

Neuroimaging Clearance in Thrombolysis

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Abstract: *We all know that time is brain. Per stroke in one second, 32, 000 neurons are lost. In one minute 1.9 million neurons are lost. In one hour 120 million neurons are lost. If left untreated, 1.2 billion neurons are lost. Here comes the early identification of stroke and salvaging the Ipenumbra which is the surrounding region of the infarcted area. Thrombolysis being decentralized now every physician should be aware of the early signs of ischemia in neuroimaging. To familiarise these grey and white magical signs to the residents is the main aim of writing this article. CT is the primary imaging modality used for selecting appropriate treatment in patients with acute stroke.*

Keywords: infarct, artery, CT, MRI, Thrombolysis

Awareness of the typical findings, pearls, and pitfalls of CT image interpretation is therefore critical for physicians, stroke neurologists, and emergency department providers to make accurate and timely decisions regarding both (a) immediate treatment with 3 intravenous tissue plasminogen activator up to 4.5 hours after a stroke at primary stroke centers and (b) transfer of patients with large-vessel occlusion (LVO) at CT angiography to comprehensive stroke centers for endovascular 4thrombectomy (EVT) up to 24 hours after a stroke.

Inclusion criteria for Thrombolysis (Must be all YES)

- 1) Age >18 years
- 2) Time of onset well established to be less than 4.5 hours
- 3) Clinical diagnosis of ischemic stroke with neurologic deficit
- 4) Head non-contrast CT scan (NCCT) without hemorrhage.
- 5) Consent form/risks/benefits: Discussed and documented in chart
- 6) Premorbid modified ranking scale < 3

Exclusion Criteria

SBP >185mm Hg or DBP > 110mm Hg despite simple measures to lower it acutely. (i.e. after 2 doses of labetalol 10-20mg)

- 1) Coma or severe obtundation
- 2) Stroke or head trauma in last 3 months
- 3) Symptoms of subarachnoid hemorrhage/ history of intracranial hemorrhage
- 4) Gastrointestinal/urinary or respiratory hemorrhage in last 21 days.
- 5) Known bleeding diathesis/peritoneal or hemodialysis
- 6) Major surgery within last 14 days
- 7) Arterial puncture at a non-compressible site within last 7 days
- 8) MI in the last 6 weeks
- 9) INR > 1.7

Introduction

An in-depth discussion of the pathophysiology of acute ischemic stroke is beyond the scope of this review, the concepts of ischemic core and penumbra are essential to understanding the role of the different components of imaging evaluation.

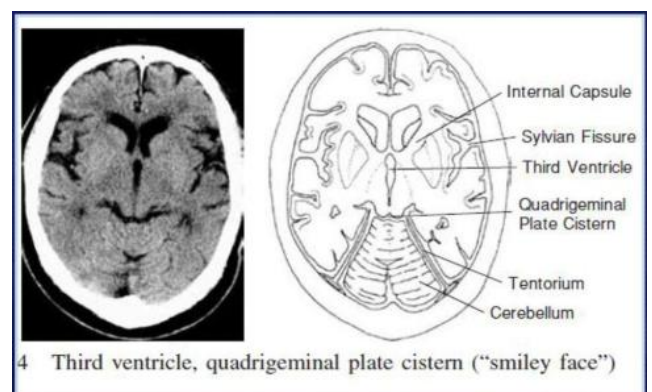
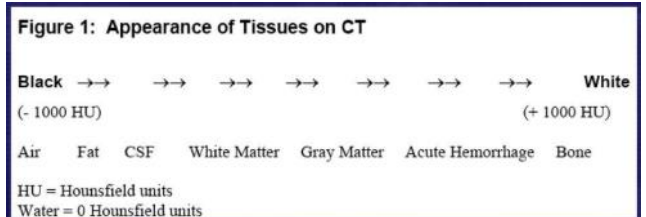
In ischemic stroke, arterial occlusion due to embolism or in situ thrombosis leads to a cascade of cellular events. These events cause local dysfunction and ultimately cell death, leading to development of an infarct core, which is defined as brain tissue likely to die despite immediate reperfusion.

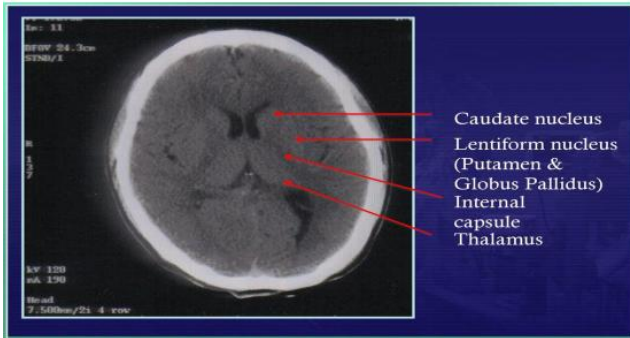
The infarct core is surrounded by the ischemic penumbra, the ischemic tissue at risk for infarction that may be salvaged with timely reperfusion. In the setting of a proximal MCA or ICA LVO, if there is persistent insufficient tissue reperfusion as time passes, there is continued core infarct growth with penumbral loss.

CT Brain:

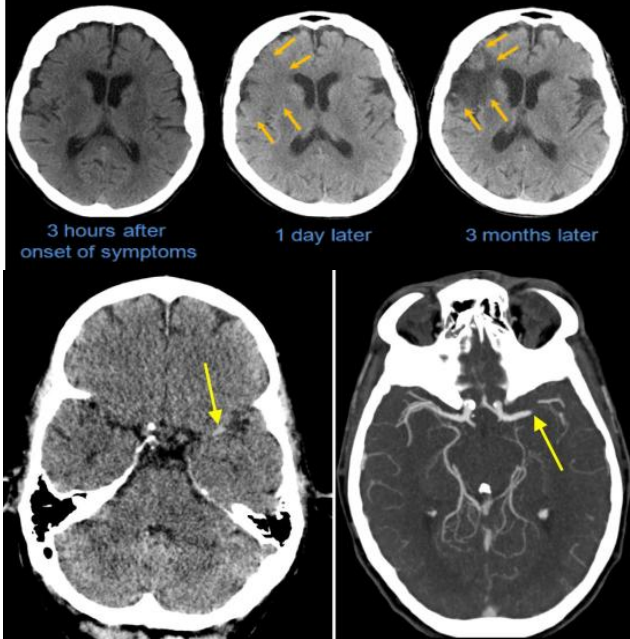
The 5 sensitivity and specificity for acute infarction at non-enhanced CT likely depend on the duration, infarct size, and degree of ischemia.

Sensitivity and specificity for depiction of early ischemic change are also likely better for the anterior circulation than the posterior circulation, primarily owing to artifact at the skull base from thick surrounding bone.

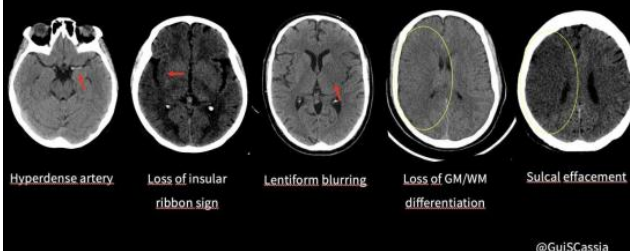




Natural Progression of Infarction



Early ischemic stroke signs



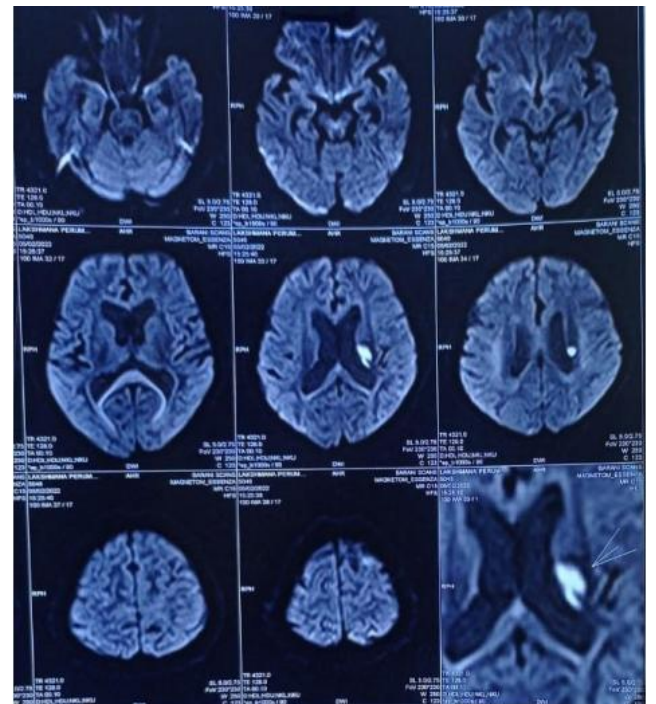
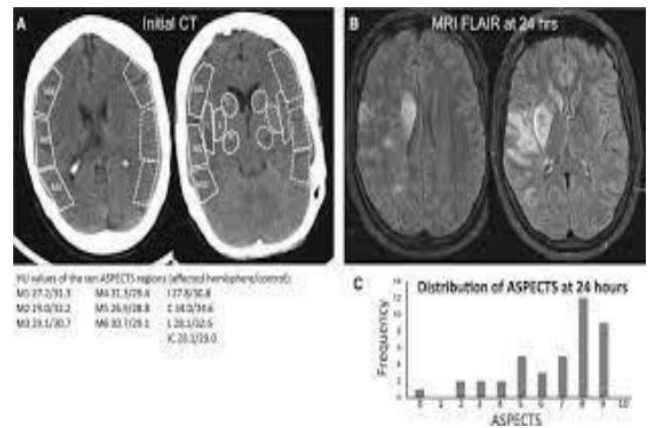
MCA INFARCT

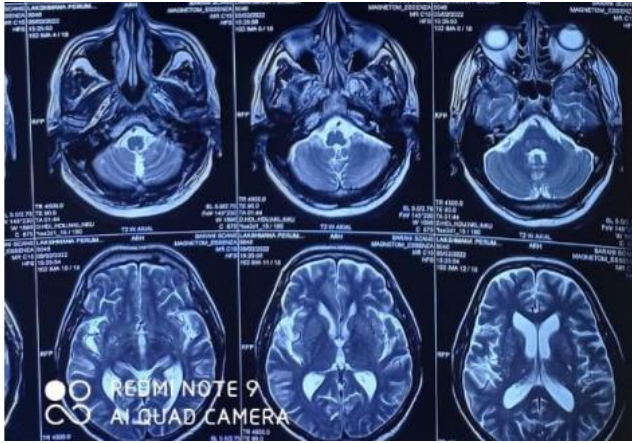


Aspects Score: Alberta Stroke Program Early CT Score



ASPECT score may be used to predict the risk of complications in stroke patients. (ASPECT <6 maybe indicative of high risk and patient may be referred to higher center).





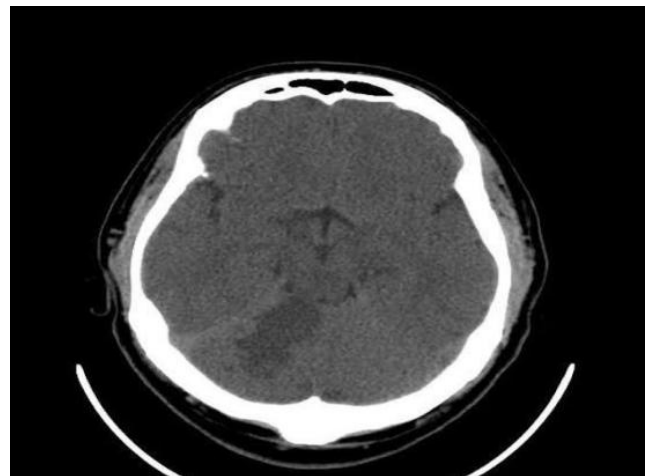
Anterio Cerebral Artery Infarct



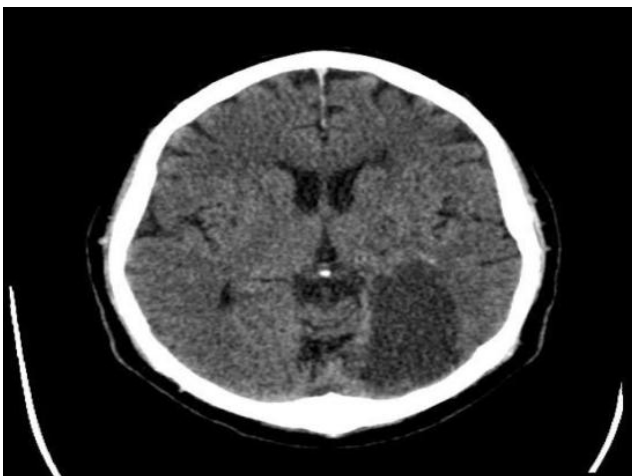
Left Cerebellar Infarct



Posterior Cerebral Infarct



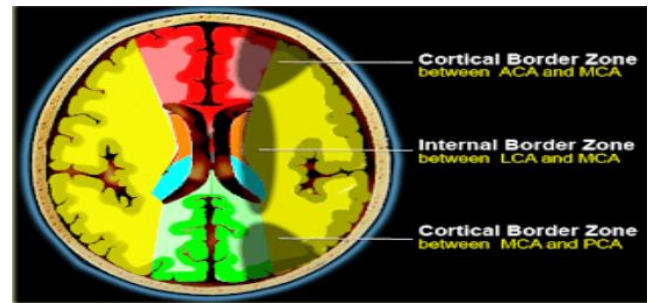
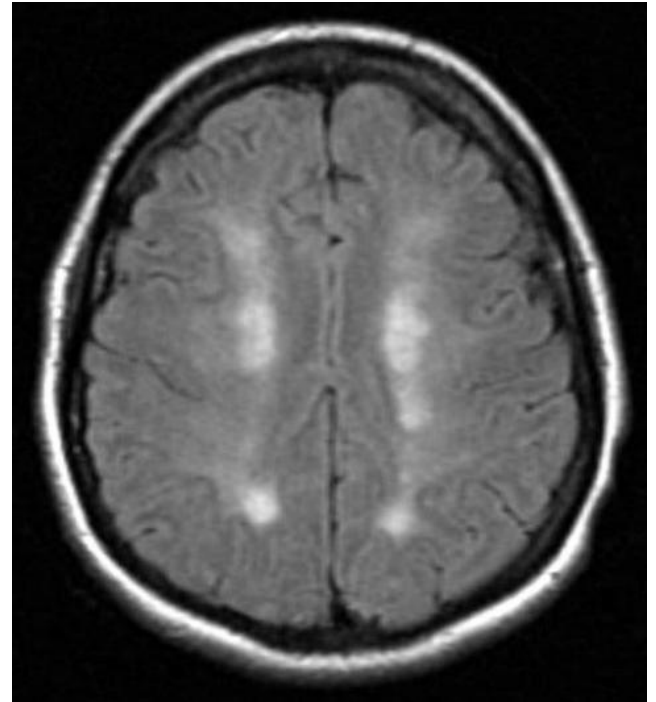
Right Midbrain Infarct



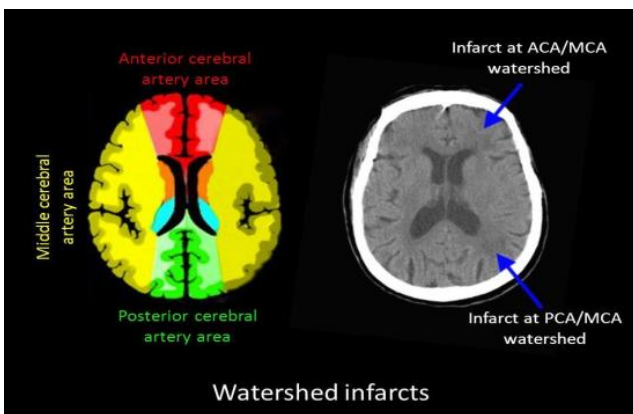
Left Thalamic Infarct



Left Pontine Infarct



Watershed Infarcts



PITFALLS

Challenges in evaluation of nonenhanced CT images in stroke are often caused by artifact from patient motion that can be associated with alterations in consciousness as well as from streak artifact from bone or hardware. Skull base artifact at the petrous apices or posterior fossa often limits the visibility of subtle hypodensities in acute infarct. Skull base streak artifact can also mimic the hyperattenuating MCA sign. Comparison with the contralateral side will typically help resolve any ambiguity.

Coronal and sagittal reformations can improve visualization and help one distinguish artifact, infarct, and hemorrhage in some cases, especially at locations such as the sylvian fissure, where axial volume averaging can mimic GSD loss. In situations where axial images are tilted off axis, coronal and sagittal reformations can display areas of nonanatomic linear hyperattenuation that cross through different brain regions and structures.

MRI

Positive diagnosis of ischaemic stroke has increased the use of magnetic resonance imaging—for example, diffusion weighted imaging, which shows early ischaemic changes as a bright white lesion (“lightbulb”).

More ischaemic strokes show up on diffusion weighted



imaging than on computed tomography or conventional magnetic resonance imaging in the first few hours⁹ and weeks later, which makes this technique especially useful for positive identification of an ischaemic stroke in patients presenting up to eight weeks after stroke.

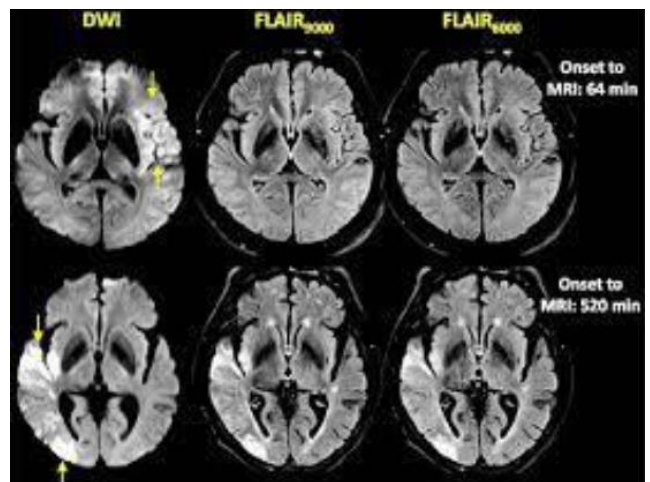
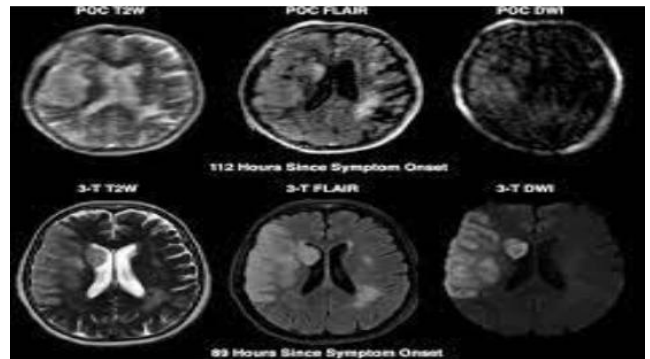
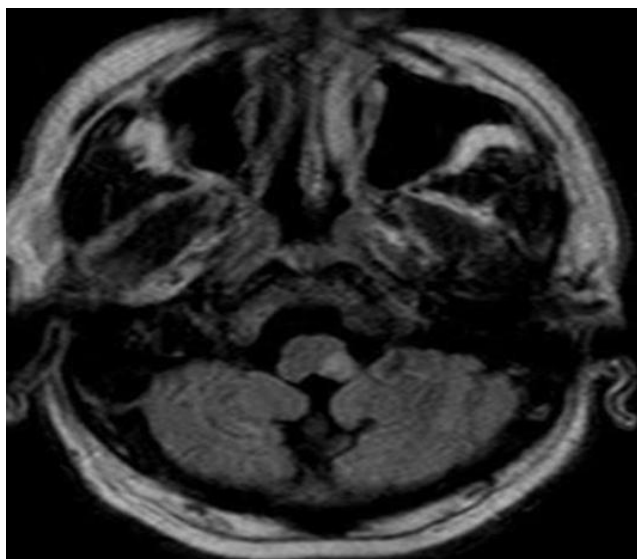
Previous intracerebral haemorrhage is visible indefinitely as a low signal (black) ring or dot on gradient echo magnetic resonance sequences, which makes this technique useful in patients presenting too late for computed tomography.

However, magnetic resonance imaging may not identify hyperacute intracerebral haemorrhage correctly; is difficult to use routinely in acute, particularly severe stroke; is less often available than computed tomography; requires more cooperation by the patient for a longer time; is very noisy and upsets confused patients; and about a fifth of patients cannot undergo magnetic resonance imaging (because they are too ill or confused or have an intraocular or intracerebral metallic foreign body or pacemaker).

Whether lesions seen on diffusion weighted imaging indicate permanent neuronal damage or include some tissue that could recover is not clear. Magnetic resonance perfusion imaging shows under-perfused brain, often over a larger area of brain than the lesion seen on diffusion weighted imaging.

The difference between the lesions seen on diffusion weighted imaging and perfusion scans may indicate potentially salvageable brain (“ischaemic penumbra”), although many issues remain—for example, defining the imaging boundaries of recoverable tissue, the “point of no return,” and the relation with the duration of ischaemia.

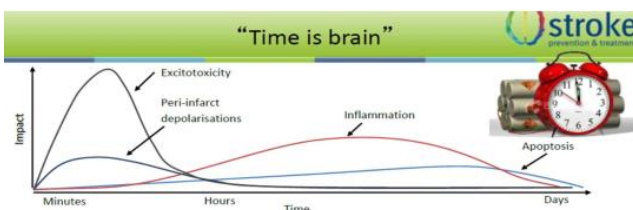
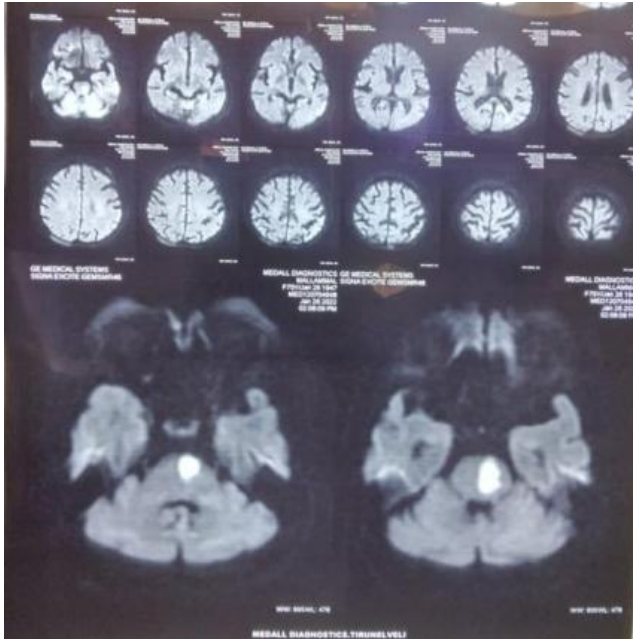
Left Medullary Infarct



Pontine Infarct

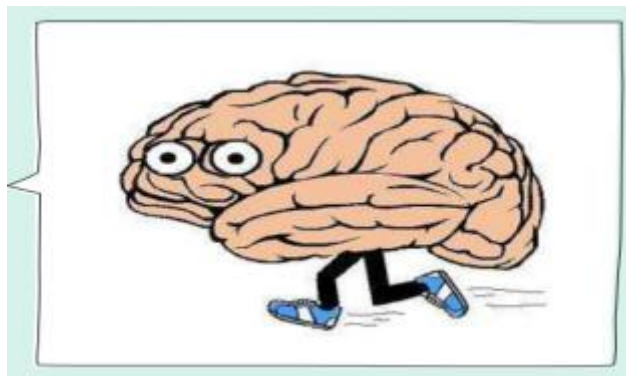


Pontine Infarct



Estimated Pace of Neural Circuitry Loss in Typical Large-Vessel Supratentorial Acute Ischaemic Stroke

	Neurons Lost	Synapses Lost	Myelinated Fibres Lost	Accelerated Aging
Per Stroke	1.2 billion	8.3 trillion	7140 km	36 y
Per Hour	120 million	830 billion	714 km	3.6 y
Per Minute	1.9 million	14 billion	12 km	3.1 wk
Per Second	32,000	230 million	200 m	8.7 h



Conclusion

Awareness of common findings, pearls, and pitfalls of multimodal stroke CT evaluation and interpretation has therefore become essential for providers caring for patients with acute stroke. CT evaluation of acute ischemic stroke is robust, rapid, and widely available.

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