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ISHEMIC STROKE

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Abstract:
Ischemic stroke is an acute neurological syndrome caused by the sudden interruption of cerebral blood flow due to thrombus formation or atherosclerotic occlusion of intracranial vessels. This article reviews the pathogenesis, key clinical manifestations, diagnostic modalities, and stepwise treatment protocols for ischemic stroke. Timely recognition and appropriate management significantly reduce mortality and long-term disability.

Keywords: ischemic stroke, cerebrovascular disease, thrombosis, neuroprotection, thrombolytic therapy

INTRODUCTION

Ischemic stroke occurs when a thrombus or embolus blocks a cerebral artery, leading to acute interruption of oxygen and nutrient delivery to brain tissue. In many patients, minor warning signs (prodromal symptoms) such as transient weakness or numbness may precede a full-blown event; however, once a major vessel is completely occluded, neurological deficits appear suddenly. According to the World Health Organization, approximately 85 percent of all stroke cases worldwide each year are of the ischemic type [1].

PATHOGENESIS AND ETIOLOGY

Ischemic stroke arises through one or more of the following mechanisms:

1. Thrombosis: Formation of a clot on an atherosclerotic plaque within a large or small cerebral artery, leading to local arterial occlusion.
2. Embolism: Migration of a clot (often from the heart, for example in atrial fibrillation) to a cerebral vessel, abruptly blocking flow.
3. Hemodynamic compromise: Systemic hypotension or severe cardiac output reduction causing global-widespread cerebral hypoperfusion.

Major risk factors include hypertension, diabetes mellitus, cardiac arrhythmias (particularly atrial fibrillation), hyperlipidemia, smoking, obesity, and a sedentary lifestyle

CLINICAL FEATURES

The clinical presentation of ischemic stroke depends on the vascular territory affected. Common signs and symptoms include:

Facial paralysis (often affecting one side of the face)
Arm and/or leg weakness or paralysis (usually hemiparesis or hemiplegia)
Visual disturbances (hemianopsia or monocular vision loss)
Sudden, severe headache
Speech impairment (aphasia or dysarthria)
Vertigo or loss of balance
Excessive somnolence or insomnia
Sudden onset of generalized weakness, possibly with syncope

Early recognition—ideally within the first 4.5 hours of symptom onset—is critical, as it directly influences treatment options and outcomes.

DIAGNOSTICS

Rapid, accurate diagnosis is essential to distinguish ischemic from hemorrhagic stroke and to guide acute therapy. Recommended investigations include:

1. Noncontrast Computed Tomography (CT): First-line imaging to exclude intracranial hemorrhage.
2. Magnetic Resonance Imaging (MRI) with Diffusion-Weighted Imaging (DWI): Highly sensitive for detecting acute ischemic changes within minutes of onset.
3. Carotid and Transcranial Doppler Ultrasound (TCD): Evaluates flow in carotid and vertebral arteries to identify stenosis or occlusion.
4. Cerebral Angiography (Digital Subtraction Angiography, DSA): Gold standard for defining the exact location and extent of arterial occlusion (used when mechanical thrombectomy is considered).
5. Electrocardiogram (ECG) and Echocardiography (EchoCG): Assess cardiac rhythm, left ventricular function, and identify potential cardioembolic sources.
6. Laboratory Tests: Complete blood count, coagulation profile, blood glucose, lipid panel, renal function tests, and inflammatory markers to identify modifiable risk factors and rule out mimics of stroke.

TREATMENT APPROACHES

Time is brain: the sooner reperfusion and neuroprotective strategies begin, the better the prognosis. Management can be divided into the following phases:

1. Neuroprotective and Decongestive Therapy

Mannitol 200 mL IV + Furosemide 4 mL IV

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– Reduces intracranial pressure in cases of cerebral edema.
Urinary catheterization to monitor output and fluid balance.
2. Antioxidant and Metabolic Agents
Xavron 20 mL + 0.9% NaCl 100 mL IV
– Scavenges free radicals, reducing reperfusion injury.
L-Lysine Essinate 10 mL + 0.9% NaCl 100 mL IV
– Improves microcirculation and protects endothelial function.
Citicoline 1000 mg/4 mL + 0.9% NaCl 100 mL IV (alternatively, Cerebrolysin)
– Promotes neuronal membrane repair and supports neurotransmitter synthesis.
Meldonium (Mildronate) 10 mL IV
– Optimizes mitochondrial function and cerebral energy metabolism.
3. Antithrombotic Therapy
Heparin 5000 IU IV every 6 hours (× 4 doses)
– Immediate anticoagulation in selected patients at high risk for early recurrent thrombosis.
Pentoxifylline (Trental) 5 mL + 0.9% NaCl 100 mL IV
– Improves blood rheology and increases erythrocyte flexibility.
Aspirin 100 mg PO once daily
– Initiated after CT excludes hemorrhage; inhibits platelet aggregation.
Atorvastatin (Atoris) 20 – 40 mg PO once daily
– Lowers cholesterol and stabilizes atherosclerotic plaques.
4. Supportive Measures and Adjuncts
Vitamin B1 (Thiamine) + Vitamin B6 (Pyridoxine): Supports neuronal health.
Oxygen therapy: Maintains adequate oxygen saturation (target $SpO_2 \geq 94$ %).
> Note: These medications are not administered simultaneously in every case. Individualization of the regimen depends on each patient's clinical condition, comorbidities, and risk profile.

COMPLICATIONS AND REHABILITATION

After an acute ischemic stroke, potential complications include:
Persistent motor deficits (e.g., hemiplegia or spasticity)
Speech and swallowing disorders (aphasia, dysphagia)
Depression and cognitive impairment
Post-stroke seizures
Pressure ulcers in bedridden patients
Early, multidisciplinary rehabilitation is essential and should begin as soon as the patient is medically stable.
Key components of rehab include:
Physiotherapy: To restore gross motor function and prevent joint contractures.
Occupational therapy: To improve activities of daily living (ADLs) and fine motor skills.
Speech therapy: To address aphasia and dysphagia.
Psychological support: To manage mood disorders and encourage social reintegration.

CONCLUSION

Ischemic stroke remains a leading cause of death and disability worldwide. Prompt recognition of early warning signs, rapid neuroimaging, and timely initiation of reperfusion-based and neuroprotective therapies can dramatically improve outcomes. Comprehensive post-stroke rehabilitation is equally vital to maximize functional recovery and reduce the socioeconomic burden. Clinicians must maintain high vigilance and adhere to evidence-based protocols to optimize patient care.

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