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CEREBRAL VENOUS SINUS THROMBOSIS : CLINICAL PROFILE, COAGULATION ABNORMALITIES, AND PROGNOSTIC FACTORS IN AN INDIAN SINGLE-CENTRE COHORT

Key Words: Cerebral Venous Sinus Thrombosis, Puerperium, Coagulation Profile, Modified Rankin Scale.

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ABSTRACT

Background: Cerebral venous sinus thrombosis (CVT) is an uncommon but important cause of stroke, predominantly affecting younger adults and women of reproductive age. Its variable clinical presentation often delays diagnosis, yet early recognition and anticoagulant therapy can significantly improve outcomes.**Aim** To evaluate the clinical, laboratory, and radiological profiles of patients with cerebral venous sinus thrombosis and to identify factors associated with short-term functional outcomes.**Methods:**A prospective observational study was conducted over 18 months in the Department of Internal Medicine, Lokmanya Tilak Municipal Medical College and General Hospital, Mumbai. Fifty patients aged over 18 years with radiologically confirmed CVT were enrolled. Detailed demographic, clinical, laboratory, and imaging data were collected. Functional outcomes were assessed using the modified Rankin scale (mRS) at admission and after 12 weeks of follow-up.**Results:**The mean age group most affected was 41–50 years (42%), with a female predominance (56%). Headache (42%) and altered sensorium (32%) were the most common symptoms, while papilledema was the leading clinical sign (48%). The superior sagittal sinus was the most frequently involved vessel (42%). The puerperal state accounted for 44% of cases, followed by dehydration (12%) and oral contraceptive use (8%). Mortality occurred in 8% of patients, and 90% achieved a good functional outcome (mRS ≤2) at 12 weeks.**Conclusion:**CVT continues to be a major postpartum neurological complication in developing regions. Early neuroimaging and timely anticoagulation are crucial for improving recovery and reducing mortality. Enhanced awareness and broader diagnostic access can facilitate earlier intervention and better prognosis.

INTRODUCTION

Cerebral venous sinus thrombosis (CVT) represents a relatively rare form of cerebrovascular disease that predominantly affects younger adults and women of reproductive age. The condition was first described in the nineteenth century, when it was typically identified post-mortem and characterized by haemorrhagic lesions at the sites of venous occlusion. Modern epidemiological data estimate an annual incidence of around 12 cases per million individuals, though the true rate varies across regions and populations. Women are affected approximately three times more frequently than men, largely due to sex-specific risk factors such as pregnancy and hormonal exposure. Recent observations from North America and South Asia indicate an increasing incidence among older adults and males, suggesting a shift in demographic patterns.[1]

The underlying causes of CVT are diverse and may involve inherited or acquired prothrombotic conditions. Commonly reported risk factors include systemic infections, malignancy, dehydration, hematologic disorders, and abnormalities in the coagulation cascade. Genetic predispositions such as factor V Leiden mutation or deficiencies of protein C, protein S, and antithrombin III also contribute to disease susceptibility. Autoimmune disorders, including systemic lupus erythematosus and polyarteritis nodosa, are recognized causes in younger populations. In many cases, transient hypercoagulable states—such as those occurring in the puerperium or during dehydration—are implicated. Infectious diseases like bacterial or tuberculous meningitis remain relevant etiologies in low- and middle-income countries, while COVID-19–associated coagulopathy has recently emerged as an additional concern.[2]

The clinical presentation of CVT is highly variable, which often complicates timely diagnosis. Septic thrombosis secondary to ear or mastoid infections typically follows an acute course, characterized by fever, systemic illness, and focal neurological deficits. While many patients with CVT may experience headaches, seizures, cranial nerve palsies, urinary disturbances, or signs of elevated intracranial pressure. Because initial non-contrast computed tomographic (CT) scans can appear normal, advanced imaging techniques such as magnetic resonance venography (MRV) or CT venography are recommended to confirm venous occlusion. Early recognition allows prompt anticoagulant therapy, reducing the likelihood of venous infarction and improving neurological outcomes.[2]

Prognosis in CVT ranges from complete recovery to severe disability or death. Most patients achieve favourable outcomes when diagnosed and treated early. However, adverse prognostic factors include coma at presentation, extensive sinus involvement, intracerebral haemorrhage, and concurrent central nervous system infections.[2] The present study seeks to characterize the clinical and laboratory profiles of patients with CVT and to identify variables that predict clinical outcomes.

MATERIALS AND METHODS

Study Design and Setting –

This is a prospective observational study conducted over a

period of 18 months in the Department of Internal Medicine at Lokmanya Tilak Municipal Medical College and General Hospital, Mumbai, a tertiary care centre in India. The study protocol received approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants before enrolment. The research was carried out in accordance with the principles of the Declaration of Helsinki. Reporting of the study adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. [3]

Inclusion and Exclusion Criteria –

Patients aged above 18 years, including postnatal women, who presented for the first time with clinical manifestations suggestive of CVT and whose diagnoses were subsequently confirmed through clinical evaluation and neuroimaging (CT or MRI brain) were eligible for inclusion.

The study excluded antenatal women, patients with coagulopathy secondary to diabetes mellitus, those with multiorgan failure who died before data collection, and individuals with arteriovenous malformation, intracranial space-occupying lesion with haemorrhage, ischemic stroke with haemorrhagic transformation, or haemorrhagic encephalitis. Patients who did not provide written informed consent were also excluded.

Sample Size and Sampling Technique –

A total of 50 consecutive patients fulfilling the inclusion and exclusion criteria were enrolled during the study period. Each patient underwent neuroimaging with CT or MRI of the brain, which was interpreted by qualified radiologists. The diagnosis of CVT was confirmed according to standard radiological criteria. Comprehensive demographic, clinical, laboratory, and radiological details were documented for every patient.

Laboratory Investigations -

All participants underwent a series of baseline laboratory tests, including complete blood count, urine analysis, serum creatinine, alanine aminotransferase, random blood glucose, glycated haemoglobin, serum electrolytes, chest radiography, and a 12-lead electrocardiogram. Selected patients underwent specialized investigations such as coagulation profile, thyroid-stimulating hormone, echocardiography, reverse transcription–polymerase chain reaction (RT-PCR) test for COVID-19, factor V Leiden mutation analysis, protein C and S levels, antithrombin III, serum homocysteine, complement levels (C3, C4), anti-neutrophil cytoplasmic antibody, anti-phospholipid antibody, and antinuclear antibody testing.All patients received supportive care and standard evidence-based management for CVT according to institutional protocols.

Follow-up and Outcome Measures -

Functional outcomes were assessed using the modified Rankin scale (mRS) at the time of admission and again at 12-week follow-up. Outcomes were dichotomized as good (mRS score 0–2) or poor (mRS score ≥3).Follow-up at 12 weeks included reassessment using the mRS, Glassgow coma scale (GCS) and mortality status. In-hospital mortality was recorded separately.Data were collected prospectively using a standardized, pre-validated, semi-structured case record proforma.

Statistical Analysis -

All collected data were coded and entered using Microsoft Excel, and statistical analysis was performed with SPSS version 22.Descriptive statistics were expressed as frequencies, percentages, and means, standard deviations (SD), and medians to describe the central tendency and dispersion of quantitative variables. Appropriate inferential statistical tests were applied as required to analyse associations between clinical and outcome variables. Results were presented in the form of tables and charts for clarity.

RESULTS

Demographic Profile -

Among the 50 patients studied, the most common age group was 41–50 years (42%), followed by 31–40 years. Females constituted 56% of the cohort, while males accounted for 44%.

Clinical Presentation -

As seen in table 1, the most frequent presenting symptom was headache (42%), followed by altered sensorium (32%), motor deficit (26%), seizures (24%), and delirium (8%).

Clinical Signs (table 1) -

On examination, papilledema was the most common sign, observed in 48% of patients. Other findings included motor deficit (26%), cranial nerve palsy (24%), stupor (16%), and coma (4%).

Table 1. Baseline clinical characteristics of patients with CVT

Clinical Presentation	Percentage Frequency
Altered sensorium	32
Headache	42
Motor deficit	26
Seizures	24
Delirium	8
Clinical Signs	
Papilledema	48
Motor deficit	26
Cranial nerve palsy	24
Stupor	16
Coma	4

Etiological Factors -

As seen in table 2, the predominant cause of CVT was puerperium (44%), followed by dehydration (12%), oral contraceptive (OCP) use (8%), and systemic lupus erythematosus (SLE) (6%). Less frequent etiologies included antithrombin III deficiency (4%), antiphospholipid antibody syndrome (APLA) (4%), hyperhomocysteinemia (4%), factor V Leiden mutation (2%), protein C deficiency (2%), and protein S deficiency (2%).

Neuroimaging Findings (table 2) -

On MRI, the superior sagittal sinus (SSS) was most commonly affected (42%), followed by the transverse sinus (26%), combined SSS and transverse sinus involvement (16%), sigmoid sinus (10%), and straight sinus (6%).

Table 2. Etiology & involved sinus of patients with CVT

Etiology	Percentage Frequency
Puerperal	44
Dehydration	12
OCP	8
SLE	6
Antithrombin III deficiency	4
APLA	4
Hyperhomocysteinemia	4
Factor V Leiden mutation	2
Protein C deficiency	2
Protein S deficiency	2
Involved sinus	
Superior sagittal sinus (SSS)	42
Transverse sinus	26
SSS & Transverse sinus	16
Sigmoid sinus	10
Straight sinus	6

Treatment Profile -

Both unfractionated heparin (UFH) and low molecular weight

heparin (LMWH) were used equally (50% each) for initial anticoagulation.For oral therapy, Nicoumalone was prescribed in 96% of patients, while dabigatran was used in 4%.

Hospital Stay and Outcomes –

The mean duration of hospitalization was 12.22 ± 3.97 days. Mortality occurred in 8% of cases, while 92% were discharged in stable condition.

As seen in table 3, at admission, 24% of patients had a Modified Rankin Scale (mRS) score 3-6, which improved to 10% at 12-week follow-up.

Based on the Glasgow Coma Scale (GCS), 62% of patients scored between 11–14, and 26% had a score of 15 on admission.

Table 3. Outcome of patients with CVT

Modified Rankin scale (mRS)	Percentage frequency (on admission)	Percentage frequency (on follow up)
mRS 0-2	76	90
mRS 3-6	24	10

DISCUSSION

Comparison with existing literature –

In this prospective observational study of 50 patients with cerebral venous sinus thrombosis (CVT), the majority belonged to the 41–50 year age group (42 %), and females comprised 56 %. Our demographic findings are consistent with the established epidemiological profile of CVT. The International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) reported that women constituted approximately 74.5 % of cases, with a mean age of 37 years.[4] The slightly lower female predominance (56 %) in our series may reflect regional variability or referral bias. Recent studies have observed an increasing incidence of CVT in older men, possibly related to improved diagnostic access and lifestyle-associated risk factors.[5,6] A Finnish nationwide study similarly demonstrated a trend toward higher median age and a narrowing male-to-female ratio.[7] These findings parallel our data, in which the most affected age group was 41–50 years and female-to-male ratio was roughly 1.3:1.

Our clinical profile mirrors prior reports in the literature with headache (42 %) and altered sensorium (32 %) being the most frequent symptoms, while papilledema (48 %) was the leading clinical sign. Headache remains the most frequent presenting complaint, occurring in roughly 80–90 % of cases in large registries.[8,9] Seizures occurred in 24 % of our cohort, comparable to rates reported by Muir and Coutinho [10], who noted seizure occurrence in approximately one-third of patients. Altered sensorium (32 %) and motor deficits (26 %) were also consistent with data from other prospective cohorts. [11]

The superior sagittal sinus was the most commonly affected vessel in our study (42 %), similar to the frequency reported in several imaging-based series. [8,9] Hu et al. documented SSS involvement in nearly 60 % of patients with non-infectious CVT.[8]

Etiologically, the puerperal state was the leading cause in our cohort (44 %), reflecting the persistent burden of postpartum CVT in developing regions, followed by dehydration (12 %) and oral contraceptive use (8 %). This contrasts with Western series, where hormonal contraception, thrombophilia, and malignancy are more prevalent causes. [4,9,12] Nevertheless, our data corroborate the findings of the CMC Vellore CVT Registry, which also identified puerperium as the dominant cause among Indian women. [5] The incidence of inherited thrombophilia markers—such as antithrombin III deficiency (4 %) and antiphospholipid antibody syndrome (4 %)—was comparable with previous reports, which note these abnormalities in a minority of patients.[12,13]

Prognostic implications -

Our short-term mortality rate (8 %) aligns closely with ISCVT (8.3 %) [4] and other recent cohorts reporting mortality between 5 % and 12 %. [8,14] The reduction in poor functional outcomes (mRS 3-6) from 24 % at admission to 10 % after 12 weeks indicates significant neurological recovery. Previous analyses have shown that early anticoagulation markedly improves outcomes, with up to 85–90 % of survivors achieving functional independence.[9,14] Predictors of adverse prognosis include coma, intracerebral haemorrhage, deep venous involvement, and infection. [4,13] Our findings of favourable recovery rates likely reflect early recognition and treatment at a tertiary centre. The mean hospital stay of 12.2 days in our cohort is similar to other Asian studies (10–14 days) [5, 12], suggesting comparable hospital resource utilisation.

CONCLUSION

Cerebral venous sinus thrombosis remains a significant yet under-recognized cause of stroke in adults, particularly among women in the puerperal period. In this prospective cohort, headache and altered sensorium were the most frequent presentations, with the superior sagittal sinus being the most commonly involved site. Early diagnosis through neuroimaging and prompt initiation of anticoagulation resulted in favourable neurological recovery in the majority of patients. Mortality and poor outcomes were confined to a small subset presenting with severe neurological impairment. The findings highlight the continuing predominance of postpartum CVT in developing regions and underscore the importance of clinician awareness, early detection, and standardized management to improve patient outcomes.

Study strengths and limitations

The prospective design and standardized imaging confirmation of CVT enhance the reliability of our findings. The study provides region-specific data from India, where postpartum and dehydration-related CVT remain common. Limitations include the modest sample size, single-centre setting, and short follow-up duration (12 weeks). Additionally, thrombophilia testing was performed selectively, which may underestimate inherited risk factors.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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