Section: Radiology

Original Research Article

# MAGNETIC RESONANCE IMAGING IN CEREBRAL VENOUS THROMBOSIS

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## Abstract

**Background:** This study investigates the critical role of Magnetic Resonance Imaging (MRI), particularly advanced sequences like Diffusion-Weighted Imaging (DWI), in diagnosing and evaluating Cerebral Venous Thrombosis (CVT). Advanced sequences aid in tailoring treatment strategies by distinguishing between cytotoxic and vasogenic edema. The prevalence and characteristics of sinus thrombosis are examined, emphasizing the correlation between thrombus age, clinical presentation, and parenchymal imaging findings. **Materials and Methods:** A prospective observational study involving 50 CVT patients was conducted over 18 months, employing conventional MRI, DWI, and MR venogram. The study was carried out using a 1.5 T MRI unit, and data analysis was performed using statistical software. Continuous variables were represented as mean± SD and categorical variables were represented by frequency. Statistical significance was set at $p \leq 0.05$. **Result:** The superior sagittal sinus was the most frequently affected (70%), followed by the transverse (42%) and sigmoid sinuses (22%). The age of the sinus thrombus correlated with clinical presentation and parenchymal imaging in 90% of cases. Hemorrhagic infarct, intraparenchymal hematoma, and non-hemorrhagic infarct were primary parenchymal changes related to sinus thrombosis. Notable phase-specific trends were observed. **Conclusion:** MRI, especially DWI, plays a pivotal role in diagnosing and evaluating CVT. The study provides valuable insights into the application of MRI in CVT, contributing to improved patient management by offering a deeper understanding of thrombus characteristics and their clinical implications.

## INTRODUCTION

Cerebral venous thrombosis (CVT) is a condition that has gained increasing recognition in recent years due to its complex nature and diverse clinical presentations. Unlike arterial strokes, CVT is associated with a unique pathophysiology characterized by vasogenic and interstitial edema resulting from venous congestion.[1] Magnetic Resonance Imaging (MRI) has emerged as a crucial tool for the diagnosis and management of CVT, with advanced techniques like Diffusion-weighted Imaging (DWI) providing valuable insights into differentiating cytotoxic and vasogenic edema.[2]

Diagnosing CVT is often challenging both clinically and radiologically. Unlike arterial strokes, CVT can manifest with a wide range of symptoms, from subacute headaches and raised intracranial pressure to severe multifocal deficits, seizures, and even coma.[3] Accurate and timely diagnosis is crucial as effective treatments, including anticoagulation and intrasinus thrombolysis, are available. Furthermore, even when treatment is delayed, many CVT patients show remarkable recoveries, emphasizing the importance of accurate diagnosis beyond the hyperacute phase.[4]

CVT differs from arterial infarctions in several ways, particularly in its pathophysiological characteristics. Acute venous occlusion in CVT is believed to result in acute vasogenic edema due to increased capillary filtration pressure and fluid extravasation. It can also lead to cytotoxic edema due to inadequate perfusion pressure, resulting in a combination of both vasogenic and cytotoxic edema. Additionally, early hemorrhagic transformation can complicate MRI interpretation in CVT cases. DWI, which is highly sensitive to the diffusion of water protons, has proven valuable in differentiating venous from arterial infarctions and predicting tissue outcomes.[5,6]

DWI, a well-established MRI modality for diagnosing arterial strokes, is now being applied to CVT.[7] DWI is based on the random motion or diffusion of water molecules, providing apparent diffusion coefficients (ADC) to differentiate between cytotoxic and vasogenic edema. Vasogenic



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edema appears hypo-intense to slightly hyper-intense on DWI but is hyperintense on ADC maps. In contrast, cytotoxic edema appears hypointense on ADC maps and hyperintense on exponential images.[8,9]

A study by Chu et al identified three general patterns of MRI abnormality in CVT patients. The most common pattern is heterogeneous signal intensity on DWI with normal or increased ADC values, likely representing vasogenic and cytotoxic edema. DWI, coupled with ADC mapping, can discriminate between acute and chronic ischemia and vasogenic and cytotoxic edema, a significant advancement over conventional MRI techniques.[10]

**Table 1: Risk factors for cerebral venous thrombosis**

| Risk factors | Description |
|---|---|
| Infection | Para nasal sinusitis<br>Intracranial infection abscess<br>Meningitis |
| Trauma | Head trauma<br>Neurosurgical interventions<br>Internal jugular catheter |
| Medical/ Surgical conditions | Dehydration<br>Pregnancy and puerperium<br>Coagulation disorders: factor V Leiden (activated protein C resistance), protein C deficiency, protein S deficiency, antithrombin III deficiency, hyperhomocysteinemia, antiphospholipid syndrome<br>Hematologic disorders: polycythemia, sickle cell disease, thrombotic thrombocytopenic purpura, Polycythemia, Paroxysmal nocturnal hemoglobinuria<br>Malignancies, inflammatory bowel disease, Nephrotic syndrome, dehydration, liver cirrhosis, collagen vascular diseases, including systemic lupus erythematosus, Wegener granulomatosis, and Behcet syndrome<br>Previous surgical procedures |
| Medication | Oral contraceptives<br>Hormone replacement therapy<br>L-asparaginase<br>Aminocaproic acid, corticosteroids |

Tissue damage and stasis (trauma, surgery, and immobilization), hematologic disorders (protein C, S deficiencies, increased resistance to activated protein C), malignancies, collagen vascular disease (systemic lupus erythematosus, Behcet syndrome), pregnancy, and some medications (oral contraceptives, hormone replacement therapy, and corticosteroids) have been reported to be predisposing factors for CVT [Table 1]. Despite multiple known risk factors, approximately 20% of patients with CVT do not have any known risk factors.

The clinical presentation of CVT is variable but often includes headaches and seizures [Table 2]. In addition, the clinical presentation of CVT is closely related to the location and the extent of the thrombosis (cortical versus dural sinus, superficial versus deep). The most frequently occurring symptoms and signs of CVT are headaches, vomiting, and papilledema, reflecting increased cerebral venous pressure. Although the symptoms are nonspecific, papilledema should prompt neuroimaging and CVT should be considered in the differential diagnosis. Patients may go on to develop seizures, decreased level of consciousness, or focal neurologic deficit. This progression of clinical signs and symptoms justifies neuroimaging on an emT as a diergency basis, and CVagnosis that must be ruled out.[16]

The prognosis of CVT has traditionally been considered poor. Two prospective reports found that 41% of patients had a poor outcome, defined as death or a Barthel index score of less than 15%.[17,18] Recent reports, however, have shown mortality rates to be less than 10%.[19] CVT in pregnancy and the puerperium has a more acute onset and a better prognosis (9% versus 33%) compared with thrombosis from other causes.[10]

Imaging plays a key role in the diagnosis of CVT. Non-contrast enhanced CT (NECT) and structural MRI provide essential information. The "delta" sign is a characteristic feature on CT indicating thrombosed sinuses or veins. MRI, especially susceptibility-weighted imaging (SWI), is useful for assessing venous congestion and hemorrhage. The distribution of parenchymal lesions depends on the thrombosed vessels, and their pattern can provide insights into the affected venous territories.

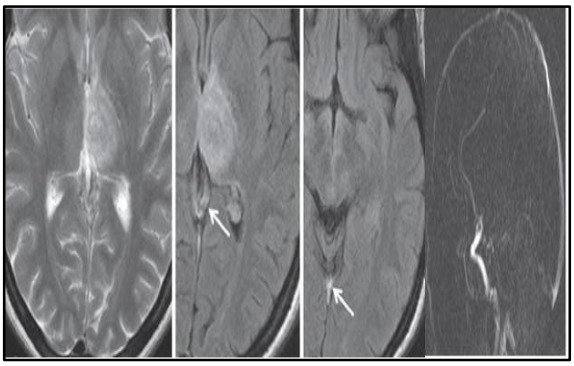

**Figure 7: Deep venous thrombosis. This young female patient presented with headache and right-sided hemiparesis whilst playing in a prolonged hockey competition in the summer, followed by reduced consciousness. The initial MRI examination (A, C) demonstrates signal abnormality and swelling consistent with venous hypertension, edema, and ischemia in the left thalamus. There is an abnormal signal within and expansion of the left internal cerebral vein and straight sinus on FLAIR (B, C, white**



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**arrows) in keeping with the thrombus. The MR venogram does not show any flow in the deep venous system (D).**

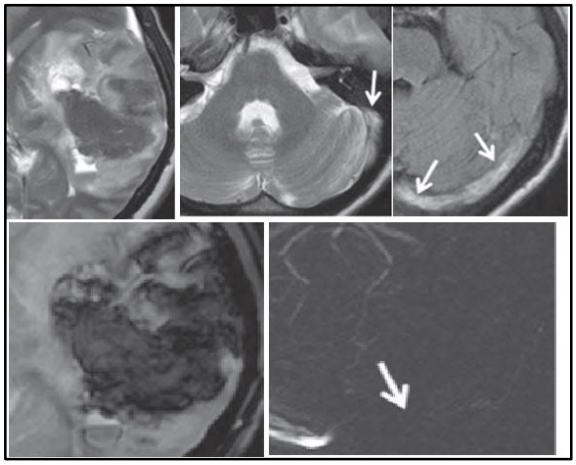


**Figure 8: Cerebral venous thrombosis. This young male patient presented with headache and seizures—in this case, there is a small area of early venous ischemia in the anterolateral aspect of the right temporal lobe on T2-weighted MRI (C, white arrow). Lateral venous thrombosis. (A-E) A large acute parenchymal hemorrhage in the left frontal and temporal lobes was discovered in this patient who presented with seizures and encephalopathy. Hypointensity on the T2-weighted MRI (A) and SWI (D) are consistent with acute blood products. There is a considerable degree of surrounding parenchymal edema shown as high signal intensity on T2W (A). A causative left lateral sinus thrombosis is identified on T2W (B, white arrow) and FLAIR (C, white arrows) with loss of the normal related signal void and confirmed by the absence of flow-related signal in this sinus on MR venography (E, white arrow).**

The study aims to investigate cerebral venous thrombosis using both conventional and advanced MR imaging techniques. The objectives are to analyze different types of brain tissue involvement, determine the prevalence of various symptoms, and establish a connection between cerebral tissue changes and the location of thrombosis.

## MATERIALS AND METHODS

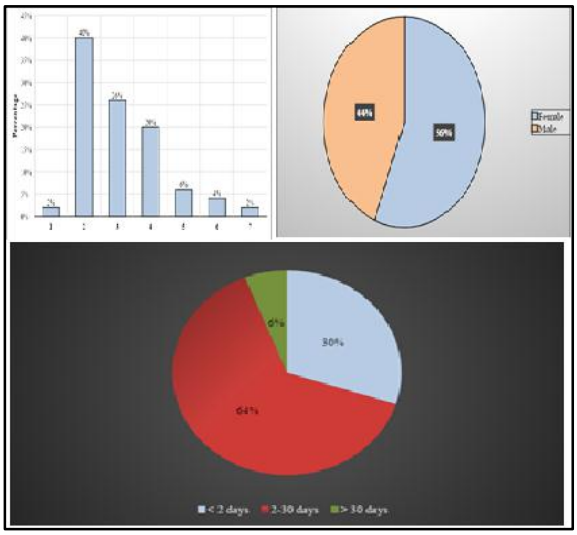
This is a prospective observational study of 50 patients with cerebral venous thrombosis (CVT). Patients had undergone conventional MRI, diffusion-weighted imaging, and MR venogram. The diagnosis of CVT was confirmed with MR venogram and other conventional MR sequences in 48 patients. MR contrast venography was done in 2 patients.

The study was conducted in the Department of Radio Diagnosis, Krishna Institute of Medical Sciences and Hospital, Karad over 18 months from October 2019 to March 2021. The patients were examined with a 1.5 T MRI unit Siemens Magnetom Avanto Machine using surface/body coils. Diffusion-weighted images with echo-planar imaging were obtained using two b values. MR Venogram was done using the TOF (time of flight) technique in oblique sagittal and coronal planes.

Data is analyzed using statistical software R version 4.1.1 and Microsoft Excel. Continuous variables were represented by mean± SD and categorical variables were represented by frequency. Shapiro-Wilk's test is used to check the normality of variables. Two sample t-tests were used to compare the mean between the groups. The chi-square test will be used to check the association between two categorical variables. A P-value less than or equal to 0.05 indicates statistical significance.

## DISCUSSION

Cerebral venous thrombosis (CVT) is more common than previously believed, affecting the deep or cortical venous system and leading to venous stroke. It presents with various symptoms, sometimes resembling arterial strokes or a mass lesion. Radiological examinations play a crucial role in diagnosis and prognosis, with MRI and MR Venography being valuable tools. Conventional MR imaging shows similar signal intensities for areas of venous congestion and infarction, making it challenging to differentiate between cytotoxic and vasogenic edema. Diffusion-weighted imaging provides ADCs that can distinguish the type of cerebral edema. However, further analysis is needed for dural sinus thrombosis progression. A study involving 50 subjects (age range: 19-71 years, mean age: 36.72±12.41 years) showed a slight female preponderance (56% females, 44% males). The highest number of patients were in the 21-30 age bracket (40%), followed by 31-40 years (26%) and 41-50 years (20%).



**Graph 1: (a) Distribution of subjects by age. (b) Distribution of subjects by gender (c) Distribution of subjects by time to MRI.**

We divided patients according to the time of onset of symptoms. Patients who presented within 2 days were classified as acute, within 2 to 30 days were



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classified as subacute, and more than 30 days as chronic. The most common presentation was found to be subacute, which was 64% (32 patients), whereas acute presentation was seen in 30 % (15 patients) and chronic in 6% (3 patients). The mean time of MR imaging from the onset of symptoms was ~ 7.46 +/- 7.65 days (Graph 3).

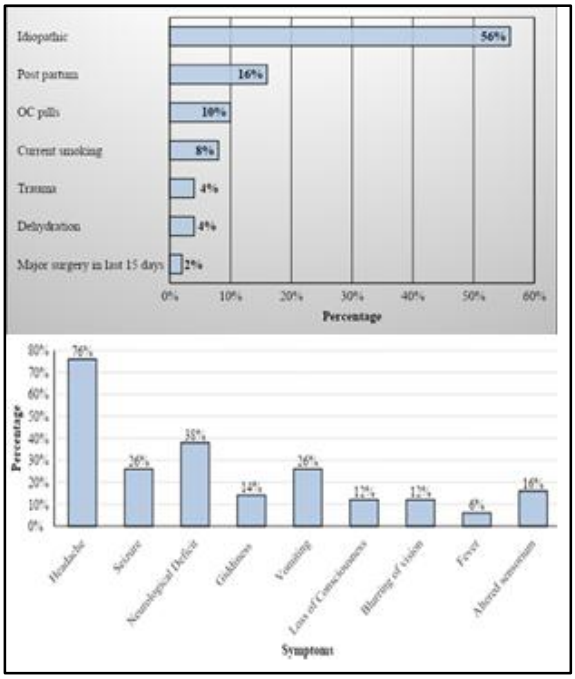
**Table 1: Summary of variables in the study.**

| Variables | | Number of subjects (%) |
|---|---|---|
| Age (in years) | ≤ 20 | 1 (2%) |
| | 21-30 | 20 (40%) |
| | 31-40 | 13 (26%) |
| | 41-50 | 10 (20%) |
| | 51-60 | 3 (6%) |
| | 61-70 | 2 (4%) |
| | ≥ 71 | 1 (2%) |
| Age (in years) | 36.72±12.41 | 33 (19, 71) |
| Gender | Female | 28 (56%) |
| | Male | 22 (44%) |
| Presentation | Acute | 15 (30%) |
| | Chronic | 3 (6%) |
| | Subacute | 32 (64%) |
| Time to MRI imaging in days | < 2 days | 15 (30%) |
| | 2-30 days | 32 (64%) |
| | > 30 days | 3 (6%) |
| Time to MRI imaging in days | 7.46±7.65 | 6 (1, 35) |
| Past history of risk factors | Idiopathic | 28 (56%) |
| | Current smoking | 4 (8%) |
| | Dehydration | 2 (4%) |
| | Major surgery in last 15 days | 1 (2%) |
| | OC pills | 5 (10%) |
| | Post-partum | 8 (16%) |
| | Trauma | 2 (4%) |

Various risk factors are seen associated with cerebral venous thrombosis.. Eight cases (16%) of peripartum thrombosis were found in our study. Hypercoagulable state (anti-cardiolipin) associated with puerperium may be one of the major factors.

In this study, 5 patients (10%) were using OCP from 6 months to 14 months duration. OCP has a prothrombotic effect and this is proved in the laboratory by Vandenbroucke et al,[22] other control studies done by Martinelli et al. revealed an increased risk of CVT in women of younger age group who were using OCP.[23] In our study, 4 patients (8%) had given current smoking history.

In this study, 2 cases of trauma were found [4%]. Miller et al studied 400 cases of depressed skull fractures and concluded that cerebral venous sinus involvement was seen in 11% of cases.[24] They depicted that the sinus lumen may get damaged or obliterated with disruption of normal flow increasing intracranial pressure and along with infection may produce CVST.




**Graph 2: (a) Distribution of subjects by past history (b) Distribution of subjects by symptoms.**

In CVST, large numbers of symptoms are associated with variable presentations. Most of these may be nonspecific like headaches, seizures, focal neurological deficit, blurring of vision, nausea, vomiting, and even decreased level of consciousness. Depending upon the availability of collateral venous pathways, it can result in significant brain involvement or may be well tolerated.[25] In this study, the headache was the most common symptom in 38/50 patients (76%), followed by the focal neurological deficit in 19/50 (38%), seizures and vomiting in 13/50 patients (26% of each) This is similar to previous study of D. Karthikeyen et al. who stated that headache is the most presenting and non-specific symptom seen in 70-90% of cases.[26]. Focal neurological deficits (hemiparesis, hemi sensory disturbance), seizures, impairment of level of consciousness, and papilledema occur in one-third to three-quarters of cases.

. The superior sagittal sinus, followed by transverse, sigmoid, and straight sinuses, were commonly affected, aligning with previous research findings. Additionally, single sinus involvement was seen in 48% of subjects, with the most frequent combination being the superior sagittal sinus and transverse sinus.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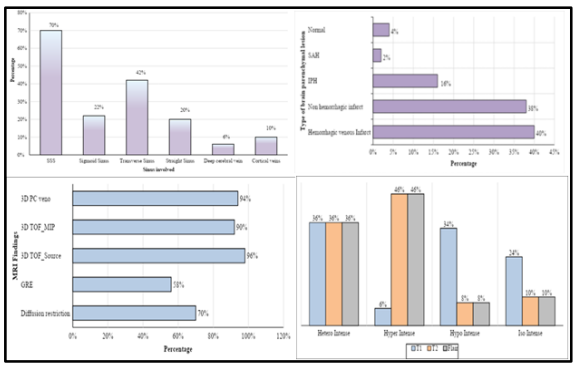

**Graph 3: (a) Distribution of subjects by sinus involved. (b) Distribution of subjects by brain parenchymal lesion (c) Distribution of subjects by MRI findings (d) Distribution of subjects by T1, T2, Flair**

Tsai et al. described the MRI findings in cerebral venous sinus thrombosis (CVST), noting that initially, there's a mild increase in dural venous sinus pressure, primarily affecting sinuses without parenchymal abnormalities. In this study, 4% of patients showed no parenchymal involvement. As intracranial pressure rises, it leads to focal neurological deficits and the affected brain region may exhibit edema or infarction, with or without hemorrhage, in 10%-50% of cases. The most common parenchymal finding was hemorrhagic venous infarct, observed in 40% of patients, in line with findings from a study in India.

**Table 2: Distribution of subjects by age and gender**

| | | Presentation | | | p-value |
|---|---|---|---|---|---|
| | | Acute (n=15) | Chronic (n=3) | Subacute (n=32) | |
| Age (in years) | ≤ 20 | 0 (0%) | 0 (0%) | 1 (3.13%) | 0.4683MC |
| | 21-30 | 9 (60%) | 0 (0%) | 11 (34.38%) | |
| | 31-40 | 2 (13.33%) | 1 (33.33%) | 10 (31.25%) | |
| | 41-50 | 3 (20%) | 2 (66.67%) | 5 (15.63%) | |
| | 51-60 | 0 (0%) | 0 (0%) | 3 (9.38%) | |
| | 61-70 | 1 (6.67%) | 0 (0%) | 1 (3.13%) | |
| | ≥ 71 | 0 (0%) | 0 (0%) | 1 (3.13%) | |
| Age (in years) | | 35.2±12.71 | 43.33±4.93 | 36.81±12.79 | - |
| Gender | Female | 6 (40%) | 2 (66.67%) | 20 (62.5%) | 0.4053MC |
| | Male | 9 (60%) | 1 (33.33%) | 12 (37.5%) | |
| Days of presentation | | 1.53±0.52 | 32±2.65 | 7.94±4.07 | - |
| T1 | Hetero Intense | 1 (6.67%) | 0 (0%) | 17 (53.13%) | 0.01499*MC |
| | Hyper Intense | 0 (0%) | 0 (0%) | 3 (9.38%) | |
| | Hypo Intense | 6 (40%) | 2 (66.67%) | 9 (28.13%) | |
| | Iso Intense | 8 (53.33%) | 1 (33.33%) | 3 (9.38%) | |
| T2 | Hetero Intense | 1 (6.67%) | 0 (0%) | 17 (53.13%) | 0.01499*MC |
| | Hyper Intense | 9 (60%) | 1 (33.33%) | 13 (40.63%) | |
| | Hypo Intense | 3 (20%) | 1 (33.33%) | 0 (0%) | |
| | Iso Intense | 2 (13.33%) | 1 (33.33%) | 2 (6.25%) | |
| Flair | Hetero Intense | 1 (6.67%) | (0%) | 17 (53.13%) | 0.01499*MC |
| | Hyper Intense | 9 (60%) | 1 (33.33%) | 13 (40.63%) | |
| | Hypo Intense | 3 (20%) | 1 (33.33%) | 0 (0%) | |
| | Iso Intense | 2 (13.33%) | 1 (33.33%) | 2 (6.25%) | |
| Hemorrhagic venous Infarct | Negative | 13 (86.67%) | 0 (0%) | 14 (43.75%) | 0.007496*MC |
| | Positive | 2 (13.33%) | 0 (0%) | 18 (56.25%) | |
| Non hemorrhagic infarct | Negative | 8 (53.33%) | 3 (100%) | 20 (62.5%) | 0.3348MC |
| | Positive | 7 (46.67%) | 0 (0%) | 12 (37.5%) | |
| IPH | Negative | 10 (66.67%) | 2 (66.67%) | 30 (93.75%) | 0.04698*MC |
| | Positive | 5 (33.33%) | 1 (33.33%) | 2 (6.25%) | |
| SAH | Negative | 15 (100%) | 3 (100%) | 31 (96.88%) | 1MC |
| | Positive | 0 (0%) | 0 (0%) | 1 (3.13%) | |
| Normal | Negative | 15 (100%) | 1 (33.33%) | 32 (100%) | 0.0004998*MC |
| | Positive | 0 (0%) | 2 (66.67%) | 0 (0%) | |

By the Chi-square test, there is no significant difference in the distribution of age between the genders, and by

By the Chi-square test, there is no significant association present between age, gender, hemorrhagic infarct, and SAH with the presentation. However, there is a significant association present between all other variables.

Our study showed that the age of the clot was the same as the clinical presentation at the time of imaging in 90% of cases. In the rest 10% of cases, it was seen that the conventional sequences (T1W, T2W, and FLAIR) had the clot signal representing that of the subacute phase, whereas the patient presented in the acute phase.



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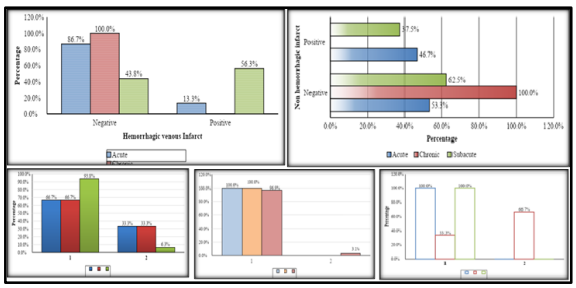

**Figure 14: Distribution of subjects by Hemorrhagic venous infarct and presentation. (b) Distribution of subjects by Non-Hemorrhagic infract and presentation. (c) Distribution of subjects by IPH infract and presentation. (d) Distribution of subjects by SAH and presentation. (e) Distribution of subjects by Normal lesions and presentation.**

In the acute phase, the study found that most patients had non-hemorrhagic infarcts, followed by intraparenchymal hemorrhages in 5 patients.[7] In the subacute phase, there were 18 cases of hemorrhagic venous infarcts, 12 cases of non-hemorrhagic infarcts, and only 2 cases of intraparenchymal hemorrhages.

The sinus thrombus clot age was found to be in sync with clinical presentation and parenchymal imaging findings in 90 % of cases, whereas in 10% of cases, the sinus thrombus age was older than the parenchymal findings. The parenchymal changes related to sinus thrombus were recognized as hemorrhagic infarct in 40% of patients, intraparenchymal hematoma in 16% of patients, and non-hemorrhagic infarct in 38% of patients.

The presence of hemorrhagic infarct in the subacute phase and intraparenchymal hematoma in the acute were observed to have a significant correlation (with p-values less than 0.05).

**Limitation of Study**

- The sample size was small so the observations would not always hold true when projected for larger populations.
- The demographic distribution of our study population was predominantly rural which caused non-conformity of findings such as only a slight preponderance of female patients, the prevalence of the most common age group being 3rd decade, and the inability to narrow down the etiology of the sinus thrombus in the majority of the patients even on extensive history elicitation.
- No pediatric patient was included in our study.
- ADC values were not calculated in the majority of patients.
- Post-contrast MR venogram was not performed in the majority of patients due to financial and time constraints.
- The follow-up data could not be collected for many patients as they were lost in follow-up.

## CONCLUSION

MRI, especially DWI, plays a pivotal role in diagnosing and evaluating CVT.

The sinus thrombus clot age, associated parenchymal changes and to differentiate between cytotoxic and vasogenic edema MRI studies found extremely helpful.

The study provides valuable insights into classifying the phases of the disease process by the application of MRI and MR venogram in CVT improving the patient management.

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