

Original Research Article

A STUDY ON CLINICAL, ETIOLOGICAL AND RADIOLOGICAL PROFILE OF CEREBRAL VENOUS THROMBOSIS IN MALE PATIENTS

B Dinesh Kumar¹, Ande Adarsh², Ronanki Priyanka³

¹Assistant Professor, Department of Neurology: Gandhi medical college/ Hospital, Secunderabad, Telangana, India.

²Senior Resident, Department of Neurology: Gandhi medical college/ Hospital, Secunderabad, Telangana, India.

³DrNB Neurology, Care hospital banjara hills, Banjara Hills, Hyderabad, Telangana, India.

Received : 17/04/2025
Received in revised form : 05/06/2025
Accepted : 29/06/2025

Corresponding Author:

Dr. Ande Adarsh,
Senior Resident, Department of
Neurology, Gandhi Medical College/
Hospital, Secunderabad, Telangana,
India.
Email: dradharshmdmch232@gmail.com

DOI: 10.70034/ijmedph.2025.3.18

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (3); 103-110

ABSTRACT

Background: Aim: To study the clinical, etiological and radiological profile of Cerebral Venous Thrombosis in male patients.

Materials and Methods: This cross-sectional study was carried out in the Neurology department. study subjects were adult male patients who were admitted with acute headache and other neurological features with CT/ MRI/ MRV (brain) findings of Cerebral Venous Thrombosis

Results: The majority of patients were of 3rd and 4th decade, with a mean age of 35.5 + 11.75 years. Headache was the most common symptom, reported by all patients, followed by seizures (74%), vomiting (62%), and blurred vision (32%). Focal neurological deficits (45%) and papilledema (32%) were the most frequently observed clinical signs. Elevated serum homocysteine levels were found in 59% of patients, of which 52% were alcoholics. A statistically significant link was found between alcoholism and elevated serum homocysteine. Additional risk factors for CVT in males include infections (10%), connective tissue disorders (6%), protein C deficiency (6%), protein S deficiency (5%), dehydration due to diarrhea (3%), anemia (3%), antithrombin-III deficiency (1%), and antiphospholipid antibody syndrome (1%). The cause was unknown in 6% of cases. The superior sagittal sinus (SSS) was the most frequently affected sinus (69%), in males followed by the transverse sinus (45%), either individually or in combination.

Conclusion: There is a need to increase the awareness among people regarding the effects of alcohol and necessary steps to reduce the consumption of alcohol.

Keywords: Superior sagittal sinus (SSS), Straight sinus(SS), Magnetic Resonance Venography (MRV).

INTRODUCTION

Cerebral venous thrombosis (CVT) is an important cause of stroke in young adults with a two-thirds female preponderance. It is caused by partial or complete occlusion of the major cerebral venous sinuses (cerebral venous sinus thrombosis) or the smaller feeding cortical veins (cortical vein thrombosis). Venous thrombosis can cause venous infarction due to congestion and inadequate blood flow which results in brain tissue damage. Though CVT is rare and constitutes less than 1% of strokes, in recent years, with the advent of imaging modalities like CT (Computerized Tomography) scan and recently Magnetic Resonance Imaging (MRI) and

Magnetic Resonance Venography (MRV), the diagnosis of CVT has improved significantly.^[1,2]

Patient with CVT usually presents with headache, seizure, papilledema, altered sensorium and focal neurological deficits due to thrombosis of intracranial veins and sinuses resulting in hemorrhagic infarctions and raised intracranial tension. Based on the studies it is estimated that prevalence of CVT is 5 per million population and it contributes to 0.5 percent of all strokes. Studies have shown that the prevalence is higher in Asians compared to the western population.^[3,4]

CVT, even though seen in both sexes, more common in women due to gender-specific risk factors like puerperium, usage of oral contraceptive pills (OCP)

and hormone replacement therapy (HRT). Even the earlier studies from India showed an increased incidence of CVT in female population. However recent studies have shown an equal or even higher incidence of CVT in male population.^[5,6] The number of male patients presenting with CVT at our hospital has been on the rise. Therefore, this study was undertaken to identify the etiology, clinical presentations, and radiological patterns of CVT in male patients, which may aid in understanding the disease in this population and assist in planning effective management strategies.

MATERIALS AND METHODS

The study subjects were adult male patients who were admitted with acute headache and other neurological features with CT/ MRI/ MRV (brain) findings of Cerebral Venous Thrombosis in the Neurology department of Gandhi General Hospital, Secunderabad.

Study design: Cross-sectional study **Sample size:** 100 patients

Duration of study: This study was done between December 2022 to May 2024 for a period of 18 months.

Inclusion Criteria: Patients included in this study were adult male patients with features of CVT confirmed by CT/MRI/MRV (brain).

Exclusion Criteria: All cases of CVT due to trauma and neoplastic diseases.

Methodology

During the period of admission, data was collected pertaining to demographic profile of patients. A detailed history with special importance to risk factors like alcohol consumption and clinical examination findings for various neurological presentations were noted. Data was collected in a pretested proforma meeting the objectives of the study after taking the informed consent from the patients and/or attendants. These patients were evaluated with complete blood picture and other blood investigations that are implicated in the pathogenesis of CVT.

Basic Investigations done as CBP, ESR, Blood urea, Serum Creatinine, Viral markers-HIV, Hepatitis B, Hepatitis C, Liver function tests, Anti-Nuclear Antibody (ANA), Prothrombotic profile: Protein C, Protein S, Antithrombin III, serum homocysteine, antiphospholipid antibodies, Electroencephalogram (EEG) and Radiological investigations as CT Brain, MRI Brain with MRV, CT Venogram whenever necessary

Statistical methods

The data was analysed using descriptive statistics. Continuous variables were presented as mean and standard deviation, while categorical variables were reported as frequencies and percentages. The Chi-square test was used to compare categorical data between groups, with a p-value of less than 0.05 considered statistically significant.

RESULTS

This was a cross-sectional study done at Gandhi Hospital, Secunderabad between December 2022 to May 2024. The results of the study are as follows.

Table 1: Age and clinical features distribution in present study

Age Group (years)	Number of patients	Percentage
<20	3	3%
21-25	22	22%
26-30	16	16%
31-35	19	19%
36-40	9	9%
41-45	16	16%
46-50	5	5%
51-55	3	3%
56-60	4	4%
>60	3	3%
Total	100	100%
Clinical feature		
Headache	100	100%
Vomiting	62	62%
Seizures	74	74%
Blurring of vision	32	32%
Diplopia	11	11%
Papilledema	38	38%
Focal neurological deficits	45	45%

Based on the current study, the average age of onset for CVT in males is 35.5 + 11.75 years, with an age range spanning from 18 to 75 years. The highest

incidence was observed in the 21-25 and 31-35 age groups. Headache was the most common clinical manifestation noted in our patients. All of our patients

(100%) presented with headache. Vomiting was present in 62% of the patients. Seizures were present in 74% of the patients. Both focal and generalised

seizures were noticed. Blurred of vision was present in 32% of patients.

Table 2: Risk factors and seasonal trends in study

Risk factor	Number of patients	Percentage
Alcohol	74	74%
Elevated serum homocysteine	59	59%
Connective tissue disease	6	6%
Protein C deficiency	5	5%
Protein S deficiency	4	4%
Antithrombin III deficiency	1	1%
APLA	1	1%
Anemia	3	3%
Dehydration secondary to diarrhoea	3	3%
Infections	10	10%
Idiopathic	6	6%
Seasonal trends		
January	2	2%
February	4	4%
March	11	11%
April	21	21%
May	25	25%
June	10	10%
July	6	6%
August	7	7%
September	4	4%
October	3	3%
November	4	4%
December	3	3%

Alcohol consumption was the predominant risk factor, observed in 74% of the patients. Elevated serum homocysteine levels were observed in 59% of the patients, with 52% of these individuals being alcoholics. Local infections, such as sinusitis, mastoiditis, and granuloma, were noted in 10% of the cases. Connective tissue disease was present in 6% of the patients. Deficiencies in Protein C and Protein S were identified in 5% and 4% of patients, respectively. Antiphospholipid antibodies (APLA) and Antithrombin III deficiencies were each observed in 1% of the patients. anemia was seen in 3%, and acute diarrhea leading to dehydration was reported in three patients. In 6% of cases, the etiology remained unknown. Most of the cases (57%) occurred during the summer months, from March to May, when temperatures are high.

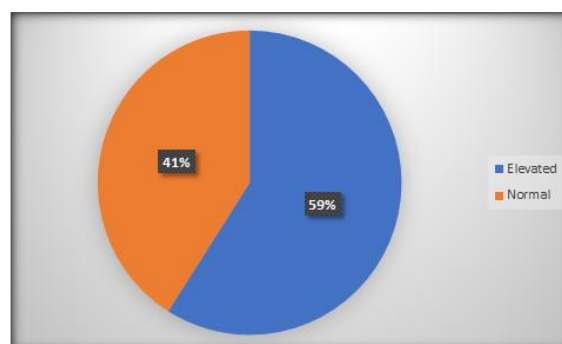


Figure 1: Serum homocysteine levels

Serum homocysteine levels were elevated in 59 of our patients. Out of these 59, 52 patients were alcoholics, with a history of binge alcohol in most of them. Only seven patients were non alcoholics. With these values hyperhomocysteinemia can be considered as an important cause of the hypercoagulable state which might be contributory to the pathogenesis of CVT in this subset of patients.

Table 3: Alcohol consumption and homocysteine

Alcohol consumption	Elevated Homocysteine		Total
	Present	Absent	
Present	52	22	74
Absent	7	19	26
	59	39	100

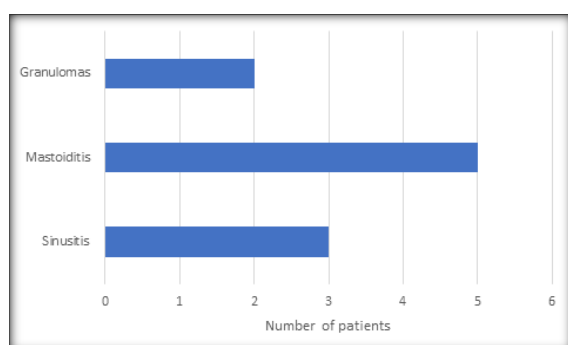
Chi-square statistic: 14.9448, p-value: 0.00011

Chi-square test calculated based on values of homocysteine and alcohol showed a significant p-value of 0.00011 (significant level $p < 0.05$) between the two indicating a statistically significant association

Table 4: Sinus involvement with other sinuses involvement

Sinus involved	Number of patients	Percentage
SSS	69	69%
TS	45	45%
SG	21	21%
DEEP	9	9%
HE	23	23%
CAV	3	3%
SSS with other sinuses involvement		
SSS	26	26%
SSS+TS	14	14%
SSS+SG	2	2%
SSS+TS+SG	5	5%
SSS+TS+SG+HE	1	1%
SSS+TS+HE	2	2%
SSS+HE	13	13%
SSS+DEEP	6	6%
Multiple sinus involvement with Transverse sinus		
TS	5	5%
TS+SSS+SG	5	5%
TS+SG	8	8%
TS+SSS	14	14%
TS+SSS+HE	2	2%
TS+SSS+SG+HE	1	1%
TS+HE	7	7%
TS+DEEP	3	3%

The superior sagittal sinus was the most frequently affected, involved in 69% of patients, either alone or in combination with other sinuses. The transverse sinus was affected in 45% of cases, while the sigmoid sinus was involved in 21% of patients. Of the 69 cases, 26 had isolated involvement of the superior sagittal sinus (SSS). Additionally, 14 patients had SSS involvement along with transverse sinus involvement. Transverse sinus (TS) involvement, either alone or in combination with other sinus involvement, was observed in 45 patients. Among these, 8 patients had thrombosis of both the TS and sigmoid sinus (SG), while 2 patients presented with TS involvement along with SSS involvement and hemorrhage. Isolated TS thrombosis with hemorrhage was noted in 7 patients.

**Figure 2: MRI Brain findings in infectious causes of CVT**

Mastoiditis was present in 5 patients. All these patients had transverse sinus thrombosis either alone or in combination with other sinus involvement. Two patients had an associated granuloma with sinus thrombosis. Sinusitis directly related to causing cavernous sinus thrombosis in 3 patients.

DISCUSSION

Most previous studies on CVT have focused either on the general population, including both males and females, or exclusively on puerperal CVT. Multiple studies, such as those by Mohammed Najib et al,^[7] and Vembu et al,^[8] indicate a higher prevalence of CVT in females. However, research by Brig.S. Kumaravelu et al,^[9] and Narayan D et al,^[6] both conducted in India, shows a higher occurrence in males. An increasing number of male patients with CVT have been presenting at Gandhi Medical College, Secunderabad. Hence, this study was undertaken to investigate CVT in male patients in this region.

The age of incidence in the study ranged from 18 to 75 years, with an average onset age of 35.5 + 11.75 years and a peak incidence in the third and fourth decades. These findings align with studies by Narayan D et al,^[6] Mohammed Najib et al,^[7] Brig. S. Kumaravelu et.al,^[8] Algatani et al,^[10] and Dr. C. Kanaga Raju et.al.^[11] which also report similar age incidences.

Table 5: Comparison of patients with other studies

Mean Age of the patients	Mean + SD (years)
Narayan D et al ^[6]	31.8 + 10.9
Dr. C. Kanaga Raju et.al ^[11]	23.5 + 3.5

Present study	35.5 + 11.75
Comparison of headache	Percentage
Narayan D et.al ^[6]	88.3 %
Mohammed Najib et al ^[7]	100 %
Vembu et al ^[8]	80 %
Brig. S. Kumaravelu et al, ^[9]	75 %
Dr.C.Kanaga Raju et.al ^[11]	84 %
E.A. Ashok Kumar et.al ^[12]	59.5 %
Abdulkader Daif et al ^[13]	82 %
Present study	100 %
Comparison of vomiting	
Narayan D et al ^[6]	74%
Dr. C. Kanaga Raju et.al ^[11]	70%
Wasay et.al ^[14]	35%
A. Rajendran et.al ^[15]	75.7%
Present study	62%
Comparison of blurred vision	
Narayan D et al ^[6]	22.1%
Wasay et al. ^[14]	23.1%
Ferro JM et.al ^[16]	13.5%
Present study	32%
Comparison of seizures	
Narayan D et.al ^[6]	45.1%
Brig. S. Kumaravelu et al ^[9]	36%
Dr.C.Kanaga Raju et.al ^[11]	59%
E.A. Ashok Kumar et.al ^[12]	35.7%
Breteau et al ^[17]	50.9%
Present study	74%
Comparison of papilledema	
Narayan D et al ^[6]	63%
Mohammed Najib et al ^[7]	86%
Vembu et al. ^[8]	28%
Brig. S. Kumaravelu et al ^[9]	66%
Wasay et al ^[14]	32%
Abdulkader daif et al ^[13]	80%
A. Rajendran et.al ^[15]	45.5%
Present study	38%
Comparison of Focal neurological deficits	
Narayan D et al ^[6]	25%
Vembu et al ^[8]	37%
Brig. S. Kumaravelu et al ^[9]	63%
Dr. C. Kanaga Raju et.al ^[11]	26%
AbdulKader daif et al ^[13]	27%
Wasay et al ^[14]	66%
Ferro JM et.al ^[16]	37.2%
Breteau et al. ^[17]	47.3%
Present study	45%
Comparison of alcohol use	
A. Rajendran et.al ^[15]	72.7%
Pratibha P study ^[18]	32%
Present study	74%
Comparison of seasonal trends	
E.A. Ashok Kumar et.al ^[12]	61.9.%
Mustansir et al ^[19]	32.8%
Present study	57%

Headache emerged as the primary symptom in this study, with all patients reporting a recent onset of headache. In comparison, other studies also highlighted headache as a common presenting feature: 88.3% in Narayan D et al,^[6] 00% in Mohammed Najib et al,^[7] 93% in Vembu et al,^[8] 75% in Brig. S. Kumaravelu et al,^[9] 84% in Dr.C.Kanaga

Raju et al,^[11] 59.5% in E.A. Ashok Kumar et al,^[12] and 82% in Abdulkader Daif et al,^[13]

Vomiting was present in 62% of our patients. Comparatively, Wasay et al,^[14] reported vomiting in 35% of patients, while Narayan D et al,^[6] noted it in 74%, Dr. C. Kanaga Raju et al,^[11] reported in 70% and A. Rajendran et al,^[15] reported in 75.7%.

Blurred vision affected 32% of our patients and diplopia occurred in 11%, particularly in those with increased intracranial pressure and cavernous sinus thrombosis with cranial nerve palsies. Wasay et al,^[14] found blurred vision in 23.1% of cases, Ferro JM et al,^[16] reported 13.5%, and Narayan D et al,^[6] reported 22.1%.

Seizures, both focal and generalized, occurred in 74% of our patients. Similarly high incidence of seizures was noted in studies by Dr. C. Kanaga Raju et al,^[11] at 59% and Breteau et al,^[17] at 50.9%. This contrasts with the findings of Brig. S. Kumaravelu et al,^[9] at 36%, Narayan D et al,^[6] at 45%, and E.A. Ashok Kumar et al,^[12] at 35.7%.

There is a wide variation in the reported incidence of seizures across various studies, with our study indicating the highest occurrence. Several factors may contribute to the high incidence of seizures in our study. The significant prevalence of alcoholism among our patients could be a contributing factor. Seizures may result from binge drinking, known as rum fits, or from alcohol withdrawal following the onset of headaches and general malaise, which could prompt patients to suddenly stop drinking, thereby triggering withdrawal seizures. Additionally, multiple sinus thromboses and their extensive involvement, leading to hemorrhage, may also play a role in causing seizures in these patients.

The incidence of papilledema in our study was 38%. This rate varies widely across other studies, ranging from 28% to 86%. Specifically, Vembu et al,^[8] reported an incidence of 28%, Mohammed Najib et al,^[7] found 86%, Brig. S. Kumaravelu et al,^[9] reported 66%, Abdulkader daif et al,^[13] reported 80%, Wasay et al,^[14] noted 32%, and Narayan D et al,^[6] observed in 63%.

In our study, 45% of patients presented with focal neurological deficits, such as monoparesis, hemiparesis, aphasias or cranial nerve palsies. The incidence of these deficits varies in the literature, with reports ranging from 25% to 66%. For instance, Narayan D et al,^[6] reported an incidence of 25%, Vembu et al,^[8] found 37%, Brig. S. Kumaravelu et al,^[9] observed 63%, Dr. C. Kanaga Raju et al,^[11] reported 26%, Abdulkader Daif et al,^[13] found 27%, Wasay et al,^[14] reported 66%, Ferro JM et al,^[16] documented 37.2%, and Breteau et al,^[17] found 47.3%.

Among focal neurological deficits 14% of patients developed aphasias, with speech difficulties ranging from slurred speech to pure aphasias. The presentation varied based on the location and side of the infarct or hemorrhage. In the study by Ferro JM et al,^[16] aphasia was present in 19% of patients, while Wasay et al,^[14] reported a 16% incidence of speech abnormalities. These findings are consistent with our data.

This study identifies alcoholism as a significant risk factor for CVT, with 74% being alcoholics. Although some patients reported substance abuse, these substances were not included in the study due to unknown and variable compositions. However, their

potential contribution to a hypercoagulable state cannot be ignored.

Heavy alcohol consumption contributes to cerebral venous thrombosis (CVT) by creating a state of dehydration, increased blood viscosity, and enhanced platelet reactivity. Additionally, it may independently increase the risk of endothelial dysfunction through the nitric oxide (NO) pathway. Chronic alcohol use, particularly in high concentrations, interferes with NO production or release from endothelial cells, reducing NO synthesis and hindering endothelial proliferation. High levels of ethanol also activate the proapoptotic caspase pathway, leading to a higher procoagulant state and impaired fibrinolytic activity, which predisposes individuals to thrombosis. Moreover, acute ingestion of a large but tolerable dose of alcohol transiently increases thromboxane-mediated platelet activation, resulting in hyperaggregation.

Most of the cases in the study were reported in the months of summer where there were high environmental temperatures. About 57% of the cases were clustered in the three months from March to May. This trend is comparable with other similar studies by E.A. Ashok Kumar et al,^[12] and Mustansir et al.^[19]

Cerebral venous thrombosis (CVT) is more prevalent in males during the summer months, largely due to dehydration and increased outdoor activity in the heat. Beyond dehydration, hyperthermia from heat exposure can lead to the release of endotoxins from intestinal mucosal cells and certain interleukins (IL-1 and IL-6). These chemical mediators enter the systemic circulation, triggering a systemic inflammatory response syndrome.⁶¹ The widespread inflammation, combined with the direct effects of heat, causes injury to the endothelial cells of blood vessels, which may predispose vulnerable individuals to developing CVT. Recent research by Aaron et al. has also reported a significantly higher incidence of CVT cases during the summer in India. As mentioned earlier alcohol and dehydration were the most important risk factors predisposing to CVT as well as directly effecting the coagulation parameters. Apart from these other prothrombotic states considered in our study included connective tissue disorders, APLA, and elevated serum homocysteine levels and deficiencies of antithrombin III, protein C and protein S.

ANA was positive in 6% of our patients, compared to 7% in the study by Abdulkader daif et al,^[13] 5% in the study by Brig. S. Kumaravelu et al,^[9] 4% in the study by Wasay et al,^[14] and 7% in the study by Ferro JM et al.^[16] Antithrombin III was positive in 1% of our patients similar to Wasay et al,^[14] study. Protein C and Protein S deficiencies were present in 5% and 4% of our patients, respectively, whereas they were positive in 9.1% and 12.3% in the study by Narayan D et al,^[6] Anticardiolipin antibody was present in 2% of our patients. Infection in the form of sinusitis, mastoiditis and granuloma of brain was the cause of CVT in 10% of patients. Acute diarrhea leading to

dehydration caused CVT in 3% of patients and anemia contributed to CVT in 3% of patients. The etiology was unknown in 6% of patients.

Table 6: Comparison of Protein C and Protein S deficiency with other studies

Study	Protein C deficiency	Protein S deficiency
Narayan D et al ^[6]	9.1%	12.3%
Present study	6%	5%

Serum homocysteine levels were elevated in 59% of our patients, a significantly higher percentage than in other studies. Mustansir et.al,^[19] reported in 37.5%,

Pratibha P study,^[18] in 24%, Wasay et al,^[14] in 5% of patients, Ferro JM et.al,^[16] in 7%, and the study by Narayan D et al,^[6] in 18% of patients.

Table 7: Comparison of elevated serum homocysteine levels with other studies

Study	Percentage
Narayan D et.al ^[6]	18%
Pratibha P study ^[18]	24%
Mustansir et.al ^[19]	37.5%
Present study	59%

A chi-square test comparing homocysteine levels and alcohol consumption in our study showed a significant p-value, indicating a statistically significant association between the two.

Homocysteine, a thiol-containing amino acid derived from methionine, is produced through the methylation cycle and is not present in dietary sources. It can be remethylated to methionine by methionine synthase or converted to cysteine via transsulphuration, a process catalysed by cystathionine β -synthase. Vitamin B12, B6, and folic acid are essential for these conversions. Folate levels are crucial for regulating homocysteine metabolism, and homocysteine levels inversely correlate with these vitamins.

A study by Bleich et al,^[64] found that alcohol consumption led to elevated homocysteine levels and decreased folic acid levels. They also observed a gradual decrease in homocysteine levels following alcohol withdrawal. They proposed that withdrawal symptoms might be due to increased levels of aspartate, glutamate, homocysteic acid, and cysteine sulphinic acid, combined with upregulation of NMDA receptors, leading to excitotoxicity. Robert M. Russell et al,^[65] used radiolabeled studies to document increased urinary excretion of homocysteine during ethanol ingestion. Charles H. Halsted et al,^[66] similarly found decreased intake of radiolabeled folate in alcoholics.

In the study "Hyperhomocysteinemia in Cerebral Venous Thrombosis," Ida Martinelli et al,^[24] reported

that hyperhomocysteinemia was associated with a fourfold increased risk of cerebral venous sinus thrombosis. Given these findings, the 59% incidence of hyperhomocysteinemia in our study may be linked to the high prevalence of alcoholism and binge drinking among our patients. The alcohol-related disruptions in folate metabolism could be contributing to hyperhomocysteinemia, which may manifest as cerebral venous sinus thrombosis.

All patients underwent CT and MRI brain imaging, including MR Venogram, diffusion-weighted sequences, ADC mapping, and gradient echo sequences. Superior Sagittal Sinus (SSS) involved in 69% of patients, with 26 having isolated SSS involvement. SSS was associated with transverse sinus (TS) in 14 patients, and with both transverse and sigmoid sinuses (SG) in 8 patients. SSS involvement was also present with deep vein thrombosis in 6 patients and with hemorrhage or hemorrhagic infarct in 13 patients.

Transverse Sinus (TS) involvement was observed in 45 patients, either alone or in combination with other sinuses. Isolated TS thrombosis was noted in 5 patients. TS was associated with sigmoid sinus (SG) in 8 patients and with deep venous sinus thrombosis in 3 patients. Hemorrhage was present in 7 patients with TS involvement.

This distribution of sinus involvement is consistent with findings from various studies on cerebral venous thrombosis (CVT).

Table 22: Comparison of sinus involved with other studies

Study	Sinus involved (Percentage)					
	SSS	TS	SG	CAV	DEEP CVT	HE
Narayan D et al ^[6]	54.3%	47.7%	20.6%	2.4%	5.8%	-
Vembu et.al ^[8]	59%	54%	-	-	-	18%
Brig. S. kumaravelu et al ^[9]	73%	33.3%	-	3.3%	-	15%
A. Rajendran et.al ^[15]	81.8%	69.7%	-	-	-	-
E.A. Ashok Kumar et.al ^[12]	23.8%	88%	23.8%	-	-	-
Ferro JM et.al ^[16]	37%	18%	-	1.3%	11%	-
Present study	69%	45%	21%	3%	9%	23%

In our study, the superior sagittal sinus (SSS) was the most commonly involved sinus, observed in 69% of patients. This finding is consistent with other studies. Narayan D et al,^[6] reported SSS involvement in 54.3% of cases, Vembu et al,^[8] found 59%, Brig. S. Kumaravelu et al,^[9] reported 73%, and A. Rajendran et al,^[15] documented 81.8%. The transverse sinus was the second most commonly involved sinus in these studies.

Mastoiditis was detected in 5 % of patients. All the cases of mastoiditis were associated with adjacent transverse sinus involvement. Granuloma was identified as the cause of adjacent cortical vein thrombosis in two patients. Sinusitis was linked to cavernous sinus thrombosis in 3% of patients.

CONCLUSION

Most of the patients in the study belonged to third and fourth decades. Most cases occurred during the summer months, particularly between March and May where the temperatures are high and chances of dehydration is more. Alcoholism emerged as the most common risk factor for CVT in males and there is a trend of increasing consumption of alcohol in this part of the country. Elevated serum homocysteine, contributing to CVT, was frequently observed in alcoholics. Headache was the most common symptom reported by all patients in the study. High index of suspicion of CVT is indicated if chronic alcoholic presenting with headache. The superior sagittal sinus (SSS) was the most commonly affected sinus in males followed by the transverse sinus (TS). Efforts to increase awareness and reduce alcohol consumption are essential.

REFERENCES

1. Coutinho JM, Zuurbier SM, Stam J. Declining mortality in cerebral venous thrombosis: a systematic review. *Stroke*. 2014; 45:1338–41.
2. Saadatnia M, Fatehi F, Basiri K, Mousavi SA, Mehr GK. Cerebral venous sinus thrombosis risk factors. *Int J Stroke*. 2009;4(2):111–23.
3. Saposnik G, Barinagarrementeria F, Brown RD Jr, Bushnell CD, Cucchiara B, Cushman M, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2011;42(4):1158. Epub 2011 Feb 3.
4. Jeyaram VK, Ranganathan LN. The clinical profile in male CVT patients admitted to the Institute of Neurology, Madras Medical College, India. *J Curr Res*. 2018;11(7):5746–9.
5. Dash D, Prasad K, Joseph L. Cerebral venous thrombosis: an Indian perspective. *Neurol India*. 2015;63(3):318–28.
6. Narayan D, Kaul S, Ravishankar K, Suryaprabha T, Bandaru VCSS, 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(2):154–9.
7. Idris MN, Gasmelseed DE, Elsadig YI, Elamin EH. Clinical presentation and outcome in a prospective series from Sudan. *Neurosciences*. 2008;13(4):408–11.
8. Vembu P, John JK, Kuruvilla MJ, El Gohary MA. Cerebral venous thrombosis in Kuwait: Clinical presentation, risk factors, and management. *Neurosciences (Riyadh)*. 2011;16(2):129–36.
9. Kumaravelu S, Gupta A, Singh KK, et al. Cerebral venous thrombosis. *Med J Armed Forces India*. 2008; 64:355–60.
10. Algahtani HA, Aldarmahi AA, Shirah BH, et al. Cerebral venous sinus thrombosis in Saudi Arabia: Demographic, clinical, and radiological characteristics. *Neurosciences (Riyadh)*. 2011;16(4):329–34.
11. Kanaga Raju C, Shakthi Raja Guru G, Ravikumar T, et al. A prospective study on CVT in young males. *Int J Sci Res*. 2018;7(12):17–20.
12. Ashok Kumar EA, Manisha A, Hanvitha M. A study of clinical profile, etiology, and therapeutic outcomes of patients with cerebral sinus venous thrombosis in men. *IAIM*. 2022;9(9):13–20.
13. Daif A, Awada A, Al-Rajeh S, et al. Cerebral venous thrombosis in Saudi Arabia: A retrospective study of 40 cases. *Stroke*. 1995; 26:1193–5.
14. 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(2):49–54.
15. Rajendran A, Bose JA, S DCH, Jayasankar VR, Shameer P. Clinical and etiological profile of cerebral venous sinus thrombosis. *Int J Contemp Med Res*. 2020;7(1): A1–A4.
16. Ferro JM, Canhão P, Stam J, Boussier MG, Barinagarrementeria F. 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.
17. Breteau G, Mounier-Vehier F, Godefroy O, Gournay P, Mackowiak-Cordoliani MA, Leys D. Cerebral venous thrombosis: 3-year clinical outcome in 55 consecutive patients. *J Neurol*. 2003; 250:29–35.
18. Pratibha P. Alcohol as a provoking factor in male cerebral venous thrombosis: A prospective analysis in Western Rajasthan. *J Clin Neurol Neurosurg*. 2022; 5:006.
19. Mustansir F, Inam M, Darbar A. The effects of temperature and prothrombotic conditions on cerebral venous sinus thrombosis frequency: An institutional experience. *Asian J Neurosurg*. 2021; 16:719–24.