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ORIGINAL ARTICLE

Perioperative complications in endovascular neurosurgery: Anesthesiologist's perspective

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ABSTRACT

Background: Endovascular neurosurgery is known to be associated with potentially serious perioperative complications that can impact the course and outcome of anesthesia. We present here our institutional experience in the anesthetic management of various endovascular neurosurgical procedures and their related complications over a 10-year period.

Methods: Data was obtained in 240 patients pertaining to their preoperative status, details of anesthesia and surgery, perioperative course and surgery-related complications. Information regarding hemodynamic alterations, temperature variability, fluid-electrolyte imbalance, coagulation abnormalities and alterations in the anesthesia course was specifically noted.

Results: Among the important complications observed were aneurysm rupture (2.5%), vasospasm (6.67%), thromboembolism (4.16%), contrast reactions, hemodynamic alterations, electrolyte abnormalities, hypothermia, delayed emergence from anesthesia, groin hematomas and early postoperative mortality (5.14%).

Conclusion: Awareness of the unique challenges of endovascular neurosurgery and prompt and appropriate management of the associated complications by an experienced neuroanesthesiologist is vital to the outcome of these procedures.

Key words: Endovascular neurosurgery, interventional neuroradiology, neuroanesthesia, perioperative complications

Introduction

Endovascular neurosurgery, also known as interventional neuroradiology, involves the diagnosis and treatment of neurovascular disease by fluoroscopy-guided endovascular delivery of drugs and devices into the cranial and spinal circulation. Endovascular techniques of coiling, embolization, angioplasty, and stenting are used to treat some common lesions like cerebral aneurysms, arterio-venous malformations (AVM), vascular tumors, carotico-cavernous fistulae (CCF), carotid artery stenoses, venous angiomas, and thromboembolic strokes.[1] Progress in this field is continuing and evolving neurovascular technology now enables the management of complex-architecture aneurysms and recurrent intracranial atherosclerotic lesions.[2] The International Subarachnoid aneurysm trial (ISAT) and Cochrane Collaboration have even reported better outcomes with endovascular coiling compared to craniotomy and clipping, particularly in anterior circulation aneurysms in good grade subarachnoid hemorrhage (SAH) patients.[3,4] However, despite the advantage of avoiding the ill effects of craniotomy, endovascular neurosurgical procedures have their own potentially serious complications. These include aneurysm rupture, thromboembolism, intracranial vessel injury or dissection, coil displacement or fracture causing parent artery compromise, vasospasm, cerebral edema, hemorrhage and ischemia, contrast reactions, contrast nephropathy, groin hemorrhage or hematoma, hypothermia,

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and fluid-electrolyte imbalance. The adverse consequences of these complications like hemodynamic instability, respiratory insufficiency, delayed emergence from anesthesia, need for postoperative ventilation, neurological deterioration, seizures, stroke, and even death can affect the anesthesia course and outcome;[1,2,5-11] it is hence important for the neuroanesthesiologist to have a thorough understanding of these procedure-related problems. We present here our 10-year-long institutional experience in the anesthetic management of various endovascular neurosurgical procedures (excluding stroke thrombolysis) and their related perioperative complications. These procedures in our institute, unlike in most other centers, are done by neurosurgeons in our regular neurosurgery operating theaters (OT).

Materials and Methods

Following institutional ethics committee approval, prospective data were collected from consecutive patients undergoing elective or emergency therapeutic endovascular neurosurgery under general anesthesia (GA); the study period was from January 2002 to December 2011. The data pertained to their pre-anesthetic check-up (PAC), anesthesia and endovascular procedures, and perioperative course and complications. The PAC included documentation of modified Hunt and Hess (HH) grading of SAH, aneurysm characteristics, patient's neurological grading, and presence of co-morbidities, especially allergy to iodine and protamine. The comorbidities were maximally treated before surgery; poor neurological grade patients were put on ventilator support and those with preoperative cerebral vasospasm were started on intravenous (IV) nimodipine infusion @ 2 mg/hour. Informed consent for the procedures was obtained.

Anesthesia was administered with IV thiopentone (4-5 mg/kg), vecuronium (0.1 mg/kg initial bolus, 0.8-1.0 μg/kg/min infusion), IV fentanyl (2 μg/kg initial bolus, repeated1-2 μg/kg hourly), midazolam (0.05 mg/kg), ondansetron (0.1 mg/kg), and propofol infusion (100-150 μg/kg/min). The patients were intubated and ventilated with an oxygen (O_2) and nitrous oxide (N_2O) mixture (FiO_2 0.4) to achieve end-tidal CO_2 ($EtCO_2$) levels of 30-33 mmHg. Intraoperative monitoring included electrocardiogram (ECG), heart rate (HR), arterial blood pressure (ABP), central venous pressure (CVP), oxygen saturation (SpO_2), $EtCO_2$, arterial blood gases (ABG), nasopharyngeal temperature, urine output, and activated clotting time (ACT). Fluids given were Ringer's lactate, normal saline, and colloids. Patient temperature was maintained with forced-air blankets and pre-heated fluids, infusions, and irrigation fluids. At the end of the procedure, anesthesia was reversed with IV neostigmine (0.05mg/kg) and glycopyrrolate (0.01mg/kg). The patients were extubated on table and subsequently monitored in the intensive care unit (ICU) till they stabilized. Deliberate hypotension (ABP ~ 15-20% below baseline) was used in post-AVM embolization patients while deliberate hypertension was used after aneurysm coiling. Nimodipine was administered as infusion @ 2 mg/hour for the initial 48 hours following aneurysm coiling and later continued orally at 60 mg 4 hourly doses for 5-7 days.

Surgery involved trans-femoral catheterization of the feeding artery by Seldinger's technique using digital subtraction angiography and road mapping under high-resolution fluoroscopy (Seimens AG, Muenchen, Germany); non-ionized contrast Ioxehol (GE Healthcare Inc, Buckinghamshire, UK) was used. All OT personnel used protection against radiation hazards. The femoral artery was punctured by a 6-8 Fr check-flow cannula, through which, a 5-7 Fr end-hole guiding catheter was negotiated into the feeder artery of the vascular lesion. Super-selective catheterization of the vascular lesions was done using 1-2.4 Fr micro-catheters through which drugs and devices like Guglielmi detachable coils and polyvinylalcohol particles (Boston Scientific, Massachusetts, USA), Onyx (EV3 Neurovascular, California, USA), N-butyl cyanoacrylate (Dr Reddy's Labs, Hyderabad, India), and revascularization stenting device (Covidien, Massachusetts, USA) were delivered. Anticoagulation was achieved with IV heparin boluses (60-70 IU/kg initially at the time of check-flow insertion, 1/3rd initial dose repeated hourly) to maintain ACT at 2-3 times baseline values. Heparinized normal saline infusion (1000 IU heparin/l), delivered at 200-250 mmHg pressure was used for continuous catheter irrigation. The check-flow sheath was removed after the procedure only when the postoperative ACT normalized, and the cannulation site was manually pressed till bleeding stopped. IV ceftriaxone 50 mg/kg/day was used for perioperative antibiotic coverage for 2 days.

The following information was collected in all cases:

- Hemodynamic variables (HR and mean ABP): Bradycardia and tachycardia were defined as decreases and increases, respectively, in HR of 20% or more over baseline values. Hypotension and hypertension were defined as decreases or increases, respectively, in mean ABP of 20% or more over the baseline
- Fluid intake-output, CVP, serial ABG, and serum electrolytes. Hyponatremia and hypernatremia were defined as decreases and increases, respectively, in serum sodium outside 130-145 meq/l. Hypokalemia and hyperkalemia were defined as similar changes in serum potassium outside 3.5-5 meq/l
- ACT values pre-heparinization (baseline), post-heparinization, hourly, and post-procedure till baseline reached. This measurement was not carried out routinely
- Nasopharyngeal temperature: Hyperthermia and hypothermia were defined as an increase or decrease, respectively, in temperatures outside 35-38^{0}C

- Surgical complications like technical failure, aneurysm rupture, cerebral vessel injury/dissection, thromboembolism, vasospasm (narrowing of vessels more than 25% of normal), coil migration/fracture, catheter entrapment, etc
- Contrast reactions such as bronchospasm, hypoxia, hypercarbia, cutaneous eruptions, hemodynamic collapse, etc
- Delayed awakening from anesthesia (preoperative consciousness obtained at ≥45 minutes following reversal) and need for postoperative ventilation
- Postoperative complications within 2-3 days of surgery like, groin hemorrhage or hematoma, neurological deterioration, seizures, stroke, vasospasm, fluid-electrolyte imbalance, infection/meningitis, nephropathy, death, etc.

Table 1: Demographic profile and preoperative patient data

	No. of patients, *n* (%)
Sex	
Male	126 (52.5)
Female	114 (47.5)
Age (years)	
<20	20 (8.33)
20-50	145 (60.41)
50-70	70 (29.17)
>70	5 (2.08)
Diagnosis	
Cerebral aneurysms	167 (67.61)
Arterio-venous malformations	42 (16.59)
Carotico-cavernous fistulae	20 (8.33)
Venous angiomas	2 (0.83)
Vascular tumors	3 (1.25)
Carotid artery stenoses	6 (2.5)
Modified Hunt and Hess grading in cerebral aneurysm patients (167)	
Grade I	40 (23.95)
Grade II	74 (44.31)
Grade III	38 (22.75)
Grade IV	11 (6.59)
Grade V	4 (2.39)
Preoperative patient co-morbidities/ abnormalities	
Hypertension	42 (17.5)
COPD/asthma	34 (14.17)
Diabetes mellitus	14 (5.83)
ECG abnormalities (ST-T changes, U and Q waves, brady/tachycardia)	94 (39.17): attributed to SAH: 65 (27.08) and to CAD: 29 (12.08)
Hyponatremia (seen in poor grade SAH patients)	3 (1.25)
Abnormal echocardiography (LV dysfunction, low EF)	32 (13.33)
Poor GCS patients requiring preoperative mechanical ventilation	8 (3.33)

SAH: Subarachnoid hemorrhage, COPD: Chronic obstructive pulmonary disease, ECG: Electrocardiogram, CAD: Coronary artery disease, GCS: Glasgow coma scale, LV: Left ventricle, EF: Ejection fraction

Results

Table 1 depicts the demographic profile and preoperative data of the total 240 patients studied; the youngest was a 5-year-old boy for AVM embolization while the oldest was an 84-year-old woman for aneurysm coiling. Majority of patients were for aneurysm coiling and belonged to a good HH grade; 8 of these had stent-assisted coiling for wide-neck aneurysms. Hypertension was the commonest comorbidity observed. The duration of endovascular procedures ranged between 25 to 320 minutes (mean, 104 minutes); procedures were longer during the initial 2-3 years of the study. In adults, average 3 l (maximum 5 l) of irrigation fluid, 3000 IU (maximum 5000 IU) of heparin in the irrigation fluid and 60-159 IU/kg of IV heparin was used.

Table 2 lists the various perioperative complications observed. Patients in whom aneurysm coiling was abandoned due to technical failure were ventilated and later underwent craniotomy and aneurysm clipping. Intraoperative hemodynamic changes primarily included transient bradycardia, hypotension, and hypertension with tachycardia. Aneurysm rupture was mostly seen during insertion of coils and was managed with IV protamine administration, lowering of ABP using propofol, isoflurane and fentanyl, postoperative ventilation, and external ventricular drain insertion (2 patients). Cerebral vasospasm occurred more in patients of aneurysm coiling and was treated by instituting deliberate hypertension, intra-arterial instillation of nimodipine (0.1 mg/min), IV nimodipine infusion, and postoperative triple-H therapy in patients with successful coiling. Thromboembolism occurred more in patients of AVM embolization; it was treated with mechanical removal of thrombi and chemical thrombolysis; 5 of these patients were ventilated postoperatively under propofol and fentanyl sedation. Occlusion of the middle cerebral artery (MCA) due to migration of glue during AVM embolization was treated successfully with mechanical lysis by the surgeon.

Two elderly patients had contrast reactions during aneurysm coiling manifesting as hypotension and bronchospasm, but no accompanying hypoxia or hypercarbia; there was no prior history of allergic predisposition in them. They were effectively managed with steroids, bronchodilators, and anti-histaminic drugs.

No significant coagulation abnormalities were observed in any patient. However, one patient developed a minor vessel rent during surgery which resulted into a large postoperative cerebral hematoma, presumably due to heparin-induced exaggerated bleeding.

Fluid-electrolyte abnormalities, requiring treatment with diuretics and electrolyte replacement, were observed during the earlier phase of our study when the procedures were of

Table 2: Perioperative complications observed during endovascular neurosurgery

Complications	No. of patients, *n* (%)
Technical failure	5 (2.08)
Bradycardia	3 (1.25), during carotid stenting
Hypertension with tachycardia	6 (2.5), secondary to aneurysm rupture
Hypotension	10 (4.16), in carotid artery procedures and secondary to contrast reactions
Aneurysm rupture	6 (2.5)
Arterial dissection	1 (0.42)
Intraoperative vasospasm	16 (6.67), including 10 patients of aneurysm coiling
Thromboembolism	10 (4.16), including 3 patients of aneurysm coiling and 7 patients of AVM embolization
Parent artery occlusion due to migration of glue into the MCA during AVM embolization	2 (0.83)
Coil migration	4 (1.67)
Micro-catheter entrapment	1 (0.42)
Major contrast reactions	2 (0.83)
Intraoperative electrolyte abnormalities (hyponatremia, hypokalemia), fluid overload (raised CVP)	16 (6.67)
Hypothermia	10 (4.16)
Delayed emergence from anesthesia	16 (6.67), including 10 patients of hypothermia, 4 patients of intraoperative vasospasm, and 2 sedated elderly patients
Need for postoperative ventilation	26 (10.83), including 5 patients of ruptured aneurysms, 5 patients of technical failure, 1 patient with pre-existing respiratory disease, 5 patients of thromboembolism, 1 patient with severe vasospasm, 8 patients with poor preoperative GCS, and 1 elderly cardiac patient with evidence of fluid overload
Postoperative vasospasm	17 (7.08)
Postoperative neurological deterioration	28 (11.67), including 17 patients of vasospasm, 2 patients of cerebral infarction, 1 patient with cerebral hematoma, 7 patients with cerebral edema, and 1 patient with stroke
Excessive groin hemorrhage	8 (3.33)
Postoperative electrolyte abnormalities	12 (5)
Postoperative pulmonary edema	1 (0.42)
Postoperative mortality	13 (5.14), including 3 patients of ruptured aneurysms, 2 patients of thromboembolism, 1 patient of cerebral hematoma, and 7 patients with poor preoperative GCS

AVM: Arteriovenus malformation, MCA: Middle cerebral artery, CVP: Central venous pressure, GCS: Glasgow coma scale

longer duration and excess irrigation fluids were used; one elderly cardiac patient had intraoperative evidence of fluid overload and subsequently developed frank pulmonary edema necessitating extended postoperative ventilation.

On table extubation was followed in all patients but those already on preoperative ventilation and those with abandoned procedures or intraoperative neurological insults were ventilated postoperatively. Vasospasm and cerebral edema were the main causes for the development of early postoperative neurological deterioration necessitating intubation and mechanical ventilation in 11 patients. Some patients had delayed awakening from anesthesia; hypothermia was the main factor responsible in these cases.

Excessive groin bleeding (8 patients) led to large hematoma formation in 2 patients but no patient developed impaired perfusion in the distal limb. Groin hemorrhage was managed with immediate removal of check-flow cannulas, applying local pressure for 20-30 minutes and pressure bandaging of the site.

There was no intraoperative mortality but early postoperative deaths occurred in 13 patients (5.14%) with ruptured aneurysms, cerebral infarction, cerebral hematoma, and poor preoperative neurological status.

Discussion

The main challenge for the anesthesiologist in endovascular neurosurgery is to provide a well-titrated, procedure-specific anesthesia to patients and to manage the sudden and often serious perioperative complications, especially if occurring in remote and inadequately equipped areas of the radiology suites. Ensuring safe intra-hospital transport of critically sick patients, maintaining good hemodynamic control, manipulating systemic and cerebral blood flows in accordance with the procedure, managing adequate anticoagulation, ensuring complete patient immobility during procedures and subsequent rapid awakening at the end to facilitate early neurological examination, and prompt diagnosis and management of the procedure-related complications are among the important anesthetic considerations in these patients.[1,2,5-11]

The complications of endovascular neurosurgery, occurring inside a closed cranium, can be catastrophic and necessitate rapid initial management by the surgeons, usually by modifying the procedure. The neuroanesthesiologist's role is vital in limiting the severity of complications by managing anticoagulation, maintaining systemic and cerebral hemodynamics, and instituting early neuro-protective measures.[5] Hemorrhagic complications like aneurysm rupture and perforation or dissection of major blood vessels may manifest with severe hemodynamic alterations like abrupt hypertension, widened pulse pressure, bradycardia, tachycardia, and arrhythmias. Aneurysms may rupture during micro-catheter negotiation, coil delivery, or blood pressure surges while guide wire or catheters can induce perforation/dissection of the feeding artery. Reversal of heparin action with protamine (1:1 ratio), lowering of ABP,

initiation of neuro-protective measures, rapid packing of the ruptured aneurysm with additional coils by the surgeon, emergency craniotomy and aneurysm clipping if coil packing fails, and venticulostomy if acute hydrocephalus develops are among the urgently deployed treatment measures.[2,5] Postoperative ventilation following aneurysm rupture helps to manage the adverse consequences like cerebral edema, hematoma, and acute hydrocephalus. Small vessel perforations may be treated conservatively or the catheter itself may be used to occlude the perforation. We observed a 2.5% incidence of aneurysm rupture during coiling, which is comparable to earlier reported incidence of 1.69 to 8.8%.[12-17]

Hyperemic complications may occur after AVM embolization secondary to "normal perfusion pressure breakthrough hyperemia" or to "occlusive hyperemia" due to venous thrombosis, and can lead to cerebral edema and hemorrhage during or after surgery;[5,6,11] achieving good systemic blood pressure control is important here. Thromboembolism, vasospasm, coil displacement causing parent artery compromise (incidence ~ 2.9%), and coil fracture are among the occlusive complications causing serious postoperative problems like thromboembolic stroke, cerebral ischemia/infarction, cerebral edema, and death.[5,14] Despite systemic heparinization, thrombi can develop either during guide wire manipulations inside the catheter or during insertion and withdrawal of coils and stents. Disruption of an atherosclerotic plaque by the catheter or guide wire or rare coagulation disorders like heparin-induced thrombocytopenia (HIT) and anti-phospholipid antibody syndrome can also cause occlusive complications.[18] Quick-acting glues and particulate material used for AVM embolization can pass into the systemic circulation causing inadvertent embolization of the normal brain vasculature;[6] pulmonary embolism from particulate material is also reported.[19] Minimizing guide wire manipulations and continuous catheter flushing with heparinized saline are useful preventive measures. Treatment of thromboembolic complications includes deploying deliberate hypertension, mechanical lysis of angiographically visible thrombi by mechanical devices or saline infusion, use of thrombolytic agents like local tissue plasminogen activator or local and IV antiplatelet agent Abciximab (GPIIb/IIIa inhibitor), and institution of urgent neuroprotection.[5] Malpositioned coils compromising the parent artery can be retrieved or repositioned by the endovascular method itself and rarely require craniotomy. Aneurysm coiling is reported to have a 1.69 to 5% incidence of thromboembolic complications[5,13-15] while AVM embolization has a 1 to 8% incidence of potentially fatal ischemic/hemorrhagic complications;[20] our 4.16% incidence of thromboembolism is comparable to these reports. Vasospasm is a commonly encountered complication of aneurysm coiling and if untreated, can result in delayed awakening from anesthesia and more importantly, postoperative neurological deterioration. Triple-H therapy (hypertension, hypervolemia and hemodilution) with target mean ABP ~20 to 30% above baseline, hematocrit <30% and CVP ~8-12 mmHg is often deployed for treatment, though a good outcome is unproven and risks of therapy are well-recognized.[2,9,21] Use of intra-arterial nimodipine, nicardipine, and papaverine have shown clinical improvement; however, the latter has a transient action and is associated with multiple side effects.[5] Balloon angioplasty, if performed within 2 hours of symptomatic ischemia, is an effective treatment method for vasospasm.[2] The incidence of intra-procedure vasospasm in patients undergoing aneurysm coiling has been reported variously as 2.94%,[14] 5.94%,[12] 9.52%,[22] and 9%,[18] and of post-procedure vasospasm as 6%;[18] we observed 6.67% intra-procedure and 7.08% post-procedure vasospasm.

Abrupt perioperative hemodynamic alterations in ABP, HR, and heart rhythm may signify development of serious complications like intracranial hemorrhage and raised intracranial tension, though transient bradycardia, hypotension, and even asystole may also be normal physiological responses in carotid artery procedures secondary to carotid body stimulation. Reported incidence of hemodynamic perturbations during aneurysm coiling has ranged from 4 to 6%.[12,18] Management requires temporarily ceasing surgical stimulation, treating the underlying cause, and using cardiac drugs and fluids as indicated.[7] Hypotension is especially worrisome in patients with bilateral internal carotid occlusion, severe vertebro-basilar disease, or additional intracranial stenosis; preoperative placement of transcutaneous pacing leads may be considered for prevention.

Besides the above problems, non-neurological complications of endovascular neurosurgery also need to be considered. Fatal contrast reactions are known and can be prevented by using non-ionic contrasts (Iohexol) and pre-treatment of predisposed patients with anti-histaminics and steroids; protamine use can also lead to anaphylaxis and acute pulmonary hypertension.[2] Early postoperative contrast nephropathy can develop secondary to large volumes of high osmolar contrast, volume depletion, nephrotoxic drugs, and pre-existing renal disease and diabetes mellitus. Preventive measures include maintaining good hydration and using small, diluted volumes of contrast; combined use of isotonic sodium bicarbonate and N-acetylcysteine may be beneficial.[2,5]

Mismanaged heparin anticoagulation can lead to disastrous consequences like cerebral thromboembolism or hemorrhage; perioperative serial ACT monitoring is hence valuable; direct thrombin inhibitor Argatroban holds promise in patients predisposed to HIT.[10] Patients with SAH are already prone to develop electrolyte abnormalities (hyponatremia, hypokalemia, hypocalcemia, and hypomagnesemia) secondary to inappropriate antidiuretic hormone secretion or cerebral salt wasting

syndrome.[2,8] Use of large amounts of irrigation fluid can further lead to electrolyte imbalance and fluid overload with deleterious cardiac and respiratory effects, especially in elderly patients and those with prior cardiac disease. Though CVP monitoring is not strongly advocated in endovascular neurosurgery,[8] we find its routine use beneficial in monitoring and managing intraoperative fluid therapy. Hypothermia can develop due to the often low ambient temperatures of the radiology rooms, continuous fluid infusions, and incomplete patient cover due to requirement for groin exposure; use of pre-warmed infusions and IV fluids and heated patient mattresses are helpful in maintaining normothermia. Though hypothermia has been used for cerebral neuro-protection for many decades, there is no conclusive human evidence to support its use in SAH;[8] conversely, hypothermia can cause delayed awakening from anesthesia and impede early post-surgery neurological evaluation. Delayed awakening can also develop secondary to electrolyte abnormalities or intra-procedure neurological complications. Delayed emergence was reported in 2% patients secondary to intracranial bleeding by Lai *et al*.,[17] while we report a 6.67% delayed emergence mostly due to hypothermia and vasospasm. Elective postoperative ventilation may be warranted in patients with neurological insults like aneurysm rupture, severe vasospasm, or thromboembolic episodes. Early postoperative problems include nausea, vomiting, groin hemorrhage/hematoma, fluid-electrolyte imbalance, pulmonary edema, osmotic diuresis leading to dehydration, transient ischemic attacks, stroke, seizures, neurological deterioration, infection/meningitis, and even death.[5]

In addition to the procedure-related complications, a good knowledge of radiation hazards during endovascular neurosurgery is also important for the neuroanesthesiologist.[1,2,8] Some of the recommended protective measures against radiation hazards include limiting maximum total body dose to 20 mSv/year, using glass lead screens, thyroid collars, and lead aprons (with minimum 0.5 mm lead equivalent thickness), stationing of personnel at least 4 feet away from radiation source, operating the fluoroscope at the lowest dose rate that yields adequate images, and monitoring radiation time and periodically notifying the surgeon.[2,8]

Anesthesia in the neuroradiology suites can be risky due to their remote location, small working areas with inadequate monitoring, anesthesia and resuscitation facilities, and insufficient-trained staff to handle neuro-emergencies.[7,23] Placing neuroradiology suites near the neurosurgery OT and ICU[7] and presence of an experienced and permanent neuroanesthesia team in these suites has been advocated.[24,25] At our institute, these procedures are done in the familiar set-up of our well-equipped neurosurgery OT in close proximity to the ICU, which also obviates the risks of intra-hospital transport of sick patients to far-away radiology suites.[7] Also, unlike in conventional neurosurgery, procedural complications in endovascular neurosurgery usually develop without overt manifestations. The hemorrhagic complications in particular may not be accompanied with visible excessive blood loss or circulatory collapse, and hence may be easily missed by the anesthesiologist if there is a lack of communication with the operator. We believe when neurosurgeons, who are already well-versed with complications of intracranial operations, perform endovascular procedures like in our set-up, there is a better management of the complications because of a good mutual understanding and communication between the surgeons and anesthesiologists.

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Conflicts of interest

There are no conflicts of interest.

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