CONTINUING EDUCATION

Identification and Management of Ischemic Stroke in the Postanesthesia Care Unit
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A thromboembolic stroke is a debilitating event that can occur with little or no warning. This report details the case of a 63-year-old male experiencing a stroke in the immediate postoperative period after total knee arthroplasty. Risk for perioperative stroke is influenced by age, sex, ethnicity, comorbidities, and some medications. The depressed neurocognitive state of patients recovering from anesthesia warrants special consideration for the identification and management of perioperative stroke.

Keywords: stroke, arthroplasty, paresis, aphasia, PACU.
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OBJECTIVES—1. Identify risk factors for ischemic stroke. 2. Review the practical physiology and clinical presentation of ischemic stroke. 3. Identify interventions to manage ischemic stroke in a postanesthesia care unit (PACU) setting.

Perioperative ischemic stroke can impart devastating, life-changing consequences for patients and their families. Risk factors associated with comorbidities, surgical procedure, and type of anesthesia delivery will be explored, along with techniques to identify and manage these critical events. This report highlights a case where a nurse in the PACU identified symptoms of perioperative stroke.

Clinical Case

Preoperative Phase

A 63-year-old male presented for an elective right total knee arthroplasty at a medium-sized hospital. In addition to osteoarthritis, his medical history was significant for hypertension and diabetes mellitus type II. His daily home-medication regimen included oral lisinopril-hydrochlorothiazide, 20-25 mg; amiodipine, 10 mg; atorvastatin, 40 mg; metformin, 1000 mg; meloxicam, 15 mg; and aspirin, 81 mg. Eight days before surgery, he visited his orthopedic surgeon for a preoperative evaluation. Historically, the patient’s blood pressure range normally was 120-130/65-75 mmHg, but he had not taken his antihypertensive medication that day, and his blood pressure was 197/87 mmHg. He was referred to the emergency department for control of hypertensive
urgency. The consulting cardiologist recommended the patient resume his existing medication regimen. His blood pressure returned to his baseline. The cardiologist recommended that the patient proceed with surgery as scheduled.

**Day of Surgery**

The patient reported no interval changes to his health status since his preoperative visit and continued compliance with prescribed antihypertensive medications. He reported that he took lisinopril-hydrochlorothiazide, amlodipine, and atorvastatin before arrival at the hospital for his procedure. On clinical presentation, the following were observed: body mass index of 37.6 kg/m²; afebrile; blood pressure, 120/73 mmHg; heart rate, 78; respiratory rate, 16; SpO₂, 97% on room air. His medical record revealed a normal stress echocardiogram less than 12 months earlier with an ejection fraction of 60-65%, normal sinus rhythm, hemoglobin A1C (HgbA1C) 3 months prior 6.7%, and fasting glucose on arrival was 130 mg/dL.

The patient had received 975 mg of oral acetaminophen and 200 mg of celecoxib the morning of surgery. The patient received a combined spinal epidural for the primary anesthetic. After the spinal anesthetic was administered, an indwelling epidural catheter was inserted to facilitate postoperative analgesia delivery to be started in the PACU. Deep sedation was maintained with a propofol and ketamine infusion. Before skin incision, the patient received 2 g of cefazolin intravenously for infection prophylaxis. One gram of tranexamic acid was given before tourniquet inflation and repeated before skin closure. His blood pressure was maintained within 20% of his baseline, and there was no evidence of dysrhythmia. After an unremarkable 103-minute surgical course, the patient emerged from anesthesia with bilateral upper extremity arm movement and was transferred onto a hospital bed.

**Postoperative Course**

Immediately after handoff to the perianesthesia nurse was completed, the perianesthesia nurse observed the new onset of asymmetrical facial droop, left hemiparesis, and slurred speech. Although the patient had a combined spinal epidural anesthetic, he had begun to regain motor control and sensation in his right lower extremity upon assessment. Oxygen was administered at 10 L/minute, and a 12-lead electrocardiogram (ECG) showed normal morphology and sinus rhythm. The nurse notified the supervising anesthesia practitioner who then consulted internal medicine to coordinate transfer to a stroke center. Both providers arrived at the bedside within 20 minutes of the initial onset of symptoms.

The providers were ruling out potential differential diagnoses with a high suspicion for an acute thromboembolic or hemorrhagic stroke. Within 40 minutes of symptom onset, the patient underwent noncontrast computed tomography (CT) scan of the head and was transferred to the emergency department. Because the patient was not admitted to the hospital at that time, the anesthesia provider felt the patient was safe to be continuously monitored in the emergency department (Administratively at this facility, the patient is not considered admitted to the hospital as an inpatient until they arrive on the medical ward.). In addition, if the patient needed to be transferred to a stroke center, it could be expedited through the hospital’s emergency department. The CT scan revealed no sign of hemorrhage. Stroke-like symptoms can also be caused by acute coronary syndrome or hypoglycemia, but ECG changes were not present and bedside blood glucose was 175 mg/dL, eliminating the likelihood of an acute coronary syndrome or hypoglycemic event.

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**Table 1. Stroke Risk Factors**

<table>
<thead>
<tr>
<th>Modifiable Risk Factors</th>
<th>Nonmodifiable Risk Factors</th>
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<tr>
<td>Smoking</td>
<td>Surgical procedure</td>
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<tr>
<td>Hypertension</td>
<td>Type of anesthetic technique</td>
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<tr>
<td>Dyslipidemia</td>
<td>Age &gt;55 years</td>
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<tr>
<td>Cerebrovascular disease</td>
<td>Ethnicity</td>
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<tr>
<td>Cardiac disease</td>
<td>Race</td>
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<td>Dysrhythmias</td>
<td>Gender</td>
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<td>Excessive alcohol</td>
<td>Previous stroke or transient ischemic attacks</td>
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<td>consumption</td>
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<tr>
<td>Diabetes mellitus</td>
<td>Some genetic disorders</td>
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<td>Systemic inflammation</td>
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<tr>
<td>Dietary habits</td>
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<tr>
<td>Physical inactivity</td>
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<td>Waist-to-hip ratio</td>
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Emergency department providers and a neurologist evaluated the patient in the emergency department within 60 minutes from onset of symptoms. A thromboembolic stroke remained as the likely cause. The patient was not considered a candidate for intravenous thrombolytic therapy before transfer to the stroke center because of his recent major surgery and the presence of an indwelling epidural catheter. The patient was transported to a certified stroke center 80 minutes after symptom onset.

Upon arrival to the stroke center, a CT angiography scan revealed a complete right internal carotid artery and middle cerebral artery thromboembolism. The patient immediately underwent a thrombectomy and stent placement by an interventional radiologist. Later studies revealed a normal transesophageal echocardiogram, no evidence of a pulmonary embolism or venous thromboembolism, and no heart rhythm abnormalities. He continued to receive medical and physical therapy at a rehabilitation center upon discharge. Three months after the event, the patient had regained the ability to swallow and move his left toes; however, functional deficits continued to impair his speech, self-efficacy, and ability to ambulate.

Pathophysiology

Ischemic stroke occurs when cerebral blood flow is compromised. Thrombi or emboli may occlude one or more cerebral arteries, resulting in ischemia distal to the occlusion. The signs and symptoms correspond to the area of ischemia in the brain. Some brain structures normally receive only a small amount of blood flow, and further decreased blood flow can result in focal symptoms such as visual disturbances, aphasia, or weakness in one extremity. Transient ischemic attacks are temporary, lasting less than 24 hours, focal neurological deficits resulting from ischemia to tissues of the central nervous system that may give some warning as they often precede an ischemic stroke.1,2 Large thromboembolic stroke symptoms include sudden onset of asymmetrical numbness and/or weakness of the face, arms, or legs; altered mental status; receptive or expressive aphasia; dysphagia; severe headache; gait disturbances; or visual changes. Fortunately, some cases may be reversible when treatment is rapidly provided.3

Risk Reduction Strategies

Although stroke is not always preventable, a useful step in managing risk is identifying risk factors when screening patients for surgery (Table 1).4,5 Health care providers can work with their patients to better mitigate modifiable risk factors associated with strokes such as smoking, hypertension, dysrhythmias, cardiac disease, alcohol consumption, systemic inflammation, and diabetes.4,7 Nonmodifiable risks for stroke include patients older than 55 years, males, those with prior transient ischemic attack or stroke, and individuals having African, Native American, or Hispanic ancestry.3 Body mass index alone does not influence risk for perioperative stroke.6 Preoperative assessments such as ECGs, echocardiograms, cardiac stress tests, carotid ultrasounds, chest x-rays, HgbA1C results, and metabolic function tests are advocated to identify comorbidities such as dysrhythmias, decreased cardiac function, carotid disease, diabetes, or renal dysfunction that might increase the risk for perioperative stroke.5

The risk for a perioperative stroke can be related to the surgical procedure being performed, anesthetic technique, and patient-related factors. Surgical procedures that pose the highest risk for perioperative stroke are cardiac and vascular surgeries, at 2-10%, whereas noncardiovascular procedures are associated with a much lower risk, between 0.08 and 0.7%, including total joint replacement, and any emergent procedure.5,8-10 General anesthesia is associated with a significantly higher risk for stroke than regional anesthesia, 5.89 odds ratio, or odds of occurrence, during a total joint replacement.5 Patient factors that are associated with higher risk for stroke include the following: age ≥55 years doubles risk from 0.2% to 0.4% and intraoperative dysrhythmia (1.06 odds ratio).7 Regional anesthesia has been suggested to reduce risk for an ischemic stroke.5 Elective cases should be delayed when comorbidities are not medically optimized.11

Intraoperative factors can impact risk for perioperative stroke. Reduction in mean arterial pressure of more than 30% below baseline values has been shown to increase the risk for the development of an ischemic stroke secondary to hypoperfusion.12 Tissues distal to vascular structures that are dependent on collateral circulation are not
able to be perfused sufficiently at low pressures, resulting in ischemia unless adequate perfusion pressure is restored. Persistent systemic hypotension can worsen a stroke caused by an existing cerebrovascular occlusion, and severe intraoperative cerebrovascular hypotension can directly induce cerebral ischemia. Patient positioning also can cause relative hypoperfusion to the brain when the head is elevated above the heart. It is important to maintain blood pressures as close as possible to the patient’s baseline to ensure adequate cerebral perfusion, especially while adjusting for the difference in the patient’s head height above the heart.

Medications and Risk for Stroke

Tranexamic acid prevents the lysis of clots and is not associated with an increased risk for stroke or other venous thromboembolic events in healthy patients. Patients who are in a hypercoagulable state have an increased potential for additional thromboembolic events. Tranexamic acid administration is contraindicated for patients with a history of thromboembolic events such as ischemic stroke, myocardial infarction, or coronary artery stent placement.

Celecoxib and meloxicam are often used to treat pain associated with osteoarthritis. These and other cyclooxygenase-2 inhibitors are associated with increased risk for adverse cardiovascular events, including ischemic stroke. Hyperlipidemia is weakly associated with an increased risk for stroke. Statin therapy has not been shown to reduce this risk.

Angiotensin-converting enzyme (ACE) inhibitors treat hypertension by disrupting the renin-angiotensin-aldosterone system. ACE inhibitors are also known to halt atherosclerotic plaque progression, reduce systemic inflammation, improve endothelial function, reduce vascular smooth muscle hypertrophy, and attenuate compensatory left ventricular hypertrophy. ACE inhibitors may even prevent vascular disease progression and reduce the risk for stroke.

Calcium channel blockers (CCBs) help improve coronary artery circulation by reducing myocardial contractility and peripheral vascular resistance. They also reduce inflammation and confer the highest risk reduction for stroke compared with other antihypertensive agents. When combined with ACE inhibitors or angiotensin II receptor blockers, CCBs reduce the mortality associated with vascular disease.

Identification and Treatment

The American Heart Association and American Stroke Association have developed recommendations for rapid identification of a suspected stroke. These recommendations include the following: ensure or establish a patent airway for effective respirations, assess vital signs, and provide supplemental oxygen when needed. Intra-venous access will facilitate laboratory analysis of blood glucose, complete blood counts, and coagulation evaluation. Performing a neurological assessment, such as the National Institutes of Health Stroke Scale (Supplementary Figure 1), and recording the score enables the onset and severity of symptoms to be clearly documented. The scale captures the level of consciousness, best gaze, visual fields, facial palsy, and upper and lower motor function. A 12-lead ECG should also be obtained to rule out an acute coronary event. If a cause for the suspected stroke is identified during this process, it should be promptly treated. The institution’s stroke team should be consulted, if available, or other expert consultation should be sought. The patient should be prepared for an emergency CT scan of the brain to rule out a hemorrhagic stroke within 20 minutes of identification or arrival to emergency department. The American Heart Association has detailed its recommendations in its Advanced Cardiac Life Support Stroke Algorithm that closely mirrors the aforementioned recommendations jointly published with the American Stroke Association. Once the diagnosis of a stroke is confirmed, the institution’s protocol should be implemented as soon as possible. When the patient is hemodynamically stable, he or she should be transferred to a stroke center or the intensive care unit where close hemodynamic, neurologic, and blood glucose monitoring and management will be instituted.

When an intraoperative stroke occurs during deep sedation or general anesthesia, most signs or symptoms will likely not be apparent. Incomplete recovery from anesthesia in the PACU further
delays identification of stroke symptoms in the postoperative period.\textsuperscript{11} Cardiac rhythm changes, hemodynamic instability, delayed emergence from anesthesia, or new focal deficits may be the only indicators of an intraoperative stroke.\textsuperscript{11} If intraoperative stroke is suspected in an anesthetized patient, it may still be possible for anesthesia providers to discover impaired cerebral perfusion. Although such detection may be difficult, near-infrared spectroscopy and other cerebral oxygenation monitors are available to help alert anesthesia practitioners about focal deficits from regional cerebral ischemia.\textsuperscript{9} Intraoperative electroencephalography, somatosensory evoked potentials, and motor evoked potentials can be used for patients receiving general anesthesia.\textsuperscript{9} This is considered a critical intraoperative event, and the anesthesia provider would communicate this finding to the operating room team and determine the best course of action to mitigate further risks to the patient.

When the exact onset of symptoms is known, thrombolytic medications, such as alteplase or tenecteplase, can be administered within 180 minutes of symptom onset. The treatment window may be extended to 270 minutes after onset of symptoms when the risk of intracranial hemorrhage does not outweigh the potential benefit of receiving one of these agents.\textsuperscript{24,26} Contraindications for thrombolytic therapy are often associated with either a risk of uncontrolled hemorrhage, such as after major surgery, or potential for greater harm, such as within 3 months of a prior ischemic stroke. These contraindications for thrombolytic therapy include major surgical procedure within the preceding 14 days; head trauma or stroke within the previous 3 months; hemorrhagic stroke, multilobar infarct, intracranial neoplasm, aneurysm, arteriovenous malformation, coagulopathy, and noncompressible arterial puncture within previous 7 days; blood glucose less than 50 mg/dL; or blood pressure greater than 185/110 mmHg.\textsuperscript{5,24}

Other treatments may be considered when risk for hemorrhage is not acceptable. Focused intraarterial thrombolytic interventions have been proposed, but their safety and efficacy have not been established.\textsuperscript{27,28} When thrombolytic medications are contraindicated, endovascular treatments such as stent placement or a thrombectomy may be considered. A thrombectomy is a minimally invasive procedure performed in an interventional radiology setting or operating room to remove a thrombus or embolus and restore blood flow distal to the occlusion.\textsuperscript{27,28}

**Conclusion**

Unfortunately, there are no risk reduction strategies known to completely eliminate a stroke risk. Despite strategies such as regional anesthesia being used in this case, this patient still suffered a perioperative stroke. Postoperative nurses familiar with risk factors for stroke are best positioned to recognize when strokes occur. Perianesthesia nurses are often the only health care staff at the bedside, being the first to notice even subtle deficits. Such vigilance will reduce the time between symptom onset and definitive treatment and likely lead to better outcomes for these patients. Nurses can facilitate identification of possible causes and appropriate interventions to treat these uncommon but potentially devastating events.

**Supplementary Data**

Supplementary data related to this article can be found at https://doi.org/10.1016/j.jopan.2019.01.011.

**References**

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1.0 Contact Hours

Purpose of the Journal of PeriAnesthesia Nursing: To facilitate communication about and deliver education specific to the body of knowledge unique to the practice of perianesthesia nursing.

Outcome of this CNE Activity: To enable the nurse to increase knowledge on the care of the pediatric patient with ischemic stroke.

Target Audience: All perianesthesia nurses.

Article Objectives

1. Identify risk factors for ischemic stroke
2. Review the practical physiology and clinical presentation of ischemic stroke
3. Identify interventions to manage ischemic stroke in a postanesthesia care unit (PACU) setting

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