REVIEW ARTICLE

Anesthetic Management of Intraoperative Aneurysm Rupture: A Narrative Review

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Intraoperative aneurysm rupture (IOAR) is a serious and potentially life-threatening complication that can occur during intracranial aneurysm surgery. Understanding the perioperative risk factor contributing to IOAR may help prevention and better handle if it occurs. The anesthesiologist should be familiar with the management of IOAR to facilitate bleeding control and improve perioperative outcome. There is controversy surrounding the management of IOAR. The main goal of management includes promptly securing for control of bleeding as well as maintaining adequate cerebral perfusion and providing neuroprotection. The purpose of the present article was to review the anesthetic management during IOAR. The authors focused on the hemodynamic and intracranial pressure control for optimizing cerebral perfusion, neuroprotection during temporary arterial occlusion, monitoring of cerebral ischemia, and recent techniques for controlling bleeding.

Keywords: Intraoperative aneurysm rupture; Anesthetic management; Intracranial aneurysm; Surgical clipping

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Intraoperative aneurysm rupture (IOAR) is a potentially lethal complication in patients undergoing aneurysm clipping that associate with unfavorable outcome and high morbidity and mortality[1-3]. The incidence of IOAR ranges from 1.2% to 61%, varies with size, location of the aneurysm, surgical techniques, neurosurgeons' experience, anesthetic technique, and different definitions[2,4-6]. IOAR might occur at any time during the intraoperative procedure and is frightening for both the surgeon and the anesthesiologist. Anesthetic management may be complicated if IOAR occurred, thus, the prompt early diagnosis and appropriate management are as important as the optimal intraoperative anesthetic management.

The present review primarily addresses anesthetic management of IOAR during surgical intracranial aneurysm clipping.

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Risk factors for IOAR

The understanding of the various risk factors that may contribute to IOAR is important. Studies have reported the factors associated with IOAR (Table 1). However, the association remains unclear[2,5,7-9]. The anesthesiologists should focus on understanding the natural course and morphology of disease, including factors such as co-morbidities (hypertension, chronic obstructive pulmonary disease, coronary artery disease, and hyperlipidemia), size, type, and location of the aneurysm. Additionally, it is crucial to consider the factors that influence changes in the transmural pressure (TMP) of aneurysms.

The anterior communicating artery and internal carotid artery aneurysms are more liable to rupture intraoperatively[8,9]. The rate of IOAR was greater in ruptured aneurysms and irregular aneurysm shape[1,2,10,11]. Although IOAR may occur at any time during the procedure, it mostly occurs during microdissection of aneurysm and artery adhering to the aneurysm[5,8,10,12].

The rate of IOAR during endovascular coil embolization is lower than open surgery, however, the chances of intraoperative rupture were higher in a younger patient, chronic obstructive pulmonary disease, middle cerebral artery (MCA) aneurysm, and very small aneurysms[7,13,14]. During the procedure, IOAR can occur if the catheter or coils caused damage to the vascular walls during the insertion process.

Table 1. Perioperative risk factors contributing intraoperative aneurysm rupture

Preoperative factors	
Comorbidities	• Smoking/COPD • Hyperlipidemia/CAD • Hypertension
Aneurysm-related factors	• Large size (clipping), very small size (coiling) • Shape: blister-like aneurysms • Morphology: multilobed/irregular • Location: Acom, Pcom, ICA • Rupture status: ruptured > unruptured
Anesthetic factors	• Pain: skull pin fixation, incision • Light anesthesia • Coughing/gagging • Intubation/extubation response
Fluctuations in TMP	• High blood pressure • Sudden changes in ICP: large and bolus mannitol, hyperventilation, CSF drain
Intraprocedural factors	• Experience of surgeon • Dural opening, arachnoid opening • Hematoma removal • Brain retraction • Dissection of the aneurysm • Dissection of the artery adhering to aneurysm • Clip application • Coil protrusion • Microcatheter perforation

COPD=chronic obstructive pulmonary disease; CAD=coronary artery disease; Acom=anterior communicating artery; Pcom=posterior communicating artery; ICA=internal carotid artery; TMP=transmural pressure; ICP=intracranial pressure; CSF=cerebrospinal fluid

Anesthetic related intraoperative aneurysm rupture and its prevention

Prevention of IOAR is essential to increase survival rate and improve the outcome in patients undergoing aneurysm surgery. IOAR might cause massive blood loss and lead to irreversible brain damage. Moreover, it is difficult to clip the aneurysm due to bleeding in the surgical field and may cause secondary damage during the procedure.

The sudden changes in hemodynamics during induction of anesthesia or surgical manipulation may be responsible for IOAR. An abrupt increase in TMP such as acute increases or decreases in blood pressure and intracranial pressure (ICP), should be avoided. The anesthetist should be aware of anesthesia related factors such as intubation response or light anesthesia, coughing or gagging, rapid boluses of mannitol, and excessive hyperventilation. A gradual increase of pain due to light anesthetic depth or inadequate pain control during surgical manipulation such as skull pin fixation and skin incision can increase mean arterial pressure (MAP). A sudden decrease in ICP caused by rapid mannitol administration, hyperventilation, or rapid cerebrospinal fluid (CSF) drainage can lead to IOAR. However, there is limited data to support ICP fluctuation as a major cause of IOAR. Prophylaxis for excessive sympathetic stimulation throughout operation should be instituted by increasing depth of anesthesia, adequate pain control, and administration of antihypertensive agents, such as esmolol, labetalol, or nicardipine.

IOAR during induction of anesthesia occurs infrequently. However, if it does happen, it would associate with poor outcomes and high mortality rate[14-16]. During induction and intubation, anesthetist should balance between MAP, ICP, and TMP. In general, the patient blood pressure should be maintained at 20% reduction of baseline to prevent abrupt increased TMP. However, in patients with poor clinical grades, signs and symptoms of cerebral ischemia, or who have impaired autoregulation, they may not tolerate transient hypotension. Therefore, the duration and degree of blood pressure decrease should be kept minimum[17]. A titratable anesthetic drug should be used to balance the risk of premature aneurysm rupture and brain ischemia.

Regarding the endovascular procedure, to minimize the risk of IOAR, the patient's movement should be restricted. General anesthesia with endotracheal intubation is preferred for controlled ventilation and complete muscle paralysis. Deliberately lowering blood pressure to minimize blood flow into the aneurysm may be employed during coiling procedure to reduce the risk of IOAR.

Detection of aneurysm rupture

The intraoperative premature rupture of aneurysms may be diagnosed by a gradual unexplained hypertension or abrupt onset of bradycardia. As a consequence of a rise in ICP and herniation, the dilation of pupils, hemodynamic derangement, and early signs of ischemic on neurophysiologic monitoring can be detected.

After craniotomy, IOAR is suspected if the dura appeared suddenly tense, became dark in color or the brain became stony hard and bulging. However, the factors that could contribute to raised ICP have to be excluded.

In coiling embolization, the extravasation of the contrast media is seen when the intraprocedural rupture occurs.

Anesthetic management of intraoperative aneurysm rupture

For premature aneurysm rupture before dural

Table 2. Anesthetic management of Intraoperative aneurysm rupture

Hemodynamics	• Maintenance of normovolemia and normal circulating blood volume • Maintain normotension • Transient control hypotension to facilitate surgical control (MAP to 40 to 50 mmHg)
Reduction of ICP	• Maintain depth of anesthesia with intravenous anesthetics • Discontinue volatile and nitrous oxide • Mild to normoventilation (30 to 35 mmHg) • Brief period of $PaCO_2$ less than 30 mmHg if other techniques fail • Correct other cause of increase ICP (hypoxia, hypercarbia, high airway pressure, hyperthermia, etc.) • Optimize positioning with slightly head elevation 10 to 30 degree • CSF drainages
Neuroprotection	• Optimize systemic physiology and glycemic control (blood glucose less than 180 mg/dL) • Avoidance of hyperthermia • Maintain hemoglobin concentration above 8 to 10 g/dL • Minimize temporary clipping time
During temporary arterial clipping	• Use 100% oxygen • Induced hypertension (10% to 20% above baseline blood pressure) • Use intravenous anesthetics titrating to achieve electroencephalogram burst suppression
Facilitate surgical exposure and clip placement	• Adenosine-induced cardiac arrest • Rapid ventricular pacing

MAP=mean arterial pressure; ICP=intracranial pressure; CSF=cerebrospinal fluid

opening, the main focus is to rapidly reduce ICP as well as protection of the brain. Good communication between the surgical and anesthesia team is very important. The primary goal is to promptly secure the aneurysm for control of bleeding together with correcting the hemodynamic derangement. The anesthetic management is described in Table 2.

In coiling embolization of an aneurysm, the management in intraprocedural rupture include 1) immediately assess the patient's condition and vital signs to determine the severity of rupture and the extent of the bleeding, 2) ensure adequate oxygenation and ventilation, 3) rapidly reverse anticoagulant such as protamine, if indicated, 4) maintain cerebral perfusion and minimize ischemic injury, 5) control increased ICP and maintain hemodynamic stability, especially blood pressure, and 6) collaborate closely with the surgical team and prepare for possible emergent conversion to open surgery. With the use of intravascular stents, the antiplatelet agents are commonly administered preprocedural and during the procedure. The prompt reversal of antiplatelet activity can be attempted by platelet transfusion if an intraprocedural rupture occurs.

Hemodynamic management

In the setting of IOAR, hypotension combined with hypovolemia may result in cerebral ischemia. The goal of hemodynamic management in IOAR is maintenance of intravascular volume with isotonic fluids.

Preoperative typed and crossmatch blood for two units should be available. Regarding the transfusion threshold, there were no randomized controlled trials comparing liberal with hemoglobin (Hb) level at 10 to 12 g/dL and restrictive transfusion trigger with Hb level at 7 to 9 g/dL in subarachnoid hemorrhage patients. However, the Hb concentration should be maintained above 10 g/dL to reduce the risk of cerebral vasospasm and improve clinical outcomes[18,19].

Blood pressure management during aneurysm rupture is poorly defined. Literature recommends that MAP should be transiently decreased to 40 to 50 mmHg or lower to reduce bleeding and facilitate surgical control[17]. However, the effect on cerebral perfusion can worsen cerebral ischemia particularly in patients with impaired cerebral autoregulation. A brief period of deep hypotension is allowed when the bleeding is uncontrollable. Moreover, if a temporary arterial occlusion was applied or clinically relevant blood loss occurred, maintenance of normotension may be appropriate[20].

In coiling embolization of an aneurysm, maintaining normal blood pressure is more important to ensure adequate cerebral perfusion while minimizing the risk of increased bleeding. The extravasated blood can increase ICP and high blood pressure can enlarge bleeding and make it worse.

Intracranial pressure reduction and brain relaxation

In the patients with brain edema and increased ICP, providing reduction of ICP and brain relaxation strategies should be applied to decrease the risk of brain injury and facilitate securing the aneurysm. The factors to increase ICP have to be instituted, such as light anesthesia or higher concentration of minimum alveolar concentration (MAC) of volatile anesthetic, hypoxia, hypercarbia, high airway pressure or central venous pressure, or hyperthermia.

To maintain of adequate depth of anesthesia, intravenous anesthetics should be considered, and inhalation anesthetics and nitrous oxide (N_2O) should be discontinued. Thiopental or propofol can be used to provide both a reduction in cerebral blood flow and cerebral metabolism and may also produce brain protection. Propofol infusion rate 6 to 12 mg/kg/hour or intermittent low doses of thiopental at 1 to 3 mg/kg follow by infusion rate 4 to 5 mg/kg/hour is useful in lower ICP and brain bulging[17,21].

A brief period of moderate to severe hyperventilation may be reasonable for prompt reduction in ICP and facilitate surgical exposure if other techniques failed to reduce ICP. The partial pressure of carbon dioxide ($PaCO_2$) at 25 to 30 mmHg during opening of the dura and 30 to 35 mmHg before opening the dura may be considered[17,22,23]. Owing to the theoretical concern of hyperventilation-induced vasoconstriction, intraoperative monitoring such as cerebral oxygenation may be useful in assessing the adequacy of cerebral perfusion[24].

Other strategies can be applied if the brain swelling did not respond to the aforementioned strategies, for example, optimal positioning and drainage of CSF. The head elevation at 10 to 30 degrees position with avoidance of excessive flexion or rotation of the neck is optimized to facilitate cerebral venous return. To decrease the CSF volume, the lumbar drain or ventriculostomy may be performed before surgery. An external ventricular drain (EVD) can also be used for ICP monitoring, and CSF drainage for facilitating brain relaxation while aneurysm rupture occurs perioperatively. The intraoperative ventriculostomy by direct ventricular puncture after dural opening is a safe technique that allows an immediate brain relaxation, removes bloody CSF, and provides a port for ICP monitoring[25,26].

Neuroprotection

Although strategies for prophylactic and therapeutic neuroprotective have been explored, the effectiveness in human studies are inconclusive and none have been clearly shown to improve the outcome. Additionally, there has not been any study on the neuroprotective effect during IOAR.

The neuroprotection should be initiated early before the occurrence of brain ischemia. A traditional technique to gain control over an aneurysm rupture is temporary arterial occlusion.

The potential strategies to provide neuroprotection from brain ischemia during the temporary arterial occlusion or hemodynamic derangement include:

1) The duration of temporary clipping should not be more than 20 minutes[21],

2) The maintenance of hyperoxygenation with 100% oxygen,

3) The maintenance of adequate cerebral perfusion pressure by induced hypertension at 10% to 20% above preinduction baseline, to recruit collateral blood flow if bleeding is controlled,

4) The reduction of cerebral metabolism if temporary clipping was required for more than 10 minutes[21,27]. The intravenous administration of propofol, thiopental, or etomidate titrated to achieve electroencephalogram (EEG) burst suppression may be an option[21,27,28],

5) The maintenance of blood glucose 80 to 180 mg/dL[31,32]. Of note, the glucose concentrations greater than 152 mg/dL has been associated with long-term gross neurological deficits[31],

6) The avoidance of hyperthermia. Although induced mild hypothermia at 32 to 35°C may be an option in poor grade patients, its routine use is not recommended and there is a lack of data regarding the role of hypothermia during IOAR.

Even though, there are no evidence of pharmacological brain protection and mild hypothermia during temporary occlusion, some non-pharmacological strategies for optimizing the global cerebral homeostasis such as adequate control of MAP at more than 80 mmHg, normoglycemia, maintain intraoperative Hb levels at 8 to 10 g/dL, and level of PaO_2 and $PaCO_2$ can be beneficial[32,33].

Intraoperative monitoring

During surgery, a reduction of systemic blood pressure might be performed to control bleeding and facilitate perianeurysmal dissection and permanent occlusion of aneurysms. Moreover, the prolonged application of temporary arterial clipping (TC) can result in ischemic complications, especially in the injured brain[34]. In addition to standard monitoring, intraoperative modalities may be used for detection

of IOAR and early recognition of cerebral ischemia.

Invasive ICP monitoring, whether it be preoperative EVD or intraoperative ventriculostomy, can be employed to assess the patient's cerebral hemodynamics and guide management.

Cerebral hypoxia and ischemia are associated with changes in electrical activity of the brain. Neurophysiologic monitoring involving somatosensory-evoked potentials (SSEPs) and motor-evoked potentials (MEPs) helps detect reversible ischemia and allows timely corrective measures, such as surgical technique adjustment and hemodynamic augmentation to prevent postoperative neurological impairment. However, the benefit remains to be defined due to its diagnostic accuracy being unreliably recorded, and the influence of general anesthesia[35,36]. EEG can be used to monitor brain activity and detect signs of ischemia. However, when using EEG to titrate neuroprotectants to the point of burst suppression, the ability to detect cerebral ischemia may be obscured.

The bispectral index (BIS) monitoring may be used to identify the lower limit of cerebral autoregulation during aneurysm surgery and maintain the level of blood pressure to prevent ischemia insult[37], in addition to monitoring the depth of anesthesia.

Transcranial doppler ultrasound (TCD) is a valuable tool for evaluating cerebral blood flow and can be used as an indirect method for detecting cerebral ischemia[38,39]. The change in the velocity flow pattern shows pathological morphology. In the case of IOAR, TCD displays a retrograde flow during diastole and is consistent with a high ICP[40].

Continuous jugular venous oxygen saturation ($SjvO_2$) monitoring may help to determine the adequate cerebral perfusion pressure and early recognition of cerebral ischemia associated with hyperventilation, blood pressure management, and temporary feeding artery occlusion[17]. The $SjvO_2$ should be maintain at level 55% to 75%. Near-infrared spectroscopy (NIRS) that is non-invasive cerebral oximetry, has been used for detection of cerebral ischemic/hypoxic events during surgery. The intraoperative abrupt desaturation deserves attention as it indicates the early sign of IOAR[41,42].

Despite the aforementioned methods having a useful role to play in the early detection of cerebral ischemia and representing the sign of IOAR, the use as a routine monitor specifically for aneurysm surgery is not warranted.

Method of transient flow arrest

There are techniques to decrease the risk of IOAR and decrease bleeding during IOAR. Examples of this include TC of the proximal vessel, temporary cross-clamping of the extracranial carotid artery in the neck, endovascular balloon occlusion with suction, and cardiac standstill[43].

TC is a traditional technique for prevention and dealing with IOAR[44-46]. Nowadays, alternatives including adenosine-induced cardiac arrest (AiCA)[47-49] and rapid ventricular pacing (RVP)[50-52] have been used to assist in control sudden IOAR when temporary occlusion is not achievable (Table 3).

Adenosine-induced cardiac arrest

AiCA has been used as an option in cases where temporary clipping is infeasible or to facilitate bleeding control when unexpected IOAR occurs. The administration of adenosine can induce a brief asystole that is adequate for clearing the surgical field and allows the implementation of definitive clipping or TC to secure the aneurysms. Studies have demonstrated the successful use of AiCA, both in cases with or without IOAR[47-49,52-58]. It helps decrease intra-aneurysmal tension, reduces the need for TC and the overall duration of TC use, and minimizes blood loss if IOAR occurred[57].

AiCA is a handy tool and has a rapid onset and very short half-life, with negative effects on sinoatrial and atrioventricular (AV) node. After a bolus injection, AV node is transient blocked, leading to bradycardia, sinus pauses, and cardiac arrest. After that, it returns to baseline in 20 to 30 seconds.

An estimated dose of 0.2 to 0.4 mg/kg ideal body weight is recommended to provide a short period of asystole that is sufficient for assessing the rupture site and bleeding control[56,58-59]. Repetitive dose of adenosine can be administered as requested by surgeon. Nevertheless, heart rate and blood pressure should be allowed to return to baseline between doses.

Although the use of AiCA has an acceptable safety profile, it should be used cautiously in patients with a history of severe coronary artery disease, severe reactive airway disease, and pre-existing cardiac connection abnormalities[43]. The transcutaneous pacing pads should be placed as a precaution for prolonged bradycardia or asystole. Moreover, in the setting of IOAR, the risk of AiCA-induced global cerebral ischemia, albeit of transient duration of hypotension, must be weighed against the benefits of its use.

Table 3. Methods for aneurysm softening and facilitating permanent clip placement

	Action	Technique	Advantage	Disadvantage
Temporary clipping	• Obstruction of the parent vessel to reduce blood flow through the aneurysm	• One or multiple clips	• Handy tool • Simple to use • Non-invasive • Repeatable	• Timing-related cerebral ischemia • Thromboembolic stroke • Vessel injury • Not feasible in some situations: large or deep-seated aneurysm
Adenosine-induced cardiac arrest	• Negative effect on SA and AV node • Rapid onset and short duration of action causing bradycardia and brief asystole	• Place transcutaneous pacing pads before operation • Prepare antecubital large-bore IV line for adenosine administration • Dose 0.2 to 0.4 mg/kg IBW	• Short half-life and recovery of normal circulation • Non-invasive • Synergy with temporary clipping, especially during IOAR • Repeatable after recovery from initial dose • Decrease risk of premature rupture	• Need close communication with the surgeon • Precaution in patients with coronary artery disease or abnormalities of cardiac conduction system, reactive airway disease • Unpredictable response
Rapid ventricular pacing	• Enforce ventricular tachycardia and ventricular filling is compromised because of the high HR and absent AV synchrony	• Preoperative cardiologist work up of the patient • Place external defibrillating pads • Prepare anti-arrhythmia drugs • Introduce bipolar pacing electrode through the internal jugular vein into the right ventricle under fluoroscopy • Dose 130 to 160 beats/minute, titrate to the desired effect	• Better control of start time and length of pacing • Predictable flow/pressure reduction • Repeatable after recovery of hemodynamics • Decrease risk of premature rupture	• Need an experienced neurosurgery and anesthesiologist team • Reserve for selected elective case and highly specialized center • Not suitable in patients with coronary heart disease and cardiac arrhythmias • Complicated-relation with pacing probes

SA=sinoatrial node; AV=atrioventricular node; IV=intravenous; IBW=ideal body weight; IOAR=intraoperative aneurysm rupture; HR=heart rate

Rapid ventricular pacing

The use of RVP has been described as an advanced technique to facilitate clip reconstruction of complex aneurysm surgery[50,51,60-62], and Khan SA et al. reported the first use of RVP assisted hypotension to control sudden intraoperative bleeding when temporary arterial occlusion was not achievable[51].

RVP induces ventricular tachycardia and reduces ventricular filling, leading to decrease blood pressure with near flow arrest for a short period of time during dissection or rupture of the aneurysm. Compared with adenosine, RVP has a time predictable, which is immediate, and significantly lowers the blood pressure at the start time and normal sinus rhythm returns instantaneously, without prolonged hypotension after RVP is terminated. Global cerebral parenchyma is still perfused, and cerebral oxygenation is not affected[61,63].

The patients considered for RVP during surgery should be evaluated by cardiologist before surgery. A bipolar pacing electrode is advanced through the internal jugular or subclavian vein into the right ventricle, by an experienced anesthetist. The external defibrillating pads are applied to the chest. RVP is initiated upon the neurosurgeon's request. The pacing rate is started at 130 to 160 beats/minute and titrated by a reduction of blood pressure. The frequency and duration of RVP have to be limited to ensure adequate recovery of left ventricular function and hemodynamics prior to further pacing.

The complication of RVP is very rare, which is mostly related to the placement and use of the pacing electrodes, such as cardiac perforation or cardiac tamponade. The other complications including atrial fibrillation and ventricular arrhythmia have been reported, which are transient and resolved after adequate intraoperative measures[60,61]. Noteworthy, caution should be taken with the patients with severe left ventricular dysfunction, coronary heart disease, cardiac arrhythmias, and severe valvular heart disease as it may increase the risk of myocardial ischemia and ventricular arrhythmias, especially in concomitant hypovolemia[52,61]. In addition, this technique should be considered in the cardiac centers for handling emergency situations.

Because there are limited case reports of the use of RVP during IOAR, its safety in case of IOAR needs to be further investigated.

Postoperative period

Patients experiencing IOAR might maintain intubation and require prolonged mechanical ventilator, owing to cerebral infarction. However, the tracheal extubation at the end of procedure should

be evaluated on an individual basis. Likewise, if the patient's preoperative status was unstable, the surgery was prolonged and difficult, the patient had a brain swelling, or after procedure in infratentorial or posterior fossa, these patients should be intubated and mechanically ventilated in an intensive care unit. The intubated patient should be kept sedated with short-acting agents such as propofol 25 to 100 mcg/kg/minute. The standard systemic examination, standard monitoring, and multimodal neuromonitoring should be performed for early recognition and management of any complications such as aneurysm rebleeding, cerebral infarction, symptomatic vasospasm, hydrocephalus, and electrolyte imbalance.

Conclusion

The goal of management of IOAR includes securing promptly the aneurysm for control of the bleeding as well as control of hemodynamic and ICP, and providing neuroprotection. In general, smooth induction and extubation, maintenance of normotension and intravascular volume, and optimizing global cerebral homeostasis are required. Recent techniques including AiCA and RVP have been used to assist in control of bleeding due to IOAR, which is determined by the safety profile. There are controversies in management of IOAR. Further study based on the clinical trials and high-quality data, is still required for the dilemma affecting the perioperative management.

What is already known on this topic?

IOAR is a serious complication that can occur during intracranial aneurysm surgery. The primary goal is promptly securing the aneurysm, as well as, protection of brain ischemia, and handling of hemodynamic derangement.

What does this study add?

This article reviewed the perioperative risk factors contributing IOAR and the anesthetic management during IOAR, including hemodynamic and ICP control for optimizing cerebral perfusion, neuroprotection during temporary clipping, and monitoring of cerebral ischemia. Moreover, the authors reviewed the recent techniques for dealing with IOAR. AiCA and RVP have been used to assist in control of bleeding with safety profile, in addition to temporary clipping, which is the traditional method.

Conflicts of interest

The authors declare no conflict of interest.

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