"COLLABORATIVE USE OF SUSCEPTIBILITY WEIGHTED IMAGING WITH DIFFUSION WEIGHTED IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY IN THE ASSESSMENT OF ACUTE ARTERIAL STROKE USING 3T MRI– A ONE YEAR HOSPITAL BASED CROSS -SECTIONAL STUDY"

BY

REG. NO. BS01201015

Dissertation

Submitted to
KAHER, Belagavi, Karnataka,
In partial fulfilment of the requirements for the degree of

M.D.

In

RADIO-DIAGNOSIS

J. N. MEDICAL COLLEGE,
BELAGAVI -590010. KARNATAKA

DEC – 2024/JAN – 2025

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,

BELAGAVI, KARNATAKA

Endorsement by the HOD/Principal/ Head of the Institution

This is to certify that the dissertation entitled "– **COLLABORATIVE USE OF SUSCEPTIBILITY WEIGHTED IMAGING WITH DIFFUSION WEIGHTED IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY IN THE ASSESMENT OF ACUTE ARTERIAL STROKE USING 3T MRI - A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY"** is a bonafide research work done by REG NO. **BS0121015.**

Dr. SANTOSH D. PATIL
M.D.(Radio-Diagnosis)
Professor & HOD
Dept. of Radio-diagnasis
J.N. Medical College, BELAGAVI-10.
KMC Reg No. 58456

Dr SANTOSH D. PATIL
M.D. RADIO-DIAGNOSIS

Professor and Head,
Department of Radio Diagnosis,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date: 26/06/24
Place: Belagavi

Dr N. S. MAHANTASHETTI
M. D. PEDIATRICS

PRINCIPAL
J.N. Medical College
BELAGAVI- 590 010

KAHER
J. N. MEDICAL COLLEGE
BELAGAVI

Principal,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date : 26/06/24
Place : Belagavi

UNDERTAKING

I, Reg. No. BS0121015 hereby declare that the information and the data mentioned in my dissertation entitled **"COLLABORATIVE USE OF SUSCEPTIBILITY WEIGHTED IMAGING WITH DIFFUSION WEIGHTED IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY IN THE ASSESSMENT OF ACUTE ARTERIAL STROKE USING 3T MRI– A ONE YEAR HOSPITAL BASED CROSS -SECTIONAL STUDY"** belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date: 25/06/2024

Place: Belagavi

(REG. NO. BS0121015)

PLAGIARISM CERTIFICATE

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

JAWAHARLAL NEHRU MEDICAL COLLEGE

(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)

(Recognized by National Medical Commission, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) **Placed in Category 'A' by MoE (GoI)**

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 – 2471350 0831 – 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 25-06-2024

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: **"COLLABORATIVE USE OF SUSCEPTIBILITY WEIGHTED IMAGING WITH DIFFUSION WEIGHTED IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY IN THE ASSESSMENT OF ACUTE ARTERIAL STROKE USING 3T MRI- A ONE YEAR HOSPITAL BASED CROSS -SECTIONAL STUDY"** has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **06%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER J. N. MEDICAL COLLEGE BELAGAVI

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

To,
Reg. No. BS0121015
Postgraduate Student,
2021-22 Batch,
Department of Radio-Diagnosis
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE CERTIFICATE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 45 **Date: 27/09/2022**

To
REG. NO. BS01201015
PG Student in Radiodiagnosis,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"COLLABORATIVE USE OF SUSCEPTIBILITY WEIGHTED IMAGING WITH DIFFUSION WEIGHTED IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY IN THE ASSESSMENT OF ACUTE ARTERIAL STROKE USING 3T MRI- A ONE YEAR HOSPITAL BASED CROSS- SECTIONAL STUDY."** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVIATIONS

SWI	Susceptibility weighted imaging
DWI	Diffusion weighted imaging
ADC	Apparent diffusion coefficient
TOFMRA	Time of flight – magnetic resonance angiography
MRI	Magnetic resonance imaging
FLAIR	Fluid attenuated inversion recovery
SVS	Susceptibility vessel sign
AIS	Acute ischemic stroke
ATP	Adenosine triphosphate
BBB	Blood brain barrier
GRE	Gradient-echo
TE	Echo time
MINIP	Minimum intensity projection
RT	Repetition times
SVS	Susceptibility vessel sign
CADASIL	Cerebral autosomal dominant arteriopathy, subacute infarcts, and leucoencephalopathy
CAA	Cerebral amyloid angiopathy
CMBS	Cerebral microbleeds
ICH	Intracranial hemorrhage
MHV	Multiple Hypointense vessels
PVS	Prominent vessel sign

CVSs	Cortical vessel signs
DMV	Deep medullary veins
HVS	Hyperintense vascular sign
DPM	Diffusion-perfusion mismatch
DHV	Distal hyperintense vessels
PLPCA	Prominent laterality of posterior cerebral arteries
PLACA	Prominent laterality of anterior cerebral arteries

LIST OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

ABSTRACT

INTRODUCTION

Acute stroke is characterized by sudden onset of localized neurologic impairment, often with progressive worsening of symptoms. Instead of relying on stroke duration, imaging modalities like MRI can be used to measure tissue viability and estimate the time of stroke onset, known as the "tissue clock." This approach helps determine intervention timeframes. Thrombolytic treatment must be delivered within six hours of the onset of stroke to dissolve clots and restore blood circulation. Hence, for patients with an unknown stroke onset time, the most effective way to estimate it is using Diffusion weighted imaging (DWI) - Fluid attenuated inversion recovery (FLAIR) mismatch. The SWI-DWI mismatch is a significant indicator in acute ischemic stroke, indicating the presence of an ischemic penumbra. Patients with this mismatch have a higher rate of favourable outcomes after thrombolytic therapy compared to those without the mismatch, suggesting it can be used as an alternative method to select appropriate candidates for thrombolysis. SWI can detect cerebral microbleeds, which may increase the risk of bleeding with thrombolytic or antithrombotic therapy. Patients with a small number of microbleeds can be safely treated, while multiple microbleeds may indicate a higher risk. SWI can also identify the susceptibility vessel sign, which helps locate the precise position of the intra-arterial thrombus, complementing the information provided by time-of-flight MRA on arterial occlusion.

OBJECTIVES

- To study the synergistic effect of SWI with DWI and MRA in the assessment of Acute Ischemic Stroke
- To evaluate if SWI and DWI are more efficient than conventional MRI sequences for evaluating stroke and selecting patients for thrombolytic treatment.

METHODOLOGY

120 patients presenting within 12 hours of symptom onset for acute ischemic stroke were evaluated clinically and then underwent an MRI brain using a 3 Tesla MRI scanner (Magnetom Avanto TIM, 18 channel; Siemens, Erlangen, Germany) for assessing the collaborative use of SWI, DWI, and MRA for comprehensive evaluation of acute ischemic stroke, allowing for improved patient selection and management, including guiding thrombolytic therapy.

RESULTS

- DWI positive FLAIR negative (DWI-FLAIR mismatch) was seen in patients presenting with < 6 hours of symptom onset hence DWI-FLAIR mismatch is the most effective method for patients with unknown onset time.
- The susceptibility vessel sign seen on SWI revealed a highly significant correlation with TOF MRA abnormality on TOF-MR Angiography.
- Microhemorrhagic foci were present in 38 (31.67%) cases out of 120 patients, Significant correlation was observed between microhaemorrhagic foci and time since onset of symptoms more than 6 hours (p-value = 0.0018) and also in infarcts with a volume cut-off of 27 ml on the DWI sequence.
- A SWI-DWI mismatch was observed in 26 (21.6%) cases out of 120 patients, and 16 of the 26 patients received thrombolytic therapy, all of these patients showed a three-grade improvement in their muscle power thus indicating SWI-DWI mismatch provides an alternative method for selecting patients who are appropriate candidates for thrombolytic therapy.

CONCLUSION

- Patients with ischemic symptoms present with a diffusion-FLAIR mismatch within 6 hours of symptom onset. The difference in tissue volume implicated on DWI and FLAIR can be used to estimate the period since ischemia's onset.
- SWI's susceptibility vessel sign had a sensitivity of 89.74%, specificity of 85.71%, PPV of 92.11% NPV of 81.82% for identifying occlusion of main arteries away from the skull base compared to TOF-MRA.
- SWI is more effective than conventional MRI sequences in detecting micro haemorrhages and early hemorrhagic transformation.
- Standard stroke imaging protocols should include both DWI and SWI sequences

INTRODUCTION

Acute stroke is characterized by a quick onset of a localized neurologic impairment, while some patients experience progressive worsening of symptoms. Ischemic brain injury is assumed to be caused by a series of events, beginning with energy depletion and resulting with cell death. Intermediate causes include an overabundance of extracellular excitatory amino acids, free radical production and inflammation [1].

Instead of relying on stroke duration as a surrogate for tissue pathology, it may be more effective to measure tissue viability regardless of imaging time from stroke onset, hence the concept of using MR imaging to build a "tissue clock" rather than a "wall clock" to determine intervention timeframes is closely related to the idea of estimating the time of stroke start[2].

The key to providing thrombolytic therapy to patients who experienced unwitnessed strokes is to distinguish between the period when the patient was last known well and the time when the patient was discovered with symptoms. For strokes with unwitnessed onsets, the variation in timings is unknown (by definition) and could represent the difference between being eligible or ineligible for therapeutic intervention [2].

In order to dissolve clot and restore blood supply, thrombolytic treatment needs to be provided within 3 hours of onset of stroke as part of the recanalization efforts [1].

Hence, for patients with an unknown stroke onset time, the most effective way to estimate it is using Diffusion weighted imaging (DWI) - Fluid attenuated inversion recovery (FLAIR) mismatch. Many individuals with a DWI-FLAIR mismatch may benefit from reperfusion treatments. If these findings are reproduced and verified in future investigations, an MRI signature could be used to select wake-up stroke patients for acute reperfusion trials[3].

Patients who had a Susceptibility weighted imaging- Diffusion weighted imaging (SWI-DWI) mismatch had a higher rate of positive outcomes than those who did not, As a result, the SWI-DWI mismatch may provide an alternative method for selecting patients who are appropriate candidates for thrombolytic therapy. [4]

SWI helps in detection of previous microbleeds (defined as hypointense patches on T2*-weighted gradient echo MR) which may increase the risk of bleeding following thrombolysis or other antithrombotic therapy. Stroke patients with a small number of microbleeds on pre-treatment MRI can be safely treated with thrombolytic therapy. Multiple microbleeds, on the other hand, could indicate a diffuse hemorrhagic vasculopathy [5].

Susceptibility-weighted imaging (SWI) allows the visualization of thrombotic material in acute ischemic stroke (AIS) hence shows significant associations between thrombus location on SWI and time of flight – MR angiography (TOF-MRA) [6]

SWI is more effective than CT scans in detecting spontaneous hemorrhagic transformation of ischemic stroke and determining the need for thrombolytic therapy, potentially lowering the incidence of symptomatic haemorrhage [5]

In addition, SWI can identify the susceptibility vessel sign (SVS) in several main intracranial artery areas, which helps identify intra-arterial thrombus in AIS cases and pinpoint its precise location. However, since occluded or restricted arteries are regarded to be the primary cause of AIS development, time of flight (TOF)-MRA is a routinely used technique in the evaluation of patients suffering from acute ischemic stroke (AIS). In light of these, the purpose of this study was to evaluate the synergistic impact of MRA, DWI, and SWI in the evaluation of AIS.

AIMS & OBJECTIVES

- To study the synergistic effect of SWI with DWI and MRA in the assessment of Acute Ischemic Stroke
- To evaluate if SWI and DWI are more efficient than conventional MRI sequences for evaluating stroke and selecting patients for thrombolytic treatment.

REVIEW OF LITERATURE

STROKE PATHOPHYSIOLOGY:

Stroke is the major cause of death and morbidity globally. Acute ischemia accounts for over 80% of all strokes and is a leading cause of death and morbidity[7].

An embolic or thrombotic obstruction of a cerebral artery can cause a significant reduction in blood flow, resulting in focal cerebral ischemia. The principal mechanisms, include complex cellular and molecular cascades. Damage mechanisms include excitotoxicity, peri-infarct depolarization, generation of reactive oxygen and nitrogen species and tissue acidosis. Later on, Inflammation and apoptosis can lead to brain tissue damage. Endogenous mechanisms of protection, regeneration, vascular remodelling and repair can mitigate brain injury, but post-stroke inflammation has both detrimental and restorative effects [8].

Cerebral blood flow below 10 mL/100 g of brain tissue depletes oxygen and glucose, resulting in a significant drop in adenosine triphosphate (ATP) at the cellular level. The sodium-potassium pump fails as ATP levels drop. This failure induces passive diffusion of Na^+ ions and fluid inside cells, resulting in cytotoxic edema. Loss of membrane potential leads to activation of voltage gated calcium channel resulting in high intracellular Ca^{2+} levels which activates enzymes such as protein kinase C, phospholipase A2, phospholipase C, cyclooxygenase, calcium dependent nitric oxide synthase, calpain, and endonucleases, resulting in irreversible mitochondrial damage, inflammation, necrosis and apoptosis [9].

The combination of hypoxic damage to the vascular endothelium, toxic damage of inflammatory molecules and free radicals and destruction of the basal lamina by matrix metalloproteinase (MMP) damages the blood brain barrier (BBB). The breakdown of the BBB causes vasogenic edema, inflammation and hemorrhagic change[9].

DIFFUSION WEIGHTED IMAGING:

Over the last two decades, improvements in MR imaging (magnetic resonance imaging) neuroimaging have heightened dramatically due to the development of stronger magnetic systems and biomedical technology, enabling in early stroke detection and treatment.

Conventional brain MRI studies take time and are not very specific at demonstrating cytotoxic or intracellular edema which is seen in the acute or less than 24 hour phase of stroke. Conventional sequences (T2, FLAIR) can demonstrate vasogenic edema which appears in the sub-acute phase. DWI can exhibit cytotoxic edema as early as ten minutes and is more conspicuous than conventional sequences[10].

Water molecules within biological tissues move randomly (Brownian motion).

When there is an acute infarct or irreversible ischemia, the intracellular water accumulation and disturbance of membrane ionic homeostasis cause cytotoxic oedema or swelling of neurons and glial cells, which reduces the diffusion of water molecules. This results in an increase in signal intensity, which in turn causes a decrease in ADC. ADC is only "apparent" because the true measurement cannot be acquired by DWI because of the presence of unmeasured variables, such as tissue temperature and the diffusing molecule's actual route, that affect the rate of diffusion. Three orthogonal planes are used to take images consecutively along the diffusion sensitization axis; the average of these planes produces an image with the least amount of anisotropy-related hyperintensity. In contrast to normal brain tissue, infarcted tissue appears brighter as a result. This significantly affects the ability to distinguish between areas of acute ischemia and chronic ischemia, allowing for the stratification of treatment options. In humans, restricted diffusion with a raised DWI signal and a decreased ADC value was observed as early as 30 minutes after the onset of ischemia. ADC decreases for 3-5 days, reaching a low value of roughly 50% between 24 and 48 hours after the onset of symptoms, and then returns to baseline at roughly 1-4 weeks—a process known as pseudonormalization. A slightly hyperintense DWI signal with a normalised ADC value will be visible at this point. After that, the ADC rises to a value that is higher than usual and is linked to a fluctuating DWI signal for up to months. This is probably due to an increase in the amount of extracellular water. It is noteworthy that the DWI signal can exhibit hyperintense behaviour for extended periods and is not a reliable indicator of infarct age alone. Conversely, a lower ADC value nearly invariably indicates an acute infarction. As a result, we must interpret DWI and ADC maps jointly with caution [(7)].

Role of diffusion weighted imaging compared to conventional MRI sequences for acute stroke:

Marten G. Lansberg et al. **[(11)] (2000**) studied the efficacy of combining diffusion-weighted imaging (DWI) with a conventional magnetic resonance imaging (MRI) protocol for identifying acute stroke. They discovered that conventional MRI properly detected at least one acute lesion in 71% to 80% of individuals with acute stroke, but this figure rose to 94% when DWI was included. Conventional MRI had modest sensitivity and specificity (50%-60% and 49%-69%, respectively), but DWI had higher interrater reliability ($\kappa = 0.8$) and observer confidence in recognizing acute lesions. Adding DWI greatly enhanced the accuracy of detecting acute stroke, especially in terms of lesion conspicuity and observer confidence[11].

Everdingen KJV et al [12] (1998) examined the predictive usefulness of the ADC and the amount of ischemic lesions on DWI images. 98% of the ischemic lesions were found with DWI, and 91% were found with fluid-attenuated inversion recovery. Clinical outcome evaluations were substantially linked with lesion size on early DWI images. With a 75% sensitivity and 100% specificity, a lesion volume greater than 22 mL on DWI predicted a favourable outcome in individuals who were having their first stroke. Compared to areas of the brain with normal appearance, mean ADC of ischemia lesions was 29% lower. There was a strong correlation between the ADC ratio and clinical outcome. They stated that for the purpose of identifying early ischemia lesions in stroke cases, DWI is a more effective imaging technique than traditional MRI. Potential indicators for forecasting clinical outcome in AIS cases include lesion size as determined by DWI scans and to a lesser extent, ADC readings.

Association between DWI lesion volume and patient outcomes :

Albert J. Yoo et all [13] (2009) retrospectively studied 34 consecutive patients with anterior circulation stroke who underwent pretreatment diffusion-weighted imaging and subsequent intra-arterial therapy. Patients were stratified based on initial infarct volume, recanalization status, and time to recanalization.

Among patients with initial infarcts >70 cc , all had poor outcomes despite a 50% recanalization rate with mean infarct growth of 114 cc. Patients with initial infarct volumes <70 cc who recanalized early had the best clinical outcomes (P<0.008) with a 64% rate of modified Rankin Scale score <or=2 and the least infarct growth (P<0.03) with mean infarct growth of 18 cc.

Liang Jiang et al. [14] (2020) studied the relationship between diffusion-weighted imaging (DWI) volume and fluid-attenuated inversion recovery vascular hyperintensities (FVH) and evaluated the value of FVH-DWI mismatch for determining the functional consequences and revascularization in cases of stroke with occlusion of large vessels after endovascular thrombectomy(EVT) . The study found that patients with good functional outcomes had smaller DWI volumes on admission, follow-up, and lower DWI volume growth, as well as a higher FVH-DWI mismatch ratio compared to those with poor outcomes. ROC analysis showed that DWI volume on admission, follow-up, and DWI volume growth could predict functional outcome with varying degrees of sensitivity and specificity(65% and 96.97%, 80% and 87.88% ,70% and 87.88% respectively). The results suggest that DWI volume and DWI volume growth can provide valuable prognostic information for acute stroke patients undergoing thrombectomy(14).

Diffusion weighted imaging - fluid attenuated inversion recovery (DWI - FLAIR) mismatch :

Studies have shown that a mismatch in the appearance of acute ischemic lesions between diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) , known as the DWI-FLAIR mismatch - could be utilized to determine the time since onset in patients with AIS for thrombolytic therapy.

FIGURE 1

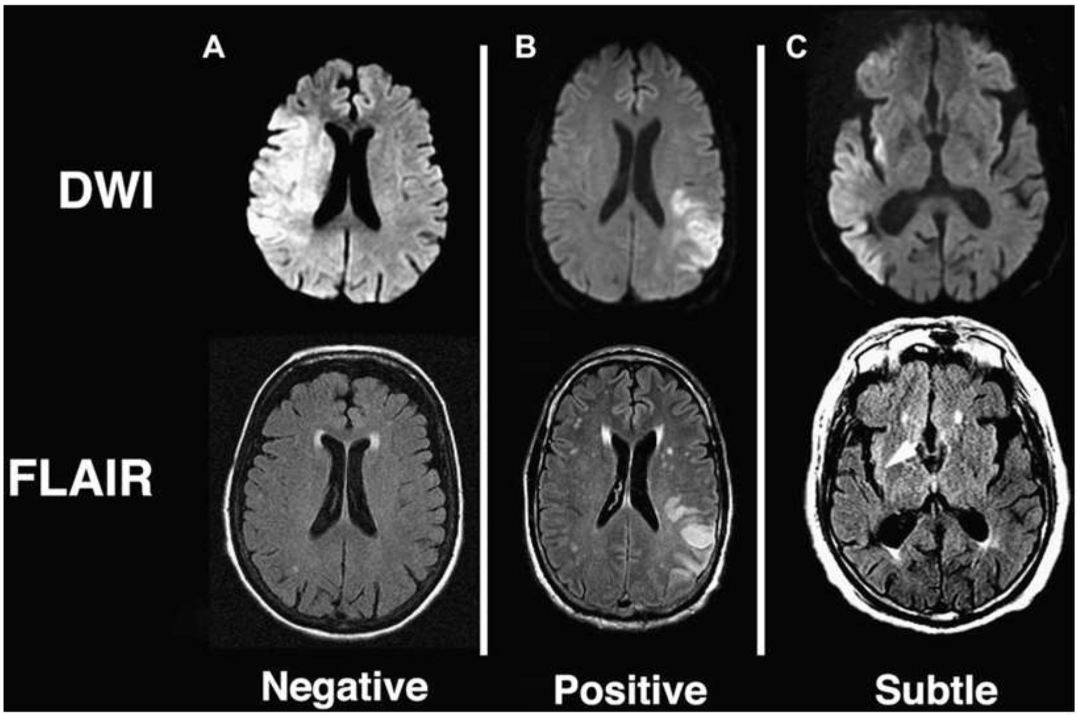


Image courtesy : ona wu et al [2](2011)

Image a : Diffusion positive , FLAIR – negative , patient imaged 1.5 hours from symptom onset
Image b : Diffusion positive , FLAIR – positive , patient imaged 6.2 hours from symptom onset ,
Image c : Diffusion positive , FLAIR – subtle hyperintensity , patient imaged 2.5 hours from symptom onset[2]

Branko et al. [3] (2012) compared 19 patients with an unknown time of start (wake-up stroke) to 22 patients with an onset time of 6 hours or less (group A) and 19 patients with an onset time of 6 to 12 hours (group B). The study discovered that the DWI-FLAIR mismatch was evident in the unknown group and group A, but was rare in group B (about 10.5%). This implies that a considerable proportion of nocturnal stroke patients with an unclear time of stroke onset had a DWI-FLAIR mismatch, indicating a recent onset stroke [3].

FIGURE 2

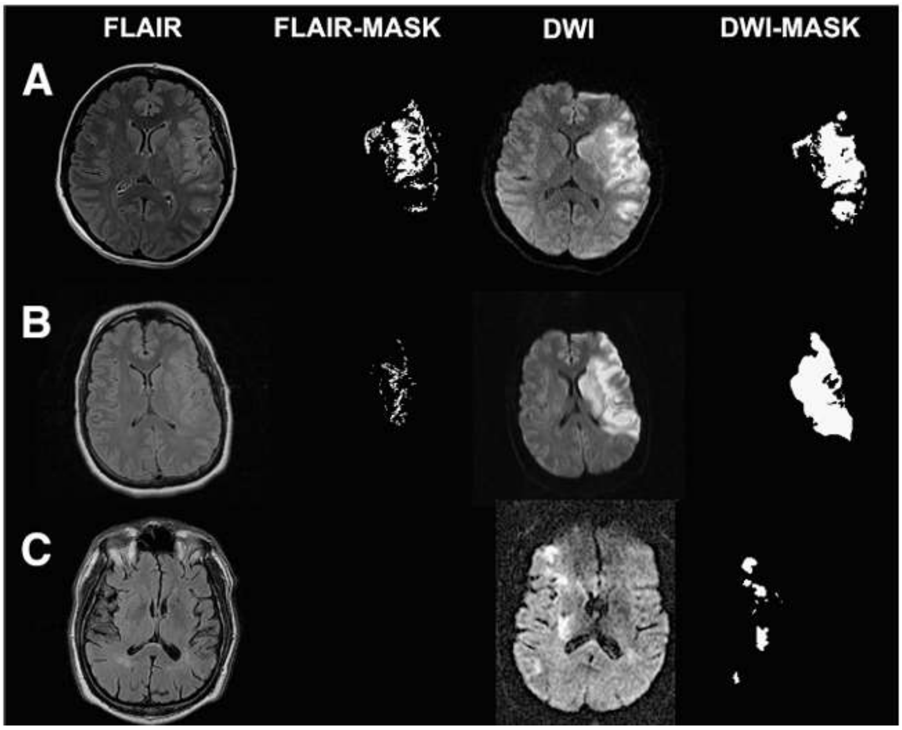


Image courtesy : Branko N. Huisa et all [3]

The patient A underwent imaging at 6 hours and 50 minutes after acute stroke onset. All raters noticed an aberrant fluid attenuated inversion recovery (FLAIR) signal. The FLAIR/DWI volume ratio was 8.2%. Row B shows a patient who had imaging 5 hours after symptom start. Two physicians graded FLAIR as normal, whereas two others judged it abnormal. The calculated FLAIR/DWI volume ratio was 2.2%. The FLAIR imaging in row C was deemed normal by all physicians. Neither automated nor manual thresholds produced a normal signal. [3]

Gotz Thomalla et al. [15] (2012) investigated the efficacy of employing DWI-FLAIR mismatch in choosing patients for thrombolysis treatment. They discovered that patients with acute ischemic lesions detectable on DWI but not on FLAIR imaging were more likely to fall within the 3-hour window for safe and efficient thrombolysis. The study revealed that the DWI-FLAIR mismatch can be used to identify individuals who are extremely likely to be within the three-hour time frame, with high specificity and positive predictive value

Junya Aoki et al. [16] (2010) investigated a method to determine the onset time of acute ischemic stroke (AIS) within 24 hours of onset. The study enrolled AIS patients with clearly defined onset times and used diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) to analyze 389 MRI studies. The results showed that when DWI was positive and FLAIR was negative (DWI+/FLAIR-), the onset time was estimated to be within 3 hours with high sensitivity and specificity, and within 4.5 and 6 hours with lower but still significant sensitivity and specificity. This method can help identify patients who are eligible for thrombolysis treatment within the 6-hour window

SUSCEPTIBILITY-WEIGHTED IMAGING (SWI)

SWI has progressed from basic 2D T2*-weighted sequences to 3D sequences with superior spatial resolution and susceptibility contrast (17).

Susceptibility-weighted imaging (SWI) is a advanced MRI technique that improves contrast based on differences in magnetic susceptibility between tissues. It is especially useful in neuroimaging. SWI's physics involves exploiting variations in magnetic susceptibility, which is the degree to which a material becomes magnetized in an external magnetic field. These differences, primarily caused by deoxygenated blood, iron, and calcium, cause local magnetic field inhomogeneities, resulting in phase shifts in the MRI signal (18)(19).

SWI captures these phase shifts by using a high-resolution 3D gradient-echo (GRE) sequence with a long echo time (TE), which allows the susceptibility effects to develop fully. The technical process includes phase unwrapping to correct phase wrapping artifacts, high-pass filtering to remove background field variations, and creating a phase mask from the filtered phase images. This mask is then multiplied with the magnitude images to enhance the susceptibility contrast. The resulting images often undergo minimum intensity projection (minIP) in order to better visualize minute features such as veins and microbleeds. Clinically, SWI is invaluable for detecting microbleeds, small vessel disease, venous malformations, and neurodegenerative changes, as well as assessing trauma and differentiating hemorrhagic from non-hemorrhagic tumors [18][19]

Role of SWI in acute ischemic stroke:

When evaluating patients who have acute stroke symptoms, neuroimaging is essential. Brain imaging tests are the only ways to reliably distinguish between haemorrhage and ischemia, despite the fact that clinical examinations and patient symptoms may point to a diagnosis. This distinction is crucial since it influences treatment choices. It has been discovered that SWI is a helpful imaging sequence for imaging stroke patients.

Two general categories can be used to classify the uses of SWI in stroke patients:

1. SWI in the evaluation of acute stroke
2. SWI in work-up of stroke patients. The many clinical uses of SWI in stroke are reviewed in brief in the ensuing sections.

Role of SWI in the assessment of intra-arterial thrombus and its correlation with TOF- MR angiography in acute ischemic stroke:

Thrombus is paramagnetic due to to the presence of a larger quantity of deoxyhemoglobin, which creates blooming artifact, and hence intra arterial thrombus can be identified on SWI[20] . When the diameter of a hypointense artery on susceptibility-weighted imaging (SWI) pictures is greater than the diameter of the contralateral artery, the susceptibility sign is considered positive[21]

MR Angiography also acts as a helpful tool for revealing the location and extent of occlusive thrombus . Rapid treatment of thrombus in acute stroke patients is extremely important. Therefore, timely and efficient imaging improves the likelihood of good clinical outcomes in individuals suffering from an acute stroke [22].

Magnetic resonance angiography (MRA) refers to a variety of imaging techniques based on magnetic resonance imaging (MRI) that were created to investigate the arterial and venous systems. An MRA has several advantages over standard angiography, including the fact that it is noninvasive, does not expose patients to ionizing radiation, allows for non-contrast examinations, and can produce high-resolution volumetric images [23].

The TOF approach is based on the "inflow effect." When protons are subjected to RF pulses with very short repetition times (RT), the signal generated by them is zero due to the continual saturation of their longitudinal magnetization. Blood protons entering the acquisition volume are not subjected to this saturation, therefore they provide a high signal, indicating vascular enhancement due to the "inflow effect." The stair-step artifact, in which the image seems pixelated for obliquely oriented vessels, is produced by the comparatively thick slices in 2D (1mm to 3mm) compared to the in-plane spatial resolution of 0.5mm to 1.0mm. They can be reduced by overlapping slices, however this requires a significant increase in scan time[23][24].

This technique is the first diagnostic option for studying cerebral circulation and the second option for studying epi-aortic arteries when a contrast medium investigation is not possible. It enables for the investigation of vessels with high flow rates, such as arteries, while ignoring much of the data from the veins. Images are prone to motion distortions and proton dephasing caused by very slow flow velocity, as seen in severe stenosis or tortuosity in vessels, with the danger of overestimating the degree of stenosis. TOF can be accomplished using both 2D and 3D acquisitions. 2D acquisitions are particularly sensitive to proton dephasing artifacts caused by the very slow flows at the level of vascular stenosis. The adoption of 3D acquisitions resulted in a significant improvement in diagnostic accuracy[(23)(24).]

In a study by **Radbruch et al.** **[(25) (2013)]**, 87 out of 94 patients showed a clear susceptibility vessel sign (SVS) on SWI. The SVS correlated with occlusion or stenosis on time-of-flight (TOF) angiography in 72 patients. Fifteen patients exclusively displayed SVS on SWI, with 14 in the M2/M3 segments and 1 in the M1 segment, while no patient showed occlusion or stenosis exclusively on TOF-angiography. The sensitivity for detecting embolic occlusion in major vessel segments (M1, M1/M2, ACA, and PCA) was similar between SWI (97%) and TOF-angiography (96%). However, SWI had significantly higher sensitivity (84%) compared to TOF-angiography (39%, $p < 0.00012$) for detecting embolic occlusion in the M2/M3 segments. Concluding that SWI and TOF-angiography have similar sensitivity for detecting central thrombi, but SWI is more effective for detecting peripheral thrombi in smaller arterial segments.

In a study conducted by **Christian W et al** **[(6) (2014)]** 88 patients with acute ischemic stroke due to middle cerebral artery (MCA) occlusions undergoing endovascular recanalization were evaluated. The study compared thrombus visibility and location on susceptibility weighted imaging (SWI) with those on time-of-flight magnetic resonance angiography (TOF-MRA), gradient echo magnetic resonance angiography (GE-MRA), and digital subtraction angiography (DSA). The results showed that 95.5% of patients had an MCA thrombus visible on SWI. Strong correlations were observed between thrombus location on SWI and those on TOF-

MRA, GE-MRA, and DSA. The study concluded that SWI was superior to the other techniques in assessing the distal end of the thrombus.

Ehab Ali Abdelgawad et al [(26) (2021)] stated that SWI is a valuable tool for detecting peripheral thrombi in patients with acute ischemic stroke, and thus the study proposed incorporating SWI into routine acute stroke MRI protocols. SWI and MRA may complement effectively to detect occluded vessels visually. In a study of 97 patients with acute ischemic stroke, 57.6% of the thrombi were located in the M1 segment of the MCA. SWI detected intra-arterial thrombus in 122 patients compared to 97 patients detected by magnetic resonance angiography (MRA), with a statistically significant difference (P=0.0002). All patients showed a positive susceptibility sign. While 88.8% of patients with positive thrombus in SWI had solitary thrombi, 11.2% had multiple thrombi. In contrast, MRA failed to detect distant thrombi. The study also found that 81% of patients with abnormally prominent vessel signs (APVS) showed parenchymal changes in these areas, and all patients with APVS had arterial occlusion. SWI plays an important role in the detection of peripheral thrombi in patients with acute ischemic stroke.

FIGURE 3

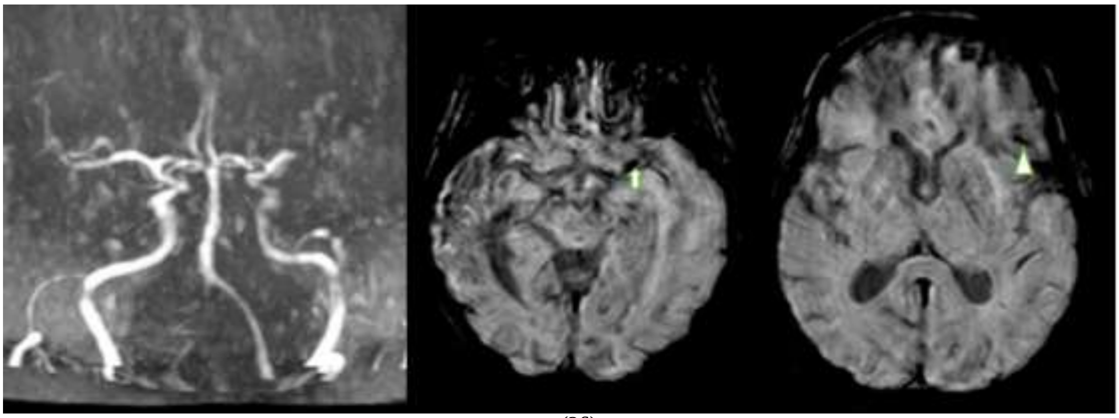


Image courtesy : Ehab ali abdelgawad et al [(26)]
A 61-year-old woman. MRA (a) indicates an abrupt non visualization of the left MCA at the M1 segment.
SWI (b, c) shows two susceptibility vessel signs along the M1 and M3 segments of the left MCA, indicating two thrombi.

Halefoglu AM et al [27] (2019) evaluated the diagnostic performance of TOF MRA and SWI in identifying arterial thrombotic occlusion in individuals suffering from AIS. SVS on SWI in the major intracranial artery areas and a corresponding blockage or severe stenosis of the arteries on TOF MRA were found to be concordant in 50 out of 63 cases. TOF MRA in five individuals did not show evidence of stenosis or occlusion in the relevant arterial area, despite the availability of the SVS. Conversely, three cases whose SVS was negative on TOF MRA revealed stenosis or occlusion. Ultimately, neither SVS on SWI nor TOF MRA were visible in the five acute infarct cases who remained. In AIS, intra-arterial thrombus was detected with marginally greater sensitivity using SVS on SWI compared to TOF MRA.

Dayanand Lingegowda et al [28](2011)compared the accuracy of susceptibility sign with contrast enhanced MRA (gold standard) and found that the susceptibility sign had a sensitivity and specificity for detecting MCA occlusion were 77% and 100%, respectively, based on 13 cases. For anterior cerebral artery (ACA) occlusion, the sensitivity was 50% (1 of 2), while for posterior cerebral artery (PCA) and basilar artery, it was 66.6% (2 of 3) and 75% (3 of 4), respectively. The susceptibility sign detected all vertebral artery occlusions (6 of 6). Overall, the sensitivity and specificity for all acute major intracranial arterial occlusions were 82% and 100%, respectively.

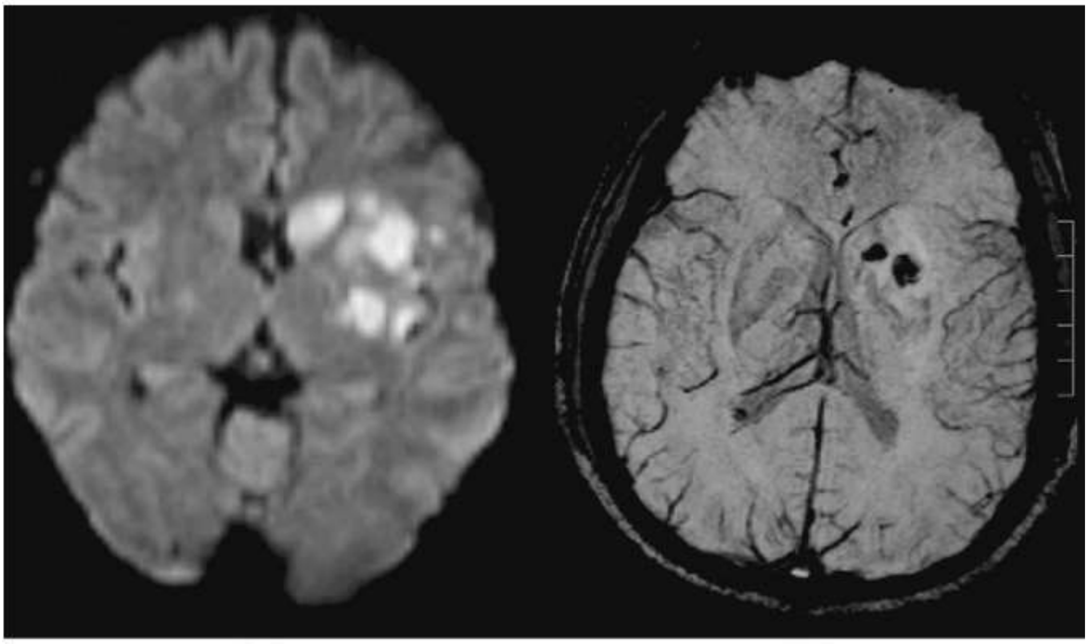
Min-Gyu Park et al [29] (2016) conducted a study to Investigate the utility of SWI in the detection of an intravascular thrombus in acute cardioembolic stroke by comparing the SVS on SWI to the vessel status on time-of-flight magnetic resonance angiography. The study enrolled 122 patients and found that the susceptibility vessel sign (SVS) was observed in 75.4% of them. Magnetic resonance angiography (MRA) showed occlusion in 57% of the patients. The SVS was identified in all 70 patients with occlusion on

MRA, indicating a high sensitivity for detecting occlusion. Additionally, the SVS was observed in 22 (42.3%) of 52 patients without occlusion on MRA, primarily in the post-bifurcation segments of the middle cerebral artery. This suggests that the SVS can be a useful marker for detecting thrombotic occlusion even when MRA does not show any occlusion and therby providing significant information about the thrombus location, the existence of a single or numerous thrombi, particularly in the distal intracranial arteries, and the thrombus burden in acute cardioembolic stroke.

Role of SWI in the detection of cerebral microbleeds and its correlation with hemorrhagic transformation

Cerebral microbleeds appear as hypointense spots of less than 5 mm in SWI sequences, caused by the increased T2* effect of paramagnetic compounds such hemosiderin or deoxyhaemoglobin. SWI is more effective than T2 or T2*W sequences for detecting tiny microbleeds . Microbleeds can occur in stroke-causing conditions like chronic hypertension, cerebral autosomal dominant arteriopathy, subacute infarcts, and leucoencephalopathy (CADASIL) , cerebral amyloid angiopathy (CAA), vasculitis, infective endocarditis, and Binswangers disease[(30).]

FIGURE 4



(a) (b)

Image courtesy : k santosh et al (30)

(A) Diffusion weighted image after thrombolysis reveals left basal ganglia restriction indicative of an acute infarct. (b) An axial SWI reveals hemorrhage within the infarct. The hemorrhagic change following thrombolysis (30)

Abd Elaziz Elnekeidy et al (31)(2013) concluded that susceptibility-weighted imaging is an important approach for accurately detecting early hemorrhagic changes within acute infarctions as well as detecting old microbleeds, alerting the treating physician to the severe side effects of anticoagulant therapy. Thus, SWI should be a routine sequence in the protocol for stroke imaging. Among the 50 patients studied, 92% had infarctions, 6% had hematomas, and 2% had transverse sinus thrombosis without infarctions or hematomas. CT detected hemorrhagic transformation in 4.3% of infarctions, while SWI detected it in 21.7%. Both CT and SWI detected hematomas in 6% of patients. SWI detected old micro-bleeds in 16% of patients, which included those with non-hemorrhagic infarctions, hematomas, and early hemorrhagic transformation.

Additionally, SWI detected sinus thrombosis in 4% of patients, with one patient having hemorrhagic infarction and the other having no visible brain insults on CT. Management was tailored to each group based on the presence or absence of hemorrhagic transformation and associated old microbleed

In a study by **Ah ling Cheng et al[32] (2013)** , 1146 cerebral microbleeds (CMBs) were identified on gradient echo (GRE) images and 1432 CMBs on susceptibility-weighted imaging (SWI) in 9 cerebral amyloid angiopathy cases. In 22 healthy control subjects, 6/22 had ≥1 CMBs on GRE (total 9 CMBs) and 5/22 on SWI (total 19 CMBs). The inter-rater reliability for CMB counts was higher for SWI compared to GRE (in cerebral amyloid angiopathy cases. In controls, agreement on the presence or absence of CMBs was moderate to good on both SWI and GRE. The study found that increased detection and reliability on SWI were related to both increased contrast and higher resolution, allowing better discrimination of CMBs from the background and better anatomic differentiation from pial vessels. The conclusion was that SWI confers greater reliability as well as greater sensitivity for CMB detection compared with GRE, and should be the preferred sequence for quantifying CMB counts.

Chelsea Kidwell et al. [33] (2002) looked into the relationship between microbleeds on pre treatment MRI and hemorrhagic transformation in individuals undergoing thrombolytic therapy for acute ischemic stroke. The study discovered that one out of every five patients with prior microbleeds experienced significant symptomatic bleeding, compared to four out of every 36 patients without microbleeds. Notably, just one patient in the entire group developed hemorrhagic transformation outside of the acute ischemic field, and it occurred immediately at the site of a previous microbleed, which was contralateral to the acute ischemic event. The study reveals that the presence of microbleeds on pretreatment MRI may indicate an elevated risk for hemorrhagic transformation in individuals following thrombolytic therapy

A study conducted by **Yannie O Y Soo et all [34] (2008)** examined the relationship between cerebral microbleeds (MB) and the risk of subsequent intracranial hemorrhage (ICH), recurrent cerebral infarct (CI), and mortality in patients with acute ischemic stroke. The results showed that the risk of ICH increased significantly with the quantity of MB, with rates of 0.6%, 1.9%, 4.6%, and 7.6% for no MB, 1 MB, 2-4 MB, and ≥5 MB, respectively. The risk of mortality from ICH also increased with the quantity of MB. However, the rate of recurrent CI did not significantly increase with the quantity of MB. The study concluded that while anti-thrombotic agents are still warranted due to the risk of recurrent CI, patients with ≥5 MB may have a higher risk and mortality of ICH that outweighs the modest benefit of antithrombotic agents, suggesting extra precautions should be taken to minimize the risk of ICH.

Role of SWI in the detection of venous congestion and SWI – DWI mismatch and their correlation with patient outcome

Susceptibility-weighted imaging (SWI) is a useful method for predicting infarct size in the ischemic brain. Ischemic zones have a larger oxygen extraction fraction and slower flow, which leads to higher amounts of deoxyhemoglobin and vein dilatation. The increased deoxyhemoglobin and vein dilatation increase the visibility of vessels on SWI, indicating venous congestion. This process can assist detect areas of elevated oxygen demand and possible infarct progression, making SWI a viable alternative for predicting infarct growth [35]

SWI is highly sensitive to both intravascular and extravascular deoxygenated blood. In acute ischemic cerebral hemispheres, cerebral veins on SWI appear as multiple hypointense vessels (MHV) [35].

The study by **Min-Gyu Park et al [35] (2014)** investigated the clinical significance of multiple hypointense vessels (MHV) on susceptibility-weighted imaging (SWI) in patients with acute ischemic stroke. Out of 80 patients, MHV were observed in 68 (85%), with 44 patients showing extensive MHV. The results showed that patients with more extensive MHV on SWI had:

Smaller diffusion lesion volume ($p < 0.001$)

Larger diffusion-perfusion mismatch (DPM) ($p < 0.001$)

Lower initial National Institutes of Health Stroke Scale (NIHSS) scores ($p = 0.022$)

Prominent distal hyperintense vessels (DHV) on FLAIR ($p < 0.001$)

Good collateral flow on time-of-flight MRA source images ($p < 0.001$)

The study concluded that more extensive MHV on SWI in acute ischemic stroke is associated with lower initial NIHSS scores, smaller diffusion lesion volume, better collateral flow, and larger diffusion-perfusion mismatch (DPM). The study concluded that MHV on SWI may be a useful marker for predicting increased oxygen extraction fraction and diffusion-perfusion mismatch in acute ischemic stroke

FIGURE 5

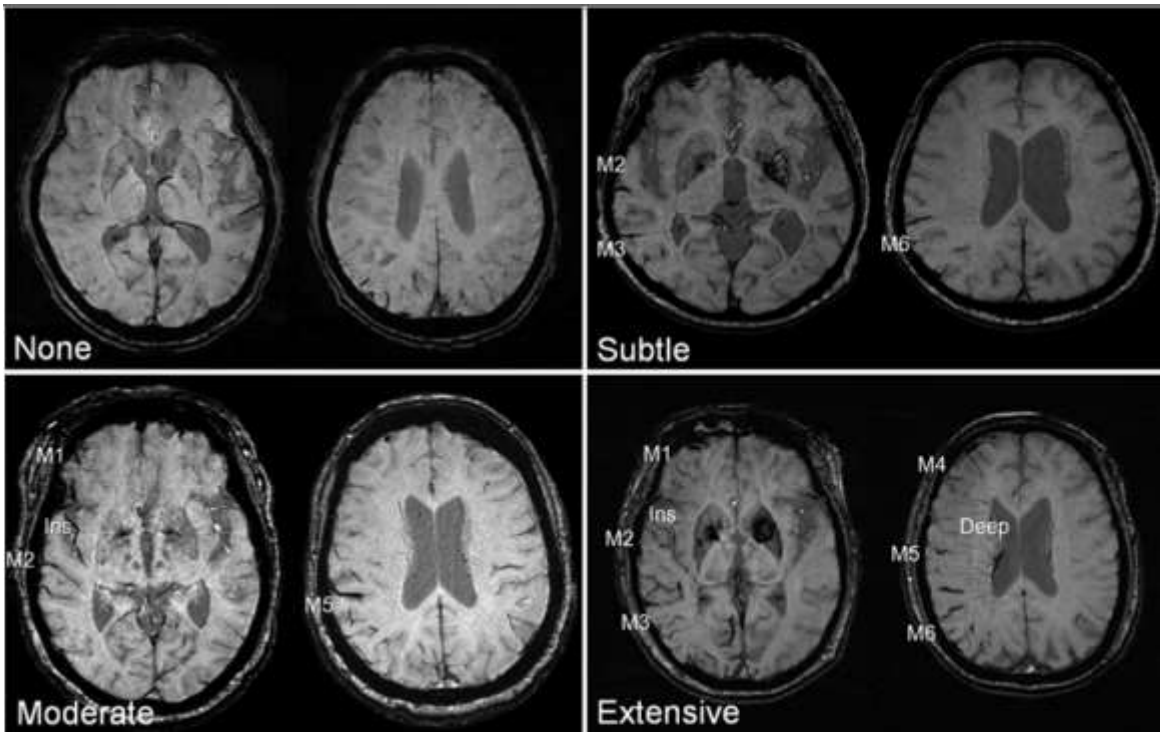


Image courtesy: **Min-Gyu Park et al [35]**

The grading of multiple hypointense vessels (MHV) on susceptibility-weighted imaging (SWI) is based on the number of defined areas where these hypointense vessels are observed. The grading system includes:

- None: No MHV are seen in any middle cerebral artery (MCA) territory.
- Subtle: MHV are observed in 1-3 defined areas.
- Moderate: MHV are seen in 4-6 defined areas.
- Extensive: MHV are observed in 7-8 defined areas, which include specific regions such as the insular cortex, anterior MCA cortex, and other areas of the MCA territory[35] .

Susceptibility-weighted imaging (SWI) is a promising method for predicting infarct size in acute stroke. In the ischemic brain, greater oxygen extraction fraction and sluggish flow cause larger amounts of deoxyhemoglobin and vein dilatation, making vessels more visible on SWI. This prominent vessel sign (PVS) on SWI corresponds well with venous and capillary deoxyhemoglobin levels, suggesting enhanced oxygen extraction. The area displaying PVS can be a target for acute therapy because it is connected with the final infarction area after 72 hours[36]

Chen et al. [36] (2015) studied the impact of the prominent vessel sign (PVS) on susceptibility-weighted imaging (SWI) and infarct growth in 22 individuals with middle cerebral artery (MCA) infarction. The study discovered that infarct expansion occurred on the second MRI in 13 of 15 patients with PVS on the initial SWI, whereas no infarct growth was identified in any of the 7 patients without PVS. The extent of PVS was significantly associated to the degree of infarct development. The researchers found that the presence of PVS on the initial SWI following acute MCA stroke is an accurate predictor of early infarct development. Extensive PVS inside the broad MCA zone is related with poor early-stage outcomes and may be useful for clinical assessment and management of acute stroke patients

FIGURE 6

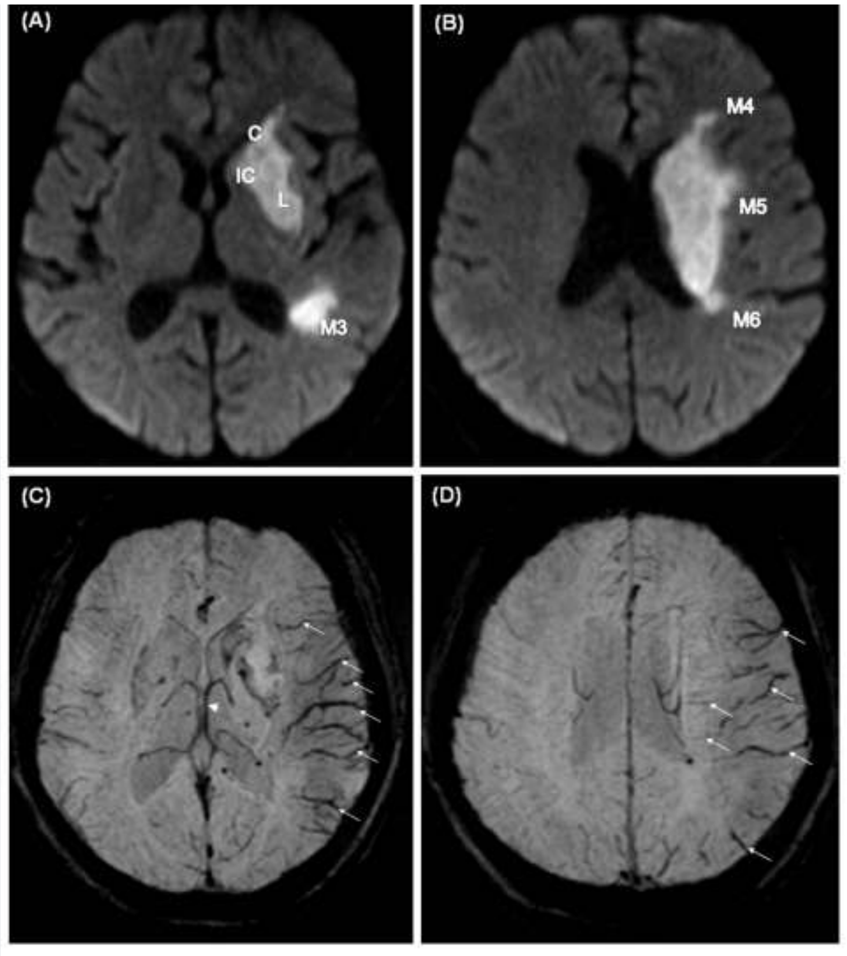


Image courtesy : chia yuen chen et al[36]

(A,B) Infarctions in the middle cerebral artery territory's internal capsule, lentiform nucleus, caudate nucleus and zones M3, M4, and M5[36].

(C, D) SWI indicates prominent hypointense cortical and medullary veins in the insula, as well as the M1-M6 zones of the left MCA [36]

Seung Kug Baik et al [37] (2012) investigated the change in cortical vessel signs (CVSs) on susceptibility-weighted imaging (SWIs) following complete recanalization in individuals with hyperacute ischemic stroke. The study comprised 19 patients with acute major artery blockage who received immediate SWI following thrombolysis. The study found that all ten individuals with pre-recanalization SWIs had significant CVSs on their afflicted side. CVSs disappeared in all patients following thrombolysis. Post-recanalization SWIs revealed equal, less, or equal and less cortical veins in the afflicted area. Patients with equal cortical veins exhibited mild lesions on diffusion-weighted imaging (DWI), whereas those with fewer cortical veins had medium to large lesions The study indicated that significant CVSs on SWI can suggest acute thromboembolic blockage, and their variation following recanalization can reflect metabolic state. The presence of equal CVSs on SWI following recanalization was related with a better clinical outcome

FIGURE 7

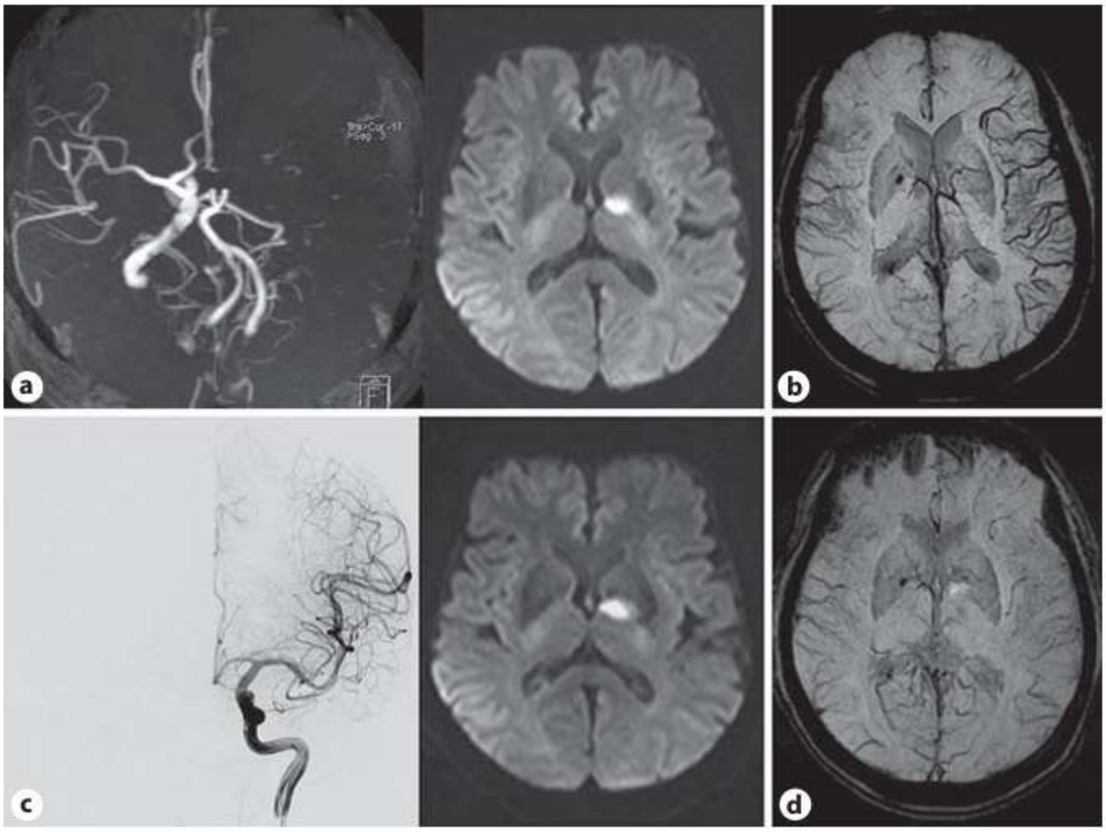


Image courtesy : Seung Kug Baik et al [37] (2012)

Image a : TOF MRA showing occlusion of left ICA and MCA
Image b : SWI image showing multiple hypointense vessels in left MCA territory
Image c : Post recanalization status showing complete recanalization of left ICA and MCA
Image d : SWI image showing non visualisation of multiple hypointense vessels

The periventricular white matter contains long and tiny vessels known as deep medullary veins (DMV). These vessels drain the cerebral hemispheres' white matter. They run perpendicular to the lateral ventricle's long axis and form collecting trunks that drain into the subependymal veins. Normal deep medullary veins couldn't be visible. Pathologic DMV can be recognized from normal DMV due to earlier visibility by contrast-enhanced imaging, an increased calibre and length, an irregular course with frequent diameter changes, and a tendency to present in clusters[38].

In a study by **Johanna M et al** [38] **(2015)**., 86 patients with acute middle cerebral artery (MCA) stroke were examined using susceptibility-weighted imaging (SWI). The study found that 55 patients displayed asymmetric deep medullary veins (DMVs), while 31 did not. The asymmetric group had a higher median National Institute of Health Stroke Scale (NIHSS) score on admission (17, 11-21) compared to the non-asymmetric group (9, 5-15), with a statistically significant difference ($p = 0.001$). Similarly, on discharge, the median NIHSS score for the asymmetric group was 11 (5-20), while it was 5 (2-14) for the non-asymmetric group, again with a statistically significant difference ($p = 0.005$). The study concluded that the asymmetry of deep medullary veins on SWI is a predictor of poor outcome in patients with acute MCA territory stroke.

A difference in the extent of venous congestion on SWI and infarct on diffusion-weighted imaging (DWI) is referred to as susceptibility-diffusion mismatch. This mismatch occurs when the area of venous congestion is greater than the area of diffusion restriction, indicating that the venous congestion is more extensive than the actual infarcted tissue.

M. Lou et al. [4] **(2014)** investigated whether patients with substantial asymmetry of cerebral veins on susceptibility-weighted imaging (SWI) and comparatively small diffusion-weighted imaging (DWI) lesions (SWI-DWI mismatch) would benefit more from thrombolytic therapy. The study found that patients with a SWI-DWI mismatch had a much higher rate of positive outcomes (78% vs. 44%) than those without the mismatch.

Patients with a SWI-DWI mismatch had a higher chance of receiving a good result following reperfusion (91% vs. 43%) and recanalization (100% vs. 40%). SWI-DWI mismatch was more accurate in predicting a positive outcome than perfusion-diffusion mismatch (63% vs 48.1%). The study proposes that the presence of SWI-DWI mismatch could identify patients with ischemia who would benefit from early reperfusion therapy

Zhihua Xu et al. (39) (2021) investigated the relationship between diffusion-weighted imaging (DWI) and susceptibility-weighted imaging (SWI) mismatch, collateral circulation, and prognosis in patients with blocked M1 segments of the middle cerebral artery (MCA). The study found that DWI-SWI mismatch was significantly associated with prominent laterality of the posterior (PLPCA) and anterior (PLACA) cerebral arteries, as well as hyperintense vascular sign (HVS) prominence, which indicated circulatory collaterals.

Following MCA M1-segment blockage, the DWI-SWI mismatch, NIHSS, and DWI-ASPECTS scores altered considerably in relation to patient prognosis (good vs. bad). In binary logistic regression analysis, NIHSS and DWI-SWI mismatch were found to be independent predictor variables.

The study demonstrates that collateral circulation may be a critical feature of DWI-SWI mismatch, which is related to the prognosis

The study demonstrates that collateral circulation may be a critical part of DWI-SWI mismatch, which is associated to the prognosis of MCA M1-segment blockage

RESULTS

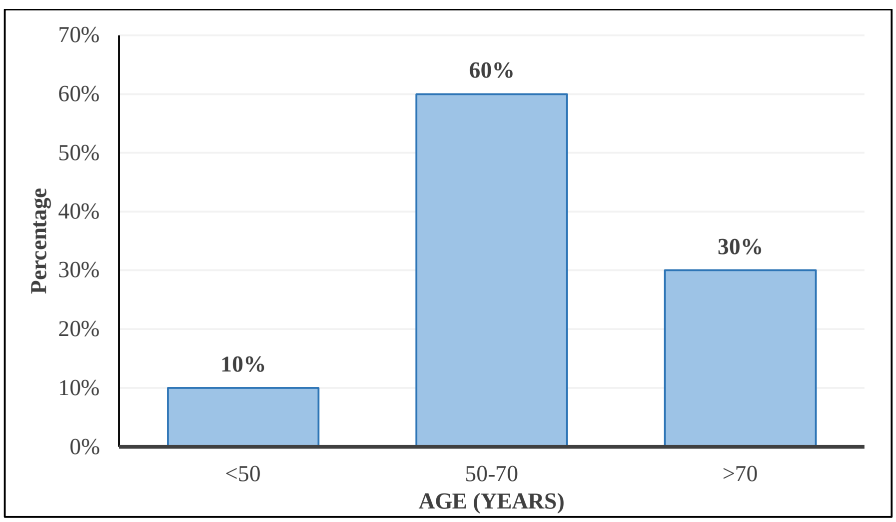
In this present study, 120 patients with symptom onset of acute ischaemic stroke within 12 hours were studied in the age group above 25 years.

1) Majority of the cases (60 %) were in the age group of 50-70 years followed by 30% of cases in the age group of > 70 years and 10 % of cases were in the age group of < 50 years.

Table 1: Distribution of subjects according to age.

Age group	Frequency	Percentage
< 50 years	12	10
50-70 years	72	60
>70 years	36	30
Total	120	100

Graph 1: Distribution of subjects according to age.

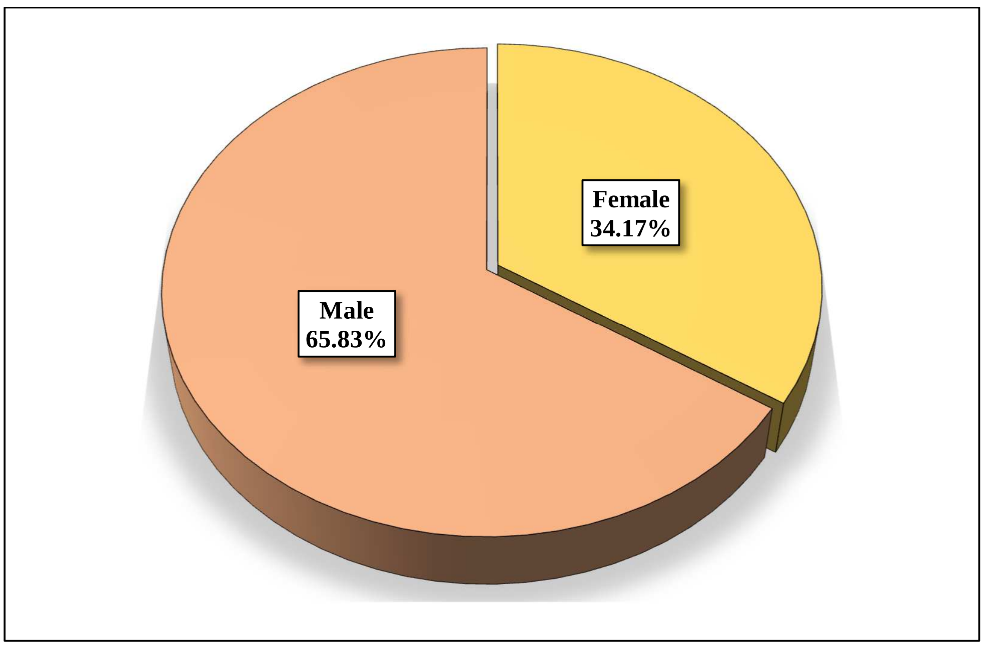


2) This study showed male predominance with 65.8% of males and 34.2% of females.

Table 2: Distribution of subjects according to gender.

Gender	Frequency	Percentage
Male	79	65.8
Female	41	34.2
Total	120	100.0

Graph 2: Distribution of subjects according to gender.



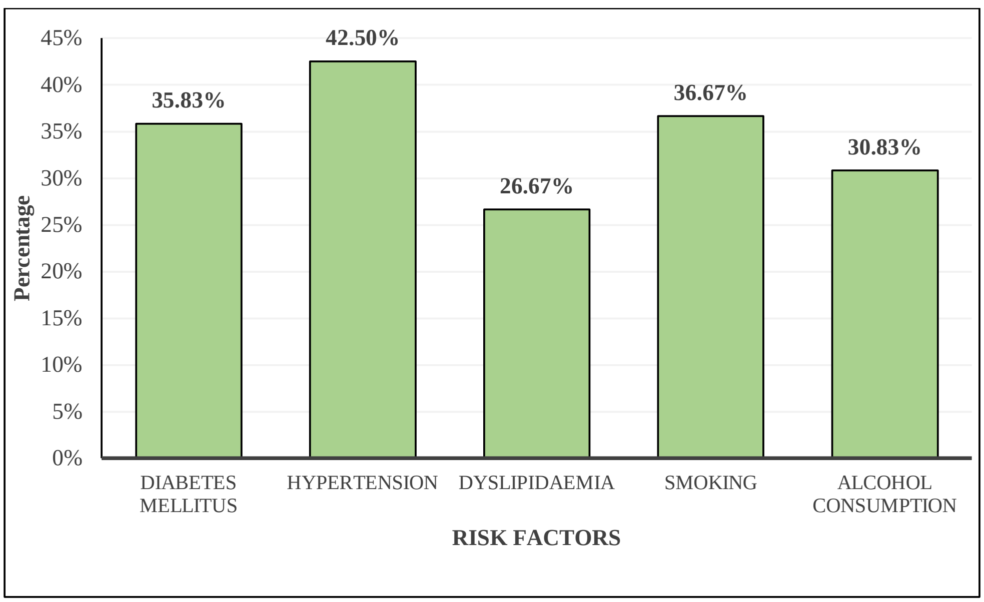
3) Risk factors

In this study, Diabetes mellitus (DM) was present in 35.8%, hypertension in 42.5%, dyslipidaemia in 26.7%, habit of smoking in 36.7% and alcohol consumption in 30.8% cases.

Table 3: Distribution of subjects according to risk factors.

RISK FACTORS	FREQUENCY	PERCENTAGE (%)
DIABETES MELLITUS	43	35.8
HYPERTENSION	51	42.5
DYSLIPIDAEMIA	32	26.7
SMOKING	44	36.7
ALCOHOL CONSUMPTION	37	30.8

Graph 3: Distribution of subjects according to risk factors.

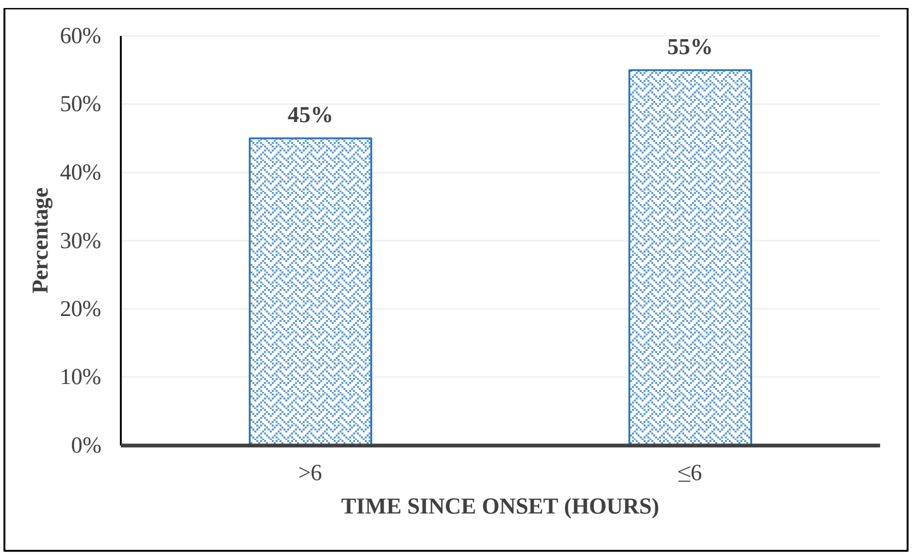


4) On assessing the time since symptom onset 55% of cases reported within 6 hours of symptom onset and the rest 45 % of cases reported after 6 hours of symptom onset.

Table 4: Distribution of subjects according to time since onset of symptoms.

Time since onset	Frequency	Percentage
≤ 6 hours	66	55
> 6 hours	54	45
Total	120	100

Graph 4: Distribution of subjects according to time since onset of symptoms.



5) The following table gives the distribution of subjects according to FLAIR.

Table 5: Distribution of subjects according to FLAIR.

Variables	Mean ± SD	Median (Min, Max)
FLAIR	23.35 ± 18.57	22 (0, 88)

The mean volume of hyperintense brain tissue was 23.35 ± 18.57 cc. The median volume was 22 cc, with the range spanning from 0 to 88 cc.

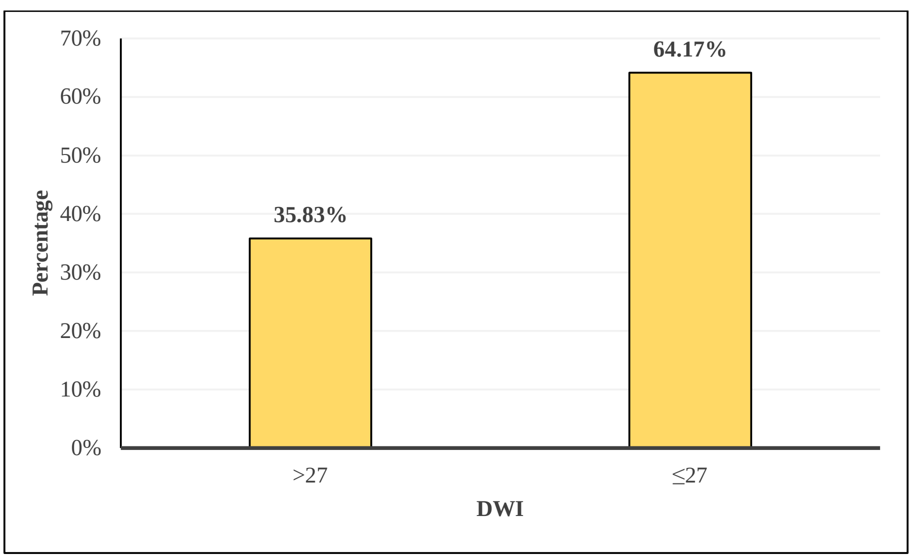
6) The following table gives the distribution of subjects according to DWI.

Table 6: Distribution of subjects according to DWI.

DWI	Number of subjects (%)
>27	43 (35.83%)
≤27	77 (64.17%)
Mean ± SD Median (Min, Max)	25.02 ± 18.95 21.5 (6, 90)

Among the subjects, 43 (35.83%) exhibited diffusion restriction volumes greater than 27cc, whereas 77 (64.17%) subjects had volumes of 27cc or less. The mean volume of brain tissue with diffusion restriction was 25.02 ± 18.95cc. The median volume was 21.5cc, with a range from 6 to 90cc.

Graph 5: Distribution of subjects according to DWI.



7) The following table gives the comparison of volume of brain tissue from FLAIR and DWI among patients with symptom onset of more than 6 hours.

Table 7: Comparison of volume of brain tissue from FLAIR and DWI among patients with symptom onset of more than 6 hours.

Variables	Mean ± SD	Median (Min, Max)	p-value
FLAIR	32.06 ± 17.1	30 (10, 88)	**0.0019^{MW*}**
DWI	24.91 ± 14.09	24 (8, 72)	

Abbreviation: MW – Mann Whitney U test, * indicates statistical test.

The volume of brain tissue displaying hyperintensity on FLAIR was greater than the volume of brain tissue showing diffusion restriction in patients with symptom onset of more than 6 hours, (p-value = 0.0019).

8) The following table gives the result of regression analysis of time since onset in predicting change in F-D.

Table 8: Result of regression analysis of time since onset in predicting change in F-D.

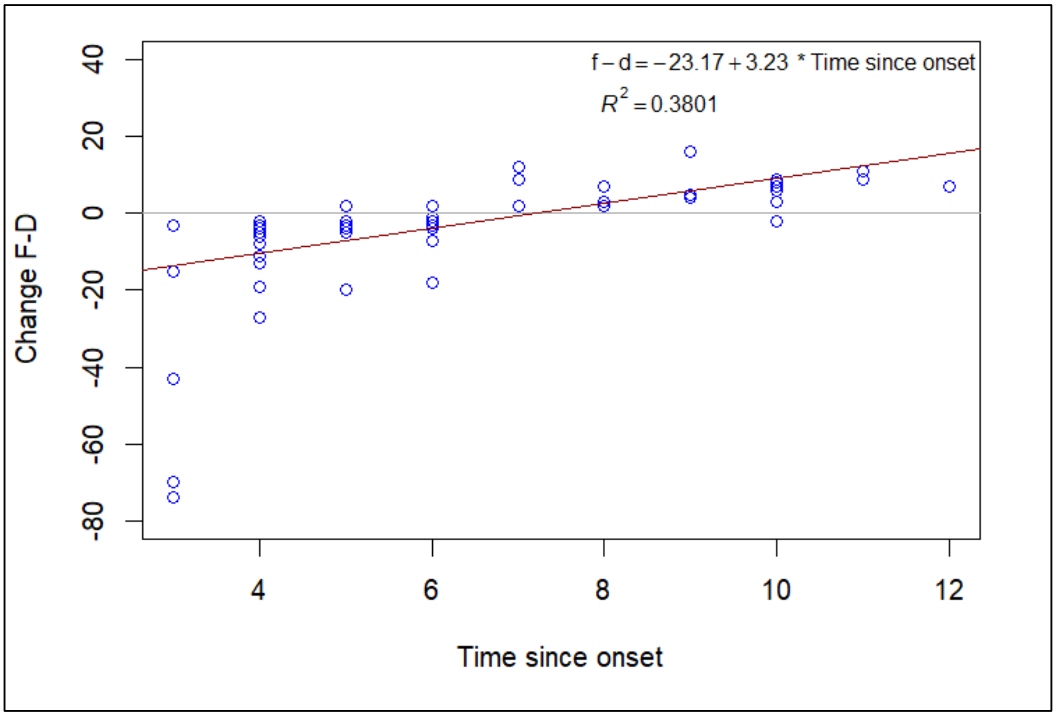
Variables	Coefficient	p-value
Intercept	-23.17	**< 0.001***
Time since onset	3.23	**< 0.001***

*Abbreviation: * indicates statistical significance.*

Time since onset of symptoms has significant effect on change F-D (p-value < 0.001). The relationship between the volume of brain tissue displaying hyperintensity (f), volume of brain tissue showing diffusion restriction on DWI (d), and time of onset:

$$f - d = -23.17 + 3.23 * \text{ Time since onset}$$

Graph 6: Plot of change F-D with time since onset.



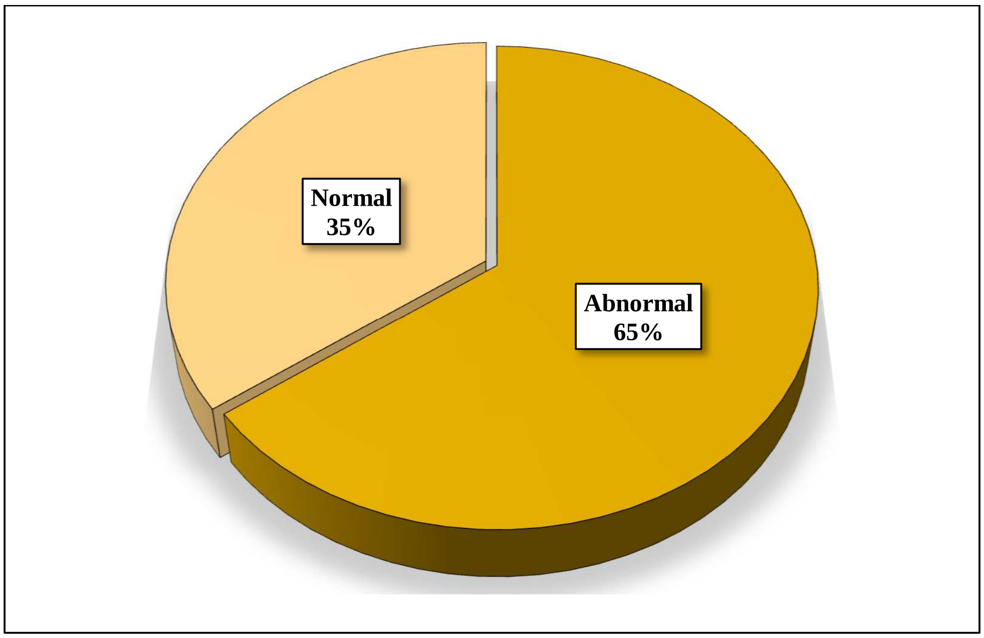
9) In the present study, 65% (78) of cases had TOF MRA abnormalities and only 35 % of cases had normal TOF MRA. Out of them SWI hypointensity vessel sign (SVS) on SWI was present in 70 out of 78 (89.7%) of cases with a sensitivity of 89.74% , specificity of 85.71% , PPV of 92.11% NPV of 81.82%.

The hypointensity sign on SWI demonstrated a highly significant correlation with abnormal findings on TOF- MR Angiography (p-value < 0.001).

Table 9: Distribution of subjects according to TOF MRA.

TOF MRA	Frequency	Percentage
Abnormal	78	65
Normal	42	35
Total	120	100

Graph 7: Distribution of subjects according to TOF MRA.



10) The following table gives the comparison of hypointensity sign with TOF MRA

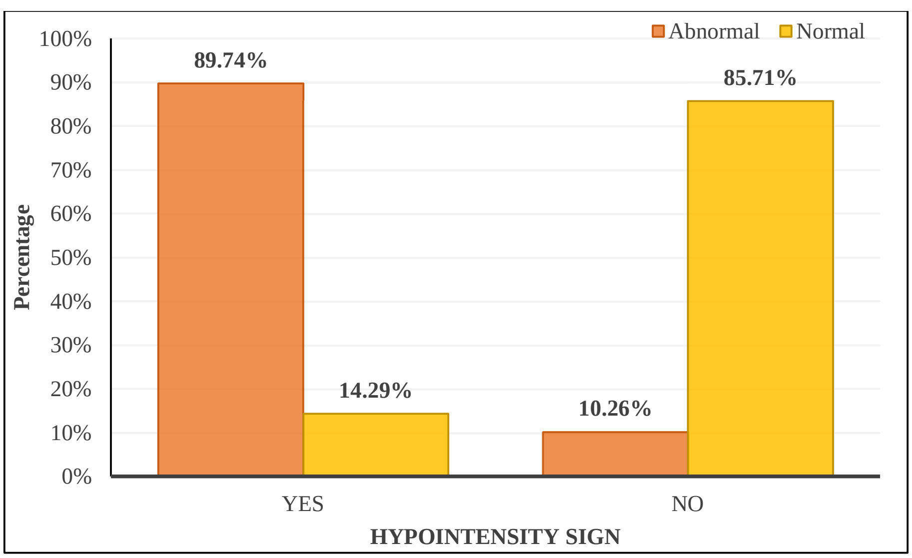
Table 10: Distribution of subjects with Hypointensity sign (SVS) ON SWI among the cases who showed TOF-MRA abnormality

Hypointensity sign	TOF MRA		p-value
	Abnormal	**Normal**	
Yes	70 (89.74%)	6 (14.29%)	**< 0.001^{C*}**
No	8 (10.26%)	36 (85.71%)	
Total	78	42	

Abbreviation: C – Chi square test, * indicates statistical significance.

Among those with abnormal TOF MRA findings, a significant majority (89.74%) exhibited the Hypointensity sign. Conversely, among subjects with normal TOF MRA results, a much smaller proportion (14.29%) had the Hypointensity sign. From Chi square test, it is observed that there is significant association of hypointensity sign with TOF MRA (p-value < 0.001).

Graph 8: Distribution of subjects with Hypointensity sign (SVS) ON SWI among the cases who showed TOF-MRA abnormality

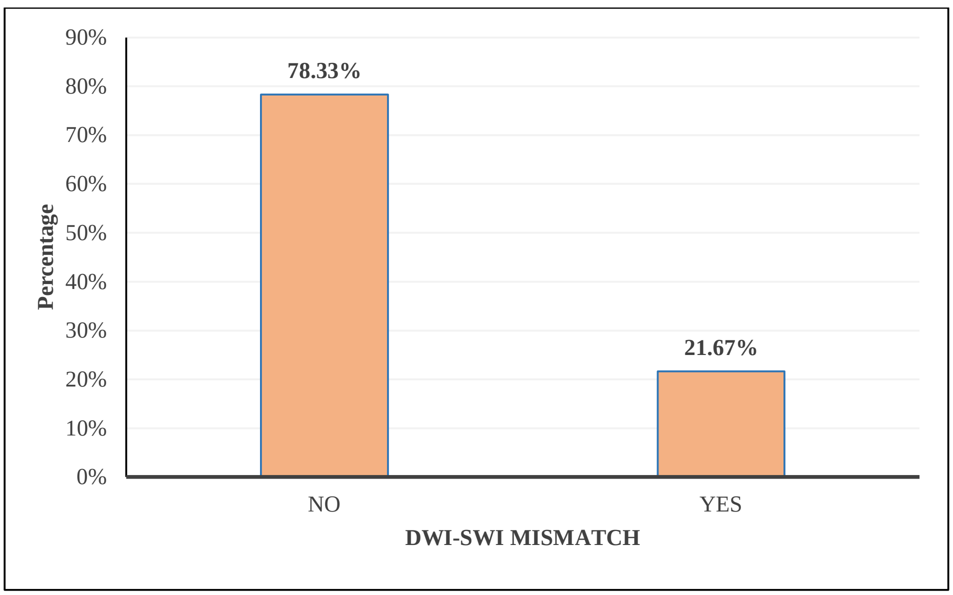


11) Out of 120 patients, 26 (21.6%) patients had diffusion-susceptibility mismatch, which showed no correlation with infarcted brain tissue volume or time since onset of symptoms .

Table 11: Distribution of subjects with DWI-SWI mismatch

DWI – SWI Mismatch	Frequency	Percentage
Yes	26	21.6
No	94	78.3
Total	120	100

Graph 9: Distribution of subjects with DWI-SWI mismatch

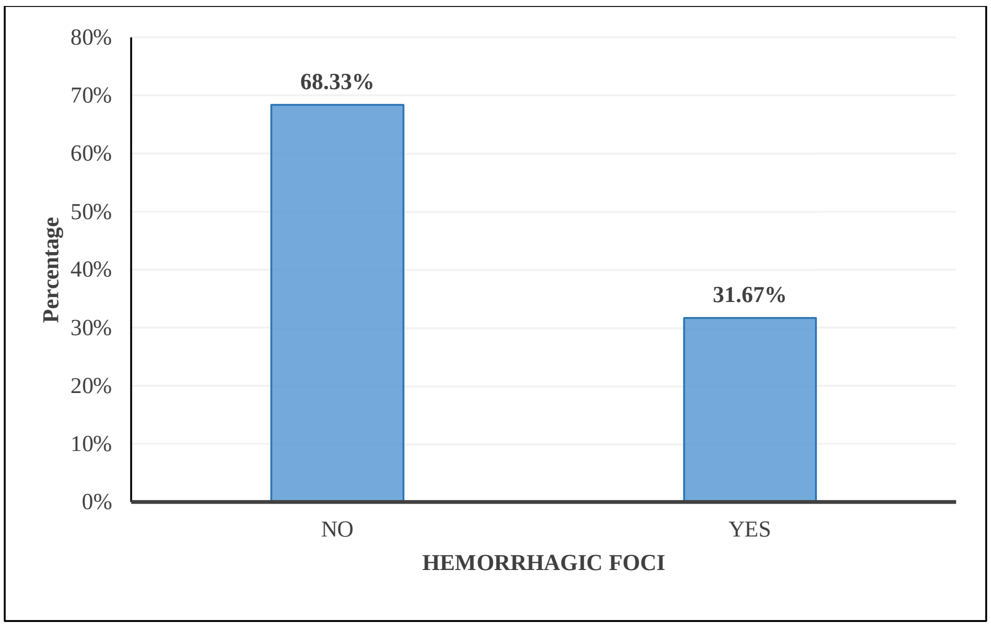


12) Among the subjects, 82 (68.33%) did not exhibit hemorrhagic foci, while 38 (31.67%) showed hemorrhagic foci.

Table 12: Distribution of subjects according to Hemorrhagic Foci.

Hemorrhagic foci	Frequency	Percentage
Present	38	31.6
Absent	82	68.3
Total	120	100

Graph 10: Distribution of subjects according to Hemorrhagic Foci.



13)

a) The following table gives the comparison of time since onset of symptoms with Hemorrhagic Foci.

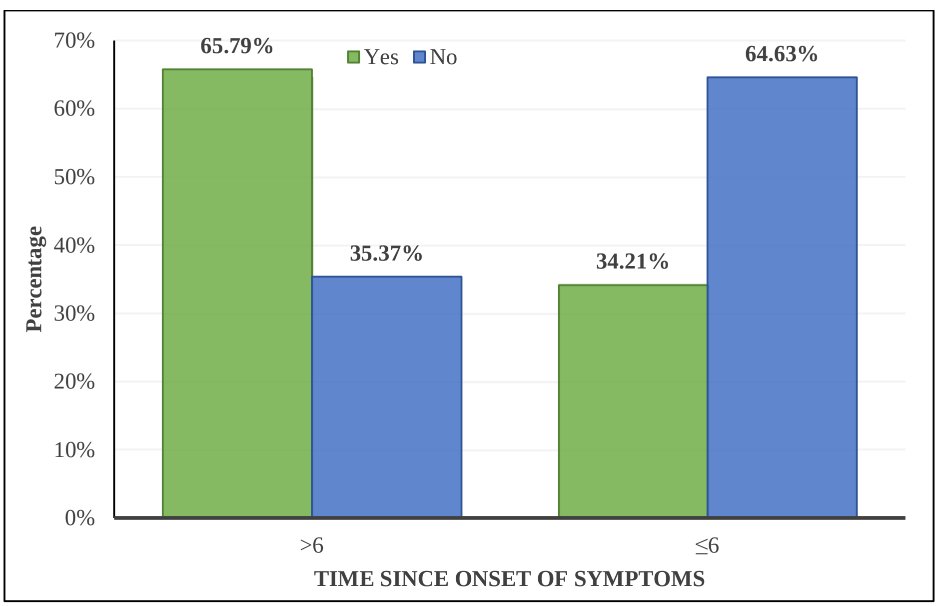
Table 13: Distribution of time since onset of symptoms with Hemorrhagic Foci.

Time since onset	Hemorrhagic Foci		p-value
	Yes	No	
>6	25 (65.79%)	29 (35.37%)	**0.0018^C***
≤6	13 (34.21%)	53 (64.63%)	

Abbreviation: C – Chi square test, * indicates statistical significance.

Among those with Hemorrhagic Foci, 65.79% had time since onset >6. Conversely, among subjects without Hemorrhagic Foci, only 35.37% had time since onset >6. From Chi square test, it is observed that there is significant association of time since onset of symptoms with Hemorrhagic Foci (p-value = 0.0018).

Graph 11: Distribution of time since onset of symptoms with Hemorrhagic Foci.



b) The following table gives the comparison of DWI with Hemorrhagic Foci.

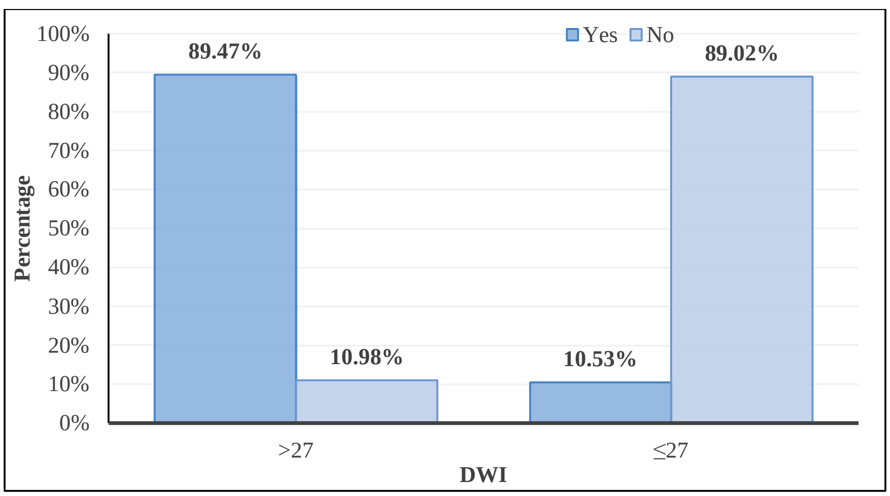
Table 14: Distribution of DWI with Hemorrhagic Foci.

DWI	Hemorrhagic Foci		p-value
	Yes	No	
>27	34 (89.47%)	9 (10.98%)	< 0.001[C*]
≤27	4 (10.53%)	73 (89.02%)	

Abbreviation: C – Chi square test, * indicates statistical significance.

Among those with Hemorrhagic Foci, a significant majority (89.47%) exhibited the DWI greater than 27. Conversely, among subjects without Hemorrhagic Foci, a much smaller proportion (10.98%) had the DWI greater than 27. From Chi square test, it is observed that there is significant association of DWI with Hemorrhagic Foci (p-value < 0.001).

Graph 12: Distribution of DWI with Hemorrhagic Foci.



Microhemorrhagic foci and the volume of infarcted brain tissue exhibiting diffusion restriction showed highly significant association (sensitivity (89.47%), specificity (89.02%), positive predictive value (PPV) of 79.07% and a negative predictive value (NPV) of 94.81%).

ROC analysis revealed microhemorrhagic foci in infarcts with a cut-off volume of 27 cc on the DWI sequence

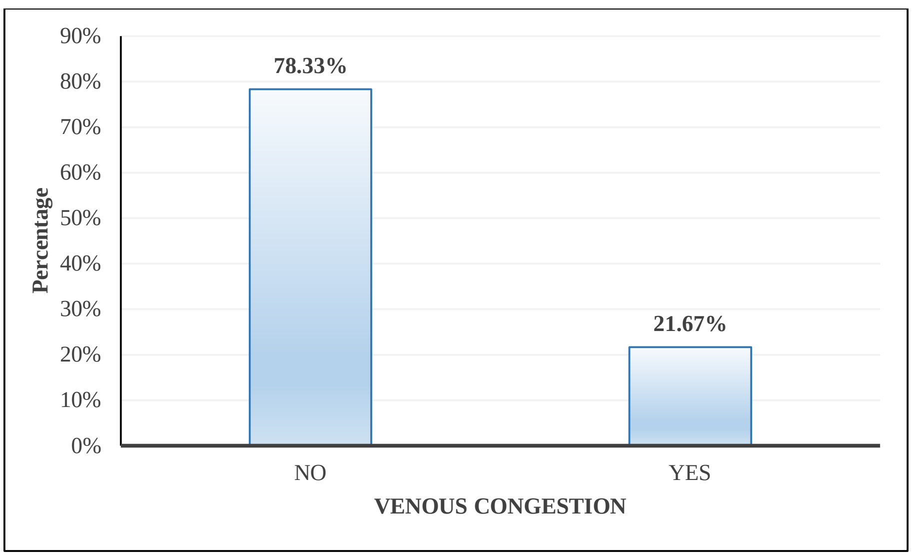
14) In this study, 26 (21.6%) cases had venous congestion however the rest 78.4% of cases revealed no venous congestion.

There was no significant statistical correlation between cortical venous congestion and volume of brain tissue showing diffusion restriction or time since onset of symptoms. The resolution of cerebral venous congestion was achieved with thrombolysis.

Table 15: Distribution of subjects according to Venous Congestion.

Venous congestion	Frequency	Percentage
Present	26	21.6
Absent	94	78.4
Total	120	100

Graph 13: Distribution of subjects according to Venous Congestion.



15) 38 (31.6 %) patients out of the 120 received intravenous thrombolysis. In ten patients, post-thrombolysis hemorrhagic transformation developed. Eight individuals experienced early spontaneous hemorrhagic transformation, which was represented by SWI.

A SWI-DWI mismatch was found in 26 (21.6 %) out of 120 patients. 16 of the 26 patients with SWI-DWI mismatch, underwent thrombolytic treatment and all of these patients showed improvement by three grade in their power of the muscles. The increase in muscle strength in the rest of the 22 patients who did not exhibit diffusion–susceptibility mismatch who underwent thrombolysis, ranged between 1 and 3 grades.

Table 16: Distribution of subjects who underwent I.V. thromboysis

I.V. THROMBOLYSIS	Frequency	Percentage
Yes	38	31.6
No	82	68.4
Total	120	100

Graph 14: Distribution of subjects who underwent I.V. thromboysis

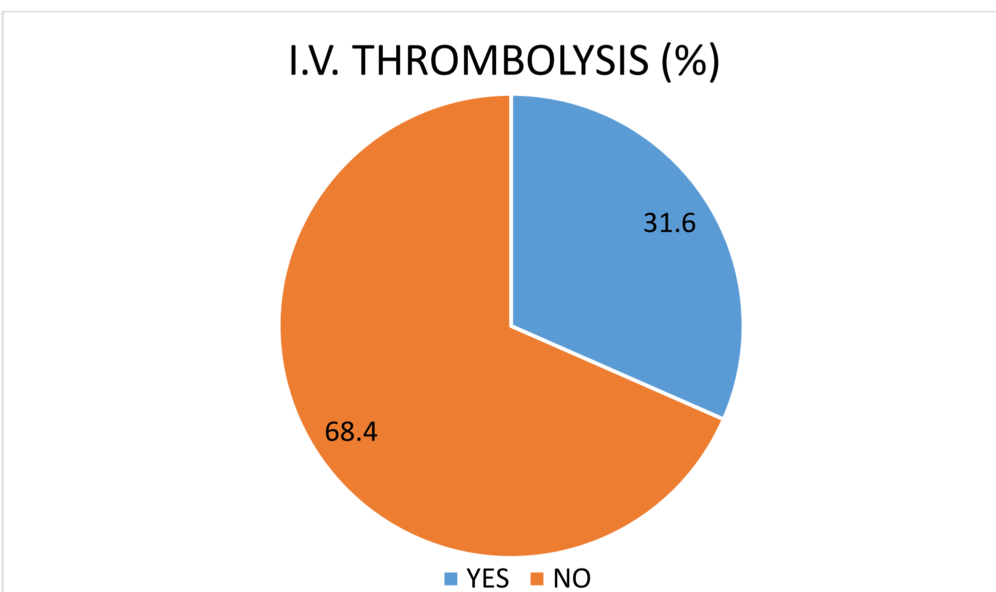


Table 17: Distribution of subjects who developed post-thrombolysis haemorrhagic transformation

HEMORRHAGIC TRANSORFATION	Frequency	Percentage
Yes	10	26.3
No	28	73.6
Total	38	100

Graph 15: Distribution of subjects who developed post-thrombolysis haemorrhagic transformation

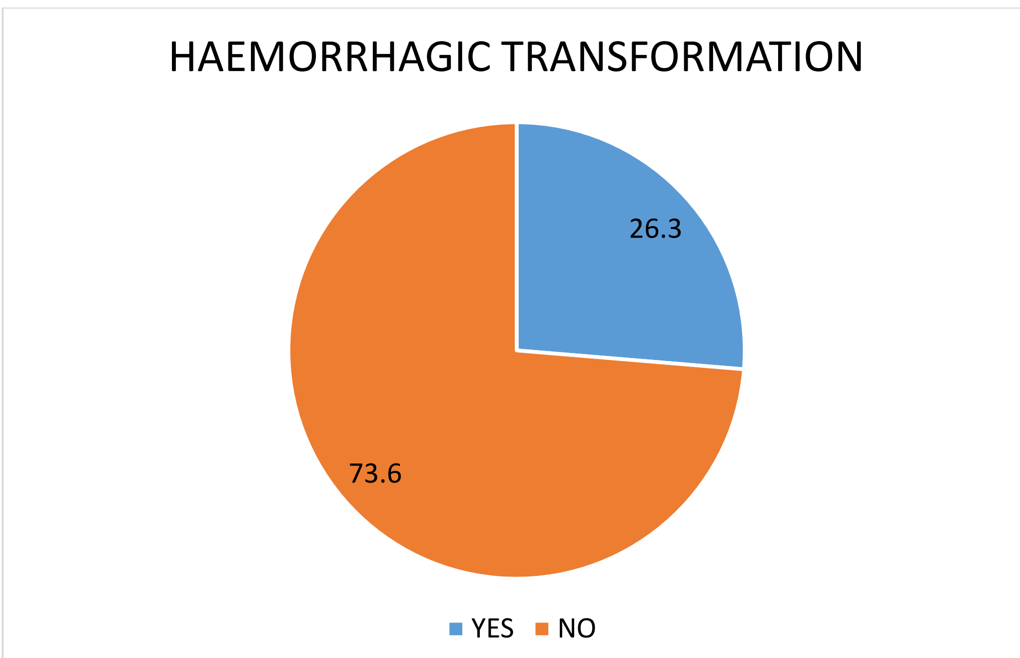


Table 18: Distribution of subjects who developed early spontaneous hemorrhagic transformation

EARLY SPONTANEOUS HEMORRHAGIC TRANSFORMATION	Frequency	Percentage
Yes	8	6.6
No	112	93.3
Total	120	100

Graph 16: Distribution of subjects who developed early spontaneous hemorrhagic transformation

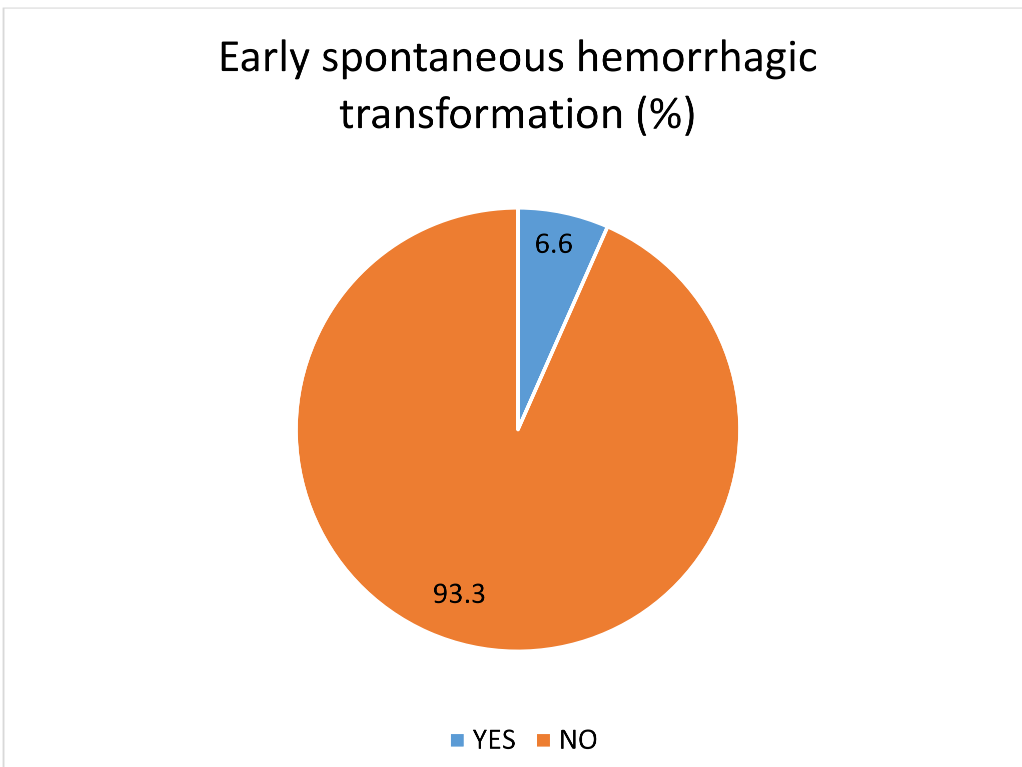
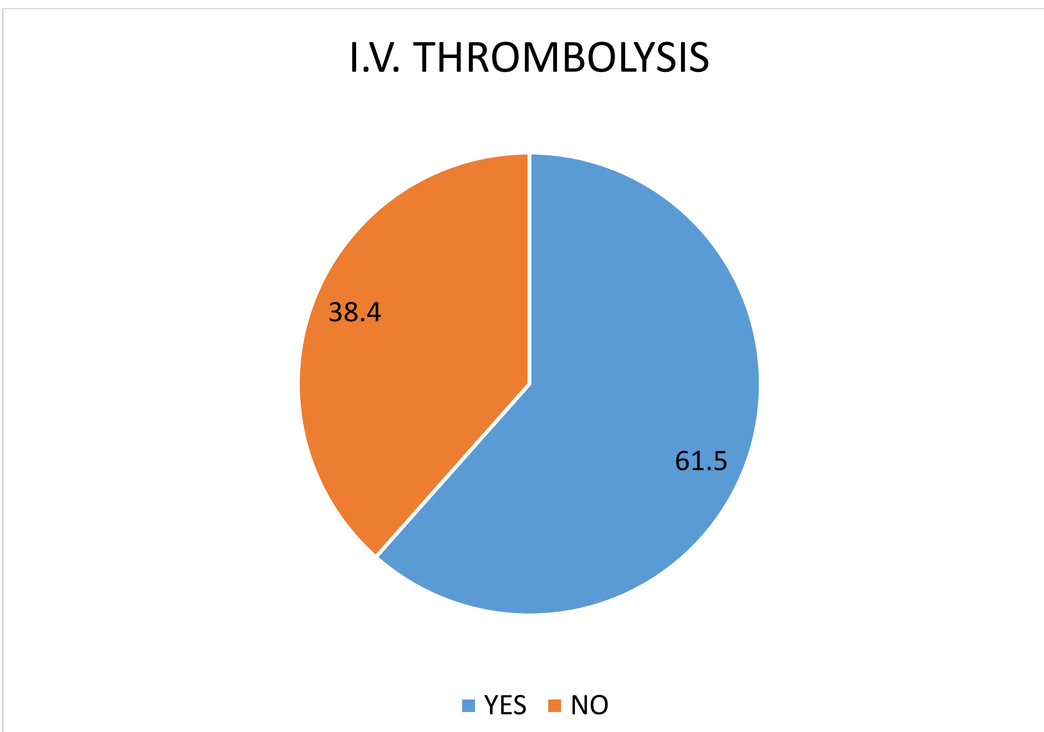


Table 19: Distribution of subjects who underwent I.V. thrombolysis among the cases that showed DWI-SWI mismatch

I.V. THROMBOLYSIS	Frequency	Percentage
Yes	16	61.5
No	10	38.4
Total	26	100

Graph 17: Distribution of subjects who underwent I.V. thrombolysis among the cases that showed DWI-SWI mismatch



In our study it was observed that in patients with pre-thrombolysis microhemorrhages on SWI imaging were associated with post thrombolysis hemorrhagic transformation.

DISCUSSION

Magnetic resonance neuroimaging has expanded substantially during the last two decades as a result of improved magnetic and biomedical technologies. This has led to improved skills for early stroke diagnosis and treatment

To provide thrombolytic therapy to individuals with unwitnessed strokes, it's important to distinguish between when the patient was last known to be healthy and when they were diagnosed with symptoms. Witnessed strokes are identical. Unwitnessed strokes have uncertain onset times, which may impact eligibility for therapeutic interventions[2].

DWI and SWI are essential techniques for imaging acute stroke patients. The present research of 120 individuals suffering from acute ischemic stroke addresses several elements of DWI and SWI features with TOF- MR Angiography correlation.

In our study of 120 acute ischemic stroke patients presenting within 12 hours of onset of symptoms, 66 (55%) cases reported within 6 hours of onset of symptoms and the rest 54 (45 %) cases reported after 6 hours of onset of symptoms.

It was noted that the volume of brain tissue displaying hyperintensity on FLAIR was larger than the volume of brain tissue showing diffusion restriction in patients with symptom onset of more than 6 hours (p-value < 0.001). Our study revealed a relationship between volume of brain tissue showing hyperintensity on FLAIR (f), volume of brain tissue showing diffusion restriction on DWI (d), and time of onset as:

$$f - d = -23.17 + 3.23 * \mathit{Time\ since\ onset}$$

Similarly a study conducted by **Seyedmehdi Payabvash et al** **[40]** **(2016)** confirms previous findings on using DWI-FLAIR mismatch to identify ischemic infarcts within the thrombolytic therapy window. It uniquely shows that the effect of symptom onset-to-scan time gap on DWI-FLAIR mismatch loss varies depending on the DWI infarct lesion volume. The study also demonstrates that the specificity and predictive accuracy of DWI-FLAIR mismatch improve when applying lesion volume thresholds, reaching 100% specificity and PPV for infarct lesions larger than 15 mL on DWI. This suggests that thrombolytic therapy may be administered in patients with unknown symptom onset if the infarct lesion volume on DWI is larger than 15 mL and there is no corresponding FLAIR hyperintensity.

Using our technique for determining time of onset based on DWI-FLAIR mismatch, patients with ambiguous time of onset can be administered thrombolysis, which would otherwise not be administered if a stringent time requirement is followed.

Xiao wei et all [41] (2016) conducted the study investigated the feasibility of using DWI-FLAIR mismatch to identify ischemic stroke patients who may benefit from thrombolytic therapy within 4.5-6 hours of symptom onset. The results showed that a mismatch between positive DWI and negative FLAIR images identified patients within 4.5 hours of symptom onset with moderate sensitivity (40.9%), specificity (76.9%), positive predictive value (75%), and negative predictive value (43.5%). Additionally, patients with FLAIR-negative images within the 4.5-6-hour window had better outcomes in terms of recanalization rate, NIHSS, and modified Rankin Scale (mRS) scores compared to those with FLAIR-positive images

Hence, the aforementioned studies revealed that DWI positive - FLAIR negative (DWI- FLAIR mismatch) had superior results with thrombolysis, because their time from start of symptoms was less than 6 hours and the infarcted brain tissue volume was over a defined threshold level

SWI can detect the susceptibility vessel sign (SVS) in numerous major intracranial artery locations, allowing it to identify intra-arterial thrombus in AIS cases and pinpoint its specific location. However, since obstructed or limited arteries are regarded to be the primary cause of AIS development, time of flight (TOF)-MRA is a routinely used technique in examination of individuals suffering from AIS.

In the light of these, the purpose of this study was to evaluate the collaborative impact of SWI and TOF-MRA in the assessment of AIS.

The SVS is a blooming artifact at the site of thrombus. The presence of deoxyhemoglobin in the thrombus in turn indicates the existence of red blood cells . The chemical components of the thrombus, changes over time postulating that the presence of an SVS correlates with the age of the thrombus[42]

In a study conducted by **Jean Darcourt et al [43] (2019)**, it was concluded that the visualization of SVS is a reliable and easily accessible predictive factor of recanalization success and early clinical improvement.

In our study, 78 (65%) of the 120 patients had abnormal TOF MRA. SWI hypointensity vessel sign (SVS) on SWI was present in 70 out of 78 (89.7%) of cases with a sensitivity of 89.74% , specificity of 85.71% , PPV of 92.11% NPV of 81.82% for intracranial arterial occlusions . Significant correlation was found between hypointensity sign (SVS) on SWI and intracranial arterial occlusions on TOF-MRA (p-value < 0.001)

In another study similarly , Thrombus location as seen on SWI correlated well with MR angiographic findings [6].

Sangmin lee et all[44] (2016) conducted a study aimed to evaluate the diagnostic value of SWI for detecting hyperacute MCA occlusion. The results showed that SWI was highly sensitive (88.2%) and specific (97.1%) for detecting MCA occlusion, with a PPV of 96.8% and a NPV of 89.5%. The diagnostic accuracy was 92.8%. Additionally, SWI detected the susceptibility vessel sign (SVS) in 88.2% of patients with MCA occlusion. The study concluded that SWI is a sensitive, specific and accurate method for detecting hyperacute MCA occlusion, making it a valuable tool for diagnosing and managing acute stroke patients.

Similar to the above mentioned study the ‘susceptibility sign' accurately identified all acute major intracranial artery occlusions with a 82% sensitivity and 100% specificity in a study conducted by **D. Lingegouda [28]** .

However the sensitivity and specificity for the same was found to be lower in our study as The previous research utilized CE MRA as a reference standard versus TOF-MRA which was used in our study.

Blooming does not happen with all acute thrombi. The composition of the thrombus determines how the paramagnetic effect changes in comparison to the rest of the circulating blood. Clots dominated by white blood cells exhibit less significant changes in the paramagnetic effect compared to clots dominated by red blood cells[45].

Cerebral microbleeds (CMBs) are increasingly detected in patients with stroke. They are more common in patients with recurrent stroke than in those with first-ever stroke, indicating a link to the progression of small vessel cerebrovascular disease. CMBs are likely caused by previous small areas of blood leakage from damaged small vessels [46].

The evidence suggests that cerebral small vessel disease (SVD) and cerebral microbleeds (CMBs) are associated with an increased risk of intracranial hemorrhage (ICH) in patients receiving antithrombotic therapy, particularly after ischemic stroke. The presence of SVD and CMBs can make small vessels brittle and fragile, increasing the risk of ICH. Anticoagulant use alone should not cause ICH if cerebral vessels are intact, but the underlying SVD and CMBs can be a significant predictor of future ICH risk. This highlights the need for careful risk-benefit assessments in patients with SVD and CMBs, as the benefits of antithrombotic therapy must be weighed against the potential risks of ICH[46].

In our study, the proportion of cases with microhemorrhagic foci was 38 (31.65%). A significant correlation was seen between hemorrhagic foci and time since onset of symptoms of more than 6 hours (p-value = 0.0018). Microhemorrhagic foci and the volume of brain tissue showing infarction with restricted diffusion had a highly significant correlation. ROC analysis revealed microhemorrhagic foci in infarcts with a volume cut-off of 27 cc on the DWI (sensitivity (89.47%), specificity (89.02%)).

In a study performed by **Soo et al.** **[34]** The risk and mortality of intracranial hemorrhage (ICH) increase with the quantity of microbleeds (MB). While the tendency to recurrent cerebral infarction exceeds that of ICH, anti-thrombotic agents are still necessary. However, in patients with ≥ 5 microbleeds, the high risk and mortality of ICH outweigh the modest benefits of anti-thrombotic agents, suggesting that these patients may not benefit from anti-thrombotic therapy.

In a study conducted by **Nayab Z Dar et all.** **[47]** 30 of 118 patients experienced hemorrhagic transformation. Most microbleeds were found in the parietal region with a 50% transformation rate. The size and grade of microbleeds were statistically associated with hemorrhagic transformation, with 33% of Grade 3 microbleeds in patients aged 55-65 years having haemorrhagic transformation and 93.3% of Grade 4 microbleeds having haemorrhagic transformation.

In contradiction, A study by **Ho Sung Kim et al.** **[48]** found no statistically significant difference in the occurrence of hemorrhagic transformation between patients with a large number of microbleeds and those without microbleeds. This result was attributed to the small sample size of patients with a large number of microbleeds receiving thrombolytic treatment.

SWI could be a useful tool for predicting infarct growth(35). The signal drop in prominent a cortical vein (PCV) on susceptibility-weighted imaging (SWI) is related to the higher oxygen extraction fraction (OEF) in the critically hypoperfused parenchyma. This increased OEF leads to a locally higher ratio of deoxyhemoglobin to oxyhemoglobin in capillaries and veins, indicating the stress that the tissue-at-risk is under. The degree of signal drop in the PCV on SWI is thought to vary according to the degree of hypoperfusion and oxygen extraction increasing the conspicuity of vessels on SWI and hence the venous congestion (35) (49) .

In our study, 26 (21.6%) cases had venous congestion .The resolution of cerebral venous congestion was achieved with thrombolysis.

Chen et al. (36) (2015) studied the impact of the prominent vessel sign (PVS) on susceptibility-weighted imaging (SWI) and infarct growth in 22 individuals with middle cerebral artery (MCA) infarction. The study discovered that infarct expansion occurred on the second MRI in 13 of 15 patients with PVS on the initial SWI, whereas no infarct growth was identified in any of the 7 patients without PVS. The extent of PVS was significantly associated to the degree of infarct development. The researchers found that the presence of PVS on the initial SWI following acute MCA stroke is an accurate predictor of early infarct development. Extensive PVS inside the broad MCA zone is related with poor early-stage outcomes and may be useful for clinical assessment and management of acute stroke patients.

The term "susceptibility-diffusion mismatch" refers to a difference in the extent of venous congestion on susceptibility-weighted imaging (SWI) and infarct volume on diffusion-weighted imaging (DWI). This mismatch occurs when the area of venous congestion is greater than the area of diffusion restriction, indicating that the venous congestion is more extensive than the actual infarcted tissue. Asymmetric hypointensity of cerebral veins on SWI has been demonstrated to indirectly represent tissue hypoxia following cerebral ischemia. As a result, the SWI-DWI mismatch, which indicates salvageable tissue, may identify individuals who may benefit from early reperfusion treatment, such as thrombolytic therapy[4] [50].

In our study out of 120 patients, 26 (21.6%) patients had diffusion-susceptibility mismatch, which showed no correlation with volume of brain tissue showing infarction or time since symptom onset .

The aforementioned findings were similar to a study by **M.Lou et al.**[4] (2014) who investigated whether SWI-DWI mismatch were more likely to benefit from thrombolytic therapy. The study found that patients with a SWI-DWI mismatch had a much higher rate of positive outcomes (78% vs. 44%) than those without the mismatch. Patients with a SWI-DWI mismatch had a higher chance of receiving a good result following reperfusion (91% vs. 43%) and recanalization (100% vs. 40%). SWI-DWI mismatch was more accurate in predicting a positive outcome than perfusion-diffusion mismatch (63% vs 48.1%). The study proposes that the presence of SWI-DWI mismatch could identify patients with ischemia who would benefit from early reperfusion therapy

In our study 38 (31.6 %) patients out of the 120 received intravenous thrombolysis. In ten patients, post-thrombolysis hemorrhagic transformation developed. Eight individuals experienced early spontaneous hemorrhagic transformation, which was represented by SWI.

As mentioned earlier , in our study SWI-DWI mismatch was found in 26 (21.6%) out of 120 patients. 16 of the 26 patients who had SWI-DWI mismatch underwent thrombolytic treatment and all of these patients showed improvement by three grade in their power of the muscles. The increase in muscle strength in the rest of the 22 patients who did not exhibit diffusion–susceptibility mismatch but underwent thrombolysis , ranged between 1 and 3 grades. This suggests that SWI-DWI mismatch may be a useful indicator for predicting the effectiveness of thrombolysis in improving muscle power in patients with acute ischemic stroke.

CONCLUSION

The DWI-FLAIR mismatch is a surrogate for time since onset of stroke in acute ischaemic stroke patients. It is used for identifying patients within the stipulated timeframe for thrombolysis treatment. Patients with a DWI-FLAIR mismatch are more likely to be within 6 hours of symptom onset, which is the recommended time window for thrombolytic therapy.

The hypointense susceptibility vessel sign (SVS) on SWI is a significant indicator in acute ischemic stroke. It is associated with the presence of a thrombus and can be used to detect peripheral thrombi more effectively than TOF magnetic resonance angiography (MRA). The SVS is caused by the increased magnetic susceptibility of deoxyhemoglobin in the thrombus, which is more visible on SWI due to its higher sensitivity to these substances.

The detection of microbleeds on susceptibility-weighted imaging (SWI) is a reliable and accurate predictor of hemorrhagic transformation in acute ischemic stroke patients. Increased number, size, and grade of microbleeds on SWI are linked with an increased risk of hemorrhagic transformation. SWI should be included in the MRI stroke protocol to identify microbleeds that may otherwise go undetected on routine imaging, allowing better risk stratification and management of acute ischemic stroke patients

The SWI-DWI mismatch is an important imaging indicator in acute ischemic stroke that can help guide thrombolytic therapy. It denotes a mismatch between DWI and SWI, indicating the presence of an ischemic penumbra with misery perfusion. This mismatch represents the viability of brain tissue at risk of infarction if not reperfused promptly. The SWI-DWI mismatch is an excellent diagnostic marker for assessing the ischemic penumbra and can aid in predicting the efficacy of thrombolytic therapy by identifying areas of viable brain tissue that may benefit. Furthermore, the SWI-DWI mismatch is linked to the presence of collateral circulation, which is an important characteristic that improves prognosis in acute ischemic stroke.

The collaborative use of SWI, DWI, and MRA provides a comprehensive evaluation of acute ischemic stroke, allowing for improved patient selection and management, including guiding thrombolytic therapy

MATERIALS AND METHODS

Source of data: Patients referred for MRI of brain above the age of 25 years, who presented within 12 hours of symptom onset for acute ischaemic stroke to the department of Radio-Diagnosis at The KLE'S Dr. Prabhakar Kore hospital & MRC, Belgaum.

Method of collection of data:

1. **Study design**: a one year hospital based cross sectional study
2. **Sample size:** Sample size was calculated as below

Formula used for sample size calculation is,

$$n = \frac{\widehat{Se}\left(100 - \widehat{Se}\right) {Z_{\propto/2}}^2}{d^2\, P}$$

where n is the sample size required, P is the occurrence of a state or condition (prevalence), $\widehat{Se}$ is sensitivity, d is the maximum error required, $Z_{\propto/2}$ is the value corresponding to level of confidence required.

Susceptibility vessel sign on SWI sequence showed a sensitivity of 66.6% compared with TOF-MRA. Considering similar result in our study, at 95% confidence level, 5% maximum error and assuming the prevalence to be 72%, the sample size is given by,

$$n = \frac{0.667 \text{ x } (1 - 0.667) \text{ x } 1.96^2}{0.1^2 \text{x } 0.72}$$

$$n = 118.5086 \approx 119$$

Hence, minimum sample size required in the study was 119. As sample size increases, accuracy of result increases.

Statistical Analysis:

Data is analysed using statistical software R version 4.4.0. and Microsoft Excel. Categorical variables given in the form of frequency tables. Continuous variables given in Mean ± SD /Median (Min, Max) form. Chi square test is used to check the association of categorical variables with groups. Normality of variable is checked by Shapiro Wilk test and QQ plot. If data follows normal distribution, parametric tests will be used. Otherwise, non-parametric tests will be used. Mann Whitney U test is used to compared the distribution of variables over FLAIR and DWI. Linear regression analysis is used to check the effect of time since onset of symptoms on change F-D. P-value less than or equal to 0.05 indicates statistical significance.

(1) **Sampling method:** All patients were evaluated clinically and then underwent an MRI brain using a 3 Tesla MRI scanner (Magnetom Avanto TIM, 18 channel; Siemens, Erlangen, Germany) within 12 hours of symptom onset for acute ischemic stroke.

(2) **Duration**: One year – between January 2023 to December 2023

(3) **Inclusion Criteria:**

i) Patients above 25 years of age who presented within 12 hours of symptom onset for acute ischemic stroke

ii) Patients / attenders consenting to take part in the study

(4) Exclusion criteria:

Contraindications to MRI like patients with metallic medical implants (intraocular metallic foreign body, cardiac pace makers, MR non compatible intracranial clips of arterial brain aneurysms etc).

(5) Methodology: The study was conducted using 3 T MRI scanner (Magnetom Avanto TIM , 18 channel ; Siemens, Erlangen, Germany) Standard scan protocol was followed for all the patients presenting with the symptoms of acute ischemic stroke. Once the MRI was done, findings were noted and analysed. Consent was taken from all the patients / attenders. All the data collected was entered in to MS Excel sheet, data was tabulated & tables, charts and graphs were prepared.

MRI sequences obtained:

1. Axial **–** T1 weighted, T2 weighted, FLAIR
2. Sagital - T1 weighted
3. Coronal - T2 weighted
4. Diffusion weighted imaging and apparent diffusion coffecient
5. Susceptibility weighted imaging
6. Magnetic resonance angiography

Ethical committee approval: Ethical committee approval was obtained for this study from the Ethics Committee of Jawaharlal Nehru Medical college, Belgaum. Informed and written consent was obtained from patients whenever possible, or family members of patients who were unable to give formal consent.

SUMMARY

1. In the present study, 120 patients with onset of ischemic symptoms within 12 hours were studied in the age group ranging from 25 to 85 years.
2. Majority of the cases (60 %) were in the age group of 50-70 years followed by 30% of cases in the age group of > 70 years and 10 % of cases were in the age group of < 50 years.
3. This study showed male predominance with 65.8% of males.
4. In this study hypertension was the present in 42.5% cases
5. On assessing the time since symptom onset 55% of cases reported within 6 hours of symptom onset and the rest 45 % of cases reported after 6 hours of symptom onset
6. In patients with symptom onset lasting more than 6 hours, the volume of brain tissue exhibiting hyperintensity on FLAIR was larger than the volume of brain tissue demonstrating diffusion restriction (p-value < 0.001). For an infarct volume more than 27 cc on DWI, regression analysis indicated the following association between hyperintensity (f), diffusion restriction (d), and time of onset:

$$f - d = -23.17 + 3.23 * Time\ since\ onset$$

7. In the present study, 65% (78) of cases had TOF MRA abnormalities. Out of them SWI hypointensity vessel sign (SVS) on SWI was present in 70 out of 78 (89.7%) of cases with a sensitivity (89.74%), specificity (85.71%), PPV (92.11%), and NPV (81.82%). The hypointensity sign on SWI demonstrated a highly significant correlation with abnormal findings on TOF- MR Angiography (p-value < 0.001).

8. Out of 120 patients, 26 (21.6%) patients had diffusion-susceptibility mismatch, which showed no correlation with volume of brain tissue showing infarction or time since symptom onset.
9. On assessing the proportion of cases with microhemorrhagic foci, there were 38 (31.67%) of cases with hemorrhagic foci.
10. Significant correlation was observed between hemorrhagic foci and time since onset of more than 6 hours (p-value = 0.0018) and in infarcts with a volume cut-off of 27 ml on the DWI sequence (sensitivity (89.47%) & specificity (89.02%).
11. In this study, 26 (21.6%) cases had venous congestion. There was no significant statistical correlation between cortical venous congestion and volume of brain tissue showing diffusion restriction or time since onset of symptoms.

    The resolution of cerebral venous congestion was achieved with thrombolysis.\
12. A SWI-DWI mismatch was found in 26 (21.6 %) out of 120 patients. 16 of the 26 patients , underwent thrombolytic treatment and all of these patients showed improvement by three grade in their power of the muscles .Patients who did not exhibit diffusion–susceptibility mismatch who underwent thrombolysis , ranged from 1 to 3 grades.

LIMITATIONS

1. Limited sample size, due to which further characterization of few of the cases was not possible.
2. The study did not take into account the role of CT

BIBLIOGRAPHY

1. van der Worp, H. Bart, and Jan van Gijn. “Acute Ischemic Stroke.” New England Journal of Medicine, vol. 357, no. 6, 9 Aug. 2007, pp. 572–579,
2. Wu, Ona, et al. “Imaging Stroke Patients with Unclear Onset Times.” Neuroimaging Clinics of North America, vol. 21, no. 2, May 2011, pp. 327–344,
3. Branko Huisa, et al. “Diffusion-Weighted Imaging–Fluid Attenuated Inversion Recovery Mismatch in Nocturnal Stroke Patients with Unknown Time of Onset.” DWI – FLAIR Mismatch in Nocturnal Strokes Patients with Unknown Time of Onset, vol. 22, no. 7, 1 Oct. 2013, pp. 972–977
4. Lou, M., et al. “Susceptibility-Diffusion Mismatch Predicts Thrombolytic Outcomes: A Retrospective Cohort Study.” American Journal of Neuroradiology, vol. 35, no. 11, 1 Nov. 2014, pp. 2061–2067, www.ajnr.org/content/35/11/2061
5. Hermier, Marc, and Norbert Nighoghossian. “Contribution of Susceptibility-Weighted Imaging to Acute Stroke Assessment.” Stroke, vol. 35, no. 8, Aug. 2004, pp. 1989–1994
6. Weisstanner, Christian, et al. “Thrombus Imaging in Acute Stroke: Correlation of Thrombus Length on Susceptibility-Weighted Imaging with Endovascular Reperfusion Success.” European Radiology, vol. 24, no. 8, 16 May 2014, pp. 1735–1741
7. Srinivasan, Ashok, et al. “State-of-The-Art Imaging of Acute Stroke.” RadioGraphics, vol. 26, no. suppl_1, Oct. 2006, pp. S75–S95
8. Mergenthaler, P., and A. Meisel. “Do Stroke Models Model Stroke?” Disease Models & Mechanisms, vol. 5, no. 6, 31 Oct. 2012, pp. 718–725

9. Kanekar, Sangam G., et al. “Imaging of Stroke: Part 2, Pathophysiology at the Molecular and Cellular Levels and Corresponding Imaging Changes.” American Journal of Roentgenology, vol. 198, no. 1, Jan. 2012, pp. 63–74
10. Moseley ME, Kucharczyk J, Mintorovitch J, Cohen Y, Kurhanewicz J, Derugin N, et al. Diffusion-weighted MR imaging of acute stroke: correlation with T2-weighted and magnetic susceptibility-enhanced MR imaging in cats. AJNR American journal of neuroradiology [Internet]. 1990 May 1;11(3):423–9
11. Lansberg MG, Norbash AM, Marks MP, Tong DC, Moseley ME, Albers GW. Advantages of Adding Diffusion-Weighted Magnetic Resonance Imaging to Conventional Magnetic Resonance Imaging for Evaluating Acute Stroke. Archives of Neurology. 2000 Sep 1;57(9).
12. van Everdingen KJ, van der Grond J, Kappelle LJ, Ramos LMP, Mali WPTM. Diffusion-Weighted Magnetic Resonance Imaging in Acute Stroke. Stroke. 1998 Sep;29(9):1783–90.
13. Yoo AJ, Verduzco LA, Schaefer PW, Hirsch JA, Rabinov JD, GonzálezRG. MRI-Based Selection for Intra-Arterial Stroke Therapy. Stroke. 2009 Jun;40(6):2046–54.
14. Jiang L, Peng M, Chen H, Geng W, Zhao B, Yin X, et al. Diffusion-weighted imaging (DWI) ischemic volume is related to FLAIR hyperintensity-DWI mismatch and functional outcome after endovascular therapy. Quantitative Imaging in Medicine and Surgery. 2020 Feb;10(2):356–67.
15. Thomalla G, Cheng B, Ebinger M, Hao Q, Tourdias T, Wu O, et al. DWI-FLAIR mismatch for the identification of patients with acute ischaemic stroke within 4·5 h of symptom onset (PRE-FLAIR): a multicentre observational study. The Lancet Neurology. 2011 Nov;10(11):978–86.

16. Aoki J, Kimura K, Iguchi Y, Kensaku Shibazaki, Sakai K, Iwanaga T. FLAIR can estimate the onset time in acute ischemic stroke patients. Journal of the Neurological Sciences. 2010 Jun 1;293(1-2):39–44.

17. Haller S, Haacke EM, Thurnher MM, Barkhof F. Susceptibility-weighted Imaging: Technical Essentials and Clinical Neurologic Applications. Radiology. 2021 Apr;299(1):3–26.

18. Haacke EM, Mittal S, Wu Z, Neelavalli J, Cheng YC . N. Susceptibility-weighted imaging: technical aspects and clinical applications, part 1. AJNR American journal of neuroradiology [Internet]. 2009 Jan 1;30(1):19–30

19. Mittal S, Wu Z, Neelavalli J, Haacke EM. Susceptibility-Weighted Imaging: Technical Aspects and Clinical Applications, Part 2. American Journal of Neuroradiology. 2009 Jan 8;30(2):232–52.

20. Flacke S, Urbach H, Keller E, Träber F, Hartmann A, Textor J, et al. Middle Cerebral Artery (MCA) Susceptibility Sign at Susceptibility-based Perfusion MR Imaging: Clinical Importance and Comparison with Hyperdense MCA Sign at CT1. Radiology. 2000 May;215(2):476–82.

21. Rovira A, Orellana P, Alvarez-Sabín J, Arenillas JF, Aymerich X, Grivé E, et al. Hyperacute ischemic stroke: middle cerebral artery susceptibility sign at echo-planar gradient-echo MR imaging. Radiology [Internet]. 2004 Aug 1;232(2):466–73

22. Gasparian GG, Sanossian N, Shiroishi MS, Liebeskind DS. Imaging of Occlusive Thrombi in Acute Ischemic Stroke. International Journal of Stroke. 2014 Dec 25;10(3):298–305.

23. De Leucio A, De Jesus O. MR Angiogram [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2022

24. Miyazaki M, Lee VS. Nonenhanced MR Angiography. Radiology. 2008 Jul;248(1):20–43.

25. Radbruch A, Mücke J, Schweser F, Deistung A, Ringleb P, Ziener CH, et al. Comparison of Susceptibility Weighted Imaging and TOF-Angiography for the Detection of Thrombi in Acute Stroke. PLOS ONE. 2013 May 23;8(5):e63459–9.

26. Abdelgawad EA, Amin MF, Abdellatif A, Mourad MA, Abusamra MF. Value of susceptibility weighted imaging (SWI) in assessment of intra-arterial thrombus in patients with acute ischemic stroke. Egyptian Journal of Radiology and Nuclear Medicine. 2021 Nov 10;52(1).

27. Halefoglu AM. Comparison of Susceptibility Weighted Imaging and Time of Flight MR Angiography in the Detection of Intra-Arterial Thrombus in Acute Ischemic Stroke Patients. ACTA MEDICA IRANICA. 2020 Jun 30;

28. Thomas B, Vaghela V, Hingwala D, Kesavadas C, Lingegowda D, Sylaja P. ′Susceptibility sign′ on susceptibility-weighted imaging in acute ischemic stroke. Neurology India. 2012;60(2):160.

29. Park MG, Oh SJ, Baik SK, Jung DS, Park KP. Susceptibility-Weighted Imaging for Detection of Thrombus in Acute Cardioembolic Stroke. Journal of Stroke [Internet]. 2016 Jan 31;18(1):73–9

30. Santhosh K, Kesavadas C, Thomas B, Gupta AK, Thamburaj K, Kapilamoorthy TR. Susceptibility weighted imaging: a new tool in magnetic resonance imaging of stroke. Clinical Radiology. 2009 Jan;64(1):74–83.

31. Elnekeidy AE, Yehia A, Elfatatry A. Importance of susceptibility weighted imaging (SWI) in management of cerebro-vascular strokes (CVS). Alexandria Journal of Medicine. 2014 Mar 1;50(1):83–91.

32. Cheng AL, Batool S, McCreary CR, Lauzon ML, Frayne R, Goyal M, et al. Susceptibility-Weighted Imaging is More Reliable Than T2*-Weighted Gradient-Recalled Echo MRI for Detecting Microbleeds. Stroke. 2013 Oct;44(10):2782–6.
33. Kidwell CS, Saver JL, Villablanca JP, Duckwiler G, Fredieu A, Gough K, et al. Magnetic Resonance Imaging Detection of Microbleeds Before Thrombolysis. Stroke. 2002 Jan;33(1):95–8.
34. Soo YOY, Yang SR, Lam WWM, Wong A, Fan YH, Leung HHW, et al. Risk vs benefit of anti-thrombotic therapy in ischaemic stroke patients with cerebral microbleeds. Journal of Neurology. 2008 Nov;255(11):1679–86.
35. Park MG, Yang TI, Oh SJ, Baik SK, Kang YH, Park KP. Multiple Hypointense Vessels on Susceptibility-Weighted Imaging in Acute Ischemic Stroke: Surrogate Marker of Oxygen Extraction Fraction in Penumbra? Cerebrovascular Diseases. 2014;38(4):254–61.
36. Chen CY, Chen CI, Tsai FY, Tsai PH, Chan WP. Prominent Vessel Sign on Susceptibility-Weighted Imaging in Acute Stroke: Prediction of Infarct Growth and Clinical Outcome. Jiang Q, editor. PLOS ONE. 2015 Jun 25;10(6):e0131118.
37. Baik SK, Choi W, Oh SJ, Park KP, Park MG, Yang TI, et al. Change in cortical vessel signs on susceptibility-weighted images after full recanalization in hyperacute ischemic stroke. Cerebrovascular Diseases (Basel, Switzerland) [Internet]. 2012 [cited 2024 Jun 14];34(3):206–12.
38. Mucke J, Möhlenbruch M, Kickingereder P, Kieslich PJ, Bäumer P, Gumbinger C, et al. Asymmetry of Deep Medullary Veins on Susceptibility Weighted MRI in Patients with Acute MCA Stroke Is Associated with Poor Outcome. Hendrikse J, editor. PLOS ONE. 2015 Apr 7;10(4):e0120801.

39. Xu Z, Tong Z, Duan Y, Xing D, Song H, Pei Y, et al. Diffusion- and Susceptibility Weighted Imaging Mismatch Correlates With Collateral Circulation and Prognosis After Middle Cerebral Artery M1-Segment Occlusion. Frontiers in neurology. 2021 Jul 22;12.

40. Payabvash S, Taleb S, Benson JC, Rykken JB, Oswood MC, McKinney AM, et al. The Effects of DWI-Infarct Lesion Volume on DWI-FLAIR Mismatch: Is There a Need for Size Stratification? Journal of Neuroimaging. 2016 Nov 23;27(4):392–6.

41. Wei XE, Zhou J, Li WB, Zhao YW, Li MH, Li YH. MRI based thrombolysis for FLAIR-negative stroke patients within 4.5–6 h after symptom onset. Journal of the neurological sciences. 2017 Jan 1;372:421–7.

42. Dillmann M, Bonnet L, Fabrice Vuillier, Moulin T, Biondi A, Charbonnier G. Factors That Influence Susceptibility Vessel Sign in Patients With Acute Stroke Referred for Mechanical Thrombectomy. Frontiers in neurology. 2022 May 11;13.

43. Darcourt J, Withayasuk P, Vukasinovic I, Michelozzi C, Bellanger G, Guenego A, et al. Predictive Value of Susceptibility Vessel Sign for Arterial Recanalization and Clinical Improvement in Ischemic Stroke. Stroke. 2019 Feb;50(2):512–5.

44. Lee S, Cho SB, Choi DS, Park SE, Shin HS, Baek HJ, et al. Susceptibility Vessel Sign for the Detection of Hyperacute MCA Occlusion: Evaluation with Susceptibility-weighted MR Imaging. Investigative Magnetic Resonance Imaging. 2016;20(2):105.

45. Kim HS, Lee DH, Choi CG, Kim SJ, Suh DC. Progression of middle cerebral artery susceptibility sign on T2*-weighted images: its effect on recanalization and clinical outcome after thrombolysis. AJR Am J Roentgenol [Internet]. 2006 Dec [cited 2015 Aug 201;187(6): W650-7

46. Charidimou A, Shakeshaft C, Werring DJ. Cerebral Microbleeds on Magnetic Resonance Imaging and Anticoagulant-Associated Intracerebral Hemorrhage Risk. Frontiers in Neurology. 2012;3.

47. Dar NZ, Ain QU, Nazir R, Ahmad A. Cerebral Microbleeds in an Acute Ischemic Stroke as a Predictor of Hemorrhagic Transformation. Cureus. 2018 Sep 15;

48. Kim HS, Lee DH, Ryu CW, Lee JH, Choi CG, Kim SJ, et al. Multiple Cerebral Microbleeds in Hyperacute Ischemic Stroke: Impact on Prevalence and Severity of Early Hemorrhagic Transformation After Thrombolytic Treatment. American Journal of Roentgenology. 2006 May;186(5):1443–9.

49. Verma RK, Hsieh K, Gratz PP, Schankath AC, Mordasini P, Zubler C, et al. Leptomeningeal collateralization in acute ischemic stroke: Impact on prominent cortical veins in susceptibility-weighted imaging. European Journal of Radiology. 2014 Aug;83(8):1448–54.

50. Liu H, Mei W, Li Y, Huang Y, Ruan S, Zhang Q, et al. Original Article Susceptibility-diffusion mismatch: an effective method by which to detect perfusion-diffusion mismatch in acute ischemic stroke. Int J Clin Exp Med [Internet]. 2016 [cited 2024 Jun 25];9(10):19691–9.

ANNEXURES – I

INFORMED CONSENT FORM

"COLLABORATIVE USE OF SUSCEPTIBILITY WEIGHTED IMAGING WITH DIFFUSION WEIGHTED IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY IN THE ASSESSMENT OF ACUTE ARTERIAL STROKE USING 3T MRI– A ONE YEAR HOSPITAL BASED CROSS -SECTIONAL STUDY"

Name of Student/Principal Investigator: REG. NO. BS01201015

Name of Guide/Co Investigators: DR. ______________________

Objective:

- To study the synergistic effect of susceptibility weighted imaging with diffusion weighted imaging and magnetic resonance angiography in the assessment of acute ischemic stroke
- To evaluate whether susceptibility -weighted imaging (SWI) and diffusion -weighted imaging (DWI) are more effective than conventional MR sequences for evaluation of stroke and selection of candidates for thrombolytic therapy

Introduction: Stroke is a major cause of mortality and morbidity and thrombolysis has served as a catalyst for major changes in the management of acute ischemic stroke. While the licensed time window extends to 3hours from symptom onset, recent data suggest that the trial window can be extended up to 4.5 hours with overall benefit. Nonetheless, 'time is brain' and every effort must be made to reduce the time delay to thrombolysis.(1)

By defining "stroke symptom onset" in the most conservative manner, namely the time the patient was last known to be well, many patients whose onsets are unwitnessed are automatically ineligible for thrombolytic therapy even if their true time of onset would make them eligible.[2]

Advances in magnetic resonance imaging (MRI) neuroimaging have exponentially increased with the advent of stronger magnet systems and biomedical technology during the past two decades, leading to early detection and treatment of stroke. With the advent of newer imaging techniques such as diffusion-weighted imaging (DWI) and susceptibility-weighted imaging

(SWI), management of stroke is moving toward imaging-based approaches rather than by strict time criteria. Studies have shown good results of the use of imaging criteria to select patients for thrombolytic therapy.[3]

DWI is used to detect early ischemic changes with greater conspicuity than standard MRI. DWI uses the principle of ischemia induced restriction of water movement. In order to help identify areas of ischemia, apparent diffusion coefficient (ADC) maps are used and areas that are bright on diffusion and dark on ADC are consistent with acute infarct.[4]

SWI exploits the magnetic susceptibility artifacts generated by local inhomogeneities of the magnetic field. SWI identifies previous microbleeds in acute ischemia, which is associated with a higher risk of post thrombolytic haemorrhagic tranformation. SWI also allows intravascular clot detection at the acute stage.[5]

In this study, I will evaluate the role of diffusion weighted imaging and susceptibility weighted imaging in relation to conventional MR sequences and summarize the synergistic use of susceptibility weighted imaging, diffusion weighted imaging and magnetic resonance angiography in the assessment of acute arterial stroke.

Explanation of procedure:

I request you to kindly participate in the study titled study ‘‘**COLLABORATIVE USE OF SUSCEPTIBILITY WEIGHTED IMAGING WITH DIFFUSION WEIGHTED IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY IN THE ASSESSMENT OF ACUTE ARTERIAL STROKE USING 3T MRI– A ONE YEAR HOSPITAL BASED CROSS -SECTIONAL STUDY**’’ at Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belgaum" is being conducted by **REG. NO. BS01201015**, Post Graduate in Radio- Diagnosis at J. N. Medical College Belgaum, Karnataka, under the guidance of Dr ________________ , Department of Radio-Diagnosis, J. N. Medical College, Belgaum.

We request you to participate in this study as you are eligible to be included. During the study you will be asked questions regarding your present and past medical history and you will be required to answer to the best of your knowledge. You will also be crucially examined as per the protocol drawn as and when required. Study will be conducted over a period of one year. Once the patient signs the informed consent history and examination will be recorded as per proforma. Magnetic resonance imaging (MRI) uses a large magnet and radio waves to look at structures inside your body. You will have to undertake an MRI scan which is done in a closed environment. During the scan, you lie on a table that slides inside a tunnel-shapedmachine. Doing the scan can take a long time, and you must stay still. The scan is painless. The MRI machine makes a lot of noise.

If you agree to participate in the study, please furnish the details pertaining to the study.

Benefits:

Results will help in early detection of acute ischemic stroke with the use of SWI ,DWI sequences and TOF- MRA when done in addition to conventional MRI sequences and will help in selecting patients who are appropriate candidates for thrombolytic therapy.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

QUESTION:

If any enquiries in the future or in case of research related injury illness, you may contact following person.

REG. NO. BS01201015	**DR. ________________**	**Dr. Harsha Hegade**
Post-Graduate, Department of Radio-Diagnosis. J.N.Medical College, Belagavi	Guide , Professor and head of department , Department of Radio-Diagnosis J.N.Medical College, Belagavi	Professor Chairman, J.N. Medical College Institutional Ethical Committee For Human Subjects Research, Belagavi

Legal rights: By signing this consent form, we are not waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**COLLABORATIVE USE OF SUSCEPTIBILITY WEIGHTED IMAGING WITH DIFFUSION WEIGHTED IMAGING AND MAGNETIC RESONANCE ANGIOGRAPHY IN THE ASSESSMENT OF ACUTE ARTERIAL STROKE USING 3T MRI– A ONE YEAR HOSPITAL BASED CROSS -SECTIONAL STUDY**". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator

ANNEXURES – III

PROFORMA FOR DATA COLLECTION

NAME________________________________

AGE___________ **OP/IP NO ___________**

MOBILE_______________

ADDRESS ____

____

HYPERTENSION, DIABETES AND ANY OTHER COMORBIDITIES

____

HISTORY OF TRAUMA ____________________________________

HISTORY OF SURGERY ______________________________________

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS

PAST HISTORY

FAMILY HISTORY

ANNEXURE IV: IMAGES / FIGURES

PHOTOGRAPHS OF CASES

CASE 1

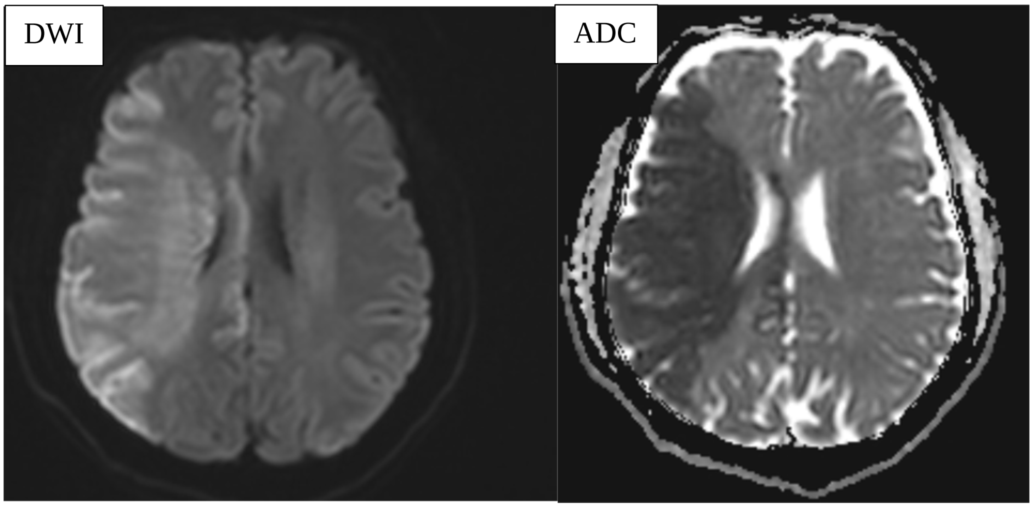


FIGURE 1. INFARCT IN RIGHT MCA TERRITORY SHOWING DIFFUSION RESTRICTION

CASE 2

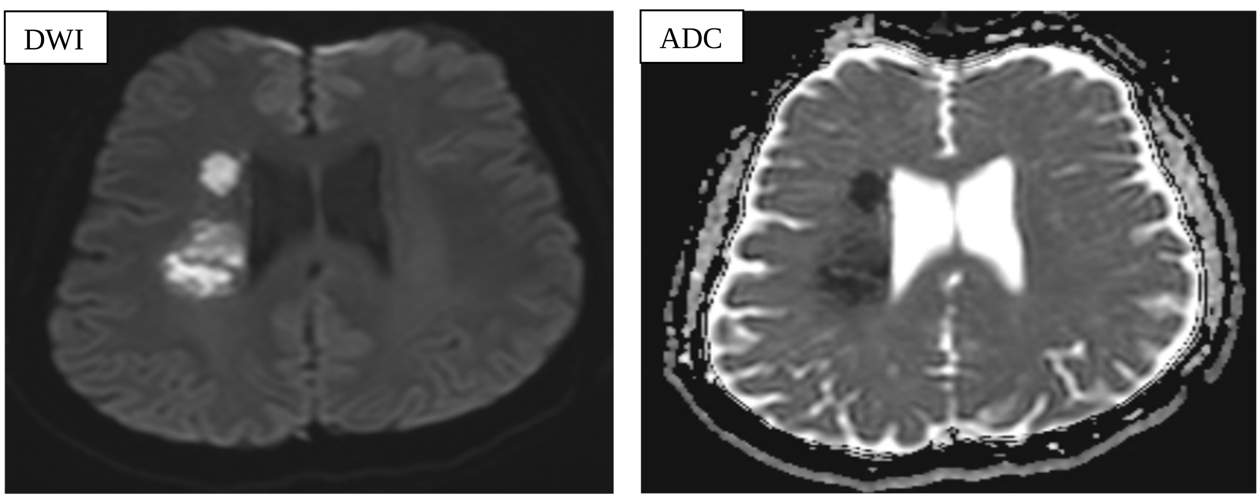


FIGURE 2. INFARCT IN RIGHT MCA TERRITORY SHOWING DIFFUSION RESTRICTION

CASE 3

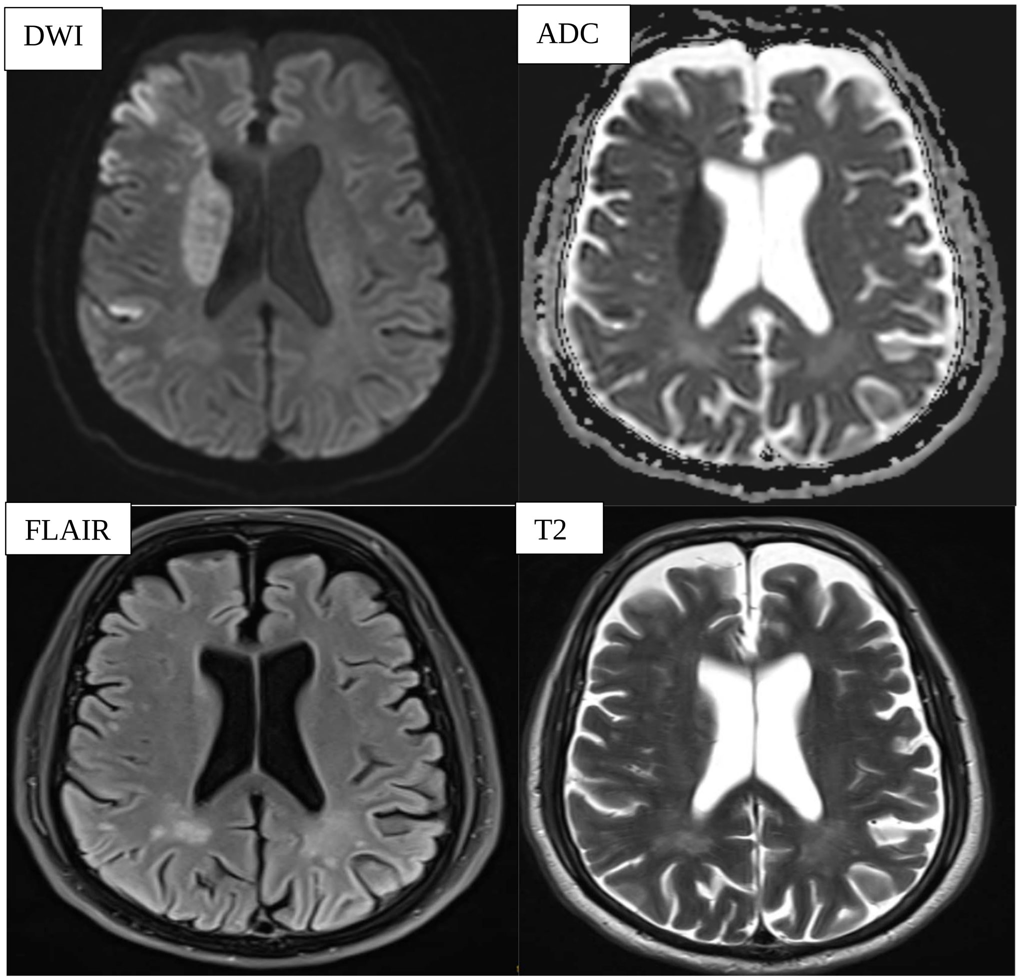


FIGURE 3. INFARCT IN RIGHT MCA TERRITORY SHOWING DIFFUSION RESTRICTION; HOWEVER FLAIR AND T2 WEIGHTED IMAGING SHOWING NO ALTERED SIGNAL INTENSITIY – ‘DWI-FLAIR MISMATCH’

CASE4

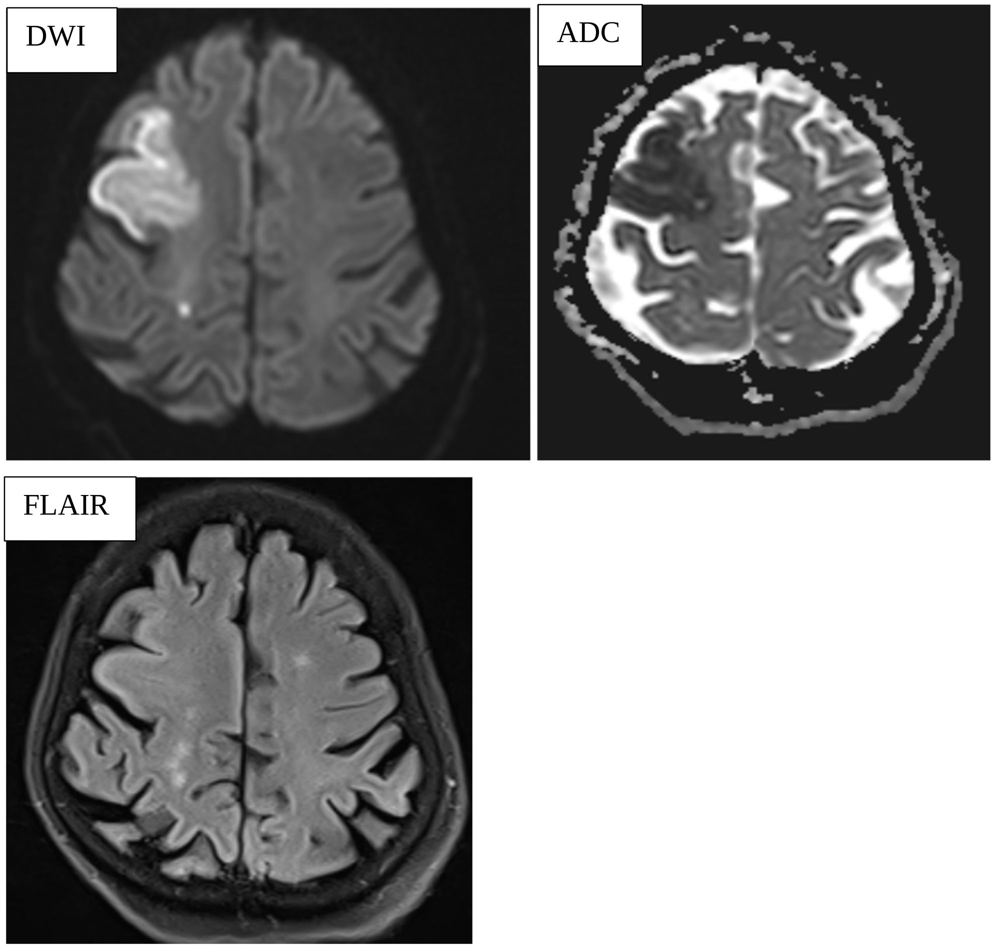


FIGURE 4. INFARCT IN RIGHT MCA TERRITORY SHOWING DIFFUSION RESTRICTION; HOWEVER FLAIR IMAGING SHOWING NO ALTERED SIGNAL INTENSITIY – 'DWI-FLAIR MISMATCH'

CASE 5

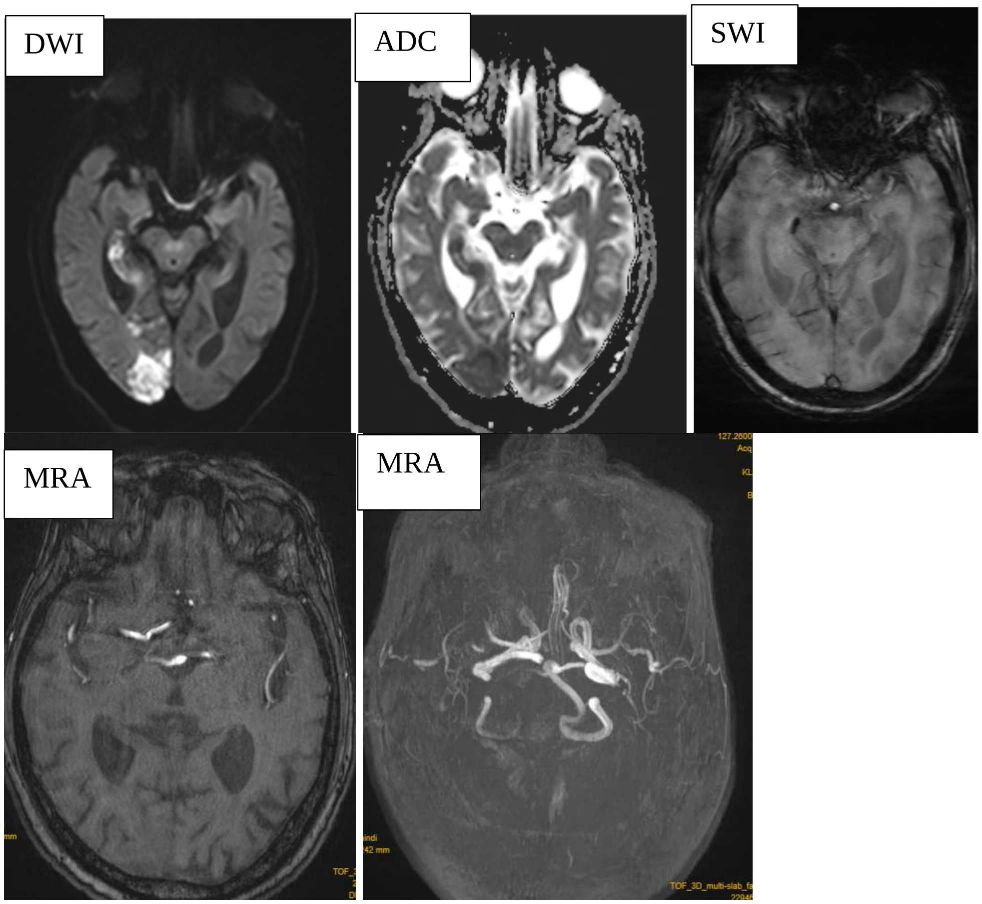


FIGURE 5. INFARCT IN RIGHT PCA TERRITORY SHOWING DIFFUSION RESTRICTION; SUSCEPTIBILITY WEIGHTED IMAGING SHOWING HYPOINTENSITY IN THE RIGHT PCA – 'SUSCEPTIBILITY VESSEL SIGN' NON VISUALISATION OF P2 SEGMENT OF RIGHT PCA ON TIME OF FLIGHT MR ANGIOGRAPHY

CASE 6

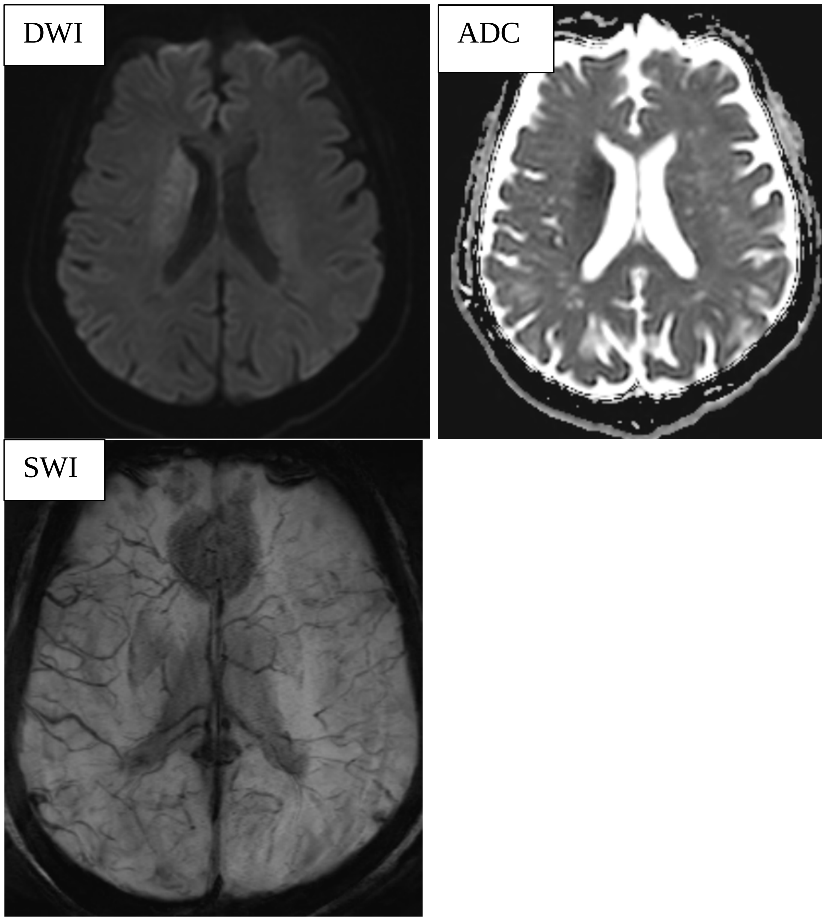


FIGURE 6. INFARCT IN THE RIGHT MCA TERRITORY; SUSCEPTIBILITY WEIGHTED IMAGING SHOWING PROMINENT HYPOINTENSE VESSEL SIGN

CASE 7

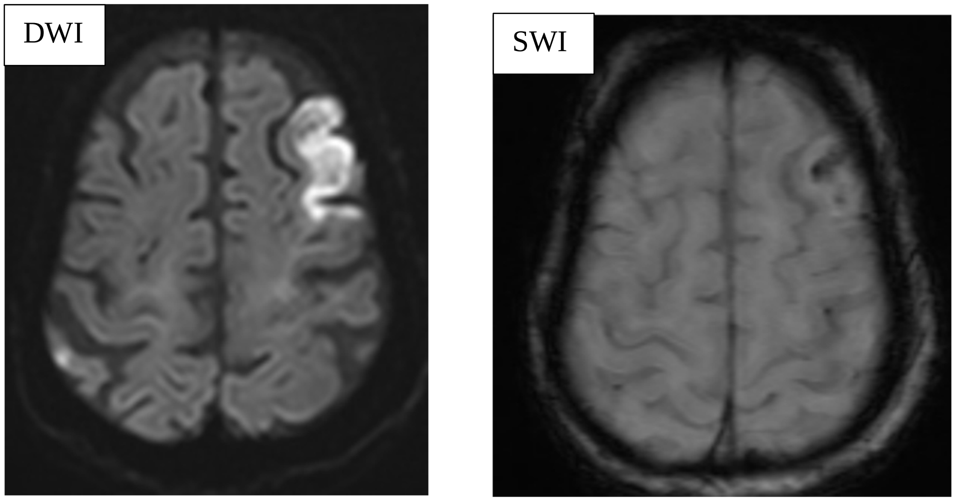


FIGURE 7. SUSCEPTIBILITY WEIGHTED IMAGING SHOWING MICROHEMORRHAGES IN THE INFARCT IN LEFT MCA TERRITORY

CASE 8

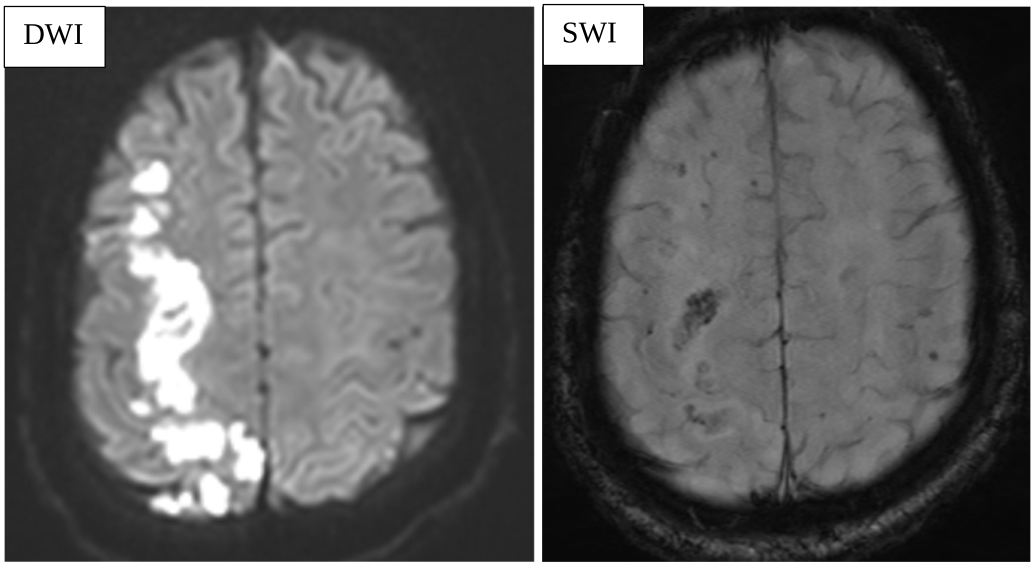


FIGURE 8. SUSCEPTIBILITY WEIGHTED IMAGING SHOWING MICROHEMORRHAGES IN THE INFARCT IN RIGHT MCA TERRITORY

CASE 9

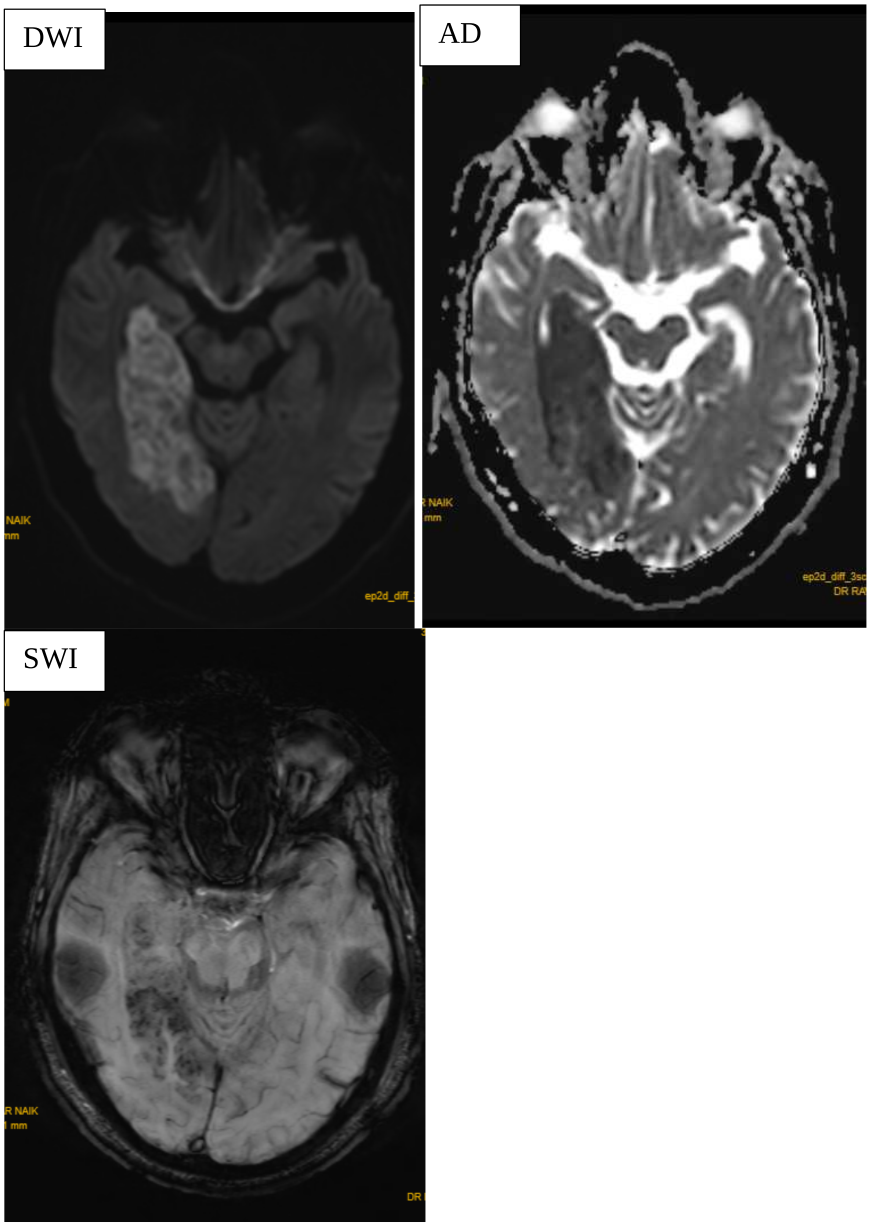


FIGURE 9. DIFFUSION WEIGHTED IMAGING SHOWING RIGHT PCA TERRITORY INFARCT ; SUSCEPTIBILITY WEIGHTED IMAGING SHOWING BLOOMING IN THE SAME TERRITORY SUGGESTIVE OF HEMORAGIC TRANSFORMATION

ANNEXURE–V–KEY TO MASTERCHART

MALE	**M**
FEMALE	**F**

DIABETES MELLITUS	**DM**
HYPERTENSION	**HTN**
DYSLIPIDAEMIA	**DL**
SMOKING	**SM**
ALCOHOL CONSUMPTION	**ALC**

DWI-SWI MISMATCH PRESENT	**Y**
DWI - SWI MISMATCH ABSENT	**N**

HEMORRHAGFIC FOCI PRESENT	**Y**
HEMORRHAGFIC FOCI ABSENT	**N**

VENOUS CONGESTION PRESENT	**Y**
VENOUS CONGESTION ABSENT	**N**

RIGHT	**RT**
LEFT	**LT**
BILATERAL	**BL**

ANTERIOR CEREBRAL ARTERY	**ACA**
MIDDLE CEREBRAL ARTERY	**MCA**
POSTERIOR CEREBRAL ARTERY	**PCA**
SUPERIOR CEREBELLAR ARTERY	**SCA**
POSTERIOR INFERIOR CEREBELLAR ARTERY	**PICA**

S NO.	AGE	GENDER	RISK FACTORS	STROKE TERRITORY	FLAIR (cc)	DWI (cc)	TIME SINCE ONSET (HRS)	HYPOINTENSITY SIGN	TOF	DWI-SWI MISMATCH	HEMORRHAGIC FOCI	VENOUS CONGESTION
1	62	F	DM, HTN,SM	LT MCA	88	72	9	DISTAL M1. PROXIMAL M2	DISTAL M1.M2 AND M3	Y	Y	Y
2	56	F	DM, HTN,SM	LT PCA	24	26	5	LT P2	LT ICA ORIGIN TO BIFURCATION	Y	N	Y
3	55	M	HTN,SM	LT MCA	8	15	6	NO	NORMAL	N	N	N
4	85	M	HTN, DL,SM	RT HEMIPONS	0	15	3	PROXIMAL BASILAR ARTERY	RT VERTEBRAL, BASILAR	Y	N	Y
5	81	M	HTN, SM	LT MCA	13	10	10	NO	NORMAL	N	N	N
6	79	M	DM, HTN,SM	RT ACA	33	35	10	A1 SEGMENT RT ACA	A1 SEGMENT RT ACA	N	Y	N
7	71	M	HTN,SM	RT HEMIPONS	4	10	4	NO	NORMAL	N	N	N
8	67	M	DM, HTN,SM	RT MCA	30	24	10	DISTAL M1.M2 SEGMENT	DISTAL M1.M2	N	N	N
9	60	F	DM, HTN,SM	RT MCA	0	20	5	DISTAL M1.M2 SEGMENT	DISTAL M1.M2	N	N	N
10	72	F	DM, HTN,SM	RT MCA	10	28	6	M2	M2	Y	Y	Y
11	61	F	DM,SM,AC	RT PICA & BL PCA	22	15	12	BL PICA	BL PICA	N	N	N
12	60	M	DM, HTN,SM	LT ACA.MCA	37	28	11	LT M1.M2	LT M1.M2.M3	N	Y	N
13	70	M	DM, HTN,SM,AC	RT PCA	22	17		NO	CORTICAL BRANCHES OF RT PCA	Y	N	Y
14	80	M	DM, HTN, DL, SM,AC	RT PCA. RT SCA.LT PCA	38	30	10	RT P2	RT P2 AND P3	N	Y	N
15	53	F	DM,SM,AC	RT MCA. RT PCA	24	17	10	DISTAL RT P2	DISTAL MCA	N	N	N
16	64	M	SM	LT MCA	7	8	6	NO	NORMAL	N	N	N
17	73	F	DM, HTN,SM,AC	RT MCA.RT ACA.LT PCA	45	34	11	DISTAL M1.PROXIMAL M2 OF RT MCA	DISTAL M1.M2.SUPERIOR BRANCH OF RT MCA	N	Y	N
18	59	M	DL,SM	LT MCA	10	8	7	NO	NORMAL	N	N	N
19	62	M	DL, SM,AC	RT PCA. RT MCA	28	21	8	RT P2.P3 . SUPERIOR BRANCH OF RT MCA	RT P2.P3.SUPERIOR BRANCH OF RT MCA	Y	N	Y
20	67	M	SM	RT MCA	38	36	8	RT M3	NORMAL	N	Y	N
21	65	M	HTN, DL,SM	RT MCA	31	22	7	RT DISTAL M1	RT DISTAL M1 M2	N	N	N
22	60	M	HTN,DL,SM,AC	RT PICA . RTSCA. LT SCA	42	30	7	NO	NORMAL	N	Y	N
23	54	M	HTN,SM	RT MCA	53	55	6	NO	NORMAL	N	Y	N
24	73	F	DM	LT THALAMUS	5	8	3	DISTAL LT P1 & P2	LT P1, P2,P3	N	N	N
25	67	M	HTN, DL, AC	RT SCA	24	35	4	RT SCA	RT SCA	N	N	N
26	55	M	HTN, DL, SM	RT HEMISPHERE	45	88	3	RT MCA. RT PCA	RT ICA, ACA, MCA, PCA	N	N	N
27	83	M	DM, HTN	LT ACA. LT MCA. RT PCA	50	53	6	RT P2	LT P1, LT PETROUS, CAVERNOUS AND SUPRACLINOID	N	Y	N
28	63	M	SM	LT PCA. MCA(SMALL)	5	8	4	RT MCA	SUPERIOR BRANCH OF RT MCA	Y	N	Y
29	80	M	HTN,SM,AC	RT PCA. BASILAR TOP	42	30	7	M1	M1	Y	Y	Y
30	39	M	DL,SM,AC	RT MCA	30	28	7	RT M2	RT M1.M2 OF MCA	Y	Y	Y
31	69	M	AC	RT THALAMUS	8	10	4	NO	NORMAL	N	N	N
32	35	F	HTN, DL,AC	RT MCA	28	25	8	DISTAL M1, PROXIMAL M2 OF RT MCA	RT M1.M2 OF MCA	N	Y	N
33	57	M		LT HEMIPONS	3	6	5	NO	NORMAL	N	N	N
34	63	M	DL	RT MCA	10	14	5	SUPERIOR BRANCH OF RT MCA	SUPERIOR BRANCH OF RT MCA	N	N	N
35	78	M	DM, HTN	LT PCA	29	25	9	LT P1 P2	LT P1 P2	N	Y	N
36	53	M		LT MCA	11	8	8	NO	NORMAL	N	N	N
37	49	M	DL,AC	RT MCA	6	10	5	RT DISTAL M1 AND PROXIMAL M2	RT M1 M2 OF MCA	N	N	N
38	76	M	SM	RT MCA	3	7	5	RT MCA	RT MCA	Y	N	Y
39	25	F		BL ACA-MCA AND MCA-PCA	55	68	4	BL ACA	NORMAL	N	N	N
40	25	M		LT ACA	2	8	4	NO	NORMAL	N	N	N
41	62	F	DM	LT MCA	88	72	9	DISTAL M1. PROXIMAL M2	DISTAL M1.M2 AND M3	Y	Y	Y
42	56	F	DM	LT PCA	24	26	5	LT P2	LT ICA ORIGIN TO BIFURCATION	Y	N	Y
43	55	M		LT MCA	8	15	6	NO	NORMAL	N	N	N
44	85	M	DL	RT HEMIPONS	0	15	3	PROXIMAL BASILAR ARTERY	RT VERTEBRAL, BASILAR	N	N	N
45	81	M	SM	LT MCA	13	10	10	NO	NORMAL	N	N	N
46	79	M	DM	RT ACA	33	24	10	A1 SEGMENT RT ACA	A1 SEGMENT RT ACA	N	N	N
47	71	M		RT HEMIPONS	4	10	4	NO	NORMAL	N	N	N
48	67	M	DM	RT MCA	30	24	10	DISTAL M1.M2	DISTAL M1.M2	N	N	N
49	60	F	DM	RT MCA	0	20	5	DISTAL M1.M2 SEGMENT	DISTAL M1.M2	N	N	N
50	72	F	DM, HTN	RT MCA	10	28	6	M2	M2	Y	Y	Y
51	61	F	DM,AC	RT PICA & BL PCA	22	15	12	BL PICA	BL PICA	N	N	N
52	60	M	DM, HTN	LT ACA.MCA	37	28	11	LT M1.M2	LT M1.M2.M3	N	Y	N
53	70	M	DM, HTN,AC	RT PCA	22	17	9	NO	CORTICAL BRANCHES OF RT PCA	N	N	N
54	80	M	HTN, DL,SM,AC	RT PCA. RT SCA.LT PCA	38	30	10	RT P2	RT P2 AND P3	N	Y	N
55	53	F	DM,AC	RT MCA. RT PCA	24	17	10	DISTAL RT P2	DISTAL MCA	N	N	N
56	64	M		LT MCA	10	8	6	NO	NORMAL	N	N	N

57	73	F	DM, HTN,AC	RT MCA.RT ACA.LT PCA	45	34	11	DISTAL M1.PROXIMAL M2 OF RT MCA	DISTAL M1.M2.SUPERIOR BRANCH OF RT MCA	N	Y	N
58	59	M		LT MCA	10	8	7	NO	NORMAL	N	N	N
59	62	M	DL,SM,AC	RT PCA. RT MCA	28	21	8	RT P2.P3 . SUPERIOR BRANCH OF RT MCA	RT P2.P3.SUPERIOR BRANCH OF RT MCA	Y	N	Y
60	67	M		RT MCA	35	36	6	RT M3	NORMAL	N	Y	N
61	65	M	HTN, DL	RT MCA	31	22	7	RT DISTAL M1 M2	RT DISTAL M1 M2	N	N	N
62	60	M	HTN,AC	RT PICA . RTSCA. LT SCA	42	30	7	NO	NORMAL	N	Y	N
63	54	M	HTN	RT MCA	52	55	6	NO	NORMAL	N	Y	N
64	73	F	DM	LT THALAMUS	5	8	3	DISTAL LT P1 & P2	LT P1, P2,P3	N	N	N
65	67	M	HTN, DL, AC	RT SCA	29	35	4	RT SCA	RT SCA	N	N	N
66	55	M	HTN, DL,SM	RT HEMISPHERE	20	90	3	RT MCA. RT PCA	RT ICA, ACA, MCA, PCA	N	N	N
67	83	M	DM, HTN	LT ACA. LT MCA. RT PCA	51	53	6	RT P2	LT P1, LT PETROUS, CAVERNOUS AND SUPRACLINOID	N	Y	N
68	63	M	SM	LT PCA. MCA(SMALL)	5	8	4	M2	M2	Y	N	Y
69	80	M	HTN,SM,AC	RT PCA. BASILAR TOP	42	30	7	NO	NORMAL	Y	Y	Y
70	39	M	DL,SM,AC	RT MCA	26	28	6	RT M2	RT M1.M2 OF MCA	Y	Y	Y
71	69	M	AC	RT THALAMUS	6	10	4	NO	NORMAL	N	N	N
72	35	F	HTN, DL,AC	RT MCA	17	25	4	DISTAL M1, PROXIMAL M2 OF RT MCA	RT M1.M2 OF MCA	N	N	N
73	57	M		LT HEMIPONS	3	6	5	NO	NORMAL	N	N	N
74	63	M	DL	RT MCA	10	14	5	SUPERIOR BRANCH OF RT MCA	SUPERIOR BRANCH OF RT MCA	N	N	N
75	78	M	DM, HTN	LT PCA	29	25	9	NO	LT P1 P2	N	Y	N
76	53	M		LT MCA	11	8	8	NO	NORMAL	N	N	N
77	59	M	DL	RT MCA	7	10	5	RT DISTAL M1 AND PROXIMAL M2	RT M1 M2 OF MCA	N	N	N
78	76	M	SM	RT MCA	2	7	5	NO	NORMAL	Y	N	Y
79	25	F		BL ACA-MCA AND MCA-PCA	49	68	4	BL ACA	NORMAL	N	N	N
80	25	M		LT ACA	4	8	4	NO	NORMAL	N	N	N
81	62	F	DM	LT MCA	88	72	9	DISTAL M1. PROXIMAL M2	DISTAL M1.M2 AND M3	Y	Y	Y
82	56	F	DM	LT PCA	24	26	5	LT P2	LT ICA ORIGIN TO BIFURCATION	Y	N	Y
83	55	M		LT MCA	8	15	6	NO	NORMAL	N	N	N
84	85	M	DL	RT HEMIPONS	0	15	3	PROXIMAL BASILAR ARTERY	RT VERTEBRAL, BASILAR	N	N	N
85	81	M	SM	LT MCA	13	10	10	NO	NORMAL	N	N	N
86	79	M	DM	RT ACA	33	24	10	NO	A1 SEGMENT RT ACA	N	N	N
87	71	M		RT HEMIPONS	4	10	4	NO	NORMAL	N	N	N
88	67	M	DM	RT MCA	30	24	10	DISTAL M1.M2	DISTAL M1.M2	N	N	N
89	60	F	DM	RT MCA	0	20	5	DISTAL M1.M2 SEGMENT	DISTAL M1.M2	N	N	N
90	72	F	DM, HTN	RT MCA	10	28	6	M2	M2	Y	Y	Y
91	61	F	DM,AC	RT PICA & BL PCA	22	15	12	BL PICA	BL PICA	N	N	N
92	60	M	DM, HTN	LT ACA.MCA	37	28	11	LT M1.M2.M3	LT M1.M2.M3	N	Y	N
93	70	M	DM, HTN,AC	RT PCA	22	17	9	NO	CORTICAL BRANCHES OF RT PCA	N	N	N
94	80	M	HTN, DL,SM,AC	RT PCA. RT SCA.LT PCA	38	30	10	RT P2 AND P3	RT P2 AND P3	N	Y	N
95	53	F	DM,AC	RT MCA. RT PCA	24	17	10	DISTAL RT P2	DISTAL MCA	N	N	N
96	64	M		LT MCA	7	8	6	NO	NORMAL	N	N	N
97	73	F	DM, HTN,AC	RT MCA.RT ACA.LT PCA	45	34	11	DISTAL M1.PROXIMAL M2 OF RT MCA	DISTAL M1.M2.SUPERIOR BRANCH OF RT MCA	N	Y	N
98	59	M		LT MCA	10	8	7	NO	NORMAL	N	N	N
99	62	M	DL,SM,AC	RT PCA. RT MCA	28	21	8	RT P2.P3 . SUPERIOR BRANCH OF RT MCA	RT P2.P3.SUPERIOR BRANCH OF RT MCA	Y	N	Y
100	67	M		RT MCA	33	36	6	RT M3	NORMAL	N	Y	N
101	65	M	HTN, DL	RT MCA	31	22	7	NO	RT DISTAL M1 M2	N	N	N
102	60	F	HTN,AC	RT PICA . RTSCA. LT SCA	42	30	7	NO	NORMAL	N	Y	N
103	54	F	HTN	RT MCA	51	55	6	NO	NORMAL	N	Y	N
104	73	F	DM	LT THALAMUS	5	8	3	DISTAL LT P1 & P2	LT P1, P2,P3	N	N	N
105	67	F	HTN, DL, AC	RT SCA	30	35	4	RT SCA	RT SCA	N	N	N
106	55	M	HTN, DL,SM	RT HEMISPHERE	16	90	3	RT MCA. RT PCA	RT ICA, ACA, MCA, PCA	N	N	N
107	83	M	DM, HTN	LT ACA. LT MCA. RT PCA	49	53	6	RT P2	LT P1, LT PETROUS, CAVERNOUS AND SUPRACLINOID	N	Y	N
108	63	F	SM	LT PCA. MCA(SMALL)	3	8	4	M3	M3	Y	N	Y
109	80	F	HTN,SM,AC	RT PCA. BASILAR TOP	42	30	7	NO	LT MCA	Y	Y	Y
110	39	M	DL,SM,AC	RT MCA	26	28	6	RT M2	RT M1.M2 OF MCA	Y	Y	Y
111	69	F	AC	RT THALAMUS	8	10	4	NO	NORMAL	N	N	N

112	35	F	HTN, DL,AC	RT MCA	20	25	4	DISTAL M1, PROXIMAL M2 OF RT MCA	RT M1.M2 OF MCA	N	N	N
113	57	F		LT HEMIPONS	8	6	5	NO	NORMAL	N	N	N
114	63	M	DL	RT MCA	10	14	5	SUPERIOR BRANCH OF RT MCA	SUPERIOR BRANCH OF RT MCA	N	N	N
115	78	F	DM, HTN	LT PCA	29	25	9	NO	LT P1 P2	N	Y	N
116	53	F		LT MCA	11	8	8	NO	NORMAL	N	N	N
117	64	M	DL	RT MCA	8	10	5	RT DISTAL M1 AND PROXIMAL M2	RT M1 M2 OF MCA	N	N	N
118	76	F	SM	RT MCA	5	7	5	NO	NORMAL	Y	N	Y
119	25	F		BL ACA-MCA AND MCA-PCA	41	68	4	BL ACA	NORMAL	N	N	N
120	66	F		LT ACA	6	8	4	NO	NORMAL	N	N	N
TOTAL												