Cerebral Venous Sinus Thrombosis Score and its Correlation with Clinical and MRI Findings

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Background: It is plausible that extent of cerebral venous sinus thrombosis (CVST) may determine clinical severity, magnetic resonance imaging (MRI) lesion, and outcome, therefore this study was undertaken. *Methods*: A total of 160 CVST patients were included and their clinical details, risk factors, Glasgow Coma Scale score, and parenchymal lesion on MRI were noted. The extent of venous sinus thrombosis on magnetic resonance venography was assessed by using CVST score which was computed giving 1 point for each thrombosed sinus and 3 points to superior sagittal sinus (SSS). Death and outcome at 6 months were assessed using modified Rankin Scale (mRS) as good (≤2) and poor (mRS 3-5). *Results*: Their median age was 29.5 years, and 76 (47%) were females. The median CVST score was 3 (range 1-9). CVST score did not correlate with clinical severity and risk factors. Insignificantly higher proportion of patients had parenchymal lesion with a CVST score of more than 2 (76.5% versus 64.2%). Superficial venous system thrombosis, however, correlated with seizure, papilloedema, and frontal lobe lesion. Frontal, temporal lesion correlated with SSS thrombosis, frontal and temporal with transverse sinus, temporal and cerebellar involvement in sigmoid, and basal ganglia and thalamus in straight sinus thrombosis. Seventeen patients (11%) died, and at 6 months, 132 (82%) had good and 11 (7%) poor recovery. Death and 6 months outcomes were not related to CVST score. *Conclusions*: Extent of CVST does not determine clinical severity, MRI lesion, and outcome. The location of parenchymal lesion however is related to thrombosis of draining sinus.

Key Words: Cerebral venous thrombosis—MRI—MRV—CVST score—stroke—outcome

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Introduction

Cerebral venous sinus thrombosis (CVST) is a rare but important cause of stroke like illness and accounts for less than 1% of all strokes. Females and neonates have higher frequency of CVST. In a study, the incidence of CVST in children below 18 years was .6 of 100,000, 43% of whom were neonates. The incidence of CVST patients peaks in the third decade with a female to male ratio of 5:1.5.[1] In the developing countries especially in South-East Asia, the incidence of CVST has been reported to be high in puerperal period.[2] Unlike arterial stroke, the presentation of CVST is subacute (2-30 days) or chronic (>30 days) in majority of the patients.[3,4] It is plausible that the extent of thrombosis and collateral status may determine the severity of clinical presentation, parenchymal lesion on magnetic resonance imaging (MRI), and outcome. The commonest clinical feature of CVST is headache occurring in 88%-93%, seizure in 37%-71%, focal neurological deficit in 20%-54%, and isolated raised intracranial pressure in 23% patients.[3-5] Focal neurological deficit and seizure

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may be the manifestation of underlying parenchymal lesion. CVST may undergo spontaneous thrombolysis due to innate thrombolytic system.[6,7] Patients with evidence of collateral at presentation may have lesser neurological and better outcome both in CVST and thrombotic stroke. There is paucity of studies evaluating the extent of CVST and collateral in determining clinical severity, MRI findings, and outcome.[8-10] Some authors reported correlation of extent of CVST on magnetic resonance venography (MRV) with parenchymal lesion, death, and poor outcome; whereas other did not find such association.[10,11] The risk factor of CVST differs in different ethnic and geographical regions; pregnancy and puerperium are commoner risk factor in South East Asia, and oral contraceptive pills in European countries.[3,5,12-18] Variation in risk factors may also be reflected in the extent of thrombosis and collateral formation. In this communication, we therefore report the role of extent of CVST and collaterals on MRV in determining clinical severity, MRI findings, and outcome from Northern India.

Methods

Inclusion

Consecutive patients with CVST admitted during 1996-2018 in a tertiary teaching hospital in North India were included from the prospectively maintained CVST registry whose MRI and MRV were available for review. The diagnosis of CVST was based on MRV. The study was approved by the Institute Ethics Committee (PGI/BE/774). All these patients were personally managed by 2 of the senior neurologist (J.K. and U.K.M.), and MRI and MRV were reviewed by 2 neuroradiologists (S.K., N.J.) who were unaware about the clinical details.

Exclusion Criteria

The patients with malignancy, vascular malformation, arterial stroke, primary intracerebral hemorrhage, paranasal sinus infection, and those with hepatic or renal failure were excluded.

Clinical Evaluation

A detailed clinical history including demographic information, duration of illness, and symptoms were included. The patients were enquired about headache, seizure, focal weakness, visual impairment, and memory impairment. Presence of altered sensorium was noted. The CVST risk factors were also noted including oral contraceptives, fever, sepsis, pregnancy, arthritis, deep vein thrombosis, anemia, paranasal infection, and malignancy.

General physical and neurological examination was done. Consciousness was assessed by Glasgow Coma Scale (GCS), and cognitive function was assessed by Mini Mental State Examination in the patients who could cooperate for the test. Presence of papilloedema, cranial nerve palsy, focal weakness, tendon reflex, and coordination were tested. Joint position, pinprick, and cortical sensation were also tested.

MRI and MRV

Cranial MRI and contrast MRV were done within 48 hours of admission, and the patients were admitted after a median 10 days of illness. We had used 1.5 T MRI machine (Magnetom SP, Siemens, Erlanger, Germany) during 1992-2002, 1.5 T (Sigma, GEMS, Milwaukee, USA) during 2002 to 2009, and 3T (Sigma HDxT, GEMS, Milwaukee, WI) from 2009 till now. Diffusion weighted image was performed since 2009. Before 2012, 2-D time of flight (TOF) venography was done in most of the patients with supplementation by phase contrast 3D in some. After 2012, contrast enhanced 3D TOF venography was done. The number of patients undergoing DWI was 109 patients, 2D TOF venography in 86 patients, and contrast enhanced 3D TOF venography in 74 patients. T1W, T2W, FLAIR, and DWI sequences were obtained in axial, coronal, and sagittal planes. The location and extent of parenchymal lesions were noted. The infarcts were considered small if it involves less than one-third of a lobe, medium one-third to whole lobe, and large more than a lobe.[10] Hemorrhagic transformation of infarction was also noted.

CVST Score

On contrast MRV, the extent of venous sinus thrombosis was scored based on the distribution of clots in the sinuses. The CVST score was calculated assigning 1 point for each thrombosed sinus except superior sagittal sinus which was given 3 points. Superior sagittal sinus was divided into 3 parts and was assigned 1 point for each third involvement. One point was also given to each internal cerebral vein and vein of Galen thrombosis.[11] The total score in a patient was noted. Sinus thrombosis was also classified as superficial, deep, or both venous system involvement. Presence of collaterals was noted and classified into grade I, grade II, and grade III.[8] A repeat MRV was done in 20 patients only and their CVST score was also calculated.

Treatment

The patients were treated with low molecular heparin (100 unit/kg subcutaneously twice daily) or unfractionated heparin (5000 IU intravenously followed by 18 unit/kg/hour infusion to keep activated partial thromboplastin time at 2.5 times of control) for 2 weeks followed

by oral anticoagulants. The dose of anticoagulant was adjusted to maintain INR of 2.5-3. The average INR and percentage of number of therapeutic INR (number of therapeutic INR/total number of INR done × 100) during 6 months were also computed. The patients with seizures received levetiracetam or sodium valproate with or without clobazam. The patients with respiratory failure were intubated and mechanically ventilated. Underlying treatable disorder was also managed.

Outcome

In hospital death and its causes were noted. The functional outcomes at 3 and 6 months were assessed using modified Rankin scale (mRS) and categorized into good (mRS ≤ 2), poor (mRs > 2).[4]

Statistical Analysis

Continuous and normally distributed data are expressed as mean (standard deviation), whereas continuous but skewed data are expressed as median (range). The relationship of CVST score on MRV was correlated with age, gender, seizure, focal motor deficit, papilloedema, GCS score, and MRI lesions using various parametric and nonparametric tests. Presence or absence of parenchymal lesions were also compared with the location of venous sinus thrombosis, clinical severity (focal deficit, GCS score, seizure), and outcome using chi-square or Mann-Whitney *U* test. Death and disability were also compared with the type of CVST (superficial, deep, or both), and CVST score using chi-square test. Repeat CVST score was compared with average INR and percentage of therapeutic INR during follow-up period using Mann-Whitney *U* test, and mRS at 6 months using Spearman correlation test. Statistical analysis was done with SPSS (Statistical Package of Social Sciences, IBM, Chicago, IL) version 16 and variables with a P value < .05 were considered significant.

Results

There were 160 patients with CVST who were admitted during the study period, and their age ranged between 3 and 76 (median 29.5) years, 76 (47%) of whom were females. The median duration of illness was 10 (range 1-180) days; 10 (6%) had acute, 130 (81%) subacute, and 20 (13%) had chronic presentation. Ninety-two (57%) patients had seizure, 110 (69%) had papilloedema, and 77 (52%) had focal motor deficit (hemiplegia in 62, monoplegia in 4, paraplegia in 1, and quadriplegia in 10). Their GCS score ranged between 3 and 15 (median 14). The risk factor of CVST was prothrombotic state in 52, prothrombotic with other risk factors in 65, idiopathic in 29, infection in 10, and female specific in 12 (pregnancy in 4, puerperium in 4, and oral contraceptive 4) patients.

MRI and MRV Findings

MRI was abnormal in 119 (74%) patients, and revealed infarction in 26 and hemorrhagic infarction in 93 patients. The majority of infarctions were in parietal in 65, followed by frontal in 53, occipital in 30, temporal in 30, thalamus in 13, cerebellum in 6, and brainstem in 3 patients. The infarctions were large in 88, medium in 13, and small in 18 patients. The most frequently involved sinus was transverse sinus in 108 (67%) followed by superior sagittal in 105 (66%), sigmoid sinus in 76 (47%), and straight sinus in 37 (23%). Isolated superficial system was involved in 127 (79%), deep system in 8 (5%), and both superficial and deep systems in 25 (16%) patients.

Cerebral Venous Sinus Thrombosis Score and its Clinical and MRI Correlation

The median CVST score was three (range 1-9). CVST score did not correlate with GCS score (P = .91), duration of illness (P = .36), risk factors of CVST (P = .85), motor deficit (P = .73), raised intracranial pressure (P = .15), and seizure (P = .68). The presence of parenchymal lesion on MRI however was not related to CVST score (Table 1). Twenty-eight patients had involvement of less than 2 sinuses and of them 18 had infarction/hemorrhagic infarction; whereas out of 44 patients with more than 4 sinuses involvement, 29 had infarction/hemorrhagic infarctions (P = .058; Fig 1). Superficial venous system thrombosis, however correlated with seizure (P = .047), papilloedema (P = .046), and frontal lesion (P < .01). The details are shown in Table 2. CVST score however was insignificantly higher in the patients with bilateral parenchymal lesions (P = .07). In 20 patients, MRV was repeated at 6 months, and the CVST score was 0-2 in 16 patients and more than 2 in 4 patients. The CVST score (0-2 versus >2) was not related to average INR (2.28 ± .34 versus 2.42 ± .30; P = .32) and percentage of therapeutic INR (70 ± 48.4 versus 52.5 ± 9.5; P = .49).

Location of Parenchymal Lesion and Sinus Involved

Frontal and temporal lesion correlated with superior sagittal sinus thrombosis, frontal and temporal with transverse sinus, temporal and cerebellum with sigmoid, and basal ganglia and thalamus with straight sinus thrombosis (Table 3 and Fig. 2 and 3; Supplementary Fig 1). Collaterals were seen in 6 of 42 (14%) patients at baseline imaging. Presence of collateral was not related to rapidity of onset, GCS score, focal deficit, seizure, papilloedema, and parenchymal lesions (Supplementary 1).

Outcome

At 3 months, 17 (11%) died (16 in hospital), 130 (81%) had good recovery, and 13 (8%) poor recovery. At 6 months, 132 (82%) had good and 11 (7%) poor recovery (mRS 3-5). Death and functional outcome at 3 and 6 months were not related to CVST score (Supplementary

Table 1. *Relationship of cerebral venous sinus thrombosis (CVST) score with clinical and MRI findings*

Parameters (n)	CVST score median (range)	*P* value
Onset		
Acute (10)	3 (2-4)	.36
Subacute (129)	3 (1-9)	
Chronic (21)	2 (1-7)	
Seizure		
Present (92)	3 (1-9)	.68
Absent (68)	3 (1-9)	
Glasgow Coma Scale score		.91
≥9 (136)	3 (1-9)	
<9 (24)	3 (1-9)	
Motor deficit		.73
Present (83)	3 (1-9)	
Absent (77)	3 (1-9)	
Papilloedema		.004
Present (110)	4 (1-9)	
Absent (50)	3 (1-8)	
Risk factors		.85
Prothrombotic (52)	3 (1-8)	
Prothrombotic plus (65)	3 (1-9)	
Idiopathic (29)	3 (1-9)	
Infection (10)	3 (1-9)	
Pregnancy (4)	2 (1-8)	
MRI findings		
Parenchymal lesion		.59
Present (119)	3(1-9)	
Absent (41)	4(1-9)	
Type of lesion		.91
Infarct (26)	4 (1-9)	
Hemorrhagic infarct (93)	3 (1-8)	
Bilateral lesion		.07
Yes (42)	4 (1-8)	
Unilateral (77)	3 (1-9)	

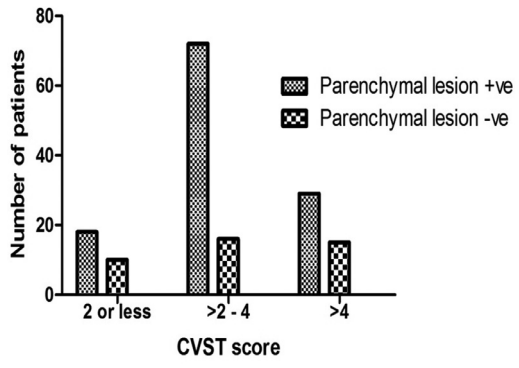

Figure 1. *Bar diagram shows relationship of parenchymal lesion and cerebral venous sinus thrombosis score (CVST). Higher proportion of patients had parenchymal lesion who had involvement of more than 2 sinuses. +ve, positive, −ve, negative.*

Fig 2). During 6 months follow-up, the average INR was 3 (1-5) and percentage of therapeutic INR 50 (20-80). There was no significant correlation between 6 months outcome with average INR (r = .17; *P* = .05) and percentage of therapeutic INR (r = .12; *P* = .16).

Discussion

In this study, CVST score correlated with papilloedema, but not with other clinical parameters, MRI findings, and outcome. Superficial venous system thrombosis correlated with seizure, papilloedema, and frontal parenchymal lesion. The location of parenchymal lesion however correlated with blockage of draining sinus such as frontal in superior sagittal sinus or transverse sinus thrombosis, temporal and cerebellar infarctions in sigmoid sinus, and basal ganglia and thalamus in straight sinus thrombosis. Collaterals were found only in 14% patients at baseline and were not related to clinical severity, MRI findings, and outcome. Zubkov et al reported role of CVST score in determining parenchymal lesion in 56 patients. Nineteen (34%) patients had parenchymal lesion; none of the patients with CVST score of 1.9 or below had a parenchymal lesion, but all the patients with a score of 3.1 or more have parenchymal lesion. CVST score was not related to age, gender, and thrombotic state. Outcome was however related to CVST score and parenchymal lesion.[11] Another study however did not find relationship of CVST score with parenchymal lesion.[10] In our study, 18 of 28 (64%) patients had parenchymal lesion with CVST score of less than 2, and 29 out of 44 (66%) patients had parenchymal lesion with a CVST score of more than 4.

In a study on 128 patients with CVT, prothrombotic risk factor was present in 46 (35.9%), prothrombotic with other risk factors in 36 (28.1%), nonprothrombotic risk factors in 20 (15.6%), and no risk factors in 26 (2.3%) patients. More than 2 risk factors were present in 33 (25.8%). On MRV, more than 2 sinuses were involved in 35 (27.3%) patients and 94 (73.4%) patients had parenchymal lesions on MRI. The number of risk factors was not related to clinical severity and extent of MRI or MRV abnormality.[19] This may be due to recanalization of sinuses following anticoagulation treatment. Recurrent venous thrombosis however are more common with more potent prothrombotic conditions such as factor V Leiden mutation and APLA syndrome.[20] Correlation of CVST score with papilloedema in our study may be due to elevated intracranial pressure as a result of impaired venous drainage and venous congestion. Same correlation has also been observed in the location of parenchymal lesion and thrombosis of draining sinus. Thrombosis of a sinus leads to stagnation of venous stasis proximally, which in turn forms a conducive environment for thrombus formation. Most of the superficial cerebral veins from both the hemispheres drain in the superior sagittal sinus, petrosal sinus drains from temporal lobe to transverse sinus, and the deeper structure of brain through

Table 2. *Comparison of clinical and MRII findings with the superficial, deep and both cerebral venous system thrombosis*

Parameters	Superficial (n = 127 [A])	Deep (n = 8 [B])	Both (n = 25 [C])	*P* value A vs B	*P* value B vs C	*P* value A vs C
Age	33.98 ± 13.9	33.88 ± 12.7	28 ± 14.72	.983	.321	.055
Female gender	61 (48%)	4 (50%)	11 (25%)	.914	.767	.712
Etiology				.131	.841	.092
Prothrombotic (52)	40 (31%)	3 (38%)	9 (36%)			
Prothrombotic plus (65)	53 (42%)	3 (38%)	9 (36%)			
Idiopathic (29)	27 (21%)	0	2 (8%)			
Infection (10)	5 (4%)	1 (13%)	4 (16%)			
Pregnancy (4)	2 (2%)	1 (13%)	1 (4%)			
Presentation				.40	.38	.74
Acute	7	1	2			
Subacute	104	5	21			
Chronic	16	2	2			
Seizure	78 (61.4%)	4 (50%)	10 (40%)	.521	.618	.047
Focal deficit	68 (53.5%)	4 (50%)	11 (44%)	.846	.767	.383
GCS				.806	.970	.633
<9	20 (16%)	1 (12.5%)	3 (12%)			
>9	107 (84%)	7 (87.5%)	22 (88%)			
Raised ICP	90 (71%)	6 (75%)	20 (80%)	.802	.763	.351
Papilloedema	43 (34%)	0	7 (28%)	.046	.902	.569
MRI brain						
Parenchyma involved	96 (75%)	6 (75%)	17 (68%)	.970	.708	.427
Type of lesion				.053	.526	.097
Infarct	17/96	3/6	6/17			
Hemorrhagic infarct	79/96	3/6	11/17			
Bilateral	29 (23%)	3 (37.5%)	10 (40%)	.374	.900	.089

Abbreviations: ICP, intracranial pressure; MRI, magnetic resonance imaging.

inferior sagittal sinus and great cerebral vein to straight sinus.[21] These may explain correlation of frontal lesion with superior sagittal sinus thrombosis, temporal with sigmoid, and basal ganglia and thalamus with straight sinus. Some studies however did not find correlation of location of focal brain lesion with site of sinus thrombosis.[10]

Death and disability is lesser in CVST compared to thrombotic and intracerebral hemorrhage.[22-24] In the present study 10 % died during hospital stay and only 7 % had poor recovery at 6 months. Death and disability were not related to CVST score in our study, but Zubkov et al reported CVST score and parenchymal lesion as independent predictors of outcome.[11] Male gender, old age, altered mental state, low GCS score, deep venous sinus thrombosis, intracerebral hemorrhage, and presence of malignancy and infections were identified as risk factor for poor outcome.[3,5,8,25,26] Various scoring including clinical, MRI, and MRV have been developed to predict death

Table 3. *Relationship of parenchymal lesion with location of thrombosis*

Parameters	SSS n = 105	*P*	TS n = 108	*P*	Sigmoid n = 76	*P*	SS n = 37	*P*
Frontal (53)	44 (83%)	.001	27 (51%)	.002	22 (41%)	.345	13 (24%)	.815
Parietal (65)	42 (65%)	.82	44 (68%)	.99	31 (48%)	.98	13 (20%)	.46
Temporal (30)	11 (37%)	<.01	29 (97%)	<.0001	26 (87%)	<.001	8 (27%)	.68
Occipital (30)	16 (53%)	.12	24 (80%)	.11	17 (57%)	.28	7 (23%)	.96
Cerebellum (6)	2 (33%)	.09	6 (100%)	.08	6 (100%)	.009	5 (83%)	<.001
Brainstem (3)	1 (33%)	.23	3 (100%)	.23	2 (67%)	.51	2 (67%)	.07
Internal capsule (3)	1 (33%)	.23	2 (67%)	.97	2 (67%)	.51	1 (33%)	.67
Basal ganglion (9)	3 (33%)	.03	7 (78%)	.50	6 (67%)	.24	7 (78%)	<.001
Thalamus (13)	4 (31%)	.006	9 (69%)	.90	7 (54%)	.65	7 (54%)	.006

Abbreviations: SS, straight sinus; SSS, superior sagittal sinus; TS, transverse sinus.
Chi-square test was done between location of parenchymal lesion and sinus involved.

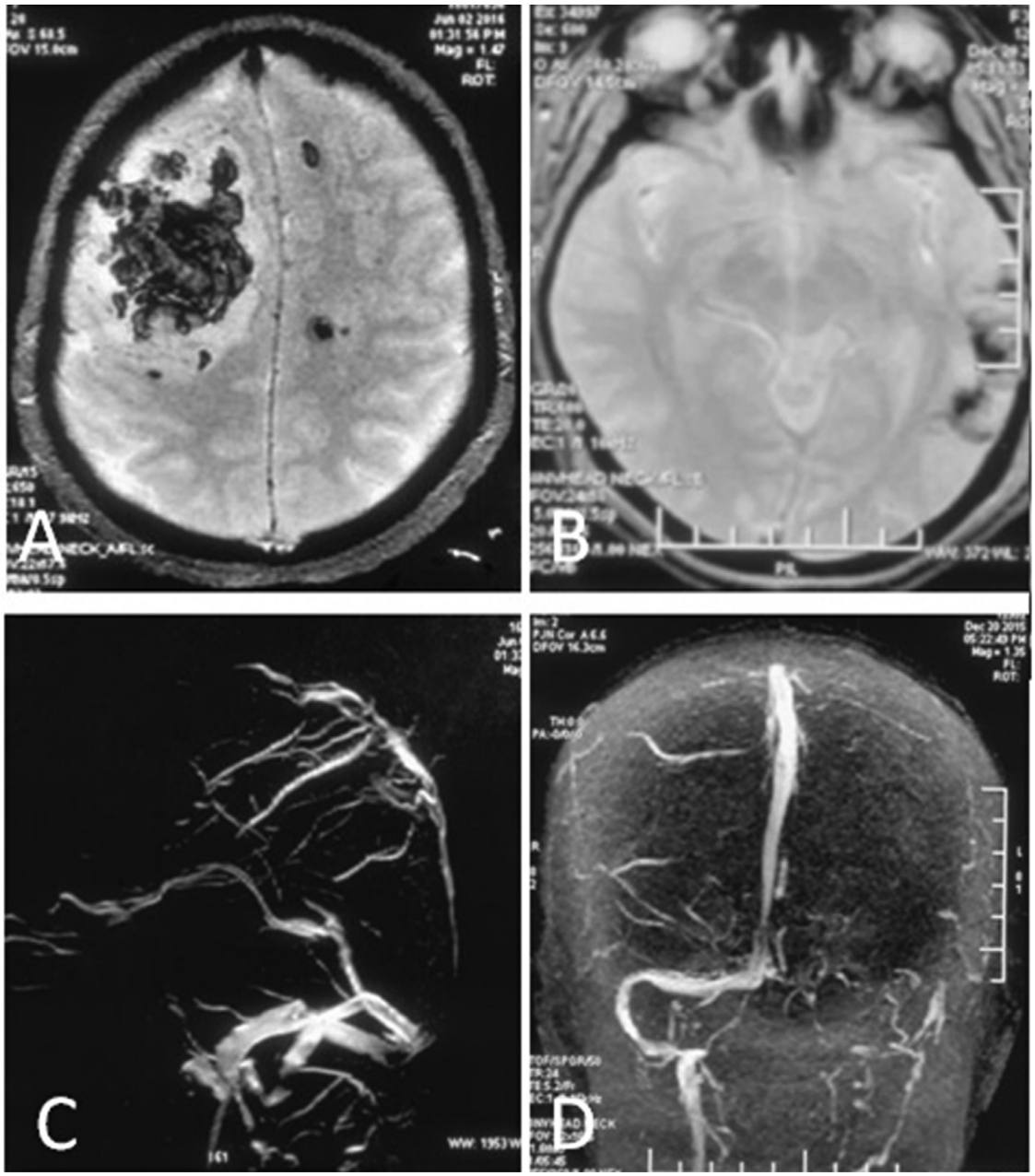

Figure 2. *MRI in susceptibility weighted sequence and MRV showing location of parenchymal lesion and site of venous sinus thrombosis. (A) MRI of a male patient shows hemorrhagic infarction in both frontal lobes, predominantly on the right with (C) thrombosis of anterior half of superior sagittal sinus. (B) MRI of a female patient shows left temporal hemorrhagic infarct with (D) left transverse and sigmoid sinus thrombosis.*

and disability of CVST.[27-29] Barboza et al described a CVT-grading scale including gender, level of consciousness, bilateral Babinski sign, parenchymal lesion, and intracerebral hemorrhage, which have an accuracy of 91.6% for the prediction of 30-day mortality and 85.3% for mRS more than 2.[30] ISCVT-risk score is a 0-9 point score including coma, malignancy, deep CVT, mental status disturbance, male gender, and parenchymal hemorrhage. For predicting mortality, this score has a sensitivity of 48% and specificity of 89%; and that of poor outcome 67% and 81%.[28]

This study highlights elevated intracranial pressure in more extensive thrombosis and location of parenchymal lesion due to blockage of draining sinus. However extent of thrombosis does determine rapidity of onset, clinical severity, MRI findings, and outcome.

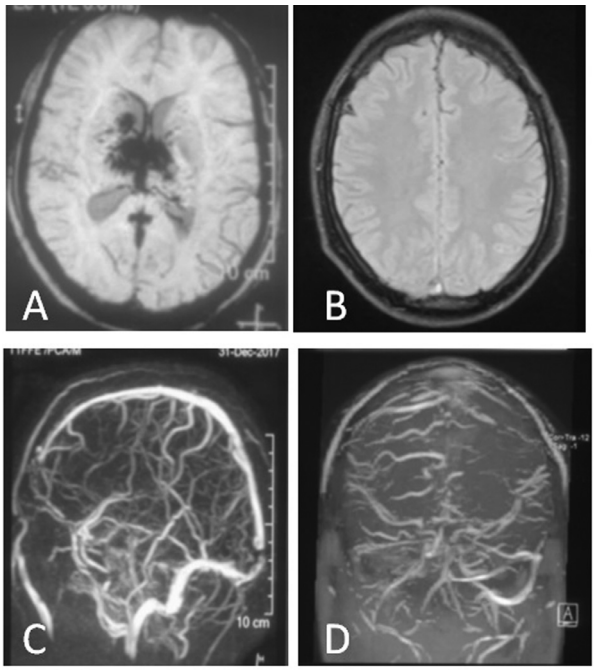

Figure 3. *MRI in susceptibility weighted sequence and MRV showing location of parenchymal lesion and site of venous sinus thrombosis. (A) MRI of a 44 years old lady shows bilateral thalamic and right caudate hemorrhagic infarct, who had (C) straight sinus thrombosis. (B) MRI of a 23-year-male in FLAIR sequence shows normal parenchyma, who had (D) thrombosis of superior sagittal sinus, right transverse, right sigmoid, and straight sinus.*

Ethical Statement

We confirm that we have read Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines. This study was approved by Institutional Ethics Committee, Lucknow, India.

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Declaration of Competing Interest

The authors declare that they have no conflict of interests.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jstrokecerebrovasdis.2019.104324.

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