Microsurgical placement of a clip across the neck and endovascular coiling are two main therapeutic options for securing a ruptured aneurysm. Surgical clipping has been assessed for a long period and has high acceptability with neurosurgeons. Aneurysms that are not amenable to clipping can be dealt with other sophisticated techniques, such as vascular bypass grafting, hypothermic cardiac arrest, and surgical or endovascular occlusion of the proximal vessel. Current consensus is for early surgery (within 72 hours after the ictus) because of high risk of rebleed in first week and its effectiveness in preventing vasospasm[21]. Late surgery is complicated by presence of vasospasm and brain edema around the clot.

Endovascular Therapy

In selected cases, endovascular treatment is emerging as a promising alternative to surgical clipping. The goal of endovascular coiling is to thrombose the aneurysmal sac with transarterial placement of small and soft platinum or titanium coils[22]. Elderly patients, patients in poor medical condition, aneurysms at difficult locations and those associated with large intracerebral hematoma are better treated by an endovascular approach. Aneurysms with wide necks are less amenable to endovascular treatment than those with narrow necks. Though this procedure carries a lower risk, its long-term effectiveness is yet to be proved.

Surgery for Unruptured Intracranial Aneurysm

Choosing surgery for patients with an unruptured intracranial aneurysm is a difficult therapeutic decision. One must weigh the risk of intracranial hemorrhage against the risks associated with surgery. Co-morbid medical conditions, severe cardiac, pulmonary, or renal disease or cancer weighs against prophylactic surgery. Patient's feelings, experiences, biases, and personal preferences are equally important. Size and site of unruptured aneurysms matters a lot in arriving at a decision.

PROGNOSIS

Nearly 15-20% patients die before admission, and another 40% die during the first month. More than one-third of survivors have major neurological deficits.

Common reasons for death are vasospasm (32%), direct effect of bleed (25%), rebleeding (18%), brain compression and shift (5%), hydrocephalus (4%), myocardial ischemia/ arrhythmia, and hyponatremia. The major prognostic factors associated with poor outcome are level of consciousness at admission (Table 6) and amount of blood shown at first CT study (Table 5). The outcome of surgery depended heavily on age. The surgery-related morbidity and mortality at one year is 6.5% for those < 45 years, 14.4% for 45-64 years, and 32% > 64 years. Other prognostic factors related to surgery are experience of surgeon, team and centre; size and location of the aneurysm; and morphologic features of the aneurysm.

CONCLUSION

Non-traumatic subarachnoid hemorrhage (SAH) accounts for 5-10% of all strokes and it has an incidence of 10.5/100000 persons/year. The most common cause of non-traumatic SAH is rupture of an intracranial aneurysm. Other causes include vascular malformations, tumors, and infection. Intensive critical care support and prompt diagnosis with high-resolution CT remain key aspects of good patient management. In patients presenting with a suspected non-traumatic SAH, CT within 12 hours will reliably show 98% of SAH. In patients who present after 12 hours with a negative CT scan, formal CSF spectrophotometry will detect SAH for the next two weeks with a reliability of 96%. The natural history of untreated aneurysmal subarachnoid hemorrhage carries a dismal prognosis. Case fatalities range between 32% and 67%. Today, with improved techniques for coil embolization and clip occlusion of aneurysms, a great majority of patients are successfully treated through the acute phase after SAH. Given the complexity of evaluation, treatment and management of aneurysmal subarachnoid hemorrhage, a team approach to the problem has proved useful.

We are still to answer many questions related to this disease, e.g. epidemiological pattern, reasons for rupture of aneurysm, reasons and best management for vasospasm and rebleed, role of neuroprotection, intracisternal application of thrombolytic therapy, use of biologically active coils and stents for endovascular treatment.

KEY POINTS

- Early diagnosis of SAH is crucial to good results.
- All patients presenting with sudden, severe headache, warrant further investigation
- A CT scan within 12 hours of presentation is 98% sensitive for SAH
- Lumbar puncture must be performed more than 12 hours after presentation.
- Visual analysis of CSF is not acceptable and spectrophotometery detection of bilirubin in CSF is indicative of SAH
- Prompt angiography identifies the cause of SAH.
- Early obliteration of the aneurysm prevents rebleed.
- Early recognition and management of cerebral vasospasm minimizes stroke.

REFERENCES

1. Fogelholm R, Hernesniemi J, Vapalahti M. Impact of early surgery on outcome after aneurysmal subarachnoid hemorrhage. A population-based study. Stroke 1993; 24: 1649-54.
2. Hop JW, Rinkel GJ, Algra A, van Gijn J. Case-fatality rates and functional outcome after subarachnoid hemorrhage: a systematic review. Stroke 1997; 28: 660-4.
3. McCormick WF, Nofzinger JD. Saccular intracranial aneurysms: an autopsy study. J Neurosurg 1965; 22:155.
4. Inagawa T, Hirano A. Autopsy study of unruptured incidental intracranial aneurysms. Surg Neurol 1990; 34:361-5.
5. Juvela S, Porras M, Heiskanen O. Natural history of unruptured intracranial aneurysms: A long-term follow-up study. J Neurosurg 1993; 79:174-82.
6. Linn FH, Rinkel GJ, Algra A, van Gijn J. Incidence of subarachnoid hemorrhage: role of region, year, and rate of computed tomography: a meta-analysis. Stroke 1996; 27: 625-9.
7. Pakarinen S. Incidence, aetiology, and prognosis of primary subarachnoid haemorrhage. A study based on 589 cases diagnosed in a defined urban population during a defined period. Acta Neurol Scand 1967;43 (suppl 29):1-28.
8. Lindsay KW, Teasdale GM, Knill-Jones RP. Observer variability in assessing the clinical features of subarachnoid hemorrhage. J Neurosurg 1983; 58:57-62.
9. Fisher M, Davidson RI, Marcus EM. Transient focal cerebral ischemia as a presenting manifestation of unruptured cerebral aneurysms. Ann Neurol 1980; 8:367-72.
10. Bassi P, Bandera R, Loiero M, et al. Warning signs in subarachnoid haemorrhage: a cooperative study. Acta Neurol Scand 1991; 84:277-81.
11. Ostergaard JR. Warning leak in subarachnoid haemorrhage. BMJ 1990; 301:190.
12. Mitchell P, Wilkinson ID, Hoggard N, et al. Detection of subarachnoid haemorrhage with magnetic resonance imaging. J Neurol Neurosurg Psychiat 2001; 70:205-11.
13. Barrows L J, Hunter FT. The nature and clinical significance of pigments in the cerebrospinal fluid. Brain 1955; 78:59-80.
14. Cruickshank AM. CSF spectrophotometry in the diagnosis of subarachnoid haemorrhage. J Clin Pathol 2001; 54:827-30.

15. Preda L, Gaetani P, Baena R, et al. Spiral CT angiography and surgical correlations in the evaluation of intracranial aneurysms. Eur J Radiol 1998; 8:739-45.
16. Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg 1968; 28:14-20.
17. Report of World Federation of Neurological Surgeons committee on a universal subarachnoid hemorrhage grading scale. J Neurosurg 1988; 68:985-6.
18. Suarez JI, Tarr RW, Selman WR. Aneurysmal subarachnoid hemorrhage. NEJM 2006; 354:387-96.
19. Lynch JR, Wang H, McGirt MJ, et al. Simvastatin reduces vasospasm after aneurysmal subarachnoid hemorrhage: results of a pilot randomized clinical trial. Stroke 2005;36:2024-6.
20. Brisman JL, Song JK, Newell DW. Cerebral aneurysms; NEJM 2006; 355:928-39.
21. Suzuki J, Yoshimoto T. Early operation for the ruptured intracranial aneurysm-especially the cases operated within 48 hours after the last subarachnoid hemorrhage. Neurol Surg 1976; 4:135-41.
22. Molyneux A, Kerr R, Stratton I, et al. International subarachnoid aneurysm trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. Lancet 2002; 360:1267-74.