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A Cross-Sectional Study Using MR Imaging To Evaluate Cerebral Venous Thrombosis

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Keywords
CVT, MRI, DWI,intracranial venous system,T2, FLAIR

Abstract
Background:Dural sinus thrombosis, along with the thrombosis of the deep or cortical cerebral venous system and the venous stroke resulting from it, is more common than once thought. Cerebral venous thrombosis (CVT) is a cause of stroke with diverse etiologies and varied clinical presentations. Its manifestations may simulate an acute arterial stroke or a mass lesion. The pathophysiology of CVT with associated venous strokes appears to differ from arterial strokes. Acute arterial strokes show cytotoxic edema, whereas venous strokes are thought to contain vasogenic and interstitial edoema due to venous congestion. MR imaging has been deemed to play a crucial role in diagnosing and evaluating this complex disease and, hence, has been progressively used to guide and direct its management.

Purpose:To study the various aspects of cerebral venous thrombosis in conventional and advanced MR imaging sequences

Methodology: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 an MR venogram and other conventional MR sequences in 48 patients. MR contrast venography was done in 2 patients.

Result:In our study, headache was the most common symptom in 38/50 patients (76%), followed by a focal neurological deficit in 19/50 (38%), seizures, and vomiting in 13/50 patients (26% of each). In our study, the most common site of sinus thrombosis was found to be the superior sagittal sinus (70%), followed by the transverse and sigmoid sinuses (42% and 22%, respectively). The involvement of the deep venous sinus was only present in three (6%) patients. 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. Hemorrhagic infarct was identified in 40% of sinus thrombosis patients, intraparenchymal hematoma in 16% of patients, and non-hemorrhagic infarct in 38% of patients. In the acute phase, it was seen that in our study, most patients (7 patients) presented with a non-hemorrhagic infarct, whereas in the subacute phase, 18 cases were of hemorrhagic venous infarct. There was a significant correlation (with p-values less than 0.05) between the presence of hemorrhagic infarction in the subacute phase and intraparenchymal hematoma in the acute phase.

Conclusion:Evaluation of CVT is often a difficult task, but after the introduction of newer MR imaging techniques, it is possible to predict if brain lesions, detected clinically and using conventional MR imaging methods, may lead to full recovery, as expected in arterial infarcts and even a hematoma. Our research concludes that MRI is the best tool to evaluate CVT.

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1. Introduction

The diagnosis of cerebral venous thrombosis is often difficult, both clinically and radiologically. There is currently no way to predict whether brain lesions detected clinically and using conventional MR imaging methods will result in full recovery, as expected in arterial infarcts and even hematomas[1].

Accurate diagnosis is difficult but important because effective therapies, including anticoagulation and possibly intrasinus thrombolysis, are available. Patients with CVT often make dramatic recoveries after anticoagulation, even when treatment is delayed. As a result, accurate diagnosis is critical even after the hyperacute period has passed [2].

Dural sinus thrombosis, along with the thrombosis of the deep or cortical cerebral venous system and the venous stroke resulting from it, are more common than once thought [(3)]. Cerebral venous thrombosis (CVT) is a cause of stroke with diverse etiologies and varied clinical presentations. Its manifestations may simulate an acute arterial stroke or a mass lesion [(3, 4)]. The pathophysiology of CVT with associated venous strokes appears to differ from arterial strokes. Acute arterial strokes show cytotoxic edema, whereas venous strokes are thought to contain vasogenic and interstitial edoema due to venous congestion.

MR imaging has been deemed to play a crucial role in diagnosing and evaluating this complex disease and, hence, has been progressively used to guide and direct its management.

It has also been suggested that advanced sequences such as DWI are better at evaluating cases of CVT than conventional ones, due to their increased ability to differentiate between cytotoxic and vasogenic edema, thus enabling the treating physician to direct the treatment accordingly [(5, 1)].

This study looks at the different facets of the application of the MRI technique to cerebral venous thrombosis.

Aim and objectives

Aim:

To study the various aspects of cerebral venous thrombosis in conventional and advanced MR imaging sequences.

Objectives: To study the various types of parenchymal involvement in patients with cerebral venous thrombosis. To find the prevalence of different symptoms of cerebral venous thrombosis in our subgroup. To find out the correlation of cerebral parenchymal changes with the site of involvement of venous thrombosis.

2. 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 an MR venogramand 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 andHospital, Karad over a period of 18 months from October 2019 to March 2021.

The patients were examined with a 1.5 T MRI unit Siemens MagnetomAvanto Machine using surface/body coils. Diffusion-weighted images with echo-planar imaging were obtained using two b values.

MR Venogram was done using the 2D Phase contrast technique in oblique sagittal and coronal planes.

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1.5 T MRI unit Siemens MagnetomAvanto (Tim + Dot)System - Technical parameters of all the sequences are as follows:

MRI Sequences	TR (Ms)	TE (Ms)	TI (Ms)	Flip Angle (Degree)
T1W	400	10	-	90
T2W	4240	100	-	150
FLAIR	8640	92	2456	150
DWI	3400	101	-	90
GRE	700	20	-	20
SWI	49	40	-	15
2D-Phase Contrast Venogram	74	10	-	15
Post contrast Venogram (2 D-TOF)	25	07	-	60

Data analysis

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-testswere 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.

3. Results

The study includes 50 people with an average age of 36.72 ±12.41 years.There were 22 males and 28 females in the study. The 50 subjects were chosen between the ages of 19 and 71, with a mean age of 36.72 ±12.41 years.A slight female preponderance was found: females (28, 56%), and males (22, 44%). The maximum number of patients belonged to the age bracket of 21–30 years (40%). The second and third most commonly affected age groups were 31–40 years (26%) and 41–50 years (20%), respectively.

Among male patients, the most common age of presentation was 41–50 years, and that among female patients was 21–30 years. However, by the Chi-square test, there is no significant difference in the distribution of age between the genders, and by the t-test, there is no significant difference in the mean of age between genders.

Out of 50 subjects, 15 subjects had acute and only 3 had a chronic presentation. In 56% of the subjects, idiopathic disease was found. The plots below depict the same information as shown in graph 1.

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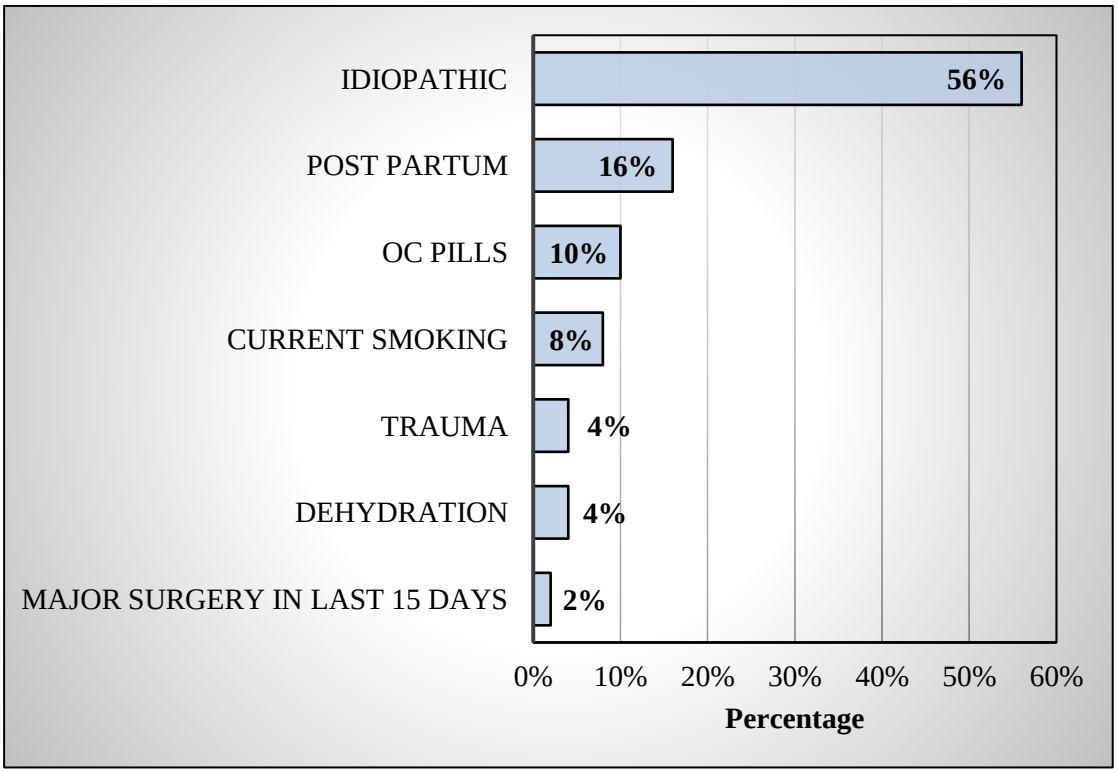

Graph 1: Distribution of subjects by history.

The subjects involved in the study present common symptoms including headache, Seizure, Neurological Deficit, Giddiness, Vomiting, Loss of Consciousness, Blurring of vision, Fever, and Altered sensorium. The below table gives the distribution of symptoms observed in the study.

Table 1: Distribution of symptoms observed in the study.

Symptoms	Positive
Headache	38 (76%)
Seizure	13 (26%)
Neurological Deficit	19 (38%)
Giddiness	7 (14%)
Vomiting	13 (26%)
Loss of Consciousness	6 (12%)
Blurring of vision	6 (12%)
Fever	3 (6%)
Altered sensorium	8 (16%)

The distribution of sinuses involved in the study is shown in table 2.

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Table 2: Distribution of Sinuses involved in the study**.**

Sinus Involved	Positive
SSS	35 (70%)
Sigmoid sinus	11 (22%)
Transverse Sinus	21 (42%)
Straight Sinus	10 (20%)
Deep cerebral vein	3 (6%)
Cortical veins	5 (10%)

During our study Single sinus was observed in 24 (48%) subjects and all others had more than one sinus. The most common combination of sinus observed was SSS and transverse sinus (16%) as shown in table 3.

Table 3: Distribution of subjects according to a combination of different Sinuses involved.

Sinus Involved	Number of subjects(%)
SSS	13 (26%)
Sigmoid Sinus	3 (6%)
Transverse Sinus	8 (16%)
SSS, Sigmoid Sinus	2 (4%)
Straight Sinus, Deep cerebral vein	2 (4%)
SSS, Cortical veins	2 (4%)
SSS, Transverse Sinus	4 (8%)
SSS, Straight Sinus	2 (4%)
SSS, Deep cerebral vein	3 (6%)
Transverse Sinus, Straight Sinus	1 (2%)
Transverse Sinus, Straight Sinus, Deep cerebral vein	1 (2%)
SSS, Sigmoid Sinus, Deep cerebral vein	1 (2%)
SSS, Transverse Sinus, Cortical veins	1 (2%)
SSS, Sigmoid Sinus, Cortical veins	1 (2%)
SSS, Straight Sinus, Deep cerebral vein	1 (2%)

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SSS, Sigmoid Sinus, Straight Sinus	1 (2%)
SSS, Transverse Sinus, Deep cerebral vein	1 (2%)
SSS, Straight Sinus, Deep cerebral vein, Cortical veins	1 (2%)
SSS, Sigmoid Sinus, Transverse Sinus, Straight Sinus	2 (4%)

The below tables give the output of MRI diagnosis.

Table 4. Distribution of MRI outputs.

	Hetero Intense	**Hyper Intense**	**Hypo Intense**	**Iso-Intense**
T1	18 (36%)	3 (6%)	17 (34%)	12 (24%)
T2	18 (36%)	23 (46%)	4 (8%)	5 (10%)
FLAIR	18 (36%)	23 (46%)	4 (8%)	5 (10%)

Table 5. Distribution of MRI outputs.

MRI findings	**Positive**
Diffusion restriction	35 (70%)
GRE	29 (58%)
2D PC Source	48 (96%)
2D PC MIP	45 (90%)

Below table and plots give the distribution of parenchymallesions observed in the study.

Table6: Distribution of subjects by brain parenchymal lesion.

Type of brain parenchymal lesion	**Positive**
Hemorrhagic venous Infarct	20 (40%)
Non-hemorrhagic infarct	19 (38%)
IPH	8 (16%)
SAH	1 (2%)

Hemorrhagic venous Infarct was observed in 40% of the subjects and Non-Hemorrhagic Venous Infarct was observed in 38% of the subjects.

The below table gives the distribution of age over gender.

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Table7: Distribution of subjects by age and gender

		Gender		p-value
		Male	Female	
Age (in years)	≤ 20	0 (0%)	1 (3.57%)	0.06347[MC]
	21-30	5 (22.73%)	15 (53.57%)	
	31-40	6 (27.27%)	7 (25%)	
	41-50	8 (36.36%)	2 (7.14%)	
	51-60	2 (9.09%)	1 (3.57%)	
	61-70	1 (4.55%)	1 (3.57%)	
	≥ 71	0 (0%)	1 (3.57%)	
Age (in years)		40.27±10.66	33.93±13.14	0.07239[t]

Abbreviations: MC: Monte-Carlo's simulation used in Chi-square test, t: two-sample t-test.

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

The below table compares the different variables with the presentation.

Table 8: Comparison of parameters over presentation.

		Presentation			p-value
		Acute (n=15)	Chronic (n=3)	Subacute (n=32)	
Age (in years)	≤ 20	0 (0%)	0 (0%)	1 (3.13%)	0.4683[MC]
	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%)	0.4053[MC]

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	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%)	0.3348[MC]
	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%)	1[MC]
	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%)	

Abbreviations: MC: Monte-Carlo's simulation used in the Chi-square test

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

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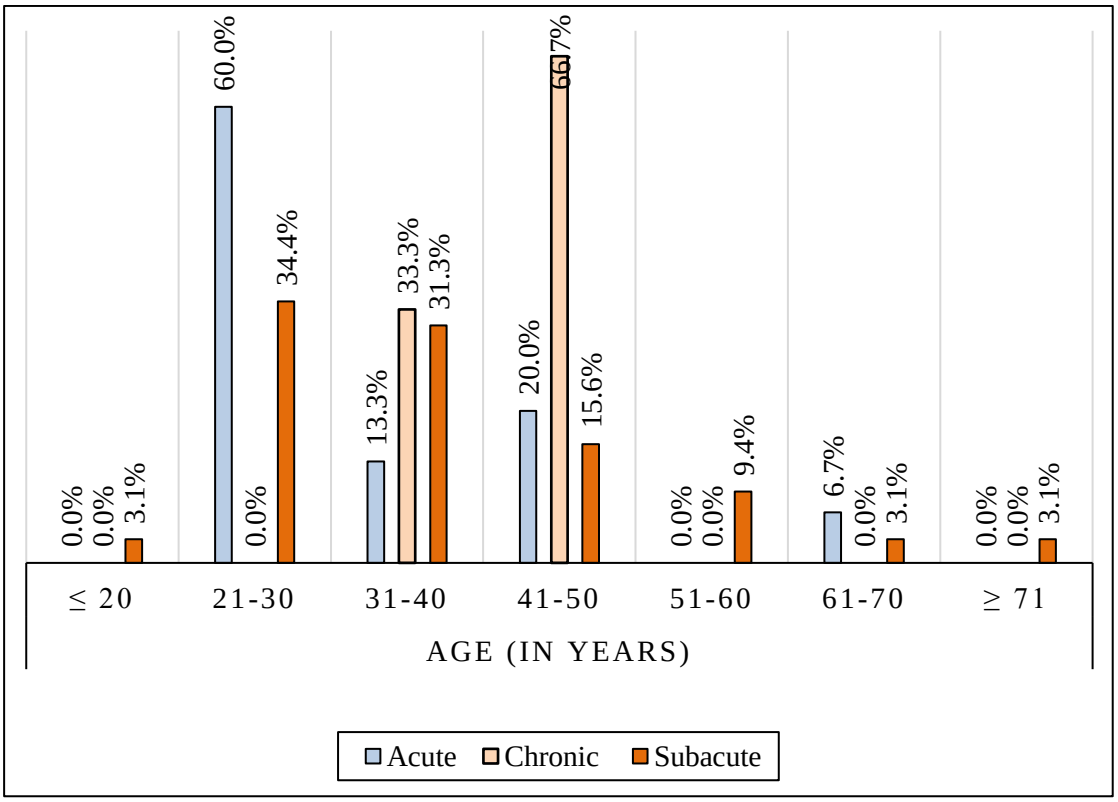
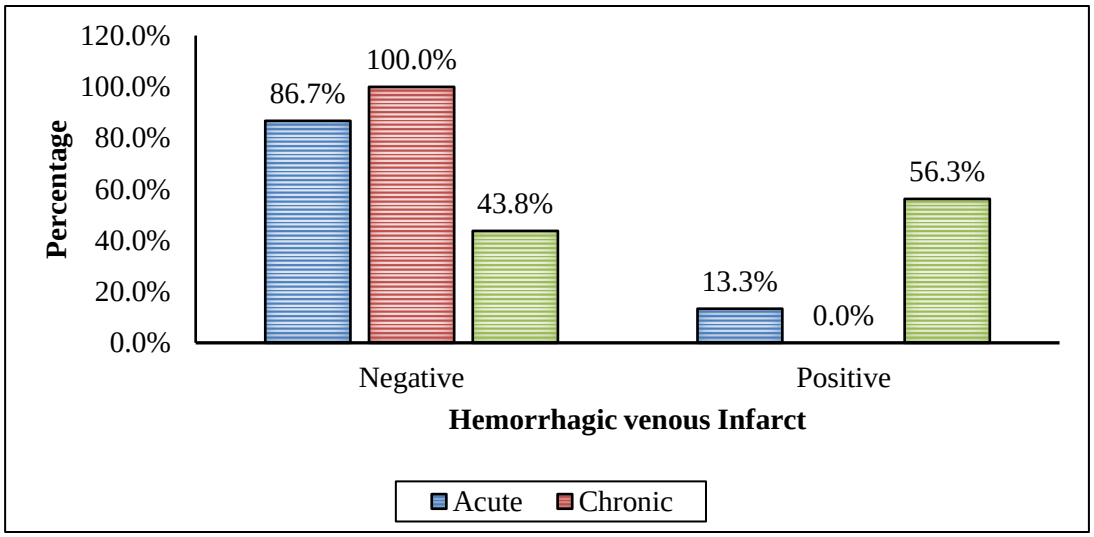


Graph 2: Distribution of subjects by age and presentation.

In the subacute presentation, the most affected age group was 21-30 years whereas 41-50 years in chronic presentation.

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.



Graph 3: Distribution of subjects by Hemorrhagic venous infarct and presentation.

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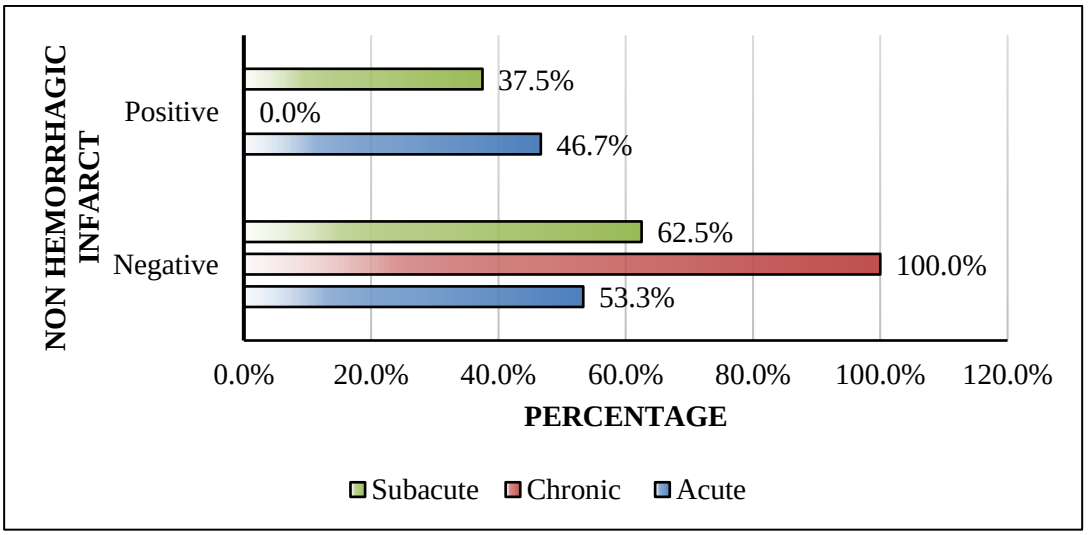
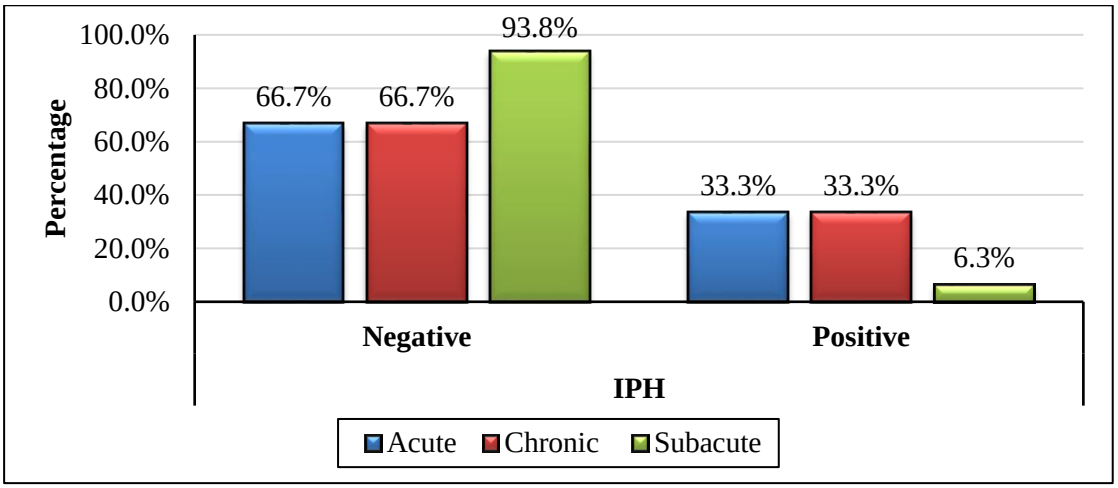
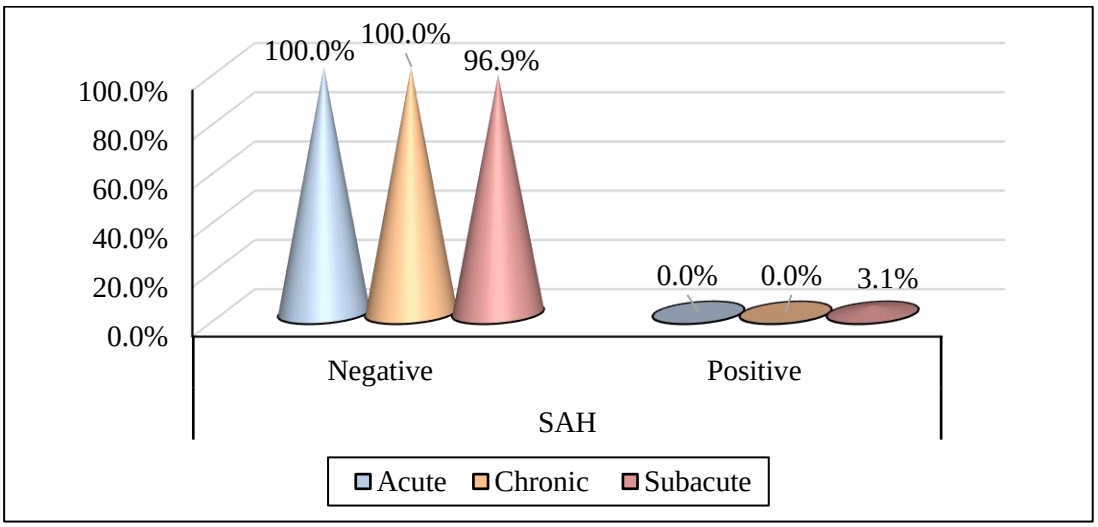


Graph 4: Distribution of subjects by Non-Hemorrhagic infract and presentation.



Graph 5: Distribution of subjects by IPH infract and presentation.



Graph 6: Distribution of subjects by SAH and presentation.

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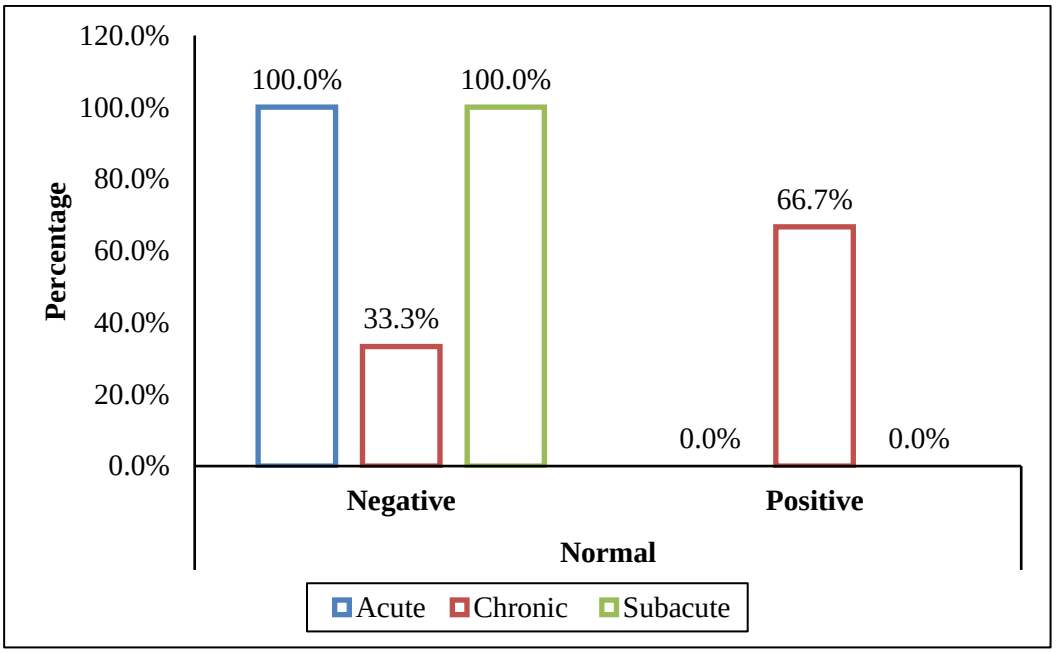
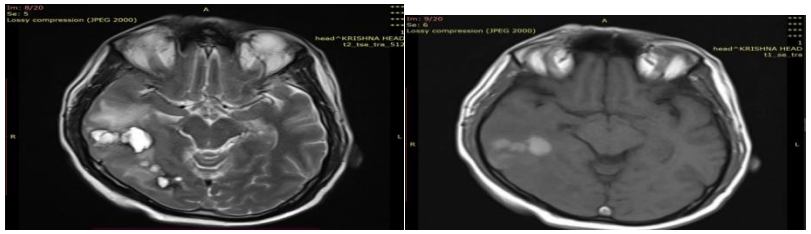
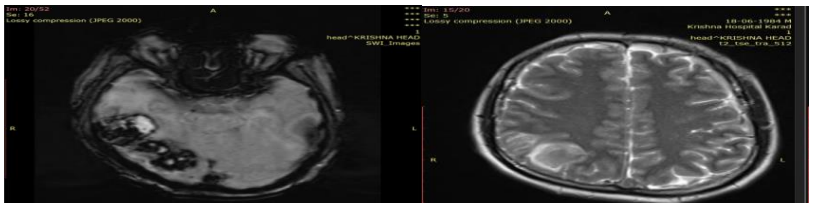


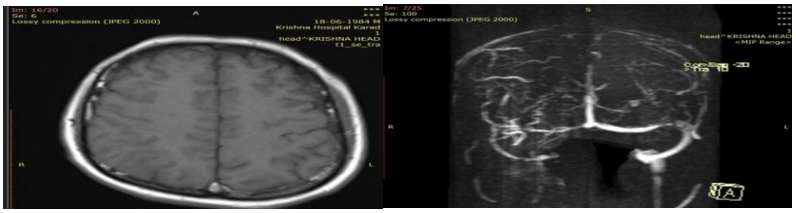
Graph 7: Distribution of subjects by Normal lesions and presentation.



A **B**



C **D**



E **F**

Figure 1: Area of altered signal intensity (~6.5 x 3.0 x 2.6 cm) hyperintense on T2WI/FLAIR (A), hyperintense on T1WI (B) noted in the right parietal-temporal region with the corresponding area showing blooming on SWI (C) suggestive of **Intra parenchymal hematoma** (blood in acute to the subacute stage) with surrounding edema.

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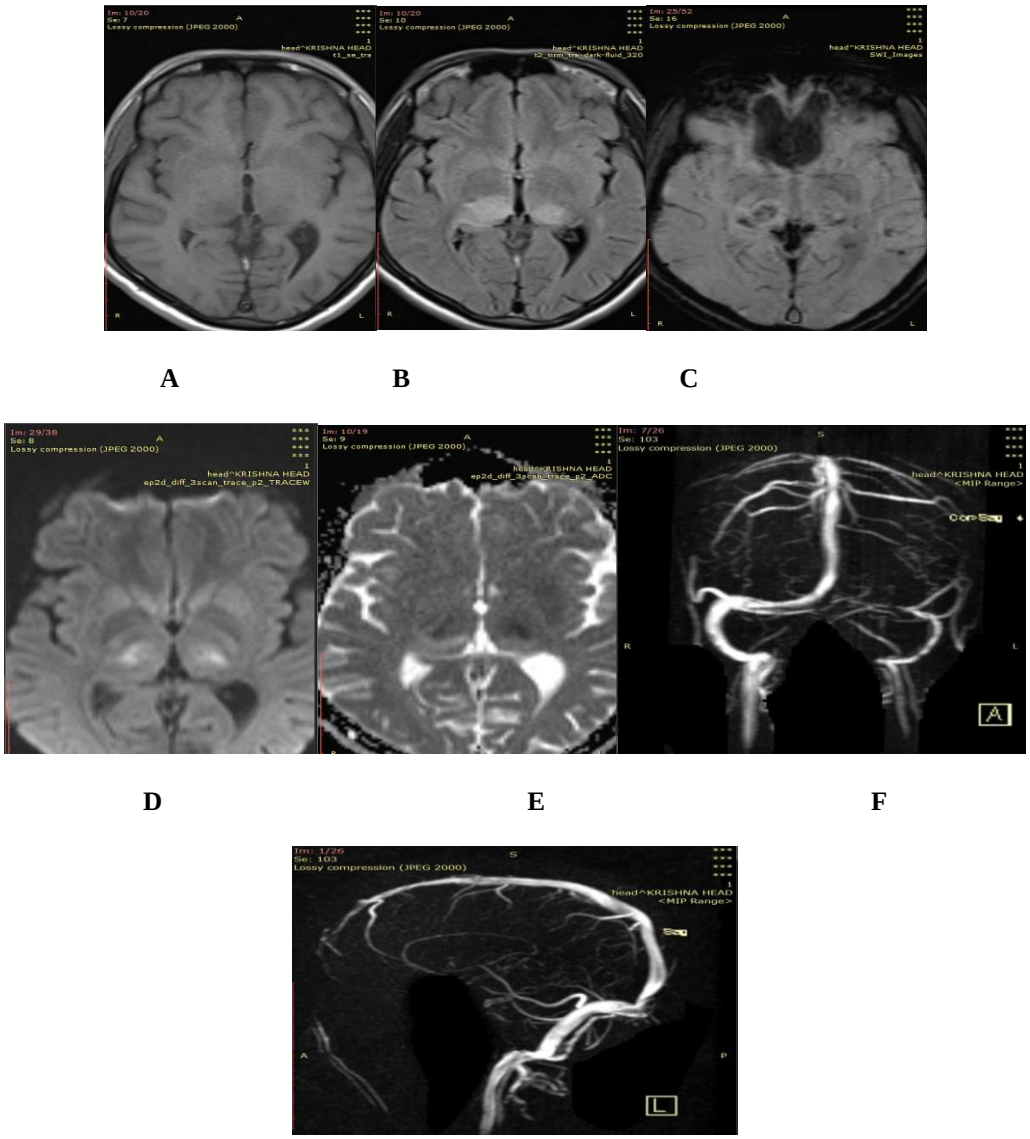
Loss of flow void of the superior sagittal sinus with Intra luminal hypo intensity on T2W (D) and hyperintensity on T1W (E).

2D PC MIP image (F) shows Superior sagittal sinus thrombosis with thinned-out right transverse sinus, sigmoid sinus & jugular bulb.



A **B** **C**

D **E** **F**

G

Figure 2: Bilateral thalami show hypointense areas on TIWI (A) which are heterogeneously hyperintense on FLAIR (B), with areas of blooming on SW images (C), hyperintense on DWI (D)with corresponding low ADC values (E)– acute hemorrhagic infarct. 2D PC MR Venogram (MIP sequences) (F, G)- Thrombosis of Straight sinus, internal cerebral veins and left transverse sinus. Partial thrombus in proximal 1/3rd of the right transverse sinus.

Large areas with surrounding edema appearing hyperintense on T2WI/FLAIR (B), showing areas of blooming on hemo (C) sequences, patchy areas of diffusion restriction (D) involving the cortex and subcortical white matter of bilateral frontal lobes – Acute hemorrhagic venous infarct.

4. Discussion

Dural sinus thrombosis, as well as thrombosis of the deep or cortical cerebral venous system, are more common than previously thought.

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Cerebral venous thrombosis (CVT) is a cause of stroke with diverse etiologies and a spectrum of clinical presentations. Its presentation may mimic acute arterial strokes or a mass lesion; thus, radiological examinations have a crucial role in the diagnosis of CVT and help to determine the prognosis. MRI and MR venography are useful tools in this regard.

Conventional MR imaging (T2 and FLAIR) depicted similarly high signal intensities for the areas of venous congestion and infarction, thus inadequately distinguishing between cytotoxic and vasogenic edema. Diffusion-weighted imaging provides ADCs that can differentiate whether the concurrent cerebral edoema is of cytotoxic or vasogenic origin. However, the findings on diffusion-weighted imaging and the ADC value in patients with Dural sinus thrombosis require further investigation.

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 classified as subacute, and more than 30 days as chronic as shown in table 9. [6]

Table 9: 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%)

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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%)

The most common presentation was found to be subacute (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.

Among the cases that presented in the acute phase, the most common age of presentation was 21–30 years; similarly, for the subacute phase, it was again 21–30 years; and for the chronic phase, it was 41–50 years. However, the p-value was found to be statistically insignificant.

Most patients in both genders presented in the subacute phase, with an average day after which imaging was done at 7.94±4.07.

Various risk factors are seen as being associated with cerebral venous thrombosis.

In this study, no definite cause of the CVT was found in 28 (56%) patients despite their extensive histories. This figure (56%) is higher than the study conducted by Gates et al. [7]. They stated that in 20–30% of cases, the underlying predisposing factor could not be revealed. It indicates that close follow-up with patients is required.

Eight cases (16%) of peripartum thrombosis were found in our study. Hypercoagulable states (anti-cardiolipin) associated with puerperium may be one of the major factors.

In this study, 5 patients (10%) were using OCP for 6 months to 14 months. OCP has a prothrombotic effect, which was proven in the laboratory by Vandenbroucke et al. [8] Other control studies done by Martinelli et al. revealed an increased risk of CVT in the women in the younger age group who were using OCP. [9]

Four patients (8%) in our study disclosed their current smoking history.

In this study, 2 cases of trauma were found (4%). Two of them had closed head injuries, and one had a depressed skull fracture present over the sagittal area. Miller et al. studied 400 cases of depressed skull fractures and concluded that cerebral venous sinus involvement was seen in 11% of cases.[10] They illustrated how disruption of normal flow, increased intracranial pressure, and infection can cause damage or obliteration of the sinus lumen.

In CVT, large numbers of symptoms are associated with variable presentations. Most of these may be nonspecific, like headaches, seizures, focal neurological deficits, blurring of vision, nausea, vomiting, and even a decreased level of consciousness. Depending upon the availability of collateral venous pathways, it can result in significant brain involvement or may be well

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tolerated.[11] In this study, headache was the most common symptom in 38/50 patients (76%), followed by a focal neurological deficit in 19/50 (38%), seizures and vomiting in 13/50 patients (26% of each) This is similar to the previous study of D. Karthikeyan et al. who stated that headache is the most presenting and non-specific symptom seen in 70-90% of cases.[12] In most cases, a headache is unilateral.Focal neurological deficits (hemiparesis, hemisensory disturbance), seizures, impairment of level of consciousness, and papilledema occur in one-third to three-quarters of cases.

Venous sinus involvement and correlation of cerebral parenchymal abnormalities:

In most of the patients, multiple segments of the dural venous sinuses were involved at a time. In the present study, the most common sinus involved was the superior sagittal sinus in 35 patients (70%) and, in these cases, parenchymal involvement was unilateral or bilateral frontoparietal lobes. The other most commonly involved sinuses were the transverse sinus in 21 patients (42%), and the sigmoid sinus in 11 patients (22%), which was associated with parenchymal involvement in the ipsilateral parieto-temporal lobes.

The deep venous system was involved in 3 patients (6%). The superior sagittal and transverse or sigmoid sinuses were found to be the most frequently associated.These findings are similar to the findings of Greiner et al. [13] They concluded that in veno-occlusive stroke, the superior sagittal sinus, followed by the transverse, sigmoid, and straight, were generally involved.

The parenchymal abnormalities that occurred with deep venous occlusion were in the thalami and deep periventricular regions. In our study, the deep venous system was found to be affected in 6 percent of cases. These findings are similar to those of Bousser et al., who stated that thrombosis of the deep cerebral venous system is rare and difficult to diagnose. [14]

However, the prediction of parenchymal changes with the extent of venous sinus involvement was variable. Isolated cortical venous thrombosis was not found in our study and was always associated with superior sagittal sinus thrombosis. This infers that isolated cortical venous thrombosis is an extremely rare entity. Fewer than 20 cases have been reported in the imaging literature [15].

Different types of brain parenchymal findings in cerebral venous thrombosis:

They are classified into 1 of 3 subgroups, depending on the radiological findings [16]:

(1) Intraparenchymal hematoma (IPH), is considered when the patient had a confluent hemorrhagic lesion >1 cm on MRI.

(2) Non-hemorrhagic ischemia (NHI), is characterized by an area of homogeneous hyperintensity on a FLAIR sequence, diffusion restriction, and no signs of hemorrhage on datasets.

(3) Hemorrhagic ischemia (HI), is characterized by a mixture of nonischemic and ischemic signs, that is, an area of hemorrhage on a T2* sequence (greatest dimension < 1 cm) within a large area of hyperintensity on a FLAIR sequence.

Tsai et al. [17] described the spectrum of MRI findings in CVST. In the initial stages, there is a mild increase in Dural venous sinus pressure, and only sinuses are affected without parenchymal abnormalities. In our study, 2 patients (4%) were without any parenchymal involvement. With further increase in intracranial pressure, focal neurological deficit takes place and the affected brain region shows edema or infarction with or without hemorrhage in 10%–50% of cases. In a study from India, a hemorrhagic venous infarct was present in 45.6% [7] which was consistent with our findings that the most common brain parenchymal finding was hemorrhagic venous infarct seen in 20 (40%) patients.

In the present study, intraparenchymal hematoma was seen in 8 (16%) patients. The mechanism of haemorrhage is multifactorial. Hemorrhage may be precipitated by continued arterial perfusion in areas of cell death, as can be seen at reperfusion in arterial ischemia. Elevation of venous pressure beyond the limit of the venous wall was also believed to be the cause [18].

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Hemorrhagic venous infarction was determined by heterogeneous signal intensities on T1, T2, and FLAIR, as well as blooming on GRE sequences and patchy areas of diffusion-weighted imaging. The signal intensities were attributed to hemorrhage. The bright signal intensity of the hemorrhagic clot on DWI was due to the paramagnetic effect of the intracellular methemoglobin, and the surrounding low SI with high ADC values was due to vasogenic edema. A thin rim of low signal was observed between these, indicating the presence of hemosiderin. These findings were similar to those reported by Kon Chu et al. [19].

Diffusion-weighted imaging and ADC measurement of intracranial hematoma were recently reported by Atlas et al. [20]; however, in our study, ADC values of hematoma were avoided. The reason is that the determining factors of ADC values in hematoma may be due to the paramagnetic effects of methemoglobin rather than the true restriction of water movement.

In the present study, non-hemorrhagic venous infarction was seen in 19 (38%) patients. The findings were focal or multifocal high signal intensities in DWI, with no evidence of blooming on GRE and hyperintense signals in T1, T2, and FLAIR.

SAH has rarely been reported in association with CVST. One patient was described by Sztajzel et al. who presented with right cerebellar SAH secondary to thrombosis of the right TS or SS. [21] The mechanism behind the development of SAH in isolated cortical venous thrombosis is not certain. In a large study from India, 3 out of 392 patients (0.7%) had SAH. [7] In the present study, one (2%) patient had SAH along with an intraparenchymal hematoma. However, we did not encounter an isolated finding of SAH in our study group.

In the acute phase, it was seen that in our study, most patients (7 patients) presented with a non-hemorrhagic infarct followed by intraparenchymal haemorrhage (seen in 5 patients). In the subacute phase, it was found that 18 cases were of hemorrhagic venous infarct, 12 cases were of non-hemorrhagic infarct, and only 2 cases were of intraparenchymal hemorrhage.

It was also discovered that the majority of patients (18 cases) who presented with hemorrhagic venous infarction were imaged in the subacute phase.The p-value was found to be significant for this correlation.

In all cases presenting with a non-hemorrhagic infarct, 12 cases were imaged in the subacute phase; however, this correlation was not statistically significant (p-value was greater than 0.05).

In all cases presenting with intraparenchymal hemorrhage, the greatest number of cases were imaged in the acute phase. The p-value was found to be statistically significant.

The signal intensity of clot in sinuses

Diffusion-weighted imaging also showed the high signal intensity of the intravascular clot, a finding that has been reported by **Kon Chu et al.** [19]. This finding was believed to be due to the paramagnetic effect of the clot (intracellular methemoglobin) and the T2 shine-through effect. The T1 and T2W SI were also high, and these findings were similar to those of the early subacute stage thrombus. This means that diffusion-weighted imaging is not required for direct imaging of a clot within a cerebral sinus because conventional MR sequences and MR venography can identify these lesions. However, **Favrole et al.**[22] reported that the movements of water molecules are more or less restricted within the venous clot according to the stage of thrombus formation in CVT. However, in our study, we did not acquire data on DWI sequences.

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 remaining 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. Acute thrombus showed an isointense signal on T1W with a hypointense signal on T2W. Subacute thrombus

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showed a hyperintense signal on both T1W and T2W.

Along with parenchymal changes, venous thrombosis was confirmed on a 2D phase contrast MR venogram using the source as well as MIP images. 48/50 cases showed evidence of thrombosis on the source images of the MR venogram in the form of non-visualization of the involved sinus or decreased and partial flow-related enhancement. The two cases in which we could not elicit evidence of thrombosis included one chronic case (> 30 days) with the involvement of a small area of thrombosis in the left transverse sinus. Likely due to collateral formation, it was not appreciated on the source images of the 2D PC/MR venogram. Another case involved thrombosis of the anterior most portion of the superior sagittal sinus as well as the cortical vein.Though the parenchymal changes were present, the source images were not promising. So, in both cases, we performed contrast MR venography first, and the thrombosis of involved segments was noted.So, contrast venography is more accurate in the diagnosis of CVT than 2D phase contrast venography. However, because conventional MR sequences can often be used to diagnose CVT, post-contrast venography should be used with caution.

Detection of an early venous infarct:

In our study, all cases of cerebral venous infarction manifesting hyperintensity on diffusion-weighted images also showed T2 signal changes. This is probably explained by image timing because we did not image any subjects hyperacutely, when diffusion restriction might have been present in the absence of T2 hyperintensity. This also explains why the time from the onset of the disease to DWI was variable and non-homogeneous. This can be attributed to the diverse clinical manifestations of CVT.

Limitations Of Study

- The sample size was small so the observations would not always extrapolate similarly when projected for larger populations.
- The demographic distribution of our study population was predominantly rural which caused nonconformity of findings such as only a slight preponderance of female patients, the prevalence of the most common age group being 3rd decade (probably active healthy males and females working in the farm sector) and the inability to narrow down the etiology of the sinus thrombus in a majority of the patients even on extensive history elicitation and further work up.
- 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.

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