The Role imaging on stroke

Dr. Melita SpRad NKL (K)

RS PON Prof Dr.dr Mahar Mardjono Jakarta

PIT Neurororadiologi XX1 , May 22 , 2025 , Makassar

STROKE

- Acute episodic neurological deficit caused by ischemia or hemorrhage in brain
- World Health Organization (WHO) defines stroke as the sudden onset of neurological symptoms attributable solely to a brain disorder and caused by a circulatory disorder lasting more than 24 h

- TIA (transient ischemic attack) is caused by a temporary clot , focal neurological deficit that resolves in 24hrs

Stroke facts

Stroke is the second leading cause of death and the third leading cause of disability worldwide

Each year, nearly 795 000 people experience a new or recurrent stroke

A stroke happens every 40 seconds

Every 4 minutes someone dies from stroke

Stroke is a major cause of morbidity and permanent disability

Benjamin EJ., et al. *Circulation* 2019;139:e56–e66.
Johnston SC., et al. *Lancet Neurol* 2009;8:345–354.

Stroke facts

No 1 cause of death in Indonesia (2019)

Majority are ischemic in origin

Neuro imaging is to identify potentially salvageable brain tissue for the aim of extension of time window for safe and effective treatment

- Di Indonesia **stroke** menjadi penyebab kematian utama yaitu **19,42%** dari total kematian berdasarkan IHME tahun **2019** dan berdasarkan hasil riskesdas prevalensi stroke di Indonesia meningkat dari 7/1000 penduduk pada tahun 2013 menjadi 10,9/1000 penduduk pada tahun 2018.

- (Direktur Pengendalian dan Pencegahan Penyakit Tidak Menular, Dr. Eva Susanti, S.Kp., M.Kes)

MENTERI KESEHATAN
REPUBLIK INDONESIA

KEPUTUSAN MENTERI KESEHATAN REPUBLIK INDONESIA
NOMOR HK.01.07/MENKES/1336/2023
TENTANG
RUMAH SAKIT JEJARING PENGAMPUAN PELAYANAN STROKE

DENGAN RAHMAT TUHAN YANG MAHA ESA

MENTERI KESEHATAN REPUBLIK INDONESIA,

Menimbang : a. bahwa stroke sebagai salah satu penyakit katastropik yang membutuhkan perawatan medis yang lama dan berbiaya tinggi memiliki angka kesakitan (morbiditas) dan angka kematian (mortalitas) yang masih tinggi, sehingga diperlukan optimalisasi pelayanan dengan meningkatkan kemampuan sumber daya, penatalaksanaan, dan rujukan melalui jejaring pengampuan pelayanan rumah sakit;

b. bahwa Keputusan Menteri Kesehatan Nomor HK.01.07/MENKES/1948/2022 tentang Rumah Sakit Jejaring Pengampuan Pelayanan Stroke sudah tidak sesuai dengan kebutuhan pelayanan kesehatan dalam pelaksanaan rumah sakit jejaring pengampuan pelayanan stroke sehingga perlu diganti;

c. bahwa berdasarkan pertimbangan sebagaimana dimaksud dalam huruf a dan huruf b, perlu menetapkan Keputusan Menteri Kesehatan tentang Rumah Sakit Jejaring Pengampuan Pelayanan Stroke;

Mengingat : 1. Undang-Undang Nomor 29 Tahun 2004 tentang Praktik Kedokteran (Lembaran Negara Republik Indonesia Tahun 2004 Nomor 116, Tambahan Lembaran Negara Republik Indonesia Nomor 4431);

Jakarta Sebagai Pusat Rujukan Nasioal Penyakit Otak dan Sistem Persyarafan;

MEMUTUSKAN:

Menetapkan : KEPUTUSAN MENTERI KESEHATAN TENTANG RUMAH SAKIT JEJARING PENGAMPUAN PELAYANAN STROKE.

KESATU : Menetapkan Rumah Sakit Jejaring Pengampuan Pelayanan Stroke.

KEDUA : Rumah sakit jejaring pengampuan pelayanan stroke sebagaimana dimaksud dalam Diktum KESATU, terdiri atas:
a. rumah sakit pengampu, dengan stratifikasi kemampuan paripurna dan utama; dan
b. rumah sakit diampu, dengan stratifikasi kemampuan utama dan madya;

KETIGA : Dalam rangka penyelenggaraan jejaring pengampuan pelayanan stroke dapat terlaksana secara komprehensif, efektif, efisien, dan memenuhi indikator pengampuan, menunjuk Rumah Sakit Pusat Otak Nasional Prof. Dr. dr. Mahar Mardjono Jakarta sebagai koordinator jejaring pengampuan pelayanan stroke.

KEEMPAT : Ketentuan mengenai stratifikasi kemampuan pelayanan, tugas rumah sakit jejaring pengampuan pelayanan stroke termasuk koordinator jejaring pengampuan pelayanan stroke, indikator keberhasilan pengampuan, dan daftar rumah sakit jejaring pengampuan pelayanan stroke tercantum dalam Lampiran yang merupakan bagian tidak terpisahkan dari Keputusan Menteri ini.

KELIMA : Dalam rangka mendukung jejaring pengampuan pelayanan stroke, pemerintah daerah membuat pernyataan komitmen dan/atau nota kesepahaman dukungan terhadap rumah sakit di daerahnya, meliputi dukungan terhadap pemenuhan sumber daya manusia, sarana dan prasarana, dan dukungan lainnya.

CODE Stroke

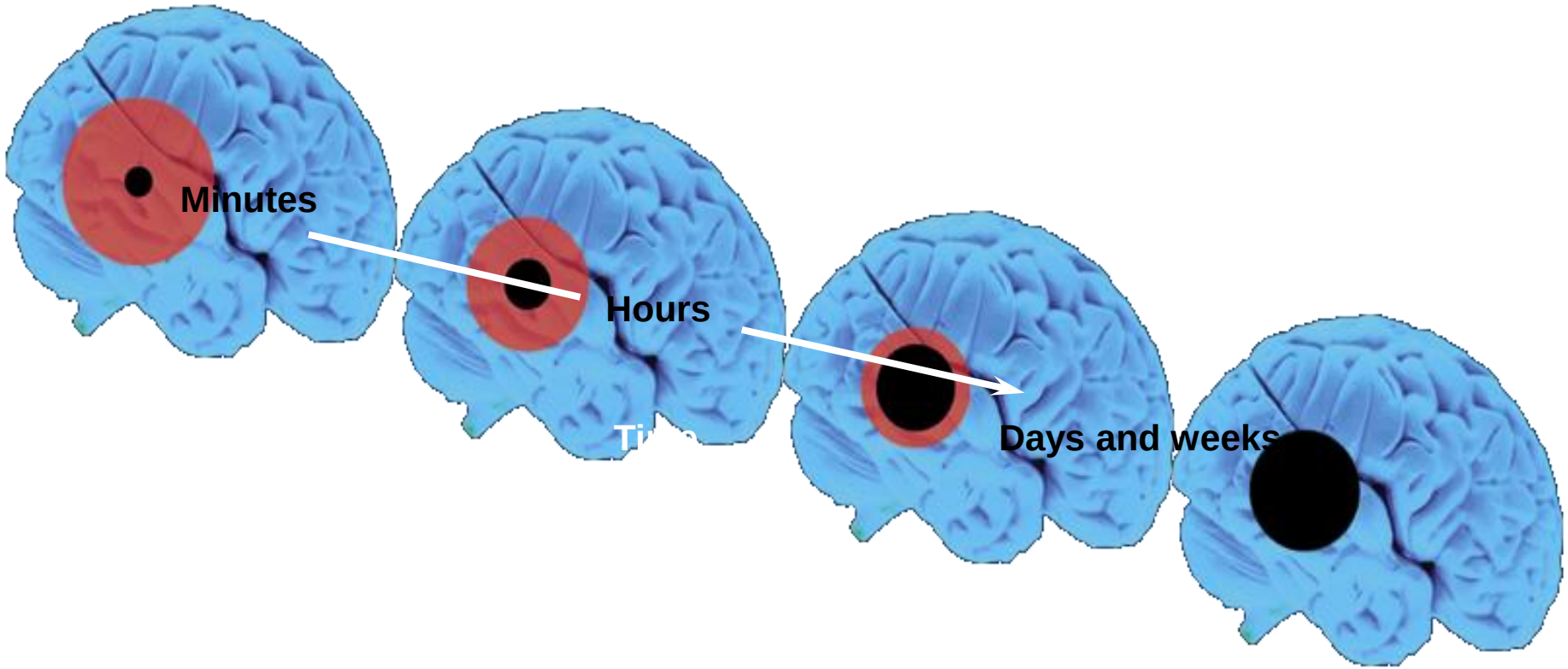
The Code Stroke Emergency Response refers to a coordinated team based approach to stroke patient care that requires rapid and accurate assessment, diagnosis, and treatment in an effort to save the brain and minimize permanent damage.

(The Code Stroke handbook 2022)

- Estimated that nearly two million neurons die each minute that elapses during the evolution of an average acute ischemic stroke
- Each hour without treatment the brain loses on average as many neurons as 3.6 years of normal aging

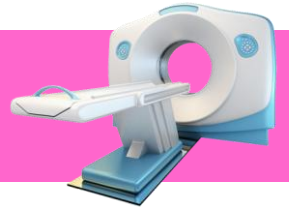
“time is brain.”

The transition from ischemia to irreversible infarction depends on both the severity and the duration of the diminution of blood flow



Emergency department response times NINDS recommendations

Door-to-MD	10 minutes
Door-to-Stroke Team Notification	15 minutes
Door-to-CT Scan	25 minutes
Door-to-Needle (80% threshold)	60 minutes
Door-to-Admission	3 hours



Bock. NINDS Stroke symposium proceedings 1996; Updated 2011.

CODE Stroke Team RS PON

- Nurse
- GP Emergency room
- Neurologist
- Neurosurgeon
- Radiologist
- Pharmacy
- Laboratory

- Door to Practioner: < 10 min.
- Door to Stroke Team: < 15 Min.
- Door to CT scan: < 25 min.
- Door to CT Interpretation:< 20 min.
- Door to Needle : < 60 min

Sumber: *Riskesdas 2007 & **Riskesdas 2013
***World Stroke Organization

WASPADA STROKE MENGINCAR ANDA

KEMENTERIAN KESEHATAN REPUBLIK INDONESIA

GERMAS Gerakan Masyarakat Hidup Sehat

Asian Games Jakarta Palembang 2018

TIPS MUDAH MENGENALI
GEJALA DAN TANDA-TANDA STROKE

Ingat Slogan
SeGeRa Ke RS

Senyum tidak simetris (mencong ke satu sisi), tersedak, sulit menelan air minum secara tiba-tiba

Se

Gerak separuh anggota tubuh melemah tiba-tiba

Ge

bica**Ra** pelo / tiba-tiba tidak dapat bicara / tidak mengerti kata-kata / bicara tidak nyambung

Ra

Kebas atau baal, atau kesemutan separuh tubuh

Ke

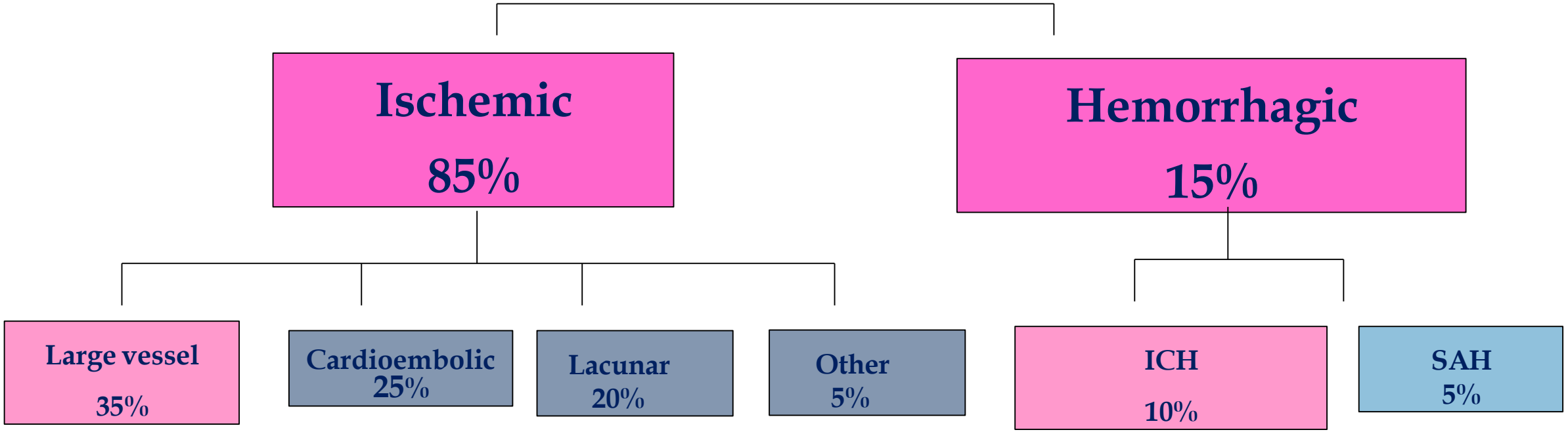
Rabun, pandangan satu mata kabur, terjadi tiba-tiba

R

Sakit kepala hebat yang muncul tiba-tiba dan tidak pernah dirasakan sebelumnya, Gangguan fungsi keseimbangan, seperti terasa berputar, gerakan sulit dikoordinasi.

S

Types of Stroke



Ischemic Stroke

- **The most common cause of stroke**

Common etiologies

- Atherosclerosis
- Cardiogenic
- Hemodynamic
- Cryptogenic

Less common etiologies

- Vasculopathies
- Immune-related diseases
- Hypercoagulable states
- Arterial dissection
- Global hypoperfusion
- Venous infarction
- Mitochondrial disorders

GOALS OF IMAGING

- To establish the diagnosis **as early as possible**
- Define **location, extent and age of infarct**
- Give accurate information about **intracranial vasculature and brain perfusion** for guidance in selecting the appropriate therapy
- To identify the **penumbra**
- Rule in or out **other disease processes**

ROLE OF NON CONTRAST CT IN ACUTE STROKE

- Identify haemorrhage
- Hyperacute Stroke Imaging Signs /Early Ischemic Changes sign (EICs)

  - Hyperdense vessel sign
  - Insular Ribbon sign
  - Obscuration of lentiform nucleus
  - Focal hypodense area : cortical, subcortical or deep GM/WM → vascular territory / watershed area

Imaging Workup of Acute Ischemic Stroke

- **Goals in acute stroke imaging are to assess the 4 "Ps" :**

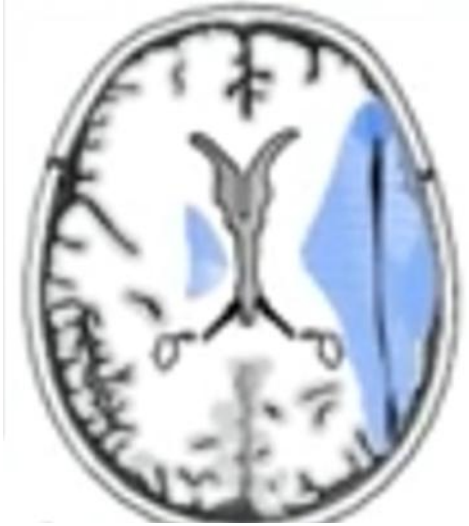
New Goals of Acute Stroke Imaging

1. P : Parenchyma : Assess early signs of acute stroke, rule out hemorrhage
2. P : Pipes : Assess extracranial circulation (carotid and vertebral arteries of the neck) and intracranial circulation for evidence of intravascular thrombus
1. C : Collaterals : Collateral circulation status assessment
2. C : Core : Core/Penumbra size estimation: Assess tissue at risk of dying if ischemia continues with out re-canalization of intravascular thrombus
3. C : Clot : Clot assessment

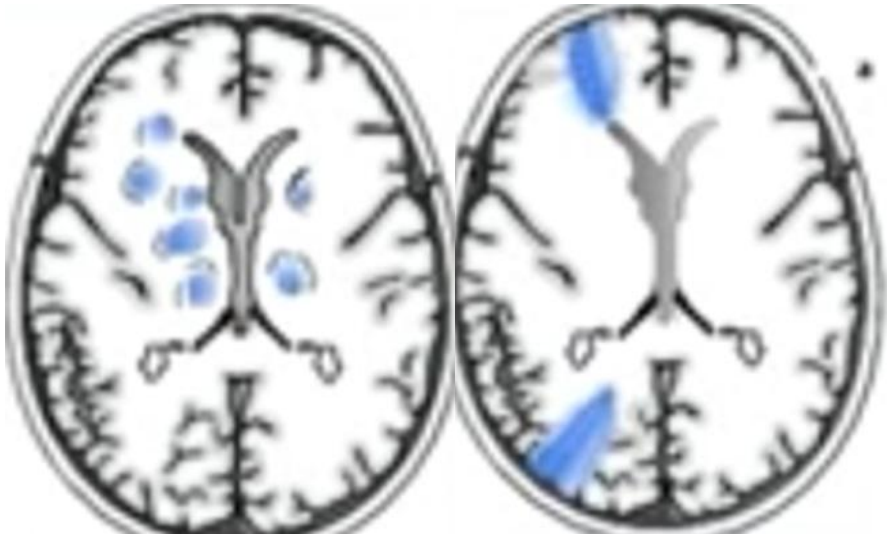
European Journal of Radiology 96 (2017) 162–172

Arterial Stroke Location

- Large Vessel → Territorial infracts
  - wedge shape
  - extends to cortex
  - follows defined vascular distribution



- Small Vessel → Lacunar /Hemodynamic infarcts
  - white matter
  - Small lesions/ Lacunar (deep brain)
  - Watershad areas



Watershed Stroke /infarct

- Ischemic strokes that occur in the brain tissues bordering two main arteries (border zones) :

  Anterior cerebral artery (ACA) & Middle cerebral artery (MCA) & Posterior cerebral artery (PCA)

- 2 Type :
- **Cortical and subcortical watershed** strokes → depending on where they occur in the border zones

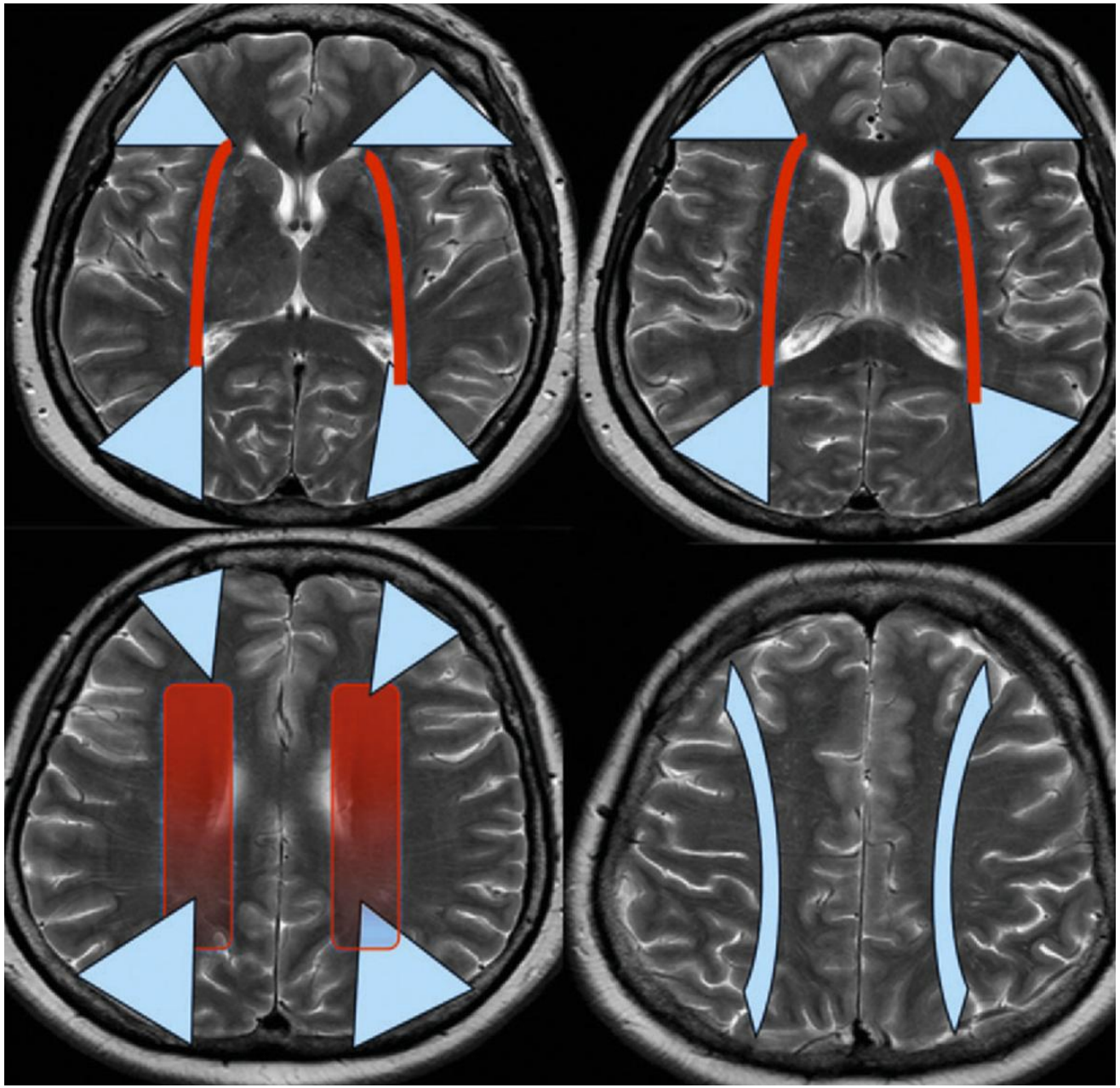
Type of watershed infarct

- **Cortical watershed (CWS) /Inner brain**

  Located between the cortical territories of the ACA, MCA and PCA

- **Subcortical watershed / Internal watersheds (IWS) brain**

  Located in the white matter white matter along and slightly above the lateral ventricle , between the deep and the superficial arterial systems of the MCA, or between the superficial systems of the MCA and ACA



Cortical watershed

Frontal (beteween ACA, MCA)
- Parietooccipital (MCA, PCA)
- Triple WS (ACA, MA , PCA)

Also includes vertex subcortical WM

Subcortical watershed

Between penetrating small arteries
- Lenticulostriate
- Thalamo perforatting
- Medullary WM perforating
- Choroidal arteries
- Penetrating branches of major arteries

Pathogenesis Watershed Infarct

- Systemic hypotension
- Micro emboli
- Severe arterial stenosis
- ICA occlusion
- or a combination of these

Cortical / External Border Zone Infarcts

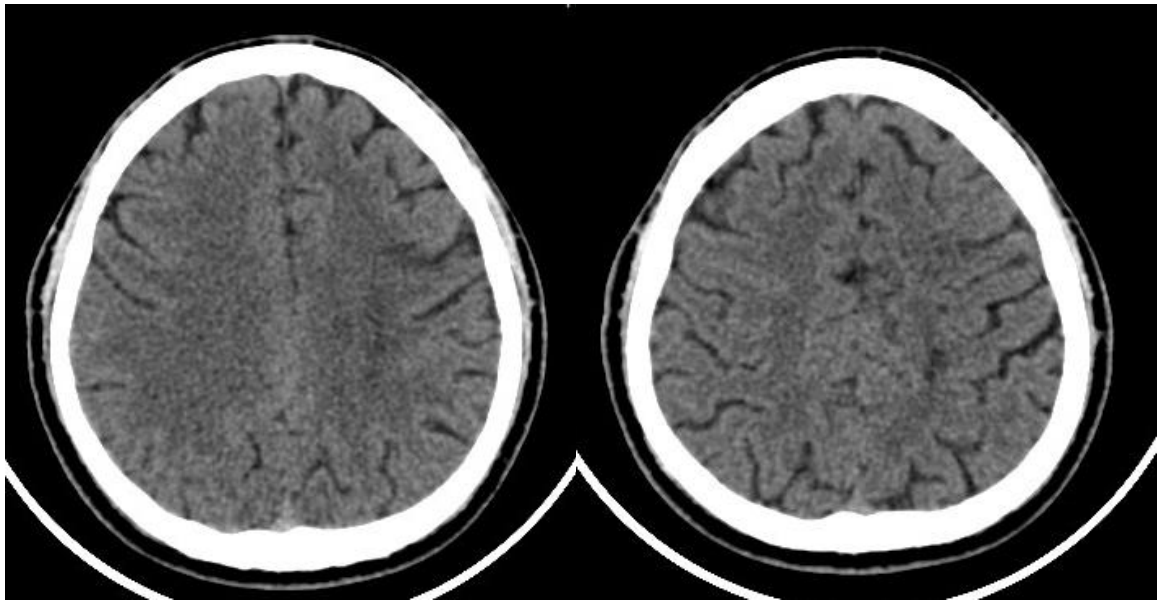
- Located at the junctions of the anterior, middle and posterior cerebral artery territories
- Wedge shaped or ovoid shaped
- Size is variable because of anatomic variation and minimal/maximal distribution of each of the large vessel territories (MCA, ACA, PCA) from person to person
- Moderate or severe narrowing of the carotid or proximal cerebral arteries
- Recent intraplaque hemorrhage/plaque rupture may play a role
- Micro emboli from the heart or atheroembolic events can propagate to the cortical border zones → these areas have a lower perfusion than more proximal regions, so less ability to "wash out" the micro emboli

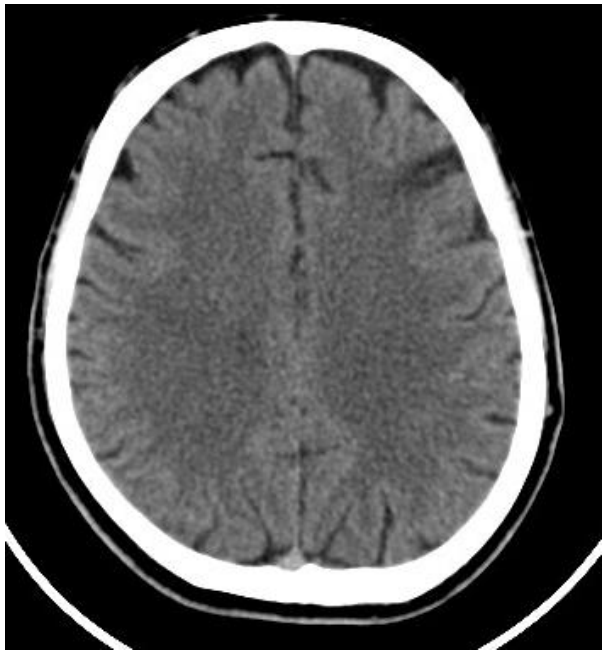
Subcortical / Internal border zone infarcts

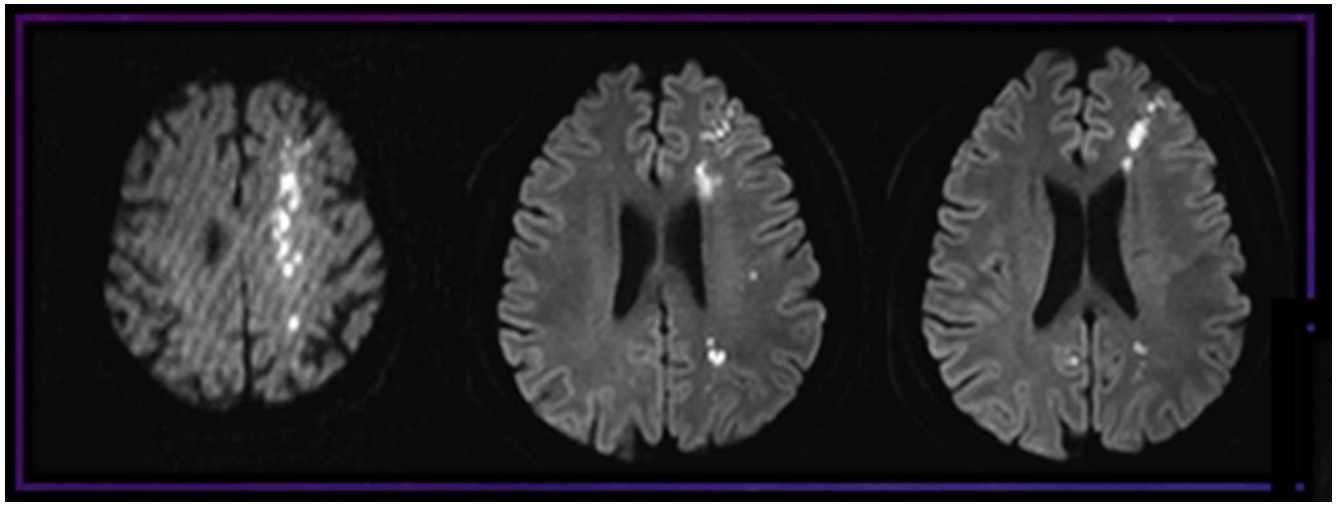
- Located at the junctions of the anterior, middle and posterior cerebral artery territories with the Recurrent Artery of Heubner, lenticulostriate and anterior choroidal artery territories
- Manifests as :
  - Series of 3 or more lesions, each with a diameter of 3 mm or more
  - A linear fashion parallel to the lateral ventricle in the centrum semiovale or corona radiata (rosary/bead)
  - Can be confluent or partial

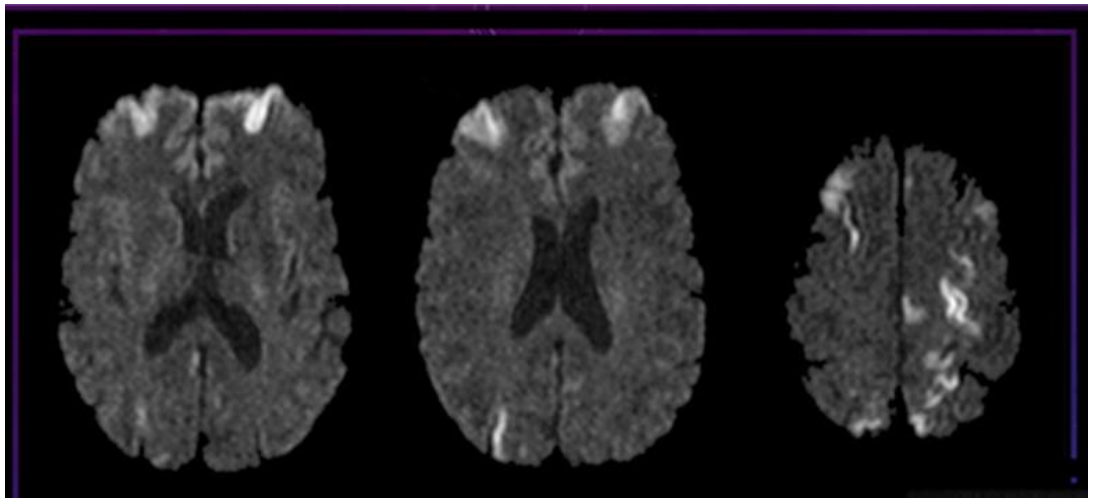
    Partial infarct : short episode of hemodynamic compromise

    Confluent infarct : longer period of hemodynamic compromise ec. by arterial stenosis or occlusion
  - Less likely than external border zone infarcts to be secondary to emboli
  - Poor prognosis and clinical deterioration when compared with cortical/external border zone infarcts



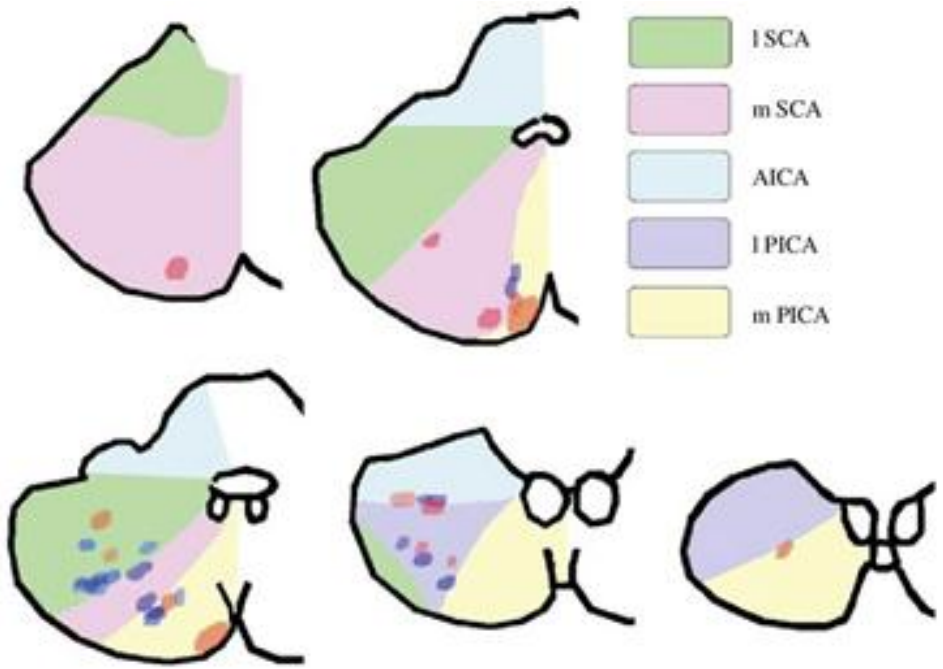


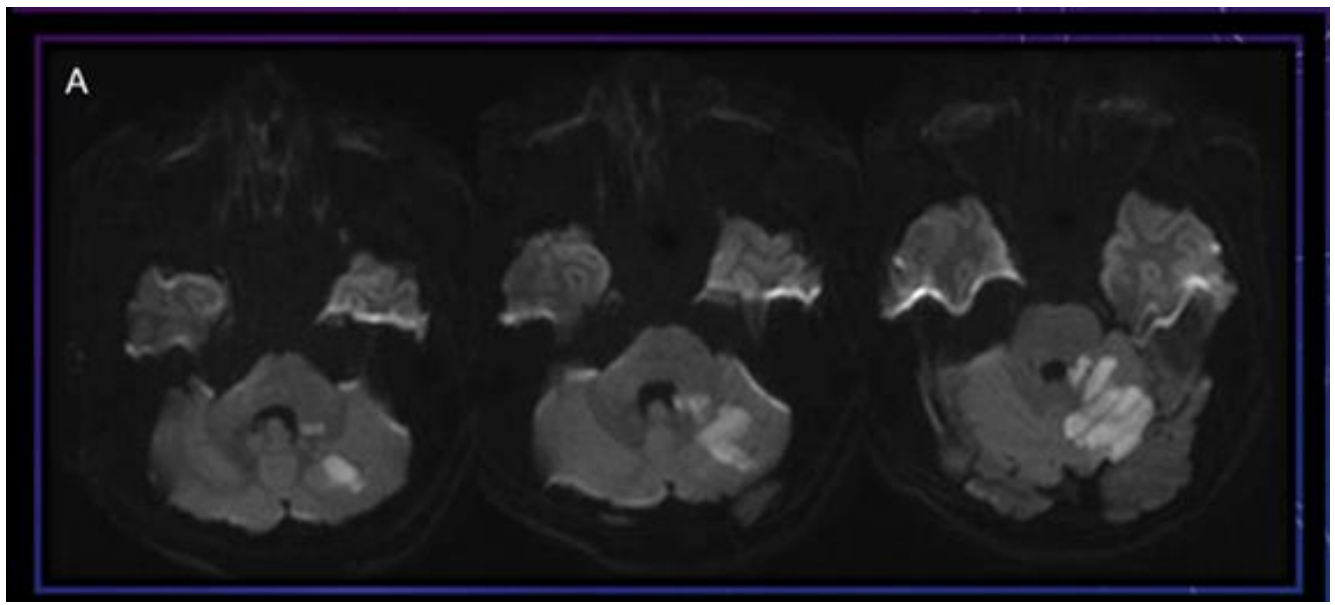


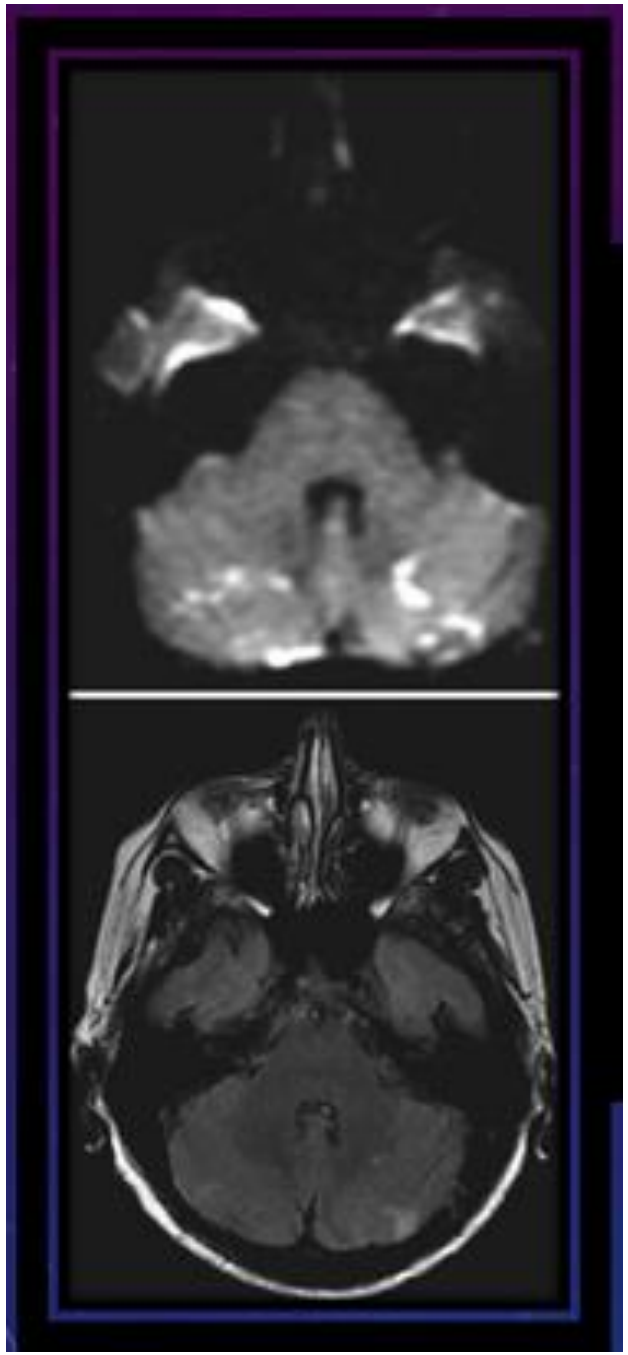


Border Zone Infarcts in the Cerebellum

- Usually < 2 cm in size and are seen at the borders of the AICA, SCA and PICA (and their branches)
- Due to stenosis or embolism. Embolic events can come from the heart or atherosclerotic disease
- May be see in vertebral dissections
- Typically not disabling in an of themselves, but often coexist with large territorial lesions
- They can be silent if small, or similar in presentation to larger cerebellar infarcts, with vertigo and ataxia


l SCA
m SCA
AICA
l PICA
m PICA


A



CT scan

STANDARD DIAGNOSTIC for Acute Stroke

(2018 AHA/ASA Guideline for AIS)

- CT >> available
- Non Contrast CT seen in 3-6 hours after onset stroke with an

Early ischemic changes (EICs)

- Sensitive for acute ischemic stroke 3- 6 hours onset stroke :

  45% and 88% (mean 55.3%)

- Sensitive >> for haemorrhagic (ICH / Vascular malformation)
- Exclusion of cases that could resemble stroke (Mimic Stroke/Strokelike syndr)

Early Ischemic Changes sign (EICs) on NCCT

- Hyperdense artery ec. clot of thrombus >> proximal artery
  Medial Cerebral A (HMCA) and Basilar art (HBA)

➢Early signs of cytotoxic edema:

Loss of GM-WM differentiation & hypoattenuation of deep nuclei :

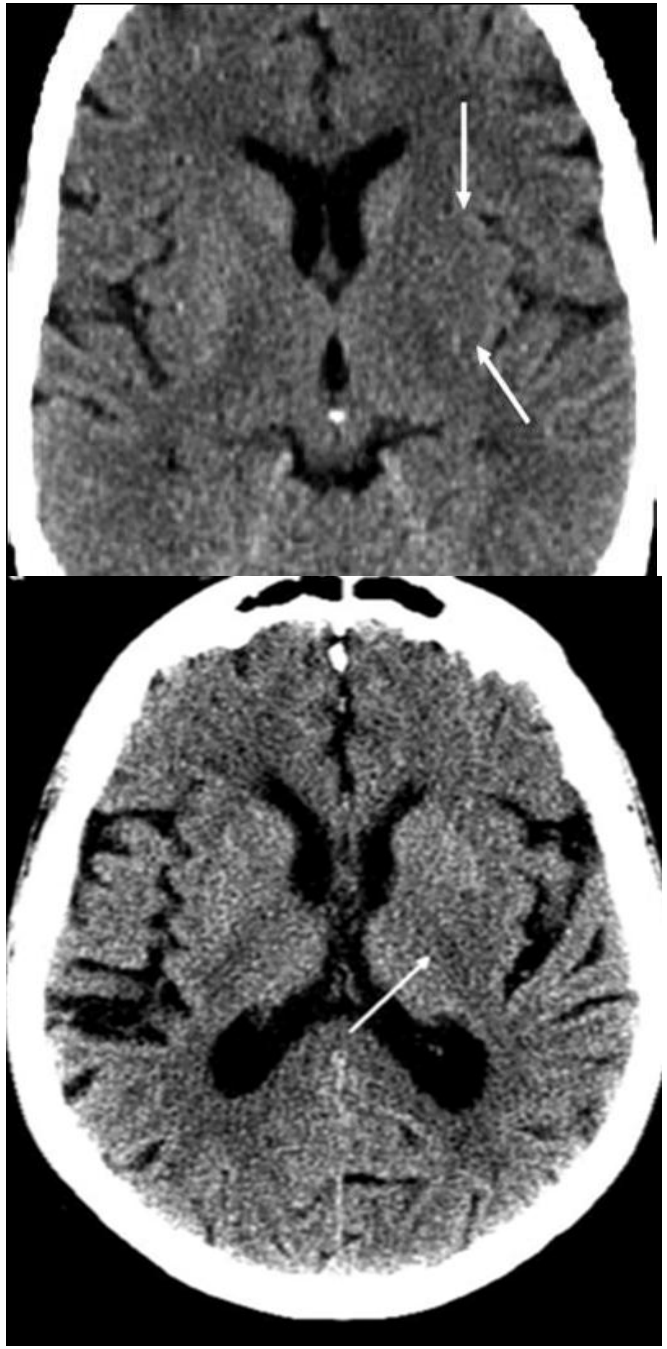
- Insular (insular ribbon sign)
- Cortical gyrus (gyral effacement)
- Basal ganglia (lentiform nucleus)

➢Focal hypodense area : cortical, subcortical or deep GM/WM vascular territory / watershed area

Stage of stroke

Time	Pathology
Hyperacute (< 6 hrs)	**- Vascular stasis/ thrombosis - Diffusion restricted - Early edema**
Acute (6 hrs -3/4 days)	**- Edema**
Subacute (days – 3 mnth)	**- BB Disruption - Edema - Enhancement - Hemorrhage**
Chronic (> 3 month)	**- Gliosis - Encephalomalacia - Atrophy**

TIME	CT
Minute	No Change / Normal
6 Hrs	Insular Ribbon sign Hyperdens vessel sign
12 Hrs	Sulcal effacement +/- Decrease attenuation
12- 24 Hrs	Decrease attenuation
7 Days	Maximum swelling
20 Days	Gyral enhacement



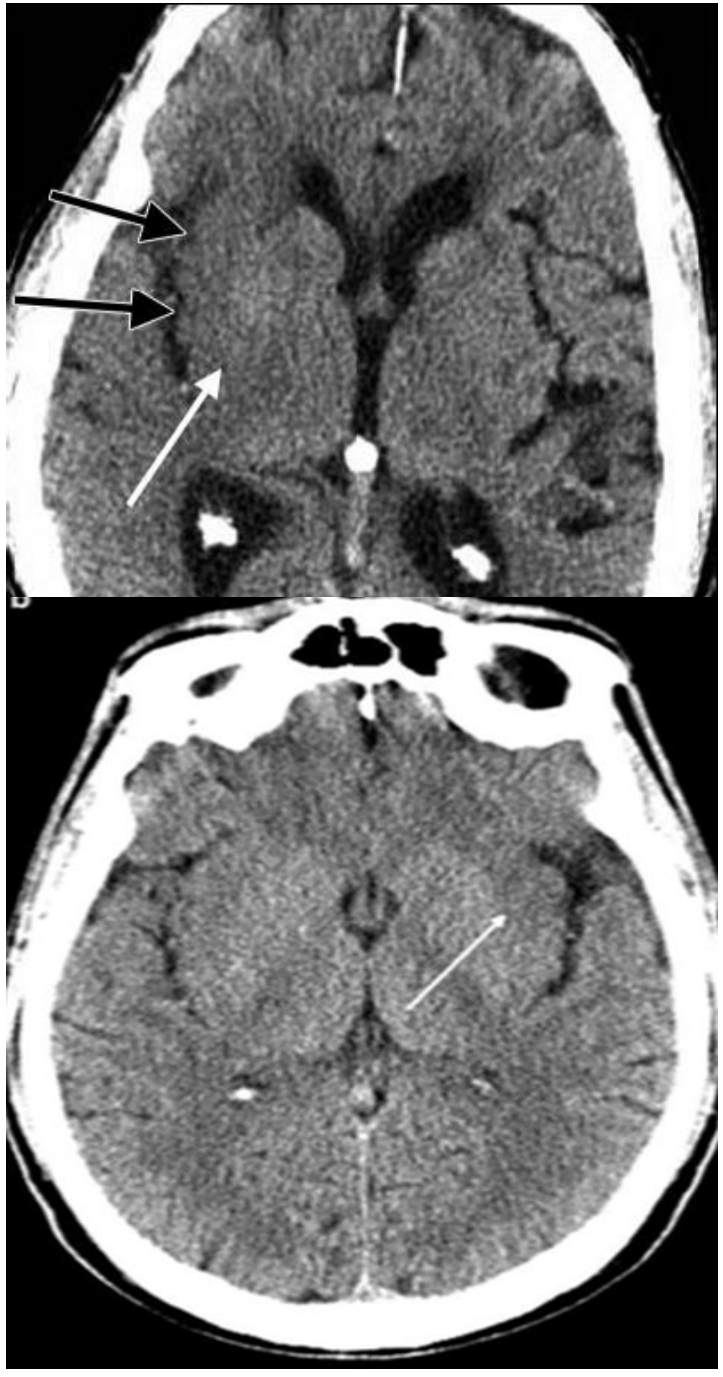
OBSCURATION OF LENTIFORM NUCLEOUS

- Lentiform nucleus appears hypo attenuated because of acute ischemia of the lenticulostriate territory , resulting in obscuration of the lentiform nucleus

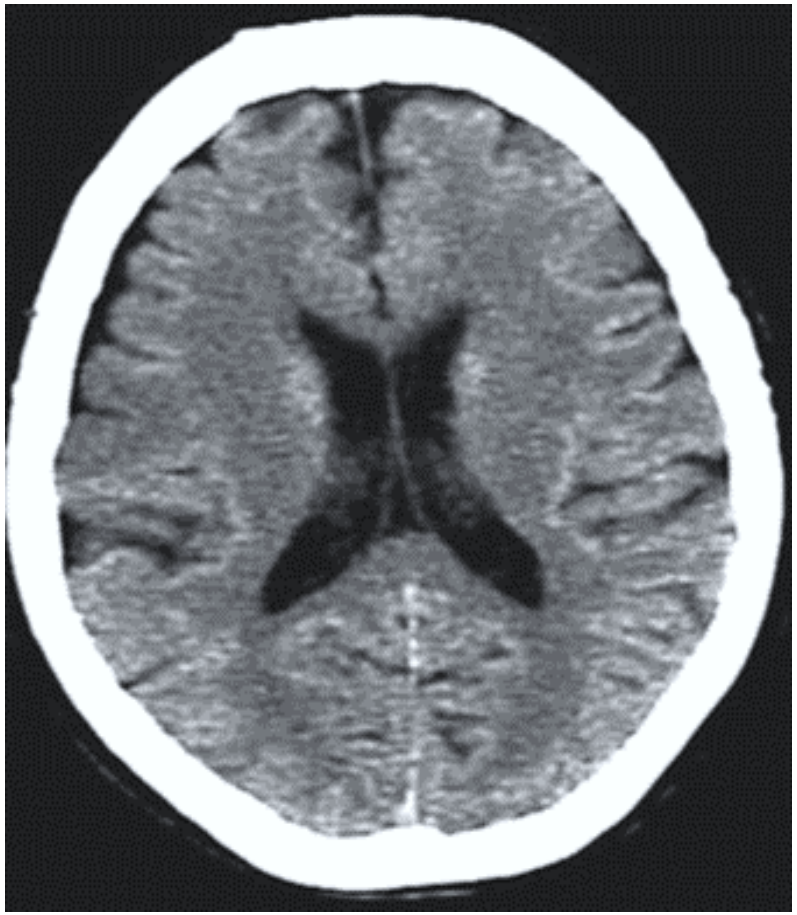
- May be seen on CT images within 2 hours after the onset of a stroke

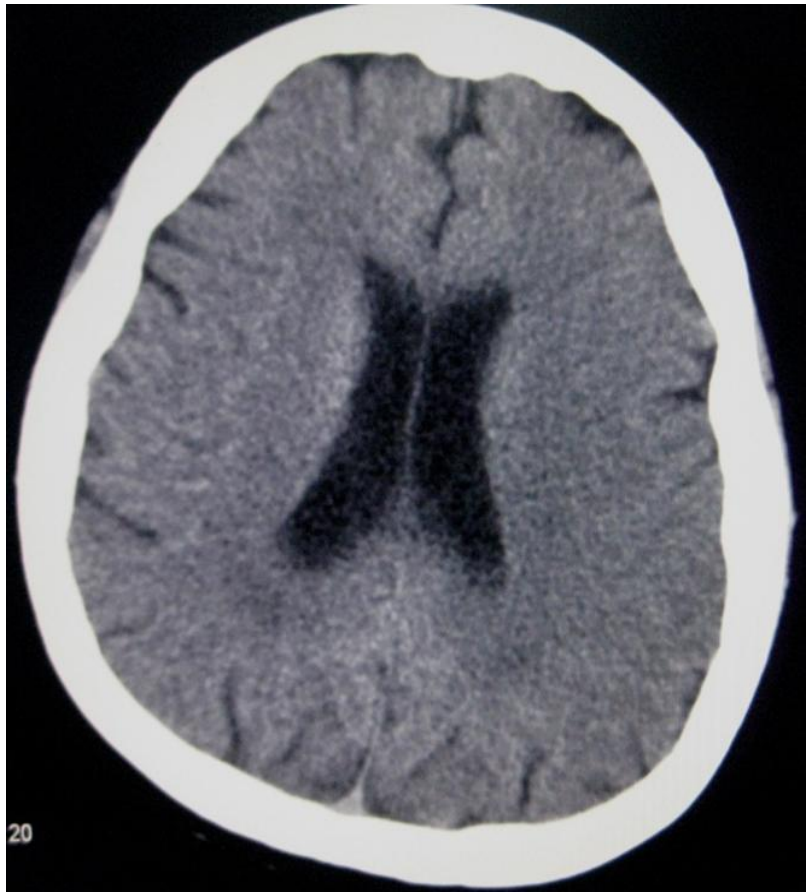
INSULAR RIBBON SIGN

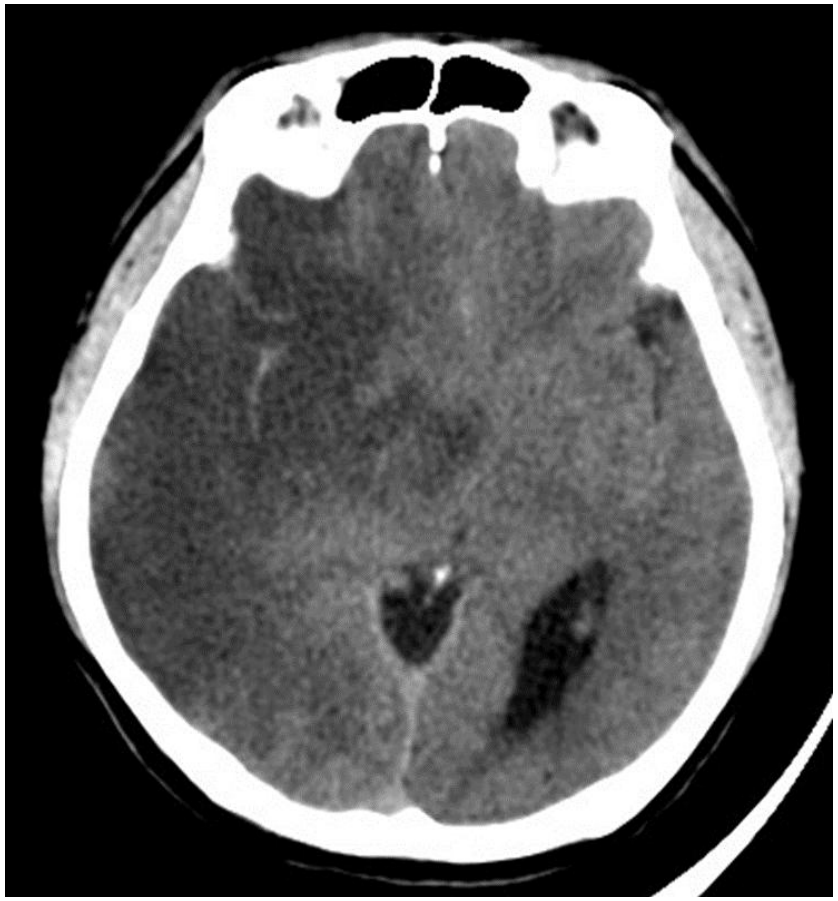
- Local hypoattenuation of the insular cortex region due to cytotoxic edema as this region is susceptible to early and irreversible ischemic damage



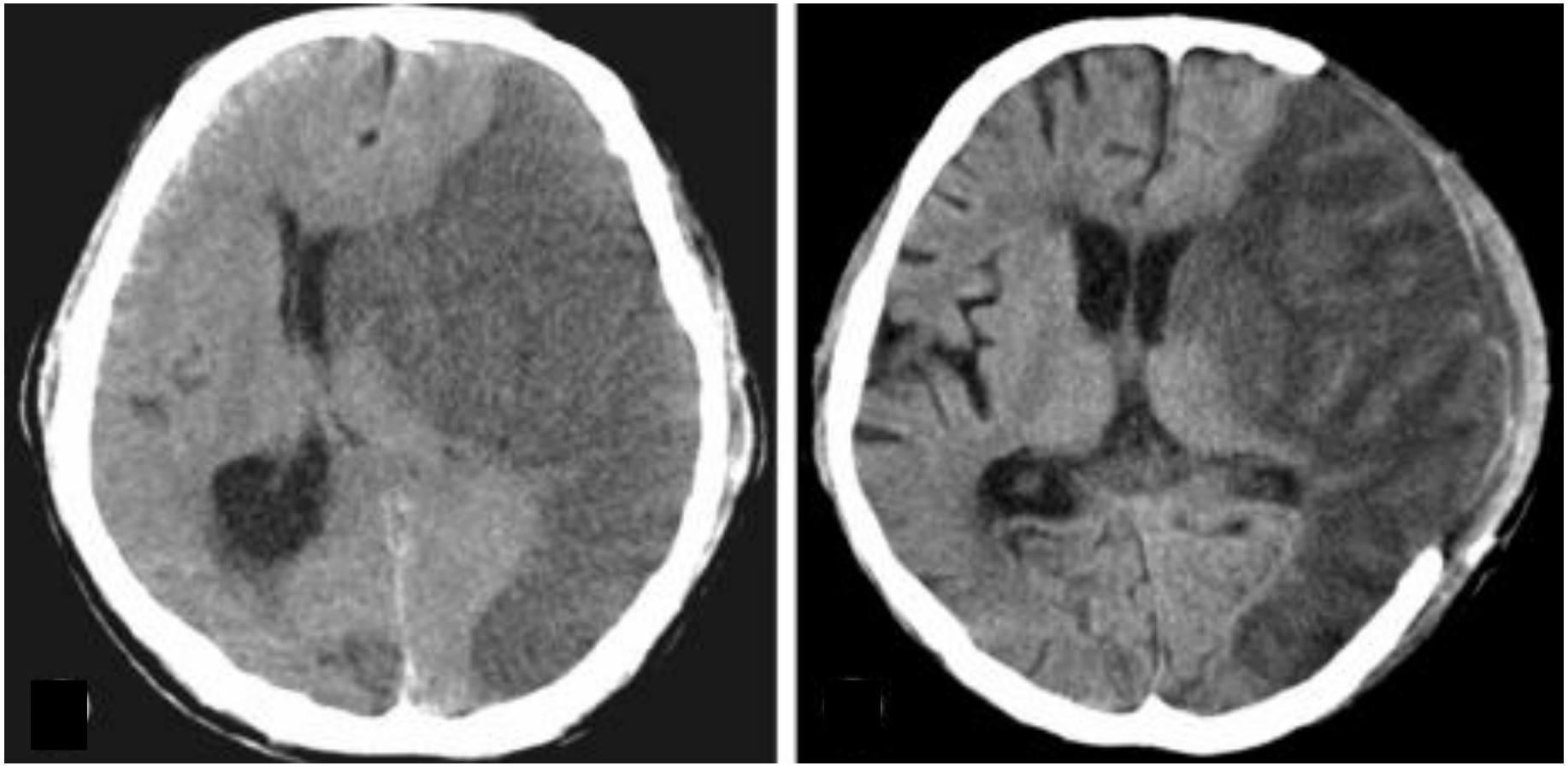
Focal hypodense area



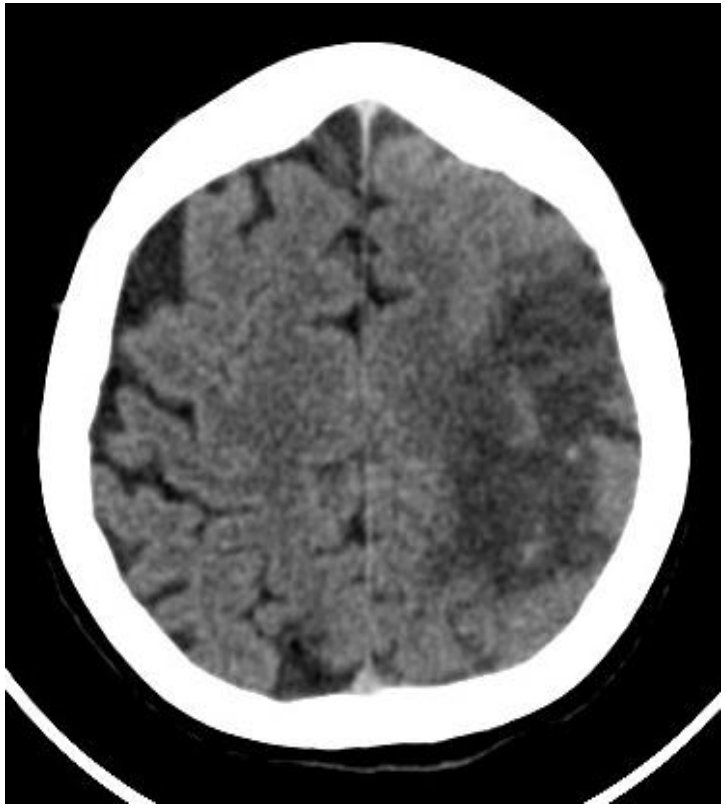


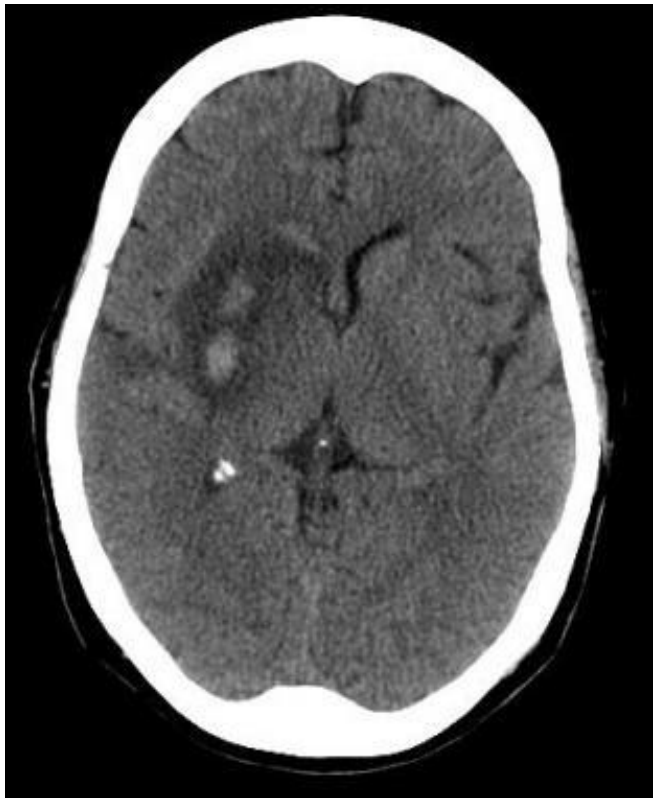


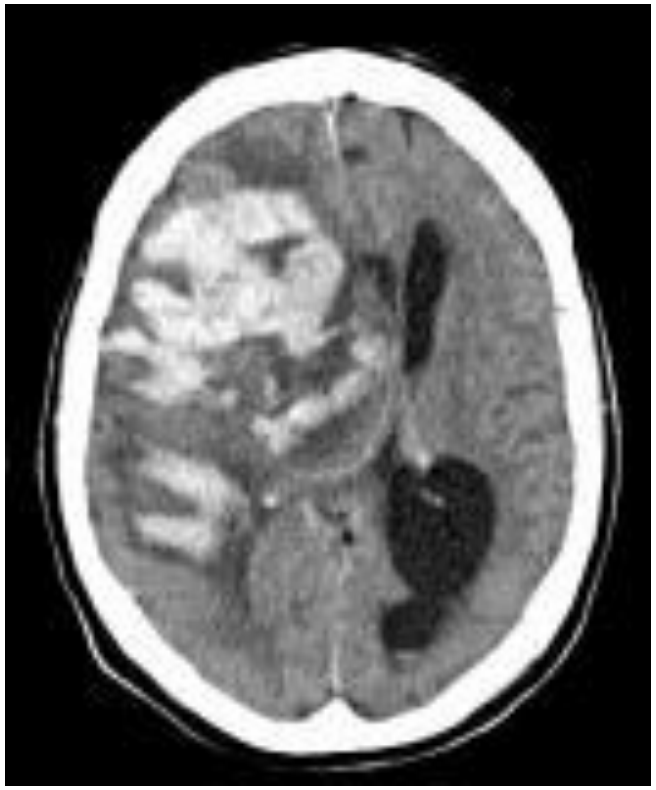
Subacute stroke
Mass effect



Subacute with haemorrhagic transformation







Hemorrhagic infarction type 1 (HI-1)—small petechiae along the margins of the infarct

Hemorrhagic infarction type 2 (HI-2)—more confluent petechiae within the infarcted area, but without space-occupying effect

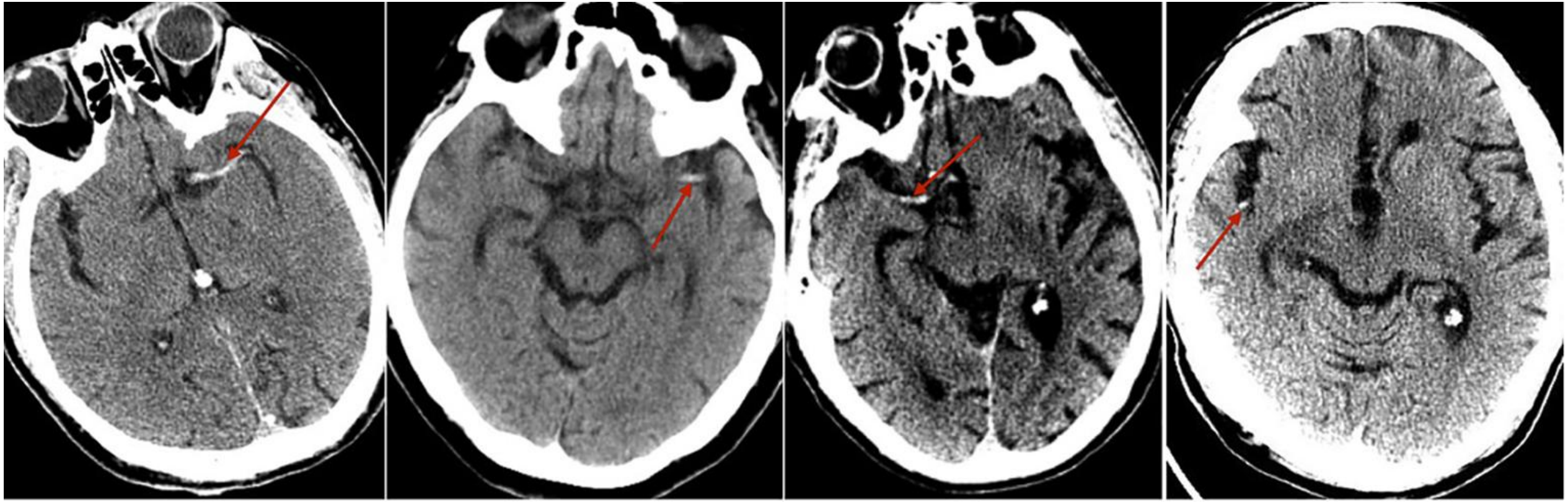
Parenchymal hematoma type 1 (PH-1)—hematoma in 30% or less of infarcted area with some slight space-occupying effect

Parenchymal hematoma type 2 (PH-2)—a dense hematoma in more than 30% of the infarcted area with substantial space-occupying effect, any hemorrhagic lesion outside the infarcted area.

Hyperdense vessel sign

- Both clot length and type → prognostic value
- Quantitative scores for evaluating the extension of the clot : the Clot burden Score
- Clots : lengths over 8 mm and/ or hypodense (fibrin-rich) compared to smaller and hyperdense (red blood cell-rich) thrombus → lower recanalization with IV thrombolysis

Hyperdense MCA sign



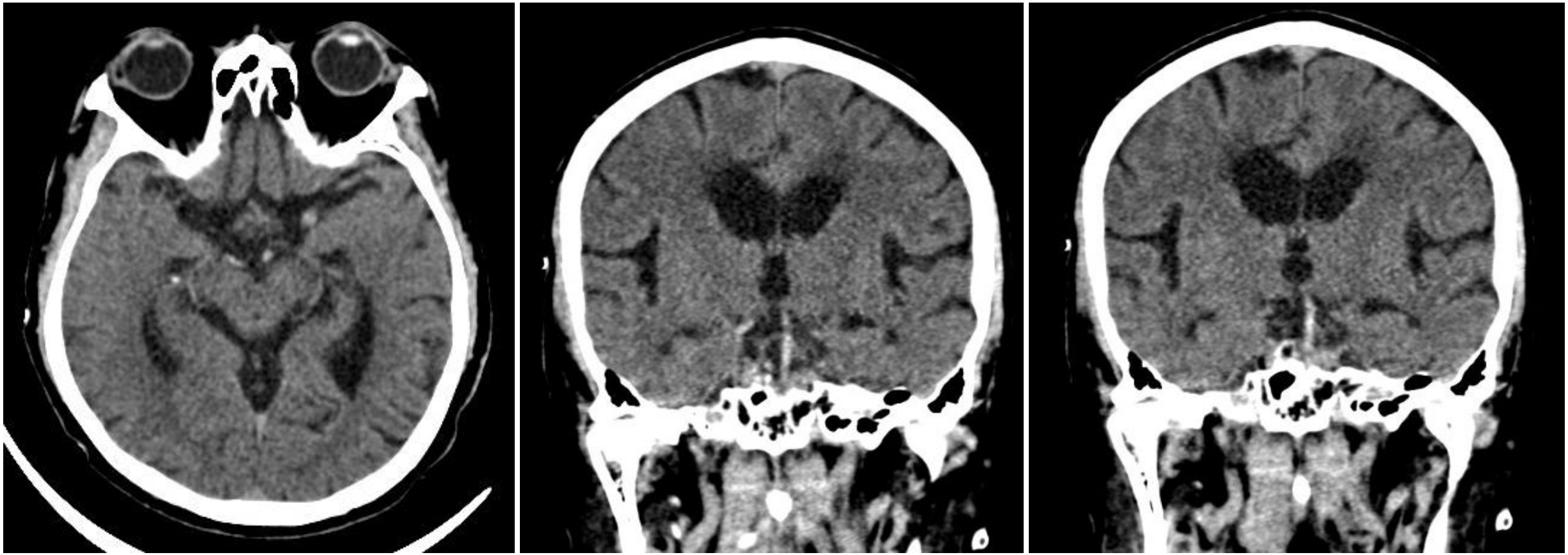
Left proximal hyperdense M1

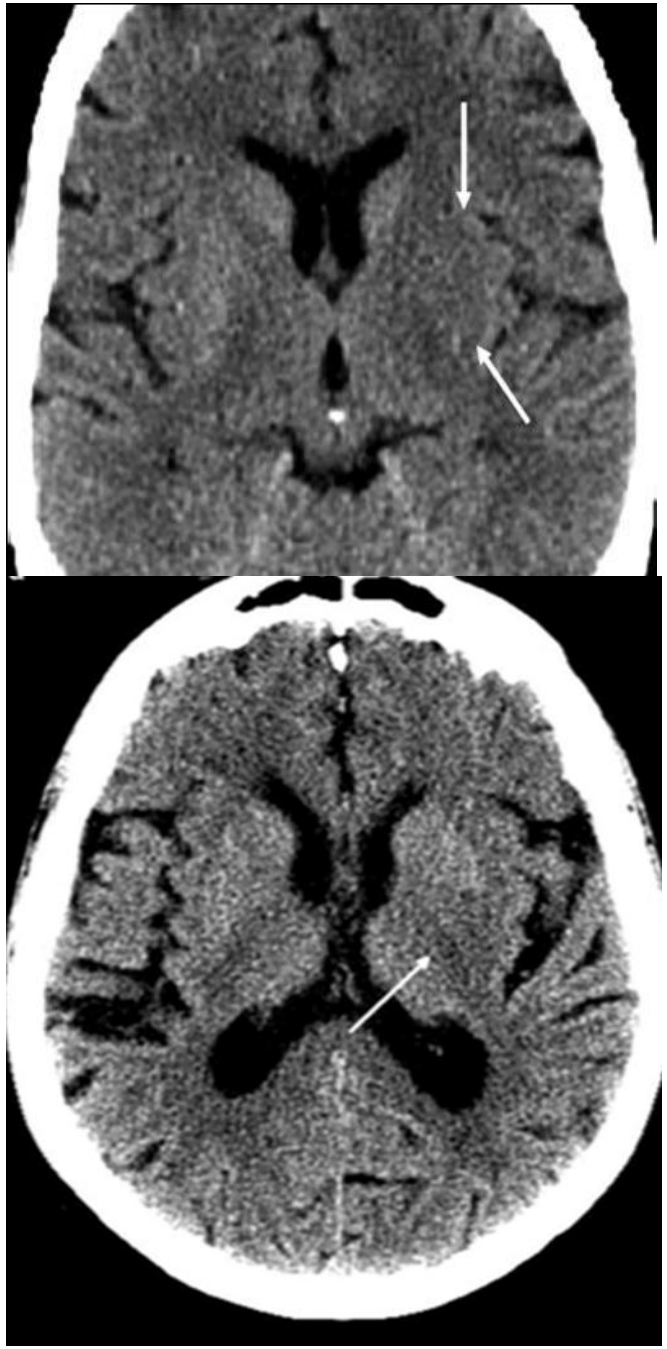
Left distal hyperdense M1

Right proximal hyperdense M1

Right hyperdense M2 in the sylvian fissure ("sylvian dot sign")

Hyperdense Basilar A sign





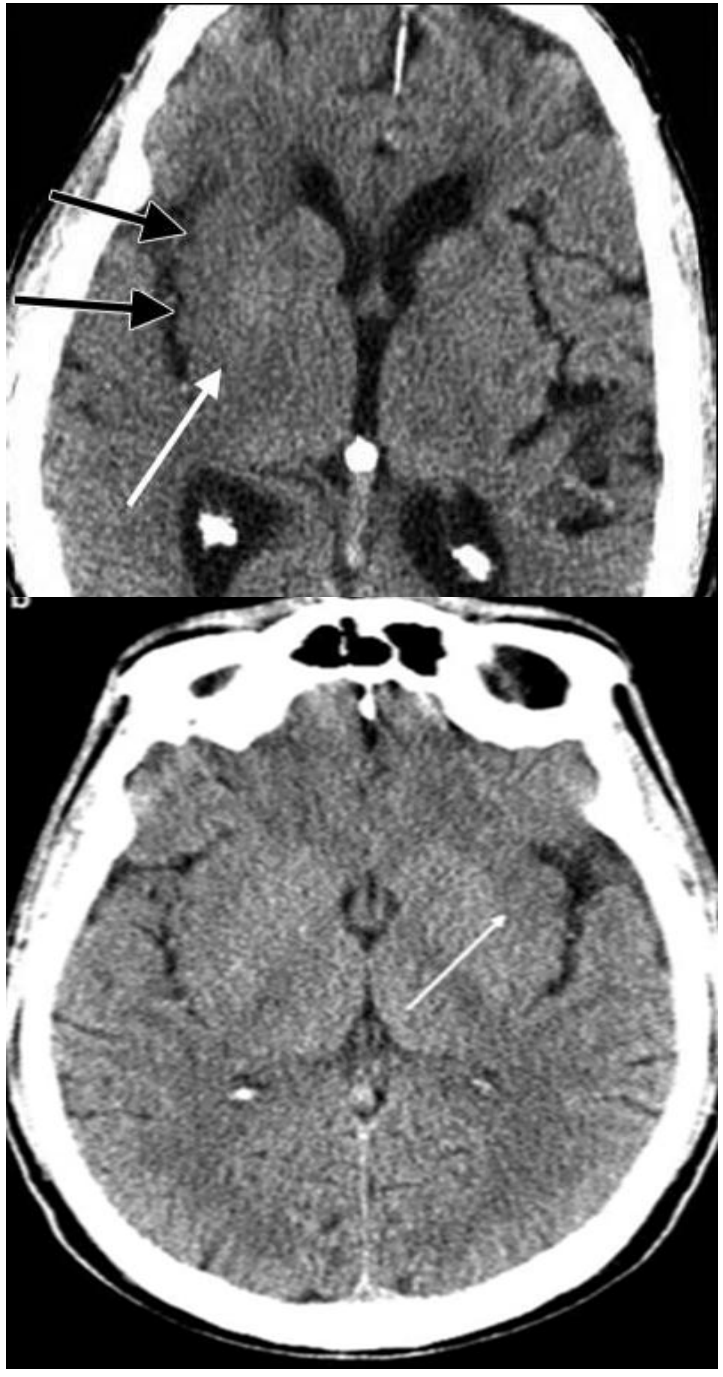
OBSCURATION OF LENTIFORM NUCLEOUS

- Lentiform nucleus appears hypo attenuated because of acute ischemia of the lenticulostriate territory , resulting in obscuration of the lentiform nucleus

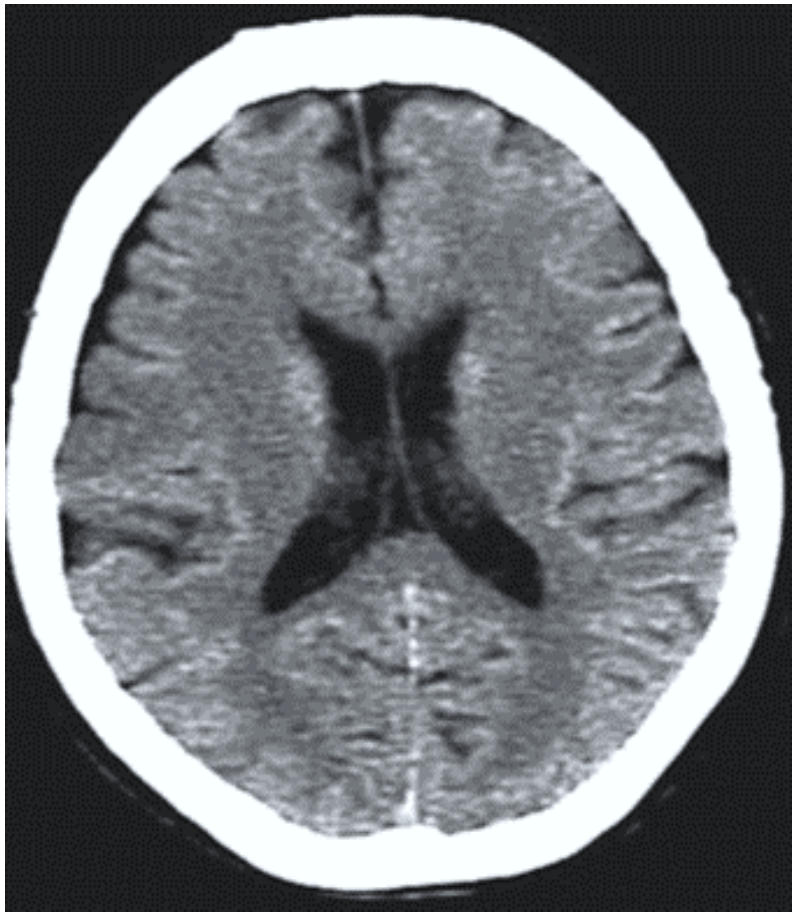
- May be seen on CT images within 2 hours after the onset of a stroke

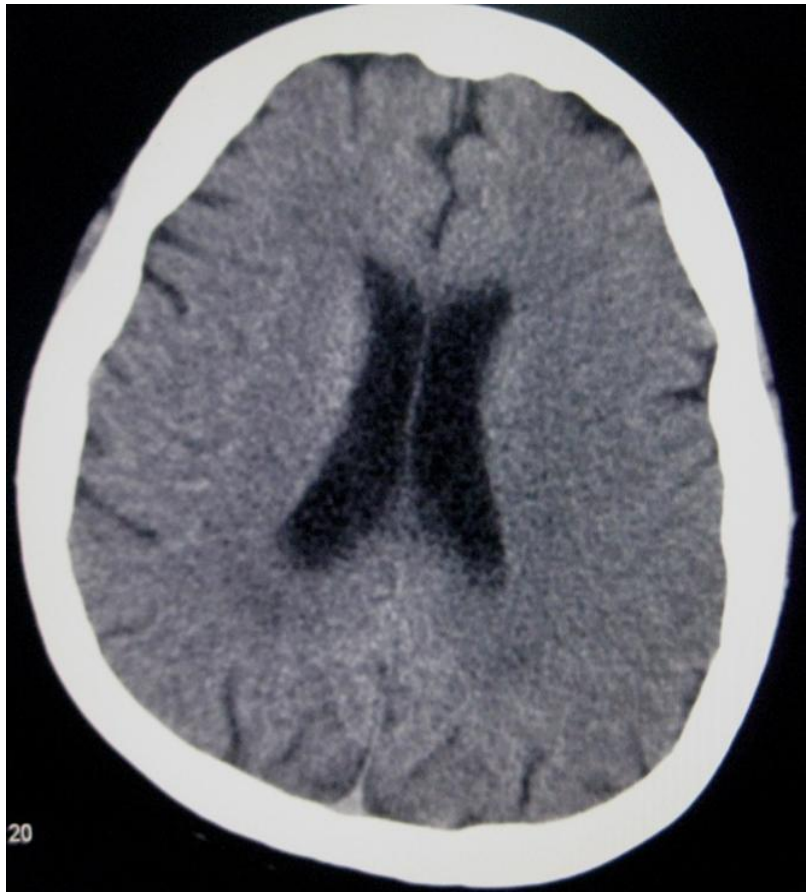
INSULAR RIBBON SIGN

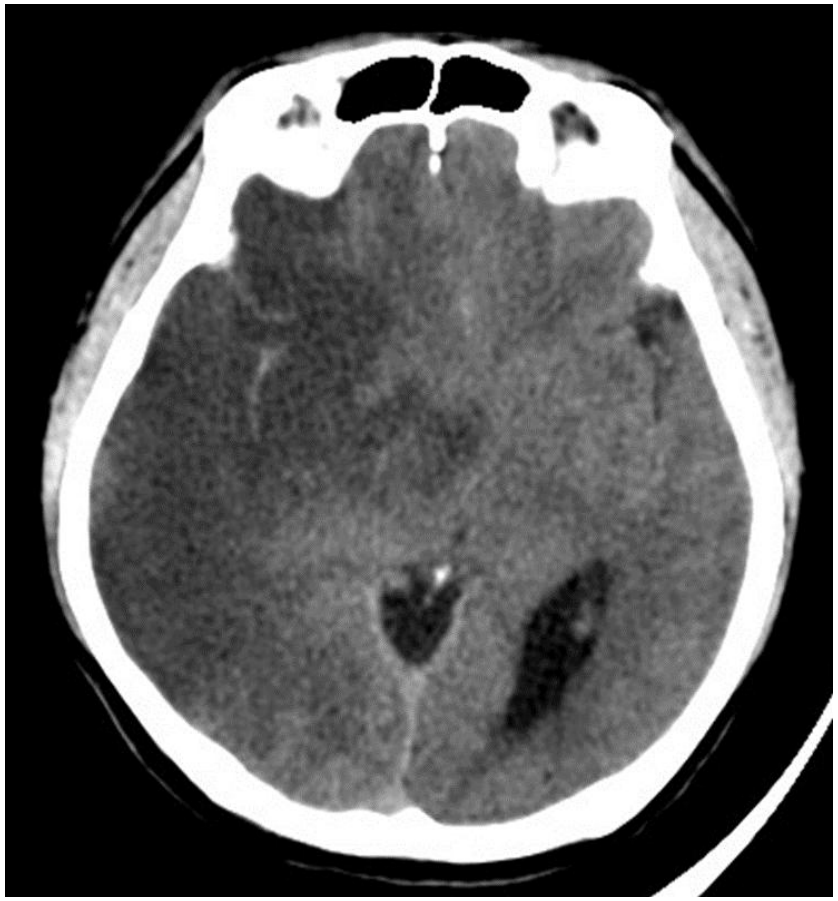
- Local hypoattenuation of the insular cortex region due to cytotoxic edema as this region is susceptible to early and irreversible ischemic damage



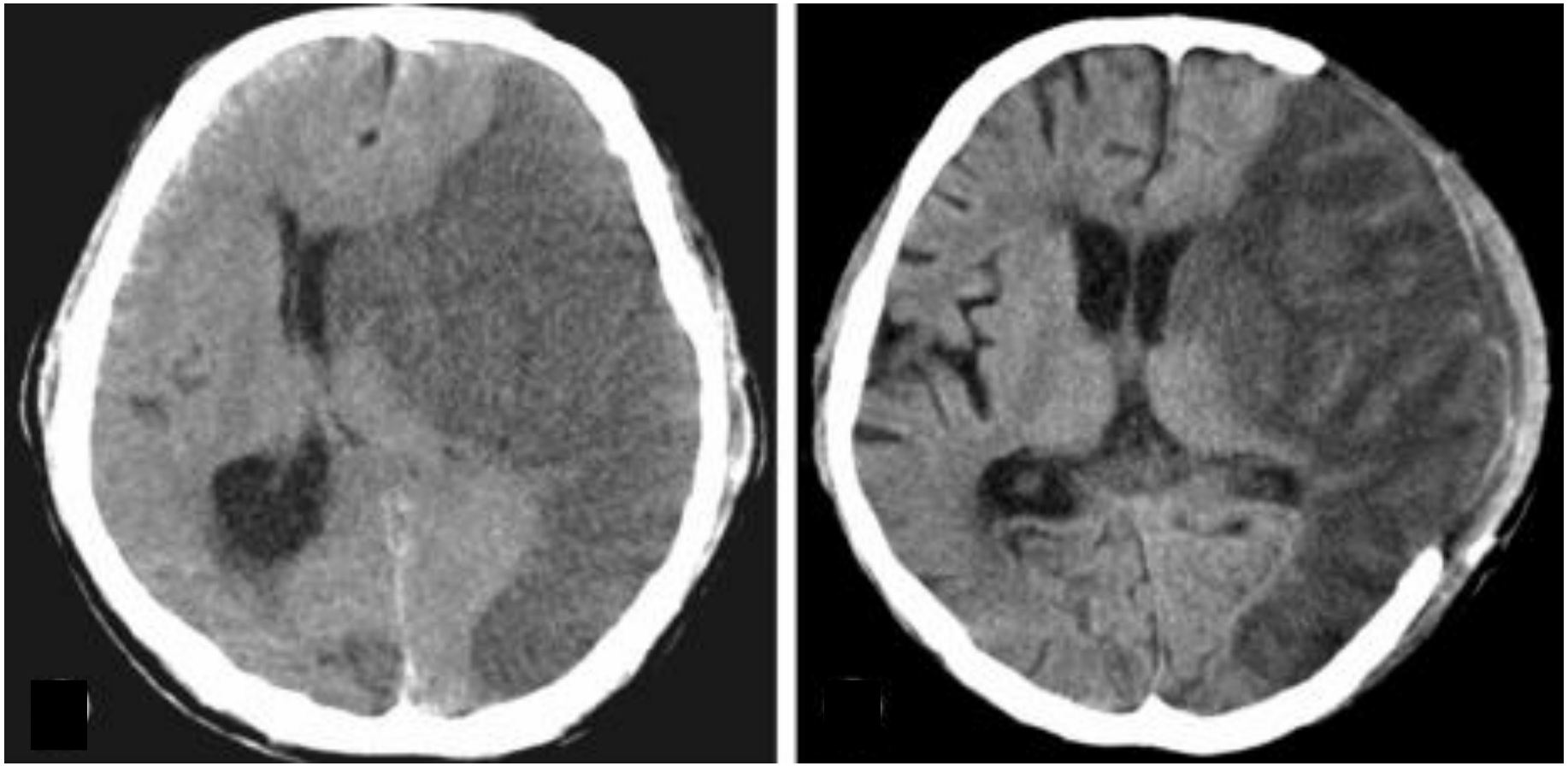
Focal hypodense area



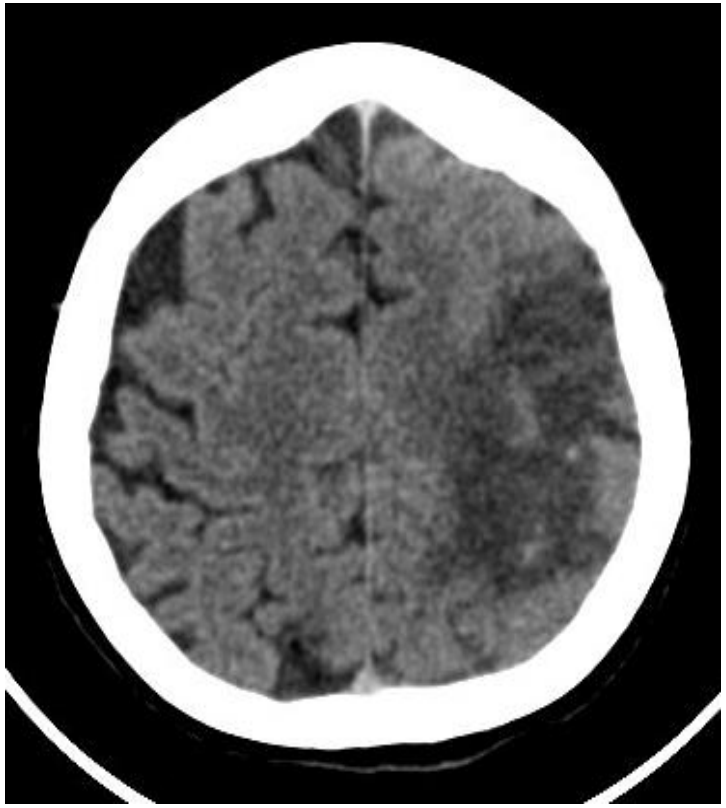


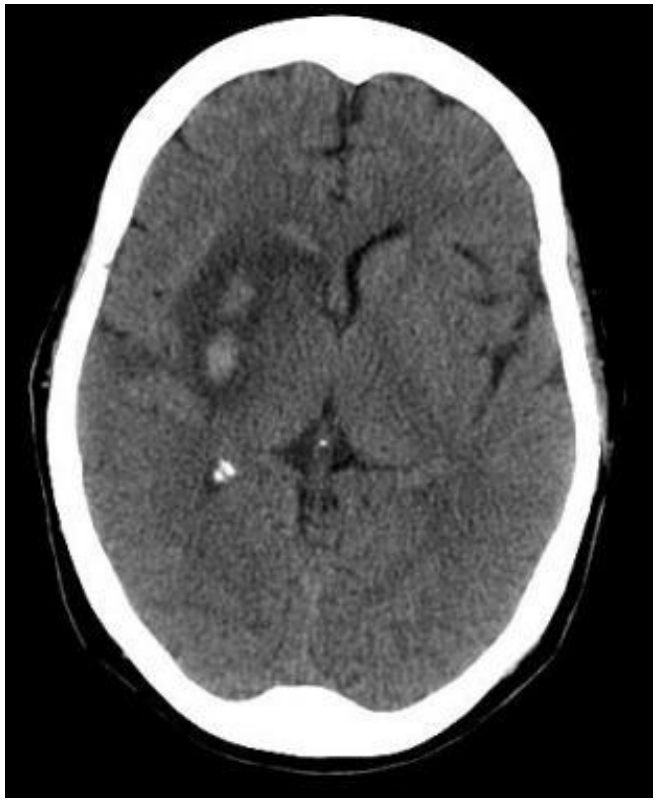


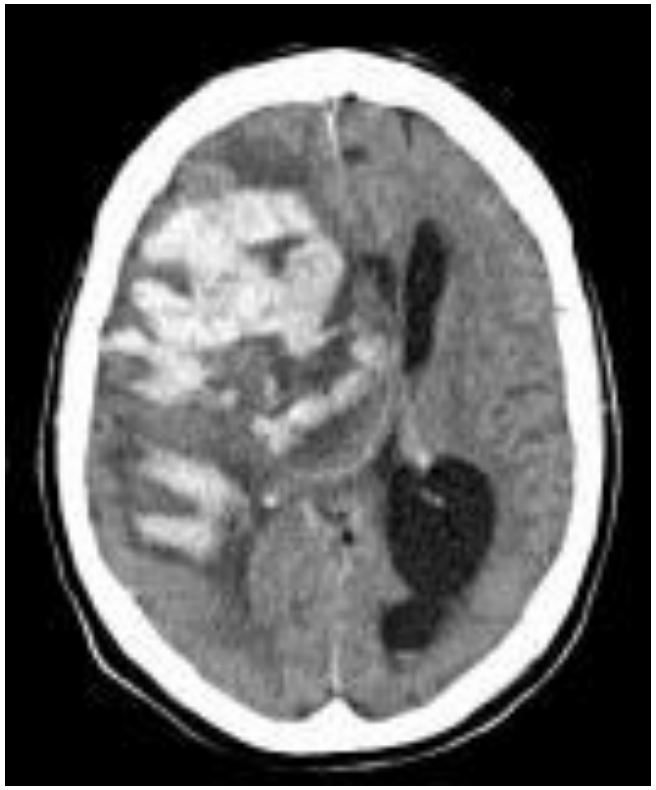
Subacute stroke
Mass effect



Subacute with haemorrhagic transformation







Hemorrhagic infarction type 1 (HI-1)—small petechiae along the margins of the infarct

Hemorrhagic infarction type 2 (HI-2)—more confluent petechiae within the infarcted area, but without space-occupying effect

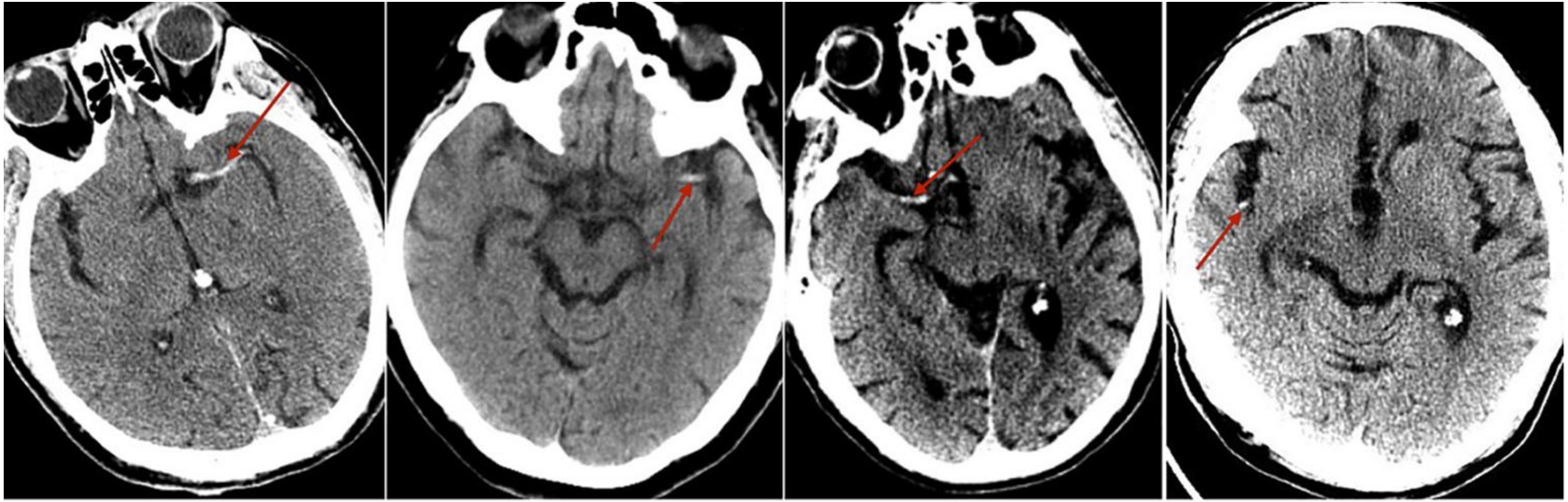
Parenchymal hematoma type 1 (PH-1)—hematoma in 30% or less of infarcted area with some slight space-occupying effect

Parenchymal hematoma type 2 (PH-2)—a dense hematoma in more than 30% of the infarcted area with substantial space-occupying effect, any hemorrhagic lesion outside the infarcted area.

Hyperdense vessel sign

- Both clot length and type → prognostic value
- Quantitative scores for evaluating the extension of the clot : the Clot burden Score
- Clots : lengths over 8 mm and/ or hypodense (fibrin-rich) compared to smaller and hyperdense (red blood cell-rich) thrombus → lower recanalization with IV thrombolysis

Hyperdense MCA sign



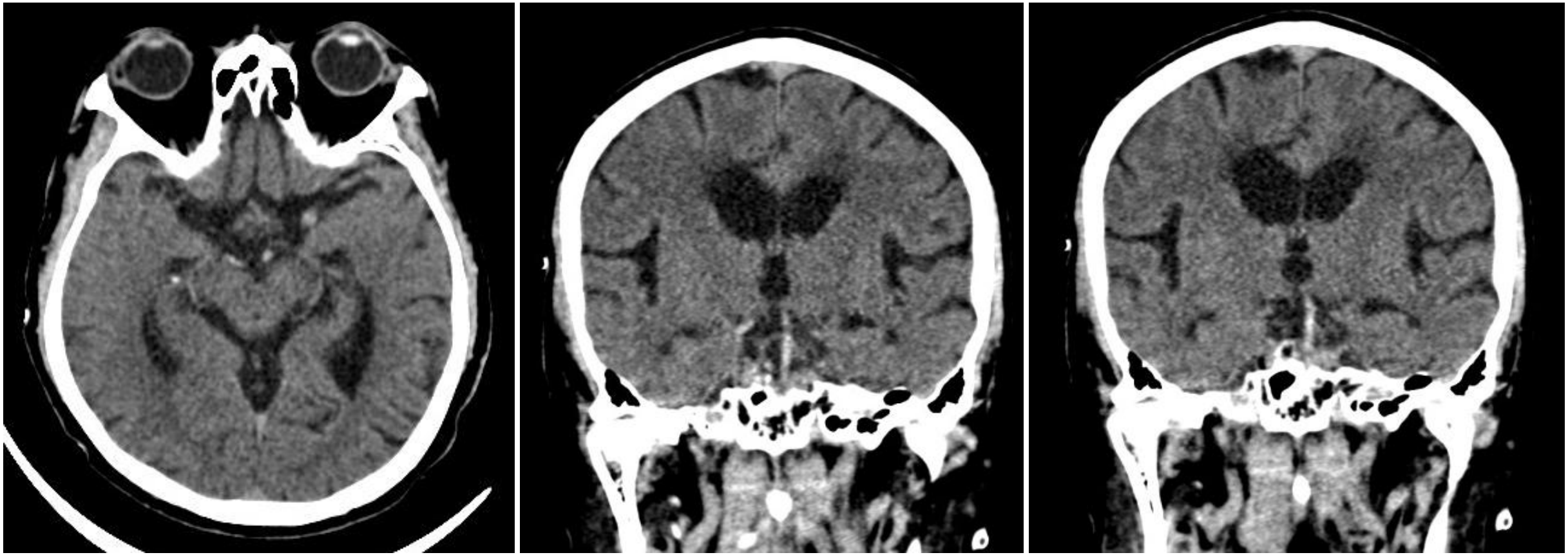
Left proximal hyperdense M1

Left distal hyperdense M1

Right proximal hyperdense M1

Right hyperdense M2 in the sylvian fissure ("sylvian dot sign")

Hyperdense Basilar A sign



Imaging of Stroke

Four-step approach to reading a non-contrast head CT for acute stroke

1. First look for the presence/absence of hemorrhage (intracerebral, subarachnoid, or subdural)
   - Acute blood is hyperdense (bright)
   - Subacute blood is isodense (similar density to brain parenchyma)
   - Chronic blood is hypodense (darker than the brain)

(2) Look for the presence/absence of any hyperdense vessel signs suggestive of an acute clot in a major artery (i.e., MCA sign, ACA, PCA, basilar) or vein (dural venous thrombosis or deep vein thrombosis)

(3) Identify the presence/absence and extent of any early acute ischemic changes, which can be rated on the ASPECTS scale

(4) Exclude other structural brain pathology

Evaluating extend of Stroke

- ECASS (European Cooprative Acute Stroke Study
  - > 1/3 MCA territory significant increase of hemorrhage
  - Exclusion criteria for thrombolysis
  - Problem : Interobserver variability

- ASPECTS (anterior & posterior circulation)
  - Systematic assesment of MCA territory & BA territory
  - 10 point subdivision of MCA & BA territory

Alberta Stroke Program Early CT Score (ASPECTS)

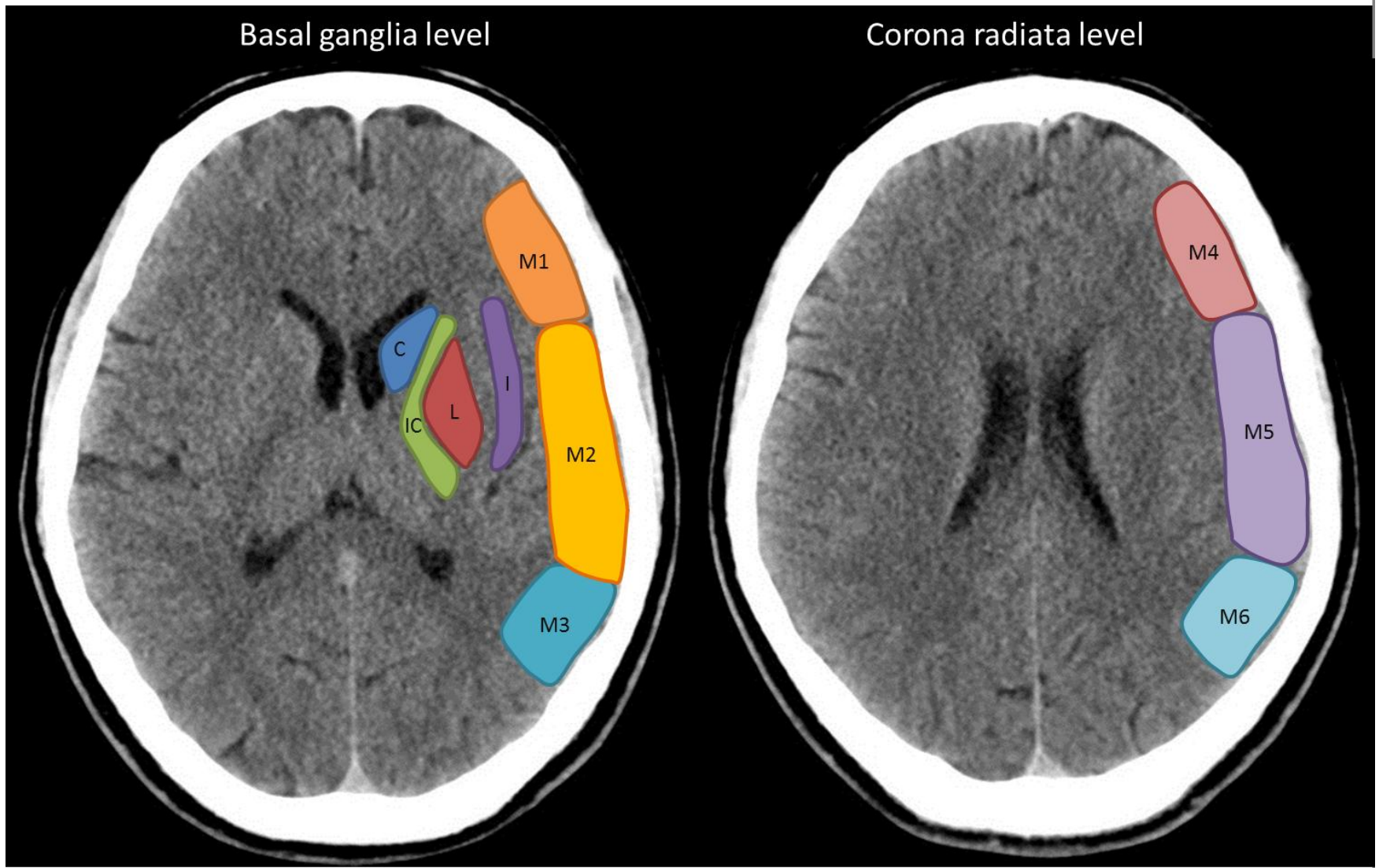
- To offer the reliability and utility of a standard CT → grading system to assess EICs pretreatment → acute ischemic anterior circulation (MCA) & posterior circulation (BA)

- ASPECT Score :
  10 point → quantitative topographic CT score

ASPECTS Anterior circulation

- Segmental estimation of the middle cerebral artery (MCA) vascular territory is made and 1 point is deducted from the initial score of 10 for every region involved:
- Nucleus caudatus
- Putamen
- Internal capsule
- Insular cortex
- M1: "anterior MCA cortex," corresponding to the frontal operculum
- M2: "MCA cortex lateral to insular ribbon" corresponding to the anterior temporal lobe
- M3: "posterior MCA cortex" corresponding to the posterior temporal lobe
- M4: "anterior MCA territory immediately superior to M1"
- M5: "lateral MCA territory immediately superior to M2"
- M6: "posterior MCA territory immediately superior to M3"

MCA Alberta stroke program early CT score (ASPECTS)



C: Caudate; IC: internal capsule; L: lentiform nucleus; I: Insular Cortex.

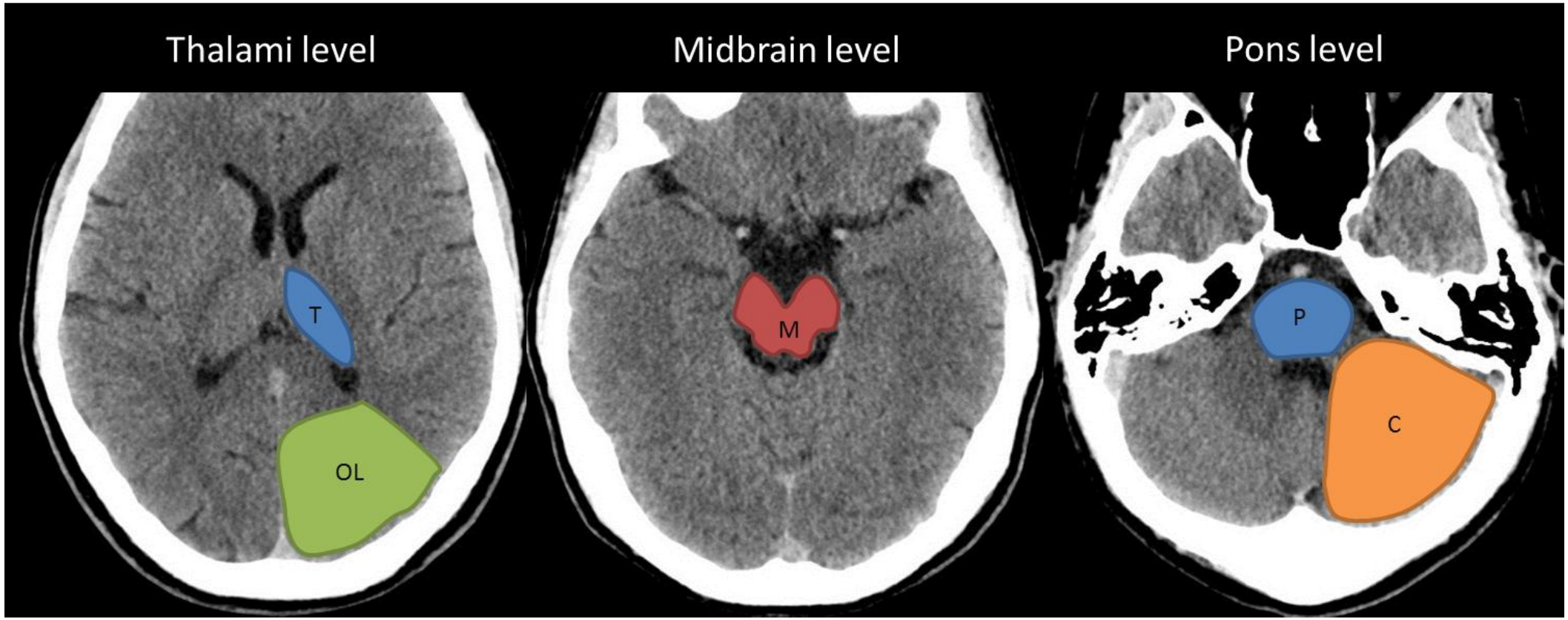
ASPECTS Posterior circulation

The pc-ASPECTS is a 10 point scale, where points are lost for each region affected

Pons and the midbrain → 2 points each ,regardless of whether or not the changes are bilateral . (any involvement of the pons, for example, deducted 2 points).

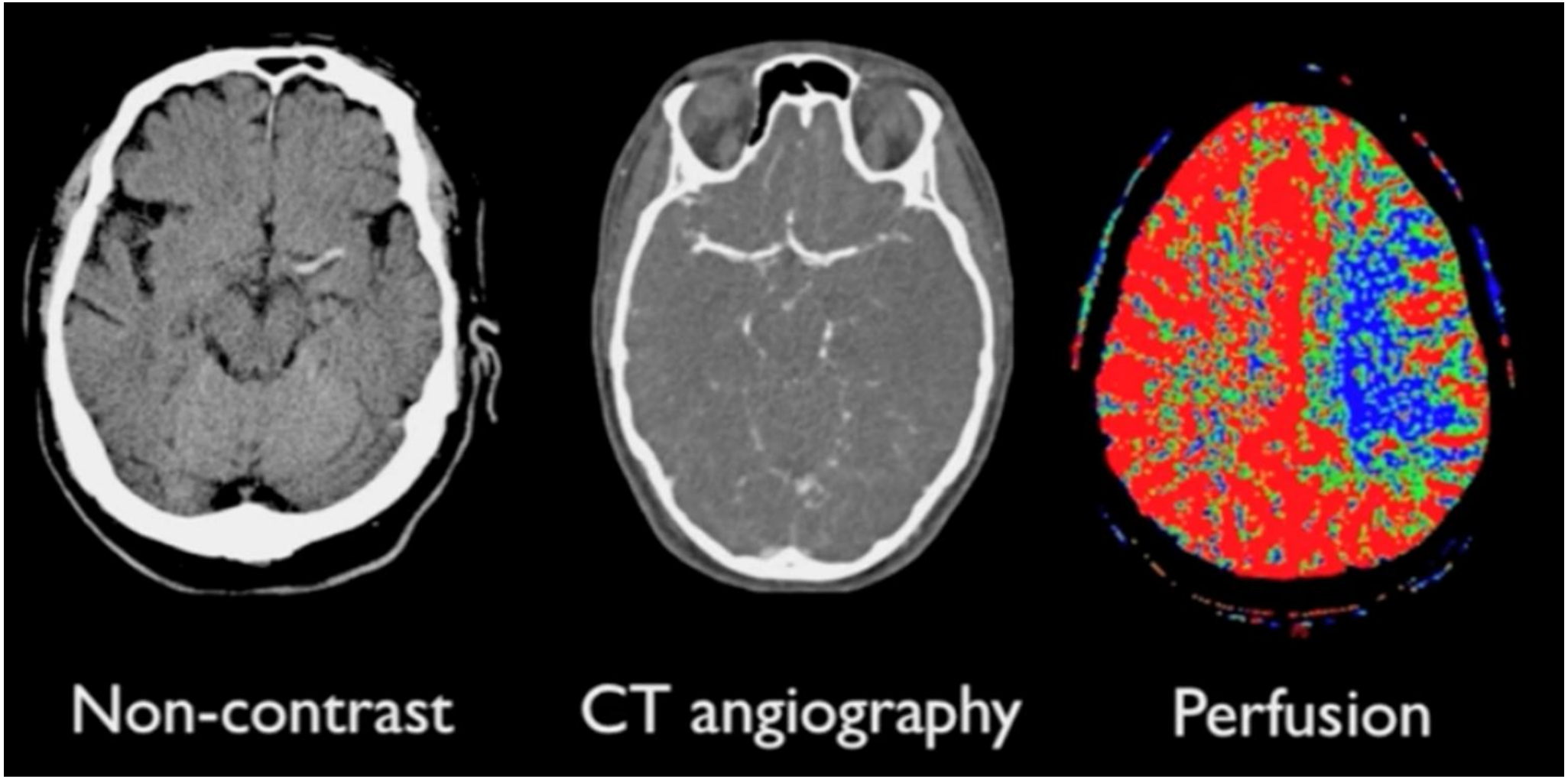
- thalami (1 point each)
- occipital lobes (1 point each)
- midbrain (2 points)
- pons (2 points)
- cerebellar hemispheres (1 point each)

Posterior circulation Acute stroke prognosis early CT score (pc-ASPECTS)



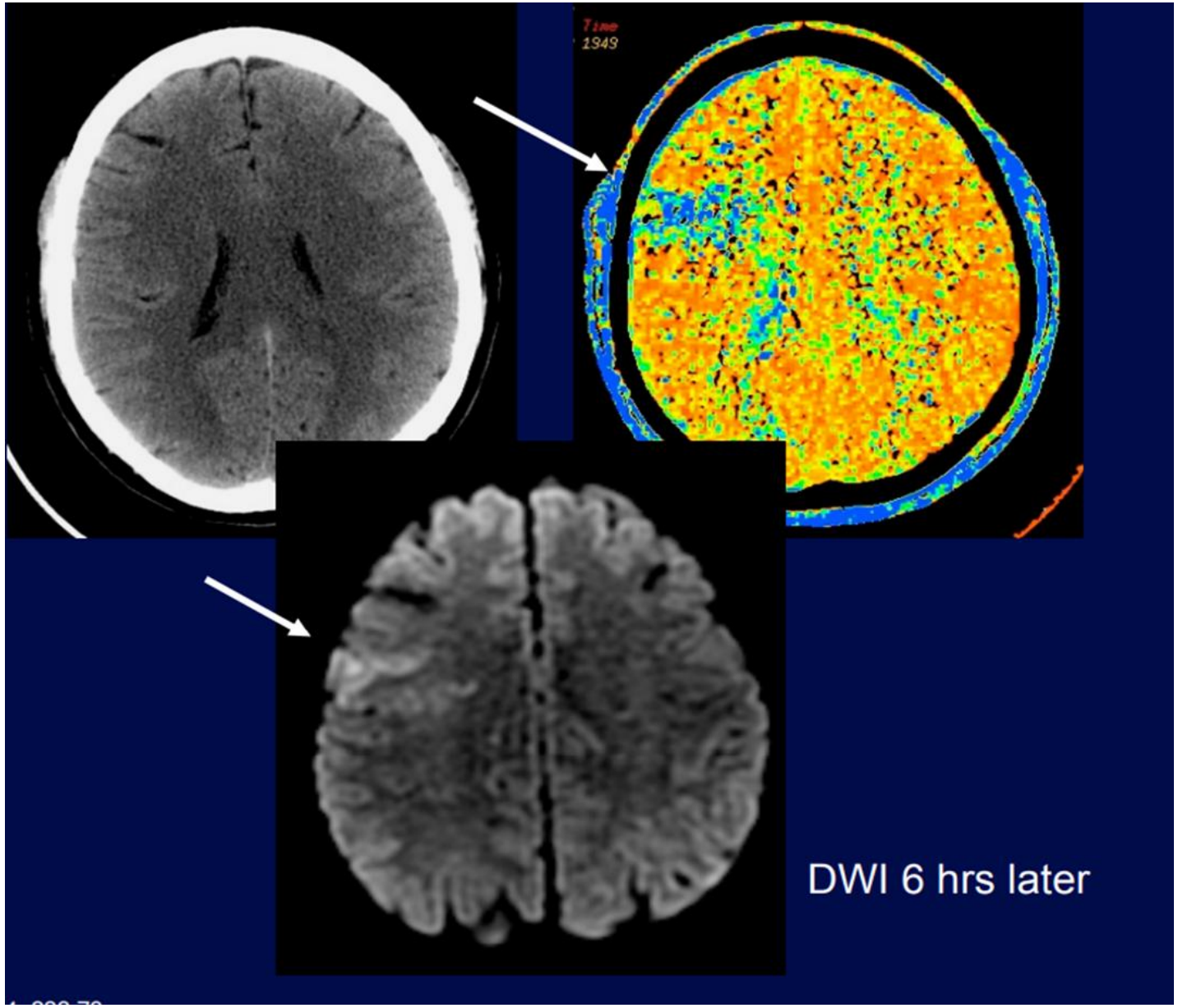
T: thalamus; OL: occipital lobe; M: any part of the midbrain; P: any part of the pons;
C: cerebellar hemisphere.

CT in Acute Stroke



CT Perfusion

- Early stroke hard to see
- NCCT : sensitivity 15-20%
- CTP : sensitivity 55-76%



MRI in Acute Ischemic Stroke

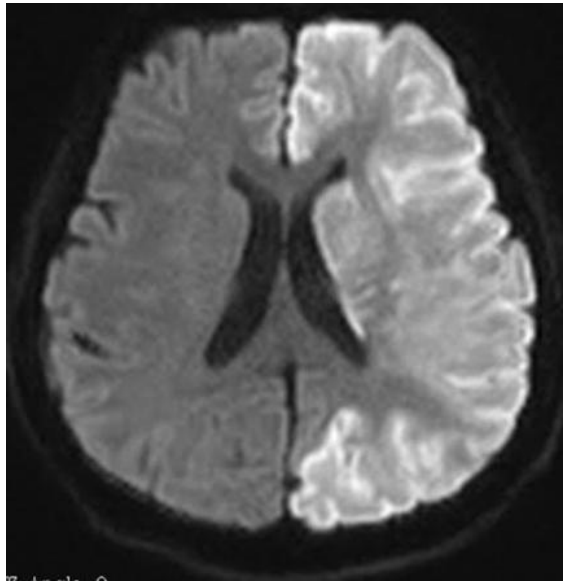
- MRI more sensitive than CT in hyperacute ischaemic
- To classify mechanism, prognosis & treatment
- Provides different info from the clinical & pathological

'Stroke' Sacco et al, AHA/ASA, Stroke, 2013

Standard Protocol MRI in AIS

- DWI & ADC, T1WI, T2WI, FLAIR, GRE/SWI
- PWI (MR perfusion contrast)
- 3 D TOF MRA

- 6 Min (DWI&ADC, FLAIR , SWI ,MR Perfusion, MRA)

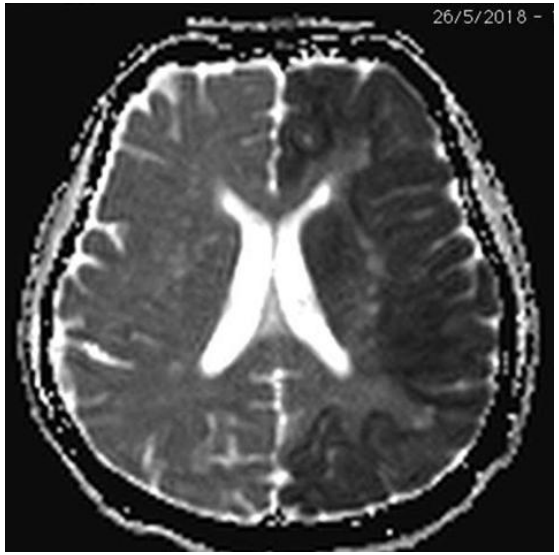


DWI & ADC

Acute ischemic stroke → Very high signal on DWI

→ Marked reduction in ADC val.

DWI : reduced diffusion → minutes to < 30 min. after ictus



ADC : decrease : 8 to 32 hrs to ≤ 50 of normal % values

reduced : 3 to 5 days

MRI – T2WI

- Slight increased detection rate over CT in early stroke
- T2 hyperintensity visible at 12- 24 hrs (80%) represents edema

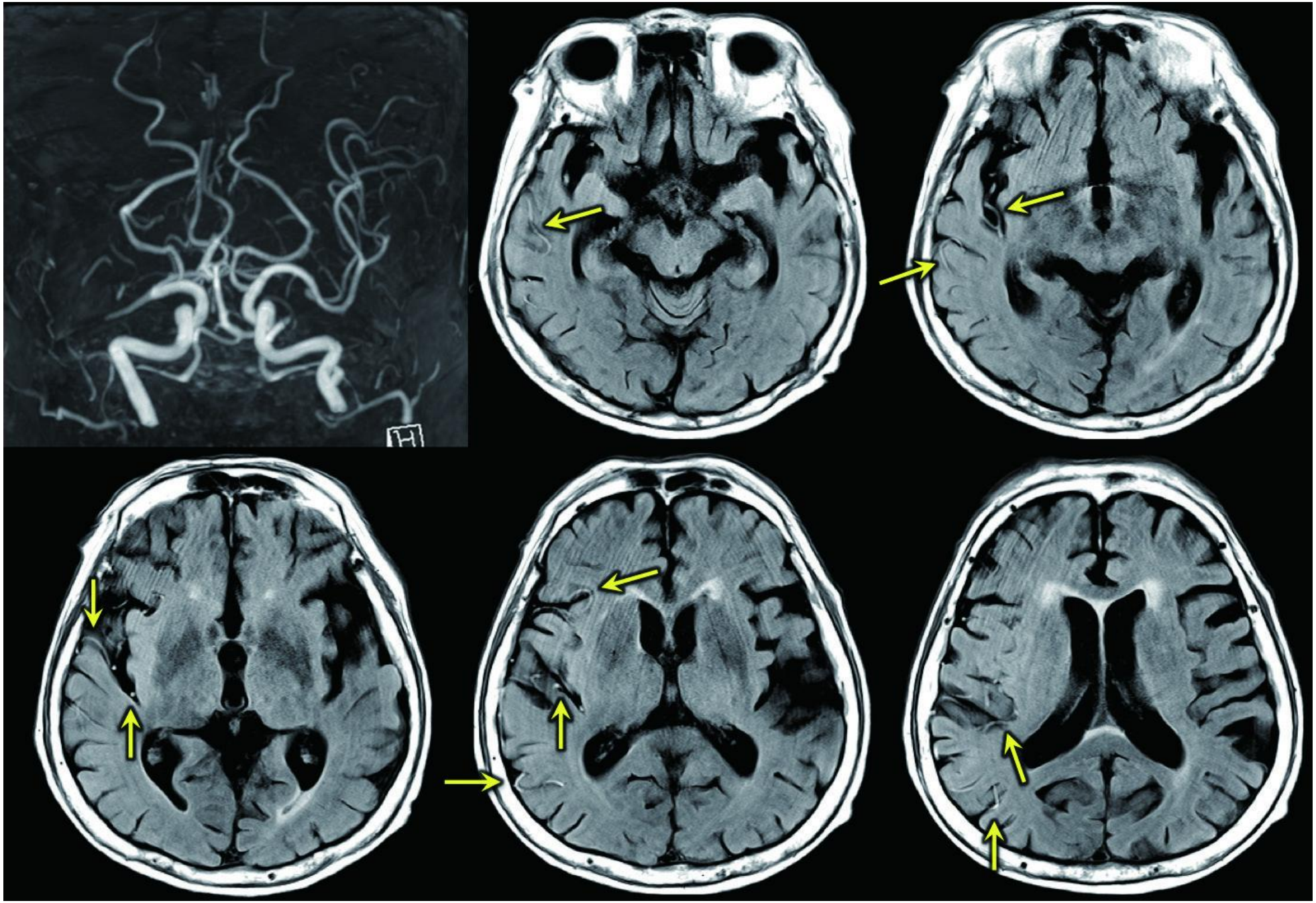
MRI signs :

- Increased signal intensity
- Swollen cortical gyri
- Increased signal intensity in the lumen of vessels → due to vessel occlusion,
  severe stenosis
- Vasculitic pattern
- Collateral leptomeningeal circulation

MRI –FLAIR

- Detect hyperintense vessels sign (HVs) → slow flow beyond the occluded site or detection proximal vascular occlusion

  (specificity 86% & sensitivity 76%)
- Arterial hyperintensity : early in stroke within 0–2 hours after onset of symptoms
- Indentify proximal occlusion or severe stenosis → presence of collaterals



DWI vs CT

- Sencitivity and specifity in detected infarct :

3 hrs onset : DWI : 77% vs CT : 16%
6 hrs onset : DWI : 91% vs CT : 61%

- Superior > CT in AIS in 12 hrs onset

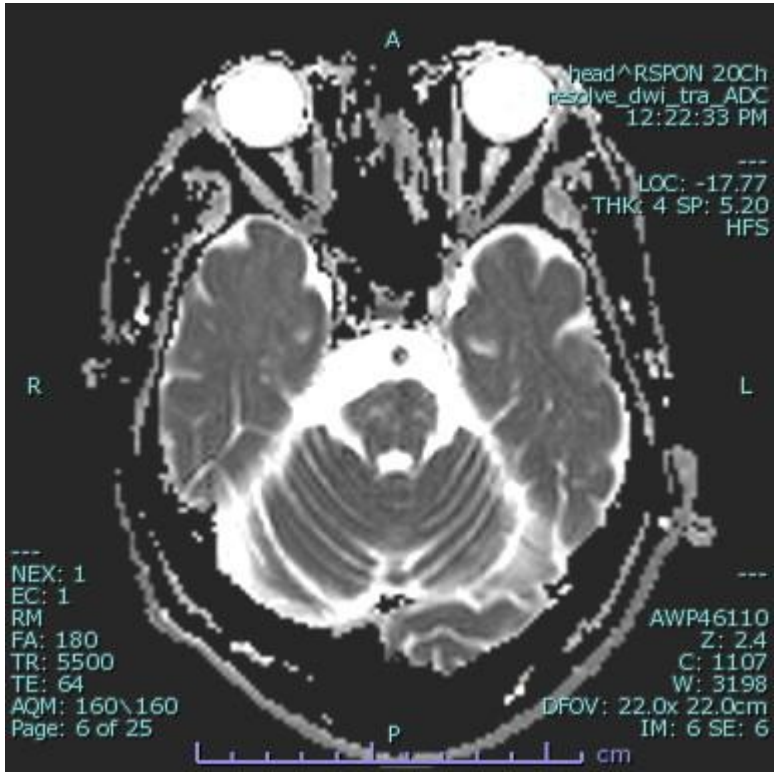
DWI vs FLAIR

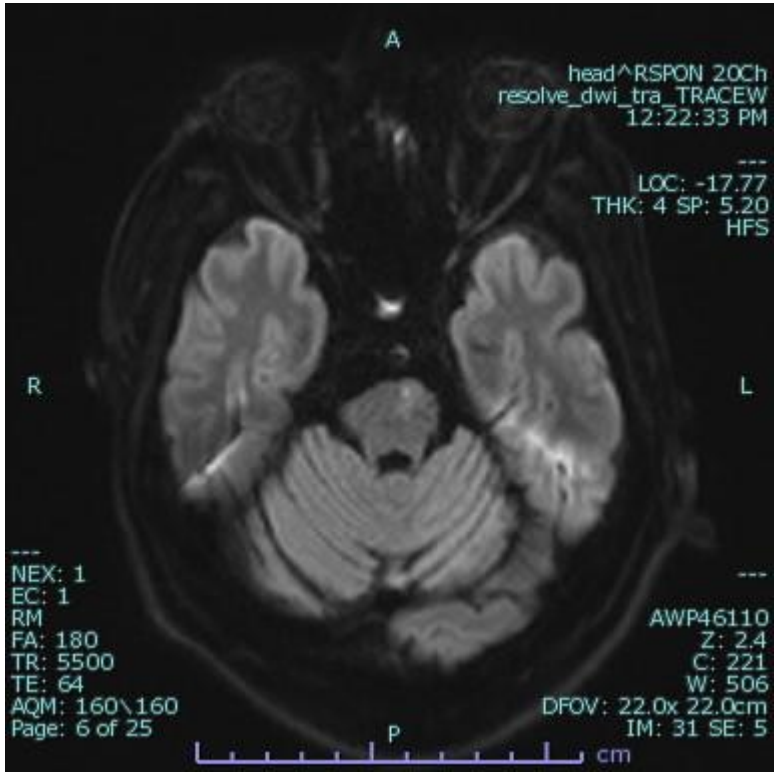
- DWI-FLAIR mismatch :
  determine onset time in AIS (high PPV)

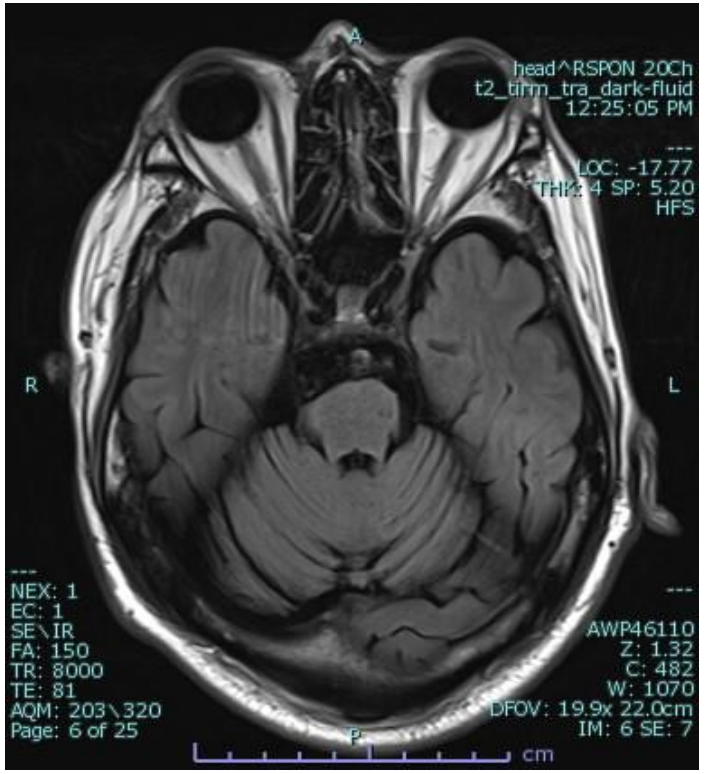
- DWI-FLAIR mismatch :
  DWI (+) and FLAIR (-) :
  77% → << than 3 hours
  96% → << than 4.5 hours
  100% → << than 6 hours

DWI-FLAIR mismatch determine→ improve the outcome in patients with unknown onset (wake up stroke) → intravenous rtPA

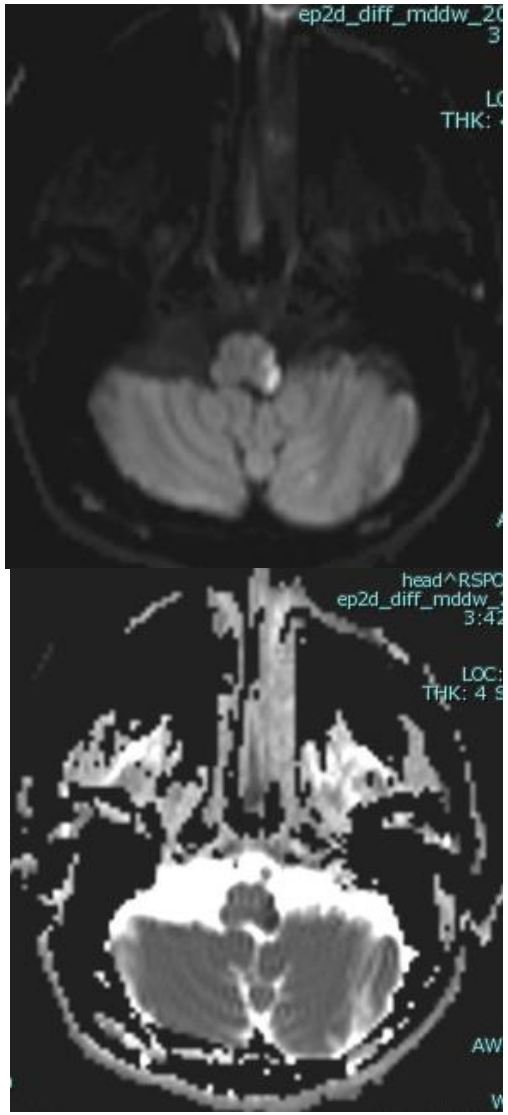
DWI FLAIR Mismatch
Hyperacute Infarct

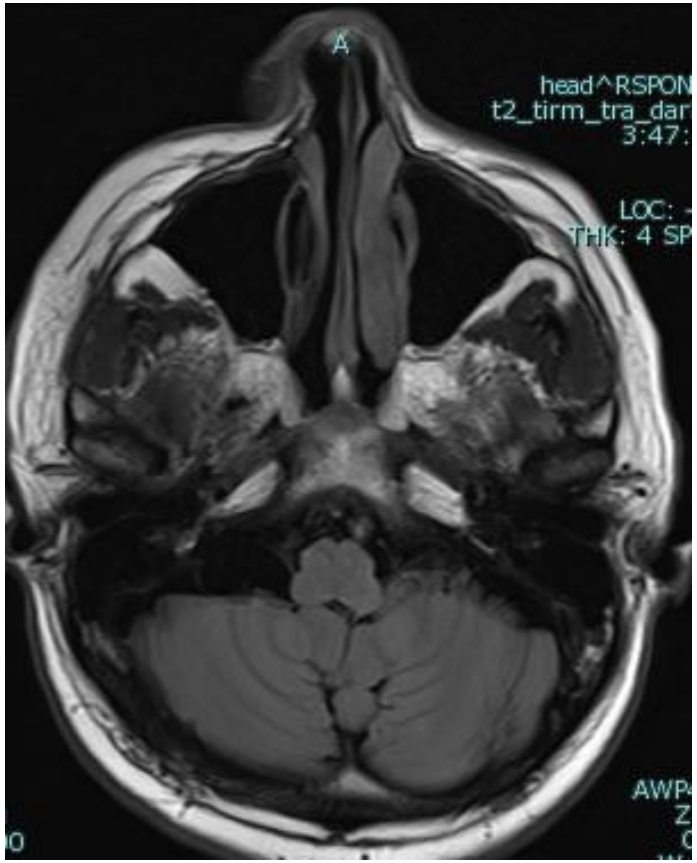


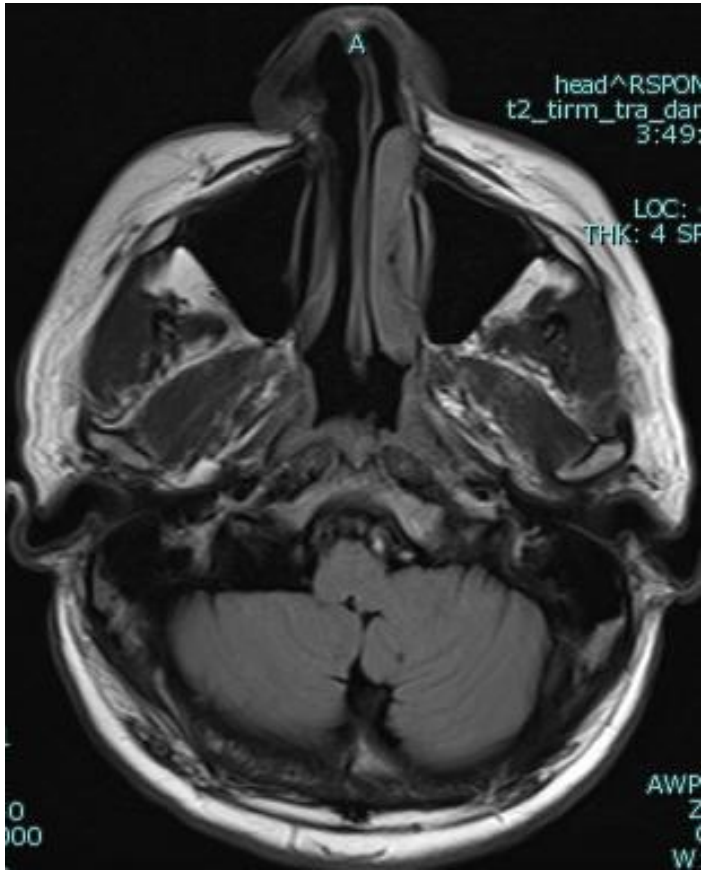


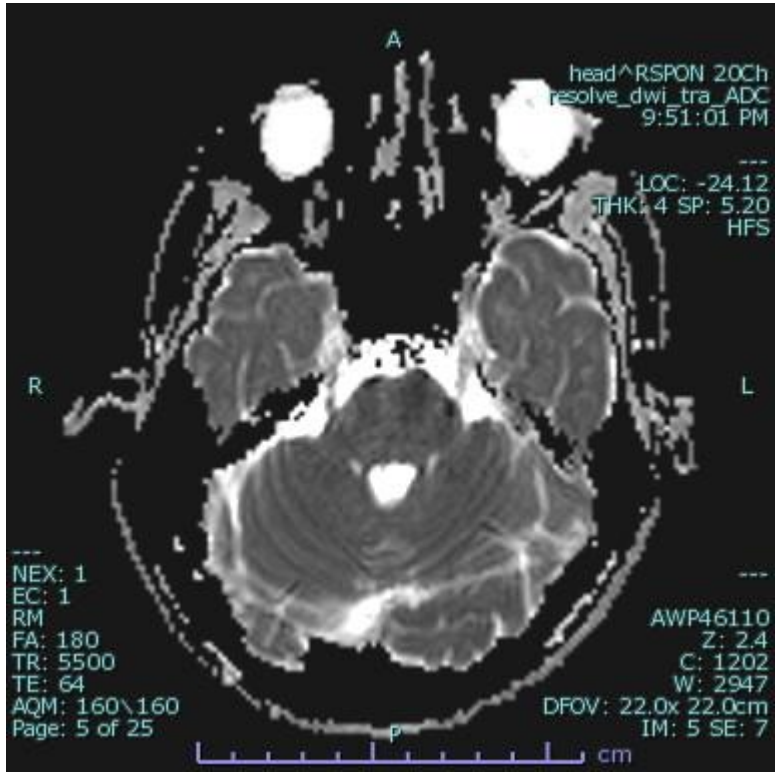


Hyperacute infarct with hiperdense vessel sign





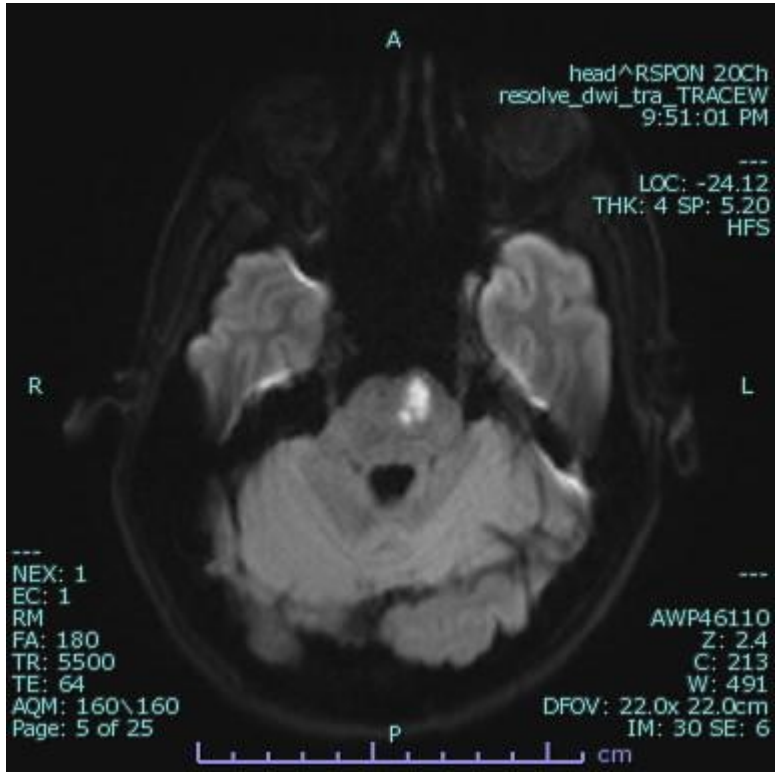



A
head^RSPON 20Ch
resolve_dwi_tra_ADC
9:51:01 PM

LOC: -24.12
THK: 4 SP: 5.20
HFS
R
L

NEX: 1
EC: 1
RM
FA: 180
TR: 5500
TE: 64
AQM: 160\160
Page: 5 of 25

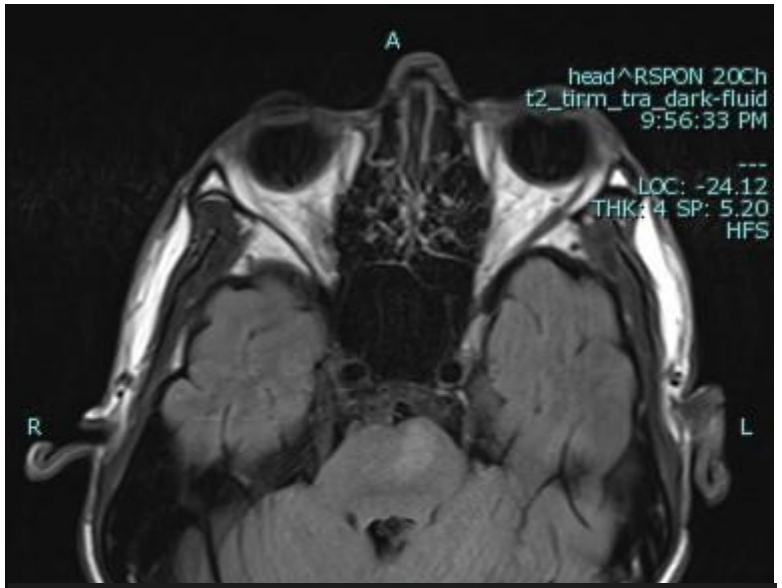
AWP46110
Z: 2.4
C: 1202
W: 2947
DFOV: 22.0x 22.0cm
IM: 5 SE: 7
P
cm


A
head^RSPON 20Ch
resolve_dwi_tra_TRACEW
9:51:01 PM

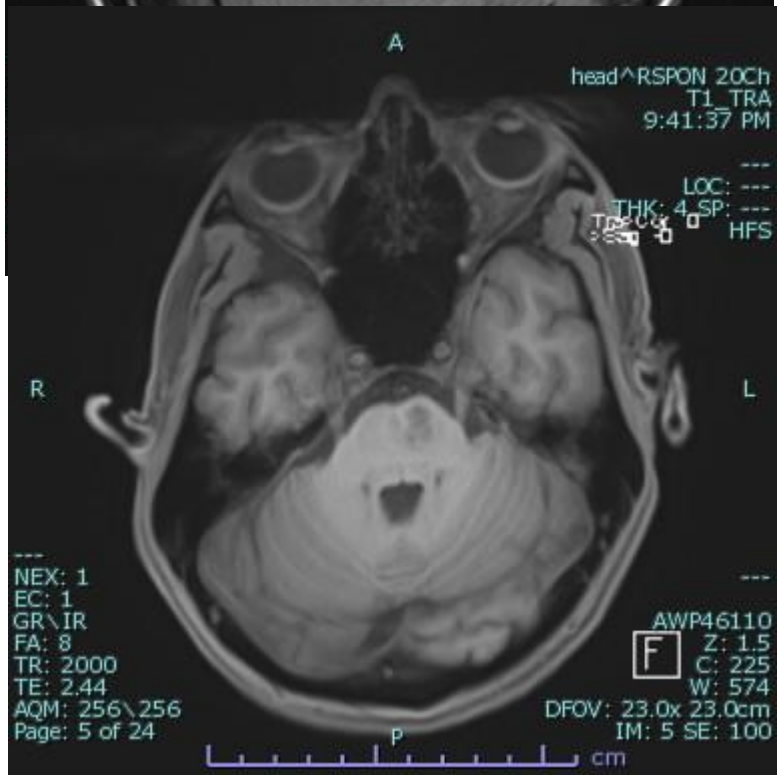
LOC: -24.12
THK: 4 SP: 5.20
HFS
R
L

NEX: 1
EC: 1
RM
FA: 180
TR: 5500
TE: 64
AQM: 160\160
Page: 5 of 25

AWP46110
Z: 2.4
C: 213
W: 491
DFOV: 22.0x 22.0cm
IM: 30 SE: 6
P
cm


A
head^RSPON 20Ch
t2_tirm_tra_dark-fluid
9:56:33 PM

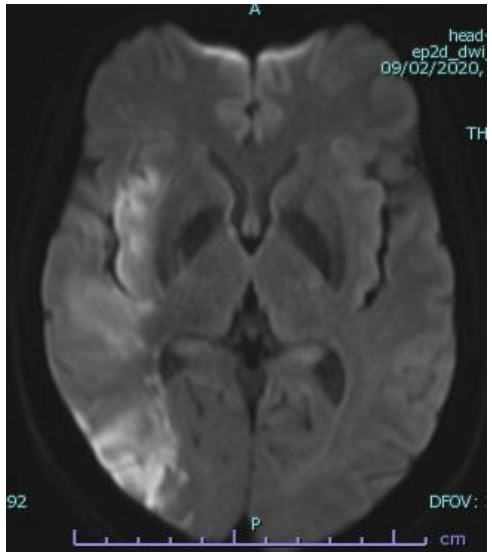
LOC: -24.12
THK: 4 SP: 5.20
HFS
R
L

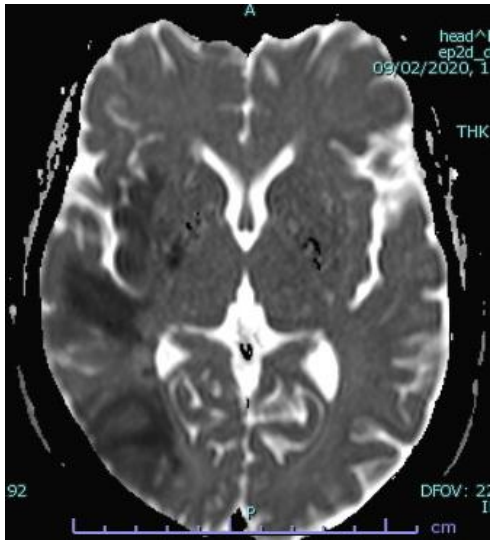

A
head^RSPON 20Ch
T1_TRA
9:41:37 PM

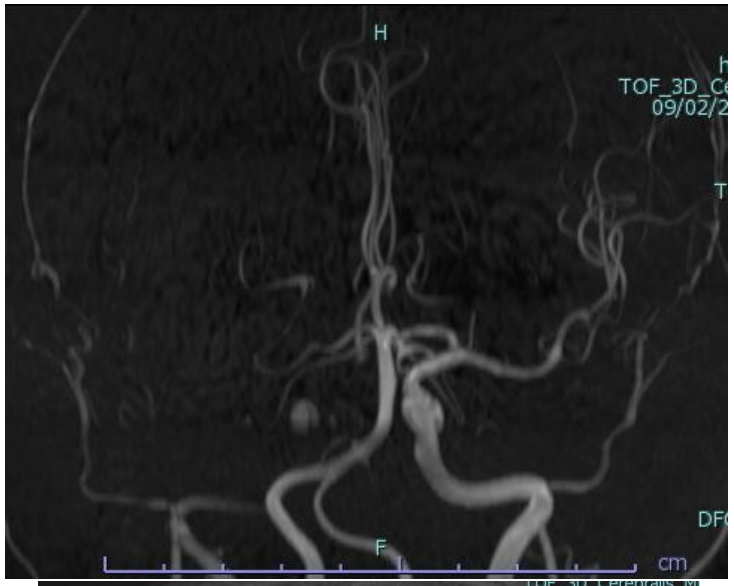
LOC: ---
THK: 4 SP: ---
HFS
R
L

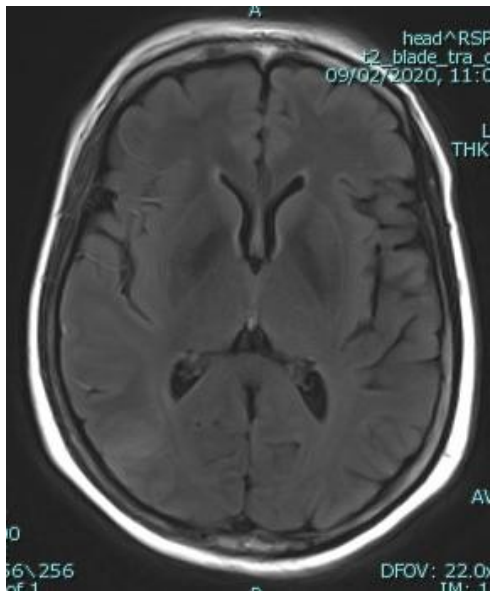
NEX: 1
EC: 1
GR\IR
FA: 8
TR: 2000
TE: 2.44
AQM: 256\256
Page: 5 of 24

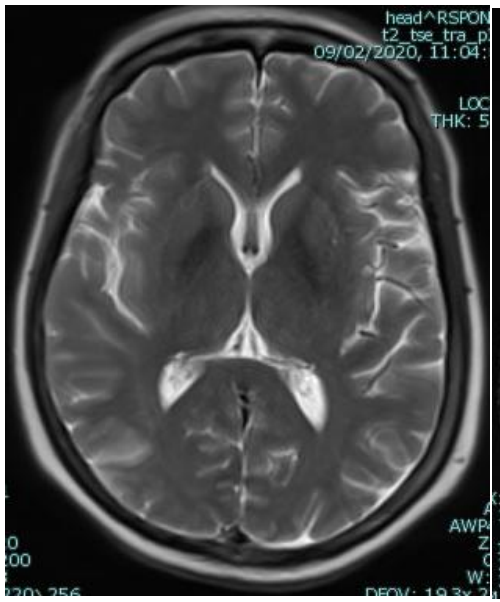
AWP46110
F
Z: 1.5
C: 225
W: 574
DFOV: 23.0x 23.0cm
IM: 5 SE: 100
P
cm

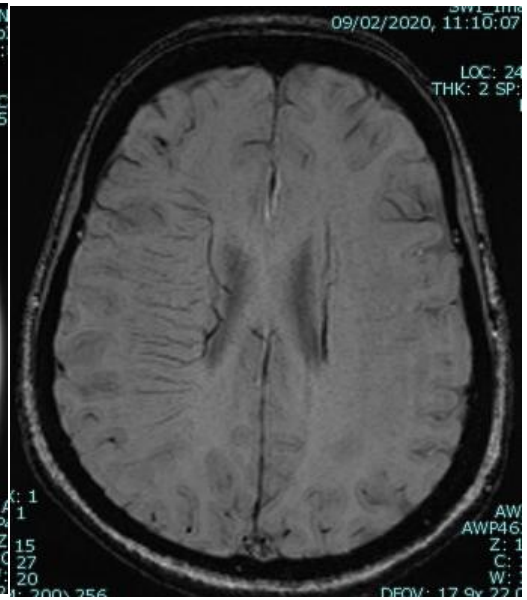

A
P
cm

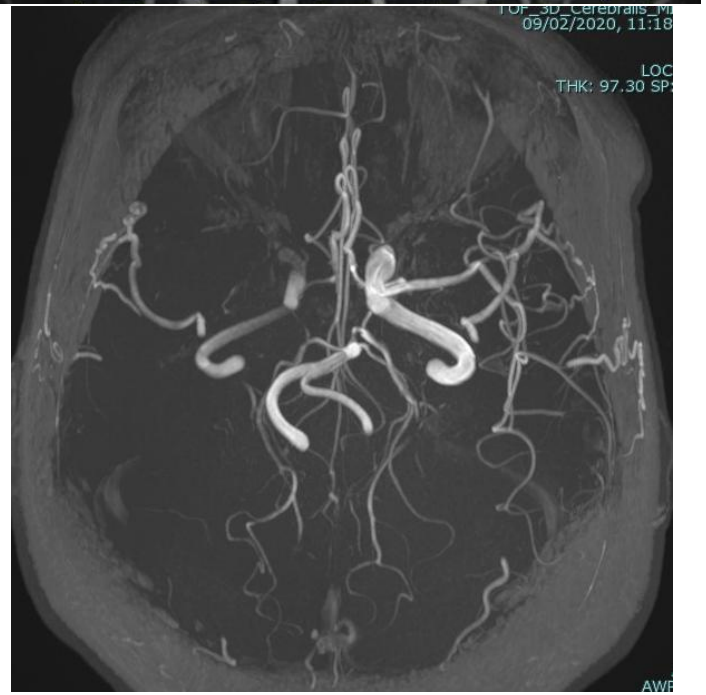

A
P
cm

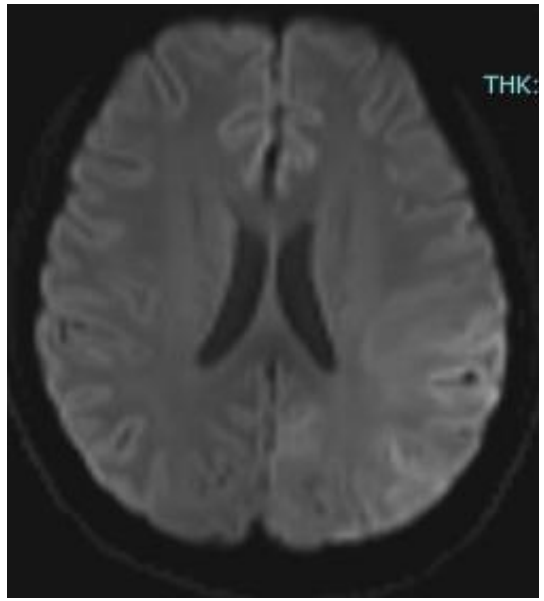

H
F
cm


A

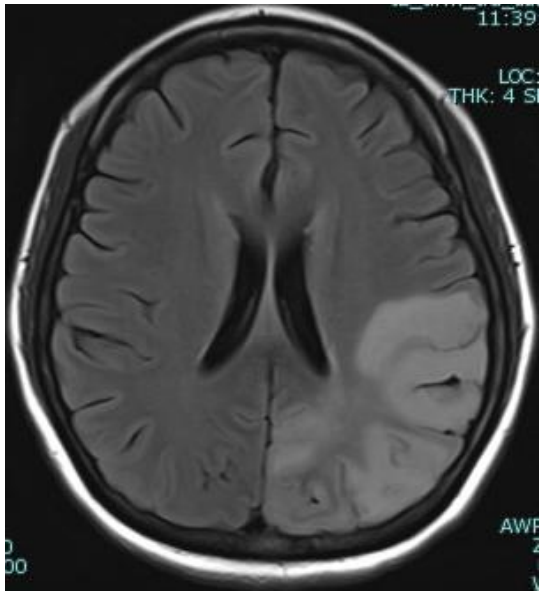

A

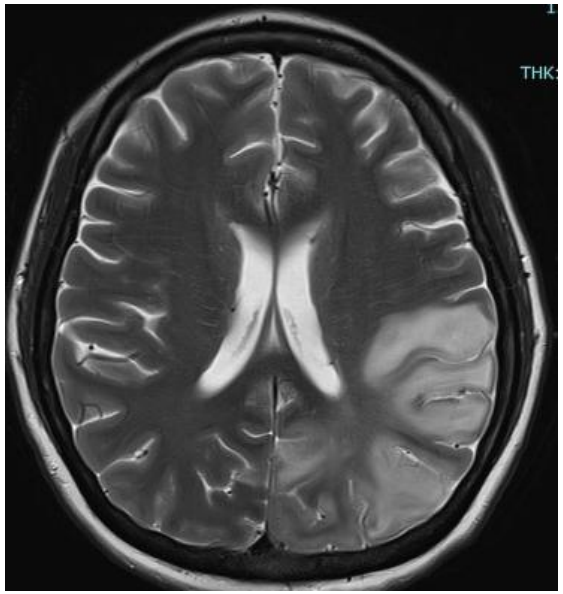


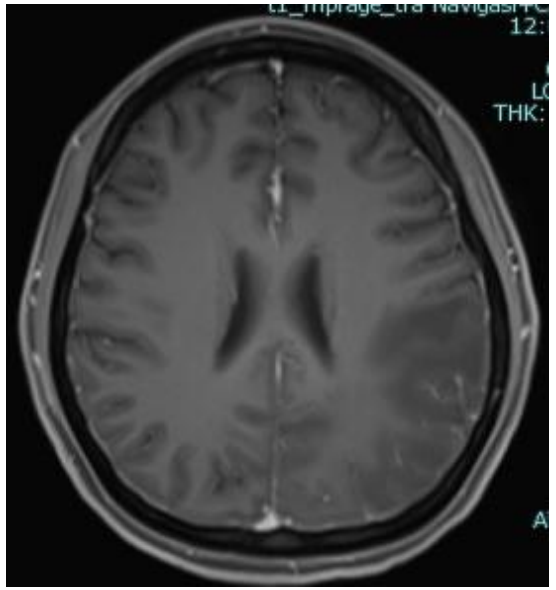


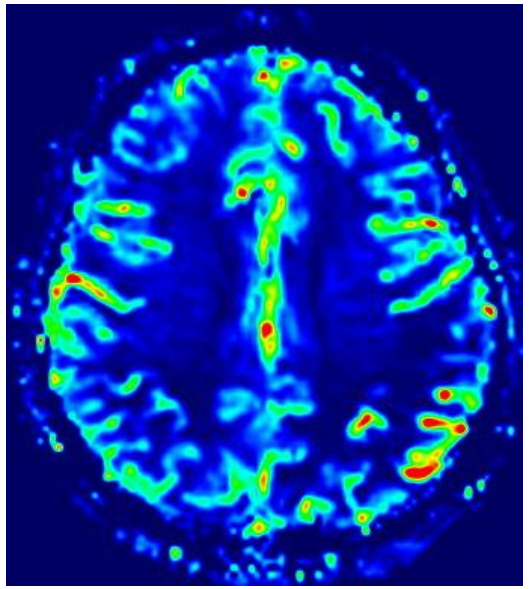


Subacute Infarct with luxery perfusion









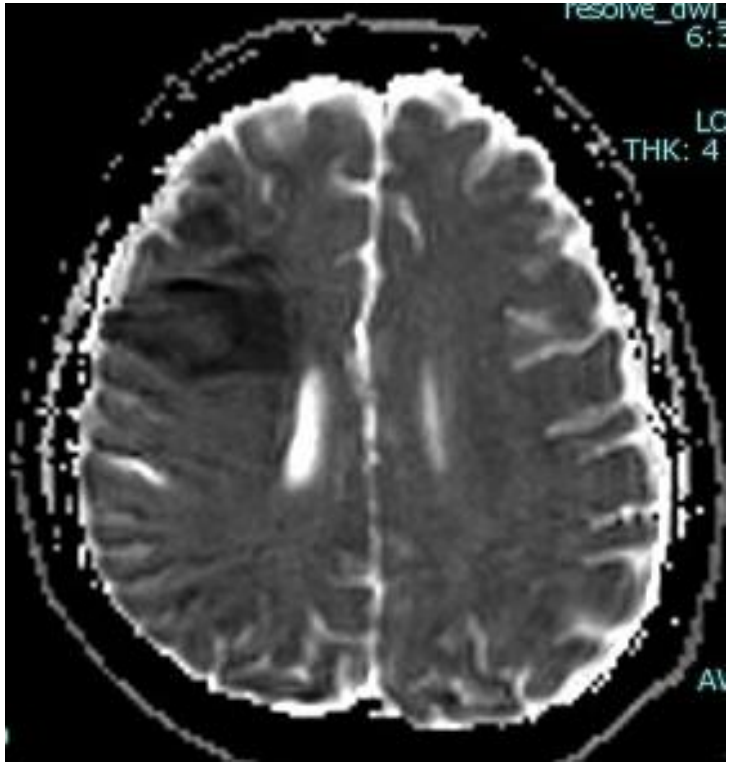
Susceptibility weighted sequences (SWI)

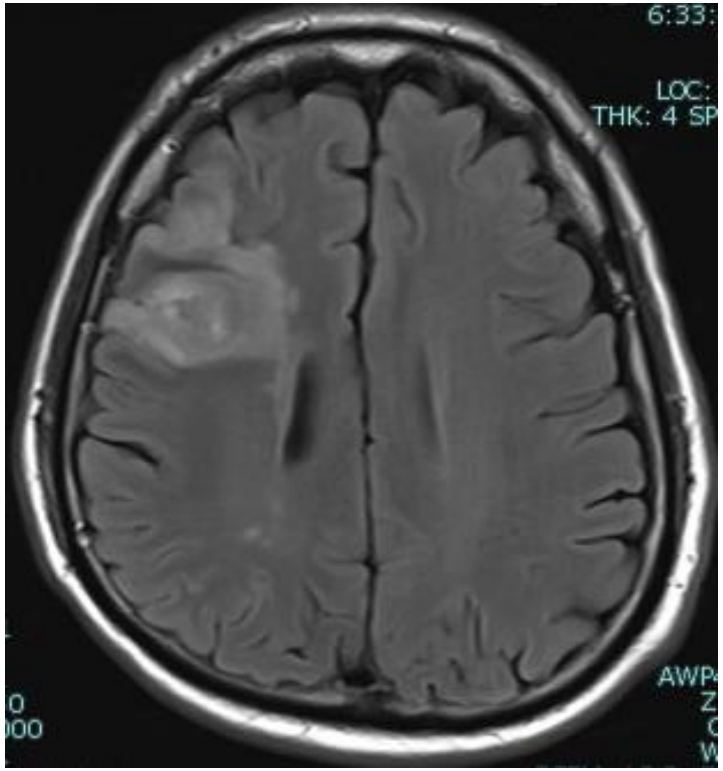
- Sensitive → hemorrhage (deoxyhemoglobin) , superparamagnetic hemosiderin, diamagnetic calcium , iron storage, slow venous blood
- Detecting bleeding from vessels as small as 200 µm

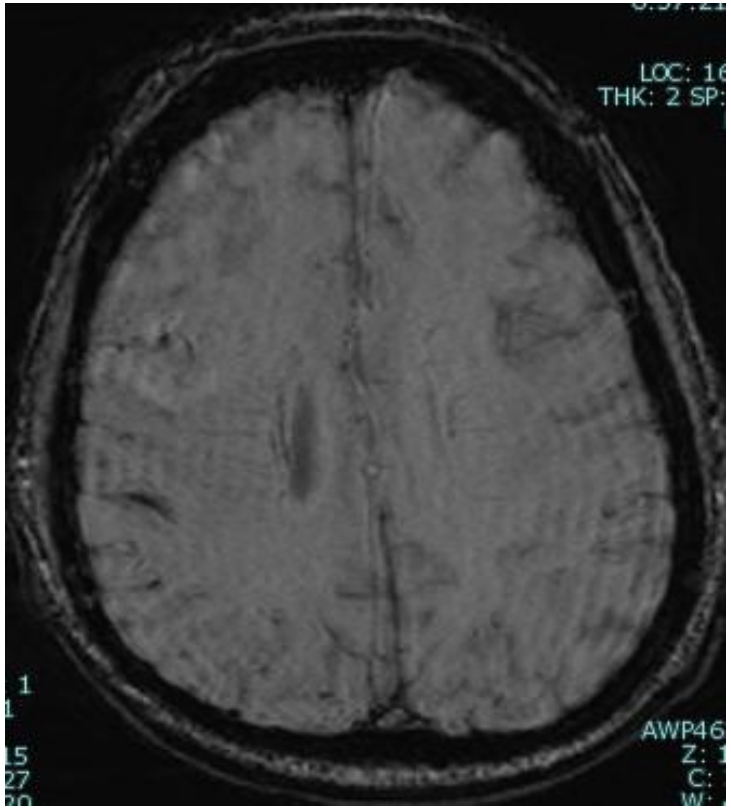
- Microbleeds : small, rounded, homogeneous, hypointense foci
- Hemorrhagic transformation : small petechial areas of micro bleeding to large parenchymal hematoma

- Can evaluate collaterals

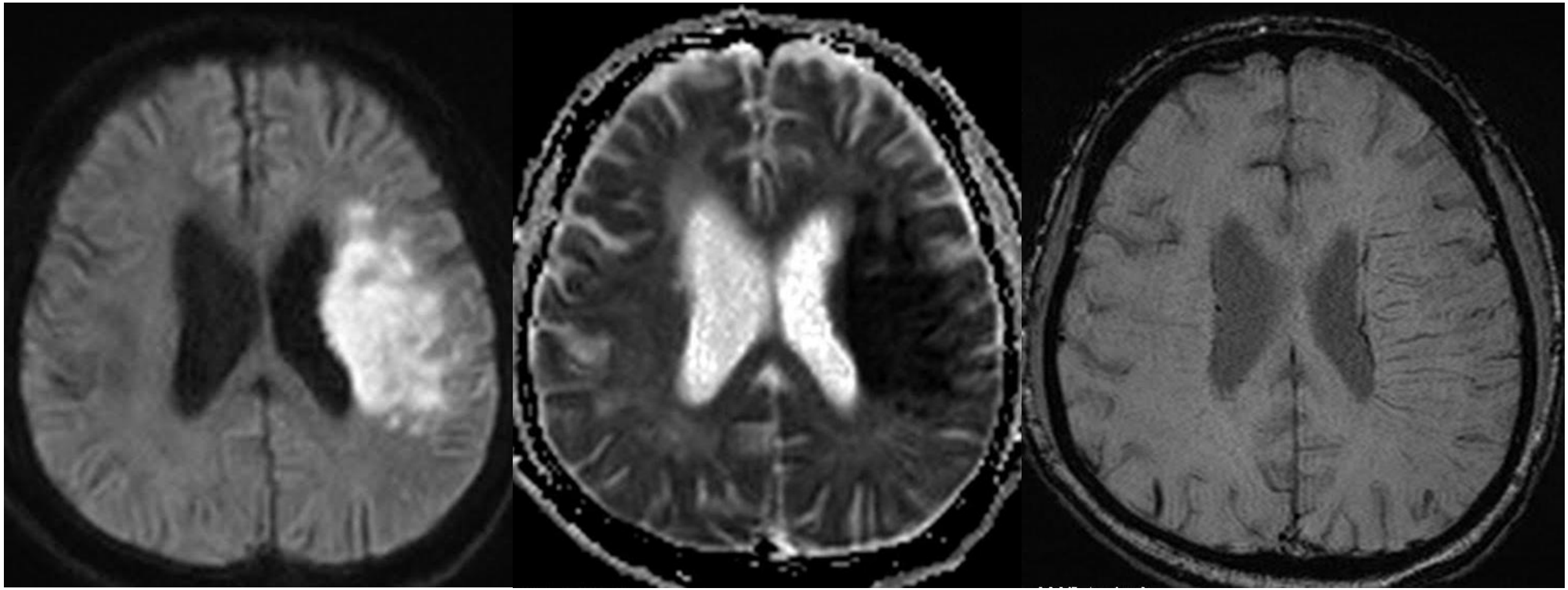
Acute infarct with hemorrhagic transformation



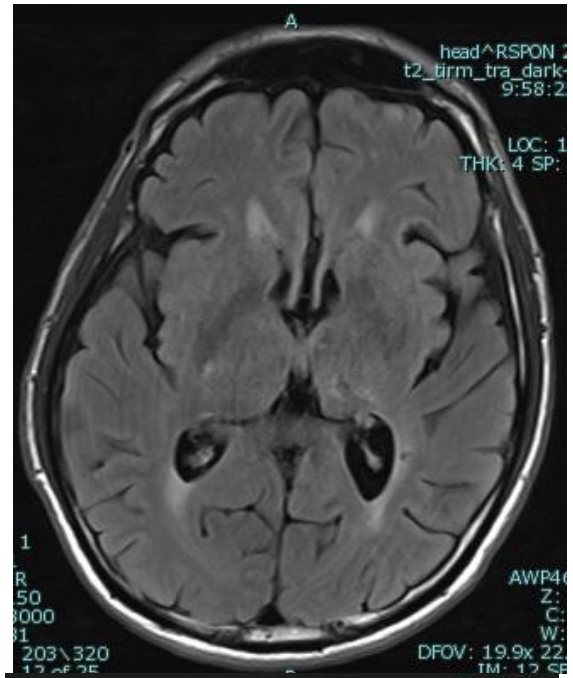


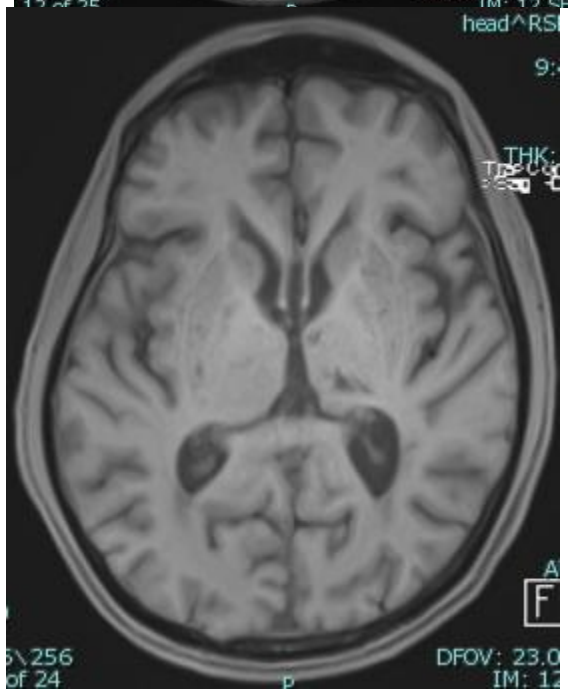


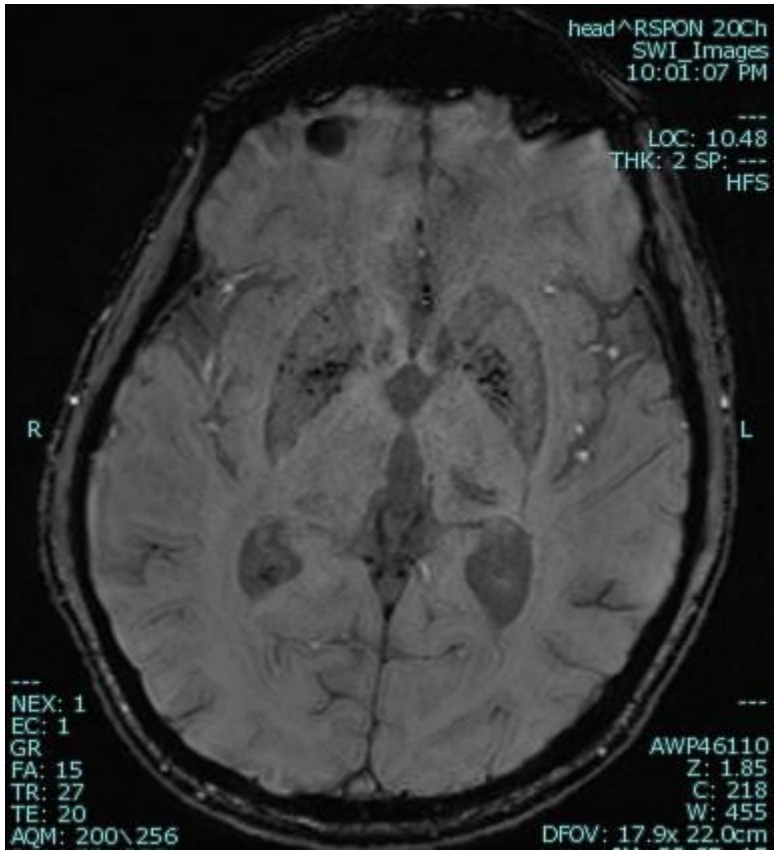
Acute Infarct with collateral

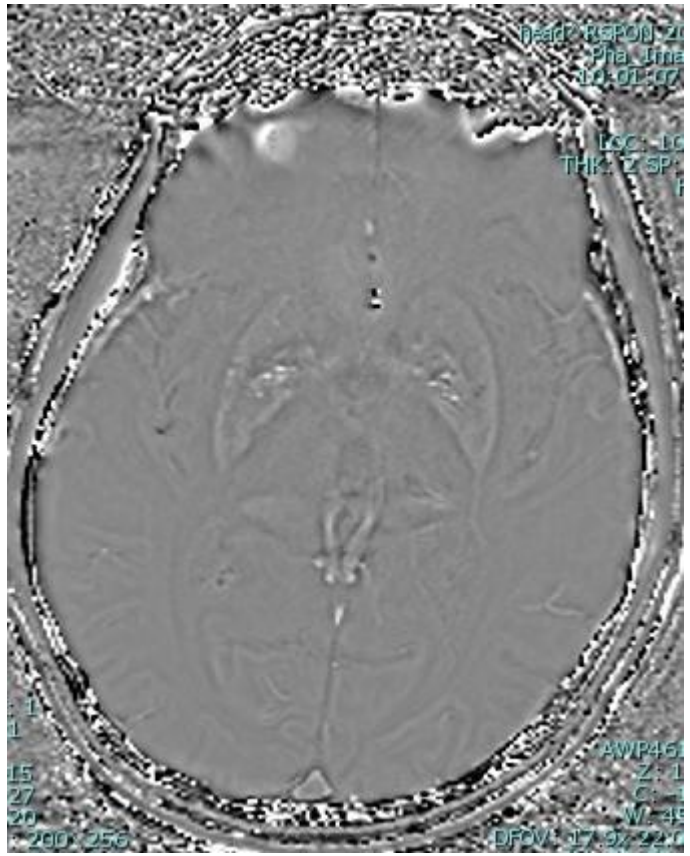


Ensefalomalacia dengan post bleeding









MRI in AIS

Time	DWI	ADC	T2/FLAIR
Hyperacute (1-6 hrs)	Hyperintense	Hypointense	Normal
Early Acute Infarct (6 – 24 hrs)	Hyperintense	Hypointense	Hyperintense approx . 2 to 4 hrs after ictus
Late Acute infarct (1- 3 days)	Hyperintense	Hyperintense	Hyperintense T1: Hypointense