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Case Series

Internal Medicine Section

Clinical Spectrum and Outcomes of Deep Cerebral Venous Sinus Thrombosis: A Case Series

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ABSTRACT

Cerebral Venous Sinus Thrombosis (CVST) is a rare disorder that can lead to significant morbidity and mortality. Thrombosis can occur in superficial veins, the deep venous system or the cortical veins of the brain. The diagnosis of CVST is often missed because of its heterogeneous presentation. Headache is the most common presenting symptom. Hemiparesis, seizures, and impaired level of consciousness can occur in many cases. Herein, the authors present four cases of CVST with distinct clinical presentations. One of our patients presented with fever and altered sensorium, which initially misled the diagnosis as viral encephalitis. All these cases were diagnosed to have Deep CVST (DCVST), having bilateral thalamic involvement on Magnetic Resonance Imaging (MRI) brain. The risk factors for provoked CVST include pregnancy, early postpartum period, use of oral contraceptive drugs, infections and dehydration. Use of oral contraceptives was identified as the underlying risk factor in one of the four cases described in this case series. In unprovoked CVST, underlying procoagulant conditions (Factor VIII, Protein C/Protein S, and Antithrombin III deficiency) are the aetiological factors. MRI of the brain with Magnetic Resonance Venogram (MRV) of the Brain is the current diagnostic modality of choice for CVST, with very high sensitivity and specificity. All of our patients had favourable outcome with conservative management, so early diagnosis and prompt management is helpful in reducing morbidity and mortality in DCVST.

Keywords: Deep cerebral veins, Headache, Intracranial sinus thrombosis

INTRODUCTION

The CVST, which includes thrombosis of the cerebral veins and the dural sinuses, is a rare disorder that can lead to significant morbidity and mortality. CVST accounts for 10-20% of strokes in young adults in India. Young adults account for nearly 30% of all stroke cases in India [1]. The overall incidence of CVST is less than 0.05%, indicating that it is a relatively rare neurological condition. The incidence of CVST before 2010 was 4.9 per 10,000 cases, which increased to 9.6 per 10,000 cases after 2010 [2]. An increase in the overall incidence of CVST cases is probably due to a higher index of clinical suspicion and improved diagnostic imaging.

Global data from multiple registries indicate a strong female gender predilection. However, recent data have shown a reversal in gender predilection from being a condition with female preponderance to one where equal or more men are being affected. Improved maternal health and lower incidence of postpartum CVST cases could be the driving factors [2]. Important clinical features that suggest CVST include new-onset focal headache, headache onset with seizures, papilledema, or focal deficits. Prothrombotic conditions are the most common risk factor for unprovoked CVST. It is one of the treatable and reversible causes of stroke in young patients. Management with Low Molecular Weight Heparin (LMWH), overlapping with oral anticoagulation, shows excellent clinical outcomes [1]. Herein, we report four cases of CVST, who presented with different clinical scenario and all of them responded well with treatment.

CASE SERIES

Case 1

A 46-year-old female presented to the Medical Emergency Department with complaints of slurring of speech followed by altered sensorium. She also had low-grade fever (99.8°F) of one day duration. She took various over-the-counter medications for these complaints, but her symptoms did not abate. She did not have any other co-morbidities. However, she had history of oral contraceptive pill intake for last three months.

On examination, she had normal vitals, Glasgow Coma Scale (GCS) of 10 (E2V4M4), and NIHSS (NIH stroke scale) score was 19. On neurological examination there was no motor deficit with the presence of a lateralising sign (extensor plantar response on right). The rest of the systemic examination was normal.

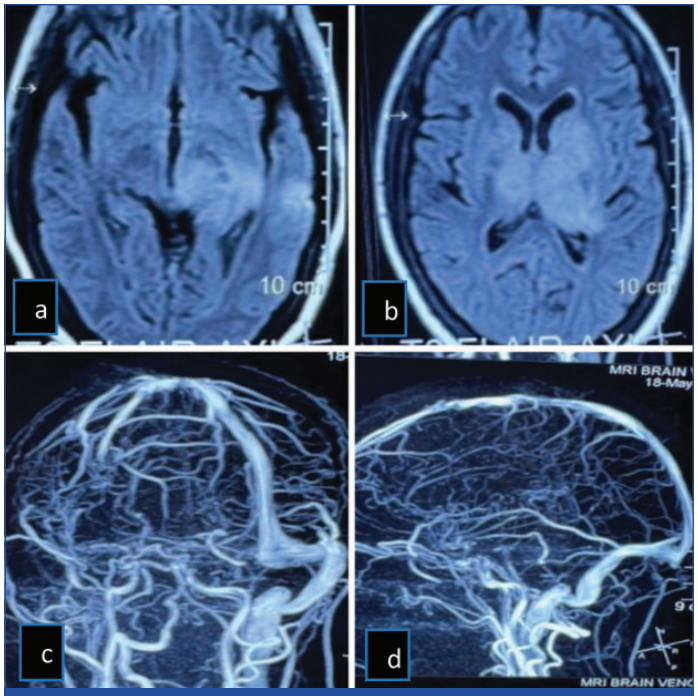
Routine blood investigations like complete haemogram, renal function tests, liver function tests, and serum electrolytes were normal. Non Contrast Computed Tomography (NCCT) of the head was normal. A possibility of viral encephalitis was kept. A lumbar puncture was performed, and Cerebrospinal Fluid (CSF) analysis showed slightly elevated proteins (52 mg/dl). Other tests of CSF, such as Adenosine Deaminase (ADA) for tuberculosis, Japanese Encephalitis (JE), and Herpes Simplex Virus (HSV) were also normal.

The MRI of the brain showed multiple hyperintense lesions on T2-weighted and Fluid Attenuated Inversion Recovery (FLAIR) sequences in bilateral thalami, left cerebral peduncle, basal ganglia, left temporal lobe and periventricular white matter showing diffusion restriction on Diffusion Weighted Imaging (DWI). Subsequently, MRV Brain was done, which clinched the diagnosis. MRV showed thrombosis of the straight sinus, Left transverse sinus, sigmoid sinus and proximal Internal Jugular Vein (IJV) thrombosis, the posterior aspect of the inferior sagittal sinus and the great vein of Galen [Table/Fig-1].

Prothrombotic work-up (Protein C, Protein S, Anti thrombin III, Anti Phospholipid Antibody, Factor VIII, serum homocysteine) was done, which came out to be normal. She was started on bridging therapy of anticoagulation with Enoxaparin 60 mg subcutaneous twice daily and oral Warfarin 5 mg daily. She showed a marked improvement in sensorium and had a GCS of 15 by the seventh day of treatment. She was discharged on oral warfarin (5 mg once daily) and was advised to maintain an International Normalised Ratio (INR) between two to three. The patient is in regular follow-up and is stable till six months follow-up visit.

Case 2

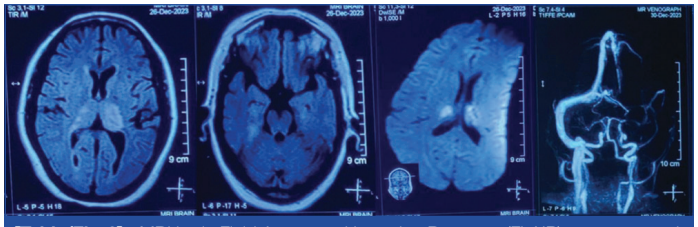
A 45-year-old male presented with severe headache, and left-sided weakness for five days, followed by altered sensorium and two



[Table/Fig-1]: (a,b) MRI Brain FLAIR Sequence showing hyperintense lesions involving bilateral thalami, left cerebral peduncle , basal ganglia, left temporal lobe and periventricular white matter; (c,d): MR venogram brain showing absent flow in straight sinus, left transverse sinus, sigmoid sinus and proximal IJV thrombosis, posterior aspect of inferior sagittal sinus and great vein of galen.

episodes of Generalised Tonic Clonic Seizures (GTCS). There were no associated comorbidities.

On examination, his pulse rate was 94/minute and regular, and his blood pressure was 110/70 mmHg, he was afebrile, had a GCS of 11(E3V4M4) and NIHSS score was 18. On neurological examination cranial nerves were normal including fundus. He had a power of 3/5 in left upper and lower limb along with extensor plantar response on the left. The rest of the systemic examination was normal. Routine blood investigations like complete haemogram, renal function tests, liver function tests, and serum electrolytes were normal. NCCT of the head was done, which was normal. Further MRI of the brain was done, which showed multiple areas of T2 and FLAIR hyperintensities in the right medial temporal lobe and bilateral thalami along with haemorrhagic conversion. Subsequently, an MR Venography Brain was done, which clinched the diagnosis. MRV showed thrombosis of the Inferior sagittal sinus, Left transverse and left sigmoid sinus [Table/Fig-2].



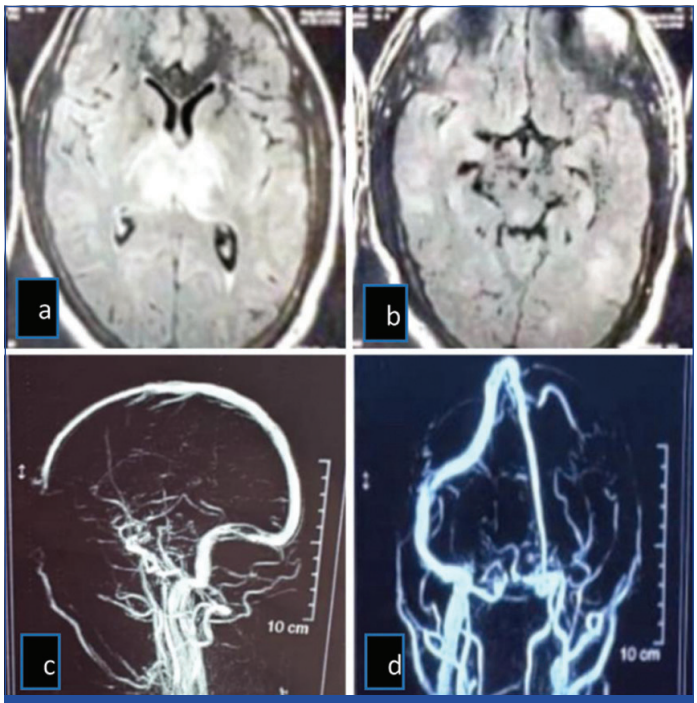
[Table/Fig-2]: MRI brain Fluid Attenuated Inversion Recovery (FLAIR) sequence and Diffusion Weighted Imaging (DWI) sequence showing multiple area of acute infarct seen in right medial temporal lobe, bilateral thalami with haemorrhagic conversion. MR venogram brain showing absent flow in left transverse sinus and sigmoid sinus.

Prothrombotic work-up (Protein C, Protein S, anti-thrombin III, Anti-Phospholipid antibody, Factor V, Factor VIII, serum homocysteine) was done, which showed elevated factor VIII. He was immediately started on bridging therapy of anticoagulation with Enoxaparin 60 mg subcutaneous twice daily and anti-epileptic therapy with Levetiracetam 500 mg twice daily. He showed a marked improvement in sensorium and had a GCS of 15 by the fifth day of anticoagulation. Patient was discharged on oral Warfarin (7.5 mg once daily) and was advised to maintain an INR between two to three. The patient is in regular follow-up and is stable at nine months follow-up.

Case 3

A 35-year-old male presented to the emergency department with complaints of headache and double vision for three days, followed by abnormal behaviour. No comorbidities were present.

On examination, his vitals were normal. He was afebrile, had a GCS of 12 (E3V4M5) and NIHSS score was 16. On neurological examination, he had bilateral lateral gaze palsy with bilateral papilloedema. Routine blood investigations like complete haemogram, renal function tests, liver function tests, and serum electrolytes were also normal. NCCT of the head was normal. MRI of the brain was done, which showed T2 and FLAIR hyperintensities involving bilateral thalami and left medial temporal lobe. Subsequently, an MR venography brain was done, which showed thrombosis of the straight sinus, left transverse sinus, sigmoid sinus and the great cerebral vein of Galen [Table/Fig-3].



[Table/Fig-3]: (a,b): MRI brain showing hyperintensities involving bilateral thalami and left medial temporal lobe; (c,d): MR venogram brain showing absent flow in straight sinus, left transverse sinus, sigmoid sinus, and great vein of Galen.

Prothrombotic work-up (Protein C, Protein S, anti-thrombin III, Anti Phospholipid antibody, Factor V, Factor VIII, serum homocysteine) was done, which showed elevated serum homocysteine level (62 micromol/L).

He was started on bridging therapy of anticoagulation with Enoxaparin 60 mg subcutaneous twice daily and oral Warfarin 5 mg daily. He showed a marked improvement in sensorium. He was discharged on oral Warfarin (5 mg once daily) and was advised to maintain an INR between 2 to 3. The patient is in regular follow-up and is stable at six months follow-up.

Case 4

A 19-year-old female presented with a dull aching, continuous and holocranial headache for 15 days, blurred vision for 10 days and vomiting for three days.

The patient was a student and was unable to continue her routine studies due to discomfort from a persistent headache. Her headache aggravated by exposure to bright light and was occasionally associated with blurred vision. She also had three to four episodes of vomiting per day for last three days. On detailed history taking, we came to know that the patient had been suffering from repeated episodes of headache for the last one year, for which she had been taking over-the-counter medicines from a local chemist.

Patient had a history of irregular and heavily bleeding menstrual cycles for the last three years. She was diagnosed as a case of Polycystic Ovarian Syndrome (PCOS), for which she was taking levonorgestrel 0.15 mg + ethinyl estradiol 0.03 mg for last 12 months. She did not have any other comorbidities.

On general physical examination, she had normal vitals, moderate pallor, no icterus, cyanosis, clubbing, lymphadenopathy or oedema. On neurological examination, higher mental functions were normal. All the cranial nerves functions were intact. There was no motor deficit or sensory loss. All other systems examinations were normal.

On routine investigations, Hb was 6.7 g/dL. The remaining routine blood and urine investigations were normal. Ultrasound pelvis showed bilateral enlarged ovaries with multiple peripherally enlarged small follicles and increased stromal echogenicity suggestive of polycystic ovarian disease.

At this juncture, MRI Brain with MR Venogram Brain was ordered. MRI Brain study was normal. MR Venogram revealed loss of flow in the straight sinus, suggestive of straight sinus thrombosis.

The patient was immediately started on bridging therapy of anticoagulation with Enoxaparin 60 mg subcutaneous twice daily and oral Warfarin 5 mg daily. The patient showed improvement in symptoms by the fifth day of anticoagulation and was discharged on oral Warfarin (5 mg once daily). The patient was advised to maintain an INR between two to three. Patient is in regular follow-up and is stable at six months follow-up. [Table/Fig-4] summarises the clinicodemographic profile, neuroradiological analysis, treatment and outcome of all the four patients.

Case	Age	Sex	Presenting symptoms	MRI brain and MR venogram brain	Aetiology/ risk factor	Treatment	Outcome
1	46 y	Female	Slurring of speech and altered sensorium	Multiple hyperintense lesions in bilateral thalami, left cerebral peduncle, basal ganglia, left temporal lobe and periventricular white matter on MRI Brain with thrombosis of straight sinus, left transverse sinus, sigmoid sinus, proximal IJV, posterior aspect of inferior sagittal sinus and Great cerebral vein of Galen on MR Venogram Brain	Oral contraceptive pills	Anticoagulation with Enoxaparin and Oral Warfarin	Significant improvement within 7 days.
2	45 y	Male	Headache, left sided weakness, altered sensorium and seizures	Hyperintensities in right medial temporal lobe, bilateral thalami with haemorrhagic conversion on MRI brain and thrombosis of inferior sagittal sinus, left transverse sinus and left sigmoid sinus on MR Venogram Brain.	Elevated Factor VIII levels	Antiepileptic treatment, Anticoagulation with Enoxaparin and oral Warfarin	Significant improvement within 5 days.
3	35 y	Male	Headache, double vision and abnormal behaviour	Hyperintensities involving bilateral thalami and left medial temporal lobe on MRI Brain with thrombosis of straight sinus, Left transverse, sigmoid sinus and the great cerebral vein of Galen on MR Venogram Brain	Elevated serum Homocysteine levels.	Anticoagulation with Enoxaparin and oral Warfarin	Complete improvement within 5 days
4	19 y	Female	Headache, blurring of vision and vomiting	Straight sinus thrombosis on MR Venogram Brain	Tablet levonorgestrel 0.15 mg + ethinyl estradiol 0.03 mg	Anticoagulation with Enoxaparin and oral Warfarin	Complete improvement within 5 days.

[Table/Fig-4]: Clinicodemographic profile, neuroradiological analysis, treatment and outcome of the patients.
MRI: Magnetic resonance imaging; MR: Magnetic resonance

DISCUSSION

The CVST is an uncommon cause of stroke. Although there are no population-based studies from India estimating the incidence of CVST, various hospital-based studies have found that CVST comprises 10-20% of all stroke cases in young adults in our country. [1,2]. The superior sagittal sinus is the most affected, followed by the transverse and sigmoid sinuses. The term DCVST refers to thrombosis of the internal cerebral vein, the vein of Galen, the basal vein of Rosenthal, and their tributaries, and is seen in 10% of cases [3]. The parenchymal changes in CVST result from increased venous pressure, which leads to vasogenic oedema, cytotoxic oedema and haemorrhagic infarction. NCCT findings in DCVST often show hypoattenuation of the bilateral thalami and basal ganglia. However, the venous system, which includes the vein of Galen, the vein of Rosenthal, the internal cerebral vein, and the straight sinus, appears hyper attenuated on NCCT. The most sensitive imaging technique for early diagnosis of DCVST is MRI with MRV. The deep venous system drains the thalamus, basal ganglia, corpus callosum, inferior frontal lobes and deep white matter of the parietal and temporal lobes. So, thrombosis of the deep cerebral vein causes vasogenic oedema and ischemic changes in these areas, which appear hyperintense on T2-weighted images and hypointense on T1-weighted images. Based on signal intensity on T1- and T2-weighted images, venous thrombosis can be divided into three stages: initial, intermediate, and late. In the first five days (initial stage), there is no flow-void, and vessels appear isointense on T1-weighted images and hypointense on T2-weighted images. Beyond five days (intermediate stage), there is absent flow with hyperintensity on both T1- and T2-weighted images. After two weeks (late stage), recanalisation occurs, with the resumption of flow-void in abnormal vessels [3,4].

Various predisposing factors combine to result in thrombus formation in cerebral veins. Inherited thrombophilic states, hyperhomocystinemia, dehydration, drugs, pregnancy, malignancy and autoimmune conditions are often found in CVST patients. However, in 15% of cases of CVST, no direct cause or predisposing factor could be identified [5].

Amongst the four cases described, headache was the most common presenting symptom. Headache, being the commonest presentation, is often seen in >80% of patients with CVST and may be the only presentation without any signs of raised intracranial pressure for patients who report early [6]. In present case series, headache was the presenting symptom in three out of four patients. The postulated mechanisms behind headache in CVT includes aseptic inflammatory changes in the course of trigeminal nerve innervation which leads to vasodilatation and dural stretching [6].

The commonest deficit in DCVST is limb weakness, which may present as monoparesis, hemiparesis, or bilateral involvement [7]. One of our patients had left hemiparesis. Due to varied clinical presentations, CVST is often missed or delayed. Headache, hemiparesis, seizures, loss of consciousness, papilloedema and visual disturbances are other commonly observed presentations in CVST. The clinical presentation of DCVST is non specific, and the patient can present with a short history of deteriorating consciousness, aphasia, seizures, nausea, vomiting, coma or death. It is often challenging to differentiate DCVST clinically from other common causes of altered sensorium, like metabolic encephalopathy or meningoencephalitis [7]. Altered level of consciousness or drowsiness has been reported in several studies. A study by Terazzi E et al., described altered consciousness in 61.5% of patients [8].

Another study by Pfefferkorn T et al., described altered sensorium in 72% of cases [9]. In present case series, we had two out of four patients with altered sensorium. The mechanism behind altered sensorium being the extensive involvement of deep grey matter and cortex.

Two out of four patients included here were females. MRI brain and MRV brain confirmed the diagnosis of CVST and DCVST in all these cases. All the patients showed excellent improvement with anticoagulation therapy. MRI involvement of the bilateral thalamus is seen in various other conditions like JE, Wilson's disease, Wernicke's encephalopathy, hypoxic-ischaemic encephalopathy and autoimmune demyelinating conditions like multiple sclerosis. MRI findings of JE closely resemble those of CVST and show a hyperintense lesion in the bilateral thalami on T2-weighted images, while haemorrhagic transformation appears hyperintense on T1-weighted images [10]. The oedema is predominantly cytotoxic rather than vasogenic in CVST. MRI often shows a thrombus in the deep cerebral veins in cases of CVST, which MRV can further confirm. MRV shows abnormal signal intensity in the corresponding sinus, with absent flow. CSF analysis in CVST is often abnormal, showing increased protein levels and lymphocytic pleocytosis that almost mimics the CSF picture of viral meningoencephalitis [11]. So, the initial clinical picture and CSF analysis of CVST can be easily confused with that of meningoencephalitis. However, a negative CSF viral antigen test and MRI help identify DCVST. Any delay in the diagnosis of CVST can be lethal, and the mortality rate increases several-fold if anticoagulation is not started within the initial days. An untreated patient often develops features of raised intracranial pressure and brain herniation, which can lead to permanent residual disability and often death [12].

Acute thrombosis decreases levels of antithrombin, Protein C, and Protein S, so tests for thrombophilic states should be performed at least 6 weeks after an acute thrombotic event [13]. Treatment of CVST remains anticoagulation with heparin/LMWH initially, followed by Warfarin to maintain an INR between two and three. Any underlying correctable cause of hypercoagulability should be treated accordingly. For provoked CVST, the duration of vitamin K antagonist is three to six months. Indefinite therapy is recommended in the presence of severe thrombophilia (protein C or S deficiency, antithrombin deficiency, homozygous factor V Leiden mutation, antiphospholipid syndrome), recurrent CVST and venous thromboembolism after CVST. For remaining cases of CVST, six to twelve months of anticoagulation is recommended [14].

CONCLUSION(S)

A high-index of suspicion is needed for early diagnosis of CVST. It has a favourable outcome if treated early. An MRI of the brain should be performed early in cases of unexplained altered sensorium. If MRI is suggestive of CVST, then MRV must be obtained for confirmation. Any reversible cause of thrombosis should be treated. A study of the clinical profile, early diagnosis using MRI of the brain and MR venogram of the brain, and evaluation for an underlying aetiology helps in better understanding and management of this treatable and reversible disease.

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