GSC Advanced Research and Reviews

eISSN: 2582-4597 CODEN (USA): GARRC2

Cross Ref DOI: 10.30574/gscarr

Journal homepage: https://gsconlinepress.com/journals/gscarr/

(REVIEW ARTICLE)

# Imaging strategy for acute stroke patients, a review of literature

Moustafa E. Radwan [1, 2, *]

[1] *Department of Radiology, Taibah University, Almadinah Almunawarah, Saudi Arabia*
[2] *Department of Radiology, Assiut University, Egypt*

GSC Advanced Research and Reviews, 2021, 06(01), 037–046

Publication history: Received on 28 December 2020; revised on 20 January 2021; accepted on 22 January 2021

Article DOI: https://doi.org/10.30574/gscarr.2021.6.1.0134

## Abstract

Background: This review article focused on the utilization and impact of current neuroimaging techniques for the patient with acute stroke, emphasizing how imaging builds upon clinical assessment to establish diagnosis or etiology and guide therapeutic decisions.

When requesting imaging examinations in patients with stroke symptoms; it is crucial to evaluate four significant parameters of stroke; parenchyma, vessels, perfusion, and penumbra. Evaluation of all these four parameters, in their right request are essential to grasp the explanation and potential therapy decisions for stroke in a specific patient. Extensive neurovascular imaging conventions utilizing multimodality CT (NCCT, CT Angiography, and CT Perfusion) or multimodality MRI (DWI-perfusion mismatch or DWI-FLAIR mismatch, and MR Angiography) might be utilized to evaluate the acute stroke patients and provide all the needed data for treatment of them inside minutes after the patient lands at the emergency clinic. Using this approach will help to discriminate between hemorrhagic and ischemic stroke as presence of frank intracerebral hemorrhage contraindicates reperfusion treatment, permits the choice of patients with large vessel occlusion for endovascular treatment and answer the important "tissue clock" within 6 hours from symptom and even with late-presenting (> 6 h) or wake-up stroke.

Conclusions: As patients with acute cerebral stroke might be critically ill, the initial imaging scanning for acute stroke patients should be constrained to the procurement of useful data only, considering the accessible therapeutic options at a given place at any given time.

**Keywords:** Stroke; Imaging Strategy; Wake-Up Stroke; Endovascular Treatment; cerebral Hemorrhage

## 1. Background

Cerebrovascular strokes usually subdivided into two major types: ischemic due to insufficient blood flow to the brain and hemorrhagic due to bleeding[1]. Recently, a major change in the management of acute stroke has occurred, from a hopeful bedside "keep a watch out" disposition towards dynamic treatment[2]. Stroke recovery teams have worked cautiously and altogether to reduce the door-to-needle time from emergency clinic admission to the organization of intravenous thrombolytics with recombinant tissue plasminogen activator (iv-rtPA) for acute stroke patients as the treatment adequacy is time-dependent, hence the saying "time is brain" [3].

The utilization of advanced neuroimaging strategies; Multimodality CT [Non-contrast CT (NCCT), CT Angiography (CTA), and Perfusion CT (CTP)] or Multimodality MRI [utilizing Diffusion-Weighted Imaging (DWI)- MRI perfusion (PWI) mismatch or DWI- Fluid Attenuated Inversion Recovery (FLAIR) mismatch] permits the choice of patients with

* Corresponding author: Moustafa E. Radwan
Department of Radiology, Taibah University, Almadinah Almunawarah, Saudi Arabia.

large vessel occlusion for endovascular treatment (EVT) and might be utilized to evaluate the "tissue clock" within 6 hours from symptom and even with late-presenting (> 6 h) or wake-up stroke (WUS)[2, 4].

## 2. Main text

When requesting imaging examinations in patients with stroke symptoms; it is crucial to evaluate four significant parameters of stroke; parenchyma, vessels, perfusion, and penumbra[5]. Thought and estimation of all these four parameters, in their right request, are essential to grasp the explanation and potential therapy decisions for stroke in a specific patient. Extensive neurovascular imaging conventions utilizing CT and/or MRI would now have the option to check all these parameters within minutes after the patient lands at the emergency clinic[6, 7].

### 2.1. I-Parenchyma

Imaging of the brain gives data that can guide patient management by:

*2.1.1. Identifying the lesion "Is it a stroke".*

It is essential to identify the nature of the lesion to be either ischemic or hemorrhagic stroke or due to other lesions that can cause the same clinical picture of stroke; like the trauma of the craniocerebral and cervical regions, meningitis, and encephalitis, intracranial lesions like masses or subdural hematoma. It also may be due to metabolic causes; hyperglycemia and hypoglycemia, ischemia following cardiac arrest, and drug/narcotic overdose[8].

*2.1.2. Determining the type of stroke.*

Initially, it is important to discriminate between hemorrhagic and ischemic stroke as presence of frank intracerebral hemorrhage contraindicates reperfusion treatment.

*2.1.2.1. Hemorrhagic stroke:*

CT is the standard method in detecting intra cerebral hemorrhage (ICH). A spot sign at CT angiography which is a focus of enhancement inside a hematoma due to active contrast media extravasation helps to predict the danger of hematoma extension[9].

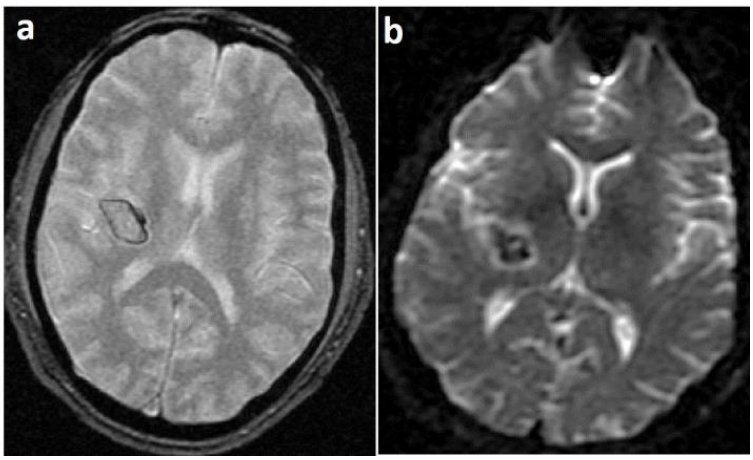


**Figure 1** (a&b) (a) T2*WI and (b) DWI-b0 showed hypo intense signal signifying acute Rt. sided cerebral hemorrhage.

Nowadays MRI has been progressively utilized in the evaluation of acute stroke patients. However, MRI must have the option to identify early hemorrhage to be utilized as a single imaging investigation used before treatment for example, thrombolysis. Gradient T2*- weighted MRI sequences (including Gradient-recalled echo (GRE) and susceptibility-weighted imaging (SWI) sequences) are similarly sensitive for the detection of acute intracerebral hemorrhage as a diffuse signal loss (hypointensity). SWI and GRE can show ICH in patients scanned somewhere in the range of 2.5 and 5 hours from the onset of symptoms (Figure 1) [10].

DWI-b0 EPI image that is obtained with DWI sequence show signal loss similar to that in T2*-weighted GRE sequence, so DWI-b0 image may thus be useful in the detection of hemorrhage without the requirement for additional examining time (Figures 1&2) [11].

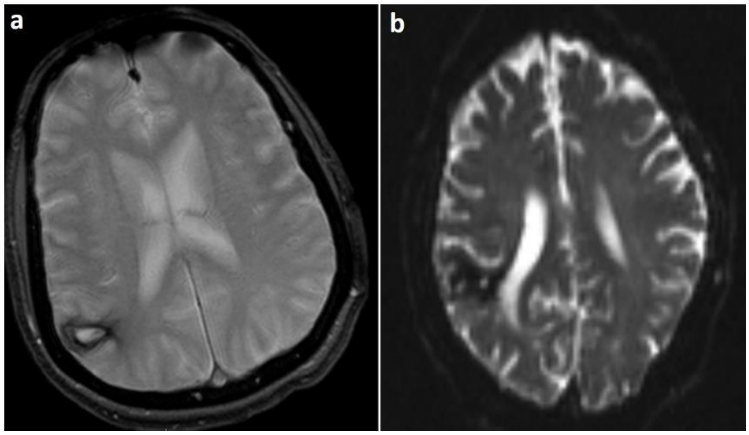


**Figure 2** (a&b) (a) T2*WI and (b) DWI-b0 showed hypo intense signal signifying acute Rt. Posterior parietal hematoma.

Both DWI b0 image and GRE sequences are less sensitive for identifying subarachnoid hemorrhages (SAH), which is best depicted on FLAIR images [12]. FLAIR shows acute and sub-acute SAH as a high signal against a dark CSF [13].

*2.1.3. Localizing the stroke "Where is it?"*

Exact confinement of ischemia is not vital, yet deciding responses to the accompanying two inquiries is valuable in the indicative assessment of stroke:

Is the ischemic lesion located in the anterior [i.e., carotid, middle cerebral artery (MCA), or anterior cerebral artery (ACA)] or posterior (i.e., vertebrobasilar) circulation? This data is significant in figuring out which circulation to image. The ACA usually has a great collateral flow from its paired contra lateral vessel, and thus lone ACA infarcts are uncommon (0.6% of all infarcts)[14]. Brain stem circulation lacks collateral protection, so even small brain stem infarcts can produce extreme deficits. Because of the beam-hardening artifact, Non-contrast brain CT (NCCT) and CTP is very constrained in the location of posterior fossa infarcts, which are every now and again observed with MRI [15].

*2.1.4. Is the ischemic incident cortical or subcortical?*

*2.1.5. Quantifying the lesion "How large is it?"*

*2.1.6. Determining the age of the lesion "How old is it?"*

NCCT stays a first-line imaging procedure in the assessment of suspected acute stroke as it is a reliable, universally available, and cost-effective means of assessment. It is utilized to quickly assess for any mimics of acute stroke and to identify proof of an ICH which would contra- indicate IV rtPA treatment.

The findings of early ischemic stroke demonstrate on NCCT as "diminished attenuation of tissue, loss of gray-white matter differentiation, and focal swelling which can lead to the effacement of the sulci or mass effect". In MCA infarct, early recognition of the loss of tissue attenuation can be seen in the basal ganglia, especially in the lentiform nucleus. Loss of gray-white differentiation at the insular cortex can be also early seen with MCA territory infarction which is known as the "insular ribbon sign". A hyper dense thrombus in a blocked artery can be directly visualized early in NCCT, known as the "dense vessel sign" [16](Figure 3). These findings are usually subtle and difficult to be seen in NCCT in early stroke, narrow window settings can be utilized to recognize them [17].

Within minutes of the onset of ischemic events, standard MRI spin-echo sequences show loss of normal vascular flow void phenomena, however identifying ischemia in the first few hours after onset can't rely on the conventional T2-weighted sequences (T2WI) [18]. Published data suggest that FLAIR images are more sensitive than that of T2WI to subtle increases in bulk water observed in the setting of acute cerebral infarction[19]. Conspicuity of infarct lesions are more clearly seen with FLAIR images in comparison with that appeared with conventional T2WI. Also, ischemic lesions

at areas of T2 prolongation (at the brain-CSF interfaces) are better seen with FLAIR sequences than conventional T2WI as at these sites, the signal alteration can easily be hidden because of partial volume artifact with CSF on conventional T2WI. Occluded arteries can be seen in FLAIR sequences as hyper intense or iso-intense to brain parenchyma (Figure 3) [20].

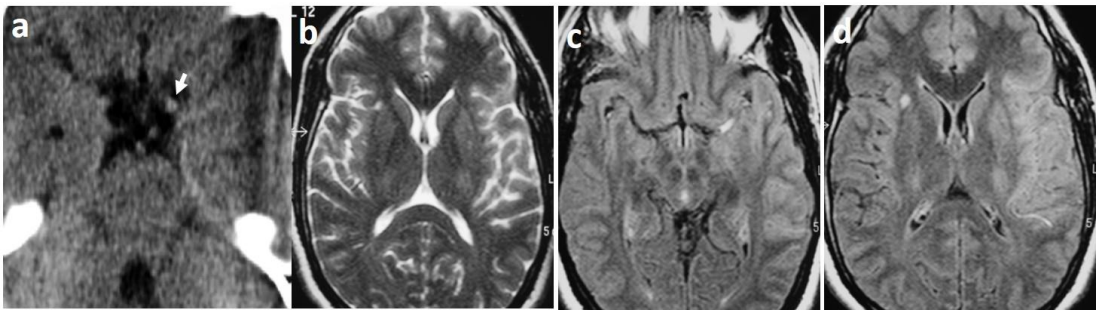


**Figure 3** [a-d] (a) Axial NCCT brain shows hyper dense left MCA sign (white arrow). (b): Axial T2WI shows no definite brain signal abnormalities in Lt. Hemisphere. (c&d) FLAIR MRI show hyper intense Lt. MCA sign with effacement of cortical sulci and faint hyper intense area in the Lt. temporo-parietal region in a case with Rt. sided hemiplegia of 5-hours duration.

With hyper acute ischemic infarction there is diminished free diffusion caused by cytotoxic edema, this leads to diminished apparent diffusion coefficient (ADC) and increased signal on DWI (Figure 4).

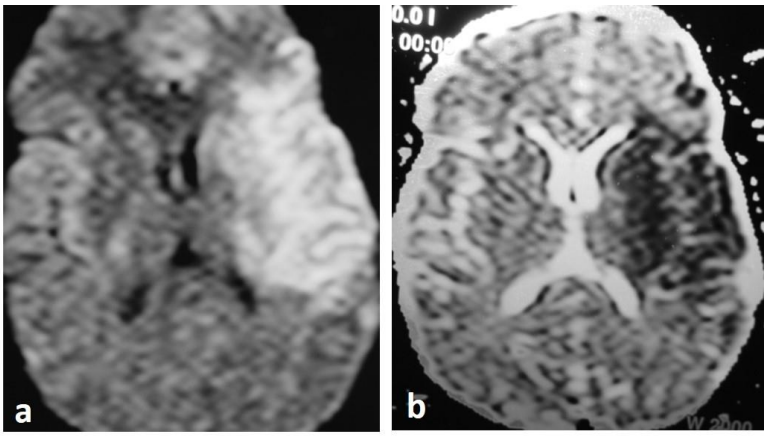


**Figure 4** (a) DWI-b1000, a large zone of hyper intense signal (restricted diffusion) in the Lt. temporo-parietal region. (b) ADC Map shows hypo intense signal of the same lesion (same case as in figure 3).

The area of diffusion restriction represents the “core” or irreversible damage which can extend over 24 hours if there is no reperfusion of conceivably salvageable brain tissue[16]. During the hyper acute stage, DWI recognizes a lesion that is absent on T2WI or FLAIR images, this frequently happens in patients whose brains are imaged within 6 to 12 hours of the start of symptoms[21].

Overall lesion conspicuity on DWI is better than that on both FLAIR and T2WI images even after the passage of the initial 48-hour time from the beginning of ischemic stroke symptoms. Consequently, DWI stays beneficial outside the hyperacute/acute period. The improved conspicuity is because of a higher lesion to background signal intensity ratio, which results from the concealment of signal from freely mobile water protons[22].

Symptoms of stroke appear in a significant number (between 8 and 28%) of patients when they wake (WUS) or with unwitnessed time of onset, this subgroup is commonly summarized as wakeup strokes. The actual time of the start of symptoms is not known in these patients, and symptom onset is estimated from the moment when the patient was most recently seen well. Thus, most WUS patients are not qualified for IV-rtPA or EVT. Imaging may assist in identifying WUS patients who would get profit from therapy and exclude patients with already extreme ischemia [23].

In acute ischemic stroke (AIS), a positive DWI and negative FLAIR, or DWI-FLAIR mismatch, can be utilized to confirm the onset of stroke is within 4.5 hours. This is on the grounds that the tissue damage which causes FLAIR abnormality has not yet happened inside that window. The theory of DWI-FLAIR mismatch can be utilized to recognize potential thrombolysis candidates when there is an unknown symptom onset or a "wake-up" stroke[24].

DWI could identify acute lesions in patients who have multiple chronic ischemic lesions. T2WI and FLAIR sequences could not differentiate acute from chronic ischemic lesions because all lesions will appear with similar hyper intense signal. DWI together with T2WI gives a method for defining the age of lesions (Figure 5). Similarly, DWI could recognize the new expansion of old ischemic lesions, while expansions of previously settled infarcts are as often as possible hard to recognize on T2WI or FLAIR images as a result of the similarity in signal characteristics [25].

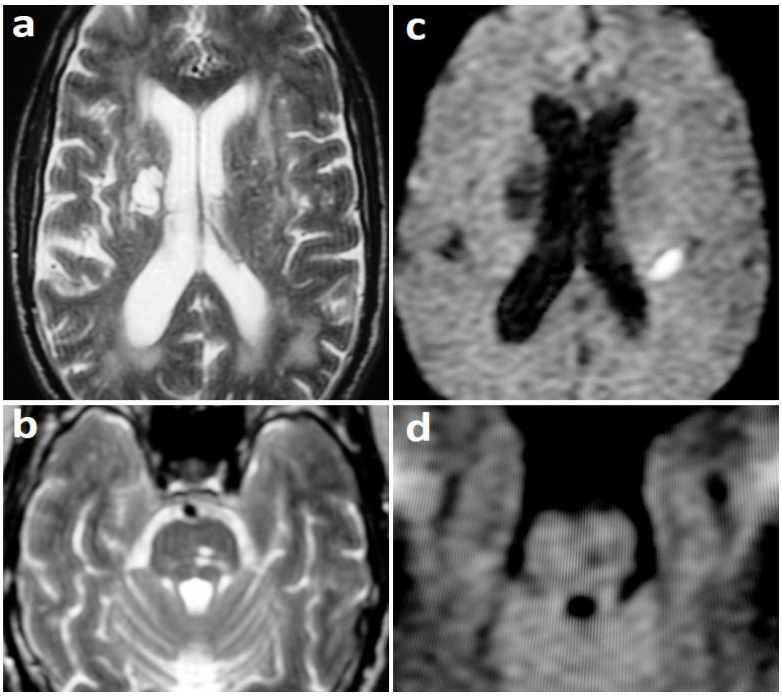


**Figure 5** (a-d); Multiple ischemic infarctions of variable chronological ages mostly embolic in nature. (a&b) Axial T2WI; (c&d) Axial DWI-b1000. (1) Old ischemic infarctions at left pontine isthmus, right periventricular region exhibiting dark signals in DWI b1000 with hyper intense signal in T2WI. (2) Hyper acute ischemic infarction in the left parietal region exhibiting bright signal in DWI b1000, not visualized in T2WI.

However, MR imaging is not accessible 24 hours consistently 7 days out of each week in many stroke centers. MR imaging is additionally susceptible to patient motion. MR imaging, despite its exactness, can possibly take additional time than un-enhanced CT within the acute stroke workflow[15].

### 2.2. II- Vessels

Vessels is one of the most important parameters to be imaged while assessing patients with AIS, as the findings will help to address inquiries regarding the mechanism of the stroke, whether thrombotic, embolic, or hemodynamic, and the danger of future occasions by; (1) Defining occlusive arterial disease (Is there blockage?), (2) Locating the site of occlusion in extra-cranial or intra-cranial vessels (Where? Carotid? Vertebrobasilar? Intra-cranial? Extra-cranial? Other?), (3) Determining the pathology (atherosclerosis? dissection? other?), and (4) Detecting other vascular lesions "malformation, aneurysm, arterial compression, venous thrombosis". This data will help decide the decision of treatment, particularly IV-rTPA or EVT or surgical intervention to prevent future strokes[26].

Currently, non-invasive vascular imaging is generally utilized in the diagnostic examination of AIS, especially when endovascular therapy is planned. The significant objectives of CTA and Magnetic resonance angiography (MRA) are to identify proximal vessel occlusion, evaluate collateral circulation, and to help in getting ready for EVT [16].

Therefore, invasive cerebral angiography can be saved for those patients who fulfill the clinical and radiological criteria of submission to subsequent intraarterial thrombolysis and who have a significant more prominent probability to profit by such treatment[27] (Figures 6-8).

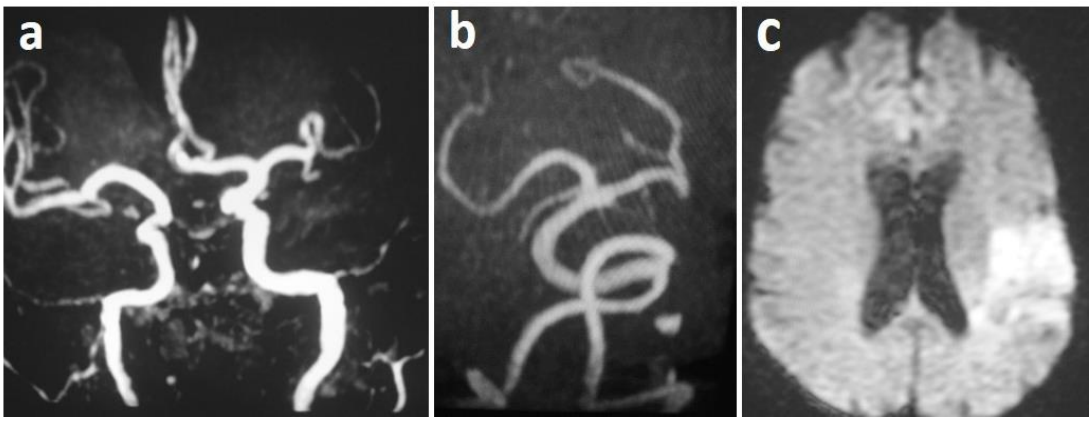


**Figure 6** (a-c); (a&b) 3D TOF MRA show non visualized Lt. MCA M3 branches, associated absent A1 segment of Rt. ACA (normal variant) and Dolichoectasia of basilar artery (c) DWI b1000 shows a bright signal of acute infarction in the Lt. posterior parietal region.

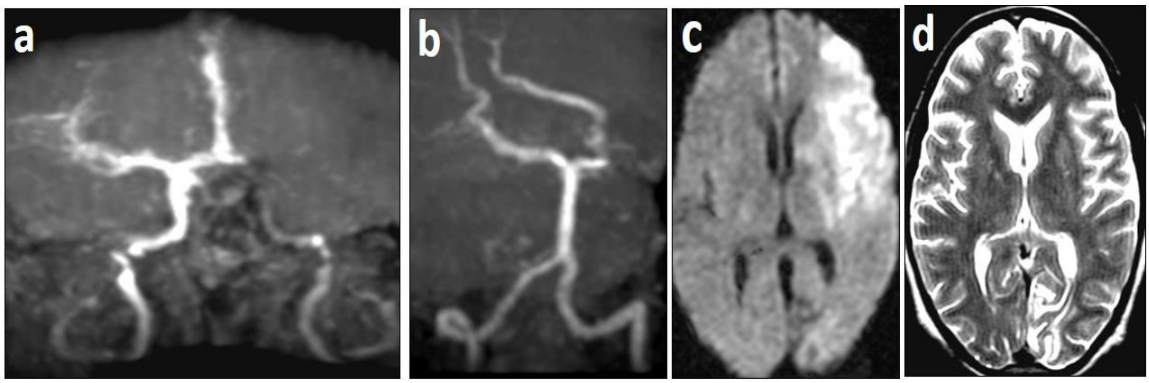


**Figure 7** (a-d); (a&b) 3D TOF MRA show occluded Lt. ICA with no flow through Lt. MCA, a plaque at Rt. ICA and a stenotic segment at Lt. PCA. (c) DWI b1000 shows a bright signal in the distribution of left MCA. (d) T2WI shows no abnormal signal in the same area with a bright signal encephalomalacic area at the left occipital region.

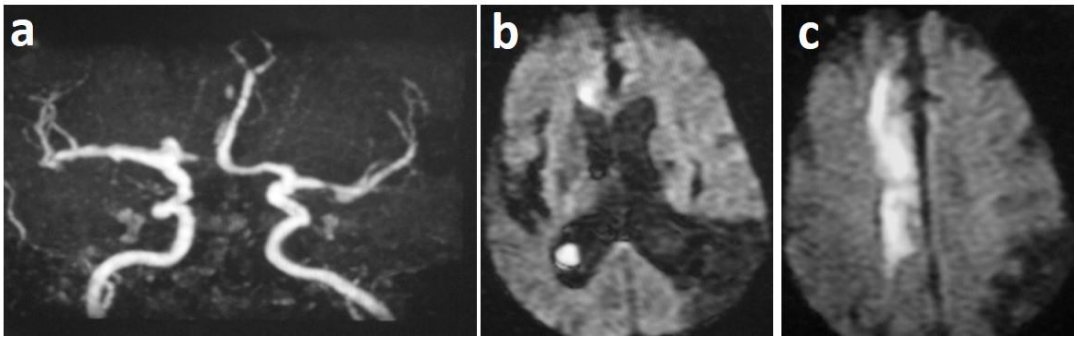


**Figure 8** (a-c); (a) 3D TOF MRA shows thrombosis of the A1 segment of Rt. ACA, with stenosis at M1 segment of Lt. MCA. (b&c) DWI-b1000, showing acute infarction in the territory of Rt. ACA, exhibiting bright signal with old infarction in the Lt. parieto-occipital region exhibiting hypo intense signal.

But why CTA or MRA when conventional catheter-based angiography is truly at its best today? The term conventional angiography is synonymous today with very sophisticated catheter techniques, high-resolution digital subtraction image acquisition technology, and a wide choice of safe contrast media, as well as a considerable amount of collective know-how and experience. So why try to supplant such an excellent method, which is viewed as the gold standard in

vascular imaging? The appropriate response is obvious. Catheter angiography, even in the best hands, stays intrusive with potential hazards of morbidity and even mortality [14, 28].

Discovery of vessel occlusion with Doppler ultrasonography is time-consuming, profoundly reliant on examiner experience, and may technically not be conceivable, though CTA and MRA can provide this data. Compared with conventional angiography and Doppler sonography, CTA and MRA have the advantages of not being operator dependent[29].

### 2.3. III-Perfusion

The primary objective of perfusion imaging is to discriminate between “penumbra” and the ischemic “core. The perfusion demonstrates the entire cerebral blood flow (CBF) landing at a specific brain area at a given moment in time, both by means of normal routes and recruited collateral. It is not adequate to know at what level the "vessels" are occluded: it is the individual variety in collateral, vascular autoregulation, and resulting net perfusion that means brain survival or infarction. An ICA or MCA occlusion in one patient might be a coincidental finding if there are associated good collaterals; however, in the other, it might prompt an overwhelming or deadly infarction. The distinction lies in the time course of occlusion and the potential collateral pathways, not the site of vessel occlusion per se [6].

Both Perfusion CT (CTP) and MRI perfusion (PWI) are obtained through the injection of a tracked bolus of IV contrast with sequential scanning of a volume of brain parenchyma during the injection. The determined maps for perfusion scanning include cerebral blood volume, cerebral blood flow, mean transit time, and time-to-max (Tmax) or time-to-peak contrast concentration. The mismatch between the total area of critically hypoperfused tissue and the ischemic core is utilized to assess the penumbra [30].

MRI perfusion can show equivalent data as CT-perfusion and when it is based on the utilization of contrast MRI perfusion has impressive preferences particularly in getting absolute measures of perfusion. It is likewise conceivable to apply perfusion sequences without the utilization of contrast, i.e., arterial spin labeling (ASL) techniques. ASL has demonstrated as solid as difference contrast-enhanced perfusion techniques in identifying zones with decreased perfusion in acute brain ischemia and with the broader utilization of 3T MRI technology, the resolution has improved[7]. With MRI, the greatest advantage is that the ischemic core is dependably represented by the acutely infarcted area on DWI scanning. In contrast, the core is indirectly evaluated in CTP either utilizing thresholds of cerebral blood volume (CBV) or the use of decline in CBF compared to normal tissue[31].

Perfusion imaging gives a response to a major inquiry before the initiation of therapy: Is the ischemic brain parenchyma already reperfused, inadequately perfused, or totally avascular? [32].

The presence of arterial occlusion with diminished CBV suggests sudden arterial occlusion prompting ongoing infarction; the presence of arterial occlusion with increased CBV suggests gradually progressive chronic arterial occlusion, giving collaterals the opportunity to create and causing ischemia without ongoing infarction yet with the danger of future stroke; while absence of arterial occlusion with normal CBV suggests small arterial occlusion or early reperfusion of the occluded artery [33].

#### *2.3.1. Reperfusion*

The brain's reaction to acute cerebral ischemia is to activate mechanisms to lyse the clot. Spontaneous thrombolysis occurs in the first 24 hours in approximately 20% of studied cases. Perfusion MRI can play an especially important job in identifying reperfusion, since DWI and T2WI may still be either normal or abnormal in such areas [34].

Identifying proof of reperfusion is significant for two therapeutic reasons; First, if reperfusion has happened, then thrombolytic therapy should be avoided. Second, other types of treatment, such as hypertensive therapy, might also be contraindicated, since once reperfusion has occurred there appears to be an increased risk of hemorrhagic transformation [35].

### 2.4. IV-Penumbra

The penumbra is eventually the most significant in ischemic stroke and it is the focus of all the previous parameters. One working meaning of the penumbra is brain tissue that is ischemic but not yet infarcted, in other words it is characterized as hypoperfused yet potentially salvageable brain tissue and is therefore in danger for additional harm except if the flow is quickly restored. The present treatment strategies are not expected to reverse infarction that has already happened, however, detection of a PWI/DWI mismatch gives us a sensible objective for potential intervention.

No single imaging feature can define the penumbra but can be obtained through the integration of each of the three former parameters: the site of vessel occlusion (the vessels), the extent and degree of oligemia at that moment (perfusion), and the mismatch between this perfusion defect and the brain already infarcted (parenchyma)[36].

There are different possible patterns of combined PWI and DWI lesion, which have been classified as "penumbral" and "non-penumbral" patterns based on the mismatch between PWI and DWI lesion volume in hyper acute stroke patients [37]. The defined patterns are as follows; *(Pattern 1)* PWI deficit that is larger than DWI imaging deficit, a so-called PWI-DWI mismatch, this suggests the presence of at risk hypoperfused tissue (ischemic penumbra), which may be salvageable with the therapeutic restoration of blood flow (reperfusion therapy e.g., thrombolysis) up to 6 hours after stroke beginning [38]. *(Pattern 2)* PWI and DWI deficits of similar size, this implies that there is no mismatch with no surrounding tissue at risk and those patients are in need for neuroprotective therapy only as there is no tissue at risk that needs thrombolysis. *(Pattern 3)* DWI deficit only but no PWI deficit and this means that early reperfusion had happened before scanning but after the beginning of irreversible tissue damage. The patients of patterns 3 would not be expected to benefit from thrombolytic or revascularization therapies, with the additional dangers of such treatment, but may be candidates for neuroprotective strategies only. *(Pattern 4)* Neither PWI nor DWI lesions despite clinical deficit and this is associated with TIAs. The patients of this pattern were not in need for acute interventional therapy[37].

## 3. Conclusion

As patients with acute cerebral stroke might be critically ill, the initial imaging scanning for acute stroke patients should be constrained to the procurement of useful data only, considering the accessible therapeutic options at a given place at any given time. It is essential to emphasize the role of the medical community in expanding the awareness of the population to the extreme importance of seeking medical care in a specialized cerebral stroke center as fast as possible once an ictus occurs. This is crucial in rescuing as much of the brain tissue as possible once the patient arrives during the window of opportunity; as during a stroke, every minute has a great value and rapid treatment can lessen the brain damage that stroke can cause.

## Compliance with ethical standards

*Acknowledgments*

Not applicable

*Disclosure of conflict of interest*

The author declares no conflict of interest.

## References

[1] Menecie TI, Khedr SA-E-f, Hassan MAS, Zaghloul MHM. Role of susceptibility weighted imaging in acute ischemic stroke %J The Egyptian Journal of Hospital Medicine. 2019; 74(2):375-81.

[2] Van der Zijden T, Mondelaers A, Yperzeele L, Voormolen M, Parizel PM. Current concepts in imaging and endovascular treatment of acute ischemic stroke: implications for the clinician. Insights Imaging. 2019; 10(1):64-73.

[3] Hansen CK, Christensen A, Rodgers H, Havsteen I, Kruuse C, Christensen H. Does the Primary Imaging Modality-Computed Tomography or Magnetic Resonance Imaging-Influence Stroke Physicians' Certainty on Whether or Not to Give Thrombolysis to Randomized Acute Stroke Patients? J Stroke Cerebrovasc Dis. 2018; 27(4):926-35.

[4] Thomalla G, Ebinger M, Fiehler J, Fiebach JB,Endres M, Gerloff C. EU-funded treatment study: WAKE-UP: a randomized, placebo-controlled MRI-based trial of thrombolysis in wake-up stroke. Nervenarzt. 2012; 83(10):1241-51.

[5] Beauchamp NJ, Jr., Barker PB, Wang PY, vanZijl PC. Imaging of acute cerebral ischemia. Radiology. 1999; 212(2):307-24.

[6] Rowley HA. The Four Ps of Acute Stroke Imaging: Parenchyma, Pipes, Perfusion, and Penumbra. American Journal of Neuroradiology. 2001; 22(4):599.

[7] Christensen AF, Christensen H. Editorial: Imaging in Acute Stroke—New Options and State of the Art. Front Neurol. 2018; 8(736).

[8] Culebras A, Kase CS, Masdeu JC, Fox AJ, Bryan RN, Grossman CB, et al. Practice guidelines for the use of imaging in transient ischemic attacks and acute stroke. A report of the Stroke Council, American Heart Association. Stroke. 1997; 28(7):1480-97.

[9] Tan CO, Lam S, Kuppens D, Bergmans RHJ, Parameswaran BK, Forghani R, et al. Spot and Diffuse Signs: Quantitative Markers of Intracranial Hematoma Expansion at Dual-Energy CT. Radiology. 2019; 290(1):179-86.

[10] Tong E, Hou Q, Fiebach JB, Wintermark M. The role of imaging in acute ischemic stroke. Neurosurg Focus. 2014; 36(1):E3-E.

[11] Linfante I, Llinas RH, Caplan LR, Warach S. MRI features of intracerebral hemorrhage within 2 hours from symptom onset. Stroke. 1999; 30(11):2263-7.

[12] Lin DD, Filippi CG, Steever AB, Zimmerman RD. Detection of intracranial hemorrhage: comparison between gradient-echo images and b(0) images obtained from diffusion-weighted echo-planar sequences. AJNR Am J Neuroradiol. 2001; 22(7):1275-81.

[13] Toyoda K, Ida M, Fukuda K. Fluid-attenuated inversion recovery intraarterial signal: an early sign of hyperacute cerebral ischemia. AJNR Am J Neuroradiol. 2001; 22(6):1021-9.

[14] Moustafa E.M. Radwan, Aboshaera KO. Magnetic resonance angiography in evaluation of acute intracranial steno-occlusive arterial disease. The Egyptian Journal of Radiology and Nuclear Medicine. 2016; 47(3):903-8.

[15] Hwang DY, Silva GS, Furie KL, Greer DM. Comparative sensitivity of computed tomography vs. magnetic resonance imaging for detecting acute posterior fossa infarct. J Emerg Med. 2012; 42(5):559-65.

[16] Pavlina AA, Radhakrishnan R, Vagal AS. Role of Imaging in Acute Ischemic Stroke. Semin Ultrasound CT MR. 2018; 39(5):412-24.

[17] Young JY, Schaefer PW. Acute ischemic stroke imaging: a practical approach for diagnosis and triage. Int J Cardiovasc Imaging. 2016; 32(1):19-33.

[18] Geijer B, Brockstedt S, Lindgren A, Ståhlberg F, Norrving B, Holtås S. Radiological diagnosis of acute stroke. Comparison of conventional MR imaging, echo-planar diffusion-weighted imaging, and spin-echo diffusion-weighted imaging. Acta Radiol. 1999; 40(3):255-62.

[19] Flemming KD, Brown RD, Jr. Cerebral infarction and transient ischemic attacks. Efficient evaluation is essential to beneficial intervention. Postgrad Med. 2000; 107(6):55.

[20] Perkins CJ, Kahya E, Roque CT, Roche PE, Newman GC. Fluid-attenuated inversion recovery and diffusion- and perfusion-weighted MRI abnormalities in 117 consecutive patients with stroke symptoms. Stroke. 2001; 32(12):2774-81.

[21] Gomez CR, Schneck MJ, Biller J. Recent advances in the management of transient ischemic attacks. F1000Res. 2017; 6:1893-.

[22] Ricci PE, Burdette JH, Elster AD, Reboussin DM. A comparison of fast spin-echo, fluid-attenuated inversion-recovery, and diffusion-weighted MR imaging in the first 10 days after cerebral infarction. AJNR Am J Neuroradiol. 1999; 20(8):1535-42.

[23] Dankbaar JW, Bienfait HP, van den Berg C, Bennink E, Horsch AD, van Seeters T, et al. Wake-Up Stroke versus Stroke with Known Onset Time: Clinical and Multimodality CT Imaging Characteristics. Cerebrovasc Dis. 2018; 45(5-6):236-44.

[24] Thomalla G, Boutitie F, Fiebach JB, Simonsen CZ, Nighoghossian N, Pedraza S, et al. Stroke With Unknown Time of Symptom Onset: Baseline Clinical and Magnetic Resonance Imaging Data of the First Thousand Patients in WAKE-UP (Efficacy and Safety of MRI-Based Thrombolysis in Wake-Up Stroke: A Randomized, Doubleblind, Placebo-Controlled Trial). Stroke. 2017; 48(3):770-3.

[25] Swan JS, Sainfort F, Lawrence WF, Kuruchittham V, Kongnakorn T, Heisey DM. Process utility for imaging in cerebrovascular disease. Acad Radiol. 2003; 10(3):266-74.

[26] Klufas RA, Hsu L, Barnes PD, Patel MR, Schwartz RB. Dissection of the carotid and vertebral arteries: imaging with MR angiography. AJR Am J Roentgenol. 1995; 164(3):673-7.

[27] Katz DA, Marks MP, Napel SA, Bracci PM, Roberts SL. Circle of Willis: evaluation with spiral CT angiography, MR angiography, and conventional angiography. Radiology. 1995; 195(2):445-9.

[28] Spies JB. Complications of Diagnostic Arteriography. Semin intervent Radiol. 1994; 11(02):93-101.

[29] Qureshi AI, Isa A, Cinnamon J, Fountain J, Ottenlips JR, Braimah J, et al. Magnetic resonance angiography in patients with brain infarction. J Neuroimaging. 1998; 8(2):65-70.

[30] Paciaroni M, Caso V, Agnelli G. The concept of ischemic penumbra in acute stroke and therapeutic opportunities. Eur Neurol. 2009; 61(6):321-30.

[31] Heit JJ, Wintermark M. Perfusion Computed Tomography for the Evaluation of Acute Ischemic Stroke: Strengths and Pitfalls. Stroke. 2016; 47(4):1153-8.

[32] Cosnard G, Duprez T, Grandin C, Dechambre S, Mataigne F, Smith A. Diffusion- and perfusion-weighted MR imaging during the hyperacute phase of stroke. J Radiol. 2000; 81(8):858-69.

[33] Schaefer PW, Ozsunar Y, He J, Hamberg LM, Hunter GJ, Sorensen AG, et al. Assessing tissue viability with MR diffusion and perfusion imaging. AJNR Am J Neuroradiol. 2003; 24(3):436-43.

[34] Shih LC, Saver JL, Alger JR, Starkman S, Leary MC, Vinuela F, et al. Perfusion-weighted magnetic resonance imaging thresholds identifying core, irreversibly infarcted tissue. Stroke. 2003; 34(6):1425-30.

[35] Butcher K, Parsons M, Baird T, Barber A, Donnan G, Desmond P, et al. Perfusion thresholds in acute stroke thrombolysis. Stroke. 2003; 34(9):2159-64.

[36] Copen WA, Rezai Gharai L, Barak ER, Schwamm LH, Wu O, Kamalian S, et al. Existence of the diffusion-perfusion mismatch within 24 hours after onset of acute stroke: dependence on proximal arterial occlusion. Radiology. 2009; 250(3):878-86.

[37] Darby DG, Barber PA, Gerraty RP, Desmond PM, Yang Q, Parsons M, et al. Pathophysiological topography of acute ischemia by combined diffusion-weighted and perfusion MRI. Stroke. 1999; 30(10):2043-52.

[38] Uno M, Harada M, Yoneda K, Matsubara S, Satoh K, Nagahiro S. Can diffusion- and perfusion-weighted magnetic resonance imaging evaluate the efficacy of acute thrombolysis in patients with internal carotid artery or middle cerebral artery occlusion? Neurosurgery. 2002; 50(1):28-35.