

Ischemic and Hemorrhagic Stroke in Young Adults: Etiological Considerations, Risk Factors, and Management Strategies

Meyboll Edily Rodriguez Medina¹, Araceli Martínez Cervantes¹, Jesus Miguel Valencia Correa¹, Carlos Emmanuel Guzman Solorzano¹, Elsa Itzel calderón Tapia¹

¹Hospital Regional Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado. Leon Guanajuato, México. Servicio de medicina interna.

ABSTRACT

Cerebrovascular events in young adults constitute a significant and often underestimated cause of long-term disability and mortality. While ischemic stroke (IS) remains the predominant subtype in this population, hemorrhagic stroke (HS) also represents a considerable burden. The etiological spectrum in younger individuals differs markedly from that in the elderly, encompassing non-traditional risk factors such as arterial dissection, prothrombotic states, cardiac embolism due to structural anomalies (e.g., patent foramen ovale), vasculitis, and substance abuse. Genetic predisposition and autoimmune conditions further complicate the diagnostic and therapeutic landscape.

This review provides an in-depth analysis of the pathophysiological mechanisms underlying stroke in young adults, highlighting the distinct clinical presentations, diagnostic modalities, and therapeutic options. The role of advanced neuroimaging, thrombophilia screening, and cardiac workup is emphasized in optimizing diagnostic accuracy. Additionally, we discuss the implications of early intervention strategies, including thrombolysis and endovascular therapy, alongside secondary prevention approaches tailored to this demographic. A comprehensive understanding of the unique aspects of stroke in young adults is imperative for improving clinical outcomes and reducing long-term disability in this patient population.

KEYWORDS: Young stroke, ischemic stroke, hemorrhagic stroke, arterial dissection, cardioembolism, thrombophilia, vasculitis, cerebrovascular disease, stroke risk factors, endovascular therapy

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INTRODUCTION

Stroke in young adults, defined as a cerebrovascular event occurring in individuals under the age of 50, is an emerging public health concern with increasing incidence worldwide. Unlike stroke in older populations, which is predominantly linked to atherosclerotic disease and conventional vascular risk factors such as hypertension, diabetes, and hyperlipidemia, young-onset stroke presents a unique and diverse etiological profile. The presence of non-traditional risk factors, including arterial dissection, genetic thrombophilic disorders, autoimmune diseases, and cardioembolic sources, underscores the complexity of diagnosis and management in this age group.^{1,2}

The distinction between ischemic and hemorrhagic stroke in young adults is crucial, as the underlying mechanisms, prognostic implications, and therapeutic approaches differ significantly. Ischemic stroke in the young is frequently attributed to paradoxical embolism via a patent foramen ovale, hypercoagulable states, or arterial dissection secondary to trauma or spontaneous intimal tears. Conversely, hemorrhagic stroke often results from intracranial aneurysms, arteriovenous malformations, or coagulopathies, necessitating a distinct therapeutic approach.^{2,3}

Given the long life expectancy of young stroke survivors, the socioeconomic impact of stroke in this demographic is profound, with substantial implications for quality of life, long-term morbidity, and reintegration into daily activities.

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Early recognition of atypical stroke presentations, comprehensive diagnostic workup, and individualized management strategies are paramount in mitigating disability and optimizing functional recovery.^{4,5}

This article aims to provide a thorough review of the epidemiology, risk factors, pathophysiology, and management strategies for cerebrovascular events in young adults. By delineating the latest advancements in stroke diagnostics, acute interventions, and secondary prevention strategies, we aim to enhance the understanding and clinical approach to this increasingly recognized entity in vascular neurology.⁴

EPIDEMIOLOGY

Stroke in young adults, typically defined as a cerebrovascular event occurring in individuals younger than 50 years, represents a significant yet often overlooked subset of stroke epidemiology. Traditionally considered a disease of the elderly, the incidence of stroke in young adults has been increasing in recent decades, posing substantial challenges to public health systems due to its long-term socioeconomic and functional consequences. While stroke remains less common in younger individuals compared to the elderly, its impact is disproportionately high, as it affects individuals in their most productive years, leading to a considerable burden of disability, dependency, and reduced quality of life.⁵

Incidence and Prevalence

Epidemiological studies have demonstrated that stroke in young adults accounts for approximately 10–15% of all stroke cases worldwide. However, regional variations exist, with a higher prevalence in low- and middle-income countries, where the incidence is increasing more rapidly than in high-income nations. The estimated annual incidence ranges from 7 to 15 per 100,000 individuals aged 18–50 years, though this varies based on geographic, ethnic, and socioeconomic factors.⁵

The proportion of ischemic stroke (IS) versus hemorrhagic stroke (HS) differs across populations. Ischemic stroke constitutes 70–80% of cases, while hemorrhagic stroke—including both intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH)—accounts for 20–30%. Notably, in contrast to older adults, where large-artery atherosclerosis is the dominant mechanism, strokes in younger patients more frequently result from non-atherosclerotic causes, such as arterial dissection, cardioembolism, and hypercoagulable states.⁵

A sex disparity has been observed in young stroke patients, with men experiencing a slightly higher overall incidence of stroke than women in most population-based studies. However, sex-specific risk factors play a pivotal role in the epidemiology of young-onset stroke. Women of reproductive age exhibit a higher relative risk of stroke during pregnancy, the puerperium, and in the context of hormonal contraceptive use or hormone replacement therapy. Additionally,

conditions such as migraine with aura, which is more prevalent in women, have been linked to an increased stroke risk, particularly in conjunction with smoking or oral contraceptive use.⁵

Trends and Risk Factor Evolution

Over the past several decades, there has been a concerning rise in the incidence of stroke among younger individuals, attributed to a combination of lifestyle-related factors, increasing prevalence of traditional cardiovascular risk factors, and improved stroke recognition and diagnostic capabilities. Factors contributing to this trend include:

- Rising prevalence of obesity and metabolic syndrome: Young adults are increasingly affected by obesity, insulin resistance, type 2 diabetes mellitus (T2DM), and hypertension—conditions historically more common in the elderly but now recognized as key contributors to early cerebrovascular disease.⁵
- Increased detection of cryptogenic stroke: Advances in neuroimaging and cardiac diagnostics, including prolonged cardiac monitoring, transesophageal echocardiography, and thrombophilia screening, have led to a greater recognition of previously undiagnosed stroke etiologies, such as paroxysmal atrial fibrillation, patent foramen ovale (PFO), and inherited hypercoagulable states.⁶
- Substance use and lifestyle factors: The consumption of cocaine, amphetamines, cannabis, and other recreational drugs has been associated with both ischemic and hemorrhagic stroke in young adults. These substances can induce vasospasm, endothelial dysfunction, arterial dissection, and hypertensive surges, all of which contribute to cerebrovascular pathology.⁶
- Increased awareness and access to healthcare: Greater public awareness, expanded use of emergency stroke pathways, and improved acute stroke care, including thrombolysis and mechanical thrombectomy, have enhanced stroke detection and management, leading to better outcomes but also a perceived rise in cases.⁶

Ethnic and Geographic Variability

The incidence of young-onset stroke demonstrates significant ethnic and geographical variation. Studies indicate that stroke occurs at a younger age and with higher prevalence among individuals of African, Hispanic, and South Asian descent compared to Caucasian populations. The reasons for this disparity include higher rates of hypertension, sickle cell disease, and metabolic syndrome, as well as disparities in healthcare access and preventive measures.⁷

In high-income countries, the increasing burden of ischemic stroke in young adults has been largely linked to modifiable risk factors such as smoking, physical inactivity, and poor dietary habits, whereas in lower-income regions, infections

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and non-communicable diseases (e.g., rheumatic heart disease, tuberculosis-associated vasculitis, HIV-related vasculopathy) play a more prominent role in stroke pathogenesis.⁷

Mortality and Long-Term Outcomes

Despite improvements in acute stroke care, young stroke patients experience significant long-term morbidity and mortality. While case-fatality rates in young adults are generally lower than in older populations—largely due to better cerebral plasticity and fewer preexisting comorbidities—stroke remains a leading cause of acquired disability. Studies estimate that up to 30–40% of young stroke survivors suffer from persistent neurological deficits, and many face difficulties returning to employment or independent living.⁸

The risk of recurrent stroke in young adults is another major concern. Data suggest that approximately 15–20% of young stroke survivors experience recurrent stroke within five years, particularly in those with underlying vasculopathy, untreated atrial fibrillation, or inadequately managed risk factors. Long-term secondary prevention strategies, including anticoagulation in cardioembolic stroke, aggressive lipid management, and lifestyle modifications, are essential to mitigate recurrent events.⁸

The epidemiology of stroke in young adults reveals an evolving landscape characterized by increasing incidence, a shifting profile of risk factors, and significant regional disparities. Unlike stroke in the elderly, where traditional atherosclerotic mechanisms predominate, young-onset stroke is frequently driven by heterogeneous and often non-traditional causes, necessitating a tailored approach to diagnosis, prevention, and management. Given the profound personal, social, and economic consequences of stroke at a young age, further research is required to elucidate the underlying mechanisms, improve early detection, and optimize long-term outcomes for this unique patient population.⁸

Clinical Manifestations of Stroke in Young Adults

The clinical presentation of stroke in young adults is highly variable, influenced by multiple factors including the underlying etiology, the specific vascular territory involved, and the presence of comorbid conditions. Compared to older individuals, young stroke patients are more likely to present with non-traditional symptoms, experience delayed recognition, and have atypical stroke syndromes due to the heterogeneous nature of stroke mechanisms in this population.⁹

While the classic sudden-onset neurological deficits, such as hemiparesis, aphasia, and visual disturbances, remain the hallmark of acute cerebrovascular events, young-onset stroke frequently exhibits subtle, fluctuating, or non-specific symptoms, which can lead to misdiagnosis, particularly in emergency settings. Furthermore, because of their generally better cerebrovascular reserve and fewer pre-existing

comorbidities, young stroke patients may experience partial symptom recovery in the hyperacute phase, further complicating timely diagnosis.⁹

Common Neurological Presentations

The clinical manifestations of stroke are primarily determined by the affected vascular territory, which dictates the specific neurological deficits observed.⁹

Ischemic Stroke Syndromes

Ischemic stroke (IS) is the predominant stroke subtype in young adults, often caused by arterial dissection, cardioembolism, vasculitis, hypercoagulable states, or paradoxical embolism. The neurological deficits correspond to the occluded artery:

- Middle Cerebral Artery (MCA) Infarction (Most Common Presentation)
 - Contralateral hemiparesis and hemisensory loss (face and upper extremity more affected than lower extremity)
 - Aphasia (if dominant hemisphere is involved)
 - Hemineglect or anosognosia (if non-dominant hemisphere is affected)
 - Homonymous hemianopia
 - Gaze deviation toward the side of the infarct
- Anterior Cerebral Artery (ACA) Infarction
 - Contralateral lower limb weakness and sensory loss
 - Abulia, apathy, or akinetic mutism
 - Frontal release signs (grasp reflex, perseveration)
- Posterior Cerebral Artery (PCA) Infarction
 - Homonymous hemianopia with macular sparing
 - Cortical blindness (if bilateral PCA involvement)
 - Thalamic syndromes (contralateral sensory loss, pain syndromes)
- Vertebrobasilar Stroke (Posterior Circulation Syndromes)
 - Dizziness, vertigo, ataxia
 - Dysarthria, dysphagia, Horner's syndrome
 - Crossed sensory/motor deficits (ipsilateral cranial nerve palsies with contralateral body weakness)
 - Locked-in syndrome (bilateral pontine infarcts affecting corticospinal and corticobulbar tracts)
- Lacunar Syndromes (Small Vessel Disease or Embolism)
 - Pure motor stroke (internal capsule infarction)
 - Pure sensory stroke (thalamic infarction)
 - Dysarthria-clumsy hand syndrome

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- Ataxic hemiparesis

Hemorrhagic Stroke Syndromes

Hemorrhagic strokes in young adults, including intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH), tend to have more severe initial presentations, with a higher risk of coma, increased intracranial pressure (ICP), and acute deterioration.

- Intracerebral Hemorrhage (ICH)
 - Abrupt onset of focal neurological deficits
 - Severe headache, nausea, vomiting (due to raised ICP)
 - Rapid progression to altered consciousness (particularly with deep hemorrhages involving the basal ganglia, thalamus, or brainstem)
 - Seizures (more common in lobar hemorrhages)
 - Hypertensive emergency findings (e.g., malignant hypertension, encephalopathy)
- Subarachnoid Hemorrhage (SAH) (Most Commonly Due to Aneurysmal Rupture)
 - Thunderclap headache (“worst headache of my life”)
 - Meningeal signs (neck stiffness, photophobia, nausea, vomiting)
 - Transient loss of consciousness
 - Third nerve palsy (if posterior communicating artery aneurysm rupture)
 - Sudden focal deficits if parenchymal extension occurs

Atypical Presentations and Stroke Mimics

Young adults with stroke frequently present with non-traditional symptoms, leading to initial misdiagnoses such as migraine, seizure, functional disorders, or vestibular syndromes. Some atypical presentations include:

- Isolated cognitive impairment (stroke in the medial frontal or parietal lobes)
- Fluctuating or progressive neurological deficits (suggesting arterial dissection or vasculitis)
- Headache-dominant presentations (commonly seen in dissection-related stroke, vasculitis, and SAH)
- Seizures as the initial manifestation (higher incidence in young stroke patients, particularly those with cortical infarctions)
- Transient ischemic attack (TIA)-like symptoms (commonly associated with paradoxical embolism, PFO, or cardioembolic sources)

In many cases, arterial dissection—a leading cause of ischemic stroke in young adults—presents with neck pain, headache, and Horner’s syndrome, preceding the neurological deficits by hours or days. Similarly, vasculitic and autoimmune-related strokes often manifest with relapsing-remitting neurological symptoms, mimicking multiple sclerosis or other inflammatory conditions.¹⁰

Systemic and Non-Neurological Features

Since many young-onset strokes are secondary to systemic diseases, extracranial symptoms can provide critical diagnostic clues:

- Fever, weight loss, and arthralgias → Suggests vasculitis, systemic lupus erythematosus (SLE), or endocarditis
- Palpitations, exertional dyspnea, or syncope → Suggests cardioembolic sources (e.g., atrial fibrillation, cardiomyopathy, PFO)
- Livedo reticularis, Raynaud’s phenomenon → Associated with antiphospholipid syndrome and hypercoagulability
- Unexplained deep vein thrombosis (DVT) or pulmonary embolism → Raises suspicion for paradoxical embolism in the setting of PFO

The clinical manifestations of stroke in young adults encompass a broad spectrum of neurological and systemic symptoms, often differing from the classical stroke presentation seen in older populations. The higher frequency of non-traditional risk factors, the atypical and fluctuating presentation of symptoms, and the frequent overlap with stroke mimics pose significant diagnostic challenges. Early recognition of these unique features is crucial to prevent delays in acute management, optimize neuroprotection, and improve long-term outcomes.¹⁰

Diagnostic Methods for Stroke in Young Adults

The evaluation of stroke in young adults necessitates a comprehensive and systematic approach due to the heterogeneous etiological spectrum and atypical presentations often encountered in this population. Unlike older patients, in whom atherosclerosis, hypertension, and atrial fibrillation predominate as causative factors, young adults frequently present with stroke secondary to arterial dissection, cardioembolism, vasculitis, hypercoagulability, genetic disorders, or paradoxical embolism. Consequently, a multimodal diagnostic workup integrating neuroimaging, vascular studies, cardiac evaluation, and specialized laboratory testing is essential for accurate etiological classification and secondary prevention strategies.¹⁰

Neuroimaging Modalities

Neuroimaging constitutes the cornerstone of acute stroke diagnosis, providing crucial information regarding infarct location, vascular involvement, and potential underlying mechanisms.¹⁰

Non-Contrast Computed Tomography (NCCT) of the Brain NCCT is the first-line imaging modality in suspected acute stroke, primarily to exclude hemorrhage and identify early ischemic changes. While hyperacute ischemic infarcts may not be readily apparent, certain key findings suggest early infarction:¹¹

- Loss of gray-white matter differentiation in the insular ribbon and basal ganglia
- Cortical sulcal effacement due to cytotoxic edema

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- Hyperdense artery sign, indicative of intraluminal thrombus

However, NCCT has limited sensitivity for detecting small cortical or posterior fossa infarcts in young patients, necessitating further imaging.¹²

Computed Tomography Angiography (CTA) and CT Perfusion (CTP)

- CTA provides rapid, high-resolution vascular imaging to evaluate arterial occlusions, dissections, atherosclerotic plaques, and stenotic lesions. It is particularly useful in detecting:
 - Carotid and vertebral artery dissection, a major cause of stroke in young adults
 - Intracranial large vessel occlusion (LVO), guiding endovascular treatment decisions
 - Intracranial vasculopathy, including vasculitis, reversible cerebral vasoconstriction syndrome (RCVS), and moyamoya disease
- CTP assesses cerebral perfusion dynamics, helping differentiate ischemic core from salvageable penumbra, which is critical in thrombolytic and endovascular treatment decisions.¹³

Magnetic Resonance Imaging (MRI) and Magnetic Resonance Angiography (MRA)

MRI is superior to CT in detecting early infarcts, particularly in the posterior circulation and small cortical or subcortical infarctions.¹³

- Diffusion-Weighted Imaging (DWI) is the most sensitive sequence for acute ischemia, revealing cytotoxic edema within minutes of stroke onset.¹³
- Apparent Diffusion Coefficient (ADC) Mapping helps differentiate acute infarction (restricted diffusion) from chronic ischemic changes or stroke mimics.¹³
- Susceptibility-Weighted Imaging (SWI) detects microbleeds, hemorrhagic transformation, and vascular malformations, aiding in hemorrhagic stroke evaluation.¹³
- MRA (Time-of-Flight or Contrast-Enhanced) provides detailed arterial anatomy, detecting stenosis, dissection, and vasculopathy.¹³
- MR Venography (MRV) is indicated when cerebral venous sinus thrombosis (CVST) is suspected, particularly in young women with hypercoagulable states.¹⁴

Vascular Studies

Since young-onset stroke is frequently related to arterial dissection, vasculopathy, or embolic mechanisms, a thorough vascular assessment is imperative.

Carotid and Vertebral Duplex Ultrasonography

- Evaluates extracranial arterial pathology, detecting:
 - Arterial dissection, characterized by an intimal flap or double lumen

- Atherosclerotic plaques, though less common in young patients
- High-risk embolic sources, such as mobile thrombi

- Transcranial Doppler (TCD) assists in identifying:
 - Intracranial stenosis or vasospasm
 - Microembolic signals, suggesting cardioembolic or paradoxical embolism
 - Right-to-left shunt detection (Bubble Study for PFO Assessment)

Digital Subtraction Angiography (DSA)

As the gold standard for cerebrovascular imaging, DSA is reserved for cases with suspected:

- Dissection or vasculitis requiring definitive confirmation
- Moyamoya disease or RCVS, to assess collateral circulation
- Embolic stroke of undetermined source (ESUS), to identify occult vascular abnormalities

Cardiac Evaluation

Cardioembolism is a leading etiology in young stroke patients, necessitating detailed cardiac workup to rule out atrial fibrillation, intracardiac thrombi, structural defects, or valvular pathology.¹⁴

Electrocardiography (ECG) and Holter Monitoring

- Detects atrial fibrillation, atrial flutter, or paroxysmal supraventricular tachycardia, common embolic sources.¹⁴
- Prolonged monitoring (24-72 hours or implantable loop recorders) is indicated in cryptogenic strokes.¹⁴

Transthoracic Echocardiography (TTE) and Transesophageal Echocardiography (TEE)

- TTE evaluates left ventricular function, valvular disease, and left atrial appendage thrombi.
- TEE is superior for detecting:
 - Patent Foramen Ovale (PFO) with Bubble Study (paradoxical embolism)
 - Aortic arch atherosclerosis
 - Endocarditis or cardiac masses

Hematologic and Laboratory Testing

Young patients require extensive laboratory workup to identify prothrombotic conditions, metabolic disorders, and systemic diseases contributing to stroke.¹⁴

Hypercoagulability Panel

- Antiphospholipid antibodies (Lupus anticoagulant, anticardiolipin, β_2 -glycoprotein-I)
- Factor V Leiden mutation, Prothrombin gene mutation
- Protein C, Protein S, Antithrombin III levels
- Homocysteine levels

Vasculitis and Autoimmune Panel

- Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) (inflammatory markers)

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- Antinuclear antibodies (ANA), Rheumatoid factor (RF), Complement levels
- Antineutrophil cytoplasmic antibodies (ANCA) for vasculitis

Infectious and Metabolic Testing

- HIV, Syphilis (RPR, FTA-ABS), Hepatitis panel (infectious stroke causes)
- Thyroid function tests (hyperthyroidism-induced embolism)
- Toxicology screen (cocaine, amphetamines, methamphetamine-related vasculopathy)

Genetic and Advanced Biomarker Testing

For suspected genetic stroke syndromes, targeted testing includes:

- Mitochondrial disorders (MELAS - Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like Episodes)
- CADASIL (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy)
- Fabry disease (α -galactosidase deficiency, lysosomal storage disorder-related stroke)

Biomarkers such as D-dimer (for hypercoagulability) and BNP (cardioembolic stroke marker) are also under investigation.¹⁴

The diagnostic evaluation of stroke in young adults requires a multidisciplinary, stepwise approach incorporating advanced neuroimaging, vascular studies, cardiac evaluation, and laboratory testing to identify rare but treatable causes. Given the high prevalence of non-traditional risk factors, early and accurate diagnosis is essential to prevent recurrent events and guide optimal management strategies.¹⁵

Therapeutic Approaches for Stroke in Young Adults

The management of stroke in young adults necessitates a multifaceted therapeutic approach encompassing acute revascularization strategies, secondary prevention measures, and tailored interventions based on the underlying etiology. Given the diverse and often non-traditional risk factors contributing to stroke in this population—such as arterial dissection, cardioembolism, hypercoagulability, vasculitis, and genetic disorders—a personalized, etiology-driven treatment paradigm is essential. The therapeutic strategies for young stroke patients can be broadly categorized into acute phase management, secondary prevention, and rehabilitation strategies.

Acute Stroke Management

The primary objective in the acute phase of ischemic stroke is rapid revascularization to restore cerebral perfusion and minimize neuronal damage. The therapeutic armamentarium includes intravenous thrombolysis, endovascular therapy, and supportive neurocritical care measures.¹⁵

Intravenous Thrombolysis (IVT)

- Alteplase (Recombinant Tissue Plasminogen Activator, rtPA)
 - Standard dosing: 0.9 mg/kg (maximum 90 mg), with 10% as a bolus and the remainder infused over 60 minutes.¹⁵
 - Indicated for patients presenting within 4.5 hours of symptom onset, provided they meet eligibility criteria.¹⁵
 - Caution in arterial dissection, as its safety remains controversial in this subgroup.¹⁵
 - Thrombophilia screening is not necessary before IVT, except in patients with known antiphospholipid syndrome, where an increased bleeding risk exists.¹⁵
- Tenecteplase (TNK-tPA)
 - Emerging as a potential alternative due to its longer half-life and ease of administration (single bolus).¹⁵
 - Evidence suggests non-inferiority to alteplase, with potential advantages in large vessel occlusion (LVO) settings.¹⁵

Endovascular Thrombectomy (EVT)

- Indicated for large vessel occlusions (LVOs) of the anterior circulation (ICA, M1 segment of MCA) within 6 hours.¹⁵
- Extended window (6-24 hours) based on imaging criteria (DAWN, DEFUSE-3 trials).¹⁵
- Particularly beneficial in young patients, who have greater collateral circulation and higher rates of favorable functional outcomes.¹⁵
- Aspiration thrombectomy and stent retrievers remain the standard approaches.¹⁵

Special Considerations for Uncommon Stroke Etiologies

- Arterial Dissection: EVT is preferred over IVT if there is an associated thrombus.
- Cerebral Venous Sinus Thrombosis (CVST): Treated with systemic anticoagulation, with thrombectomy reserved for severe cases.¹⁵
- Moyamoya Disease: Revascularization surgery (e.g., direct or indirect bypass) is required in recurrent ischemic events.¹⁵
- Hypercoagulability-Related Stroke: Anticoagulation therapy (heparin → warfarin or DOACs) is prioritized in antiphospholipid syndrome, malignancy-associated thrombosis, or inherited thrombophilias.¹⁶

Secondary Prevention Strategies

Antithrombotic Therapy

- Antiplatelet Therapy
 - Aspirin (81-325 mg/day) is the cornerstone for non-cardioembolic stroke prevention.¹⁶

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- Clopidogrel (75 mg/day) or aspirin-dipyridamole combination may be alternatives.¹⁶
- Dual antiplatelet therapy (DAPT) (aspirin + clopidogrel for 21-90 days) is used in high-risk TIA, minor stroke, or intracranial atherosclerosis.¹⁶
- Anticoagulation Therapy
 - Indicated for cardioembolic stroke (e.g., atrial fibrillation, left atrial thrombus, mechanical valves, PFO with embolism, hypercoagulable states).¹⁶
 - Direct Oral Anticoagulants (DOACs) (e.g., apixaban, rivaroxaban, dabigatran) preferred over warfarin for non-valvular AF.¹⁶
 - Low Molecular Weight Heparin (LMWH) → Warfarin is required for antiphospholipid syndrome.¹⁶

Patent Foramen Ovale (PFO) Closure

- Young patients with cryptogenic stroke and a high-risk PFO (large shunt, atrial septal aneurysm) benefit from percutaneous closure.¹⁶
- Reduces recurrent stroke risk by ~45-50% compared to medical therapy alone.¹⁶

Risk Factor Modification

- Hypertension: Target BP <130/80 mmHg (ACE inhibitors, ARBs, thiazides).
- Dyslipidemia: High-intensity statins (atorvastatin 40-80 mg, rosuvastatin 20-40 mg).¹⁶
- Diabetes Mellitus: HbA1c goal <7%, SGLT2 inhibitors, GLP-1 receptor agonists.
- Lifestyle modifications: Smoking cessation, weight loss, Mediterranean diet.¹⁶

Rehabilitation and Long-Term Recovery

Neurorehabilitation

- Early mobilization and physical therapy improve functional outcomes.
- Constraint-induced movement therapy, mirror therapy, and robotic-assisted rehabilitation benefit motor recovery.¹⁶
- Cognitive therapy and neuropsychological support are crucial for post-stroke depression, aphasia, and executive dysfunction.¹⁶

Neuromodulation Therapies

- Repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS) are being explored for motor and speech rehabilitation.¹⁶

Surgical and Interventional Strategies

- Decompressive hemicraniectomy is lifesaving in malignant middle cerebral artery infarction.¹⁷
- Carotid endarterectomy (CEA) or stenting may be required in symptomatic carotid stenosis >50%.¹⁷

The therapeutic landscape for stroke in young adults is characterized by a diverse range of treatment strategies, requiring a precision medicine approach that integrates acute revascularization, tailored secondary prevention, and personalized rehabilitation interventions. Given the unique stroke etiologies in this population, multidisciplinary collaboration among neurologists, cardiologists, hematologists, and rehabilitation specialists is essential to optimize long-term outcomes.¹⁷

CONCLUSION

Stroke in young adults represents a distinct clinical entity characterized by aetiological heterogeneity, unique risk factor profiles, and diverse prognostic implications. Unlike stroke in the elderly, which is predominantly driven by atherosclerosis and atrial fibrillation, young-onset cerebrovascular events frequently stem from non-traditional mechanisms such as arterial dissection, cardioembolism due to structural heart disease, prothrombotic states, vasculitis, and genetic syndromes. The atypical presentation and lower index of suspicion in this age group often contribute to delayed diagnosis, underscoring the necessity for heightened clinical vigilance and an expeditious, systematic diagnostic approach.

The management of acute ischemic stroke (AIS) in young patients aligns with conventional thrombolytic and endovascular strategies; however, individualized therapeutic considerations are paramount, particularly in cases involving dissection, hypercoagulability, or PFO-associated embolism. While intravenous thrombolysis (IVT) and mechanical thrombectomy (MT) remain the cornerstone of acute revascularization therapy, adjunctive interventions—such as PFO closure in cryptogenic stroke, anticoagulation for thrombophilic states, and surgical revascularization in moyamoya disease—necessitate a personalized, pathophysiology-driven treatment paradigm. Secondary prevention strategies must be tailored to the underlying etiology, incorporating targeted antithrombotic therapy, optimal cardiovascular risk management, and lifestyle modification strategies.

From a prognostic standpoint, young stroke patients generally exhibit better functional recovery compared to older populations due to greater neuroplasticity and lower burden of chronic vascular pathology. However, long-term morbidity remains significant, with a substantial proportion experiencing recurrent strokes, post-stroke cognitive impairment, epilepsy, and psychiatric sequelae such as depression and anxiety disorders. The integration of early, intensive neurorehabilitation, including robotic-assisted rehabilitation, neurostimulation techniques, and cognitive therapy, is pivotal in maximizing neurological recovery and enhancing quality of life.

Future research must focus on advancing precision medicine approaches, particularly through the identification of novel

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biomarkers, genetic predispositions, and individualized therapeutic targets. Furthermore, the advent of machine learning algorithms and artificial intelligence-driven risk stratification tools holds promise for improving early detection and optimizing treatment paradigms. Given the significant socioeconomic impact of stroke in young adults, public health initiatives must emphasize primary prevention, early recognition, and timely intervention to mitigate long-term disability and optimize functional outcomes.

In conclusion, young-onset stroke necessitates a multidisciplinary, comprehensive, and patient-centric approach that extends beyond conventional stroke management algorithms. By refining diagnostic precision, optimizing therapeutic interventions, and enhancing long-term rehabilitation strategies, clinicians can significantly improve the prognosis and quality of life of affected individuals. The continued evolution of personalized medicine, advanced neuroimaging modalities, and novel therapeutic innovations will play a pivotal role in shaping the future landscape of stroke care in young adults.

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