Cerebrovascular Accident (CVA)

Ischemic stroke

Overview

Is the 3rd leading cause of death and the most common cause of permanent disability in adult.

- Ischemic strokes represent 85%
- hemorrhagic strokes 14%
- venous strokes 1%

Ischemic Strokes

- Ischemic stroke occurs from lack of sufficient arterial blood flow in the territory of a specific cerebral artery to maintain neuronal viability.

- Tow third of the ischemic stroke are due to intrinsic vascular occlusion (***thrombotic***).

- one third are due to occlusion with material originating elsewhere (***embolic***).

- Of the total number of ischemic strokes
- 80% involve the anterior circulation .
- 20% involve the posterior circulation.

Definitions

- *Stroke* is a syndrome characterized by the acute onset of neurological deficit that persist for at least 24 hours, reflects focal involvement of the CNS, and is the result of disturbance of the cerebral circulation.

- *TIA* has been defined as a brief episode of neurologic dysfunction caused by focal brain ischemia, with clinical symptoms typically lasting less than 1 hour (24hours) and without evidence of acute infarction.

- *reversible ischemic neurological deficit (RIND) or minor stroke,* deficits last longer than 24 hours, but resolves completely or almost completely within a few days.

- *Stroke on evolution or progressing stoke* a stroke that is progressing as a direct consequences of the underlying vascular disorder (but not because of cerebral edema).

Table 15.1 Stroke risk factors

Nonmodifiable	Modifiable
Age	Prior stroke/TIA
Sex	Hypertension
Race/ethnicity	Diabetes
Family history	Hyperlipidemia
	Atrial fibrillation
	Homocystinemia
	Carotid stenosis
	Smoking
	Alcohol abuse
	Obesity
	Physical inactivity

Pathophysiology of Ischemic Stroke

- normally cerebral arterial blood flow is 50 mL/100 g of brain tissue per minute.
- A decrease in cerebral blood flow to zero causes death of brain tissue within 4–10 minutes.
- values <16–18 mL/100 g cause infarction within an hour.
- values >20 mL/100 g cause ischemia without infarction unless prolonged for several hours or days.
- If blood flow is restored prior to a significant amount of cell death, the patient may experience only transient symptoms, and the clinical syndrome is called a TIA.

- Tissue surrounding the core region of infarction is ischemic but reversibly dysfunctional and is referred to as the ***ischemic penumbra***.

- The ischemic penumbra will eventually infarct if no change in flow occurs, and hence saving the ischemic penumbra is the goal of revascularization therapies.

Hemorrhagic transformation of ischemic stroke

- represents the conversion of an ischemic infarction into an area of hemorrhage.
- estimated to occur in 5% of uncomplicated ischemic strokes, in the absence of fibrinolytic treatment.
- Proposed mechanisms include reperfusion of ischemically injured tissue (from recanalization or collateral) or disruption of the blood-brain barrier.
- Hemorrhagic transformation of an ischemic infarct occurs within 2-14 days postictus, usually within the first week.

Poststroke cerebral edema:

Cytotoxic edema

- occurs very early in cerebral ischemia.
- Result from intracellular increase in sodium and water content.

Vasogenic edema.

- flooding of the extracellular space by protein and water due to dysfunction of the cerebral vasculature and with breakdown of the blood-brain barrier.

- occurring within 4-6 hours after infarction.

- produces greater levels of brain swelling and mass effect that peak at 3-5 days and resolve over the next several weeks with resorption of water and proteins.

Seizures

- Seizures occur in 2-23% of patients within the first days after ischemic stroke.

- A fraction of patients who have experienced stroke develop chronic seizure

Etiology of ischemic stroke.

A. Vascular Disorders

Atherosclerosis
Inflammatory disorders
Carotid or vertebral artery dissection
Lacunar infarction
Drug abuse
Migraine
Venous or sinus thrombosis

B. Cardiac Disorders

Mural thrombus
Rheumatic heart disease
Arrhythmias
Endocarditis
Mitral valve prolapsed

C. Hematological disorders

Polycythemia
Leukocytosis
Hypercoagulable state
Sickle cell disease

Clinical Diagnosis

- The diagnosis of stroke is ***not difficult***.
- Symptoms typically begin ***suddenly*** and are ***referable to the region*** of brain that is ischemic.
- Thus, the diagnossis of stroke is ***clinical***, and laboratory studies including brain imaging are used to ***support*** the diagnosis

There are no reliable clinical findings that conclusively separate ischemia from hemorrhage, although:

- a more depressed level of consciousness
- higher initial blood pressure
- worsening of symptoms after onset

favor hemorrhage

- a deficit that is maximal at onset, or remits

suggests ischemia.

Neurological symptoms and signs of cerebral ischemia and infarction

- The following suggest ***middle cerebral artery*** ischemia:
  - loss of use of the contralateral face and arm;
  - loss of feeling in the contralateral face and arm;
  - dysphasia;
  - dyslexia, dysgraphia, dyscalculia.

- The following suggests ***anterior cerebral artery*** ischemia:
  - loss of use and/or feeling in the contralateral leg.

- The following suggests ***posterior cerebral artery*** ischemia:
  - contralateral homonymous hemianopia.

Involvement of face, arm and leg with or without a homonymous hemianopia suggests: ***internal carotid artery*** occlusion

Combinations of the following suggest ***vertebrobasilar artery*** ischemia:

- double vision (cranial nerves 3, 4 and 6 and connections).
- facial numbness (cranial nerve 5) .
- facial weakness (cranial nerve 7) .
- vertigo (cranial nerve 8) .
- dysphagia (cranial nerves 9 and 10).
- dysarthria .
- ataxia .
- loss of use or feeling in *both* arms or legs.

Table 11.1. Clinical Deficits Associated With the Anterior and Posterior Cerebral Circulations

Anterior (internal carotid) circulation	Posterior (vertebrobasilar) circulation
Hemiparesis	Hemiparesis
Contralateral body	Contralateral body
Contralateral face	Ipsilateral face
Hemisensory loss (cortical type)	Hemisensory loss
Contralateral body	Brainstem
Contralateral face	Contralateral body
Aphasia (left hemisphere)	Ipsilateral face
Monocular loss of vision	Thalamus—all modalities
	Contralateral body
	Contralateral face
	Homonymous hemianopia
	Diplopia
	Dysarthria
	Dysphagia
	Ataxia

Brain Imaging

- Brain CT scan can demonstrate *very early* hemorrhage, and Because of its *speed* and *wide availability*:

  noncontrast head CT is the imaging modality of choice in patients with acute stroke.

- Brain MRI is superior to CT in detecting infarction from small-vessel occlusion at all stages: acute, subacute, and chronic.

- In cases of acute stroke involving posterior fossa structures (i.e., the brainstem and/or cerebellum), artifact from adjacent bone may impair the sensitivity and the specificity of CT. A MRI will provide clearer images of this region.

Table 15.6 Tests commonly performed in the evaluation of suspected stroke

Routine	Variable
Brain CT without contrast	Brain MRI
Electrocardiogram	Carotid duplex
Complete blood count, platelet count, prothrombin time/International Normalized Ratio (INR), partial thromboplastin time	Cerebral angiography
	Echocardiography
	Transcranial Doppler
	Chest x-ray
Blood chemistries	More extensive blood testing: erythrocyte sedimentation rate, fasting homocysteine, antiphospholipid antibody screen, protein C & S levels, etc.
Fasting lipid profile	Arterial blood gas levels
	Lumbar puncture
	Electroencephalography

Transient Ischemic Attacks

- TIAs are episodes of stroke symptoms that *last only briefly*; the standard definition of duration is <24 hours, but most TIAs last <1 hour.

- The causes of TIA are similar to the causes of ischemic stroke, but with a TIA, the occluded blood vessel *reopens* and neurologic function is *restored*.

- The risk of stroke after a TIA is about 10–15% in the *first 3 months*, with most events occurring in the *first 2 days*.

- This risk can be directly estimated using the **ABCD^2 method**.

- The *improvement characteristic* of TIA is a *contraindication to thrombolysis*.

- However, since the risk of subsequent stroke in the first few days after a TIA is high, the opportunity to give rtPA rapidly if a stroke occurs probably justifies hospital admission for most patients.

- Acute *antiplatelet therapy* after TIA is likely to be effective.

TABLE 1-1 ABCD2 Score and Prognosis After TIA

Score	Factor Assessed at Time of TIA
1	Age ≥60 years
1	Blood pressure ≥140/90 mm Hg on first evaluation
2	Clinical symptoms of focal weakness with the spell (or) 1 Speech impairment without weakness
2	Duration ≥60 minutes (or) 1 10 to 59 minutes
1	Diabetes

TABLE 1-2 ABCD2 Score and Risk of Stroke

ABCD2 Score	2-Day Risk of Stroke (%)
0 to 1	0
2 to 3	1.3
4 to 5	4.1
6 to 7	8.1

Small-Vessel Stroke

- ***lacunar infarction*** refers to infarction following occlusion of small penetrating arteries in the brain.
- These infarcts range in size from *3 mm to 2 cm* in diameter.
- because of *their small size* and *their location* in a relatively *silent area of the brain*, many lacunar infarctions are *not recognized clinically*.
- Hypertension and age are the principal risk factors.
- Recognition of lacunar stoke is important, because the prognosis for complete or near complete recovery is good. In addition, the likelihood of further lacunar stoke can be reduced by treating hypertension.
- Lacunar infarcts are most common in *deep nuclei* of the brain (putamen, thalamus, pons, internal capsule).

Management

Medical support

- Attend to the patient's airway, breathing, circulation (ABC's)
- Treat hypoglycemia or hyperglycemia if identified.
- Perform an emergency noncontrast head CT scan to differentiate between ischemic stroke and hemorrhagic stroke.

Principles of Management

A- Reverse or lessen the amount of tissue infarction
B- Improve functional recovery.
C- Prevention of subsequent stokes.

(1) *Intravenous Thrombolysis*

- Treatment with *IV rtPA* within *3 hours* of the onset of ischemic stroke improved clinical outcome.

- The *time of stroke onset* is defined as the time the patient's symptoms began or the time the patient was last seen as normal. Patients who awaken with stroke have the onset defined as when they went to bed.

Indications

- Clinical diagnosis of stroke
- Onset of symptoms to time of drug administration <3 h
- CT scan showing no hemorrhage or edema of >1/3 of the MCA territory
- Age >18 years
- Consent by patient or surrogate

Contraindications

- Sustained BP >185/110 mm Hg despite treatment
- Platelets <100,000; HCT <25%; glucose <50 or >400 mg/Dl
- Use of heparin within 48 h and prolonged PTT, or elevated INR
- Rapidly improving symptoms
- Prior stroke or head injury within 3 months; prior intracranial hemorrhage
- Major surgery in preceding 14 days
- Minor stroke symptoms
- Gastrointestinal bleeding in preceding 21 days
- Recent myocardial infarction
- Coma or stupor

2) Endovascular techniques

- Occlusions in such large vessels (middle cerebral artery or the internal carotid artery) generally involve a *large clot volume* and often fail to open with IV rtPA alone. Therefore, there is growing interest in using thrombolytics via an *intraarterial route* to increase the concentration of drug at the clot and minimize systemic bleeding complications.

- *Endovascular mechanical thrombectomy* has recently shown promise as an alternative or adjunctive treatment of acute stroke in patients who are ineligible for, or have contraindications to, thrombolytics or in those who have failed to have vascular recanalization with IV thrombolytics

(3) Antithrombotic treatment

Platelet Inhibition

Aspirin is the only antiplatelet agent that has been proven effective for the acute treatment of ischemic stroke.

Anticoagulation

There is no benefit of anticoagulation, unless in case of atrial fibrillation and where a cardiac source of embolization has been found.

The use of low-molecular-weight heparin (anti–factor Xa enoxaparin or nadroparin) given subcutaneously (approximately 4,000 U tid) within the first 48 h of the onset of symptoms appears to be safe and is possibly beneficial.

To improve the functional recovery, we should consider the following:

•Systematic assessment of swallowing, to prevent choking and aspiration pneumonia, with percutaneous gastrostomy feeding if necessary.

•Early mobilization to prevent the secondary problems of pneumonia, deep vein thrombosis, pulmonary embolism, pressure sores, frozen shoulder and contractures.

- Management of blood pressure, with avoiding of over-treatment of hypertension in the first 2 weeks or so (when cerebral perfusion of the ischemic area is dependent upon blood pressure because of impaired autoregulation), moving towards active treatment to achieve normal blood pressure thereafter,

•Early involvement of physiotherapists, speech therapists.

Prevention of subsequent strokes aims at;

- Identifying and treating the cause of the stroke.
  - Lowering modifiable risk factors.
  - Blood pressure reduction, with a thiazide diuretic and (ACE) inhibitor, even in patients with normal blood pressure.
  - Statin therapy even if the cholesterol level lies within the normal range.
- taking oral platelet aggregation inhibitors such as daily aspirin, clopidogrel or dipyridamole.
- Warfarin has not been shown to be better than aspirin in preventing strokes unless the patient has a cardiac cause for the stroke.
- Patients with anterior circulation strokes and a corresponding high-grade (70%–99%) carotid artery stenosis may be considered for carotid endarterectomy

Table 11.5. Antiplatelet Agents

Medication	Dose	Clinical comment
Aspirin	50 mg once daily to 650 mg twice daily	Lower dose reduces risk of side effects Onset of action is immediate Favorable cost
Aspirin/dipyridamole (Aggrenox)	25 mg/200 mg twice daily	Common side effects are headache, gastrointestinal symptoms, and dizziness
Clopidogrel (Plavix)	75 mg once daily	When combined with aspirin, may increase risk for bleeding Adverse effects include rash, diarrhea, and hematologic effects
Ticlopidine (Ticlid)	250 mg twice daily	Potential adverse hematologic effects limit use

Natural recovery from stroke occurs over 3 to 6months. In general, 70% of motor recovery occurs in the first month and 90% occurs by 3 months. Recovery of speech is slower, with 90% recovery by 6 months. In hemiparetic patients, 80% walk again but only 10% regain full use of the paretic hand.

Factors associated with a good recovery include;

- Young age.
- Mild stroke severity.
- High level of consciousness.
- Previous independence.
- Living with a partner.
- Positive mood.