

ISCHEMIC STROKE: CONTEMPORARY INSIGHTS INTO PATHOGENESIS, DIAGNOSIS, AND MANAGEMENT

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Abstract

Ischemic stroke is a major contributor to mortality and long-term disability across the globe. It develops when cerebral blood flow is reduced or completely obstructed, leading to tissue ischemia and neuronal death. This article presents an overview of causes, pathophysiology, clinical features, diagnosis, and treatment approaches.

Keywords: ischemic stroke, cerebral infarction, thrombosis, embolism, reperfusion therapy, neurology

Introduction

Ischemic stroke, or cerebral infarction, occurs due to a sudden focal interruption of blood supply to the brain, leading to irreversible neuronal loss and neurological deficits. Accounting for approximately 85% of all stroke cases, its management is a race against time.

Etiology and Classification

According to the TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification, ischemic stroke is categorized into five subtypes:

1. Large-artery atherosclerosis: Plaque rupture or stenosis in major cerebral arteries.
2. Cardioembolism: Often associated with atrial fibrillation, where a thrombus migrates from the heart to the brain.
3. Small-vessel occlusion (Lacunar stroke): Infarcts in the deep penetrating arteries.
4. Stroke of other determined etiology: Such as vasculitis, arterial dissection, or hypercoagulable states.
5. Stroke of undetermined etiology (Cryptogenic): Cases where no definitive cause is found despite evaluation.

Pathophysiological Mechanisms

Reduced cerebral blood flow leads to ATP depletion, ion imbalance, calcium influx, and neuronal damage. The affected area includes the ischemic core and penumbra.

The cessation of blood flow triggers a complex biochemical sequence known as the ischemic cascade:

- Energy Failure: Depletion of ATP leads to the failure of ion pumps (Na⁺/K⁺-ATPase).
- Excitotoxicity: Massive release of glutamate results in excessive calcium (Ca²⁺) influx into neurons.
- Oxidative Stress: Generation of reactive oxygen species (ROS) causes membrane lipid peroxidation.
- Inflammation and Apoptosis: Subsequent microglial activation and programmed cell death further expand the lesion.

Clinical Presentation

Symptoms include unilateral weakness, speech disturbances, visual impairment, coordination loss, and altered consciousness.

Diagnostic Approaches

CT and MRI are primary imaging techniques. Additional tests include blood analysis, ECG, and Doppler ultrasound.

Modern diagnostics focus on rapid differentiation between ischemic and hemorrhagic stroke. Key modalities include Non-contrast Computed Tomography (NCCT) to exclude hemorrhage,



and Magnetic Resonance Imaging (MRI), specifically Diffusion-Weighted Imaging (DWI), which is highly sensitive for detecting hyperacute ischemia. CT Angiography (CTA) is essential for identifying Large Vessel Occlusion (LVO).

Treatment and Management

Acute treatment includes thrombolysis and thrombectomy. Supportive care and secondary prevention are essential.

1. Intravenous Thrombolysis (IVT): Administration of Alteplase (rtPA) or Tenecteplase within a 4.5-hour window. 2. Mechanical Thrombectomy (MT): Endovascular removal of clots in patients with LVO, effective up to 6–24 hours in selected patients. 3. Neurocritical Care: Optimization of blood pressure, glycemic control, and normothermia. 4. Secondary Prevention: Use of antiplatelets, statins, and anticoagulants to reduce recurrence risk.

Conclusion

The prognosis of ischemic stroke has significantly improved with the advent of endovascular therapies. However, the burden remains high, necessitating further research into neuroprotective agents. Early recognition of symptoms and specialized stroke unit care remain the cornerstones of management.

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