Postprocedure Intensive Care Management of Stroke Patients

Appropriate postprocedure management improves outcomes in stroke patients.

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Patients with cerebrovascular disease who undergo endovascular procedures are at risk for a variety of complications. They can experience new neurologic insults, or their deficits may be worsened by a host of systemic abnormalities. The goals of pre- and postprocedure management of these patients should be to minimize risk of complications and to maximize recovery potential. Although the principles of neurologic critical care are not specifically different for patients undergoing endovascular procedures, there are certain therapeutic goals that must be kept in mind for patients with cerebrovascular disease.

BEFORE AND DURING THE PROCEDURE

One of the most effective ways to prevent complications and confusion after the case is to know the patient well before the procedure. Familiarity with each patient's comorbidities allows preparedness for certain complications. This set of information is not only essential during the procedure, but needs to be relayed to the personnel in charge of postprocedure care. In particular, diabetes mellitus, chronic obstructive pulmonary disease, coronary disease, heart failure, renal failure, and coagulopathy all necessitate special attention after the procedure. These risk factors should be ascertained before considering intervention and conveyed to the team providing postprocedure care. In a study of 2,899 patients undergoing cerebral angiography, complications were more common in patients over 55 years of age, those with cardiovascular disease, and when fluoroscopy times were longer than 10 minutes.[1] Therefore, age and the presence of cardiovascular disease deserve special attention.

Probably the most important aspect of preprocedure care is an accurate and well-documented neurologic examination. The details of the exam are crucial in neurologic surveillance for worsening deficits or the development of new deficits. These complications can sometimes be reversed, but only when they are recognized promptly.

Likewise, events during the procedure must be conveyed to the critical care team. Hemodynamic status, blood loss, fluid shifts, anesthesia or sedation protocols, drugs used, details of the procedure such as the approach, blood vessels traversed and involved, procedural techniques, devices utilized, and procedure-related complications all have important ramifications for postprocedure care.

AFTER THE PROCEDURE

The postprocedure care of stroke patients undergoing endovascular treatment is largely similar to the care of acute stroke patients who did not receive endovascular treatment. However, there are differences that should influence management. For example, endovascular patients may have recently recanalized vessels and require individually tailored blood pressure goals for optimal cerebral perfusion. They are at risk for rethrombosis or bleeding and require more intensive neurologic monitoring. They may also be anticoagulated and require more frequent monitoring of bleeding parameters. For all these reasons and more, patients undergoing endovascular procedures should be admitted to an intensive care unit, ideally a specialized neuroscience critical care unit (NCCU), after the procedure. As time is of the essence in the prevention or reversal of acute brain injuries, the need for specialized training to facilitate early recognition of neurologic deterioration is crucial not only for the physician staff, but more importantly for the nursing staff that has direct contact with patients during this critical period. The use of an NCCU has been associated with reduced mortality and offers specialized neurologic multimodal monitoring.[2] The critical care of patients with neurologic injuries was further advanced recently with the formation of the multidisciplinary Neurocritical Care Society (www.neurocriticalcare.org) that includes neurointerventional specialists in its membership.

MONITORING

Postprocedure monitoring should focus on neurologic status and physiologic parameters, such as vital signs and intracranial pressure (ICP). Surveillance of neurologic status is of particular importance because as many as 37% of stroke patients will deteriorate within the first 24 hours.[3] An initial postprocedure exam should be performed and documented by the accepting physician and nursing team and compared to the preprocedure exam. A difference between the pre- and postprocedure exams should be discussed with the interventional team immediately. This may signify an expected deficit given the lesion and intervention or a procedure-related complication, and prompt investigation may be necessary. After the initial exam, serial neurologic checks can be performed by the nursing staff. A detailed evaluation, including mental status, cranial nerve, motor, and sensory system assessments, may be necessary to detect early or subtle deficits. Changes here may reflect reocclusion, hemorrhagic transformation, expanding infarct area, worsening edema, rising ICP, or other serious but potentially reversible conditions. Therefore, changes in neurologic function should be reported to the physician immediately and investigated. At our own institution, NCCU nurses perform neurologic assessments every 15 minutes for 2 hours, then every 30 minutes for 6 hours, then every 1 hour for 16 hours after thrombolysis for acute stroke.

It is also vitally important to monitor key physiologic parameters after neurologic intervention. With decreased level of consciousness, the airway and respiratory status may be compromised. The physician should have a low threshold for intubation and mechanical ventilation. There is also potential for cardiac arrhythmia after stroke, and continuous cardiac monitoring should be employed. Strict blood pressure control is of particular importance in stroke patients, and therefore direct continuous monitoring via an arterial line is frequently employed. In appropriate cases, additional parameters that may be monitored in the NCCU include temperature, ICP, cerebral perfusion pressure, and the electroencephalogram.[2]

BLOOD PRESSURE

Blood pressure is often significantly elevated in the setting of acute stroke. This pathophysiologic response can be helpful in the short-term, providing some perfusion to the ischemic penumbra despite a highly stenotic or thrombosed vessel. Therefore, aggressive treatment of hypertension in the setting of acute stroke is not recommended and may in fact be harmful.[4] The American Heart Association (AHA) has recommended observation without treatment for blood pressure <220/120 mm Hg in the absence of other organ dysfunction.[5] If blood pressure rises above this level or other organ damage occurs, the AHA recommends treatment with either intravenous labetalol or nicardipine, or nitroprusside if the diastolic pressure is >140 mm Hg. We tend to avoid nitroprusside as it can cause cerebral vasodilation and potentially increase ICP.[6] Labetalol can be effective in lowering blood pressure but may lead to significant bradycardia. In our experience, intravenous nicardipine, a calcium channel blocker, seems more effective in safely lowering blood pressure with less likelihood of bradycardia or increased ICP.

In patients who receive thrombolytic therapy, excessive blood pressure elevation may cause further complications and should therefore be avoided. In the NINDS study of intravenous rt-PA for acute stroke, blood pressure was kept below 185 mm Hg systolic or 110 mm Hg diastolic.[7] Although there are no specific guidelines for patients receiving intra-arterial rt-PA, we generally adhere to similar goals for patients undergoing intervention. In addition, the rapid recanalization of previously stenotic vessels may lead to relative hyperperfusion and further brain injury in the form of edema or hemorrhage. Therefore, even lower blood pressures may be targeted if large vessels are recanalized or if the patient exhibits signs of reperfusion injury.[8,9]

Caution must be exercised when treating hypertension acutely. The reason is that chronic hypertension can lead to a shift in the cerebral autoregulation curve, the mechanism that keeps cerebral blood flow constant over a wide range of mean arterial pressures (Figure 1). With longstanding hypertension, the curve becomes shifted to the right, and therefore lower, or normal, blood pressures may be inadequate for cerebral perfusion. As a result, reducing the blood pressure too quickly in the setting of chronic hypertension can result in cerebral ischemia. In practice, we generally do not treat hypertension up to 220 mm Hg systolic in acute ischemic stroke not treated with thrombolysis for the first 24 hours. Thereafter, hypertensive therapy is slowly initiated. In acute ischemic stroke treated with intravenous rt-PA, we target blood pressure between 120/80 and 180/105 mm Hg. These goals are similar for patients who have undergone endovascular intervention, although individual goals are tailored to each patient depending on baseline blood pressure and the nature of the intervention.

ANTICOAGULATION AND ANTIPLATELET AGENTS

Based on multiple large trials, both the AHA and the American Academy of Neurology (AAN) have concluded that most patients should not receive anticoagulants in the setting of acute stroke.[10,11] Specifically, unfractionated heparin, heparinoids, low-molecular-weight heparin, and abciximab have not been shown to improve outcomes or to decrease the chance of stroke recurrence when adminis-

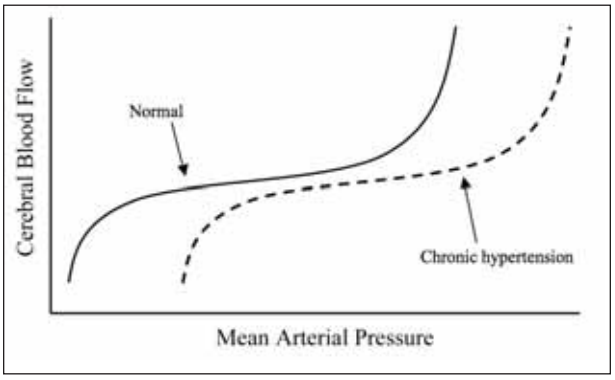

Figure 1. The autoregulation of cerebral blood flow. Normally, cerebral blood flow remains stable across a range of mean arterial pressure of approximately 50 to 150 mm Hg (solid line). In chronic hypertension, autoregulation accommodates to elevated blood pressure and shifts to the right (dotted line).

tered within 48 hours of stroke. Their use for these purposes in the setting of acute stroke is discouraged by the AHA and AAN because of a high risk of intracranial hemorrhage. However, stroke patients are at significant risk for the development of deep vein thrombosis (DVT). The use of subcutaneous heparin or low-molecular-weight heparin for DVT prophylaxis is supported by clinical evidence, and it is recommended by the AHA along with early mobilization and intermittent external compression stockings. In the postendovascular procedure setting, the absence of well-controlled studies regarding the use of anticoagulants has led to wide practice variability across institutions. Well-designed clinical studies addressing anticoagulation after endovascular procedures are therefore needed.

In contrast to anticoagulation, antiplatelet agents should be used to treat ischemic stroke. There is strong evidence that 160 mg to 325 mg of aspirin administered within 24 to 48 hours of ischemic stroke improves outcomes and reduces the risk of recurrence,[5] despite a small increase in the risk of hemorrhagic stroke.[11] However, aspirin should not be used as an adjunctive therapy within 24 hours of thrombolysis. In most cases, we begin aspirin therapy 24 hours after intervention for acute stroke, and we consider the use of additional antiplatelet agents at that time.

TEMPERATURE

Hyperthermia is associated with poor neurologic outcomes in ischemic stroke,[12] as it is with subarachnoid hemorrhage.[13] The source of fever should be aggressively sought, and empiric antibiotic therapy should be initiated without delay. Additionally, febrile patients should be actively cooled using pharmacologic antipyretics as well as adjunctive cooling procedures, with a goal of normothermia. Therapeutic hypothermia has been shown to improve neurologic outcomes only in comatose survivors of cardiac arrest, but it has not yet been proven effective in stroke patients. Until definitive clinical trials show outcome benefit of therapeutic hypothermia in patients with acute ischemic strokes, the temperature management goal should focus on fever control or maintenance of normothermia.

GLUCOSE

Diabetes mellitus is an important risk factor for stroke, and hyperglycemia at the time of stroke may lead to poor outcomes.[14] Although intensive insulin therapy has not been studied for stroke patients in particular, maintaining blood glucose levels of 80 mg/dL to 110 mg/dL significantly reduced morbidity and mortality in a cohort of 1,548 critically ill patients.[15] Tight glucose control has also been shown to reduce secondary injury to the central and peripheral nervous systems of intensive care patients.[16] Therefore, we aggressively treat hyperglycemia in the setting of acute stroke.

COMMON NEUROLOGIC COMPLICATIONS

Complications in stroke patients who have received intervention may be divided into those that are related to stroke and those that are related to the procedure itself. Complications in the latter category include iatrogenic dissection and stroke, hematoma formation, and allergic reaction.[1] These complications are commonly considered by the interventionist and are therefore generally recognized during or immediately after the procedure. In the remaining section, we will focus on the stroke-related complications.

Sudden neurologic deterioration in the postprocedure period may indicate a complication related to the stroke itself. The most common etiologies include recurrent stroke, intracerebral hemorrhage, cerebral edema, elevated ICP, and seizure. The management of any change in neurologic status should always begin with a bedside evaluation of airway, breathing, and circulation, followed by neurologic assessment and institution of supportive measures. Once a diagnosis is reached, specific therapies may be initiated for definitive treatment.[17]

Postprocedure intracerebral hemorrhage is usually associated with an acute decline in neurologic function and is diagnosed by a head CT. Once hemorrhage is identified, the clinician should rapidly obtain blood coagulation studies and a platelet count. Coagulopathy should be reversed using fresh frozen plasma, cryoprecipitate, protamine, or platelet transfusion, depending on the etiology. Of particular importance is early intervention to lower the blood pressure, which may prevent expansion of the hematoma. The AHA recommends systolic pressures <180 mm Hg,[18] and other studies have suggested that systolic pressure <150 mm Hg is associated with decreased risk of hematoma expansion.[19]

Surgical decompression or evacuation of the hematoma may be warranted cases of cerebellar hemorrhage, but it is less helpful in hemorrhage involving other areas of the brain.[20]

Seizures occur in approximately 5% of acute strokes and are usually of the generalized tonic-clonic type, although they are occasionally subclinical. Given the relative infrequency of seizures, prophylactic therapy should be individualized based on each patient's seizure risk. When seizures do occur, they should be treated acutely with an intravenous benzodiazepine, such as lorazepam. A longer-acting anticonvulsant, such as phenytoin or valproate, may subsequently be administered. It is also important that the cause for seizure be investigated, as it may be a sign of intracerebral hemorrhage or a new stroke. Typical investigations include bedside neurologic examination, repeat brain imaging, and electroencephalography.

Brain edema and elevated ICP frequently occur as a result of ischemic stroke. Specific causes include hemorrhagic transformation and hyperperfusion after thrombolysis. Symptoms and signs of elevated ICP include headache, nausea, vomiting, visual changes, ataxia, and abducens nerve palsy. Severely elevated ICP may lead to Cushing's triad of bradycardia, respiratory distress, and hypertension, which often precedes death. Elevated ICP may also cause brain herniation, which can present as mental status change, cranial nerve palsies, and brainstem dysfunction. Any of these signs or symptoms may indicate a catastrophic neurologic event and should be aggressively treated. Acute treatment of elevated ICP begins with mild hyperventilation with a goal pCO_2 of 25 mm Hg to 30 mm Hg. Osmolar therapy may also be initiated, either in the form of intravenous mannitol or hypertonic saline. External ventricular drainage can be used in cases of obstructive hydrocephalus, or when ICP monitoring is warranted. Surgical decompression may be considered in cases of cerebellar infarction and hemispheric strokes. Corticosteroids are not useful in treating cytotoxic edema associated with infarction and should be avoided. Prompt recognition and immediate aggressive management of cerebral herniation syndromes may lead to clinical reversal and favorable outcome in a subset of patients.[21]

CONCLUSION

Appropriate postintervention care for stroke patients begins before the procedure, with a detailed history and neurologic exam. After the procedure, the patient should be monitored in an intensive care unit—preferably one specialized for neurocritical care. Important goals of management include normothermia, glycemic control, appropriate use of antiplatelet agents, DVT prophylaxis, and blood pressure control. Common complications include recurrent stroke, intracerebral hemorrhage, seizure, and elevated ICP, and can be treated if recognized swiftly. ■

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1. Willinsky RA, Taylor SM, TerBrugge K, et al. Neurologic complications of cerebral angiography: prospective analysis of 2,899 procedures and review of the literature. Radiology. 2003;227:522-528.
2. Suarez JI. Outcome in neurocritical care: advances in monitoring and treatment and effect of a specialized neurocritical care team. Crit Care Med. 2006;34(9 suppl):S232-S238.
3. Davalos A, Toni D, Iweins F, et al. Neurological deterioration in acute ischemic stroke: potential predictors and associated factors in the European cooperative acute stroke study (ECASS) I. Stroke. 1999;30:2631-2636.
4. Urrutia VC, Wityk RJ. Blood pressure management in acute stroke. Crit Care Clin. 2006;22:695-711.
5. Adams HP, Jr, Adams RJ, Brott T, et al. Guidelines for the early management of patients with ischemic stroke: a scientific statement from the Stroke Council of the American Stroke Association. Stroke. 2003;34:1056-1083.
6. Weiss MH, Spence J, Apuzzo ML, et al. Influence of nitroprusside on cerebral pressure autoregulation. Neurosurgery. 1979;4:56-59.
7. Brott T, Lu M, Kothari R, et al. Hypertension and its treatment in the NINDS rt-PA Stroke Trial. Stroke. 1998;29:1504-1509.
8. Pan J, Konstas AA, Bateman B, et al. Reperfusion injury following cerebral ischemia: pathophysiology, MR imaging, and potential therapies. Neuroradiology. 2007;49:93-102.
9. Coutts SB, Hill MD, Hu WY. Hyperperfusion syndrome: toward a stricter definition. Neurosurgery. 2003;53:1053-1058.
10. Adams H, Adams R, Del Zoppo G, et al. Guidelines for the early management of patients with ischemic stroke: 2005 guidelines update a scientific statement from the Stroke Council of the American Heart Association/American Stroke Association. Stroke. 2005;36:916-923.
11. Coull BM, Williams LS, Goldstein LB, et al. Anticoagulants and antiplatelet agents in acute ischemic stroke: report of the Joint Stroke Guideline Development Committee of the American Academy of Neurology and the American Stroke Association (a division of the American Heart Association). Neurology. 2002;59:13-22.
12. Hajat C, Hajat S, Sharma P. Effects of poststroke pyrexia on stroke outcome: a meta-analysis of studies in patients. Stroke. 2000;31:410-414.
13. Fernandez A, Schmidt JM, Claassen J, et al. Fever after subarachnoid hemorrhage: risk factors and impact on outcome. Neurology. 2007;68:1013-1019.
14. Bruno A, Biller J, Adams Jr HP, et al. Acute blood glucose level and outcome from ischemic stroke. Trial of ORG 10172 in Acute Stroke Treatment (TOAST) Investigators. Neurology. 1999;52:280-284.
15. van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med. 2001;345:1359-1367.
16. van den Berghe G, Schoonheydt K, Becx P, et al. Insulin therapy protects the central and peripheral nervous system of intensive care patients. Neurology. 2005;64:1348-1353.
17. Finley CA, Wijman CA. Critical care of acute ischemic stroke. Crit Care Clin. 2006;22:581-606.
18. Broderick JP, Adams Jr HP, Barsan W, et al. Guidelines for the management of spontaneous intracerebral hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Stroke. 1999;30:905-915.
19. Ohwaki K, Yano E, Nagashima H, et al. Blood pressure management in acute intracerebral hemorrhage: relationship between elevated blood pressure and hematoma enlargement. Stroke. 2004;35:1364-1367.
20. Mendelow AD, Gregson BA, Fernandes HM, et al. Early surgery versus initial conservative treatment in patients with spontaneous supratentorial intracerebral haematomas in the International Surgical Trial in Intracerebral Haemorrhage (STICH): a randomised trial. Lancet. 2005;365:387-397.
21. Qureshi AI, Geocadin RG, Suarez JI, et al. Long-term outcome after medical reversal of transtentorial herniation in patients with supratentorial mass lesions. Crit Care Med. 2000;28:1556-1564.