# Overview of Stroke

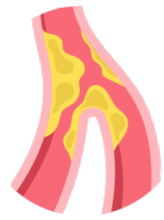
Stroke is a clinically defined syndrome of **acute, focal neurological deficit** attributed to **vascular injury** of the central nervous system.

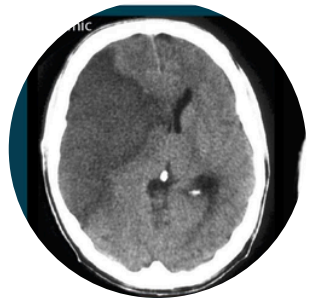
## Ischemic Stroke (85%)

Occurs when there is a blockage in a blood vessel, resulting in restricted blood supply to the brain

**Causes**

- Large vessel disease (e.g. thromboembolism secondary to atherosclerosis in carotid artery)
- Cardioembolic (e.g. atrial fibrillation)
- Small vessel disease (e.g. lacunar infarcts)





Infarct is hypodense!

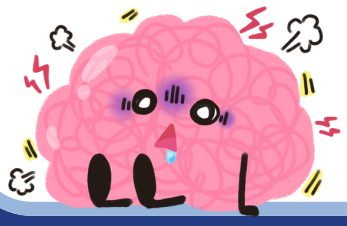
## Hemorrhagic Stroke (15%)

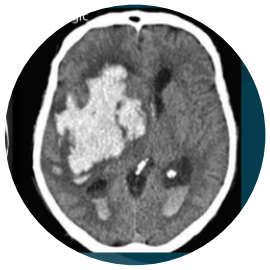
Occurs when a blood vessel ruptures, causing blood to leak into the intracranial cavity

### Intracerebral hemorrhage

**Causes**

- Hypertensive vasculopathy
- Arteriovenous malformations
- Cerebral amyloid angiopathy
- Coagulopathies
- Substance misuse

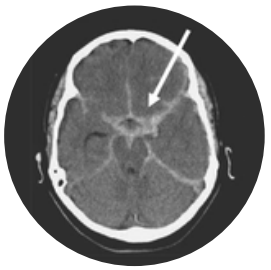




Acute blood is hyperdense!
Chronic blood is hypodense!

### Subarachnoid hemorrhage

- Ruptured berry aneurysm in the circle of Willis
- Arteriovenous malformations
- Substance misuse



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Chronic blood is hypodense!

# Localisation of Stroke

The clinical presentation of stroke depends on which **arterial territory** is involved and the size of the lesion.

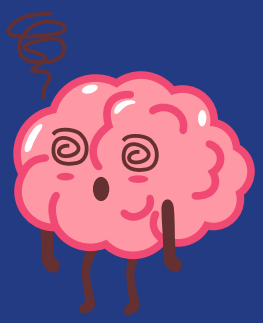
## Anterior circulation stroke (carotid artery)

| Artery | Affected ares | Clinical features |
|---|---|---|
| Anterior cerebral artery | Frontal lobe<br>Parietal lobe | • Contralateral paralysis/weakness of LL > UL<br>• Contralateral sensory loss of LL > UL<br>• Urinary incontinence<br>• Transcortical motor aphasia<br>• Abulia (slowness and delays to perform acts) |
| Middle cerebral artery | Frontal lobe<br>Temporal lobe<br>Parietal lobe | • Contralateral paralysis/weakness of face & arms > LL<br>• Contralateral sensory loss of face & arms > LL<br>• Contralateral homonymous hemi- or quadrantonopia<br>• Conjugate gaze deviation to the side of lesion<br>• Aphasia (involvement of Broca's and Wernicke's areas) |

## Posterior circulation stroke (vertebrobasilar system)

Frequently misdiagnosed, causes a wide range of non-specific presenting symptoms!

Most common symptoms: dizziness, unilateral limb weakness, dysarthria, headache, nausea & vomiting.

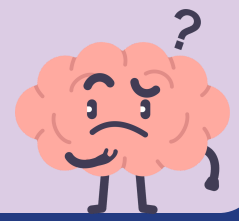


| Affected ares | Clinical features |
|---|---|
| Occipital lobe (supplied by posterior cerebral artery) | • Contralateral homonymous hemianopia<br>• Cortical blindness |
| Cerebellum | • Dizziness or vertigo<br>• Ataxia<br>• Nystagmus<br>• Other cerebellar signs |
| Brainstem | • Ipsilateral cranial nerve palsies (diplopia, dysphagia, dysarthria)<br>• Contralateral hemiparesis<br>• Contralateral hemisensory loss |

# Investigations for Acute Stroke

"All patients with suspected acute stroke should receive emergency brain imaging evaluation on first arrival to a hospital before initiating any specific therapy to treat AIS." (2019 AHA/ASA)

| Objectives | Diagnostic questions | Investigations |
|---|---|---|
| Confirm the diagnosis of stroke | • Is it a vascular lesion?<br>• Is it ischemic or hemorrhagic? | Non-contrast CT brain<br>• Emergency neuroimaging of choice for all patients<br><br>MRI<br>• Limited availability and high cost |
| Rule out stroke mimics | • Metabolic encephalopathy (hypoglycemia, hyperglycemia, hyponatremia)?<br>• Postictal Todd's paresis?<br>• Brain tumours?<br>• Extradual / subdural hematoma?<br>• Brain abscess?<br>• Encephalitis?<br>• Migrainous aura? | Full blood count<br><br>Random blood glucose<br><br>Urea & electrolytes |
| Identify underlying vascular disease | • Is there a cardiac source of embolism?<br><br>• Is there any extracranial vessel disease? | 12-lead ECG<br><br>Carotid duplex ultrasound |
| Identify risk factors | • Hyperlipidemia?<br>• Diabetes mellitus? | Fasting lipid profile (next day)<br>Fasting blood glucose (next day) |



*Advanced imaging (e.g. CT/MR angiography, CT/MR perfusion) may be required in selected cases

*Management of Acute Ischemic Stroke*

# Reperfusion Therapy

**All patients with a confirmed diagnosis of stroke should be admitted for care in a dedicated stroke unit**

## Intravenous thrombolysis (IVT)

### Regimen

IV alteplase 0.9 mg/kg (max 90 mg), 10% given as bolus, remaining dose infused over 1 hour

### Treatment window

Symptom onset of acute ischemic stroke within

**4.5 hours**

### Contraindications

- Acute intracranial hemorrhage
- Systemic coagulopathy
- Severe uncontrolled high blood pressure (BP 185/110 mmHg)


### General care

- Keep NBM and put on IV drip
- Monitor neurological status deterioration and bleeding
- Keep BP < 185/110
- Keep blood glucose between 7.8 - 10
- No antiplatelet and anticoagulants for the first 24 hours after alteplase
- No NG tube insertion, catheterization, and invasive procedure for 24 hours

## Endovascular thrombectomy (EVT)

### Devices

- Aspiration devices
- Stent retriever

### Treatment window

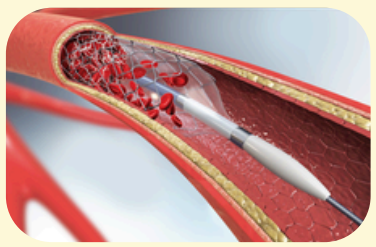
Symptom onset of acute ischemic stroke within

**6 hours**

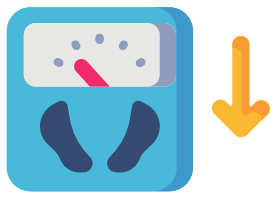
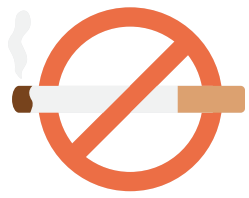
*Can be extended up to 24 hours in selected patients

### Indications

- Generally ADL independent
- Acute ischemic stroke with large vessel occlusion
- Patients who have received IV alteplase (EVT bridging with alteplase "drip and ship")
- Patients not eligible for IV alteplase
- For patients arriving between 6-24 hours of stroke onset, evidence of salvageable brain tissue on CT/MR perfusion (penumbra-core mismatch)

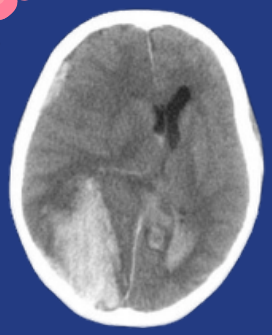


# Prevention of Stroke

| | PRIMARY PREVENTION | SECONDARY PREVENTION |
|---|---|---|
| **Lifestyle** |  Weight loss<br>  Physical activity<br>  Healthy diet<br>  Smoking cessation<br>  Limit alcohol | |
| **Hypertension** | • BP > 140/90 mmHg → treat medically | • Lower BP with ACE-inhibitors<br>• Systolic BP target 120–130 mmHg |
| **Diabetes** | Maintain good glycemic control | |
| **Hyperlipidemia** | • Treatment is stratified based on risk<br>• High-risk group: lower LDL to < 1.8 mmol/L<br>• Intermediate- and low-risk group: lower LDL to < 3.4 mmol/L | • Consider lipid reduction with statins<br>• LDL target < 1.8 mmol/L |
| **Aspirin** | Aspirin is not recommended for primary prevention of stroke in diabetics, moderate risk individuals or elderly patients in view of the high risk of bleeding | • Aspirin 100mg oral OD<br>or<br>• Clopidogrel 75mg oral OD |
| **Anticoagulants** | DOAC to prevent cardioembolic stroke in all atrial fibrillation patients with CHA2DS2-VASc score of ⩾2 (male) or ⩾3 (female) | |
| **Re-vascularization procedures** | • Carotid endarterectomy may be considered in asymptomatic carotid stenosis ⩾70% | • Carotid endarterectomy indicated for most patients with carotid stenosis ⩾70% after a recent ischemic event (benefit greatest within 2 weeks) |

*Hemorrhagic Stroke*

# Intracerebral Hemorrhage

A hyperdense collection of blood, often with surrounding hypodense edema

Hematoma expansion and perilesional oedema leads to mass effect and ↑ ICP

The distinction between brain hemorrhage and ischemia cannot be made based on the basis of clinical characteristics alone! Neuroimaging is mandatory!

## Clinical Features

- Neurologic symptoms & signs may be progressive over minutes or a few hours
- Stroke symptoms: sudden onset loss of function in speech, vision, movement, sensation
- ↑ ICP: headache, vomiting, dilated pupil, progressive drowsiness

## Investigations

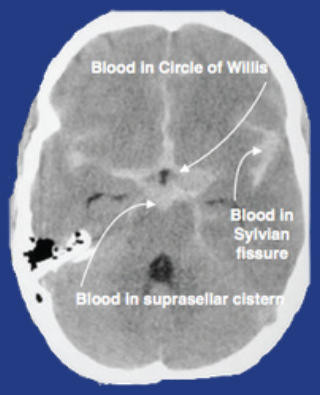
- Non-contrast head CT / MRI brain
  - Confirm diagnosis of ICH
  - Exclude ischemic stroke and stroke mimics
  - Estimate hemorrhage volume
  - Predict hemorrhage expansion

## Management

- Admission to ICU or stroke unit
- Discontinue all anticoagulants and antiplatelets, give reversal agents immediately
- Manage elevated blood pressure
- Manage intracranial pressure
  - Osmotic therapy
    - Hypertonic saline
    - IV mannitol
- Surgical options
  - External ventricular drain for obstructive hydrocephalus
  - Craniectomy and hematoma evacuation

*Hemorrhagic Stroke*

# Subarachnoid Hemorrhage




Stellate-shaped hyperdense blood filling subarachnoid space

Most nontraumatic SAH are caused by ruptured berry aneurysms

Although aneurysmal SAH occurs commonly on physical exertion, straining, sexual excitement; but aneurysms can rupture at any time

## Clinical Features

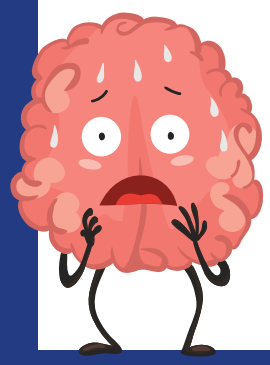
- Sudden-onset, severe headache "worst headache of my life" "thunderclap headache"
- Nausea and vomiting
- Meningism: neck stiffness
- Lower back pain (circulation of bloody CSF down spinal axis)
- Loss of consciousness
- Prodromal headache days to weeks prior (minor hemorrhage "a warning leak")

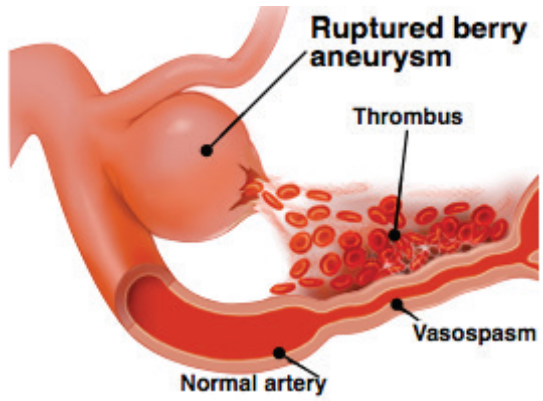
## Investigations

- Non-contrast head CT
- Lumbar puncture if head CT normal & suspicion high
  - ↑ Opening pressure
  - Elevated RBC count not diminishing from CSF tube 1 to tube 4
  - Xantochromia

## Management

- Admit to neurocritical care unit
- Discontinue all anticoagulants and antiplatelets, give reversal agents immediately
- Control blood pressure
- Constant hemodynamic, cardiac, neurologic monitoring
- Nimodipine (CCB) to prevent vasospasm
- Aneurysm repair with surgical clipping or endovascular coiling
- Others: pain control, bed rest, DVT prophylaxis





# Test your Knowledge!

A 67-year-old male presents to the emergency department with sudden onset right-sided weakness and slurred speech that began 2 hours ago. His past medical history is significant for hypertension and type 2 diabetes mellitus. On examination, his blood pressure is 190/110 mmHg, and he has a right facial droop with 2/5 strength in the right upper and lower extremities. His speech is slurred, and he has difficulty understanding complex commands.

Given the patient's presentation and history, which of the following is the most appropriate next step in management?

A. Administer intravenous thrombolytics (tPA) immediately

B. Perform a non-contrast head CT scan

C. Administer aspirin 325 mg orally

D. Refer for carotid endarterectomy

# References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