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Role of MRV in Cerebral Venous Sinuses Pathology and Its Normal Variants

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Abstract: *Cerebral venous Sinus thrombosis (CVST) is a rare type of cerebrovascular disease that can occur at any age, including in neonates. CVST is a multifactorial condition with gender-related specific causes, with wide clinical presentations.Cerebral venous sinus thrombosis is a challenging condition because of its variability of clinical symptoms and signs. It is very often unrecognised at initial presentation. The widespread use of neuroimaging now allows for early diagnosis and has completely modified our knowledge on this disorder.*

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I. Introduction

Cerebral venous sinus thrombosis occurs when a blood clot forms in the brain's venous sinuses. This prevents blood from draining out of the brain. As a result, blood cells may break and leak blood into the brain tissues, forming a hemorrhage. This chain of events is part of a stroke that can occur in adults and children. It often affects young- to- middle- aged patients, and more commonly women, particularly in the age group of 20 to 35, due to pregnancy, puerperium and oral contraceptive use. Although recognized for more than 100 years, it has only in recent years come to be diagnosed frequently ante- mortem. This is partly due to greater awareness among physicians and neurologists, and partly to improved non- invasive imaging techniques.

Cerebral venous sinus thrombosis is a challenging condition because of its variability of clinical symptoms and signs. It is very often unrecognised at initial presentation. All age groups can be affected. Large sinuses such as the superior sagittal sinus are most frequently involved. Extensive collateral circulation within the cerebral venous system allows for a significant degree of compensation in the early stages of thrombus formation2.

The widespread use of neuroimaging now allows for early diagnosis and has completely modified our knowledge on this disorder. CVST is more common than previously thought and it is recognised as a non-septic disorder with a wide spectrum of clinical presentations, numerous causes, and usually a favourable outcome with a low mortality rate.

Numerous conditions can cause or predispose to CVST and often more than one cause will be found in an individual patient. A principal distinction can be made between infective and non-infective causes. Infective causes have declined and were responsible for only 8% of cases in recent series. Amongst the non-infective causes, systemic conditions such as connective tissue diseases, other granulomatous or inflammatory disorders and malignancies are most common. In young women, CVST occurs more frequently during the puerperium than during pregnancy. Oral contraceptives and various coagulation disorders have frequently been implicated recently.

Hereditary prothrombotic conditions such as Factor V Leiden (leading to increased resistance to activated protein C), deficiency of proteins C and S and antithrombin III as well as prothrombin gene mutations may account for 10–15% of cases of CVST. The risk of a carrier of any of these prothrombotic conditions developing CVST is increased by the coexistence of other predisposing factors2.

The most common causes and risk factors associated with CVST can be classified or grouped as follows:

- Genetic conditions such as antithrombin deficiency, protein C and protein S deficiency, Factor V Leiden, Antiphospholipid antibody.
- Acquired conditions includes nephritic syndrome, pregnancy and pueperium.
- Infections: otitis, mastoiditis, sinusitis and meningitis

- Collagen vascular disease: systemic lupus erythematosus , sarcoidosis and Behcet syndrome.
- Hematological condition: polycythemia, leukemia and sickle cell disease, paroxysomal nocturanal hemoglobinuria, TTP.
- Drugs: oral contraceptives and tamoxifen.
- Mechanical causes: trauma and neurosurgical procedure
- Miscellaneous causes: dehydration and cancer.
- Local causes: penetrating septic head injury, intracranial infection (abscess, emphyema, meningitis)
- Systemic causes: septicaemia, endocarditis, typhoid, tuberculosis.
- Non infective cause: Local head injury, neurosurgery, stroke and hemorrhage, space occupying lesions idiopathic intracranial hypertension.
- Other: Thyrotoxicosis, Electrocution, idiopathic.

II. Material and Methods

A prospective study of 100 cases was performed at our department of radio diagnosis. Patients with known CVST were subjected to (0.4 T) by using open MRI unit (Hitachi)medical system. The age group of the patient ranges between 11 – 70 years. Mean age of the patient was taken as 32.3 years.

Inclusion Criteria: Known cases of CVST and clinically suspected case of intra-cranial vascular lesions who were referred to radiology department for MRI brain with venography.

Exclusion Criteria: Patient with the history of metallic implant, foreign body, pacemaker, aneurysm clip, recently implanted prosthetic valve. Patient who are unstable to undergo MRI (patients on ventilator)
Claustrophobic patients

Total of 100 cases were studied. The patients advised intracranial vascular study for suspicion of involvement of intracranial venous system we subjected to MRI of brain with venography, O.4 T Whole body MR imager Hitachi MR system machine were used for the study. Multi sequential study in coronal, sagittal and axial sections were taken. These patient underwent MRV which included use of method/sequence 2D TOF. Intracranial venous system were studied after standard reconstruction were prepared using maximum intensity projection (MIP).

In our study the slice thickness for 2DTOF method was 3mm with distant factor 33%, 2D TOF technique was optimized by choosing a sagittal slice orientation titled slightly towards coronal and axial directions. Examination time for 2DTOF in our study was 7 minutes 20 seconds. Usually, it is desirable to set the slice thickness as small as possible, typically on order of 1.0 to 1.5mm to overcome limitation of 2DTOF method but that would have increased the time of scan to 10mm and therefore was not adopted in this study.

The appearance of various intracranial venous structures were assessed in the context of their signal intensity, uniformity and extent of dimension of the structures visible on three different MRV methods. Accordingly, values were assigned to each of them in different methods.

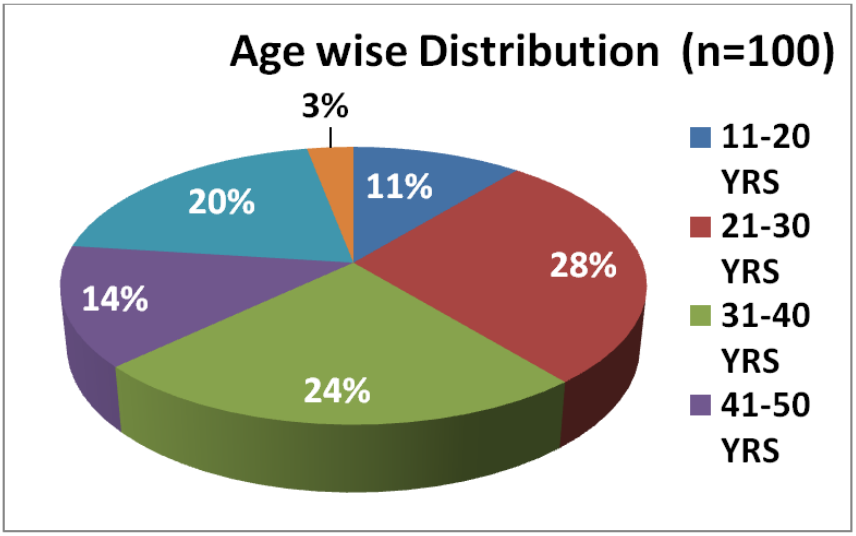
III. Result

The study includes 100 patients with CVST presenting a wide spectrum of symptoms. The data has been assimilated for a period of over one year and has been deduced as follows. Of the 100 patients 64% were female and remaining 36% were male. An agewise analysis indicated a higher no of cases in the age group of 21-30 years constituting 28% of the cases as shown in Table 1. And the same pattern continues to follow among both the sexes. Table 2 clearly shows that 28.1% among females and 27.8% among males were falling in the age group of 21-30 years with CVST. Whereas, only 3% were in the age group of 61-70 in both cases.

Table1: Age wise distribution

AGE YRS	NUMBER OF SUBJECTS	RELATIVE FREQUENCY
11-20 YRS	11	0.11
21-30 YRS	28	0.28
31-40 YRS	24	0.24
41-50 YRS	14	0.14
51-60 YRS	20	0.2
61-70 YRS	3	0.03

Chart 1: A pie chart representing distribution of CVST among different age group



Of the known causes of CVST, Puerpurium is the dominating cause for the formation of thrombosis with 18% of the reported cases falling in that category. However, in majority of cases the causes for the formation of CVST are largely unknown. In general, we noticed that the causes were acquired and non-infective.

Table 2: Distribution of patient depending on the cause of CVST

S.NO	CAUSES	NUMBER OF SUBJECTS	RELATIVE FREQUENCY
1	UNKNOWN	20	20.0%
2	TRAUMA	6	6.0%
3	PUERPURIUM	18	18.0%
4	DEHYDRATION	12	12.0%
5	ALCOHOL	13	13.0%
6	SEPSIS	10	10.0%
7	MASTODITIS	5	5.0%
8	MALIGNANCY	5	5.0%
9	MENINGIOMA	4	4.0%
10	HEMOTOLOGICAL DISORDER	7	7.0%
TOTAL		100	100.0%

Table 3: Distribution of patient with CVST depending on parenchymal changes

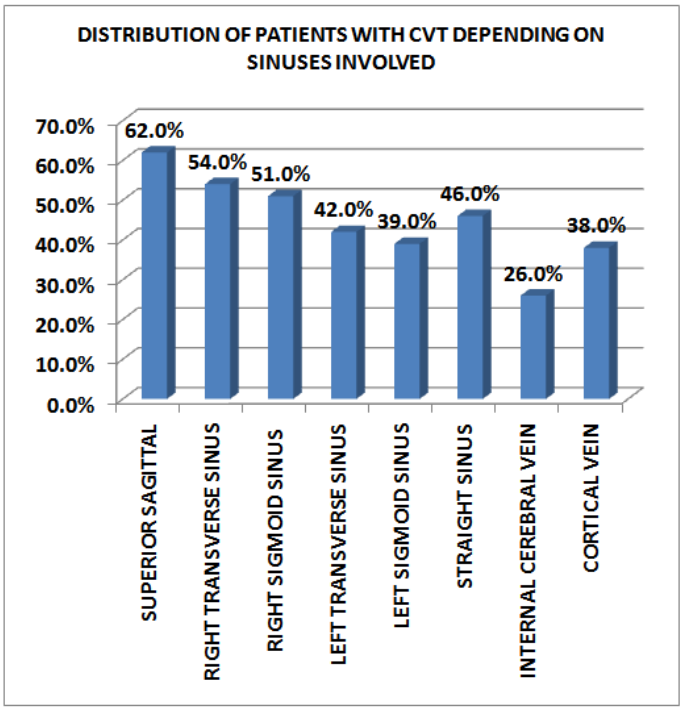
FOCAL PARENCHYMAL CHANGES	NUMBER OF CASES	PERCENTAGE
ABSENT	22	22.0%
HEMORRHAGIC INFARCT	53	53.0%
NON - HEMORRHAGIC INFARCT	25	25.0%
TOTAL	**100**	100.0%

53% of the cases were found to have hemorrhagic infarct and 25% of the cases had non-hemorrhagic infarct.

Table 4: Distribution of patients with CVST depending on sinuses involved

SINUSES INVOLVED	NO.OF PATIENTS	PERCENTAGE
SUPERIOR SAGITTAL SINUS	62	62.0%
RIGHT TRANSVERSE SINUS	54	54.0%
RIGHT SIGMOID SINUS	51	51.0%
LEFT TRANSVERSE SINUS	42	42.0%
LEFT SIGMOID SINUS	39	39.0%
STRAIGHT SINUS	46	46.0%
INTERNAL CEREBRAL VEIN	26	26.0%
CORTICAL VEIN	38	38.0%

Chart 2: Distribution of patients with CVST depending on sinuses involved

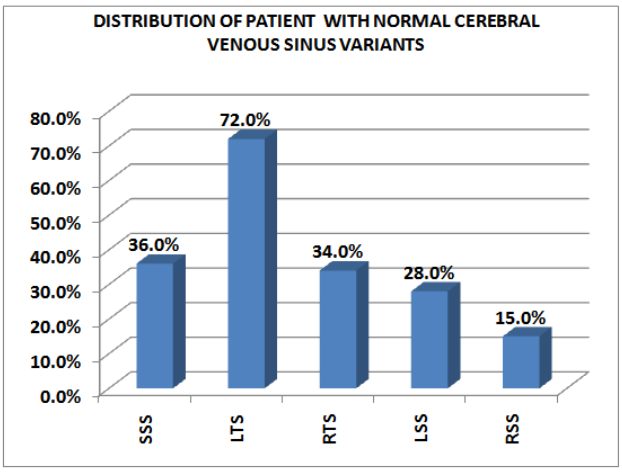


Though CVST is highly variable, in our study we encountered 62% of cases with superior sagittal sinus thrombosis. Right transverse sinus thrombosis was 54% followed by left transverse sinus thrombosis at 42%. And the cortical vein thrombosis was found to be in 38% of cases.

Table 5: Distribution of patient with normal cerebral venous sinus variants

SINUSES INVOLVED	NO.OF CASES WITH HYPOPLASTIC VENOUS SINUS	PERCENTAGE
SSS	36	36.0%
LTS	72	72.0%
RTS	34	34.0%
LSS	28	28.0%
RSS	15	15.0%

Chart 3: Bar diagram showing distribution of patients with normal cerebral venous sinus variants



Hypoplastic Left Transverse Sinus (LTS) was found to be more predominant 72% (Table 5) among other sinuses of the brain. Followed by Superior Sagittal Sinuses (SSS) 36% and Right Transverse Sinuses (RTS) 34%

Table 6: Detection of thrombus cases with MRV, T2* and T1 & T2 sequences

Method	Number of cases
Detected by MRV	99
Detected by T1, T2	94
Detected by T2*	98

Table 7: Detection of venous thrombosis with MRV & T2* and MRV, T1 & T2 sequence

Method	Using ROC curve	
	Area under the curve	P value
Detection by MRV and T1 & T2	0.975	0.103
Detection by MRV and T2*	0.995	0.09

IV. Discussion

The true incidence of CVST is unknown. Thrombosis of the cerebral veins and sinuses is a distinct cerebrovascular disorder that, unlike arterial stroke, most often affects young adults and children. The symptoms and clinical course are highly variable. During the past decade, increased awareness of the diagnosis, improved neuroimaging techniques, and more effective treatment have improved the prognosis. More than 80 percent of all patients now have a good neurologic outcome.

Although CVST can occur at any age (from neonates to the elderly), it is most commonly seen in young individuals. Nearly 80% of patients are younger than 50 years of age. The most common and earliest clinical presentation in this study was headache followed by convulsion, disturbed consciousness, weakness of one limb, blurring of vision and papilledema. Higher occurrence of CVST was observed between the age interval of 25 -35 years. Headache is the presenting symptom in 70–90% of cases. Focal deficits such as hemiparesis and hemisensory disturbance, seizures, impairment of level of consciousness and papilloedema occur in one-third to three-quarters of cases. An even less frequent presentation is a rapidly progressive illness with deepening coma, headache, nausea and pyramidal signs, due to extensive involvement of the deep cerebral veins.

In our study, 100 patients with Cerebral Venous Sinus Thrombosis were analysed with the help of MRI and MRV. It was generally noticed that the age group of 21-40 were comparatively more prone to CVST. And, it was also noticed that the incidence was more in case of female compared to male. This is due to the fact that women are exposed to predisposing factors like pregnancy, use of oral contraceptives and pueperium. In studies conducted on female population during the course of pregnancy and the puerperium they found that CVT may occur anytime, but the risk seems to be highest during the first 2 weeks of the puerperium.

The main MRV finding in our study were non-visualization of occluded veins or sinuses due to absent signal and flow defect. Hemorrhagic infarct is common among CVST. In our study, hemorrhagic infarct was noticed in 53% of cases whereas non-hemorrhagic infarct was noticed in 25% of cases.

Parenchymal Abnormalities: Parenchymal lesions are better depicted and more commonly identified at MR imaging than at CT. Parenchymal changes may be secondary to cytotoxic edema, vasogenic edema, or intracranial hemorrhage. Vasogenic and cytotoxic edema patterns may coexist.

In view of the variable nature of the parenchymal abnormalities that may occur in cerebral venous thrombosis, the use of the term venous infarct in reference to these lesions should be discouraged because that term implies irreversibility. In contrast with arterial ischemic states, many parenchymal abnormalities secondary to venous occlusion are reversible. Although parenchymal changes may occur in areas of the brain that are directly drained by the occluded venous sinus, in some patients the parenchymal changes may not closely correlate with the location of venous occlusion.

Unlike conventional MR images, Diffusion Weighted (DW) MR images can differentiate between vasogenic and cytotoxic edema. Cytotoxic edema is characterized by markedly decreased diffusion. Vasogenic edema, with increased interstitial water, demonstrates increased diffusion .

In a study by Mullins et al, they found that DWI, in combination with the clinical history (i.e., seizure), may be useful in differentiating parenchymal lesions that resolve from those that progress to permanent injury in the setting of acute CVT. In their retrospective cohort, lesions with elevated diffusion resolved, lesions with decreased diffusion resolved when the patient had seizures, and lesions with decreased diffusion in the absence of clinical seizure demonstrated abnormality on follow-up images. The ADC values were similar between the lesions with low ADC values that resolved and the lesions with low ADC values that persisted.

Isolated Cortical Venous Thrombosis: Isolated cortical venous thrombosis is relatively a rare entity; fewer than 20 cases have been reported in the imaging literature disease. Typical parenchymal findings are areas of focal cortical edema or hemorrhage, which may be nonspecific. The finding of an adjacent thrombosed venous structure, may be the most specific sign of this disorder.

On CT images, this finding has been referred to as the "cord sign"; on MR images, it has been called the "hyperintense vein sign ". Blooming artifacts within the thrombosed veins can be a very useful adjunct finding on GRE images.

False-Negative Cases On MRI: False-negative cases on MRI result from simulation of normal signal intensity (normal brain tissue or normal blood flow) by thrombosed dural sinuses.

When gadolinium-containing contrast agents are administered, thrombus is expected to result in a filling defect within the enhancing venous sinus . However, on occasion, contrast-enhanced T1-weighted images of DST in chronic stages can also show homogeneous contrast enhancement of the thrombosed dural sinus because of the presence of capillary channels in the thrombus, making it difficult to distinguish from a patent dural sinus

False-Positive Cases On MRI: False-positive cases on MRI are seen on spin-echo MR sequences when the signal intensity of a dural sinus simulates that of thrombus.

The primary causes are slow flow, in-plane flow, and entry-slice phenomena causing loss of the expected flow void within a dural sinus

False-Positive Cases on MR Venography: False-positive cases on MR venography. It is associated with 9% of false positive rate. Anatomic causes of false-positive findings can result from variations in the size of the dural sinuses, hypoplastic/ aplastic dural sinus and anatomic structures that produce filling defects ie septations and arachnoid granulations. Artifacts related to flow and those specific to various MR venography techniques can also cause signal loss that simulate thrombus.

False-Negative Cases On MR Venography: It is associated with 24% of false negative rate. Signal produced by hyperintense thrombus on unenhanced T1-weighted images can simulate flow within the dural sinus on TOF MR angiography and cause a false-negative finding.

Subarachnoid hemorrhage (SAH) associated with cerebral venous thrombosis (CVST) is rarely reported in the literature. The exact cause of SAH associated with CVST is unknown. Venous hemorrhagic infarct can be responsible for secondary rupture into subarachnoid spaces and cause SAH. Dural sinus thrombosis with secondary venous hypertension may lead to SAH into the subarachnoid space due to the rupture of fragile, thin-walled cortical vein. With spin-echo sequences, flowing blood typically produces a signal void; stationary blood on thrombus has been observed to produce higher signal intensity. This phenomenon is not entirely reliable, however, as a variety of flow-related artifacts can occasionally give rise to increased intraluminal flow signal that can mimic thrombus. Spin-echo MR images for detection of CVST yields variable results, showing some degree of dependence on the age of the thrombus.

CVST is a potentially fatal condition which if recognized and treated appropriately has excellent prognosis. MRI and MRV is the best diagnostic modality for CVST. The combination of hyperintense T1 and T2 signals from the sinus, associated with a defect seen on the MR venogram, in the appropriate clinical setting are diagnosed of CVST. MRI in conjunction with MRV is both sensitive and specific enough to provide the best noninvasive method of diagnosing cerebral venous thrombosis. The diagnosis usually can be made without intravenous contrast although contrast enhancement can aid in confirming the diagnosis.

Normal Variants: Normal variations in venous anatomy are common on MR Venography. The superior sagittal sinus usually drains in to the right transverse sinus. The transverse sinus which receives superior sagittal sinus is considered dominant. Hypoplastic Left Transverse Sinus (LTS) was found to be more predominant 72% among other sinuses of the brain. Followed by Superior Sagittal Sinuses (SSS) 36% and Right Transverse Sinuses (RTS) 34%. Hypoplasia and atresia of transverse sinuses occur frequently. Technical artifacts on MR Venography occur due to misregistration data and during reconstruction. These are also frequently seen in clinical MR imaging. Flow gaps in transverse sinuses Flow gaps can be misdiagnosed as cerebral venous thrombosis.

V. Conclusion

Cerebral venous thrombosis is a relatively uncommon but serious neurologic disorder. Imaging plays a primary role in diagnosis. Prompt and appropriate medical therapy is important because brain parenchymal alterations and venous thrombus formation are potentially reversible. MR imaging, TOF 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 achieving an accurate diagnosis. MRV in conjunction with MRI can accurately diagnose CVST without risk of invasive procedures and with no significant limitations.

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