Print ISSN: 2321-6379
Online ISSN: 2321-595X
DOI: 10.17354/ijss/2018/215

Original Article

# Role of Magnetic Resonance Venography, Diffusion-Weighted Imaging, and Gradient-Recalled Echo in Cerebral Sinovenous Thrombosis

**Nadeem Ahmed[1], Alle Praveen Kumar[2], Swapna Chouhan[3], Shaik Ismail[4], Uzma Afreen[4]**

[1]Professor, Department of Radiology, Osmania General Hospital, Hyderabad, India, [2]Assistant Professor, Department of Radiology, Mahatma Gandhi Memorial Hospital, Warangal, India, [3]Associate Professor, Department of Radiology, Mahatma Gandhi Memorial Hospital, Warangal, India, [4] Postgraduate, Department of Radiology, Mahatma Gandhi Memorial Hospital, Warangal, India

## Abstract

**Background:** Cerebral venous thrombosis (CVT) is a cause of stroke with inconspicuous pathophysiological properties that differ from arterial stroke. It is an elusive diagnosis because of its non-specific presentation and its subtle imaging findings. Diagnosis is often missed on initial imaging. Patients with CVT often make dramatic recoveries after effective therapies including anticoagulants and intrasinus thrombolysis. Accurate diagnosis is difficult as conventional magnetic resonance imaging (MRI) which cannot differentiate between cytotoxic and vasogenic edema. The use of magnetic resonance venography (MRV), diffusion-weighted imaging (DWI), and gradient echo (GRE) combines the strengths of individual imaging sequences in improving diagnostic yield. Accurate and timely imaging diagnosis is essential because it facilitates patient triage, guides clinical management, and helps determine patient prognosis. Diffusion-weighted MRI (DWI) has been reported to detect early ischemic damage (cytotoxic edema) as bright high-signal intensity (SI) and vasogenic edema as heterogeneous SI.

**Aim:** The aim of this study was as follows: (1) To study the extent of venous sinus involvement and associated cerebral parenchymal changes on MRV, (2) to study the pattern of diffusion-weighted images and apparent diffusion coefficient (ADC) mapping in patients with CVT, (3) to study the role of GRE in patients with CVT, and (4) to characterize the clinical applications of DWI in CVT.

**Methods:** A study was conducted on 20 patients diagnosed to have CVT on imaging, over the period of 6 months.

**Results:** Imaging analyses of 20 patients (9 males, 11 females, and age range 19–65 years) were done. Thrombus on MRV was seen as loss of high-flow signal from the sinus in cases of total occlusion of the sinus and frayed or patchy flow signal in the cases of non-occlusive thrombus. 10 patients with hemorrhagic infarct showed heterogeneous SI on DWI and blooming on GRE sequence. Five patients with non-hemorrhagic infarct showed multifocal high-signal intensities in DWI with variable ADC values and no blooming on GRE sequence. Two patients with intracerebral hematoma showed areas of heterogeneous signals on DWI with blooming on GRE sequence, corresponding ADC values were variable. Two patients with no parenchymal changes and one patient with chronic venous thrombosis presented with benign intracranial hypertension.

**Conclusions:** MRV, DWI, and GRE can be used to evaluate the extent of thrombus, differentiate between types of edema, detect intracerebral hematoma, hemorrhagic and non-hemorrhagic infarcts, and give time-saving information for early diagnosis of CVT.

**Key words:** Cerebral venous thrombosis, Diffusion-weighted imaging, Gradient echo, Magnetic resonance venography

## INTRODUCTION

Cerebral venous thrombosis (CVT) differs from arterial infarction in several ways. First, the clinical presentation is variable and ranging from headache, raised intracranial pressure to severe multifocal deficits, seizures, and coma.[1] Headache is the most common presentation, occurring in nearly 90% of cases. The headache is usually non-focal, often slowly increasing in severity over several days to weeks. Its main mechanisms of pathophysiology are the breakdown of the blood–brain barrier and the coexistence of cytotoxic and vasogenic edema. Rother *et al.*[2] summarized the mechanisms: (1) Increased pressure in superior sagittal sinus results in reduced capillary perfusion pressure[3] and increased cerebral blood volume,[4] (2) obstruction of venous flow leads to increased intracranial

Access this article online

www.ijss-sn.com

Month of Submission : 06-2018
Month of Peer Review : 07-2018
Month of Acceptance : 08-2018
Month of Publishing : 08-2018

**Corresponding Author:** Dr. Alle Praveen Kumar, Assistant Professor, Department of Radiology, Mahatma Gandhi Memorial Hospital, Mattewada, Warangal, Telangana 506007, India. Phone: 9666060506, E-mail: allepravi@gmail.com.

pressure and blood–brain barrier disruption, resulting in decreased cerebral blood flow,[5] (3) the net capillary filtration increases, leading to progressive cerebral edema, and (4) intracerebral and subarachnoid hemorrhage additionally compromising the brain tissue.[6]

There have been a few case reports on the application of diffusion-weighted imaging (DWI) in patients with CVT.[7-9] These reports revealed that the most striking feature of DWI findings was that reversible apparent diffusion coefficient (ADC) changes (decrease[7,8] or increase[9]) were evident during the acute period of CVT. More recently, various DWI results have been reported[10,11] such as heterogeneous findings and the possibility that cytotoxic edema is a feature of CVT. We report various DWI findings in CVT and evaluate the prognostic value of DWI. Furthermore, the mechanisms of venous stroke were characterized by analyzing ADC maps.

## MATERIALS AND METHODS

This prospective study was performed on 20 patients diagnosed to have CVT over the period of 6 months, from January to June 2018, in the Department of Radiodiagnosis of MGM Hospital, Warangal, Telangana, India.

After an informed consent was taken, patients in this study were scanned with 1.5 T *magnetic resonance imaging* (MRI) (GE 1.5T) in the department of radiodiagnosis and examined with the following sequences:

- T1- and T2-weighted sequence.
- Fluid-attenuated inversion recovery (FLAIR) sequence.
- DWI sequence with corresponding ADC mapping.
- Gradient echo (GRE) sequence.
- 3D time-of-flight imaging sequence.

ADC values were calculated by the software and then displayed as a parametric map that reflected the degree of diffusion of water molecules through different tissues. Then, ADC measurements were recorded for a specified region by drawing regions of interest on the ADC map. ADC values of the region of interest were compared with ADC values of the normal brain parenchyma.

Role of DWI with ADC mapping is to distinguish the type of edema, GRE to diagnose the presence of any bleed and MR venography (MRV) to determine the extent of thrombus are also evaluated.

### Inclusion Criteria

Any patient diagnosed to have CVT on computed tomography or MRI.

### Exclusion Criteria

All patients with metallic implants and pacemakers and claustrophobic patients are excluded from the study.

## RESULTS

A total of 20 patients who were diagnosed with CVT on imaging are included in the present study [Table 1].

In the present study, the female: male ratio is 1.2:1.

The peak incidence of CVT is seen in the age group of 21–40 years in both males and females (55.5% and 45.4%, respectively) [Table 2].

Headache was the most common symptom seen in 6 patients (30%), the next common clinical feature being seizures in 5 patients (25%) followed by hemiparesis in 4 patients (20%) [Table 3].

Hemorrhagic infarct was the most common associated manifestation seen in 10 patients (50%) followed by non-hemorrhagic infarct in 5 patients (25%), intracerebral

**Table 1: Sex-wise distribution in patients with CVT**

| Sex | Number of cases (%) |
|---|---|
| Male | 9 (45) |
| Female | 11 (55) |
| Total | 20 (100) |

CVT: Cerebral venous thrombosis

**Table 2: Sex-wise and age-wise distribution in patients with CVT**

| Age (years) | Males | Cases % | Females | Cases % | Total number of cases |
|---|---|---|---|---|---|
| 0–20 | 0 | 0 | 2 | 18.1 | 2 |
| 21–40 | 5 | 55.5 | 5 | 45.4 | 10 |
| 41–60 | 2 | 22.2 | 3 | 27.2 | 5 |
| >61 | 2 | 22.2 | 1 | 9.0 | 3 |
| Total | 9 | 100 | 11 | 100 | 20 |

CVT: Cerebral venous thrombosis

**Table 3: Distribution of patients based on clinical history in patients with CVT**

| Clinical feature | Cases (%) |
|---|---|
| Headache | 6 (30) |
| Hemiparesis | 4 (20) |
| Giddiness | 2 (10) |
| Seizures | 5 (25) |
| Vomiting | 1 (5) |
| Coma | 1 (5) |
| Raised ICT | 1 (5) |
| Total | 20 (100) |

CVT: Cerebral venous thrombosis, ICT: Intracranial tension

hematoma in 2 patients (10%), without parenchymal changes in 2 patients (10%), and chronic venous thrombosis with benign intracranial hypertension in 1 patient (5%) [Table 4].

Transverse sinus is most commonly involved in dural venous thrombosis which is seen in 6 patients (30%). The next most common sinus involved is superior sagittal sinus in 4 patients (20%) followed by straight sinus in 2 patients (10%) [Table 5].

Coexistence of cytotoxic and vasogenic edema was seen in 10 patients (50%), vasogenic edema in 4 patients (20%), and cytotoxic edema in 3 patients (15%); no edema was seen in 3 patients (15%) [Table 6].

## DISCUSSION

Cerebral venous infarction is uncommon form of stroke, secondary to CVT. CVT is the occlusion of venous channels in the cranial cavity including dural venous thrombosis, superficial vein thrombosis, and deep cerebral vein thrombosis. They often exist together and the clinical presentation among them is very similar and nonspecific.

**Table 4: Distribution of patients with CVT depending on associated manifestations**

| Associated manifestation | Number of cases (%) |
|---|---|
| Hemorrhagic infarct | 10 (50) |
| Non-hemorrhagic infarct | 5 (25) |
| Intracerebral hematoma | 2 (10) |
| Without parenchymal changes | 2 (10) |
| Chronic venous thrombosis with benign intracranial hypertension | 1 (5) |

CVT: Cerebral venous thrombosis

**Table 5: Distribution of sinuses involvement in patients with CVT**

| Sinus | Number of cases (%) |
|---|---|
| Superior sagittal sinus | 4 (20) |
| Inferior sagittal sinus | 1 (5) |
| Straight sinus | 2 (10) |
| Transverse sinus | 6 (30) |
| Cortical veins | 1 (5) |
| Cavernous sinus | 2 (10) |
| Vein of Galen | 2 (10) |
| Sigmoid sinus | 1 (5) |
| Internal jugular vein | 1 (5) |

CVT: Cerebral venous thrombosis

**Table 6: Distribution of pattern of edema in patients with CVT (Cerebral venous thrombosis)**

| Types of edema | Number of cases (%) |
|---|---|
| Cytotoxic edema | 3 (15) |
| Vasogenic edema | 4 (20) |
| Cytotoxic and vasogenic edema | 10 (50) |
| No edema | 3 (15) |

Correct diagnosis depends on neuroimaging. The diagnostic imaging features can be subtle.[12] This disorder is potentially lethal but treatable, oftenly underdiagnosed in both clinical and radiologic in routine practice. MRV, GRE, and DWI with ADC mapping are a useful method to establish the diagnosis.

These images may reveal either direct sign (visualization of intraluminal clot) or indirect signs (parenchymatous change and intracranial hemorrhage). Early diagnosis and effective treatment will improve the prognosis of the patient.

Parenchymal hemorrhages were seen in 17 patients with CVT. James *et al.* concluded in a study in 2006 that the mechanism of hemorrhage is multifactorial. Hemorrhage may be because of 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 venous wall is also believed to be a cause.[13]

Favrole *et al.* found that the movements of water molecules are restricted within the venous clot according to the stage of thrombus formation in CVT. Some authors have suggested that the migration of fibroblasts into the clot and incorporation of collagen may render the fibrin less accessible to fibrinolytic enzymes.[14] Others say that this resistance may be related mainly to an abnormal fibrin polymerization.[15]

DWI and ADC measurement of intraparenchymal hematoma were reported by Atlas *et al.*, but in our study, ADC values of hematoma were avoided because the determining factors of ADC values in hematoma may be due to paramagnetic effect of the methemoglobin rather than the true restriction of water movement.[16]

CVT is more common in women than in men. In a study of 110 cases, Ameri and Bousser reported a female-to-male ratio of 1.29:1.

Ferro *et al.* made the same observations in a prospective study from 1995 to 1998. This females preponderance is probably due to specific causes such as oral contraceptives, pregnancy, and puerperium.[17] This preponderance of females did not exist before the era of the oral contraceptive pills. Female predominance was also seen in our study where female-to-male ratio was 1.2:1.

Carrol *et al.* reported seizures in 29.83% of patients, Srinivasan and Natarajan found seizures in 66% of patients.[18,19] Mehta *et al.* found seizures in 26.6%. In our study, 25% of patients had convulsion of which generalized tonic-clonic type

was the most common. Our study had 20% of patients with hemiplegia. Hemiplegia was the most common form noticed in various series.[20] Headache was the most common symptom seen in our study which was seen in 6 patients (30%) Khaladkar *et al.* found that the most common sinus involved was superior sagittal sinus with almost equal involvement of transverse and sigmoid sinuses, and the deep venous system was affected in 17.5% of patients, and superficial venous system affected in 2.5% of cases. Most of the patients had involvement of more than one sinus.[21]

The most common association was noticed between superior sagittal sinus and transverse sinuses. Greiner *et al.* found that in veno-occlusive stroke, the superior sagittal sinus followed by transverse, sigmoid, and straight was generally involved.[22] In our study, transverse sinus was the most common sinus involved in 30% of cases followed by superior sagittal sinus in 20% of cases, straight sinus in 10%, and sigmoid sinus in 5% of cases.

## CONCLUSION

Cerebral venous sinus thrombosis is a frequently misdiagnosed condition because of its variability of clinical symptoms and signs. It is very often missed on initial imaging. All age groups can be affected. The prognosis of cerebral venous sinus thrombosis depends on early and accurate diagnosis of CVT.

Our study shows the importance of role of MRI with MRV, DWI with ADC mapping, and gradient-recalled echo in early and precise diagnosis of CVT.

Thrombus on MRV was seen either as loss of high-flow signals from the sinus in cases of total occlusion of the sinus or frayed or patchy flow signal in non-occlusive thrombus. MRV is also useful for demonstrating the extent of cerebral venous system involvement, recanalization of thrombosis in venous sinuses.

DWI with ADC maps can be used to differentiate between vasogenic and cytotoxic edema for tissue viability and to gives information about stages and diagnostic clues in CVT.

Although both vasogenic and cytotoxic edema are identified in the early phase of CVT, vasogenic edema develops more frequently. Our study showed coexistence of increased and decreased ADCs in hemorrhagic infarcts. Increase in ADC suggests predominance of vasogenic edema and decrease in ADC suggests cytotoxic edema.

Gradient-recalled echo is an important technique that detects early hemorrhagic transformations within acute infarctions accurately. It also detects chronic microbleeds and intracerebral hematomas, thus warning the treating physician about the devastating complication of anticoagulant and revascularization therapies.

### Case 1

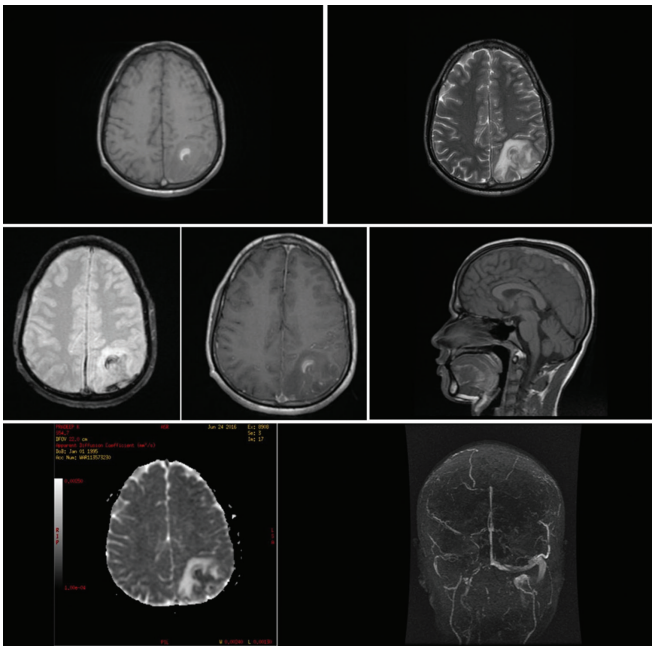
T1, T2, GRE, T1+C, T1 SAGGITAL, DWI, ADC, and MRV

Heterogeneous signal lesion with hyperintense areas in it, which shows blooming on GRE, restricted diffusion on DWI in the left posterior parietal region suggestive of hemorrhagic infarct with vasogenic edema.

Loss of flow void noted in superior sagittal sinus suggestive of superior sagittal sinus thrombosis.

MRV shows superior sagittal sinus and right transverse sinus thrombosis.

On contrast, mild gyral enhancement noted.



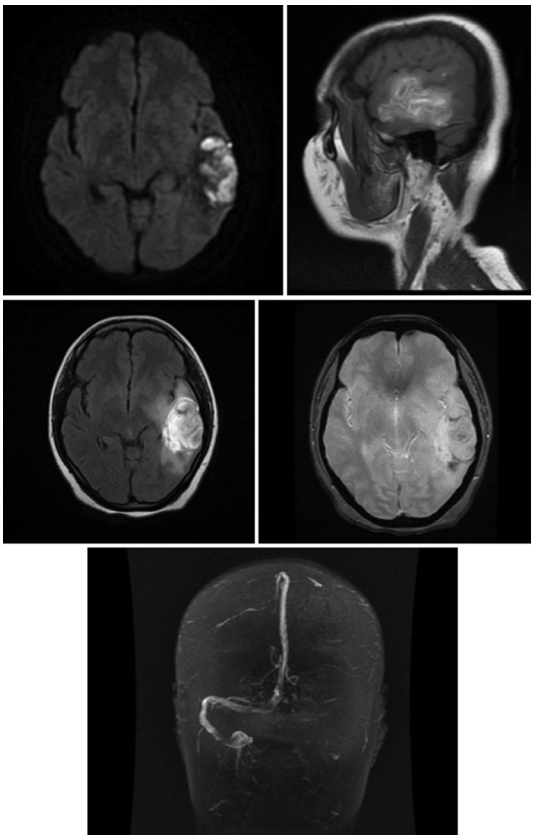
### Case 2

T1, T2, FLAIR, GRE, T1 SAGGITAL, DWI, and MRV

T1, T2, and FLAIR hyperintense lesion showing restriction on DWI and blooming areas on GRE in the left temporoparietal convexity noted.

MRV shows the left transverse and sigmoid sinus thrombosis.

Hemorragic infarct with vasogenic edema.



## REFERENCES


1. Benamer HT, Bone I. Cerebral venous thrombosis: Anticoagulants or thrombolytic therapy. J Neurol Neurosurg Psychiatry 2000;69:427-30.
2. Rother J, Waggie K, Van Bruggen N, De crispigny AJ, Moseley ME. Experimental cerebral venous thrombosis: Evaluation using magnetic resonance imaging. J Cereb Blood Flow Metab 1996;16:1353-61.
3. Wagner E, Traystman R. Effects of cerebral venous and cerebrospinal fluid pressure on cerebral flow. In: Auer L, Low F, editors. The Cerebral Veins. New York: Springer; 1983. p. 223-30.
4. Gotoh M, Ohmoto T, Kuyama H. Experimental study of venous circulatory disturbance by dural sinus occlusion. Acta Neurochir 1993;124:120-6.
5. Kurokawa Y, Hashi K, Okuyama T, Uede T. Regional venous ischemia in cerebral venous hypertension due to embolic occlusion of superior sagittal sinus in the rat. Surg Neurol 1990;34:390-5.
6. Garcia J. Thrombosis of cerebral veins and sinuses: Brain parenchymal effects. In: Einhaupl K, Kempski O, Baethmann A, editors. Cerebral Sinus Thrombosis. New York: Plenum Press; 1990. p. 27-37.
7. Corvol JC, Oppenheim C, Manaï R, Logak M, Dormont D, Samson Y, *et al.* Diffusion-weighted magnetic resonance imaging in a case of cerebral venous thrombosis. Stroke 1998;29:2649-52.
8. Manzione J, Newman GC, Shapiro A, Santo-Ocampo R. Diffusion-and perfusion-weighted MR imagings of dural sinus thrombosis. AJNR Am J Neuroradiol 2000;21:68-73.
9. Keller E, Flacke S, Urbach H, Schild HH. Diffusion-and perfusion-weighted magnetic resonance imaging in deep cerebral venous thrombosis. Stroke 1999;30:1144-6.
10. Ducreux D, Oppenheim C, Vandamme X, Dormont D, Samson Y, Rancurel G, *et al.* Diffusion-weighted imaging patterns of brain damage associated with cerebral venous thrombosis. AJNR Am J Neuroradiol 2001;22:261-8.
11. Forbes KP, Pipe JG, Heiserman JE. Evidence for cytotoxic edema in the pathogenesis of cerebral venous infarction. AJNR Am J Neuroradiol 2001;22:450-5.
12. Van Gijn J. Cerebral venous thrombosis: Pathogenesis, presentation and prognosis. J R Soc Med 2000;93:230-3.
13. Hermier M, Nighoghossian N. Contribution of susceptibility-weighted imaging to acute stroke assessment. Stroke 2004;35:1989-94.
14. Favrole P, Guichar JM, Crassard I, Bousser MG, Chabriat H. Diffusion weighted imaging of intravascular clots in cerebral venous thrombosis. Stroke 2004;35:199-203.
15. Soria C, Soria J, Mirshahi M, Desvignes P, Bonnet P, Caen JP. Importance of the structure of the clot in thrombolysis. Ann Biol Clin 1987;45:207-11.
16. Atlas SW, Dubois P, Singer MB, Lu D. Diffusion measurement in intracranial hematoma: Implications of MR imaging in acute stroke. Am J Neuroradiol 2000;21:1190-4.
17. Ferro JM, Lopes MG, Rosas MJ, Femo MA, Fontes J. Long-term prognosis of cerebral vein and dural sinus thrombosis. Results of the venoport study. Cerebrovasc Dis 2002;13:272.
18. Carrol JD, Lead D, Lee HA. Cerebral thrombo phlebitis in pregnancy and puerperium. QJ Med 1966;35:347-8.
19. Srinivasan K, Natarajan M. Cerebral venous and arterial thrombosois in pregnancy and puerperium. Neurol India 1974;22:131-40.
20. Nagaraja D, Taly AB. Puerperalcerebralsino venous thrombosis. Prog Clin Neurosci 1986;16:165-177.
21. Khaladkar SM, Thakkar DK, Thakkar DK, Shrotri H, Kulkarni VM. Cerebral venous sinus thrombosis on MRI: A case series analysis. Med J DY Patil Univ 2014;7:296-303.
22. Greiner FG, Takhtani D. Neuroradiology case of the day. Superior sagittal sinus thrombosis and infarcts. Radiographics 1999;19:1098-101.


**How to cite this article:** Ahmed N, Kumar AP, Chouhan S, Ismail S, Afreen U. Role of Magnetic Resonance Venography, Diffusion-Weighted Imaging, and Gradient-Recalled Echo in Cerebral Sinovenous Thrombosis. Int J Sci Stud 2018;6(5):1-5.

**Source of Support:** Nil, **Conflict of Interest:** None declared.