

The Use of Adenosine for Temporary Cardiac Arrest during Intraoperative Cerebral Aneurysmal Re-Bleeding

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Abstract

Intraoperative cerebral aneurysmal re-bleeding has a deleterious effect that is associated with high mortality and morbidity. Neurosurgeons and anesthesiologists should be familiar with all methods including hypothermia, cardiopulmonary bypass, pharmacologically-induced hypotension and cardiac standstill to manage intraoperative aneurysmal rupture or clip complex aneurysms. Specifically, adenosine induced profound hypotension or cardiac arrest has been used recently. We present our experience with the use of adenosine during a case of incidentally developed intraoperative cerebral aneurysmal rupture.

Keywords: Adenosine; Cardiac arrest; Aneurysmal subarachnoid hemorrhage; Intraoperative care

Introduction

Rupture of an intracranial aneurysm during surgery may have extremely serious consequences. In such a situation, adenosine may be a very useful management tool through decreased blood flow or complete cardiac asystole. Low blood volume in the affected area enables to provide clear surgical field, and surgeon is able to identify the focus of bleeding and control the problem. We report the successful use of intravenously administered adenosine in suddenly developed intraoperative cerebral aneurysmal re-bleeding.

Case Report

A 70-year-old woman came to the emergency room because of a headache that occurred the day before. She had been diagnosed with a subarachnoid hemorrhage caused by cerebral aneurysm rupture at a local hospital. At the time she arrived at the emergency room, her mental status was drowsy and her Glasgow Coma Scale (GCS) score was 14 points but her vital signs were stable. Her medical history included well-controlled hypertension for which she had been taking medication including a calcium-channel blocker, a beta-blocker, and aspirin for 5 years. Preoperative laboratory tests, an electrocardiogram, and a chest radiograph revealed no significant abnormalities. A brain computed tomography (CT) and CT angiography scan (Figures 1 and 2) was showed that acute subarachnoid hemorrhage (SAH) in both sylvian fissures, the interhemispheric fissure, the basal cistern and a small amount of acute intraventricular hemorrhage (IVH) in all ventricles, with a 9 mm saccular aneurysm in the right anterior communicating artery. It was determined that she should undergo an emergency operation with aneurysm clipping. After she arrived at

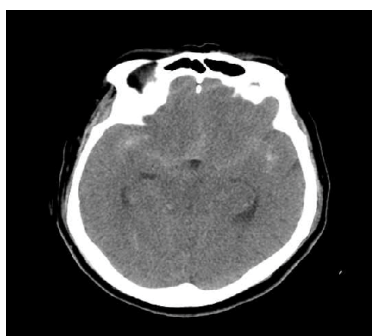


Figure 1: Pre-operative CT.

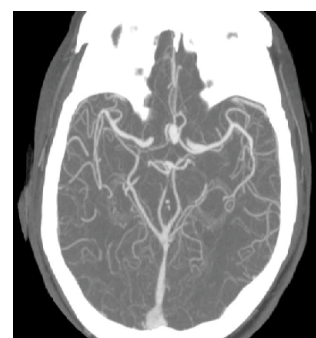


Figure 2: Pre-operative CT angiography.

the operating room, she was closely monitored via electrocardiogram, pulse oximetry, and non-invasive arterial pressure. At that time her vital signs were: blood pressure (BP), 128/52 mmHg; heart rate (HR), 52 beats/min; and oxygen saturation measured by pulse oxymetry, 98%. Her mental status was still drowsy. The patient was administered 100% oxygen through a face mask. General anesthesia was induced intravenously with 3.0 µg/mL propofol (Fresofol 2% MCT®, Fresenius Kabi, Germany) and 3.0 ng/mL remifentanyl (Ultiva®, Glaxo Smith Kline, Germany), and maintained with propofol (3.0-5.0 µg/mL) and remifentanyl (3.0-5.0 ng/mL) using a target-controlled infusion device (Orchestra Base Primea®, Fresenius Kabi, Germany). Endotracheal intubation was facilitated with 0.8 mg/kg rocuronium (Esmeron®, MSD, USA) administered intravenously. The patient was mechanically ventilated with an initial tidal volume of 7-8 ml/kg, a respiratory rate of 12 breaths/min, and a fraction of inspired oxygen (FiO₂) of 0.45, which were adjusted in order to maintain arterial carbon dioxide between 30-35 mmHg. In addition, arterial catheter and central venous catheter were placed into the left radial artery and the right internal jugular vein,

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Figure 3: Post-operative CT.

respectively, to continuously monitor the arterial blood pressure and administer drug or fluid. After the anesthesia was induced and head pinning was accomplished, 200ml of 15% mannitol was infused for management of increased intracranial pressure. Since the scalp incision started, blood pressure and heart rate were maintained 100-130/50-60 mmHg and 60-80 beats/min, respectively. Intraoperative baseline arterial blood gas analysis showed that pH 7.43, carbon dioxide partial pressure (PaCO_2) 34 mmHg, oxygen partial pressure (PaO_2) 181 mmHg, hematocrit 23.5%, hemoglobin 9.5 g/dL. 90 minutes after scalp incision, during tissue dissection to find the aneurysm sac, sudden massive bleeding occurred in the surgical field. The neurosurgeon requested the anesthesiologist to induce artificial cardiac arrest in doubting re-bleeding of aneurysm. At that time, the patient's BP was 110/50 mmHg and HR was 80 beats/min. The anesthesiologist injected 6mg of adenosine (adenocor®, Famar Health Care Services, Spain) rapidly via a central intravenous line, the patient's blood pressure and heart rate decreased sharply to 50/30 mmHg and 30-40 beats/min, respectively. This allowed the neurosurgeon to find the bleeding focus, an aneurysm in the right anterior communicating artery, and he controlled ruptured aneurysm with multiple clips. The patient's blood pressure rapidly returned to 90-100/40-45 mmHg. At that time, arterial blood gas analysis showed that pH 7.43, PaCO_2 37 mmHg, PaO_2 121 mmHg, hematocrit 19%, hemoglobin 7.5 g/dL. Thereafter, dopamine was infused and two units of packed red cells were transfused in order to produce a high-normal mean arterial pressure (MAP) to prevent critical reduction of cerebral blood flow. The patient's blood pressure and heart rate were 120-140/60-80 mmHg and 70-90 beats/min, arterial blood gas analysis showed that pH 7.41, PaCO_2 36 mmHg, PaO_2 120 mmHg, hematocrit 23%, hemoglobin 9.3 g/dL, and the operation concluded without any additional problems. After the operation, the patient was transferred to the intensive care unit after CT (Figure 3) was checked. In the intensive care unit, 5mg of midazolam was injected intravenously. The next day, the patient's mental state had recovered and tracheal extubation was done. Postoperative 38 days, she had one more surgery, a ventricular-peritoneal shunt to correct hydrocephalus. And postoperative 52 days later, she was transferred to a local hospital for conservative treatment. She had no specific neurologic sequelae except occasional confusion.

Discussion

Aneurysmal subarachnoid hemorrhage occurs in 2–16 people per 100,000 and has a high rate of morbidity and mortality. Aneurysms presenting with subarachnoid hemorrhage tend to re-bleed at a rate of 4-13.6% within the first 24 hours after the initial episode [1]. Aneurysm re-bleeding is associated with very poor outcomes [2]. To reduce the risk of re-bleeding, the ruptured aneurysm must be treated as early as possible either by neurosurgical clipping or endovascular coiling. Without prompt intervention, ruptured aneurysm is often fatal [1].

In this case, intraoperative aneurysmal re-bleeding was happened in aneurysmal subarachnoid hemorrhage patient, we used adenosine for cardiac arrest in order to provide clear surgical field.

Intraoperative aneurysmal rupture can occur under any situation during aneurysm surgery. It remains a potentially catastrophic event that can adversely affect a patient's outcome from aneurysm surgery [3,4]. In the past, several management strategies including carotid artery ligation, hypothermia, cardiopulmonary bypass, pharmacologically-induced hypotension, and cardiac standstill have been used to treat intraoperative rupture or to clip complex aneurysms that are large or deep in location or difficult to control. Adenosine has been used since the 1980's to induce hypotension and complete circulatory arrest [5]. In 1984, Alf Sollevi and colleagues studied adenosine-induced hypotension using continuous infusion during clipping of a cerebral aneurysm and showed no problems during surgery [6]. In 1999, Michael Groff and colleagues reported a case of adenosine-induced transient asystole during the clipping of a basilar arterial aneurysm with no complications [7].

Adenosine is a purine nucleoside with a very short intravascular half-life of less than 10 seconds. It has various effects including negative chronotropic and dromotropic effects on the heart, vasodilation, and modulation of sympathetic nervous system activity. Traditionally, intravascular injection of adenosine (6 mg) has occasionally been used to treat paroxysmal supraventricular tachycardia [8]. Also, continuous infusion of adenosine (150-300 $\mu\text{g}/\text{kg}/\text{min}$) in anesthetized patients is able to induce hypotension via dilation of arterial resistance vasculature, and adenosine-induced hypotension rapidly reverses after discontinuation. Furthermore, administration of high doses of adenosine in patients with normal sinus rhythms induces rapidly-reversible cardiac arrest [8,9]. By adenosine's very short half-life and few adverse effects, it has been used during cardiac surgery, embolization of arteriovenous malformations, intracranial aneurysms interventions by induced facilitating hypotension or cardiac arrest [7,10-14].

As mentioned previously, adenosine has been used during intracranial aneurysm surgery since the 1980's [6,7,12-14]. However, the dose, regimen, efficacy, and risks of adenosine have not yet been clearly established. According to one retrospective review, the use of adenosine during intraoperative rupture of an intracranial aneurysm, the median (range) single bolus dose used was 12 (6-18) mg and the median total dose given in multiple boluses was 27 (18-89) mg [12]. In another study, the dose of adenosine required to flow arrest during aneurysm surgery reported a starting dose of 0.3 to 0.4 mg/kg ideal body weight (IBW), with additional administration as needed. The median dose of adenosine was 24 mg, and the range of doses was from 6 to 60 mg [13]. In 2015, Sung-Ho Lee and colleagues recommended that the dose of adenosine that required for transient asystole during intracranial aneurysm surgery is 0.3 to 0.4 mg/kg IBW [14].

In our case, because of suddenly developed intraoperative aneurysm re-bleeding, the surgeon had poor visibility in the surgical field. So, we used 6 mg adenosine, although it didn't enough to induce cardiac arrest, it caused profound decrease in blood pressure and heart rate. We prepared additional dose of adenosine for cardiac arrest but we didn't use it because of marked decrease in blood pressure and heart rate were sufficient to allow the neurosurgeon to find the focus of the bleeding.

Conclusion

In conclusion, aneurysmal subarachnoid hemorrhage intervention makes the surgeon's and anesthesiologist's tasks more difficult. Neurosurgeons have accurate knowledge of the anatomy of the

aneurysm and the surrounding vasculature and are able to carefully apply microneurosurgical techniques such as the application of temporary clips [15]. The general goals of anesthetic management include hemodynamic control to minimize the risk of re-bleeding and strategies to protect the brain against ischemic injury [16,17]. Adenosine may be useful in the management of dangerous situations during intracranial aneurysm surgery as it induces profound hypotension or transient cardiac arrest. As the necessary dose of adenosine is dependent on many factors, adverse effects should be closely monitored. Also it is needed adequate communication between anesthesiologist and neurosurgeon.

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