

---

**DIAGNOSTIC ACCURACY OF SUSCEPTIBILITY-WEIGHTED  
IMAGING (SWI) TO DIAGNOSE THE CASES OF CEREBRAL  
VENOUS THROMBOSIS (CVT) COMPARED TO MAGNETIC  
RESONANCE VENOGRAPHY (MRV) - ONE YEAR HOSPITAL  
BASED CROSS SECTIONAL STUDY"**

---

**BY**

**REGISTRATION NO. BS0119003**

**Dissertation**

**Submitted to the**

**KLE Academy of Higher Education and Research, Belagavi,  
Karnataka**

**In partial fulfillment**

**of the requirements for the degree of**

**M.D.**

**IN**

**RADIO-DIAGNOSIS**

**DEPARTMENT OF RADIO-DIAGNOSIS,**

**J. N. MEDICAL COLLEGE,**

**BELAGAVI -590010. KARNATAKA**

---

**APRIL – 2022**

---

**KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,  
BELAGAVI, KARNATAKA**

**Endorsement by the HOD/Principal/  
Head of the Institution**

This is to certify that the dissertation entitled “**DIAGNOSTIC ACCURACY OF SUSCEPTIBILITY-WEIGHTED IMAGING (SWI) TO DIAGNOSE THE CASES OF CEREBRAL VENOUS THROMBOSIS (CVT) COMPARED TO MAGNETIC RESONANCE VENOGRAPHY (MRV) - ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY**” is a bonafide research work done by **REGISTRATION NO. BS0119003**.

**Dr. Ashwin S. Patil MD,**  
Professor and Head,  
Department of Radio Diagnosis,  
J. N. Medical College,  
Nehru Nagar, Belagavi – 10  
Date:  
Place: Belagavi

**Dr. N. S. Mahantshetti MD**  
Principal,  
J. N. Medical College,  
Nehru Nagar,  
Belagavi – 10  
Date:  
Place: Belagavi

# PLAGIARISM ACCEPTED LETTER



**JAWAHARLAL NEHRU MEDICAL COLLEGE**



(Recognized by Medical Council of India, New Delhi)

Accredited 'A' Grade by NAAC (2<sup>nd</sup> Cycle)

Placed in Category 'A' by MHRD (Govt)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

☎ 0831-2471350

☎ 0831-2478759

🌐 www.jnmc.edu

✉ principal@jnmc.edu

Ref No: MDC/PG/

Date: 25-11-2021.

## ACCEPTANCE LETTER

The softcopy of thesis entitled: "DIAGNOSTIC ACCURACY OF SUSCEPTIBILITY WEIGHTED IMAGING (SWI) TO DIAGNOSE THE CASES OF CEREBRAL VENOUS THROMBOSIS (CVT) COMPARED TO MAGNETIC RESONANCE VENOGRAPHY (MRV) - ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY" has been submitted for Anti-Plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of 07% which is within the acceptable limits of 10% as per the guidelines given by UGC.

Guide.



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

To,  
Reg. No. BS0119003,  
Postgraduate Student,  
2019-20 Batch,  
Department of Radiodiagnosis,  
J. N. Medical College, Belagavi.

## LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
3T	3 Tesla
ADC	Apparent diffusion coefficient
AUC	Area under the curve
CBM	Chinese biomedical
CECT	Contrast-enhanced CT
CI	Confidence interval
CT	Computed tomography
CVST	Cerebral venous sinus thrombosis
CVT	Cerebral venous thrombosis
DOR	Diagnostic odds ratio
DSA	Digital subtraction angiography
DTI	Diffusion tensor imaging
DVST	Dural venous sinus thrombosis
DVT	Deep Venous Thrombosis
DWI	Diffusion-weighted imaging
FLAIR	Fluid-attenuated inversion recovery
FMRI	Functional MRI
FSE	Fast spin-echo
GRE	Gradient echo
ICH	Intracerebral haemorrhage
ICoVT	Isolated Cortical Venous Thrombosis
IJV	Internal Jugular Vein
ISCVT	International study on cerebral vein and dural sinus thrombosis
MDCTA	Multidetector CT Angiography
MIP	Maximum intensity projection
MRA	Magnetic resonance angiography
MRBTI	MR black blood imaging

MRDTI	MR direct thrombus imaging
MRI	Magnetic resonance imaging
MRV	MR venography
MSE	Magnetic susceptibility effect
NCCT	Non-contrast CT
NCE	Non-Contrast Enhanced
PC	Phase-contrast
PDW	Proton density-weighted
RBCs	Red blood cells
SAH	Subarachnoid haemorrhage
SE	Spin echo
SPAIR	Spectral attenuated inversion recovery
SWI	Susceptibility weighted imaging
T1w	T1 weighted
T2 w	T2 weighted
TFE	Transforaminal
TOF	Time of flight
TR	Time to repeat
TSE	Turbo spin-echo

## ABSTRACT

**Background:** Cerebral venous thrombosis (CVT)/Cerebral Venous Sinus Thrombosis (CVST) is a rare presentation of venous thromboembolism. Clinical presentation is highly variable in CVT. The diagnosis of CVT could be missed on T1 and T2 Weighted sequences. Magnetic susceptibility signals produced by degraded products in the blood such as deoxyhaemoglobin can be visualised in Susceptibility Weighted Imaging (SWI) as hypointense signal which is termed as magnetic susceptibility effect (MSE). On MR Venogram - Time of flight (MRV- TOF) the intraluminal thrombosis is detected by absence of flow signal. Hence the present study was carried out to compare the diagnostic accuracy of SWI with MRV-TOF for CVT.

**Materials and methods:** A hospital based observational study was done on 30 patients referred for MRI (Magnetic Resonance Imaging) brain and MRV-TOF to a tertiary care teaching institute in Belgaum. All patients were evaluated clinically and then MRI of the brain was done using a 3 Tesla MRI scanner. The primary outcome variables in this study were diagnostic accuracy of SWI sequence and MRV-TOF. P value <0.05 was considered statistically significant. The data were analysed by using SPSS software V.22.

**Results:** The mean age was  $39.93 \pm 16.1$  years. Majority were males (80%). 53.33% had headache, 26.67% had seizures, 16.67% had right hemiparesis and 10% had a loss of consciousness. In SWI, the Superior sagittal sinus was abnormal in 50% with MSE noted in its entire length was seen in 33.33%. The inferior sagittal sinus had no MSE. In the Straight sinus, MSE was seen in 10%. Sensitivity was 100% for SWI on comparison with MRV. Specificity also varied between 80% to 100% for all the

vessels. The total diagnostic accuracy also varied between 90% to 100% for all the vessels.

**Conclusion:** On SWI sequence, the thrombus is visualized as an area of hypo intensity within the lumen of the affected vein or sinus. The sensitivity of SWI for detecting CVT has far exceeded than that of routine T1 and T2 weighted images and was comparable with MRV-TOF.

## TABLE OF CONTENT

<b>S. NO</b>	<b>TABLE OF CONTENT</b>	<b>PAGE NO</b>
<b>1</b>	<b>INTRODUCTION</b>	<b>1-3</b>
<b>2</b>	<b>AIMS &amp; OBJECTIVES</b>	<b>4</b>
<b>3</b>	<b>REVIEW OF LITERATURE</b>	<b>5-31</b>
<b>4</b>	<b>MATERIALS &amp; METHODS</b>	<b>32-34</b>
<b>5</b>	<b>RESULTS</b>	<b>35-51</b>
<b>6</b>	<b>DISCUSSION</b>	<b>52-60</b>
<b>7</b>	<b>CONCLUSIONS</b>	<b>61-62</b>
<b>8</b>	<b>SUMMARY</b>	<b>63-65</b>
<b>9</b>	<b>LIMITATIONS AND RECOMMENDATIONS</b>	<b>66</b>
<b>10</b>	<b>BIBLIOGRAPHY</b>	<b>67-75</b>
<b>11</b>	<b>ANNEXURES</b>	
	<b>I. ETHICAL CLEARANCE</b>	<b>76</b>
	<b>II. INFORMED CONSENT FORM</b>	<b>77-84</b>
	<b>III. STUDY PROFORMA</b>	<b>85-86</b>
	<b>IV. CLINICAL IMAGES</b>	<b>87-96</b>
	<b>V. KEY TO MASTER CHART</b>	<b>97</b>

## LIST OF TABLES

S. NO	TABLE DESCRIPTION	PAGE NO
1	Clinical presentation in CVT as described by Bousser MG et al	7
2	Presentation of cerebral sinus venous thrombosis	10
3	Signs of CVT on CT	13
4	Time intervals in MRI	17
5	Descriptive analysis of age (in years) in study population (n=30)	35
6	Descriptive analysis of gender in the study population (n=30)	35
7	Descriptive analysis of history in the study population (n=30)	36
8	Descriptive analysis of signal intensity within the venous sinuses on T1 W in the study population (n=30)	36
9	Descriptive analysis of signal intensity within the venous sinuses on T2 W in the study population (n=30)	38
10	Descriptive analysis of signal intensity within the venous sinuses on FLAIR in the study population (n=30)	39
11	Descriptive analysis of SWI Sequence in the study population (n=30)	40
12	Descriptive analysis of MR Venogram (MRV-TOF) in the study population (n=30)	42
13	Descriptive analysis of parenchymal abnormalities in the study population (n=30)	44
14	Descriptive analysis of cortical veins showing magnetic susceptibility effect (MSE) in the study population (n=30)	45
15	Comparison of MR Venogram (MRV-TOF) with SWI Sequence (n=30)	46
16	Predictive validity of SWI Sequence in diagnosing CVT (n=30)	47

17	Comparison of MR Venogram (MRV-TOF) with SWI Sequence (n=30)	49
18	Predictive validity of SWI Sequence in diagnosing CVT (n=30)	50
19	Comparison of baseline characteristics across the studies	55
20	Comparison of diagnostic accuracy across the studies	56

## LIST OF FIGURES

S. NO	FIGURE DESCRIPTION	PAGE NO
1	Normal major cerebral veins and Sinuses (as seen in MRV-TOF).	6
2	Axial CT image in Acute Sinus Thrombosis	15
3	MR black blood imaging (MRBTI) of a 27-year-old male patient with sub-acute CVT	23
4	Pie chart of gender in the study population (n=30)	35
5	Pie chart of signal intensity within the venous sinuses on T1 W in the study population (n=30)	37
6	Pie chart of signal intensity within the venous sinuses on T2 W in the study population (n=30)	38
7	Pie chart of signal intensity within the venous sinuses on FLAIR in the study population (n=30)	39
8	Graphical presentation of SWI Sequence in the study population (n=30)	41
9	Graphical presentation of MR venogram in the study population (n=30)	43

## **INTRODUCTION**

Cerebral venous thrombosis (CVT) / Cerebral Venous Sinus Thrombosis (CVST) is a rare presentation of venous thromboembolism. CVT comprises of thrombosis of cortical and deep veins. Though it's rare, but a possibly fatal neurological condition which is often missed because of its vague clinical and radiological presentation.<sup>1</sup> It is considered as one of the rare cases of stroke in young with increased mortality. Differentiating it from other neurological conditions is difficult, because of the variable clinical presentation.

Around 0.5% to 1% of stroke cases are due to clots in the Dural Venous Sinuses.<sup>2,3</sup>

CVT can be classified based on the timing of the symptom onset as

1. Acute (Less than or equal to 48 hours),
2. Subacute (more than 48 hours to 30 days) and
3. Chronic (more than one month).

Subacute type is the most frequent type as compared to chronic form.<sup>4</sup> It contributes to around 50% of all cases. The ISCVT (International Study on Cerebral Vein and Dural Sinus Thrombosis) determined the occurrence of CVT in various sites – transverse sinus (86%), superior sagittal sinus (62%), straight sinus (18%), cortical veins (17%), jugular veins (12%), a vein of Galen, and internal cerebral vein (11%).<sup>5</sup>

CVT is mostly associated with pregnancy and the use of oral contraceptives. It is more common in females, especially during puerperium.<sup>2</sup> Pregnant women have five times increased risk of developing DVT.<sup>6</sup> It could be due to the hyper coagulable

state in pregnancy. The incidence of CVT is 202 per one lakh deliveries to pregnant women. The CVT-associated mortality rate is also high in pregnancy at 11.63%.<sup>2</sup>

Clinical presentation is highly variable in CVT besides being nonspecific, hence clear-cut valid diagnostic algorithms are not available in its diagnosis. Neuroimaging plays an important role in diagnosing, evaluating the complications. It also helps in deciding the course of management and the prognosis.<sup>7</sup>

Diagnosing CVT early can help in improving the overall outcome. Due to its non-specific presentation, the diagnosis of CVT is difficult, besides the radiological findings which are subtle. A delay in the diagnosis and further management can lead to disability, which may be permanent and sometimes even to death. Hence the main deciding factor in the course of management of CVT is Neuroimaging.<sup>7</sup>

MRI is considered an investigation of choice, because of its non-invasiveness and highly sensitivity in detecting CVT. Besides, it is a painless day-care procedure, with the added benefit of lack of hazard due to ionizing radiation.

**NEED FOR THE STUDY:**

The rate of occurrence of CVT is low on comparison with arterial stroke. But the younger population is commonly affected with CVT in developing countries, with higher mortality due to CVT.<sup>8, 9</sup> The dilemma associated with the diagnosis of CVT makes it more important. Because of the lack of valid protocols to diagnose it clinically, the outcome can be poor. In the past few years, various MRI sequences and MRV are being used extensively for making a diagnosis of CVT. It is identified by the signal changes arising from blood flow alterations and signal changes arising due to the degraded haemoglobin products in the veins which are thrombosed. These changes can be identified in the T1, T2 W MRI – spin-echo images.<sup>10-12</sup> The thrombosed vessels shows a hyperintense signal on the T1 & T2 W sequence and non-visualization on MRV.<sup>13, 14</sup> The diagnosis of CVT could be missed on T1 and T2 W sequences. In cases without any clinical suspicion, the routine sequences performed without venography can miss the diagnosis. There has been a recent emergence of literature on the increased diagnostic accuracy of SWI for detection of the magnetic susceptibility signals produced by degraded products in the blood such as deoxyhaemoglobin in subjects with acute intraparenchymal hemorrhage.<sup>12, 15</sup> But there is a lack of sufficient evidence in the present region to make clinical recommendations. Hence there is a need to study the diagnostic accuracy of SWI in CVT diagnosis compared to MRV-TOF.

## **OBJECTIVES**

### **AIM AND OBJECTIVES OF THE STUDY:**

1. To assess the diagnostic accuracy of susceptibility weighted imaging (SWI) in diagnosing the cases of cerebral venous thrombosis (CVT) compared to magnetic resonance venography (MRV)

## REVIEW OF LITERATURE

### **Definition**

Cerebral vein thrombosis (CVT), also known as Cerebral Venous Sinus Thrombosis (CVST) is a rare presentation of venous thromboembolism. CVT encompasses thrombosis of both Dural sinus as well as cerebral veins (cortical vein and also deep vein). Though it's rare, but a possibly fatal neurological condition which is often missed because of its vague clinical and radiological presentation.<sup>1</sup>

### **Anatomy of Cerebral Venous System –:**

The cerebral venous system is divided into

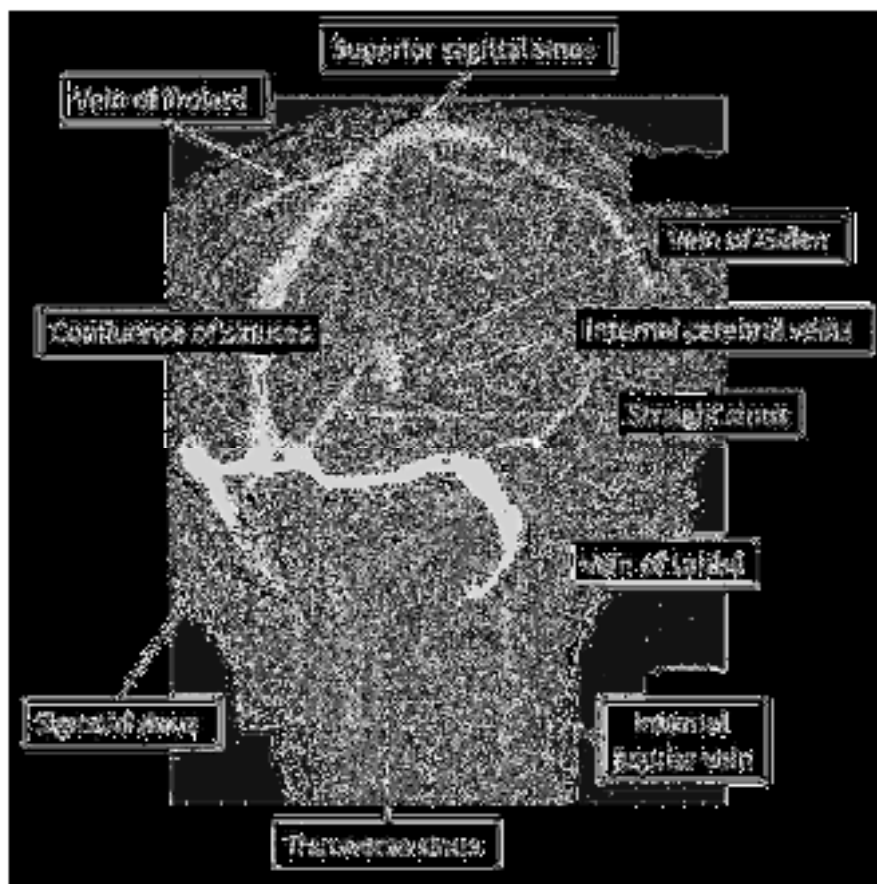
1. Superficial venous system and
2. Deep venous system.<sup>16</sup>

These are intracranially placed collections of venous channels and are placed between the meningeal, endosteal layer of the dura mater. The cerebral veins don not have valves as compared to systemic veins. They also don't follow the territory of the cerebral arteries. The superior sagittal sinus empties CSF from the subarachnoid space.

The superficial system consists of cortical veins and Dural sinuses. They drain the superficial white matter and the cerebral cortex. Superior sagittal drains the dorsolateral area while the anteroventral region is drained by the cavernous sinus. They are the major Dural sinuses. The superior sagittal sinus drains into the transverse sinus which then drains into the straight sinus. The cavernous sinus drains into the transverse sinus posterolateral and sigmoid sinus inferolateral, along the superior and inferior petrosal sinuses, respectively.<sup>16</sup> Veins draining superiorly and inferiorly are superficial cortical veins (vein of Labbe and Sylvian or superficial middle cerebral veins).

The deep cortical veins are included in the deep venous system. They comprise the sigmoid sinus, straight sinus, transverse sinus, a vein of Galen, internal cerebral veins, Rosenthal or basal vein, medullary, and subependymal veins. The deep venous system drains deep white matter of the brain, upper part of the brain stem, thalamus, and basal ganglia. The internal jugular vein is the ultimate drainage point for the contents from both the venous systems.

**Figure 1: Normal major cerebral veins and Sinuses (as seen in MRV-TOF).**



**Classification:**

CVT can be classified based on the timing of the clinical symptom onset as

1. Acute (Less than or equal to 48 hours),
2. Subacute (more than 48 hours to 30 days) and
3. Chronic (more than one month).

Subacute type is the most frequent type.<sup>4</sup> It contributes to around 50% of all cases. The chronic form is less frequent.

**Table 1: Clinical presentation in CVST as described by Bousser MG et al.<sup>3</sup>**

**Courtesy:** Alvis-Miranda HR et al<sup>4</sup>, Bousser MG et al<sup>3</sup>

Type of presentation	Clinical presentation characteristics
Focal syndrome	Characterized by Focal signs like seizures, headache, or mental state abnormalities
Diffuse sub-acute encephalopathy	Abnormalities in the Mental state
Isolated Intra Cranial Hypertension	Papilledema, vomiting, nausea, Headache
Cavernous sinus syndrome	Painful ophthalmoplegia, proptosis, chemosis

**Epidemiology:**

Around 0.5% to 1% of stroke cases are due to clots in the Dural Venous Sinuses.<sup>2, 3</sup> CVT is mostly associated with hormonal factors such as pregnancy and oral contraceptives. It is more common in females, especially during puerperium.<sup>2</sup> Pregnant women have five times increased risk of developing DVT.<sup>6</sup> It could be due to the hyper coagulable state in pregnancy. The incidence of CVT in pregnancy has been reported as 202 per one lakh deliveries. The CVT-associated mortality rate is also high in pregnancy at 11.63%.<sup>2</sup> The incidence of CVT is estimated at around 1.32 per one lakh person-years.<sup>17</sup> Devasagayam S et al.<sup>18</sup> (2016) in their retrospective population based study observed the rate of occurrence of new cases of CVT was 15.7 million cases per year. They observed that in about 48% of subjects, there was a possibility of procoagulant predisposition. They also observed that the majority (55/105) of the subjects were females. Although not statistically significant, a woman

in the reproductive age group has increased relative risk as compared to males. (1.18 [95% confidence interval, 0.94-1.48]). It is considered as the rare cause of stroke in young with increased mortality.

The ISCVT determined the occurrence of CSVT in various sites – transverse sinus (86%), superior sagittal sinus (62%), straight sinus (18%), cortical veins (17%), jugular veins (12%), a vein of Galen, and internal cerebral vein (11%).<sup>5</sup>

### **Pathophysiology:**

The clinical manifestations in CVT are explained by two theories.<sup>19, 20</sup>

First, thrombosis of the cerebral veins leads to increased venous and capillary pressure, which leads to a decrease in cerebral perfusion. Decreased cerebral perfusion results in ischemic injury, manifested by cytotoxic edema, which damages the energy-dependent cellular membrane pumps and leads to intracellular swelling. Disruption of the blood-brain barrier leads to vasogenic edema and leakage into the interstitial space. The increased pressure in the venous system can lead to an intraparenchymal hemorrhage.

The second is obstruction of the cerebral sinuses, particularly when thrombus does not resolve. Normally, the cerebrospinal fluid found in the cerebral ventricles is transported through the subarachnoid space to the arachnoid granulations and absorbed into the venous sinuses. Thrombosis of the venous sinuses results in impaired cerebrospinal fluid absorption and ultimately leads to increased intracranial pressure. Increased intracranial pressure leads to cytotoxic and vasogenic edema and ultimately may lead to parenchymal hemorrhage.

The abnormalities in the vessel wall, abnormalities of blood composition, and blood stasis (Virchow's triad) cause an imbalance in prothrombotic and fibrinolytic

processes. It leads to progressive venous thrombosis. Due to the increase in the pressure in the venous system, leads to vasogenic as well as cytogenic edema. Because of widespread anastomosis, among the cortical veins, the territories of the venous system are not well defined. This in turn leads to the development of alternative pathways for venous drainage when there is a block or an occlusion. The absorption of CSF via arachnoid villi is also blocked in CVT, leading to increased intracranial tension, due to obstruction of the superior sagittal sinus.

**Risk factors:**

The female gender is a risk for CVT, specifically during the postpartum period, because of the associated hormonal factors.<sup>2</sup>

The risk factors for CVT include factors contributing to the Virchow triad.

The causative factors are broadly classified as

1. Inherited/Genetic factors such as Inherited thrombophilia
2. Acquired risk factors (surgery, trauma, pregnancy, puerperium, antiphospholipid syndrome, cancer, exogenous hormones).

Other risk factors include medications like steroids, medicines used for cancer treatment, medicines used as hormonal therapy for contraception, etc.

The risk factors can also be classified as Systemic conditions, Infections, Gender-based factors, Inherited prothrombotic conditions, Acquired prothrombotic conditions, Medications, Vascular abnormalities, and Mechanical factors.<sup>5, 16</sup>

**Clinical presentation and diagnosis:**

CVT is a rare, but possibly fatal neurological condition which is often missed because of its vague clinical and radiological presentation.<sup>1</sup> Clinical presentation is highly variable in CVT besides being nonspecific. Clear-cut valid diagnostic algorithms are not available for the diagnosis of CVT. The non-specific nature of clinical presentation leads to a delay in the diagnosis. The median time for arriving at a diagnosis taken from the onset of clinical features is usually around Seven days.<sup>14</sup> A diagnosis is arrived at earlier when a patient gets admitted with changes in his consciousness and/or mental state and/or seizures.<sup>21</sup> Chronic onset is seen in around nearly twenty percentage of the subjects. Delay in diagnosis and delay in prompt management has poorer outcome besides the elevated risk of deficits visually.<sup>21</sup> In subjects with ICH, delay in diagnosis can lead to mortality or severe disability. Subjects with chronic CVT require extra attention for avoiding misdiagnosis as the presentation may occur with an untraditional onset.<sup>21</sup> The common symptoms with which patients present in a suspected case of CVT are headache, seizure, and hemiparesis.

**Table 2: Presentation of cerebral sinus venous thrombosis.**<sup>4, 22</sup>

<b>Type of presentation in CVST</b>	<b>Duration for development of symptoms</b>	<b>Rate of occurrence</b>
1. Acute onset	Less than 48 hours	30%
2. Sub-acute onset	48 hours to 30 days	50%
3. Chronic onset	30 days to 6 months	20%

The majority (50%) of subjects with CVT present sub-acutely, while 30% present in an acute fashion. 20% present chronically. In CVT, Headache is the commonest clinical symptom.<sup>23</sup> It is seen in around 80–90% of subjects with CVT. >80 % of patients have headaches acute to subacute in nature while only a few are chronic headaches. The headache has a diverse type of presentation in CVT. They can manifest as throbbing, band-like, or burning or as a thunderclap and other types of headache.<sup>24, 25</sup> Headache associated with CVT is generally persistent and it correlates generally with the disease severity.<sup>26</sup> CVT should be suspected, in case of aggravation of a headache that is chronic intermittent or with the appearance of a new chronic headache. Other than for thrombosis of the sigmoid sinus, no obvious association noted between the thrombosed site and headache.<sup>26</sup>

A **thunderclap headache** is a type of very grave headache which can occur rapidly and then peaks in a span of a minute. It is seen in about 5–13% of subjects presenting with CVT.<sup>25, 26</sup> It can be seen in conditions such as Subarachnoid Haemorrhage (SAH). In CVT subjects presenting with thunderclap headaches, the headache can occur without or with SAH.

**Migraine-like headaches** can also occur in CVT. It can be pulsatile and unilateral analogous to a migraine occurring without or with aura.<sup>26</sup>

There are also reports of **post-Dural puncture headache** in CVT.<sup>27</sup> It should be considered in headache, occurring after lumbar puncture.

**Headaches in CVT** are generally go together with clinical features like Localised neurological deficits, altered sensorium, and seizures.

CVT can also occur without a headache. They are more likely to be older and males, with the occurrence of paralysis, and seizures.<sup>28</sup> CVT occurring without

headache is most commonly associated with diseases like isolated cortical venous thrombosis, parenchymal lesions, and malignant diseases.

**Seizures** can occur in around 40% of subjects with CVT.<sup>29, 30</sup> They can occur as a single incident or as a cluster incident.

**Neurological deficits** such as palsies of the cranial nerves, motor impairment or sensory impairment, and cortical blindness can also be a presentation of CVT.<sup>31</sup> They are generally seen in CVT of the non-inflammatory type. In CVT associated with infections, cavernous sinus syndrome is more likely.<sup>32</sup> In CVT the commonest neurological deficits were in the form of motor deficits (19 to 39%). They were more frequently found in CVT subjects with involvement of superior sagittal sinus, cortical veins, and cerebral deep venous system.<sup>31, 32</sup> Aphasia was also reported in 19 to 24% of subjects with CVT.<sup>5, 26</sup>

**Ophthalmological symptoms** such as vision loss, papilledema can also occur in CVT. The most common manifestation among them is papilledema (28% to 67.5%). Usually, they do not occur without a headache.

**Altered consciousness** has been observed in 20% to 30% of subjects with CVT.<sup>5, 33</sup>

CVT can also present with **psychological symptoms, abnormal behaviour**, and several other manifestations.<sup>34</sup>

### **Diagnosis:**

For the diagnosis of CVT, there are no clear-cut valid diagnostic algorithms available. Diagnosis is mainly based on neuroimaging, which plays an important role in the evaluation of complications, deciding the management and prognosis.<sup>7</sup>

The historical diagnostic standard for CVT is Digital subtraction angiography. It is not used nowadays. MRI has replaced it in the past few decades. But there is a

lack of evidence with respect to diagnostic accuracies of modern imaging modalities for CVT.

**Imaging modalities in diagnosing CVT:**

1. Computed tomography.
2. Computed tomography venography.
3. Magnetic resonance imaging (MRI).
4. MR black blood thrombus imaging.

**Computed Tomography (CT):**

Computed tomography is the commonest modality used on subjects, who present with localized and acute neurological symptoms.<sup>35</sup> CT uses a series of X-rays and produces a 3D image of soft tissues and bones with the help of a computer. Besides being non-invasive, it is a painless technique for detecting health conditions. It is a cost-effective, widely accessible, and available tool for ruling out the common neurological diagnosis.<sup>36</sup> There may be a variable presentation of CVT on CT.

**Table 3: Signs of CVT on CT.**

Type of CT	Direct signs	Indirect signs
Non-contrast CT (NCCT)	1. Dense clot sign	1. Haemorrhagic infarction
	2. Cord or string sign (dense vessel sign):	2. Brain edema
		3. Mass effect
		4. SAH
Contrast-enhanced CT (CECT)	1. Empty delta sign	Similar findings as seen in NCCT

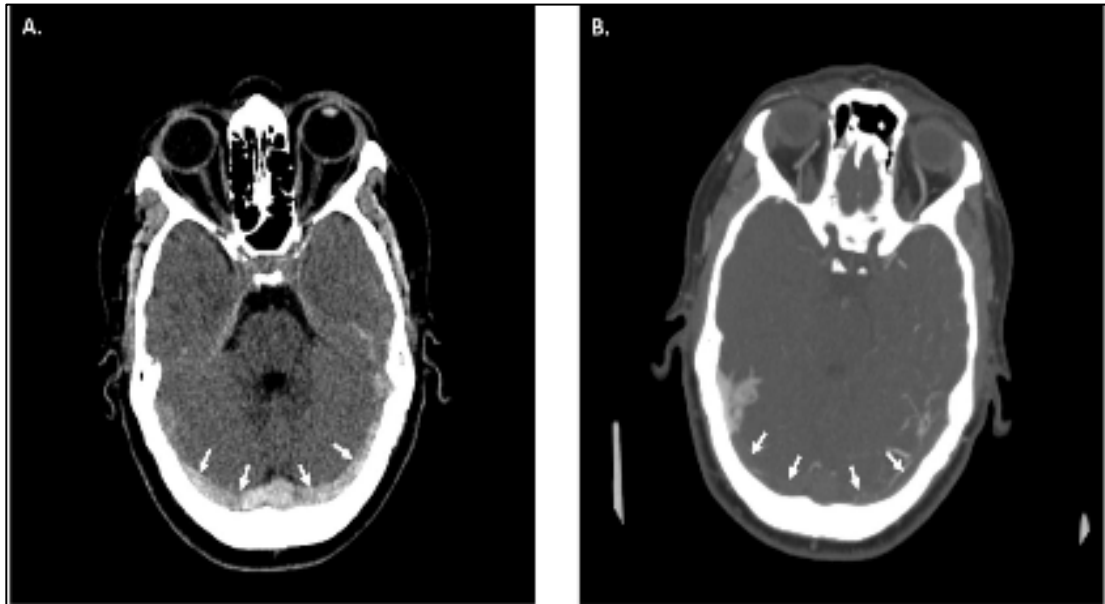
In NCCT, hyperattenuating noted within the lumen of cerebral sinus and veins, suggestive of thrombus (dense clot sign). It is due to the protein factor associated with haemoglobin inside the RBC'S (Red Blood Cells). Another direct sign is the dense vessel sign or cord sign, where the cortical vein, which is thrombosed appears like a cord. Indirect signs on CT include Haemorrhagic infarction, Oedema of the brain, SAH, and Mass effect. They occur in around 60 to 80% of cases.<sup>36</sup> The lesions in the parasagittal hemisphere indicate superior sagittal sinus thrombosis. Edema in the thalamus bilaterally, lesions of the temporo-occipital area indicate thrombosis of the transverse sinus.<sup>36,37</sup> In deep cerebral vein thrombosis also, bilateral thalamic edema is seen.

Empty delta sign is the characteristic sign of CVT seen in CECT, the thrombus is seen as a filling defect. It is specific for thrombosis of the superior sagittal sinus. In this sign, thrombosed sinus seen as a triangular area of enhancement with relatively low-attenuation center. Indirect signs are similar as seen in Non-contrast CT. A study done in 2018 observed in their meta-analysis assessed the role of CT and MRI for CVT/CVST diagnosis.<sup>38</sup> Overall sensitivity for CVT and CVST groups with CT was 79% and 81% respectively. The pooled specificity was 90% and 89%. With regards to MRI, the pooled sensitivity was 82% and 80% and pooled specificity was 92% and 91% for CVT and CVST respectively. They concluded that the diagnostic accuracy of CT and MRI was very high. It was excellent, regardless of stage, methods of analysis, and targets for it. They also observed that assessment of direct or indirect signs had more validity on comparison with attenuation assessment.

In a suspected case of acute CVT, CT can be used as a primary imaging modality, but there is a need for additional imaging to diagnose accurately and rule out CVT.<sup>7, 38, 39</sup>

**Figure 2: Axial CT image in Acute Sinus Thrombosis.**

- A. NCCT showing hyper dense areas in Transverse sinus (indicated by arrows)
- B. CT venography showing Filling defects in both transverse sinus (indicated by arrows)



**CT venography:**

It is a CECT, which is done with the help of a time-optimized contrast bolus for enhancing the cerebral venous system. The commonly used imaging modalities for diagnosing CVT. It is widely available besides being cost-effective.<sup>40</sup> Filling defect is noted within the thrombosed vein as shown in figure .<sup>40</sup> Indirect signs in CT venography like SAH and brain edema can also help in arriving at the diagnosis.

Axial thin-section contrast-enhanced source images of a helical CT scan are used to make the diagnosis of CVT. 2D and 3D reformation techniques like maximum intensity projection, integral display, and volume rendering can be used to provide detailed anatomic images of the deep and superficial cerebral veins free from over projecting bones and brain parenchyma.<sup>41</sup> In certain studies CT venography is a dependable substitute for CVT diagnosis as compared to Digital Subtraction

Angiography.<sup>38, 42</sup> But the role of CT venography in cortical vein thrombosis is limited.<sup>43</sup> The diagnostic sensitivity was variable between 6 to 75%.<sup>43, 44</sup> It can be due to the contrast filling defect known as 'missing vein' is hard to discriminate from the variations seen physiologically in the anatomy of the venous system.

### **MAGNETIC RESONANCE IMAGING:**

MRI is considered an appropriate imaging modality and is most commonly used for the brain.<sup>43</sup> But the quality of the image obtained depends on several technical factors. They include a selection of sequences and imaging planes, positioning of the patient, and selection of proper coil. The various types of MRI sequences<sup>6, 36, 43, 45</sup> that can be obtained are T1 and T2 weighted imaging, SWI, DWI, MRA, MRV, FLAIR, DTI, and fMRI.

In MRI one can visualize the anatomy in all three planes (i.e. axial, sagittal, and coronal). It has the ability to detect demyelinating disease, vascular malformations in comparison with CT. There is no ionizing radiation exposure with MRI as compared to CT. MRI sequences also have some disadvantages. They are subject to motion artifacts and require prolonged acquisition time for many images. They are inferior to CT in detecting acute hemorrhage and bony injuries.

The magnetization properties of atomic nuclei is the principle of MRI is based on.<sup>43, 44</sup> Normally the protons are randomly oriented within the water nuclei of tissue. The powerful external magnetic field is applied uniformly and the protons are aligned. Further, the alignment is disturbed by the application of external radiofrequency energy. Now the nuclei return to their resting alignment through relaxation processes and emit radiofrequency energy which is measured. By using Fourier transformation, the signals are converted to corresponding intensity levels. Different types of images

are created by changing the sequence of Radio frequency pulses applied and then collected.

**Table 4: TIME INTERVALS IN MRI.**

<b>S. No</b>	<b>TIME INTERVAL</b>	<b>DEFINITION</b>
1	Repetition Time	Time interval between successive pulse sequences which are applied to the same slice.
2	Time to Echo	Time interval between the delivery of the RF pulse and the receipt of the echo signal.
3	T1 – Longitudinal relaxation time	Time is taken for spinning protons to realign with the external magnetic field.
4	T2 – Transverse relaxation time	Time is taken for spinning protons to lose phase coherence among the nuclei spinning perpendicular to the main field.

T1 weighted MRI is a standard imaging procedure. It gives a clear picture of the structure and anatomy of the brain. It also shows brain damage, but only when there is significant damage. Like T1, T2 weighted MRI is also a standard imaging procedure. Short TE and TR times are used to produce T1-weighted images. Long TE and TR times are used to produce T2-weighted images. CSF appears dark on T1W & bright on T2-W imaging.

The conditions involving vascular structures and which cause disruption of the blood-brain barrier, T1- weighted Gadolinium-enhanced images are very useful.

Diffusion-weighted imaging is extremely sensitive in diagnosing conditions such as acute stroke. It works by detecting the random movement of water protons. The movement of water molecules is significantly restricted in the intracellular space but can diffuse freely in the extracellular space. In ischemic brain tissue, the spontaneous movements of water protons are restricted. The sodium-potassium pump during ischemia shuts down and sodium starts to accumulate intracellularly, as a

result, there is a shift of water from extracellular to intracellular space because of the osmotic gradient. There is an increase in sodium resulting in an increase of water in the tissues. There is an extremely bright signal on DWI in ischemia.

FLAIR imaging is very sensitive to water content in brain tissue. It is used to visualize alterations in conditions of tissues such as multiple sclerosis. It is also useful in conditions where there is a reduction in brain tissue following injury.

Susceptibility-weighted imaging (SWI) is an increasingly important adjunct in diagnosing a variety of neurologic diseases and provides a powerful tool to depict and help characterize microbleeds, veins, and other sources of susceptibility.

Susceptibility-weighted imaging (SWI) evolved from simple two-dimensional T2\*-weighted sequences to three-dimensional sequences with improved spatial resolution and enhanced susceptibility contrast. SWI is an MRI sequence sensitive to compounds that distort the local magnetic field (eg, calcium and iron), in which the phase information can differentiate.

Compounds that have paramagnetic, diamagnetic, and ferromagnetic properties all interact with the local magnetic field distorting it and thus altering the phase of local tissue which, in turn, results in a change of signal. Paramagnetic compounds include deoxyhemoglobin, ferritin and hemosiderin. Diamagnetic compounds include bone minerals and dystrophic calcifications. Initially, SWI and related sequences were mostly used to improve the depiction of findings already known from standard two-dimensional T2\*-weighted neuroimaging: more microbleeds in patients who are aging or with dementia or mild brain trauma; increased conspicuity of superficial siderosis in Alzheimer disease and amyloid angiopathy; and iron deposition in neurodegenerative diseases or abnormal vascular structures, such as capillary telangiectasia. But SWI also helps to identify findings not

visible on standard T2\*-weighted images: the nigrosome 1 in Parkinson disease and dementia with Lewy bodies, the central vein and peripheral rim signs in multiple sclerosis, the peripheral rim sign in abscesses, arterial signal loss related to thrombus, asymmetrically prominent cortical veins in stroke, and intratumoral susceptibility signals in brain neoplasms.<sup>71</sup>

Typically the images presented are:

- magnitude
- filtered phase
- SWI (combined post-processed magnitude and phase)

A fourth set of images is provided, minimum intensity projection (minIP) which is just a thick slab of the conventional SWI images and is better able to demonstrate venous anatomy.

Diffusion Tensor Imaging visualizes the white matter tract in brain tissues.

Functional MRI – depends on the magnetic properties of blood and helps to see images of blood flow in the brain. A functional MRI scan can produce images of brain activity as fast as every second. fMRI helps to determine the active brain regions and how long they remain active. It produces high-quality images and helps to know which areas of the brain are being activated.

Indications for MRI of Brain are.<sup>46</sup> Trauma (epidural hematoma, subdural hematoma, contusion), Infection (abscess, encephalitis, meningitis), Tumor (primary CNS and metastatic), Vascular (ischemic & hemorrhagic stroke, venous thrombosis, aneurysm, arteriovenous malformation), Inflammatory/Demyelinating Lesions, other conditions such as Congenital Malformations and Hydrocephalus. The Contraindications are implanted metallic devices and magnetic electronic devices in

the body like pacemakers, artificial heart valves, etc. Due to unknown effects on the fetus, pregnancy is considered a relative contraindication.

### **MRI SEQUENCES IN CVT:**

For depicting the thrombus in CVT, MRI techniques can be divided into three groups

1. Non-contrast enhanced flow-related MRI
2. Native contrast thrombus MRI
3. Contrast-enhanced MRI

**Non-contrast enhanced flow-related MRI** includes 2D-TOF MRV, 3D TOF MRV, 2D phase-contrast (PC) MRV, 3D PC MRV.

TOF MRV depends on Flow-related enhancement. They are sensitive to slow flow (especially 2D) and they do not require the use of a contrast agent and have a relatively short acquisition times (5–8 min). The principle used here is the flow-related enhancement of spins entering into an imaging slice. These spins give more signal on comparison with surrounding stationary spins, because they are unsaturated. A 3D image can be by combining these images.<sup>72</sup> But their main disadvantage is False positives (loss of signal due to in-plane saturation) and False negatives (high signal from background tissue with short T1 values that can mimic flowing blood).

Phase-contrast MRV depends on Velocity-induced phase shift of spins. Its advantages include suppression of background tissues, no false negatives due to methaemoglobin, detection of flow direction and quantifying flow, detecting flow in orthogonal planes. It has long acquisition times (>15 min). It is also sensitive to motion artifacts and velocity & turbulent blood flow.

NCE flow-related MRI on comparison with DSA has high sensitivity (100%), specificity (71%) for diagnosis of CVT.<sup>47, 48</sup> But they were less accurate in identification of thrombosis of cortical veins.<sup>43, 46</sup>

The thrombus is directly visualized in native contrast thrombus MRI. The following are the sequences used:

1. **Spin echo (SE):** T1-WI FSE (fast spin-echo) /TSE (turbo spin-echo), T2-WI FSE/TSE, FLAIR, PDW (proton density-weighted), MR Black Blood Imaging (MRBTI: T1-WI 3D SPACE), 3DT1 TSE SPAIR.  
SPACE - variable-flip-angle-turbo spin echo,  
SPAIR - Spectral Attenuated Inversion Recovery
2. **Gradient echo (GRE):** DWI, MR Direct Thrombus Imaging (MRDTI: T1-WI magnetization prepared 3D gradient TFE).  
TFE – turbo field echo
3. **Gradient echo susceptibility-weighted:** T2\*WI, T2\*WI SE EPI, T2\*SW (susceptibility weighted), T2\*GRE, GRE.

In the first 5 days (acute thrombus) the concentration of deoxyhaemoglobin is high, on T1W the thrombus appears isointense and hypointense on T2W. Between 6 and 15 days, due to a high methaemoglobin concentration on T1WI and T2WI, the clot may appear hyperintense. After 15 days the thrombus may appear isointense on T1WI and iso to hyperintense on T2WI. On GRE SW images, the methaemoglobin, deoxyhaemoglobin (blood breakdown products) can cause exaggerated signal drop-out, termed as magnetic susceptibility effect (MSE). Because of that intraluminal thrombi can be depicted in stages, where the clot may be subtle in other sequences.<sup>7,40</sup>

By using the various combination of native contrast thrombus MRI techniques, the overall sensitivity & specificity for the diagnosis of CVT is 84–97% & 28–96% respectively.<sup>47-49</sup>

For cortical vein thrombosis, GRE SW MRI had adequate sensitivity of 97–98% and specificity of 100%.<sup>43,45</sup>

CE MRV includes 3D CE MRV (static) and 4D CE MRV (dynamic). They are based on the T1 shortening of Gadolinium. Advantages include good suppression of background signals, fast acquisition time, ability to assess partial recanalization and no in-plane saturation effects. In 4D CE MRV, there is no need for a sophisticated triggering system for injection of contrast as compared to 3DCE MRV. The disadvantages with CE MRV include the need for contrast administration.

DSA was used as the reference standard, contrast-enhanced MRI was more accurate for diagnosing CVT with a sensitivity (83%) and specificity (100%) than non-contrast-enhanced flow-related and native contrast thrombus MR sequences with sensitivity (8–51%) and specificity (80–93%).<sup>50</sup> Contrast-enhanced MRI was also more sensitive for CVT than non-contrast-enhanced MRI, with a sensitivity and specificity of 86–97% and 52–100% versus 55–97% and 28–95%, respectively.<sup>51,52</sup> In a meta-analysis, by using various MRV techniques the diagnostic performance of contrast-enhanced MRV was better than that of non-contrast-enhanced TOF and PC MRV.<sup>53</sup>

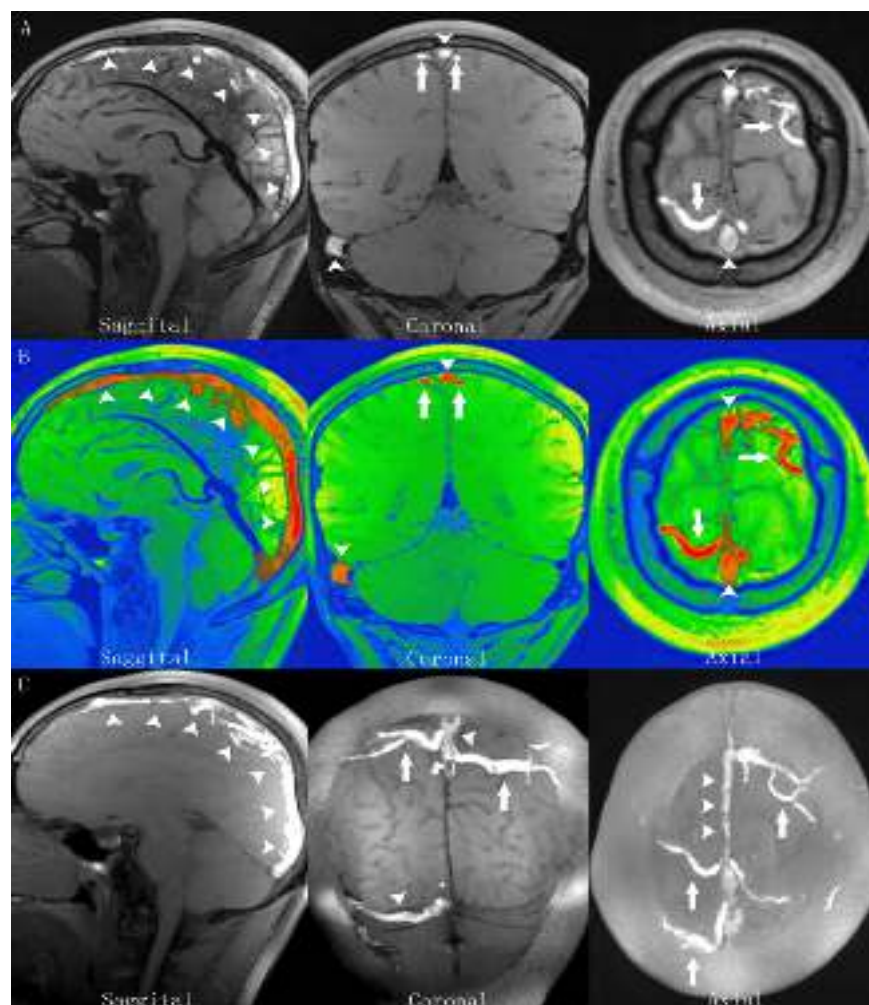
#### **MAGNETIC RESONANCE BLACK BLOOD THROMBUS IMAGING:**

Magnetic resonance black-blood thrombus imaging (MRBTI) is a native contrast thrombus MR technique. It provides a non-invasive visualization of thrombi in cerebral veins and sinuses. It is used to distinguish the probable ages of thrombi (acute, subacute, or chronic) for deciding customized treatment. Studies done

previously have also confirmed the sensitivity of MRBTI in the context of investigating thrombi in acute ischemic stroke<sup>54</sup>, deep venous thrombosis<sup>55</sup>, and myocardial infarction is high.<sup>56</sup> It has been used in the evaluation of suspected CVT.<sup>57, 58</sup> It has yielded 100% sensitivity, 96% specificity, accurate to the individual venous segments level on comparison with CT and MRI but not with DSA as diagnostic standard.<sup>58</sup>

MR Direct Thrombus Imaging (MRDTI), is another similar technique with high accuracy for diagnosis of DVT and helps in differentiating acute from chronic DVT in legs.<sup>59</sup> There is a need for evidence-based studies before MRBTI is incorporated in daily clinical practice for CVT diagnosis.

**Figure 3: MRBTI of a 27-year-old male patient with sub-acute CVT.**



A: MRBTI demonstrated hyper-intense signal intensity in the superior sagittal sinus (arrowheads), the right transverse and sigmoid sinuses (arrowheads), and the cortical veins (arrows) suggesting intraluminal thrombus formation. B: All thrombi semi-automatically outlined by software based on their high signal contrast were rendered with red color and volume was 21.5 cc. C: sagittal, coronal and axial sections of maximum intensity projection (MIP) reformations of MRBTI better depicted the thrombosed segments with hyper-intense signals.<sup>58</sup>

**Image courtesy:** Yang Q et al.<sup>58</sup>

#### **DIAGNOSTIC ACCURACY OF SWI VERSUS MR VENOGRAPHY- TOF IN CVT DIAGNOSIS:**

Cerebral venous thrombosis is an important cause of stroke in young adults. The intraluminal clot within the veins is detected by The Susceptibility Weighted Imaging (SWI). The affected vein or sinus appears hypointense which is termed as magnetic susceptibility effect (MSE) due to the presence of a clot. The sensitivity of SWI for detecting CVT has far exceeded that of routine T1 and T2 weighted images. Thrombosed veins and sinuses are more easily visualized on SWI than on any other MR sequence. Several studies have confirmed the usefulness of MRI for diagnosis of CVT.<sup>48, 60, 61</sup> The main sign of CVT on a standard MRI protocol is the lack of expected signal flow void on standard spin echo T1 & T2 sequences. The various signal changes on T1 and T2 weighted images are due to different chemical products of blood breakdown in thrombosed veins and sinuses. MRV is almost always required to confirm the diagnosis of CVT. The intraluminal thrombosis is identified by the absence of flow signal on MRV-TOF. However, MRV-TOF is also subject to artifacts, which may result in a false negative diagnosis.

A study done in the year 2020, provided an overview of the best available evidence regarding the diagnostic performance of CT and MRI for the diagnosis of CVT. Notably, other available studies are observational, mostly small, outdated, and with a high risk of bias. Therefore, direct comparison between studies is difficult due to the large diversity in study design, imaging method, reference standard, patient selection, and sample size. In general, contrast-enhanced techniques are more accurate for the diagnosis of CVT than non-contrast-enhanced techniques. CT venography and MRI have been both reported to be adequate for establishing a final diagnosis of CVT, but the choice of modality as used in clinical practice depends on availability, local preference, and experience, as well as patient characteristics. Their review underlines the need for high-quality diagnostic studies comparing CT venography and MRI in specific settings, to improve clinical care and standardize clinical trials.<sup>1</sup>

The MR properties of hemoglobin catabolism and their products are well characterized.<sup>10-12</sup> When the arterial blood with high oxygen saturation passes into the venous blood with lower oxygen saturation, hemoglobin becomes deoxygenated.<sup>62</sup> This results in the formation of deoxyhemoglobin, which has paramagnetic properties. In CVT, the loss of the T2\* -weighted signal is due to deoxyhemoglobin which produces a non-uniform magnetic field and rapid dephasing of proton spins. The paramagnetic molecules have this property and its termed as ‘magnetic susceptibility effect (MSE)’ and results in a signal loss (darkening) best seen in T2\*/SWI.<sup>63-65</sup> Thus T2\*/ SWI not only helps in the detection of thrombosed sinus but also helps in the detection of the hemorrhagic venous infarct.

**MOST RELEVANT STUDIES:**

A study done in the year 2020, observed that Digital subtraction angiography (DSA) is the historical diagnostic standard for CVT, but is rarely used nowadays and replaced by computed tomography (CT) and magnetic resonance imaging (MRI). They concluded that contrast-enhanced techniques have more validity than non-contrast-enhanced techniques.<sup>1</sup>

A study done in the year 2018, meta-analysis assessed the role of CT, MRI for CVT and CVST diagnosis. Overall sensitivity was 79% (95% C.I. of 76% to 82%) and 81% (95% C.I. of 78% to 84%) with CT for CVT and CVST respectively. The pooled specificity was 90% (95% C.I. of 89% to 91%) and 89% (88% to 91%), with an AUC of 0.93 and 0.91 respectively. For the pooled sensitivity for MRI-CVT and MRI-CVST was 82% (95% C.I. of 78% to 85%) and 80% (95% C.I. of 76% to 83%), and pooled specificity was 92% (95% C.I. of 91% to 94%) and 91% (95% C.I. of 89% to 92%), with an AUC of 0.92 and 0.93 respectively. They concluded that both CT and MRI have excellent validity.<sup>66</sup>

A study done in the year 2018, assessed the diagnostic accuracy of MRV for CVT diagnosis. They searched the Embase, PubMed, and Chinese Biomedical (CBM) databases up to Mar 31, 2018. The meta-analysis synthesized 12 articles containing 27 cohorts with a total of 1933 cases. The pooled sensitivity and specificity were 0.86 (95% CI: 0.83, 0.89) and 0.94 (95% CI: 0.93, 0.95), respectively. The pooled diagnostic odds ratio (DOR) was 75.24 (95% CI: 38.33, 147.72). They concluded that MRV had excellent diagnostic accuracy for CVST diagnosis.<sup>53</sup>

A study done in the year 2016, on 17 subjects with cerebral venous thrombosis (CVT) for evaluating various gradient echo (GRE) MRI sequences compared to conventional MRI sequences for characterizing CVT. The MRI sequences included

T<sub>1</sub>-weighted spin-echo (SE) imaging, T<sub>2</sub>\*-weighted turbo SE (TSE), fluid-attenuated inversion recovery (FLAIR), T<sub>2</sub>\*-weighted conventional GRE, and diffusion-weighted imaging (DWI). MR venography (MRV) images were obtained as the golden standard. They observed that venous sinus thrombosis was best detectable in T<sub>2</sub>\*-weighted conventional GRE sequences in all patients except in one case. Venous thrombosis was undetectable in DWI. T<sub>2</sub>\*-weighted GRE sequences were superior to T<sub>2</sub>\*-weighted TSE, T<sub>1</sub>-weighted SE, and FLAIR. Enhanced MRV was successful in displaying the location of thrombosis.<sup>67</sup>

A study done in the year 2016, a retrospective population-based study observed the CVT incidence, that is the rate of occurrence of new cases was 15.7 million cases per year with a 95% confidence interval ranging from 12.9 million to 19.0 million. They observed that in about 48% of subjects, there was a possibility of procoagulant predisposition. They also observed that the majority (55/105) of the subjects were females. Although not statistically significant, the relative risk of CVT was higher in females belonging to the reproductive age group on comparison with males. (1.18 with 95% confidence interval of 0.94 to 1.48).<sup>18</sup>

A study done in the year 2016, assessed the diagnostic accuracy of routinely used MRI sequences without and with contrast enhancement for DVST. The seven MRI sequences that were used (axial unenhanced T1-weighted, T1-weighted CE, T2-weighted, DWI, T2-weighted FLAIR, T2-weighted gradient-recalled echo [GRE], and sagittal 3D T1-weighted GRE CE sequences) were randomized, anonymized, and reviewed independently by two neuroradiologists who were blinded to the final diagnosis. Ten separate venous sinus segments were evaluated. CE MRV was the reference standard for determining the presence or absence of DVST, and it was performed using the following imaging parameters: TR/TE, 4.1-77/1.4-9.5; flip angle,

12-35°; and slice thickness, 0.8-1.4 mm. The diagnostic performance of and interobserver variability for each sequence was assessed per patient and per segment. Thirty-six patients with DVST (72% of whom had acute thrombosis and 28% of whom had chronic thrombosis) and 29 patients without DVST were included in the study. For each sequence, the AUC values for the detection of DVST per patient, as determined by reviewer 1 and reviewer 2, respectively, were as follows: for T1-weighted unenhanced sequences, 55% and 61%; for T1-weighted CE sequences, 79% and 80%; for T2-weighted sequences, 77% and 76%; for DWI sequences, 59% and 64%; for T2-weighted FLAIR sequences, 70% and 72%; for T2-weighted GRE sequences, 64% and 66%; and for the 3D T1-weighted GRE CE sequence, 77% and 81%. The diagnostic performance of the 3D T1-weighted GRE CE sequences was statistically significantly greater than that of the other sequences. Interobserver variability ranged from 0.26 (for T1-weighted unenhanced sequences) to 0.73 (for the DWI sequence). Overall, for each reviewer and with the use of all evaluated sequences, MRI had a high sensitivity (> 99% for both reviewers) but low specificity (14% for reviewer 1 and 48% for reviewer 2) for the detection of DVST. They concluded that Sequences used in routine brain MRI performed with and without contrast enhancement have varying strengths that are important to recognize when the likelihood of DVST is assessed, but they do not replace the utility of dedicated CE MRV.<sup>52</sup>

A study done in the year 2010, observed that for the diagnosis of CVT, T2\* weighted imaging is superior. They observed besides T2 weighed imaging, only T1weighted imaging had a sensitivity of more than 50% for CVT. It was followed by FLAIR and MRV. They suggested that NCCT and also MDCTA may not be appropriate for CVT diagnosis.<sup>43</sup>

A study done in the year 2009, described MR imaging features, including T2\*gradient-echo (GE) sequence, in 8 subjects with presumed Isolated Cortical Venous Thrombosis. MR venography was performed in all patients and digital subtraction angiography in 4) at the time of diagnosis and during the follow-up at 15 days (4 patients) and at 3 (8 patients), 6 (6 patients), 12 (3 patients), and 18 months (1 patient). Magnetic susceptibility effect (MSE) was detected on T2\*GE imaging at the site of a cortical vein in all subjects at the first MR imaging examination. The occluded vein appeared as hyperintense in 3 patients, iso- to slightly hyperintense in 1 on T1, hypointense in 6 on FLAIR images, and as a signal-intensity loss on DWI in 3. At follow-up, persisting signal-intensity abnormalities on T2\*GE imaging were detected at the venous sites in all patients, whereas signal-intensity changes on T1- and T2-weighted images were no longer present. Parenchymal hyperintensities on FLAIR and DWI (increased apparent diffusion coefficient [ADC]) were observed in close vicinity to the thrombosis in 6/8 patients. Petechial hemorrhages ( $n = 3$ ) or hematoma ( $n = 2$ ) was present on T2\*GE imaging in 5/8 patients. During the follow-up, all cerebral tissue signal-intensity changes on T1, T2, and FLAIR images decreased both in volume and intensity. ADC values normalized within the tissue after 3 months in all patients. They concluded that T2\*GE imaging, MSE of hemoglobin products within the thrombus was observed both at the early and late phases of ICoVT and appears to be of high diagnostic value compared with the other signal intensity changes detected on standard MR imaging.<sup>13</sup>

A study done in the year 2009, in their review article observed that SWI enhances contrast in MRI. “Conventional imaging relies on the magnitude information to generate the image; the phase information on the other hand has typically been discarded except for a few applications in flow imaging. Historically,

phase images have been difficult to interpret, as the valuable information about susceptibility changes between tissues was hidden by background field inhomogeneities caused by air/tissue interfaces and main magnetic field effects. It has been shown, however, that by using a special high-pass filter it is possible to remove most of these unwanted effects, leaving behind only the valuable information about susceptibility changes between tissues. The contrast in the phase image is complimentary to the magnitude contrast and the two can be combined to create what is now referred to as susceptibility-weighted (SW) images. SWI is a new type of contrast that is complementary to conventional spin-density, T1-, and T2-weighted imaging methods. SWI is particularly suited for imaging venous blood as it is very sensitive to deoxyhaemoglobin, making it useful in imaging haemorrhages from trauma, visualizing blood products and the vascularization of tumours, and high-resolution MR venography. It has also proven useful in other applications relating to iron such as measuring iron content in multiple sclerosis lesions, and aging.<sup>68</sup>

A study done in the year 1994, explored 53 patients with CVT by MRI. They observed 3 types of signal abnormalities in thrombosed sinuses: 1) iso signal on T1-weighted sequence and low-intensity signal on T2-weighted sequence (early stage); 2) high-intensity signal on T1-and T2-weighted sequences (intermediate stage); 3) isosignal on T1-weighted sequence and high-intensity signal on T2-weighted sequence (late-stage). Signal abnormalities in Dural sinuses enabling CVT to be diagnosed were absent in 2 out of 53 cases. Twenty-six out of 53 patients had venous infarction. These lesions were haemorrhagic in 20 cases. MRI made it possible to follow the course of CVT in 15 cases, showing partial or complete recanalization of the occluded sinuses in 14 cases. They concluded that MRI is the best modality for exploring CVT due to its non-invasiveness.<sup>61</sup>

**LACUNAE OF LITERATURE:**

As compared to non-enhanced MRI & CT, contrast-enhanced MRI & CT venography has more accuracy in the diagnosis of CVT. In order to make a diagnosis of CVT, both contrast-enhanced MRI and CT venography appear to be satisfactory. But there is no high-quality evidence to make recommendations for clinical practice. Clinical availability, experience, and local preference determine the modality which is used. Hence there is a necessity for multi-centric studies of high quality, to be done on a large scale.

## METHODOLOGY

### Source of data:

Patients referred for MRI brain and MRV-TOF to the Department of Radio-Diagnosis at The KLE'S Dr. Prabhakar Kore Hospital & MRC, Belgaum.

### Method of collection of data:

(a) **Study design:** Hospital-based observational study

(b) **Sample size:**

The following formula was used to calculate the minimum sample size:

$$n = \frac{Z_{\alpha}^2 \text{ sensitivity (100- sensitivity)}}{L^2 \times P}$$

Were,

n = Sample size

$Z_{\alpha}$  = 1.96 at 95% confidence interval

L = Standard error (25%)

P = Prevalence of the disease = 20%

Taking Sensitivity = 90%

L = 25%

P = 20%

We get n = 28.

The sample size is calculated to be 28

(c) All patients were evaluated clinically and then undergo an MRI of the brain performed using a “3 Tesla MRI scanner (Magnetom Avanto TIM, 18 channel; Siemens, Erlangen, Germany)”.

**DURATION: One year – between January 2020 to December 2020**

**Inclusion criteria:**

1. Patients of cerebral venous thrombosis suspected clinically or detected by CT Brain scan and referred to the radiology department for MRI.
2. Patients who give consent to take part in the study.

**Exclusion criteria:**

1. Contraindications to MRI: cardiac pacemaker, prosthesis, joint replacement, Claustrophobia.

**METHODOLOGY:** Study was done using a 3T MRI scanner (MagnetomAvanto TIM, 18 channel; Siemens, Erlangen, Germany)

Standard scan protocol was followed for all the patients undergoing MRI.

Once the MRI was done, Findings was noted and analysed.

**MRI sequences that were obtained:**

1. Axial – T 1 W, T 2 W, FLAIR.
2. Sagittal – T 1 W.
3. Coronal - T 2 W.
4. DWI & ADC.
5. Susceptibility weighted imaging (SWI)– (Magnitude – axial, Phase – axial, Minimum intensity projection – axial, SWI – axial, sagittal & coronal).
6. 3D TOF (MRV-TOF).

**STATISTICAL METHODS:**

The primary outcome variables in this study were SWI sequence and MR venogram. Demographic and clinical-related parameters were study-related variables. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagrams and pie diagrams. Categorical outcomes were compared between study groups using the Chi square test. MRV–TOF was considered the gold standard. SWI sequence was considered a screening test. The sensitivity, specificity, predictive values, and diagnostic accuracy of the screening test along with their 95% CI were presented. P value <0.05 was considered statistically significant. The data were analysed by using SPSS software V.22.<sup>69</sup>

## RESULTS

### Result:

A total of 30 subjects were included in the study.

**Table 5: Descriptive analysis of age (in years) in study population (n=30)**

Parameter	Mean $\pm$ SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Age (in years)	39.93 $\pm$ 16.1	36.50	16.00	72.00	33.92	45.94

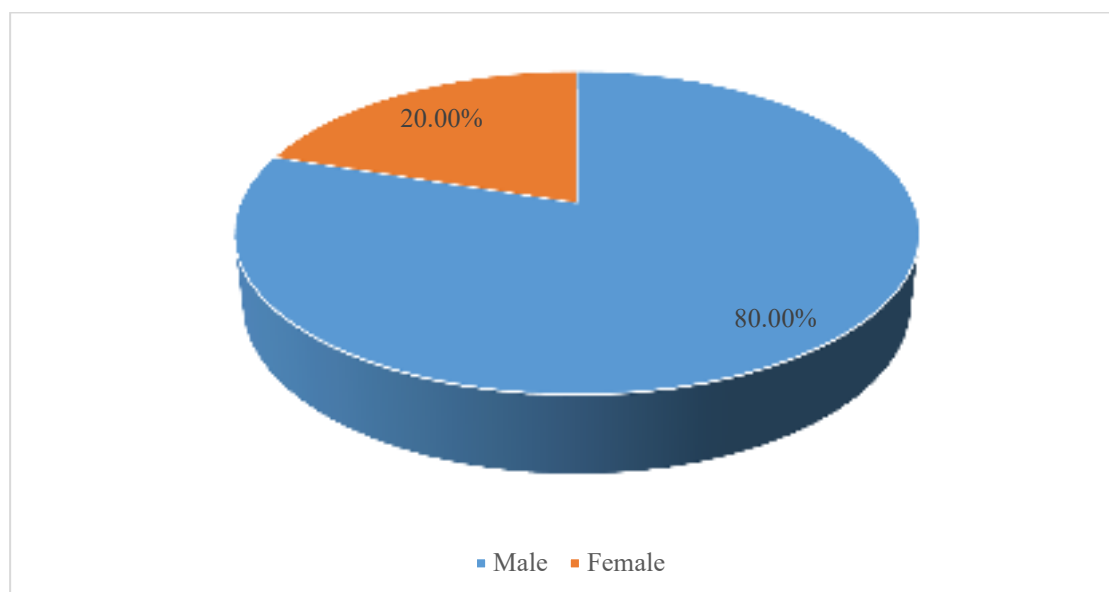
The mean age of the study population was 39.93  $\pm$  16.1 years, ranging from 36.50 years to 72 years. (Table 5)

**Table 6: Descriptive analysis of gender in the study population (n=30)**

Gender	Frequency	Percentages
Male	24	80.00%
Female	6	20.00%
Total	30	100%

Among the study population, the gender was male for 24 (80.00%) participants and female for 6 (20.00%) participants. (Table 6 & Figure 4)

**Figure 4: Pie chart of gender in the study population (n=30)**



**Table 7: Descriptive analysis of history in the study population (n=30)**

<b>History</b>	<b>Frequency</b>	<b>Percentages</b>
Headache	16	53.33%
Loss of consciousness	3	10%
Right hemiparesis	5	16.67%
Seizures	8	26.67%
Vertigo and right hemiparesis	1	3.33%

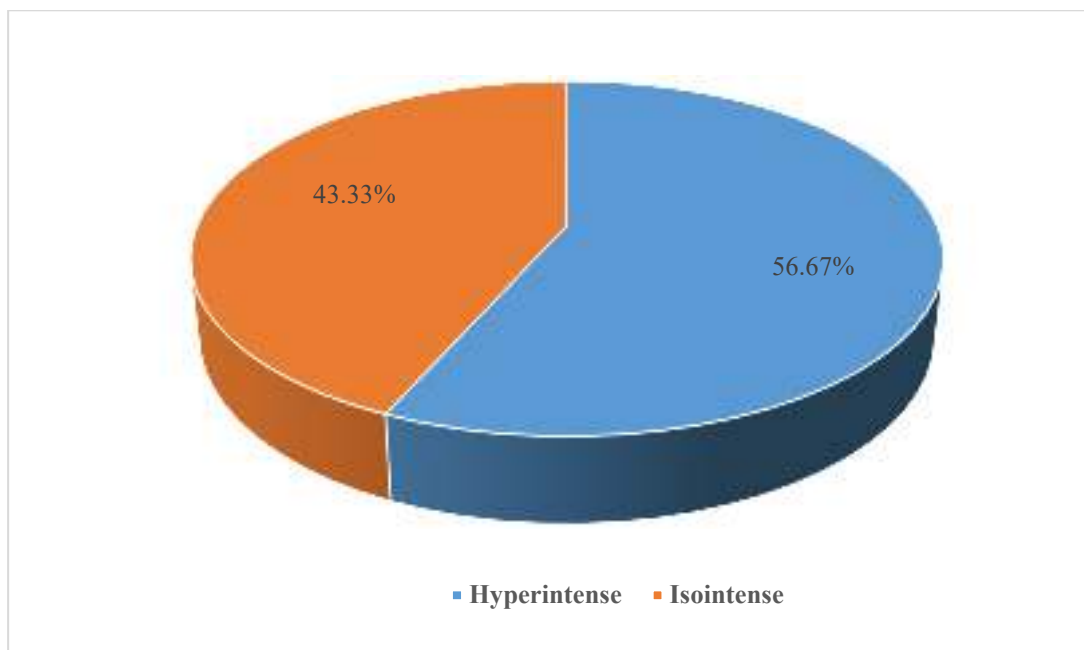
Among the study population, 16(53.33%) participants had a headache, 3(10%) had a loss of consciousness,5(16.67%) had right hemiparesis, 8(26.67%) had seizures and 1(3.33%) participant had vertigo and right hemiparesis. (Table 7)

**Table 8: Descriptive analysis of signal intensity within the venous sinuses on T1 W in the study population (n=30)**

<b>Signal intensity within the venous sinus on T1 W</b>	<b>Frequency</b>	<b>Percentages</b>
Hyperintense	17	56.67%
Isointense	13	43.33%
Total	30	100%

Among the study population, 17(56.67%) had hyperintense signal within the venous sinuses on T1 W and 13(43.33%) had isointense signal. (Table 8 & Figure 5)

**Figure 5: Pie chart of signal intensity within the venous sinuses on T1 W in the study population (n=30)**

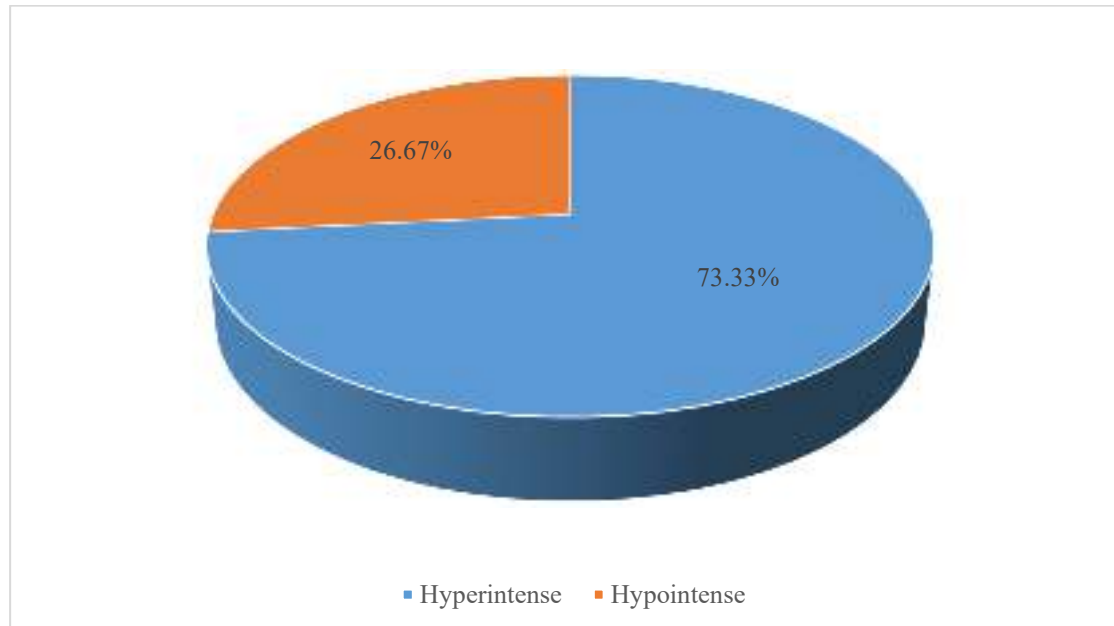


**Table 9: Descriptive analysis of signal intensity within the venous sinuses on T2 W in the study population (n=30)**

Signal intensity on T2 W	Frequency	Percentages
Hyperintense	22	73.33%
Hypointense	8	26.67%
Total	30	100%

Among the study population, 22(73.66%) had hyperintense signal within the venous sinuses on T2 W and 8(26.67%) had hypointense signal. (Table 9 & Figure 6)

**Figure 6: Pie chart of signal intensity within the venous sinuses on T2 W in the study population (n=30)**

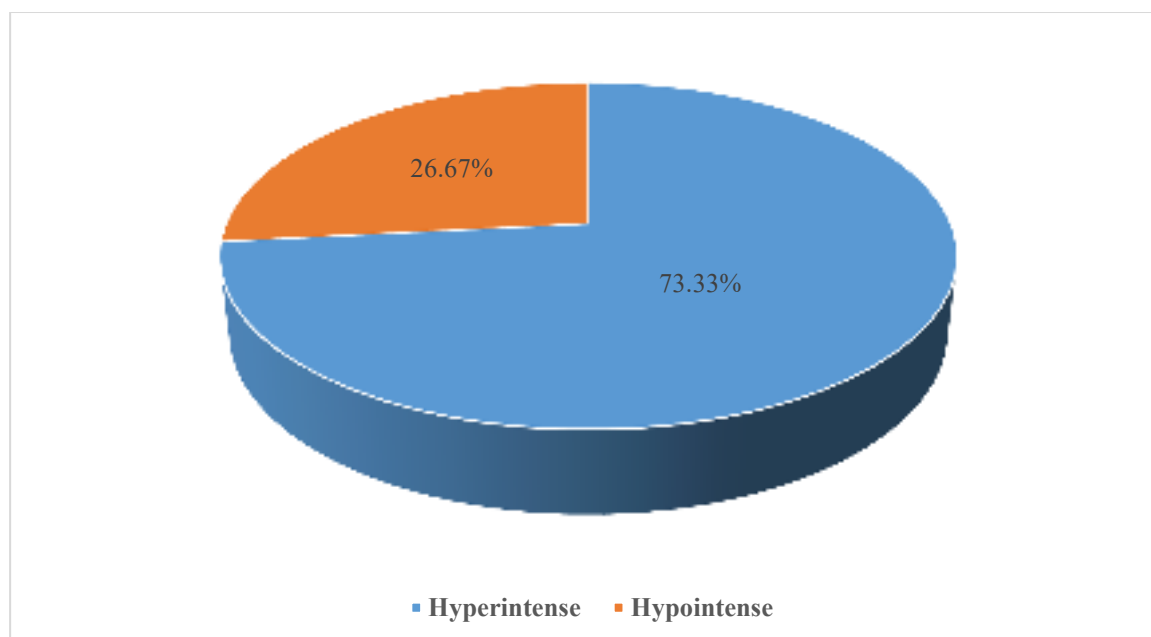


**Table 10: Descriptive analysis of signal intensity within the venous sinuses on FLAIR in the study population (n=30)**

Signal intensity on flair	Frequency	Percentages
Hyperintense	22	73.33%
Hypointense	8	26.67%
Total	30	100%

Among the study population, 22(73.33%) had hyperintense signal within the venous sinuses on FLAIR and 8(26.67%) had hypointense signal. (Table 10& Figure 7)

**Figure 7: Pie chart of signal intensity within the venous sinuses on FLAIR in the study population (n=30)**

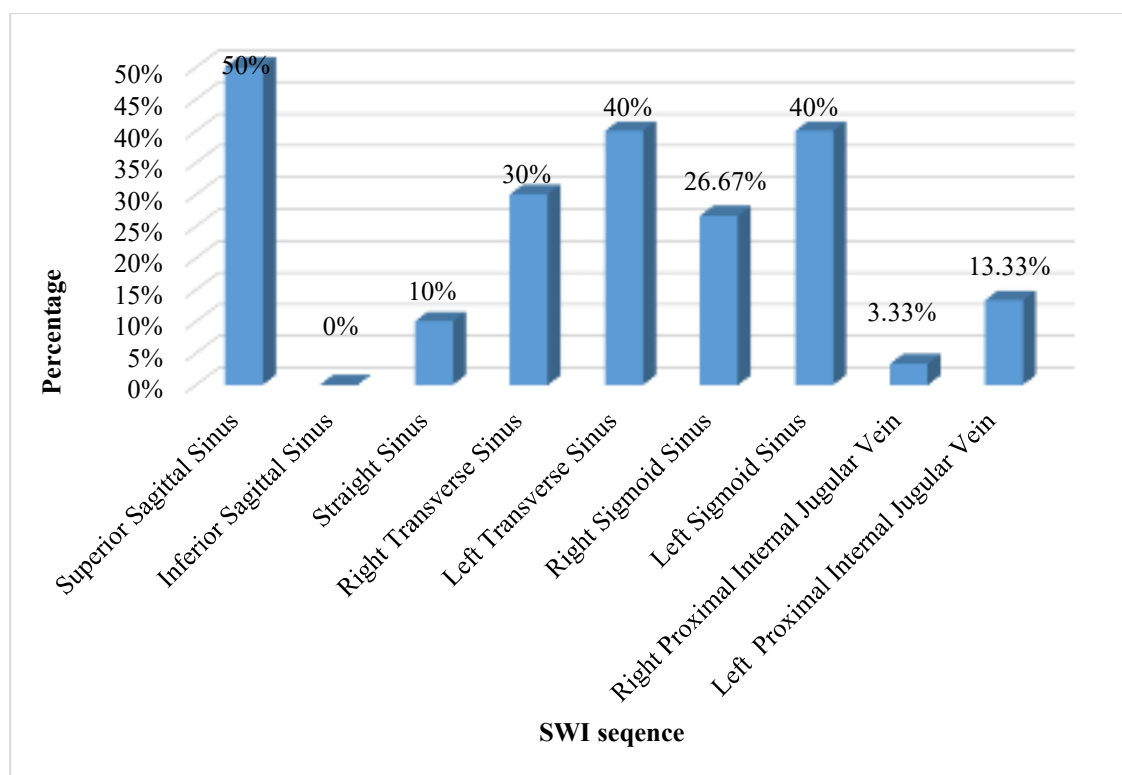


**Table 11: Descriptive analysis of SWI Sequence in the study population (n=30)**

SWI Sequence	Frequency	Percentages
<b>Superior Sagittal Sinus</b>		
Normal	15	50.00%
Abnormal	15	50.00%
<b>Inferior Sagittal Sinus</b>		
Normal	30	100%
<b>Straight Sinus</b>		
Normal	27	90.00%
Abnormal	3	10.00%
<b>Right Transverse Sinus</b>		
Normal	21	70.00%
Abnormal	9	30.00%
<b>Left Transverse Sinus</b>		
Normal	18	60%
Abnormal	12	40.00%
<b>Right Sigmoid Sinus</b>		
Normal	22	73.33%
Abnormal	8	26.67%
<b>Left Sigmoid Sinus</b>		
Normal	18	60%
Abnormal	12	40.00%
<b>Right Proximal Internal Jugular Vein</b>		
Normal	29	96.67%
Abnormal	1	3.33%
<b>Left Proximal Internal Jugular Vein</b>		
Normal	26	86.67%
Abnormal	4	13.33%

As per the SWI sequence, 15(50%) superior sagittal sinus, 30(100%), 3(10%) straight sinus, 9(30%) right transverse sinus, 12(40%) left transverse sinus, 8(26.67%) right sigmoid sinus, 12(40%) left sigmoid sinus, 1(3.33%) right proximal IJV and 4(13.33%) left proximal IJV were abnormal, i.e they showed MSE in the study. (Table 11 & Figure 8)

**Figure 8: Graphical presentation of SWI Sequence in the study population (n=30)**

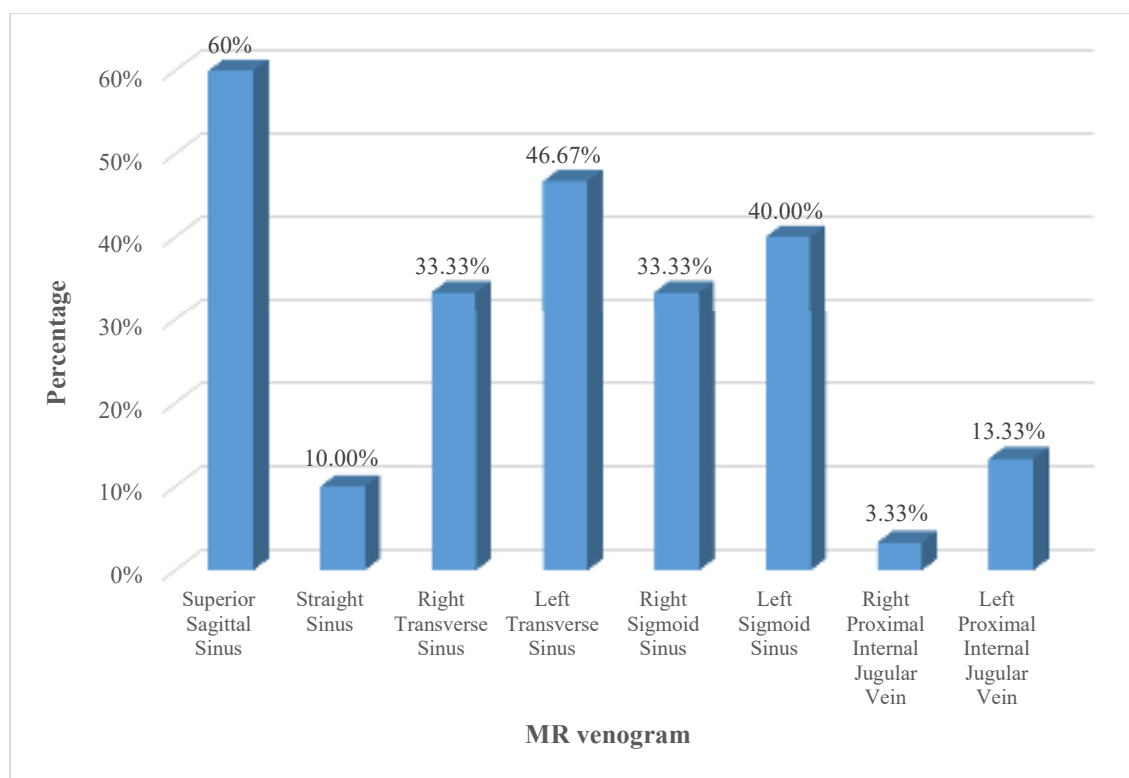


**Table 12: Descriptive analysis of MR Venogram (MRV-TOF) in the study population (n=30)**

<b>MR Venogram</b>	<b>Frequency</b>	<b>Percentages</b>
<b>Superior Sagittal Sinus</b>		
Normal	12	40.00%
Abnormal	18	60%
<b>Inferior Sagittal Sinus</b>		
Normal	30	100%
<b>Straight Sinus</b>		
Normal	27	90.00%
Abnormal	3	10.00%
<b>Right Transverse Sinus</b>		
Normal	20	66.67%
Abnormal	10	33.33%
<b>Left Transverse Sinus</b>		
Normal	16	53.33%
Abnormal	14	46.67%
<b>Right Sigmoid Sinus</b>		
Normal	20	66.67%
Abnormal	10	33.33%
<b>Left Sigmoid Sinus</b>		
Normal	18	60.00%
Abnormal	12	40.00%
<b>Right Proximal Internal Jugular Vein</b>		
Normal	29	96.67%
Abnormal	1	3.33%
<b>Left Proximal Internal Jugular Vein</b>		
Normal	26	86.67%
Abnormal	4	13.33%

As per the MR venogram, 18(60%) superior sagittal sinus, 30(100%), 3(10%) straight sinus, 10(33.33%) right transverse sinus, 14(46.67%) left transverse sinus, 10(33.33%) right sigmoid sinus, 12(40%) left sigmoid sinus, 1(3.33%) right proximal IJV and 4(13.33%) left proximal IJV were abnormal, i.e they showed absence of signal in the study. (Table 12 & Figure 9)

**Figure 9: Graphical presentation of MR venogram (MRV-TOF) in the study population (n=30)**



**Table 13: Descriptive analysis of parenchymal abnormalities in the study population (n=30)**

<b>Parenchymal Abnormalities</b>	<b>Frequency</b>	<b>Percentages</b>
<b>Edema</b>		
Normal	26	86.67%
Bilateral high frontal and left high parietal	1	3.33%
Left frontal and high frontal	1	3.33%
left temporal	1	3.33%
Right frontal	1	3.33%
<b>Haemorrhagic Venous Infarct</b>		
Normal	22	73.33%
Left temporal-parietal	2	6.67%
Left temporal -occipital	1	3.33%
Left frontal - temporal-parietal	1	3.33%
Left frontal, high frontal	1	3.33%
Left parietal	1	3.33%
Left temporal	1	3.33%
Right frontal-temporal-parietal	1	3.33%

Among the study population, 4(13.32%) patients had edema and 8(26.65%) patients had haemorrhagic venous infarct. (Table 13)

**Table 14: Descriptive analysis of cortical veins showing magnetic susceptibility effect (MSE) in the study population (n=30)**

<b>Cortical Veins showing MSE</b>	<b>Frequency</b>	<b>Percentages</b>
Normal	19	63.33%
Bilateral high frontal	6	20.00%
Left high frontal	2	6.67%
Bilateral high parietal	1	3.33%
Left temporal	1	3.33%
Right frontotemporal	1	3.33%
Total	30	100%

Among the study population, 19(63.33%) showed no MSE of cortical veins, 6(20%) had bilateral high frontal, 2(6.67%) had left high frontal and 1(3.33%) had bilateral high parietal, one 1(3.33%) left temporal and one 1(3.33%) right frontotemporal regions. (Table 14)

**Table 15: Comparison of MR Venogram(MRV-TOF) with SWI Sequence (n=30)**

SWI sequence	MR Venogram	
	Normal	Abnormal
<b>Superior Sagittal Sinus</b>	<b>(N=12)</b>	<b>(N=18)</b>
Normal	12 (100%)	3 (16.67%)
Abnormal	0 (0%)	15 (83.33%)
<b>Straight Sinus</b>	<b>(N=27)</b>	<b>(N=3)</b>
Normal	27 (100%)	0 (0%)
Abnormal	0 (0%)	3 (100%)
<b>Right Transverse Sinus</b>	<b>(N=20)</b>	<b>(N=10)</b>
Normal	20 (100%)	1 (10%)
Abnormal	0 (0%)	9 (90%)
<b>Left Transverse Sinus</b>	<b>(N=16)</b>	<b>(N=14)</b>
Normal	16 (100%)	2 (14.29%)
Abnormal	0 (0%)	12 (85.71%)

\*No statistical test was applied- due to 0 subjects in the cells

Taking MR venogram as the gold standard, 18 (60%) superior sagittal sinus were detected as abnormal and 12 (100%) superior sagittal sinus were normal, whereas on SWI sequence 15 (83.33%) were detected as abnormal. 3 (10%) straight sinus were detected as abnormal and 27 (100%) straight sinus were normal, whereas on SWI sequence 3 (100%) were detected as abnormal. 10 (33.33%) right transverse sinus were detected as abnormal and 20 (100%) right transverse sinus was normal, whereas on SWI sequence 9 (90%) were detected as abnormal. 14 (46.67%) left transverse sinus were detected as abnormal and 16 (100%) left transverse sinus were normal, whereas on SWI sequence 12 (85.71%) were detected as abnormal. (Table 15)

**Table 16: Predictive validity of SWI Sequence in diagnosing cerebral venous thrombosis (CVT) (n=30)**

<b>Parameter</b>	<b>Superior sagittal sinus Value</b>	<b>Straight sinus Value</b>	<b>Right transverse sinus Value</b>	<b>Left transverse sinus Value</b>
Sensitivity	100.00% (73.54% - 100.00%)	100.00% (87.23% - 100.00%)	100.00% (83.16% - 100.00%)	100.00% (79.41% - 100.00%)
Specificity	83.33% (58.58% - 96.42%)	100.00% (29.24% - 100.00%)	90.00% (55.50% - 99.75%)	85.71% (57.19% - 98.22%)
False positive rate	16.67% (3.58% - 41.42%)	0.00% (0 - 70.76%)	10.00% (0.25% - 44.50%)	14.29% (1.78% - 42.81%)
False negative rate	0.00% (0 - 26.46%)	0.00% (0 - 12.77%)	0.00% (0 - 16.84%)	0.00% (0 - 20.59%)
Positive predictive value	80.00% (51.91% - 95.67%)	100.00% (87.23% - 100.00%)	95.24% (76.18% - 99.88%)	88.89% (65.29% - 98.62%)
Negative predictive value	100.00% (78.20% - 100.00%)	100.00% (29.24% - 100.00%)	100.00% (66.37% - 100%)	100.00% (73.54% - 100.00%)
Diagnostic accuracy	90.00% (73.47% - 97.89%)	100.00% (88.43% - 100.00%)	96.67% (82.78% - 99.92%)	93.33% (77.93% - 99.18%)

---

The SWI sequence (SSS, SS, TSR and TSL) had sensitivity of 100.00% (95% CI 73.54% - 100.00%), 100.00% (95% CI 87.23% - 100.00%), 100.00% (95% CI 83.16% - 100.00%) and 100.00% (95% CI 79.41% - 100.00%) in diagnosing cerebral venous thrombosis(CVT). specificity was 83.33% (95% CI 58.58% - 96.42%), 100.00% (95% CI 29.24% - 100.00%), 90.00% (95% CI 55.50% - 99.75%) and 85.71% (95% CI 57.19% - 98.22%), false positive rate was 16.67% (95% CI (3.58% - 41.42%), 0% (95% CI 0% - 70.76%), 100.00% (95% CI 0.25% - 44.50%) and 14.29% (95% CI 1.78% - 42.81%) ,false negative rate was 0% (95% CI (0% - 26.46%), 0% (95% CI 0% - 12.77%), 0% (95% CI 0% - 16.84%) and 0% (95% CI 0% - 20.59%), positive predictive value was 80% (95% CI (51.91% - 95.67%), 100.00% (95% CI 87.23% - 100.00%), 95.24% (95% CI 76.18% - 99.88%) and 88.89% (95% CI 65.29% - 98.62%), negative predictive value was 100.00% (95% CI (78.20% - 100.00%), 100.00% (95% CI 29.24% - 100.00%), 100.00% (95% CI 66.37% - 100.00%) and 100.00% (95% CI 73.54% - 100.00%) ,and the total diagnostic accuracy was 90% (95% CI (73.47% - 97.89%), 100.00% (95% CI 88.43% - 100.00%), 96.67% (95% CI 82.78% - 99.92%) and 93.33% (95% CI 77.93% - 99.18%). (Table 16)

**Table 17: Comparison of MR Venogram (MRV-TOF) with SWI Sequence (n=30)**

SWI Sequence	MR Venogram	
	Normal	Abnormal
<b>Right sigmoid sinus</b>	<b>(N=20)</b>	<b>(N=10)</b>
Normal	20 (100%)	2 (20%)
Abnormal	0 (0%)	8 (80%)
<b>Left sigmoid sinus</b>	<b>(N=18)</b>	<b>(N=12)</b>
Normal	18 (100%)	0 (0%)
Abnormal	0 (0%)	12 (100%)
<b>Right Proximal Internal Jugular Vein</b>	<b>(N=29)</b>	<b>(N=1)</b>
Normal	29 (100%)	0 (0%)
Abnormal	0 (0%)	1 (100%)
<b>Left Proximal Internal Jugular Vein</b>	<b>(N=26)</b>	<b>(N=4)</b>
Normal	26 (100%)	0 (0%)
Abnormal	0 (0%)	4 (100%)

\*No statistical test was applied- due to 0 subjects in the cells

Taking MR venogram as the gold standard, 10 (33.33%) right sigmoid sinuses were detected as abnormal and 20 (100%) right sigmoid sinus was normal, whereas on SWI sequence 8(80%) were detected as abnormal. 12 (40%) left sigmoid sinus were detected as abnormal and 18 (100%) left sigmoid sinus were normal, whereas on SWI sequence 12 (100%) were detected as abnormal. 1 (33.33%) right proximal IJV were detected as abnormal and 29 (100%) right proximal IJV were normal, whereas on SWI sequence 1 (100%) were detected as abnormal. 4 (13.33%) left proximal IJV were detected as abnormal and 26 (100%) left proximal IJV were normal, whereas on SWI sequence 4 (100%) were detected as abnormal. (Table 17)

**Table 18: Predictive validity of SWI Sequence in diagnosing cerebral venous thrombosis (CVT) (n=30)**

<b>Parameter</b>	<b>Right sigmoid sinus Value</b>	<b>Left sigmoid sinus Value</b>	<b>Right Proximal IJV Value</b>	<b>Left Proximal IJV Value</b>
Sensitivity	100.00% (83.16% - 100.00%)	100.00% (81.47% - 100.00%)	100.00% (88.06% - 100.00%)	100.00% (86.77% - 100.00%)
Specificity	80.00% (44.39% - 97.48%)	100.00% (73.54% - 100.00%)	100.00% (2.50% - 100.00%)	100.00% (39.76% - 100.00%)
False positive rate	20.00% (2.52% - 55.61%)	0.00% (0 - 26.46%)	0.00% (0 - 97.50%)	0.00% (0 - 60.24%)
False negative rate	0.00% (0 - 16.84%)	0.00% (0 - 18.53%)	0.00% (0 - 11.94%)	0.00% (0 - 13.23%)
Positive predictive value	90.91% (70.84% - 98.88%)	100.00% (81.47% - 100.00%)	100.00% (88.06% - 100.00%)	100.00% (86.77% - 100.00%)
Negative predictive value	100.00% (63.06% - 100.00%)	100.00% (73.54% - 100.00%)	100.00% (2.50% - 100.00%)	100.00% (39.76% - 100.00%)
Diagnostic accuracy	93.33% (77.93% - 99.18%)	100.00% (88.43% - 100.00%)	100.00% (88.43% - 100.00%)	100.00% (88.43% - 100.00%)

The SWI sequence (SS right, SS left, PIJV right and PIJV left) had sensitivity of 100.00% (95% CI 83.16% - 100.00%), 100.00% (95% CI 81.47% - 100.00%), 100.00% (95% CI 88.06% - 100.00%) and 100.00% (95% CI 86.77% - 100.00%) in diagnosing cerebral venous thrombosis(CVT). Specificity was 80% (95% CI 44.39% - 97.48%), 100.00% (95% CI 73.54% - 100.00%), 100% (95% CI 2.50% - 100%) and

100% (95% CI 39.76% - 100%), false positive rate was 20% (95% CI (2.52% - 55.61%), 0% (95% CI 0% - 26.46%), 0% (95% CI 0% - 97.50%) and 0% (95% CI 0% - 60.24%), false negative rate was 0% (95% CI (0% - 16.84%), 0% (95% CI 0% - 18.53%), 0% (95% CI 0% - 11.94%) and 0% (95% CI 0% - 13.23%), positive predictive value was 90.91% (95% CI (70.84% - 98.88%), 100.00% (95% CI 81.47% - 100.00%), 100% (95% CI 88.06% - 100%) and 100% (95% CI 86.77% - 100%), negative predictive value was 100.00% (95% CI (63.06% - 100.00%), 100.00% (95% CI 73.54% - 100.00%), 100.00% (95% CI 2.50% - 100.00%) and 100.00% (95% CI 39.76% - 100.00%) and the total diagnostic accuracy was 93.33% (95% CI (77.93% - 99.18%), 100.00% (95% CI 88.43% - 100.00%), 100% (95% CI 88.43% - 100%) and 100% (95% CI 88.43% - 100%). (Table 18)

## **DISCUSSION**

CVT/CVST is a rare presentation of venous thromboembolism. It is considered a rare cause of stroke in young with increased mortality in that group. It is associated with severe disability and neurological deficits. The clinical presentation is highly variable and nonspecific. Hence, the diagnosis mainly relies on neuroimaging. MRI is considered as the investigation of choice for diagnosis because of its non-invasiveness and highly sensitivity in detecting CVT. Various MRI sequences and MRV are being used extensively for making a diagnosis of CVT.<sup>13, 14</sup> There has been a recent emergence of interest on the SWI for detection of the magnetic susceptibility signals produced by degraded products.<sup>12, 15</sup> In order to make high-quality recommendations for diagnosis, there is a lack of evidence. Hence the present hospital-based observational study was carried out to compare the diagnostic accuracy of SWI with MRV-TOF in the diagnosis of CVT.

### **Baseline demographic and clinical characteristics at presentation:**

The present study is a tertiary hospital-based observational study. The sample size was 30 subjects, who were suspected clinically or detected by CT and then referred to the Radio-diagnosis department for MRI. A 3T MRI machine manufactured by SIEMENS was used in the study. The following MRI sequences were done for each subject –

1. Axial – T 1 W, T 2 W, FLAIR.
2. Sagittal – T 1 W
3. Coronal - T 2 W
4. DWI and ADC

5. SWI – (Magnitude-axial, Phase-axial, Minimum intensity projection -axial, SWI- axial, sagittal, and coronal)
6. 3D TOF (MRV)

The objectives of the present study were similar to that of several other authors. Xu W et al.<sup>66</sup> in their meta-analysis assessed the diagnostic validity of CT, MRI for diagnosis of CVT, CVST. Gao L et al.<sup>53</sup> in their meta-analysis assessed the diagnostic validity of MRV for CVST diagnosis. Bidar F et al.<sup>67</sup> assessed the diagnostic validity of various gradient echo (GRE) MRI sequences against Conventional MRI for CVT. Linn J et al.<sup>43</sup> and Dormont D et al.<sup>61</sup> also assessed the diagnostic accuracy of various MRI sequences for CVT. Sadigh G et al.<sup>52</sup> assessed the diagnostic accuracy of various MRI sequences for DVST.

### **a. Age and Gender:**

CVT tends to commonly occur in old age and female gender, especially during the postpartum period. The mean age of the present study population was  $39.93 \pm 16.1$  years. The minimum age of the included subject was 16 years while the maximum age was 72 years. This median age of 39.9 years also underlines the role of CVT as an etiological factor in Young stroke.<sup>70</sup> (Stroke occurring in people aged less than 45 years). Devasagayam S et al.<sup>18</sup> observed the median age of the subjects with DVT in their retrospective record-based study to be higher than the present study at around 49 years with a range of 40 to 61 years. They reviewed the records of hospital-admitted adults in Australia in their study while the present study did not have an age restriction for including the subject into the study. The present study was also based only on a single centre compared to their multi-centric study. The difference in the use of oral contraceptives, hormones used as medications between the countries could have also influenced the difference.

The majority of the subjects were males (80%) in the present study. This is not in line with the predilection that CVT is more common in females, especially in the puerperium. But Bidar F et al.<sup>67</sup> in their study observed that 75.5% of the subjects were females. But their study included only 17 subjects in total and hence their results may not be comparable. Devasagayam S et al.<sup>18</sup> in their study observed that 52% of the subjects were female. In their study, there was no statistically significant difference across gender with regards to CVT presentation. It was a more representative, record-based study (2005 to 2011) than the present study.

### **b. Clinical presentation:**

Clinical presentation is highly variable in CVT besides being nonspecific, so it's difficult to diagnose. Diagnosing CVT early can help in improving overall outcomes.

In CVT, the most common clinical symptom is Headache<sup>23</sup>, seen in around 80–90% of subjects. In the present study, 53.33% of subjects had a headache. The headache can present with various characteristics such as throbbing pain or migraine-like or thunderclap headache or band-like. It gives a clue to the etiological factor. CVT can also occur without a headache. They are more likely to be older and males, with the occurrence of paralysis, and seizures.<sup>28</sup> In the present study, 26.67% had seizures at presentation. Neurological deficits such as palsies of the cranial nerves, motor impairment or sensory impairment, and cortical blindness can also be a presentation of CVT.<sup>31</sup> They are generally seen in CVT of the non-inflammatory type. In subjects with ICH, delay in diagnosis can lead to mortality or severe disability. In the present study, 16.67% had right hemiparesis at presentation. Altered consciousness has been observed in 20% to 30% of subjects with CVT.<sup>5, 33</sup> In the present study, 10% of subjects had a loss of consciousness. A delay in the diagnosis

and further management can lead to disability, which may be permanent and sometimes even to death. Hence the main deciding factor in the course of management of CVT is Neuroimaging.<sup>7</sup> MRI is considered as the investigation of choice for diagnosis and is highly sensitive in detecting CVT.

**Table 19 : Comparison of baseline characteristics across the studies.**

S. No	Author	Published year or year of reporting	Country	Sample size	Major inclusion criteria	Gender (Male)	Mean age or common age group
1	Present study	2021	India	30	CT confirmed or clinically suspected CVT for various MRI sequences	80%	39.93 ± 16.1 years
2	Xu W et al. <sup>66</sup>	2018	Meta-analysis	4595	CT, MRI for diagnosis of CVT, CVST	-	-
3	Gao L et al. <sup>53</sup>	2018	Meta-analysis	1933	MRV for CVST diagnosis	-	-
4	Bidar F et al. <sup>67</sup>	2016	Iran	17	various gradient echo (GRE) MRI sequences vs Conventional MRI for CVT	24.5%	45 ± 18.76 years
5	Linn J et al. <sup>43</sup>	2010	Germany	30 CVT patient's vs 20 controls	Various MRI sequences for CVT	-	-
6	Sadigh G et al. <sup>52</sup>	2016	United States	36	Various (Seven) MRI sequences for DVST	-	-
7	Dormont D et al. <sup>61</sup>	1994	France	53	T1, T2 weighted MRI for CVT	-	-

**Table 20: Comparison of diagnostic accuracy across the studies.**

S. No	Author	Gold standard	Imaging technique being studied	Sensitivity	Specificity	Total diagnostic accuracy
1	Present study	MR venogram	SWI sequences 1. SSS 2. SS 3. RTS 4. LTS 5. RSS 6. LSS 7. RPIJV 8. LPIJV	100% 100% 100% 100% 100% 100% 100% 100%	83% 100% 90% 85.71% 80% 100% 100% 100%	90% 100% 96.67% 93.33% 93.3% 100% 100% 100%
2	Xu W et al. <sup>66</sup>	-	MRI-CVT  MRI-CVST	82%  80%	92%  91%	Total AUC – 0.92  0.93
3	Gao L et al. <sup>53</sup>	-	MRV	86%	94%	AUC – 0.947
4	Linn J et al. <sup>43</sup>	-	i. T2* WI ii. T1*WI iii. FLAIRw iv. vMRA v. DWI	i. 97.5% ii. 70% iii. 50% iv. 41.7% v. <30%	100% for all	
5	Sadigh G et al. <sup>52</sup>	CE MRV for DVST	i. T1*WI ii. T2*CE iii. T1*CE iv. T2 FLAIR v. T2 GRE vi. DWI vii. 3D T1 CE GRE	Overall sensitivity for MRI > 99% for both reviewers	Overall specificity (MRI) 14% for reviewer 1 48% for reviewer 2	55%, 61% 77%, 76% 79%, 80% 70%, 72% 64%, 66% 59%, 64% 77%, 81%

### **Characteristics of Neuroimaging:**

CT is the most commonly used imaging modality in subjects, who present with localized and acute neurological symptoms.<sup>35</sup> In a suspected case of acute CVT, CT can be used as a primary imaging modality but there is a need for additional imaging to diagnose and rule out CVT accurately.<sup>7, 38, 39</sup> MRI is the most commonly used and the best available imaging modality for the brain.<sup>43</sup> In the literature, the most common site involved in CVT was Transverse sinus followed by Superior sagittal sinus. The ISCVT determined the occurrence of CVT in various sites – transverse sinus (86%), superior sagittal sinus (62%), straight sinus (18%), cortical veins (17%), jugular veins (12%), a vein of Galen, and internal cerebral vein (11%).<sup>5</sup> Several technical factors affect the quality of the image obtained. They include a selection of proper sequences, imaging planes, positioning of the patient, and selection of proper coil. The various types of MRI sequences<sup>6, 36, 43, 45</sup>, that can be obtained are T1 & T2 W, SWI, DWI, MRA, MRV, FLAIR, DTI, and fMRI.

#### **a. T1, T2 W, and FLAIR MRI characteristics:**

In the present study, on T1 W, 56.67% had hyperintense signal intensity while 73.66% had hyperintense signal intensity on T2 W within the venous sinuses. On FLAIR imaging, 73.33% had hyperintense signal intensity within the venous sinuses.

Brain parenchymal findings were in the form of edema or haemorrhagic venous infarct. 13.32% of patients had edema of the cerebral parenchyma and 26.65% had haemorrhagic venous infarct. There was no predilection for the occurrence of these events in any single site.

### **b. SWI Sequence characteristics:**

On SWI sequence, the thrombus is visualized as an area of hypo intensity, termed as magnetic susceptibility effect(MSE) within the lumen of the affected vein or sinus. The sensitivity of SWI for detecting CVT has far exceeded that of routine T1 and T2 weighted images. There is easy visualization of thrombosed sinus or vein on SWI sequence as compared with other sequences Several studies have confirmed the usefulness of MRI for diagnosis of CVT.<sup>48, 60, 61</sup> On susceptibility-weighted imaging, Superior sagittal sinus, MSE was seen in 50% with MSE seen in its entire length was 33.33%. With regards to the inferior sagittal sinus, there was no MSE noted. In the Straight sinus, MSE was seen in 10% while in the right transverse sinus, MSE was seen in 30%. Left transverse sinus showed MSE in 40%. In right sigmoid sinus MSE was seen in 26.67% while in the left sigmoid sinus MSE was seen in 40%. In right Proximal IJV, MSE was seen in 33.33% while in left Proximal IJV MSE was seen in 13.3%.

With regards to cortical veins, 20% had MSE in bilateral high frontal region while 6.67% had MSE in left high frontal region.

### **c. MR venogram (MRV-TOF) characteristics:**

On MRV-TOF, the intraluminal thrombosis is identified by the absence of a flow signal. In the present study, in MRV-TOF, Superior sagittal sinus was not entirely visualized in 43.33% while Inferior sagittal sinus was normal in all the subjects. Straight sinus was not visualized in 10%, Right transverse sinus was not visualized in 33.33% while the left transverse sinus was not visualized in 46.67%. The right sigmoid sinus was not visualized in 33.33% while the left sigmoid sinus was not

visualized in 40%. The right proximal IJV was not visualized in 3.33% while the left proximal IJV was not visualized in 13.33%.

### **Comparison of Diagnostic accuracy of MR Venogram (MRV-TOF) with SWI Sequence:**

In the present study, MRV-TOF is considered as gold standard while SWI sequence is considered as a screening test. SWI sequence and MRV-TOF were considered as primary outcome variables in the analysis.

Taking MRV-TOF as the gold standard, the SWI sequence (for Superior sagittal sinus, Straight sinus, Right transverse sinus and left transverse sinus respectively) had

1. A sensitivity of 100% (for all) in diagnosing CVT.
2. Specificity was 83%, 100%, 90% and 85.71% respectively.
3. Total diagnostic accuracy was 90%, 100%, 96.67% and 93.33% respectively.

The SWI sequence (for Right sigmoid sinus, left sigmoid sinus, Right Proximal IJV, and Left Proximal IJV) had

1. A sensitivity of 100% (for all) in diagnosing CVT.
2. Specificity was 80%, 100%, 100% and 100% respectively.
3. Total diagnostic accuracy was 93.33%, 100%, 100% and 100% respectively.

Overall, in the present study, sensitivity was 100% for SWI on comparison with MRV-TOF. Specificity also varied between 80% to 100% for all the vessels. The total diagnostic accuracy also varied between 90% to 100% for all the vessels. The present study results were comparable with that of Sadigh G et al.<sup>52</sup> Sadigh G et al.<sup>52</sup> in their study observed that as compared to other sequences, the diagnostic performance of the 3D T1-weighted GRE CE sequences was statistically significantly more. They observed that the Area Under the Curve (AUC) values for T2-weighted

GRE sequences were 64% and 66% by two reviewers respectively; and for the 3D T1-weighted GRE CE sequence, was 77% and 81% by two reviewers respectively. Similar to the present study, Bidar F et al.<sup>67</sup> also observed that “for the CVT diagnosis, T<sub>2</sub>\*-weighted conventional GRE sequences are best. They also observed that for demonstration of the thrombus, its exact location, small cortical vein details, collaterals, extension CE-MRV is perfect. T<sub>2</sub>\*-weighted GRE sequences are more sensitive to paramagnetic effects as compared to other sequences. T<sub>2</sub>\*-weighted GRE sequences are effective in the detection of CVT, particularly in acute stage”. But Linn J et al.<sup>43</sup> in their study observed that T2\*w showed the highest sensitivity for the detection of CVT (97.4%), followed by T1w (70%). FLAIR and MRV had a sensitivity of 50% and 41.7%, respectively. In a meta-analysis, subgroup level analysis of different MRV techniques confirmed that CE-MRV had better diagnostic accuracy than NCE-TOF and PC MRV”.<sup>53</sup> Gao L et al.<sup>53</sup> in their meta-analysis observed that MRV has excellent diagnostic accuracy in the diagnosis of CVST. In CVT, the loss of the T<sub>2</sub>\* -weighted signal is due to deoxyhemoglobin which produces a non-uniform magnetic field and rapid dephasing of proton spins. The paramagnetic molecules have this property and its termed as ‘magnetic susceptibility effect’ and results in a signal loss (darkening) best seen in T<sub>2</sub>\*/SWI.<sup>63-65</sup> Thus T<sub>2</sub>\*/ SWI not only helps in the detection of thrombosed sinus but also helps in the detection of hemorrhagic venous infarct.

MRI is the investigation of choice for diagnosis as it is non-invasive besides being highly sensitive in detecting CVT. Various MRI sequences are used extensively for making a diagnosis of CVT.<sup>13, 14</sup> The sensitivity of SWI for detecting CVT has far exceeded than that of routine T1 and T2 weighted images.

## **CONCLUSION**

1. One of the rare presentations of venous thromboembolism is CVT, but with possibly fatal consequences
2. The clinical presentation is highly variable and nonspecific.
3. The present study is a tertiary hospital-based observational study which was carried out on a sample of 30 subjects presenting with CVT suspected clinically or detected by CT and then referred to the radiology department for 3T MRI with various sequences.
4. The mean age of the present study population was  $39.93 \pm 16.1$  years.
5. The majority of the subjects were males (80%)
6. 53.33% of subjects had a headache, 26.67% had seizures, 16.67% had right hemiparesis and 10% had a loss of consciousness
7. In T1 MRI, 56.67% had hyperintense signal intensity while 73.66% had hyperintense signal intensity on T2 MRI within the venous sinues. On FLAIR imaging, 73.33% had hyperintense signal intensity within the venous sinues.
8. 13.32% of patients had edema of the cerebral parenchyma and 26.65% had haemorrhagic venous infarct. There was no predilection for the occurrence of these events in any single site.
9. In SWI, the Superior sagittal sinus showed MSE in 50% with MSE in its entire length was seen in 33.33%. The inferior sagittal sinus had no MSE. In the Straight sinus, MSE was seen in 10% while in the right transverse sinus, MSE was seen in 30%. Left transverse sinus MSE was seen in 40%.
10. In MRV, Superior sagittal sinus was not entirely visualized in 43.33% while Inferior sagittal sinus was normal in all the subjects. Straight sinus was not

visualized in 10%, Right transverse sinus was not visualized in 33.33% while the left transverse sinus was not visualized in 46.67%.

11. Overall, in the present study, sensitivity was 100% for SWI on comparison with MRV. Specificity also varied between 80% to 100% for all the vessels. The total diagnostic accuracy also varied between 90% to 100% for all the vessels.
12. Taking MR venogram as the gold standard, the SWI sequence (for Superior sagittal sinus, Straight sinus, Right transverse sinus and left transverse sinus respectively) had a sensitivity of 100% (for all) in predicting MR venogram, specificity was 83%, 100%, 90%, and 85.71% respectively and total diagnostic accuracy was 90%, 100%, 96.67%, and 93.33% respectively.
13. The SWI sequence (for Right sigmoid sinus, left sigmoid sinus, Right Proximal IJV, and Left Proximal IJV) had a sensitivity of 100% (for all) in predicting MR venogram, specificity was 80%, 100%, 100%, and 100% respectively and total diagnostic accuracy was 93.33%, 100%, 100%, and 100% respectively.

## **SUMMARY**

Cerebral venous thrombosis (CVT) / Cerebral Venous Sinus Thrombosis (CVST) is a rare presentation of venous thromboembolism. Clinical presentation is highly variable in CVT besides being nonspecific. Neuroimaging plays an important role in diagnosing, evaluating the complications and in the prognosis. The diagnosis of CVT could be missed on T1 and T2 W sequences. In cases without any clinical suspicion, the routine sequences performed without angiography can miss the diagnosis. There has been a recent emergence of literature on the increased diagnostic accuracy of SWI for detection of the magnetic susceptibility effect (MSE) produced by degraded products in the blood such as deoxyhaemoglobin in subjects with acute intraparenchymal hemorrhage. But there is a lack of sufficient evidence in the present region to make clinical recommendations.

Hence the present study was carried out with the objective of comparing the diagnostic accuracy of SWI with MRV-TOF for CVT.

A hospital based observational study was done on 30 patients referred for MRI brain and MRV to the Department of Radio-Diagnosis at The KLE'S Dr. Prabhakar Kore Hospital & MRC, Belgaum. The major inclusion criteria was patients of cerebral venous thrombosis suspected clinically or detected by CT Brain scan and referred to the radiology department for MRI. All patients were evaluated clinically and then MRI of the brain was performed using a 3 Tesla MRI scanner (Magnetom Avanto TIM, 18 channel; Siemens, Erlangen, Germany). The MRI sequences that were obtained were Axial – T 1 W, T 2 W, FLAIR, Sagittal – T 1 W, Coronal - T 2 W, DWI & ADC, SWI (Magnitude – axial, Phase – axial, Minimum intensity projection – axial, SWI – axial, sagittal & coronal) and 3D TOF (MRV). The primary outcome variables in this study were SWI sequence and MR venogram. Descriptive analysis

was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagrams and pie diagrams. Categorical outcomes were compared between study groups using the Chi square test. MR venogram was considered the gold standard. SWI sequence was considered a screening test. The sensitivity, specificity, predictive values, and diagnostic accuracy of the screening test along with their 95% CI were presented. P value <0.05 was considered statistically significant. The data were analysed by using SPSS software V.22.

The mean age of the present study population was  $39.93 \pm 16.1$  years. Majority of the subjects were males (80%). 53.33% of subjects had a headache, 26.67% had seizures, 16.67% had right hemiparesis and 10% had a loss of consciousness. In T1 MRI, 56.67% had hyperintense signal intensity within the venous sinues while 73.66% had hyperintense signal intensity within the venous sinues on T2 MRI. On FLAIR imaging, 73.33% had hyperintense signal intensity within the venous sinues. 13.32% of patients had edema of the cerebral parenchyma and 26.65% had haemorrhagic venous infarct. There was no predilection for the occurrence of these events in any single site.

In SWI, the Superior sagittal sinus showed MSE in 50% with MSE in its entire length was seen in 33.33%. The inferior sagittal sinus had no MSE. In the Straight sinus, MSE was seen in 10% while in the right transverse sinus, MSE was seen in 30%. Left transverse sinus MSE was seen in 40%. In MRV-TOF, Superior sagittal sinus was not entirely visualized in 43.33% while Inferior sagittal sinus was normal in all the subjects. Straight sinus was not visualized in 10%, Right transverse sinus was not visualized in 33.33% while the left transverse sinus was not visualized in 46.67%.

Taking MR venogram as the gold standard, the SWI sequence (for Superior sagittal sinus, Straight sinus, Right transverse sinus and left transverse sinus respectively) had a sensitivity of 100% (for all) in diagnosing CVT, specificity was 83%, 100%, 90%, and 85.71% respectively and total diagnostic accuracy was 90%, 100%, 96.67%, and 93.33% respectively.

The SWI sequence (for Right sigmoid sinus, left sigmoid sinus, Right Proximal IJV, and Left Proximal IJV) had a sensitivity of 100% (for all) in diagnosing CVT, specificity was 80%, 100%, 100%, and 100% respectively and total diagnostic accuracy was 93.33%, 100%, 100%, and 100% respectively.

Overall, in the present study, sensitivity was 100% for SWI on comparison with MRV. Specificity also varied between 80% to 100% for all the vessels. The total diagnostic accuracy also varied between 90% to 100% for all the vessels.

MRI is the investigation of choice for diagnosis as it is non-invasive besides being highly sensitive in detecting CVT. Various MRI sequences are used extensively for making a diagnosis of CVT. The sensitivity of SWI for detecting CVT has far exceeded than that of routine T1 and T2 weighted images. On SWI sequence, the thrombus is visualized as an area of hypo intensity, termed as MSE within the lumen of the affected vein or sinus. There is easy visualization of thrombosed sinus or vein on SWI sequence as compared with other sequences. Several studies have confirmed the usefulness of MRI for diagnosis of CVT.

The main limitation of the present study is that, it is only a single-center observational study of a small sample size. Future multi-centric studies involving a large sample size and the use of randomized sampling techniques could increase the validity of the results. It can further help in generating clinical and radiological evidence for making recommendations in the day-to-day practice.

## **LIMITATIONS AND RECOMMENDATIONS**

The present study is only a single-center observational study. Because of the practical limitations, only consecutive sampling was possible. This hospital-based consecutive sample is a non-probability sampling method and the internal validity of the present study results is questionable. The external validity is also low and the study results cannot be generalized because of the small sample size and single center sampling. MRI sequences also have some disadvantages. Besides the problems such as accessibility and cost, they are Subject to motion artifacts and require prolonged acquisition time for many images. They are inferior to CT in detecting acute haemorrhage and bony injuries.

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.

## **BIBLIOGRAPHY**

1. van Dam LF, van Walderveen MAA, Kroft LJM, Kruyt ND, Wermer MJH, van Osch MJP, et al. Current imaging modalities for diagnosing cerebral vein thrombosis - A critical review. *Thromb Res.* 2020;189:132-9.
2. Liang ZW, Gao WL, Feng LM. Clinical characteristics and prognosis of cerebral venous thrombosis in Chinese women during pregnancy and puerperium. *Sci Rep.* 2017;7:43866.
3. Bousser M-G, Ferro JM. Cerebral venous thrombosis: an update. *The Lancet Neurology.* 2007;6(2):162-70.
4. Alvis-Miranda HR, Milena Castellar-Leones S, Alcalá-Cerra G, Rafael Moscote-Salazar L. Cerebral sinus venous thrombosis. *J Neurosci Rural Pract.* 2013;4(4):427-38.
5. Ferro JM, Canhao P, Stam J, Bousser MG, Barinagarrementeria F, Investigators I. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke.* 2004;35(3):664-70.
6. Devis P, Knuttinen MG. Deep venous thrombosis in pregnancy: incidence, pathogenesis and endovascular management. *Cardiovasc Diagn Ther.* 2017;7(Suppl 3):S309-S19.
7. Saposnik G, Barinagarrementeria F, Brown RD, Jr., Bushnell CD, Cucchiara B, Cushman M, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2011;42(4):1158-92.

8. Borhani Haghghi A, Edgell RC, Cruz-Flores S, Feen E, Piriyaawat P, Vora N, et al. Mortality of cerebral venous-sinus thrombosis in a large national sample. *Stroke*. 2012;43(1):262-4.
9. Borhani Haghghi A, Ashjazadeh N, Safari A, Cruz-Flores S. Cerebral venous sinus thrombosis in iran: cumulative data, shortcomings and future directions. *Iran Red Crescent Med J*. 2012;14(12):805-10.
10. Hayman LA, Taber KH, Ford JJ, Bryan RN. Mechanisms of MR signal alteration by acute intracerebral blood: old concepts and new theories. *AJNR Am J Neuroradiol*. 1991;12(5):899-907.
11. Bradley WG, Jr. MR appearance of hemorrhage in the brain. *Radiology*. 1993;189(1):15-26.
12. Linfante I, Llinas RH, Caplan LR, Warach S. MRI features of intracerebral hemorrhage within 2 hours from symptom onset. *Stroke*. 1999;30(11):2263-7.
13. Boukobza M, Crassard I, Bousser MG, Chabriat H. MR imaging features of isolated cortical vein thrombosis: diagnosis and follow-up. *AJNR Am J Neuroradiol*. 2009;30(2):344-8.
14. Luo Y, Tian X, Wang X. Diagnosis and Treatment of Cerebral Venous Thrombosis: A Review. *Front Aging Neurosci*. 2018;10:2.
15. Vymazal J, Brooks RA, Baumgarner C, Tran V, Katz D, Bulte JW, et al. The relation between brain iron and NMR relaxation times: an in vitro study. *Magn Reson Med*. 1996;35(1):56-61.
16. Ulivi L, Squitieri M, Cohen H, Cowley P, Werring DJ. Cerebral venous thrombosis: a practical guide. *Pract Neurol*. 2020;20(5):356-67.
17. Coutinho JM, Zuurbier SM, Aramideh M, Stam J. The incidence of cerebral venous thrombosis: a cross-sectional study. *Stroke*. 2012;43(12):3375-7.

18. Devasagayam S, Wyatt B, Leyden J, Kleinig T. Cerebral Venous Sinus Thrombosis Incidence Is Higher Than Previously Thought: A Retrospective Population-Based Study. *Stroke*. 2016;47(9):2180-2.
19. Tadi P, Behgam B, Baruffi S. Cerebral Venous Thrombosis. [Updated 2021 Jan 31]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK459315/>.
20. Itrat A, Shoukat S, Kamal AK. Pathophysiology of cerebral venous thrombosis--an overview. *J Pak Med Assoc*. 2006;56(11):506-8.
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.
22. Guenther G, Arauz A. Cerebral venous thrombosis: a diagnostic and treatment update. *Neurologia*. 2011;26(8):488-98.
23. Agostoni E. Headache in cerebral venous thrombosis. *Neurol Sci*. 2004;25 Suppl 3:S206-10.
24. Gunes HN, Cokal BG, Guler SK, Yoldas TK, Malkan UY, Demircan CS, et al. Clinical associations, biological risk factors and outcomes of cerebral venous sinus thrombosis. *J Int Med Res*. 2016;44(6):1454-61.
25. Wasay M, Kojan S, Dai AI, Bobustuc G, Sheikh Z. Headache in Cerebral Venous Thrombosis: incidence, pattern and location in 200 consecutive patients. *J Headache Pain*. 2010;11(2):137-9.
26. Sparaco M, Feleppa M, Bigal ME. Cerebral Venous Thrombosis and Headache--A Case-Series. *Headache*. 2015;55(6):806-14.

27. Guner D, Tiftikcioglu BI, Uludag IF, Oncel D, Zorlu Y. Dural puncture: an overlooked cause of cerebral venous thrombosis. *Acta Neurol Belg.* 2015;115(1):53-7.
28. Coutinho JM, Stam J, Canhao P, Barinagarrementeria F, Bousser MG, Ferro JM, et al. Cerebral venous thrombosis in the absence of headache. *Stroke.* 2015;46(1):245-7.
29. Masuhr F, Busch M, Amberger N, Ortwein H, Weih M, Neumann K, et al. Risk and predictors of early epileptic seizures in acute cerebral venous and sinus thrombosis. *Eur J Neurol.* 2006;13(8):852-6.
30. Ferro JM, Canhao P, Bousser MG, Stam J, Barinagarrementeria F, Investigators I. Early seizures in cerebral vein and dural sinus thrombosis: risk factors and role of antiepileptics. *Stroke.* 2008;39(4):1152-8.
31. Paciaroni M, Palmerini F, Bogousslavsky J. Clinical presentations of cerebral vein and sinus thrombosis. *Front Neurol Neurosci.* 2008;23:77-88.
32. Korathanakhun P, Petpichetchian W, Sathirapanya P, Geater SL. Cerebral venous thrombosis: comparing characteristics of infective and non-infective aetiologies: a 12-year retrospective study. *Postgrad Med J.* 2015;91(1082):670-4.
33. Sassi SB, Touati N, Baccouche H, Drissi C, Romdhane NB, Hentati F. Cerebral Venous Thrombosis: A Tunisian Monocenter Study on 160 Patients. *Clin Appl Thromb Hemost.* 2017;23(8):1005-9.
34. Hassan KM, Kumar D. Reversible diencephalic dysfunction as presentation of deep cerebral venous thrombosis due to hyperhomocysteinemia and protein S deficiency: Documentation of a case. *J Neurosci Rural Pract.* 2013;4(2):193-6.
35. Linn J, Ertl-Wagner B, Seelos KC, Strupp M, Reiser M, Brückmann H et al. Diagnostic value of multidetector-row CT angiography in the evaluation of

- thrombosis of the cerebral venous sinuses. *AJNR Am J Neuroradiol.* 2007;28(5):946-52.
36. Ferro JM, Canhão P, Aguiar de Sousa D. Cerebral venous thrombosis. *La Presse Médicale.* 2016;45(12):e429-e50.
37. Herrmann KA, Sporer B, Yousry TA. Thrombosis of the internal cerebral vein associated with transient unilateral thalamic edema: a case report and review of the literature. *AJNR Am J Neuroradiol.* 2004;25(8):1351-5.
38. Ferro JM, Boussier MG, Canhao P, Coutinho JM, Crassard I, Dentali F, et al. European Stroke Organization guideline for the diagnosis and treatment of cerebral venous thrombosis - endorsed by the European Academy of Neurology. *Eur J Neurol.* 2017;24(10):1203-13.
39. Buyck PJ, Zuurbier SM, Garcia-Esperon C, Barboza MA, Costa P, Escudero I, et al. Diagnostic accuracy of noncontrast CT imaging markers in cerebral venous thrombosis. *Neurology.* 2019;92(8):e841-e51.
40. Dmytriw AA, Song JSA, Yu E, Poon CS. Cerebral venous thrombosis: state of the art diagnosis and management. *Neuroradiology.* 2018;60(7):669-85.
41. Rodallec MH, Krainik A, Feydy A, Helias A, Colombani JM, Julles MC, et al. Cerebral venous thrombosis and multidetector CT angiography: tips and tricks. *Radiographics.* 2006;26 Suppl 1:S5-18; discussion S42-3.
42. Wetzel SG, Kirsch E, Stock KW, Kolbe M, Kaim A, Radue EW. Cerebral veins: comparative study of CT venography with intraarterial digital subtraction angiography. *AJNR Am J Neuroradiol.* 1999;20(2):249-55.
43. Linn J, Michl S, Katja B, Pfefferkorn T, Wiesmann M, Hartz S, et al. Cortical vein thrombosis: the diagnostic value of different imaging modalities. *Neuroradiology.* 2010;52(10):899-911.

44. Gaikwad AB, Mudalgi BA, Patankar KB, Patil JK, Ghongade DV. Diagnostic role of 64-slice multidetector row CT scan and CT venogram in cases of cerebral venous thrombosis. *Emerg Radiol.* 2008;15(5):325-33.
45. Altinkaya N, Demir S, Alkan O, Tan M. Diagnostic value of T2\*-weighted gradient-echo MRI for segmental evaluation in cerebral venous sinus thrombosis. *Clin Imaging.* 2015;39(1):15-9.
46. Fellner FA, Fellner C, Aichner FT, Molzer G. Importance of T2\*-weighted gradient-echo MRI for diagnosis of cortical vein thrombosis. *Eur J Radiol.* 2005;56(2):235-9.
47. Ozturk K, Soyulu E, Parlak M. Dural venous sinus thrombosis: The combination of noncontrast CT, MRI and PC-MR venography to enhance accuracy. *Neuroradiol J.* 2018;31(5):473-81.
48. Lafitte F, Boukobza M, Guichard JP, Hoeffel C, Reizine D, Ille O, et al. MRI and MRA for diagnosis and follow-up of cerebral venous thrombosis (CVT). *Clinical Radiology.* 1997;52(9):672-9.
49. Rizzo L, Crasto SG, Ruda R, Gallo G, Tola E, Garabello D, et al. Cerebral venous thrombosis: role of CT, MRI and MRA in the emergency setting. *Radiol Med.* 2010;115(2):313-25.
50. Liang L, Korogi Y, Sugahara T, Onomichi M, Shigematsu Y, Yang D et al. Evaluation of the intracranial dural sinuses with a 3D contrast-enhanced MP-RAGE sequence: prospective comparison with 2D-TOF MR venography and digital subtraction angiography. *AJNR Am J Neuroradiol.* 2001;22(3):481-92.
51. Sari S, Verim S, Hamcan S, Battal B, Akgun V, Akgun H, et al. MRI diagnosis of dural sinus - Cortical venous thrombosis: Immediate post-contrast 3D GRE T1-

- weighted imaging versus unenhanced MR venography and conventional MR sequences. *Clin Neurol Neurosurg.* 2015;134:44-54.
52. Sadigh G, Mullins ME, Saindane AM. Diagnostic Performance of MRI Sequences for Evaluation of Dural Venous Sinus Thrombosis. *AJR Am J Roentgenol.* 2016;206(6):1298-306.
53. Gao L, Xu W, Li T, Yu X, Cao S, Xu H, et al. Accuracy of magnetic resonance venography in diagnosing cerebral venous sinus thrombosis. *Thromb Res.* 2018;167:64-73.
54. Jang W, Kwak HS, Chung GH, Hwang SB. Three-dimensional black-blood contrast-enhanced MRI improves detection of intraluminal thrombi in patients with acute ischaemic stroke. *Eur Radiol.* 2018;28(9):3840-7.
55. Xie G, Chen H, He X, Liang J, Deng W, He Z, et al. Black-blood thrombus imaging (BTI): a contrast-free cardiovascular magnetic resonance approach for the diagnosis of non-acute deep vein thrombosis. *J Cardiovasc Magn Reson.* 2017;19(1):4.
56. Holtackers RJ, Van De Heyning CM, Nazir MS, Rashid I, Ntalas I, Rahman H, et al. Clinical value of dark-blood late gadolinium enhancement cardiovascular magnetic resonance without additional magnetization preparation. *J Cardiovasc Magn Reson.* 2019;21(1):44.
57. Niu P-P, Yu Y, Guo Z-N, Jin H, Liu Y, Zhou H-W, et al. Diagnosis of non-acute cerebral venous thrombosis with 3D T1-weighted black blood sequence at 3T. *J Neurol Sci.* 2016;367:46-50.
58. Yang Q, Duan J, Fan Z, Qu X, Xie Y, Nguyen C, et al. Early Detection and Quantification of Cerebral Venous Thrombosis by Magnetic Resonance Black-Blood Thrombus Imaging. *Stroke.* 2016;47(2):404-9.

59. Westerbeek RE, Van Rooden CJ, Tan M, Van Gils AP, Kok S, De Bats MJ, et al. Magnetic resonance direct thrombus imaging of the evolution of acute deep vein thrombosis of the leg. *J Thromb Haemost.* 2008;6(7):1087-92.
60. Bianchi D, Maeder P, Bogousslavsky J, Schnyder P, Meuli RA. Diagnosis of cerebral venous thrombosis with routine magnetic resonance: an update. *Eur Neurol.* 1998;40(4):179-90.
61. Dormont D, Anxionnat R, Evrard S, Louaille C, Chiras J, Marsault C. MRI in cerebral venous thrombosis. *J Neuroradiol.* 1994;21(2):81-99.
62. Li D, Wang Y, Waight DJ. Blood oxygen saturation assessment in vivo using T2\* estimation. *Magn Reson Med.* 1998;39(5):685-90.
63. Edelman RR, Johnson K, Buxton R, Shoukimas G, Rosen BR, Davis KR, Brady TJ. MR of hemorrhage: a new approach. *AJNR Am J Neuroradiol.* 1986;7(5):751-6.
64. Schellinger PD, Jansen O, Fiebich JB, Hacke W, Sartor K. A Standardized MRI Stroke Protocol. *Stroke.* 1999;30(4):765-8.
65. Patel MR, Edelman RR, Warach S. Detection of hyperacute primary intraparenchymal hemorrhage by magnetic resonance imaging. *Stroke.* 1996;27(12):2321-4.
66. Xu W, Gao L, Li T, Ramdoyal N, Zhang J, Shao A. The Performance of CT versus MRI in the Differential Diagnosis of Cerebral Venous Thrombosis. *Thromb Haemost.* 2018;118(06):1067-77.
67. Bidar F, Faeghi F, Ghorbani A. Assessment of cerebral venous sinus thrombosis using T2 (\*)-weighted gradient echo magnetic resonance imaging sequences. *Iran J Neurol.* 2016;15(2):96-9.

68. Barnes SR, Haacke EM. Susceptibility-weighted imaging: clinical angiographic applications. *Magn Reson Imaging Clin N Am*. 2009;17(1):47-61.
69. IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.
70. Griffiths D, Sturm J. Epidemiology and etiology of young stroke. *Stroke Res Treat*. 2011;2011:209370.
71. Sven Haller, E. Mark Haacke, Majda M. Thurnher, Frederik Barkhof. Susceptibility-weighted Imaging: Technical Essentials and Clinical Neurologic Applications. *Radiology*. 2021 ; 299(1). Available from : <https://doi.org/10.1148/radiol.2021203071>
72. Willinek WA, Born M, Simon B, Tschampa HJ, Krautmacher C, Gieseke J, Urbach H, Textor HJ, Schild HH. Time-of-flight MR angiography: comparison of 3.0-T imaging and 1.5-T imaging--initial experience. *Radiology*. 2003 Dec;229(3):913-20. doi: 10.1148/radiol.2293020782. PMID: 14657322

**ANNEXURE I. ETHICAL CLEARANCE.**

	<p>K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH (Karnatak - to - University) Accredited 'A' Grade by NAAC 12<sup>th</sup> Cycle Placement Category 'A1' by MHRD (Govt)</p> <p><b>JAWAHARLAL NEHRU MEDICAL COLLEGE,</b> NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)</p> <p>Website: <a href="http://www.jnmc.edu">http://www.jnmc.edu</a> E-Mail : <a href="mailto:dmc@jnmc.edu">dmc@jnmc.edu</a></p>	<p>Phone: (+ 91-0831) Office : 2472550 Principal: 2471701 Fax No. +91 (0)831 – 2470759</p>
<hr/>		
<p>Ref: MDC/DOME/ 263</p>	<p>Date: 24/12/2019</p>	
<p>To,</p> <p><b>REGISTRATION NO. BS0119003</b> PG student in Radio-diagnosis J.N.Medical College, BELAGAVI.</p>		
<p>Sub: Institutional Ethical Clearance for the study.</p>		
<p>With reference to the above, we wish to inform you that your proposed research project titled "DIAGNOSTIC ACCURACY OF SUSCEPTIBILITY WEIGHTED IMAGING (SWI) TO DIAGNOSE THE CASES OF CEREBRAL VENOUS THROMBOSES (CVT) COMPARED TO MAGNETIC RESONANCE VENOGRAPHY (MRV) – ONE YEAR 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.</p>		
<p> (Dr. Anita Dalal) Member Secretary JNMC Institutional Ethics Committee on Human Subjects Research, J.N.Medical College, Belagavi.</p>	<p> (Dr. Roopa M Bellad) Chairman, JNMC Institutional Ethics Committee on Human Subjects Research, J.N.Medical College, Belagavi.</p>	
<p>94</p>		

**ANNEXURE II**

**INFORMED CONSENT**

**TITLE OF THE STUDY: “DIAGNOSTIC ACCURACY OF SUSCEPTIBILITY-WEIGHTED IMAGING (SWI) TO DIAGNOSE THE CASES OF CEREBRAL VENOUS THROMBOSIS (CVT) COMPARED TO MAGNETIC RESONANCE VENOGRAPHY (MRV) - ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY**

**PRINCIPAL INVESTIGATOR: REGISTRATION NO. BS0119003**

**INTRODUCTION AND PURPOSE:**

Cerebral venous thrombosis can lead to devastating disability, and even death, if not timely diagnosed and treated .Magnetic Resonance Imaging (MRI) is the current imaging tool of choice in the investigation of patients with cerebral venous thrombosis. The advent of high resolution MRI has significantly increased the chances of identifying a cause resulting in a positive clinical impact on the management of these patients.

**PROCEDURE:**

I request you to kindly participate in the study titled – “**DIAGNOSTIC ACCURACY OF SUSCEPTIBILITY-WEIGHTED IMAGING (SWI) TO DIAGNOSE THE CASES OF CEREBRAL VENOUS THROMBOSIS (CVT) COMPARED TO MAGNETIC RESONANCE VENOGRAPHY (MRV) - ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY**” at Dr. Prabhakar Kore charitable hospital and Medical Research Centre, Belgaum” is being conducted by **REGISTRATION NO. BS0119003**, 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. U will also be clinically examined as per the protocol drawn.

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

**BENEFITS:**

- Noninvasive modality

**COMPLICATIONS:**

- No risk to the patient has been documented from MR imaging of the brain conducted earlier.

**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 MRI as a investigation for low backache 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.

<b>REG. NO. BS0119003</b>	<b>Dr _____</b>	<b>Dr. Roopa M Bellad</b>
Post-Graduate, Department of Radio-Diagnosis. J.N.Medical College, Belagavi	Guide, Professor, Department of Radio-Diagnosis J.N.Medical College, Belagavi	Professor of Pediatrics Chairman, J.N. Medical College Institutional Ethical Committee for Human Subjects Research

**CONSENT TO PARTICIPATE IN RESEARCH STUDY:**

1. I understand that I am participating in the study, which includes Magnetic Resonance Imaging of brain.
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 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:



1. मैं समझता हूँ कि मैं अध्ययन में भाग ले रहा हूँ जिसमें एम्.आर.आई शामिल है |
2. मैं पुष्टि करता हूँ कि मैंने मरीज सूचना शीट में जानकारी पढ़ ली है और समझ ली है। अध्ययन में समझाया गया है कि अध्ययन में भाग लेने के फायदे और नुकसान के बारे में जानकारी के साथ मुझे विस्तार से बताया गया है। मुझे परीक्षण के सभी पहलुओं पर चर्चा करने का अवसर दिया गया है, प्रश्न पूछें और इस तरह से ऊपर दिए गए मुकदमे में सहभागिता की सहमति है।
3. यह समझें कि इस अध्ययन में भाग लेने का निर्णय पूरी तरह से स्वैच्छिक है और मुझे पता है कि मैं चुन सकता हूँ एक समय पर अध्ययन से वापस लेने के लिए
4. मेडिकल, वैज्ञानिक या शैक्षिक उद्देश्यों के लिए मेरे शरीर के उपयुक्त भाग सहित कार्य करने के लिए प्रक्रिया की तस्वीर या रिकॉर्डिंग के लिए सहमति दी गई है, बशर्ते मेरी पहचान चित्रों में या उन के साथ आने वाली वर्णनात्मक ग्रंथों में प्रकट नहीं हुई है।
5. मैं समझता हूँ कि इस अध्ययन में किए गए किसी भी महत्वपूर्ण जोखिम को शामिल नहीं किया गया है।
6. कोई गारंटी या आश्वासन किसी भी व्यक्ति द्वारा दिए गए परिणाम के रूप में नहीं दिया गया है।
7. इस फार्म पर मेरा हस्ताक्षर दर्शाता है कि मैंने ऊपर की जानकारी समझने के बाद खुशी-खुशी भाग लेने का फैसला किया है।

प्रतिभागी के नाम / कानूनी तौर पर अधिकृत प्रतिनिधि

हस्ताक्षर नाम

गवाह के हस्ताक्षर

साक्षात्कारकर्ता का नाम और हस्ताक्षर

दिनांक :

स्थान :

संशोधन अभ्यास क्रमांत सहभागी होण्यासाठी संमती

1. मलासमजतेकीमीयाअभ्यासातभागघेतआहे, ज्यातएम्आरआईसमाविष्टआहे.
2. मीपुष्टीकरतोकीमीरुग्णमाहितीपत्रकातमाहितीवाचलीआहेआणिसमजूनघेतलीआहे.  
अभ्यासातभागघेण्याच्याफायदेआणितोटेयाविषयीमाहितीसहप्रक्रियातपशीलानेमलासमजावूनसांगितलेगेले  
आहे. मलाचाचणीच्यासर्वपैलूंवरचर्चाकरण्याची, प्रश्नविचारण्याद्वारेआणिउपरोक्तदिलेल्याचाचणीतसहभा  
गीहोण्याससंमतीदेण्याचीसंधीदिलीगेलीआहे.
3. समजूनघ्याकीयाअभ्यासातभागघेण्याचानिर्णयपूर्णपणेस्वयंसेवीआहेआणिमलायाचीजाणीवआहेकीमी  
निवडूशकतोएकावेळेसअभ्यासातूनबाहेरपडण्यासाठी
4. वैद्यकीय, वैज्ञानिककिंवाशैक्षणिकहेतूसाठीमाझ्याशरीराच्यायोग्यभागांसहितकार्यासाठीछायाचित्रकाढ  
णेकिंवारेकॉर्डिंगकरण्याससंमतीदेणेम्हणजेमाझीओळखचित्रांमध्येकिंवात्यांच्यासोबतअसलेल्यावर्णनात्मक  
ग्रंथांमध्येउघडझालीनाही.
5. मलाहेसमजतेकीयाअभ्यासातकेलेल्याचाचणीमध्येकोणतेहीलक्षणीयधोकासमाविष्टनाही.
6. कोणतीहीहमीकिंवाआश्वासनकोणीहीमिळवूशकतीलअसेपरिणामम्हणूनदेतनाही.
7. याफॉर्मवरमाझेस्वाक्षरीअसेदर्शवतेकीमीउपरोक्तमाहितीसमजल्यानंतरसहभागीहोण्याचानिर्णयघेतला  
आहे.

सहभागीचेनाव / कायदेशीरपणेअधिकृतप्रतिनिधी

स्वाक्षरीचेनाव

साक्षीदारांचीसहीनाव :

मुलाखतकाराचेनावस्वाक्षरी

दिनांक :

ठिकाण

**ANNEXURE III  
STUDY PROFORMA**

NAME \_\_\_\_\_

AGE \_\_\_\_\_

OP/IP NO \_\_\_\_\_

ADDRESS \_\_\_\_\_

\_\_\_\_\_

MRI NUMBER:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS

PAST HISTORY

FAMILY HISTORY

MRI :

Signal intensity of venous sinuses on T1 w:

Signal intensity of venous sinuses on T2 w:

Signal intensity of venous sinuses on FLAIR:

MSE on SWI sequence :

- Superior sagittal sinus
- Inferior sagittal sinus
- Straight sinus
- Transverse sinus (right and left)
- Sigmoid sinus (right and left)
- Proximal IJV (right and left)

Non visualization of venous sinuses on MRV :

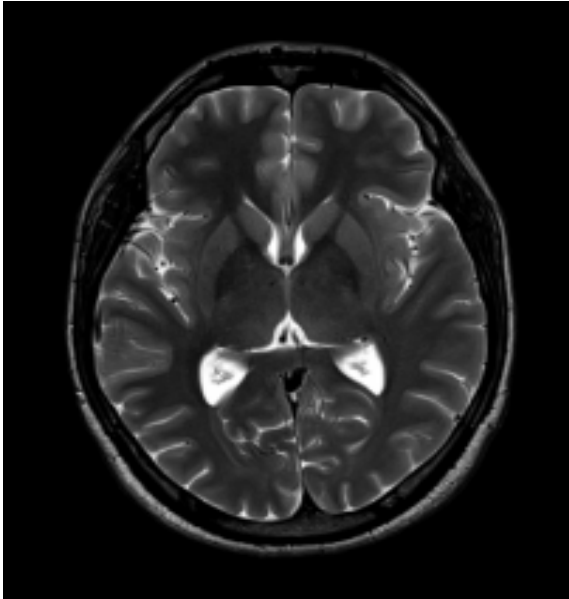
- Superior sagittal sinus
- Inferior sagittal sinus
- Straight sinus
- Transverse sinus (right and left)
- Sigmoid sinus (right and left)
- Proximal IJV (right and left)

Parenchymal abnormalities :

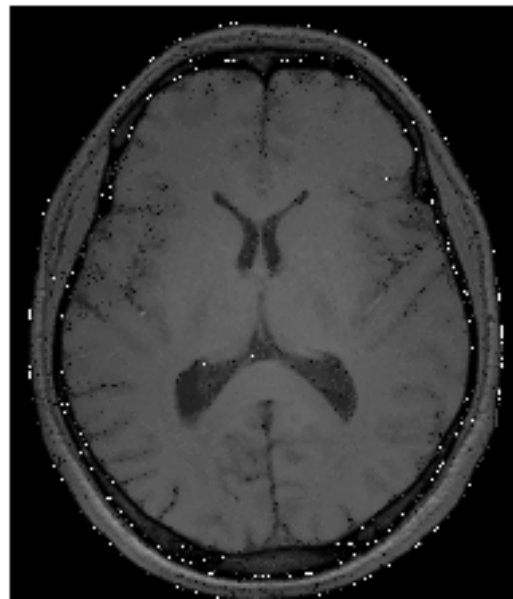
- Edema
- Haemorrhagic venous infarct
- MSE of Cortical veins

**ANNEXURE IV**

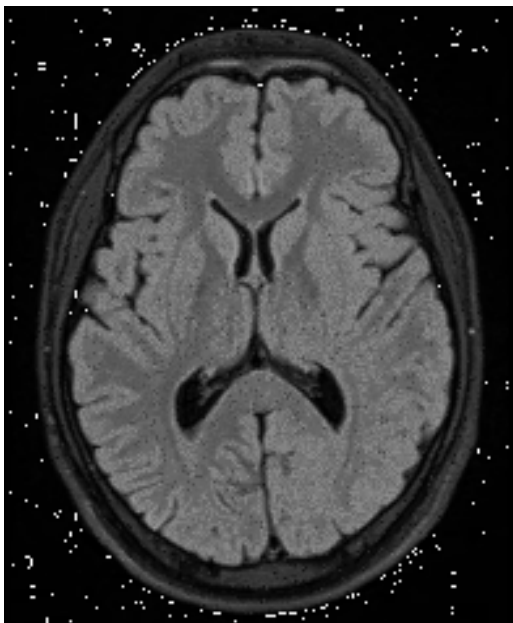
**CLINICAL IMAGES**



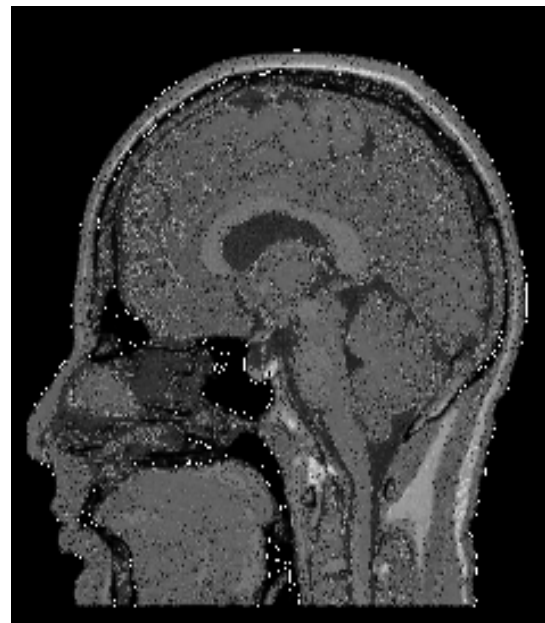
**T2-W axial**



**T1-W axial**



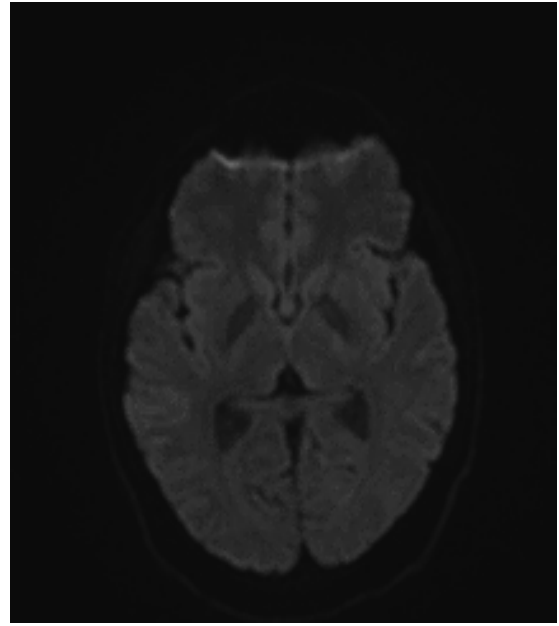
**FLAIR - axial**



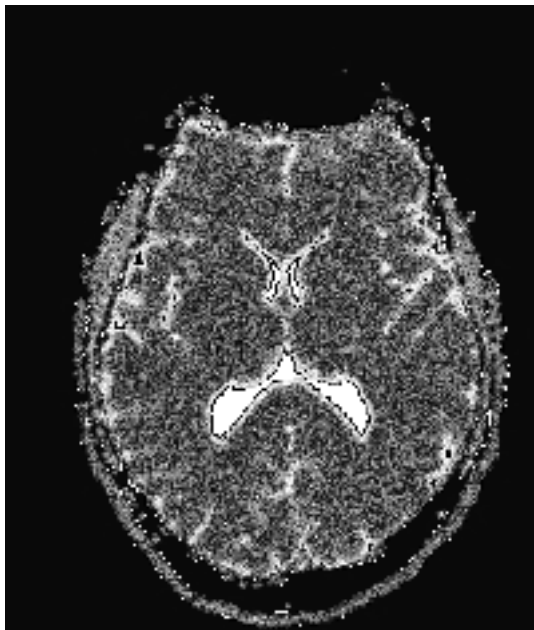
**T1W - Sagittal**



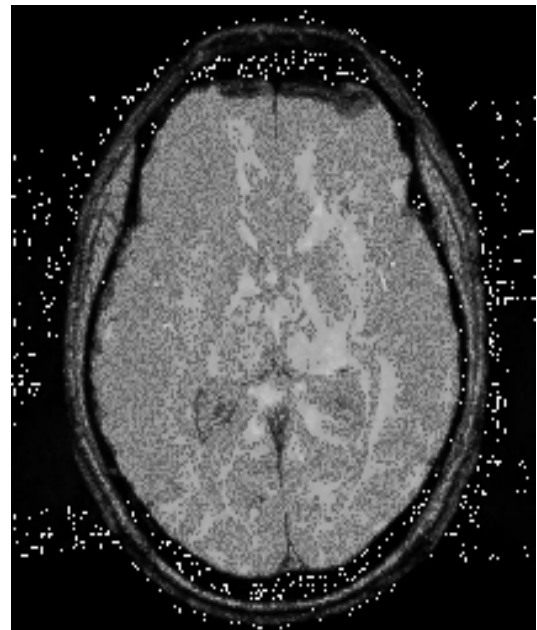
**T2W- coronal**



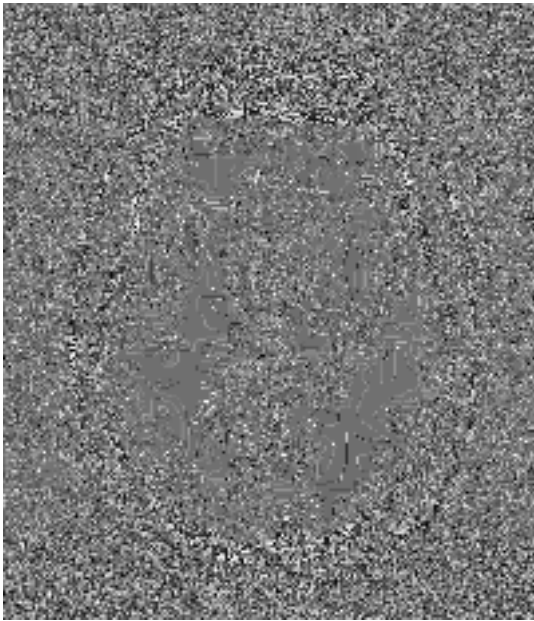
**DWI**



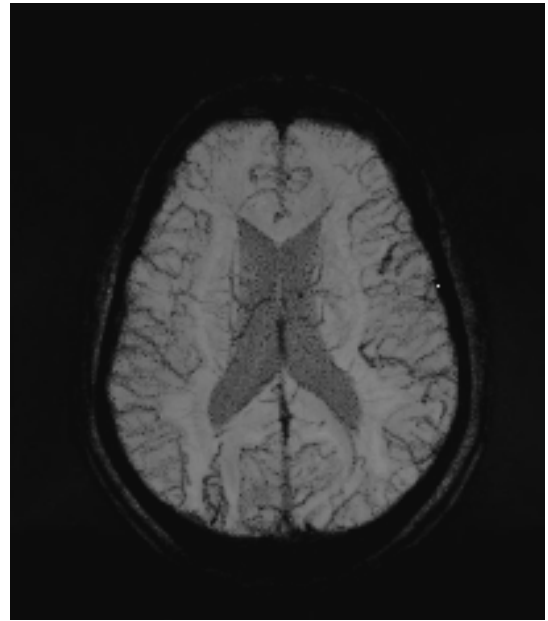
**ADC**



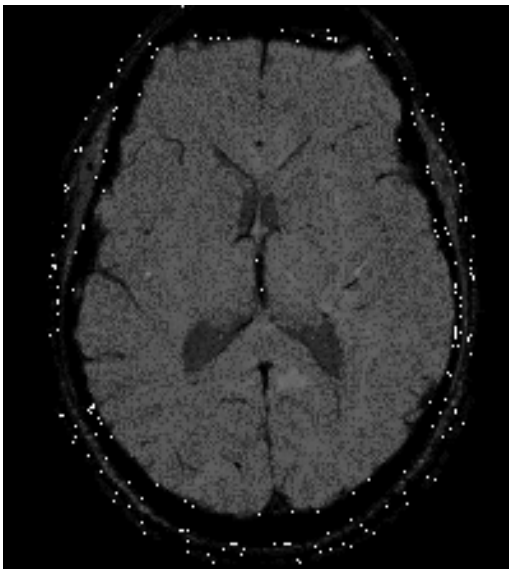
**Magnitude – axial**



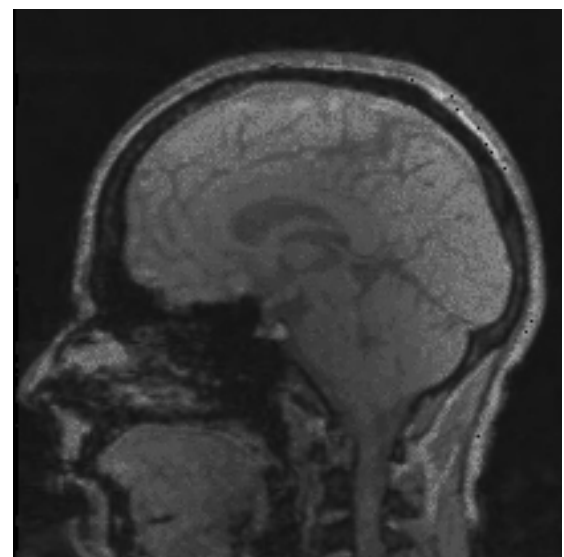
**Phase- axial**



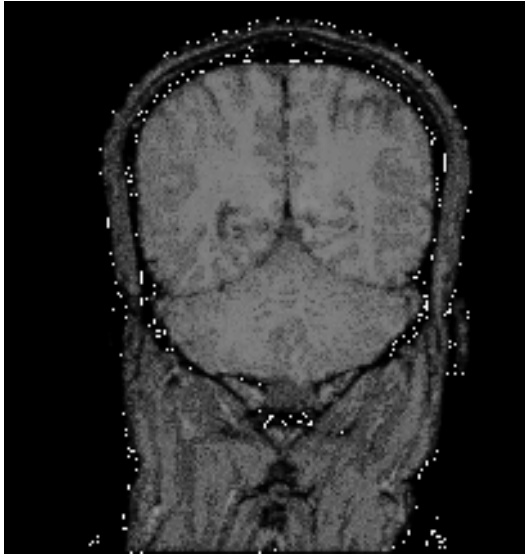
**Minimum intensity projection- axial**



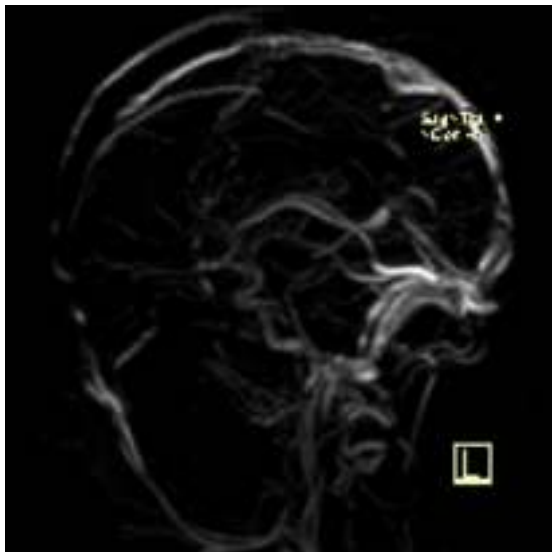
**SWI – axial**



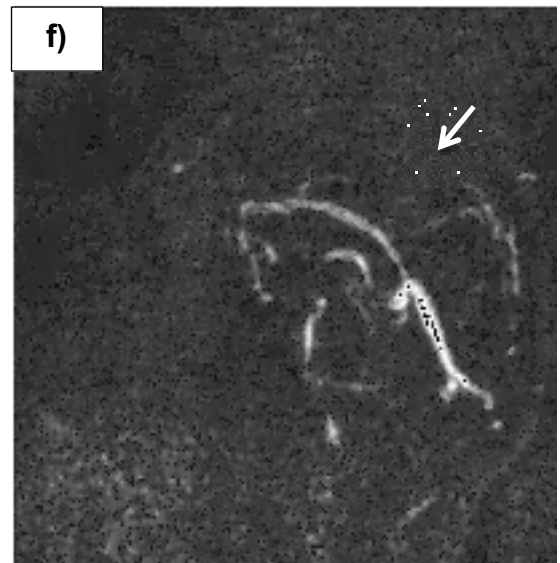
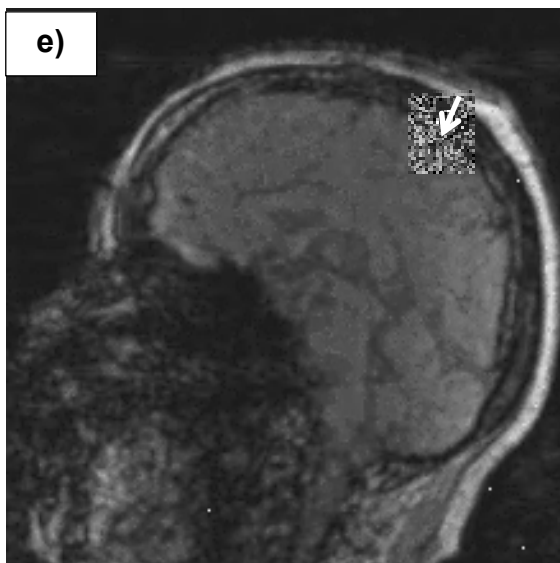
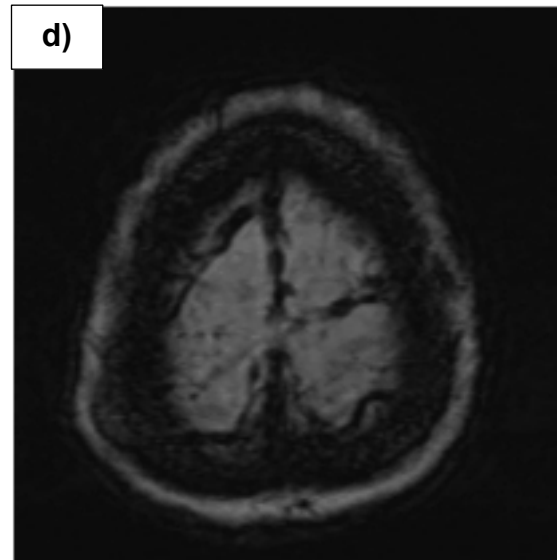
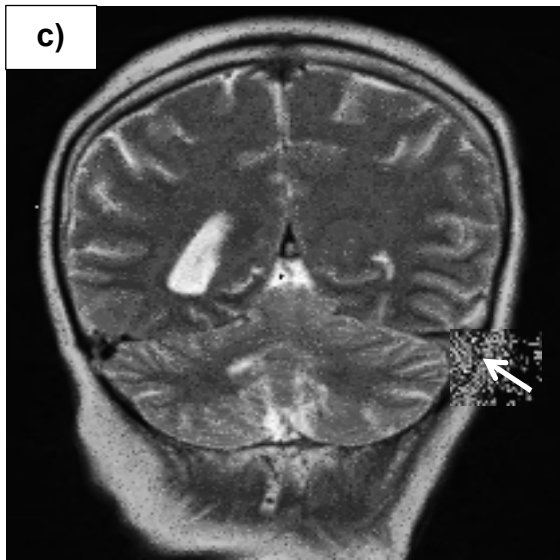
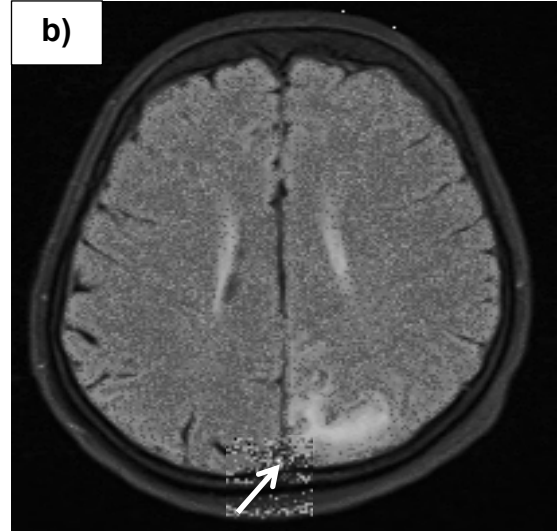
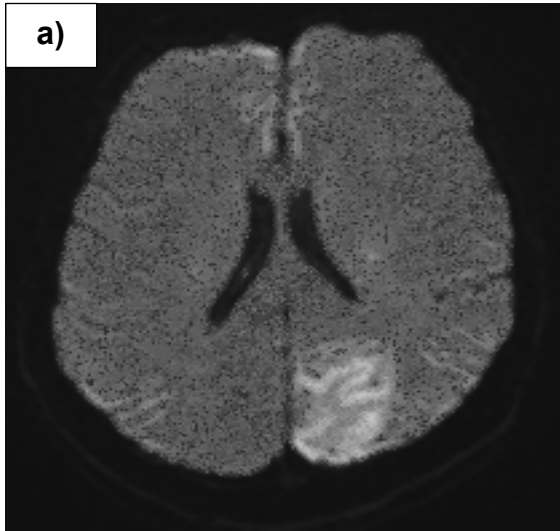
**SWI – sagittal**



**SWI – coronal**

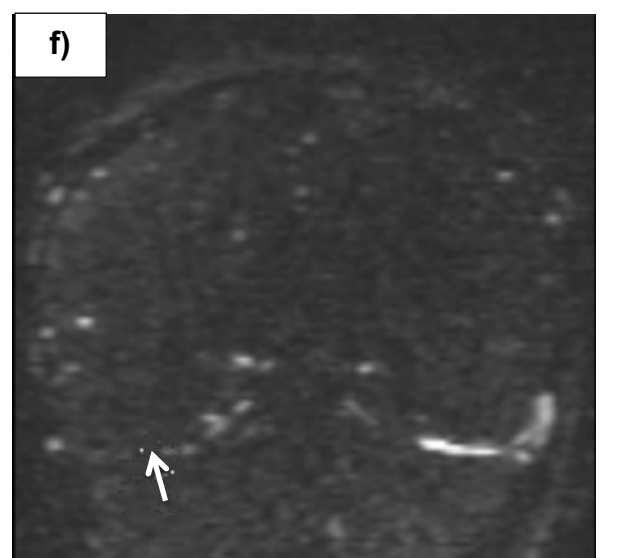
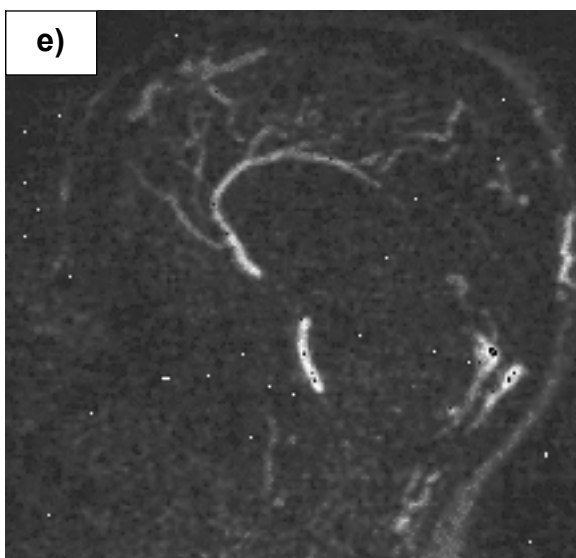
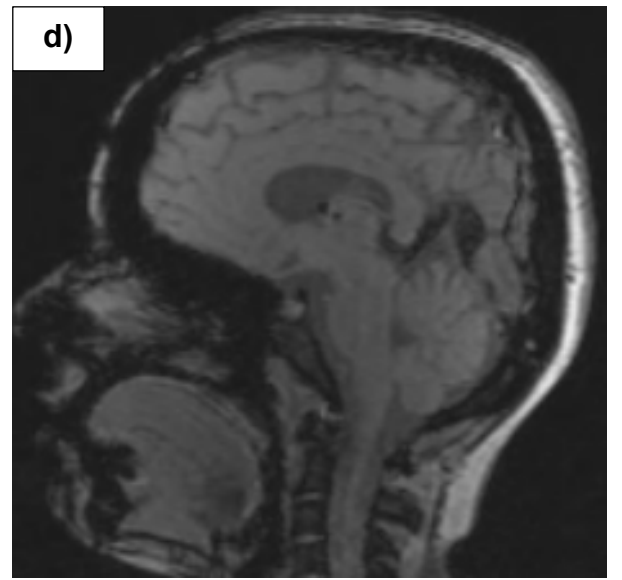
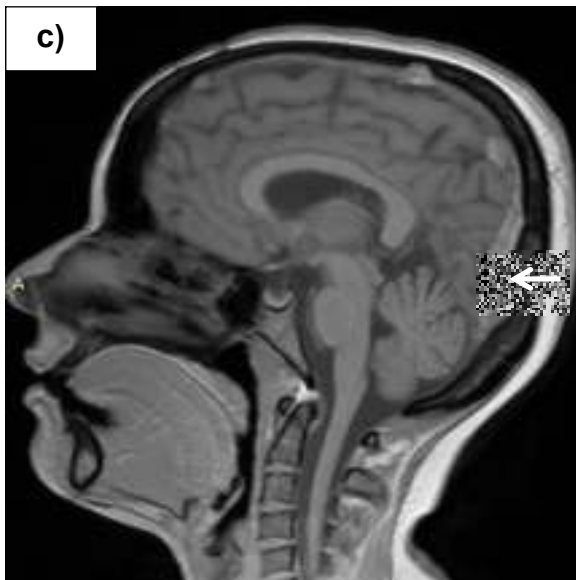
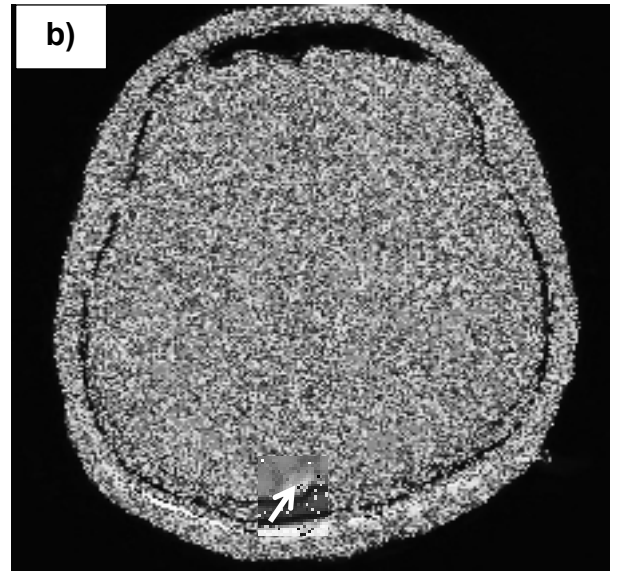
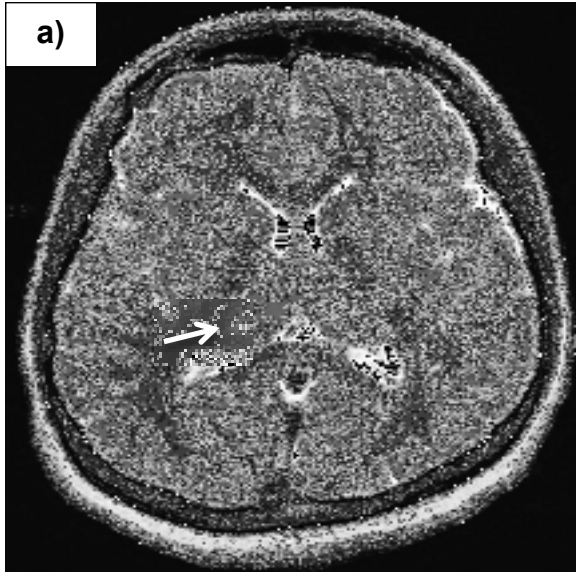


**3D – TOF MRV**



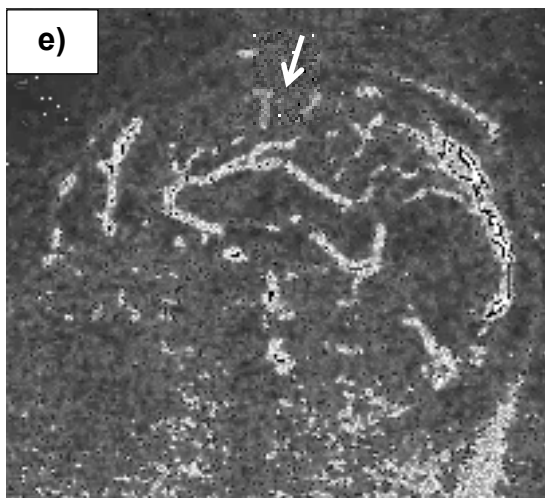
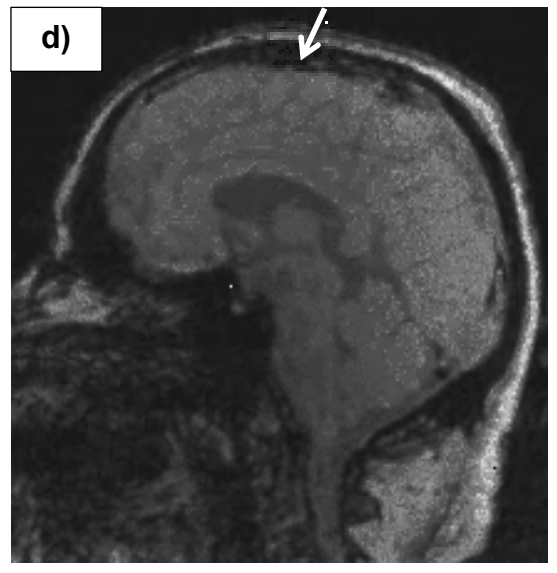
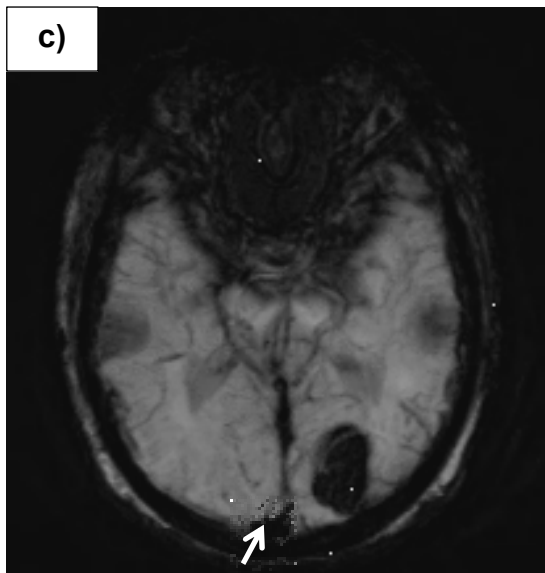
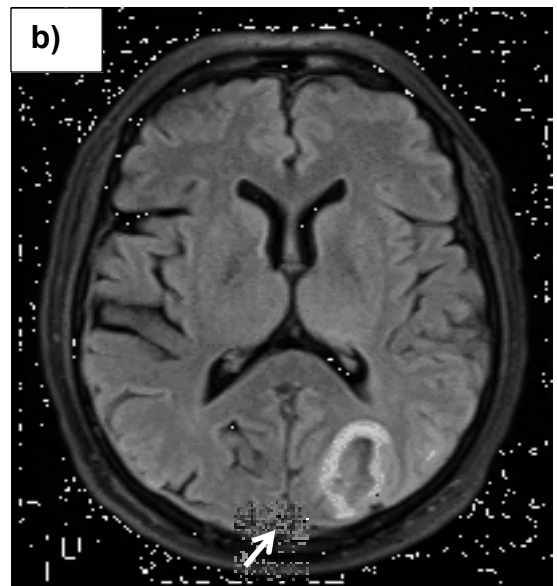
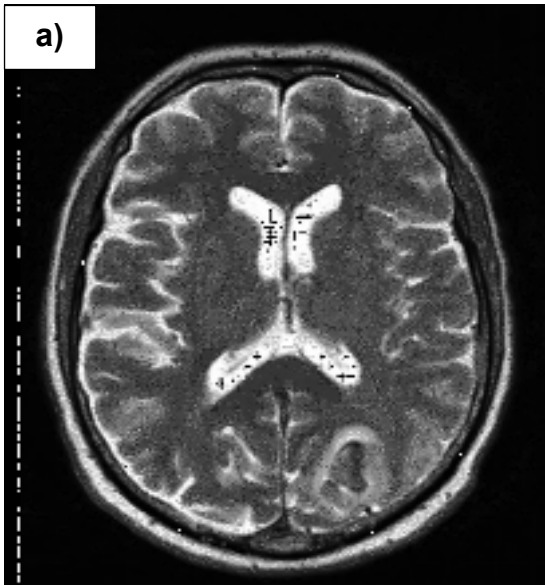
**21 years old male, complains of severe headache**

- a. Diffusion weighted MR image shows high signal in the left parietal region, corresponding ADC showed low signal (not shown), suggestive of restricted diffusion
- b. Axial FLAIR image shows hyperintensities involving the cortical and subcortical white matter of left parietal region suggestive venous infarct and (white arrow) shows iso to hyperintense signal within the lumen of superior sagittal sinus
- c. Coronal T2-W image shows iso to hyperintense signal involving the left transverse sinus (white arrow)
- d. Axial SWI image shows MSE in the cortical veins in bilateral high frontal region
- e. Sagittal SWI image shows MSE involving the superior sagittal sinus in its entire length (white arrow), suggestive of thrombosis
- f. Sagittal MR venogram image shows non visualization of superior sagittal sinus(white arrow) and left transverse sinus (not shown in the image), suggestive of thrombosis



**40 years female, complains of headache**

- a. Axial T2-W image shows hyperintensities involving the thalamus on right side (white arrow)
- b. Axial T1-W image shows hypointensities involving the thalamus on right side. T1 hyperintense signal noted involving the superior sagittal, suggestive of thrombosis (white arrow)
- c. Sagittal T1-W image shows T1 hyperintensities involving the superior sagittal sinus, suggestive thrombosis (white arrow)
- d. Sagittal SWI image shows MSE involving the superior sagittal sinus, right internal cerebral vein and vein of Galen, suggestive of thrombosis
- e. Sagittal MR venogram shows non visualization of superior sagittal sinus and right internal cerebral vein and vein of Galen, suggestive of thrombosis
- f. Coronal MR venogram shows non visualization of right transverse sinus, suggestive of thrombosis (white arrow)



**65 years old male, complains of headache and seizures**

- a. Axial T2-W image shows mixed intense area in the left parietal region, suggestive of hemorrhagic venous infarct
- b. Axial FLAIR image shows mixed intense area in the left parietal region, suggestive hemorrhagic venous infarct. Iso to hyperintense signal noted involving the superior sagittal sinus (white arrow), suggestive of thrombosis.
- c. Axial SWI image shows hypointense signal in the left parietal region, suggestive of hemorrhage. MSE noted involving the superior sagittal sinus, suggestive of thrombosis (white arrow)
- d. Sagittal SWI image shows MSE involving the anterior and mid portion of superior sagittal sinus, suggestive of thrombosis (white arrow)
- e. Sagittal MR venogram shows non visualization of superior sagittal sinus in its anterior & mid portion, suggestive of thrombosis (white arrow)

**ANNEXURE V - KEY TO MASTERCHART**

T1	T1 weighted
T2	T2 weighted
FLAIR	Fluid attenuated inversion recovery
ISO	Isointense
HO	Hypointense
HR	Hyperintense
SSS	Superior sagittal sinus
ISS	Inferior sagittal sinus
TS	Transverse sinus
IJV	Internal jugular vein
RT	Right
LT	Left
NV	Not visualized
H	Headache
S	Seizures
RH	Right hemiparesis
LH	Left hemiparesis
LOC	Loss of consciousness
V	Vertigo
MSE	Magnetic susceptibility effect
HG	High
FT	Frontal
PT	parietal
TP	Temporal
OT	Occipital
Ant	Anterior
Post	Posterior