Review Article

Perioperative Management of Intracranial Aneurysms

Gopalakrishnan Raman*, Mohanarangam T**

*Consultant Neuroanaesthetist, **Consultant Anaesthetist, Chettinad Super Speciality Hospital, Kelambakkam, Chennai

Dr.Gopalakrishnan Raman did his undergraduation from Government Medical College, Calicut. He completed his post graduation [D.A, MD (Anaesth) and DNB (Anaesth)] from the King Edward VII Memorial Hospital (KEM), Mumbai. He did his post-doctoral certification in Neuroanaesthesia from the prestigious Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram. He has had extensive experience in Neuroanaesthesia, both in India and the United Kingdom, over a period of 20 years. He is presently working as a Consultant Neuronaesthetist in Chettinad Super Speciality Hospital. He was conferred the Membership of the National Academy of Medical Sciences (MNAMS) in 2013.

Corresponding author - Dr.Gopalakrishnan Raman (gopalraman1000@hotmail.com)

Abstract

A ruptured intra-cranial aneurysm and the ensuing subarachnoid haemorrhage have a dramatic clinical presentation and are associated with high morbidity and mortality. The diagnosis and perioperative management of this condition extending from the intensive care unit to the neurosurgical OT or catheterization laboratory (for surgical clipping or endovascular coiling) are discussed in detail. Recent recommendations in the anaesthetic management of this condition are also covered in this review article.

Key Words: Aneurysmal SAH, Anaesthesia, Surgical clipping, Endovascular coiling.

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Epidemiology

Intracranial aneurysms affect 5-10% of the general population[1]. The rate of rupture of an intracranial aneurysm is 0.05% to 6% per year. Smoking, hypertension, heavy alcohol intake and sympathomimetic drug abuse are risk factors for aneurysm rupture. Aneurysmal rupture may occur during exertion although in most cases there is no associated relationship. They are 11 times more likely to rupture in patients with a history of previous subarachnoid haemorrhage(SAH) than in those who do not. Around 20% of patients have more than one aneurysm. There is a male preponderance in the age group below 40 years but females are more affected than males in the age group above 40 years with a ratio of 3:2[1]. The main mode of presentation of a ruptured intracranial aneurysm is subarachnoid haemorrhage (SAH).

The overall incidence of SAH is around 9.0 – 10.5 / 100000 population / year[2]. Finland and Japan have the highest incidence of SAH in the world[2,3,4]. The incidence in these countries varies from 15.1 – 29.8 / 100000 population / year. The incidence in India is reported to be around 3 – 4 /100000 population / year[5]. The incidence of SAH is 1.24 – 1.60 times more common in women compared to men. It is higher in Hispanics and blacks compared to caucasians. The incidence of SAH is similar in males and females under 50 years, but is higher in females over 50 years of age.

Types of Aneurysms:

- Saccular aneurysms are usually less than 2.5 cm in diameter. They are the most common.
- Fusiform aneurysms are usually associated with severe atherosclerosis.
- Dissecting aneurysms are due to an endothelial tear
- Traumatic aneurysms usually follow traumatic brain injury
- Mycotic aneurysms are caused by vessel wall infection due to haematogenous spread
- Giant aneurysms are those over 2.5cm in diameter.

Genetics

Connective tissue disorders which have been associated with cerebral aneurysms include polycystic kidney disease, Ehler-Danlos syndrome type IV, Marfans syndrome, Neurofibromatosis type I, Pseudoxanthoma elasticum and Alpha-1 antitrypsin deficiency. There is defective synthesis of collagen and elastin in the arterial walls in these patients.

Aetiology of SAH

Traumatic brain injury is the most common cause of SAH. Aneurysmal rupture constitutes 70 – 75 % of all non-traumatic causes of SAH, as listed in Table 1. All the rare causes together constitute less than 5 %.

Table – 1: Non-Traumatic causes of SAH	
Vascular – Ruptured Aneurysms	70 – 75 %
AV Malformations	5 %
Haemorrhage from Tumours	Rare
Pituitary Apoplexy	Rare
Vasculopathy–Collagen vascular disease	Rare
Amyloid angiopathy	Rare
Arterial dissection	Rare
Haematological – Anticoagulant Treatment	Rare
Leukaemia	Rare
Coagulopathy (Liver disease)	Rare
Drugs – Ephedrine, Cocaine, Amphetamine	Rare
Undefined	15 %

Clinical features

Ruptured aneurysms present as subarachnoid haemorrhage. Sentinel symptoms like headache, retro-bulbar pain and nuchal pain and are seen in 60-70% of patients before rupture.Some patients however have a minor bleed before the major haemorrhage. This is associated with mild headache, nausea and vomiting. These sentinel bleeds occur in 40 – 50 % of cases of SAH and are usually seen 2 – 8 weeks prior to the major haemorrhage[6,7]. Only 49% of unruptured aneurysms were symptomatic[8]. Neurological symptoms of unruptured aneurysms are given in table 2.

Table 2 : Neurological symptoms of unruptured aneurysms	
Acute Symptoms (17 %)	
Headache	~ 37 %
Cerebral ischaemia	~37 %
Seizures	~18 %
Cranial neuropathies	~12 %
Chronic Symptoms (32 %)	
Headache	51 %
Visual defects	29 %
Weakness	11 %
Facial pain	9 %

The severity of symptoms in SAH is related to the severity of the bleed. The most common and classical symptom of aneurysmal SAH is a severe headache of sudden onset (Thunderclap headache). A period of loss of consciousness or epileptic seizures may immediately follow. Nausea and vomiting occur commonly. The headache is acute in onset, peaks rapidly and persists for days in spite of treatment. In some patients, neck pain may predominate over other symptoms. Fifty percent of patients suffering from SAH have nuchal rigidity due to meningeal irritation by the blood. It develops 3 – 12 hours after the SAH. The Kernigs sign may also be positive. Examination of the fundus may reveal papilloedema as well as retinal, pre-retinal or subhyaloid haemorrhages. Visual symptoms may occur due to SAH from ACOM aneurysms while rupture of PCOM aneurysms cause third cranial nerve palsy. Diabetes insipidus and hypothalamic dysfunction may be seen in patients with ruptured ACOM aneurysms. A reactive systemic hypertension may develop even in previously normotensive individuals and the BP may take several days to return to normal levels. A severe fluctuating pyrexia may reflect ischaemic hypothalamic damage. SAH is a true medical emergency. 10 – 15 % of patients suffering from SAH die before reaching the hospital. About 40 % die within 30 days[9]. Of all patients suffering from SAH, roughly one third die acutely, one third survive with significant neurological deficits and one third recover well[10].

Clinical grading of SAH

Botterell in 1956 introduced a system for grading patients after SAH to facilitate assessment of surgical risk, prediction of outcome and prompt evaluation of the patient's condition. This system was modified in 1968 by Hunt and Hess to include a provision for the effect of serious systemic illness[11] (Table-3). When there is any associated serious systemic illness like hypertension, diabetes, COPD or angiographically-demonstrated vasospasm, the patient is placed in the next less favourable Hunt and Hess grade.

Table 3: Hunt and Hess clinical grading of SAH		
Grade	Description	Mortality
Grade 0	Unruptured aneurysm	0
Grade 1	Asymptomatic or minimal headache with normal neurological examination	2 %
Grade 2	Moderate to severe headache, nuchal rigidity, no other neurological deficit other than cranial nerve palsy	5 %
Grade 3	Lethargy, confusion or mild focal deficit	15-20 %
Grade 4	Stupor, moderate to severe hemiparesis, early decerebrate rigidity, vegetative disturbances	30-40 %
Grade 5	Deep coma, decerebrate rigidity, moribund state	50-80 %

The World Federation of Neurological Surgeons (WFNS)grading scale, based on the GCS was introduced in 1988, demonstrated that the preoperative level of consciousness correlated most directly with outcome[12] (Table-4).

Table 4: WFNS Grading Scale		
Clinical Grade	**GCS Score**	**Motor Deficit**
I	15	Absent
II	14-13	Absent
III	14-13	Present
IV	12-7	Present or absent
V	6-3	Present or absent

Investigative approach to aneurysmal SAH

CT Scan – Any patient suffering from a severe headache of acute onset for the first time in their life should undergo an immediate CT scan of the brain. A subarachnoid bleed will be evident in 95-98 % of patients in the first 24 hours post bleed[13]. This declines to 58 % by the fifth day and to 50 % by the seventh day[14].The location of SAH may give information about the probable aneurysm site (Table-5).

Table – 5: Correlation of CT scan location of SAH and site of aneurysm	
Location of SAH on CT Scan	**Probable site of ruptured aneurysm**
Anterior Interhemispheric fissure	ACA aneurysm
Distal Interhemispheric fissure	Distal ACA aneurysm
Unilateral sylvian fissure	MCA aneurysm
Suprasellar cisterns	ICA aneurysm
Interpeduncular cistern	Basilar top aneurysm
Prepontine/Cerebellopontine cisterns	Basilar trunk/Vertebrobasilar aneurysm
Isolated intraventricular bleed	Distal PICA aneurysm, Basilar top aneurysm, Post.superiorly directed ACOM aneurysm
Frontal ICH	A1, ACOM, M1, IC bifurcation aneurysm
Temporal ICH	MCA aneurysm

Fischer et al have graded subarachnoid haemorrhage based on the CT scan appearance (Table-6) and this can be used to predict the outcome of patients with SAH[15]. The amount of blood in the subarachnoid space directly correlates with the risk of vasospasm with Fischer grade 3 SAH carrying the highest risk.

Table 6: Fischer Grading of SAH based on CT scan appearance	
Grade 1	No detectable blood on CT scan
Grade 2	Diffuse thin SAH (vertical layers < 1 mm thickness)
Grade 3	Localised clot / thick SAH (vertical layers > 1 mm thickness)
Grade 4	Intraventricular / Intracerebral clot with diffuse or no SAH

Lumbar Puncture

CT scan is often considered the investigation of first choice since it has a high sensitivity for detecting SAH. A lumbar puncture is indicated when a clinical diagnosis of SAH has been made and CT scan turns out to be negative. It is usually done 6 – 12 hours after SAH to allow formation of haemoglobin metabolites. CSF that is uniformly blood stained when collected in four separate tubes is highly suggestive of SAH. If the lumbar puncture is done a few days after SAH, CSF analysis will reveal xanthochromia, RBC's, WBC's and bilirubin. A sentinel bleed can be ruled out and a favourable outcome expected if both CT scan and CSF results are negative after a history of severe headache of sudden onset.

Cerebral Angiography

Four vessel catheter angiography is the gold standard for detection of aneurysms. It gives the exact location, size and configuration of the aneurysm with good visualisation of the neck. It establishes the cause of SAH in 85 % of cases. Cerebral angiography does not delineate thrombosed aneurysms. The procedure carries a complication rate of 7 %, the majority being puncture site haematomas.

CT Angiography

This is a rapid, non-invasive technique for the detection of aneurysms. The procedure has a sensitivity of 95-100% for detecting aneurysms of over 5 mm in size. The sensitivity is significantly lesser in detecting aneurysms of less than 5 mm in size[16]. CT angiography is less effective in detecting mild to moderate vasospasm and is not useful in patients whose aneurysms have already been clipped or coiled because of artefacts. CT angiography in combination with catheter angiography can demonstrate intraluminal thrombosis in aneurysms, calcification of the aneurysmal wall and its relationship to intracerebral haemorrhage and bony landmarks.

MRI Scan / MR Angiography

MR angiography is useful in demonstrating the size and shape of the aneurysm and its relationship to important structures, the amount of clot inside the aneurysmal lumen and any recent or old haematomas. MR angiography has a sensitivity of 85-100 % in detecting aneurysms over 5 mm in size. This falls to 56 % for aneurysms smaller than 5 mm[17]. MR angiography does not require ionizing radiation or radiocontrast and hence is safe to use in pregnant women.

MRI scans performed several days after the bleed may provide greater sensitivity than CT scans in detecting small areas of subarachnoid clot and help determine the particular lesion responsible in patients with multiple aneurysms. MRI scans are also useful in the detection of arteriovenous malformations of the brain and spine resulting in SAH.

Management of SAH

Mortality following SAH is high even in those patients reaching hospitals with adequate facilities to manage these cases. Approximately 10 % die before reaching the hospital and 25 % die in the first 24 hours[18]. The main causes of death are re-bleeding, vasospasm induced ischaemia, raised ICP, acute hydrocephalus, intra-cerebral haemorrhage, myocardial ischaemia, cardiac arrhythmias, pulmonary oedema and respiratory failure. The Hunt & Hess or WFNS score at admission is the most important predictor of outcome and mortality.

Patients with SAH have to be managed in intensive care units by trained nurses in hospitals with adequate expertise to treat these cases. The ICU's should be dimly lit since most of these patients suffer from photophobia. Complete bed rest is an essential part of the management of these cases. Sedatives may be given to control agitation and analgesics administered for treating headache. Laxatives should be prescribed to avoid straining at stools. Pneumatic compression devices and TED stockings may be used for DVT prophylaxis.

SAH adult patients require 3000 to 4000 ml of fluids to maintain normovolaemia. In addition to hypovolaemia, these patients also demonstrate hyponatremia, hypokalemia, hypocalcemia and hypomagnesemia. Hypomagnesemia may be associated with vasospasm and poor outcome. A large placebo controlled trial of treatment with a continuous infusion of magnesium sulphate suggested that the administration of magnesium may reduce delayed cerebral ischaemia by 34 % and decrease poor outcome at 3 months by 23 %[19]. Hyponatremia, which occurs due to release of atrial natriuretic peptide from the hypothalamus, should be corrected with normal or hypertonic saline.

Hypothalamic disturbances following SAH result in elevated levels of catecholamines and renin which causes arterial hypertension. Moderate hypertension with a MAP of less than 120 mm Hg need not be treated. Control of pain and agitation may be sufficient to lower the blood pressure in these patients.

Following SAH, responsiveness of the cerebral vasculature to changes in CO_2 tension is usually preserved. However SAH interferes with cerebral autoregulation. The main CNS complications following SAH are seizures and hydrocephalus.

The incidence of seizures in patients with SAH is 3 – 26% and usually occurs during the first 24 hours. The associated rise in blood pressure may precipitate re-bleeding from the aneurysm. Patients who have lobar intra-cerebral haemorrhage or a thick layer of cisternal blood have the highest risk of developing seizures. Other risk factors are given in Table 7.

Table-7 :Risk factors for developing seizures in SAH
Re-bleeding
Vasospasm and delayed ischaemic deficit
MCA aneurysms
Subdural Haemorrhage
Intraparenchymal haematoma
Cerebral infarction
Medical hypertensive disease
Chronic neurological impairment

What is more disturbing is the development of non-convulsive seizures in stuporous patients which can be detected only by EEG monitoring. Hence it is common practice to provide seizure prophylaxis in these patients with fosphenytoin or levetiracetam, since phenytoin administration is associated with a worsened cognitive function at 3 months post SAH.

Acute hydrocephalus is seen in 20 – 30 % of patients with SAH and usually occurs within 72 hours of the bleed. A communicating hydrocephalus is due to blood clot within the basal cisterns and obstruction of the arachnoid villi. An obstructive hydrocephalus is due to a blood clot within the ventricular system. Associations of acute hydrocephalus are given in Table 8.

Table – 8: Associations of acute hydrocephalus in SAH
Early onset of lethargy and coma
Lower WFNS grade
Higher Fischer grade
Thicker subarachnoid clot
Intraventricular haemorrhage
Alcoholism
Female gender / Elderly
Larger aneurysms
Medical Hypertensive disease
Meningitis

Symptoms of acute hydrocephalus include headache, impaired consciousness, dementia, incontinence and gait ataxia. Subacute hydrocephalus occurs a few days after the bleed and is characterised by a gradual decline in the sensorium. Delayed hydrocephalus occurs weeks to months after the bleed and the main manifestation is subacute dementia. Acute hydrocephalus resolves spontaneously in most patients. However some may require temporary CSF diversion (EVD insertion). There is however a risk of re-bleeding on sudden decompression of the ventricle. Predictors of the requirement of shunting include a lower WFNS grade, intra-ventricular haemorrhage and re-bleeding. Delayed hydrocephalus develops in 25 % of SAH survivors and is treated by permanent shunting.

Raised ICP following SAH causes a depression in the sensorium. The causes include cerebral oedema, intra-cerebral haematoma, intra-ventricular haematoma and hydrocephalus. Raised ICP is managed by head end elevation, fluid balance, antioedema measures, correction of hyponatremia, prevention of hypoventilation and hypocapnia and treatment of fever and agitation. Specific measures include evacuation of intra-cerebral haematoma and temporary or permanent shunting for hydrocephalus.

Re-bleeding after the initial SAH is one of the main causes of neurological deterioration in these cases. It is usually more severe than the original bleed. The size of the re-bleed haematoma is the most important factor determining outcome. Other indicators of a poor prognosis are a marked midline shift, a large subdural haematoma, intra-cerebral haematoma and intra-ventricular haemorrhage. Re-bleeding is characterised by deterioration in the level of consciousness, development of focal neurological deficits like hemiplegia and aphasia, hypertension, bradycardia, irregular breathing and presence of haemorrhages on fundoscopic examination. Factors predisposing to re-bleeding include the female gender, elderly patients, systemic hypertension, abnormal clotting parameters, posterior circulation aneurysms, increased time to treatment, intra-cerebral and intra-ventricular haematomas and large volume of blood in the sub-arachnoid space after the initial bleed. Approximately 20 – 30 % of SAH patients re-bleed within the first 30 days and upto 50 % by 6 months. The definitive prevention of re-bleeding is by early aneurysm clipping or endovascular coiling (within 24 – 48 hours of SAH). Systolic hypertension and interventions that cause an increase in the transmural pressure gradient (TMPG) should be avoided. These patients are usually on nimodipine for vasospasm and this has to be taken into consideration when adding an infusion of labetalol or esmolol for BP control. Hypotension, which can lead to cerebral ischaemia in the presence of vasospasm, should be avoided at all costs.

Antifibrinolytic drugs like tranexamic acid reduce the rate of re-bleeding following SAH. There is however an increase in the incidence of cerebral ischaemia and infarction. Currently it is recommended to administer a short course of antifibrinolytic therapy in combination with early definitive treatment of the aneurysm. Management of re-bleeding after SAH is designed to maintain CPP, limit ICP, decrease intracranial volume, control systemic BP, reduce TMPG across the wall of the aneurysm and maintain cerebral oxygen delivery.

Vasospasm is a dreaded complication following SAH and mainly affects the larger conducting arteries in the sub-arachnoid space. It is induced by the breakdown products of red blood cells in clots. It causes ischaemic deficits and cerebral infarction and is a major cause of disability and death. The incidence of vasospasm peaks between the 4th and 9th day after SAH and subsequently decreases. Vasospasm is suspected by the appearance of new focal neurological deficits and progressive impairment in the level of consciousness and is usually seen four days after the initial SAH and which cannot be linked to a structural or metabolic cause.

Transcranial Doppler studies reveal blood flow velocities > 120 cm / sec which correlates with angiographically demonstrated vasospasm. A rapid

increase in blood flow velocity of 50 cm / sec from baseline in 24 hours also indicates development of vasospasm. A peak flow velocity of 140 – 200 cm / sec indicates moderate vasospasm while values > 200 cm / sec indicates severe vasospasm. Cerebral angiography is the most reliable investigation for diagnosing and evaluating vasospasm. Angiographically severe vasospasm is defined as a reduction in the diameter of the arterial lumen by > 50 %. Xenon enhanced CT demonstrates decreases in regional blood flow in patients with vasospasm. Jugular bulb oximetry detects an increase in AV DO2 as early as 24 hours before vasospasm sets in and a decreasing value reflects the patient's response to treatment. Positron Emission Tomography(PET) may be used to measure CBF and shows a fall in CMRO2 after SAH. Single Photon Emission Computed Tomography (SPECT) may reveal regions of hypoperfusion associated with vasospasm.

The definitive treatment of vasospasm involves early clipping or coiling of the aneurysm. If the surgical modality is used, fresh clot may be removed by irrigation and suction. Recombinant TPA may be instilled to remove residual clot keeping in mind that normal clot may also get lysed causing re-bleeding.

The prophylactic use of nimodipine is universally accepted in the prevention of development of vasospasm following SAH. Enoxaparin in a dose of 20 mg / day has been shown to improve overall outcome at one year after SAH. Statins like simvastatin and pravastatin have been shown to reduce vasospasm and associated mortality.

Hypertensive Hypervolaemic Haemodilution (Triple-H therapy) is used to augment perfusion in the vasospastic areas of the brain. Crystalloids are infused to attain a CVP of 10 – 12 mm Hg. Haemodilution to a haematocrit of 30 – 35 % helps to improve blood flow through the cerebral microvasculature. An infusion of vasopressor drugs like dopamine or phenylephrine maybe required to increase the blood pressure, the target being 120 – 160 mm Hg before the aneurysm is clipped or coiled and then a higher level of 160 – 200 mm Hg afterwards. The complications of Triple-H therapy are given in Table 9.

Table-9 Complications of Triple-H therapy
Re-bleeding
Haemorrhagic transformation of infarct
Cerebral / Pulmonary oedema
Raised intra cranial pressure
Hypertensive encephalopathy
Myocardial Infarction
Congestive cardiac failure
Dilutional hyponatremia
Coagulopathy
Complications of CVP access

In transluminal balloon angioplasty, the vasospastic segment is selectively catheterised and dilated. Selective intra-arterial injection of vasodilators like verapamil, nimodipine, nicardipine and papaverine may be done. The last drug is falling out of favour since it is neurotoxic.

Cardiac abnormalities following SAH include T-wave inversion, ST segment depression, U waves, QT prolongation and prominent Q waves in the ECG. Rhythm disturbances include PVC's, Sinus bradycardia, Sinus tachycardia, AV blocks, Atrial ectopics, Atrial fibrillation, Brady/Tachyarrythmias, Ventricular tachycardia and Ventricular fibrillation. They are usually seen during the first 7 days following SAH with a peak occurrence on the 2nd and 3rd day. Cardiogenic pulmonary oedema may also be seen in some cases.

Medical complications are responsible for 25 % of all deaths following SAH out of which 50 % are due to pulmonary causes. Pulmonary complications following SAH include neurogenic pulmonary oedema, pneumonia, ARDS and pulmonary embolism. Hepatitis and hepatic failure are seen in 25 % of cases, renal dysfunction in 8 % of cases and thrombocytopaenia in 4 % of cases following SAH. Gastrointestinal bleeding is seen in 5 % of cases of SAH and should be suspected if unexplained hypotension and tachycardia occur.

Management of Aneurysms: 85 % of all intracranial aneurysms originate in the anterior circulation and 10 % in the vertebro-basilar (Posterior) circulation. About 4% of aneurysms arise from the superior cerebellar artery and anterior inferior cerebellar artery. The common locations of aneurysms are given in Table – 10.

Table – 10 Common locations of intra-cranial aneurysms	
Location	Incidence
ACOM	~ 30 %
Junction of ICA and PCOM	~ 25 %
Bifurcation of MCA	~ 20 %
Bifurcation of ICA	~ 7.5 %
Basilar artery bifurcation	~ 7 %
Pericallosal-Callosomarginal artery Junction	~ 4 %
Origin of PICA	~ 3 %

Ruptured intracranial aneurysms should be treated as soon as possible after the haemorrhage to prevent re-bleeding and provide adequate medical treatment of vasospasm. Aneurysms can be managed surgically by clipping or endovascularly using Gugliemi Detachable Coils (GDC). Patients with large intra- parenchymal haematomas (> 50 ml) and MCA aneurysms benefit from clipping[20]. Early surgical clipping allows removal of blood clots from the sub-arachnoid space and the safe institution of Triple H therapy (Fig 1,2,3) .

Table-11 Indications for endovascular coiling
Age > 70 Years
Aneurysm is not amenable to surgical clipping
Patient is unable to tolerate surgery
Poor clinical grades (WFNS IV / V)
Basilar top aneurysm
Partially clipped aneurysm
Patient refusal for surgery

Fig 1: Aneurysm clips and applicator

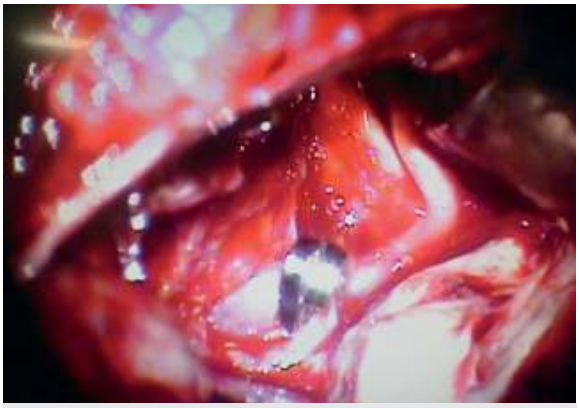

Fig 2: Clipped ACOM aneurysm

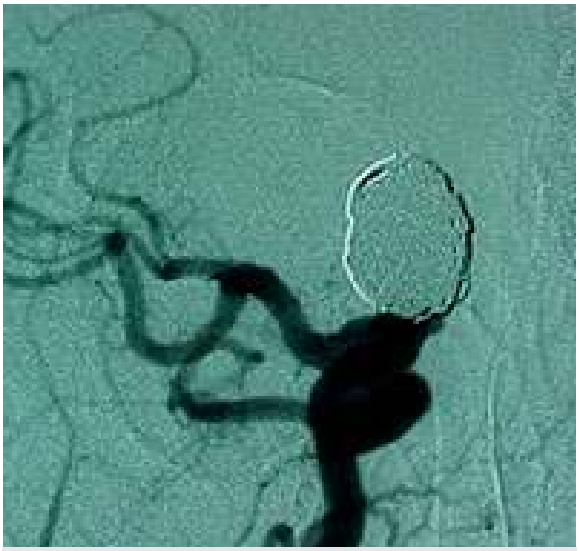

Fig 3: Coiled ICA aneurysm

The goal of endovascular coiling is to fill the aneurysmal lumen with Gugliemi Detachable Coils (GDC) which then initiates secondary thrombosis of the aneurysm. These are soft platinum coils which are placed inside the aneurysm. The coils are threaded through a micro-catheter placed in the aneurysmal lumen through the femoral artery. If the aneurysm is amenable to both clipping and coiling, coiling should be preferred. Elderly patients, patients with poor WFNS grades (IV and V) and those with a basilar top aneurysm benefit from endovascular coiling[20]. Broad necked aneurysms may be managed with balloon remodelling techniques and using bioactive coils which promote complete neck endothelialisation.

The complications of endovascular coiling include aneurysmal perforation, thromboembolic events and occlusion of the parent vessel from coil herniation. New technologies like enhanced coil construction, intracranial stents, balloon remodelling and two catheter techniques have evolved to enhance safety during endovascular coiling.

The management of aneurysmal SAH has changed considerably since the publication of the International Subarachnoid Aneurysm Trial (ISAT)[21]. This is a randomized controlled trial which compared endovascular coiling with surgical clipping in patients with ruptured aneurysms who were suitable for either treatment. A total of 2143 patients with ruptured intracranial aneurysms were included in the study. Primary outcome was death or dependence at 1 year. Secondary outcomes included re-bleeding from the treated aneurysm and risk of seizures. 23.5 % of patients (250 / 1063) allocated to endovascular coiling were dead or dependant at 1 year compared to 30.9 % of patients (326 / 1055) allocated to neurosurgery, an absolute risk reduction of 7.4 % (p=0.0001). The early survival advantage was maintained up to 7 years (p=0.03). The risk of epilepsy was substantially lower in patients allocated to endovascular treatment, but the risk of re-bleeding was higher. There were 13 re-bleeds from the treated aneurysms on long term follow up out of which 10 were in the endovascular group and 3 in the neurosurgical group (p=0.06)[22]. ISAT was criticised on a number of factors mainly related to the randomisation of the patient population. The patient population was on average younger and the majority had aneurysms under 10 mm and in the anterior circulation. In order to address these criticisms, a new randomised multi-centric trial (ISAT 2) is currently under way.

Anaesthesia for surgical clipping of aneurysms

Anaesthesia in these patients should ensure the preservation of adequate cerebral perfusion and oxygenation, avoidance of sudden changes in blood pressure and ICP, satisfactory surgical exposure and prompt emergence for early neurological assessment. Pre-operative evaluation of these patients includes a review of the radiological investigations, detailed history and a focused neurological examination. The relationship between hypotension and appearance of symptoms of neurological deterioration should be evaluated. The fluid and electrolyte balance and the cardiac function should be assessed. The patient's drug regimen should be evaluated.

Premedication in these patients includes drugs to reduce gastric acidity and prokinetic agents like metoclopramide. Calcium channel blockers, anticonvulsants and steroids should be continued in the perioperative period. Sedatives, hypnotics, anxiolytics and opioids are generally avoided since they may cause respiratory depression and mask neurological deterioration.

Monitoring in the perioperative period includes the usual monitors along with direct arterial blood pressure, central venous pressure, PCWP (if indicated), Trans-oesophageal echocardiography, Trans-cranial Doppler, Electroencephalogram, Jugular venous oximetry and arterial blood gases. The exact combination of monitoring may be selected based on requirement and availability.

Two large bore intravenous cannulae should be inserted and checked for free flow. A femoral venous sheath may be inserted in addition for rapid replacement of large volumes. During induction of anaesthesia, the hypertensive response to laryngoscopy and intubation should be blunted in all patients using adequate depth of anaesthesia, Beta-blockers [Esmolol (0.5 mg/Kg); Metoprolol (2-5 mg); Labetalol (2.5-5.0 mg/Kg)] or additional doses of Propofol (1-2 mg/Kg), Fentanyl (3-5 mcg/Kg), Lignocaine (1.5 mg/Kg) or Midazolam (0.1-0.2 mg/Kg).Sevoflurane or Isoflurane may be introduced to deepen the plane of anaesthesia before laryngoscopy. Other maneuvers that increase the blood pressure include application of skull pins, positioning for surgery and elevation of the bone flap.

Mild hypotension can be used in good grade patients (WFNS Grades 0, I, II) during induction since their ICP is usually not elevated and there is no evidence of cerebral ischaemia. It should be avoided in poor grade patients (WFNS Grades IV, V). Good grade patients may be ventilated normally, but poor grade patients with elevated ICP benefit from moderate hyperventilation to a pCO_2 of 30 mm Hg.

Maintenance of anaesthesia may be done with intravenous or inhalational agents or a combination of the two. An infusion of Fentanyl (bolus 25-50 mcg followed by an infusion of 1-2 mcg / Kg / Hr) may be combined with an infusion of Propofol (40-60 mcg/Kg/mt) and/or an inhalational agent like sevoflurane or isoflurane. Thiopentone at a dose of 3 mg/Kg/hr may be used in the presence of a tight brain. However its use is associated with delayed recovery and difficulty in early neurological assessment. Sevoflurane has minimal effects on cerebral haemodynamics and ICP. Isoflurane and desflurane can increase ICP when used at higher concentrations. Nitrous oxide is avoided in patients with poor intracranial compliance as it can further increase the ICP.Muscle relaxants that have minimal effect on cerebral haemodynamics like vecuronium, rocuronium and cis-atracurium may be used for intubation and maintenance.

In order to achieve brain relaxation to facilitate surgical access to the aneurysm, various measures may be adopted. These include moderate hyperventilation to a pCO_2 of 30-35 mm Hg (unless vasospasm is present), Mannitol at a dose of 0.25-1.0 GM/Kg (given after the dura is opened), Furosemide at a dose of 0.25-1.0 mg/Kg (potentiates mannitol), drainage of CSF through a lumbar sub-arachnoid catheter and using total intravenous anaesthesia (TIVA).

Patients with SAH are hypovolaemic and require more fluids compared to a patient with an unruptured aneurysm. Normovolaemia should be maintained in good grade patients till the aneurysm is clipped and then Triple-H therapy guided by invasive monitoring of the volume status should be instituted. Dextrose containing solutions are generally avoided since ischaemic deficits are exacerbated by hyperglycaemia. Ringers lactate is hypotonic to plasma and can contribute to cerebral oedema, hence it should be avoided. Blood and blood products should be utilized to maintain a haematocrit of 30 – 35 %. Blood should be available in the operation theatre before the dissection of the aneurysm starts. Hetastarch can interfere with haemostasis and might cause intracranial bleeding.

Good grade patients may be reversed and extubated at the end of the procedure taking care to avoid coughing, straining, hypercapnia and hypertension. Hypertension in the immediate post-operative period, unless existing before surgery, usually returns to normal within 12 hours. The various reasons for post-operative hypertension include pain, urinary retention and hypercapnia secondary to residual anaesthesia. The blood pressure should be maintained 10-20 % above preoperative values in patients at risk of developing delayed ischaemic deficits. The blood pressure of patients whose aneurysms have been wrapped rather than clipped should be maintained within 20 % of their normal range during recovery from anaesthesia. Patients with poor grades and who have brain swelling or those with intraoperative rupture of their aneurysm should be sedated and electively ventilated post-operatively. If a patient fails to wake up from anaesthesia or develops a new neurological deficit, all administered sedative, analgesic and anaesthetic drugs should be reversed, the pCO_2 normalized and hypoxia and hyponatremia ruled out or treated. If the patient still doesn't recover, an urgent CT scan should be ordered to diagnose a haematoma, hydrocephalus, pneumocephalus, cerebral infarct or cerebral oedema.

Most modern neurosurgeons prefer to avoid induced hypotension during dissection of the aneurysm because of increased incidence of vasospasm, cerebral ischaemia, infarcts and post-operative neurological deficits. Induced hypotension is however indicated if the aneurysm ruptures during dissection in order to gain control of the parent vessel. Use of induced hypotension could lead to increased risk of early and delayed neurological deficits[23,24]. Induced hypotension is relatively contraindicated in the presence of cerebrovascular occlusive disease, coronary artery disease, renal dysfunction and anaemia.

Temporary proximal clipping of the parent artery of the aneurysm may be used to reduce the risk of rupture during dissection. Blood flow in the collateral blood vessels may be enhanced during temporary (if required for > 120 seconds) by maintaining the patient's blood pressure in the high normal range. An infusion of dopamine or phenylephrine may be used for achieving this. The duration of temporary occlusion should be less than 20 minutes in order to minimize the risk of cerebral ischaemia and infarction, cerebral oedema and damage to the parent artery. A dose of Propofol or Thiopentone may be administered to achieve EEG burst suppression immediately before temporary occlusion. Mannitol may also be administered to reduce the incidence of cerebral oedema. None of the pharmacological agents

used for cerebral protection have been shown to improve neurological outcome in these patients.

Mild hypothermia (32-34°C) during surgery is not associated with a beneficial effect in mortality or neurological outcome among patients with good grade SAH[25]. In addition, intraoperative hypothermia had no beneficial effects on neuro-psychological function after SAH[26]. Intra-operative hyperglycaemia is associated with long term decline in cognition and gross neurological function[27].

The incidence of aneurysm rupture during induction of anaesthesia is 0.5 – 2.0 % while intraoperative rupture occurs in 6 – 18 %. The causes of intraoperative rupture include aneurysmal dissection, brain retraction, haematoma evacuation and dural opening. Aneurysm rupture during induction of anaesthesia and surgical dissection of the aneurysm carries a very high mortality and morbidity because of cerebral ischaemia from hypotension and attempts to clip the aneurysm on an urgent basis. Rupture during induction is associated with a sudden rise in ICP and systemic hypertension with or without bradycardia. A ruptured aneurysm is treated by normalizing the intravascular volume using crystalloids, colloids or cross matched blood, maintaining cerebral perfusion, controlling ICP and reducing bleeding by lowering the systemic BP. Definitive surgical repair should be carried out immediately and the aneurysm clipped after reducing the MAP to 40-50 mm Hg or using temporary proximal and distal occlusion of the parent vessel.

Anaesthesia for endovascular coiling of aneurysms

Patients undergo endovascular coiling in catheterization laboratories that are often located in remote areas of the hospital. The anaesthetist must be familiar with the intervention planned for the patient as well as the anticoagulation plan (degree, duration and timing of reversal). The baseline renal function should be evaluated as IV contrast will be used. The anaesthetist should also be ready to provide intra-procedural hypotension, hypertension and hypercapnia when required. Intravenous access and monitoring should be similar to that used in the neurosurgical operation theatre. Direct arterial BP monitoring is mandatory if manipulation of the blood pressure is anticipated.

The patient should be given a comfortable pillow and pressure points should be padded if conscious sedation is used since they may have to stay immobile for long periods of time. The main advantage of conscious sedation is that it allows the physician to perform periodic neurological examinations. Conscious sedation is also used to check adequacy of collateral circulation during trial balloon occlusion of parent vessel in patients with giant aneurysms. This is followed by trapping the aneurysm by coiling the parent vessel both proximal and distal to the aneurysm. General anaesthesia negates patient movement and improves image quality on radiological screening. Since access to the airway is difficult once the procedure starts, it is important to intubate the patient beforehand.

Induction and maintenance of anaesthesia is similar to that described for aneurysm clipping. Total intravenous anaesthesia may be used if the ICP is elevated. The systemic BP should be adjusted to maintain CPP and wide swings that can cause the aneurysm to rupture, should be avoided. Use high normal levels of blood pressure if the patient has vasospasm. Heparin should be given at the start of the procedure and reversed once it ends. This will help prevent thromboembolic events.

If the patient has a perforation during the procedure, it may manifest as headache, nausea, vomiting, systemic hypertension, bradycardia and change in the level of consciousness. This should be immediately treated with protamine (to reverse the heparin) and then maintaining the blood pressure in the normal range. Since aggressive treatment of hypertension may induce ischaemia, only extreme elevations should be treated. Platelets may be transfused if the patient is on aspirin, clopidogrel or glycoprotein IIb / IIIa receptor antagonists. If occlusive problems like thromboembolism or vasospasm develop, deliberate hypertension guided by neurological examination with or without direct thrombolysis or angioplasty to improve distal perfusion, should be undertaken.Other measures include volume expansion, head up tilt, hyperventilation, diuretics, anticonvulsants, mild hypothermia, cerebral protection and cerebral vasodilators.

Conclusion

The perioperative management of intracranial aneurysms has evolved over the past decades with advances in endovascular coiling and management of subarachnoid haemorrhage. Evidence based recommendations published by the American Heart Association and the American Stroke Association (most recently updated in 2012) have resulted in standardisation of the management of aneurysmal SAH[20]. The initial Hunt & Hess or WFNS score is a good indicator of the outcome following aneurysmal SAH. Since re-bleeding is associated with high mortality, all measures should be taken to prevent it.

Recovery following treatment of an intracranial aneurysm depends whether it has bled or not and the type of treatment administered. For patients with ruptured aneurysms, the hospital stay and recovery is typically stormy compared to unruptured aneurysms. Endovascular therapy, being less invasive compared to surgical clipping, is associated with a shorter hospital stay and a more rapid return to previous activities. Because of the higher risk of recurrence of aneurysms following endovascular therapy, these patients have to undergo follow up angiography at periodic intervals. Many of the patients recovering from SAH go on to develop mood disorders like depression and anxiety and this should also be addressed.

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