Adenosine-induced transient asystole for management of a basilar artery aneurysm

Case report

MICHAEL W. GROFF, M.D., DAVID C. ADAMS, M.D., RONALD A. KAHN, M.D., UDAY M. KUMBAR, M.D., BO-YI YANG, PH.D., AND JOSHUA B. BEDERSON, M.D.

Departments of Neurosurgery and Anesthesiology, Mount Sinai School of Medicine, New York, New York

Recent advances in anesthetic and surgical management, such as induced deep hypothermic circulatory arrest and application of temporary clips, have improved outcome for patients with basilar artery aneurysms. Nonetheless, these techniques are associated with significant risks. The authors report a case in which three transient periods of cardiac asystole were induced during basilar artery aneurysm surgery. Adenosine-induced asystole facilitated the safe clipping of the aneurysm by producing consistent periods of profound hypotension and collapse of the aneurysm without the need for temporary clipping. This technique provided unencumbered identification of perforating arteries, precise definition of the local anatomy, and an ideal environment for the safe placement of the aneurysm clip.

KEY WORDS • cerebral aneurysm • asystole • hypotension • basilar artery • pharmacology • adenosine

Case Report

This 57-year-old woman with a history significant for hypertension was involved in a motor vehicle accident 6 months prior to presentation. Several months after the accident the patient reported persistent memory difficulty, although she was otherwise neurologically intact.

Examination. Magnetic resonance imaging suggested the presence of a BA aneurysm. This diagnosis was confirmed by cerebral angiography (Fig. 1), and the patient was scheduled to undergo elective right perirional craniotomy for aneurysm clipping.

Operation. To prepare the patient for surgery, standard monitors were placed, radial artery and central venous catheterization were performed, and general anesthesia was induced using propofol, remifentanil, and pancuroni-
The patient’s temperature was monitored by means of an esophageal probe, and bladder catheterization was performed. Anesthesia was maintained using nitrous oxide, oxygen, isoflurane (\(0.1-0.4\%\) endtidal), and remifentanil (0.1–0.3 \(\mu\)g/kg/minute). External pacing/defibrillator pads (Zoll, Burlington, MA) were placed and one-to-one ventricular pacing was confirmed to ensure pacemaker function (in the event that adenosine administration resulted in a prolonged asystole) or electrical cardioversion (in the event that it resulted in a hemodynamically significant supraventricular arrhythmia). A cooling blanket was applied to the patient to induce mild systemic hypothermia (32.5˚C) before aneurysm clipping commenced.

Needle electrodes were placed at F3, F4, F7, C3, C4, and Cz (international 10–20 system) for monitoring of somatosensory evoked potentials (SSEPs), brainstem auditory evoked potentials, and the electroencephalography (EEG) data. Neurophysiological data were collected and stored using a commercially available system (Computational Diagnostics Incorporated, Pittsburgh, PA).

A standard right pterional craniotomy was performed, and a transsylvian approach was followed with extensive mobilization of the temporal lobe. When the aneurysm was exposed, its posteriorly directed dome prevented complete visualization of the origin of the perforating arteries emanating from the dorsal surface of the BA. Deliberate hypotension to a target mean arterial pressure (MABP) of 60 mm Hg was induced by infusion of sodium nitroprusside (0.1–0.3 \(\mu\)g/kg/minute). An initial dose of 6 mg adenosine was administered through the central venous catheter and followed by a 10-ml saline flush. During the resulting 8-second period of asystole, the patient’s MABP decreased to approximately 15 mm Hg. On spontaneous return of the sinus rhythm, her MABP gradually returned to 60 mm Hg during an approximately 15-second period. During asystole, the dome of the aneurysm collapsed, allowing final dissection of the perforating vessels from the dorsal surface of the aneurysm dome. After the aneurysm neck had been defined, an additional 12 mg adenosine was given, resulting in a 13-second period of asystole that was followed by a gradual return to baseline MABP during the subsequent 25 to 30 seconds (Figs. 2 and 3). This transient period of profound systemic hypotension facilitated placement of the aneurysm clip. Subsequently, a third dose of adenosine (12 mg) facilitated final clip positioning. After inspection of the clip, the patient was actively rewarmed (Warmtouch; Mallinckrodt Medical, St. Louis, MO) to a temperature of 35.1˚C as the operation was completed.

Continuous recording of EEG data during adenosine administration showed a loss of faster frequency activity and loss of amplitude consistent with cerebral ischemia approximately 10 seconds after each episode of asystole was induced (Fig. 4). The EEG values recovered to baseline within 15 to 20 seconds after each asystolic period. The SSEP recordings did not show a significant change.

**Postoperative Course.** At the conclusion of surgery, the patient was extubated and transported to the neurological intensive care unit. The patient was neurologically intact. A follow-up angiogram demonstrated no residual aneurysm (Fig. 5), and a computerized tomography scan obtained on postoperative Day 4 was normal. The remainder of the patient’s postoperative course was unremarkable, and she was discharged from the hospital on postoperative Day 5.

**Discussion**

Adenosine is an endogenous nucleoside analog that...
Adenosine and basilar artery aneurysm

exerts negative dromotropic and chronotropic effects at both the sinoatrial and atrioventricular nodes. Its major therapeutic use is to terminate supraventricular tachycardias. Its interaction with the cardiac A₁ receptor reduces cyclic adenosine monophosphate (cAMP) accumulation by antagonizing catecholamine-stimulated adenylate cyclase conversion, decreasing inward calcium conductance and, thus, diminishing the pacemaker current. It also interacts with the A₂ receptor by stimulating adenylate cyclase and thus increasing intracellular cAMP.²

Because of its rapid enzymatic breakdown and reuptake into cells, the half-life of adenosine is less than 10 seconds. Potential adverse effects of adenosine administration are transient and may include flushing, chest pain, and shortness of breath. Bronchoconstriction, which can cause the sensation of shortness of breath, warrants caution when adenosine is used in patients with reactive airway disease. Adenosine-induced high-degree atrioventricular block is usually short lived and only rarely requires treatment. Because adenosine may cause atrial arrhythmia, facilities for direct-current cardioversion should be available.

Adenosine administration is associated with sedation and decreased seizure activity, resulting from an inhibitory effect on neurotransmitter release that is mediated by the A₁ receptors. Adenosine also exerts an A₂ receptor-mediated effect on smooth muscle in the cerebral vasculature and appears to minimize calcium influx, thereby decreasing ischemic free radical and excitotoxic neuronal injury.¹⁰ Nonetheless, it is unlikely that administration of adenosine for transient cardiac asystole is associated with a significant cerebral protective effect. Rather, as in other clinical situations, such as ventricular fibrillation for implantation of a cardioverter defibrillator,¹ transient profound hypotension is not associated with postoperative neurological dysfunction. Although we observed ischemic EEG changes during each asystolic period, simultaneously recorded SSEPs failed to show changes in latency or amplitude, reflecting the transient nature of the cerebral ischemia. However, even a brief period of cerebral ischemia might be less well tolerated by a patient with recent subarachnoid hemorrhage, limiting the use of adenosine to patients with unruptured or remotely ruptured aneurysms.

The current application of adenosine has been expanding beyond its use as an antiarrhythmic agent. Adenosine-induced transient asystole has been used during minimally invasive coronary artery bypass grafting,⁹ endovascular repair of aortic aneurysms,⁴ and embolization of cerebral arteriovenous malformations.⁸ The ultimate clinical utility of adenosine-induced asystole for aneurysm clipping remains to be defined. Moreover, adenosine-induced asystole should not be used as an alternative to standard cerebrovascular techniques of proximal and distal control because intraoperative aneurysm rupture would be inadequately prevented or treated using cardiac asystole alone.

Recently, techniques such as temporary clipping and DHCA have been used to facilitate the microsurgical dissection of aneurysms.⁷,¹¹ However, in a recent series, there was postoperative computerized tomography evidence of cerebral infarction in 19% of patients undergoing tempo-

![Fig. 3. Arterial blood pressure electrocardiographic recordings showing the spontaneous return of a normal sinus rhythm after a 13-second period of asystole. Baseline blood pressure was achieved approximately 30 seconds after the return of the normal sinus rhythm. S = second.](image)

![Fig. 4. Recording of EEG data and the computerized spectral analysis (CSA) during adenosine-induced asystole. Approximately 17 seconds after adenosine (12 mg) is administered (A), asystole and profound hypotension are noted (B), resulting in a slowing of the EEG trace and a corresponding leftward shift in the CSA (C). As baseline arterial blood pressure is restored, the EEG trace returns to baseline (D).](image)
Conclusions

In this case, three transient periods of adenosine-induced cardiac asystole facilitated direct clipping of a BA aneurysm and there were no complications resulting from its use. We believe this is the first time that adenosine has been used to facilitate clipping of a cerebral aneurysm. As experience with this drug accumulates, more precise indications for adenosine-induced asystole during neurosurgery will emerge.

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References


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Address reprint requests to: David C. Adams, M.D., Department of Anesthesiology, Mount Sinai Medical Center, One Gustave L. Levy Place, New York, New York 10029. email: David_Adams@smi@mssm.edu.

Fig. 5. Postoperative angiogram demonstrating successful clipping of the aneurysm.