Original Article

Clinical Presentation, Neuroimaging Findings, and Predictors of Brain Parenchymal Lesions in Cerebral Vein and Dural Sinus Thrombosis: A Retrospective Study

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Abstract

Introduction: Cerebral venous sinus thrombosis (CVST) is an unusual cause of stroke with potentially serious consequences. This study was designed to investigate the clinical and neuroimaging features in patients with CVST and to analyze the predictors of brain parenchymal lesions. **Materials and Methods:** A retrospective study of 181 patients with CVST was conducted in a tertiary care hospital. **Results:** Of 181 patients (age range 14–96 years, mean age: 34.64 ± 14.66 years), 121 were female (66.9%). Most of the patients were in their third decade of life. Headache (47.51%) was the most common clinical presentation followed by seizures (24.31%). Transverse sinus (TS) (77.9%) was the most common site of venous sinus thrombosis. Brain parenchymal lesions were present in 63%, and each patient had subarachnoid and intraventricular hemorrhage. Hemorrhagic venous infarct was the most common brain parenchymal lesion (37.57%). Frontal region (25.4%) was the most common site of brain parenchymal lesions followed by frontoparietal region (21.9%). Women were more likely to have brain parenchymal lesions (72.4%, $P = 0.034$). Headache was the most common clinical presentation in patients without brain parenchymal lesions while seizures with brain parenchymal lesions. Straight sinus thrombosis was more likely to be associated with brain parenchymal lesions ($P = 0.009$). **Conclusion:** CVST presents in young and more commonly in females. TS was the most common site of venous sinus thrombosis. Female gender, seizures, altered sensorium and focal neurological deficit at presentation, and straight sinus thrombosis were more likely associated with the presence of brain parenchymal lesions.

Keywords: Magnetic resonance venography, straight sinus, superior sagittal sinus, transverse sinus

Introduction

Cerebral venous sinus thrombosis (CVST), once considered to be rare, is increasingly diagnosed with advancement in the diagnostic neuroimaging techniques and increasing awareness of the disease. As opposed to arterial stroke, CVST is primarily a disease of the young, with young adults and children being most often affected. CVST accounts for 0.5% of all strokes and its annual incidence is estimated to be 3–4 cases per million population and up to 7 cases per million children.[1] About 75% of the adult patients are women.[2] Clinical presentation of CVST varies considerably. While some patients have relatively mild symptoms such as headache, others develop devastating complications, including hemorrhagic venous infarctions and severe intracranial hypertension. Headache is the most common presentation and sometimes the only feature, making it difficult to diagnose.[2,3] Along with headache, seizure (focal or generalized), hemiparesis, and aphasia (though less common) may also present. Focal brain abnormalities are present in approximately 50%–60% of patients with CVST.[4,5] The pathophysiologic mechanism of parenchymal injuries in CSVT is not fully understood. In experimental CVST models, severity of parenchymal injuries has been considered proportional to the degree of venous occlusion.[6] The lack of correlation between the extent and site of thrombosis in the dural sinuses and location of brain lesions has been suggested

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in adults.[7] Magnetic resonance imaging (MRI) is the modality of choice for diagnosis and when combined with magnetic resonance venography (MRV) increases its sensitivity.[5,8] There is lack of data regarding predictors of brain parenchymal lesions in patients with CVST in the literature.

The present study is designed to study the clinical and neuroimaging features in patients with CVST and to analyze the predictors of brain parenchymal lesions.

Materials and Methods

Medical records of the patients, sent for MRV examination in a tertiary care teaching hospital from January 2011 to December 2013, were reviewed retrospectively. MRI and MRV data of 2000 patients during the study period were reviewed. Patients with CVST confirmed by MRV were included in this study. Patients with any congenital intracranial abnormality, head trauma, arteriovenous malformation, or previous surgery were excluded from the study. One hundred and eighty-one patients with CVST confirmed by MRI and MRV were included in the final analysis. Clinical indication for MRI and MRV was noted in each patient.

Magnetic resonance imaging

MRI was performed with superconducting 1.5-T magnetic resonance (MR) machine (Achieva version 1.3; Philips, Best, the Netherlands) and standard head coil. Three-dimensional MR venography was performed in the coronal plane using the following parameters: time to echo (TE) – 50, time to repeat (TR) – 500, field of view (FOV) – 230–250, slice thickness – 1 mm, matrix – 240 × 256, and flip angle – 50°. Additional routine T2-weighted image coronal and axial fluid-attenuated inversion recovery sequences were also performed.

The slice thickness of 1.2 mm was acquired with contiguous sections using matrix size of 256 × 256 and a nominal FOV of 27 cm with TR − 22 ms, TE − 7.4 ms, and flip angle – 15°, TRITE – 50/8.4. The velocity encoding applied was 30–40 cm/s. The images were displayed as 20 maximum intensity projection (MIP) images reconstructed from the source images at 9° increments.

Image analysis

MRI included diffusion-weighted images, axial T1, axial fast spin-echo (FSE) T2, coronal and sagittal FSE T2, and gradient-echo sequences. MIPs were created at the MR operating console for 3D-MR venography dataset. The MIP images were viewed in the sagittal, transverse, and coronal planes.

The dural venous sinuses included in this study are superior sagittal sinus (SSS), sigmoid sinus (SS), transverse sinus (TS), and straight sinus.

Statistical analysis

Numeric values were shown as the mean ± standard deviation. Chi-square contingency analysis was used to explore the statistically significant difference of demographic, clinical, and radiological variables among patients with CVST with or without brain parenchymal lesions. A difference was considered statistically significant at a $P < 0.05$. The analysis was performed using Epi Info-7 statistical software (developed by Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia (US)).

Results

This study included a total of 181 patients (age range 14–96 years, mean age: 34.64 ± 14.66 years). The majority of the patients were women (121, 66.9%). Most of the patients were in their third decade of life. Distribution of CVST patients according to age (decade years) has been shown in Figure 1. The mean age for men was 36.85 ± 17.91 years (range 16–96 years) and for women was 33.55 ± 12.69 years (range 14–80 years).

Clinical presentation

The most common clinical presentation of CVST was headache (47.51%). Other presentations were seizures (24.31%), altered sensorium (14.92%), focal neurological deficit (12.15%), and vertigo (1.1%).

Brain parenchymal findings

Of 181 patients of CVST, 116 (64.09%) had brain parenchymal lesions. One patient had subarachnoid hemorrhage (SAH) without other brain parenchymal lesions and other had intraventricular hemorrhage. Eight patients had SAH along with other brain parenchymal abnormalities. Four patients with SAH had nonhemorrhagic venous infarcts, three had hemorrhagic venous infarcts and one had intraparenchymal hemorrhage. Sixty-five patients had no brain parenchymal lesions. Hemorrhagic venous infarct was the most common brain parenchymal lesion (37.57%). Eleven patients had intraparenchymal hemorrhage. Type of brain parenchymal lesions has been tabulated in Table 1. The most common site of brain parenchymal lesions was frontal region (25.4%) followed by the frontoparietal region. Distribution of site of brain parenchymal lesions has been summarized in Table 2. Neuroimaging findings of two patients have been illustrated in Figure 2.

Distribution of venous sinus thrombosis

TS (77.9%) was the most common site of venous sinus

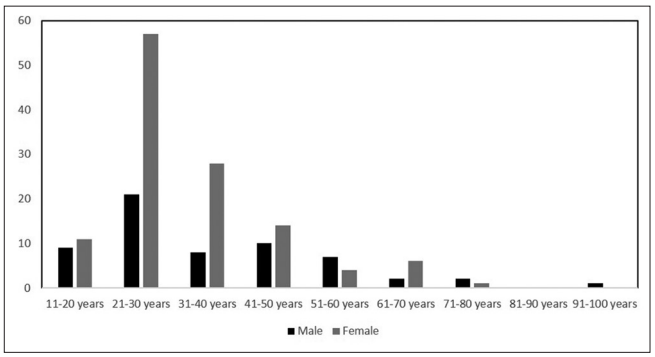

Figure 1: Distribution of cortical venous sinus thrombosis patients according to age (decade years)

thrombosis followed by SS (66.9%) and SSS (60.2%). SSS was the most common thrombosed sinus in isolation among 29 patients. Deep venous sinus system was involved in 39 patients. Superficial cortical veins were involved in five patients. One hundred and thirty-five patients had involvement of more than one venous sinus, and among them, 20 patients had involvement of both superficial and deep venous sinuses. The most common association was noticed between SSS and TS or SS. Distribution of venous sinus thrombosis has been summarized in Table 3.

Predictors of brain parenchymal lesions in cerebral venous sinus thrombosis

Patients were subdivided into two groups depending on the presence of parenchymal abnormalities on neuroimaging. There were 65 patients without evidence of parenchymal lesions (Group 1) and 116 patients with parenchymal lesions (Group 2). There was no significant difference in age between both the groups ($P = 0.33$). Women predominated in Group 2 (72.4% in Group 2 versus 56.9% in Group 1, $P = 0.034$). Headache was the most common presentation in Group 1, while seizure was the most common in Group 2. None of the patients in Group 1 had focal neurological deficit. Majority had TS thrombosis among both groups, although straight sinus thrombosis was more common in Group 2 ($P = 0.009$). Female gender, seizures, altered sensorium and focal neurological deficit at presentation, and

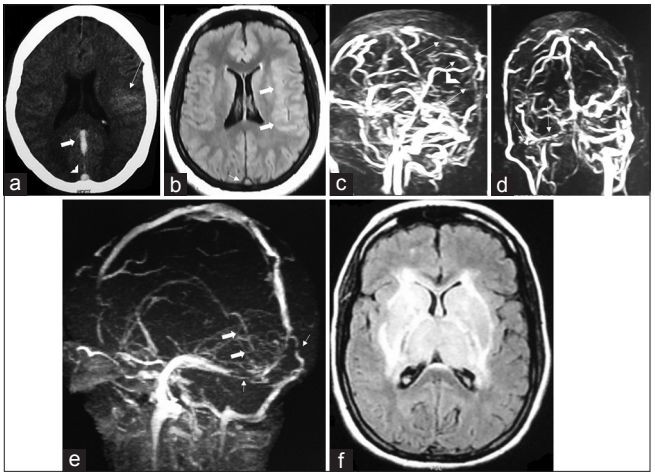

Figure 2: A 34-year-old female with seizure had (a) Hyperdensity of straight sinus (thick arrow) and superior sagittal sinus (arrowhead) and subarachnoid hemorrhage of the left parietal region (long arrow) on noncontrast computed tomography head. (b) Thrombus at superior sagittal sinus (short arrow) and subarachnoid hemorrhage of the left parietal region (thick arrows) on fluid-attenuated inversion recovery. (c) Magnetic resonance venography showed thrombosis of superior sagittal sinus (long arrows), straight sinus (arrowhead) and (d) Left transverse sinus and sigmoid sinus (long arrows). (e) A 23-year-old female (postpartum) with altered sensorium had thrombosis of deep venous system (thick arrows) and bilateral transverse sinus (short arrows) on magnetic resonance venography, and (f) nonhemorrhagic venous infarcts of bilateral thalami and basal ganglia on fluid-attenuated inversion recovery sequence

Table 1: Type of brain parenchymal lesion

Brain parenchymal lesion	Frequency (%)
Hemorrhagic venous infarct	68 (37.57)
Nonhemorrhagic venous infarct	35 (19.34)
Intraparenchymal hemorrhage	11 (6.17)
Intraventricular hemorrhage	1 (0.55)
Subarachnoid hemorrhage	1 (0.55)
Normal	65 (35.9)

Table 2: Distribution and types of brain parenchymal lesions

Site of lesion	Hemorrhagic venous infarct (*n*)	Nonhemorrhagic venous infarct (*n*)	Intraparenchymal hemorrhage (*n*)	Total, *n* (%)
Frontal	14	12	3	29 (25.4)
Parietal	4	2	0	6 (5.3)
Occipital	3	1	0	4 (3.5)
Frontotemporal	2	2	0	4 (3.5)
Frontoparietal	16	6	3	25 (21.9)
Temporoparietal	3	1	2	6 (5.3)
Parieto-occipital	7	1	0	8 (7.0)
Temporo-occipital	4	2	2	8 (7.0)
Frontotemporoparietal	1	1	0	2 (1.7)
Temporo-parieto-occipital	5	0	1	6 (5.3)
Bilateral thalamic	4	2	0	6 (5.3)
Bilateral thalamocapsuloganglionic	1	3	0	4 (3.5)
Bilateral caudate nuclei	0	1	0	1 (0.9)
Bilateral thalamic and frontoparietal	1	0	0	1 (0.9)
Cerebellum	1	1	0	2 (1.7)
Cerebellum and fronto-parieto-occipital	2	0	0	2 (1.7)
Intraventricular hemorrhage	-	-	-	1 (0.9)
Subarachnoid hemorrhage	-	-	-	1 (0.9)
Total	68	35	11	116 (100)

straight sinus thrombosis were more likely associated with the presence of brain parenchymal lesions. Predictors of brain parenchymal lesions in patients with CVST have been summarized in Table 4.

Discussion

This study was conducted to evaluate the clinical presentation, neuroimaging findings, and predictors of brain parenchymal lesions in cerebral vein and dural sinus thrombosis. Of 181 patients, majority were women (66.9%) in their third decade of life. The most common clinical presentation was headache (47.51%). Hemorrhagic venous infarct was the most common brain parenchymal lesion (37.57%). One patient had isolated SAH secondary to CVST. The most common site of venous sinus thrombosis was TS.

In the present study, the majority of the patients were in their economically productive years of life. Women were more often affected than men. This observation is consistent with other studies. The International Sinus Cerebral Vein Thrombosis (ISCVT) trial, a collaborative study that enrolled more than 600 patients with CVST, showed higher incidence of CVST in females and in their third decade.[2] Pregnancy and postpartum status are known to result in increased incidence of the CVST due to the normal physiological changes in the pregnancy that results in hypercoagulability. This gender bias is usually attributed to gender-specific risk factors such as the usage of oral contraceptives and the influence of other factors such as pregnancy, postpartum status, and hormone replacement therapy. Comparing the age group involved, 20–40 years is the most common age groups involved in various studies (Mehta *et al*., 77.8%; Ameri and Bousser, 61%; and Mangshetty and Reddy, 87.5%).[9-11] The present study also showed similar finding with 62.9% in the same age group.

The clinical presentation of CVST is varied, and this mandates the high index of suspicion in order not to overlook it. The most frequent, but the least-specific clinical presentation of CVST is headache, which is present in 75%–90% of adult patients.[2,3] Even in the present study, headache (47.51%) was the most common clinical presentation. Similar results have been shown in different series.[11-15] Depending on the availability of collateral venous pathways, CVST can result in significant brain involvement or may be presented as nonspecific symptoms like headache. The mechanism of headache is postulated to be the stretching of nerve fibers in the walls of the occluded sinus and local inflammation, as suggested by the evidence of contrast enhancement of the sinus wall surrounding the clot. Other mechanism of headache may be increased intracranial pressure due to impaired venous drainage. In the presence of cerebral cortical lesions because of venous ischemia or hemorrhage, neurological signs and symptoms referable to the affected region are often present. The most common are seizures, hemiparesis, and aphasia, but other cortical signs and sensory symptoms may occur. Focal neurological deficit was present in 12.15% patients in the present study. Other studies have shown higher incidence of focal deficit compared to the present study. In a study by

Table 3: Distribution of venous sinus thrombosis

Venous sinus	Frequency (%)
Isolated SSS	29 (16)
SSS with other venous sinuses	80 (44.2)
Isolated TS	7 (3.9)
TS with other venous sinuses	134 (74)
Isolated SS	2 (1.1)
SS with other venous sinuses	119 (65.7)
Isolated straight sinus	1 (0.6)
Straight sinus with other venous sinuses	33 (18.2)
Straight sinus, internal cerebral veins, and vein of Galen	3 (1.7)
Internal cerebral veins and vein of Galen	2 (1.1)
Isolated superficial cortical veins	2 (1.1)
Superficial cortical veins with other sinuses	3 (1.7)
All superficial and deep venous sinuses	20 (11.1)

Superficial venous sinuses include SSS, TS, and SS. SSS=Superior sagittal sinus, TS=Transverse sinuses, SS=Sigmoid sinuses

Table 4: Comparison of patients with cerebral venous sinus thrombosis with/without brain parenchymal lesions

	Group 1 (without brain parenchymal lesions=65)	Group 2 (with brain parenchymal lesions=116)	*P*
Age (years)	36.06±14.89	33.84±14.54	0.33
Male/female	28/37	32/84	0.034
Headache	57	29	<0.001
Seizure	4	40	<0.001
Altered sensorium	3	24	0.007
Focal neurological deficit	0	22	<0.001
Vertigo	1	1	0.75
SSS thrombosis	34	75	0.14
TS thrombosis	51	90	0.96
SS thrombosis	44	77	0.99
Straight sinus thrombosis	6	31	0.009
Internal cerebral veins and vein of Galen thrombosis	1	11	0.08
Superficial cortical veins thrombosis	0	5	0.22

SSS=Superior sagittal sinus, TS=Transverse sinuses, SS=Sigmoid sinuses

Mangshetty and Reddy, 54% of patients had focal deficits.[11] Among them, 20 had hemiparesis and 9 had aphasia. Seizures are far more frequently seen in CVST than in arterial stroke with a frequency of 35%–50%.[16-18] Seizures were present in 24.31% in the present study. In a retrospective study by Kalita *et al*., the presence of a supratentorial parenchymal lesion on MRI was independently associated with a higher risk of presenting seizures on multivariate analysis.[19] The mental status in CVST may vary from no change in alertness, developing mild confusion, or progressing to coma. Earlier case series reported higher incidence of altered sensorium at presentation (43%–93%).[20-22] Similar results have been shown in some other studies (57.53% and 52% by Nagaraja *et al*. and Mangshetty and Reddy, respectively).[11,23] Lesser number of patients had altered sensorium at presentation in some recent studies.[12] Altered sensorium is less common in the present study (14.92%). The reason for this decline is probably patient's awareness and early diagnosis.

Brain parenchyma was normal in 35.9% patients in the present study. Hemorrhagic venous infarct was the most common brain parenchymal lesion followed by nonhemorrhagic venous infarct. Eleven patients had intraparenchymal hemorrhage. In a study from India, hemorrhagic venous infarct was present in 45.6% and 0.7% had SAH.[12] Khaladkar *et al*. reported 40 patients of CVST, half of them had normal brain parenchyma.[13] Hemorrhagic venous infarct was the most common brain parenchymal lesion (35%). Tsai *et al*. described the spectrum of MRI findings in CVST. In initial stages, there is a mild increase in dural venous sinus pressure, and only sinuses are affected without parenchymal abnormalities.[24] With further increase in intracranial pressure, focal neurological deficit takes place and affected brain region shows edema or infarction with or without hemorrhage in 10%–50% of cases. In a recent study of 44 patients with CVST, concomitant cortical vein thrombosis was associated with severe clinical manifestations, poor short-term outcomes, and brain lesions.[25] SAH has rarely been reported in association with CVST. One patient was described by Sztajzel *et al*. who presented with the right cerebellar SAH secondary to thrombosis of the right TS or SS.[26] This patient subsequently developed hemorrhagic infarction of the cerebellum. Three patients of SAH associated with isolated cortical venous thrombosis were reported by Chang and Friedman.[27] No associated parenchymal abnormalities were identified in all three patients. All the three patients had isolated thrombosis of a cortical vein, probably the vein of Trolard. The mechanism behind the development of SAH in isolated cortical venous thrombosis is not certain. In a large study from India, 3 out of 392 patients (0.7%) had SAH.[12] In the present study, one (0.55%) patient had SAH without other brain parenchymal lesions and eight had SAH along with other brain parenchymal abnormalities.

Multiple dural venous sinuses were involved in most of the patients. The involvement of deep venous sinuses and cortical veins is much less than superficial dural venous sinuses. The most common involved sinus is the SSS followed by TS in most of the studies. In a study by Narayan *et al*., SSS was involved in 54.3% and TS in 47.7%.[12] In contrary, TS was the most common involved sinus (83.9%) followed by SS (44.6%) and SSS (35.7%) in a study by Zubkov *et al*.[28] Similar findings were observed in the present study. The ISCVT trial had similar observation.[2] Lateral sinus thrombosis was present in 85.9% and SSS thrombosis in 62%. Deep cerebral venous system includes the vein of Galen, internal cerebral vein, and straight sinus. Approximately 10%–26% of patients with CVST have thrombosis of the deep cerebral venous system, which can lead to thalamic or basal ganglia infarction.[2,11-13] Deep venous system was involved in 21.5% and superficial venous system in 2.8% of cases in the present study. Cortical vein thrombosis is uncommon, and specific clinical syndromes related to the larger cortical veins are rarely seen (e.g., temporal lobe hemorrhage associated with vein of Labbe thrombosis).[29]

Recently, 7048 reported cases of cortical veins and sinus thrombosis were analyzed from all around the world.[30] In this largest pooled published data, CVST cases' demographics, etiology, clinical features, radiological presentation, and mortality were compared from one continent to other. Overall, male-to-female ratio was 1:2.2 and headache was the most common presenting symptom. The most common site of thrombosis was TS. In Asian subcontinent, females were more commonly affected and headache was the most common presentation. The most common site of thrombosis was SSS followed by TS-SS-Internal jugular vein.[30] Similarly, in the present study, females were most commonly affected and headache was the most common presenting symptoms. In the contrary, TS was most commonly affected sinus in this study compared to SSS. This difference of involved sinus cannot be explained as there is small sample size in the present study compared to large pooled published data.

In the study by Zubkov *et al*., the comparison between CVST patients with (Group 2) and without brain parenchymal lesions (Group 1) was done.[28] Thirty-seven (66.1%) out of 56 patients had no brain parenchymal lesions. Women predominated in both the groups. There was no significant age difference between both groups. Internal cerebral veins, vein of Galen, and isolated cortical vein thrombosis were always associated with brain parenchymal lesions in that study.[28] On the contrary, the present study had significantly more women in Group 2. The mean age of both groups had no significant difference. Headache as the presenting symptom was more common in Group 1, while seizures, altered sensorium, and focal neurological deficit were significantly more common in Group 2. In contrast to the previous study, straight sinus thrombosis was more common in Group 2. Involvement of rest of cortical venous sinuses and cortical veins was not significantly different among both the groups. Female gender, seizures, altered sensorium and focal neurological deficit at presentation, and straight sinus thrombosis were found to be associated with brain parenchymal lesions and serve as predictors in the present study. Intracranial venous hypertension undoubtedly plays an important role in the pathophysiology of brain parenchymal changes. Increased

intraluminal venous pressure causes decrease in cerebral blood flow and cerebral perfusion pressure. This might induce an energy failure and a disruption of the blood–brain barrier that results in vasogenic edema and hemorrhagic transformation from increased venous pressure. This can explain more severe clinical presentation in Group 2 in the present study.

Limitations

The retrospective nature of the study is the major limitation. Other limitations are single-center study, lack of etiology, outcome, and follow-up data. Single-center study may cause case selection bias. Reporting by single radiologist in the present study may also cause bias in the interpretation.

Conclusion

CSVT is an important and treatable cause of stroke with a wide spectrum of symptoms and signs. Women are affected more, especially in the third decade of life. Headache is the most frequent presenting symptom followed by seizures. Hemorrhagic venous infarct is the most common brain parenchymal lesion, and the frontal region is the most common site followed by frontoparietal. TS is the most common site of venous sinus thrombosis. Headache is the most common presentation in patients with CVST without brain parenchymal lesions, while seizure is the most common with brain parenchymal lesions. Female gender, seizures, altered sensorium and focal neurological deficit at presentation, and straight sinus thrombosis are predictors of brain parenchymal lesions. Prompt diagnosis and treatment of CSVT may prevent potential serious damage to the brain parenchyma.

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Conflicts of interest

There are no conflicts of interest.

References

1. Stam J. Thrombosis of the cerebral veins and sinuses. N Engl J Med 2005;352:1791-8.
2. Ferro JM, Canhão P, Stam J, Bousser MG, Barinagarrementeria F; ISCVT Investigators, *et al.* 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.
3. Wasay M, Bakshi R, Bobustuc G, Kojan S, Sheikh Z, Dai A, *et al.* Cerebral venous thrombosis: Analysis of a multicenter cohort from the United States. J Stroke Cerebrovasc Dis 2008;17:49-54.
4. Yuh WT, Simonson TM, Wang AM, Koci TM, Tali ET, Fisher DJ, *et al.* Venous sinus occlusive disease: MR findings. AJNR Am J Neuroradiol 1994;15:309-16.
5. Dormont D, Anxionnat R, Evrard S, Louaille C, Chiras J, Marsault C, *et al.* MRI in cerebral venous thrombosis. J Neuroradiol 1994;21:81-99.
6. Fries G, Wallenfang T, Hennen J, Velthaus M, Heimann A, Schild H, *et al.* Occlusion of the pig superior sagittal sinus, bridging and cortical veins: Multistep evolution of sinus-vein thrombosis. J Neurosurg 1992;77:127-33.
7. Bergui M, Bradac GB, Daniele D. Brain lesions due to cerebral venous thrombosis do not correlate with sinus involvement. Neuroradiology 1999;41:419-24.
8. Lafitte F, Boukobza M, Guichard JP, Hoeffel C, Reizine D, Ille O, *et al.* MRI and MRA for diagnosis and follow-up of cerebral venous thrombosis (CVT). Clin Radiol 1997;52:672-9.
9. Mehta SR, Varadarajulu R, Gupta A, Kumaravelu S. In: Joshi SR, Sainani GS, Joshi VR, Anand P, Mynadkar, Rao M, *et al*., editors. abstracts of 59th annual conference of API 2004. JAPI 2003;51:1196.
10. Ameri A, Bousser MG. Cerebral venous thrombosis. Neurol Clin 1992;10:87-111.
11. Mangshetty B, Reddy KN. Clinical and neuroimaging correlation in patients with cerebral sinus venous thrombosis. Al Ameen J Med Sci 2015;8:64-71.
12. Narayan D, Kaul S, Ravishankar K, Suryaprabha T, Bandaru VC, Mridula KR, *et al.* Risk factors, clinical profile, and long-term outcome of 428 patients of cerebral sinus venous thrombosis: Insights from Nizam's Institute Venous Stroke Registry, Hyderabad (India). Neurol India 2012;60:154-9.
13. Khaladkar SM, Thakkar DK, Thakkar DK, Shrotri H, Kulkarni VM. Cerebral venous sinus thrombosis on MRI: A case series analysis. Med J DY Patil Univ 2014;7:296-303.
14. Daif A, Awada A, Al-Rajeh S, Abduljabbar M, Al-Tahan AR, Obeid T, *et al.* Cerebral venous thrombosis in adults. A study of 40 cases from Saudi Arabia. Stroke 1995;26:1193-5.
15. Karthikeyan D, Vijay S, Kumar T, Kanth L. Cerebral venous thrombosis-spectrum of CT findings. Indian J Radiol 2004;14:129-37.
16. Ferro JM, Correia M, Pontes C, Baptista MV, Pita F; Cerebral Venous Thrombosis Portuguese Collaborative Study Group (Venoport), *et al.* Cerebral vein and dural sinus thrombosis in Portugal: 1980-1998. Cerebrovasc Dis 2001;11:177-82.
17. Ferro JM, Correia M, Rosas MJ, Pinto AN, Neves G; Cerebral Venous Thrombosis Portuguese Collaborative Study Group[Venoport], *et al.* Seizures in cerebral vein and dural sinus thrombosis. Cerebrovasc Dis 2003;15:78-83.
18. Masuhr F, Busch M, Amberger N, Ortwein H, Weih M, Neumann K, *et al.* Risk and predictors of early epileptic seizures in acute cerebral venous and sinus thrombosis. Eur J Neurol 2006;13:852-6.
19. Kalita J, Chandra S, Misra UK. Significance of seizure in cerebral venous sinus thrombosis. Seizure 2012;21:639-42.
20. Bansal BC, Gupta RR, Prakash C. Stroke during pregnancy and puerperium in young females below the age of 40 years as a result of cerebral venous/venous sinus thrombosis. Jpn Heart J 1980;21:171-83.
21. Nagpal RD. Dural sinus and cerebral venous thrombosis. Neurosurg Rev 1983;6:155-60.
22. Nagaraja D, Taly AB, Das S. Puerperal cerebral venous thrombosis. In: Sinha KK, Chandra P, editors. Progress in Clinical Neurosciences. Ranchi: NSI Publication; 1987. p. 325-38.
23. Nagaraja D, Sarma GR. Treatment of cerebral sinus/venous thrombosis. Neurol India 2002;50:114-6.
24. Tsai FY, Wang AM, Matovich VB, Lavin M, Berberian B, Simonson TM, *et al.* MR staging of acute dural sinus thrombosis: Correlation with venous pressure measurements and implications for treatment and prognosis. AJNR Am J Neuroradiol 1995;16:1021-9.
25. Liang J, Chen H, Li Z, He S, Luo B, Tang S, *et al.* Cortical vein thrombosis in adult patients of cerebral venous sinus thrombosis correlates with poor outcome and brain lesions: A retrospective study. BMC Neurol 2017;17:219.
26. Sztajzel R, Coeytaux A, Dehdashti AR, Delavelle J, Sinnreich M. Subarachnoid hemorrhage: A rare presentation of cerebral venous thrombosis. Headache 2001;41:889-92.
27. Chang R, Friedman DP. Isolated cortical venous thrombosis presenting as subarachnoid hemorrhage: A report of three cases. AJNR Am J Neuroradiol 2004;25:1676-9.
28. Zubkov AY, McBane RD, Brown RD, Rabinstein AA. Brain lesions in cerebral venous sinus thrombosis. Stroke 2009;40:1509-11.
29. Jones BV. Case 62: Lobar hemorrhage from thrombosis of the vein of labbé. Radiology 2003;228:693-6.
30. Maali L, Khan S, Qeadan F, Ismail M, Ramaswamy D, Hedna VS, *et al.* Cerebral venous thrombosis: Continental disparities. Neurol Sci 2017;38:1963-8.