Ischemic Stroke

Adel Alhazzani MD ,FRCPC , FAAN
Associate Professor of Neurology
College of Medicine , King Saud University

Outlines

- What and why ?
- Pathophysiology and subtypes
- Clinical presentation
- Diagnosis (Neuroradiology 101)
- Management
- TIA

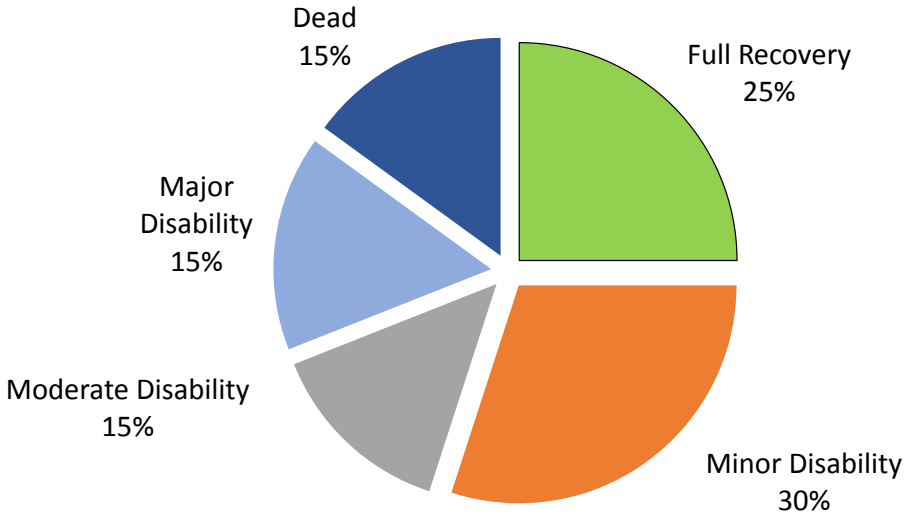
Worldwide Burden of Stroke

- 1, 2, 3, 4, 5, 6,
- Leading cause of adult disability
- 2nd cause of death
- 20 million people worldwide suffer a stroke each year.
- 1/4
- 5 million deaths/year
- Every 6 seconds

Kim AS, Johnston C. Circulation 2011

Lozano et al. Global Burden of Disease. Lancet. 2012 May 27;367(9524):1747-57Lancet 2006

Outcome of Ischemic Stroke



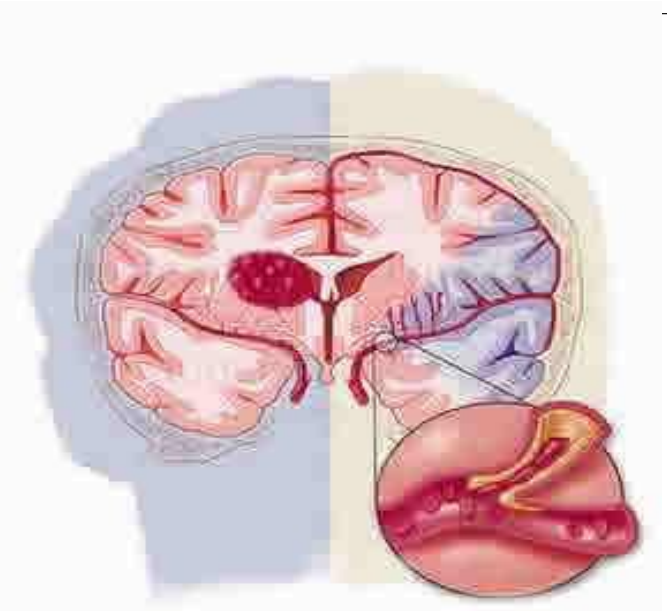
Adapted from Stegmayr B, et al. *Stroke* 1997;**28**:1367-1374

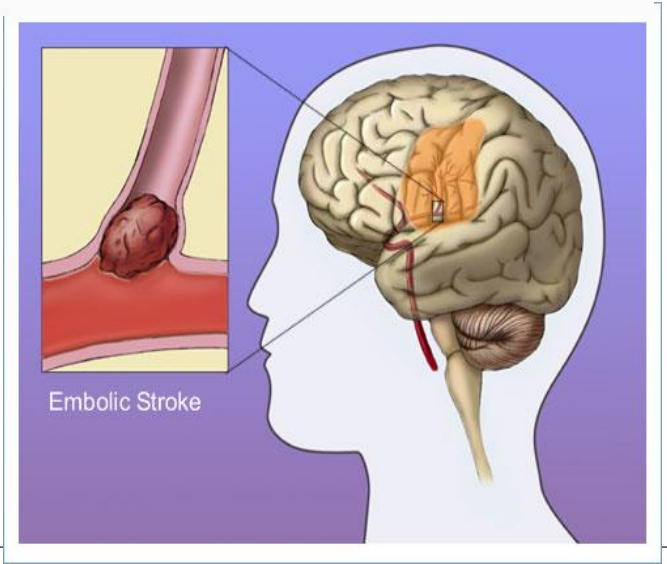
In Saudi Arabia

- 20-25,000 new strokes
- 4000-5000 deaths (estimate)
- 8000 - disabilities
- Incidence 58/100,000 new
- Total 70/100,000 total “recurrence ”
- Cost to patient, family , community

Definition

- Abrupt onset
- Focal neurological deficits
- Due to interruption of vascular supply
- Can be ischemic (blockage) or hemorrhagic (bleeding)





Ischemic Stroke

- 85% of all strokes
- Acute onset of neurologic deficits caused by impaired blood flow to CNS
- Stroke
  - persisting neurologic deficit after 24hrs and/or
  - infarct on CT or MRI

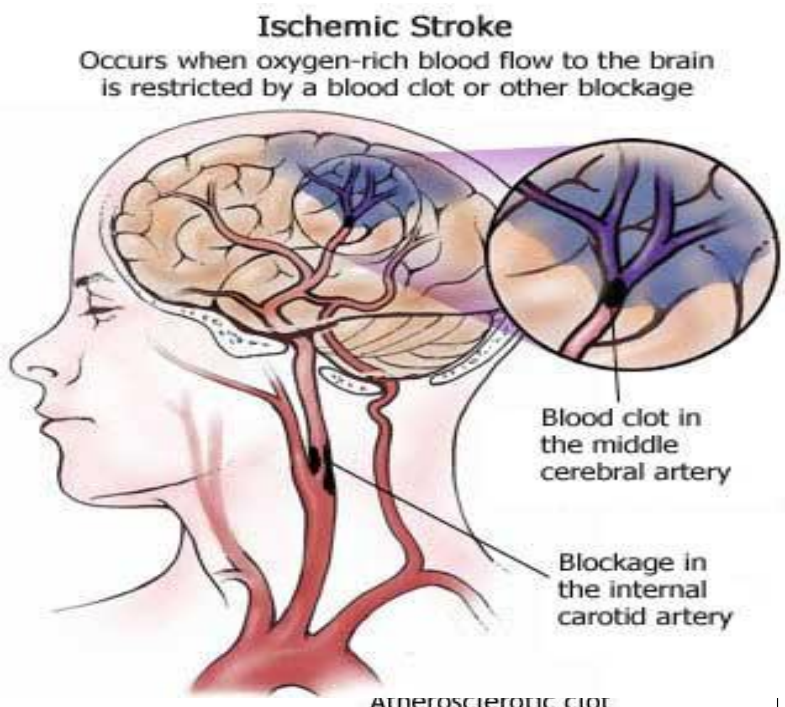
- Transient ischemic attacks (TIAs) AKA "mini strokes" or "warning strokes" stroke-like symptoms that last for a very short time(<1hr) with complete recovery (most are <5 min)
- A TIA indicates that conditions for an ischemic stroke are present

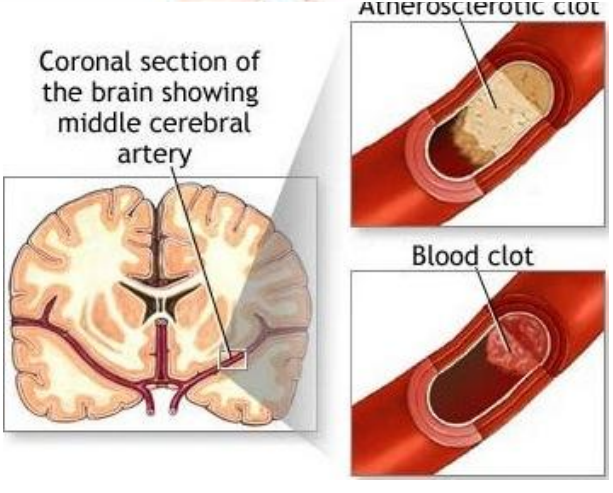
Ischemic Stroke Mechanisms

Due to ***blockage*** from :

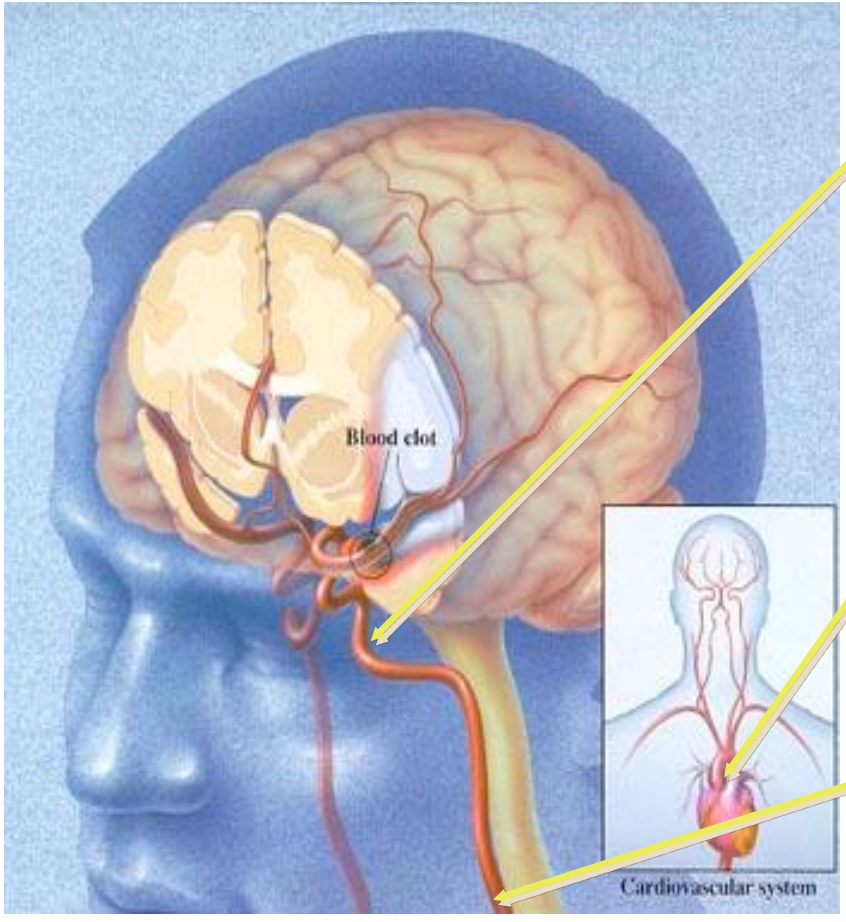
- Cerebral thrombosis: a thrombus (blood clot) that develops at the blocked part of the artery
- Cerebral embolism: typically caused by a blood clot that forms at another location and breaks loose and enters the bloodstream

Hypoperfusion (Narrow vessels reduced flow)





Ischemic Stroke: Mechanisms



BLOOD VESSELS

-Atheromatous (large or small vessels)

-Non atheromatous (vasculitis, dissection)

HEART

Cardioembolic

BLOOD

Coagulo and heamoglobinpathies

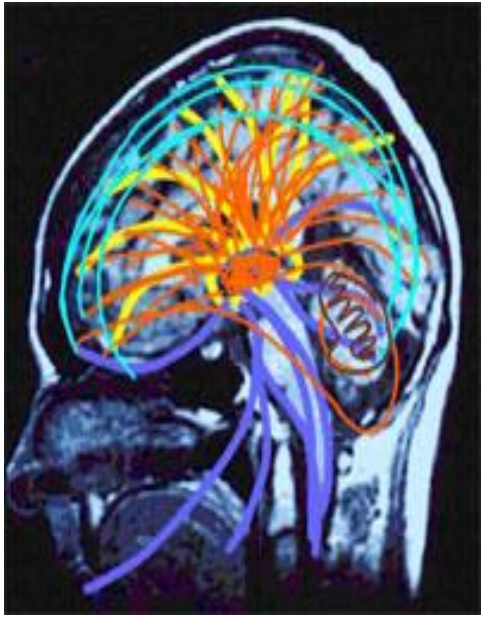
Risk Factors

Non-modifiable .

Age, Sex, Ethnicity, and genetic determinants

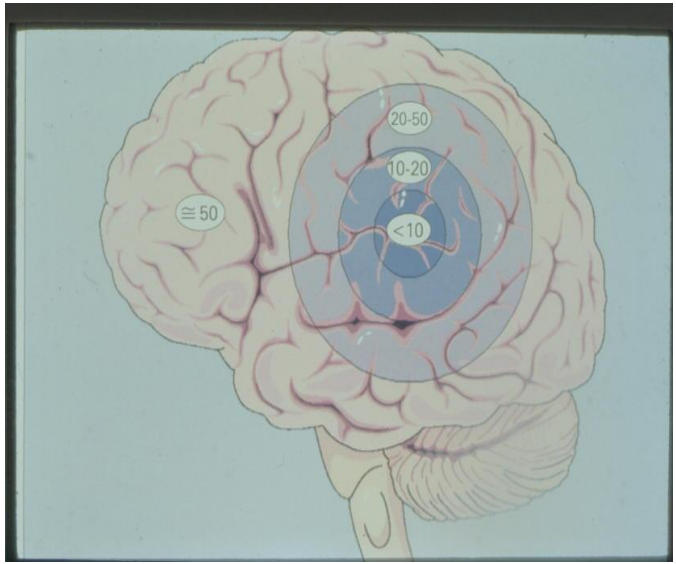
Modifiable :

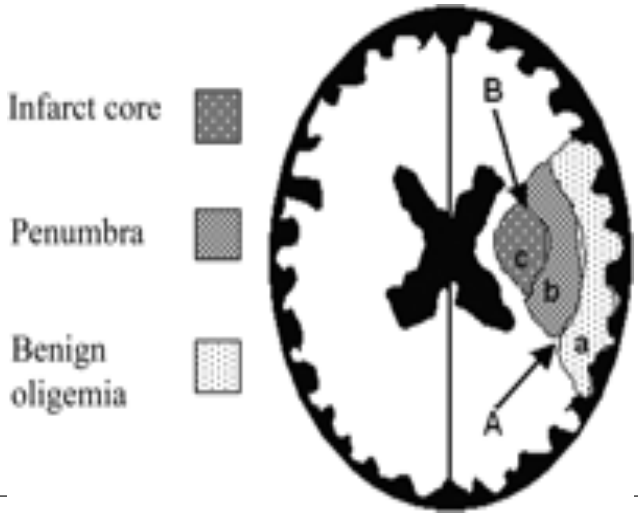
- **HTN**
- DM
- SMOKING .
- Hyperlipidemia .
- cardiac disease (particularly atrial fibrillation [AF]
- Stroke , TIA , carotid artery stenosis.
- Sedentary lifestyle



Pathophysiology

- Active and does not store energy.
- the brain is not adequately perfused , cells begins to die.
- Core (area of irreversible damage)
- Penumbra (tissue at risk can





History

- *ONSET* (Last time seen normal)
  - Symptoms (analysis of symptoms)
  - progression
  - Headache (sudden and severe)*
  - Neck pain/ trauma*
  - Previous HX of stroke or TIA,

- PMHX : Risk factors/medication
- HX from others

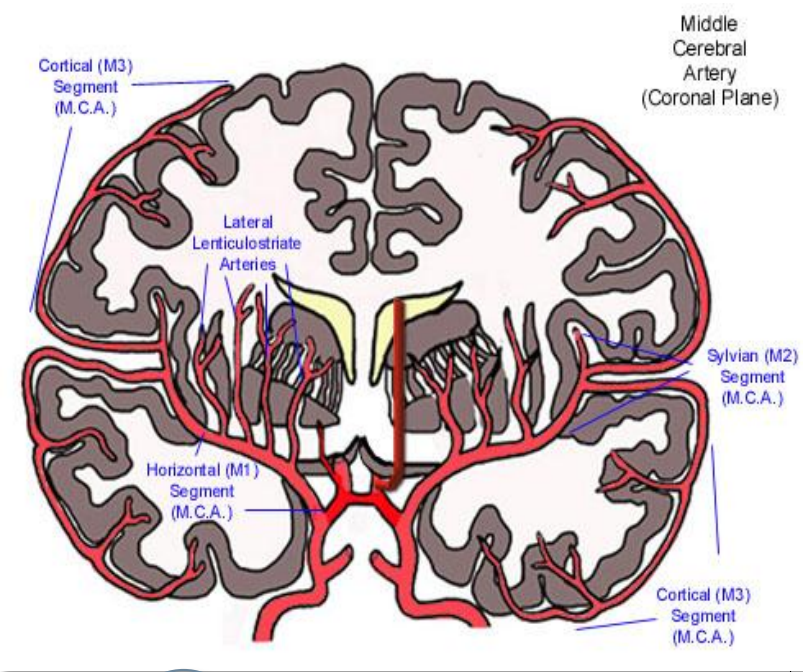
Physical examination

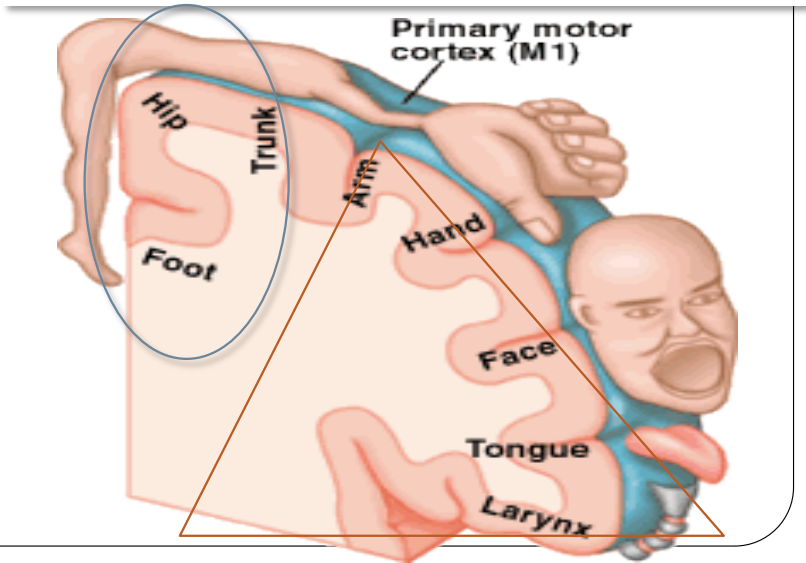
- ABC
- General examination
- Pulse (A.fib)
- BP
- Hand
- listen for heart murmur , carotid bruits

- Cortical infarcts are suspect based on the presence of
  - language impairment
  - neglect or anosognosia
  - graphethesia or stereoagnosia
  - visual field impairment
- CN involvement and crossed motor
- Tone –decreased on side of weakness early on, later on increased
- Pyramidal pattern weakness (UMN)
  – UE extensor > flexor
  – LE flexor > extensor
- Reflexes –hypereflexic on side of weakness, with up-going toe

Clinical presentation

- Depends on location
  - Middle Cerebral Artery MCA (arm + face > leg weakness and sensory loss aphasia, neglect, homonymous hemianopia)
  - Anterior Cerebral Artery ACA (weakness LE >UE , emotional disturbance)
  - Internal Carotid (above and ophthalmic)
  - Lacunar syndrome (small penetrating arteries)



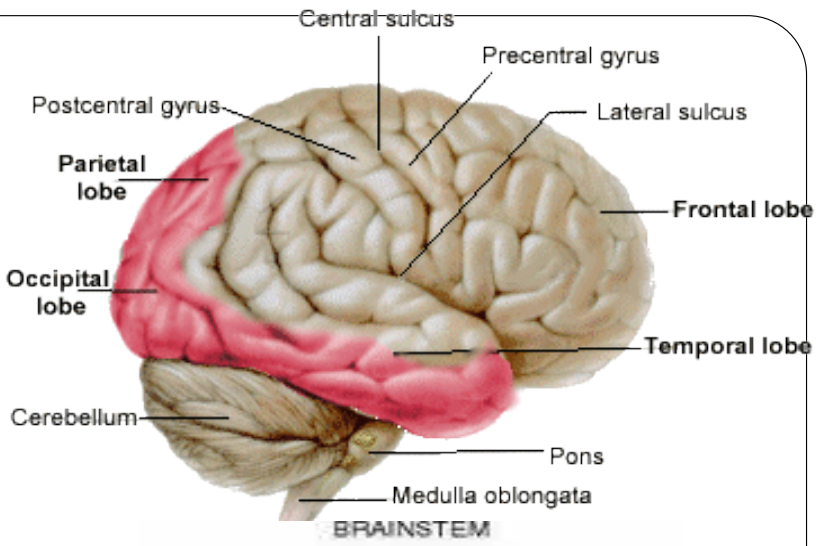


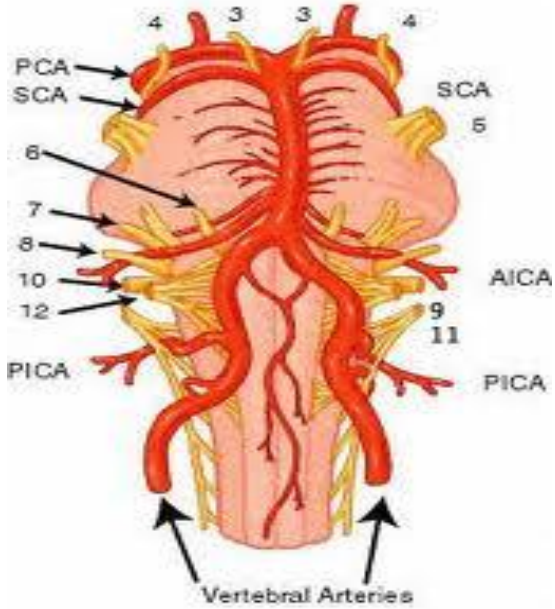
Clinical presentation

- Posterior cerebral artery PCA

(vision-visual fields and memory)

- Vertebrobasilar : CN with crossed motor , cerebellum, altered LOC
- Midbrain
  - CN III –, dilated pupil
- Pons
  - CN V –facial numbness, weakness jaw movements
  - CN VI –lateral rectus palsy
  - CN VII –facial weakness
- Medulla
  - CN VIII –vertigo, hearing loss
  - CN IX, X –dysphagia
  - CN XII –tongue weakness





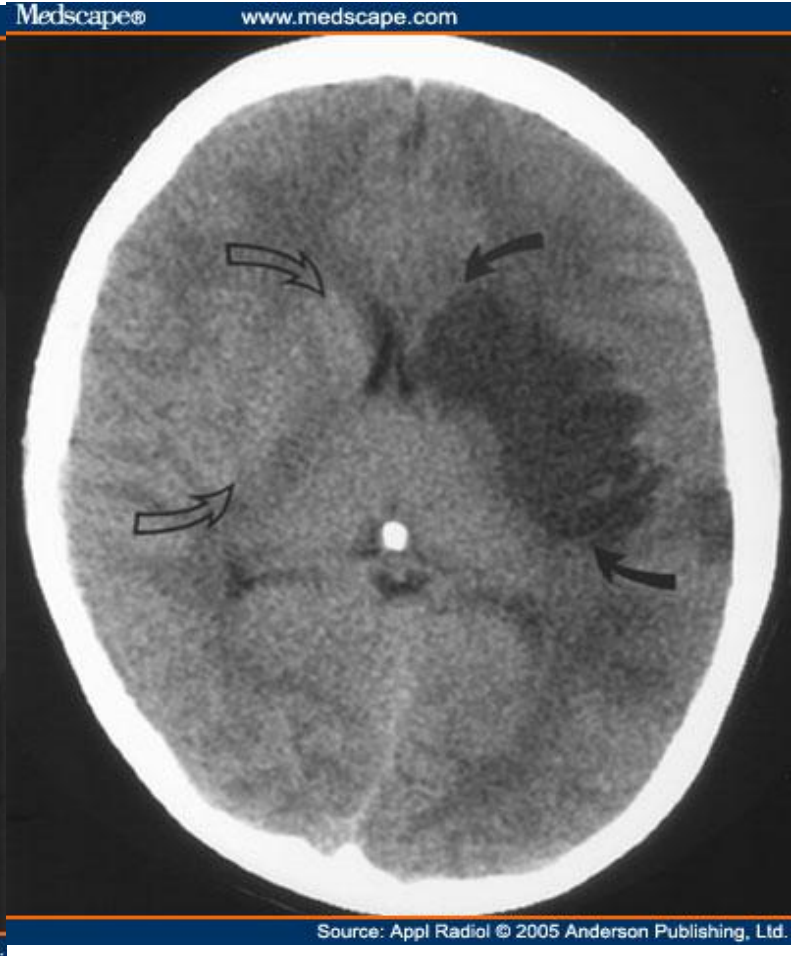
Investigation

- CBC
- Coagulation profile (PT , PTT and INR)
- Chemistry
  - Fasting glucose, Hba1c,Lipids
- Specific cases (Hb electrophoresis , hypercoagulable work up, CTD screen, HIV and syphilis)
- Imaging
  - CT scan
  - MRI
  - Vascular imaging (Carotid U/S , CTA , MRA , cerebral Angio)
- Cardiac work up
  - ECG
  - Echo (TTE or TEE)
  - Holter

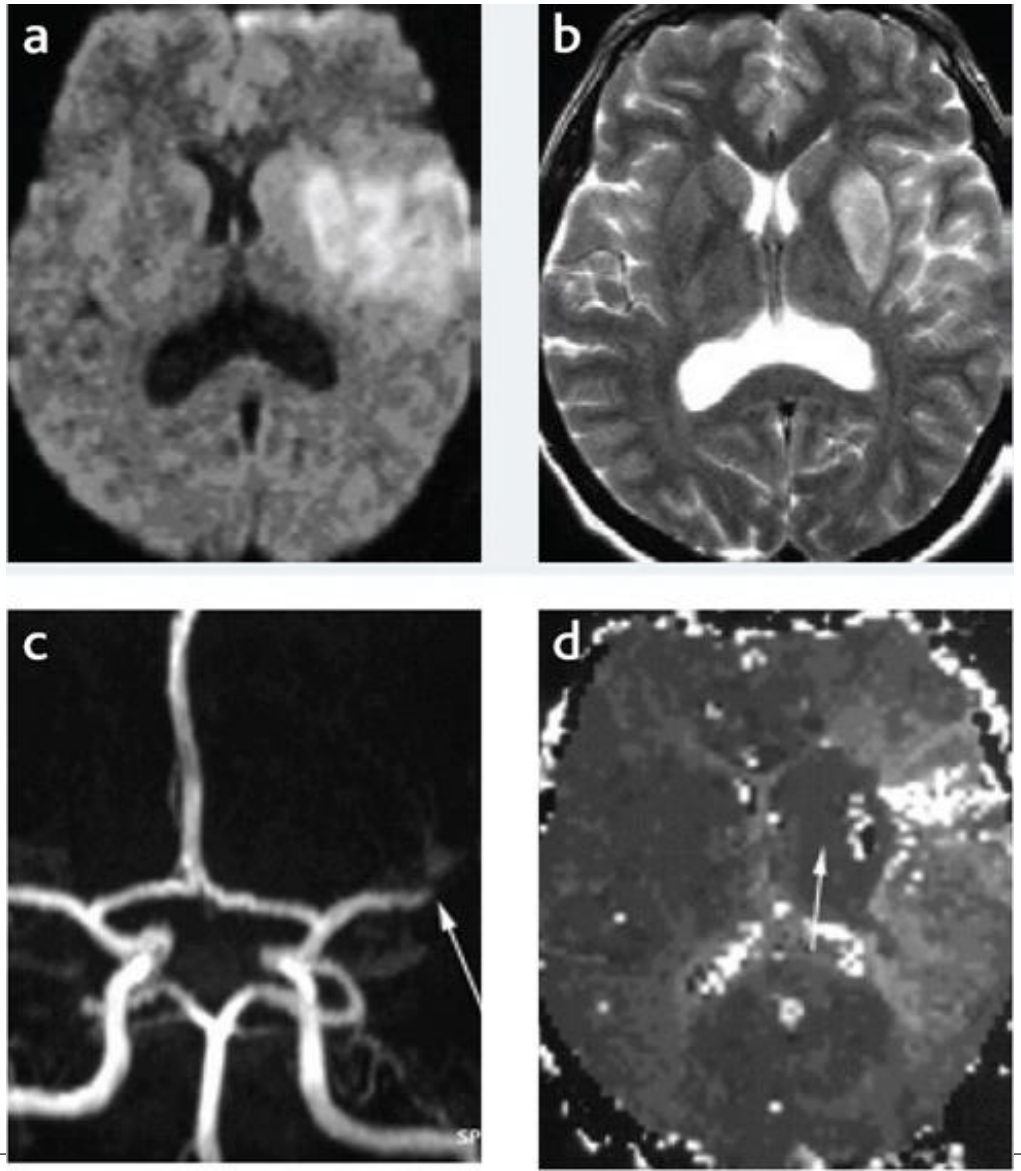
CT scan

2hrs

20 hrs



36 HRS



MRI acute stroke

- More sensitive
- C/I

Management

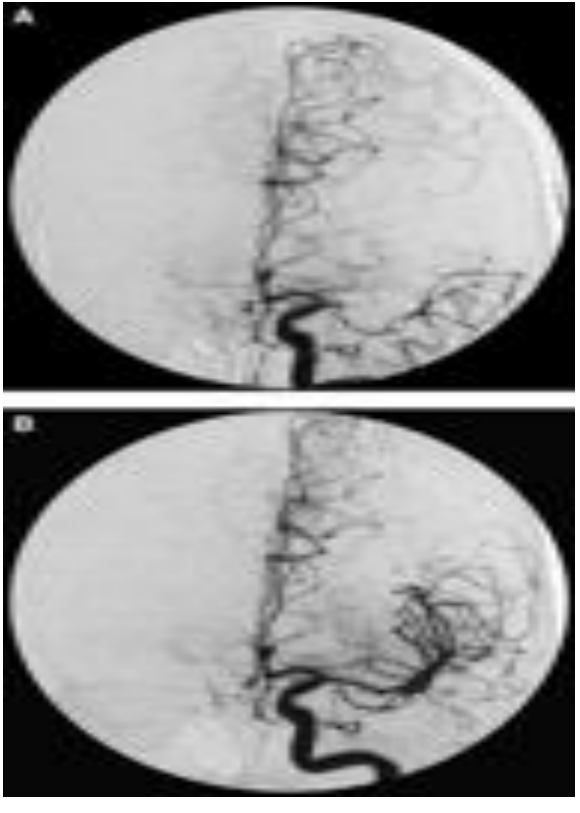
- **Acute Stroke Management**
  - ABC
  - Reperfusion
  - Prevent progression and complication
- **Long Term Management**
  - Risk Factor: HTN, DM, lipid, smoking, A-fib
  - Anti-platelet (atherosclerosis) or Anticoagulant (afib or hypercoagulability)
  - Rehabilitation

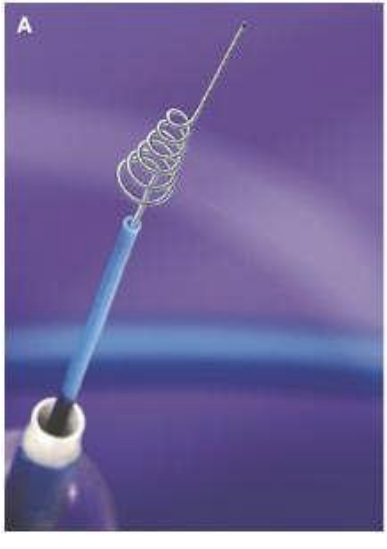
Reperfusion

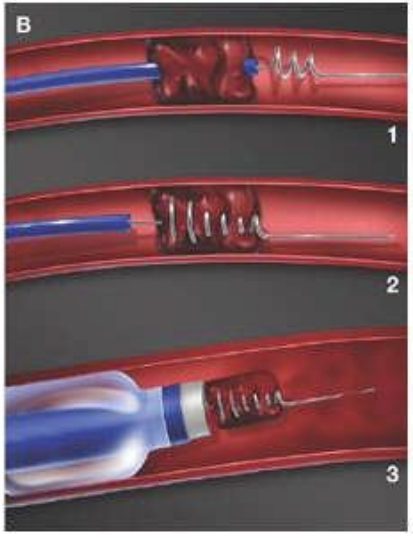
- Intravenous thrombolysis (IV t-PA) *Tissue plasminogen activator*
  - Effective up to 4.5 hrs from onset
  - Sooner the better (time= brain)
  - 30% chance of improvment 1/3, 1 out of 8 complete recovery
  - Risk of bleeding (ICH) = 6%

- Exclusion criteria:
  - ICH
  - prior ICH, Hx suggests of SAH, stroke past 3mts
  - GI or GU hmg in past 3wks, recent MI, major surgery 14d
  - platelet <100 000,
  - INR >1.7, PT >15
  - SBP >186 or DBP >110, Hg<100?

Intra-arterial thrombolytic







Management

- Stroke unit
  - BP and glycemic control
  - NPO, Avoid aspiration
  - Dx and Rx Temp .
  - PT , OT and early rehab.
  - DVT prophylaxis
- Aspirin or other antiplatlets (started within 48 hours reduces the risk of early recurrent ischemic stroke without a major risk Hge and improves long-term outcome)
- **Long Term Management**
  - HTN
  - DM
  - Stop smoking
  - Lipid lowering agent
  - Exercise

Secondary prevention

- Treat underline condition (Carotid SX, cardio-embolic and hypercoagulable rx with Coumadin)

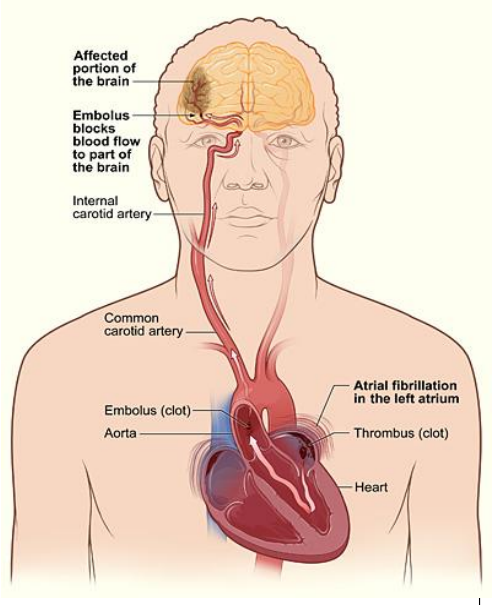
TRANSIENT ISCHEMIC ATTACKS

- Brief and temporary reduction in blood flow to a focal region within the brain with no evidence of infarction on imaging.
- Is a stroke that did not finish YET
- Up to 1/3 with have stroke (usually first 48 hrs)
- most TIA's last 5-20 minutes
- if >1hr usually small infarction on MRI
- DDX (Seizure, migraine , Syncope , Labyrinthine

SDH,

Approach to TIA

- Needs urgent assessment (ER)
- Rule out other causes of transient events (by HX and PE) TIA rarely march across body
- Work up (labs , CT scan or MRI ,
  - ***vascular imaging of carotid CTA , MRA, US***
  - ***Cardiac work up (EKG, echo +/-Holter***
- Start stroke prevention measures (like ischemic stroke) ASA , control HTN ,DM and lipids, stop smoking and exercise .



Take Home Message

- Stroke can be ischemic or hemorrhagic
- Every acute stroke patient should be viewed as an eminently treatable neuroemergency.
- Time window for effective therapy in stroke is brief (Time is brain)
- TIA Is a stroke that did not finish YET
- Any one present with sudden severe Headache should be presumed to be SAH *until proven otherwise* .

Questions