Anesthetic considerations for Endo-Vascular Management of Intracranial Aneurysms

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ABSTRACT

Intra cranial aneurysms are acquired lesions responsible for about 80% of non-traumatic sub arachnoid hemorrhage. Treatment of the condition in the past has relied on craniotomy and clipping of the aneurysm to prevent a recurrent hemorrhage. Nowadays endovascular coiling is the best primary treatment. The anesthesia in interventional radiology room has special arrangement and precautions. Intra operative management of endovascular cerebral aneurysm from the start including: arrangement of the room, monitoring, induction, maintenance and emergence of the patients. Post-operative care is very important and good management of potential perioperative complications like: aneurysm rupture, cerebral infarction, cerebral vasospasm, contrast reaction and nephropathy is mandatory.

Aim of the Study: reviewing the current medical literature as regards the anesthetic considerations and problems of endo-vascular management of intracranial aneurysm.

Conclusion: anesthesia in interventional radiology room should have special arrangements and precautions. Intra operative management of endovascular cerebral aneurysm from the start including: arrangement of the room, monitoring, induction, maintenance and emergence of the patients. Post-operative care plays a key role in mitigating potential perioperative complications like: aneurysm rupture, cerebral infarction, cerebral vasospasm, contrast reaction and nephropathy are mandatory.

Keywords: anesthesia , Post-operative care, Intracranial Aneurysms.

INTRODUCTION

Stroke and cerebrovascular diseases are the second leading cause of death worldwide and account for significant healthcare costs and morbidity among survivors [1].

Hemorrhagic strokes result from the rupture of weakened blood vessel walls, usually aneurysms or arteriovenous malformations. Often a subarachnoid hemorrhage (SAH) results from the rupture of these vessels [2]. The incidence of cerebral aneurysms in the general population is 1.5–8.0%. Multiple aneurysms exist in 20% of patients diagnosed with aneurysm. There is an increased incidence in first degree relatives, and an estimated lifetime risk of 2–5%.

The significant increase in the number of asymptomatic aneurysms diagnosed is most likely a consequence of screening policies. Patients can present with symptoms of subarachnoid haemorrhage (SAH), cranial nerve palsies, seizures, cerebral compression, and hydrocephalus. Cerebral aneurysm is responsible for 77% of acute spontaneous SAH. Patients who survive a SAH have a 4% risk of a further bleed in the first 24 h and a 1% risk per day thereafter. The morbidity and mortality (3%) rates related to embolization of an acute aneurysm are lower than those associated

with an untreated acute ruptured aneurysm [3]. Treatment of aneurysm involves obliterating the aneurysm by surgical clipping via the intracranial route or coiling via the endovascular route. If both treatment options are possible for a particular patient, endovascular coiling is the preferred method [4].

Endovascular coil embolisation of unruptured intracranial aneurysm (UIA) is associated with a 5–10% risk of morbidity and nearly zero mortality from the procedure [5].Complete or near complete occlusion is usually achieved in >90% of cases, and endovascular therapy seems to reduce the risk of future rupture significantly. Endovascular therapy appears to be a safe and effective treatment for selected unruptured intracranial aneurysm [6].

The SAH is often complicated by re-bleeding, hypertension, cerebral edema, delayed cerebral ischemia (DCI), electrolyte abnormalities, hydrocephalus, seizure activity, and cardiopulmonary dysfunction. Monitoring for and consideration of each of these complications should guide anesthetic management from preoperative through postoperative treatment [1].

The study was approved by the Ethics Board of Ain Shams University.

PREOPERATIVE ANESTHETIC CONSIDERATIONS

History

It is most important part of preoperative assessment. It include: Current and past medical history, surgical history, a social history (use of alcohol, tobacco and illegal drugs), a family history, current and recent drug therapy, history of allergies, abnormal reactions or responses to drugs and any complications accompany with previous anesthesia. A family history of abnormal reactions associated with anesthesia should also be obtained [7]. The history should include review of all systems to discover undiagnosed diseases or improper controlled chronic disease like: hypertension and diabetes, renal impairment especially, diseases of respiratory systems and cardiovascular systems are the most relevant in respect of fitness for surgery and anesthesia [7].

Endovascular management of cerebral aneurysms would be associated with exposure to significant amount of radiation, so the possibility of pregnancy in female patients of childbearing period should be explored by history taking or pregnancy test as pregnant ladies need extra protection against radiation during procedure [8]. In children, we should ask about birth history, specifically risk factors like: prematurity at birth, perinatal complication and congenital chromosomal or anatomic malformations and special attention for recent infections, especially upper and lower respiratory tract infections [7].

Physical examination

(1) Central nervous system:

Physical examination should be carried out and comparison and documentation of the pre and post procedure of neurological status is absolutely essential [9].

For better assessment of global cerebral dysfunction, interventional risk, and prognosis of outcome. Many scales have been proposed and the most widely used & accepted scales are:

(1) The Glasgow Coma Scale (GCS)

The Glasgow Coma Scale (GCS) used in assessment of global cerebral dysfunction which result from increase ICP (table 1). The existence of arterial vasospasm or increase ICP will place limitations on circulatory and ventilatory manipulations that the radiologist needs, and perhaps change the approach used. As result of increase in intracranial pressure, the patient become progressively more confused, lethargic, and eventually comatosed. When patient has GCS 8 or less the patient should has direct Monitoring of ICP [10].

Table (1): Glasgow Coma Score (ranging from 3 to 15) [10]

Test	Finding score	Score
Motor response	- No movement.	1
	- Abnormal extension.	2
	- Abnormal flexion.	3
	- Withdraws to pain.	4
	- Localizes to pain.	5
	- Obeys commands.	6
Verbal response	- No response.	1
	- Incomprehensible sounds.	2
	- Inappropriate words.	3
	- Confused conversation.	4
	- Oriented and appropriate.	5
Eye opening	- No response.	1
	- Response to pain.	2
	- Response to voice.	3
	- Spontaneously.	4

(2) The Hunt and Hess scale

In cases of ruptured aneurysm the grades of the Hunt and Hess subarachnoid hemorrhage scale, which has prognostic value should be noted **(table 2)** [11].

Table (2): Hunt and Hess Classification for Aneurysms [11].

Grade V	Deep coma and decerebrate rigidity
Grade IV	Stupor, hemiparesis, early decerebrate rigidity and vegetative disturbances
Grade III	Drowsiness, confusion, and mild focal deficits
Grade II	Moderate to severe headache and nuchal rigidity but no focal or lateralizing signs
Grade I	Asymptomatic or with slight headache

(2) World Federation of Neurological Surgeons (WFNS) grade.

Most widely used and accepted scales are the WFNS clinical scale which is used for grading patients with SAH [8].

(3) The Fisher grading scale.

Which uses CT to assess the amount of blood detected, gives the best indication of the likelihood of the development of cerebral vasospasm and patient outcome (Table **3**).

Table (3): Fisher grading scale of cranial computerized tomography (CCT) [12]

Grade	Findings on CCT
1	No subarachnoid blood detected
2	Diffuse or vertical layers ≤ 1mm
3	Localized clot and/or vertical layer >

	1mm
4	Intracerebral or intraventricular clot with diffuse or no subarachnoid haemorrhage

(4) Cardiovascular system

Assessment as usual in any procedure with special attention to certain points:

(A)ECG changes following SAH:

Due to hyperactivity of the sympathetic system with increased levels of norepinephrine. Abnormalities in both morphology of ECG and rhythm have been observed in up to 100% of patients with SAH [8].

• The types of dysrhythmia range from benign such as, sinus tachycardia, sinus bradycardia, atrioventricular dissociation, to potentially life threatening rhythms for instance ventricular tachycardia and ventricular fibrillation.

• ECG Morphological changes include: ST segment depression, T wave inversion, and appearance of Q waves and U waves. Also prolonged QT interval may appears in about one-third of patients, which in turn is associated with dangerous ventricular dysrhythmia. During the first 48h after SAH These ECG changes usually occur and may return to normal from 10 days to 6 weeks, while changes in T wave can persist for months [13].

(B) Pathological changes in cardiac structures with SAH: Include left ventricular (LV) dysfunction and elevated serum levels of cardiac enzymes including troponin I.

(C) Neurogenic Pulmonary Edema (NPE): Massive sympathetic discharge resulting from neurological injury may be the cause of NPE and is associated with reduced global and segmental left ventricular systolic function [14].

(D)Hypertension

To avoid lowering blood pressure markedly, cerebral perfusion pressure (CPP) must be maintained at adequate levels. In 2012, The American Heart Association (AHA) Guidelines for the Management of aneurysmal Subarachnoid Hemorrhage (aSAH) do not give any recommendation about any specific blood pressure. But, generally it is recommended to keep systolic blood pressure less than 160 mmHg because of the risk of re-bleeding: administering titratable, short acting, continuous intravenous (IV) agents with a favorable safety profile such as nicardipine,esmolol, labetalol [15].Nimodipine is recommended by AHA for every patients with aSAH for vasospasm prophylaxis. Although it has not been shown to improve cerebral vasospasm by angiogram, nimodipine has decreased delayed ischemia and improved neurologic outcomes [16]. Verapamil has been shown to improve neurologic outcomes

without increasing ICP [17]. Vasodilators such as hydralazine, sodium nitroprusside and nitroglycerine should be avoided because vasodilatation may increase cerebral blood flow and is likely to worsen ICP [15].

(3) Respiratory system: It should be assessed as usual as in any procedure with special attention to certain points:

(A) Cigarette smoking : Cigarette smokers have five times the risk of SAH as compared to non-smokers [18].

(B) Pulmonary edema: in patient with SAH the incidence of pulmonary edema range from 23% in patients who survived to more than 90% in patient who died [19]. Both types of cardiogenic and non-cardiogenic pulmonary edema participate in the formation of pulmonary edema Sympathetic stimulation, as result of hypothalamic ischemic stress, lead to hydrostatic pressure injury to the pulmonary capillaries causing pulmonary edema. Also, neurogenic pulmonary edema (NPE) can develop within seconds of neurological event or up to 2 weeks after SAH. This time scale corresponds to catecholamine hyper secretion that may last for 10 days or more after SAH [20].

INVESTIGATIONS

In addition to the general preanesthetic laboratory testing, evaluation of the hemostatic function should be considered. The Following laboratory tests should be available:

1. **Complete Blood Count (CBC)** including platelets count.

2. **Partial Thromboplastin time (PTT), Prothrombin Time (PT), bleeding time and clotting time** [21]

Since the use of anticoagulant is a part of procedure mostly and about 4% of SAH patients were reported to be thrombocytopenic within the first 2 weeks [19].

3- **Renal function tests**.

4- Intravascular volume assessment.

In 36% to 100% of SAH patient's intravascular volume was low which may reflect into renal dysfunction. Many factors may contribute including: bed rest, negative nitrogen balance, supine diuresis [22].

5-Electrolytes assessment: as there are disturbances such as: hyponatremia, hypokalemia, hypocalcemia, hypomagnesmia.

6-CT, CTA, MRI:

The first imaging of choice is non contrast head computed tomography (CT)**,** if SAH requires more evaluation we need CTA or cerebral angiography or even MRI diuresis [23].

Corticosteroids, Anticonvulsants, antibiotics etc. may be used as premedication drugs according to the patient's condition and requirements. In patients with SAH, who suffer from gastro esophageal reflux or obesity, H2 receptor antagonists like ranitidine and metoclopramide are used to decline the risk of aspiration [24].

INTRA OPERATIVE ANESTHETIC MANAGEMENT

Pre-procedure Preparation

One of the important tasks of the anesthesiologist before the procedure is checking the equipment and medication trolley and ensure that all essential medications are available and all equipments needed are perfect functioning [25]. Confirm continuous supply of oxygen and working suction is very important. Anesthetic drugs, Emergency resuscitation drugs and intravenous fluid should be kept on the drug trolley. Emergency trolley for difficult airway tools and cardiopulmonary resuscitation Equipment should be immediately available. The circuits of anesthesia must have extension tubing. Standard monitoring should be available and in perfect functioning [25].

MONITORING

Routine:

Usual monitoring in INR is similar to standard Monitoring in operating rooms [26]. It should consist of: pulse oximetry, blood pressure (BP) monitoring, ECG, nerve stimulator, inspired and expired gas analysis and temperature monitoring. Due to the duration of the procedure and ambient temperature of INR room. It may be necessary in some cases to use active warming devices to prevent hypothermia. Whereas hypothermia has been used as neuro-protective method, there is no definite human data to support the use of hypothermia as neuroprotective method in case of SAH. So, normothermia should be maintained during the procedure. Urine output is measured regularily and frequently assessed in order to ensure adequate hydration and electrolyte balance since the contrast medium produces an osmotic load that leads to a vigorous diuresis [75].

Arterial line: Monitoring blood pressure invasively is preferable from the start till the end of the procedure. Wide fluctuations in BP should be avoided to preserve adequate cerebral perfusion. So, it is better to insert an arterial cannula in an awake patient with local anesthetic before induction of anesthesia. In cases of un-ruptured aneurysms with good WFNS grade, it is possible to monitor BP non-invasively until the patient is asleep. We can also transduce the femoral artery introducer sheath to monitor invasive BP [21].

Central venous pressure:

Endovascular treatment of aneurysms is not commonly associated with marked blood loss, fluid shifts or air embolism as compared to surgical clipping. So, central venous pressure (CVP) monitoring is less indicated. Central venous line is needed when close monitoring and assessment of fluid status and postoperative hemodynamic maintenance are required especially in the patients with pre-existing medical problems, significant cerebral vasospasm, focal neurological deficit, and low GCS. It could be inserted with help of fluoroscopy [8].

Neurophysiological monitoring (NPM):

Intraoperative neurophysiological monitoring consists of EEG, somatosensory evoked potentials (SSEPs) and/or brain stem auditory evoked potentials (BAEPs), depends on the site of the aneurysm and vascular territory at risk. It is not commonly used during the procedure, but it is used in attempts to minimize neurological morbidity from operative manipulations. The aim of this monitoring is to know changes in the brain function prior to irreversible damage as the procedure can alter regional CBF in the arterial distribution of the aneurysm and cause attendant ischemic complications [28]. Also, intra aneurysmal management may result in thromboembolic complications or may partially hinder blood flow in the parent vessel. NPM can provide an indirect measure of regional ischemia produced in the cerebral circulation and direct assessment of the functional state of specific cerebral regions [28].

Transcranial Doppler (TCD):

It has been used widely to assess vasospasm after SAH. It is even more sensitive than NPM, which depends on change in CBF. The primary aim of TCD is to detect early development of a significant (high moderate or severe) vasospasm enabling these patients to receive suitable treatment. Potential TCD obstacles comprise poor sensitivity to mild (<20 mmHg) ICP increases, limited ability to detect distal branch vasospasm, high resistance flow patterns appearing due to increased cardiac output (CO) vs. increased ICP and difficult grading of spasm severity in arteries other than MCA. The other problems include the fact that the accuracy of interpretation of TCD data relies on skill, knowledge and experience of the Technician and the interpreter [29].

Techniques of anesthesia

1) Monitored Anesthesia Care (MAC): (Monitored Intravenous sedation/ Conscious Sedation)

At the first the patient should be comfortable as possible. The Targets of IV sedation are to relieve pain and discomfort, decrease anxiety, and ensure

that the patient remains motionless, but at the same time be able to rapidly reduce sedation when neurologic testing is required. It is suggested that carefully titrated sedation is a suitable technique for therapeutic endovascular neuroradiology procedures as it allows for intraprocedure neurological examination and assessment [30], thus allowing any new neurological deficit during the procedure to be rapidly diagnosed.

* Propofol is commonly used in numerous procedures that require sedation for the reason that it demonstrates a fast onset, rapid recovery and short half-life. In spite of these advantages, Propofol has a critical disadvantage, as it may induce severe respiratory depression, even apnea [31].

* On the other hand, Dexmedetomidine (Precedex), the highly selective Alpha-2 agonist, demonstrates both hypnotic and analgesic properties and with slight effect on respiration. Additionally, Dexmedetomidine decreases stress responses to surgery by reducing the sympathetic tone, so that Dexmedetomidine can be selectively used for INR procedures for which general anesthesia is not absolutely required [32].

2) General anesthesia: Most centers prefer general anesthesia (GA) versus sedation due to many factors.

Advantages of General Anesthesia:

1. With newer inhalation and induction agents, GA can be rapidly induced with minimal hemodynamic alterations, the depth of anesthesia freely controlled and a rapid and smooth emergence obtained.

2. Under GA, $PaCO_2$ can be regulated, controlled and management of induced hypotension is facilitated.

3. The patients are motionless with better images during the procedure.

4. In case of neurological emergency such as thrombosis or hemorrhage,

No waste time in securing the airway and the anesthesiologist can immediately begin to control hemodynamics [33].

Disadvantages of GA:

(1) Inability to perform neurological assessment immediately.

(2) Endotracheal intubation can cause hypertension, while extubation can lead to straining, airway obstruction, coughing and both intubation and extubation can lead to increase ICP. However, with careful anesthetic management, these disadvantages can be minimized [33].

* For airway control with less hemodynamic stress and smoother emergence the laryngeal mask airway (LMA) may be used as an alternative to endotracheal intubation for the airway management.

However, the protection and security of the airway by LMA are not as complete as that by endotracheal intubation during this long procedure [32].

Induction of anesthesia:

An aneurysm integrity is dependent on the transmural pressure (TMP). TMP is calculated by the difference between the pressure within the aneurysm (equivalent to MAP) and the pressure outside the aneurysm (equivalent to ICP) So, TMP = MAP - ICP[8].

• **One practical method** is to aim for a moderate (nearly 20%) initial reduction in baseline blood pressure during induction of anesthesia, administer drugs that blunt or abolish the hypertensive response to endotracheal intubation process (e.g.I.V. lidocain, labetalol and esmolol), and then go on with tracheal intubation [34].

• **Alternative approach** is to provide a deep level of anesthesia (Achieved by the use of high doses of anesthetic drugs, or confirmed by monitoring of depth of anesthesia) while counteracting the expected reduction in blood pressure (and thus in CPP) by a continuous infusion of a vasopressor drugs (E.g. norepinephrine or phenylephrine) from the beginning of induction of anesthesia. The deep level of anaesthesia will reduce the hypertensive response to laryngoscopy and endo tracheal intubation (reducing risk of rupture aneurysm), while the continuous infusion of a vasopressor drugs will guarantee adequate CPP (reducing risk of cerebral ischemia) [34].

• **Another method** is the balance according to patient's clinical grade between the risk of rupture of the aneurysm versus risk of inadequate cerebral perfusion.

patients with normal ICP like clinical grades I and II of Hunt and Hess Classification for Aneurysms and don't manifest acute ischemic deficits: These patients can be expected to tolerate nearly more than 20% transient reduction in CPP without becoming acutely ischemic. So in these patients considerably decreasing the risk of rupture of the aneurysm takes priority [34].

Maintenance of anesthesia

The aim of maintenance of anaesthesia include the need to maintain a stable BP, ablate the response to painful stimuli and avoid any further increases in ICP. This can be achieved by using a volatile or total intravenous anaesthesia (TIVA) anaesthetic and opioids. As there is no skull decompression during endovascular treatment, the risk of aneurysm rupture is present till it is coiled successfully [8].

Sevoflurane appears to be the vapour closest to the 'ideal'. The change in CBF secondary to a

change in arterial CO2 tension is preserved at 1 MAC (minimal alveolar concentration) end-tidal sevoflurane anaesthesia and 'coupling' of CBF and Cerebral metabolic rate of oxygen (CMRO2) is maintained. However, isoflurane and desflurane in less than 1 MAC concentrations have also been used safely [8].

The routine use of nitrous oxide for these cases is not encouraged since nitrous oxide increases cerebral blood flow from 60 to100%; and can increase ICP to a variable degree. Nitrous oxide could also theoretically enlarge air bubbles that may be accidentally introduced into the arterial circulation by the radiologist [21].Opioids are lacking intrinsic cerebrovascular effects. Increases in ICP that have been measured after opioid boluses are probably secondary to a vasodilatory response to hypotension.

A combination of low dose of propofol and remifentanil infusions supplemented with sevoflurane in oxygen enriched air can also be used to maintain anesthesia. This combination of low doses of I.V. agents and a vapour minimises individual drugs' side-effects and allows 'fine tuning' of the depth of anaesthesia by varying vapour concentration [35].

Cannulation of the femoral artery is usually the most stimulating part of the procedure, although, manipulation of the microcatheter in the intracranial blood vessels and injection of the contrast material can also cause varying amounts of discomfort. Overall, the anaesthetic requirements are not high, and the relative overdose may lead to difficulty in the maintenance of cardiovascular stability [8].

Intraoperative Management

(1) Intraoperative Management of Ventilation

By changing the PaCO2, cerebral blood flow, cerebral blood volume and intracranial pressure can be modified. A high PaCO2 causes cerebral vasodilatation, increasing CBF and to a lesser extent CBV. Between a PaCO2 of 18mmHg and 80mmHg, CBF varies directly with the PaCO2. The lowest CBF that can be obtained by hyperventilation occurs at a PaCO2 of 18-20 mmHg[36].

To manage ventilation, the $PaCO_2$ can be obtained from a blood gas, or deduced from the $PetCO_2$. Ventilatory adjustments should be made by changing respiratory rate, keeping tidal volume constant, because changes in tidal volume can alter physiologic dead space and change the $PaCO_2$-$PetCO_2$, relationship. Increasing the ventilation by using large tidal volumes can also elevate intracerebral venous pressure. Most patients for endovascular neurosurgery are ventilated to a $PaCO_2$ of low normal around 35 mmHg [37].

(2) Intraoperative Management of blood pressure:

The most important physiological goals in the management of Arterial pressure during endovascular neurosurgical procedures are:
- To maintain cerebral perfusion pressure.
- To prevent vessel rupture.
- To avoid cerebral edema.

Once the radiologist reaches the AVM or aneurysm, controlled hypotension is required to a MAP of 50-70 mmHg and continue until embolization is complete. This is the most critical part of the procedure, and invasive monitoring of the blood pressure is mandatory. After the embolization is complete, MAP is allowed to increase, But keep it 10 -20% lower than basal levels during emergence and in the Neurological intensive care unit [9].

(3) Intraoperative Management of Anticoagulation:

Heparin: Heparinisation is commenced at Different times by different centers, but is usually between femoral cannulation and insertion of first coil. The aim is to maintain activated clotting time (ACT) between 2.3 times normal. This may be achieved by 3000.5000 IU I.V. in adults, followed by boluses. Other regimes for administration of heparin include a loading dose of (100 IU/kg) followed by hourly additions of 1000 U to maintain ACT at twice the baseline value [35], or (70 IU/kg) followed by continuous infusion or intermittent bolus with hourly monitoring of ACT [33].

Direct Thrombin Inhibitors: Heparin-induced thrombocytopenia is a rare but important adverse event for heparin anticoagulation. Development of heparin-dependant antibodies after initial exposure leads to a prothrombotic syndrome. In high-risk patients, direct thrombin inhibitors can be applied, recognizing that there are inherent adverse events, such as anaphylaxis, associated with their use [38].Bivalirudin was also identified a potential alternative during endovascular neurosurgical procedures to heparin for IV anticoagulation and intra-arterial thrombolysis [38].

Antiplatelet Agents

Antiplatelet agents (aspirin, Thienopyridine derivatives, and the glycoprotein IIb/IIIa receptor antagonists e.g. abciximab) have been extensively used in patients with coronary stents, and are especially useful in patients who have developed thromboembolic vascular occlusion leading to reduced blood [33]. Abciximab, Eptifibatide and Tirofiban are glycoprotein IIb/IIIa receptor antagonists. The long duration and potent effect of Abciximab also increase the likelihood of major bleeding. The smaller molecule agents, Eptifibatide

and Tirofiban, are competitive blockers and have a shorter half-life of approximately 2 hours [33].

Reversal of Anticoagulation: At the end of the procedure or at occurrence of hemorrhagic complication, heparin may be reversed with Protamine. The usual dose is a 3000 –5000 unit bolus and 1000 u/hour to maintain ACT at around 2.5 times the baseline [39].

(4) Intraoperative Management of Blood transfusion: Perioperative blood transfusion has been associated in one study with the development of angiographically confirmed vasospasm after SAH and worse outcome in patients with SAH and hence some centers prefer to avoid blood transfusion unless strongly indicated [40].

Recovery: After assessing the reversibility of the muscle relaxant, patient is extubated while still under deep anesthesia, reverse the muscle relaxant and put the patient breathing oxygen via face mask. If a laryngeal mask airway was used, the patient is allowed to wake up with the device in place to be removed once the patient responds to commands. This awake extubation with LMA is remarkably well tolerated, with little cough, straining or [9].

POSTOPERATIVE MANAGEMENT
Post-Procedure Management:

Awakening period after AVM embolization and coiling of aneurysms is very critical. Rapid and smooth emergence without hemodynamic stress, cough, or strain to prevent increases in intracranial pressure is very important [41].

Many of the patients undergoing endovascular neuroradiological procedures require intensive care and are transported directly to intensive care unit (ICU), or main operating room PACU. The transfer to these units should be supervised by the anesthesiologist and the patient's peripheral oxygen saturation, blood pressure and ECG should be monitored during transfer [42].

Post procedure blood pressure control is important, i.e. modest hypotension in the case of AVM embolization or relative hypertension in the patient with occlusive disease or cerebrovascular vasospasm [43]. Maintenance of modest hypotension is required post AVM embolization to prevent cerebral oedema and haemorrhage. The mean arterial pressure should be kept 15–20% below the baseline for 24h *(Connors and Wojak, 1999)*.

Antihypertensive agents such as labetolol or esmolol, which have minimal effect on cerebral physiology, can be used to control pressure. A mean arterial pressure 20–30% above normal may be required in patients with occlusive conditions or vasospasm to maintain cerebral perfusion pressure. This can be achieved with the use of phenylephrine

or norepinephrine. Nimodipine, is used in aneurysmal SAH until the patient can take oral medication and continued for 3 weeks [45]. Most patients receive aspirin 75 mg for 3 months afterwards. Maintenance of heparinization in the post-procedure period is recommended if a large surface area of coil is exposed in the parent vessel, or if an embolic complication was encountered during the procedure. Patients should remain supine until the femoral sheath is removed [45]. Postoperative nausea and vomiting can be a problem due to contrast and anaesthetic agents used during the procedure. Maintenance of hydration is important, as there can be a large osmotic diuresis due to hyperosmolar contrast used during the procedure. Post-procedure ischemia and swelling from contrast can be symptomatic after procedures performed in the posterior fossa [45].

Complications of interventional neuroradiological procedures:

Complications during the INR procedures could happen rapidly and may be catastrophic. There should be good communication between the neuroradiologist, anaesthetist, and the radiographer for the prompt management of complications that may occur. The primary responsibility of the anaesthetist is for the airway and gas exchange. It is important to know whether the complication is occlusive or haemorrhagic as these require a different approach for successful management [45].

(A)CNS complications:
1- Hemorrhagic
(a)Aneurysm rupture:

May be occur during introduction of micro catheter. Also, perforation of proximal vessels may also occur. There may be a sudden and massive rise in BP with or without a reduction in heart rate, due to raised ICP, and extravasation of contrast material may be noted by neuroradiologist. Therefore, it is important to maintain good communication between the anesthetist and the neuroradiologist. Prompt management is required in this situation [8].

Aggressive treatment of surges of BP predisposes to a risk of ischaemia in areas with loss of autoregulation, Hence it seems best to reserve antihypertensive therapy for patients with extreme elevations of BP as well as clinical or laboratory evidence of rapidly progressive end organ deterioration [46]. A common practice is to maintain BP at the same level as prior to the bleed. Anticoagulation should be reversed as soon as possible followed by rapid delivery of coils to seal the breach. Hyperventilation and osmotically active agents like hypertonic saline or mannitol may be required to control raised ICP. If the endovascular management is incomplete or unsuccessful,

craniotomy and surgical clipping may be required and operative conditions may be less than optimal [8].

2. Occlusive:

(a) Cerebral infarction:

Cerebral infarction occurs due to vascular occlusion which may be caused by coil misplacement, thromboembolism, catheters or vasospasm. These will need to be treated to ensure adequate blood supply to the ischaemic area of the brain. Misplaced coils should be attempted to be retrieved by the neuroradiologist, while thromboembolic event will necessitate anticoagulation with heparin or antiplatelet drugs and an increase in BP [8].

(b) Cerebral vasospasm:

Vasospasm reduces CBF to the cerebral tissue, Causing cerebral ischemia and subsequent delayed ischemic neuro-deficits[8].

Treatment of established vasospasm
*Nimodipine:

Nimodipine is a calcium channel blocker which is highly lipophilic and so effectively crosses the blood–brain barrier. Prophylactic administration of nimodipine significantly improves outcomes following aneurysmal SAH .It can be administered orally, I.V. or by the intra-arterial route [47].

*Triple 'H' therapy:

Triple 'H' therapy stands for Hypertension, hypervolaemia, and haemodilution. The aim of this treatment is to augment CBF by increasing BP, expanding the blood volume and reducing blood viscosity [48].

Parameters set for triple 'H' therapy vary slightly between studies, but generally include CVP of 8–12mmHg, MAP> 20mmHg higher than preoperative BP and a hematocrit of 0.30–0.35 mL [49].

*Papaverine:

Papaverine-a non-specific muscle relaxant has also been used to treat vasospasm . The resulting vasodilatation is often referred to as **'Chemical angioplasty'**. Papaverine is administered into the affected artery under angiography by the neuroradiologist. The drawback to its use is that it is very short acting and often requires subsequent doses to be administered. Numerous other reported complications include seizures, change in mental status, cerebral haemorrhage and new focal neurological deficits [50].

*Angioplasty:

Cerebral angioplasty is the mechanical dilatation of the vasospastic blood vessels using compliant balloons that conform to the vessel wall. It is an invasive procedure and usually reserved for patients who have not responded to triple 'H' therapy and are at a high risk of developing cerebral infarction [51].

B. Non-CNS complications:
(1) Contrast reaction

The most commonly used contrast for INR nowadays is iohexol (non-ionic) with an osmolality of 672 mOsm /kg. Although fatal reactions occur at the same frequency of ionic agents (1:10 000 exposures), non-ionic agents have a lower incidence of mild and moderate reactions [52].

(2) Contrast nephropathy

This is the third most common cause of hospital-acquired renal failure, and accounts for 12% of patients. The risk factors include diabetes mellitus, high dose of contrast, volume depletion, co-administration of nephrotoxic medications, and pre-existing renal disease [53].

N-acetylcysteine, 600–1200 mg twice daily, two doses before and after the procedure has shown significant reduction in the incidence and it is acceptable for use in High-risk patients [54].

Isotonic bicarbonate infusion may also reduce the incidence of contrast-induced nephropathy, by alkanizing renal tubular fluid and thereby minimizing tubular damage [54].

CONCLUSION

Anesthesia in interventional radiology room should have special arrangements and precautions. Intra operative management of endovascular cerebral aneurysm from the start should be followed. It includes: arrangement of the room, monitoring, induction, maintenance and emergence of the patients. Post-operative care plays a key role in mitigating potential perioperative complications like: aneurysm rupture, cerebral infarction, cerebral vasospasm, contrast reaction and nephropathy.

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