ORIGINAL RESEARCH PAPER

Volume-7 | Issue-1 | January-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CEREBRAL VENOUS SINUS THROMBOSIS – A REVIEW OF 90 CASES

Medicine

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ABSTRACT

INTRODUCTION : Cerebral venous sinus thrombosis is a life threatening and rare condition, responsible for less than 1% of strokes. It is an under-diagnosed condition and prompt diagnosis requires a high degree of suspicion and use of relevant neuroimaging modalities.

OBJECTIVES : To evaluate patients with Cerebral venous sinus thrombosis (CVST) and characterize the risk factors, clinical manifestations, treatment and clinical course.

METHODS : In a retrospective cross-sectional study, 90 patients with proven diagnosis of CVST admitted in a tertiary care hospital from 2014 to 2017 were evaluated. The diagnosis of CVST was based on MRI, MRV, CT, CTV and conventional angiography. Statistical analysis was performed using R software**.**

RESULTS : The most common presenting complaint was headache, followed by vomiting and focal neurological deficit. In most of the patients the diagnosis of CVST was made on MRI and MRV. The mean time to diagnosis was 6 days. The most affected cerebral venous sinus was transverse sinus followed by superior sagittal sinus and sigmoid sinus. All patients were treated with low molecular weight heparin (LMWH) followed by warfarin. Most of the patients had complete recovery.

CONCLUSION: Strong clinical suspicion and appropriate imaging plays a key role in the diagnosis of CVST. MRI along with MRV is the diagnostic modality of choice with CT Venography a good alternative to MRI in case of non-availability of the same. The mainstay of treatment is systemic anticoagulation with LMWH in the acute phase followed by oral anticoagulation.

KEYWORDS

Cerebral venous sinus thrombosis, Magnetic resonance imaging, Magnetic resonance Venography, Low molecular weight heparin

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is thrombosis of cerebral veins and cerebral venous sinuses. It is a rare condition and is responsible for less than 1% of strokes (1). The disease is diagnosed on a strong clinical suspicion with corroborative neuroimaging. However, it has a varied clinical presentation and findings on computed tomography (CT) and magnetic resonance imaging (MRI) may differ quiet a lot. It is a disease of young adults with a male/ female ratio of 1.5-5 (2).

Headache is the commonest complaint with other common presentations being seizures, altered consciousness and focal neurological deficits (3). MRI and magnetic resonance venography (MRV) are noninvasive modalities of choice. CT venography (CTV) may be used as an alternative if MRI is not available. Digital subtraction cerebral angiography, which was previously the gold standard, has now been superseded by CTV and MRV (4). The radiologist has a key role in not only confirming the diagnosis by demonstrating the thrombus but also look for signs of cerebral venous ischemia and also attempt to show any pathology related with the thrombosis (5).

The aim of this study was to evaluate patients with CVST seen in a tertiary care hospital, characterize the risk factors, clinical manifestations, treatment and clinical course.

METHODS

This study was a retrospective cross-sectional study of patients with CVST admitted in a tertiary care hospital from 2014 to 2017. 90 patients with proven diagnosis of CVST based on MRI, MRV, CT, CTV and conventional angiography were included in the study. The data related to demographics, clinical variables, exposure to high altitude area was obtained from the medical records. Neuroimaging was done in Philips Achieva 1.5 T MR Scanner and Philips Brilliance 16 Slice CT Scanner. Data on clinical manifestations and time to diagnosis (time from the onset of symptoms to diagnosis of CVST) was determined. Further, risk factors, radiological manifestations, treatment and clinical outcome were determined.

Statistical analysis was performed using R software**.** Nominal variables were summarized by proportions and variables on continuous/discrete scale were summarised by mean and standard deviation. Approval for the study was obtained from our institutional ethics committee.

RESULTS

Of the 90 patients included in the study, 82 (91.1%) were males and 8 (8.9 %) were females. The minimum age was 6 years and the maximum 74 years with mean age of 34.3 years. The most common presenting complaint (Table 1) was headache in 68 patients (76%), with other common presentations being vomiting in 46 patients (51%) and focal neurological deficits in 39 patients (43%). The most important etiological factor was high altitude exposure, which was present in 52 patients (58%), with other risk factors being prothrombotic hematological conditions (12%), dehydration (8 %) and use of oral contraceptive pills (7%).

In most of the patients the diagnosis of CVST was made on MRI and MRV. In a few patients who were transferred to our hospital from a peripheral hospital, the diagnosis of CVST was made on CT and CTV due to non-availability of MRI. Only one patient had to undergo conventional angiography to establish the diagnosis of CVST. The mean time to diagnosis was 6 days (range 0 to 20 days).

Table 1 – Clinical Presentation

S.No.	Symptoms	Number of cases (Percentage)
1.	Headache	68 (76%)
2.	Vomiting	46 (51%)
3.	Seizures	27 (30%)
4.	Focal neurological deficit	39 (43%)
5.	Altered sensorium	25 (28%)

The most affected cerebral venous sinus (Table 2) was transverse sinus, which was thrombosed in 73 patients (81%), with left transverse sinus involved in 33 patients and right transverse sinus in 24 patients. Superior sagittal sinus and sigmoid sinus were thrombosed in 60 patients (67%) each. Cortical veins were involved in 40 patients (44%). Venous infarcts were seen in 53 patients (59%) with midline shift seen in 12 patients (13%).

All patients were treated with low molecular weight heparin (LMWH) followed by warfarin. The follow up of the patients was from 3 to 26 months. 77 patients (86%) had complete recovery and 7 patients (8%) had partial recovery. 3 patients had severe sequelae like visual loss, motor deficit, seizures and 3 patients died during the course of treatment in the hospital.

Table 2 – Site of thrombosis

S. No.	Name of the sinus	Number of cases (Percentage)
1.	Superior sagittal sinus	60 (67%)
2.	Transverse sinus	73 (81%)
2a.	Bilateral transverse sinuses	16 (18%)
2b.	Right transverse sinus	24 (27%)
3b.	Left transverse sinus	33 (36%)
3.	Sigmoid sinus	60 (67%)
3a.	Bilateral sigmoid sinuses	4 (5%)
3b.	Right sigmoid sinus	22 (24%)
3c.	Left sigmoid sinus	34 (38%)
4.	Internal jugular veins	35 (39%)
4a.	Bilateral internal jugular veins	1 (1%)
4b.	Right internal jugular vein	16 (18%)
4c.	Left internal jugular vein	18 (20%)
5.	Cortical veins	40 (44%)
6.	Straight Sinus	18 (20%)
7.	Cavernous sinus	2 (2.2%)
8.	Internal Cerebral vein	5 (5.5%)
9.	Vein of Galen	10 (11.1%)

DISCUSSION

CVST is a rare form of venous thromboembolism and its etiology is multifactorial (6). It is responsible for less than 1% of strokes and affects all age groups with estimated annual incidence of 3-4 cases per million people (7). CVST is being recognized increasingly now because of the greater awareness and availability of CT and MRI.

Many congenital and acquired risk factors have been described with commonest being congenital thrombophilia, head trauma, high altitude exposure, oral contraceptive pills, brain tumors, pregnancy and puerperium (8). In our series, majority of the patients (58 %) had history of high altitude exposure. Our hospital was the tertiary referral center of few peripheral hospitals located in high altitude area and that is why the incidence of exposure to high altitude was more in our study. Majority of the patients were males in our study because they were occupationally employed in high altitude area.

Clinical features of CVST are relatively nonspecific and the presentation is subacute with the symptoms developing over many days (9). The common presentations include headache, vomiting, seizures, focal neurological deficits and altered sensorium with the less common being blurring of vision, cranial nerve palsies and coma (10). In our study, the commonest presentation was with headache (76 %) followed by vomiting (51%).

Although the clinical suspicion needs to be high in establishing CVST, neuroimaging plays a key role in its diagnosis. About 50% cases of CVST progress to cerebral venous infarction. Obstruction of venous drainage leads to increased venous pressure and subsequently venous infarction (11). The imaging signs of CVST may be direct or indirect. The direct sign is visualization of the thrombus on imaging. The indirect signs include differential appearance of the involved cerebral parenchyma, loss of gray-white matter differentiation, focal hemorrhaging areas, white matter edema, sulcal effacement, etc (12).

CT is the initial modality to evaluate a patient with CVST because of widespread availability and lower cost. On non-contrast CT, thrombus may be visualized in the venous channel as a high attenuation lesion giving "dense triangle" or "cord" sign. The brain lesions correspond to venous distribution and venous infarction is seen as a hypodense lesion with or without hemorrhages. On contrast-enhanced CT, intraluminal thrombus surrounded by contrast gives rise to "empty delta" sign (13). CT venography with multiplanar reformatted images is a reliable and fast modality to diagnose CVST with a sensitivity of 95% (14).

On MRI, thrombus may be seen as absent flow void and as altered signal intensity in it. Depending on the age of the thrombus, the signal characteristics change, especially in the acute and sub-acute stages (Fig 1). Parenchymal abnormalities are well visualized on MRI with venous infarct appearing hypointense on T1 weighted images and hyperintense on T2 weighted images (Fig 2). The hemorrhages within the infarct may show signal blooming on gradient-recalled echo (GRE) images (Fig 2, 3). MRV (Fig 1, 2, 3, 4) along with MRI is presently the noninvasive modality of choice in diagnosing CVST. Time of flight (TOF) and phase contrast (PC) techniques use MR flow phenomena for generation of contrast and hence may show flow related artifacts. Contrast enhanced MRV is less prone to flow related artifacts as it depends on filling of lumen by contrast (7).

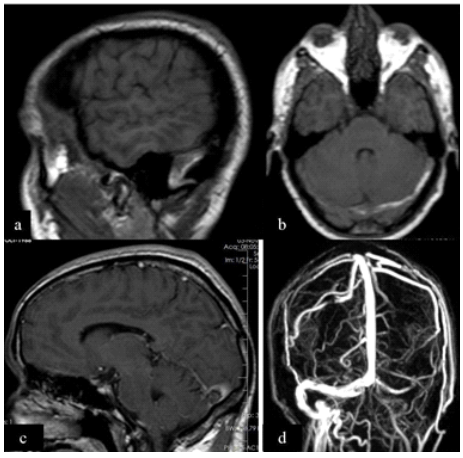

Fig 1

a- T1 W sagittal image heterogeneously hyperintense filling defects in left sigmoid sinus.
b - T1 W axial image heterogeneously hyperintense filling defects in left transverse and left sigmoid sinuses.
c – Contrast enhanced T1 W sagittal image showing filling defects in the left transverse sinus.
d – MRV showing non-visualized left transverse and left sigmoid sinuses.

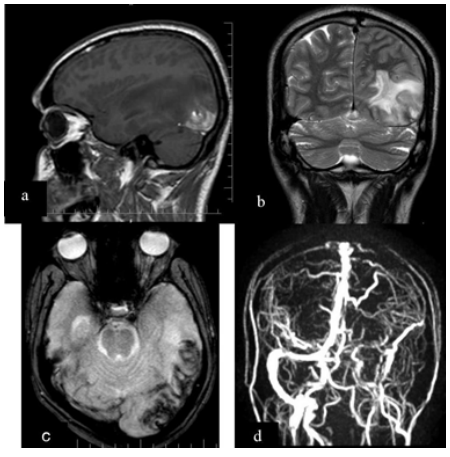

Fig 2

a – T1W sagittal image showing hyperintense filling defect in left transverse sinus with hemorrhagic infarct in left occipital lobe
b– T2 Coronal image showing hyperintense filling defect in left transverse sinus with hemorrhagic infarct in left occipital lobe
c- GRE axial image showing hemorrhagic venous infarct in left temporal and left occipital lobes with signal blooming.
d – MRV showing non-visualized left transverse sinus, left sigmoid sinus and proximal left internal jugular vein.

Digital subtraction angiography (DSA) is now rarely required for diagnosis of CVST. However, it may be of use in confirming the diagnosis of isolated cortical venous thrombosis in which indirect signs like delayed local venous drainage or collateral venous pathways may be seen (15). DSA may also be of use in patients with CVST along with dural arteriovenous malformation where it helps in appropriate treatment planning.

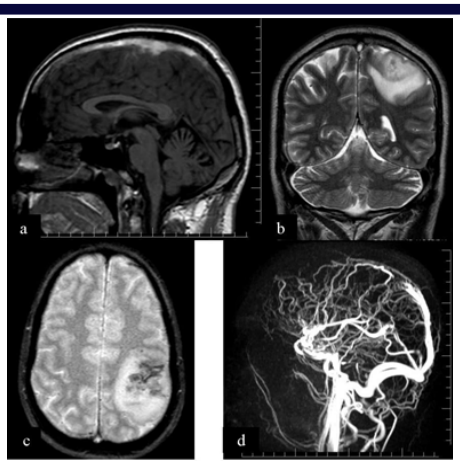

Fig 3

a – T1W sagittal image showing hyperintense filling defects in the superior sagittal sinus.
b – T2 Coronal image showing hyperintense filling defect in superior sagittal sinus with venous infarct in left fronto-parietal lobes.
c- GRE axial image showing hemorrhagic venous infarct in left fronto-parietal lobes with signal blooming.
d – MRV showing non-visualized superior sagittal sinus and some superficial cortical veins draining into the superior sagittal sinus.

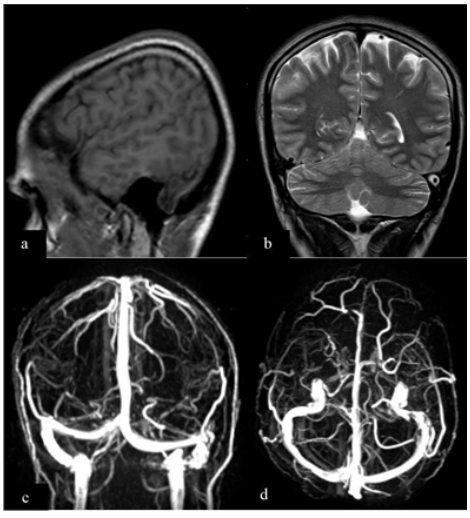

Fig 4

a – T1W sagittal image showing hypointense filling defects in the left sigmoid sinus.
b – T2 Coronal image showing hyperintense filling defects in the left sigmoid sinus with a flow void – suggestive of old CVT with partial recanalization.
c & d – MRV showing old partially recanalized CVT involving left sigmoid sinus with a few adjacent collateral channels.

The most commonly involved sinus in our study was transverse sinus (81%), followed superior sagittal sinus (67%) and sigmoid sinus (67%). Wyokinska et al (16) showed that transverse sinus was involved in 79%, sigmoid in 50% and superior sagittal sinus in 49% of their patients.

General measures in the treatment of CVST include appropriate headboard inclination, protection of airway, adequate oxygenation and seizure prevention and treatment. Medical management of CVST involves systemic anticoagulation with low molecular weight heparin (LMWH). LMWH is the drug of choice not only because of its efficacy and safety but also because it has been shown to reduce both morbidity and mortality (17). After the acute phase, the patient should be shifted over to appropriate oral antiocoaguation. Most of our patients were treated with low molecular weight heparin (LMWH) followed by warfarin.

Endovascular management is with mechanical thrombolysis or locally infused rt-PA and is advised only in cases showing worsening inspite of adequate anticoagulation (18). Surgical management is reserved for severe cases and includes thrombectomy, decompressive craniectomy and evacuation of ICH.

With the advent of neuroimaging, the mortality rates have come down drastically in cases of CVST. Strong clinical suspicion, prompt diagnosis and early initiation of anticoagulation have tremendously improved the prognosis. The outcome with regards to function is better than arterial stroke (19).

Our study has a few limitations. Being a retrospective study, the possibility of recording bias and under-evaluation of pertinent clinical information is likely. Further, there is a possibility that cases were under-represented in a few situations. Milder cases of CVST who presented with headache may not have been evaluated completely in view of low clinical suspicion. Further, some cases of extensive ICH, which subsequently had a poor prognosis, may not have been evaluated for CVST as one of the etiological factors.

CONCLUSION

The presentation of CVST is non-specific and many neurological conditions may mimic it. Strong clinical suspicion and appropriate imaging plays a key role in its diagnosis. MRI along with MRV is the diagnostic modality of choice with DSA reserved only for a few cases. The mainstay of treatment is systemic anticoagulation with LMWH in the acute phase followed by oral anticoagulation. In recent times, the morbidity and mortality associated with CVST has come down drastically.

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