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REVIEW ARTICLE

Emergency Neurological Life Support: Acute Ischemic Stroke

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Abstract Acute ischemic stroke is a neurological emergency that can be treated with time-sensitive interventions, including intravenous thrombolysis and endovascular approaches. Extensive study has demonstrated that rapid assessment and treatment are essential for improving neurological outcome. For this reason, acute ischemic stroke was chosen as an Emergency Neurological Life Support protocol. The protocol focuses on the first hour following the onset of neurological deficit.

Keywords Ischemic stroke · tPA · Embolectomy · Thrombectomy · $ABCD^2$ score

Introduction

According to 2008 World Health Organization statistics, cerebrovascular disease is the second cause of death worldwide, with an estimated 6.3 million deaths per year—coming close second to ischemic heart diseases (7.3 million deaths per year [1]). In the United States, approximately 800,000 strokes occur annually, of which nearly 25 % are recurrent strokes [2]. Although there are many new advances in the treatment of stroke, it is crucial that proper diagnosis and management occur as soon as possible, since the later therapies are instituted, the less likely the chance of successful intervention.

The Emergency Neurological Life Support (ENLS) suggested algorithm for the initial management of acute ischemic stroke is shown in Fig. 1. Suggested items to complete within the first hour of evaluating a patient with acute ischemic stroke are shown in Table 1.

Clinical Suspicion of Stroke

Acute stroke is suspected when a patient exhibits the sudden onset of a neurological deficit. In the absence of an obvious seizure, the deficit can most likely be attributed to stroke or transient ischemic attack (TIA). In countries that treat stroke as an emergency, paramedical personnel are typically the first to evaluate the patient (Fig. 2). The standard treatments are to perform routine airway, breathing, and circulation (ABC) assessments, administer supplemental oxygen, check blood glucose, perform a stroke screening examination [e.g., Cincinnati Prehospital Stroke Scale (CPSS), Los Angeles Prehospital Stroke Screen (LAPSS), or Miami Emergency Neurologic Deficit Exam (MEND)] in addition to history and physical exam, determine the time of onset of stroke symptoms, and transport the patient.

Prehospital systems should call ahead to the receiving hospital, and many transport the patient preferentially to stroke centers. Once the patient arrives to an emergency department (ED), he or she is rapidly clinically assessed and then imaged, typically with non-contrast computed tomography (CT) but many centers perform computed

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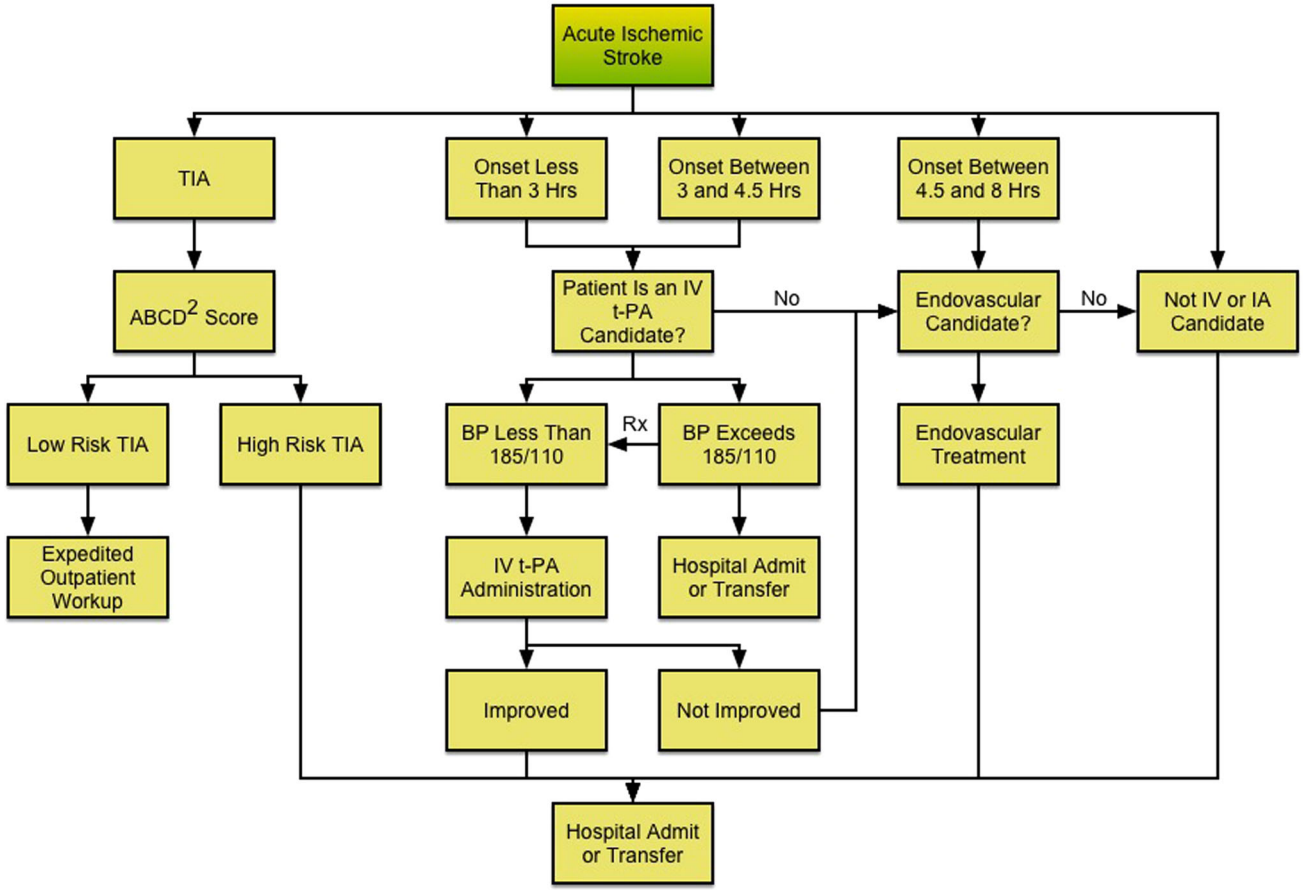

Fig. 1 ENLS acute ischemic stroke protocol

Table 1 Acute ischemic stroke checklist for the first hour

Checklist
☐ Vital signs
☐ Supplemental oxygen to maintain saturation >94 %
☐ Activate stroke code system (if available)
☐ IV access
☐ Labs: capillary glucose, CBC with platelets, PT/INR, PTT, EKG, and beta-HCG for women of child-bearing age
☐ Determine time of onset/last seen normal
☐ CT or other brain imaging study
☐ Determine NIHSS score
☐ Medication list[a]

[a] When asking about medications, be sure to ask specifically about anticoagulants: e.g., warfarin, heparin, low molecular weight heparin (enoxaparin, dalteparin, and tinzaparin), target-specific anticoagulants (e.g., dabigatran, apixaban, edoxaban, and rivaroxaban), and when medication was last taken/administered

tomography angiography (CTA) at the same time to expedite the identification of large vessel intracranial occlusion and to determine the status of the extracranial internal carotid artery. Similarly, many regional arrangements have been made for "telestroke" consultation in order to expedite the administration of thrombolytic agents, followed by transfer to a stroke center if necessary or possible.

As shown in Fig. 2, imaging is essential to confirm the correct diagnosis and exclude cerebral hemorrhage. Treatment should then proceed according to one of three ENLS protocols (shaded): Subarachnoid Hemorrhage, Intracerebral Hemorrhage, or Acute Ischemic Stroke. If the CT is free of hemorrhage or mass effect, then a presumptive ischemic stroke or TIA is present.

The diagnosis of acute ischemic stroke is based on the presence of new and typically sudden focal neurological findings (e.g., facial weakness, arm/leg weakness, aphasia, neglect, visual field disturbance, and ataxia) with an imaging study—typically CT or magnetic resonance imaging (MRI) of the brain—that excludes hemorrhage.

When confronted with a patient whose focal symptoms have begun within the preceding few hours, it should be assumed that the patient will eventually be diagnosed with stroke. Most TIAs are brief, typically lasting less than 20 min before completely resolving. Therefore, if the patient is still manifesting physical signs of a stroke in the ED, those signs likely indicate a stroke.

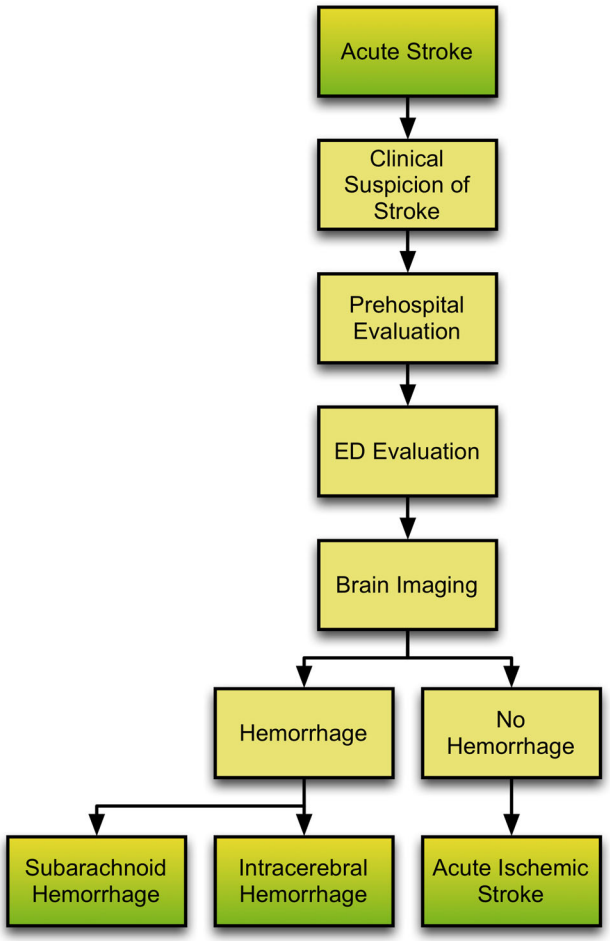

Fig. 2 Clinical suspicion of stroke algorithm: this algorithm assumes that the patient is outside of the hospital when stroke occurs. Based on the results of brain imaging, the patient can be triaged to one of the three ENLS protocols shaded (*bottom*)

In some centers, patients may be screened for clinical stability immediately upon arrival ("at the door"), then taken directly to CT or MRI based on symptoms of facial weakness, dysarthria, gaze preference, limb weakness, or other focal findings. However, there are a number of stroke mimics, including seizure, hypoglycemia, sepsis or fever, migraines, and Bell's palsy. It is not uncommon for patients with stroke mimics to be treated with tissue plasminogen activator (tPA) and this appears to be safe.

Each of the following elements should be addressed in rapid succession.

Time of Onset

One of the chief criteria used to select patients for vascular reperfusion therapy is the duration of stroke symptoms, since treatment delays lead to a lower likelihood of a good outcome and higher risk of intracranial hemorrhage. The time the patient last known to be without neurological deficits must be established from the patient or a bystander. If the patient went to bed and awoke with the stroke, the time of onset is considered to be when the patient went to bed.

Vital Signs

Pulse oximetry should guide supplemental oxygen to achieve an oxygen saturation $\geq$94 %. Hyperoxia may be detrimental in stroke, so there is no need for high-flow oxygen [3]. If the patient is not hypoxic, supplemental oxygen is not needed.

Blood pressure (BP) must be obtained. Low BP typically points away from an acute stroke and may indicate exacerbation of symptoms of a previous stroke due to poor perfusion of previously injured tissue. BP in excess of 220/120 mmHg should be lowered, regardless of the ultimate diagnosis. There are consensus suggestions of allowing permissive hypertension up to 220/120 mmHg for acute stroke patients who are not candidates for tPA, including those who have failed attempts to lower BP to allow tPA eligibility [3].

If the patient is a potential tPA candidate, interventions to control BP should be initiated immediately. The target BP for IV tPA candidates is less than 185/110 mmHg, and once tPA is initiated, BP must be maintained below 180/105 mmHg to limit the risk of intracranial hemorrhage [3]. A strategy of careful BP lowering should be employed while being mindful of not dropping the BP too much once the patient is at goal. Chronic antihypertensive medications should be reduced or temporarily withheld. Short-acting intravenous (IV) agents such as labetalol, nicardipine, or clevidipine are preferred (see Table 2) to achieve a BP $<$ 185/110 mmHg.

If the patient's BP proves refractory to the above medications, the patient is considered to be high risk for ICH and should not be treated with tPA. However, efforts to reduce BP below 220/120 mmHg should be continued. Permissive hypertension (allowing BP to naturally rise) is allowed for TIA, as it is for non-tPA treated patients, up to 220/120 mmHg.

Nitroglycerin paste (for patients with no IV access) is listed as an option in the current American Stroke Association guidelines [3]; however, this agent is not titratable and may lead to unpredictable BP responses after administration of tPA. Clevidipine is still under investigation for use in acute stroke and is not yet recommended as an alternative agent for BP control in the acute stroke setting. Therefore, titratable agents like labetalol and nicardipine are recommended as the acute antihypertensives of choice.

Table 2 IV antihypertensive agents used to lower BP to attain tPA eligibility

Labetalol
10–20 mg IV bolus over 1–2 min; may repeat every 10 min
Consider doubling dose (i.e., 20, 40, and 80 mg) to a maximum total dose of 300 mg, followed by a maintenance infusion (0.5–8 mg/min)
The importance of the maintenance infusion should not be underestimated or dismissed. If a bolus was required to lower the blood pressure, then the BP should be assumed to climb again as soon as the bolus wears off, potentially placing the patient in danger of ICH due to the uncontrolled BP. Start an infusion if labetalol boluses successfully lower the BP. If the patient is no longer deemed a candidate for tPA and permissive hypertension is being planned, then the infusion may be discontinued, provided the BP does not rise above 220/120 mmHg
Nicardipine
Begin with 5 mg/h IV infusion
Titrate by 2.5 mg/h at 5–15 min intervals to a maximum total dose of 15 mg/h to achieve goal BP

Laboratory Examination

An adequate laboratory examination includes capillary blood glucose (CBG), complete blood count (CBC) with platelets, chemistries, prothrombin time/partial thromboplastin time (PT/PTT), international normalized ratio (INR), and beta-human chorionic gonadotrophin (HCG) for women. In selected patients who are taking direct thrombin inhibitors or direct factor Xa inhibitors, it may be prudent to check a thrombin time (TT) and/or ecarin clotting time (ECT) or chromogenic anti-Xa activity assays, respectively. However, if a patient is deemed a candidate for tPA and there is no reason to suspect abnormal laboratory test results, tPA should be administered without waiting for these laboratory values to prevent further delay [3], at a minimum, a CBG (finger-stick) should be performed prior to tPA administration, since it can be completed quickly to rule out hypoglycemia as a stroke mimic.

Imaging

There should be a goal of completing a head CT scan or MRI within 25 min of the patient's arrival. Many centers couple CTA of the head and neck with this initial CT. Chest X-ray is no longer routinely recommended during the acute phase of the workup [3]. With EMS pre-arrival notification of a potential stroke patient, a doorway assessment by the clinician and simultaneous patient registration can facilitate rapid patient transport to imaging.

Activate Stroke Team

If available, the stroke code system should be activated. The team should evaluate the patient within 10–15 min of the call. Again, prehospital notification by EMS and paging the stroke team can expedite patient assessment on arrival and in CT, thereby decreasing time to determine tPA candidacy.

Table 3 Risk of intracerebral hemorrhage with IV tPA treatment [4]

NIHSS score	Risk of intracerebral hemorrhage (%)
0–10	2–3
11–20	4–5
>20	17

NIHSS

The National Institutes of Health Stroke Scale (NIHSS) is a standardized method for examiners to reproducibly and quantifiably assess a patient's stroke symptoms. This is the preferred scoring system and may be used by a variety of non-neurological medical providers [3]. Scores range from 0 (no deficit) to 42.

The scale has some limitations especially in scoring brainstem strokes. While many physicians use an NIHSS of 4 as a threshold to treat an acute ischemic stroke with tPA, a low score is not an absolute contraindication. Clinicians should carefully consider and document the individual patient's relative risks, potential benefit and premorbid status when evaluating a patient for treatment with tPA.

Health care providers wishing to learn how to perform the NIHSS and receive free certification can find these resources online through the American Stroke Association and the National Stroke Association.

The NIHSS score may also be used as a guideline to predict risk of intracerebral hemorrhage in patients who are given tPA, as shown in Table 3. However, despite this escalating risk of hemorrhage, patients in all NIHSS strata benefit from tPA.

Patients presenting with acute stroke are typically hypo- or euvolemic. Hypovolemia can worsen ischemic injury as a result of impaired perfusion to brain tissue. Euvolemia is desirable in the acute stroke setting, and most stroke patients should receive maintenance IV fluids in the form of normal saline.

Table 4 Eligibility and absolute and relative contraindications for use of IV tPA upto 3 h [3]

Eligibility
Diagnosis of ischemic stroke causing measurable neurological deficit. Neurological signs should not be minor and isolated. Neurological signs should not be clearing spontaneously
Onset less than 3 h before initiating tPA
Patient is at least 18-year old (see Sect. 2.14)
Absolute exclusion criteria if positive
No major head trauma or prior stroke in the previous 3 months
Symptoms of stroke should not be suggestive of subarachnoid hemorrhage
No arterial puncture at a non-compressible site or lumbar puncture in the previous 7 days
No history of previous intracranial hemorrhage
No history of intracranial neoplasm, aneurysm, or arteriovenous malformation
No intracranial or intraspinal surgery in the previous 3 months
Blood pressure not elevated (systolic <185 mmHg and diastolic <110 mmHg)
No evidence of active bleeding or acute trauma (fracture) on examination
Platelet count $<100{,}000$ mm^3
If receiving heparin in previous 48 h, aPTT must be in normal range
Not taking an oral anticoagulant or, if anticoagulant being taken, INR $<$ 1.7 or PT $>$ 15 s
No dabigatran, apixaban, or rivaroxaban use for chronic anticoagulation for conditions such as atrial fibrillation. There is still little information on assessing influence or levels of these medications in patients with acute stroke. There are suggestions to check an activated thromboplastin time (aPTT), INR, platelet count, thromboplastin time (TT), ecarin clotting time (ECT), and antifactor 10a level (if available). Without normalized special tests (as listed), use of tPA is NOT recommended in patients with recent use (within 48 h) of these products
Blood glucose concentration <50 mg/dL (2.7 mmol/L)
CT does not show a multilobar infarction (hypodensity $>1/3$ cerebral hemisphere)
Relative exclusion criteria if positive—use caution if recommending tPA if one or more are positive
Stroke symptoms are rapidly improving or only minor
Pregnancy
Seizure with postictal residual neurological impairments
Major surgery or major trauma in the previous 14 days
Gastrointestinal or urinary tract hemorrhage in the previous 21 days
Myocardial infarction in the previous 3 months
Some additional considerations
Caution should be exercised in treating a patient with major deficits
Caution using tPA in patients treated with low molecular weight heparin in the past 24 h
Patient or family members understand the potential risks and benefits from treatment. No written consent is required but the conversation should be documented in the clinical notes. Do not delay IV therapy if a surrogate is not readily available as this can lead to worse outcomes
tPA is not FDA approved for treatment of patients under the age of 18. However, tPA has been used off-label in selected pediatric patients with strokes, following careful counseling of parents and using identical eligibility and contraindication criteria to those used in adults. (see Special Considerations: Pediatric Stroke below)

Onset <3 h

If the time from stroke system onset is less than 3 h, it should be confirmed that there are no contraindications for IV tPA (Table 4). One relative contraindication is "clearing neurological deficit." Some patients will clear to near or full recovery without tPA, while others may improve somewhat from a severe stroke but then fail to clear further. If a patient has plateaued or still has significant stroke symptoms and no other contraindication, treatment with tPA should proceed as it would otherwise. Also some patients will present with stuttering symptoms. If symptoms completely resolve, many clinicians will reset the clock to start a new tPA candidacy window; if there are still symptoms—however mild—the time of onset remains unchanged. Patients with stuttering symptoms tend to be high risk for extending their vascular occlusions. Be aware that some patients will have pressure-dependent lesions and lowering of BP may actually exacerbate their symptoms; conversely, allowing their BP to rise above their threshold will improve their deficit.

Common reasons to avoid administering IV tPA are time (duration from first symptom >4.5 h), recent surgery,

current bleeding at a non-compressible site, as well as large area of cerebral infarction that is already apparent as low density on the brain CT or diffusion-weighted MRI study (>1/3 of the middle cerebral artery, or MCA, territory).

Patients with major neurological deficits have a high risk of poor outcome, regardless of whether tPA is administered. In these cases, realistic expectations and risks associated with either choice should be discussed with the patient's family members, and a joint decision should be made. While a glucose level greater than 400 mg/dL is not a contraindication, it should be noted that high glucose may be a stroke mimic, can be associated with worse outcome, and may increase the risk of intracerebral hemorrhage. Similarly, the presence of fever should prompt a reconsideration of the diagnosis. For example, a simple urinary tract infection can bring back old, subclinical stroke symptoms, and once corrected, these stroke-like symptoms may resolve.

Onset Between 3–4.5 h

In the U.S., tPA has not yet been approved by the Food and Drug Administration for use between 3 and 4.5 h, though it has been approved in Europe and Canada. However, tPA use in this timeframe has been endorsed by the American Stroke Association [3] and is widely used.

The inclusion criteria are similar to those of onset <3 h (discussed above), but are modified as noted in Table 5.

Patient is an IV tPA Candidate: IV tPA Administration

After placing two peripheral IV lines, the patient should be weighed rather than using an estimated body weight. Recombinant tPA 0.9 mg/kg should be mixed by swirling (rather than shaking), with the total dose not to exceed 90 mg.

The initial 10 % of the total tPA dose is given by bolus over 1 min, then the remainder is infused over 1 h. As tPA is dispensed in 50 and 100 mg bottles, excess tPA should be withdrawn from the vial and discarded to avoid accidental infusion of the excess.

During the hospital admission or transfer period, there should be continued observation for complications of tPA, including airway obstruction due to angioedema (consider rapid intubation), hemorrhage (stop tPA), and sudden deterioration in mental status.

A sudden decline in neurological status during or following tPA administration may be due to an intracranial hemorrhage. Intracerebral bleeds following IV tPA carry a 50 % or greater mortality rate. This is often accompanied by a marked rise in BP; however, a marked rise or fall in BP alone may signal an ICH. In these cases, the following steps should be immediately taken:

- Stop tPA infusion.
- Obtain a head CT scan STAT.
- Notify the neurosurgeon on call; if a neurosurgeon is not available, begin the process of transferring the patient to a facility with neurosurgical capability once CT scan results are available.
- STAT labs: PT, PTT, platelets, fibrinogen, type, and cross 2–4 unit PRBCs.
- Give the following:
  - 6–8 units of IV cryoprecipitate.

Improvement During Infusion of tPA

If the patient improves during administration of tPA (drop of 4 or more points in NIHSS), it is reasonable to proceed with patient admission or patient transfer, depending on the setting.

No Improvement Following tPA Infusion

If the patient does not improve by a drop of 4 or more points on the NIHSS following tPA (which is often the case), this does not necessarily indicate drug failure: the drug may have successfully opened the occluded intracranial vessel, but it takes time for the brain to recover function. However, the possibility still exists that the vessel remains occluded.

Based on an observed lesser ability for tPA to open larger intracranial vessels, many stroke neurologists refer the patient for endovascular treatment at this stage—especially if the stroke is due to a large vessel intracranial occlusion, demonstrated by CTA or magnetic resonance angiography (MRA), or on clinical grounds.

Onset Between 0–8 h: Endovascular Treatment

If the patient has a large vessel occlusion—e.g., middle cerebral artery (MCA), intracranial internal carotid artery (ICA), basilar or vertebral artery—and is within 6 h of the stroke onset, intra-arterial (IA) thrombolysis may be an option [6, 7]; if the patient is within 8 h, mechanical

Table 5 Additional inclusions to IV tPA use between 3 and 4.5 h [5]

Meet all criteria of <3 h since onset of stroke
Age ≤ 80 years
No anticoagulant use, regardless of INR
NIHSS ≤ 25
No combined history of prior stroke and diabetes

embolectomy treatment should be considered. Several recent randomized trials of embolectomy in acute, large vessel ischemic stroke have shown marked clinical efficacy and even reduction in mortality with or without IV tPA pre-treatment [8–12].

Large vessel occlusion can be suspected by seeing a hyper-dense sign (e.g., clot within the vessel) on non-contrast CT, but this sign is insensitive. CTA or MRA are more diagnostic, as is conventional angiography. It is prudent to contact the neuro interventional physician on call, if one is available; if the treating hospital does not have this capability, consideration may be given to transfer to a comprehensive stroke center. Some hospitals use CT perfusion or MR perfusion techniques to select appropriate patients for intervention, looking for ischemic penumbra, but this practice has not been established as a standard.

Exclusions for IA thrombolysis or embolectomy include the absence of a large vessel occlusion on CTA or MRA, or large area of infarction already present on the brain imaging study.

Hospital Admission or Transfer

Assuming there are no complications of tPA or IA therapy, Table 6 lists the orders that should be considered while waiting for the patient to be admitted.

Additional admission orders must address glucose, volume status, body temperature, and catheters:

- Keep glucose 140–180 mg/dL; consider insulin drip if the blood glucose is persistently >200 mg/dL or the patient is known to have insulin-dependent diabetes mellitus. Hyperglycemia is proven to worsen outcomes flowing ischemic strokes.
- Administer IV fluids, preferably isotonic saline, at 1.5 ml/kg/h initially, with a goal of euvolemia.
- Continue telemetry/bedside cardiac monitoring principally to detect intermittent atrial fibrillation.
- Treat fever sources with antipyretics (while occasionally used in post-cardiac arrest situations as a neuroprotective maneuver, hypothermia has not been sufficiently studied to recommend at present).
- If tPA was administered, avoid indwelling urinary catheter, nasogastric tubes, and intra-arterial catheters for 4 h, and do not give anticoagulant/antiplatelet therapy for 24 h. Urinary catheters should in general be avoided unless absolutely needed.

Table 6 Sample acute stroke admission orders

Neuro check every 15 min for 2 h, then every 30 min for 6 h, then hourly
Supplemental oxygen to keep O_2 saturation >94 %
BP check every 15 min for 2 h, then every 30 min for 6 h, then every hour for 16 h
Keep BP after tPA treatment $<180/105$ mmHg (Note: this is lower than pre-treatment values); if no tPA given, keep BP $<$ 220/120
Bedside swallow test (30 mL water PO) before anything else PO

Pediatric Stroke

While not as common as adults, pediatric stroke occurs in 1.6–13/100,000 children each year [13–15] with select populations, such as children under 1 year of age, children with sickle cell disease or cardiac disorders, having a higher incidence. Challenges in identifying stroke within the pediatric population include possible different presentations than adults and a broad differential diagnosis. Pediatric stroke may often present with seizures, particularly within the younger pediatric population. Headache, and other diffuse, non-localizing signs may also be part of the presenting symptoms. Additionally, other diagnoses, such as migraine, may be equally or even more likely in children presenting with an acute neurologic change [16]. Imaging, particularly with magnetic resonance imaging, is crucial for establishing the diagnosis. The Pediatric NIHSS (PedNIHSS), similar to the adult NIHSS, is a validated, age-appropriate tool for quantifying neurologic deficits in pediatric stroke [17].

Despite its lack of approval for children under 18 years of age, there are several case reports of tPA use in pediatric ischemic stroke cases in the literature. A recent multicenter trial to examine safety and efficacy of tPA in children 2–18 was unfortunately closed early due to insufficient enrollment [18]. This study highlights the challenge of confirming a diagnosis of stroke in children within 4.5 h of symptom onset. While individual physicians may continue to offer thrombolysis therapy with appropriate parental informed consent, one should be careful to apply similar strict inclusion and exclusion safety criteria as used in adults. Regardless of age and antithrombotic treatment, similar neuroprotective strategies should be employed, as described above. Such strategies include avoiding fever and maintaining normoglycemia, euvolemia, and adequate BP.

TIA

The diagnosis of TIA is based on the new onset of focal neurological symptoms and signs that are explainable by a vascular disease (e.g., arterial occlusion of a single or group of arteries adequately explain the patient's signs and

symptoms), and these signs and symptoms resolve within 24 h (most TIAs resolve in a much shorter period of time).

However, up to one-third of TIAs have demonstrable injury on MRI [19]. Although these cases are now classified as stroke, it is unlikely that emergency reperfusion therapy should be attempted since tissue injury is present and all symptoms have resolved. Approximately, 50 % of patients with a TIA will have CT findings of a prior ischemic stroke even though this may have been clinically silent.

TIAs present a conundrum as there clearly was an event and the patient is at some risk for a recurrence. There are several tools that may help provide guidance assessing the risk of recurrence or outright stroke at different time intervals. Each has limitations and one must assess patient compliance, available resources in one's practice environment as a decision is made with the patient and family for the best disposition and follow-up. The $ABCD^2$ score is commonly used and is presented below.

$ABCD^2$ Score

The $ABCD^2$ score is an ordinal scale that provides risk prediction of subsequent stroke following a TIA. Table 7 demonstrates how to calculate this score.

Add all of the points from above for the total $ABCD^2$ score (0–7). Table 8 lists the estimated percent risk of a stroke occurring within various time ranges.

Based on this risk stratification, some physicians choose to admit high-risk patients and discharge those at low-risk. There is controversy regarding admission of moderate-risk patients, and this decision follows local practices.

Low-Risk TIA

For low-risk patients ($ABCD^2$ scores 0–3), an outpatient workup in the 1–2 days following score calculation may be most appropriate. Alternately, observation or admission may be an option. In either case, stroke can be prevented by rapid institution of the following regimen [21]:

- Begin an antithrombotic agent (ASA 81 mg/day, clopidogrel 75 mg/day, or ASA 25 mg/extended release dipyridamole 200 mg twice daily).
- Perform carotid imaging: ultrasound, CTA, or MRA.
- Consider transthoracic echocardiography; if bilateral infarcts are present on CT or there is high suspicion of cardiac embolic source, and transthoracic echo is normal, obtain transesophageal echocardiograph (TEE).
- Consider 30-day ambulatory cardiac monitor to detect intermittent atrial fibrillation (cryptogenic A-fib). This should be strongly considered if the workup shows no other etiology for cause of stroke or TIA.
- Encourage smoking cessation.
- Initiate high-intensity statin (atorvastatin 40–80 mg/day or rosuvastatin 20–40 mg or equivalent). Consider moderate intensity statins in patients >75-year old [22].

If ECG or rhythm strip shows atrial fibrillation, consider starting anticoagulation (oral anticoagulant or low molecular weight heparin) or ASA; calculate CHADS2 or CHA2DS2-VASc [23], and HAS-BLED [24] scores to help guide long-term therapy. In these cases, referral to a vascular neurologist or cardiologist is appropriate.

Moderate and High-Risk TIA

For patients with moderate and high risk TIAs ($ABCD^2$ scores >3), hospital admission is advisable. In addition to the treatments discussed above, some physicians keep

Table 7 Calculate $ABCD^2$ score [20]

$ABCD^2$ score	Points
Age > 60 years	1
BP ≥ 140/90 mmHg at initial evaluation	1
Clinical features of the TIA	
Speech disturbance without weakness, or	1
Unilateral weakness	2
Duration of symptoms	
10–59 min, or	1
>60 min	2
Diabetes mellitus in patient's history	1

Table 8 Percent risk of stroke following TIA with various $ABCD^2$ scores [20]

Total risk	Score	2 days	7 days	90 days
Low	0–3	1.0	1.2	3.1
Moderate	4–5	4.1	5.9	9.8
High	6–7	8.1	12	18

Table 9 Acute ischemic stroke communication regarding assessment and referral

Communication
☐ Age
☐ Airways status
☐ Time of symptom onset (time of day)
☐ NIHSS
☐ CT or MRI results
☐ tPA administration or contraindication(s) to tPA
☐ Endovascular intervention(s) if applicable

patients on bed rest for a day, with the head of bed flat (in order to maintain brain perfusion). After 24 h, the patient should begin to get out of bed as tolerated with assistance. Permissive hypertension is encouraged (not to exceed 220/120 mmHg), and BP limits should be gradually lowered over 24–48 h.

Communication

When communicating to an accepting or referring physician about this patient, consider including the key elements listed in Table 9.

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