Adenosine-induced Flow Arrest to Facilitate Control of a Basilar Artery Injury during Craniotomy for Petroclival Meningioma: A Case Report

Carlo D. Monteblanco, MD, Karl Matthew C. Sy Su, MD and Geraldine Raphaela B. Jose, MD

Division of Neuroanesthesia, Department of Anesthesiology, Philippine General Hospital, University of the Philippines Manila

ABSTRACT

Intraoperative hemorrhage is a life-threatening complication during neurosurgery, especially in posterior fossa surgery, where critical vasculature and brain structures are present. Adenosine has been used in neurovascular surgery, particularly in the management of intraoperative aneurysm rupture and hemorrhage for its ability to produce transient flow arrest. This case report describes a novel application for adenosine, in which adenosine-induced flow arrest was used to facilitate control of a vascular injury sustained during meningioma surgery.

A 46-year-old female diagnosed with a petroclival meningioma after presenting with a several-month history of headache, dizziness, and loss of balance underwent craniotomy and excision of the meningioma. The patient received general endotracheal anesthesia, and was maintained on remifentanil, propofol, and low-dose sevoflurane. During posterior excision of the meningioma, the basilar artery was inadvertently lacerated, resulting in significant blood loss. Adenosine was given through a subclavian catheter, and produced severe bradycardia, hypotension, then asystole for approximately 50 seconds. Adenosine-induced flow arrest allowed for initial control of the vascular injury through coagulation, while further hemostasis was achieved through hemostatic agents and muscle tamponade.

This case report demonstrates the usefulness of adenosine-induced flow arrest in the management of intraoperative hemorrhage from an intracranial vascular injury. Adenosine-induced flow arrest has documented safety and efficacy in other neurosurgical applications. As the management of intraoperative hemorrhage is an essential component of neuroanesthesia, this technique may be considered in similar circumstances of major vascular injury to facilitate hemostasis.

Keywords: adenosine, basilar artery, meningioma, case report

Poster presentation – 8th Congress of Asian Society for Neuroanesthesia and Critical Care, March 1, 2025, InterContinental Bangkok Hotel, Thailand.

eISSN 2094-9278 (Online)
Published: March 31, 2026
https://doi.org/10.47895/amp.vi0.13711

Corresponding author: Carlo D. Monteblanco, MD
Division of Neuroanesthesia
Department of Anesthesiology
Philippine General Hospital
University of the Philippines Manila
Taft Avenue, Ermita, Manila 1000, Philippines
Email: cdmonteblanco@alum.up.edu.ph
ORCiD: https://orcid.org/0009-0004-8576-5009

INTRODUCTION

Intraoperative hemorrhage during neurosurgery is an emergent and life-threatening adverse event that may worsen perioperative morbidity, neurologic deficits, and surgical outcomes.[1] Excessive intraoperative blood loss is associated with various complications, including hemodynamic insta-bility, anemia, coagulation disorders, hypothermia, systemic inflammation, ischemic stroke, and intracranial hemorrhage.[1,2] Furthermore, intraoperative blood loss during intracranial surgery may result in prolonged duration of mechanical ventilation, prolonged hospital stay, and increased risk of mortality.[2,3]

Petroclival meningioma surgery is considered one of the most challenging procedures in neurosurgery.[4] These meningiomas tend to involve the cranial nerves, and compress and displace the brainstem and the basilar artery.[4] Surgical difficulties arise from tumor size, proximity of the brainstem, involvement of cranial nerves and vessels, and the necessity of

brain retraction.[4] Because of these, intraoperative hemorrhage is a potential complication of petroclival meningioma surgery, along with cerebrospinal fluid leakage, brainstem ischemia, cranial nerve injury, and cerebral infarction.[4]

Adenosine has been used for decades in cardiac electrophysiology to manage certain arrhythmias, but has seen growing application in neuroanesthesia for its ability to induce transient asystole and flow arrest.[5] Adenosine is currently used in neurovascular surgery, particularly in aneurysm surgery to manage intraoperative aneurysm rupture and hemorrhage, where bleeding can become substantial and difficult to control.[5] In these instances, adenosine-induced flow arrest produces a profound reduction in cerebral blood flow that assists with hemostasis and clip application.[5]

In this case report, a novel and valuable application for adenosine in neuroanesthesia is described. Here, adenosine-induced flow arrest was successfully used to assist hemostasis and facilitate control of a basilar artery injury incurred during craniotomy and excision of a petroclival meningioma.

CASE PRESENTATION

A 46-year-old female weighing approximately 55 kg presented to the emergency room with a seven-month history of intermittent headaches with associated dizziness and loss of balance. The patient had no known comorbidities, no known allergies, and no previous surgeries. Family history, and personal and social history were non-contributory. Physical examination revealed vital signs within normal limits, and unremarkable cardiovascular and pulmonary findings. On neurological examination, the patient was awake, oriented, and followed commands. She had no cranial neuropathies, but motor strength was decreased to 3/5, movement against gravity, on the right, while sensation was also decreased to 60% on the right. Airway assessment revealed Mallampati Class II, adequate mouth opening, adequate thyromental distance, and full neck mobility. Imaging revealed a 4.4 x 4.9 x 4.7 cm petroclival mass extending from the prepontine cistern more towards the left until the interpeduncular cistern (Figure 1). Preoperative complete blood count, serum chemistry, coagulation tests, and electrocardiogram were within normal limits. The benefits and potential risks of surgery and anesthesia were discussed with the patient and family, and informed consent was obtained. The patient was then brought in for a left frontotemporal craniotomy, and excision of tumor under general anesthesia.

In the operating room, electrocardiogram leads, blood pressure apparatus, and pulse oximeter were attached. General anesthesia was induced with a remifentanil infusion at 0.2 mcg/kg/min, propofol 70 mg, sevoflurane 2%, and atracurium 30 mg, then the patient was intubated through direct laryngoscopy. After induction, a scalp block using 0.25% bupivacaine and epinephrine was performed. Additional peripheral intravenous access, an arterial line at the radial artery, and a lumbar cerebrospinal fluid drain were then inserted. A right subclavian central venous catheter was also inserted under ultrasound guidance. The patient was positioned supine with the head turned towards the right, and fixed with a skull clamp. General anesthesia was maintained with a remifentanil infusion at 0.1-0.2 mcg/kg/min, propofol infusion at 50-100 mcg/kg/min, and sevoflurane at a minimum alveolar concentration <1.0. Additional doses of atracurium were only given when there were signs of spontaneous respiration.

Intraoperatively, the tumor was noted to be dural-based, extra-axial, originating from the clivus and petrous bone,

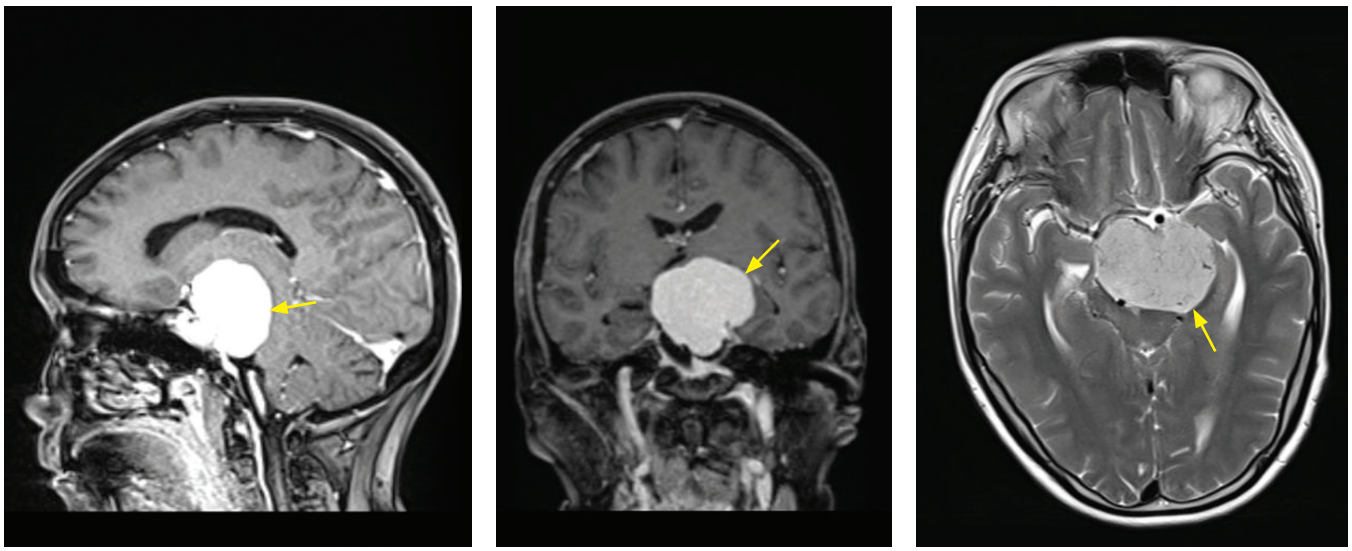

Figure 1. Selected sagittal, coronal, and transverse slices of the contrast-enhanced magnetic resonance imaging showing the petroclival meningioma *(yellow arrows)*.

and in close proximity to the left internal carotid artery and basilar artery. During posterior excision of the meningioma, the basilar artery was inadvertently lacerated, resulting in significant blood loss exceeding 50% of the patient's estimated blood volume and hemodynamic instability with lowest mean arterial pressure of ~50 mmHg. Blood products were immediately requested and transfused to replace blood loss. A norepinephrine infusion was also started and titrated to a highest rate of 0.2 mcg/kg/min to maintain blood pressure. Flooding of the surgical field despite suctioning and tamponade hindered control of the arterial injury. After discussion with the surgical team, it was decided that adenosine-induced flow arrest would be attempted to facilitate control of the vascular injury. Prior to adenosine administration, necessary surgical equipment was readied, suction was prepared, and an external defibrillator was made available. Adenosine was then given in close coordination with the surgical team. A dose of 18 mg or 0.33 mg/kg of adenosine was administered via the subclavian catheter, followed immediately by a normal saline flush. This produced profound bradycardia and hypotension, with mean arterial pressure <30 mmHg, followed by asystole for approximately 50 seconds. During this period of flow arrest, the site of the basilar artery injury was identified, but bleeding was not yet controlled. Return to sinus rhythm was spontaneous after the period of asystole, however, tachycardia and hypotension were pronounced. Heart rate and blood pressure gradually returned to pre-adenosine levels over several minutes. After vital signs had stabilized, another 18 mg of adenosine was given, and again resulted in bradycardia, hypotension, then asystole lasting around 50 seconds. During this second period of flow arrest, initial control of the basilar artery injury was achieved with coagulation. Further hemostasis was established through hemostatic agents and muscle tamponade. Return of sinus rhythm was again spontaneous with tachycardia and hypotension settling to pre-adenosine levels over several minutes. Once the basilar artery injury was controlled, further tumor resection was discontinued, and the procedure was brought to a close.

The patient was not extubated after the procedure owing to significant intraoperative blood loss and hemodynamic instability. Throughout the procedure, approximately 104% of the patient's estimated blood volume was lost for which 1200 ml of packed red blood cells, 1000 ml of fresh frozen plasma, and 200 ml of platelet concentrate were transfused. The patient was transferred to the critical care area of the post-anesthesia care unit for further management and monitoring. Neurological evaluation showed no eye opening, absence of verbal response due to intubation, and withdrawal to painful stimuli, while postoperative imaging revealed edema of the brainstem (Figure 2). On the third postoperative day, the patient developed brainstem dysfunction presenting as dysautonomia followed by progressive hypotension, which eventually led to the demise of the patient.

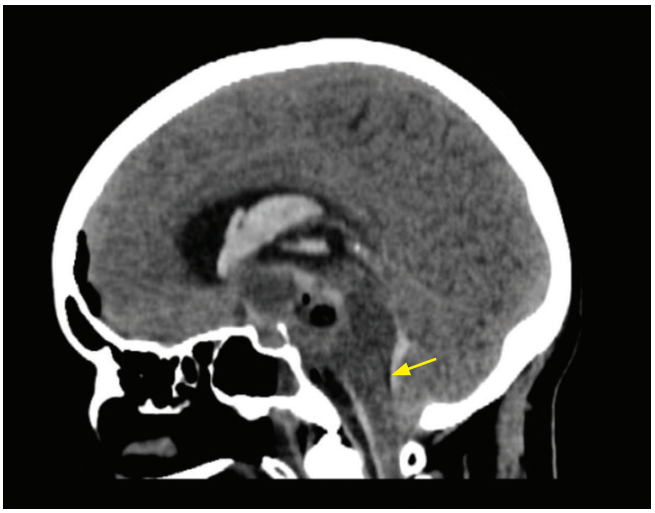

Figure 2. Postoperative contrast-enhanced cranial computed tomography scan showing edema *(yellow arrow)* of the brainstem.

DISCUSSION

Adenosine is an endogenous purine nucleoside composed of a molecule of adenine attached to a ribose sugar through a glycosidic bond.[5,6] Adenosine binds to cardiac A1 receptors and initiates a cascade through activation of adenylyl cyclase to decrease intracellular cyclic adenosine monophosphate and decrease inward calcium conductance.[6,7] This results in decreased sinoatrial node activity, atrioventricular nodal conduction, atrial contractility, and ventricular automaticity.[6,7] These pharmacologic effects produce a dose-dependent reduction in atrial and ventricular electrical activity that manifests as bradycardia, AV nodal blockade, and sinus pauses.[6-8] When administered at a sufficient dose, adenosine can produce transient asystole and flow arrest.[8] Furthermore, the negative chronotropy, inotropy, and dromotropy produce a significant decrease in cardiac output and mean arterial pressure.[6-8] Mean arterial pressure is further reduced by vasodilation in most vascular beds facilitated through binding to A2 receptors.[5,9]

The rationale behind the use of adenosine in this case is comparable to the rationale behind its use in neurovascular surgery, particularly in aneurysm surgery. In aneurysm surgery, adenosine is most commonly used to decrease aneurysm turgor and bulk, minimize the risk of rupture, and facilitate clip application.[5] This is particularly beneficial with aneurysms in unfavorable anatomical locations, such as basilar tip aneurysms, or when a temporary clip may induce an unacceptable risk of ischemia or impede visualization of important structures.[5] Adenosine has also been used in the endovascular management of arteriovenous malformations to reduce flow and prevent venous migration of the embolic agent.[10,11] More importantly, adenosine may be used in the event of intraoperative aneurysm or arteriovenous

malformation rupture, where bleeding may become significant.[7,12,13] This is an emergent and life-threatening complication of neurovascular surgery, and Wright et al. propose intraoperative aneurysm rupture as the clearest indication for adenosine.[6,7,14,15] When intraoperative aneurysm rupture occurs, blood can hinder visualization of nearby structures, and uncontrolled bleeding can induce cerebral edema and ischemia secondary to hypoperfusion.[16] Intraoperative hemorrhage may result in hemodynamic instability, neurological deficits, and unfavorable outcomes.[16] In these situations, adenosine can assist with hemostasis and securing the aneurysm.[5] The transient asystole and flow arrest from adenosine produce a global reduction in cerebral blood flow that may facilitate clearing the surgical field, improving visualization of nearby structures, and decompressing the aneurysm.[7,12,15] Adenosine-induced flow arrest is particularly useful in clearing the surgical field when conventional suctioning and temporary clip application fail.[5,6] In this case, injury to the basilar artery led to significant and continuous bleeding that obscured the surgical field and prevented timely control of the vascular injury. As in cases of intraoperative aneurysm rupture, adenosine was used to clear the surgical field and help control the basilar artery injury. Adenosine-induced flow arrest produced a cessation in cerebral blood flow that temporarily stopped bleeding at the surgical site. This interruption in blood flow, although brief, enabled the surgical team to effectively clear the operative field of blood, locate the basilar artery injury, and establish control of the bleeding.

Historically, rapid ventricular pacing and deep hypothermic circulatory arrest have also been used in neurovascular surgery to achieve flow arrest. In rapid ventricular pacing, cardiac pacing wires are advanced into the right ventricle to induce ventricular tachycardia and produce controlled hypotension.[9,17] This technique maintains the heart rate above 150 beats per minute to reduce ventricular filling, stroke volume, and blood pressure without actually producing cardiac arrest or asystole.[9] After cessation of rapid ventricular pacing, blood pressure immediately normalizes without prolonged hypotension.[9] Rapid ventricular pacing offers an on-demand and more predictable hemodynamic response, but necessitates an invasive procedure that carries a risk of cardiac perforation and cardiac tamponade.[9,17] With deep hypothermic circulatory arrest, extracorporeal circulation and systemic hypothermia are first established, after which a cardioplegic solution is administered to achieve flow arrest.[9] Cooling to below 28°C decreases the cerebral metabolic rate of oxygen consumption, and increases the tolerance to hypoxia during circulatory arrest.[9,17] Deep hypothermic circulatory arrest provides neuroprotection, longer flow arrest, and controlled hypotension, but it is invasive, resource-intensive, and associated with a higher risk of complications.[9,17] In this case, given the emergent circumstances, adenosine was used to achieve flow arrest since adenosine is readily available and easily administered. An advantage of adenosine-induced flow arrest is that adenosine is easily given in a variety of situations without need for advanced preparation or logistical coordination, requiring only an intravenous bolus.[8] Because of this, adenosine-induced flow arrest is suitable for unexpected situations such as aneurysmal rupture, or in this case, vascular injury.[17] Temporary clip application may also not have been appropriate in this case due to the narrow surgical corridor of the posterior circulation, and limited visibility from blood flooding the surgical field.

Bebawy et al. determined that an empirical adenosine dose of 0.3 to 0.4 mg/kg ideal body weight is sufficient to produce asystole and flow arrest in patients receiving a balanced anesthetic consisting of remifentanil, propofol, and sevoflurane.[8] This dose produces approximately 45 seconds of controlled flow arrest when using that particular anesthetic technique.[8] The magnitude and duration of flow arrest are dose-dependent, but there is considerable variability among patients.[8,9] Ideal body weight is used since the volume of distribution of adenosine is unlikely to change with obesity.[6] The dose of Bebawy et al. is considerably lower than that of Hashimoto et al., who used incremental dosing of adenosine to establish a dose-response relationship in each patient.[8,11] The dose-response model of Hashimoto et al. predicts that an adenosine dose of 2.15 mg/kg is required for 45 seconds of profound hypotension with a mean arterial pressure <30 mmHg.[6,11] The lower dose of Bebawy et al. was attributed to remifentanil, which depresses sinoatrial node activation and atrioventricular node conduction similar to adenosine.[6,8] Table 1 presents a comparison of the adenosine doses and corresponding durations of flow arrest reported by Bebawy et al. and Hashimoto et al.[8,11] In this case, a dose of 18 mg or 0.33 mg/kg of adenosine was sufficient to induce the flow arrest needed to facilitate control of the basilar artery injury. This dose was empirically chosen based on prior institutional experience with aneurysm surgery, and existing evidence on adenosine-induced flow arrest in different neurosurgical procedures. In both instances of adenosine-induced flow arrest, the dose used produced bradycardia, hypotension, then asystole for approximately 50 seconds while using a similar balanced anesthetic technique involving remifentanil, propofol, and sevoflurane. Tachyphylaxis, which may develop with repeated doses of adenosine, was not observed when adenosine was administered the second time.

The clinical effects following intravenous bolus administration of adenosine are apparent within 10 to 20 seconds.[5,7] Heart rate first slows over 20 to 30 seconds, and if a sufficient dose is given, a period of asystole ensues.[5] This is accompanied by profound hypotension lasting up to 2 minutes from a reduction in systemic vascular resistance.[5] Heart rate then returns to baseline over another 20 to 30 seconds.[5] Following asystole, adenosine may cause sinus tachycardia with atrial and ventricular ectopic beats secondary to a reflex increase in circulating catecholamines.[5] The cardiovascular effects of adenosine are transient due to immediate plasma and tissue metabolism, resulting in an intravascular half-

Table 1. Comparison of Reported Adenosine Doses and Corresponding Durations of Flow Arrest in Selected Studies

Author	Procedure	Anesthetic	Dose	Duration	Remarks
Bebawy et al.[8]	Aneurysm clip ligation	General anesthesia (remifentanil, desflurane or sevoflurane, propofol)	0.34 mg/kg IBW (range: 0.29-0.44 mg/kg)	57 s (range: 26-105 s)	• Recommended starting dose of 0.3-0.4 mg/kg to achieve 45 s of flow arrest.
Hashimoto et al.[11]	Endovascular embolization of AVM	General anesthesia (isoflurane, nitrous oxide, propofol)	0.98 mg/kg (range: 0.24-1.76 mg/kg)	Asystole: 8 s MAP <30 mmHg: 18 s MAP <50 mmHg: 50 s	• Developed dose-response model to predict duration of asystole and hypotension based on dose. • A dose of 2.15 mg/kg would be required to achieve 45 s of flow arrest.

IBW = ideal body weight, AVM = arteriovenous malformation, MAP = mean arterial pressure

life of only 0.6 to 20 seconds.[6-8] Exogenously administered adenosine is eliminated by rapid uptake into erythrocytes and vascular endothelial cells, where adenosine is metabolized by adenosine deaminase and adenosine kinase.[5-7] However, elimination of adenosine may be inhibited by certain drugs such as dipyridamole, nimodipine, and methylxanthines.[7] Because of the short intravascular half-life, the cardiovascular effects of adenosine are typically self-limited.[5] The clinical effects of adenosine observed in this case are consistent with those previously described. Adenosine initially produced a brief period of bradycardia and hypotension followed by asystole. In this case, flow arrest lasted approximately 50 seconds, and asystole was followed by spontaneous return of sinus rhythm. After asystole, marked tachycardia and hypotension were observed, with gradual return to baseline over several minutes. The tachycardia and hypotension were anticipated effects following adenosine administration and, as these are typically self-limited, were managed with supportive care. Overall, the observed cardiovascular effects of adenosine were transient, unfolding and resolving over a few minutes, and were not associated with any subsequent arrhythmias.

While valuable in the intraoperative management of intraoperative hemorrhage, the administration of adenosine necessitates consideration for potential adverse effects and the need for special precautions. Adenosine causes vasodilation of the coronary vasculature, and this may lead to a coronary steal phenomenon in patients with preexisting cardiac ischemia.[6,7] Because of this, it is recommended that adenosine be avoided in patients with evidence of severe left main coronary artery stenosis, or severe multi-vessel coronary artery disease.[6,8,14] Cardiac arrhythmias and elevated troponins may also occur after adenosine administration.[6,8] Arrhythmias are usually transient and recover spontaneously without any long-term sequelae, while elevated troponin levels may be asymptomatic and not necessarily indicative of cardiac dysfunction.[6,8] Nonetheless, placement of external defibrillator pads and postoperative monitoring of troponin levels are recommended in patients who have received adenosine.[6,8] Binding of adenosine to bronchial A1 receptors also produces smooth muscle contraction that may induce bronchospasm in patients with asthma or chronic obstructive pulmonary disease.[5] In patients without reactive airway disease, administration of adenosine does not significantly increase peak airway pressures.[6,8] In terms of neurological outcomes, adenosine-induced flow arrest appears to be associated with minimal morbidity in the short term.[8] Bebawy et al. found that intraoperative adenosine was not associated with an increased neurological complication rate defined as a modified Rankin score more than 2 at 48 hours postoperatively or at the time of discharge.[8] In this case, it is difficult to determine whether the administration of adenosine directly contributed to the outcome of the patient. There were no indications of cardiac or pulmonary complications attributable to adenosine observed after the administration of adenosine and in the immediate postoperative period. However, the patient did develop prolonged hypotension immediately after adenosine-induced flow arrest. This hypotension is more likely explained by hypovolemia, as blood pressure improved following the transfusion of blood products. Clinical deterioration occurred only on the third postoperative day, well after the administration of adenosine, suggesting that adenosine was less likely to have contributed to the patient's outcome.

This case report demonstrates the utility of adenosine-induced flow arrest in the management of intraoperative hemorrhage secondary to intracranial vascular injury during meningioma surgery. Although the patient eventually succumbed several days following the procedure, adenosine-induced flow arrest was effective in facilitating hemostasis and control of the vascular injury. This intervention was critical, as failure to control the vascular injury during surgery may have resulted in demise intraoperatively or shortly thereafter. While most evidence on adenosine-induced flow arrest in neurosurgery is derived from aneurysm surgery, the dosing strategy used produced comparable clinical effects, and was equally effective in improving surgical conditions and facilitating hemostasis. This case report presents valuable initial evidence supporting a novel use for adenosine-induced flow arrest, however, as it describes a single patient experience, the findings are limited in generalizability and should be interpreted within this context. Additional research is required

to establish the efficacy of adenosine-induced flow arrest, identify the optimal dosing of adenosine, and evaluate the impact of adenosine on neurological and surgical outcomes in this specific application.

CONCLUSION

Intraoperative hemorrhage is an emergent and life-threatening complication of neurosurgery, and as such, management of blood loss is an essential component of neuroanesthesia. This case report describes the successful use of adenosine-induced flow arrest to facilitate hemostasis and control of a basilar artery injury during craniotomy and excision of a petroclival meningioma. Here, adenosine-induced flow arrest produced a temporary reduction in cerebral blood flow that allowed the surgical team to clear the surgical field, identify the vascular injury, and control the bleeding. Adenosine has documented efficacy and safety in other neurosurgical applications, and thus, this technique may be considered for similar instances of major vascular injury to facilitate hemostasis. Further studies are necessary to determine the efficacy of adenosine-induced flow arrest, the appropriate dosing of adenosine, and the effects of adenosine on neurological outcome when used for this particular application.

Informed Consent

General consent for the use of clinical data was obtained from the patient at admission, and its applicability for publication has received institutional approval.

Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

All authors declared no conflicts of interest.

Funding Source

None.

REFERENCES

1. Ghare A, Wong QHW, Sefcikova V, Waraich M, Samandouras G. Handling severe intraoperative hemorrhage and avoiding iatrogenic stroke during brain tumor surgery: Techniques for prevention of hemorrhagic and ischemic complications. World Neurosurg. 2023 May 11;176:e135-50. doi: 10.1016/j.wneu.2023.05.019. PMID: 37178915.
2. Wang C, Li P. Risk factors for intraoperative blood loss in resection of intracranial meningioma: Analysis of 530 cases. PloS One. 2023 Sep 8;18(9):e0291171. doi: 10.1371/journal.pone.0291171. PMID: 37682850; PMCID: PMC10490957.
3. Hsu SY, Huang YH. Characterization and prognostic implications of significant blood loss during intracranial meningioma surgery. Transl Cancer Res. 2016 Dec 1;5(6):797–804. doi: 10.21037/tcr.2016.11.72.
4. Tahara A, De Santana PA, Maldaun MVC, Panagopoulos AT, Da Silva AN, Zicarelli CA, et al. Petroclival meningiomas: Surgical management and common complications. J Clin Neurosci. 2009 Mar 23;16(5):655–9. doi: 10.1016/j.jocn.2008.08.011. PMID: 19318254.
5. Osborn S. Adenosine for transient cardiac standstill in neurovascular surgery: A heart-stopping moment. Australasian Anaesthesia. 2017 Jan (2017):229-36.
6. Thakore B, Goraksha S, Monteiro JN. Adenosine-assisted management of intracranial aneurysm. J Neuroanaesth Crit Care. 2019 Feb 8;6(1):13–7. doi: 10.1055/s-0038-1676555.
7. Desai VR, Rosas AL, Britz GW. Adenosine to facilitate the clipping of cerebral aneurysms: literature review. Stroke Vasc Neurol. 2017 Jun 23;2(4):204–9. doi: 10.1136/svn-2017-000082. PMID: 29507781; PMCID: PMC5829927.
8. Bebawy JF, Gupta DK, Bendok BR, Hemmer LB, Zeeni C, Avram MJ, et al. Adenosine-induced flow arrest to facilitate intracranial aneurysm clip ligation: dose-response data and safety profile. Anesth Analg. 2010 Apr 23;110(5):1406–11. doi: 10.1213/ane.0b013e3181d65bf5. PMID: 20418302.
9. Wadhwa R, Singh D. Cardiac standstill and neurosurgery: A much-needed collaboration for complicated vascular procedures. J Cerebrovasc Sci. 2022 Jan 1;10(1):17–25. doi: 10.4103/jcvs.jcvs_16_22.
10. Hellstern V, Bhogal P, Pérez MA, Alfter M, Kemmling A, Henkes E, et al. Adenosine-induced asystole during AVM embolization. Clin Neuroradiol. 2021 Jun 14;32(1):39–48. doi: 10.1007/s00062-021-01035-z. PMID: 34125253; PMCID: PMC8894166.
11. Hashimoto T, Young WL, Aagaard BD, Joshi S, Ostapkovich ND, Pile-Spellman J. Adenosine-induced ventricular asystole to induce transient profound systemic hypotension in patients undergoing endovascular therapy. Anesthesiology. 2000 Oct 1;93(4):998–1001. doi: 10.1097/00000542-200010000-00021. PMID: 11020753.
12. Jang EA, Song J, Kang SW. The use of adenosine for temporary cardiac arrest during intraoperative cerebral aneurysmal re-bleeding. J Clin Case Rep. 2016 Jan 1;6(11). doi: 10.4172/2165-7920.1000884.
13. Al-Mousa A, Bose G, Hunt K, Toma AK. Adenosine-assisted neurovascular surgery: initial case series and review of literature. Neurosurg Rev. 2017 Jul 22;42(1):15–22. doi: 10.1007/s10143-017-0883-3. PMID: 28735438.
14. Wright JM, Huang CL, Sharma R, Manjila S, Xu F, Dabb B, et al. Cardiac standstill and circulatory flow arrest in surgical treatment of intracranial aneurysms: a historical review. Neurosurg Focus. 2014 Apr 1;36(4):E10. doi: 10.3171/2014.2.focus13554. PMID: 24684323.
15. Wang X, Feletti A, Tanaka R, Yamada Y, Suyama D, Kawase T, et al. Adenosine-induced Flow Arrest to Facilitate Intracranial Complex Aneurysm Clip Ligation: Review of the Literature. Asian J Neurosurg. 2018 Sep 1;13(03):539–45. doi: 10.4103/ajns.AJNS_207_16. PMID: 30283502; PMCID: PMC6159025.
16. Nager GB, Pontes JP, Udoma-Udofa OC, Gomes FC, Larcipretti AL, de Oliveira JS, et al. Efficacy and safety of adenosine, rapid ventricular pacing and hypothermia in cerebral aneurysms clipping: a systematic review and meta-analysis. Neurosurg Rev. 2024 May 11;47(1):215. doi: 10.1007/s10143-024-02450-9. PMID: 38730072.
17. Meling TR, Romundstad L, Niemi G, Narum J, Eide PK, Sorteberg AG, et al. Adenosine-assisted clipping of intracranial aneurysms. Neurosurg Rev. 2017 Aug 17;41(2):585–92. doi: 10.1007/s10143-017-0896-y. PMID: 28819885.