



FROM SIGNAL TO SIGNIFICANCE: ROLE OF DWI AS A DIAGNOSTIC MARKER IN CEREBRAL VENOUS THROMBOSIS

Radio-Diagnosis

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ABSTRACT

Introduction: Cerebral venous thrombosis (CVT), a distinct and under-recognized cause of stroke, results from thrombosis of the dural sinuses or cortical veins. Unlike arterial strokes, CVT involves a combination of cytotoxic and vasogenic edema due to venous congestion. Diffusion-weighted imaging (DWI), an advanced MRI technique, can differentiate these edema types, offering diagnostic and prognostic value. This study evaluates DWI's role in detecting parenchymal abnormalities and characterizing edema in patients with CVT. **Materials and Methods:** This prospective observational study included 40 patients clinically suspected of CVT, conducted from January 2024 to February 2025 at a tertiary care hospital. MRI scans were performed on a 1.5 Tesla Siemens Semptra scanner, incorporating conventional sequences, DWI, and MR venography. ADC values were calculated by placing regions of interest on abnormal parenchymal areas, avoiding hematomas. The final diagnosis was confirmed using MR venography and structural MRI. **Results:** Among 40 patients (mean age: 30.65 years), headache was the most common symptom (80%), with superior sagittal sinus being the most frequently thrombosed site (55.5%). DWI revealed three main patterns: heterogeneous signal in hemorrhagic infarcts, hyperintense signal in non-hemorrhagic infarcts, and high-signal intravascular clots. ADC values varied: cytotoxic edema showed decreased ADC, while vasogenic edema exhibited elevated values. All parenchymal lesions were supratentorial and were also visible on conventional MRI, suggesting limited added value of DWI for clot detection. **Conclusion:** DWI enhances the diagnostic utility of conventional MRI in CVT by characterizing parenchymal changes and distinguishing cytotoxic from vasogenic edema, aiding prognosis and treatment planning. Although DWI has limited utility in detecting thrombus directly, it offers essential insights into the underlying pathophysiology and potential reversibility of venous infarcts. Future time-sensitive studies could further elucidate DWI's prognostic value in CVT management.

KEYWORDS

Cerebral venous thrombosis ; Diffusion-weighted imaging (DWI); Vasogenic edema; Cytotoxic edema; Venous infarct

I) INTRODUCTION

Cerebral venous thrombosis (CVT), characterized by thrombosis of the dural sinuses and cortical veins, is more prevalent than previously recognized and constitutes a notable cause of The condition manifests with various symptoms, including headache, increased intracranial pressure, seizures, multifocal neurological deficits, and coma (1,2) . Accurate diagnosis is essential, as timely interventions such as anticoagulation can lead to substantial recovery, even if administered later (3)

CVT is distinct from arterial strokes in terms of pathophysiology and clinical characteristics. Venous strokes exhibit vasogenic and cytotoxic edema resulting from venous congestion, while arterial strokes predominantly display cytotoxic edema. Acute venous occlusion elevates capillary filtration pressure, resulting in vasogenic edema, whereas insufficient perfusion causes cytotoxic edema(7). Hemorrhagic transformation can complicate the interpretation of MRI results.

CVT is prevalent among young adults and children, occurring three times more frequently in females; however, this gender disparity decreases after the age of 60. (4,5,6) Conventional MRI frequently encounters challenges in differentiating vasogenic from cytotoxic edema. Diffusion-weighted imaging (DWI), an advanced MRI technique that detects water molecule motion, enhances the early identification of these edemas. In CVT, DWI aids in distinguishing tissue types: vasogenic edema is hyperintense on T2-weighted (T2W) images and Apparent diffusion coefficient (ADC) maps, whereas cytotoxic edema exhibits restricted diffusion, appearing bright on DWI and dark on ADC maps. (8,9) Interpretation may be complicated by the "T2 shine-through" effect observed in chronic infarcts or lesions.(11,13)

Some venous infarcts may resolve through collateral drainage, whereas others persist. In contrast, arterial infarcts typically lead to permanent damage. Numerous studies have assessed DWI in CVT, yet their findings remain inconclusive.(1,14) CVT presents both clinical and radiological challenges.(15,19) This study seeks to evaluate the effectiveness of diffusion-weighted MRI in enhancing diagnosis, evaluating disease mechanisms, and forecasting outcomes

II) MATERIALS AND METHODS

Between January 2024 and February 2025, a prospective research was undertaken at a Jaipur National University Institute for Medical Sciences and Research Centre, after approval from the Institutional Ethics Committee was sought before the trial started; informed permission was also acquired from every participating patient. The sample size for the study was calculated with heterogeneous signal intensity as the primary outcome variable. Based on the findings of Kon Chu et al.(14), an expected proportion of 71.4% for heterogeneous signal intensity was assumed. A relative precision of 20% and a 95% confidence level were considered for the calculation. The required sample size was determined to be 38; therefore, 40 subjects were included in the study to accommodate any potential data loss. Patients clinically suspected of cerebral venous thrombosis, who were referred to the Department of Radiodiagnosis and were willing to undergo MRI of the brain during the study period, were included. Exclusion criteria include if they had claustrophobia, the presence of a pacemaker, metallic prostheses, or any contraindications to contrast administration.

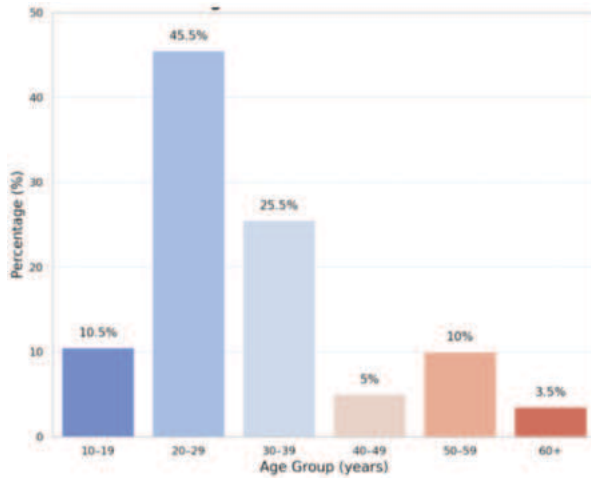
MRI scans were conducted using the Siemens Semptra 1.5 Tesla (T) scanner, featuring an ultracompact, superconducting magnet. Imaging protocol comprising of standard spin echo sequences such as axial T1, axial T2, and axial Fluid-Attenuated Inversion Recovery (FLAIR) along with Magnetic Resonance Venography (MRV), DWI with T1W and T2W sequences in sagittal and coronal planes. In all the cases, MR Venogram and other standard MR sequences verified the diagnosis of CVT. Two b-value diffusions of 50, and 800 s/mm² were used to acquire diffusion-weighted images with echo-planar imaging. Oblique, sagittal and coronal planes were used for MRV. An aberrant intensity area of interest was selected after avoiding areas of hematoma in order to calculate ADC values. When several sites of aberrant intensity were seen, the areas with highest and lowest ADC values were considered the representative lesions.

III) OBSERVATIONS AND RESULTS

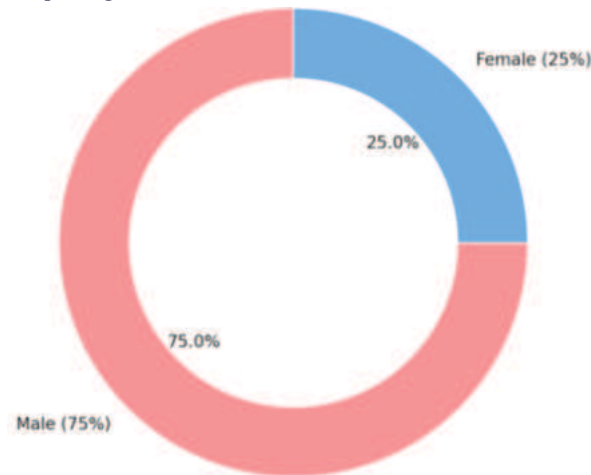
In this observational study, image analysis was performed for 40 patients diagnosed with cerebral venous thrombosis (CVT). Ranging in age from 10 to 83 years, the cohort study consisted of 28 men and 12 women with an average age of 30.65 years.

a) Age & Sex Distribution

The majority of patients were in the 20–29-year age group (45.5%), followed by the 30–39-year group (25.5%). Five patients (10.5%) were aged between 10–19 years, while two patients (5%) were aged 40–49 years. Four patients (10%) were between 50–59 years, and only one patient (3.5%) was older than 60 years.(Graph 1) A male predominance was observed, with 28 male patients (75%) and 12 female patients (25%).(Graph 2)



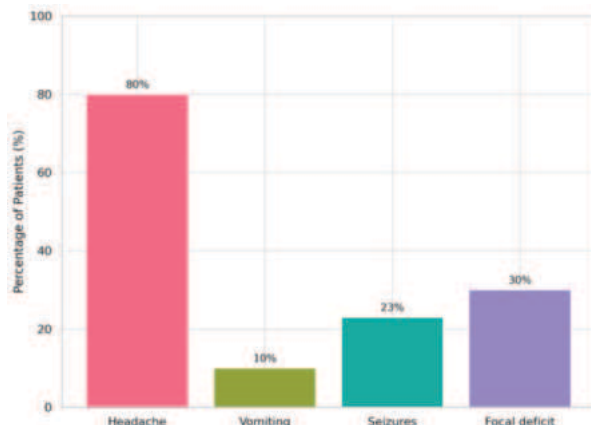
Graph 1: Age Distribution of Patients



Graph 2: Sex Distribution of Patients

b) Presenting Symptoms

The most frequent presenting ailment, reported in 80% of the patients, was headache. Other clinical signs were vomiting (10%), seizures (23%), and focal neurological abnormalities (30%). Additional symptoms included giddiness, fever, loss of consciousness, visual impairment, and poor sensorium, in a declining sequence of frequency. (Graph 3)



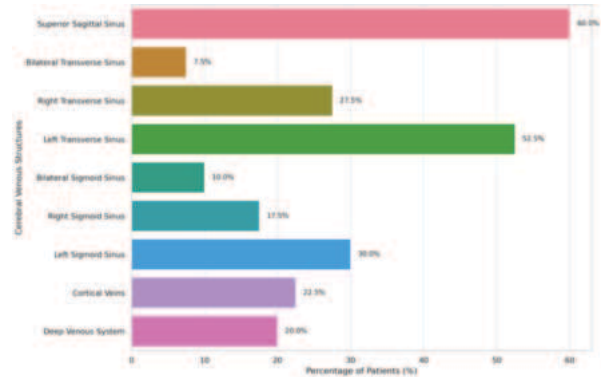
Graph 3: Distribution of Presenting Symptoms

c) Imaging Analysis

A structural assessment of the site of each blocked vein or sinus was used on the basis of results of both MR venography and conventional MRI for the goal of conducting image analysis. Visual inspection of a particular area on the DWI and identification of signal anomalies in the brain parenchyma using conventional imaging and assessment of DWI using ADC values both help to identify the evaluation of an elevated signal in veins or sinuses.

d) Distribution Pattern of CVT Based on Anatomical Locations:

Superior sagittal sinus involvement was the most common finding observed in 24 patients (60%). Transverse sinus involvement was noted in 35 patients: bilateral transverse sinus thrombosis in 3 patients (7.5%), right transverse sinus in 11 patients (27.5%), and left transverse sinus in 21 patients (52.5%). Sigmoid sinus thrombosis was seen bilaterally in 4 patients (10%), isolated right sigmoid sinus involvement in 7 patients (17.5%), and left sigmoid sinus involvement in 12 patients (30%). Cortical vein involvement was observed in 9 patients (22.5%), while the deep venous system was involved in 8 patients (20%) (Graph 4 & Table 1)



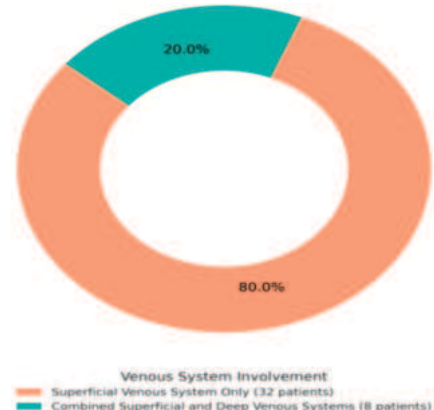
Graph 4: Distribution Pattern of CVT Based on Anatomical Locations

Table 1 – Distribution Pattern of CVT Based on Anatomical Locations in 40 Patients

CVT Location	Frequency (Percentage) of patients
Superior Sagittal sinus	24 (60%)
Transverse sinus (TS)	
Bilateral TS	3 (7.5%)
Right TS	11 (27.5%)
Left TS	21 (52.5%)
Sigmoid sinus (SS)	
Bilateral SS	4 (10%)
Right SS	7 (17.5%)
Left SS	12 (30%)
Cortical Veins	9 (22.5%)
Deep Venous System	8 (20%)

Involvement of Superficial and Deep Venous Systems

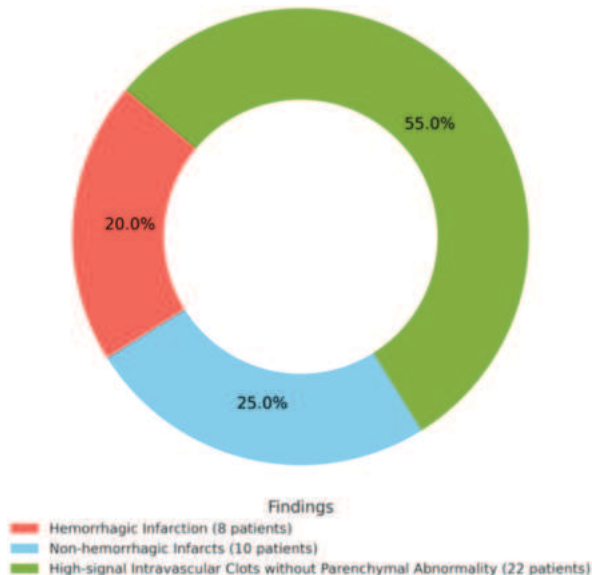
Involvement of the superficial venous system alone was observed in 32 patients. Combined involvement of both superficial and deep venous systems was seen in 8 patients (Graph 5).



Graph 5: Distribution of Superficial and Deep Venous System Involvement

d) Parenchymal Anomalies in CVT with Diffusion Profiles

Parenchymal abnormalities associated with diffusion hyperintensities were identified as follows (Graph 6). Hemorrhagic infarction was present in 8 patients. Non-hemorrhagic infarcts were observed in 10 patients. High-signal intravascular clots without associated parenchymal abnormality were seen in 22 patients. All parenchymal lesions were located supratentorially.



Graph 6: Distribution of Parenchymal Anomalies in CVT with Diffusion Profiles

Patients with superior sagittal sinus thrombosis frequently had parasagittal frontal and parietal lobe involvement. Five superior sagittal sinus occlusion patients showed bilateral parenchymal damage. Whether thrombosis was total or partial had no bearing on hemorrhagic infarcts.

Transverse sinus thrombosis was linked to parenchymal alterations confined to the temporal or occipital lobes. Thalamic and deep white matter involvement were linked to occlusion of the deep venous system, including the internal cerebral veins, straight sinus, and vein of Galen.

All cerebral venous infarctions were recognized by employing the usual sequences of magnetic resonance imaging. According to diffusion-weighted imaging, there was not a single participant who exhibited hyperintensity without concomitant abnormalities on conventional imaging.

Normal ADC values of the undamaged brain parenchyma were $0.75\text{--}0.80 \times 10^3 \text{ mm}^2/\text{s}$. Diffusion-weighted pictures in hemorrhagic infarcts showed irregular signal intensities with a typical thin black ring and surrounding low signal intensities(22). Near the hemorrhagic cavity, ADC maps revealed lower values; in the periphery low signal intensity areas, they exhibited higher values ($0.8\text{--}1.5 \times 10^3 \text{ mm}^2/\text{s}$).(22,24)

Of the patients with non-hemorrhagic infarcts, two had high ADC values that suggested vasogenic oedema, and eight had lowered ADC values that were compatible with limited diffusion, which is comparable to arterial strokes. Twenty-two individuals had high-signal intravascular clots on diffusion-weighted imaging. Eighteen of these individuals had lower ADC levels, while four of them had higher ADC values.

IV) DISCUSSION

In this cohort of patients with Dural sinus thrombosis, often extending into deep or cortical veins diverse DWI patterns are observed. While deep venous involvement impacted thalami and periventricular areas, parenchymal abnormalities were more often ipsilateral to transverse-sigmoid sinus thrombosis (parieto-temporal lobes) or linked with superior sagittal sinus blockage (fronto-parietal lobes). Emphasizing their scarcity, no separate cerebral venous thromboses were seen.

Three Principal DWI Phenotypes Emerged:

- (1) Heterogeneous SI in haemorrhagic venous infarcts,
- (2) High SI in non-haemorrhagic strokes with variable ADC, and
- (3) Hyperintense intravascular clots mirroring early subacute thrombus.

Comparison with Prior Studies

Our heterogeneous-signal subgroup (n=8) mirrored the mixed DWI appearance described by Kon Chu et al., in which intracellular methaemoglobin generates high DWI SI, vasogenic oedema yields elevated ADC, and peripheral hemosiderin appears as a low-SI rim (14). Unlike Atlas et al., who quantified ADC in hematomas, we omitted hematoma ADCs to avoid artefactual paramagnetic influences (25). The non-haemorrhagic group (n=10) exhibited both restricted (low ADC) and facilitated (high ADC) diffusion. This biphasic ADC evolution parallels Forbes et al. and Kon Chu et al., who documented an early ADC decrease—akin to cytotoxic oedema in arterial ischemia—that normalizes or overshoots baseline by day 4 (21,14). The existence of high-ADC lesions in our series likely reflects predominant vasogenic oedema or reperfusion effects, although clinical deficits often did not map precisely to DWI abnormalities, suggesting complex pathophysiology beyond simple ischemia.

Hyperintense clot within sinuses was readily visible on DWI in two patients, consistent with reports by Kon Chu et al. and Favrole et al., who linked water-movement restriction in thrombus to its maturation stage(14,22). However, since conventional T1/T2-weighted MRI and MRV reliably depict thrombus, DWI offers no clear added value for direct clot detection.

Increased venous pressure and compromised capillary perfusion most likely cause cytotoxic oedema in venous infarcts by means of restricted diffusion. On the other hand, enabled diffusion points to vasogenic oedema from blood-brain barrier damage. The combination of both inside haemorrhagic infarcts shows the interaction of haemorrhage, oedema, and blood degradation products. Heterogeneous imaging intervals might explain the lack of hyperacute DWI-only lesions (i.e., genuine diffusion restriction) without T2W changes as well as possible under-representation of really hyperacute venous infarcts.

Clinical Implication includes DWI enriches conventional MRI by characterizing oedema type (Cytotoxic or Vasogenic), which may influence therapeutic urgency(23). Early restricted diffusion could flag regions at risk of irreversible injury, underscoring the need for prompt anticoagulation. Conversely, high ADC predominance may predict reversible changes and favourable outcomes, as illustrated in cases with rapid clinical resolution after therapy. However, DWI should not supplant MRV for thrombus detection, and ADC measurements in hematoma require cautious interpretation.(22,24)

As an observational series, our study is limited by variable timing of MRI relative to symptom onset, potentially biasing diffusion patterns. The sample size, particularly for haemorrhagic and intravascular-clot subgroups, was small. ADC measurements in hematoma were deliberately omitted, which precludes direct comparison with some prior quantitative studies. Finally, clinical correlation was limited by heterogeneous presentations and the retrospective design.

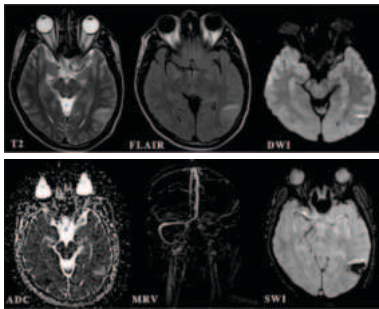
Prospects consist of prospective serial DWI investigations that ideally capture hyperacute (<24 h) time points required to track ADC progression in venous infarction.(22) Quantitative diffusion-tensor imaging (DTI) or intravoxel incoherent motion MRI might help to further define interstitial fluid changes and microvascular. (5) Combining MR angiography (MRA)and perfusion MRI (p-MRI) might clarify collateral routes affecting the development of oedema. Diffusion measures correlated with long-term neurological results might help to define prognostic use.

V) CONCLUSION

In summary, DWI complements structural MRI in CVT by differentiating cytotoxic from vasogenic oedema and may guide clinical management, but its role in direct thrombus visualization is limited. Larger, time-resolved studies are warranted to refine its diagnostic and prognostic applications. Consequently, whereas MR venography is the definitive method for thrombus detection, DWI with ADC quantification offers essential insights into the equilibrium between cytotoxic and vasogenic oedema, which may influence prognosis and treatment urgency.

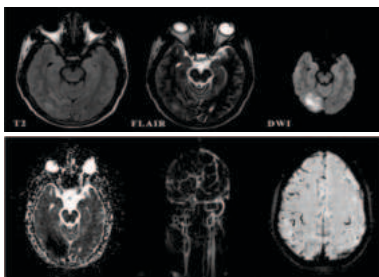
Figures

Case 1



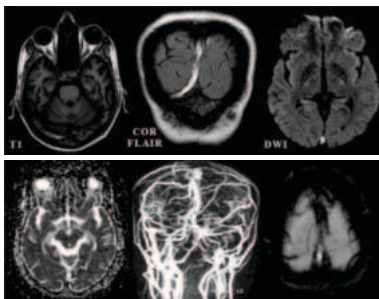
A 38Y/M patient presented with an acute onset of headache, giddiness, and limb weakness underwent MRI brain, T2-weighted and FLAIR sequences revealed hypointense signals in the left transverse and sigmoid sinuses. MR venography showed a filling defect, confirming the diagnosis of cerebral venous sinus thrombosis (CVST). Additionally, a hyperintense lesion with surrounding oedema was identified in the left temporal lobe, suggestive of parenchymal involvement. Diffusion-weighted imaging (DWI) demonstrated areas of diffusion restriction, while susceptibility-weighted imaging (SWI) revealed blooming artifacts, indicative of subarachnoid haemorrhage. These findings are consistent with venous infarction with haemorrhagic transformation secondary to left transverse and sigmoid sinus thrombosis.

Case 2



A 52Y/F patient with acute onset of headache, loss of consciousness and blurring of vision underwent MRI for evaluation of neurological symptoms. T2-weighted and FLAIR sequences revealed a poorly defined hyperintense lesion in the right occipito-parietal region, suggestive of parenchymal oedema or infarction. MR venography demonstrated multiple linear blooming foci within the sulcal spaces bilaterally, indicating cortical venous thrombosis. Diffusion-weighted imaging showed restricted diffusion with corresponding low ADC values, consistent with cytotoxic oedema. Susceptibility-weighted imaging (SWI) revealed localized blooming, suggestive of microhaemorrhages. Contrast-enhanced MR angiography showed extensive filling defects in the superior sagittal sinus, straight sinus, right transverse and sigmoid sinuses, and the right internal jugular vein—findings consistent with deep cerebral venous sinus thrombosis.

Case 3



A 28Y/M patient with acute onset of seizures and vomiting underwent MRI for evaluation of neurological symptoms. MRI revealed hyperintense signals on both T1-weighted and T2-weighted images in the superior sagittal sinus, right transverse sinus, right sigmoid sinus, and the proximal right internal jugular vein, suggesting subacute thrombus. MR venography demonstrated partial filling defects in these sinuses, consistent with partial deep venous sinus thrombosis.

Additionally, multiple linear blooming areas along the sulcal spaces of both cerebral hemispheres were observed, indicative of associated cortical vein thrombosis.

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