Neurocritical Care Aspects of Ischemic Stroke Management

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KEYWORDS

• Stroke • Fibrinolysis • Neurointensive care • Cerebral thrombectomy
• Cerebral edema • Decompressive hemicraniectomy • Poststroke hemorrhage
• Carotid endarterectomy

KEY POINTS

- Care of ischemic stroke in the intensive care unit (ICU) begins with postfibrinolytic and/or thrombectomy care, through the management of cerebral edema and hemorrhagic conversion, and finally to prolonged ICU issues such as common infections and immobility complications.
- Acute management of blood pressure (BP) is critical with goals individualized for each type of stroke treatment and revascularization score.
- Familiarity with postcraniotomy complications is important in caring for patients following surgical treatment of malignant cerebral edema in both the anterior and posterior fossa.
- Key differences in the management of embolic strokes caused by infective endocarditis include urgent initiation of antibiotics, avoiding anticoagulation, and evaluation of possible associated mycotic aneurysms.
- Following carotid endarterectomy (CEA) intensivists must anticipate and monitor for the life-threatening complications of expanding wound hematoma, hemodynamic instability, and hyperperfusion injury with hemorrhage and seizures.

INTRODUCTION

Numerous factors contribute to outcomes after acute ischemic stroke (AIS). Neurointensive care (Neuro ICU) management of patients with AIS calls for an individualized approach considering factors such as the size and location of infarct, presenting blood pressure (BP), and National Institute of Health Stroke Scale (NIHSS). The main goals of admission to specialized intensive care units (ICU) for patients with stroke are divided into supportive care and management and prevention of specific complications.

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Supportive care includes airway watch, ventilatory support, and close BP monitoring. Management of specific complications addresses cerebral edema and hemorrhagic transformation, timely recognition of indications for, and postoperative management of decompressive hemicraniectomy (DHC) and carotid endarterectomy (CEA), and prevention of postthrombectomy reocclusion, stroke expansion, and recurrence.[1,2] Additional circumstances such as the management of endocarditis-related strokes, common infectious, and cardiac complications of critically ill stroke patient are presented. All aspects discussed require intensive bedside neuromonitoring in a Neuro ICU to optimize stroke outcomes. NIHSS-trained staff is key for proper clinical monitoring and is thus a Joint Commission Association of Hospital Organization (JCAHO) requirement for primary and comprehensive stroke center certifications.

GENERAL SUPPORTIVE CARE

Respiratory Support

Intubation

Stroke volume and location play a critical role in determining the need for respiratory support. Large hemispheric infarctions (LHI) can cause impairments in the level of consciousness (LOC), respiratory drive, and protective and swallowing reflexes, thus leading to respiratory failure. Per The Neurocritical Care Society guidelines for Large Hemispheric Stroke, intubation is strongly recommended.[3]

With respect to location, brainstem and thalamic strokes affect both LOC and respiratory centers. Erratic respiratory patterns causing ventilatory issues lead to acid/base imbalance. Furthermore, strokes that impair swallowing functions, have a high risk of aspiration and associated complications. Large cerebellar strokes with cytotoxic edema can cause direct brainstem compression resulting in secondary microischemia and dysfunction, or effacement of the fourth ventricle, resulting in obstructive hydrocephalus. Cortical strokes, particularly those with hemorrhagic conversion, can cause status epilepticus with resultant loss of airway protection.[4]

One prospective study in AIS identified GCS less than 10 or respiratory failure, history of hypertension (HTN), and infarct size more than 2/3rd of the MCA territory or LHI as independent risk factors for intubation and mechanical ventilation.[4] Other risk factors frequently considered include[3]:

- Loss of airway protective reflexes
- Signs and symptoms of increased intracranial pressure (ICP)
- Midline shift
- Pulmonary edema or pneumonia
- Imminent need for surgical management

This subset of patients with stroke is at high risk of aspiration and subsequent acute hypoxic or hypercarbic respiratory failure, thus warranting monitoring in a Neuro ICU.

Extubation

Delays in extubation after successful ventilator weaning are associated with higher rates of pneumonia, increased need for tracheostomy, longer ICU length of stay, and increased mortality.[5] However, extubation failure (EF) and subsequent need for emergent reintubation are associated with similar sequelae.

The following general extubation criteria should be considered before the extubation of patients with stroke with adequate airway protection[6]:

- Glasgow Coma Scale greater than 8
- No signs/symptoms of elevated ICP

- Body temperature 36°C to 38.5°C
- Heart rate 60 to 120 bpm
- Systolic blood pressure (SBP) 90 to 185 mm Hg

Additionally, the following respiratory parameters should be considered for the same subpopulation[6]:

- Spontaneous respiratory minute volume (≤12 L)
- Positive end-expiratory pressure (≤5 mm Hg)
- Pa_{O_2}/Fi_{O_2} (>200)
- Rapid shallow breathing index (<105).

Specific criteria for extubation in LHI have been identified[3]:

- Successful spontaneous breathing trials
- Absence of oropharyngeal saliva collections
- Suctioning required less than every 4 hours
- Presence of cough reflex and tube intolerance (ie, if the patient is comfortably tolerating the endotracheal tube off sedation and analgesia, this should give pause for extubation).
- Free of analgesia and sedation

Ultimately, tracheostomy should be considered if there has been one failure of extubation that was not due to an immediately reversible cause (ie, fluid overload, inadequate time off sedation) and if extubation has not been possible in 7 days after intubation. Placing a tracheostomy should be completed by the fourteenth day of intubation, given the increased risk of injury to the posterior oropharynx, vocal cords, and trachea.[6]

Blood Pressure Goals and Monitoring

Stroke etiology and modality of treatment are key factors in setting BP goals. Commonly encountered scenarios are detailed later in discussion and summarized in **Table 1** with antihypertensive options summarized in **Table 2**.

Table 1
Blood pressure targets versus type of stroke treatment used

Stroke Treatment	Blood Pressure Target
Acute Ischemic Stroke, not eligible for tpA	Permissive hypertension (SBP up to 220, DBP up to 120) for first 24–48 h
Acute Ischemic Stroke, tpA eligible before tpA administration	SBP <180 mm Hg and DBP< 105 mm Hg
Acute Ischemic Stroke, tpA eligible post tpA administration	SBP < 185 mm Hg and DBP <110 mm Hg for 24 h
Acute Ischemic Stroke, Postthrombectomy TICI 2a or <	SBP 120–180
Acute Ischemic Stroke, Postthombectomy TICI 2b-3	SBP 120–160

Please note TICI stands for thrombolysis in cerebral infarction score.

Data from Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, et al. 2018 Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/ American Stroke Association. Stroke 2018;49:e46-110; and Matusevicius M, Cooray C, Bottai M, Maza M, Tsivgoulis G, Nunes AP, Moreira T, Ollikainen J, Tassi R, Strbian D, Toni D, Holmin S, Ahmed, N. Blood Pressure After Endovascular Thrombectomy.Stroke 2020; 51(2): 519-525.

Table 2
Antihypertensives used in acute ischemic stroke to achieve blood pressure goals

Labetalol	10 mg IV over 1–2 min. Can repeat Q 5–10 min
Nicardipine	5 mg/h IV, titrate up by 2.5 mg/h every 5–15 min, maximum 15 mg/h; when desired BP reached, adjust to maintain proper BP limits
Clevidipine	1–2 mg/h IV, titrate by doubling the dose every 90 s until desired BP reached; recommended maximum 21 mg/h
Enalapril	Starting dose 1.25 mg IV give over 5 min. Repeat every 20 min. Maximum 5 mg
Hydralazine	Starting dose 10 mg to 20 mg IV. May repeat every 15 min for a total of 3 doses

Data from Refs.[10,24,27]

Tissue plasminogen activator/tenecteplase eligible strokes

Blood pressure goals before tissue plasminogen activator (tPA) per the 2013 American Heart Association guidelines are SBP less than 185 mm Hg and diastolic BP (DBP) less than 110 mm Hg. Post-tPA BP should be SBP less than 180 and DBP less than 105. Guidelines for antihypertensive medication use in this acute phase are provided in **Table 2**. [7–12]

Frequent measurement of BP every 10 to 15 minutes for the first 60 minutes following the administration of tPA can establish a trajectory and guide management of BP before transferring to another unit.[7]

Current guideline recommendations for post-tPA monitoring are close monitoring in an ICU or stroke unit for at least 24 hours after the infusion. Parameters to be monitored include BP and neurologic examination every 15 minutes for the first 2 hours, then every 30 minutes for the next 6 hours, and then every hour for the next 16 hours.[13] The importance of BP control should prompt the initiation of a continuous antihypertensive infusion once 2 to 3 intravenous push doses have been administered.[7]

Mechanical thrombectomy eligible strokes

Current AHA/ASA guidelines recommend maintaining BP < 180/105 mm Hg during and for 24 hours after mechanical thrombectomy (MT) (class IIa, *level of evidence* B). If successful reperfusion is achieved, guidelines recommend that BP be maintained at less than 180/105 mm Hg (class IIb, *level of evidence* B).[14,15]

The protocol from the endovascular treatment of small core and anterior circulation proximal occlusion with emphasis on minimizing CT to recanalization times (ESCAPE) trial stated that if reperfusion failed, an SBP $\geq$ 150 mm Hg may be useful in maintaining adequate collateral flow, and if successful reperfusion was achieved, normal BP was then targeted.[16]

Permissive hypertension in large vessel occlusions not eligible for tissue plasminogen activator or mechanical thrombectomy

Patients may be ineligible for MT (outside of a clinical trial) despite large vessel occlusion for the following reasons: presentation beyond the 24-h time window, Alberta Stroke Program Early Computed Tomography (ASPECT) score </ = 6, and Computed Tomography Perfusion (CTP) ratio of less than 1.7. For these patients, permissive hypertension up to 220/120 for the initial 24 to 48 hours is targeted as tolerated.[7]

However, controversy exists regarding the possible contribution of such hypertension to cerebral edema and risk of hemorrhagic conversion. Several studies have

looked at lowering BP goals poststroke. The recommended goals for lowering BP vary but are consistent in limiting reduction by < 10–15% from baseline.[7]

Per AHA guidelines it is reasonable to lower BP by 15% during the first 24 hours after stroke onset. Lower BP targets are often initiated if there is evidence of end-organ damage or exacerbating of comorbid conditions.[7]

Blood pressure augmentation/induced hypertension in acute ischemic strokes

Although BP augmentation is sometimes used in AIS, no large, randomized control studies have assessed the safety and efficacy of this treatment.[7] Koenig *and colleagues* showed the relative safety of induced hypertension therapy when moderate elevation was targeted, but data on clinical outcomes were insufficient.[17]

Hypotension was associated with worse neurologic outcomes in 3 separate studies.[7] The Safety and Efficacy of Therapeutic INduced HYPERTENSION in noncardioembolic AIS (SETIN-HYPERTENSION) trial is a multicenter, randomized, open-label, prospective, phase-III trial that aims to determine safety and efficacy of induced hypertension (IH) using phenylephrine in patients with noncardioembolic AIS. There is potential for this study to further inform the practice of IH for AIS, and subjects are currently being enrolled.[7]

Hemorrhagic transformation of ischemic stroke

The frequency of hemorrhagic transformation (HT) is associated with epidemiologic factors (eg, age, prestroke treatment, and conditions), characteristics of the infarct (core volume and timing of follow-up), reperfusion techniques (intravenous thrombolysis with or without mechanical thrombectomy), radiological diagnosis (CT or MRI techniques), and subsequent use of antithrombotics.[18]

The recent Enhanced Control of Hypertension and Thrombolysis Stroke Study (ENCHANTED) trial has shown that intensive BP control potentially reduces the risk of major intracranial hemorrhage in patients with AIS receiving intravenous thrombolytic therapy.[18]

Health care providers should determine BP targets by weighing the risk of worsening ischemia versus the risk of expansion based on the severity of hemorrhage. Patients with incomplete recanalization may need higher BP targets to maintain sufficient blood flow to the ischemic bed to reduce infarct growth. Conversely, these same patients may need tighter BP control to avoid impending HT.[18]

MANAGEMENT OF SPECIFIC COMPLICATIONS

Management of Cerebral Edema

The development of cerebral edema after an AIS is secondary to cytotoxic cell injury resulting in intracellular water influx. The timeline of cytotoxic cerebral edema development begins a few hours postictus and peaks 2 to 5 days. This “watch period” necessitates close monitoring for a decline in neurologic examination due to secondary brain and brainstem compression.[5,6] Malignant cerebral edema is identified with large hemispheric strokes. The mortality rate of malignant cerebral edema is high (40%–80%) if not thoughtfully managed.[5,6] Recent studies have shown that imaging can be helpful in predicting outcomes. A midline shift of more than 3 mm can be a predictor of poor outcome.[19]

The following are risk factors for malignant edema[3]:

- Younger age
- Presenting NIHSS greater than 20 and greater than 15 for dominant and nondominant hemisphere strokes, respectively
- Early development of encephalopathy

- Systolic BP (SBP) > 180 within 12 hours of stroke onset
- CT scan showing hypodensity in 50% or more of the MCA territory
- Involvement of multiple major vascular territories
- MRI with Diffusion Weighted Imaging (DWI) volume of 82 cm or more if conducted within 6 hours of initial stroke onset

Current therapies include both medical and surgical options.[3]

Hyperosmolar Therapy

Hyperosmolar therapy, that is, mannitol or hypertonic saline (HTS), can lower elevated ICP and reverse herniation by extracting water from intracellular and interstitial spaces. This secondarily increases cerebral perfusion to peri-infarct areas at risk for secondary ischemia from compression.[1,20]

HTS and mannitol carry conditional recommendations with low-quality evidence for the initial management of cerebral edema with or without elevated ICP in AIS. Overall, there is insufficient evidence to recommend either, though both are used when neurologic worsening occurs while awaiting surgical rescue, when surgery is not offered, and in cases of impending herniation.[1,20]

Surprisingly, few clinical studies have assessed mannitol use in AIS, and no randomized clinical trial has evaluated the effect of mannitol on clinical outcomes after LHI with edema. Thus, mannitol is used based on experimental studies, or observations in small nonrandomized case series in humans.[20,21]

Historically, HTS was administered as continuous infusion titrated to serum sodium concentration; however, recent data suggest better outcomes with intermittent boluses. Desired target sodium is determined on individual bases typically ranging between 140 and 155 mEq/L. There is no therapeutic value of serum sodium levels greater than 160 mEq/L and mental status can be further negatively affected beyond this threshold.[20,22]

Temperature Management

Fever with AIS is known to worsen outcomes. Therefore, normothermia is an important goal in ICU stroke care. When cerebral edema is refractory to all measures including advanced cooling devices, hypothermia is sometimes tried. We are still lacking evidence from randomized trials regarding the effect of hypothermia on malignant MCA strokes. Five observational case series showed decreased mortality with hypothermia compared with other methods of ICP management in preliminary analysis. This data remains experimental, and no evidence-based recommendations can be provided.[1,23]

SURGICAL METHODS

Decompressive Craniectomy

Anterior fossa strokes

DHC is an option in LHI for most patients under the age of 60 and some patients under the age of 75.[3] The key to maximizing surgical outcomes is timing the decompression to avoid secondary brain injury. Secondary injury can occur from significant and prolonged mass effect, and before clinical signs of brainstem compression. Trials have shown that patients up to 60 years of age with a large stroke burden greater than 2/3 of brain volume that also presents with a decreased level of consciousness experience better outcomes if decompressed in the first 24 to 48 hours after presentation. Careful identification of those patients is essential.[1] Most recent studies have shown that DHC improves survival and functional outcome, but most survivors live with at

least a moderately severe disability. The proportion of survivors with moderately severe to severe disability increases in the elderly.[23] **Fig. 1** demonstrates a case of successful DHC.

Optimization of neurocritical care may reduce the need for surgery. It should also be noted that advancement in other therapies for AIS such as thrombolytics and thrombectomy may further mitigate the need. Another aspect of medical care that requires further evaluation is the optimal provider setting in which these patients should receive care. While most of the RCTs admitted enrolled patients to ICUs, there was a higher proportion of surgical patients in ICU, compared with medical patients. The differences in the level of care alone could have confounded outcomes.[19]

DESTINY II was a randomized controlled trial in patients 61 years and older with LHI.[1,18] The results showed a decrease in mortality rate from 70% to 33% in the DHC group, but 32% and 28% of patients who survived remained in a very poor neurologic status quantified as Modified Rankin Scale (mRS) of 4 and 5, respectively. Only 7% of patients showed a mRS of 3 as the best outcome that could be achieved. Although the authors concluded that DHC significantly increased the probability of survival without the most severe disability, data clearly show that an increased survival rate was achieved by a significant increase of patients with poor and very poor outcomes (mRS5).[23]

In summary, DHC as a potential therapy to improve survival after large hemispheric strokes is recommended with the following special considerations[3]:

- In patients older than 60 years, it is recommended to consider patient and family wishes, since in this age group, DHC can reduce mortality rate but with a higher likelihood of being severely disabled

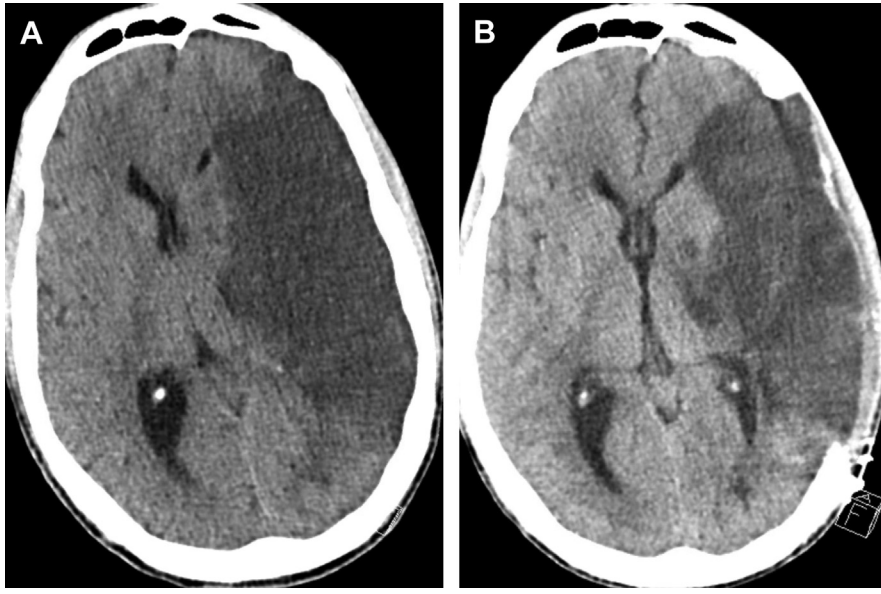

Fig. 1. (*A*) Noncontrast head CT scan showing new left MCA ischemic stroke 48 hours post-onset of symptoms, with cerebral edema and midline shift. (*B*) Noncontrast head CT of same patient 24 hours post-DHC with near resolution of shift. (*Courtesy of* The University of New Mexico, Albuquerque, NM.)

- There are insufficient data to recommend against DHC based on hemispheric dominance
- To achieve the best neurologic outcome, it is recommended to perform DHC within 24 to 48 h hours of symptom onset and before any herniation symptoms
- The minimal size for DHC should be 12 cm, with larger sizes of 14 to 16 cm being associated with better outcomes

Postoperative care for patients with DHC involves monitoring the flap site for sudden expansion which can occur with a post-DHC subdural hematoma or hemorrhagic conversion. The head should be positioned to take the weight off the DHC side. The patient should not be out of bed without an othortically fitted protective helmet. The wound should be examined daily for leakage or erythema suggesting infection.

Cerebellar Strokes

Up to 20% of patients with cerebellar infarcts are at risk of developing malignant edema. Initial infarct size on imaging helps identify those at risk. The kinetics of edema in the posterior fossa is not well characterized. The infarct–edema growth rate over the first 48 h independently predicted the need for surgical intervention in patients with cerebellar infarction.[24,25] The AHA recommends that Neuro ICU monitoring is indicated when $^{1}/_{3}^{rd}$ of one cerebellar hemisphere is affected by a stroke. The most concerning issues related to cerebellar infarcts are brainstem and fourth ventricle compression. When either is present, it is recommended that medical therapy serves as a bridge to early suboccipital craniectomy (SOC). Care must be taken when obstructive hydrocephalus is present, that ventriculostomy placement is timed with surgical decompression to avoid inducing upward herniation.[24,26]

Fig. 2A demonstrates brainstem and fourth ventricular compression in the setting of bilateral cerebellar strokes and Fig. 2B shows resolution after SOC.

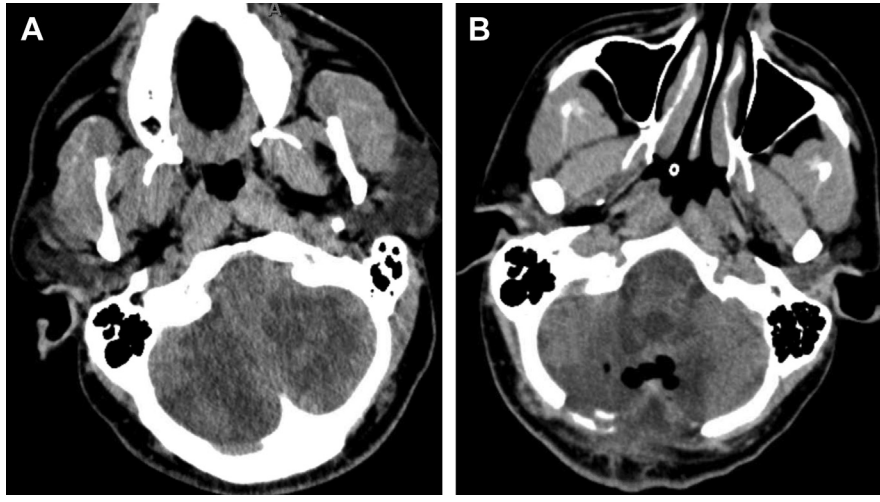

Fig. 2. (*A*) Noncontrast head CT in a patient with bilateral cerebellar infarcts with edema, fourth ventricular compression, and brainstem compression. (*B*) Noncontrast head CT in the same patient post-suboccipital decompression with a resolution of brainstem compression and ventricular compression. (*Courtesy of* The University of New Mexico, Albuquerque, NM.)

Management of Hemorrhagic Transformation

HT can be classified clinically and radiographically.

Clinical classification distinguishes symptomatic intracranial hemorrhage (sICH) from asymptomatic intracranial hemorrhage (aICH). sICH is defined as a worsening of the NIHSS by $\geq$ 4 points attributable to HT, within 36 hours of ictus. Clinical classification may be unreliable in cases of severe stroke whereby significant decline may be difficult to discern.[13]

Radiographic classification of HT distinguishes small petechial hemorrhagic infarction (HI1), confluent petechial hemorrhagic infarction (HI2), small parenchymal hemorrhage (PH1) (<30% of infarct, mild mass effect), and large parenchymal hemorrhage (PH2, >30% of infarct, marked mass effect). The Heidelberg Bleeding Classification scale has been proposed to address some of the challenges and limitations of the ECASS classification, outlined in **Table 3**.[13,27] **Fig. 3** illustrates PH1 hemorrhagic transformation of a left MCA stroke post tPA treatment.

The use and reversal of antiplatelets and anticoagulants in AIS are discussed in other chapters of this text. This article is additive by specifically addressing the reversal of fibrinolytic agents used in AIS.

Post tPA/TNK reversal: Prompt diagnosis and early correction of coagulopathy after fibrinolytic therapy has remained the mainstay of treatment.[13,28] The options for reversal of an HT after fibrinolytics are included in **Table 4** later in discussion:

Table 3
A and B: Radiologic classification of hemorrhagic conversion

ECASS Classification for Hemorrhagic Conversion	
Petechial hemorrhage	
HI1	Small petechial hemorrhages that are scattered
HI2	Petechial hemorrhages that are confluent
Parenchymal Hemorrhage	
PH1	Parenchymal hemorrhage <30% of infarct, only mild mass effect
PH2	Parenchymal hemorrhage more than 30% of infarct, marked mass effect

3B Heidelberg Bleeding Classification	
1a HI1	Scattered small petechia, no mass effect
1b HI2	Confluent petechia, no mass effect
1c PH1	Hematoma with infarcted tissue occupying <30% without mass effect
2 PH2	Hematoma occupying 30% or more of infarcted tissue with mass effect
3	ICH, outside infarcted tissue or intracranial-extracerebral hemorrhage
3a	Parenchymal hematoma away from infarction tissue
3b	Parenchymal hematoma away from infarction tissue with: IVH
3c	Parenchymal hematoma away from infarction tissue with: Subarachnoid Hemorrhage
3d	Parenchymal hematoma away from infarction tissue with: Subdural Hemorrhage

Data from Spronk E, Sykes G, Falcione S, Munsterman D, Twinkle J, Kamtchum-Tatuene J, Jickling GC. Hemorrhagic Transformation in Ischemic Stroke and the Role of Inflammation. Frontiers in Neurology vol. 12, 2021; and Yaghi S, Willey JZ, Cucchiara B, Goldstein JN, Gonzales NR, Khatri P, Kim LJ, Mayer SA, Sheth KN, Schwamm LH; Treatment and Outcome of Hemorrhagic Transformation After Intravenous Alteplase in Acute Ischemic Stroke: A Scientific Statement for Healthcare Professionals From the AHA/ASA. Stroke. 2017 Dec;48(12):e343-e361.

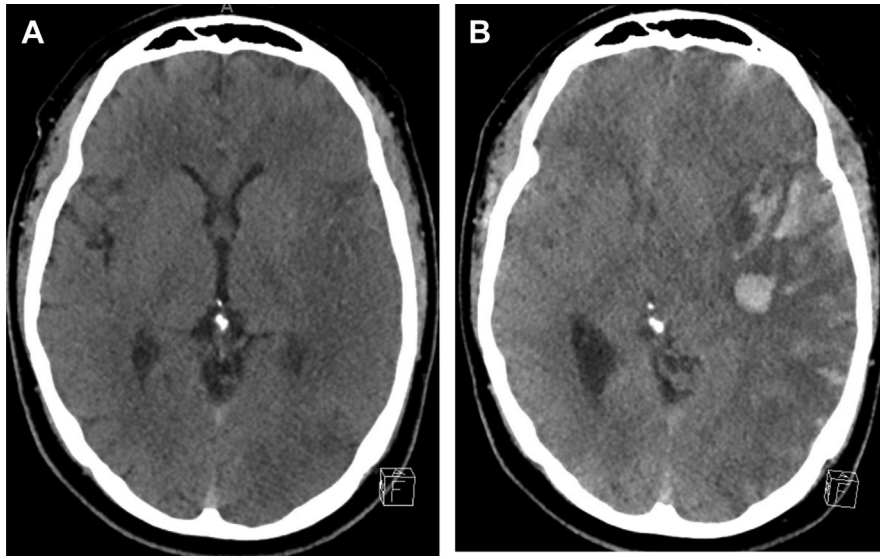

Fig. 3. (*A*): Noncontrast head CT showing early infarct signs in the left MCA territory (*B*) Noncontrast head CT showing hemorrhagic conversion within 24 hours of first head CT. Note that the ECASS score here would be PH1 using definitions in Table 3A and the Heidelberg Bleeding Classification would be 1cPH1 using definitions in Table 3B. (*Courtesy of* The University of New Mexico, Albuquerque, NM.)

Table 4
Options for reversal of an HT after fibrinolytics

Agent	Amount	Comments
Cryoprecipitate	Initial dose: 10 U cryoprecipitate. Additional given as needed	Derived from FFP and contains fibrinogen. A fibrinogen level should be sent immediately. Goal fibrinogen level >150 mg/dL. Each 10 U cryoprecipitate increases fibrinogen by nearly 50 mg/dL
Platelets	6–8 units	Unclear benefit except in thrombocytopenia <100 that was not known on admission
Fresh Frozen Plasma	12 mL/kg	INR = 1.6
Prothrombin Complex Concentrate	25–50 U/Kg	Dose depends on the INR level
Vitamin K:	10 mg IV	
Recombinant Factor VIIa	20–160 μg/kg	
Antifibrinolytic agents:		
Aminocaproic acid	4 g IV during the first hour followed by 1 g/h for 8 h	
Tranexamic acid:	1000 mg IV x once[13,14]	

Strokes Secondary to Infective Endocarditis

Cerebrovascular complications (ie, AIS, transient ischemic attack, silent cerebral embolism) frequently occur in patients with infective endocarditis (IE) and result from cerebral septic embolization of an endocardial vegetation.[29]

Treatment recommendations include[30–32]:

- Starting antibiotics as soon as possible (reduces the risk of neurologic complications by more than 50%).
- Avoid anticoagulation, if possible, due to the risk of hemorrhagic transformation
- Obtain CT angiogram to look for mycotic aneurysms warranting endovascular coiling
- If cardiac valvular surgery is indicated, timing will depend on the presence and severity of AIS.

Optimal surgical timing is unknown and is recommended to be individualized based on embolic risk factors including[30–33]:

- Age
- Diabetes
- Presence of atrial fibrillation
- Size, mobility, and location of vegetations
- Changing the size of the embolism *on* antibiotics
- Prior embolism
- Organisms such as Staphylococcus aureus

In general, it is recommended to wait 2 weeks before surgical intervention.[32]

Management of Patients Postcarotid Revascularization

Revascularization via open CEA or endovascular carotid artery stenting (CAS) is indicated for symptomatic internal carotid stenosis of greater than 70%.[34,35] Ideally, depending on the size of stroke (if one has occurred) revascularization is completed within 2 weeks following the heralding event. For the initial 2 to 48 hours postprocedure, these patients benefit from monitoring in the Neuro ICU.[14,35]

Postoperative Care for Carotid Endarterectomy Procedures

Significant postoperative complications to anticipate include wound hematoma, uncontrolled hypertension, hemodynamic depression with hypotension and bradycardia, hyperperfusion syndrome with postoperative ICH, and seizures. Due to the seriousness of these complications, perioperative care in a Neuro ICU is recommended. For those who remain stable for 24 hours after surgery, direct discharge from the ICU is often possible. However, if instability is demonstrated, continued close observation is recommended.[14]

- Wound hematoma

Wound hematomas are relatively common following CEA. In the NASCET study, 5.5% of patients had documented wound hematomas. The majority are small but large hematomas may require emergency treatment. If the trachea is not compromised, emergency evacuation of hematoma in the operating room is warranted. Once the hematoma begins obstructing the trachea, emergent bedside revision is best.[14]

- Hypertension

BP control post-CEA is vital. Neck hematoma, hyperperfusion syndrome, and intracerebral hemorrhage are associated with poor control. Preoperative hypertension is the single most important predictive factor for postoperative hypertension. Baroreflex failure syndrome with either unilateral or bilateral CEA can occur. Yet, even of those who were normotensive preprocedure, 21% had postsurgically induced abnormalities of baroreceptor sensitivity during the dissection of the common carotid artery and vagus nerve. Careful BP management in the initial post-CEA period is essential in avoiding further complications.[14]

- Hemodynamic depression: hypotension and bradycardia

Hemodynamic depression with hypotension and bradycardia occurs predominately after CAS with a frequency between 13% and 76%. Critical care management of hypotension includes fluid infusion and low dose phenylephrine or norepinephrine, dependent on heart rate. Bradycardia is frequently benign but can become symptomatic or severe (heart rate < 40) requiring treatment with atropine or glycopyrrolate. In very severe cases transient external pacing may need to be considered. Typically, the course is self-limited to 24 to 48 hours[14,36]

- Hyperperfusion syndrome

Post-CEA, hyperperfusion syndrome is a critical entity resulting from impaired cerebral autoregulation in the ipsilateral carotid territory. Chronic hypoperfusion distal to the stenotic segment causes tonic compensatory maximal collateral vasodilation. Postoperatively these beds remain unable to vasoconstrict. The ensuing luxury perfusion leads to edema followed by hemorrhage. A progression is similar to hemorrhagic PRES (Posterior Reversible Encephalopathy Syndrome) but isolated to the affected carotid territory. Tight BP control is the mainstay of treatment along with hourly observation of the neurologic exam.[14]

- Intracerebral hemorrhage

Risk factors for post-CEA and CAS hemorrhage include advanced age, presence of pre-existing hypertension, presence of poor collaterals, and evidence of slow MCA territory flow on angiography. Angiographic hypoperfusion is the strongest risk factor. The ICH that develops postrevascularization is typically large and often fatal.[14]

- Seizures

Post-CEA and CAS seizures are managed with BP control in addition to antiseizure medications and may be attributed to one or more of the following 3 mechanisms.[14]

- Postoperative cortical ischemic strokes
- Postoperative intracranial hemorrhage
- Cerebral hyperperfusion syndrome

Common Medical/Intensive Care Unit-Related Complications After Stroke

Strokes and cardiac issues

Cardiac ischemia and arrhythmias are known risk factors for complications after AIS. Involvement of the insular cortex, particularly on the right, is associated with cardiac events due to its role in autonomic control. The most common of these is subendocardial myonecrosis, or non-ST elevation myocardial infarction clinically.[21]

Screening for cardiac ischemia in AIS includes obtaining a 12-lead ECG and at least 2 sets of troponin levels 8 hours apart. If the troponin is elevated, it needs to be followed until the level peaks. All patients with AIS in the ICU should be on continuous

cardiac monitoring. Common abnormal ECG changes after AIS include prolonged QT, ST segment changes, prominent U waves, atrial fibrillation, and supraventricular tachycardia.

Other Aspects of Care for Patients with Stroke in Critical Care Unit

Deep venous thrombosis prevention

The incidence of deep venous thrombosis (DVTs) in AIS is high and typically manifests clinically between 2 and 7 days. Pulmonary Embolism is the most common cause of death (25%) in AIS.

Venous thromboembolism (VTE) prophylaxis recommendations are as follows[33]:

- Initiate VTE prophylaxis as soon as feasible in all patients with AIS.
- Prophylaxis with low molecular weight heparin (LMWH) is recommended over unfractionated heparin (UFH) in AIS with restricted mobility.
- Those undergoing hemicraniectomy or endovascular procedure are suggested to use UFH, LMWH, and/or intermittent pneumatic compression devices for VTE prophylaxis in the immediate postsurgical or endovascular epoch. Administration of thrombolytics is the exception when prophylaxis can be delayed for 24 hours.[33,37,38]

Strokes and aspiration pneumonia

Coughing and swallowing reflexes are crucial protective mechanisms to prevent aspiration pneumonia and are frequently impaired in patients with poststroke. Pneumonia impacts stroke outcomes even without respiratory failure. Although pneumonia is a common complication, antibiotic prophylaxis is not recommended. Conversely, beta-blockers and statins are linked to a lower risk of pneumonia. Other drugs, such as benzodiazepines and antiacid medications, are associated with an increased risk of pneumonia and should be used judiciously.[37]

Strokes and urinary tract infection

Patients with AIS have a high risk of infection in general. Several factors have been identified as predictors for the development of UTI, including age, elevated procalcitonin, interleukin-6, CRP levels, higher presenting NIHSS, comorbid diabetes, and presenting hemoglobin level.[39] Early removal of indwelling Foley catheters is the mainstay of prevention, as well as protocolized postvoid bladder scanning for early identification of acute retention.

Glycemic control

Among patients with AIS and hyperglycemia, treatment with intensive (blood glucose <120) versus standard glucose control (blood glucose <180) for up to 72 hours did not result in a significant difference in favorable functional outcomes at 90 days. These findings do not support using intensive glucose control in this setting.[40]

SUMMARY PARAGRAPH

AIS management in the Neuro ICU addresses several cornerstones of patient care. Management of airway compromise and BP are critical first steps. Monitoring for iatrogenic complications preoccupies the early phase of care. Management of stroke-related sequelae such as cerebral edema and secondary ischemia are critically important. Early surgical intervention for malignant poststroke edema, in appropriate cases, is also important for survival and functional outcome for some. Patients with poststroke are at risk for cardiopulmonary, thromboembolic, and infectious complications. Detailed ICU checklists to address these issues assist in the prevention of these common complications that can significantly impact stroke recovery.

CLINICS CARE POINTS

- Loss of airway control requiring intubation poststroke depends mainly on the size and/or location of stroke, GCS assessment (<10 at risk), loss of cough or gag reflex, and degree of cerebral edema with midline shift in anterior fossa or brainstem compression in posterior fossa strokes.
- Targeted BP control in patients with ischemic stroke reduces the risk of hemorrhagic conversion postfibrinolytics and interventional thrombectomy reduces the risk of hemorrhagic conversion
- Management of poststroke cerebral edema includes early surgical management, appropriate use of hypertonic saline or mannitol, temperature control, blood glucose, and BP control to improve mortality and outcome.
- Patients with post-CEA have the greatest risk of complications in the first 24 hours after the procedure. BP control will reduce the risk of neck hematoma and exacerbation of reperfusion injury complications.
- The most common ICU-related complications include aspiration pneumonia, deep venous thromboses, cardiac arrhythmias, and urinary tract infections. Implementation of mechanisms to prevent these complications is an important part of stroke ICU care.

DISCLOSURE

The authors have nothing to disclose.

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