



A COMPREHENSIVE REVIEW ON ACUTE ISCHEMIC STROKE; ADVANCES IN UNDERSTANDING, DIAGNOSIS AND MANAGEMENT

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ABSTRACT:

This review article aims to provide information regarding various etiological [risk factors], which are associated with both include, modifiable and non-modifiable. It provides and identify the clinical manifestations of acute ischemic stroke [According to American Heart Association (AHA)]. As stroke is the second most common cause of death. It ranked as the fourth most common cause of disability globally. 20 million out of 5 million people who get stroke each year will pass eventually.

This review also provides information regarding epidemiological data and pathology involving acute ischemic stroke. It provides, appropriate diagnostic criteria to evaluate stroke, according to American academy of neurological guidelines. It provides various treatment approaches to treat acute ischemic stroke, along with rehabilitation therapy and new investigational strategies.

Key words: Ischemic stroke, Diagnostic, Rehabilitation therapy

INTRODUCTION:

The World Health Organization describes stroke as a clinical syndrome that includes quickly evolving clinical indications of a focal (or global, in the case of a coma) disruption of cerebral function that lasts longer than 24 hours or results in death with no evident reason other than a vascular origin.

This is referred to as a transient ischemic attack (TIA) because the focused neurological abnormalities only last for a brief period, typically less than an hour.

CLASSIFICATION:

Strokes are the two main classes of stroke. In US, ischemic stroke accounts for 87% of conclusion; hemorrhagic stroke histories for 13%. A] Intracerebral hemorrhage, or atypical bleeding within the brain or brain parenchyma, and subarachnoid hemorrhage, or anomalous bleeding within the subarachnoid space, are both embraced in hemorrhagic stroke. Trauma or instinctive bleeding can bring about both subarachnoid hemorrhage and intracerebral hemorrhage. While there's coherent medical carefulness accessible to handle hemorrhagic stroke, the development of attestation- grounded treatments for hemorrhagic stroke has not observed up with the ascents in the treatment of ischemic stroke, which are regrettably far less effectual. B] The tenure" ischemic stroke" refers to a discontinuity in blood inflow to the brain parenchyma that results in the oxygen- starved doom of brain cells. It may result from Large-arterial atherosclerosis, small vessel occlusions, cardiac emboli, and other causes.

EPIDEMIOLOGY:

The second the most prevalent cause of death and disability globally is stroke. Over the past few decades, poor countries have grown more affected by stroke than modernized ones. Currently, developing nations account for 81% of all stroke-related disability adjusted life years (DALYs) and 75% of all stroke-related deaths. The burden of disease is believed to be altering from modernized to developing nations because of population increase, ageing, and shifting disease patterns brought on by variations in risk factors, socioeconomic status, and access to healthcare. Because of this, stroke has become a major threat to public health in developing nations.

Approximately 90 Americans suffer a stroke every hour in the United States, leading to a fatality every four minutes. After ischemic heart disease, stroke is the second greatest cause of death and disability globally and the fifth most common cause of death in the United States. The United States' southeastern region, dubbed the "stroke belt" because of its proximity to the country, has greater stroke mortality rates than other regions. Residents there have a 20% to 50% higher risk of dying from a stroke than those in other regions. The three most prevalent risk factors for acute coronary syndrome, also known as ACS, are Type 2 diabetes (30%), high blood pressure (38%), and cigarette smoking (40%), despite India experiencing the highest prevalence of ACS internationally. We might accurately assume that stroke is exceptionally prevalent in India according to the information specified above and considering that stroke and ACS share common risk variables.

CLINICAL MANIFESTATIONS:

Stroke clinical presentation varies depending on the region of the brain wherein obstruction of arteries happens. The FAST strategy for detecting ischemic stroke when in a prehospital scenario has been gaining prominence courtesy to the American Heart Association and American Stroke Association (AHA/ASA). The abbreviated form of FAST represents 1] speech limitation, 2] face droop 3] arm/ limb weakness, and 4] time of began. Another straightforward approach to pay attention to indicators of ischemic stroke is the technique known as 6S or the BEFAST strategy.

Recalling the 6S Protocol for Assessing Stroke,

- Sudden (symptoms generally appear immediately)
- Dialogue (speech) that is slurred or unidentified, seems impaired.
- Side Weak (face, arm, or leg, or a combination of the three)

- Spinning/Rolling (vertigo)
- a powerful(severe) personality headache
- Seconds (account for the onset of symptoms and head straight to the hospital)
- Be fast: Arm (weakness), Speech (slurred), Face (facial drooping), Eyes (disturbance of vision in one or both eyes), and Balance (loss of balance/dizziness) tests.



PATHOPHYSIOLOGY:

A stroke is highlighted as an unpredictable neurological break out caused by inadequate blood vessel circulation to the brain. Understanding the neurovascular structure serves as essential for assessing the stroke's neurological symptoms. Two internal carotid arteries situated anteriorly, and two vertebral arteries positioned posteriorly control blood flow to the brain (the circle of Willis). Hemorrhagic stroke has been brought about by bleeding or ruptured blood vessels, although ischemic stroke is caused by inadequate blood and oxygen reaching the brain.

Stroke patients, ischemic occlusions consider roughly 85 percent of fatalities with intracerebral hemorrhage responsible for the remainder 15%. In the brain, ischemic obstruction induces thrombotic and embolic scenarios. Atherosclerosis-related constriction of the arteries limits blood flow in thrombosis. The accumulation of plaque eventually leads to the vascular chamber narrowing down and clotting, ultimately resulting in thrombotic stroke. Impaired circulation to the brain region resulting in an embolism in an embolic stroke; such embolism causes excessive stress and premature deaths of cells (necrosis). During necrosis, the plasma membrane gets damaged, organelles expand and leak the contents of cells into the extracellular environment, acidosis, impairment of equilibrium, raised intracellular calcium levels and the excitotoxicity cytokine-mediated cytotoxicity, toxicity generated by free radicals, complement stimulation, breakdown of the blood-brain barrier, glial cell being activated, oxidative damage, and infiltrated leukocytes.

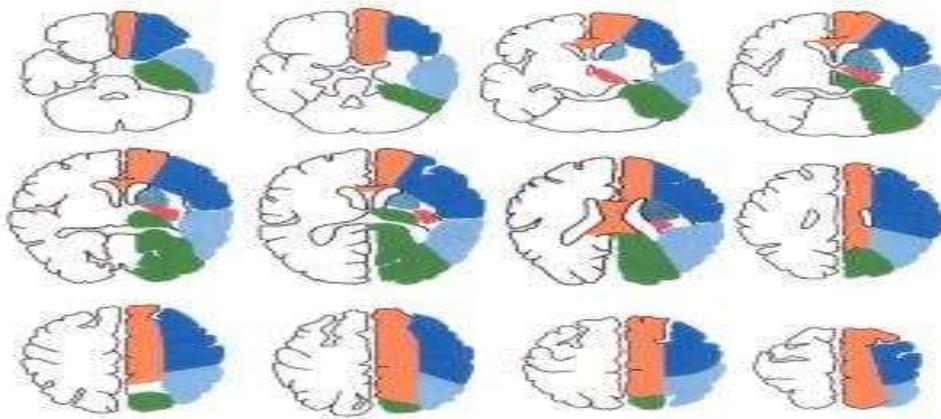
Posterior circulation Acute stroke prognosis early CT score (pc-ASPECTS)



T: thalamus; OL: occipital lobe; M: any part of the midbrain; P: any part of the pons; C: cerebellar hemisphere.



Vascular Territories



■ Middle Cerebral Artery: Superior Division ■ Posterior Cerebral Artery
■ Middle Cerebral Artery: Inferior Division ■ Anterior Cerebral Artery
■ Middle Cerebral Artery: Lenticulostriate ■ Anterior Choroidal

- Antinuclear antibody (ANA)
- RA Factor
- Serum homocysteine level and toxicological screening.

OTHERS, Fasting lipid profile and ESR.

Clinical Features and Vascular Territories of Stroke		
Clinical Finding	Vascular Territory	Additional Comments
Contralateral leg weakness	<i>Anterior circulation</i> —anterior cerebral artery (ACA)	Includes stem of Circle of Willis connecting internal carotid artery to ACA, and the segment distal to ACA and its anterior choroidal branch Largest vascular bed for stroke
Contralateral face, arm > leg weakness, sensory loss, field cut, aphasia (left MCA) or neglect, apraxia (right MCA)	<i>Anterior circulation</i> —middle cerebral artery (MCA)	
Contralateral motor or sensory deficit without cortical signs	<i>Subcortical circulation</i> *—lenticulostriate deep penetrating branches of MCA	Small vessel subcortical <i>lacunar infarcts</i> in internal capsule, thalamus, or brainstem. Four common syndromes: pure motor hemiparesis; pure sensory hemianesthesia; ataxic hemiparesis; clumsy hand—dysarthria syndrome
Contralateral field cut	<i>Posterior circulation</i> —posterior cerebral artery (PCA)	Includes paired vertebral and basilar artery, paired posterior cerebral arteries. Bilateral PCA infarction causes cortical blindness but preserved pupillary light reaction.
Dysphagia, dysarthria, tongue/palate deviation and/or ataxia with crossed sensory/motor deficits (= ipsilateral face with contralateral body)	<i>Posterior circulation</i> —brainstem, vertebral, or basilar artery branches	
Oculomotor deficits and/or ataxia with crossed sensory/motor deficits	<i>Posterior circulation</i> —basilar artery	Complete basilar artery occlusion—“locked-in syndrome” with intact consciousness but with inability to speak and quadriplegia

*Learn to differentiate cortical from subcortical involvement. *Subcortical or lacunar syndromes* do not affect higher cognitive function, language, or visual fields.
Source: Adapted from American College of Physicians. Stroke, in Neurology: Medical Knowledge Self-Assessment Program (MKSAP) 14. Philadelphia: American College of Physicians, 2006. pp. 52-68.

RISK FACTORS:

NON-MODIFIABLE:

- **Ageing:** The development of stroke is facilitated by the progressive nature of both modifiable and non-modifiable risk factors with respect to age. When a person reaches the age of 55, their risk of stroke doubles.
- **Gender:** Men are more likely than women to get a stroke, and their incidence rate is higher. In the age range of 35–44 and over 85, women experience strokes at a somewhat higher rate than males.
- **Ethnic:** Studies conducted in the United States indicate that white people are less likely to experience a stroke than Hispanic or Black people. The fact that Black people have a much greater incidence of hemorrhagic stroke than do age-matched White people.
- Previous episodes of headaches.



- **Genetics:** Another risk factor for stroke is a history of stroke in both the mother and the father. It causes stroke by a combination of variables, including cultural and religious background within families and hereditary stroke risk factors.

MODIFIABLE

- High blood pressure: There is a connection between cerebral bleeding and cerebral infarction due to hypertension. Among modifiable risk factors for cerebrovascular accidents, hypertension is the most significant one. It is responsible for roughly 50% of ischemic strokes.
- Diabetes type 2: It results in a mortality rate that is about 20% higher and increases the risk of ischemic stroke. Furthermore, the prognosis following a stroke is poorer for those with diabetes than for those without the disease; this includes a higher risk of severe disability and a slower rate of recovery.
- Heart diseases: Prosthetic heart valves, congenital heart illnesses with shunt lesions, rheumatic heart disease with valvular lesions, and dilated cardiomyopathy are other cardiac causes of stroke that result in thrombo embolism. Therefore, 20% of the cause of ischemic stroke is cardio embolism.
- Elevated levels of cholesterol: Men who have excessive cholesterol, for example, have a higher death rate. In those whose blood cholesterol levels range from 240 to 279 mg/dL, the risk ratio is 1.8 and the ratio is 2.6 when cholesterol levels are ≥ 280 mg/dL.
- Atrial fibrillation: Depending on the patient's age, atrial fibrillation (AF) can increase the risk of stroke by two to five times. It causes more severe impairment and higher death than strokes unrelated to AF, accounting for 15% of all strokes.
- A medical condition of carotid stenosis
- A high concentration of homocysteine
- Lifestyle worries: Indulging excessive in smoking, drinking, consuming illegal drugs, instead of exercise.
- Being overweight, Combination of contraceptive pills.

COMPLICATIONS:

Box 2. Long-term complications of stroke:

Late medical complications

- | | |
|------------------------|--|
| • Post-stroke seizures | Treat with conventional anticonvulsants. Seizure advice: general safety advisory, driving and operating machinery. |
| • Urinary incontinence | Exclude exacerbating/precipitating risk factors. Consider oxybutynin in selected patients and indwelling catheter as a last resort. |
| • Bowel incontinence | Review of medications, improving diet/fluid intake, using drugs such as codeine phosphate with twice weekly enema or loperamide to reduce frequency of incontinence. |
| • Cognitive impairment | Control of risk factors to prevent recurrence. Physical and cognitive stimulation. |

Musculoskeletal complications

- | | |
|--------------------------------|---|
| • Spasticity and hypertonicity | Physiotherapy, splinting, positioning of limbs. Systemic drugs include baclofen, tizanidine, dantrolene, diazepam, and botulinum toxin in selected cases. |
| • Hemiplegic shoulder pain | Proper handling and positioning, refer to physiotherapy, simple analgesic, transcutaneous electrical nerve stimulation in selected patients. |
| • Wrist and hand flexion | Physiotherapy (both active and passive), splints (ready-made splints or custom made). |

Psychosocial complications

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|--|--|
| • Post-stroke depression | Selective serotonin reuptake inhibitors (SSRIs) are commonly used. Psychological counselling is helpful but often underused. |
| • Emotional lability (pseudobulbar affect) | Therapeutic trial of antidepressants, especially SSRIs. |
| • Mood/emotional changes | Stroke and caregivers support groups may be helpful, e.g. the Singapore National Stroke Association (www.snsa.org.sg) provides education and support for stroke patients and their family in community. |

TREATMENT APPROACH:

PHARMACOLOGICAL THERAPY

FIBRINOLYTIC THERAPY:

Fibrinolytic therapy in acute ischemic stroke breaks up blood clots, preventing blood flow to the brain to restore blood flow to the brain areas that have not yet suffered an infarct. According to AHA/ASA (American Stroke Association) guidelines, Intravenous alteplase (tPA) is the mainstay of thrombolysis in acute ischemic stroke that improves functional outcomes significantly when administered within 4.5 hours of stroke onset. All patients with acute ischemic stroke who present within a 4.5-hour window from their last known well time and without any absolute contraindication should receive treatment with intravenous alteplase (tPA). The benefit is significant when treatment with thrombolytics occurs early after stroke onset and declines with time.

Mechanical thrombectomy is beneficial when an acute ischemic stroke results from a proximal intracranial arterial occlusion. According to a recent meta-analysis, patients with acute ischemic stroke, having large vessel occlusion receiving Tenecteplase have better recanalization and clinical outcomes than those receiving intravenous alteplase. Alteplase is the preferred thrombolytic agent in acute ischemic stroke. The recommended dose is 0.9 mg/kg (maximum 90 mg total dose), 10% of the total dose is administered as an initial IV bolus over one minute, and the remaining dose is infused over 60 minutes.

In situations involving acute ischemic stroke, fibrinolytic therapy breaks down blood clots, restricting the flow of blood to the brain and enabling blood to flow back to the regions of the brain that are yet to suffer an infarct. The key component of thrombolytic therapy in acute ischemic stroke is iv alteplase (tPA), which significantly enhances functional results if administered before a period of 4.5 hours of stroke onset, in accordance with guidance from the American Heart Association and the American Stroke Association. Systemic alteplase (tPA) ought to be used as a treatment for all acute ischemic stroke patients who are present within a period of 4.5 hours following their final known healthy time and lacking any essential contraindications.

A recent systematic review discovered that those suffering from acute ischemic stroke who get Tenecteplase for major blood vessel occlusion have improved medical results and recanalization compared to those whose receive alteplase systemically. During acute ischemic stroke, alteplase is the preferred thrombolytic therapy. 10 percent of the total dose is administered as an initial IV bolus over a minute, and the balance is infused for 60 mins. The suggested dosage is 0.9 mg/kg, with a maximum cumulative frequency of 90 mg.

ANTIPLATELET:

While antiplatelet medication within the first 48 hours of the assumed ischemic stroke, aspirin 160 mg to 300 mg daily, given orally (or via a nasogastric tube or per rectum in people who can't swallow), substantially decreases mortality and dependence and reduces the possibility of an early recurrent ischemic stroke with no a significant risk of early hemorrhagic complication; long-term results were enhanced All patients who have had an AIS or TIA should receive long-term antithrombotic therapy for secondary prevention

All patients who have had AIS or TIA should receive long-term antithrombotic therapy for secondary prevention.

The early use of Aspirin can reduce long-term death and disability owing to ischemic stroke.

Aspirin + Clopidogrel is now considered as first line agents for secondary stroke prevention.

Aspirin 50 – 325mg and Clopidogrel 75mg daily.

STATIN THERAPY:

It was successfully shown that statins reduced the risk of stroke among individuals with CAD and raised plasma lipids by about 30%.

The National Cholesterol Education Programmer (NCEP) considers ischemic stroke or a TIA (transient ischemic attack) as coronary "equivalent" and recommends statins for achieving LDL concentrations under 100 mg/dL. In those at increased risk, Atorvastatin, simvastatin 40 mg/day lowered the probability of stroke.

Statins reduce the potential of recurrent strokes of any sort for individuals who experienced an ischemic stroke through lowering the frequency of ischemic stroke. We observed no elevated risk factors for hemorrhagic stroke.

ANTICOAGULANTS:

Unlike with alternative anticoagulant medicines, warfarin is a potent anticoagulant that has been scientifically proven to significantly decrease the probability of stroke. In a meta-analysis, it was found that warfarin minimized the frequency of stroke by 40 percent in comparison to anti-platelet therapies and 64% in comparison to a placebo.

Because vitamin K is necessary for the synthesis of coagulation components, foods high in it can vary in their effectiveness when combined with warfarin. Several green foods, including spinach, broccoli, and sprouts, contain vitamin K. According to studies, consuming 100µg of vitamin K every day for four days in a row can reduce the INR by 0.2. Therefore, it is advised that the patient consume on a frequent basis to accurately calculate their INR and carry out any necessary dose adjustments.

ANTIHYPERTENSIVES:

An independent predictor of morbidity and mortality in ischemic stroke is blood pressure fluctuation that occurs early in the event. The neurologic outcome is adversely affected by both high and low systolic blood pressure. Permissive hypertension is encouraged by current guidelines during the early stages of acute ischemic stroke.

Treatment	Received t-PA	Did Not Receive t-PA
None	<180/105	<220/120
Labetalol IV ^a or Nicardipine IV ^b	180–230/105–120	>220/121–140
Nitroprusside ^c	Diastolic >140	Diastolic >140

t-PA, tissue plasminogen activator.

^aLabetalol IV = 10–20 mg, doubled every 10–20 minutes, to a maximum of 300 mg. Also can use an infusion of 2–8 mg/min.

^bNicardipine IV = infusion starting at 5 mg/h up to 15 mg/h.

^cNitroprusside IV = infusion starting at 0.5 mcg/kg/min, with continuous arterial blood pressure monitoring.

Adapted from Adams HP, del Zoppo G, Alberts MJ, et al. *Stroke* 2007;38:1655–1711.

It is advised that blood pressure be lowered and kept below 185 mm Hg systolic for the first twenty-four hours in patients receiving intravenous thrombolysis for acute ischemic stroke. Sodium nitroprusside, nicardipine, and labetalol continue to be the first-choice medications for decreasing blood pressure. Still, consensus rather than evidence supports these guidelines.

ANTIHYPERGLYCEMIC:

- Blood glucose
- Treat hypoglycemia with D50
- Treat hypoglycemia with insulin if serum glucose >200mg/dl.

NOR-PHARMACOLOGICAL THERAPY:

- Lifestyle modifications: cessation of smoking, alcohol
- Exercise, weight reduction.
- Reducing salt intake by 3g per day lowers blood pressure. The effect is doubled with a 6gm/day reduction and tripped with a 9gm/day reduction in stroke risk parallels reduction in salt intake.

STROKE REHABILITATION:

- Physical therapy: walking, range of movement
- Occupational therapy: Taking care of oneself.
- Speech language therapy: communication skills, swallowing, cognition
- Recreational therapy: cooking, gardening.
- Lifestyle changes for survivors and caregivers:
- Daily living skills
- Dressing and grooming
- Diet, nutrition and eating difficulties.
- Skin care problems.

SCALE: MEDICATION ADHERENCE REPORT SCALE (MARS-5)

Respondents to the MARS-5 medication adherence questionnaire were given a score on how often individuals participate in each of the five behavioral characteristics associated with non-compliance.

QUESTIONS:

- 1) I forget to take them.
- 2) I alter the dose.
- 3) I stop taking them for a while.
- 4) I decide to miss out a dose.
- 5) I take less than instructed.

INTERPRETATION: Each answer is assigned the values 1 to 5 with,

Always (1), Often (2), Sometimes (3), Seldom/Rarely(4), Never(5). Scores are summed up to a scale ranging from 5 to 25, with higher score indicates higher levels of patient compliance to treatment (better adherence). In this we considered that <20 are considered as non-adherence to treatment. Whereas 21-25 is considered as adherence to treatment.

INVESTIGATIONAL STRATEGIES:

REPERFUSION:

Numerous investigations among people with acute ischemic stroke are currently performed with the objective of clearing the obstructed cerebral artery to restore its integrity. Longer-acting fibrinolytic medications, intraarterial fibrinolysis using t-PA along with additional agents, and mechanical and laser-guided endovascular clot removal represent a few of the approaches currently being studied. Furthermore, investigators are working to establish which people might benefit from receiving reperfusion periodically outside of the permitted time range using advanced MRI techniques. There is no doubt that attempts to reperfusion the ischemic brain will be investigated further, providing this therapy accessible to more individuals.

NEUROPROTECTION AND NEURORESTORATION:

While a wide range of neuro protective treatments have been investigated in acute ischemic stroke clinical trials, the majority of these have not proven effective. Agents with several suggested protective

mechanisms, such as minocycline and albumin infusions, are among the most promising. Furthermore, there is optimism that physicians may be able to improve neural restoration—the process by which the brain repairs itself—by employing neural and cell transplantation, targeted neurorehabilitation, and membrane stabilization (citicoline).

Hypothermia is a promising nonpharmacologic approach that has been demonstrated to give patients neuroprotection.

CONCLUSION:

Stroke ranks third internationally in terms of morbidity and is the second leading cause of death worldwide. Non-modifiable and modifiable stroke risk variables are separated out. Transient ischemic attack and family history are frequent examples of non-modifiable factors that are uncontrollable. Moreover, uncontrolled diabetes and hypertension are the two most common modifiable risk factors.

Furthermore, it is thought that obesity, excessive alcohol use, cigarette smoking, and physical inactivity are modifiable risk factors for stroke. By managing or avoiding these modifiable risk factors, the frightening stroke can be reduced or even prevented.

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