Cerebrovascular Accident Under Anesthesia During Dental Surgery

Mathew Cooke, DDS, MD, MPH,* Michael A. Cuddy, DMD,† Brad Farr, DDS,‡ and Paul A. Moore, DMD, PhD, MPH§

*Assistant Professor, †Associate Professor and Residency Director, ‡Anesthesia Resident, §Professor, Department of Dental Anesthesiology, University of Pittsburgh School of Dental Medicine, Pittsburgh, Pennsylvania

Stroke, or cerebrovascular accident (CVA), is a medical emergency that may lead to permanent neurological damage, complications, and death. The rapid loss of brain function due to disruption of the blood supply to the brain is caused by blockage (thrombosis, arterial embolism) or hemorrhage. The incidence of CVA during anesthesia for noncardiac nonvascular surgery is as high as 1% depending on risk factors. Comprehensive preoperative assessment and good perioperative management may prevent a CVA. However, should an ischemic event occur, appropriate and rapid management is necessary to minimize the deleterious effects caused to the patient. This case report describes a patient who had an ischemic CVA while under general anesthesia for dental alveolar surgery and discusses the anesthesia management.

Key Words: General anesthesia; Cerebrovascular event; Complication; Dentistry.

Central nervous system infarction occurs over a clinical spectrum.¹ A cerebrovascular accident (CVA), or stroke, is a sudden interruption in the blood supply to the brain, accompanied by overt signs.¹ Most strokes (85%) are caused by an abrupt occlusion of a cerebral artery leading to loss of adequate blood supply to a specific area of the brain (ischemic stroke).² Ischemic strokes are either thrombotic or embolic. A thrombotic stroke occurs when a blood clot (thrombus) forms in one of the arteries that supply blood to the brain.³ An embolic stroke occurs when a blood clot or other debris forms away from the brain and is carried through the bloodstream to lodge in narrower brain arteries. Another cause of stroke is when bleeding occurs into brain tissue from a ruptured blood vessel (hemorrhagic stroke).³

Stroke is a leading cause of morbidity and mortality.⁴ Perioperative acute ischemic stroke is a recognized

complication of noncardiac, nonvascular surgery.⁵ Among the general population, the rate of acute ischemic stroke in the perioperative period has been reported to be as high as 0.7%.⁵ The incidence increases to 1.0% as age increases above 65 years.⁵ Other major risk factors that predispose to acute ischemic stroke in the perioperative period include: renal disease, atrial fibrillation, previous history of stroke, valvular disease, congestive heart failure, male sex, diabetes mellitus, and race.⁵⁻⁷

Adequate preoperative assessment of risk is imperative.⁸ When possible, patients should receive optimum medical treatment in the interest of attenuating the impact of risk factors in the perioperative period.⁸ However, when strokes occur, rapid, immediate treatment is required to prevent permanent brain damage.⁹

CASE PRESENTATION

A 40-year-old white woman with a diagnosis of severe dental caries and acute abscesses presented to the

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Address correspondence to Dr Cooke, University of Pittsburgh School of Dental Medicine, Department of Dental Anesthesiology, G-89 Salk Hall, Pittsburgh, PA 15261; mrc99@pitt.edu.

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University of Pittsburgh School of Dental Medicine for full mouth extraction under deep sedation/general anesthesia. The surgical plan included extraction of the remaining 25 teeth and 4 quadrants of alveoloplasty. Upon arrival, nothing by mouth (NPO) and allergy status were verified. Preoperative assessments were completed, and consent for treatment was obtained. Positive findings on the medical history and physical examination were smoking and gastroesophageal reflux disease. The patient reported noncompliance for several months with her proton pump inhibitor (Omeprazole). Although there was a suspicion of illicit drug use in the past, the patient denied any current substance abuse. Risks versus benefits for anesthesia were evaluated. Because of the extensive surgical plan and potential for bleeding near the airway, general anesthesia with tracheal intubation was planned.

The patient was brought to the operating room in the Departments of Dental Anesthesiology and Oral and Maxillofacial Surgery. Standard American Society of Anesthesiologists (ASA) monitors were placed: 3 lead electrocardiography (ECG), pulse oximetry, noninvasive blood pressure, capnography, and temperature. Intravenous access was obtained with a 22-gauge intravenous (IV) catheter, and a 0.9% normal saline infusion was connected. Baseline vital signs were obtained and included a blood pressure of 98/78 mm Hg, a heart rate of 80 beats per minute (BPM) with a normal sinus rhythm, and an SpO₂ of 98% on room air. The patient was preoxygenated with 100% oxygen for 5 minutes. General anesthesia was induced with 50 µg of fentanyl, 150 mg of propofol, and 1.0% end-tidal sevoflurane for approximately 2 minutes. Rocuronium (Zemuron) 10 mg and succinylcholine 100 mg were used to facilitate nasal endotracheal intubation with direct visual laryngoscopy. A 6.5-cuffed nasal RAE tube was placed in the right naris under direct visualization using a 3.0 Macintosh blade without difficulty. The tube's position was verified with positive end-tidal CO₂ and positive bilateral breath sounds upon auscultation of the lungs. The nasotracheal tube was secured, eyes were taped, head was wrapped, pressure points were padded, and the patient was draped. Care was then turned over to the surgeons, a throat pack was placed, and surgery was started. Local anesthesia was administered (6 cartridges of 2% lidocaine with 1 : 100,000 epinephrine and 2 cartridges of 0.5 bupivacaine with 1 : 200,000 epinephrine). Her vital signs remained stable.

Maintenance of anesthesia was with isoflurane at 1.2 minimum alveolar concentration via mechanical ventilation with flows of 1 L oxygen and 1 L nitrous oxide. During the initial 20 minutes, blood pressure readings were recorded as 110/75, 109/77, 99/68, and 92/62 mm Hg at 5-minute intervals with a heart rate in the range of 60 to 90 BPM in normal sinus rhythm.

Thirty-five minutes after baseline blood pressure readings, the blood pressure fell to 61/39 mm Hg with a heart rate of 58 BPM. Another reading was taken immediately. Proper blood pressure cuff positioning was verified, but the blood pressure remained depressed. Isoflurane was discontinued, fluids were increased, and 10 mg of ephedrine was administered intravenously. Her heart rate continued to drop to 53 BPM, while the pressure remained unchanged. Ephedrine 10 mg bolus was repeated, but the heart rate continued to drop to 39 BPM with a sinus bradycardia rhythm. Blood pressure remained depressed at 50/30 mm Hg. A 0.3 mg bolus of epinephrine was administered intravenously, and within 90 seconds, the heart rate and blood pressure elevated to 90 BPM and 150/90 mm Hg, respectively. The ECG rhythm after epinephrine was irregular with inverted T waves, widened QRS complexes, and premature ventricular contractions. A 60 mg bolus of lidocaine was administered intravenously, and her heart rate slowed and returned to a sinus rhythm. All other vital signs remained normal during the hypotensive event including a SpO₂ of 99% and end-tidal CO₂ of 31–35 mm Hg.

After appropriate management of her adverse cardiovascular events, the patient stabilized and her surgery was completed. There were no additional intraoperative events, and her vital signs remained stable. Ten minutes before the end of the case, the patient was weaned from the mechanical ventilator and began breathing spontaneously with tidal volumes greater than 400 mL per breath at a rate of 18 breaths per minute. Upon completion of surgery, the throat pack was removed and both isoflurane and nitrous oxide were discontinued. The patient was placed on 100% oxygen and continued with spontaneous ventilation. The patient's head was unwrapped, tape was removed from her eyes, mouth and oropharynx were suctioned, and drapes were removed.

About 45 minutes after termination of the volatile anesthetic, the patient did not meet criteria for extubation. During that time, the patient would respond to painful stimuli like sternal rub or manipulation of the endotracheal tube. However, she would not respond to verbal commands such as, "squeeze my hand," "lift your head," or "open your eyes." It was noted that her response to the stimuli was much more pronounced on her left side compared to her right. Her pupils were equal, round, and reactive to light bilaterally. Her skin was cool and diaphoretic. Because she did not meet criteria for extubation, emergency medical services was summoned to transport the patient for further evaluation. Emergency medical services transported the patient who was intubated on a T-piece with high-flow 100% oxygen to the emergency department (ED) at the

University of Pittsburgh Medical Center. In the ambulance, she became more alert, was following commands, and tried to pull her endotracheal tube. Significant right-sided weakness continued.

Upon arrival in the ED, reports were given to the ED physician and neurology fellow. A clinical neurologic assessment/exam was completed, which showed weakness with 4 out of 5 deficits in both limbs of the right side. Following assessment, the patient was taken emergently for a computed tomography (CT) scan. Her CT with and without contrast showed bilateral narrowing/stenosis of the carotid arteries. An intracranial bleed was ruled out. Tissue plasminogen activator for an ischemic stroke was considered as the patient was within the acceptable 6-hour time period. However, a fibrinolytic was not administered because the neurologists were unsure of the etiology. The patient was resedated with propofol and scheduled for additional testing including magnetic resonance imaging, magnetic resonance arteriography, CT (with and without contrast), and electroencephalography. Electroencephalographic testing showed normal brain function for a patient her age. Later that day the results from the imaging tests reported 80 to 90% stenosis of the right carotid artery and 90% stenosis of the left carotid artery with no intracranial hemorrhage. Her diagnosis of ischemic left-sided stroke was confirmed. A stent was placed in her left carotid, immediate perfusion was observed, and the patient was placed on a regimen of aspirin and clopidogrel (Plavix). With reperfusion came significant, albeit gradual improvement; the sedation medications were weaned, and the patient was extubated by criteria, which included following verbal commands. Over the course of a 5-day hospital stay, the patient continued to gain strength, function, and sensation of the right side. She was dismissed to physical therapy with expectation of 90% return of function. Two weeks after dismissal from the hospital, she returned to the School of Dental Medicine to have her denture delivered.

DISCUSSION

It would not be expected that a 40-year-old woman with no significant past medical history would have 80 to 90% occlusion of her carotid arteries, as was the case with our patient. The first clinical signs indicating complications in the perioperative period included hypotension followed by bradycardia. It was later learned that our patient had a perioperative acute ischemic stroke or CVA while having dental surgery under general anesthesia. In this discussion, we will evaluate the patient's intraoperative response to the acute ischemic event. We will also discuss etiology of a CVA, risk factors, preoperative

assessment, and perioperative management of a patient with an acute CVA.

Intraoperative hypotension was the first event observed. Despite efforts to raise the pressure via administering fluids, lowering the concentration of the volatile anesthetic, and administering ephedrine, the pressure continued to drop and the heart rate slowed. Ephedrine should have increased blood pressure and slightly increased heart rate due to endogenous release of catecholamines.¹⁰ The carotid sinuses of the internal carotid arteries play a major role in the maintenance of blood pressure. These sinus baroreceptors provide powerful moment-to-moment control of arterial pressure.¹¹ Diseased carotid arteries, with 80 to 90% occlusion as seen in our patient, may be the cause of the lack of self-regulation needed during maintenance of anesthesia. Studies suggest that an ischemic brain has an impaired ability to regulate blood pressure.^{12,13} Ephedrine may not have been potent enough to overcome the carotid response; however, when intravenous epinephrine was administered, the desired outcome of increase in blood pressure and heart rate resulted. A single bolus dose of 300 µg of IV epinephrine could have caused an overshoot in blood pressure and heart rate, which could have resulted in ventricular tachycardia, ventricular fibrillation, myocardial infarction, or hemorrhagic stroke. However, in this case because of the rapidity of decline in blood pressure and heart rate, even in the presence of 20 mg of ephedrine, it was felt that the benefits of a moderate IV bolus dose of 300 µg outweighed the risks in a patient not in cardiac arrest. Perhaps, a more conservative approach may have been an IV bolus of 100 µg of epinephrine, followed if needed by a doubling of that dose a minute later. Should that also not have been effective, another doubling of that larger dose to a total of 600 µg could have been administered.

This patient was having dental alveolar surgery in the head and neck region. She had general anesthesia with nasal tracheal intubation, which was placed under direct vision. Both the intubation and surgery require significant head and neck manipulation. It is possible that with this manipulation, an atherosclerotic plaque from her diseased carotid became dislodged and embolized to a smaller vessel causing ischemia and possible infarction.

One might also consider the CVA may have been a result of a drug-induced episode of hypotension combined with an 80 to 90% occlusion of both the left and right carotid arteries. This category of stroke is called hemodynamic stroke and has been documented.¹⁴ In a study of the frequency and pathogenesis of this category of stroke, the authors note that hemodynamic stroke may account for as high as 9.6% of stroke unit admissions. All patients (except 2) in their study had evidence of hypotension and watershed infarction,

Table. Ranges of Sensitivities for Detecting Ischemic and Hemorrhagic Stroke¹⁸

Neuroimaging Method*	Type of Stroke	Time Since Symptom Onset			
		>3 Hours	>6 Hours	<48 Hours	>48 Hours
Head CT without contrast	Sensitivity for hemorrhagic stroke	Evidence limited, assumed 100% in many studies	86–90%†	93%	17–58%
	Sensitivity for ischemic stroke	64–85%	47–80%	23–81%	53–74%
Brain MRI	Sensitivity for hemorrhagic stroke	No MRI studies identified for this time frame	86–90%†	46%	38–97%
	Sensitivity for ischemic stroke		65%	84–88%	94–98%

* CT indicates computed tomography; MRI, magnetic resonance imaging.

† Sensitivity is 86% when other imaging modality is used as the gold standard, 90% when discharge diagnosis of acute hemorrhagic stroke is used as the gold standard.

commonly in association with severe carotid stenosis/occlusion.¹⁴

Whatever the initial cause of the CVA, the fact remains that with an undiagnosed 80 to 90% occlusion of the right carotid artery and a 90% occlusion of the left carotid artery, the probability of having an acute cerebrovascular event was high.^{15,16} The patient was fortunate in that she was in an environment where she could be medically managed in a timely, appropriate manner. Because the patient was intubated and well-ventilated (SpO₂ 99% and end-tidal CO₂ 31–35), she never became hypoxic or acidotic. Hypoxia and acidosis could have only further complicated the patient's acute ischemic event, possibly resulting in increased areas of infarction. In addition, the hypotension was also managed in a timely fashion. It is known that hypotension following an ischemic stroke is one of the major predictors of early cardiac morbidity and mortality.¹² Hypotension only perpetuates poor perfusion, which in an ischemic brain will increase the area of infarct.¹²

Good preoperative assessment and perioperative management are paramount to prevent or minimize adverse outcomes. The anesthesia provider must always evaluate risk versus benefit when making treatment decisions. Risk factors for stroke incidence for noncardiac and nonvascular surgeries include: type of surgery, history of coronary artery disease, history of chronic congestive heart failure, chronic kidney disease, history of cerebrovascular disease, preoperative abnormal ECG, intraoperative hypotension, and blood transfusion.⁸ The goal should be to reduce risk, educate and inform patients regarding risk, make careful surgical treatment decisions, and identify high-risk patients. Perioperative antithrombotic strategies should be considered for high-risk patients.¹⁷

Patients exhibiting stroke-like symptoms should be imaged immediately, within 3 hours of onset of symptoms.¹⁸ CT without contrast is the gold standard for ruling out a stroke. Other modalities, such as CT with contrast or magnetic resonance imaging may be used if

enhancement is needed. The Table shows ranges of sensitivities for detecting ischemic and hemorrhagic stroke at >3 hours, >6 hours, <48 hours, and >48 hours.^{18–20}

Patients with acute ischemic stroke may be candidates for IV thrombolytics, which must be administered within a 3-hour window of stroke onset to be effective.¹⁸ Rapid assessment, imaging, and management will reduce morbidity and mortality. The goals of stroke management include early stroke evaluation, good general medical care, specific interventions such as reperfusion strategies for ischemic stroke, and physiological optimization for cerebral resuscitation.²¹

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