

Revascularization for acute ischemic stroke treatment

1. Revascularization Strategies The two primary revascularization approaches for AIS are:

A. Intravenous Thrombolysis (IVT)

[Intravenous recombinant human tissue plasminogen activator for ischemic stroke treatment.](#)

Standard treatment for AIS within 4.5 hours of symptom onset.

Effective for small- and medium-sized clots but limited efficacy for large vessel occlusions (LVOs).

Risk: Symptomatic intracerebral hemorrhage (sICH) in ~6% of cases.

Tenecteplase (TNK-tPA)

Emerging alternative with potential benefits, including higher fibrin specificity, longer half-life, and simpler administration (single bolus dose).

Comparable or superior efficacy to Alteplase in LVO strokes.

B. Endovascular Thrombectomy (EVT)

For AHA guidelines for [mechanical thrombectomy](#), see Practice guideline:

[Mechanical thrombectomy for large vessel occlusion](#)

Indicated for large vessel occlusions (LVOs) within the anterior circulation (ICA, M1, proximal M2, BA) up to 24 hours after onset, based on neuroimaging criteria.

Mechanical Thrombectomy Techniques: Stent Retrievers (Solitaire, Trevo): Expand within the clot, capturing it for removal. Aspiration Catheters (ADAPT technique): Suction applied to remove the thrombus directly. Combined Techniques: Stent retriever plus aspiration (Solumbra technique). 2. Patient Selection for EVT Early Window (0–6 hours): Strong evidence for EVT in patients with LVO and disabling stroke symptoms. Late Window (6–24 hours): Selection based on advanced imaging (CT perfusion or MRI-DWI mismatch) per DAWN and DEFUSE 3 trials. Exclusion Criteria: Extensive infarction (ASPECTS <6), poor functional baseline (mRS ≥5), distal occlusions. 3. Bridging Therapy (IVT + EVT) Patients presenting within 4.5 hours with LVO may receive IVT before EVT, improving clot fragmentation and recanalization rates. Ongoing trials (SWIFT-DIRECT, MR CLEAN-NOIV) aim to determine the necessity of IVT before EVT. 4. Emerging Revascularization Approaches Intra-arterial thrombolysis: Direct administration of thrombolytics during EVT for residual clot burden. Neuroprotection Strategies: Adjunctive therapies to protect penumbra tissue (hypothermia, neuroprotective agents). Next-generation devices: Improved aspiration catheters, balloon guide catheters, and robot-assisted thrombectomy. 5. Post-Revascularization Care Blood Pressure Control: <180/105 mmHg post-IVT, <140/90 mmHg post-EVT. Dual Antiplatelet Therapy (DAPT): If no

hemorrhagic risk. Statin therapy, diabetes control, and lifestyle modifications for secondary prevention.

Endovascular intervention

[Endovascular treatment for acute ischemic stroke](#).

see also [Cerebral venous sinus thrombosis treatment](#).

Remarkable developments in the field of [endovascular neurosurgery](#) have been witnessed in the last decade. The success of [endovascular therapy](#) for [ischemic stroke](#) treatment is now irrefutable, making it an accepted standard of care ¹⁾.

In [ischemic stroke](#) or patients with [TIA](#) less than five cerebral microbleeds (CMBs) should not affect [antithrombotic](#) decisions, although with more than five CMBs the risks of future [ICH](#) and ischaemic stroke are finely balanced, and antithrombotics might cause net harm. In lobar ICH populations, a high burden of strictly lobar CMBs is associated with [cerebral amyloid angiopathy](#) (CAA) and high ICH risk; antithrombotics should be avoided unless there is a compelling indication ²⁾.

American Heart Association Guidelines for the Early Management of Patients With Acute Ischemic Stroke

[American Heart Association Guidelines for the Early Management of Patients With Acute Ischemic Stroke](#)

Hypothermia

see [Hypothermia for acute ischemic stroke treatment](#).

Brain ischemia and treatment are one of the important topics in neurological science. Free oxygen radicals and inflammation formed after ischemia are accepted as the most important causes of damage. Currently, there are studies on many chemopreventive agents to prevent cerebral ischemia damage. The aim of Aras et al is to research the preventive effect of the active ingredient in [genistein](#) There is currently no promising pharmacotherapy aside from intravenous or intra-arterial [thrombolysis](#). Yet because of the narrow therapeutic time window involved, thrombolytic application is very restricted in clinical settings. Accumulating data suggest that non-pharmaceutical therapies for stroke might provide new opportunities for stroke treatment ³⁾.

Progression of focal stroke symptoms still constitutes a serious clinical problem for which heparin has insufficient effectiveness in clinical practice. New therapies, ideally preventive, are needed ⁴⁾.

[Omega 3 fatty acid](#) enhance cerebral angiogenesis and provide long-term protection after stroke ⁵⁾.

After cerebral ischemia, revascularization in the ischemic boundary zone provides nutritive blood flow as well as various growth factors to promote the survival and activity of neurons and neural progenitor cells. Enhancement of angiogenesis and the resulting improvement of cerebral microcirculation are key restorative mechanisms and represent an important therapeutic strategy for ischemic stroke.

With the emergence of new technologies in imaging, thrombolysis and endovascular intervention, the treatment modalities of acute ischemic stroke will enter a new era ⁶⁾.

Within 3 h from symptom onset, the existence of [FLAIR](#)-positive lesions on pretreatment MRI is significantly associated with an increased bleeding risk due to systemic thrombolysis. Therefore, considering FLAIR-positive lesions on baseline MRI might guide treatment decisions in ischemic stroke ⁷⁾.

Stent retriever

[Stent retriever for acute ischemic stroke treatment.](#)

Thrombolysis

see [Acute ischemic stroke thrombolysis](#)

Blood Pressure Management

see [Blood Pressure Management.](#)

Rehabilitation

Intensive rehabilitation effectively improves physical functions in patients with acute stroke, but the frequency of intervention and its cost-effectiveness are poorly studied. This study aimed to examine the effect of early high-frequency rehabilitation intervention on inpatient outcomes and medical expenses of patients with stroke.

Methods: The study retrospectively included 1759 patients with acute stroke admitted to the Kobe City Medical Center General Hospital between 2013 and 2016. Patients with a transient ischemic attack, subarachnoid hemorrhage, and those who underwent urgent surgery were excluded. Patients were divided into two groups according to the frequency of rehabilitation intervention: the high-frequency intervention group (>2 times/day, n = 1105) and normal-frequency intervention group (<2 times/day, n = 654). A modified Rankin scale score ≤ 2 at discharge, immobility-related complications and medical expenses were compared between the groups.

Results: The high-frequency intervention group had a significantly shorter time to first rehabilitation

(median [interquartile range], 19.0 h [13.1-38.4] vs. 24.7 h [16.1-49.4], $P < 0.001$) and time to first mobilization (23.3 h [8.7-47.2] vs. 22.8 h [5.7-62.3], $P = 0.65$) than the normal-frequency intervention group. Despite higher disease severity, the high-frequency intervention group exhibited favorable outcomes at discharge (modified Rankin scale, ≤ 2 ; adjusted odds ratio, 1.89; 95% confidence interval, 1.25-2.85; $P = 0.002$). No significant differences were observed between the two groups concerning the rate of immobility-related complications and total medical expenses during hospitalization.

Conclusions: High-frequency intervention was associated with improved outcomes and decreased medical expenses in patients with stroke. Our results may contribute to reducing medical expenses by increasing the efficiency of care delivery ⁸⁾.

Endogenous Neural Stem Cell-induced Neurogenesis after Ischemic Stroke

Currently, [stem cell therapy](#) is the most promising approach for inducing [neurogenesis](#) for neural repair after ischemic stroke. Stem cell treatments include transplantation of exogenous stem cells but also stimulating endogenous neural stem cells (NSCs) proliferation and differentiation into neural cells ⁹⁾.

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