

## ORIGINAL RESEARCH

### **Magnetic resonance venography evaluation in cerebral venous thrombosis – A retrospective study**

**<sup>1</sup>Dr Nipa Hathila, <sup>2</sup>Dr Harish Chandra Chaturvedi, <sup>3</sup>Dr. Shekhar Karnawat,  
<sup>4</sup>Dr. Shrinidhi Kulkarni, <sup>5</sup>Dr. Kanaram Yadav**

<sup>1,2</sup>Associate Professor, <sup>3</sup>Professor, <sup>4,5</sup>2nd year Resident, Department of Radiology, Pacific Medical College and Hospital, Bhilon Ka Bedla, Udaipur, Rajasthan, India

#### **Correspondence:**

Dr Nipa Hathila

Associate Professor, Department of Radiology, Pacific Medical College and Hospital, Bhilon Ka Bedla, Udaipur, Rajasthan, India

**Email:**[nipahathila@yahoo.com](mailto:nipahathila@yahoo.com)

#### **ABSTRACT**

**Introduction:** Cerebral venous thrombosis is a relatively uncommon disorder which has an estimated annual incidence between two to seven cases per million in the general population.<sup>1</sup> The incidence was likely underestimated before the advent of prompt non-invasive imaging methods. It is estimated that five to eight cases per year might be seen at a referral centre. Cerebral venous thrombosis or occlusion by extrinsic compression that eventually progresses to a complete occlusion is an elusive diagnosis because of its non-specific presentation and its numerous predisposing causes which can precipitate the condition. It often affects young and middle-aged patients which more commonly involved in women. It is an uncommon cause of cerebral infarction relative to arterial disease which is an important consideration because of its potential morbidity. The imaging characteristics of CVT that can be observed through MRI include: (1) brain parenchymal imaging that appears in the form of non-specific lesions, such as intracerebral hemorrhages or infarcts, edema, isolated or associated with infarcts or hemorrhages, and it can even be considered normal in about 30% of patients.<sup>9</sup> MRV features include non-visualization of the arterial & venous vessels (i.e., no flow), flow defect and presence of collaterals at the site of occlusion.

**Methodology:** Data were collected from the available clinical records in the Emergency department were used. Pre hospitalisation records, inpatient records with discharge summary and any surgical records available over the period of two years from 2017 to 2019 were evaluated. This retrospective study is carried out in a population of 122 of the 130 patients were known to be diagnosed or suspected CVO subjected to high field MRI of the brain by using closed MRI unit (Siemens Magnetom Essenza 1.5T with 8 channel receiving). Statistical analysis was done by comparing the numbers and percentages for descriptive purposes.

**Results:** This study included 130 patients with age range from 40 days to 84 years old as briefed in (Table 1) and with the mean age of 33.50 years. Eighty-seven (66.7%) were females and forty-three patients (33.3%) were males. The earliest clinical presentation in this study was headache followed by convulsion, disturbed consciousness, coma, weakness of one limb, blurring of vision and papilledema which were the most common clinical symptoms. The predisposing factors for CVO were post-partum complications

(thirty-five patients – 26.7%), followed by dehydration and post-surgery complications, (both included twenty-six patients – 20%); however, there was no proper predisposing factor in forty-three patients (33.3%). The most common cause of cerebral venous occlusion in this study was venous thrombosis, was detected in 113 patients (86.7%) followed by extrinsic compression by tumours (meningioma), that was detected in seventeen patients (13.3%). MRI features in our study were observed in the form of brain parenchymal changes and abnormal signals in thrombosed veins and sinuses in both T1 and T2, in addition to blooming of the vessels. Parenchymal changes that were observed in this study were due to thrombosis in 105 patients and tumor in sixteen patients. The commonest MRI findings were replacement of the signal void of sinuses or veins by altered signal intensity. It showed three patterns, the first was hyper intense in both T1 and T2 in sixty patients (46.2%) (i.e. late subacute stage). In twenty patients (15.4%), it was hyperintense in T1 and iso to hypo intense in T2 (i.e. early subacute stage) while in thirty-five patients (26.7%), it was hypo intense in both T1 and T2 (i.e. chronic stage). MRV successfully diagnosed all cases. The most common non-visualized sinus in this study was Superior Sagittal Sinus that was noted in 86.7%, followed by transverse sinus 73.3%, sigmoid sinus 40% and straight sinus 33.3%. There was more than one occluded sinus could be found in some patients. **Conclusion: Cerebral venous thrombosis is a relatively uncommon but serious neurologic condition. Imaging plays a unique role in diagnosis. Prompt and appropriate medical therapy is important because brain parenchymal alterations and formation of venous thrombus are potentially reversible. MR imaging, MR venography, contrast-enhanced MR venography, and CT venography are the most useful techniques for diagnosis of this condition. Knowledge of normal venous variations and potential pitfalls related to image interpretation are important for arriving an accurate diagnosis.**

**Keywords: MRV, thrombosis, imaging**

## **INTRODUCTION**

Cerebral venous thrombosis is a relatively uncommon disorder which has an estimated annual incidence between two to seven cases per million in the general population.<sup>1</sup> The incidence was likely underestimated before the advent of prompt non-invasive imaging methods. It is estimated that five to eight cases per year might be seen at a referral centre. Accurate and prompt diagnosis of cerebral venous thrombosis is very critical, because timely and appropriate therapy can revert the disease process and significantly reduce the risk of acute complications and long-term sequelae.<sup>2</sup>

Cerebral venous thrombosis or occlusion by extrinsic compression that eventually progresses to a complete occlusion is an elusive diagnosis because of its non-specific presentation and its numerous predisposing causes which can precipitate the condition.<sup>3</sup> It often affects young and middle-aged patients which more commonly involved in women. It is an uncommon cause of cerebral infarction relative to arterial disease which is an important consideration because of its potential morbidity.<sup>4</sup> The various common causes and risk factors associated with CVT are due to: genetic prothrombotic conditions: antithrombin deficiency, protein C and protein S deficiency. Acquired prothrombotic states that can precipitate this condition are: nephrotic syndrome, pregnancy, and puerperium.<sup>5</sup> Infections like otitis, mastoiditis, sinusitis and meningitis. Collagen-vascular diseases such as systemic lupus erythematosus, sarcoidosis and Behcet syndrome.<sup>6</sup> Hematologic conditions like polycythemia, leukemia, anemia and sickle cell disease. Drugs which can cause CVT are oral contraceptives and tamoxifen.<sup>7</sup> Mechanical causes: trauma and neurosurgical procedures and miscellaneous causes: dehydration and cancer.<sup>8</sup>

The imaging characteristics of CVT that can be observed through MRI include: (1) brain parenchymal imaging that appears in the form of non-specific lesions, such as intracerebral hemorrhages or infarcts, edema, isolated or associated with infarcts or hemorrhages, and it can even be considered normal in about 30% of patients.<sup>9</sup> MRV features include non-visualization of the arterial & venous vessels (i.e., no flow), flow defect and presence of collaterals at the site of occlusion.<sup>10</sup>

## METHODOLOGY

Data were collected from the available clinical records in the Emergency department were used. Pre hospitalisation records, inpatient records with discharge summary and any surgical records available over the period of two years from 2017 to 2019 were evaluated. This retrospective study is carried out in a population of 122 of the 130 patients were known to be diagnosed or suspected CVO subjected to high field MRI of the brain by using closed MRI unit (Siemens Magnetom Essenza 1.5T with 8 channel receiving). Statistical analysis was done by comparing the numbers and percentages for descriptive purposes.

Protocols that were followed throughout the procedure are. T1W1, T2W1 and flair sequences, sagittal T1W1 sequence, coronal T2W1 sequence and MRI for all patients using 3-D phase contrast technique (3D-PC).

Parameters,

Closed MRI (1.5T)	
Slice thickness – 1.2mm	
Interslice gap- 1mm	
Field of view – 240 mm <sup>2</sup>	
Rectangular Field of view-71.9%	
T1W sequence, Repetition time (TR) – 450 ms	
Echo time (TE) – 8.9 ms	
T2W sequence, TR – 3200 ms	
TE – 86 ms	
Flair sequence, TR – 9000 ms	
TE – 86 ms	
MRV,	TR – 79.6 ms
	TE – 10.3 ms
	FOV – 240 mm <sup>2</sup>
	Velocity – 15 cm/s

## RESULTS

This study included 130 patients with age range from 40 days to 84 years old as briefed in (Table 1) and with the mean age` of 33.50 years. Eighty-seven patients (66.7%) were females and forty three patients (33.3%) were males. The earliest clinical presentation in this study was headache followed by convulsion, disturbed consciousness, coma, weakness of one limb, blurring of vision and papilledema which were the most common clinical symptoms. The predisposing factors for CVO were post-partum complications (35 patients – 26.7%), followed by dehydration and post-surgery complications, (both included twenty-six – 20%); however, there was no proper predisposing factor in forty-three (33.3%). The most common cause of cerebral venous occlusion in this study was venous thrombosis, was detected in 113 patients (86.7%) followed by extrinsic compression by tumours (meningioma), that was detected in seventeen patients (13.3%) (Table 2). MRI features in our study were observed in the form of brain parenchymal changes and abnormal signals in thrombosed veins and sinuses in both T1 and T2, in addition to blooming of the vessels. Parenchymal changes that

were observed in this study were due to thrombosis in 105 patients and tumor in sixteen patients. There was one patient of thrombosed cases reported no parenchymal changes. These changes were seen in the form of non-hemorrhagic infarction in fifty-two patients (40%), hemorrhagic infarction in thirty-five patients (26.7%) and edema in sixty-one patients (46.7%) (Table 3). The commonest MRI findings were replacement of the signal void of sinuses or veins by altered signal intensity. It showed three patterns, the first was hyperintense in both T1 and T2 in sixty patients (46.2%) (i.e. late subacute stage). In twenty patients (15.4%), it was hyperintense in T1 and iso to hypo intense in T2 (i.e. early subacute stage) while in thirty-five patients (26.7%), it was hypo intense in both T1 and T2 (i.e. chronic stage). Finally, there was only one patient with equivocal signal in both T1 and T2 (Table 4) but was diagnosed by MRV. The main MRV findings in our study were non-visualization of occluded veins or sinuses due to absence of signal, flow defect and presence of collaterals that may form in the dura that surrounds the occluded sinus or vessels. These collaterals were observed in thirty patients in this study (23.1%). MRV successfully diagnosed all cases. The most common non-visualized sinus in this study was Superior Sagittal Sinus that was noted in 86.7%, followed by transverse sinus 73.3%, sigmoid sinus 40% and straight sinus 33.3%. There was more than one occluded sinus could be found in some patients. Both cortical and deep veins could not be visualized in 33.3% and torcular herophili was included in 13.3%.

**Table – 1: Distribution of patients according to age and sex**

Age in years	Male		Female		Total	
	N	%	N	%	N	%
<1 year	17	13.3	9	6.7	26	19
1 - <10	-	-	-	-	-	-
10 - <20	-	-	-	-	-	-
20 - <30	-	-	17	13.3	17	13.3
30 - <40	9	6.8	17	13.3	26	20
40 - <50	-	-	26	20	26	21
50 - <60	9	6.7	18	13.3	27	20
60 – more	8	6.7	-	-	8	6.7
Total	43	33.5	87	66.7	130	100

**Table – 2: Distribution of patients according to the causes of cerebral venous occlusion (CVO).**

Cause	No.	%
Occlusion by thrombosis	106	81.2
Occlusion by tumour compression	24	18.7

**Table – 3: Distribution of patients according to MR appearance of parenchymal changes.**

MR appearance of parenchymal findings	No. of patients	%
Non – hemorrhagic infarction	52	40
Hemorrhagic infarction	35	26.7
Edema	43	30.2

**Table – 4: Distribution of patients according to the MRI appearance of signal changes in dural sinuses and veins.**

Stage of thrombus	Intensity	PT No.	Intensity	PT No.
Subacute stage	Hyper	80	Hypo	20
Early subacute			Hyper	60

Late subacute Chronic stage	Hypo	35	Hypo	35
--------------------------------	------	----	------	----

## DISCUSSION

The diagnosis of CVT is tedious since its clinical manifestations are non-specific and may be subtle sometimes. Several factors that combine to create an extremely elusive diagnosis of CVT: wide array of clinical presentations, many predisposing factors and the fact that individuals of all age groups are equally affected.<sup>11</sup> CVT may mimic various other medical conditions as arterial stroke, tumor, encephalitis, abscess and idiopathic intracranial hypertension (pseudo tumor cerebri).<sup>12</sup> So it is a diagnosis of exclusion and imaging investigations. Although most different modalities as DSA, CT, CTV and others help in diagnosis of cerebral venous occlusion (CVO) but each one has specific side effects and some have few normal findings.<sup>13</sup>

These findings agreed with similar literature reported that the most patients with this disease present in the neonates and young–middle aged patients.<sup>4</sup> Other literature reported that it is the most common in the third decade while it may occur in all age groups.<sup>14</sup> Eleven patients in the current study were females (68.7%) and five patients were males (33.3%). This correlates with *Einha`upl et al*<sup>14</sup> that reported with 75% of patients were females and had been suggested that the use of oral contraceptives in women is behind the difference between the sexes. According to patients' complaint, they were presented with CVT or cerebral venous compression; the current study had shown that headache was the most frequent and often the earliest symptom noted in 10 patients (66.7%). This was in favour with previous studies of *Ameri and Bousser*[15] who had reported that 75% of the 110 cases complained of headache. The most common predisposing factor was puerperium and this is in concordance with *Nagaraj et al*<sup>16</sup> who had found that 200 out of 230 cases (86%) of CVT, seen over eight years, were puerperal in nature. The commonest cause of cerebral venous occlusion in the current study was identified to be intraluminal thrombus, it occurred in 13 patients (86.7%) and this agreed with *Bousser et al*<sup>16</sup> who concluded that all patients in his study of 110 cases had cerebral venous occlusion due to thrombosis.<sup>17</sup> Occlusion by extrinsic compression was seen in two patients (13.3%) and was due to meningioma and this is in different figures obtained from *Jeffrey et al*<sup>18</sup> who reported in his study that compression or invasion of cerebral sinuses from dural or calvarial metastases was the major cause in those patients with cerebral sinuses occlusion due to solid tumours. Edema was the most common brain parenchymal change found in the present study which is seen in (46.7%) of cases but *Simonds et al*<sup>19</sup> observed focal edema in 25% of cases, followed by non-hemorrhagic infarction in (40%) and hemorrhagic infarction found in (26.7%). *Nagaraj et al*<sup>16</sup> found hemorrhagic infarction in (40.9%) and non-hemorrhagic infarction in (51.6%). Eight patients (61.5%) in our study presented with subacute thrombus. Chronic thrombus was found in 4 cases (26.7%) but one patient had equivocal signal. No cases of acute thrombus could be detected. According to some estimates, 10–30% of cases of sinus thrombosis developed the thrombus at initial presentation or imaging examination is in the acute stage of formation.<sup>19</sup> Subacute thrombus stage has been found in 55% of patients and this almost agreed with our study.<sup>20,21</sup> and chronic thrombosis was found in 15% of patients.<sup>20</sup> In this study, MRV was done in all 16 patients using phase contrast technique in the axial plane with coronal reconstruction. MRV successfully diagnosed occlusion in all the 16 cases. It was diagnosed by absence of signal intensity with consequent non-visualization of occluded sinuses or veins in all patients, flow defect in one patient and three patients were presented with collaterals. The superior sagittal sinus was most commonly involved accounting for (86.7%) and this agreed with *Ameri and Bousser*<sup>15</sup> who reported in their study of 110 cases that SSS accounts for (72%). We concluded finally that MRI and MRV are valuable imaging modalities for the

diagnosis of CVO especially as the clinical presentation and CT findings are usually non-specific in most of the cases. Therefore, the radiologist in the emergency department should be familiar with the imaging findings of CVO or CVT, and if it is suspected on CT, MRI and phase contrast MRV should be recommended as they are safer, effective, non-invasive, non-ionizing and highly accurate modalities. Also phase contrast MRV has the ability to differentiate thrombosed sinuses from its normal variants and other pitfalls.<sup>19</sup>

## CONCLUSION

Cerebral venous thrombosis is a relatively uncommon but serious neurologic condition. Imaging plays a unique role in diagnosis. Prompt and appropriate medical therapy is important because brain parenchymal alterations and formation of venous thrombus are potentially reversible. MR imaging, MR venography, contrast-enhanced MR venography, and CT venography are the most useful techniques for diagnosis of this condition. Knowledge of normal venous variations and potential pitfalls related to image interpretation are important for arriving an accurate diagnosis.

## REFERENCES

1. Stam J. Cerebral venous and sinus thrombosis: incidence and causes in ischemic stroke. *Adv Neurol* 2003;92:225–232.
2. Masuhr F, Mehraein S, Einhaupl K. Cerebral venous and sinus thrombosis. *J Neurol* 2004;251: 11–23.
3. Bousser MG. Cerebral venous thrombosis: nothing, heparin or local thrombolysis? *Stroke* 1999;30:481–3.
4. Ferro JM, Lopes MG, Rosas MJ, et al. Long-term prognosis of cerebral vein and dural sinus thrombosis. Results of the venoport study. *Cerebrovasc Dis* 2002;13:272–8.
5. Ferro JM, Canhao P, Stam J, Bousser MG, Barinagarrementeria F. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke* 2004;35:664–70.
6. DeVeber G, Andrew M. Cerebral sino-venous thrombosis in children. *N Engl J Med* 2001;345:417–23.
7. Ennaifer R, Moussa A, Mouelhi L, et al. Cerebral venous sinus thrombosis as presenting feature of ulcerative colitis. *Acta Gastroenterol Belg* 2009;72:350–3.
8. Schievink WI, Maya MM. Cerebral venous thrombosis in spontaneous intracranial hypotension. *Headache* 2008;48:1511–9.
9. Lanska DJ, Kryscio RJ. Risk factors for peripartum and postpartum stroke and intracranial venous thrombosis. *Stroke* 2000;31:1274–82.
10. Van Buchem MA, Liauw L, Split A, et al. MR angiography of the intracranial venous system. *Radiology* 2000;214:678–82.
11. Khealani BA, Wasay M, Saadah M, et al. Cerebral venous thrombosis: a descriptive multicenter study of patients in Pakistan and Middle East. *Stroke* 2008;39:2707–11.
12. Bousser MG. Cerebral venous thrombosis: diagnosis and management. *Eur J Neurol* 2000;247:252–8.
13. Duddalwar VA. Multislice CT angiography: a practical guide to CT angiography in vascular imaging and intervention. *BJR* 2004;77:27–38.
14. Einhaupl K, Bousser MG, de Bruijn SF, et al. EFNS guideline on the treatment of cerebral venous and sinus thrombosis. *Eur J Neurol* 2006;6:553–9.
15. Ameri A, Bousser MG. Cerebral venous thrombosis. *Neurol Clin* 1992;10:87–111.
16. Nagaraj D, Haridas T, Taly AB, Veerendrakumar M, Subbukrishna DK. Puerperal cerebral venous thrombosis: therapeutic benefit of low dose heparin. *Neurol India* 1999;47:43–6.

17. Bousser MG, Barnett HJM. Cerebral venous thrombosis. In: Barnett HJM, Mohr JP, Stein BM, Yatsu FM, editors. Stroke pathophysiology diagnosis and management. 2nd ed. New York, NY: Churchill Livingstone Inc.; 1992.
18. Jeffrey J, Lisa M, Raiseer DM, et al. Cerebral sinus thrombosis diagnosed by MRI and MRV venography in cancer patients. *Neurology* 2000;55:309.
19. Simonds GR, Truwit CL. Anatomy of the cerebral vasculature. *Neuroimaging Clin N Am* 1994;4:691–706.
20. Bergui M, Bradac G. Clinical picture of patients with cerebral venous thrombosis and patterns of dural sinus involvement. *Cerebrovasc Dis* 2003;16:211–6.
21. Macchi PJ, Grossman RI, Gomori JM, Goldberg HI, Zimmerman LT, Bilaniuk LT. High field MR imaging of cerebral venous thrombosis. *J Comput Assist Tomogr* 1986;10:10–5