Acute ischemic stroke treatment

- The Anti-Parkinsonian A2A Receptor Antagonist Istradefylline (KW-6002) Attenuates Behavioral Abnormalities, Neuroinflammation, and Neurodegeneration in Cerebral Ischemia: An Adenosinergic Signaling Link Between Stroke and Parkinson's Disease
- Sinusoidal Extremely Low-Frequency Electromagnetic Stimulation (ELF-EMS) Promotes Angiogenesis In Vitro
- Non-Saccular Aneurysm Shape as a Poor Prognostic Factor in Younger Patients with Spontaneous Subarachnoid Hemorrhage
- The Complex Role of the miR-17-92 Cluster in Stroke: Mechanistic Insights and Biomarker Potential
- Mechanisms of mitochondrial dynamics in ischemic stroke and therapeutic strategies
- Unusual Evolution of Carotid Atherosclerosis in a Patient with Transient Ischemic Attack
- Regional Differences in Presentation, Cause, and Outcome of Reversible Cerebral Vasoconstriction Syndrome
- Postacute Discharge Destination and Major Adverse Cardiovascular Events Among Patients With Intracerebral Hemorrhage

The treatment of acute ischemic stroke is time-sensitive and involves a combination of medical interventions and, in some cases, interventional procedures. The primary goals of treatment are to restore blood flow to the affected brain tissue and minimize neurological damage.

Revascularization for acute ischemic stroke treatment

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Early Recognition and Rapid Response

Identification of acute ischemic stroke with large vessel occlusion (AIS-LVO) etiology is crucial for effective revascularization therapy.

Recognize the signs and symptoms of a stroke, such as sudden weakness, numbness, or difficulty speaking, and call emergency services immediately. Time is crucial, and early intervention can improve outcomes.

Ren et al. demonstrated national marked and sustainable improvement in adherence to door-to-needle time, door-to-puncture time, and successful reperfusion therapy from 2013 to 2017 in Japan in patients with acute ischemic stroke. Adhering to the key Quality Indicators substantially affected in-hospital outcomes, underlining the importance of monitoring the quality of care using evidence-based QIs and the nationwide Close The Gap-Stroke program [1)].

Medical Evaluation and Diagnosis

Upon arrival at the hospital, healthcare providers will perform a physical examination and order diagnostic tests, including a CT scan or MRI to confirm the diagnosis and identify the type of stroke. Intravenous Thrombolysis (tPA):

If the patient is eligible and presents within the appropriate time window (usually within 4.5 hours of symptom onset), intravenous tissue plasminogen activator (tPA) may be administered. tPA is a clot-busting medication that can dissolve the blood clot causing the stroke.

Blood pressure control

see Blood Pressure Management in Acute Ischemic Stroke

Depending on the underlying cause of the stroke (e.g., atrial fibrillation), antiplatelet agents (like aspirin) or anticoagulant medications may be prescribed to prevent further clot formation.

Supportive Care

Patients with acute ischemic stroke may require supportive care to manage complications and improve recovery. This may include treatments for associated conditions such as fever, high blood sugar, and seizures. Rehabilitation:

After the acute phase, stroke survivors often require rehabilitation, including physical therapy, occupational therapy, and speech therapy, to regain lost functions and improve their quality of life.

Secondary Prevention

Patients are usually prescribed medications to manage risk factors, such as high blood pressure, high cholesterol, and diabetes. Lifestyle modifications, such as a healthy diet, regular exercise, and smoking cessation, are also emphasized to reduce the risk of recurrent strokes.

Psychosocial Support

Stroke can have a significant impact on a person's emotional and psychological well-being. Support from healthcare professionals, family, and support groups can be essential for coping with the emotional aspects of recovery. It's important to note that the specific treatment plan may vary based on individual factors, including the cause and severity of the stroke, the patient's overall health, and the time elapsed since symptom onset. Rapid medical evaluation and intervention remain critical for improving outcomes in acute ischemic stroke cases.

Time is of the essence when diagnosing, and treating a stroke (brain attack) and restoring normal blood flow to the affected areas. The success and effectiveness of interventional stroke therapy literally depends on how quickly a patient recognizes the symptoms of stroke and seeks emergency medical care. Interdisciplinary in nature.

TeleStroke considerably improves quality of stroke care (for instance, by increasing thrombolysis) and may be valuable for the management of intracranial hemorrhages in rural hospitals and hospitals lacking neurosurgical departments, given that surgical/interventional therapy is only recommended for a subgroup of patients.

For ischemic strokes caused by blood clots, the medical team will first give clot-busting drugs, such as tPA (tissue plasminogen activator) intravenously to patients who arrive at the hospital within three hours from the onset of stroke symptoms. If the tPA does not adequately dissolve the clot or cannot be administered within the ideal timeframe, Northwestern's neuroendovascular experts can offer intra-arterial (through the artery) thrombolysis treatment. This therapy helps extend the required treatment time for tPA up to six hours by delivering the clot-busting drug directly to the blockage. This intra-arterial procedure involves inserting, under x-ray guidance, a catheter (a thin tube) into the femoral artery located in the patient's leg, near the groin area, and guiding it to the site of the clot. From there, the interventional neuroradiology team can locally administer a clot-busting agent to dissolve the blockage. The patient undergoes the entire procedure under general anesthesia.

Clots can also be broken up or removed via mechanical means. Using the same type of catheter-based technique as other intra-arterial therapies, Northwestern's interventional neuroradiologists have at their disposal advanced clot-retrieving systems for the treatment of ischemic stroke. Inserted through the catheter, a tiny corkscrew-shaped device, called the MERCI, allows the neuroendovascular specialists to capture the clot and pull it free and away from the site of the blockage. This procedure can be used up to eight hours after the onset of stroke symptoms.

Since patients with stroke frequently develop bladder dysfunction, a careful approach is required to reduce unnecessary indwelling urinary catheter (IUC) for preventing catheter-associated urinary tract infection (CAUTI).

Ikeda-Sakai et al. conducted a prospective interrupted time series study in three tertiary care hospitals in Japan. Adult patients with acute stroke were eligible. The study consisted of three phases: baseline, education and implementation. The program included an assessment of IUC indications, educational meetings among healthcare professionals, reminders for removal of inappropriate IUC and a urinary retention protocol. The primary outcome was the proportion of inappropriate IUC use to assess effectiveness. The device utilization ratio and incidence of CAUTI were examined to assess effectiveness, and incidences of urinary retention and all symptomatic urinary tract infection (UTI) were examined to assess safety.

Among 976 patients who met the inclusion criteria, 738 were analysed. Inappropriate IUC use

decreased from 50.1% in the baseline phase to 22.5% in the implementation phase (absolute risk reduction in interrupted time series analysis 42.4% [95% confidence interval, 19.2%-65.6%]). The device utilization ratio decreased from 0.302 to 0.194 ($p < 0.001$), whereas CAUTI did not change significantly (from 8.81 to 8.28 per 1000 catheter-days; incidence rate ratio 0.95 [0.44-1.94]). All symptomatic UTI decreased from 9.5% to 4.9% ($p = 0.015$), with no increase in urinary retention.

The program improved the appropriateness of IUC use in stroke care while ensuring safety [2].

As the second-leading cause of death, stroke faces several challenges in terms of treatment because of the limited therapeutic interventions available. Previous studies primarily focused on metabolic and blood flow properties as a target for ischemic stroke treatment, including recombinant tissue plasminogen activator and mechanical thrombectomy, which are the only USFDA approved therapies. These interventions have the limitation of a narrow therapeutic time window, the possibility of hemorrhagic complications, and the expertise required for performing these interventions. Thus, it is important to identify the contributing factors that exacerbate the acute ischemic stroke outcome and to develop therapies targeting them for regulating cellular homeostasis, mainly neuronal survival and regeneration. Glial cells, primarily microglia, astrocytes, and oligodendrocytes, have been shown to have a crucial role in the prognosis of ischemic brain injury, contributing to inflammatory responses. They play a dual role in both the onset as well as resolution of the inflammatory responses. Understanding the different mechanisms driving these effects can aid in the development of therapeutic targets and further mitigate the damage caused. In a review, Jadhav et al. summarize the functions of various glial cells and their contribution to stroke pathology. The review highlights the therapeutic options currently being explored and developed that primarily target glial cells and can be used as neuroprotective agents for the treatment of ischemic stroke [3].

In the complete absence of blood flow, neuronal death occurs within 2–3 minutes from the exhaustion of energy stores. However, in most strokes, there is a salvageable penumbra (tissue at risk) that retains viability for a period of time through suboptimal perfusion from collaterals. Local cerebral edema from the stroke results in a compromise of these collaterals and progression of the ischemic penumbra to infarction if the flow is not restored and maintained. Prevention of this secondary neuronal injury drives the treatment of stroke and has led to the creation of designated Primary Stroke Centers that offer appropriate and timely triage and treatment of all potential stroke patients.

Time delays from initial CTA acquisition to neuroendovascular surgery (NES) team notification can prevent expedient treatment with endovascular thrombectomy (ET). Process improvements and automated stroke detection on imaging with automated notification of the NES team may ultimately improve the time to reperfusion [4].

Restoring the circulation is the primary goal in emergency cerebral ischemia treatment. However, better understanding of how the brain responds to energy depletion could inform the time available for resuscitation until irreversible damage and advance development of interventions that prolong this span.

Finding novel agent for cerebral ischemia therapy is urgently required. In a study, Gao et al., aimed to investigate the regulatory mechanism of Ginkgolides B (GB) in hypoxia-injured PC-12 cells.

PC-12 cells were exposed to hypoxia and administrated with GB. Cell viability was detected by MTT assay. Flow cytometry assay was conducted for the detection of cell apoptosis, ROS generation and cell cycle assay. The changes of protein levels of Bax, Pro/Cleaved-Caspase-3, CyclinD1, CDK4, CDK6, PI3K/AKT and MEK/ERK pathways were detected by Western blot. Transfection was conducted for Polo-like kinase 1 (PLK1) knockdown.

Hypoxia-induced decrease of cell viability and increase of ROS generation, apoptosis and cell cycle arrest were ameliorated by GB. Hypoxia disposition hindered PI3 K/AKT and MEK/ERK signaling pathways while GB had the opposite effects. Then we observed that hypoxia exposure suppressed PLK1 expression while GB increased PLK1 expression dose-dependently. Knockdown of PLK1 attenuated the neuroprotective effects of GB on hypoxia-injured PC-12 cells and also inhibited PI3 K/AKT and MEK/ERK pathways.

The above observations corroborated that GB alleviated hypoxia-induced PC-12 cell injury by up-regulation of PLK1 via activating PI3K/AKT and MEK/ERK pathways. These findings implied the neuro-protective impacts in hypoxia-injured PC-12 cells [5].

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