"CLINICAL APPLICATIONS OF SUSCEPTIBILITY-WEIGHTED MAGNETIC RESONANCE IMAGING AMONG PATIENTS WITH NEUROGICAL DISORDERS AT 3T MRI– A HOSPITAL BASED CROSS -SECTIONAL STUDY"

BY

REG NO. BS0120004

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CLINICAL APPLICATIONS OF SUSCEPTIBILITY-WEIGHTED MAGNETIC RESONANCE IMAGING AMONG PATIENTS WITH NEUROGICAL DISORDERS AT 3T MRI – A HOSPITAL BASED CROSS SECTIONAL STUDY"**, is ethical and justifiable.

The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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LIST OF ABBREVIATIONS

SWI	Susceptibility weighted imaging
MRI	Magnetic resonance imaging
GRE	Gradient echo pulse sequence
ICH	Intracranial hemorrhage
SAH	Subarachnoid hemorrhage
DIR	Double inversion recovery
CVT	Cerebral venous thrombosis
ICP	Intracranial pressure
MRV	Magnetic resonance venography
ICoVT	Isolated cortical venous thrombosis
MSE	Magnetic susceptibility effect
CMB	Cerebral microhemorrhages
MV	Medullary veins
DAI	Diffuse axonal injury
MSA-P	Multisystem atrophy-predominant putamen
PKAN	Panthotenante kinase associated neurodegeneration
MMD	Moya-Moya disease
SWS	Sturge Weber Syndrome
SDGM	Subcortical deep grey matter
DVA	Developmental venous anomaly
BOLD	Blood oxygen level dependent imaging

ABSTRACT

BACKGROUND & OBJECTIVES: Susceptibility-weighted imaging (SWI) images are generated from two-dimensional Gradient echo pulse sequence (GRE) to three-dimensional sequences which allows thinner slices and smaller voxels with increased spatial resolution. SWI is sensitive to substances that distort the local magnetic field (such as calcium and iron), in which the phase images can differentiate. In SWI, magnitude and phase images are independently processed and can also be combined for diagnostic use.

SWI is helpful in the setting of trauma and acute neurologic presentations such as stroke & cerebral venous thrombosis, intraparenchymal hemorrhage, neurological infections, aneurysms, vascular malformations, developmental venous anomaly, cerebral microbleeds, intracranial calcifications, neurodegenerative diseases and brain tumors.

The main objective of this study is to understand the uses of SWI as an addition to the conventional MRI sequences in concluding the final diagnosis.

MATERIALS AND METHODS: One-year cross-sectional study was done in the department of radiodiagnosis at the KLES Dr. Prabhakar Kore Hospital and MRC, Belagavi. Total patients included in the study were 143. These patients with any neurological symptoms were considered.

These patients were subjected to department of radiology to undergo MRI brain in 3T Siemens MRI machine. SWI sequence was added to the routine MRI protocol in all the patients undergoing MRI brain and relevant findings were noted.

Any hypointense blooming on SWI followed by hypointense/ hyperintense blooming in the same region on Phase sequences was observed to understand the presence of bleed/ calcium.

Other conventional MR sequences were also observed to come to the final diagnosis and also to differentiate between bleed, calcium and hemosiderin.

RESULTS: The mean age ranged from 46.58 +/- 22.07 years. The minimum age was one day and the maximum age was 85. Among the study population, 102 were males and 41 were females. Most common symptom in our study was headache followed by seizures and maximum number of patients had symptoms for less than one month.

In this study 78 patients had non-vascular pathology and 65 patients had vascular pathology in the scan. Among the study group, 82 cases (57.34 %) had hemorrhage within the brain parenchyma, 35 cases (24.48 %) had calcium and 26 cases (18.18 %) had hemosiderin.

The most common diagnosis in this study was brain tumors i.e; 31 cases (21.68 %) with hemorrhage (17 cases) / calcium (12 cases) / hemosiderin (2 cases) within the lesion. Few other important diagnosis were made during this study being 26 cases of iron deposition in brain, 15 cases of neuro infections, 13 cases of cerebral venous thrombosis, 10 cases of hemorrhagic transformation of infarct, 10 cases of intraparenchymal hemorrhage, 7 cases of neurodegenerative disorders, 6 cases of hemorrhage due to trauma, 6 cases of vascular malformations, 5 cases of hypertensive microangiopathy with microbleeds, 4 cases of aneurysm, 2 cases of developmental venous anomaly, 3 cases of neurocutaneous syndromes, 2 cases of moya moya, 2 cases of acute necrotizing hemorrhagic encephalopathy, 2 cases of peri-callosal lipoma, one case of lentiform nucleus calcification and one case of colloid cyst.

INTERPRETATION & CONCLUSION: Compounds with substances that are diamagnetic, paramagnetic and ferromagnetic properties will interact with local magnetic field resulting in its destruction and hence altering the phase of local tissue which causes signal alteration. SWI combines GRE techniques with phase to create images that are more superior and that help in cases of resolving venous vasculature, hemorrhage, calcium and iron.

In this study we understood that SWI can be useful for diagnosing traumatic brain injury, tumors, neurodegeneration, stroke, venous thrombosis, vascular malformations, chromic hematomas, aneurysms and calcified lipomas. We have observed that SWI was useful in narrowing down the differential diagnosis in cases of brain tumors by helping to know the presence of haemorrhage, calcium or iron in the lesion. SWI was particularly useful in cases of chronic bleeds and microhaemorrhages which were overlooked on T1 and in cases where T1 & T2 sequences did not show any signal abnormality.

Thus, SWI can be useful for imaging mineralization, venous anatomy, and cerebrovascular disease. Though SWI cannot replace conventional MRI sequences, it can provide complementary information which helps in the final diagnostic outcome.

KEYWORDS: Susceptibility weighted imaging, phase images, Gradient echo pulse sequence, magnetic field, hemorrhage, calcium, iron, hemosiderin, stroke, neurodegeneration, cerebral venous thrombosis, trauma, vascular malformation.

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

Susceptibility Weighted Imaging (SWI) is a novel neuroimaging approach that has gained widespread use recently to gain differential diagnosis of several neurologic diseases. SWI gives information about tissues that has different magnetic susceptibility from their surroundings like iron, calcium, deoxygenated blood, and haemosiderin[1]. It utilizes the intrinsic differences in magnetic susceptibilities of various substances, such as deoxygenated blood, blood products, iron, and other tissues to enhance a unique contrast in Magnetic Resonance (MR). SWI images tend to be fast sequence hence it has considerably less scan time. The sequences when included in conventional MRI sequences can help in evaluation of different neurological disorders such as diffuse axonal injury (traumatic brain injury), cerebral infarction, neoplasms, neurodegenerative disorders and vascular malformations[2].

SWI is a particular sequencing and processing scheme created to boost contrast in T2*-weighted pictures. Originating in research on cerebral microbleeds (CMBs) and venous deoxyhemoglobin, its applications have since grown significantly. SWI has increased susceptibility sensitivity than the GRE images because it is based on high resolution, flow-compensated and long TE three dimentional GRE technique which contains filtered phase information[3]. SWI increases the effect of the local magnetic field variations caused by tissues containing blood, iron, calcium and deoxyhemoglobin and these changes that occur in the magnetic field will result in signal loss[4].

Magnetic resonance imaging recently made some helpful advancements, one of which being susceptibility-weighted imaging (SWI). Its use gave radiologists in a variety of specialties, especially neuroradiology, useful information. SWI was able to show the presence of diamagnetic and paramagnetic materials in the brain.

As a result, this imaging method might be used for a variety of purposes in both academic and clinical neuroradiology. The practising radiologist needs to be educated on how to interpret SWI and phase pictures from both right- and left-handed MRI systems in order to diagnose and characterise a wide variety of neurological and neurodegenerative disorders affecting the brain[4,5].

Dementia and neurodegenerative diseases are becoming more common as the average lifespan of people rises. Neuroimaging is a crucial component of numerous studies that are currently being conducted to help with the early identification and treatment of dementia. Conventional neuroimaging, however, frequently falls short in terms of providing sufficient diagnostic information in individuals with neurodegenerative diseases. To help with the proper diagnosis of such illnesses in this situation, various MRI sequences are now being researched[6].

SWI combines magnitude and phase images to create contrast that is sensitive to the detection of changes in tissue susceptibility. SWI can be used to detect microhemorrhages and/or fast iron accumulation in the brain, both of which are associated with numerous neurodegenerative diseases. The microhemorrhages in the cortex of a patient with cerebral amyloid angiopathy, for instance, are most often seen in the frontal and parietal lobes and may be detected by SWI. On high-resolution SWI, on the other hand, the abnormal swallow-tail sign is very diagnostic for Parkinson's disease[6]. Additionally, SWI is a helpful sequence to identify precentral cortices with low signal intensity in individuals with amyotrophic lateral sclerosis. Accurate diagnosis of neurodegenerative illnesses requires knowledge of SWI results.

There are several uses for susceptibility weighted imaging in neuroradiology practise, and it need to be incorporated into standard protocols. It can detect micro- and macrohemorrhages, pinpoint low-flow vascular anomalies, and identify cerebral

microvasculature. It has been demonstrated to be an advantageous imaging sequence that is complimentary for the treatment of stroke patients. It helps with the grading and characterisation of cerebral malignancies as well as the assessment of patients with traumatic brain injuries. By determining the iron concentration of the brain, quantitative susceptibility mapping may be used on a variety of neurodegenerative diseases.

Susceptibility-weighted imaging is a crucial technology that allows for the precise identification of early hemorrhagic changes inside acute infarctions as well as the detection of old microbleeds, notifying the treating physician about the life-threatening side effect of anticoagulant medications[7]. Additionally, SWI can identify cerebral venous thrombosis early. SWI should thus be a standard procedure in the stroke imaging regimen.

NEED FOR THE STUDY:

A new MR sequence called susceptibility weighted imaging (SWI) analyses and makes use of the magnetic characteristics of tissues like blood, iron, and bone[8]. SWI is a 3D velocity compensated gradient echo sequence that produces magnitude, phase, or a combination of these two pictures. A special combination of SWI's high resolution 3D gradient echo, full flow compensation in all three directions, thin slices, phase image filtering, high-lighting susceptibility by phase masking, and improved data interpretation make SWI stand out. SWI is frequently employed for high-resolution venous anatomy, tissue susceptibility testing, and the diagnosis of cerebral vascular disorders[8]. SWI is highly helpful in identifying brain cancers and degenerative illnesses of the brain, detecting calcifications in a variety of clinical circumstances, and detect cerebral micro-bleeds.

To distinguish between the diamagnetic effects of calcium and the paramagnetic susceptibility effects of blood, phase images are very helpful. SWI can assess iron deposit variations of several neuro-degenerative diseases[6].

In this study, I will explore the clinical uses of SWI, the ideal parameters to employ, and the interpretation of the processed SWI data and phase images, as well as the current state of the art technology, post processing, and technical considerations.

OBJECTIVES

AIM AND OBJECTIVES OF THE STUDY:

1. To illustrate the clinical objectives of SWI as a sequence of MRI Brain in neurologic disorders.
2. To collect evidence and prove that SWI is a better approach to diagnose the neurologic disorders.

REVIEW OF LITERATURE

Definition

Susceptibility weighted imaging (SWI) is a MRI sequence that has elevated spatial 3D resolution and is completely velocity compensated. It is sensitive to substances that alter the local magnetic field, making it useful in the detection of calcium, blood products, etc. Unlike the majority of other standard sequences, SWI makes use of both the effect's size and phase. Deoxyhemoglobin, ferritin, and hemosiderin are examples of paramagnetic, diamagnetic, and ferromagnetic compounds that interact with the local magnetic field to vary its phase, which changes the signal. Other examples include bone minerals and dystrophic calcifications.

Anatomy of Cerebral Venous System –:

Deep veins, superficial veins, and dural venous sinuses make up the cerebral venous system. It is divided into: superficial and deep systems[9].

Dural sinuses and cortical veins put together form the superficial system. It is responsible for draining the blood from the cerebral cortex and superficial white matter. The Superior sagittal and Cavernous sinus drains dorsolateral portion and the anteroventral areas respectively. The transverse sinus run along the lateral boarder of tentorium cerebelli and grooves the occipital and squamous temporal bones. They terminate into sigmoid sinus just as it receives the superior petrosal sinus from the cavernous sinus. The cavernous sinus drains into the posterolateral transverse sinus and the inferolateral sigmoid sinus through the superior and inferior petrosal sinuses[9]. Superior and inferior draining veins are known as superficial cortical veins (vein of Labbe and sylvian or superficial middle cerebral veins).

The veins in the deep cerebral cortex are a part of the deeper venous network. The Rosenthal or basal vein, the medullary and subependymal veins, the sigmoid sinus, the straight sinus, the transverse sinus, a vein of Galen, and the internal cerebral veins are all included. The deep venous system collects blood from the brain's deep white matter, thalamus, and basal ganglia. The final drainage site for the contents of both venous systems is the internal jugular vein.

Figure 1: Normal major cerebral veins and Sinuses (as seen in MR Venogram)[9]

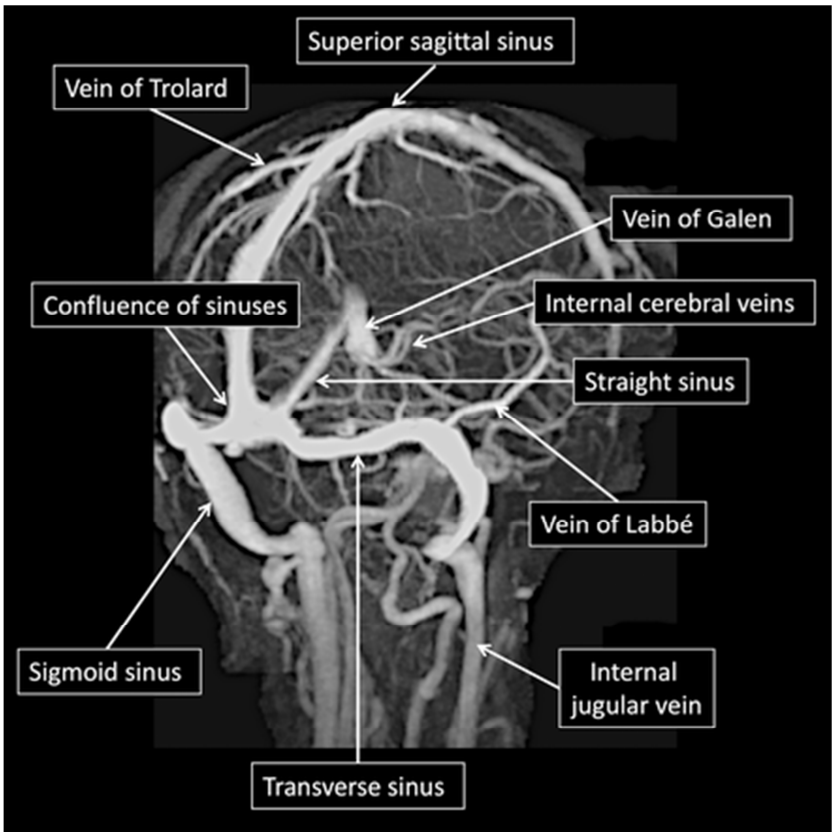


Image courtesy: Ulivi L et al[9]

Figure 2: Cortical venous vascular territories

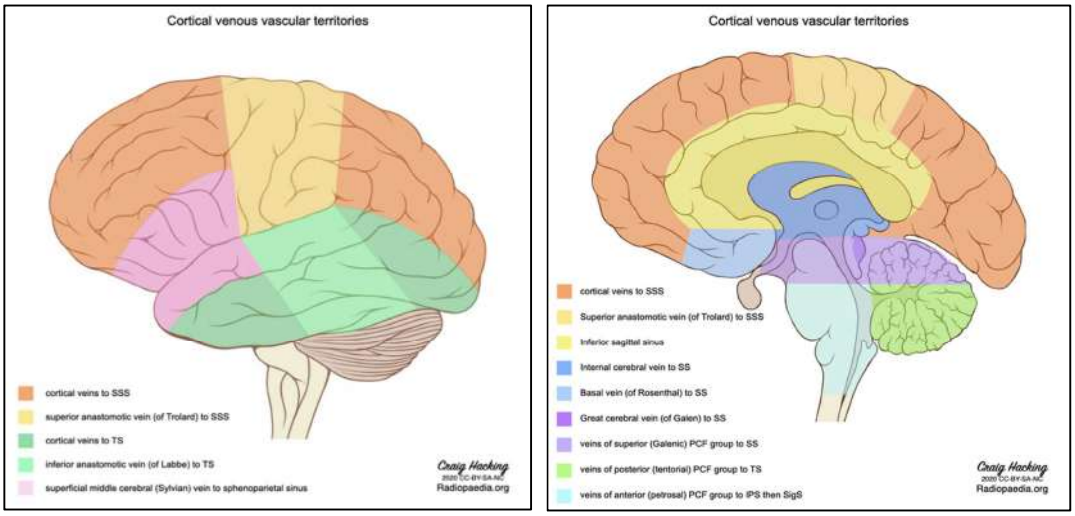
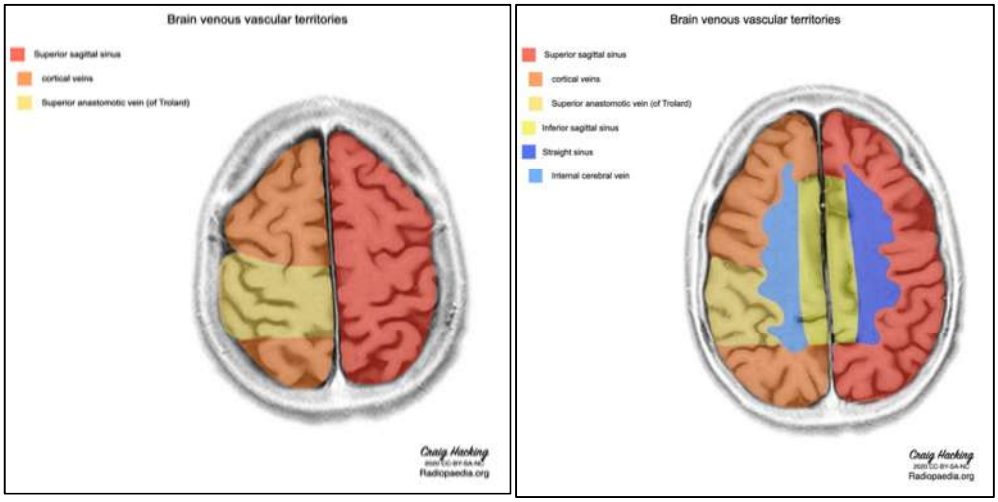


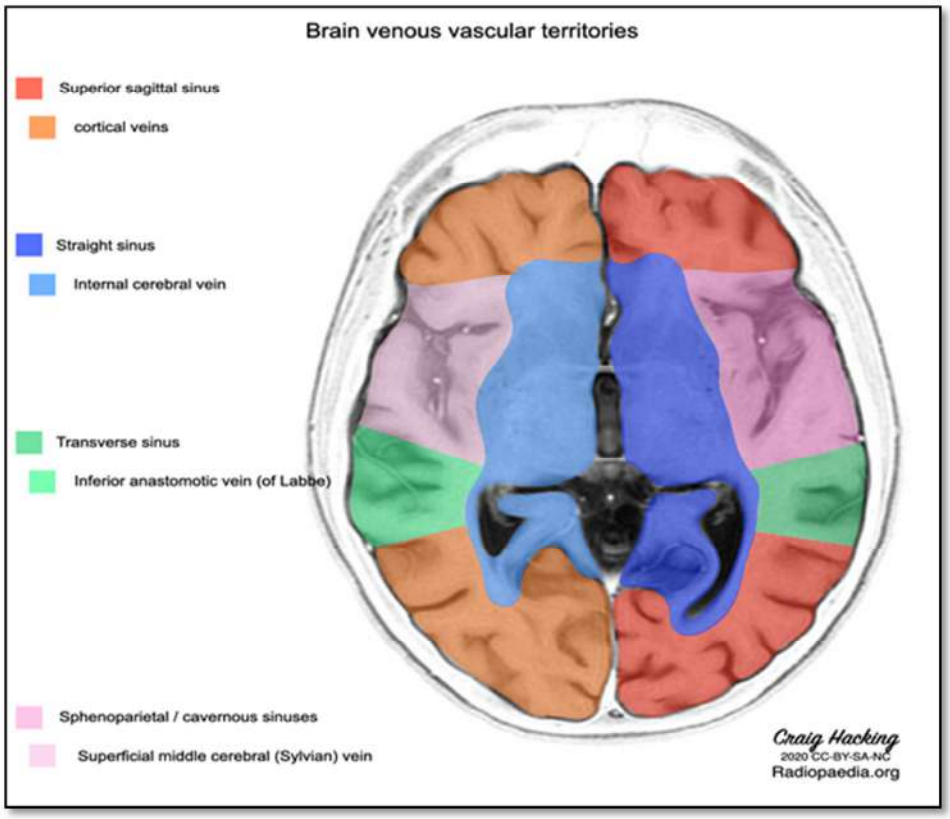
Image courtesy: https://radiopaedia.org/articles/cerebral-venous-thrombosis

A. Lateral view of the cerebral cortex

B. Medial view

Figure 3: Brain venous vascular territories.




Brain venous vascular territories
Superior sagittal sinus
cortical veins
Straight sinus
Internal cerebral vein
Transverse sinus
Inferior anastomotic vein (of Labbe)
Sphenoparietal / cavernous sinuses
Superficial middle cerebral (Sylvian) vein
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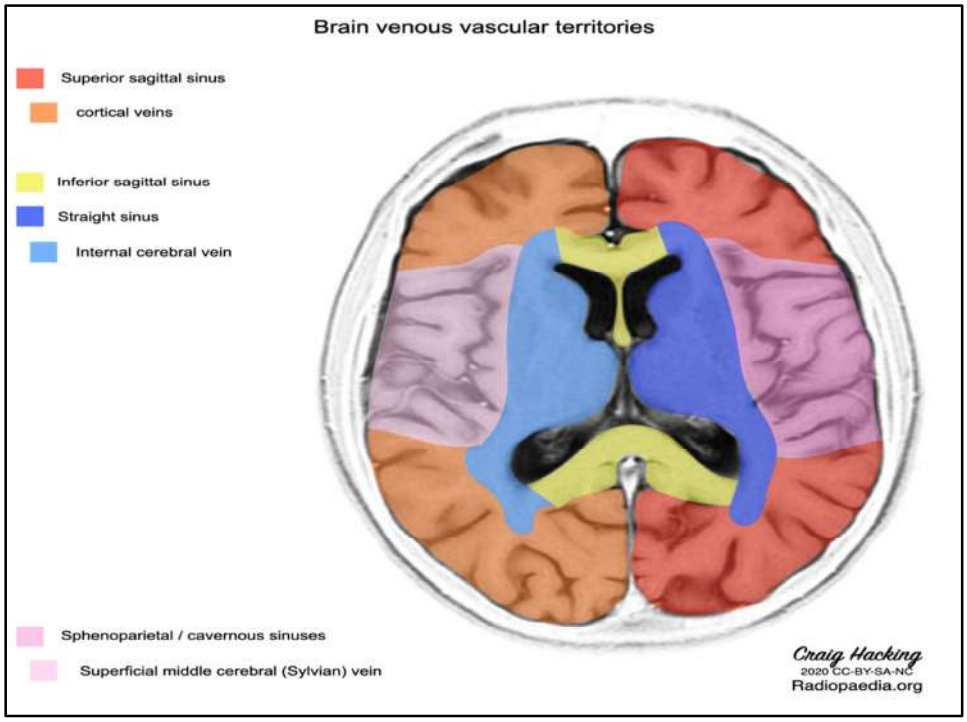
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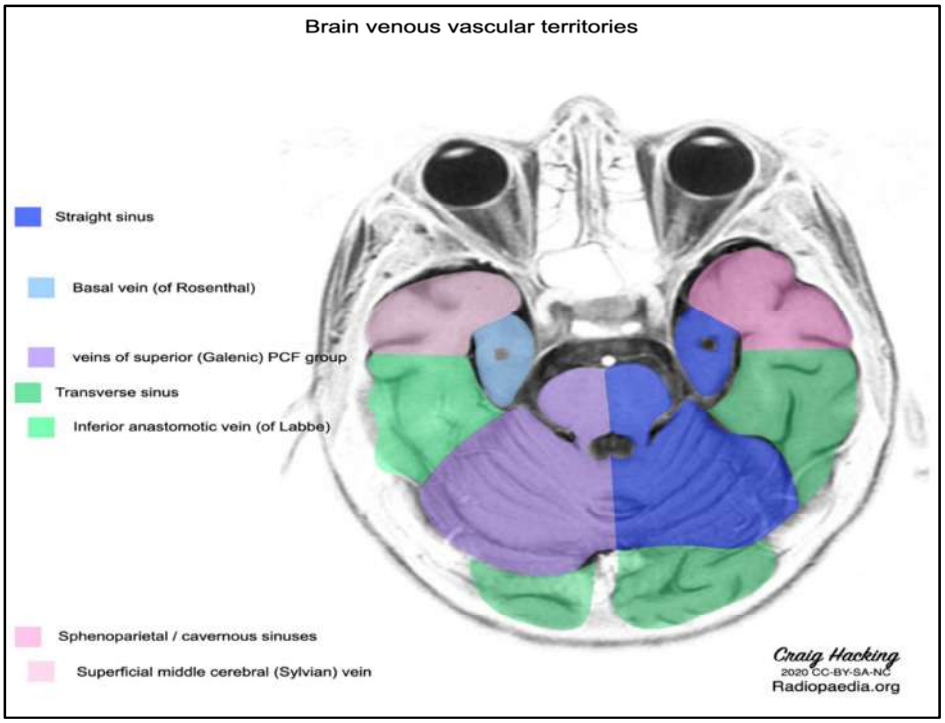
Brain venous vascular territories
Straight sinus
Basal vein (of Rosenthal)
veins of superior (Galenic) PCF group
Transverse sinus
Inferior anastomotic vein (of Labbe)
Sphenoparietal / cavernous sinuses
Superficial middle cerebral (Sylvian) vein
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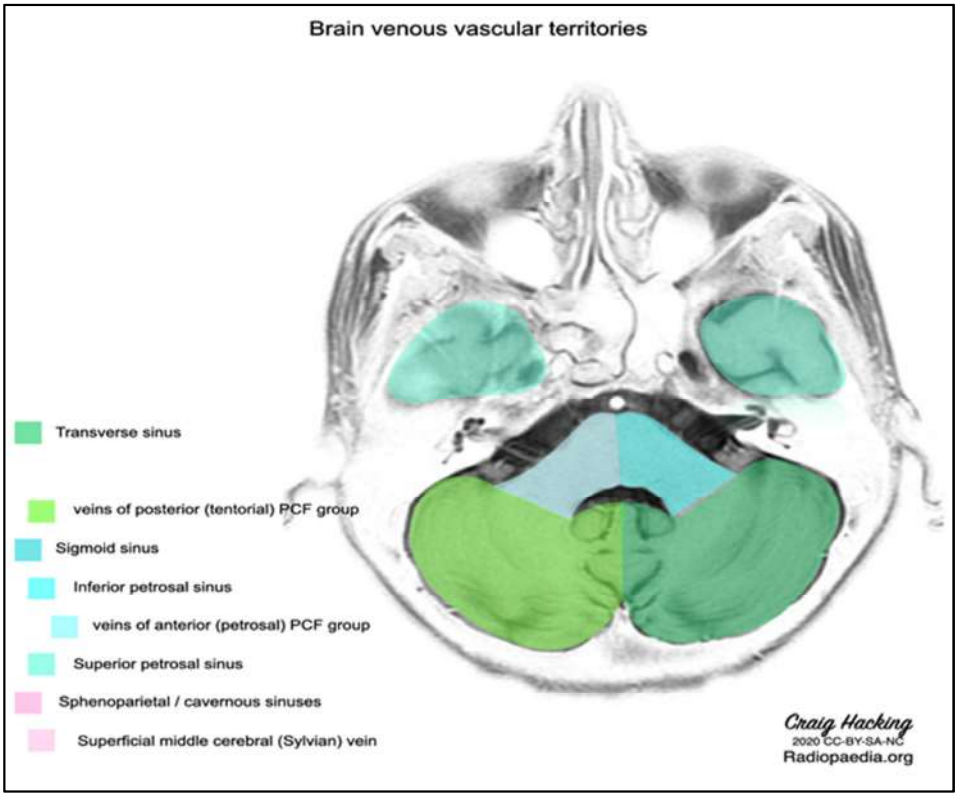
Brain venous vascular territories
Transverse sinus
veins of posterior (tentorial) PCF group
Sigmoid sinus
Inferior petrosal sinus
veins of anterior (petrosal) PCF group
Superior petrosal sinus
Sphenoparietal / cavernous sinuses
Superficial middle cerebral (Sylvian) vein
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Arterial anatomy of brain:

Internal carotid arteries (ICA) and vertebral arteries (VA) on both sides of the body provide blood to the brain. ICA along with middle and anterior cerebral arteries form the anterior circulation and vertebral along with basilar and posterior cerebral artery forms the posterior circulation[10]. Through the Circle of Willis, the anterior and posterior circulations communicate through the anterior and posterior communicating arteries. If one or more of the arteries entering the brain are damaged or dysfunctional, this circle of willishelps to enable blood circulation to the brain to continue through collaterals. However, only around a quarter of people have a full Circle of Willis[10].

Figure 4: Circle of willis

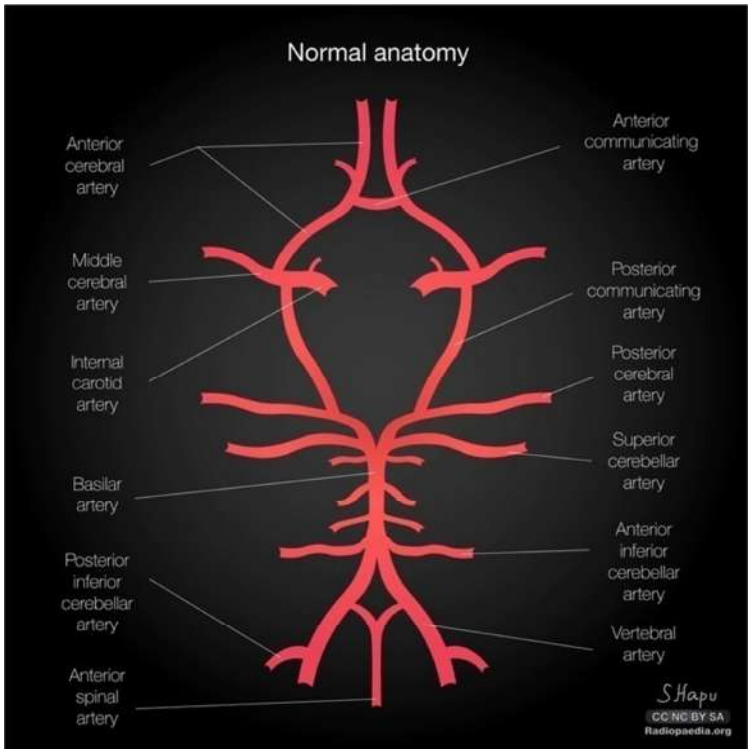


Image courtesy: https://radiopaedia.org/cases/common-variants-of-the-circle-of-willis-illustrations?lang=gb

Figure 5: Brain arterial vascular territories

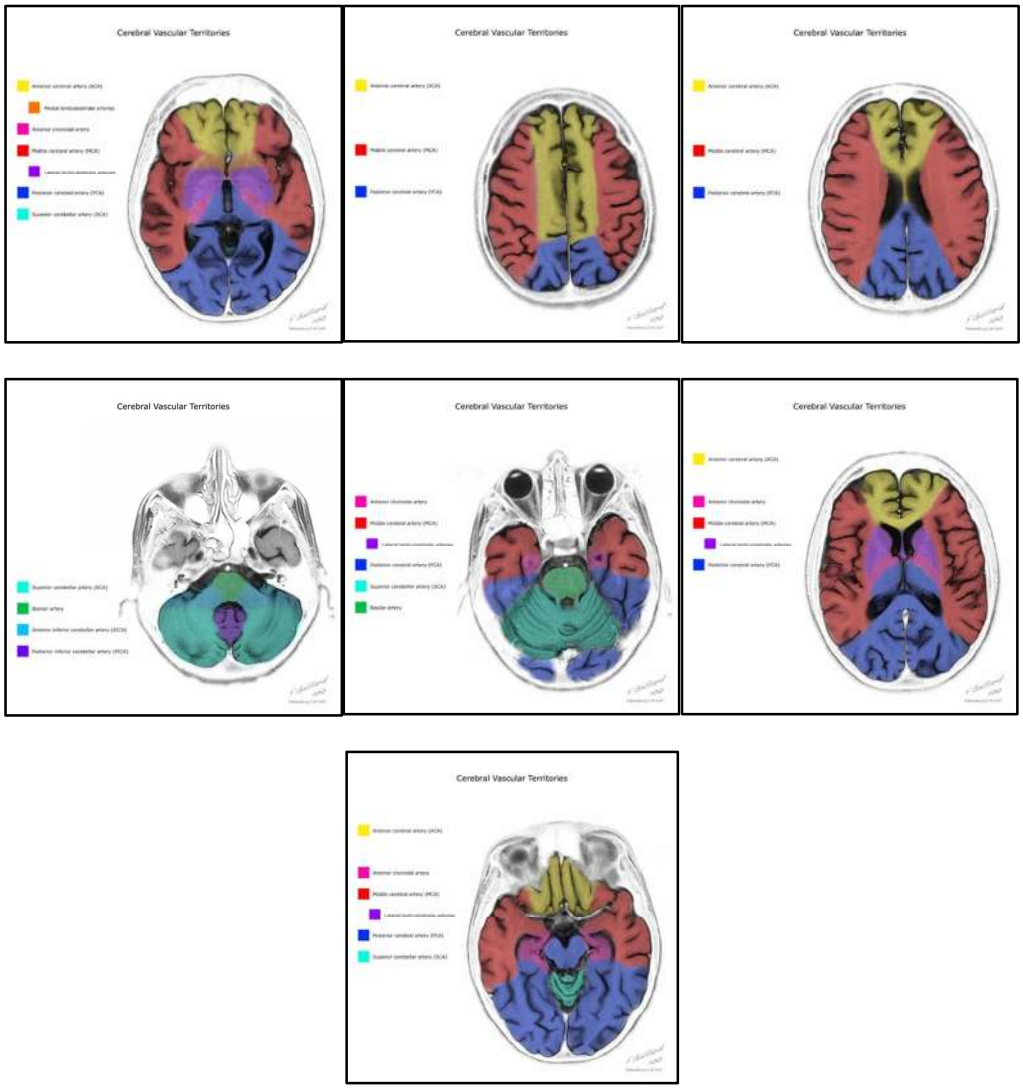


Image courtesy: https://radiopaedia.org/cases/cerebral-vascular-territories-illustration?lang=gb

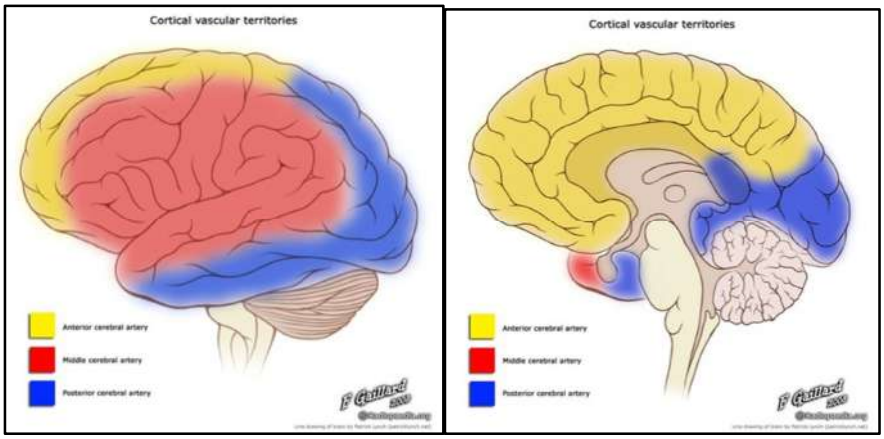


Image courtesy: https://radiopaedia.org/cases/cerebral-vascular-territories-in-the-midline-illustration?lang=gb

SWI Images -Generation and Processing

Gradient-echo (GRE) pulse sequences act as a key source for generation of SWI images. GRE sequences show high sensitivity susceptibility differences, due to its inability to refocus spins de-phased by the homogeneities in magnetic field.

Calcifications, iron, and blood products have traditionally been detected using T2*weighted GRE sequences. In order to get more finely cut voxels, SWI sequences are obtained in 3D rather than 2D mode. Three-dimensional (3D) imaging helps acquire less noise, and analogous imaging shortens imaging sessions. Within a fixed TR, SWI may produce both single and multiple echoes. SWI's ability to absorb information both individually and collectively is crucial for making diagnoses. To perform SWI imaging, a flip angle of 15–20 degrees is used in conjunction with a TR parameter interval of 25–50 ms and a TE parameter interval of 20–40 ms. In simple terms, a stronger magnetic field causes smaller flip angles[11].

A study done in the year 2009, in their review article observed that SWI enhances contrast in MRI. For the most part, phase information has been disregarded in conventional imaging, with the exception of a few niche uses in flow imaging.

Background field inhomogeneities induced by air/tissue interfaces and main magnetic field influences make phase pictures hard to understand[12]. However, these inhomogeneities also conceal important information regarding susceptibility variations across tissues. However, it has been shown that these undesired effects may be filtered out using a specialised high-pass filter, leaving just the useful data on susceptibility differences across tissues. Susceptibility-weighted pictures are the result of combining the contrast from the magnitude image with the contrast from the phase image. SWI is an innovative contrast modality that may be used in conjunction with traditional spin-density, T1-, and T2-weighted imaging techniques. SWI is especially sensitive to deoxyhaemoglobin, making it helpful in imaging haemorrhages from trauma, detecting blood products, and tracing the vascularization of tumours, as well as in high-resolution magnetic resonance venography. It has also shown promise in a variety of other iron-related contexts, such as the evaluation of MS lesion iron and the study of ageing.

SWI pictures are created by combining magnitude and phase images, establishing the various effects of calcification and bleeding on susceptibility. Phase delay in the MR signal is caused by the diamagnetic characteristics of calcium depositions. The paramagnetic properties of tissues containing ferritin or hemosiderin cause phase progression. On phase pictures, calcifications and haemorrhages have opposite signal intensities due to the various phase shifts in the voxels. On the phase pictures of SWI, calcification looks hypointense whereas haemorrhage appears hyperintense[3].

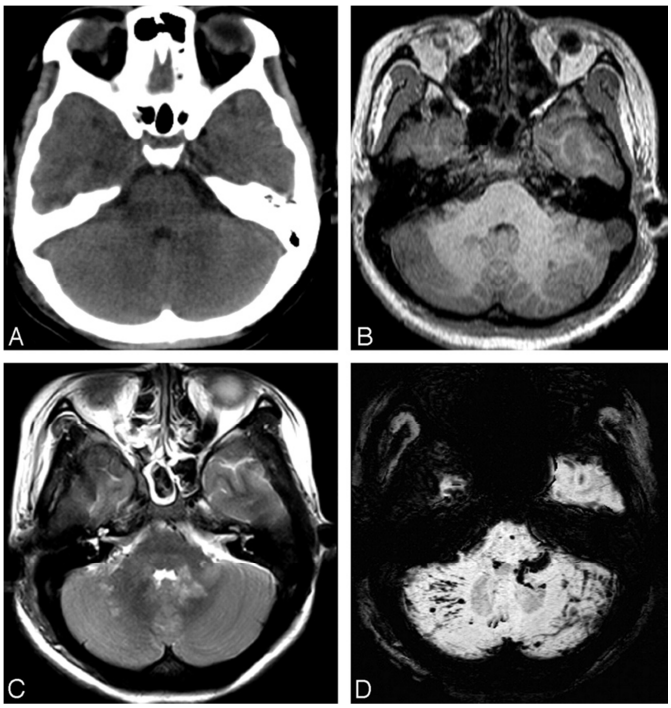
SWI for detection of Haemorrhage:

A novel magnetic resonance approach called SWI can take advantage of the variances in susceptibility of different tissues. The SWI magnitude pictures of intracranial haemorrhage (ICH) have a black blossoming appearance[13]. Hemorrhagic phase SWI images show four types of bleeding: subdural /epidural bleed, subarachnoid bleed, parenchymal bleed, and microbleed. The SWI phase value pattern varied with its kind, development stage, and size. The physiology and magnetic properties of the haemoglobin have a role in the formation (and progression) of a cerebral haemorrhage. It is common practise to classify haemorrhages into five stages based on the presence or absence of met-hemoglobin in red blood cells: hyperacute /intracellular oxyhemoglobin, acute /intracellular deoxyhemoglobin, early subacute /intracellular met-hemoglobin, late subacute /extracellular met-hemoglobin, and chronic[13]. Combining T1- and T2-weighted pictures made it possible to distinguish between various bleeding phases. SWI, which have just lately been used, have demonstrated greater sensitivity and specificity for the identification of intracranial haemorrhages than GRE.

SWI can offer extra phase information and is highly sensitive to blood products. To distinguish between bleeding phases, T1, T2, T2* weighted images shall be combined. The different cerebral haemorrhages shown on the phase picture, however, have not yet been described in detail. On the SWI, every haemorrhage displayed a blooming black signal. However, depending on the stage, location, and size of the haemorrhages, the phase images revealed various looks. These observations on the phase pictures may offer further details regarding the different cerebral haemorrhages[2,13].

Studies by Tong et al.[14,15] and Babikian et al. [16] showed that SWI is around 3 to 6 times more effective than GE sequences in detecting overall size, frequency, quantity, and dispersion of hemorrhage. Figure 6 depicts a patient who was engaged in a car accident and went into a coma as a result of significant brain trauma. Suspicious hypodensities were seen on a non-enhanced CT scan in the affected regions. The cerebral lesions could be seen more clearly using T1 and T2 weighted images. Compared to other traditional sequences, SWI had significantly identified more number of lesions of different sizes, and was even more impressively sensitive in identifying the abnormalities.

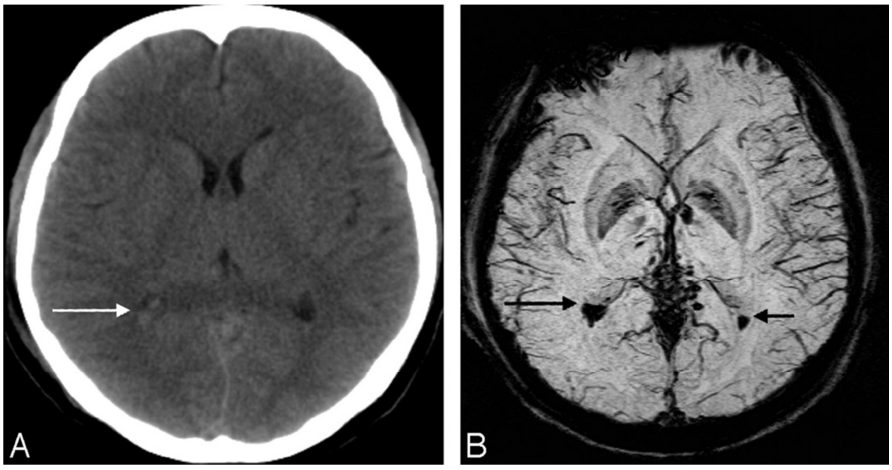
FIGURE 6:



Although larger haemorrhages are visible on CT and conventional diagnostic MR imaging sequences, Tong et al. found that some mild hemorrhagic lesions (shearing) are only evident when using SWI[15]. This conclusion is in line with ours. Additionally, Babikian et al[16] study has shown a correlation between particular

cognitive abnormalities and the magnitude and frequency of SWI hemorrhagic lesions. Lesions in the frontal white matter or parieto-temporo-occipital grey or white matter were seen in the vast majority of individuals, the thalamus area, brainstem area, the cerebellum, and basal ganglia were least affected. Recent head injuries show the SWI is particularly useful in identifying lesions in brainstem caused by trauma, as opposed to conventional MR imaging sequences. According to research, SWI is sometimes even more accurate than CT at identifying haemorrhages outside of the brain parenchyma, such as intraventricular and subarachnoid haemorrhages[2]. CT was unable to find an intraventricular haemorrhage in Fig. 7. The same-day SWI clearly demonstrated haemorrhage deposits in the lateral ventricle's posterior horn. The right lateral ventricle's occipital horn was detected on the initial CT scan by the radiologist retrospectively as having a faint, high-attenuation signal strength. It is obvious that SWI was beneficial in making clinical choices for this patient.

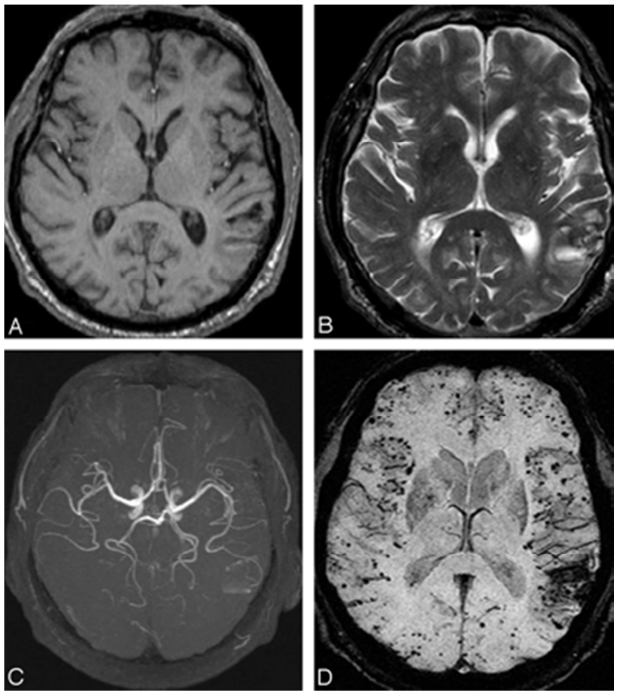
FIGURE 7:



According to several studies, SWI is the most accurate approach for finding microbleeds, exposing nearly 6 times as many micro-bleed as traditional techniques[2]. Figure 8 is of age 70, who had a stent (coronary artery) implanted having prescribed for aspirin and clopidogrel. The patient showed up with a changed mental condition

two months later. An intraparenchymal haemorrhage was seen in the left posterior temporoparietal area on a subsequent CT scan. Brain angiography and MRA on the patient revealed no signs of arterio-venous malformation or other vascular abnormalities.

FIGURE 8:



The accurate diagnosis of cerebral haemorrhages is crucial, yet utilising T2 weighted spin and gradient echo sequences, it's possible to miss it even one year after the haemorrhage started. SWI is quite hence, good at bleed detection and may eventually become more so. Using SWI determines if the duration of time following an intracranial haemorrhage affects the intracranial blood detection and degradation. After a cerebral haemorrhage, it is consistently feasible to identify blood and its products with SWI for a considerable amount of time[17,18].

Subarachnoid haemorrhage (SAH) remains a challenge to diagnose using conventional imaging techniques, especially in the subacute phase. In a research, they compared the conventional MRI sequences with a newer sequence called double

inversion recovery (DIR) for the diagnosis of subacute SAH as compared to GRE and SWI sequences[18]. The DIR sequence is indicated as a great imaging technique for identifying hemorrhagic areas.

The SWI acts as a complement to the existing conventional methods as it is more sensitive in identifying blood products and venous vasculature. SWI also showed the correlations between the blood products[19]. The authors have reported that SWI can be used for tumor characterization as it is good at highlighting the blood products and venous vasculature.

Nathaniel D. Wycliffe, MD , Judy Choe, MD et al. [20] did a comparative study on MR imaging , CT and SWI imaging techniques for identification of haemorrhage. SWI showed evidences of haemorrhage better than MR imaging and CT . The authors have suggested that SWI could be powerful approach for visualizing and detection of haemorrhage. The authors have also suggested that adding SWI sequence to MRI may help in eliminating the need for CT images to detect haemorrhage[20].

SWI for Cerebral venous thrombosis (CVT):

Around 0.5% of strokes are caused by CVT, making it a very unusual cause of brain damage. Diagnosis needs a high indicator of clinical and neuro- radiological investigative evidence due to its varied clinical practice. Few treatment options mostly rely on consensus. Therefore, it's crucial to be conversant with global standards. Although a tiny percentage of patients experience death or disability, outcomes are frequently favorable and the majority make full recoveries[21,22]. The symptom and indications of elevated intra-cranial pressure (ICP), a localized lesion in brain or both elevated ICP and focal lesion can all be present in the clinical presentation of CVT. In as much as 40% of cases, individuals appear suddenly with stroke-like symptoms within 48 hours of symptom onset, and the onset patterns are very variable. More than

50% show up within a month after the commencement of the symptoms, and just a tiny percentage (7%) show up with chronic problems that have persisted for longer than a month [8]. Most patients may benefit from a wide range of therapies, including emergency medicine, stroke care, neurology, and neurosurgery, due to the vague nature of their symptoms. SWI can detect thrombosed sinuses having low signal levels[21].

SWI can be used in detecting Cortical Vein Thrombosis in individuals having spontaneous Intracranial Hypotension. This condition is known as cerebral cortical vein thrombosis. It can occur alone or in conjunction with venous sinus thrombosis. Because the cerebral venous system has various anatomical differences, isolated CVT is challenging to identify. A highly sensitive MR sequence for both the identification of extravascular blood products and intravascular deoxygenated blood is SWI[9].

The most common cause of CVT is venous sinus thrombosis. In the case of spontaneous intracranial hypotension, it is necessary to find imaging evidence of both venous sinus thrombosis and isolated CVT. However, due to the numerous structural variances in the cerebral veins and the fact that the age of the thrombus affects the CVT's , solitary CVT is uncommon and challenging to identify. SWI was able to quickly identify the isolated CVT. According to reports, SWI is useful for identifying intravascular thrombus[23].

SWI can identify a blooming artefact caused by thrombus in and beyond the cerebral artery lumen in individuals suffering from acute stroke and major vascular blockage. It can also be helpful in determining the extent of the thrombus, which could make it easier to develop a treatment plan for thrombolysis. The cerebral veins are visualized as intense structures with dark signals on SWI because they contain a significant amount of deoxy-hemoglobin, which creates a susceptibility effect. The

thrombosed cortical vein in the experimental patient had a much big diameter than the nearby healthy veins. Thus, its easy to identify that it was caused by CVT. Additional help for the diagnosis was the strong signal strength of the associated vein on the T1 picture[23].

The exact evaluation of early haemorrhage and thrombosis involving tiny cortical veins may be greatly aided by SWI (susceptibility weighted imaging) sequences. The most reliable way to diagnose cerebral venous thrombosis is by magnetic resonance venography (MRV) of the brain. It has been noted that SWI has superior diagnostic performance to MRV in identifying CVT and in enabling early detection of CVT in cases when MRV is not practical[24].

Magnetic resonance venography (MRV) brain is the gold standard for diagnosing cerebral venous thrombosis (CVT). SWI is a high-spacial resolution technique that is exquisitely sensitive to venous blood, hemorrhage and iron storage. Study aimed to know the diagnostic performance of SWI in detecting CVT compared to MRV and to facilitate early diagnosis of CVT where MRV not feasible. Magnetic resonance venography (MRV) brain is the gold standard for diagnosing cerebral venous thrombosis (CVT).

When MRV brain is not feasible, conventional MRI with a SWI sequence added is the imaging modality of choice for evaluating individuals with CVT. SWI is more effective in diagnosing isolated cortical vein thrombosis[24].Due to its ability to show microbleeds and the veins increased sensitivity to magnetic susceptibility differences. Researches have attempted to assess the ability of SWI and MRV brain to detect CVT in patients presenting with headache. In addition to providing time-saving information for an early diagnosis of CVT, MRV, diffusion, and SWI shall be used to assess the extent of thrombus, distinguish between different types of edema, find

intracerebral hematoma, hemorrhagic and non-hemorrhagic infarcts, and detect intracerebral hematoma[9,24].

Hemoglobin catabolism and its byproducts have well-defined MR properties. When arterial blood with a high oxygen saturation is combined with venous blood with a lower oxygen saturation, haemoglobin gets deoxygenated. Deoxyhemoglobin, which possesses paramagnetic characteristics, is produced as a result. Deoxyhemoglobin causes a non rhythmic magnetic area and a quick de-phasing of proton spins, which is why the T2*-weighted signal is lost in CVT. This characteristic of the paramagnetic molecules, known as the "magnetic susceptibility effect," causes a signal loss (blooming) that is most seen in T2*/SWI. As a result, T2*/SWI aids in both the identification of hemorrhagic venous infarction and thrombosed sinus.

Thus, SWI/ GRE sequences are critical in the diagnosis of CVT, particularly cortical vein thrombosis, and are very sensitive in detecting blood breakdown products. Deoxyhemoglobin and meth-Hb, two paramagnetic compounds present in the thrombus, cause the thrombosed venous segments to exhibit blooming artefacts[25,26]. In acute thrombosis, where the SI may mistakenly be normal, SWI/GRE is more significant. In addition to sulcal SAH, well-defined tubular hypointensity on SWI may also signal cortical vein thrombosis, which can continue for weeks and be accompanied with underlying cortical/subcortical white matter petechial haemorrhages[25,26]

In young people, cerebral venous thrombosis is a significant stroke-causing factor. The SWI method detects the intraluminal clot in the veins. Due to the clot, the afflicted vein or sinus looks hypointense. SWI is an added diagnostic advantage in detecting clots in cerebral venous sinuses to the conventional T2 flow voids and MRV

particularly when the thrombosis is in its acute phase[27]. SWI offers significantly more sensitivity for diagnosing CVT than standard T1 and T2 weighted pictures. SWI is the MR sequence that makes it easiest to see thrombosed veins and sinuses. In MRI, the loss of signal flow on T1 and T2W sequences is a sign for diagnosing CVT. The distinct signal variations on T1, T2 images are caused by diverse blood breakdown chemical products in thrombosed veins and sinuses. The diagnosis of CVT nearly usually has to be confirmed by MRV. The intraluminal thrombosis is identified by the void of flow on MRV. However, MRV produces artefacts, that could lead to a false negative result[24].

In a 2009 study, researchers outlined the MR imaging characteristics, such as the T2*gradient-echo (GE) sequence, used on 8 patients suspected of having Isolated Cortical Venous Thrombosis (ICoVT)[28]. At the first MR imaging examination, T2*GE imaging revealed a magnetic susceptibility effect (MSE) at the location of a cerebral vein in all individuals. T2*GE imaging revealed either petechial haemorrhages or hematomas in 5/8 individuals. In the course of the follow-up, the volume and strength of the alterations in signal intensity shown in the brain tissues on T1, T2, and FLAIR images all reduced. After 3 months, all patients' ADC readings inside the tissue had returned to normal. The researchers came to the conclusion that T2*GE imaging, MSE of haemoglobin products within the thrombus was observed during both the asymptomatic and symptomatic phases of ICoVT, and that this finding suggests that T2*GE imaging has a higher diagnostic value

SWI is particularly useful in diagnosis of CVT by imaging the hemorrhagic venous infarct and thrombus within the sinus or thrombosed cortical veins. It helps in diagnosing secondary phenomena like venous stasis in the form of transmedullary veins, engorged cortical veins and collateral slow flow[29].Venous hypertension can be

noted at a very early stage before infarction or hemorrhage occurs. These findings will help us to come to the correct diagnosis even though primary findings like the 'empty delta sign' is missed. SWI also plays a significant role in follow up imaging of patients.

SWI for diagnosis of Hypertension:

Blood pressure's impact on venous blood flow in the brain was evaluated using SWI. In MRI, the SWI technique are sensitive to identify differences in magnetic vulnerability across tissues[30]. Deoxyhemoglobin is very reactive to the blood in venous system because of the iron it contains.

The cerebral venous vasculature can differ depending on the range of blood pressure readings, and this can be determined using the SWI approach. The SWI is a prospective method for assessing and classifying blood pressure change in the arteries under study, providing a physiological point of view for MRI and a fresh perspective for radiological vascular research[30].The variations in magnetic properties between oxyhemoglobin and the deoxyhemoglobin play a key role in the SWI phenomenon. The variable that gauges how a material responds to the influence of a B0 magnetic area is called magnetic susceptibility. The substances may be diamagnetic or paramagnetic when this field is present. If they encourage a magnetic field that is directed in the reverse direction from B0, reducing the total area of the magnetic field, they will become diamagnetic. This activity of the oxyhemoglobin results in the creation of an opposing magnetic field.

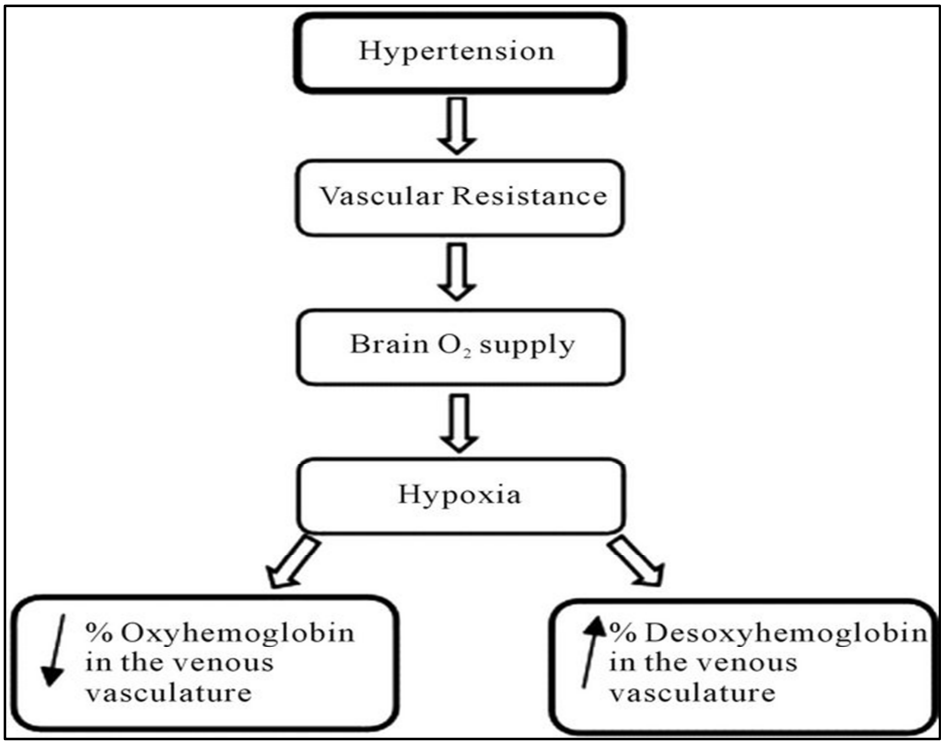


Figure 9. Flow chart showing the effect of hypertension on venous vasculature.

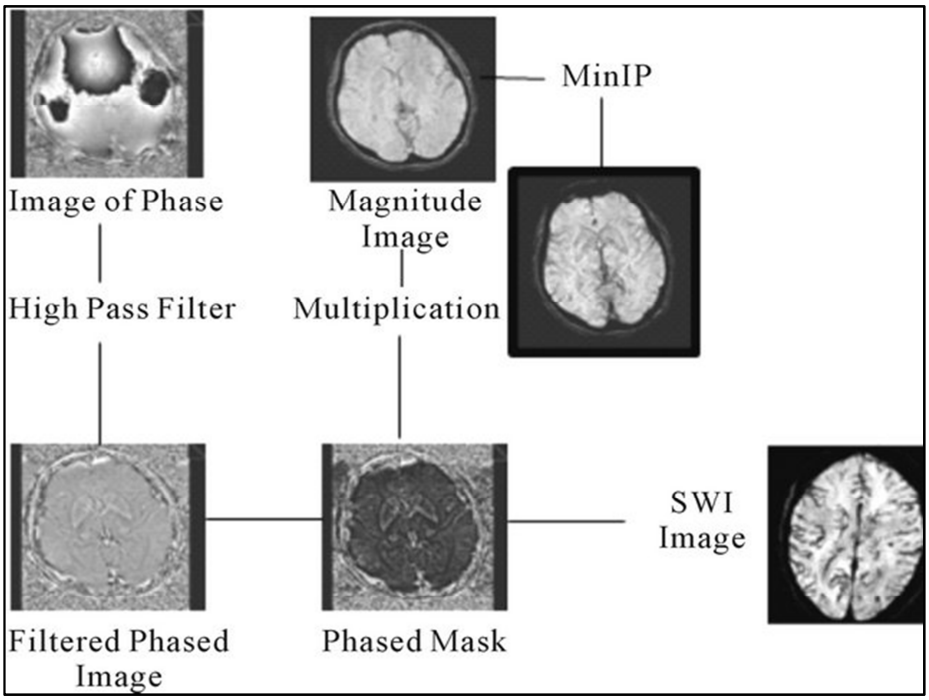


Figure 10. SWI process scheme [31].

On the other hand, because they are paramagnetic substances like deoxyhemoglobin, they may intensify the overall magnetic field of the region. These characteristics are based on the fundamental idea of the venographic impact MRI BOLD (blood oxygenation level dependent),where the SWI is an advancement of the method. It involves using the intrinsic contrast agent T2* and the deoxyhemoglobin's paramagnetic characteristics to generate a variant phase between veins holding de-oxygenated blood and close by brain tissue, balancing out the variations of signal intensities between the two. The additional SWI phase picture will enable the study of blood vessels with extremely tiny dimensions. As a result, the venous vasculature looks hypointense and sharp (reduced slice thickness)[30].

Brain traumatic microbleeds (TMBs), which are identified by susceptibility weighted imaging, have been shown to briefly go unnoticed immediately after damage in a previous rodent research (SWI). Cerebral microhemorrhages with a maximum size of up to 5 or 10 mm that may be seen on susceptibility weighted magnetic resonance imaging[32].

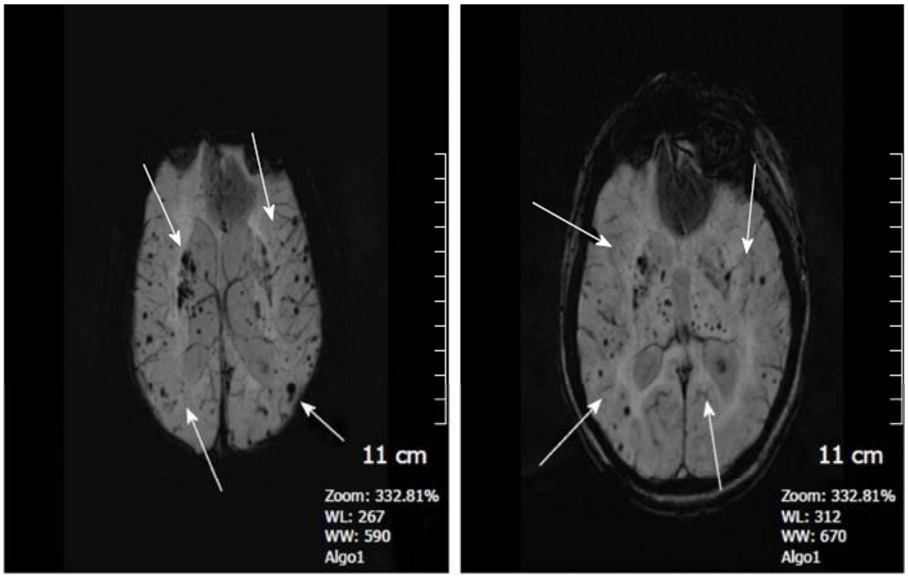
CMBs are increasingly visible on brain MR scans because of the widespread adoption of MR imaging devices and specialised imaging sequences including SW imaging. CMBs may be discovered by chance or in connection with a particular clinical feature, such cerebral amyloid angiopathy(CAA).

The prevalence of blast-induced traumatic brain injury is rising, mostly as a result of the usage of improvised explosive devices, and it is associated with undesired cognitive dysfunctions, as shown in both animal and human studies. A sensitive approach for detecting brain damage is with multi-echo SWI, blood vessels of various diameters became more visible. Increased deoxyhaemoglobin is indicated by a reduction in major vessel post-blast signal strength. Using assumed changes in

oxygen saturation from major blood arteries, relative cerebral blood flow was calculated from filtered phase SWI pictures[33].

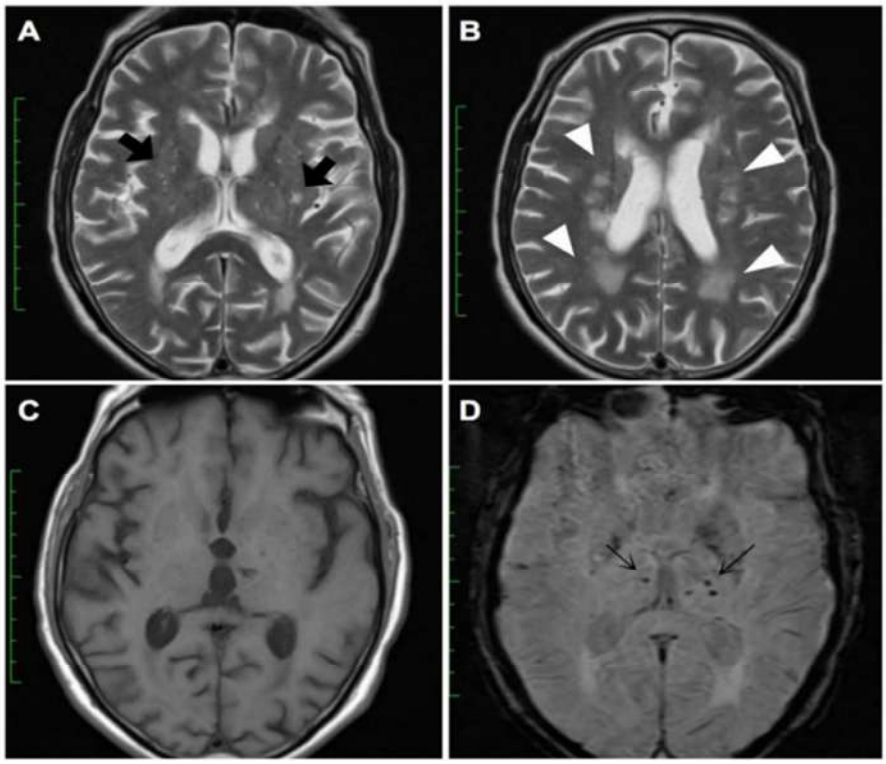
Figure 11 is an image of a 45 year old woman with chronic hypertension. SWI images show multiple microhemorrhages in the thalami, basal ganglia and subcortical white matter which are typical for hypertensive microangiopathy[3].

Figure 11 [3]:



A hypertensive subject with chronic hypertensive encephalopathy is shown in the below picture (Fig 12). Chronic hypertensive encephalopathy is confirmed by the presence of magnetic susceptibility foci in both basal ganglia, as shown on the SWI in the D area of the picture (black arrows). The results have shown that SWI is more efficient in detection and classification of lesions that that of MR imaging without SWI. The authors have also reported that time taken for scan is very less with a maximum time of 3 minutes. The authors have suggested that addition of SWI sequence as a component of brain pathology shall act as a key indicator for diagnosing various brain lesions especially not missing the hemorrhagic lesions, and will contribute to avoiding inappropriate therapeutic treatments[34].

Figure 12[34]:



SWI for detection of Aneurysm:

A brain aneurysm is a dilation of a blood vessel there (AN-yoo-riz-um). An aneurysm is often shown as a fruit dangling from a stem. Hemorrhage in the brain is caused by a ruptured or leaking aneurysm in the brain. Subarachnoid hemorrhage occurs in case of rupture of an aneurysm[35]. Aneurysm ruptures quickly turn life-threatening and need immediate medical attention. However, the majority of brain aneurysms don't rupture, harm the body, or manifest any symptoms. These aneurysms are frequently found during examinations for other disorders. In rare circumstances, treatment for a brain aneurysm that has not ruptured may be necessary in order to avoid a future rupture[35].

Using 7T time-of-flight magnetic resonance imaging and susceptibility-weighted imaging, it was found that the vessel walls of massive unruptured intracranial aneurysm were composed of `three distinct layers. Due of the high spatial resolution of the research, in vivo microstructures could be seen. When compared to TOP-MRA, the wall thickeness of the aneurysm was increased by 1.5 times due to substantial blooming effect on SWI at high magnetic strength[36,37]. However, MR vessel wall imaging can detect aneurysmal wall thickening with neovascularization and inflammation[38].

SWI for Subacute Stroke:

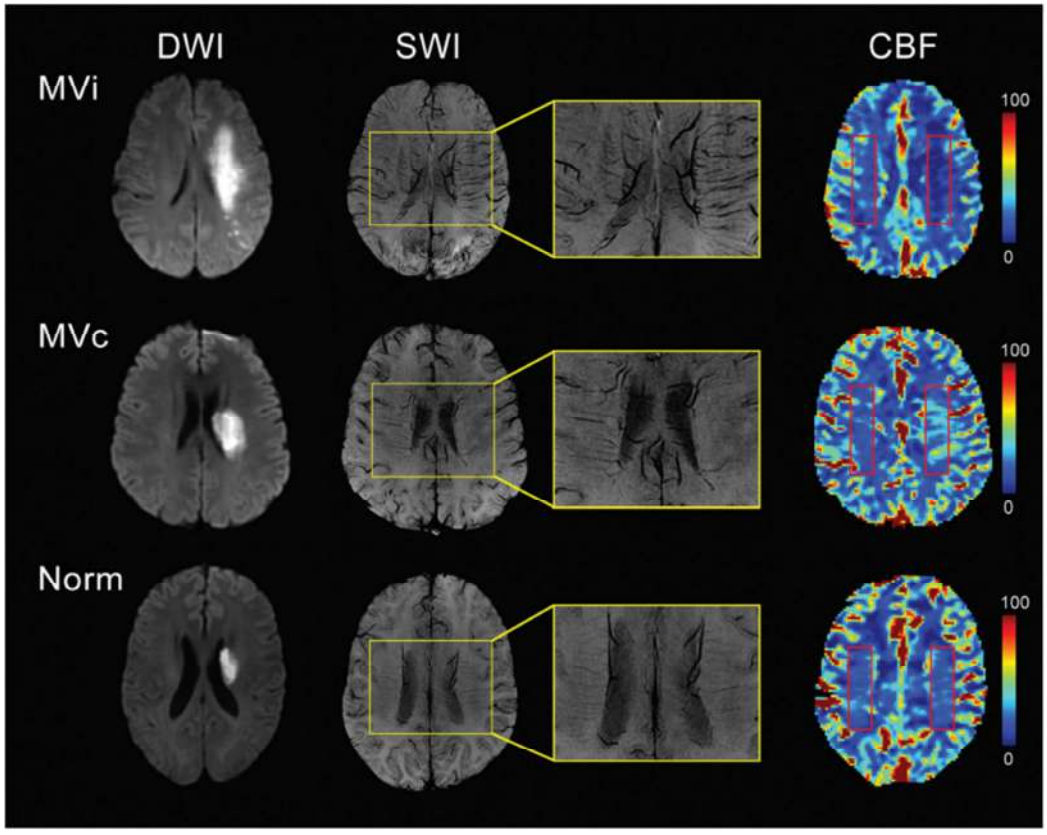
Using SWI, Morita N. et al.[39] found that individuals suffering from acute ischemic stroke had significant cerebral medullary veins (MV) in the centrum semiovale and corona radiata. Increased deoxyhemoglobin owing to localised oxygen extraction in the drainage area may account for the disease's prevalence.

According to research by Hermier M. et al.,[40] greater cerebral blood volume and a longer mean `transit time are linked to MV anomalies. Patients suffering from ischemic stroke, particularly in the subacute period, may have a pronounced MV, also known as the brush sign.

It has been observed by X.Yu et al.[41] that individuals with acute ischemic stroke show pronounced medullary veins in the white matter of the brain. Clinicians may use these imaging indications as prognostic indicators to decide whether or not to use tissue-rescue treatments like recanalization. As a result, this may help stop strokes from becoming worse in the subacute stage. The authors have employed SWI in conjunction with MR imaging to test many hypotheses and locate a small number of biomarkers. Notable ipsilateral MV have been described as predictive indicators for subacute ischemic stroke, hypoperfusion. The figure 13 below demonstrates the

medullary veins on susceptibility-weighted imaging, along with accompanying diffusion-weighted images and cerebral blood flow maps.

Figure 13 [41]:

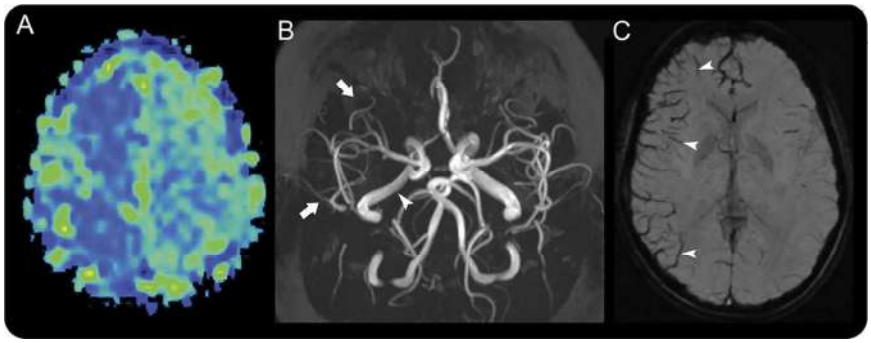


There are Ipsilateral MV in the deep white matter, as shown in the top row of the figure, CBF maps demonstrate a reduction in white matter perfusion in this region. The regions of interest (ROIs) utilised to determine CBF are denoted by red rectangles. MV may be seen in the deep white matter on the other side in the middle row. White matter perfusion is seen to be higher on CBF maps in this region. There is little to no obvious MV in the susceptibility-weighted pictures in the bottom row. The authors show that the prominent MV on SWI has high ability to predict clinical outcome in individuals with acute stroke[41].

Lamiaa G. El-Serougy et al.[42] looked at the accuracy of SWI in identifying microbleeds in cerebral area in stroke patients, can be used to assess the efficacy of thrombolytic medications and predict future recurrence. 124 patients who were referred from the stroke unit underwent traditional MRIs and SWIs. In order to determine the presence, anatomical location, and number of cerebral microbleeds, two observers independently assessed the SWI twice. Using Kappa statistics, inter- and intraobserver agreement was calculated. In SWI, there was almost complete intraobserver agreement for the existence of CMBs in any area of the brain for both observers. Guidelines for the evaluation of CMBs associated with bleeding in both acute and chronic stroke should be revised to include SWI[42].

A study done by Zamora C A et al. on a 10 year old boy who presented with left sided hemiplegia, facial drop and seizures, SWI in MRI showed the prominence of draining veins in the ipsilateral brain parenchyma as shown in the figure 14 below, which indicates raised deoxyhemoglobin in the hypoperfused territory of brain. There was no acute infarct in this case. Although the causes of Todd paralysis is unknown, research show reversible hypoperfusion. It was noted that SWI was a strong and recently developed imaging tool that may help in the assessment of perfusion problems because of its sensitivity to deoxyhemoglobin[43].

Figure 14[43]:

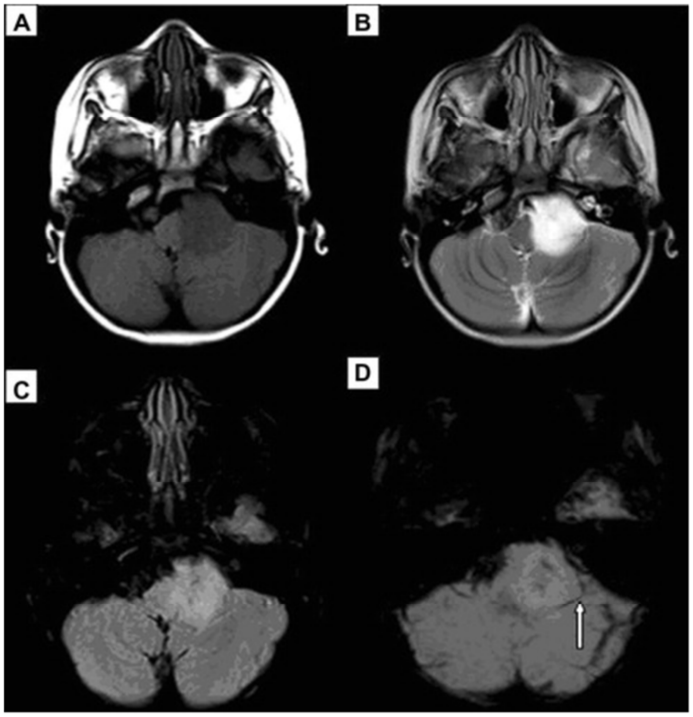


SWI in brain tumors:

Vivek Sehgal, MD, Zachary Delproposto MD et al. has investigated the importance of SWI as a diagnostic value by studying the brain masses. The study was done to evaluate the visibility of tumors, identifying the blood products and defining boundaries. Results have shown that SWI performed better than T1-weighted imaging[19].

Mohamed Masoud Radwan, Reda A. Darwish et al. [44] also evaluated the role of SWI in characterizing brain lesions[44]. SWI helps in identifying the signal loss due to disturbance in homogenous magnetic field caused by ferromagnetic, paramagnetic and diamagnetic components. The investigation was done on 30 patients. Out of 30 patients, 3 had diffuse axonal injury, 6 of them had stroke, 6 of them had brain tumor, 6 had chronic micro bleeds, 5 with venous thrombosis and 4 with extra-axial hemorrhage. The experimental results have shown that all patients SWI proved excellent demonstration of veins as well as bleeds. The below figure (fig 15) is the scan of one of the 6 subjects of brain tumor with left cerebellopontine angle lesion, on conventional imaging it was not certain whether the mass was intra or extra-axial, while on SWI a displaced vessel (arrow) is clearly seen between the mass and the cerebellum revealing the extra-axial origin of the tumor.

Figure 15[44]:



Lt Col Ravinder Sahde et al. investigated the role of SWI for intracranial lesion characterization mostly calcification and hemorrhagic foci. A systematic review was done on all brain imaging studies for a period of two years from 2014-2016. The review was done both inclusive and excusive of SWI in MR imaging. Experiments were done on 3710 patients for identification of trauma, chronic micro bleeds, tumors, venous malformations, intra-cranial calcifications, infarcts and extra axial haemorrhage. Both conventional MRI and SWI were performed on all images. SWI was able to identify positive lesion in 619 patients which was not identified in conventional MRI method. In 21 patients having traumatic brain injury, positive results from SWI were superior than those from MRI and CT. Twenty-six patients had a positive history of hypertension, and SWI indicated several foci of 'blooming' indicative of cerebral microbleeds. A greater grade of glioma was indicated by SWI's detection of widespread hemorrhagic change inside the mass[34].

SWI in Degenerative Disorders:

Dementia and other neurological diseases are becoming more common as individuals live longer. A large number of active research studies for the early detection and treatment of dementia rely heavily on neuroimaging. However, in patients with neurodegenerative diseases, traditional neuroimaging frequently falls short of delivering adequate diagnostic results[6].The proper diagnosis of such illnesses can be facilitated by the analysis of several MRI sequences. Using SWI, we can identify these diagnostic clues since many neurodegenerative diseases are associated with fast iron deposition and/or microhemorrhages in diverse brain regions.

Houman Sotoudeh , Amir Hossein Sarrami et al. reported that in hypertensive cerebral angiopathy condition or vascular dementia, SWI can detect microhemorrhages in typical locations for hypertensive cerebral angiopathy as thalami, basal ganglia, cerebellum, and pons. Microhemorrhages may be detected by SWI even while they are still rather tiny. SWI has reportedly been able to identify brain microhemorrhages more than 1 mm in size. The location of microhemorrhages and iron deposition in the brain is crucial for diagnosis, and the scientists indicate that SWI is the most sensitive sequence to identify them. Patients experiencing cognitive impairment or dementia should include SWI into their daily routine[6].

SWI in Pediatric NeuroImaging:

SWI is progressively used more in clinical practice mainly in children. SWI is used in detection of pediatric and neonatal neurological diseases. Thangamadhan Bosemani, MD et al. investigated the role of SWI in Pediatric Neuroimaging. MR imaging's sensitivity and specificity may be improved by SWI in a variety of paediatric neurological disorders. SWI imaging method can be used to assess crucial brain perfusion in young stroke patients[45]. In paediatric neuroimaging, Researchers

may utilise SWI as a biomarker to learn more about illness development and treatment outcomes. SWI can be used in the early diagnosis of conditions such as developmental venous anomaly, vein of galen aneurysm malformation, any vascular malformations, sturge weber syndrome, hemiplegic migraine, hypoxic ischemic injury, infections and pediatric brain tumor characterization[45].

SWI for identification of Calcification:

SWI filtered phase images can detect even small quantities of calcification. Although a shorter echo duration or more suitable phase conversion to local susceptibility might help, it is still challenging to display the exact form of calcification across a vast region or in high concentration due to aliasing[46]. SWI images showed calcifications that are normal for the pineal gland, both choroid plexuses in the trigone of the lateral ventricles, and the left globus pallidus. Advantages of SWI include the capacity to distinguish between bleeding and calcification inside the plaque[46].

SWI to detect abnormal iron deposition:

Histopathologic examination of multiple sclerosis patients has shown iron accumulation in the brain parenchyma. Quantifying and visualising iron deposition has been accomplished using SWI and other MR imaging modalities. Due to the fact that elevated iron levels produce decreased T2 relaxation time, resulting in hypointensity on T2-weighted images, analysing T2-intensity variations is a simple way to assess iron concentration[47]. Iron accumulation may develop in the same structures before atrophy is noticeable. Tissues with iron deposition (either ferritin, deoxyhemoglobin, or hemosiderin) can be seen using SWI, an MR imaging method. Researchers recently employed this technique to discover substantial changes in iron level between multiple sclerosis (MS) and healthy cohorts individuals with subcortical

deep grey matter(SDGM). When compared to T2 hypointensity and relaxometry studies, a SWI-filtered phase technique is more sensitive to identify tissue alterations indicative of iron[47].

LACUNAE OF LITERATURE:

As compared to conventional imaging techniques such as MRI and CT , SWI has shown more accuracy in diagnosis of various neurological conditions . SWI sequences can identify the different types cerebral hemorrhages, identify bleed in hemorrhagic lesions and separate bleed from calcification, identifying traumatic lesions in the brainstem, help in diagnosis of CVT, show blooming artifact in aneurysm, visualizing and detection of haemorrhage in acute stroke patients, detection of degenearative disorders and detect Cerebral microbleeds (CMBs), SWI is more sensitive in identifying blood products and venous vasculature.

Advantages of SWI over existing conventional method using MR imaging sequences

1. More sensitive to susceptibility than the standard T2* GRE sequence
2. Extremely reactive to microhemorrhages and blood degradation products
3. Because deoxyhemoglobin in veins is paramagnetic, this test may provide details about a patient's venous architecture, blood type variances, and even vascular malformations.

The SWI acts as a complement to the existing conventional MRI method. Hence SWI when added to brain pathological assessments can help in diagnosing neurological disorders better avoiding incorrect treatment regimen and their catastrophic results.

MATERIAL AND METHODS

Source of data: Patients referred for MRI of brain with SWI for different clinical indications including stroke, brain hemorrhage, brain tumors, headache, infection and multiple sclerosis to the department of Radio-Diagnosis at The KLE'S Dr. Prabhakar Kore hospital & MRC, Belgaum.

Method of collection of data:

1. **Study design:** Hospital based observational cross sectional study

2. **Sample size formula:** The minimum sample size formula based on prevalence rate is

$$n = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

Where P is the percentage of prevalence and d is the percentage likely difference in the prevalence.

z_α is linked with the level of significance. For 5% level of the significance $z\alpha = 1.96$.

Ref:

With P = 30% and d = 25% of P = 7.50%, the sample size is 143.

Statistical Analysis: Since the study is of cross-sectional study the plan of analysis will be as follows.

For the continuous quantitative variables mean and standard deviation will be calculated. For the purpose of comparison if the data is divided into two groups with respect to certain qualitative characteristic, the continuous variables will be compared using suitable tools of statistics like student's unpaired t test. The pre and post treatment measures will be compared using student's paired t test.

Discrete variables will be represented by median.

The categorical data will be expressed in terms of rates, ratios and percentages. The association between the outcome, clinical and demographic characteristics will be tested using Chi-square test, test of proportion or Fisher's exact test.

For discrete variables nonparametric tests will be used.

Apart from the above suitable tools like ANOVA, correlation, regression etc., will be used according to the need.

Suitable graphs will be used to depict the comparison.

For all the tests the value of p less than 5% (0.05) will be considered significant.

(1) **Sampling method:** All patients will be evaluated clinically and then undergo an SWI MRI Sequence using a 3 Tesla MRI scanner (Magnetom Avanto TIM, 18 channel; Siemens, Erlangen, Germany) within seven days from the onset of seizures.

(2) **DURATION:** One year – between January 2021 to December 2021

(3) Inclusion criteria:

1. Patients referred with variety of clinical indications including stroke, infections, brain tumors, brain hemorrhage, headache.
2. Patients in the age group of 1day to 85 years will be included in the study.

(4) Exclusion criteria:

1. Contraindications to MRI.
2. Patients with metallic medical implants (intraocular metallic foreign body, cardiac pace makers, MR non compatible intracranial clips of arterial brain aneurysms).
3. Unfit patients for examination.
4. Patients who refused to do the examination.

(5) Methodology: Study will be done using a 3T MRI machine manufactured by Siemens. Standard scan protocol will be done for all the patients with any brain pathology undergoing SWI MRI Sequence. Their SWI sequence will be evaluated and the findings on SWI will be noted and analyzed. Consent will be taken from all patients.

RESULTS

Table 1: Age wise distribution of patients

Age groups	No of patients	% of patients
<1yr	5	3.50
1-10yrs	11	7.69
11-20yrs	10	6.99
21-30yrs	14	9.79
31-40yrs	17	11.89
41-50yrs	21	14.69
51-60yrs	22	15.38
61-70yrs	24	16.78
>=71yrs	19	13.29
Total	143	100.00
Mean	46.58	
SD	22.07	

In this study population, the mean age was 46.58 +/- 22.07 years.

Among the study population, 5 cases were less than 1 year of age (3.5 %), 11 (7.69 %) cases were aged 1-10 years , 10 (6.99 %) cases were aged between 11-20 years, 14 (9.79 %) cases were aged 21-30 years, 17 (11.89 %) cases were aged between 31-40 years, 21 (14.69 %) cases were between 41-50 years, 22 (15.38 %) cases aged 51-60 years, 24 (16.78 %) cases aged between 61-70 years and 19 (13.29 %) cases aged more than or equal to 70 years

Graph 1: Age wise distribution of patients:

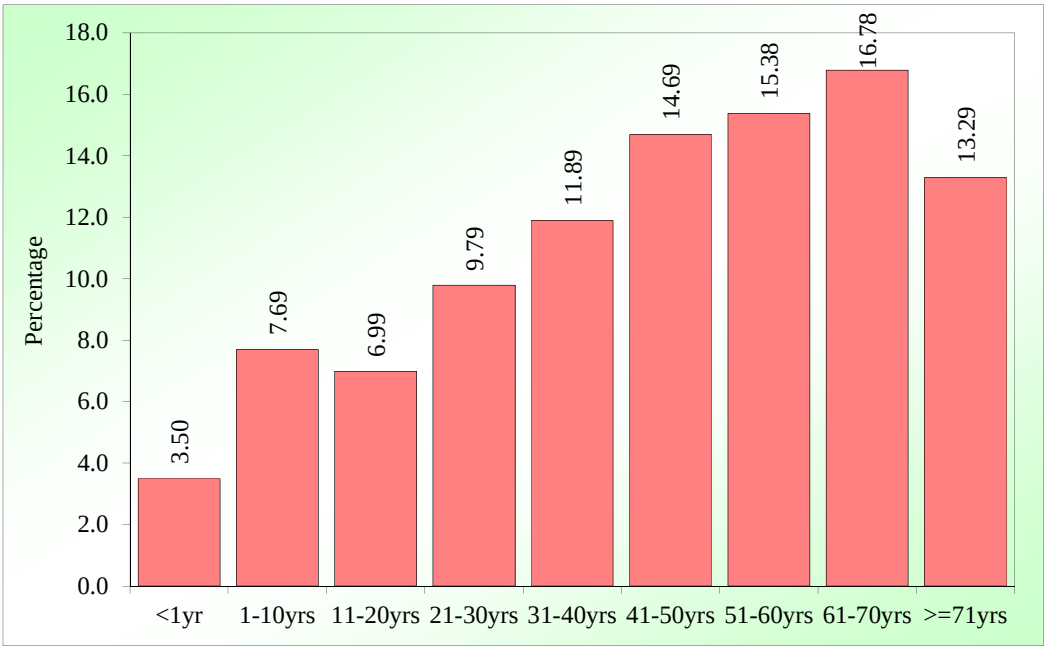


Table: 2 Gender wise distribution of patients

Gender	No of patients	% of patients
Male	102	71.33
Female	41	28.67
Total	143	100.00

Among the study population, 102 (71. 33%) were males and remaining 41 (28.67 %) were females.

Graph 2: Gender wise distribution of patients

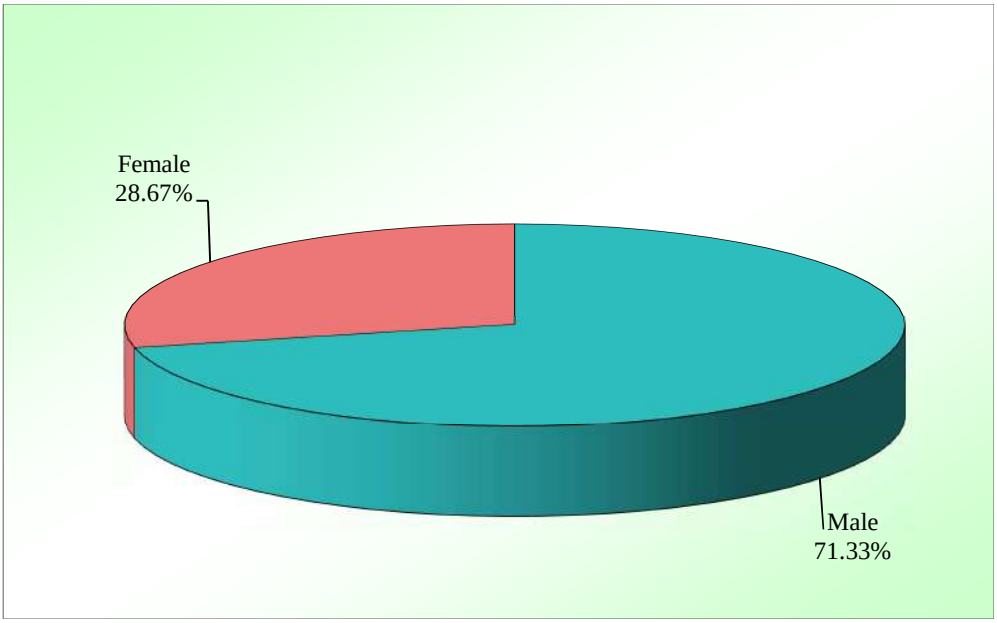


Table 3: Duration of symptoms wise distribution of patients

Duration in months	No of patients	% of patients
<1month	109	76.22
1-5months	21	14.69
>=6months	13	9.09
Total	143	100.00

Among our study population, 76.22 % (109 cases) had symptoms for less than one month, followed by 14.69 % (21 cases) had symptoms for more than one month and less than 5 months and 9.09 % (13 cases) had symptoms more than 6 months.

Graph 3: Duration of symptoms wise distribution of patients

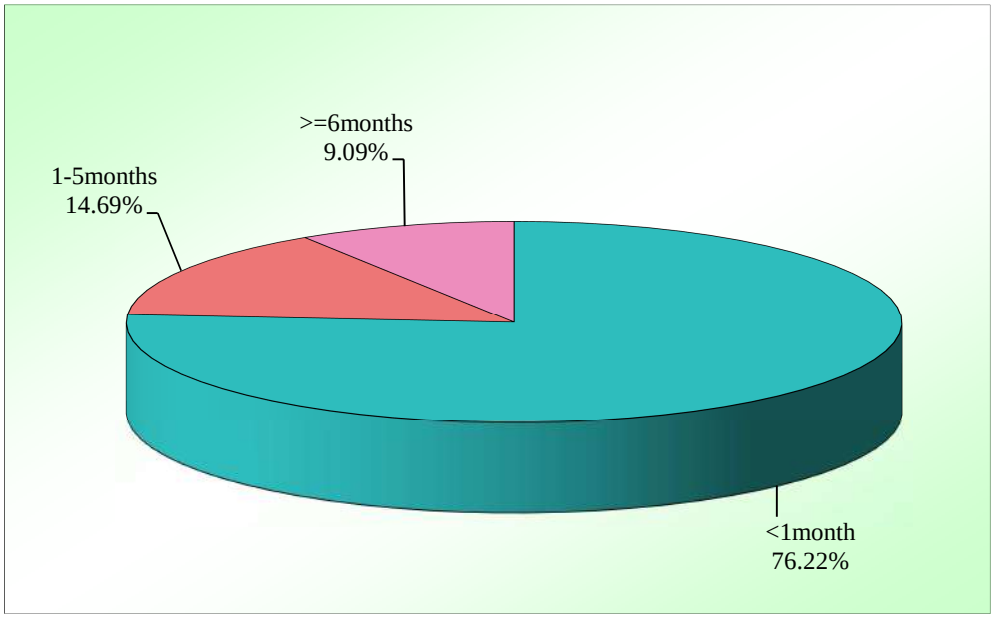


Table 4: Co-morbidities wise distribution of patients

Co-morbidities	No of patients	% of patients
Hypertension	46	32.17
Diabetic mellitus	26	18.18
Myocardial infraction	22	15.38

Among the study population, the most common co-morbidity was hypertension in 46 cases (32.17 %) followed by diabetes mellitus in. 26 cases (18.18 %) and myocardial infarction in 22 cases (15.38 %). Few of the above subjects (28 cases) were associated with more than one co-morbidities Rest of the subjects, 78 out of 143 (54.5 %) were not associated with any co-morbidities.

Graph 4 : Co-morbidities wise distribution of patients

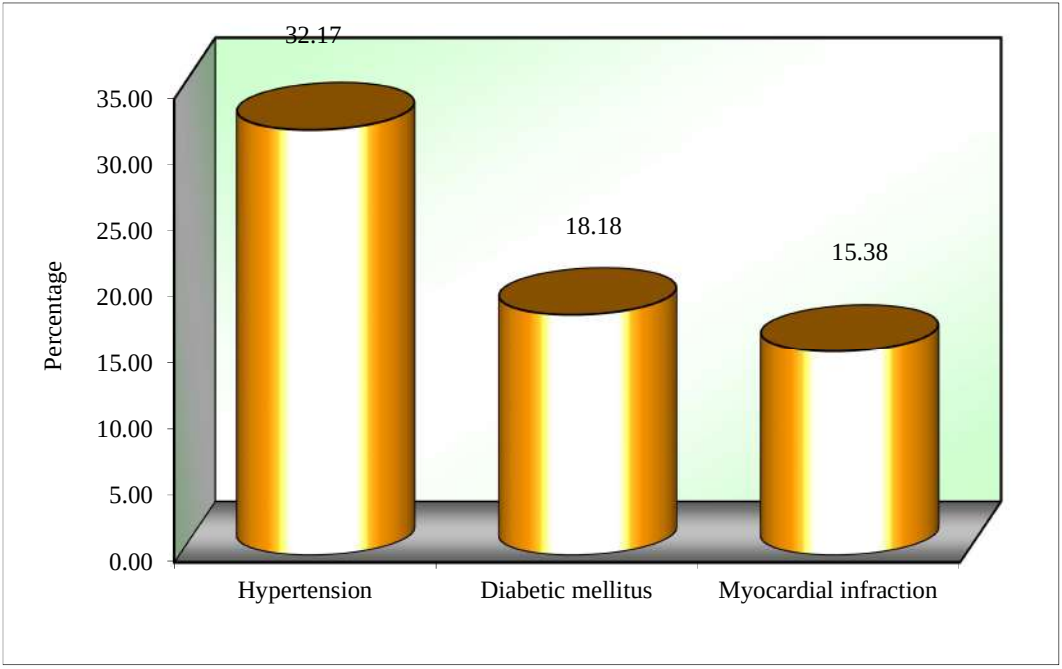


Table 5: Underlying cause wise distribution of patients

Underlying cause	No of patients	% of patients
Nonvascular	78	54.55
Vascular	65	45.45
Total	143	100.00

Among the study population, the underlying case of the pathology was non-vascular in 78 cases (54.55 %) and vascular in 65 cases (45.45 %).

Graph 5: Underlying cause wise distribution of patients

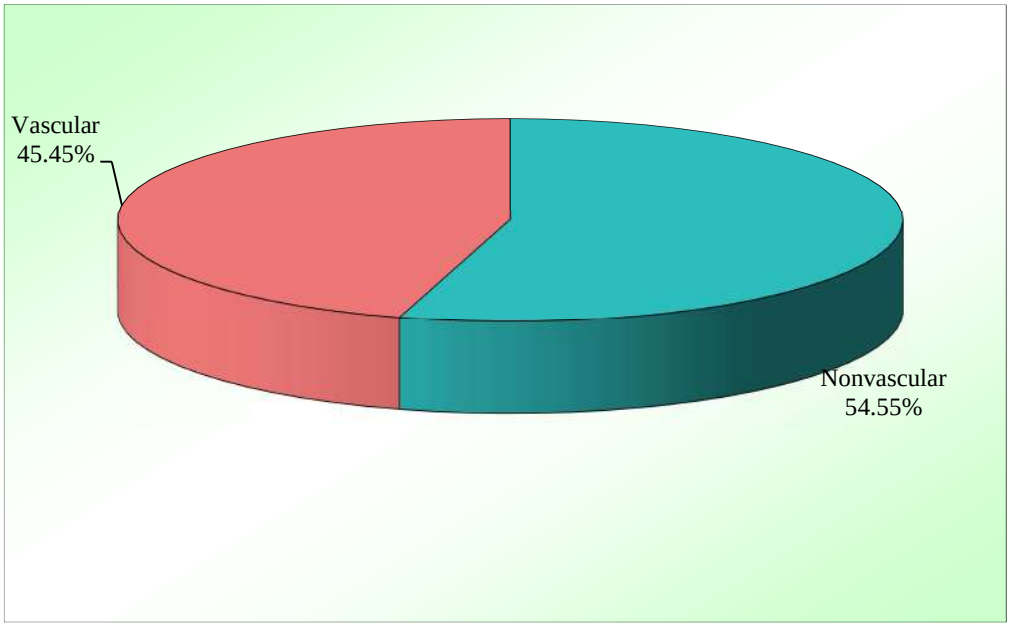


Table 6: Supra/ Infratentorial wise distribution of patients

Underlying cause	No of patients	% of patients
Infratentorial	16	11.19
Supra	125	87.41
Both	2	1.40
Total	143	100.00

Among the study population, the pathology was located in the supratentorial brain paren chyma in 125 cases (87.41 %) and infratentorial brain parenchyma in 16 cases (11.19 %) and both in 2 cases (1.40 %).

Graph 6: Supra/ Infratentorial wise distribution of patients

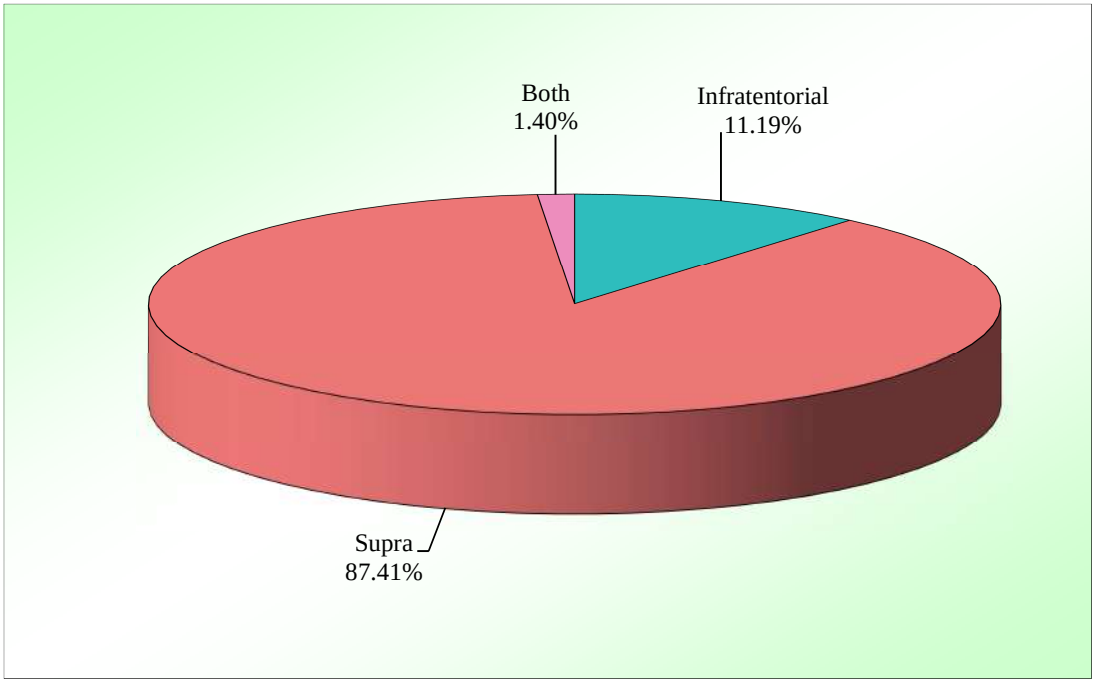


Table 7: MRI sequences (T1) wise distribution of patients

MRI sequences (T1)	No of patients	% of patients
Hyper	61	42.66
Hypo	58	40.56
ISO	20	13.99
Mixed	2	1.40
NA	2	1.40
Total	143	100.00

Among the study population, 61cases (42.66 %) had T1 hyperintensity, 58 cases (40.56 %) were hypointense on T1, 20 cases (13.99 %) were isointense on T1, 2 cases (1.40 %) showed mixed intensity and 2 cases (1.40 %) had no obvious signal intensity on T1.

Graph 7: MRI sequences (T1) wise distribution of patients

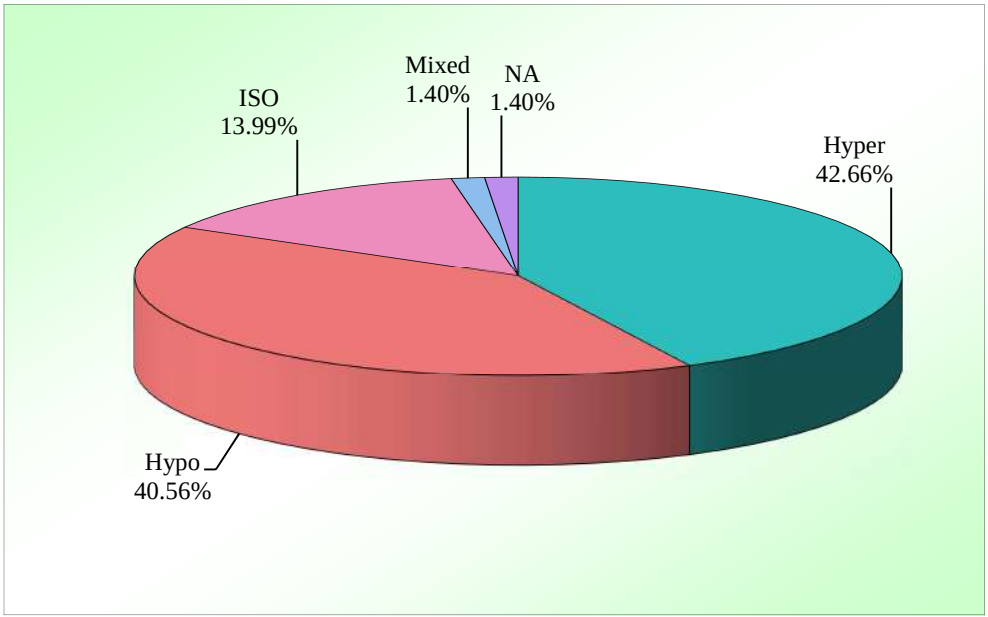


Table 8: MRI sequences (T2) wise distribution of patients

MRI sequences (T2)	No of patients	% of patients
Hyper	75	52.45
Hypo	48	33.57
ISO	4	2.80
Mixed	10	6.99
NA	6	4.20
Total	143	100.00

Among the study population, 75 cases (52.45 %) showed T2 hyperintensity, 48 cases (33.57 %) showed T2 hypointensity, 4 cases (2.80 %) showed isointensity on T2, 10 cases (6.99 %) showed mixed intensity on T2 and 6 cases (4.20 %) had no signal abnormality on T2.

Graph 8: MRI sequences (T2) wise distribution of patients

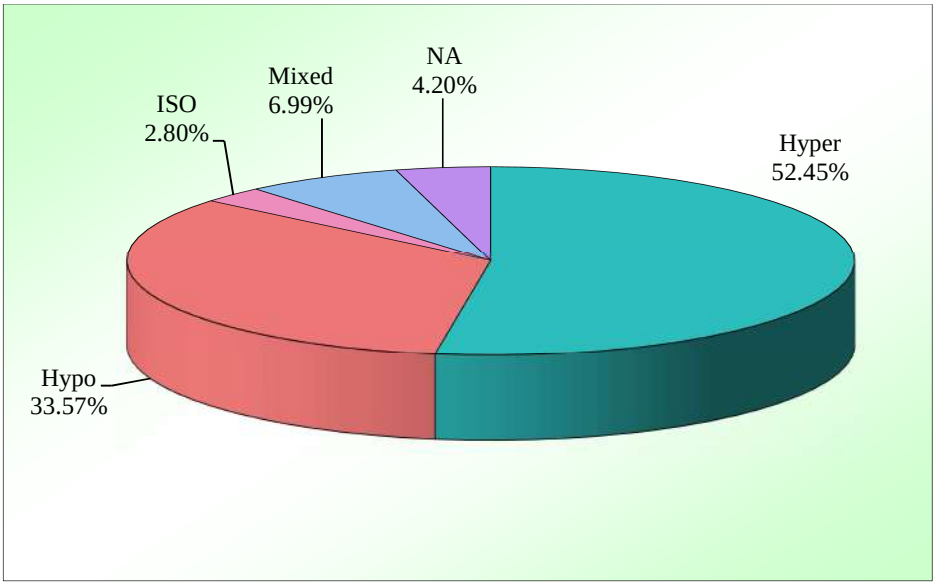


Table 9: SWI wise distribution of patients

SWI	No of patients	% of patients
Yes	143	100.00
No	0	0.00
Total	143	100.00

Since our study is based on clinical applications of SWI as an added sequence, only the patients with blooming on SWI were considered as subjects, hence all 143 subjects had blooming on SWI.

Table 10: Phase wise distribution of patients

Phase	No of patients	% of patients
Yes	108	75.52
No	35	24.47
Total	143	100.00

Among our study population, 108 cases (75.52 %) had hypointensity on SWI had hyperintensity oh phase suggestive of bleed/ hemosiderin and 35 cases (24.48 %) which had hypointensity on SWI had hypointensity in phase as well suggestive of calcium.

Table 11: MRI findings wise distribution of patients

MRI findings	No of patients	% of patients
Bleed	82	57.34
Calcium	35	24.48
Hemosiderin	26	18.18
Total	143	100.00

Among the study population, 82 cases (57.34 %) had bleed followed by 35 cases (24.48 %) and 26 cases (18.18) had hemosiderin based on the MRI findings predominantly depending on SWI & Phase sequences.

Graph 9: MRI findings wise distribution of patients

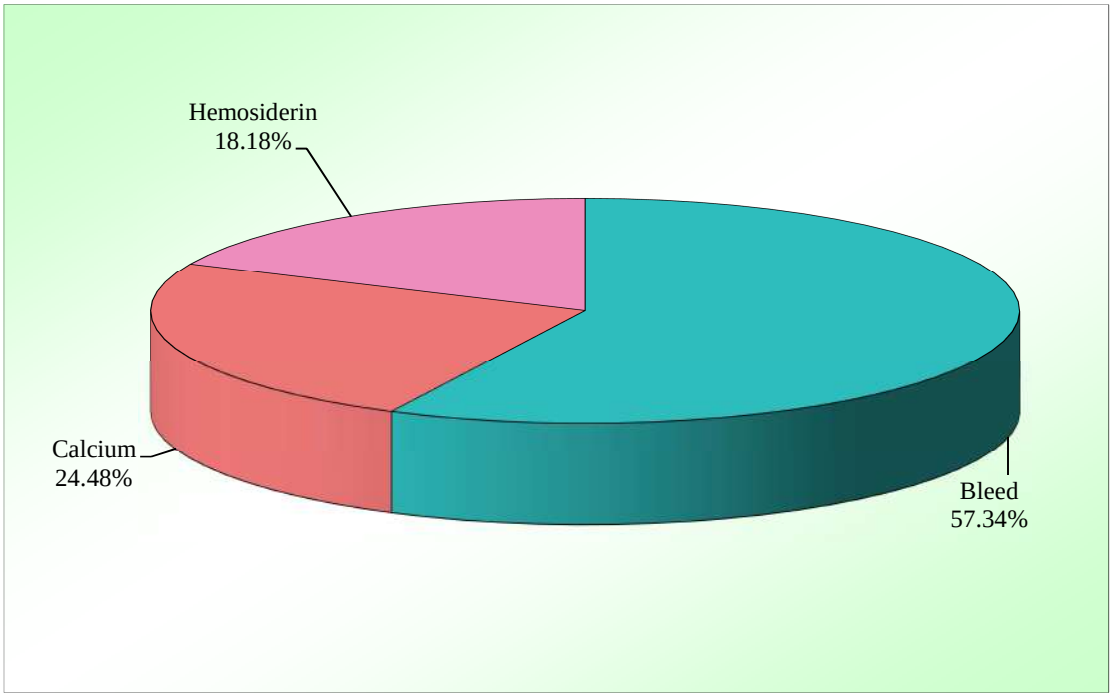


Table 12: Patients with presence of hypointensity (SWI) and (Phase)

Patients with	Patients with	No of patients	% of patients
Blooming (SWI)	Calcium	35	24.48
Blooming (Phase)			

Table 13: Patients with presence of hypointensity (SWI) and hyperintensity (Phase)

Patients with	Patients with	No of patients	% of patients
Blooming (SWI)	Bleed/ Hemosiderin	108	75.52
Non-blooming (Phase)			

The cases with bleed and hemosiderin both had hypointense blooming on SWI and hyperintensity on Phase and they are differentiated by correlating with T1 & T2 and clinical history. In our study in most of the cases, we had seen that hemosiderin (iron) deposition was seen as T2 hypointensity, however in few cases like PKAN, T2 hyperintensity was noted due to gliosis and spongiosis that is surrounded by a low signal intensity caused by iron accumulation.

Table 14: Provisional diagnosis wise distribution of patients

Provisional diagnosis	No of patients	% of patients
Hemorrhagic tranfromation of infarct	10	6.99
Cerebral venous thrombosis	13	9.09
Trauma Bleed	6	4.20
Infective Etiology	15	10.49
Iron (Hemosiderin) deposition	26	18.18
Vascular malformations	6	4.20
Developmental venous anomaly	2	1.40
Aneurysm	4	2.80
Neurodegenerative disorders	7	4.90
Brain tumors	31	21.68
Hyper tensive microangiopathy	5	3.50
Intraparenchymel hemorrhage	10	6.99
Neuro cutaneous syndrome	3	2.10
Moya Moya disease	2	1.40
Acute Necrotizing hemorrhagic encephalopathy	2	1.40
Lentiform nucleus calcification	1	0.70
Colloid cyst	1	0.70
Peri callosal lipoma	2	1.40

The above mentioned table demonstrates various diagnosis which are made during this study along with the number of patients under each diagnosis.

Among the study population, the highest number of cases with pathology on SWI were of brain tumor i.e; 31 cases (21.68 %)

Among the study population with hemosiderin , few cases were included both under their primary diagnosis and under hemosiderin deposition. For example, multiple sclerosis was included both in neurodegenerative disorders and in hemosiderin deposition.

Graph 10: Provisional diagnosis wise distribution of patients

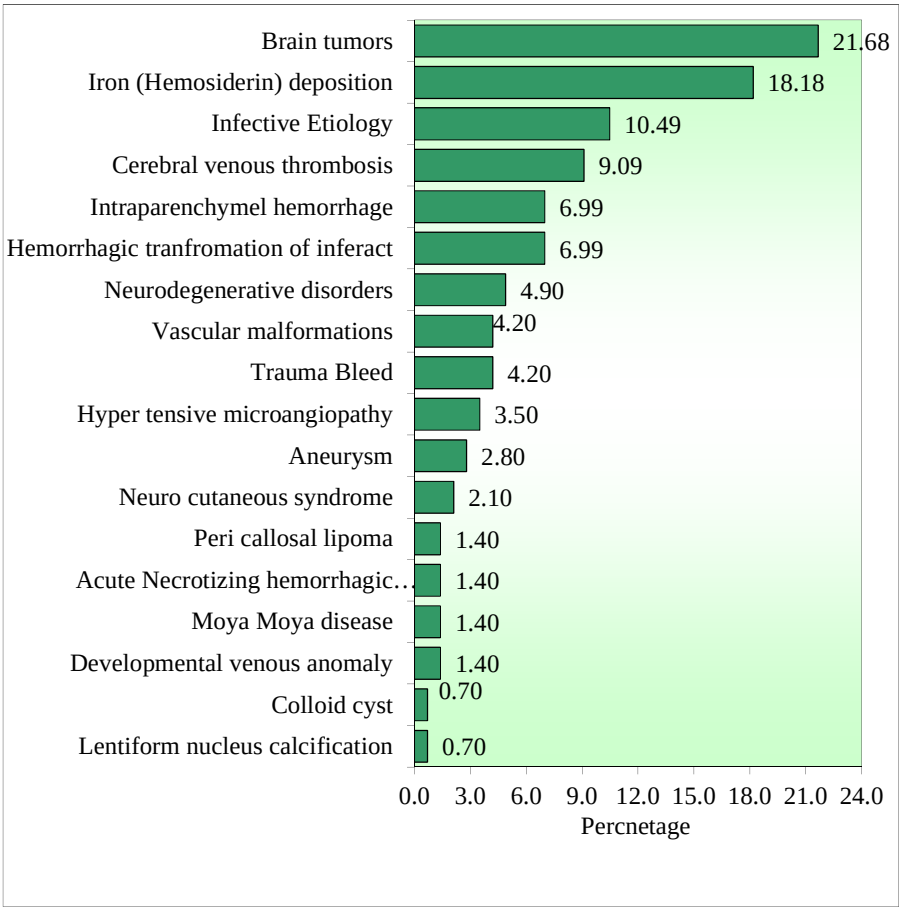


Table 15: Cerebral venous thrombosis wise distribution of patients

Cerebral venous thrombosis	No of patients	% of patients
Hemorrhagic venous infarct	6	46.15
No hemorrhagic only venous thrombosis	7	53.85
Total	13	100.00

In our study population, a total of 13 cases of cerebral venous thrombosis were noted out of which 6 cases (46.15 %) were of hemorrhagic venous infarct and 7 cases (53.85 %) did not have hemorrhagic component.

Graph 11: Cerebral venous thrombosis wise distribution of patients

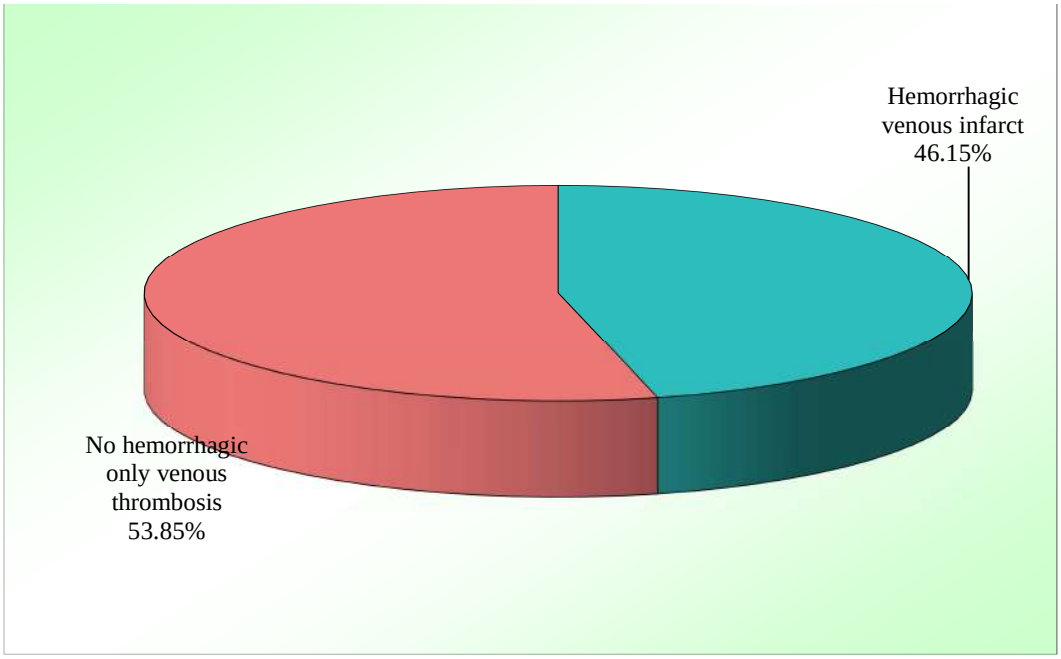


Table 16: Infective Etiology wise distribution of patients

Infective Etiology	No of patients	% of patients
Tubercular Etiology	8	53.33
Neuro cysticercosis	6	40.00
Cerebral abscess	1	6.67
Total	15	100.00

In the study population, a total of 15 cases of infection were noted, out of which 8 cases (53.33 %) were of tubercular etiology, 6 cases (40.00 %) were of neurocysticercosis and one case (6.67 %) was of cerebral abscess.

Graph 12: Infective Etiology wise distribution of patients

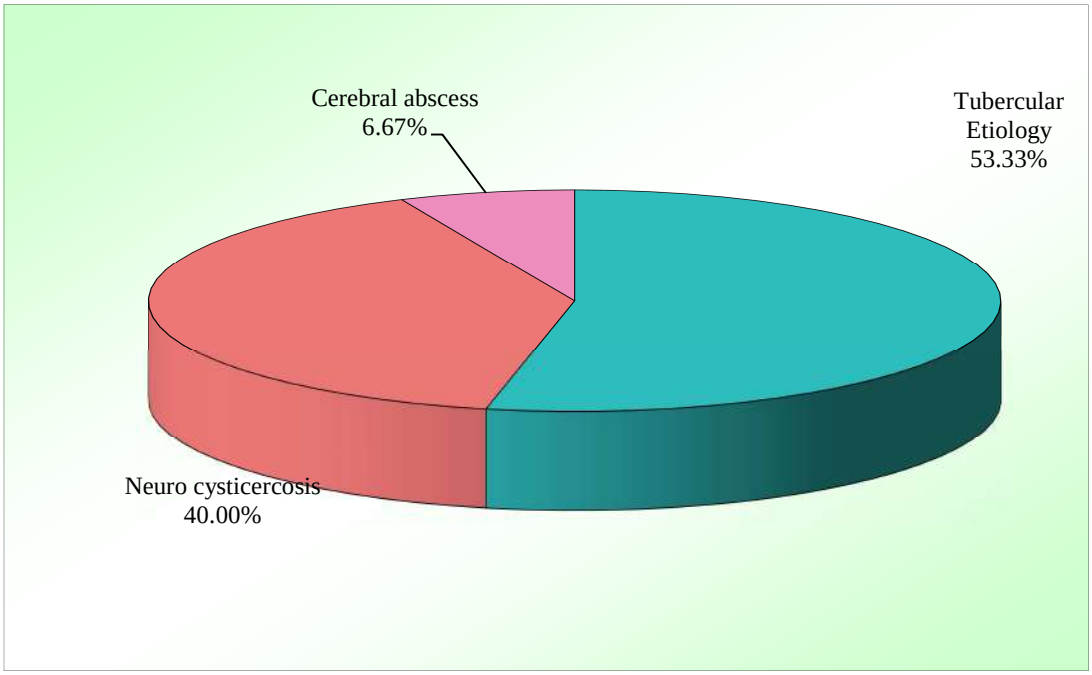


Table 17: Vascular malformations wise distribution of patients

Vascular malformations	No of patients	% of patients
Arterio venous malformation	3	50.00
Cavernous malformation	3	50.00
Total	6	100.00

A total of 6 cases of vascular malformations were noted in this study and among the 3 cases (50.00 %) were arterio-venous malformation and 3 cases (50.00 %) were cavernous malformation.

Graph 13: Vascular malformations wise distribution of patients

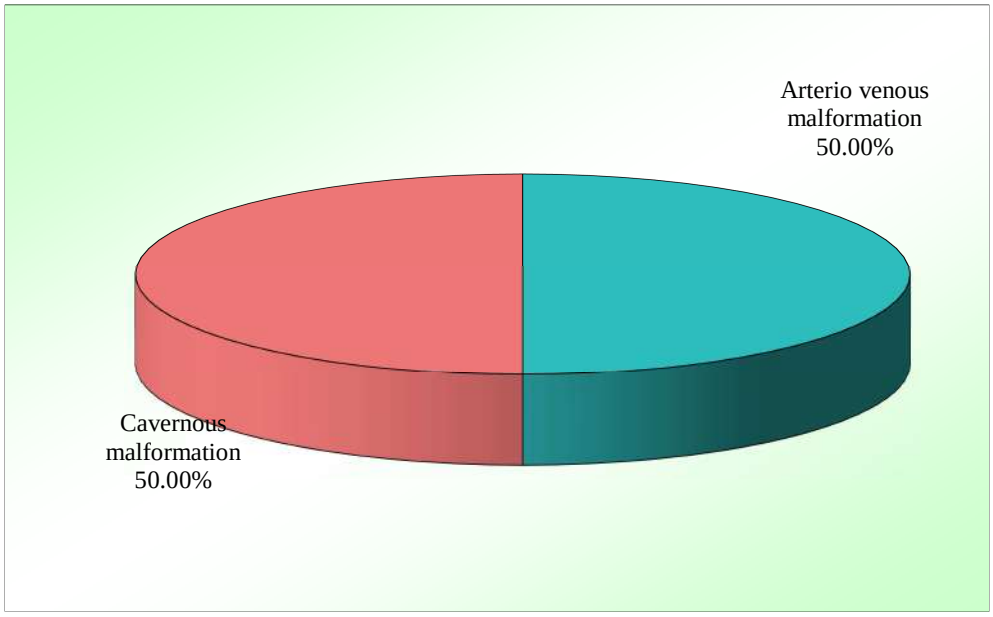


Table 18: Aneurysm wise distribution of patients

Aneurysm	No of patients	% of patients
Ruptured Aneurysm	1	25.00
Unreptured Aneurysm	3	75.00
Total	4	100.00

Among the study population, 4 cases of aneurysm were noted out of which one case (25.00 %) was ruptured aneurysm and the rest three cases (75.00 %) were unruptured aneurysms.

Graph 14: Aneurysm wise distribution of patients

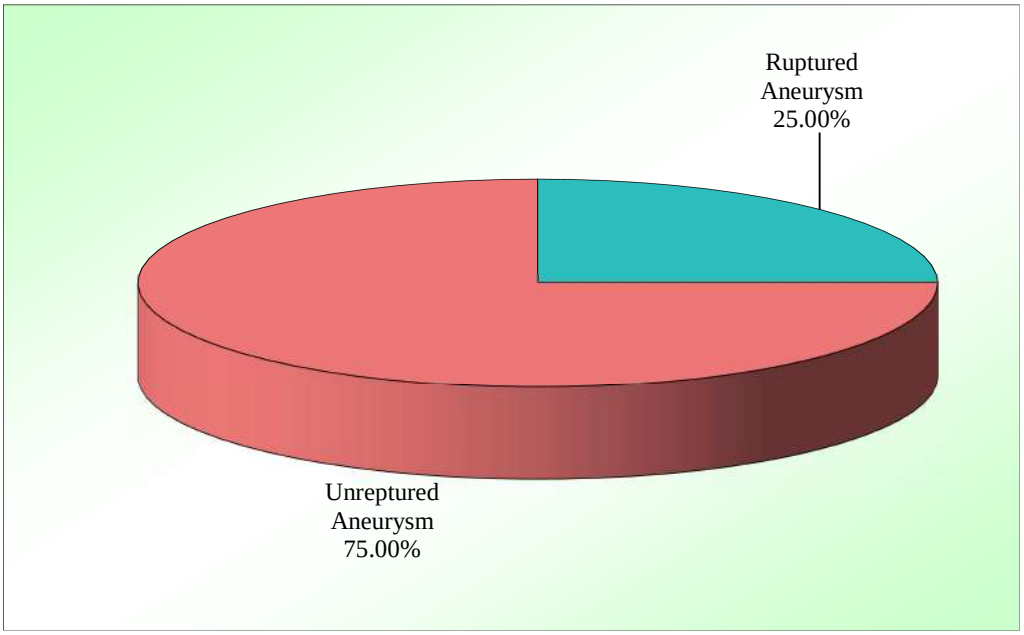


Table 19: Neurodegenerative disorders wise distribution of patients

Neurodegenerative disorders	No of patients	% of patients
Multiple scalerosis	2	28.57
Neuro-degeneration with iron accumulations	1	14.29
Panthotenate kinase associated Neuro-degeneration	1	14.29
Parkinsons	1	14.29
Demyelinating plaques	1	14.29
Multi system atrophy predominant putamen	1	14.29
Total	7	100.00

Among the study population, a total of 7 (28.57 %) neurodegenerative cases were noted, out of which 2 were cases of multiple sclerosis, one (14.29 %) was case of neuro-degeneration with iron accumulation, one (14.29 %) was case of pathotenate kinase associated neurodegeneration, one (14.29 %) was case of parkinson's disease, one (14.29 %) was case of demyelinating plaques and one (14.29 %) was case of multisystem atrophy-predominent putamen.

Graph 15: Neurodegenerative disorders wise distribution of patients

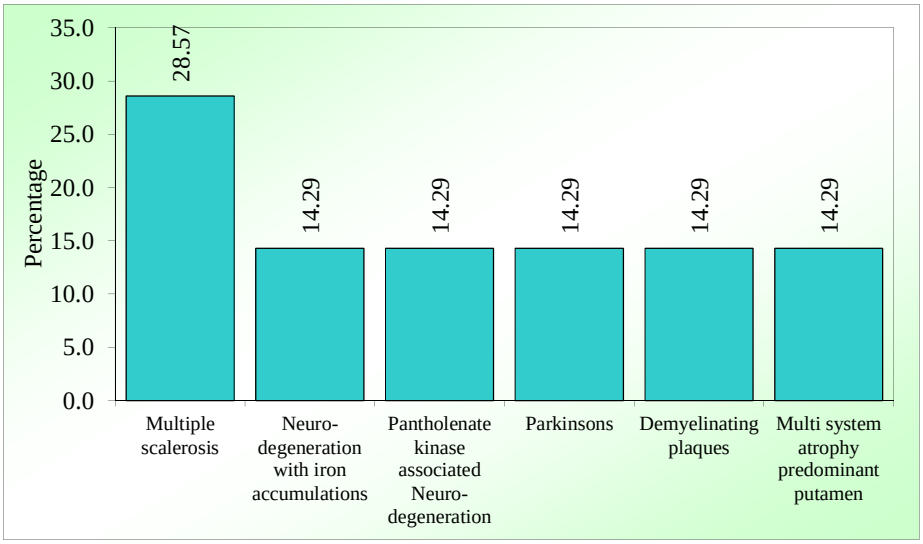


Table 20: Brain tumors wise distribution of patients

Brain tumors	No of patients	% of patients
Hemorrhage within tumor	17	54.84
Calcium within tumor	12	38.71
Hemosiderin within tumor	2	6.45
Total	31	100.00

In this study, about 31 cases of brain tumors were observed and in that 17 cases (54.84 %) had hemorrhage within the lesion, 12 cases (38.71 %) had calcium within the lesion and 2 cases (6.45 %) had hemosiderin within the lesion.

Graph 16: Brain tumors wise distribution of patients

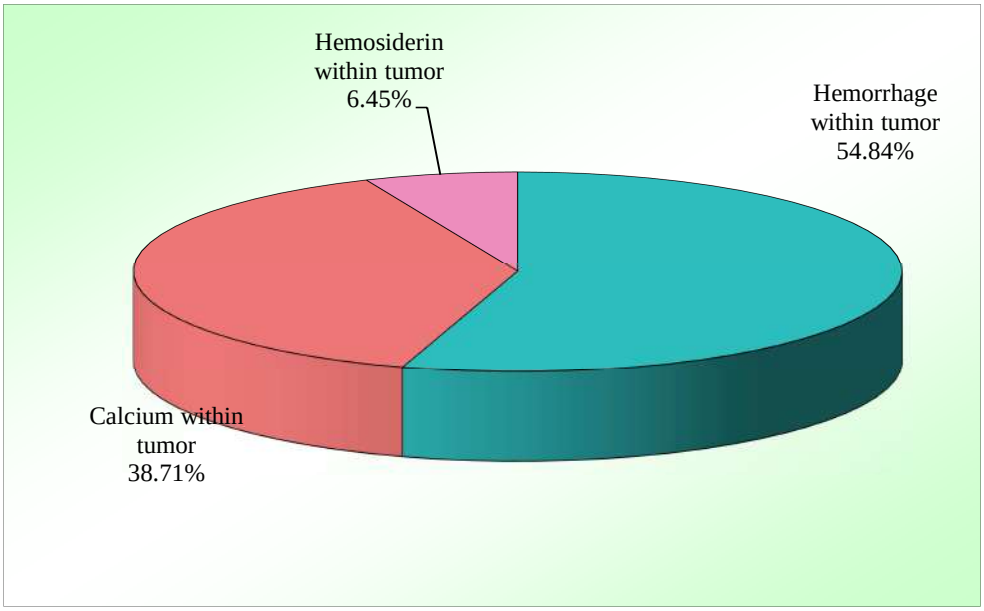
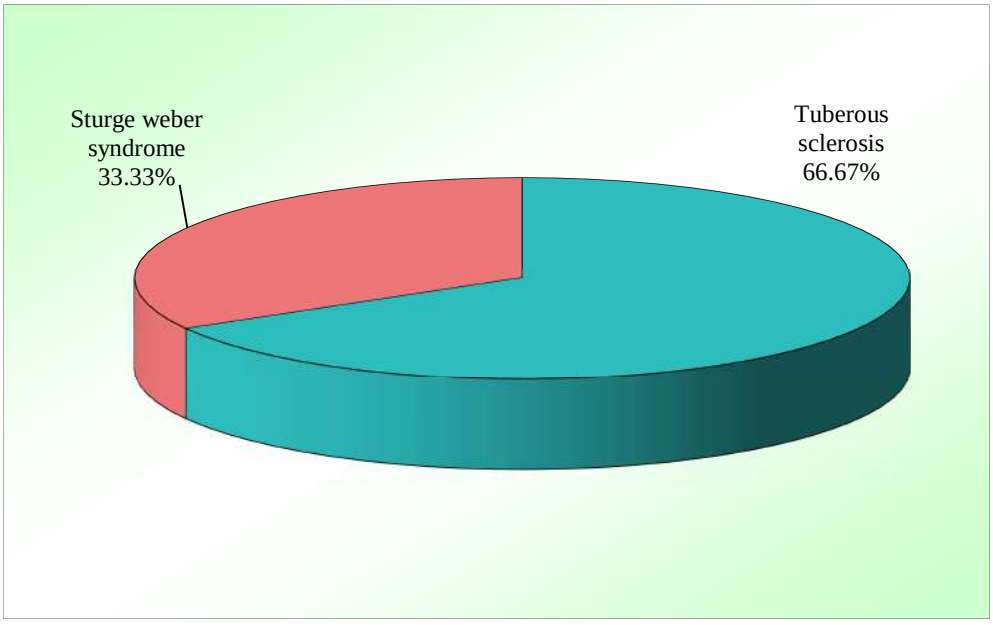


Table 21: Neuro cutaneous syndrome wise distribution of patients

Neuro utaneous syndrome	No of patients	% of patients
Tuberous sclerosis	2	66.67
Sturge weber syndrome	1	33.33
Total	3	100.00

In this study, note was made of 3 neurocutaneous syndrome cases, out of which 2 cases (66.67 %) were of tuberous sclerosis and one (33.33 %) was of sturge weber syndrome.

Graph 17: Neuro cutaneous syndrome wise distribution of patients



DISCUSSION

The purpose of this study was to investigate the therapeutic uses and benefits of SWI as an additional sequence to the usual standard sequences of the MRI protocol in patients with neurological symptoms[42,47,48]. In the present study, the sample size was 143, but in previous studies the sample size ranged from 45 to 200.

A thorough clinical history, severity of symptoms, and comorbidities were recorded. In this study, the mean age of the participants was 46.58 years, with a standard deviation of 22.07 years; however, this varied between investigations based on disease and neurological deficiency. This research is comparable to one done by Radwan MM et al. with a mean age of 45[44].

The majority of the participants in the study belonged to age group of 61-70 (16.78 %) years followed by 51-60 years (15.38% each). Least number of participants in the study belonged to the age group of less than 1 year (3.50 %).

In the present study males outnumbered females (102 males with 71.33 % and 41 males with 28.67 %) similar to the study conducted by Guna singh M et al. which constituted 71 % of males and 29 % of females. In few more similar studies as well male predominance was noted[49].

The purpose of this study was to determine the radiological significance of SWI as an addition to traditional MR sequences in aiding us to detect disease with more precision, which is crucial for patient care. Bleed, calcium, and iron in the form of hemosiderin are the top three substances listed on SWI. We detected 84 (58.7%) instances of bleeding, 33 (23.1%) instances of calcium, and 26 (18.1%) instances of hemosiderin in the current study.

The majority of the participants, 125 (87.41%), had pathology in the supratentorial brain parenchyma, while 16 (11.19%) had pathology in the infratentorial brain parenchyma, and 2 (1.40%) had pathology in both supra & infratentorial brain parenchyma.

In this study, neurological diseases were classified as either vascular or non-vascular. Approximately 78 people (54.55%) had a non-vascular aetiology, whereas 65 participants (45.5%) had a vascular abnormality.

In a research by Elnekeidy AE et al., SWI sequence revealed hemorrhagic transformation of infarct in 10 out of 50 patients (20.0%), in our investigation, hemorrhagic transformation was found in 10 out of 143 participants (6.9%). Therefore, SWI is a precise sequence that aids in the diagnosis of early hemorrhagic change of infarct in stroke patients, which might be overlooked by CT scan[50].

In our study, about 13 (9.09%) of 143 patients had cerebral venous thrombosis, and 6 (46.1%) had hemorrhagic venous infarction. SWI is extraordinarily sensitive to even minute quantities of paramagnetic compounds due to its intrinsic sensitivity, enhanced spatial resolution, and thinner slices obtained. SWI provides a distinct contrast, comparable to blood oxygen level–dependent (BOLD) imaging, which is commonly utilised in functional imaging[51].

Six (4.2%) of the 143 people in our study had a history of trauma that manifested as subdural hematoma, hemorrhagic contusions, and extradural hematoma on the SWI sequence. Eldeş T. et al. conducted a similar study on the importance of SWI in mild traumatic brain injury (mTBI) in which 7 of 58 participants with a history of trauma revealed microhemorrhages on SWI; however, 5 of those 7

participants had hyperintensity on conventional sequences of MRI and in the remaining 2 participants, the bleed was only seen on SWI sequences, demonstrating the importance of SWI in cases of mild trauma. SWI is a technology that exploits the differential in magnetic sensitivity across tissue types. SWI can detect microhemorrhage sites linked to DAI six times more sensitively than standard MRI sequences. There is a need for sequences that complement standard sequences and aid in diagnosing mild traumatic brain injuries[52].

In the present study, about 15 (10.4%) of 143 individuals developed brain infections, of which 8 (53.3%) cases were of tubercular aetiology (tuberculoma, tuberculous meningitis, and calcified granuloma), 6 (40.0%) cases were of neurocysticercosis, and 1 (6.66%) case was of cerebral abscess. In these situations, SWI was primarily employed to illustrate calcifications within these important ring-enhancing lesions (tuberculomas and neurocysticercosis).

Lai PH, Chang HC, et al. conducted a research on 14 patients with pyogenic brain abscess. SWI phase images in 6 subjects indicated moderate hypointensity, in 3 subjects indicated iso-intensity, and a mixture of iso- and mild hypointensity in 5 subjects. The study concluded that SWI phase imaging demonstrates the existence of paramagnetic compounds consistent with free radicals from phagocytosis. SWI may provide crucial new information for characterising pyogenic brain abscesses[53].

In the current study, 26 (18.2%) of 143 subjects have iron (in the form of hemosiderin) accumulation in the brain parenchyma due to various causes, including neurodegeneration, old history of trauma, gliosis with hemosiderin deposition, hemosiderin within a brain space-occupying lesion, and chronic intraparenchymal haemorrhage. Iron buildup is typically observed in neurodegenerative disease

patients. Hagemeier J et al. found in their investigation that SWI is capable of recognising tissues impacted by iron deposition as ferritin, deoxyhemoglobin, or hemosiderin. This was employed in a recent study that found significant iron content variations between multiple sclerosis (MS) patients and healthy controls in subcortical deep grey matter (SDGM). An SWI-filtered phase technique offers significant methodological benefits over T2 hypointensity and relaxometry measures, since it is more sensitive to identify tissue changes that are symptomatic of iron. Without SWI, T2 hypointensity is considered as straight-forward way to diagnose iron deposition, but now with advanced MR sequences like SWI, the diagnosis of iron deposition in brain has become more accurate[46]. However, in our study in few cases of hemosiderin deposition such as lacunar infarct with hemosiderin, cerebral abscess and gliosis with hemosiderin there was no T2 hypointensity likely due to the underlying primary pathology which is seen as T2 hyperintensity (example: gliosis which is the primary diagnosis of the patient is seen as T2 hyperintesity), however in such cases SWI played a crucial role in the visualization of hemosiderin which was not seen on T2. Clinical history of the patient also plays an important role in such cases[47].

The current study found that out of 143 participants, 6 (4.1% of the total) had vascular malformations, 3 of which were arterio-venous malformations and 3 were cavernous malformations that were not easily visible using standard MRI sequences.

SWI is accurate in diagnosing arteriovenous shunting in vascular malformations of the brain, and for some patients, it may help as a non-invasive alternative to angiography in screening for or diagnosing vascular malformations of the brain[47]. This was shown in a retrospective study by Jagadeesan BD et al., who found that SWI

was 93% sensitive and 98% specific for the detection of AVS in vascular malformations of brain (BVMs)[54].

Sultan AY's research indicated that SWI is extremely sensitive in the diagnosis and identification of the correct number of vascular malformations such as cavernomas than conventional MRI and in his study diagnosing cavernoma using the help of SWI, had a sensitivity of 100%, specificity of 60%, and accuracy of 91.9%[55].

Only two people out of 143 in our sample exhibited a developmental venous abnormality (DVA), but the SWI sequence revealed significant sensitivity to blood via susceptibility dephasing effects inside the veins, paving the way for precise diagnosis, grading, and follow-up of these abnormalities. In DVA, a group of veins was arranged in a "spoke-wheel" or "caput medusae" pattern. Abdelgawad MS found that, compared to other non-contrast MR sequences, SWI had a significantly higher sensitivity and specificity for DVA detection, ranging from 96.6 to 100%. SWI also had a significantly higher positive predictive value (PPV) of about 96.66% and a significantly higher negative predictive value (NPV) of about 93.1%. This study found that compared to other non-contrast MR sequences, SWI demonstrated outstanding DVA demonstration, with much greater sensitivity, specificity, PPV, and NPV. The SWI diagnosis of DVA identifies as a signal void lesion with the normal cerebral veins[56].

Out of 143 patients, we found that 4 (2.79%) had aneurysms, with 1 of those instances being a burst aneurysm. The term "aneurysm" refers to a localised, abnormal widening of a blood vessel. CT angiography is the gold standard for diagnosis, however MR imaging with additional sequences like SWI also provides information about aneurysms by revealing hypointense blooming on the SWI

sequence and allowing for the detection of calcium within the aneurysm on the Phase sequence. Subarachnoid haemorrhage, which may be overlooked on standard MR sequences, can be diagnosed using SWI in case of a ruptured aneurysm.

Seven participants (4.9% of the total) had diagnoses of neurodegenerative disease; two had multiple sclerosis, one had neurodegeneration with iron accumulation, one had panthotenate kinase associated neurodegeneration (PKAN), one had Parkinson's disease, one had demyelinating plaques, and one case of multisystem atrophy-predominant putamen (MSA-P) was noted. Our research shows that SWI may be used to identify diagnostic signs like rapid iron deposition and/or microhemorrhages in brain parenchyma, which are linked to numerous neurodegenerative pathologies.

The basic concept of SWI is that signals from tissues with different magnetic susceptibilities compared to their adjacent neighbouring tissue will appear out of phase if the echo time is long enough, as was stated in a similar study by Sotoudeh H et al. on the role of SWI in evaluating neurodegenerative disorders in daily practise. In Parkinson’s syndrome an aberrant swallow tail sign is a valuable marker with a 100% sensitivity and negative predictive value, 95% specificity, and 69% positive predictive value. Susceptibility artefact (poor signal) in iron deposition locations is seen on SWI scans of individuals with PKAN[6].

The MSA-P is the most well-known marker for Parkinson's disease. Putaminal atrophy was found in 51.9% of patients with parkinsonism-predominant multiple system atrophy, and it was most common in the unilateral putamen. Hwang I concluded his study by noting that SWI can show putaminal atrophy and significant signal hypointensity with high specificity, and that this demonstrates the dominant

side of putaminal changes, which correlate with the contralateral symptomatic side of patients[57].

Classification of brain tumours can benefit from SWI since it enables the detection of hemorrhagic and calcified foci inside tumours and the evaluation of the tumours' intricate internal angioarchitecture. We found that 31 people, or 21.6%, out of 143 had brain tumours, and that among those 31 people, 17 (54.8%) had blood inside the lesion, 12 (38.7%) had calcium inside the lesion, and 2 (6.4%) contained hemosiderin. Whether the lesion contains blood, calcium, or hemosiderin, using SWI played a crucial role in determining the correct diagnosis.

Sehgal V. et al. found that in a subgroup of 38 patients, SWI provided more useful information than conventional T1-weighted postcontrast pictures for viewing blood products and venous vasculature, respectively[19]. The research also noted that SWI displayed a beneficial FLAIR-like contrast and supplemented the information given by traditional T1 postcontrast sequences describing the internal architecture of the lesions, in addition to being significantly more sensitive for revealing blood products and venous vasculature[19].

Zulfiqar M. conducted a study on the detection of intratumoral calcification in oligodendrogliomas using SWI, and found that compared to paired data of MR with SWI and MR without SWI, the sensitivity of MR with SWI (86%) was significantly higher for the detection of intratumoral calcification in oligodendrogoma (by using CT as the criterion standard)[58].

Additionally, SWI can differentiate between vestibular schwannomas and cerebellopontine angle meningiomas. Schwannomas, unlike meningiomas, produce microhemorrhages[59].

Only five out of 143 patients in this research had hypertensive microangiopathy, detected by SWI as the main sequence, which manifests as a loss of signal or bloom[55]. Multiple silent cerebral microhemorrhages, which are not readily apparent on regular CT and conventional MR sequences, have been associated with hypertensive cerebral angiopathy. A hypertensive intracerebral hematoma is more likely to occur in the thalamus, basal ganglia, cerebellum, and pons than in the cortex, as is the case with congophilic amyloid angiopathy[60]. A risk factor for the development of later intracerebral macrohematomas/lobar haemorrhages makes the diagnosis of these microhemorrhages crucial[61].

Out of 143 participants, 10 developed intraparenchymal haemorrhage, which was identified as a dark blooming impact on SWI sequence. The SWI offered the diagnosis an extra benefit to confirm the diagnosis as haemorrhage, therefore aiding in boosting the diagnostic accuracy, even if in these cases IPH was also observed on traditional sequences, with intensity varied according to the age of the bleed. Researchers Punitha P et al. found that utilising the SWI sequence was more sensitive than using the GRE for detecting cerebral bleeding in individuals with amyloid angiopathy[62].

In addition, Lee YJ et al. found that the phase value of the SWI varied in a predictable fashion based on the hemorrhage's cause, severity, and duration[13].

Two instances of tuberous sclerosis and one case of sturge weber syndrome (SWS)were identified in our analysis as neurocutaneous syndromes. Multiple cortical and subcortical tubers, some of which are calcified and appear as hypointense blooming on SWI and phase sequences, are characteristic of tuberous sclerosis. There is a low risk of malignancy in these cortical tubers, which are hamartomatous lesions. The frontal lobes are the most prevalent location for these lesions, followed by the parietal, occipital, and temporal regions[63].

Multiple areas of hypointensity in the subcortical regions were seen in both SWI and phase sequences, which we attributed to calcifications, a diagnostic feature of sturge-weber syndrome. When comparing MR SWI to conventional T1 contrast MRI in characterising brain abnormalities of SWS, Hu J found that SWI was more effective than T1-Gd in detecting enlarged transmedullary veins, abnormal periventricular veins, cortical gyriform abnormalities, and grey matter/white matter junction abnormalities[64]. When it came to detecting enlarged choroid plexus and leptomeningeal abnormalities, however, T1-Gd was superior than SWI. The study concluded saying that SWI can give important and unique information supplementary to conventional contrast enhanced T1 weighted MRI for defining SWS[64].

Moya-moya disease (MMD) is a rare cerebrovascular condition defined by stenosis of the distal most bilateral internal carotid arteries, which causes formation of a compensatory network of perforating blood vessels called Moyamoya vessels[65].

Throughout our research, we came across 2 cases of MMD in which SWI revealed strong hypointense blooming signals in the draining veins within regions of poor perfusion indicating increased visibility of veins in that region, as a result.

However, Digital subtraction angiography is the gold standard for diagnosing and evaluating MMD at the moment[66].

Few other cases were seen in this investigation in which SWI was crucial to making a diagnosis, including lentiform nucleus calcification, colloid cyst, pericallosal lipoma, and acute necrotizing hemorrhagic encephalopathy.

CONCLUSION:

- SWI is based on a magnetic susceptibility difference of various substances such as deoxyhemoglobin, blood, calcium and iron.
- Sample size in this study was 143.
- Mean age in the study population was 46.58+-22.07
- In this study males outnumbered females (71.33 % vs 28.67 % respectively)
- In our study headache was the most common symptom followed by seizures.
- Among the study population, maximum number of cases observed were brain tumors i.e; 31 cases (21.68 %).
- Among 143 participants, hemorrhage was present in 82 cases (57.34 %), calcium was noted in 35 cases (24.48 %) and hemosiderin was noted in 26 cases (18.18 %).
- Observing all the conventional MRI sequences along with advanced sequences like SWI in neuroimaging, following points were noted in this study:
    (a) This study has observed that SWI is very useful in detecting micro as well as macrohemorrhages with great accuracy as compared to conventional sequences like T1.
    (b) Few microhemorrhages, calcium and iron deposition which did not show any abnormality in convetional MR sequences were noted using SWI.
    (c) SWI has helped to differentiate between calcium and hemorrhage using Phase as an addition.
    (d) SWI was helpful in characterization of brain tumors which helped to narrow down the differential diagnosis

(e) SWI shed light on few neurodegenerative disorders in a more rigorous manner by assessing the brain iron accumulation

(f) SWI gave an added accuracy in the diagnosis of vascular and ischemic conditions which has hemorrhagic transformation

(g) SWI helped in proving additional information in characterizing neuro infections.

(h) SWI has also played a key role in the detection of developmental venous anomaly, aneurysms, microangiopathies, intraparenchymal hemorrhage, neurocutaneous syndromes and so on.

Thus, in this study we observed that SWI is a very useful imaging sequence with a variety of clinical application and should be included in routine MRI protocol for increasing the accuracy of the final diagnosis.

LIMITATIONS

1. In unstable patients and in patients with claustrophobia the quality of the images was poor
2. MRI brain is an expensive investigation as compared to CT
3. MRI brain is a time-consuming investigation in case of emergencies
4. Due to limited sample size, further characterization of few of the cases was not possible.
5. Future multi-centric studies involving a large sample size and the use of randomized sampling techniques could increase the validity of the results and can further help in generating clinical and radiological evidence for making recommendations in the day-to-day practice.

SUMMARY

- The study was a prospective cross-sectional study.
- 143 cases were included in the study with neurological symptoms after observing the inclusion and exclusion criteria.
- SWI sequence was including in all these MRI brain studies along with conventional MR sequences.
- The main purpose of the study was to understand the uses and applications of SWI as an added advantage to the conventional sequences to narrow the differential diagnosis and to come to a final diagnosis.
- Susceptibility-weighted imaging (SWI) is a new neuroimaging technique.
- There are multiple neurologic conditions that can be benefitted dramatically through a very sensitive technique that monitors the bleed, calcium and amount of iron, whether in the form of deoxyhemoglobin, ferritin, or hemosiderin.
- SWI combines gradient-echo techniques with phase information to make images that are superior and complementary to other methods for resolving hemorrhage, venous vasculature, and iron
- Among the study population, the number of patients with brain tumors, stroke, cerebral venous thrombosis, hypertension, neurodegenerative disorders, vascular malformations, trauma and brain infections were noted.
- The cases which showed hypointense blooming on SWI and hyperintense blooming on phase indicate bleed/ hemosiderin.
- The cases with hypointense blooming on SWI and hypointensity on phase as well indicated calcium.
- Bleed and hemosiderin can be further differentiated based on T1 & T2 sequences.

- Among the patients with the above diagnosis the cases with bleed, calcium and hemosiderin were noted.
- About 82 cases (57.34 %) showed bleed, 53 cases (24.48 %) showed calcium and 26 cases (18.18 %) showed hemosiderin deposition in the brain parenchyma.
- The highest number of cases noted were brain tumors i.e; 31 cases (21.68 %) .
- In this study SWI and phase helped to diagonise various neurological conditions related to hemorrhage, calcium and iron deposition.
- In case of brain tumours the presence of haemorrhage or calcium is of significant importance to narrowed down the differentials such as oligodendroglioma which contain calcifications.
- SWI also played a significantly important role in the diagnosis of microbleeds which in future may cause intraparenchymal hemorrhage.
- SWI played a key role in the diagnosis of chronic hemorrhage which can be missed om conventional sequences.
- SWI helped in the diagnosis of various neurodegenerative disorders which contain iron deposition.
- In cases like various vascular malformations such as cavernoma, it is difficult to diagnose without SWI sequence.
- In cases of vascular territory based stroke SWI showed blooming of the thrombosed vessel.
- SWI was also useful in cases of hemorrhagic transformation of infarct and hemorrhagic venous infarct.
- It was useful in cases with tiny focal findings such as lentiform nucleus calcifications and calcifications in lipoma.

- Hence, the study highlighted the significance of SWI in MRI brain and its use in making the final diagnosis and it should be added to the conventional MR sequences in all the cases.
- While SWI has not replaced conventional MRI sequences, it provides clinically relevant information.

BIBLIOGRAPHY

1. Rubin A, Waszczuk Ł, Trybek G, Kapetanakis S, Bladowska J. Application of susceptibility weighted imaging (SWI) in diagnostic imaging of brain pathologies–a practical approach. Clinical Neurology and Neurosurgery. 2022 Jul 14:107368.
2. Mittal S, Wu Z, Neelavalli J, Haacke EM. Susceptibility-weighted imaging: technical aspects and clinical applications, part 2. American Journal of neuroradiology. 2009 Feb 1;30(2):232-52.
3. Halefoglu AM, Yousem DM. Susceptibility weighted imaging: clinical applications and future directions. World journal of radiology. 2018 Apr 28;10(4):30.
4. Haller S, Haacke EM, Thurnher MM, Barkhof F. Susceptibility-weighted imaging: technical essentials and clinical neurologic applications. Radiology. 2021 Apr;299(1):3-26.
5. Aker L, Abandeh L, Abdelhady M, Aboughalia H, Vattoth S. Susceptibility-weighted imaging in neuroradiology: practical imaging principles, pearls and pitfalls. Current Problems in Diagnostic Radiology. 2021 Jun 8.
6. Sotoudeh H, Sarrami AH, Wang JX, Saadatpour Z, Razaei A, Gaddamanugu S, Choudhary G, Shafaat O, Singhal A. Susceptibility-Weighted Imaging in Neurodegenerative Disorders: A Review. Journal of Neuroimaging. 2021 May;31(3):459-70.
7. Hsu CC, Kwan GN, Hapugoda S, Craigie M, Watkins TW, Haacke EM. Susceptibility weighted imaging in acute cerebral ischemia: review of emerging technical concepts and clinical applications. The neuroradiology journal. 2017 Apr;30(2):109-19.

8. Desai S. SWI, a new MRI sequence-how useful it is?. Indian Journal of Radiology and Imaging. 2006 Feb;16(01):13-4.
9. Ulivi L, Squitieri M, Cohen H, Cowley P, Werring DJ. Cerebral venous thrombosis: a practical guide. Practical neurology. 2020 Oct 1;20(5):356-67.
10. Prince EA, Ahn SH. Basic vascular neuroanatomy of the brain and spine: what the general interventional radiologist needs to know. InSeminars in interventional radiology 2013 Sep (Vol. 30, No. 03, pp. 234-239). Thieme Medical Publishers.
11. Haacke EM, Xu Y, Cheng YC, Reichenbach JR. Susceptibility weighted imaging (SWI). Magnetic Resonance in Medicine: An Official Journal of the International Society for Magnetic Resonance in Medicine. 2004 Sep;52(3):612-8.
12. Haacke EM, Mittal S, Wu Z, Neelavalli J, Cheng YC. Susceptibility-weighted imaging: technical aspects and clinical applications, part 1. American Journal of Neuroradiology. 2009 Jan 1;30(1):19-30.
13. Lee YJ, Lee S, Jang J, Choi HS, Jung SL, Ahn KJ, Kim BS, Lee KH. Findings regarding an intracranial hemorrhage on the phase image of a susceptibility-weighted image (SWI), according to the stage, location, and size. Investigative Magnetic Resonance Imaging. 2015 Jun 1;19(2):107-13.
14. Tong KA, Ashwal S, Holshouser BA, Nickerson JP, Wall CJ, Shutter LA, Osterdock RJ, Haacke EM, Kido D. Diffuse axonal injury in children: clinical correlation with hemorrhagic lesions. Annals of neurology. 2004 Jul;56(1):36-50.
15. Tong KA, Ashwal S, Holshouser BA, Shutter LA, Herigault G, Haacke EM, Kido DK. Hemorrhagic shearing lesions in children and adolescents with posttraumatic diffuse axonal injury: improved detection and initial results. Radiology. 2003 May;227(2):332-9.

16. Babikian T, Freier MC, Tong KA, Nickerson JP, Wall CJ, Holshouser BA, Burley T, Riggs ML, Ashwal S. Susceptibility weighted imaging: neuropsychologic outcome and pediatric head injury. Pediatric neurology. 2005 Sep 1;33(3):184-94.
17. Schelhorn J, Gramsch C, Deuschl C, Quick HH, Nensa F, Moenninghoff C, Schlamann M. Intracranial hemorrhage detection over time using susceptibility-weighted magnetic resonance imaging. Acta Radiologica. 2015 Dec;56(12):1501-7.
18. Mardanshahi Z, Tayebi M, Shafiee S, Barzin M, Shafizad M, Alizadeh-Navaei R, Gholinataj A. Evaluation of subacute subarachnoid haemorrhage detection using a magnetic resonance imaging sequence: Double inversion recovery. BioMedicine. 2020;10(4):29.
19. Sehgal V, Delproposto Z, Haddar D, Haacke EM, Sloan AE, Zamorano LJ, Barger G, Hu J, Xu Y, Prabhakaran KP, Elangovan IR. Susceptibility-weighted imaging to visualize blood products and improve tumor contrast in the study of brain masses. Journal of Magnetic Resonance Imaging: An Official Journal of the International Society for Magnetic Resonance in Medicine. 2006 Jul;24(1):41-51.
20. Wycliffe ND, Choe J, Holshouser B, Oyoyo UE, Haacke EM, Kido DK. Reliability in detection of hemorrhage in acute stroke by a new three-dimensional gradient recalled echo susceptibility-weighted imaging technique compared to computed tomography: a retrospective study. Journal of Magnetic Resonance Imaging: An Official Journal of the International Society for Magnetic Resonance in Medicine. 2004 Sep;20(3):372-7
21. Behrouzi R, Punter M. Diagnosis and management of cerebral venous thrombosis. Clinical Medicine. 2018 Feb;18(1):75.
22. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. The Lancet Neurology. 2007 Feb 1;6(2):162-70.

23. Kwon H, Choi DS, Jang J. Susceptibility-Weighted MR Imaging for the Detection of Isolated Cortical Vein Thrombosis in a Patient with Spontaneous Intracranial Hypotension. Investigative Magnetic Resonance Imaging. 2019 Dec 1;23(4): 381-4.

24. Manorenj S, Marupaka SK, Ramineni KK. Diagnostic Performances of Susceptibility weighted imaging Brain for the Detection of Cerebral Venous Thrombosis in Patients Presenting with Headache. headache.;100:100.

25. Raj S, Bhatia V, Bhatia P, Joshi M, Saini AG, Singh P. Imaging in Cerebral Sinovenous Thrombosis.

26. Leach JL, Fortuna RB, Jones BV, Gaskill-Shipley MF. Imaging of cerebral venous thrombosis: current techniques, spectrum of findings, and diagnostic pitfalls. Radiographics. 2006 Oct;26(suppl_1):S19-41.

27. Idbaih A, Boukobza M, Crassard I, Porcher R, Bousser MG, Chabriat H. MRI of clot in cerebral venous thrombosis: high diagnostic value of susceptibility-weighted images. Stroke. 2006 Apr 1;37(4):991-5.

28. Boukobza M, Crassard I, Bousser MG, Chabriat H. MR imaging features of isolated cortical vein thrombosis: diagnosis and follow-up. American Journal of Neuroradiology. 2009 Feb 1;30(2):344-8.

29. Hingwala D, Kesavadas C, Thomas B, Kapilamoorthy TR. Clinical utility of susceptibility-weighted imaging in vascular diseases of the brain. Neurology India. 2010 Jul 1;58(4):602.

30. Martins H, Carreiras M, Ribeiro M, Sousa P, Silva C. The influence of the blood pressure on the venous cerebral flow measured by magnetic susceptibility (SWI) technique. Journal of Biomedical Science and Engineering. 2013;6:426-34.

31. Matsushita T, Anami D, Arioka T, Inoue S, Kariya Y, Fujimoto M, Ida K, Sasai N, Kaji M, Kanazawa S, Joja I. Basic study of susceptibility-weighted imaging at 1.5 T. Acta Medica Okayama. 2008;62(3):159-68.
32. Haller S, Vernooij MW, Kuijer JP, Larsson EM, Jäger HR, Barkhof F. Cerebral microbleeds: imaging and clinical significance. Radiology. 2018;287(1):11-28
33. Verma SK, Kan EM, Lu J, Ng KC, Ling EA, Seramani S, Kn BP, Wong YC, Tan MH, Velan SS. Multi-echo susceptibility-weighted imaging and histology of open-field blast-induced traumatic brain injury in a rat model. NMR in Biomedicine. 2015 Sep;28(9):1069-77
34. Sahdev R, Rao A, Yadu N, Shukla Y. Role of susceptibility weighted imaging in characterization of intracranial Lesions, International Journal of Medical Research and Review.November, 2016; Vol 4/Issue 11; ISSN- 2321-127X
35. Rodemerk J, Junker A, Chen B, Pierscianek D, Dammann P, Darkwah Oppong M, Radbruch A, Forsting M, Maderwald S, Quick HH, Zhu Y. Pathophysiology of intracranial aneurysms: COX-2 expression, iron deposition in aneurysm wall, and correlation with magnetic resonance imaging. Stroke. 2020 Aug;51(8):2505-13.
36. Matsushige T, Chen B, Ringelstein A, Umutlu L, Forsting M, Quick HH, Sure U, Wrede KH. Giant intracranial aneurysms at 7T MRI. American Journal of Neuroradiology. 2016 Apr 1;37(4):636-41.
37. Samaniego EA, Roa JA, Hasan D. Vessel wall imaging in intracranial aneurysms. Journal of neurointerventional surgery. 2019 Nov 1;11(11):1105-12.
38. Matsushige T, Shimonaga K, Ishii D, Sakamoto S, Hosogai M, Hashimoto Y, Kaneko M, Ono C, Mizoue T, Kurisu K. Vessel wall imaging of evolving unruptured intracranial aneurysms. Stroke. 2019 Jul;50(7):1891-4.

39. Morita N, Harada M, Uno M, et al. Ischemic findings of T2*- weighted 3-TeslaMRI in acute stroke patients.Cerebrovasc Dis 2008; 26:367–75 CrossRef Medline

40. Hermier M, Nighoghossian N, Derex L, et al. Hypointense transcerebral veins at T2*-weighted MRI: a marker of hemorrhagic transformation risk in patients treated with intravenous tissue plasminogen activator. J Cereb Blood Flow Metab 2003;23:1362–7

41. Yu X, Yuan L, Jackson A, Sun J, Huang P, Xu X, Mao Y, Lou M, Jiang Q, Zhang M. Prominence of medullary veins on susceptibility-weighted images provides prognostic information in patients with subacute stroke. American Journal of Neuroradiology. 2016 Mar 1;37(3):423-9.21. Ferro JM, Canhao P, Stam J, Bousser MG, Barinagarrementeria F, Massaro A, et al. Delay in the diagnosis of cerebral vein and dural sinus thrombosis: influence on outcome. Stroke. 2009;40(9):3133-8.

42. Lamiaa G. El-Serougy , Mohamed M. El-Rakhawy , Germeen A. Ashamallah , Wessam F. Mustafa. Reliability of magnetic susceptibility weighted imaging in detection of cerebral microbleeds in stroke patients. The Egyptian Journal of Radiology and Nuclear Medicine. 2017; 48: 225–229

43. Zamora CA, Kontzialis M. Susceptibility-weighted imaging in Todd paralysis. Neurology. 2015 Jul 21;85(3):297

44. Radwan MM, Darwish RA, El Nekiedy AE, Shama SA. Role of magnetic susceptibility weighted imaging in evaluation of brain lesions. Alexandria Journal of Medicine. 2011 Dec 1;47(4):299-308

45. Bosemani T, Poretti A, Huisman TA. Susceptibility-weighted imaging in pediatric neuroimaging. Journal of Magnetic Resonance Imaging. 2014 Sep;40(3):530-44.

46. Wu Z, Mittal S, Kish K, Yu Y, Hu J, Haacke EM. Identification of calcification with MRI using susceptibility-weighted imaging: a case study. Journal of Magnetic Resonance Imaging: An Official Journal of the International Society for Magnetic Resonance in Medicine. 2009 Jan;29(1):177-82.
47. Hagemeier J, Weinstock-Guttman B, Bergsland N, Heininen-Brown M, Carl E, Kennedy C, Magnano C, Hojnacki D, Dwyer MG, Zivadinov R. Iron deposition on SWI-filtered phase in the subcortical deep gray matter of patients with clinically isolated syndrome may precede structure-specific atrophy. American journal of neuroradiology. 2012 Sep 1;33(8):1596-601.42.
48. Roh K, Kang H, Kim I. Clinical applications of neuroimaging with susceptibility weighted imaging. Journal of the Korean Society of Magnetic Resonance in Medicine. 2014 Dec 1;18(4):290-302.
49. Gunasingh M, Sharma P, Pitchandi M, Ramakrishnan KK, Muralidharan Y. Reliability in detection and differentiation between calcification and hemorrhage by 3 d GRE/SWI technique compared to CT. Journal of University of Shanghai for Science and Technology. 2021 Sep;23(9):17746.
50. 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.
51. Mittal P, Kalia V, Dua S. Pictorial essay: Susceptibility-weighted imaging in cerebral ischemia. Indian Journal of Radiology and Imaging. 2010 Oct;20(04):250-3
52. Eldeş T, Çeliker FB, Bilir Ö, Ersunan G, Yavaşi Ö, Turan A, Toprak U. How important is susceptibility-weighted imaging in mild traumatic brain injury. Ulus Travma Acil Cerrahi Derg. 2020 Jul 1;26(4):574-9.

53. Lai PH, Chang HC, Chuang TC, Chung HW, Li JY, Weng MJ, Fu JH, Wang PC, Li SC, Pan HB. Susceptibility-weighted imaging in patients with pyogenic brain abscesses at 1.5 T: characteristics of the abscess capsule. American journal of neuroradiology. 2012 May 1;33(5):910-4.
54. Jagadeesan BD, Delgado Almandoz JE, Moran CJ, Benzinger TL. Accuracy of susceptibility-weighted imaging for the detection of arteriovenous shunting in vascular malformations of the brain. Stroke. 2011 Jan;42(1):87-92.
55. Sultan AY, ElShafey KI, Hassanien OA, Dawoud RM. Magnetic resonance susceptibility weighted in evaluation of cerebrovascular diseases. Egyptian Journal of Radiology and Nuclear Medicine. 2020 Dec;51(1):1-2.
56. Abdelgawad MS, Aly RA. Value of susceptibility-weighted MR imaging (SWI) in the detection of developmental venous anomaly. Egyptian Journal of Radiology and Nuclear Medicine. 2020 Dec;51(1):1-8.
57. Hwang I, Sohn CH, Kang KM, Jeon BS, Kim HJ, Choi SH, Yun TJ, Kim JH. Differentiation of Parkinsonism-predominant multiple system atrophy from idiopathic Parkinson disease using 3T susceptibility-weighted MR imaging, focusing on putaminal change and lesion asymmetry. American Journal of Neuroradiology. 2015 Dec 1;36(12):2227-34.
58. Zulfiqar M, Dumrongpisutikul N, Intrapiromkul J, Yousem DM. Detection of intratumoral calcification in oligodendrogliomas by susceptibility-weighted MR imaging. American journal of neuroradiology. 2012 May 1;33(5):858-64.
59. Thamburaj K, Radhakrishnan VV, Thomas B, Nair S, Menon G. Intratumoral microhemorrhages on T2*-weighted gradient-echo imaging helps differentiate vestibular schwannoma from meningioma. American Journal of Neuroradiology. 2008 Mar 1;29(3):552-7.

60. Blitstein MK, Tung GA. MRI of cerebral microhemorrhages. American Journal of Roentgenology. 2007 Sep;189(3):720-5.
61. Tsushima Y, Tanizaki Y, Aoki J, Endo K. MR detection of microhemorrhages in neurologically healthy adults. Neuroradiology. 2002 Jan;44(1):31-6.
62. Punitha P, Rajamanickam K, Krishnamoorthy A, Einstein A. Imaging Cerebral Haemorrhage using MRI: Improved Sensitivity of Susceptibility Weighted Imaging (SWI) Compared to Gradient Echo Sequences (GRE). Archives of Internal Medicine Research. 2020;3(1):18-25.
63. Docampo J, Cabrini M, Bruno C, Morales C. Tuberous sclerosis: evaluation of intracranial lesions. Rev. Argent. Radiol. 2013;77(4):275-83.
64. Hu J, Yu Y, Juhasz C, Kou Z, Xuan Y, Latif Z, Kudo K, Chugani HT, Haacke EM. MR susceptibility weighted imaging (SWI) complements conventional contrast enhanced T1 weighted MRI in characterizing brain abnormalities of Sturge-Weber syndrome. Journal of Magnetic Resonance Imaging: An Official Journal of the International Society for Magnetic Resonance in Medicine. 2008 Aug;28(2):300-7.
65. Horie N, Morikawa M, Nozaki A, Hayashi K, Suyama K, Nagata I. “Brush sign” on susceptibility-weighted MR imaging indicates the severity of moyamoya disease. American journal of neuroradiology. 2011 Oct 1;32(9):1697-702.
66. Huang Y, Li J, Jin M, Sun X, Li J, Liu Y, Xi Y, Wang Q, Zhao W. Imaging of Moyamoya Disease and Moyamoya Syndrome: Current Status.

ANNEXURE – I –CONSENT FORM

TITLE OF THE STUDY: "CLINICAL APPLICATIONS OF SUSCEPTIBILITY - WEIGHTED MAGNETIC RESONANCE IMAGING AMONG PATIENTS WITH NEUROGICAL DISORDERS AT 3T MRI– A HOSPITAL BASED CROSS SECTIONAL STUDY"

PRINCIPAL INVESTIGATOR: Dr.

INTRODUCTION AND PURPOSE: The advent of high resolution SWI, the current tool, has significantly increased the chances of identifying a cause resulting in a positive clinical impact on the management of the patients with microbleeds in ageing and occult low-flow vascular malformations, in characterizing brain tumors and degenerative diseases of the brain, and in recognizing calcifications in various pathological conditions, for high-resolution venous anatomy, evaluation of tissue susceptibility and cerebro vascular diseases. SWI will help in early diagnosis and allow for a better and early treatment plan.

PROCEDURE: I request you to kindly participate in the study titled study **"CLINICAL APPLICATIONS OF SUSCEPTIBILITY-WEIGHTED MAGNETIC RESONANCE IMAGING AMONG PATIENTS WITH NEUROGICAL DISORDERS AT 3T MRI– A HOSPITAL BASED CROSS SECTIONAL STUDY"** at Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belgaum" is being conducted by Dr. _____ Post Graduate in Radio-Diagnosis at J. N. Medical College Belgaum, Karnataka, under the guidance of **DR. _____** Professor, Dept. 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 clinically examined as per the protocol drawn.

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 organs and 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-shaped machine. 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 diagnosis of the disease in patients with calcification CVT abscess, developmental venous anomaly, multiple sclerosis tumors, etc…, and hence allow for a better and early treatment plan. Non invasive, cost effective modality

COMPLICATIONS: No risk to the patient has been documented from SWI conducted earlier.

RISKS: No risks have been documented so far.

ALTERNATIVES: If patient is not willing to take part in the study, his / her treatment or any other further investigations the patient wants to undergo, in future, in KLE will not be affected by his / her decision.

VOLUNTARY PARTICIPATION/WITHDRAWAL: Taking part in this study is voluntary. I may choose not to take part in this study, or if I decide to take part I can later change my mind and withdraw from the study. My decision will not change the present or future health care or other services that I receive. The study doctor or the sponsor may stop my participation in this study. I will tell if any important new findings that may change my willingness to continue to take part. If I choose not to take part in the study I will receive the standard treatment for patients with my condition.

COSTS: NIL (The study is to be conducted on the participants who are advised for SWI as an investigation by the referring consultant and the participants will bear the charges for it).

PAYMENT FOR PARTICIPATION: No incentive will be paid to you for participating in this study.

COMPENSATION: In the event that I become injured as a result of taking part in this study, treatment whatever available at KLE Charitable Hospital, Belagavi, will be offered to me. No reimbursement, compensation or free medical care is given.

CONFIDENTIALITY: All information collected about me during the course of the study will be kept confidential to the extent permitted by the law. The code numbers will identify me in this research record. Information from this study may be published but my identity will be confidential in any publication.

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

DR. HARSHA HEGDE,
CHAIRPERSON, JNMC, IEC & Scientist D, ICMR, NATIONAL INSTITUTE OF TRADITIONAL MEDICENE, BELAGAVI
Ph. No: 9480422500

CONSENT TO PARTICIPATE IN RESEARCH STUDY:

1. I understand that I am participating in the study, which includes Susceptibility-Weighted Imaging Beyond Hemorrhage
2. I confirm that I have read and understood the information in the patient information sheet. Procedure is explained to me in detail along with information about the advantages and disadvantages of taking part in the study. I have been given the opportunity to discuss all aspects of the trial, to ask questions and hereby consent to participation in the trial outlined above.
3. I understand that the decision to take part in this study is completely voluntary and I am aware that I can choose to withdraw from the study at any point of time.
4. I consent to the photographing or recording of the procedure to be performed including appropriate portions of my body, for medical, scientific or educational purposes provided my identity is not revealed in the pictures or by the descriptive texts accompanying them.
5. I understand that there is no significant risk involved in the test that would be done in this study.
6. No guarantee or assurance has been given by anyone as to the results that may be obtained.
7. My signature on this form signifies that I have willingly decided to participate after understanding the above information.

Participant's Name/ legally authorized _____

representative

Signature _____

Name and signature of witness _____

Name and signature of interviewer _____

Date:

Place:

ANNEXURE – II –PROFORMA

NAME_____

AGE_____ OP/IP NO _____

ADDRESS _____

MRI NUMBER:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

PAST HISTORY:

FAMILY HISTORY:

MRI:

Blooming on SWI sequence:

Type of blooming on Phase: hypointense/ hyperintense

Signal intensity on T1 weighted imaging:

Signal intensity on T2 weighted imaging:

Region of blooming on SWI:

Associated Parenchymal abnormalities:

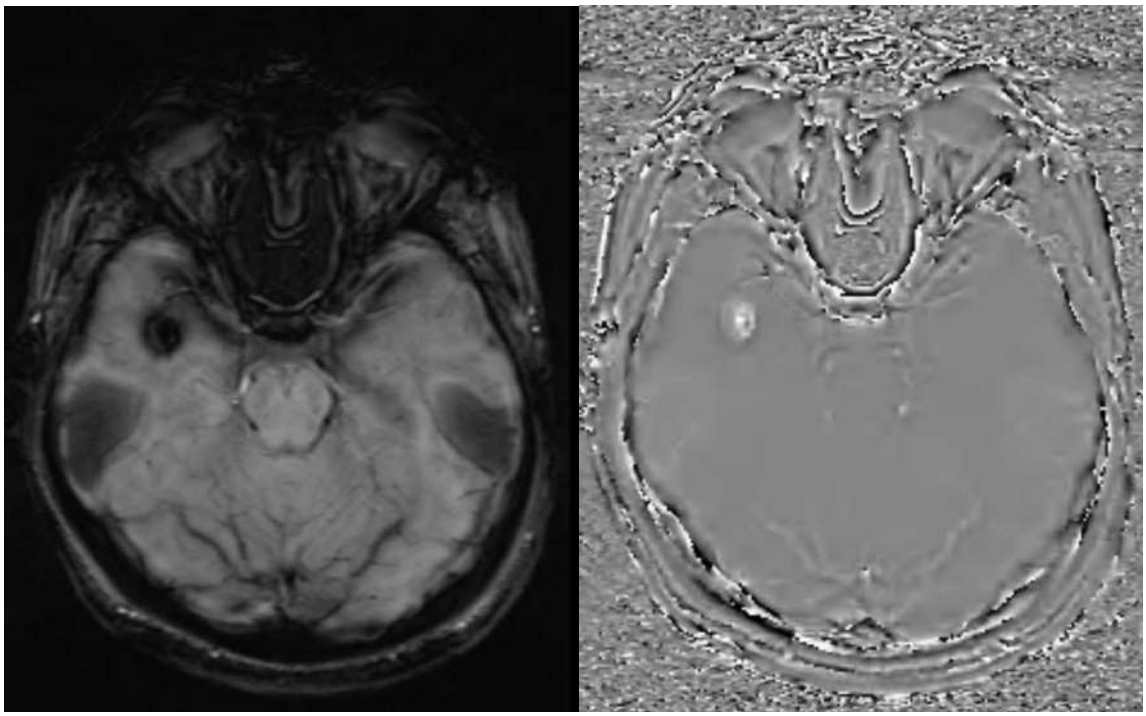
- Edema
- Cortical veins blooming
- Hypertensive changes
- Age related changes

ANNEXURE – III – PHOTOGRAPHS

PHOTOGRAPH OF CASES

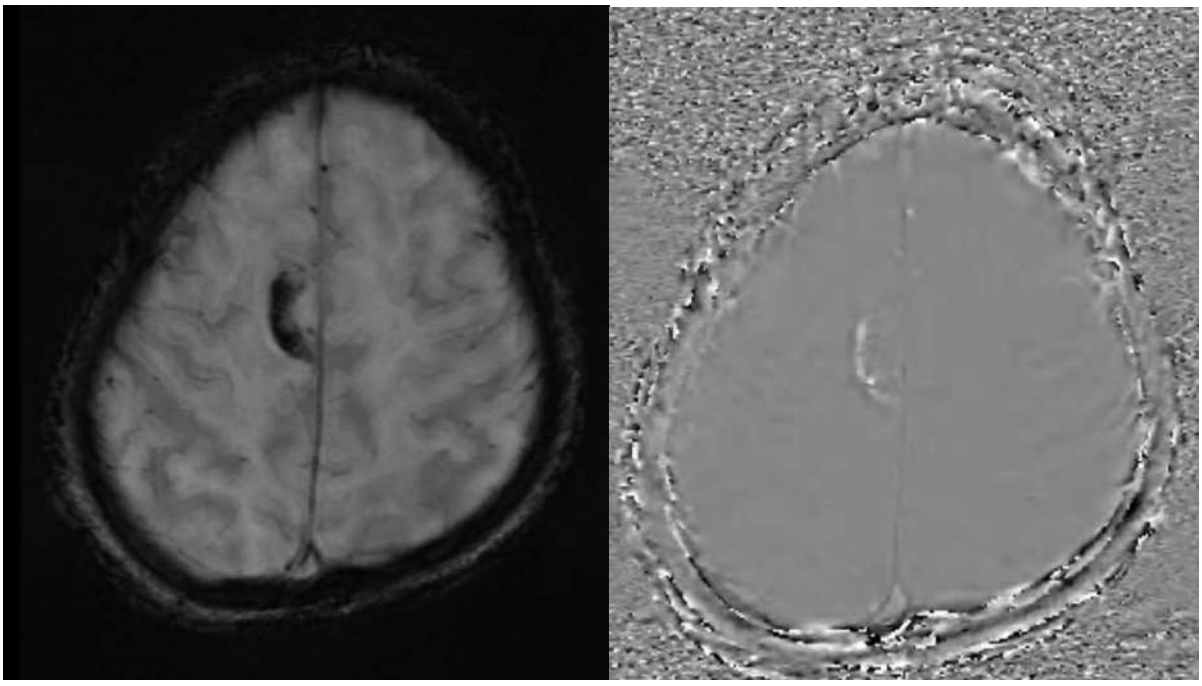
CASE 1: 17 years old male presented with confusion and new onset seizures since 4 days.

A well-defined rounded lesion in the right temporal region which shows hyperintense blooming on SWI and Phase sequences suggestive of cavernoma.



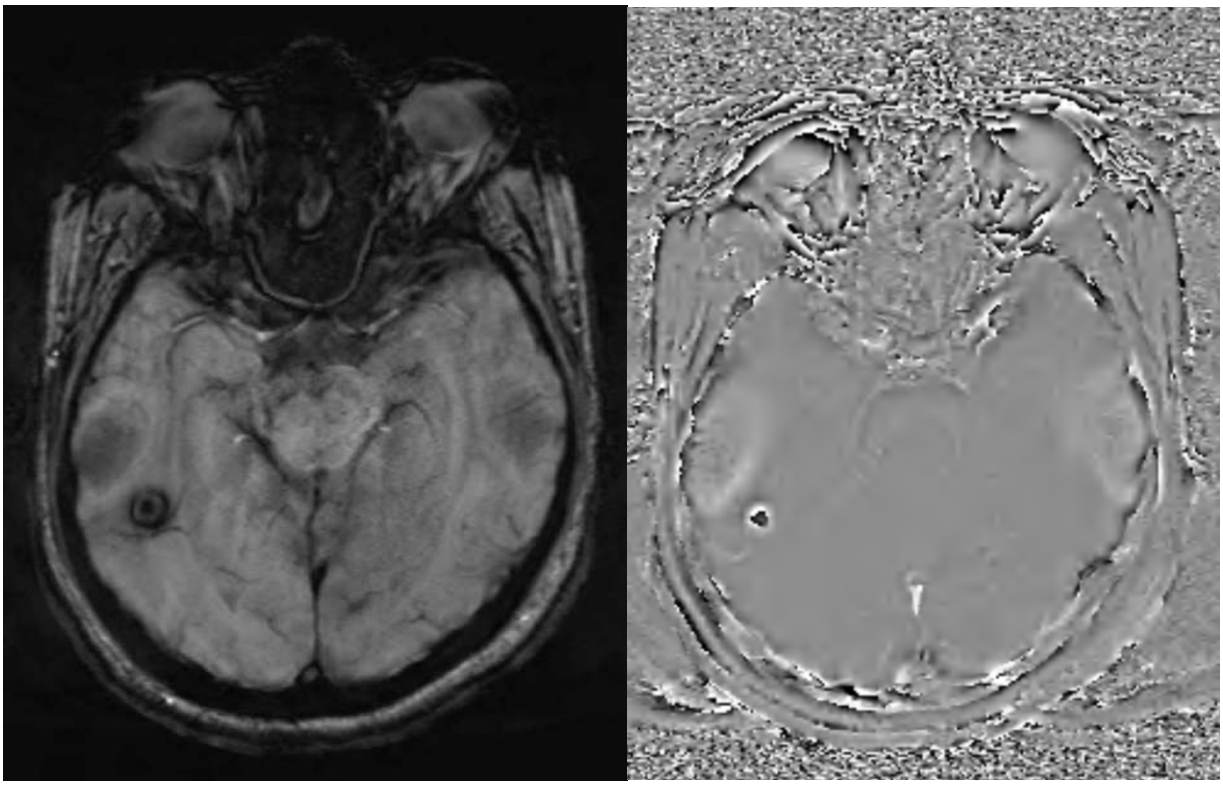
CASE 2: A 83 year old presented with blurring of vision and bilateral lower limb weakness since 4 days

SWI and Phase sequences shows hyperintense blooming (bleed) with areas of infarct in right frontal region of conventional MRI sequences suggestive of infarct with hemorrhagic transformation.



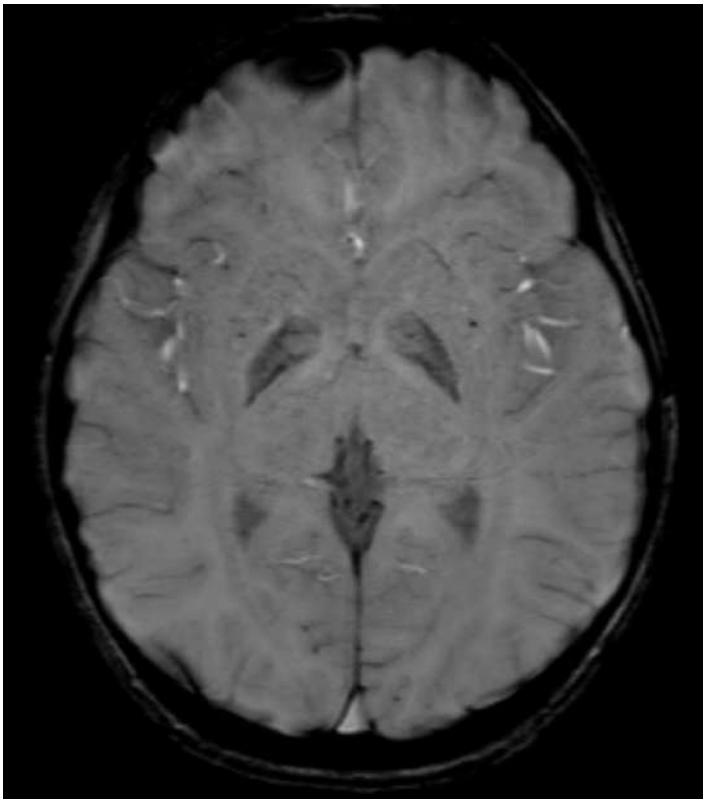
CASE 3: A 45 year old female presented with a history of seizures since 4 days

A well defined rounded lesion was seen in the right temporal region which shows hyperintense blooming on SWI and hypointensity on Phase (calcium). The lesion was seen to have scolex on T2 images suggestive of neurocysticercosis.



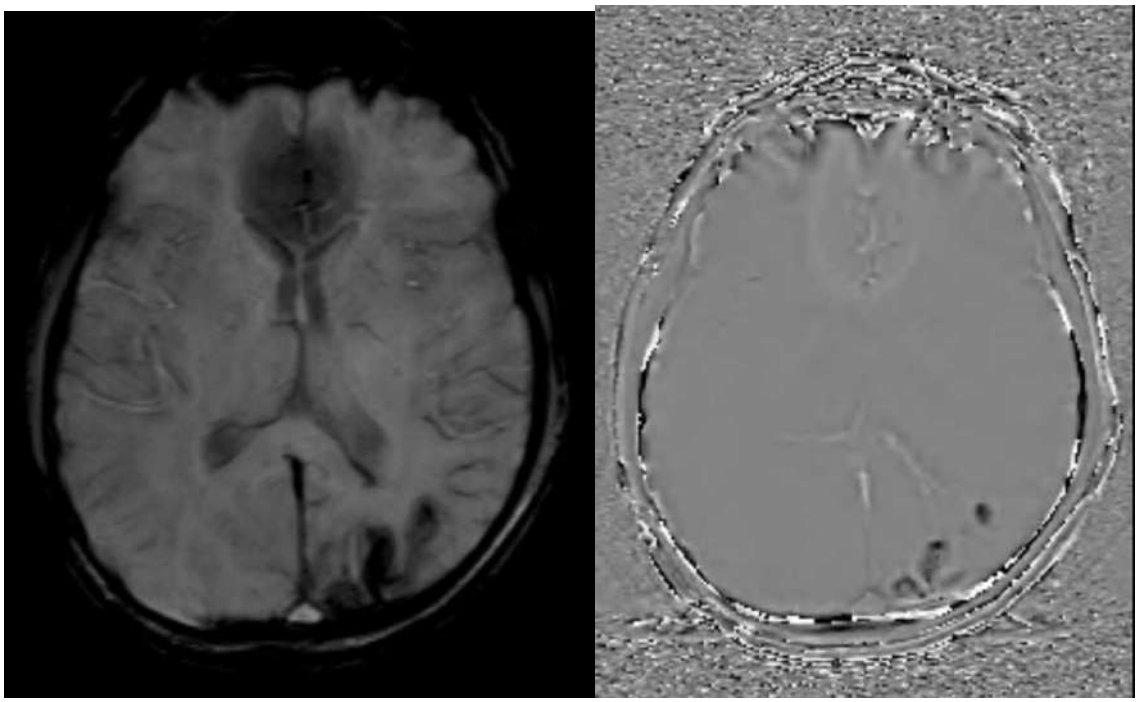
CASE 4: A 8 year old male presented with dementia, rigidity and dysarthria since 8 days.

There is seen symmetrical blooming on hyperintense SWI sequence in bilateral globus pallidi (eye of tiger appearance) suggestive of hemosiderin deposition seen in pantothenate kinase associate neurodegeneration (PKAN)



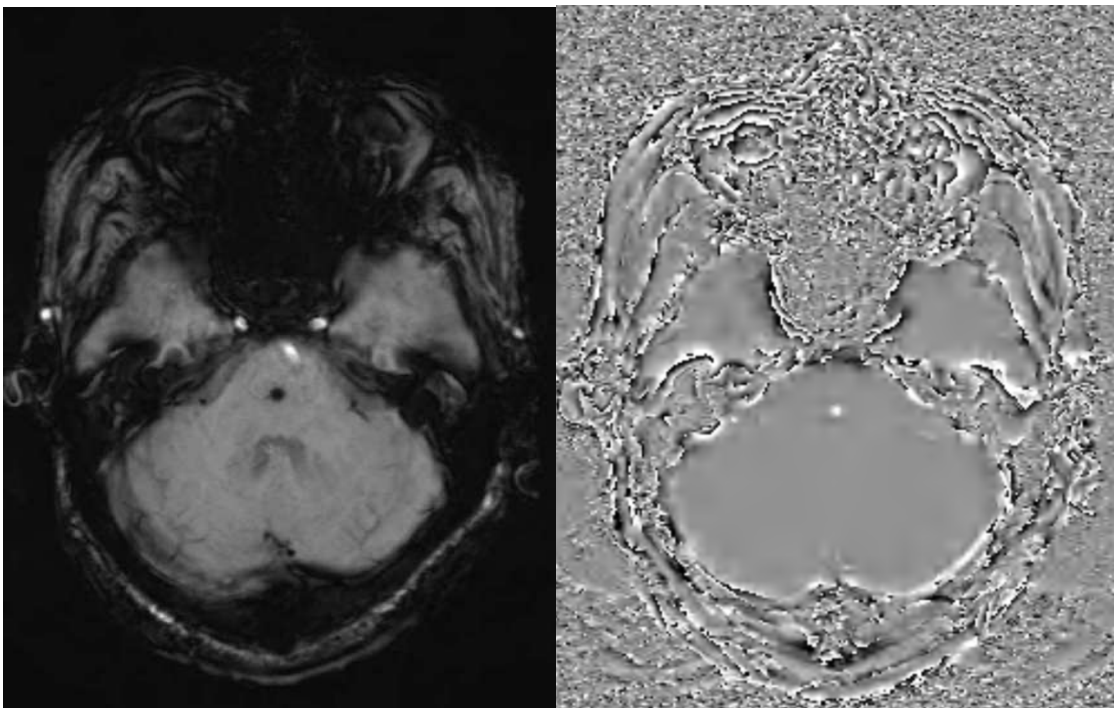
CASE 5: A 13 year old male case had port wine stain and seizures since 3 years.

Hyperintense blooming on SWI noted in the subcortical region of left parietal and occipital region which shows hypointense blooming on phase sequende (calcificantion)…based on the above mentioned history and MRI findings the case was diagnosed as sturge weber syndrome.



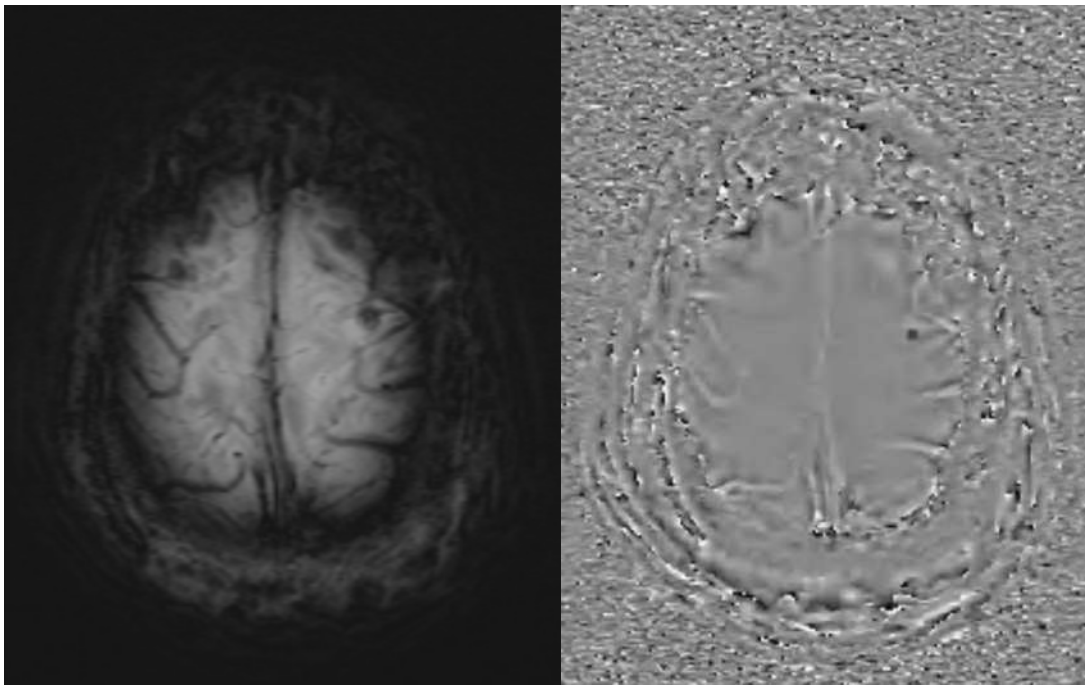
CASE 6: A 60 year old male subject had a history of hypertension since last 6 years

SWI and Phase images shows a focal area of hyperintense blooming (bleed) in the pons suggestive of hypertensive microangiopathy



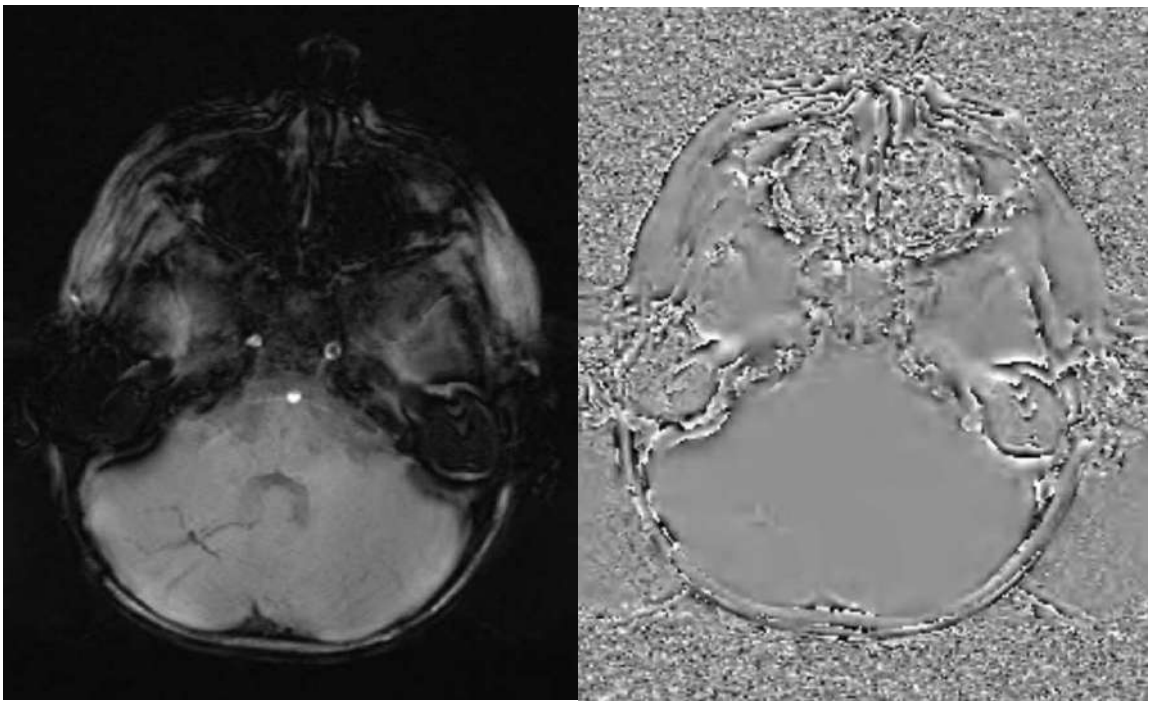
CASE 7: A 18 year of female presented with history of head and seizures since 17 days

Focal area of hyperintense blooming on SWI and hypointense blooming on Phase images (calcium) is seen in the left high frontal region, features favoring the possibility of tuberculoma



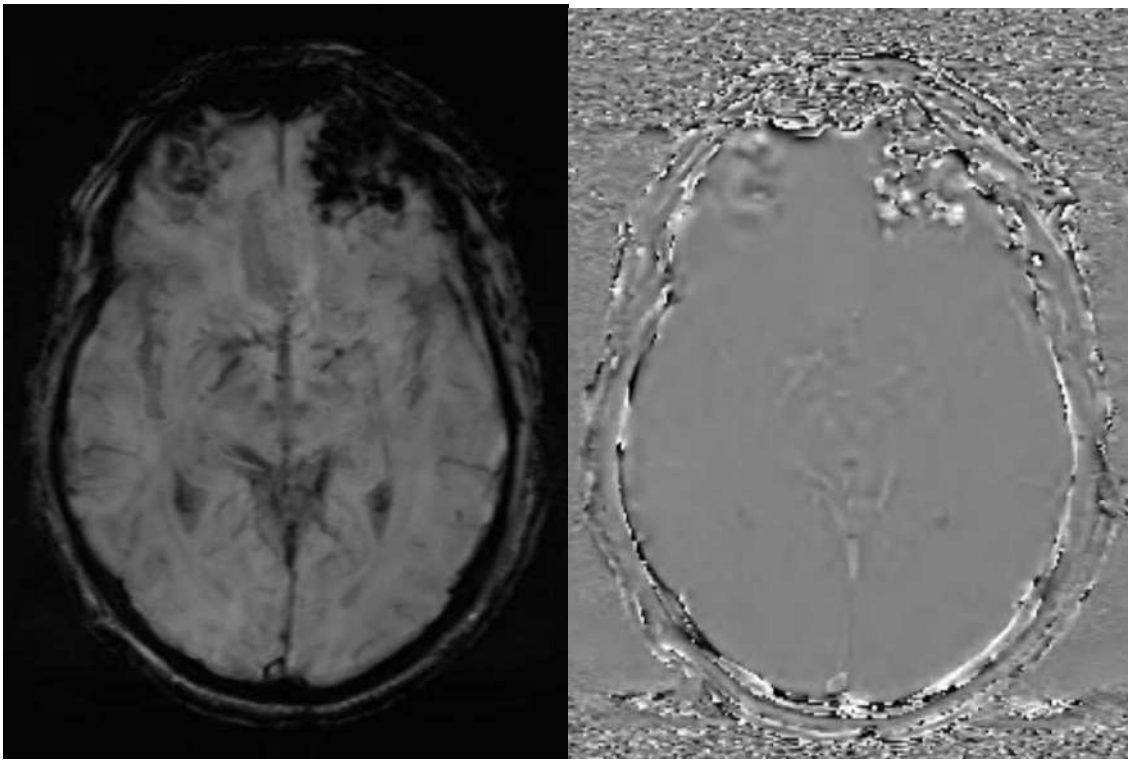
CASE 8: A one year old child presented with developmental delay of motor mile stones

Bunch of few veins noted in right cerebellar hemisphere (medusa head appearance) likely to be developmental venous anomaly.



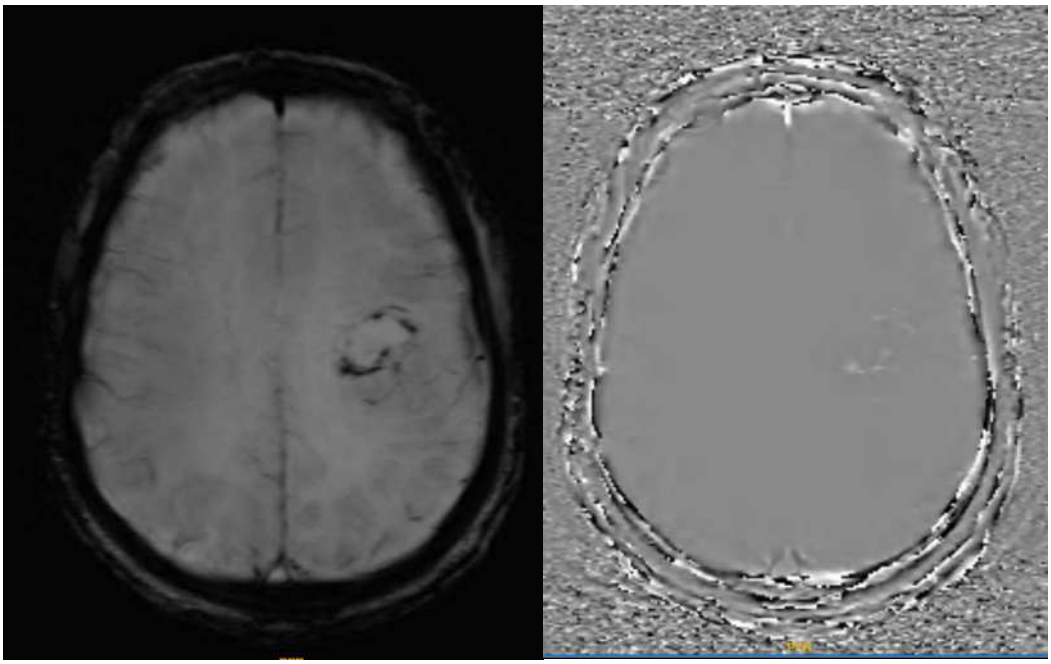
CASE 9: A 68 year old male case had history of trauma 1 day ago

Areas of hyperintense blooming are seen in the left frontal region on both SWI & Phase images (bleed) suggestive of hemorrhagic contusions.



CASE 10: A 60 year male presented with headache and altered sensorium since 20 days.

A well defined lesion was seen in the left fronto-parietal region which showed areas of hyperintense blooming on SWI & Phase images (bleed within the lesion).



ANNEXURE – IV – KEY TO MASTERCHART

Hypointense blooming on SWI and hyperintense blooming on Phase indicating: Bleed/Hemosiderin

Hypointense blooming on SWI and hypointense blooming on Phase indicating calcium

HYPOINTENSE BLOOMING ON SWI COLUMN	**Y**
HYPERINTENSE BLOOMING ON PHASE COLUMN	**Y**
HYPOINTENSE BLOOMING ON PHASE COLUMN	**N**

SUPRATENTORIAL BRAIN PARENCHYMA	**S**
INFRATENTORIAL BRAIN PARENCHYMA	**I**

YES	**+**
NO	**-**

HTN	**Hypertension**
DM	**Diabetes mellitus**
MI	**Myocardial infarction**

Male	**M**
Female	**F**

												T1	T2	SWI	PHASE	MR FINDINGS
S.NO	**AGE**	**SEX**	**MRI NO**	**CHIEF COMPLAINTS**	**DURATION**	**CO-MORBIDITIES**	**UNDERLYING CAUSE**		**SUPRA/ INFRATENTORIAL**	**REGION**	**PROV DIAG**					
							VASCULAR	NONVASCULAR								
1	46	F	25076	HEADACHE	1 month	HTN	-	+	S	LEFT PARIETO-TEMPORAL	GLIOBLASTOMA MULTFORME	ISO	HYPER	Y	Y	BLEED
2	24	M	25088	DEMENTIA AND HEADACHE	15 days	-	+	-	S	LEFT LENTIFORM NUCLEUS	LACUNAR INFARCTS	HYPO	HYPER	Y	Y	HEMOSIDERIN
3	28	M	25166	HEADCAHE AND BLURRING OF VISION	1 month	-	-	+	S	LEFT FRONTO-TEMPORAL	ANAPLASTIC ASTROCYTOMA	HYPO	HYPER	Y	Y	HEMOSIDERIN
4	11	M	27297	SEIZURES	15 DAYS	-	-	+	S	BILATERAL PARIETAL AND LEFT TEMPORAL	TUBERCULOMA	HYPO	HYPER	Y	N	CALCIUM
5	47	M	27520	HEADACHE & SEIZURES	3 WEEKS	DM	-	+	S	BILATERAL PARASAGGITAL PARAFALCINE FRONTAL	HIGH GRADE GLIOMA	ISO	MIXED	Y	Y	BLEED
6	35	F	27556	GIDDINESS	2 MONTH	-	-	+	S	BODY OF RIGHT LATERAL VENTRICLE	CENTRAL NEUROCYTOMA	ISO	HYPER	Y	N	CALCIUM
7	48	M	27568	HEADACHE	20 DAYS	-	-	+	S	RIGHT PARIETO-OCCIPITAL	GLIOBLASTOMA MULTIFORME	ISO	MIXED	Y	Y	BLEED
8	60	M	27334	GIDDINESS & IMBALANCE	4 HOURS	HTN	+	-	I	MIDBRAIN ON LEFT	HYPERACUTE INFARCT IN MB & PONS	-	-	Y	Y	BLEED
9	75	F	20774	RIGHT SIDED WEAKNESS	9 HOURS	HTN & MI	+	-	S	LEFT FRONTAL & INSULAR	LATE HYPERACUTE INFARCT, LT MCA INFARCT	-	HYPER	Y	Y	BLEED
10	44	F	27062	SEIZURES	4 DAYS	-	-	+	S	RIGHT TEMPORAL	NEUROCYSTICERCOSIS	HYPO	HYPO WITH CENTRAL HYPERINTENSITY	Y	Y	CALCIUM
11	45	M	27126	LEFT SIDED WEAKNESS	3 DAYS	-	+	-	S	RIGHT FRONTO-PARIETO-TEMPORAL	SUBACUTE INFARCT WITH HEMORRGAFIC TRANSFORMATION	HYPER	HYPER	Y	Y	BLEED
12	62	F	27131	headache & vomitings	15 DAYS	DM & MI	-	+	S	RIGHT FRONTAL	HIGH GRADE GLIOMA	HYPO	MIXED	Y	Y	BLEED
13	60	M	27152	HEADACHE & GIDDINESS	20 DAYS	HTN	-	+	S	RIGHT GANGLIOCAPSULAR	GLIOBLASTOMA MULTFORME	HYPO	HYPER	Y	Y	BLEED
14	36	M	27429	HEADACHE & COUGH	2 MONTHS	-	-	+	S	BL F-P REGION, BL SYLVIAN FISSUES, ANTERIOR FALX CEREBRI	TUBERCULAR MENINGITIS WITH BASAL EXUDATES	HYPO	HYPER	Y	N	CALCIUM
15	17	M	27523	CONFUSION & NEW ONSET SEIZURES	4 DAYS	-	+	-	S	RIGHT TEMPORAL	CAVERNOMA	HYPER	HYPO	Y	Y	BLEED
16	10	M	27924	SEIZURES	2 DAYS	-	-	+	S	BL GLOBUS PALLIDUS	MINERAL DEPOSTION	HYPO	HYPER	Y	N	HEMOSIDERIN
17	48	M	28420	LOSS OF CONSIOUSNESS	2 HOURS	-	+	-	S	LEFT POSTERIOR PARIETAL REGION	ACUTE INTRAPARENCHYMAL HEMORRHAGE	ISO	HYPO	Y	Y	BLEED
18	83	M	28415	BLURRING OF VISION & BILATERAL LL WEAKNESS	4 DAYS	MI	+	-	S	BILATERAL FRONTAL	SUBACUTE INFARCT WITH HEMORRHAGIC TRANSFORMATION	HYPER	HYPO	Y	Y	BLEED
19	72	M	28418	GENERALIZED WEAKNESS & HEADACHE	30 DAYS	HTN & MI	-	+	S	PUTAMEN	MULTISYSTEM ATROPHY-PREDOMINENT PUTAMEN (MSA-P)	HYPO	HYPO	Y	N	CALCIUM
20	60	M	28412	HEADACHE & GIDDINESS	25 DAYS	HTN & DM	-	+	S	LEFT HIGH FRONTAL	CALCIFIED MENINGIOMA	HYPO	HYPER	Y	N	CALCIUM
21	65	M	28390	GIDDINESS	7 DAYS	DM	+	-	S	RIGHT ANTERIOR TEMPORAL	GLIOSIS WITH HEMOSIDERIN DEPOSITION	HYPO	HYPER	Y	Y	HEMOSIDERIN
22	64	M	28321	PINPOINT PUPILS & BREATHLESSNESS	3 DAYS	HTN & DM	+	-	I	PONS	PONTINE HEMORRHAGE	HYPER	HYPO	Y	Y	BLEED
23	22	M	28317	HEADACHE & BLURRING OF VISION	20 DAYS	-	-	+	S	RIGHT FRONTAL PARAFALCINE	DIFFUSE ASTROCYTOMA	HYPO	HYPER	Y	N	CALCIUM
24	28	F	28313	SEIZURES & COUGH	8 DAYS	-	-	+	S	RIGHT PARIETO-OCCIPITAL	TUBERCULOMA	HYPO	HYPO	Y	N	CALCIUM
25	39	M	28301	LOSS OF CONSIOUSNESS WITH HYPERTENSION	3 HOURS	-	+	-	S	LEFT FRONTO-PARIETO-TEMPORAL	HYPERACUTE BLEED	ISO	HYPER	Y	Y	BLEED
26	60	F	28301	HEADACHE & ALTERED SENSORIUM	3 DAYS	DM	-	+	S	RIGHT TEMPORAL REGION	DURAL MENINGIOMA	ISO	ISO	Y	N	CALCIUM
27	36	M	28351	HEADACHE & SEIZURES	2 DAYS	-	+	-	S	RT PARITO-OCCIPITAL	HEMORRHAGIC VENOUS INFARCT	HYPER	HYPER	Y	Y	BLEED
28	74	M	28224	RIGHT SIDED WEAKNESS	3 DAYS	HTN & MI	+	-	S	LEFT FRONTAL	SUBACUTE INFARCT WITH HEMORRGAFIC TRANSFORMATION	HYPER	HYPER	Y	Y	BLEED
29	32	M	28208	HEADACHE & GIDDINESS	10 DAYS	-	+	-	I	RIGHT CEREBELLAR FOLIAE	OLD INTRAPARENCHYMAL HEMORRHAGE WITH SUPERFICIAL SIDEROSIS	HYPO	MIXED	Y	Y	HEMOSIDERIN
30	52	M	28154	SEIZURES & HEADACHE	15 DAYS	-	-	+	S	LEFT POSTERIOR PARIETAL REGION	TUBERCULAR RING ENHACING LESIONS	HYPO	MIXED	Y	N	CALCIUM
31	35	M	28100	FOCAL NEUROLOGICAL DEFICIT & SEIZURE	5 DAYS	-	+	-	S	BILATERAL PARIETO-TEMPORAL	CAVERNOUS MALFORMAION	HYPER	HYPO	Y	Y	BLEED
32	57	M	28947	HEADACHE & SEIZURES	10 DAYS	HTN	+	-	S	RIGHT PARIETAL (RIGHT PERICALLOSAL ARTERY DRAINING	ARTERIO-VENOUS MALFORMATION	HYPER	HYPO	Y	Y	BLEED
33	37	M	25897	HEADCHE AND DISORIENTED	15 DAYS	-	+	-	S	BILATERAL SUPRACLINOID INTERNAL CAROTID ARTERY	MOYA-MOYA	HYPER	HYPO	Y	Y	BLEED
34	46	M	26543	SEIZURES AND RIGHT SIDED WEAKNESS	4 DAYS	-	+	-	S	RIGHT FRONTO-PARIETAL REGION	DEVELOPMENTAL VENOUS ANOMALY	HYPER	HYPO	Y	Y	BLEED
35	38	F	28499	BILATERAL UPPER LIMB WEAKNESS & SPASTICITY	20 DAYS	-	-	+	S	LEFT PARIETAL (CENTRAL VEIN SIGN)	MULTIPLE SCLEROSIS	HYPO	HYPER	Y	Y	HEMOSIDERIN
36	68	M	24337	TREMORS & RIGIDITY	30 DAYS	DM & MI	-	+	S	BILATERAL GLOBUS PALLIDI, RED NUCLEUS, PUTAMEN, SUBSTANTIA NIGRA	PARKINSONS DISEASE	HYPO	HYPO	Y	Y	HEMOSIDERIN
37	32	F	29778	HEADACHE & GIDDINESS	5 DAYS	-	+	-	S	RIGHT PERI-ROLANDIC CORTICAL REGION	SUBACUTE HEMORRHAGIC VENOUS INFARCT	HYPER	HYPER	Y	Y	BLEED
38	50	M	28616	FOCAL NEIROLOGICAL DEFICIT & HEADCHE	7 DAYS	HTN	+	-	S	LEFT PARIETO-TEMPORAL	HEMORRHAGIC VENOUS INFARCT	HYPER	HYPER	Y	Y	BLEED
39	18	M	28633	HEADACHE & SEIZURES	17 DAYS	-	-	+	S	LEFT HIGHT FRONTAL	CALCIFIED GRANULOMA… TUBERCULAR ETIOLOGY	HYPO	HYPO	Y	N	CALCIUM
40	54	F	28604	CARCINOMA LUNG	26 MONTHS	-	-	+	S	RIGHT HIGH FRONTAL	CEREBRAL METASTASIS	ISO	HYPER	Y	Y	BLEED
41	38	F	28627	HEADACHE WITH LOSS OF BALANCE	5 DAYS	-	+	-	S	LEFT THALAMUS AND RIGHT CEREBELLAR LOBE	SUBACUTE VENOUS INFARCT	HYPER	HYPO	Y	Y	BLEED
42	38	M	28644	HEADACHE & PAST H/O COVID-19 PNEUMONIA	10 DAYS	-	+	-	S	STRAIGHT SINUS, LEFT TRANSVERSE SINUS, LEFT SIGMOID SINUS	CEREBRAL VENOUS THROMBOSIS	HYPER	-	Y	Y	BLEED
43	8	M	29546	DEMENTIA, RIGIDITY AND DYSARTHRIA	8 DAYS	-	-	+	S	BILATERAL GLOBUS PALLIDI (EYE OF TIGER APPEARANCE)	PANTOTHENATE KINASE ASSOCIATE NEURODEGENERATION (PKAN)	HYPO	HYPER	Y	N	CALCIUM
44	62	M	24802	HEADCHE WITH BLURRING OF VISION	15 DAYS	DM	-	+	S	RIGHT FRONTAL	HIGH GRADE GLIOMA	HYPER	MIXED	Y	Y	BLEED
45	1 DAY	M	18813	MACROCEPHALY WITH NON-RESPONSIVESS	1 DAY	-	-	+	S	LEFT CRANIAL VAULT	DESMOPLASTIC INFANTILE GANGLIOGLIOMA	ISO	ISO	Y	Y	BLEED
46	25	M	23193	SEIZURES	70 DAYS	-	-	+	S	LEFT ANTERO-MEDIAL TEMPORAL LOBE	DYSEMBYOPLASTIC NEUROEPITHELIAL TUMOR (DNET)	HYPO	HYPER	Y	N	CALCIUM
47	48	M	18851	BLURRING OF VISION	3 DAYS	-	+	-	S	LEFT OPHTHALMIC INTERNAL CAROTID ARTERY & BASILAR ARTERY	ANEURYSMS	HYPER	ISO	Y	Y	BLEED
48	15	F	17849	ATAXIA AND SLURRING OF SPEECH	35 DAYS	-	-	+	I	BILATERAL DENTATE NUCLEUS	VERMIAN AGENESIS	HYPO	HYPO	Y	N	CALCIUM
49	79	M	21615	DOWNWARD GAZE WITH LACK OF PUPILLARY RESPONSE	1 DAY	-	+	-	S	LEFT THALAMUS	HYPERACUTE TO ACUTE HEMORRHAGE IN LEFT THALAMUS	HYPER	HYPO	Y	Y	BLEED
50	51	F	21437	CARCINOMA BREAST	36 MONTHS	DM	-	+	S	LEFT PARITAL	CHRONIC BLEED	HYPO	HYPO	Y	Y	BLEED

51	85	F	21894	LOSS OF BALANCE	5 DAYS	HTN & MI	+	-	I	RIGHT CEREBELLAR LOBE	GLIOSIS WITH HEMOSIDERIN DEPOSITION	HYPO	HYPER	Y	Y	HEMOSIDERIN
52	70	M	23746	CARCINOMA OVARY	30 MONTHS	HTN & DM	-	+	S & I	LEFT FRONAL AND RIGHT CEREBELLAR LOBE	HEMORRHAGIC METASTASIS	HYPER	HYPO	Y	Y	BLEED
53	33	M	25124	HEADCHE AND DISORIENTED	2 ADYS	-	+	-	S	RIGHT HIGHT FRONTAL AND CENTRUM SEMI OVALE	HEMORRHAGIC VENOUS INFARCT	ISO	HYPER	Y	Y	BLEED
54	71	M	22250	HYPERTENSION	75 MONTHS	HTN	+	-	I	PONS	HYPERTENSVE MICROANGIOPATHY	HYPER	HYPO	Y	Y	BLEED
55	74	F	19762	RIGHT SIDED WEAKNESS	2 DAYS	HTN	+	-	S	LEFT PARIETO-TEMPORAL	SUBACUTE INFARCT WITH HEMORRHAGIC TRANSFORMATION	HYPER	HYPER	Y	Y	BLEED
56	83	M	24262	HEADACHE & BLURRING OF VISION	3 DAYS	DM & MI	+	-	S	BILATERAL THALAMUS	THALAMIC MICROBLEEDS	HYPER	HYPER	Y	Y	BLEED
57	70	M	20578	LOSS OF BALANCE AND ATAXIA	4 DAYS	HTN	+	-	I	LEFT CEREBELLAR LOBE	ACUTE TO EARCLY SUBACUTE INTRAPARENCHYMAL HEMORRHAGE	ISO TO HYPER	HYPO	Y	Y	BLEED
58	46	M	24737	FOCAL NEIROLOGICAL DEFICIT & HEADCHE	3 DAYS	-	+	-	S	LEFT PARIETO-TEMPORAL	HEMORRHAGIC VENOUS INFARCT	HYPER	HYER	Y	Y	BLEED
59	1	M	24636	DEVELOPMENTAL DELAY OF MOTAR MILESONES	20 DAYS	-	+	-	I	RIGHT CEREBELLUM (MEDUSA HEAD APPEARANCE)	DEVELOPMENTAL VENOUS ANOMALY	HYPER	HYPO	Y	Y	BLEED
60	24	F	17663	FEVER & HEADACHE	10 DAYS	-	-	+	S	LEFT PARIETAL AT GREY WHITE MATTER JUNCTION	NEUROCYSTICERCOSIS	HYPO	HYPO	Y	N	CALCIUM
61	14	M	24721	SEIZURES	10 MONTHS	-	-	+	S	BILATERAL THALAMUS	ACUTE NECROTIZING HEMORRHAGIC ENCEPHALOPATHY	HYPO	HYPER	Y	Y	BLEED
62	7	M	26277	SEIZURES	7 MONTHS	-	-	+	S	BILATERAL THALAMUS	ACUTE NECROTIZING HEMORRHAGIC ENCEPHALOPATHY	HYPO	HYPER	Y	Y	BLEED
63	70	M	20085	TRAUMA	1 DAY	DM	-	+	S	RIGHT FRONTO-PARIETAL REGION	HYPERACUTE ON ACUTE SUBDURAL HEMORRHAGE	ISO	HYPER	Y	Y	BLEED
64	66	M	18353	IRRITABILITY AND INCREASED MOVEMENTS	3 DAYS	HTN & DM	+	-	S	LEFT HEAD OF CAUDATE & LENTIFORM. NUCLEUS	SUBACUTE INFARCT WITH HEMORRGAFIC TRANSFORMATION	HYPER	HYPER	Y	Y	BLEED
65	73	F	19075	LEFT SIDED WEAKNESS	3 DAYS	HTN & MI	+	-	S	RIGHT FRONTAL REGION	SUBACUTE HEMORRHAGIC VENOUS INFARCT	HYPER	HYPER	Y	Y	BLEED
66	73	M	26400	TRAUMA	4 DAYS	MI	-	+	S	RIGHT FRONTAL & TEMPORAL	SUBACUTE SUBDURAL HEMATOMA	HYPER	HYPER	Y	Y	BLEED
67	30	M	25847	FOCAL NEIROLOGICAL DEFICIT & HEADCHE	5 DAYS	-	+	-	S	RIGHT TRANSVERSE, SIGMOIS AND INTERNAL JUGULAR VEIN	CEREBRAL VENOUS THROMBOSIS	HYPER	-	Y	Y	BLEED
68	3	F	21951	PRE B CELL ACUTE LYMPHOBLASTIC LEUKEMIA	3 MONTHS	-	-	+	S	BILATERAL PARIETO-OCCIPITAL	ATYPICAL POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES)	HYPER	HYPER	Y	Y	BLEED
69	3 MONTHS	M	21436	SEIZURES	2 DAYS	-	-	+	I	CEREBELLAR VERMIS	CHRONIC BLEED	HYPO	HYPO	Y	Y	HEMOSIDERIN
70	60	M	28688	HYPERTENSION	80 MONTHS	HTN	+	-	S & I	BILATERAL LENTIFORM NUCLEUS, LEFT THALAMUS, PONS, MEDULLA	HYPERTENSIVE MICROANGIOPATHY	HYPER	HYPO	Y	Y	BLEED
71	66	M	27705	TRAUMA	5 HOURS	HTN & MI	-	+	S	BILATERAL FRONTO-PARIETO-OCCIPITAL REGION	HYPERACUTE SUBDURAL HEMATOMA	ISO	HYPER	Y	Y	BLEED
72	84	M	29716	HEADCAHE WITH LOSS OF CONSCIOUSNESS	1 DAY	MI, HTN, DM	-	+	S	RIGHT FRONTAL REGION	INTRAPARENCHYMAL HEMORRHAGE	ISO	HYPO	Y	Y	BLEED
73	46	F	28771	MENSTRUAL IRREGULARITY WITH BLURRING OF VISION	5 MONTHS	-	-	+	S	SELLAR & SUPRASELLAR	PITUITARY MACROADENOMA	HYPO	HYPO	Y	Y	HEMOSIDERIN
74	45	M	24786	HEADACHE & GIDDINESS	2 DAYS	HTN	+	-	S	RIGHT FRONTO-PARIETO-TEMPORAL	HEMORRHAGIC VENOUS INFARCT	HYPER	HYPER	Y	Y	BLEED
75	60	M	29029	HEADACHE & ALTERED SENSORIUM	25 DAYS	HTN & DM	-	+	S	LEFT FRONTO-PARIETAL	HIGH GRADE GLIOMA	HYPO	HYPER	Y	N	CALCIUM
76	63	M	23689	FEVER & HEADACHE	10 DAYS	HTN & DM	-	+	S	RIGHT OCCIPITAL LOBE	CEREBRAL ABSCESS	HYPO	HYPER	Y	N	HEMOSIDERIN
77	3 MONTHS	F	25981	SEIZURES	15 DAYS	-	-	+	S	BILATERAL LATERAL VENTRICLES, THIRD & FOURTH VENTRICLES	INTRAVENTRICULAR HEMORRHAGE	HYPER	HYPO	Y	Y	BLEED
78	72	F	26894	HEADACHE	15 DAYS	HTN & MI	-	+	S	BILATERAL LENTIFORM NUCLEUS	BILATERAL LENTIGORM NUCLEUS CALCIFICATION	HYPO	HYPER	Y	N	CALCIUM
79	4	F	24605	SEIZURES & DELAYED MILESTONES	30 DAYS	-	-	+	S	BILATERAL FRONTO-PARIETO-TEMPORAL REGION	TUBEROUS SCLEROSIS	HYPO	HYPER	Y	N	CALCIUM
80	26	F	27649	SEIZURES & FEVER	10 DAYS	-	-	+	S	BILATERAL FRONTAL, RIGHT OCCIPITAL & LEFT PARIETAL	TUBERCULOMA	ISO	HYPO	Y	N	CALCIUM
81	46	F	26794	LOSS OF CONSIOUSNESS	1 DAY	DM	+	-	S	BILATERAL FRONTO-PARIETO-TEMPORAL	ARTERIO-VENOUS MALFORMATION	HYPER	HYPO	Y	Y	BLEED
82	62	F	28954	HYPERTENSION	70 MONTHS	HTN & DM	+	-	S	LEFT PARITAL	HYPERTENSIVE MICROANGIOPATHY	HYPER	HYPER	Y	N	BLEED
83	31	M	28965	HEADACHE	1 MONTH	-	+	-	S	LEFT FRONTAL & RIGHT CENTRUM SEMI OVALE	CHRONIC HEMATOMA	HYPO	HYPO	Y	Y	HEMOSIDERIN
84	5 MONTHS	F	29856	ABSENT HEAD HOLDING	2 MONTHS	-	-	+	S	LEFT PARITAL	GLIOSIS WITH HEMOSIDERIN DEPOSITION	HYPO	HYPER	Y	Y	HEMOSIDERIN
85	45	F	27809	HEADACHE & BLURRING OF VISION	1 MONTH	-	-	+	S	RIGHT PARAFALCINE FRONTO-PARIETAL REGION	CYSTIC GLIOBLASTOMA	HYPO	HYPER	Y	Y	BLEED
86	68	M	27911	TRAUMA	1 DAY	DM & MI	-	+	S	BILATERAL FRONTO-PARIETAL REGION	HEMORRHAGIC CONTUSIONS	ISO	HYPER	Y	Y	BLEED
87	29	F	26877	SEIZURES & COUGH	15 DAYS	-	-	+	S	LEFT PARASAGITTAL HIGH FRONTAL	CALCIFIED GRANULOMA	HYPO	HYPO	Y	N	CALCIUM
88	17	M	29800	TRAUMA	2 DAYS	-	-	+	S	LEDT FRONTO-TEMPORAL	EXTRA DURAL HEMORRHAGE	HYPER	HYPER	Y	Y	BLEED
89	50	F	22578	BLURRING OF VISION WITH WEIGHT GAIN	10 DAYS	DM	-	+	S	SELLAR & SUPRASELLAR	PITUITARY MACROADENOMA	HYPER	MIXED	Y	Y	BLEED
90	74	M	27899	TRAUMA	4 DAYS	HTN & MI	-	+	S	LEFT FRONTO-PARIETO-TEMPORAL	LATE SUBACUTE SUBDURAL HEMATOMA	HYPER	HYPER	Y	Y	BLEED
91	82	M	27766	HEADACHE & ALTERED SENSORIUM	5 DAYS	MI	+	-	S	RIGHT TEMPORAL	LATE SUBACUTE INTRAPARENCHYMAL HEMORRHAGE WITH AREAS OF HEMOSIDERIN	HYP0	HYPER	Y	Y	HEMOSIDERIN
92	3	M	24557	DELAYED MILESTONES	20 MONTHS	-	-	+	S	CORTICAL & SUBCORTICAL BILATERAL FRONTO-PARIETAL	TUBEROUS SCLEROSIS	HYPO	HYPO	Y	N	CALCIUM
93	2	F	28577	HEADACHE & SEIZURES	3 MONTHS	-	-	+	S	RIGHT FRONTAL AND PARIETAL REGION	MALIGNANT MELANOMA	HYPER	HYPO	Y	Y	BLEED
94	57	M	28500	HEADACHE & BLURRING OF VISION	1DAY	DM	+	-	S	LEFT FRONTAL	HYPERACUTE BLEED	ISO	HYPER	Y	Y	BLEED
95	36	F	28425	HEADACHE	1 MONTH	-	+	-	S	CORTICAL & SUBCORTICAL RIGHT FRONTAL	CHRONIC HEMORRHAGIC VENOUS INFARCT	HYPO	HYPER	Y	Y	HEMOSIDERIN
96	58	M	28397	FACIAL NUMBNESS	3 DAYS	-	+	-	S	POSTERIOR LIMB OF LEFT INTERNAL CAPSULE	SUBACUTE INFARCT WITH HEMOSIDERIN DEPOSITION	HYPO	HYPER	Y	Y	HEMOSIDERIN
97	53	F	28306	LOSS OF CONSIOUSNESS	1 DAY	HTN & DM	+	-	S	POSTERIOR COMMUNICATING ARTERY	RUPTURED PCOM ANEURYSM	ISO	HYPER	Y	Y	BLEED
98	66	F	27890	HEADACHE & SEIZURES	7 DAYS	HTN	-	+	S	BILATERAL FRONTO-PARIETO-OCCIPITAL REGION	TUBERCULOMA	HYPO	HYPER	Y	N	CALCIUM
99	69	M	27866	SPASTICITY WITH REDUCED POWER IN UPPER LIMBS	15 DAYS	HTN & MI	-	+	S	RIGHT FRONTAL (CENTRAL VEIN SIGN)	MULTIPLE SCLEROSIS	HYPER	HYPER	Y	Y	BLEED
100	75	M	28027	HEADACHE & SEIZURES	20 DAYS	MI	-	+	S	RIGHT PARIETAL	NEUROCYSTICERCOSIS	HYPO	HYPER	Y	N	CALCIUM
101	57	F	28500	LOSS OF CONSIOUSNESS	1 DAY	HTN	+	-	S	LEFT FRONTAL REGION	HYPERACUTE BLEED	ISO	HYPER	Y	Y	BLEED

102	31	M	29154	HEADACHE & ALTERED SENSORIUM	10 DAYS	-	+	-	S	LEFT FRONTAL REGION	GLIOSIS WITH HEMOSIDERIN DEPOSITION	HYPO	HYPER	Y	Y	BLEED
103	34	F	29195	HEADACHE & PAST H/O COVID-19 PNEUMONIA	7 DAYS	-	+	-	S	SUPERIOR SAGITTAL SINUS	CEREBRAL VENOUS THROMBOSIS	HYPER	-	Y	Y	BLEED
104	22	F	27800	LOSS OF CONSIOUSNESS WITH RIGHT SIDED WEAKNESS	2 DAYS	HTN	+	-	S	LEFT GANGLIOCAPSULAR REGION	ACUTE INTRAPARENCHYMAL HEMORRHAGE	ISO	HYPO	Y	Y	BLEED
105	45	M	29263	HEADACHE & GIDDINESS	3 DAYS	-	+	-	S	LEFT FRONTAL REGION	OLD RESOLVING HEMATOMA	HYPO	HYPO	Y	Y	HEMOSIDERIN
106	60	M	29029	HEADACHE & SEIZURES	10 DAYS	HTN	-	+	S	LEFT FRONTO-PARIETAL REGION	HIGH GRADE GLIOMA	HYPER	HYPER	Y	Y	BLEED
107	61	M	29287	HEADACHE & GIDDINESS	7 DAYS	HTN & DM	+	-	S	RIGHT FRONTAL REGION	OLD RESOLVING HEMATOMA	HYPO	HYPO	Y	Y	HEMOSIDERIN
108	28	M	29269	SEIZURES & ALTERED SENSORIUM	5 DAYS	-	-	+	S	RIGHT SUBCORTICAL PARIETAL REGION	NEUROCYSTICERCOSIS	HYPO	HYPER	Y	N	CALCIUM
109	68	M	29264	RIGHT SIDED WEAKNESS	4 DAYS	HTN & MI	+	-	S	LEFT THALAMUS AND CORONA RADIATA	OLD BLEED	HYPO	HYPER	Y	Y	HEMOSIDERIN
110	64	M	29247	LOSS OF BALANCE & BREATHLESNESS	3 DAYS	HTN	+	-	I	RIGHT CEREBELLUM & PONS	SUBACUTE INFARCT WITH HEMORRHAGIC TRANSFORMATION	HYPER	HYPER	Y	Y	BLEED
111	78	M	28755	HEADACHE & NUMBNESS OF BILATERAL UPPER LIMBS	5 DAYS	HTN & DM	+	-	I	RIGHT TRANSVERSE AND SIGMOID SINUS	CEREBRAL VENOUS THROMBOSIS	HYPER	-	Y	Y	BLEED
112	75	M	29178	FOCAL NEIROLOGICAL DEFICIT & HEADCHE	5 DAYS	HTN & MI	+	-	S	LEFT THALMUS AND POSTERIOR LIMB OF LEFT INTERNAL CAPSULE	CHRONIC BLEED WITH HEMOSIDERIN	HYPO	HYPO	Y	Y	HEMOSIDERIN
113	76	M	29136	HEADACHE & ALTERED SENSORIUM	20 DAYS	HTN	-	+	S	LEFT PARIETAL REGION	ANAPLASTIC ASTROCYTOMA	HYPER	HYPO	Y	Y	BLEED
114	1	F	29070	DELAYED DEVELOPMENTAL MILESTONES	6 MONTHS	-	-	+	S	BODY OF CORPUS CALLOSUM	PERICALLOSAL LIPOAM WITH ? POSTERIOR CORPUS CALLOSAL DYSGENESIS	HYPER	HYPER	Y	N	CALCIUM
115	25	M	24489	DYSTONIA & SPASMS	2 MONTHS	-	-	+	S	BILATERAL BASAL GANGLIA	NEURODEGENERATION WITH BRAIN IRON ACCUMULATION (NBIA)	HYPO	HYPER	Y	Y	HEMOSIDERIN
116	23	M	24555	ATAXIA & GAIT DISTURBANCE	1 MONTH	-	-	+	S	LEFT CRONA RADIATE & POSTERIOR LIMB OF INTERNAL CAPSULE	ACTIVE DEMYELINATING PLAQUES	HYPER	HYPER	Y	Y	BLEED
117	56	M	23378	HEADACHE & ALTERED SENSORIUM	3 DAYS	-	+	-	S	RIGHT CAVERNOUS INTERNAL CAROTID ARTERY	ANEURYSM	HYPER	HYPER	Y	Y	BLEED
118	58	M	24145	RIGHT SIDED WEAKNESS	4 DAYS	MI	+	-	S	LEFT FRONTO-PARIETAL	SUBACUTE INFARCT WITH HEMORRGAFIC TRANSFORMATION	HYPER	HYPER	Y	Y	BLEED
119	42	F	28813	UPWARD GAZE PALSY	10 DAYS	-	-	+	S	PINEAL GLAND	PINEAL PARENCHYMAL TUMOR	HYPO	HYPER	Y	N	CALCIUM
120	43	M	23831	HEADACHE & GIDDINESS	15 DAYS	-	-	+	S	THIRD VENTRICLE	COLLOID CYST	HYPO	HYPER	Y	N	CALCIUM
121	28	M	26780	HEADACHE	7 DAYS	-	-	+	S	LEFT PERI-ROLANDIC CORTEX	NEUROCYSTICERCOSIS	HYPO	HYPER	Y	N	CALCIUM
122	19	M	27590	FEVER & HEADACHE	15 DAYS	-	-	+	S	LEFT FRONTAL REGION	LEFT FRONTAL ABSCESS	HYPO	HYPER	Y	Y	HEMOSIDERIN
123	1	M	27880	HEADACHE & GIDDINESS	10 DAYS	-	+	-	I	BILATERAL CEREBELLAR LOBES	MICROBLEEDS	HYPER	HYPER	Y	Y	BLEED
124	58	M	24667	HEADACHE & BLURRING OF VISION	1 MONTH	HTN	-	+	S	LEFT TEMPORAL REGION	NERVE SHEATH TUMOR	HYPO	HYPER	Y	N	CALCIUM
125	3 MONTHS	M	25219	HEADACHE & SEIZURES	7 DAYS	-	+	-	S	LEFT FRONTO-PARIETAL	HEMORRHAGIC VENOUS INFARCT	HYPER	HYPO	Y	Y	BLEED
126	43	M	28703	HEARING LOSS & TINNITUS	10 DAYS	-	-	+	I	RIGHT CEREBELLO-PONTINE ANGLE	VESTIBULAR SCHWANOMMA	ISO	HYPER	Y	Y	BLEED
127	60	M	20229	HEADACHE & ALTERED SENSORIUM	1 MONTH	DM	-	+	S	RIGHT HIGH PARIETAL REGION	MENINGIOMA	HYPO	HYPER	Y	N	CALCIUM
128	65	M	28022	CARCINOMA LUNG	15 MONTHS	-	-	+	S	BILATERAL FRONTO-PARIETO-TEMPORAL	HEMORRHAGIC METASTASIS	HYPER	HYPER	Y	Y	BLEED
129	54	M	25688	LOSS OF CONSIOUSNESS	5 DAYS	-	+	-	I	LEFT CEREBELLAR LOBE	LATE SUBACUTE INTRAPARENCHYMAL HEMORRHAGE	HYPER	HYPER	Y	Y	BLEED
130	67	M	21342	HYPERTENSION	40 MONTHS	HTN	+	-	S	SUPARTENTORIAL BRAIN PARENCHYMA	HYPERTENSIVE MICROANGIOPATHY	HYPER	HYPER	Y	Y	BLEED
131	65	M	21261	HEADACHE & SEIZURES	2 DAYS	HTN	+	-	I	BASILAR ARTERY	ANEURYSM	HYPER	HYPER	Y	Y	BLEED
132	10	M	28839	SEIZURES & DOUBLE VISION	10 DAYS	-	-	+	S	ROOF OF FOURTH VENTRICLE	MEDULLOBLASTOMA	HYPER	ISO	Y	Y	BLEED
133	66	M	22332	RIGHT SIDED WEAKNESS	5 DAYS	HTN & MI	+	-	S	M2 & M3 OF LEFT MIDDLE CEREBRAL ARTERY	OCCLUSION OF LEFT MIDDLE CEREBRAL ARTERY	HYPER	-	Y	Y	BLEED
134	65	M	20779	HEADACHE	7 DAYS	HTN	-	+	I	PONS	CAVERNOMA	HYPO	HYPER	Y	N	CALCIUM
135	19	F	25054	HEADCHE AND DISORIENTED	10 DAYS	-	+	-	S	RIGHT TEMPORAL REGION	GLIOSIS WITH HEMOSIDERIN DEPOSITION	HYPO	HYPER	Y	Y	HEMOSIDERIN
136	48	F	27800	SEIZURES	20 DAYS	HTN	-	+	S	RIGHT FRONTAL	NEUROCYSTICERCOSIS	HYPO	HYPER	Y	N	CALCIUM
137	46	M	23377	HEADCHE AND DISORIENTED	25 DAYS	-	+	-	S	RIGHT M2 OF MIDDLE CEREBRAL ARTERY	MOYA-MOYA	HYPER	HYPER	Y	Y	BLEED
138	57	M	27809	HEADACHE & GIDDINESS	30 DAYS	-	-	+	S	RIGHT FRONTAL REGION	OLIGODENDROGLIOMA	HYPO	MIXED	Y	N	CALCIUM
138	15	M	29018	HEADACHE & BLURRING OF VISION	10 DAYS	-	-	+	S	SUPRASELLAR	ADAMANTINOMATOUS CRANIOPHARYNGIOMA	MIXED	MIXED	Y	N	CALCIUM
140	13	M	28971	PORT-WINE STAIN & SEIZURES	3 YEARS	-	-	+	S	LEFT PARIETO-OCCIPITAL	STURGE WEBER SYNDROME	HYPO	HYPO	Y	N	CALCIUM
141	62	M	20250	HEADACHE & SEIZURES	10 DAYS	HTN	+	-	S	RIGHT FRONTAL	ARTERIO-VENOUS MALFORMATION	HYPER	HYPO	Y	Y	BLEED
142	39	M	28885	HEADACHE & ALTERED SENSORIUM	15 DAYS	-	-	+	S	LEFT FRONTAL & GANGLIOCAPSULAR	INTRAPARENCHYMAL HEMATOMA	HYPER	HYPO	Y	Y	BLEED
143	57	F	26795	HEADACHE & SEIZURES	7 DAYS	HTN & DM	-	+	S	LEFT FRONTAL	OLOGODENDROGLIOMA	HYPER	MIXED	Y	N	CALCIUM