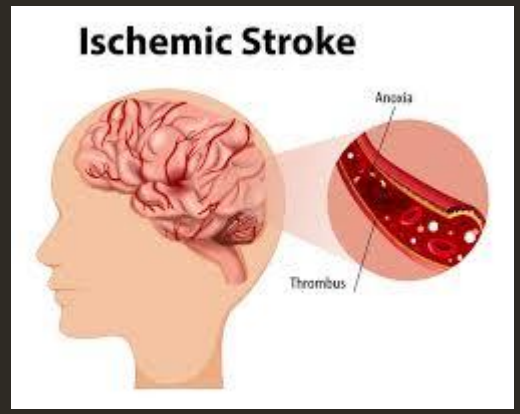

18th International Congress of Anesthesiology, Critical Care and Pain

Acute Ischemic Stroke in the Critically Ill Patient

From Reperfusion to Neurocritical Care

Shahram Amini, MD, FCCM
Professor of Anesthesiology and Critical Care
MUMS

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Objectives

1. Understand the pathophysiology and classification of ischemic stroke
2. Recognize the clinical presentation and diagnostic approach .
3. Apply evidence-based acute management strategies, including thrombolysis and thrombectomy.
4. Learn ICU-specific considerations for stroke care.
5. Explore emerging therapies and future directions1.

Pathophysiology of Ischemic Stroke

Definition: Sudden loss of blood flow to a part of the brain due to arterial occlusion.

Mechanisms:

- Thrombotic: Atherosclerosis leading to plaque rupture (e.g., carotid stenosis).
- Embolic: Cardioembolic (e.g., atrial fibrillation) or artery-to-artery embolism.
- Systemic Hypoperfusion: Global ischemia (e.g., cardiac arrest).

Core vs. Penumbra: - Core: Irreversibly damaged tissue.

Penumbra: Salvageable tissue with timely intervention. –

Epidemiology of Ischemic Stroke

- Stroke is the fifth leading cause of adult death worldwide and a major cause of disability.
- Incidence: 12.2 million new strokes annually (globally).
- Prevalence: 101 million people living with stroke (2020 data).
- 800000 people suffer strokes in the US, 82-90% of theses are ischemic
- Regional Variations: Higher incidence in low- and middle-income countries (LMICs) due to limited access to preventive care.
- High-income countries see more strokes in older populations due to aging demographics.

Epidemiology of Ischemic Stroke

- As of 2019, stroke claimed 3.29 million lives worldwide. That number is expected to increase further to 4.90 million by year 2030
- Only 3–8.5% of potentially eligible patients receive thrombolytic therapy .
- For patients with an arterial occlusion in the proximal middle cerebral artery or basilar artery, thrombectomy is another acute therapeutic intervention.
- A 2019 study performed at a comprehensive stroke center (CSC) found that only seven out of one hundred (7%) AIS patients were eligible for thrombectomy according to the 2018 guidelines by the American Heart Association (AHA).

Epidemiology of Ischemic Stroke

Risk Factors

- Modifiable: Hypertension (most significant), diabetes, smoking, obesity, atrial fibrillation, hyperlipidemia.
- Non-Modifiable: Age (>55 years), gender (men > women), race (higher in Black and Hispanic populations), family history.

ICU Relevance: ~30% of stroke patients require ICU admission due to complications (e.g., hemorrhagic transformation, cerebral edema).

Stroke categories

- Large artery: thrombotic in situ occlusion on the atherosclerotic lesions in the carotid, verterbrobasilar, and cerebral arteries typically proximal to major branches
- Small vessel or lacunar: typically in deep penetrating arteries
- Cardioembolic infarction: common cause of recurrent stroke(20% with the highest mortality)

Prognosis -Short-Term Outcomes

- Mortality: 10-20% in the first 30 days.
- Disability: 50% of survivors have moderate-to-severe disability (e.g., hemiparesis, aphasia).
- Complications

➢ Hemorrhagic transformation (5-10% with thrombolysis).

➢ Malignant cerebral edema (10-20% in large MCA strokes).

➢ Aspiration pneumonia, DVT/PE, seizures.

Prognosis -Long-Term Outcomes

- Recurrence: ~10% risk within 1 year, ~25% within 5 years.
- Functional Recovery: Early rehabilitation improves outcomes. ~30% of survivors achieve full independence at 6 months.
- Quality of Life: Persistent cognitive impairment, depression, and fatigue are common.
- Predictors of Poor Prognosis:

✓ Large infarct volume
✓ older age
✓ delayed treatment
✓ comorbidities (e.g., diabetes, heart failure)

Clinical Presentation

- FAST Mnemonic
  - F: Face drooping.
  - A: Arm weakness.
  - S: Speech difficulty.
  - T: Time to call emergency services.
- Other Symptoms: - Hemiparesis, aphasia, visual field defects, ataxia, altered mental status.
- Key Point: Time of symptom onset is critical for treatment decisions

Artery involved	Syndrome
Anterior cerebral artery	Middle portion of frontal and parietal lobes and anterior portion of the basal ganglia and anterior internal capsule Motor and/or sensory deficit (leg > face, arm) Grasp, sucking reflexes Abulia, paratonic rigidity, gait apraxia
Middle cerebral artery	Lateral portion of parietal and frontal lobes and anterior and lateral portions of temporal lobes, prforating brances to the globus pallidus, putamen, and internal capsule Dominant hemisphere: aphasia, motor and sensory deficit (face, arm > leg > foot), may be complete hemiplegia if internal capsule involved, homonymous hemianopia Non-dominant hemisphere: neglect, anosognosia, motor and sensory deficit (face, arm > leg > foot), homonymous hemianopia
Posterior cerebral artery	Thalami and brain stem and cortical branches to the posterior and middle temporal lobes and occipiyal lobes Homonymous hemianopia; alexia without agraphia (dominant hemisphere); visual hallucinations, visual perseverations (calcarine cortex); sensory loss, choreoathetosis, spontaneous pain (thalamus); III nerve palsy, paresis of vertical eye movement, motor deficit (cerebral peduncle, midbrain)
Penetrating vessels	Pure motor hemiparesis (classic lacunar syndromes) Pure sensory deficit Pure sensory-motor deficit Hemiparesis, homolateral ataxia Dysarthria/clumsy hand
Vertebrobasilar	Cranial nerve palsies Crossed sensory deficits Diplopia, dizziness, nausea, vomiting, dysarthria, dysphagia, hiccup Limb and gait ataxia Motor deficit Coma Bilateral signs suggest basilar artery disease
Internal carotid artery	Progressive or stuttering onset of MCA syndrome, occasionally ACA syndrome as well if insufficient collateral flow

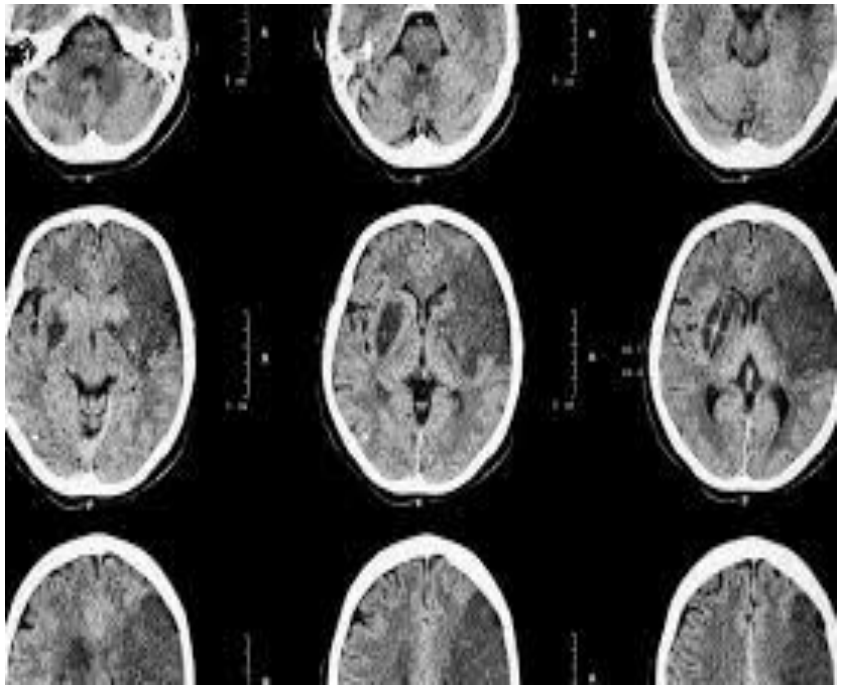
Diagnostic Workup

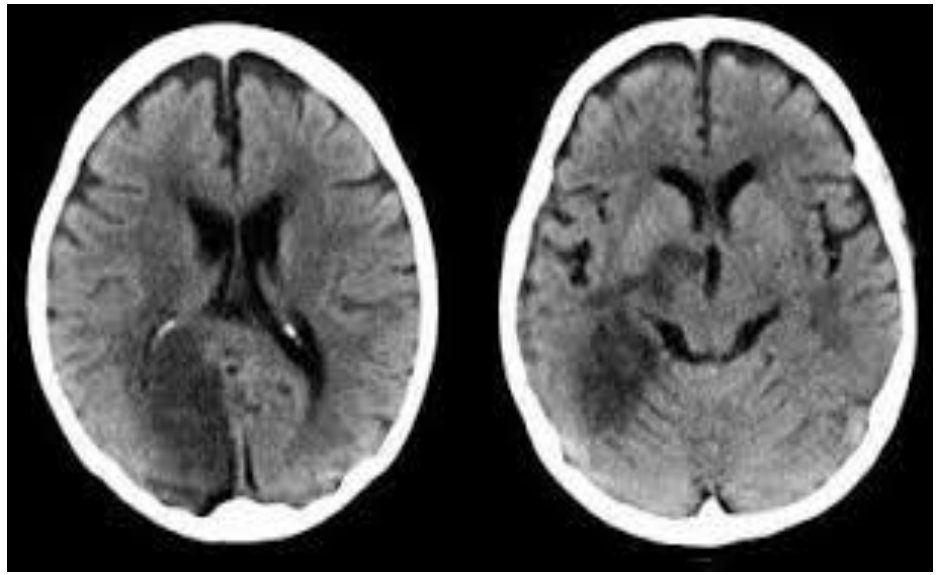
- **Imaging**
  - Non-contrast CT Head: Rule out hemorrhage (first-line).
  - CT Angiography (CTA): Identify large vessel occlusion (LVO).
  - MRI: Diffusion-weighted imaging (DWI) for acute ischemia.
- **Labs**: CBC, coagulation profile, glucose, electrolytes, cardiac biomarkers. - Hypercoagulable workup if indicated (e.g., young patient, cryptogenic stroke).
- **Cardiac Evaluation**: - ECG (atrial fibrillation), echocardiogram (cardiac source of embolism).

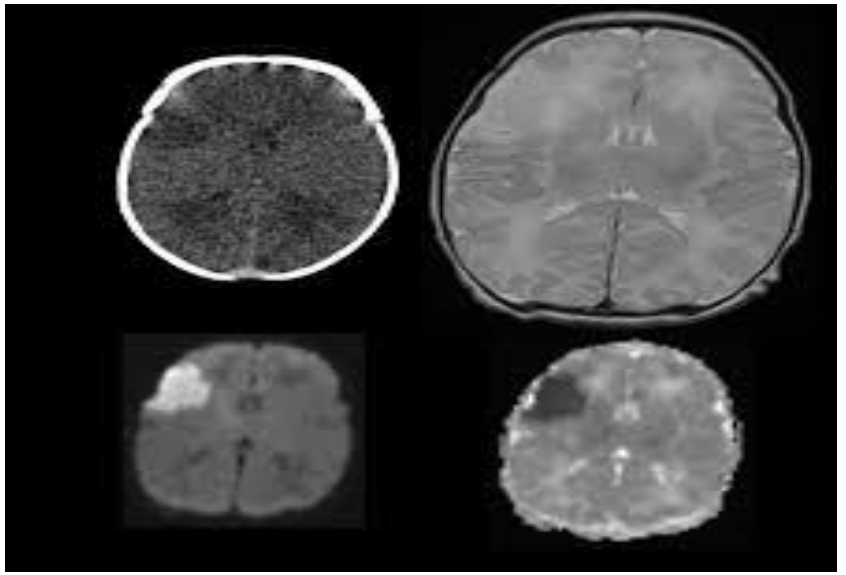
Cerebellar hemispheres

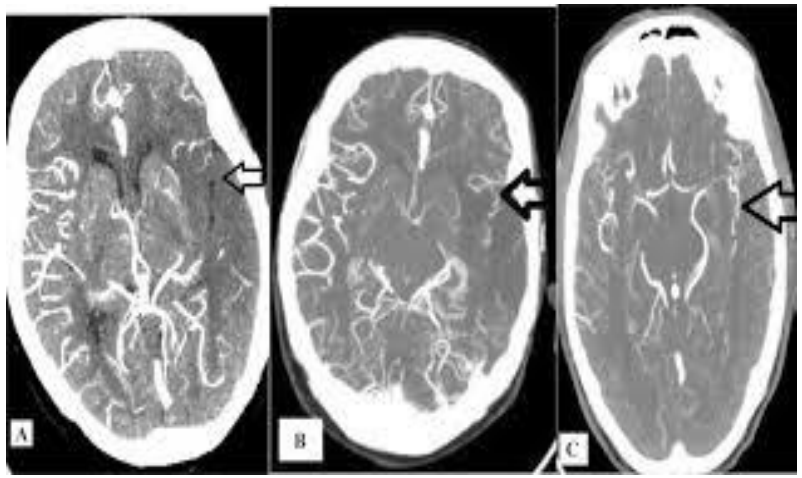
- Inferiorly by the posterior cerebeela artery arising from the vertebral artery
- Supeiorly by the superior cerebellar artery
- Anterolaterallyt by the AICA from the basilar artery



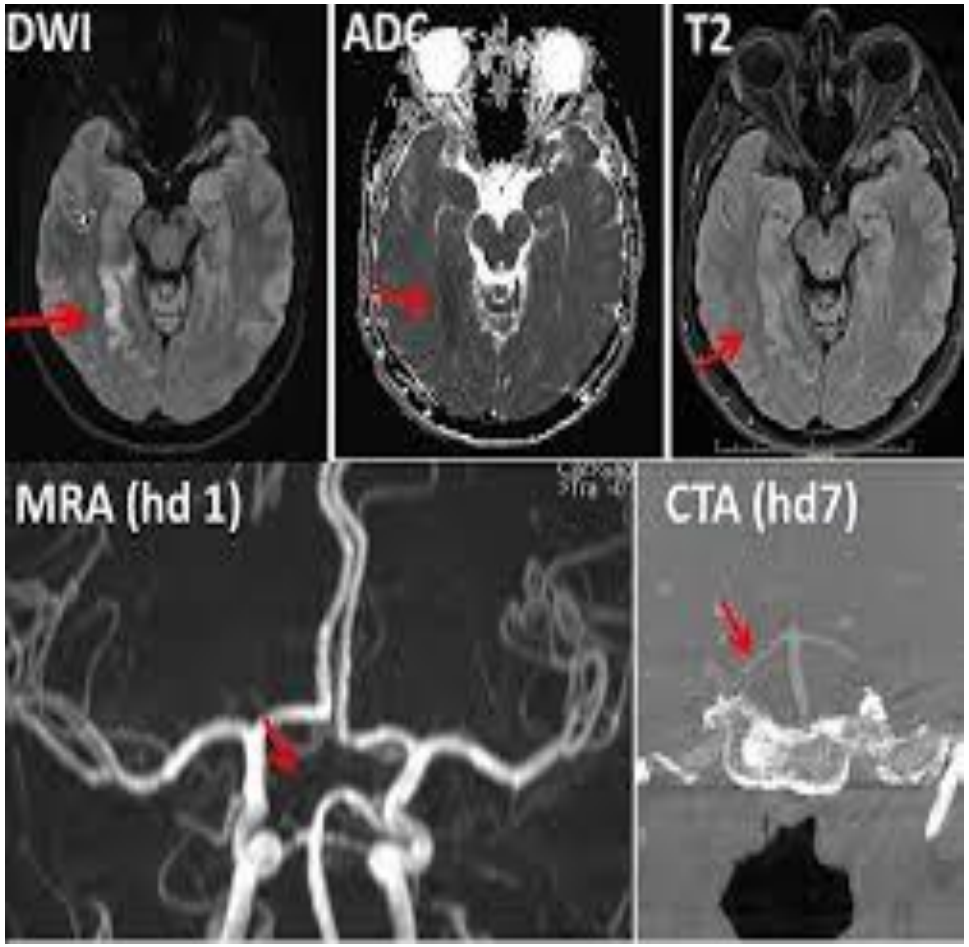







A
B
C




DWI
ADC
T2
MRA (hd 1)
CTA (hd7)



Time is Brain!

	Neurons Lost	Synapses Lost	Myelinated Fibers Lost	Accelerated Aging
Per Stroke	1.2 billion	8.3 trillion	7140 km/4470 miles	36 y
Per Hour	120 million	830 billion	714 km/447 miles	3.6 y
Per Minute	1.9 million	14 billion	12 km/7.5 miles	3.1 wk
Per Second	32 000	230 million	200 meters/218 yards	8.7 h

- **Time is Brain**

➢ Goal: Door-to-needle time <60 minutes for thrombolysis.

➢ Goal: Door-to-puncture time <90 minutes for thrombectomy.

- **Thrombolysis**

❖ Alteplase (tPA): - Dose: 0.9 mg/kg (max 90 mg), 10% as bolus, rest over 1 hour. Window: Within 4.5 hours of symptom onset (relative contraindications apply).

❖ Tenecteplase: Emerging alternative with easier administration (single bolus).

- Thrombectomy: Indication: LVO (e.g., MCA, ICA) within 24 hours of symptom onset (selected patients).
- Devices: Stent retrievers, aspiration catheters.
- Evidence: DAWN, DEFUSE-3 trials support extended window (6-24 hours). –

ACUTE ISCHEMIC STROKE CARE MAP



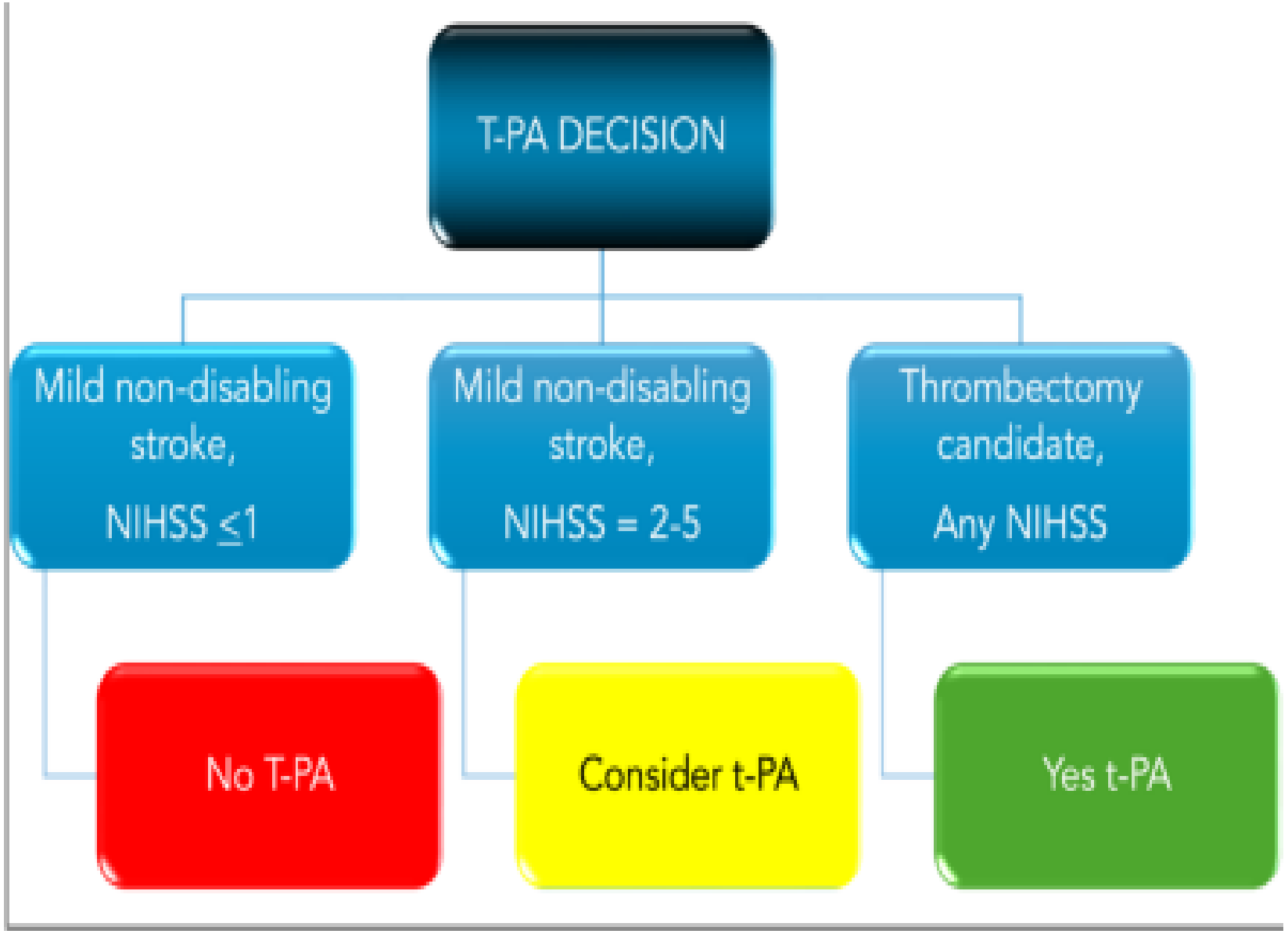
1	Determine time of onset of symptoms
2	Obtain noncontrast brain CT
3	Calculate NIHSS
4	Check glucose. If <60 mg/dL, give D50
5	Maintain oxygen >94%
6	Check temperature, treat fever
7	Obtain Neurology consult
8	Keep patient NPO, dysphagia screen
9	Give aspirin, unless ICH or true allergy
10	BP: maintain MAP> 100 mmHg with saline or pressors if necessary; do not lower BP unless > 220/110 mmHg unless concurrent emergency or tPA candidate
11	Obtain ancillary testing (labs, ECG)
12	Do not anticoagulate

Eligibility criteria for the treatment of acute ischemic stroke with intravenous thrombolysis (recombinant tissue plasminogen activator or tPA)

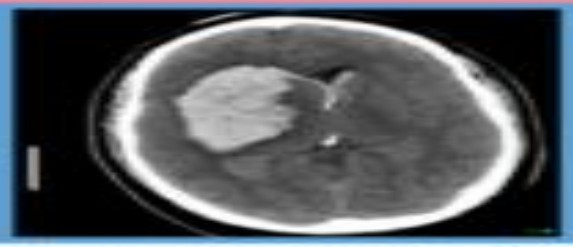
Inclusion criteria
•Clinical diagnosis of ischemic stroke causing measurable neurologic deficit •Onset of symptoms <4.5 hours before beginning treatment; if the exact time of stroke onset is not known, it is defined as the last time the patient was known to be normal or at neurologic baseline •Age ≥18 years
Exclusion criteria
•Patient history of Ischemic stroke or severe head trauma in the previous three months •Previous intracranial hemorrhage •Intra-axial intracranial neoplasm •Gastrointestinal malignancy •Gastrointestinal hemorrhage in the previous 21 days •Intracranial or intraspinal surgery within the prior three months
•Clinical Symptoms suggestive of subarachnoid hemorrhage •Persistent blood pressure elevation (systolic ≥185 mmHg or diastolic ≥110 mmHg) •Active internal bleeding •Presentation consistent with infective endocarditis •Stroke known or suspected to be associated with aortic arch dissection •Acute bleeding diathesis, including but not limited to conditions defined under 'Hematologic'
•Hematologic Platelet count <100,000/mm^3* •Current anticoagulant use with an INR >1.7 or PT >15 seconds or aPTT >40 seconds* •Therapeutic doses of low molecular weight heparin received within 24 hours (eg, to treat VTE and ACS); this exclusion does not apply to prophylactic doses (eg, to prevent VTE) •Current use (i.e, last dose within 48 hours in a patient with normal renal function) of a direct thrombin inhibitor or direct factor Xa inhibitor with evidence of anticoagulant effect by laboratory tests such as aPTT, INR, ECT, TT, or appropriate factor Xa activity assays
•Head CT Evidence of hemorrhage •Extensive regions of obvious hypodensity consistent with irreversible injury

Eligibility criteria for the treatment of acute ischemic stroke with intravenous thrombolysis (recombinant tissue plasminogen activator or tPA)

Warnings[¶]
•Only minor and isolated neurologic signs or rapidly improving symptoms[Δ] •Serum glucose <50 mg/dL (<2.8 mmol/L)[◊] •Serious trauma in the previous 14 days[§] •Major surgery in the previous 14 days[¥] •History of gastrointestinal bleeding (remote) or genitourinary bleeding[‡] •Seizure at the onset of stroke with postictal neurologic impairments[†] •Pregnancy** •Arterial puncture at a noncompressible site in the previous seven days[¶¶] •Large (≥10 mm), untreated, unruptured intracranial aneurysm[¶¶] •Untreated intracranial vascular malformation[¶¶]
•**Additional warnings for treatment from 3 to 4.5 hours from symptom onset**[ΔΔ] Age >80 years •Oral anticoagulant use regardless of INR •Severe stroke (NIHSS score >25) •Combination of both previous ischemic stroke and diabetes mellitus


T-PA DECISION
Mild non-disabling stroke, NIHSS ≤1
Mild non-disabling stroke, NIHSS = 2-5
Thrombectomy candidate, Any NIHSS
No T-PA
Consider t-PA
Yes t-PA

MANAGEMENT OF SYMPTOMATIC INTRACRANIAL BLEEDING OCCURRING WITHIN 24 HOURS OF INTRAVENOUS T-PA

STOP ALTEPLASE	**Draw labs** CBC PT/INR aPTT FIBRINOGEN TYPE and CROSSMATCH
GET NON-CONTRAST HEAD CT Look for hemorrhage [1]	
CRYO-PRECIPITATE	**Includes Factor VIII, XIII, fibrinogen, Von Willebrand factor** 10U infused over 10-30 minutes Obset in 1 hr, peaks in 12 h Administer additional dose for fibrinogen level <150
ANTI-FIBRINOLYTICS	**Tranexamic acid** 1000 mg Iv infused over 10 minutes **Aminocaproic acid** 4-5g over 1 hr, followed by 1g IV/hr until bleeding is controlled
SUPPORTIVE CARE	**Consult hematology and neurosurgery** Control BLOOD PRESSURE Treat FEVER Maintain blood GLUCOSE within normal range

[1] https://radiopaedia.org/cases/cerebral-haemorrhage-post-tpa-thrombolysis

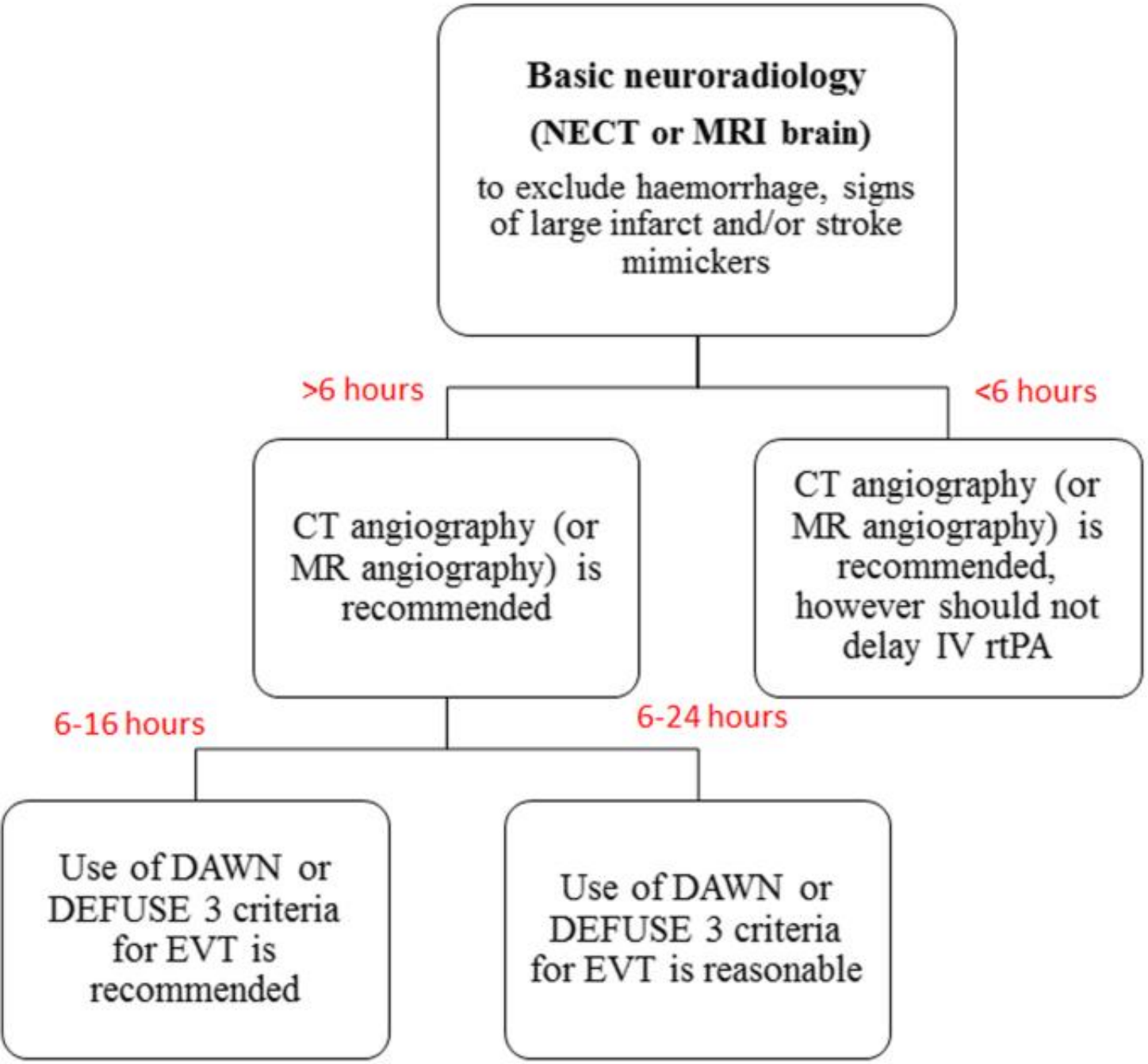
- Airway protection if indicated: prophylactic ceftriaxone 2 gr within 12 hours of intubation
- Keep SpO2> 94%
- Fluid management
- Head and body position

- In case of thrombolytics: BP< 185/110 before thrombolytics and < 180/105 for at least 24 hours thereafter
- The 2026 guideline specifically advises against an SBP target below 140 mmHg for the first 72 hours after successful anterior-circulation reperfusion in patients without another indication
- Otherwise: treat if BP>220/120 unless (15% in the first 24 h)

  - Active ischemic coronary disease
  - Heart failure
  - Aortic dissection
  - Hypertensive encephalopathy
  - Pre-eclampsia/eclampsia

Options to treat hypertension before and during reperfusion therapy for acute ischemic stroke

Patient otherwise eligible for acute reperfusion therapy except that blood pressure is >185/110 mmHg*
Labetalol 10 to 20 mg intravenously over 1 to 2 minutes, may repeat one time; **or**
Nicardipine 5 mg/hour intravenously, titrate up by 2.5 mg/hour every 5 to 15 minutes, maximum 15 mg/hour; when desired blood pressure reached, adjust to maintain proper blood pressure limits; **or**
Clevidipine 1 to 2 mg/hour intravenously, titrate by doubling the dose every 2 to 5 minutes, maximum 21 mg/hour, until desired blood pressure reached¶; **or**
Other agents (hydralazine, enalaprilat, etc) may also be considered
If blood pressure is not maintained at or below 185/110 mmHg, do **not** administer alteplase
Management to maintain blood pressure at or below 180/105 mmHg during and after acute reperfusion therapy*
Monitor blood pressure every 15 minutes for 2 hours from the start of rtPA therapy, then every 30 minutes for 6 hours, and then every hour for 16 hours
If systolic blood pressure is >180 to 230 mmHg or diastolic is >105 to 120 mmHg:
Labetalol 10 mg intravenously followed by continuous infusion 2 to 8 mg/min; **or**
Nicardipine 5 mg/hour intravenously, titrate up to desired effect by 2.5 mg/hour every 5 to 15 minutes, maximum 15 mg/hour; **or**
Clevidipine 1 to 2 mg/hour intravenously, titrate by doubling the dose every 2 to 5 minutes, maximum 21 mg/hour, until desired blood pressure reached¶
If blood pressure is not controlled or diastolic blood pressure >140 mmHg, consider intravenous sodium nitroprusside



DAWN criteria
ICA – MCA occlusion

Mismatch clinical deficit-infarct volume:

- 80 years or older: NIHSS ≥10 + <21 ml infarct core
- <80 years: NIHSS ≥ + <31 ml infarct ***or*** ≥ 20 NIHSS + 31-51 ml infarct

DEFUSE 3 criteria
ICA – proximal MCA occlusion

Mismatch clinical deficit-infarct volume:

- Initial infarct <70 ml + volume ischemic tissue / volume infarct= ≥ 1,8
- No NIHSS score threshold

- The normal cerebral blood fow (CBF) in the human brain is approximately 50–60 mL per 100 g of tissue per minute .
- When cerebral blood flow drops to 50%, synaptic transmission stops, and cerebral energy use decreases by 50%.
- If blood flow falls below 25 ml/10 g of tissue /min, it is called **pnumbra** that can remain viable for several hours because of marginal tissue perfusion. At this point, autoregulation fails and the ischemic brain becomes dependent on systemic circulation for perfusion
- If blood flow falls below 10 ml/10 g of tissue /min, it is called **ischemic core** id presumed to die within minutes.
- Post-thrombolysis: Maintain BP <180/105 mmHg.
- Post-thrombectomy: Individualized based on reperfusion status.

Drug	Dose	Onset of action	Elimination half life	Duration of action	Contra-indications	Caution
Labetalol	10–20 mg IV over 1–2 min, may repeat 1 time	2–5 min	5.5 h	4 h	Bradycardia; 2nd or third-degree heart block, severe asthma	
Clevidipine	1–2 mg/h IV, titrate by doubling the dose every 2–5 min until desired BP reached; maximum 21 mg/h	2–4 min	1 min	5–15 min		Lipid emulsification can result in hyper-triglyceridemia
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	5–10 min	40–60 min	15–90 min	Severe aortic stenosis	Cumulative doses can result in cyanide toxicity
Enalaprilat	1.25 mg/dose given over 5 min every 6 h	< 15 min	35 h	6 h	history of angioedema related to an ACE inhibitor; bilateral renal artery stenosis	Not easily titratable, long half life

- Agents that are not recommended for blood pressure treatment in acute ischemic stroke are hydralazine and nitroglycerin, due to their vasodilatory properties that can result in an extension of the area of infarct. Hydralazine raises intracranial pressure, which, together with its effect upon systemic blood pressure, reduces cerebral perfusion pressure

ICU-Specific Considerations

- Glycemic Control: - Target: 140-180 mg/dL. Avoid hypoglycemia (exacerbates brain injury).

Patients with hyperglycemia were 2.3 times more likely to be dead at 90 days compared to those with normal glucose

- Temperature Control: -Treat fever aggressively (antipyretics, cooling devices). Therapeutic hypothermia: Emerging evidence for neuroprotection.

A retrospective cohort study of 9366 patients with AIS found that peak temperature in the frst 24 h of 39 °C was associated with an increased risk of in-hospital death compared to normothermia

- Swallowing Assessment: - Screen for dysphagia to prevent aspiration pneumonia. Keep the patients NPO

Patients with infarctions of the brain stem, multiple strokes, major hemispheric lesions, or depressed consciousness are at the greatest risk for aspiration

After reperfusion: the ICU mission

- *Detect hemorrhagic transformation, re-occlusion, edema and neurological deterioration early*
- *Maintain physiological stability and protect the reperfused but injured brain*
- *Perform structured neurological assessments and escalate changes immediately*
- *Coordinate antithrombotic restart, stroke mechanism work-up and secondary prevention*
- *Avoid unnecessary sedation that obscures the neurological examination*

Neurological monitoring and escalation

- *Establish a reproducible baseline: NIHSS, pupils, motor response and level of consciousness*
- *After IVT: frequent neurological and BP checks per protocol; commonly q15 min initially*
- *Any acute headache, vomiting, hypertension or neurological decline → urgent CT and hemorrhage evaluation*
- *Use continuous EEG for unexplained impaired consciousness or suspected nonconvulsive seizures*
- *Consider repeat vascular/perfusion imaging when re-occlusion or infarct progression is suspected*

Hemorrhagic transformation and symptomatic ICH

- *Risk relates to infarct size, reperfusion injury, BP, glucose and antithrombotic exposure*
- *Recognize: abrupt neurological decline, headache, vomiting, acute hypertension or reduced consciousness*
- *Stop thrombolytic infusion if ongoing; obtain emergent CT, CBC, coagulation tests and fibrinogen*
- *Activate stroke, neurocritical care, neurosurgery and transfusion/hematology teams*
- *Use institutional reversal protocol—typically cryoprecipitate to restore fibrinogen; consider antifibrinolytic therapy*

Malignant MCA infarction: anticipate the curve

- *: Large hemispheric infarction can produce progressive edema, midline shift and herniation*
- *Clinical deterioration often evolves over 24–72 hours; risk is not excluded by early stability*
- *High-risk features: large core, poor collaterals, early swelling, declining consciousness*
- *Serial examination and imaging; involve neurosurgery early—not after fixed herniation*
- *Osmotherapy is a bridge to definitive management, not a substitute for timely surgery*

Osmotherapy and intracranial pressure

- *Treat clinical or radiological intracranial hypertension; avoid prophylactic osmotherapy*
- *Hypertonic saline or mannitol may be used for acute deterioration / impending herniation*
- *Monitor sodium, osmolality, renal function, volume status and hemodynamics*
- *Avoid hypotension and preserve cerebral perfusion during treatment*
- *Routine corticosteroids do not treat ischemic cerebral edema*

Decompressive surgery: do not miss the window

- *Decompressive hemicraniectomy reduces mortality in selected patients with malignant hemispheric infarction*
- *Benefit is greatest when performed early, generally within 48 hours of stroke onset*
- *Discuss survival with substantial disability—not mortality alone—with family and surrogate decision-makers*
- *Cerebellar infarction with brainstem compression or obstructive hydrocephalus requires urgent neurosurgical assessment*
- *Age, premorbid function, infarct extent and patient values inform shared decisions*

Posterior circulation and basilar artery occlusion

- *May present with coma, quadriparesis, dysarthria, gaze abnormalities or fluctuating deficits*
- *NIHSS can underestimate severity in posterior circulation stroke*
- *Urgent CTA/MRA is essential when basilar occlusion is suspected*
- *Selected basilar occlusion patients benefit from EVT within 24 hours; strongest evidence in more severe deficits*
- *Monitor closely for brainstem respiratory failure, dysphagia and hydrocephalus*

Seizures and EEG

- *Treat clinical seizures promptly; correct provoking metabolic disturbances*
- *Continuous EEG is appropriate for unexplained coma, fluctuating examination or suspected nonconvulsive status*
- *Routine antiseizure prophylaxis is not recommended in the absence of seizures*
- *Choose agents considering hemodynamic effects, interactions, renal/hepatic function and sedation burden*
- *Avoid allowing sedative accumulation to masquerade as neurological deterioration*

Airway, dysphagia and aspiration prevention

- *Intubate for airway failure, loss of protective reflexes or respiratory failure—not NIHSS alone*
- *Screen swallowing before oral intake, including medications*
- *Use early enteral nutrition when safe; maintain aspiration precautions and oral care*
- *Extubate when airway protection, cough, secretion management and neurological trajectory permit*
- *Tracheostomy decisions should reflect expected course, goals and multidisciplinary assessment*

Antithrombotics and secondary prevention: the ICU interface

- *After IV thrombolysis, withhold antiplatelets/anticoagulants for 24 hours and obtain follow-up imaging*
- *For minor non-cardioembolic stroke not treated with IVT, short-course dual antiplatelet therapy may be appropriate*
- *Anticoagulation timing after cardioembolic stroke depends on infarct size, hemorrhagic risk and indication*
- *Identify mechanism: large artery, cardioembolic, small vessel, dissection or other*
- *Coordinate statin, vascular risk reduction, AF evaluation and carotid management with stroke team*

Emerging Therapies

- Neuroprotection: Cerebrolysin: Peptide-based therapy with neurotrophic effects.
- Remote Ischemic Conditioning: Intermittent limb ischemia to reduce brain injury.
- Revascularization: Tenecteplase: Easier administration, non-inferior to alteplase.
- Sonothrombolysis: Ultrasound-enhanced clot lysis.
- Stem Cell Therapy: Investigational for promoting neuroregeneration.

Cerebrolysin

Disorder	Daily dosage	Initiation of treatment	Treatment Duration
Stroke	20 - 50 ml	as soon as possible	10 - 21 days
Traumatic brain injury	20 - 50 ml	as soon as possible	7 - 30 days
Alzheimer's disease	10 - 30 ml	as soon as possible	1 cycle: 5 days weekly/4 weeks 2 - 4 cycles per year
Vascular dementia	10 - 30 ml	as soon as possible	1 cycle: 5 days weekly/4 weeks 2 - 4 cycles per year

Intravenous (IV) infusion	10 ml - 50 ml	diluted to at least 100 ml total volume with Saline, Ringer solution or 5% glucose solution	Infuse within 15 – 60 min
To guarantee sterility, always use disposable one-way infusion sets and cannulas! Start the infusion immediately after dilution!			

Intravenous (IV) injection	up to 10 ml	undiluted	inject slowly over 3 minutes
Intramuscular (IM) injection	up to 5 ml	undiluted	inject slowly over 3 minutes
The IV or IM injection has to be administered immediately after opening the ampoule!			

Complications

- Hemorrhagic transformation (HT)
  - Risk increased with thrombolysis because of reperfusion.
  - Within 2-14 days (first week)
  - From pethechial hemorrhage to hematoma formation(evacuation)
  - More commonly following cardioembolic and large strokes

- Cerebral edema: Malignant MCA infarction (consider decompressive craniectomy). 10-20%

- Seizure: 2-23%, may become chronic

- Systemic: - Aspiration pneumonia, DVT/PE, cardiac arrhythmias.
- Delirium: 25%; preexisting cognitive decline, infection, greater stroke severity
- Key Point: Early recognition and management of complications improve outcomes


What's a disabling stroke?
Complete hemianopia: ≥2 on NIHSS
Severe aphasia: ≥2 on NIHSS
Visual extinction: ≥1 on NIHSS
Any weakness limiting sustained effort against gravity: ≥2 on NIHSS question 5 or 6
Any other deficit considered potentially disabling by the patient, family, or the treating clinician
Any deficit that would prevent the patient from performing basic activities of daily living (ie, bathing, walking, toileting, and eating or returning to work)

References

- 1. AHA/ASA Guidelines for Early Management of Acute Ischemic Stroke (2026).
- 2. DAWN and DEFUSE-3 Trials: Thrombectomy in Extended Windows (2018)
- 3. Tenecteplase vs. Alteplase for Acute Ischemic Stroke (2022)
- 4. UpToDate: Management of Acute Ischemic Stroke (2026).

Thank You for Your Attention

Questions and Discussion

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