Original Article

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Radiology Section

Multidetector Computed Tomography Venography versus Magnetic Resonance Venography in Cerebral Venous Thrombosis: A Cross-sectional Study

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ABSTRACT

Introduction: Cerebral Venous Thrombosis (CVT) is a relatively uncommon condition whose presentation is varied. The diagnostic imaging features can be subtle. The correct diagnosis of CVT relies on neuroimaging Non Contrast Computed Tomography (NCCT), CT Venography (CTV), Magnetic Resonance Imaging (MRI), MR Venography (MRV).

Aim: To compare 128 slice Multidetector Computed Tomography (MDCT) venography and MRV in CVT, taking MRV as gold standard.

Materials and Methods: This cross-sectional study was conducted in the Department of Radiodiagnosis, Gandhi Medical College, Secunderabad, Telangana, India, from October 2017 to September 2019. The study constituted of 56 patients, with clinical signs and symptoms of CVT who underwent MDCT venography and MRV. NCCT with MDCT venography was carried out on HITACHI 128 slice CT machine and MRI was performed in 1.5 tesla SIEMENS AVANTO systems. Imaging findings analysed on NCCT were hyperdense cord sign, delta sign, and multiple parenchymal hypodense lesions in bilateral parasagittal location, bilateral thalamic hypodensities, and isolated temporal bleed and on CTV signs like empty delta/empty triangle sign. Plain MRI was assessed for involved sinuses with thrombus showing loss of expected flow void with hyperintensity on T1-Weighted (T1W) and blooming on Gradient Recalled Echo (GRE). Parenchymal changes were assessed for presence of cytotoxic oedema, vasogenic oedema and haemorrhagic infarct. MRV was assessed for non visualisation of involved sinus or lack of normal flow related enhancement.

Results: Among the total 56 study subjects evaluated, 48 were diagnosed to have CVT on both CTV and MRV. On CTV, 147 sinuses were found to be involved, while MRV showed involvement of 142 sinuses. One case was found to be negative for thrombus on CTV but positive on Time of Flight (TOF) MRV. Both CTV and MRV were negative for CVT in seven patients. The sensitivity and specificity of NCCT in diagnosing CVT was found to be 60% and 87%, respectively.

Conclusion: The parenchymal changes in CVT were better evaluated on MRI. Venous abnormalities were better depicted in MRI as loss of flow voids in T1, T2 and with dark signal on GRE. Haemorrhagic bleeds were easily evaluated on MRI with 100% sensitivity of GRE sequence. CTV was easier to interpret, showed better and faster depiction of sinuses, with thin section reformatted images and showed higher spatial resolution compared to MRV.

Keywords: Cord sign, Delta sign, Diffusion weighted imaging, Neuroimaging, Venous abnormalities

INTRODUCTION

The Cerebral Venous Thrombosis (CVT) is a relatively uncommon condition that affects young to middle aged patients, commonly women, with an annual incidence of three to four cases per million and mortality rate of 8% [1]. CVT most commonly involves Superior Sagittal Sinus (SSS) followed by Transverse Sinus (TS), and in 30-40% of the patients more than one sinus is involved. Most of the patients present with non specific signs and symptoms, and the diagnostic imaging features can be subtle [2]. Early diagnosis and appropriate treatment are essential to prevent neurological deterioration. The accurate diagnosis of CVT relies on neuroimaging. Imaging modalities of choice in CVT are NCCT, CTV, conventional MRI and MRV. According to Indian guidelines for stroke management, patient suspected to be having stroke due to CVT should be investigated by MRI/MRV/CTV if CVT is not diagnosed by CT scan [3].

Plain CT can be normal in upto two thirds of patients and findings are often subtle and non specific in early stages [4]. MDCT scan time is less than a second, and is less susceptible to motion artefacts, so can easily be performed in unstable patients. MRI and MRV, although considered a modality of choice is not universally available and is subject to various artefacts. In a country like India, CTV is readily available and is an alternative diagnostic modality as good as MRI [4].

Plain CT shows non specific signs like venous infarcts, diffuse brain oedema and specific signs like dense sinus sign, and cord sign. The signs in favour of venous infarct are ill-defined multiple, subcortical hypodensities in non arterial territory, bilateral involvement of thalami or basal ganglia. Eccentric hypodensity at periphery of an intraparenchymal haemorrhage that is present soon after onset of neurological symptoms suggests that the bleeding developed in an area of brain oedema. Venous thrombi are seen as filling defects in CTV [2]. The direct MR sign of CVT is the lack of expected flow void on T1 and T2 sequences. In acute stage (0-5 days), there is absence of flow void and the thrombi appear isointense on T1 and hypointense on T2W images (confused with normal flow void) due to presence of deoxyhaemoglobin in intact red blood cells. In subacute stage (day 6-15), the thrombus becomes hyperintense, initially on T1W and little later on T2W images, due to presence of methaemoglobin. At the chronic stage (>15 days) the MRI signal pattern is variable [5].

On Diffusion Weighted Imaging (DWI), the clot can be seen directly as high signal intensity in the affected sinus in acute phase. DWI in venous infarct shows heterogeneous signal intensity with a normal or increased Apparent Diffusion Coefficient (ADC) corresponding mostly to vasogenic oedema [5].

Digital Subtraction Angiography (DSA) was once considered standard for CVT, but is invasive and associated with procedure related complications. Hence, it is occasionally reserved when reperfusion or thrombosuction is contemplated for therapy [6].

The primary aim of this study was to determine the reliability of CT in diagnosing CVT and compare 128 slice MDCT venography with MRV, using MRV as the gold standard. The secondary objective was to analyse the pattern of distribution of venous infarcts and parenchymal changes associated with CVT and appearance of thrombus on CT and MRI.

MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Radiodiagnosis in Gandhi Medical College, Secunderabad, from October 2017 to September 2019. The study enrolled 56 patients who underwent MDCT venography and MRV. Institutional Ethics Committee approval was obtained (Rc. No. IEC/GMC/2021) and informed consent was taken prior to the study.

Inclusion criteria: The patients with clinical suspicion of CVT like headache, vomiting, seizures, altered sensorium, papilledema, focal neurological deficits or had NCCT showing suspicious signs of CVT were included in the study.

Exclusion criteria: Patients with high renal parameters, metallic implants, claustrophobia, pregnancy, hypersensitivity to CT contrast agents and in whom CT is contraindicated due to any other reason were excluded from the study.

Study Procedure

The CTV was performed in patients with clinical suspicion of CVT and MRV was performed after two to three days. Most of the patients were conscious at the time of evaluation.

Non contrast computed tomography with CT venography: NCCT with CTV was carried out on the HITACHI 128 slice CT, at 120 kVp and 200 Milliampere seconds (mAs). The scan protocol consisted of slice thickness of 0.625 mm, Field of View (FOV) 220 mm, and reconstruction index 5.0 mm, spacing 0.5 mm using a pitch of 0.59 with smooth reconstruction filter, Window Width (WW)/ Window Level (WL) range between 450/150 for soft tissue, 200/50 for thin sections. The data was acquired by scanning from skull base to vertex in craniocaudal direction. CTV was performed with 80-100 mL of non ionic contrast at 4.0-4.5 mL/second with a saline chase of 30-35 mL at 3 mL/second. The imaging was done with a scan delay of 45 seconds.

Conventional MR with magnetic resonance venography: Conventional MR with MRV was carried out on 1.5 Tesla SIEMENS AVANTO systems. MR protocol included localiser, DWI, axial T2, Fluid-Attenuated Inversion Recovery (FLAIR), T1, GRE, sagT2 and 2D TOF MRV. 3D Phase Contrast (PC) MRV was also performed in some subjects. MRV with gadolinium was performed wherever the findings were equivocal. 2D TOF, 3D PC was planned on axial plane; angling the position blocks 10° to midline of the brain. Check the positioning block in the coronal plane and angle 10° to midline of the brain. This angulation was to reduce in-plane saturation effects. Saturation band was placed at the bottom of the block in the sagittal and coronal plane to avoid arterial signals. Slices were placed to cover the whole brain from temporal lobe to temporal lobe. Parameters included for 3D PC were Repetition Time (TR) 68-75, Time of Echo (TE) 8-9, flip angle-150, slice thickness-1 mm, Field of View (FOV)- 280 mm, phase- A>P and 2D TOF were TR- 28-35, TE5-8, flip angle- 60°, slice thickness- 2 mm, FOV- 250 mm, phase A>P. Post processing 3D MPR and Maximum Intensity Images (MIP) images were generated on both CTV and MRV.

Plain CT was evaluated for presence of parenchymal changes like multiple bilateral parasagittal hypodensities, bilateral thalamic hypodensities, isolated temporal haemorrhages, and direct sinus involvement like hyperdense cord sign and delta sign. CTV images were assessed for presence of empty delta/empty triangle sign seen as filling defects within sinus with a thin peripheral rim enhancement [2]. On MRI, evidence of parenchymal involvement like FLAIR/T2 hyperintensities and bright signal in DWI, ADC (vasogenic oedema), hypointense on ADC (cytotoxic oedema) was assessed. Blooming on GRE was suggestive of a haemorrhagic infarct. Direct evidence of sinus thrombosis was noted as a loss of expected flow void on T1W, T2W with hyperintensity on T1W which appeared bright on DWI and blooming on GRE. MRV was also assessed for non visualisation of involved sinus or lack of normal flow related enhancement [5].

The CTV and MRV images were assessed independently by two qualified radiologists, with an experience of 10 years and 3 years respectively. Both of them were trained in assessing venography images. All images were initially evaluated in conjunction with clinical data for the presence of CVT separately by each radiologist. In case of any discrepancy in findings, the images were evaluated together by both radiologists in conjunction with clinical data and arrived at diagnosis based on consensus. CTV and MRV were initially assessed on source images and later on Three Dimensional (3D) reconstructed Maximum Intensity Images (MIP) images.

STATISTICAL ANALYSIS

The MRI with MRV was taken as gold standard. Sensitivity, specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV), accuracy was calculated using Microsoft Excel and epi info software version 7.0. The data was analysed using Statistical Package for the Social Science (SPSS) software.

RESULTS

Among 56 patients, the most common age group was 21-40 years (64%) followed by 41-50 years (21%). Alcoholism was the most common risk factor (21%), while 9 (16%) of the patients did not have any known risk factor [Table/Fig-1].

Parameters		No. of patients (%)
Age (years) Mean age 34.6 (SD=11.27)	10-20	4 (7%)
	21-30	18 (32%)
	31-40	18 (32%)
	41-50	12 (21%)
	>50	4 (7%)
Gender	Male	33 (59%)
	Female	23 (41%)
Risk factors	Postpartum	5 (9%)
	Alcoholics	12 (21%)
	Hyperhomocystinaemia	2 (3.5%)
	Oral contraceptives	4 (7%)
	Mastoiditis	4 (7%)
	Human Immunodeficiency Virus (HIV)	2 (3.5%)
	Malignancy	2 (3.5%)
	Dehydration	5 (9%)
	Anaemia	5 (9%)
	Antithrombin 3, protein C and protein S	2 (3.5%)
	Other co-morbidities like Congestive cardiac failure, nephritic syndrome	4 (7%)
	No risk factor	9 (16%)

[Table/Fig-1]: Demographic profile in CVT.

Subacute presentation (48 hours to 1 month) was noted in 36 (64%) patients, followed by acute presentation (less than 48 hours) in 18 (32%) patients. Chronic presentation was noted only in two (4%) patients.

Most common symptom was headache 50 (89%), followed by vomiting 42 (75%); papilledema was the most common clinical sign at presentation, seen in 20 (37%) patients [Table/Fig-2].

Clinical features		No. of patients (%)
Symptoms	Headache	50 (89%)
	Seizures	24 (43%)
	Vomiting	42 (75%)
	Diplopia	20 (36%)
	Fever	15 (27%)
	Aphasia	23 (42%)
	Focal deficits	20 (37%)
	Altered sensorium	30 (53.5%)
Signs	Hemiparesis	20 (37%)
	Papilledema	20 (37%)
	Pallor	12 (22%)
	Cranial nerve involvement	18 (32.5%)
	Dysphasia	9 (17.5%)

[Table/Fig-2]: Clinical features in CVT.

The clinical presentation could be summarised in three main patterns, each of them simulating another neurological disease. The most frequent and homogeneous one was the progressive onset of signs of Intracranial Hypertension (ICH) (35/56) like headache, vomiting, diplopia, altered sensorium and papilledema. The second presentation was the sudden onset of focal deficits (20/56) simulating arterial strokes but with more frequent seizures. The third presentation simulated an abscess (15/56) with deficits and/or seizures with or without ICH. It is therefore clear that CVT had no single clinical presentation.

Among the 56 study subjects, 48 were diagnosed to have CVT on both CTV and MRV. On CTV, 147 sinuses were found to be involved, while MRV showed involvement of 142 sinuses. On CTV, the most common sinus involved was Transverse sinus 39 (69.6%) patients [Table/Fig-3].

Sinus/Vein		CTV	MRV
Superior sagittal sinus		18 (32%)	18 (32%)
Transverse sinus	Total	39 (69.6%)	40 (71.4%)
	Right	14 (25%)	15 (26.7%)
	Left	18 (32%)	18 (32%)
	Both	7 (12.5%)	7 (12.5%)
Sigmoid sinus	Total	34 (60.7%)	30 (53.5%)
	Right	16 (28.5%)	15 (26.7%)
	Left	14 (25%)	13 (23%)
	Both	4 (7%)	2 (3.5%)
Internal Jugular Vein (IJV)	Total	26 (46.4%)	27 (48%)
	Right	10 (17.8%)	11 (19.6%)
	Left	14 (25%)	14 (25%)
	Both	2 (3.5%)	2 (3.5%)
Vein of galen and deep veins		12 (21.4%)	12 (21.4%)
Straight sinus		14 (25%)	12 (21.4%)
Cortical veins		4 (7%)	3 (5.3%)
Total sinuses		147	142

[Table/Fig-3]: Frequency of thrombosis of major sinuses on CT Venography and 2D TOF MR Venography.

In NCCT, hyperdense sinus/cord sign was seen in 20 (35.7%) patients, while non haemorrhagic infarct was seen in 12 (21.4%) patients, and haemorrhagic infarcts in 22 (39.3%) patients [Table/Fig-4]. Out of 22 patients, 20 patients presented with parenchymal bleed of which 18 patients had isolated parenchymal bleed and two patients had parenchymal bleed with concurrent Subarachnoid Haemorrhage (SAH) and Subdural Haematoma (SDH). Two patients presented with isolated SAH.

Parietal-temporal lobe was the most common location of bleed, noted in 11 (20%) patients followed by frontal lobe in 9 (16%) patients. Incidence of superficial vein thrombosis on CTV was 44/56 (78.5%), deep vein thrombosis was 12/56 (21.4%) and cortical vein thrombosis was 4/56 (7%). In 22 patients, NCCT appeared normal, and further investigation was done based on clinical findings. Four patients with ear discharge had soft tissue density in mastoid air cells on CT [Table/Fig-1]. Two patients had hydrocephalus on CT.

In the present study, Hounsfield Unit (HU) threshold values were greater than 70 and venoarterial difference greater than 15 on NCCT was considered significant for CVT, which was later confirmed on CTV. The sensitivity and specificity of NCCT in diagnosing CVT was found to be 60% and 87% respectively. CTV had a specificity of 83-100% and sensitivity of 90-100%, depending on the sinuses involved [Table/Fig-5].

Arachnoid granulations were found in 10/56 patients most commonly (8/10) in the dominant TS, and in Superior Sagittal Sinus (SSS) in 2/10 patients. Persistent occipital sinus (2/56) and vein of galen malformation (1/56) were also seen. In 41/56 patients SSS directly drained into right TS which was the dominant sinus [Table/Fig-6].

On MRI, diffusion restriction in the sinuses was seen in 12/56 cases (21%). Diffusion restriction within the haemorrhagic infarct was also noted in 12/56 cases (21%). On MRI, incidence of cases with cytotoxic oedema in the present study was 12/56 (21%), whereas with vasogenic oedema was 36/56 (64.3%). FLAIR and T2 sequences depicted vasogenic oedema better in 36 patients, while T1 and GRE sequences were valuable in detecting haemorrhagic lesions in 28 and 31 patients respectively. Venous abnormalities were better detected on T1W and GRE as loss of flow void and blooming, respectively in 35 patients [Table/Fig-7].

The most common sinus involved in MR was TS (71.4%). Sensitivity of GRE in detecting venous abnormalities was 62.5% respectively. Phase contrast MRV was performed in 16 patients, out of which 14 patients had CVT, which was also confirmed on Two-Dimensional (2D) TOF MRV. In two cases of chronic CSVT, filling defect with loss of hyperintense signal was noted in PC MR in SSS and right TS while in 2D TOF MRV hyperintense signal with surface irregularities was noted. The loss of hyperintense signal was probably due to turbulence which affected the PC images. On CTV, partial filling of the sinuses with thin linear hypodense filling defect was noted suggesting of chronic CSVT with near total recanalisation.

Contrast Enhanced MRI (CEMR) was performed in six patients with equivocal status. On TOF two patients who showed filling defect in proximal 1/3rd of TS, were found to be hypoplastic in CEMR. Other findings were consistent with 2D TOF MRV.

CTV allowed direct visualisation of thrombus along with the extent of involvement. MIP images from both CTV and MRV were almost

Variables	No. of patients	Parenchymal abnormalities			Venous abnormalities		
		Normal (CT/MRI)	Non haemorrhagic infarct (CT/MRI)	Haemorrhagic infarct (CT/MRI)	Normal (CT/MR)	Cord sign (CT)	Loss of flow void (MR)
Superficial venous system thrombosis	38	18/15	5/5	15/18	30/18	8	20
Isolated deep venous thrombosis	3	-	3/1	0/2	0/0	3	3
Thrombosis of both superficial and deep venous system	15	4/4	4/0	7/11	6/3	9	12
Total	56	22/19	12/6	22/31	36/21	20	35

[Table/Fig-4]: Unenhanced CT and MRI findings in CVT patients in total 56 patients.

Sinus/Vein	CTV				
	Sensitivity	Specificity	PPV	NPV	Accuracy
Superior sagittal sinus	100	88.24	77.78	100	91.67
Right transverse sinus	90	100	100	92.31	95.45
Left transverse sinus	100	83.33	84.62	100	91.3
Right sigmoid sinus	100	89.47	85.71	100	93.55
Left sigmoid sinus	100	94.74	92.86	100	96.86
Straight sinus	100	100	100	100	100
Vein of galen	100	100	100	100	100

[Table/Fig-5]: Sensitivity, Specificity, PPV, NPV, and accuracy of CT venography when using MR venography as gold standard.

Anatomical variant	CTV	MRV
Hypoplastic transverse sinus	Right-4, left-18	Right-3, left-15
Hypoplastic sigmoid sinus	Right-3, left-13	Right-3, left-9
Persistent occipital sinus	2/56	2/56
Vein of Galen malformation	1/56	1/56
Arachnoid granulation	10/56	10/56
Superior sagittal sinus directly draining into right transverse sinus	41/56	41/56
Sigmoid diverticula	2/56	2/56

[Table/Fig-6]: Common anatomical variants seen on CTV and MR venography.

MRI sequence	Parenchymal abnormalities		Venous abnormalities
	Vasogenic	Haemorrhagic	
T2W	36/56 (64.3%)	27/56 (48%)	35/56 (62.5%)
FLAIR	36/56 (64.3%)	27/56 (48%)	36/56 (64.3%)
T1W	34/56 (61%)	28/56 (50%)	35/56 (62.5%)
GRE	-	31/56 (55%)	35/56 (62.5%)
DWI	-	12/56 (21%)	12/56 (21%)

[Table/Fig-7]: Conventional MRI findings in CVT in total 56 patients.

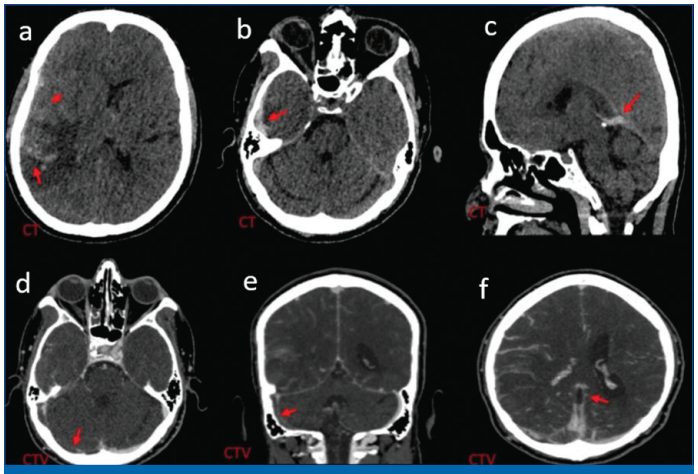
identical. When both CTV and MRV were interpreted together, CTV revealed all major cerebral veins and sinuses that are seen on MRV. However, CTV was more reliable than TOF in identifying basal vein of rosenthal, thalamostriate veins and Inferior Sagittal Sinus (ISS). When viewing PC MRV images, simultaneous review of T1W, T2W images to visualise the expected site of thrombus was done. Similarly, review of source image for 2D TOF was also done.

DISCUSSION

The CVT is characterised by thrombosis of intracranial veins and sinuses which results in parenchymal damage, following rise in intracranial pressure [7]. Radiological hallmark of this condition is thrombosis of intracranial sinuses and veins with/without haemorrhagic infarction and oedema with/without evidence of herniation.

It has been suggested that the incidence of CVT was higher in females and in the aged people, this was not confirmed in our present study, in which male:female ratio was 1.43. This data was consistent with previous Indian study by Aaron S et al., from South India and Anadure RK et al., which showed a higher proportion of CVT in males and in postpartum females [8,9]. High proportion of postpartum CVT patients were also observed by Cantu C and Barinagarrementeria F, from Mexico with similar socio-demographic characteristics and economic status of the patients as in India [10]. Alcohol was the major risk factor in 21% of patients in this study, consistent with study by Anadure RK et al., [9]. More than half (64%) of the patients were in the second and third decade of their age. The mean age of the patients was 34.6 years, similar to Indian study by Anadure RK et al., which showed a mean age of 34.4 years [9]. In the present study 5 (9%) of patients had anaemia, which was similar to study by Sharma N et al., where a higher frequency of anaemia was noted with CVT in pregnancy and puerperium [11]. Whether this is a reflection of high incidence of anaemia in Indian population particularly in pregnant females or anaemia is a real risk factor needs further evaluation. In a study done by Beri S et al., severe anaemia leading to CVT in infants was a proven risk factor [12]. No case of polycythemia was encountered in the present study. In 4 (7%) of cases, mastoiditis was an associated risk factor. As the middle ear structures and sigmoid, transverse sinuses are in contiguity, there is direct spread of inflammatory process leading to venous occlusion [13]. In 9 (16%) of cases, no cause could be found, however complete aetiological workup could not be completed. Headache with or without vomiting were the major clinical features noted at presentation. Similar findings were noted from earlier studies. Four major patterns of presentation have been described like Isolated ICH, focal neurological abnormalities, seizures and encephalopathy presenting in combination or isolation depending on extent and location of CVT [7].

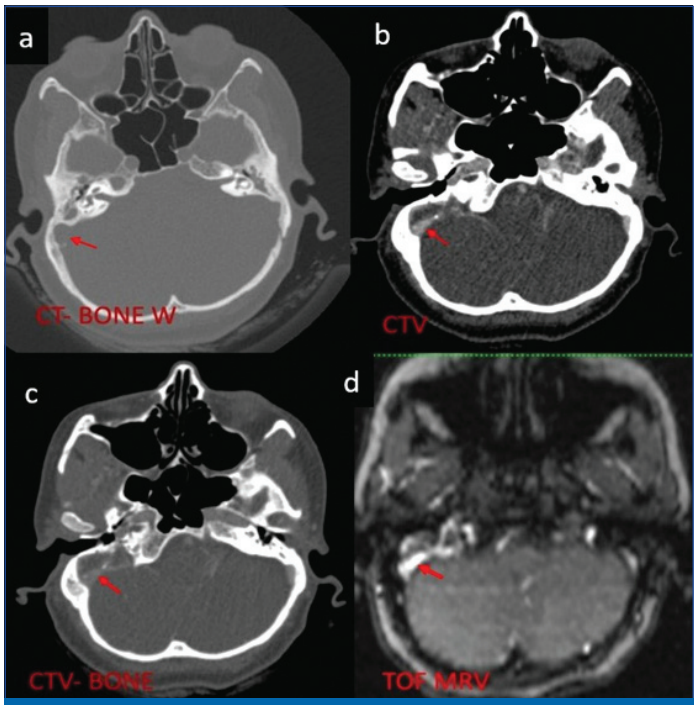
In this study, hyperdense sinus on NCCT was seen in (20/56) cases, all of which presented within three days of onset of symptoms. According to Besachio DA et al., NCCT threshold HU values of greater than 65 and venoarterial difference values greater than 15 were considered significant for CVT which is similar to findings in the present study [14]. Non haemorrhagic infarct or hypodensity was commonly bilateral and in high parasagittal location. Haemorrhagic infarcts which were seen with extensive oedema at the time of presentation and which were not seen in hypertensive locations like basal ganglia, pons, cerebellum were suspicious of venous infarcts. Two of our patients presented with SAH and SDH in addition to temporoparietal bleed while two had isolated SAH. In a study done by Akins PT et al., secondary SDH and secondary CVT following intracranial hypotension and preceding use of thrombolytic leading to iatrogenic SDH were some mechanisms proposed [Table/Fig-8] [15]. Bilateral thalamic hypodensity on NCCT, was attributed to deep CVT, confirmed on CTV.



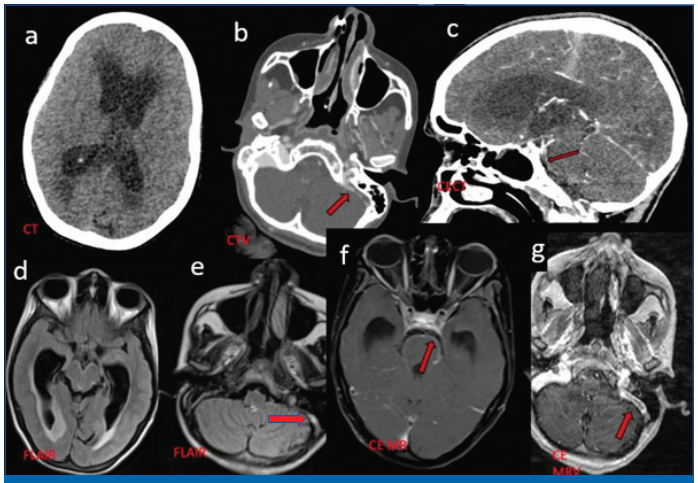
[Table/Fig-8]: A 36-year-old male presented with headache, seizures showing: (a) Plain CT axial showing bleed with oedema (arrow) in right temporal lobe; (b) Plain CT axial showing thin rim of SDH (arrow) in right middle cranial fossa; (c) Plain CT sagittal image showing hyper density/thrombus in vein of galen (arrow); (d, e, f) CTV in axial and coronal images showing filling defect in right transverse, sigmoid, straight sinuses (arrows)-s/o haemorrhagic infarct in right temporal lobe, SDH along right temporal convexity due to CVT involving right transverse, sigmoid, straight sinus, vein of galen. Diagnosis: Right transverse, sigmoid and straight sinus thrombosis with right temporal lobe haemorrhagic infarction with SDH along right temporal convexity.

The present series confirms the fact that isolated single sinus involvement was less common than multiple sinus involvement. In isolated sinus involvement, most frequently involved sinuses are SSS and TS. In the present study no case of isolated cortical CVT was encountered. Cortical veins were involved in 7% of patients, in which sinus involvement was also seen. In cases who presented with mastoiditis, involvement of ipsilateral TS and sigmoid sinus was noted [Table/Fig-9]. In patients who presented with chronic

CSVT a thin linear hypodense non enhancing, filling defect was noted in SSS. In one case of chronic CSVT we observed early filling of SSS and right TS due to formation of a dural AVF. In two cases who presented with signs of raised ICT and hydrocephalus showed basal cisternal enhancement indicating meningitis with coexisting CVT in TS, probably due to spread of infection leading to thrombosis [Table/Fig-10]. A similar case of CVST associated with Tuberculous (TB) meningitis along with proposed mechanisms was described by Verma R et al., [16]. In a study done by Khandelwal N et al., in 2006, CTV was found to have a specificity and sensitivity value of 75-100% depending on the sinus involved taking MRV as gold standard [17]. In the present study CTV was found to have specificity values of 83-100% and sensitivity values of 90-100% depending on sinuses involved [Table/Fig-5].

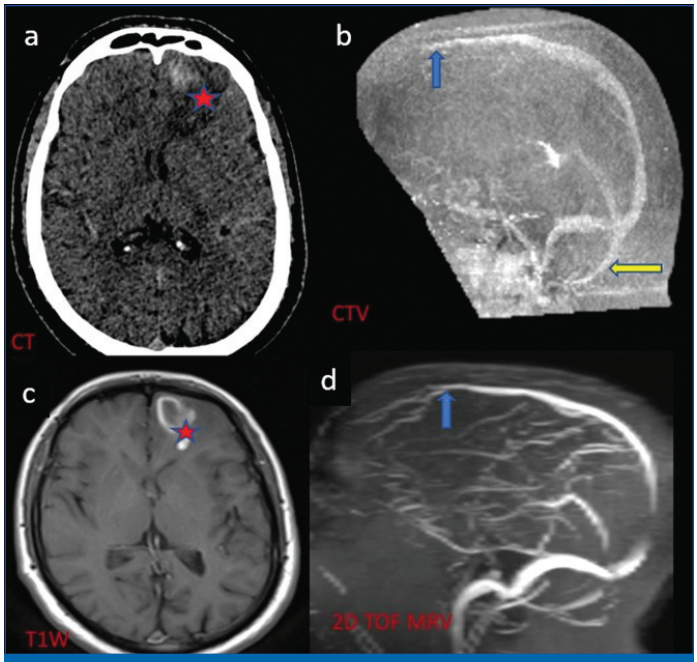


[Table/Fig-9]: A 43-year-old female presented with right ear discharge and pain showing: (a) Plain axial CT bone window showing diverticular out pouching (arrow) of right sigmoid sinus, with opacity in bilateral mastoid air cells; (b) (c) CTV axial images showing partial filling defect (arrow) in right sigmoid sinus; (d) TOF axial showing partial thrombus as isointense signal and patent lumen as hyperintensity (arrow) in right sigmoid sinus-suggestive of bilateral mastoiditis with right sigmoid sinus diverticulum and partial thrombus right sigmoid sinus.



[Table/Fig-10]: A 23-year-old female presented with fever, seizures and vomiting: (a) Plain axial CT showing dilatation of both lateral ventricles with periventricular hypodensity; (b) CTV axial image showing filling defect (arrow) along left sigmoid sinus; (c) CTV sagittal image showing prepontine cisternae enhancement (arrow); (d) FLAIR axial showing mild dilatation of both occipital horns with periventricular FLAIR hyperintensity; (e) FLAIR axial showing loss of flow void in left sigmoid sinus (arrow); (f) CEMR T1W axial image showing basal cisternae enhancement (arrow); (g) CEMR T1W axial showing filling defect (arrow) in left sigmoid sinus suggestive of Mild hydrocephalus with meningitis and left sigmoid sinus thrombosis. CSF culture showed presence of Mycobacterium tuberculosis.

Two cases were incidentally encountered of persistent occipital sinus, one who had chronic CVT of SSS and other case was normal [Table/Fig-11]. A case of congenital vein of galen malformation with persistent fascine sinus without CVT was incidentally noted. In chronic CVT, multiple flow voids accounting to Dural AVF was noted in a single patient, with no CVT at the time of imaging. Anatomical asymmetry of TS was the most commonly observed anatomical variant, seen in 40% of cases. In a study done by Goyal G et al., hypoplastic left TS was the most common variant, as also seen in the present study [18].



[Table/Fig-11]: A 38-year-old-male presented with headache showing: (a) Plain axial CT showing bleed with oedema (red star) in left frontal lobe; (b) CTV sagittal MIP image showing irregularity (blue arrow) in anterior superior sagittal sinus, persistent occipital sinus (yellow arrow); (c) axial T1 showing hyperintensity/bleed (red star) in left frontal lobe; (d) 2D TOF sagittal MIP image showing irregularity (blue arrow) in anterior superior sagittal sinus and persistent occipital sinus-s/o haemorrhage in left frontal lobe likely due to chronic thrombosis of anterior superior sagittal sinus and incidentally detected persistent occipital sinus.

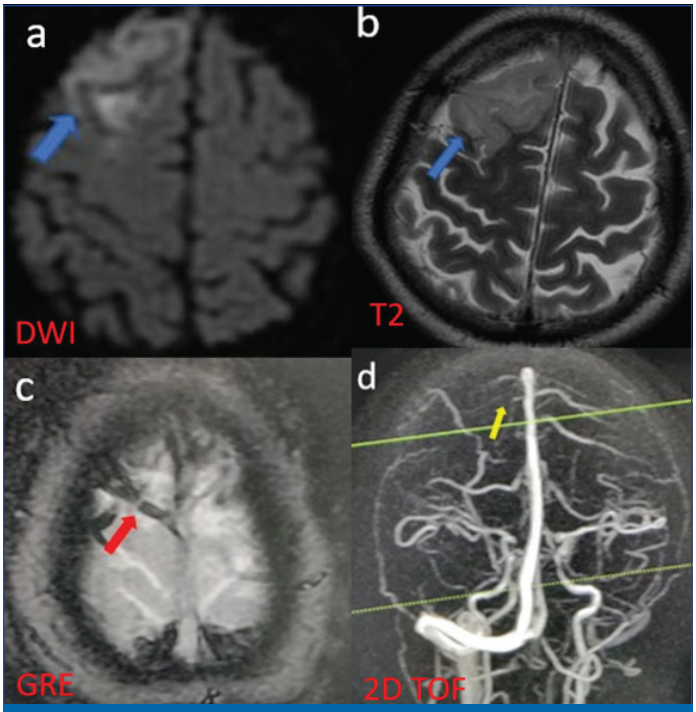
On MRI, incidence of cases with cytotoxic oedema in our study was 21%, whereas with vasogenic oedema was 64.3%. This corresponded to various stages of bleed and cytotoxic oedema within the infarction. In venous infarction, initially there is vasogenic oedema, and in later stages there is cytotoxic oedema due to venous infarcts. DWI may show regions with both raised as well as diminished ADC values depending upon whether there is cytotoxic and vasogenic oedema. Presence of both vasogenic and cytotoxic oedema is more indicative of venous infarct [5]. Flow voids were better visualised on T2, while T1 images commonly showed peripheral hyperintense content with central flow void. The plane of imaging should be perpendicular to the sinuses, for them to be assessed. Imaging plane parallel to sinuses, loss of hyperintense signal in proximal TS, entry slice phenomenon are some artefacts to be kept in mind during MRV.

In the present study, in cases of superficial sinus thrombosis, NCCT showed normal scans in 18 cases, while MRI was normal in 15 cases [Table/Fig-4]. This discrepancy was probably due to late presentation of cases with resolved bleed on CT but persistent blooming foci on GRE.

In cases presenting with thrombosis of both superficial and deep venous system, 11 cases on MRI showed haemorrhagic infarcts while it was diagnosed as non haemorrhagic infarcts in four of these cases on CT. It was observed that parenchymal changes were better evaluated on MRI especially who presented with haemorrhagic infarcts as it was easily visualised on GRE.

In a study by Gaikwad AB et al., CTV using 64 slice CT was found to have sensitivity of 88-100% and specificity of 95-100% in

diagnosing CVT [19]. It had limited diagnostic value in diagnosing cortical vein thrombosis with a sensitivity of 6-75%. This is due to contrast filling defect (the missing vein) which is difficult to distinguish from physiological variations in venous anatomy. However T2W was reported to have sensitivity of 97.4% and specificity of 100% in a study by Linn J et al., for diagnosing cortical vein thrombosis [20]. In the present study cortical vein thrombosis was visualised as linear hypointense signal/blooming involving cortical veins with underlying parenchyma showing T2/FLAIR abnormality and non visualisation of cortical veins on 2D TOF [Table/Fig-12].

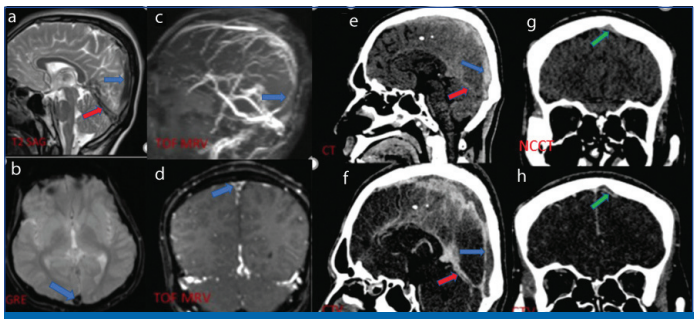


[Table/Fig-12]: A 34-year-old female presented with headache, MRI showing (a) DWI axial image showing restriction in right high frontal lobe (blue arrow); (b) T2 axial image showing hyperintensity (blue arrow); (c) GRE axial image showing blooming in right frontal cortical vein (red arrow); (d) 2D TOF coronal image showing non visualisation of right frontal cortical vein-s/o Cortical vein thrombosis with venous infrarct.

Loss of flow void on MR sequences was seen in 35/56 patients [Table/Fig-4]. Loss of flow void was better seen on T2 than T1. GRE showed dark signal void in the thrombosed sinuses. Hyperdense sinus (cord sign or delta sign) on CT was visualised in 20/56 cases. Parenchymal abnormalities were better visualised on MRI in comparison to CT. In cases of bony abnormalities, lateral sigmoid diverticulae, CT was helpful. Hypoplastic sinuses were easily visualised on CTV with smaller ipsilateral jugular foramen on bone window.

In CTV, 147 sinuses were involved, while on 2D TOF MRV 142 sinuses were involved. This disparity in five patients were due to anatomical variant of hypoplastic TS which was misinterpreted as complete loss of hyperintense signal in proximal 1/3rd of TS, this was better evaluated on CTV with small hypoplastic TS and ipsilateral small jugular foramen. Flow gaps were noted in 20 cases in the hypoplastic TS (more commonly on the left side). This also correlated with study done by Ayanzen RH et al., who observed in 31% of non dominant transverse sinuses in patients where MRI was normal [21]. In 20 cases where there was confusion of flow gaps versus thrombus on TOF in transverse sinus, CTV showed hypoplastic transverse sinus without any filling defect. A 112/112 transverse sinuses were visualised on CTV whereas 105/112 transverse sinuses were noted on TOF. A 2/7 was thought to be thrombosis on MRV and 5/7 were thought to be aplastic. However, they appeared hypoplastic on CTV. Basal Rosenthal veins, ISS and thalamostriate veins were better visualised on CTV than on 2D TOF MRV. Arachnoid granulations were equally detected on CTV and using FLAIR/T2 spin echo MR sequences.

The major technical drawbacks of TOF are saturation effects occurring at in-plane flow and the inclusion of substances, such as methaemoglobin, with a short T1 relaxation time [21,22]. In 3D phase-contrast MR angiography, the acquisition times are longer, so the technique is more susceptible to motion artefacts. A prior estimate of blood flow velocity is required to avoid aliasing. The phase-contrast technique may be more sensitive to signal loss because of turbulence or intravoxel dephasing [21]. Spin echo MRI may also be misleading, with a false diagnosis of dural sinus thrombosis resulting from flow-related enhancement or even from echo rephrasing, or a false impression of vessel patency resulting from intracellular deoxyhaemoglobin/Methaemoglobin mimicking a normal signal void on long TR sequences (T2W images) [17] [Table/Fig-13].



[Table/Fig-13]: A 25-year-old male presented with headache, vomiting and diplopia showing: (a) T2 sag image showing T2 hypointensity of SSS (blue arrow), straight sinus (red arrow); (b) GRE showing blooming of SSS (blue arrow); (c) TOF sag showing non visualisation of posterior SSS (blue arrow), (d) TOF coronal showing partial thrombus of SSS (blue arrow); (E) plain CT sag image showing hyperdensity of SSS (blue arrow), straight sinus (red arrow); (F) CTV sag showing filling defect in SSS (blue arrow), straight sinus (red arrow); (G) Cronal plain CT in anterior aspect showing hyperdense cortical vein in left high frontal region (green arrow); (H) CTV showing filling defect in cortical vein in left high frontal region (green arrow). Diagnosis- CVT involving SSS, straight sinus, left frontal cortical vein.

In a study by Ozsvath RR et al., [23], it was concluded that CTV is superior to MRV in identification of cerebral veins and dural sinuses and is atleast equivalent in establishing diagnosis of dural sinus thrombosis. Changes seen in chronic thrombosis with recanalisation are revealed well with CTV, however MRV is better suited for long-term follow-up because of lack of exposure to ionizing radiation. If MRI and MRV findings are equivocal in diagnosis of chronic sinus thrombosis, CTV should be the test of choice.

In a study by Van Dam LF et al., a new spectral CT technique called photon counting CT was shown to improve visualisation of CVT [6]. Recently, native contrast thrombus MR techniques, MR Direct Thrombus Imaging (MRDTI), MR Black Blood Thrombus Imaging (MRBTI) were evaluated in suspected CVT cases. In a study by Wetzel SG et al., both intra-arterial DSA and CTV were compared, and concluded that CTV was a reliable method for depicting the cerebral venous structures [24].

Limitation(s)

The main limitation of the study was its relatively small sample size. Delay between CT and MRI may have affected the results because of progression or resolution of thrombus in this interval period. Other limitations were the lack of follow-up for imaging recanalisation and interobserver variability.

CONCLUSION(S)

The CT Venography is superior or atleast equivalent to MR Venography in assessment of CVT. Easy accessibility, fast imaging time, high spatial resolution makes CTV a viable alternative to MRV. However, parenchymal changes and follow-up imaging can be better assessed on MRI with MRV.

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