Imaging In Acute Stroke

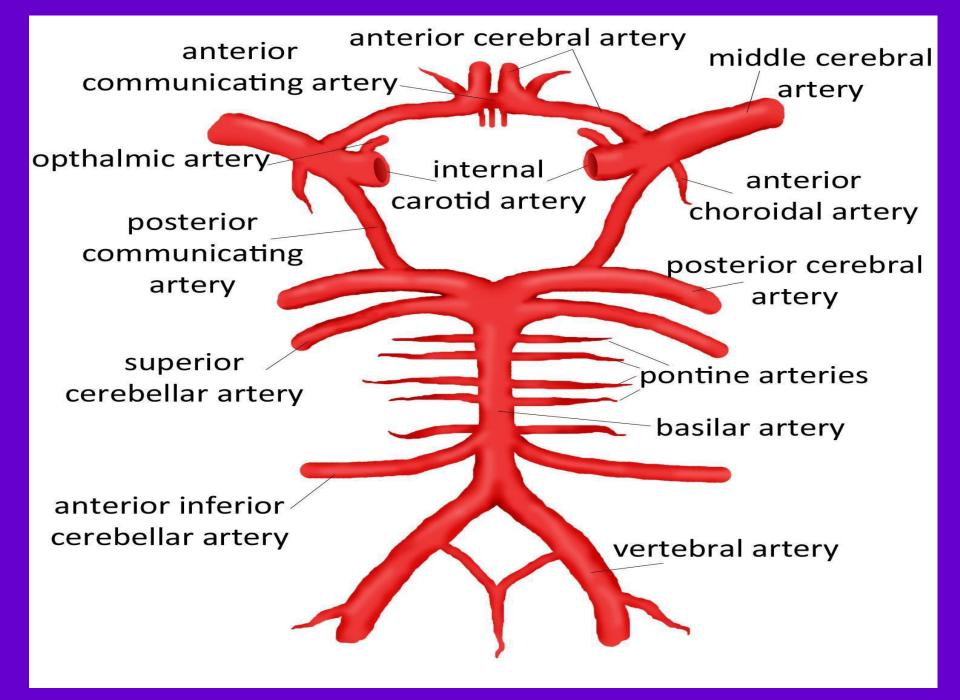
SPEAKER : Dr.YUGANDHAR S COMMON ACADEMIC PROGRAM, 10-04-2021 NARAYANA MEDICAL COLLEGE

Stroke

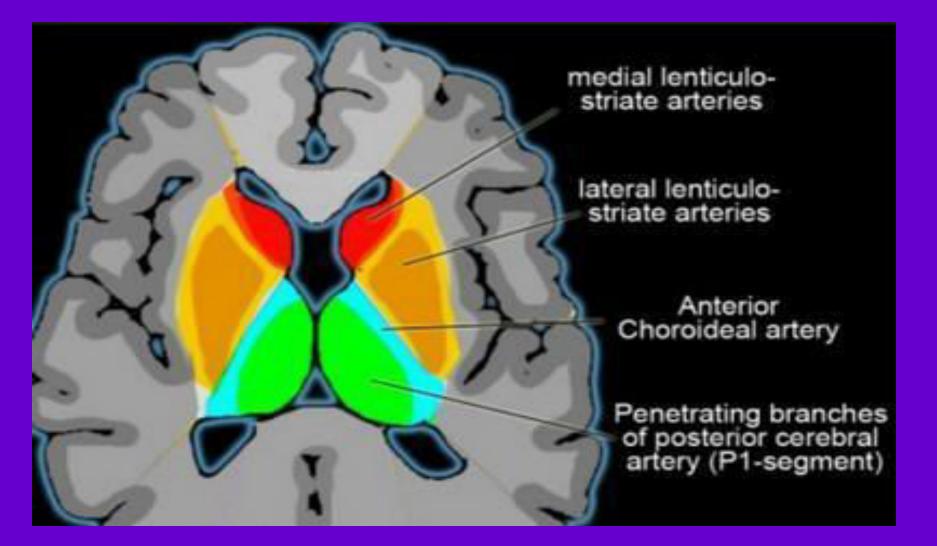
- Definition : Lack of blood supply to brain tissue leads to development of focal neurological deficit for > 24 hrs.
- TIA : Decrease blood flow to brain tissue leads to focal neurological deficit and resolves within 24 hrs , without radiological evidence.

STROKE Mechanism:

- Interruption of blood flow(Ischemia) >> Cerebral Infarction (80%) or
- Bleeding into or around the brain >> Hemorrhagic stroke (20%)



BASAL GANGLIA BLOOD SUPPLY



ISCHEMIC INFARCT

Goals of imaging :

- . To diagnose & locate the infarct ASAP.
- . To exclude hemorrhage Appropriate therapy.
- . To know information regarding intracranial vasculature and brain perfusion

4 - Ps of Acute Stroke Imaging

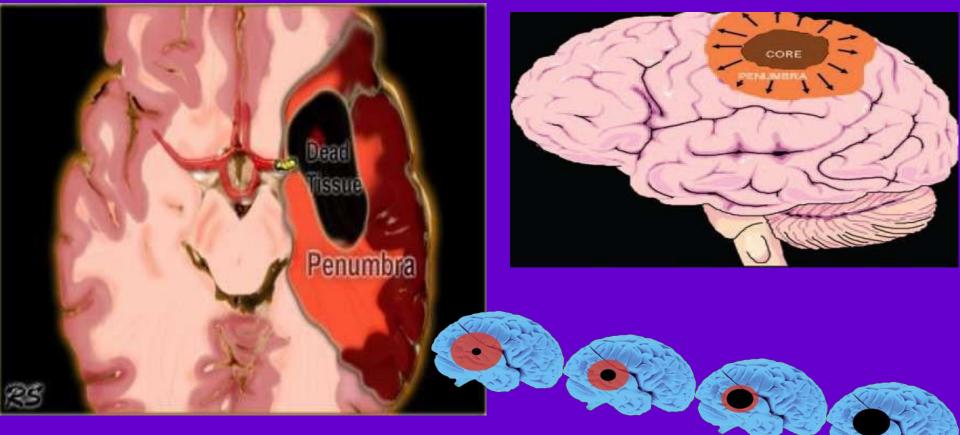
. Parenchyma: Assess early sign of acute stroke, rule out hemorrhage (NCCT)

.Pipes: Assess extracranial circulation (carotid and vertebral arteries of the neck) Assess intracranial circulation for evidence of intravascular thrombus

.Perfusion: Assess Cerebral blood volume, cerebral blood flow, and mean transit time

.Penumbra: assess tissue at risk of dying if ischemia continues without recanalization of intravascular thrombus

PENUMBRA



.Core of irreversibly infracted tissue surrounded by a peripheral region of ischemic but salvageable tissue referred to as a penumbra.

Without early recanalization, the infarction gradually expands to include the penumbra

INFARCT Terminology based on time of onset:

Immediate sign of Infarct is Hyperdense MCA Sign

Early Hyperacute(0 to 6 hrs) : Hypoattenuation of Brain tissue Hypoattenuation of Lentiform nuclei Loss of Insular ribbon sign Loss of Cortical Grey White matter differentiation Sulcal effacement.

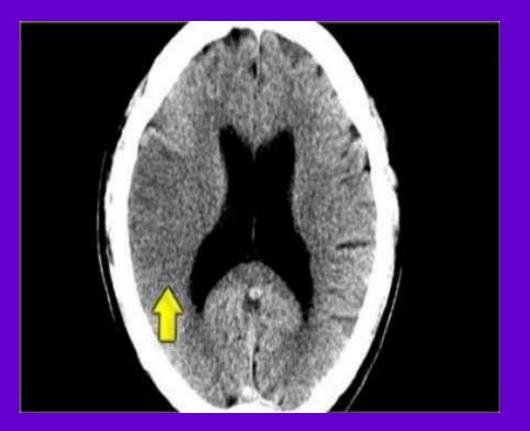
. Late Hyperacute : 6 to 24 hrs

. Acute(24 hrs to 1 wk) : Swelling become marked , shows MASS EFFECT

.Subacute(1 to 3 wks) : Swelling starts subside, small amount of petechial haem.result in elevation of attenuation of cortex-CT FOGGING PHENOMENON (Not to be confused with haemorragic transformation)

.Chronic : > 3 wks

Hypo attenuating brain tissue



Obscuration lentiform nucleus



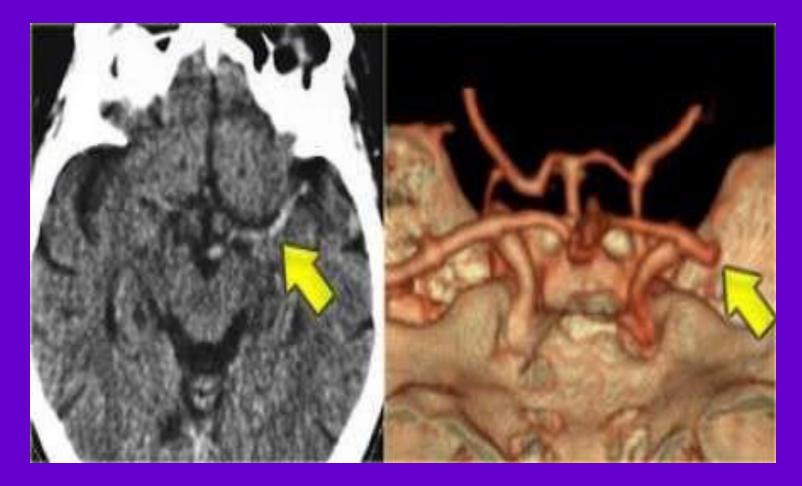
 MCA infarction: on CT an area of hypo attenuation appearing within six hours is highly specific for irreversible ischemic brain damage Axial NCCT image shows hypoattenuation obscuration of the left lentiform nucleus (arrows), comparison with the right lentiform nucleus.

LOSS OF INSULAR RIBBON SIGN

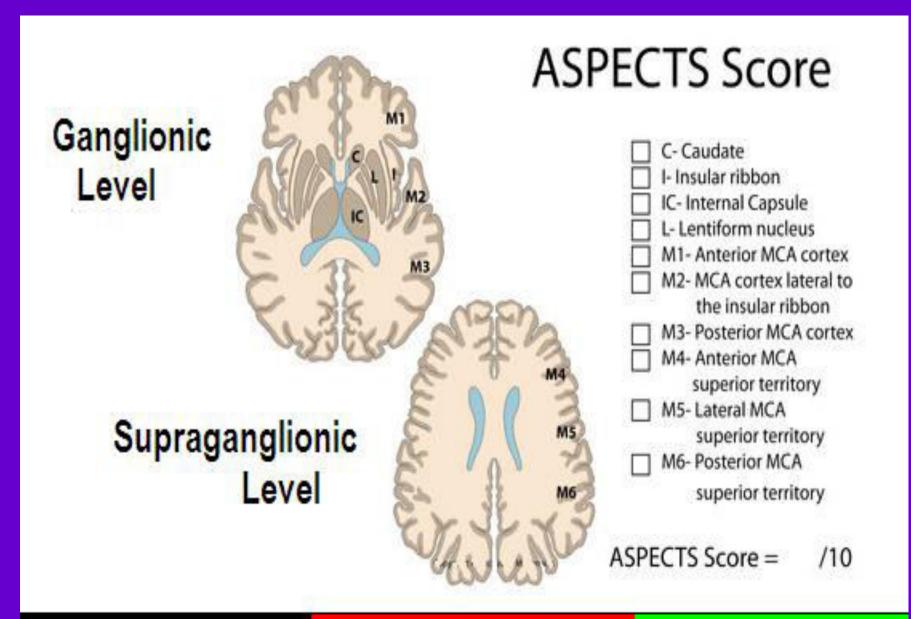
Axial unenhanced CT image, shows hypo attenuation and obscuration of the posterior part of the right lentiform nucleus (white arrow) and a loss of gray matter—white matter definition in the lateral margins of the right insula (black arrows).The latter feature is known as the insular ribbon sign.



Dense MCA sign



Thrombus or embolus in the Left MCA. On CT-angiography occlusion of the Lt. MCA is visible.

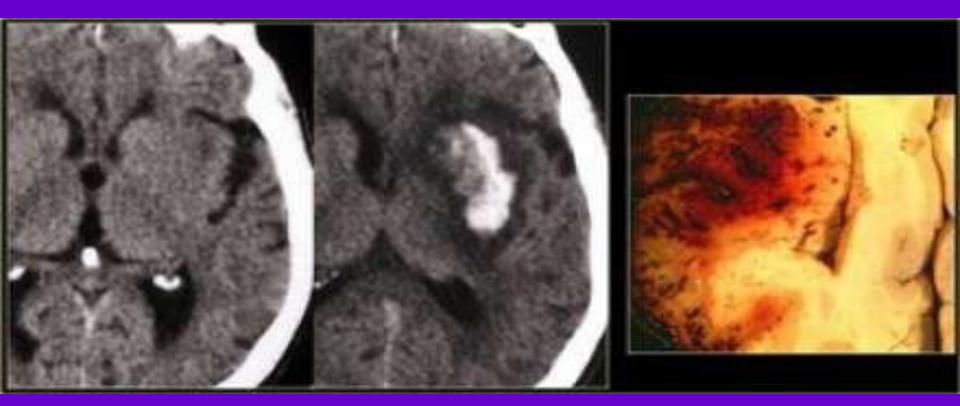


8-10 small core 6-7 moderate core

0-5 large core

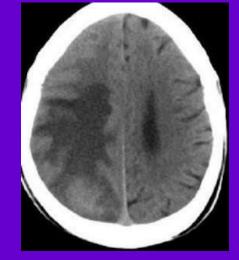
Hemorrhagic infarcts

• 15% of MCA infarcts are initially hemorrhagic.

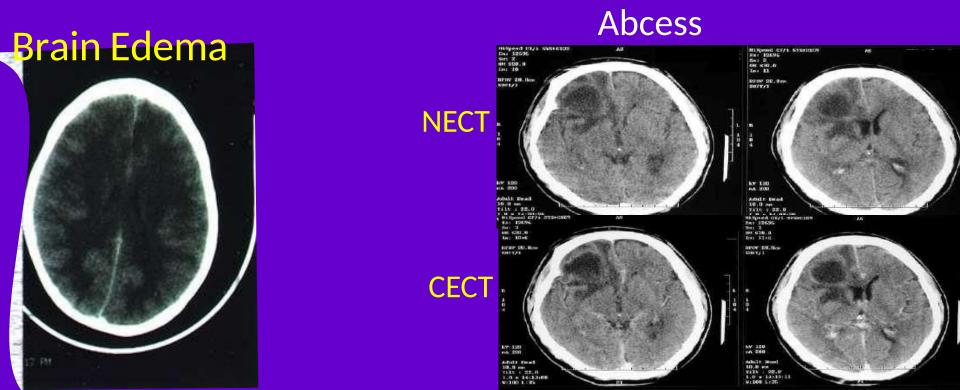


Stroke may Mimics on NECT

- Old Blood- EDH, SDH
- Traumatic contusion



Tumor Mets from CA Breast

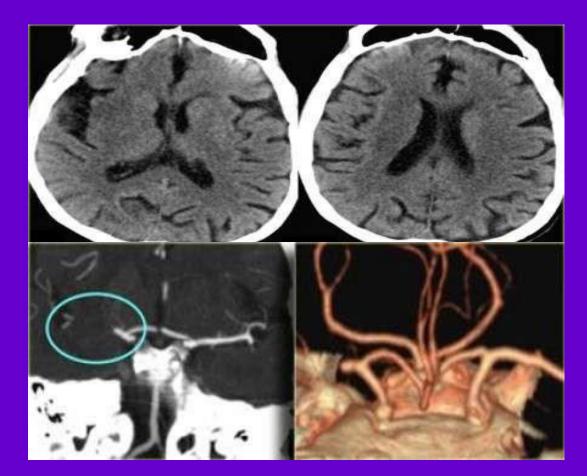




Once we have diagnosed the infarction, we want to know which vessel is involved by performing a CTA.

 Insular ribbon sign in right insular cortex

• CTA disclose thrombus in rt. MCA



CT Perfusion (CTP)

- With CT and MR imaging we can localize infarcted area, but we cannot preclude a large ischemic penumbra (tissue at risk).
- In perfusion studies iodinated contrast agent bolus through the cerebral vasculature. Perfusion will tell us which area is at risk..
- The limitation of CT-perfusion is the limited coverage.

CT PERFUSION PARAMETERS

CEREBRAL BLOOD VOLUME the volume of blood per unit of brain tissue

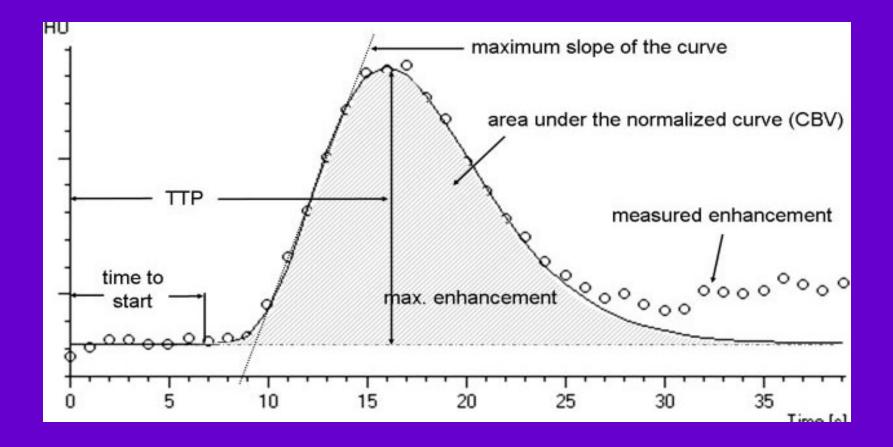
CEREBRAL BLOOD FLOW the volume of blood flow per unit of brain tissue per minute

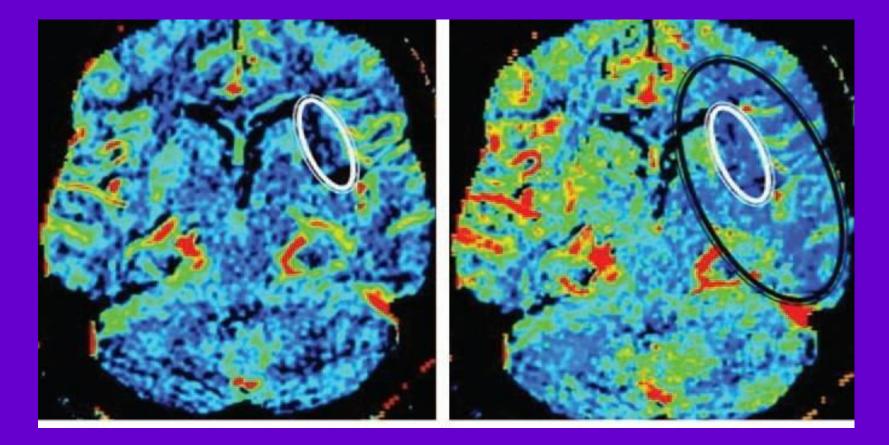
MEAN TRANSIT TIME the timedifference between the arterial inflow and venous outflow

TIME TO PEAK ENHANCEMENT the time from the beginning of contrast material injection to peak enhancement

CBF = CBV / MTT

CT perfusion - Interpretation

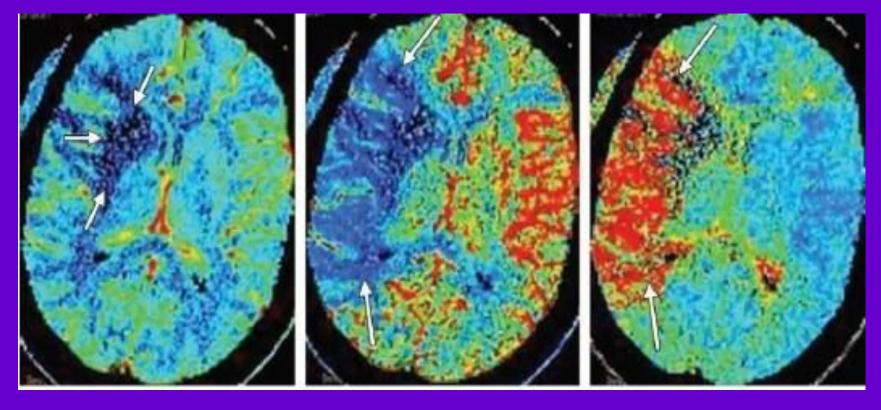








CBF-CBV= Penumbra



CBV

CBF

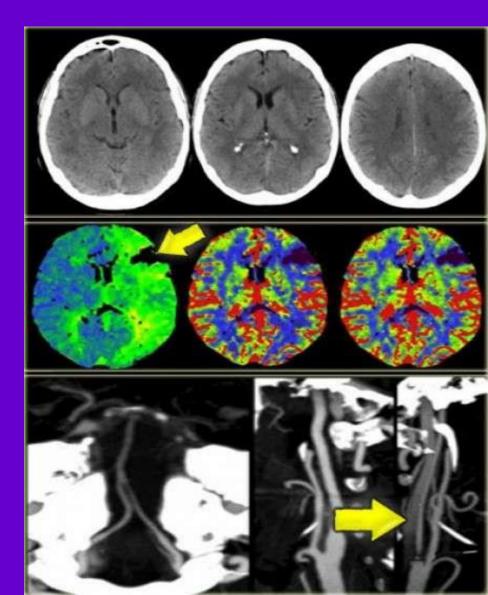
MTT

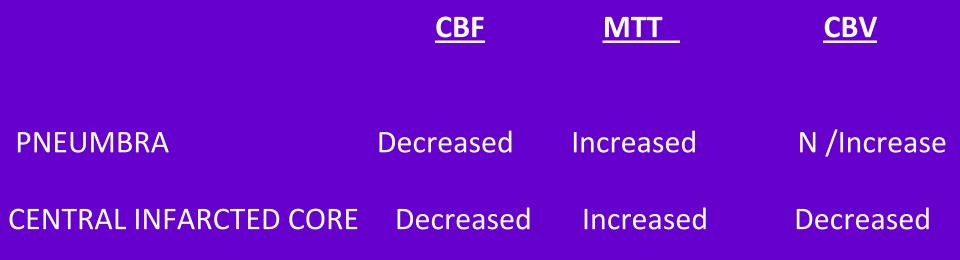
INFARCT WITH SALVAGABLE PENUMBRA

NECT, CTP and CTA

 CT is normal but patient is symptomatic

- .CTP shows a perfusion defect
- CTA was subsequently performed and a dissection of the left internal carotid was demonstrated.

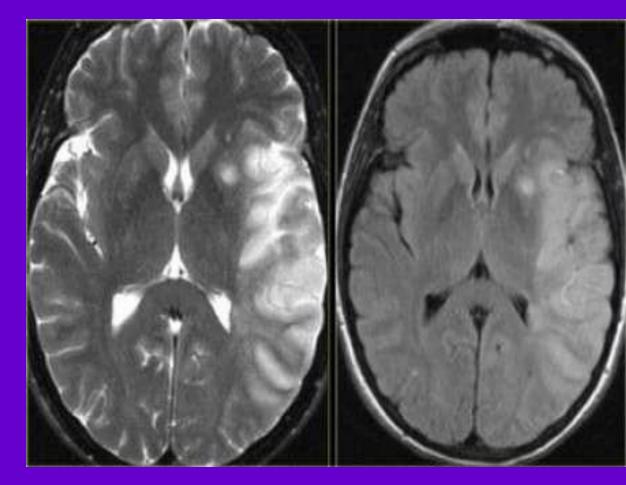




Role of MRI

- On T2WI and FLAIR infarction is seen as high SI.
- These sequences detect 80% of infarctions before 24 hours.
 - MR Hperintensity = CT Hypodensity

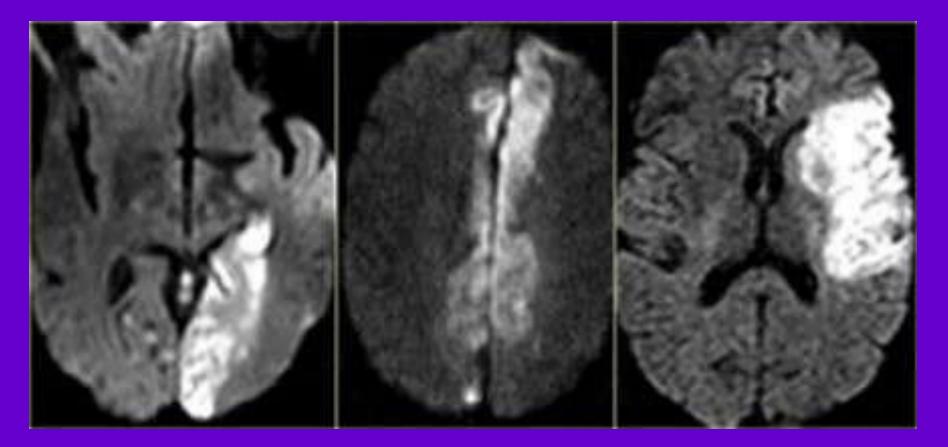
- T2WI and FLAIR demonstrating hyperintensity in the territory of the middle cerebral artery.
- Notice the involvement of the lentiform nucleus and insular cortex.



Diffusion Weighted Imaging (DWI)

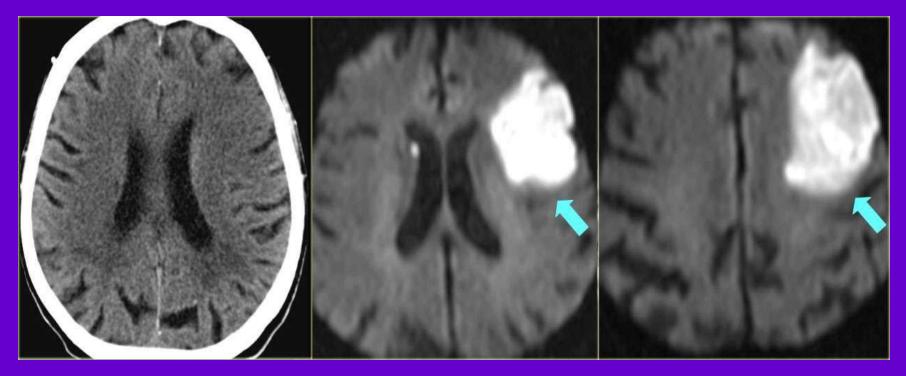
- DWI is the most sensitive sequence for stroke imaging.
- Also called Stroke sequence

Diffusion Weighted Imaging (DWI)



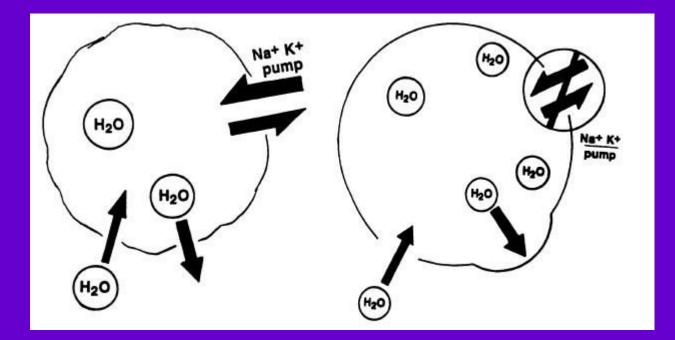
DWI in posterior, anterior and middle cerebral infarction

Diffusion Weighted Imaging (DWI)



- very subtle hypodensity and swelling in the left frontal region with effacement of sulci compared with the contralateral side.
- DWI shows marked superiority in detecting infarct

E Hyperacute Ischemic stroke- Cytotoxic edema



Lesion appears bright







Subacute

Moderate Bright

Towards Normal

Chronic

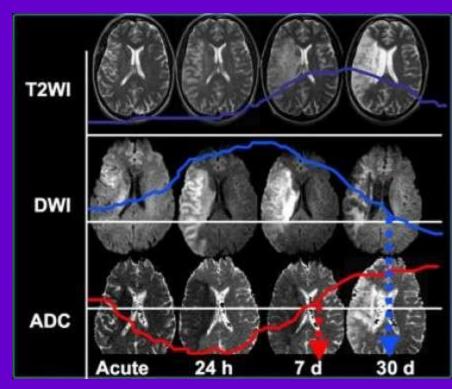
Mild Bright

Increased

PERSISTENT T2 SHINE THROUGH

Signal intensities on T2WI and DWI & ADC in time

- In the acute phase T2WI will be normal, but in time the infracted area will become hyperintense.
- The hyperintensity on T2WI reaches its maximum between 7 and 30 days. After this it starts to fade.
- DWI is already positive in the acute phase and then becomes more bright with a maximum at 7 days.
- DWI in brain infarction will be positive for approximately for 3 weeks after onset.
- ADC will be of low signal intensity with a maximum at 24 hours and then will increase in signal intensity and finally becomes bright in the chronic stage.



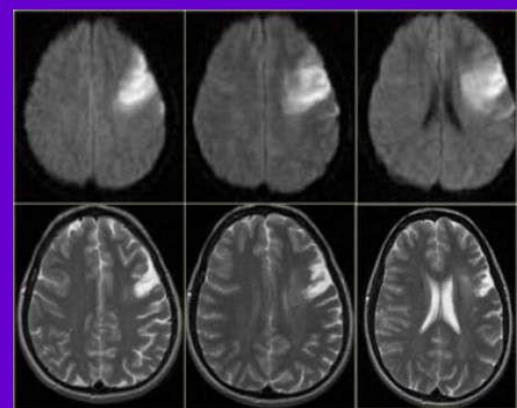
Pseudo-normalization of DWI

- This occurs between 10-15 days.
- DWI is normal.
- T2 WI shows subtle hyperintensity in rt. Occipital lobe.
- GD T1 shows gyral enhancement which suggest infarct.



Pitfall in DWI

- If we compare the DWI images in the acute phase with the T2WI in the chronic phase, we will notice that the affected brain volume in DWI is larger compared to the final infracted area (respectively 62cc and 17cc).
- So everything bright on DWI might not be irreversibly dead.



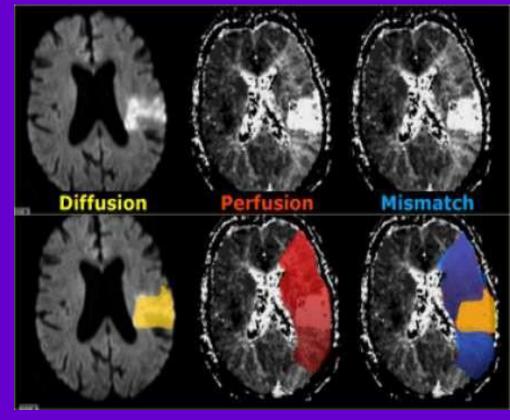
Perfusion MR Imaging

Technique

- Perfusion with MR is comparable to perfusion CT.
- A compact bolus of Gd-DTPA is delivered through a power injector.
- Multiple echo-planar images are made with a high temporal resolution.
- T2* gradient sequences are used to maximize the susceptibility signal changes.

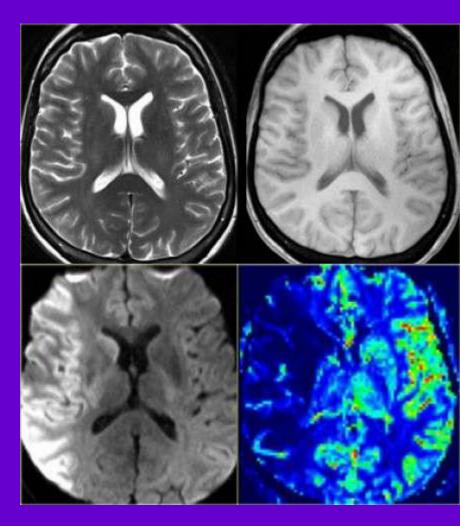
Identification of PENUBRA BY PMR

- On the left we first have a diffusion image indicating the area with irreversible changes (dead issue).
- In the middle there is a large area with hypoperfusion.
- On the right the diffusion-perfusion mismatch is indicated in blue.
- This is the tissue at risk(PENUMBRA).
- This is the brain tissue that maybe can be saved with therapy.



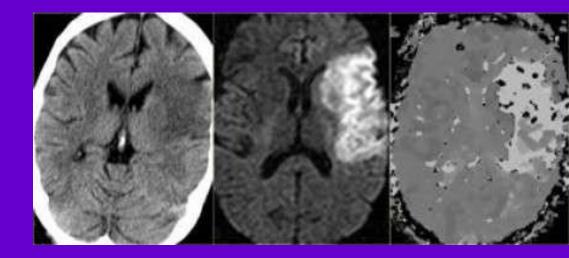
• Above images are normal and we have to continue with DWI.

- On the DWI there is a large area with restricted diffusion in the territory of the right middle cerebral artery.
- There is a perfect match with the perfusion images.
- so this patient should not undergo any form of thrombolytic therapy.



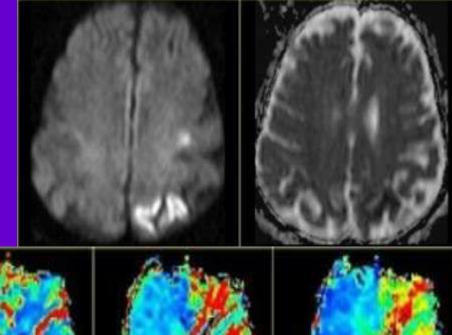
• On the left another MCA infarction.

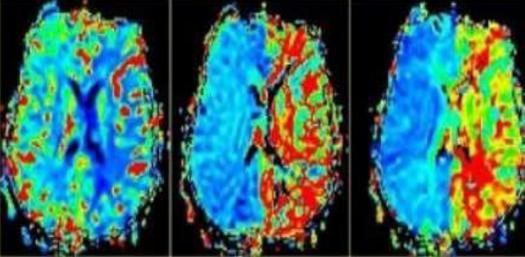
- It is clearly visible on CT (i.e. irreversible changes).
- There is a match of DWI and Perfusion, so no therapy.



The DWI and ADC map is shown which suggest infarct.

 perfusion images show that there is a severe mismatch.
Almost the whole left cerebral hemisphere is at risk due to hypoperfusion.
This patient is an ideal candidate for therapy.





Hemorrhagic Stroke

 Intracranial haemorrhage is a collective term encompassing many different conditions characterized by the extra vascular accumulation of blood within different intracranial spaces.

Hemorrhagic Stroke

Intra-axial haemorrhage

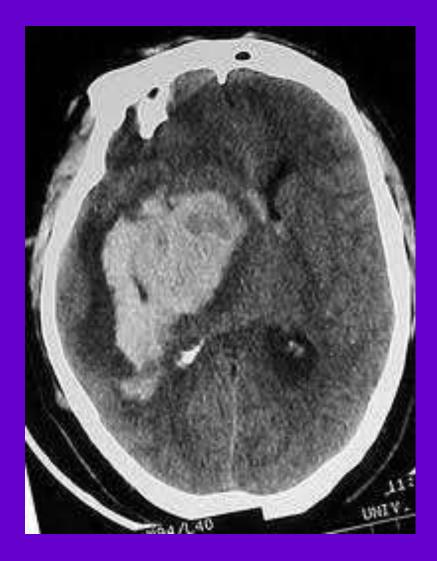
- intracerebral haemorrhage
- basal ganglia haemorrhage
- lobar haemorrhage
- pontine haemorrhage
- cerebellar haemorrhage
- Intraventricular haemorrhage (IVH)

extra-axial haemorrhage

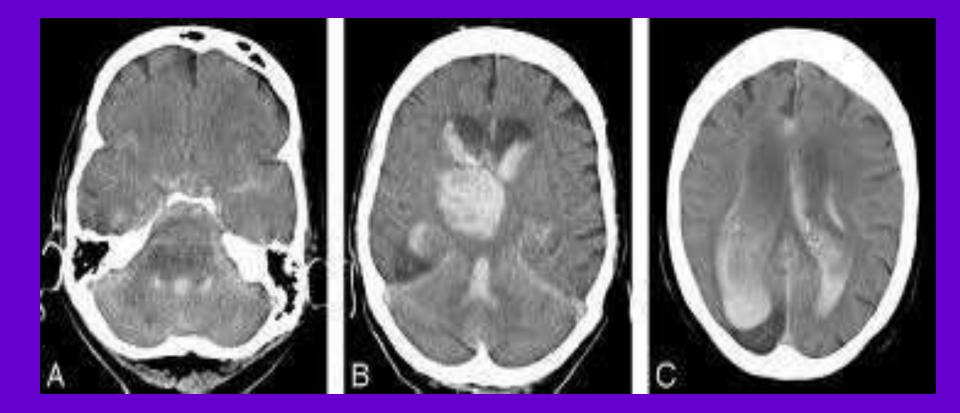
- extradural haemorrhage (EDH)
- subdural haemorrhage (SDH)
- subarachnoid haemorrhage (SAH)

Intracerebral Hemorrhage

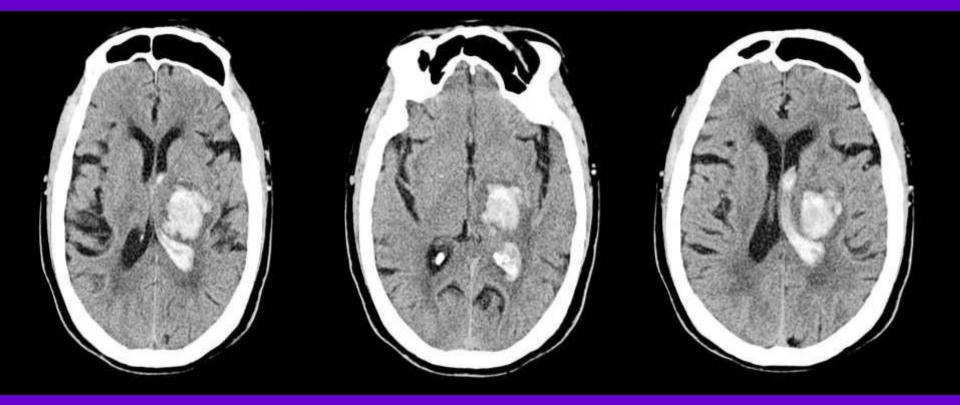
 Large intracerebral hemorrhage with midline shift



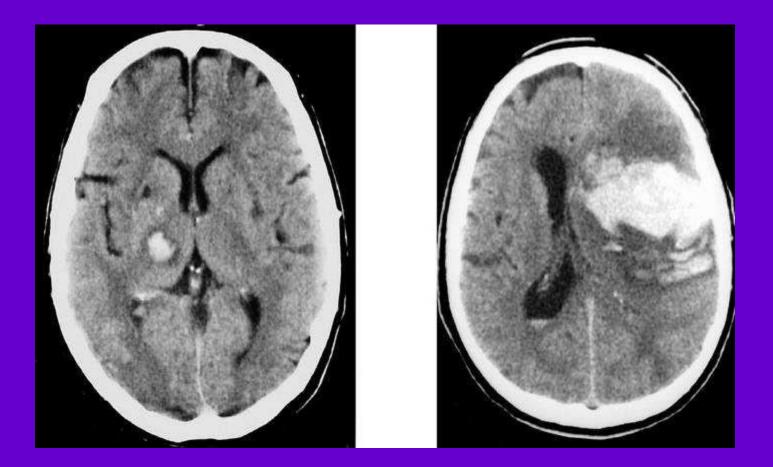
ICH with Intraventricular Extension



Basal Ganglia Hemorrhage with IC extension



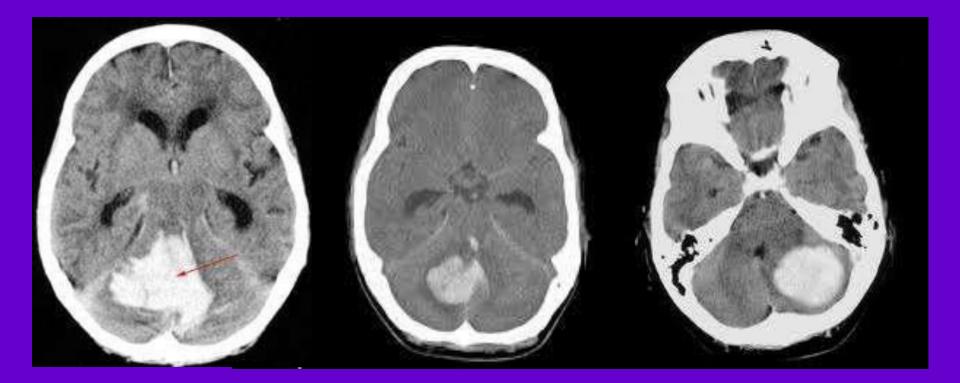
Lobar intracerebral hemorrhage.



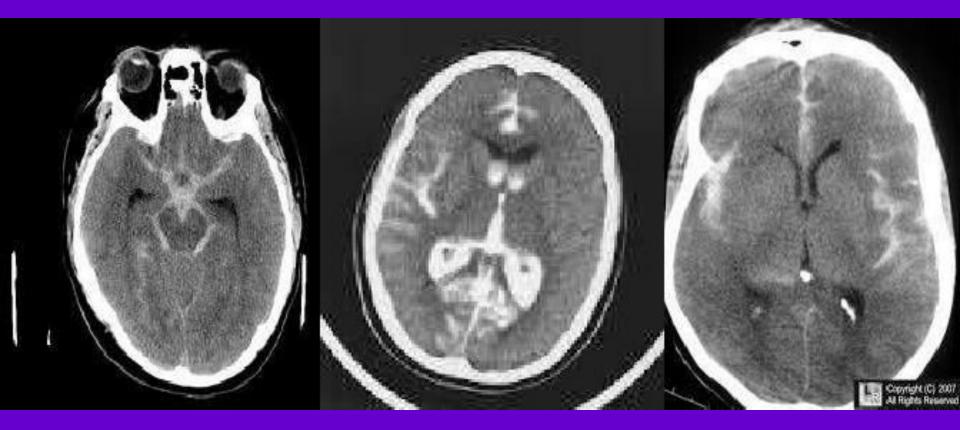
Pontine Hemorrhage



Cerebellar Hemorrhage

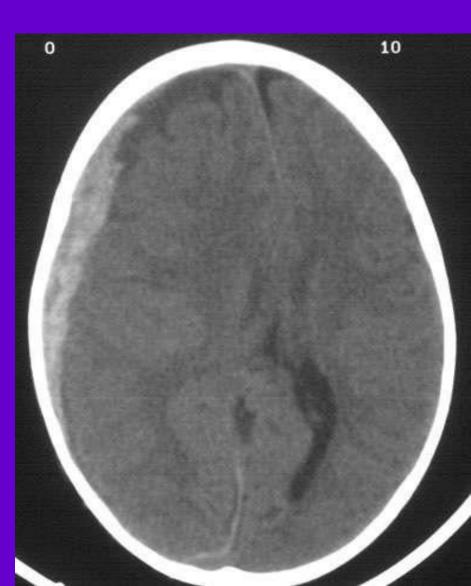


Subarachnoid Hemorrhage



Non Traumatic Subdural Hematoma

 Acute subdural hematoma. Note the bright (white) image properties of the blood on this non contrast cranial CT scan. Note also the midline shift.



References

- Acute stroke: usefulness of CT before starting thrombolytic therapy :by R von Kummer et al. Radiology 1997, Vol 205, 327-333,
- Early CT findings in Lentiform Nucleusby N Tomura et al Radiology 1988, Vol 168, 463-467
- State of the art imaging of acute stroke by Ashok Srinivasan et al RadioGraphics 2006;26:S75-S95
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THANK YOU