

Original Article

STUDY OF THE PREDICTORS OF CLINICAL OUTCOME IN PATIENTS WITH CEREBRAL VENOUS SINUS THROMBOSIS

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ABSTRACT:

Introduction: Cerebral venous sinus thrombosis (CVST) is a serious condition that requires rapid diagnosis and treatment. The prognosis of cerebral venous sinus thrombosis (CVST) is variable, and the outcome may range from complete recovery to death and disability. **Objectives:** To study the clinical, radiological and therapeutic prognostic factors in CVST that predict a poor outcome.

Materials and Methods: The patients' demographic characteristics, clinical features, laboratory investigations and brain imaging findings and treatment options were studied. Follow-up visits were performed at month 1, 3, and 6 months. Modified Rankin Scale (MRS) was applied at each visit.

Results: Ninety patients (56.7% men, 43.3% women) with mean age of 33±10.96 years were identified. Most common presenting features were headache (78%), and generalised tonic clonic seizures (42%). The most frequent clinical sign was papilledema in 68.9% of patients, followed by hemiparesis (22.2%) and aphasia (18.9%). Common risk factors were puerperium (23.3%), alcohol consumption (21.1%), hyperhomocysteinemia (16.7%), infections (8.9%), Protein C and S deficiency (3%), malignancy (3%) and trauma (1%). Mortality was seen in 6(7%) patients. The outcome was favourable with 93% survival rate. Factors related to poor outcome were identified as altered consciousness, coma, intracerebral haemorrhage, infarcts, midline shift, generalised tonic clonic seizures and involvement of parietal lobe.

Conclusion: In conclusion, coma, intracerebral haemorrhage, midline shift, generalised tonic clonic seizures and parietal lobe involvement are independent predictors for poor outcome of CVST.

Keywords: Cerebral venous thrombosis; Risk factors; Anticoagulation; Prognostic factors; Outcome

INTRODUCTION

Cerebral venous sinus thrombosis is a condition which is characterized by thrombosis of the intracranial veins and sinuses.¹

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It can present in all age groups, however, the most commonly affected are young individuals.² It results in varied clinical manifestations, ranging from headache, seizures, and features of idiopathic intracranial hypertension, focal motor deficits, cranial nerve palsies and ophthalmoplegia.³ Parenchymal damage resulting due to CVST can cause residual deficits. In severe cases, there can be raised intracranial tension, necessitating intervention. The outcome of patients with CVST can be varied, which ranges from complete recovery to permanent neurological deficit and death.⁴ Due to earlier diagnosis through imaging,

and anticoagulant treatment, the outcome has improved in the last decade. Factors determining prognosis in CVST include coma, the presence of parenchymal lesions including intracerebral haemorrhage or infarcts, midline shift, seizures, age, and neurological deficits, based on data from case reports or retrospective series.⁵ In the acute phase, it is important to identify these factors as it can influence the therapeutic strategy and enables the treating physician to give reliable information to the patient and relatives.

We conducted a prospective study in a series of 90 patients with CVST, to identify the clinical presentations, radiological features and prognostic factors that predict a poor outcome in patients with CVST.

MATERIAL AND METHODS

This study is a prospective analysis of 90 patients admitted in our institution during a period of 2 years. After obtaining a written informed consent, patients' details were recorded in the proforma. A detailed neurological examination was performed. Modified Rankin Scale (MRS) were applied to all patients. All patients were subjected to MRI on GE Signa HDxt 1.5 T machine for confirmation of the diagnosis of CVST. Conventional MRI sequences including Axial T1, Sag T1, Axial FLAIR, Axial T2, Coronal T2 FLAIR, GRE, DWI, Time of Flight Angiography and Sag inphase sequence were used. Contrast enhanced MR Venogram sequences were done whenever necessary. Pre contrast images were taken, followed by post contrast images after administration of 0.1mmol/kg body weight of gadolinium intravenously.

Relevant blood and radiological investigations were carried out on all patients. Plasma concentrations of proteins C, S, Antithrombin III and antibodies such as Anticardiolipin, Antinuclear, antidouble-stranded DNA, and hypercoagulability and vasculitis tests were done when indicated and the results were recorded. All patients with CVST, even those with hemorrhagic lesions, received intravenous heparin or enoxaparin followed by oral warfarin for a period of three to

six months and if an important etiology of thrombophilia was found, warfarin therapy continued lifelong. INR was repeated every month and oral anticoagulants were adjusted accordingly

The patients' demographic characteristics, clinical presentations, laboratory findings, cranial imaging, treatment options and outcome were studied. Clinical assessment were done during the hospital stay and follow up assessment was done at 1 week, 1 month, 3 months, 6 months and 1 year. Modified Rankin Scale (MRS) was applied at every visit. Imaging (CT or MRI) were done at 3 months when possible.

Statistical analysis:

Statistical Analyses was performed with IBM SPSS program for Windows Version 22. Categorical variables were presented as frequency and percentage. To study the association between categorical variables, Chi Square test and if the cell values were small Fisher's Exact Test was used.

Logistic regression was used to assess the risk factors. Continuous variables were represented as mean and Standard Deviation. Inter group comparison was done with Unpaired t test and within the group comparison was done with Paired t test. To assess the relation between variables, Pearson's Correlation was applied. A p value of <0.05 is considered as significant.

RESULTS:

The present study enrolled a total of 90 patients. The mean age was 33±10.60 years. Fifty-one patients (56.7%) were male and thirty-nine (43.3%) were females.

Fifty nine (65.6%) patients had an acute onset, with symptoms developing within 48 hours before the diagnosis. Twenty one (23.3%) patients had puerperal CVST. Nineteen (21.1%) patients were alcoholics and fifteen (16.7%) patients had hyperhomocystenemia. Oral contraceptive pills(OCPs) use was seen in eight (8.9%) patients. Six (5.6%) patients had polycythemia. Three (3.3%) patients had underlying malignancy.

Infectious cause of CVST was seen in eight patients, with six (6.7%) being diagnosed as TB meningitis, one patient with mastoiditis and one with HIV. Thrombophilia in the form of protein C and S deficiency was seen in 3 (3.3%) patients. One patient (1.1%) had dehydration and one (1.1%) had underlying trauma. The etiological factors are summarised in Table 1

Table 1: Predisposing conditions

Etiology	Frequency	%
Alcohol	19	21.1
Hyper homocystenemia	15	16.7
Malignancy	3	3.3
OCP	8	8.9
Puerperium	21	23.3
Polycythemia	6	5.6
TB meningitis	6	6.7
Anaemia	3	3.3
Protein C deficiency	2	2.2
APLAS	1	1.1
Dehydration	1	1.1
HIV	1	1.1
Mastoiditis	1	1.1
Protein C S Deficiency	1	1.1
Trauma	1	1.1
Unknown	1	1.1
Total	90	100

The neurological symptoms and signs at admission are summarised in table 2. Headache was the most frequent symptom, present in seventy eight (95%) of the patients. Other commonly occurring symptoms were generalised tonic clonic seizures in forty two (46.7%), focal seizures in eleven (12.2%) and fever in ten (11.1%). The most common presenting sign was papilloedema seen in sixty-two (68.9%) patients. Other signs included hemiparesis in twenty (22.2%), aphasia in seventeen (18.9%) and coma in thirteen (14.4%) patients. Other findings on clinical examination included cranial

nerve six and seven nerve involvements, in one (15.6%) and nine (10%) patients, respectively.

Thirty nine patients (43.3%) had signs of intracerebral haemorrhage(ICH) on their baseline CT or MRI. Forty six (51.1%) had infarcts on presentation. The most common lobe to be involved was the parietal lobe in thirty-nine (43.3%) of the patients, followed by frontal lobe in twenty-nine (32.2%) and temporal lobe in sixteen (17.8%) patients. Four (4.4%) patients had occipital lobe involvement and 2(2.2%) patients had involvement of the cerebellum. Midline shift was seen in thirteen (14.4%) patients. Magnetic resonance imaging showed involvement of the superior sagittal sinus (SSS) in sixty-five patients (72.2%); in sixty-one (67.85) of them the transverse sinus (TS) was also involved. The third common sinus to be involved was the sigmoid sinus in forty-one (45.6%) of the patients. Cortical veins were involved in thirty-nine (43.3%) and deep venous system was involved in eight (8.9%) patients. Majority of the patients had multiple sinus involvement. The combined involvement of the superior sagittal sinus and the cortical veins was the commonest, seen in twenty-seven (54%) patients. The next common sinuses to be involved were the superior sagittal sinus and the transverse sinus in twenty (40%) patients. The superior sagittal sinus, transverse sinus and the straight sinus were involved in fifteen (30%) patients. Five (10%) patients had involvement of the superior sagittal, transverse, sigmoid (SS), straight sinus, cortical vein and the internal jugular vein (IJV). Frequency of brain parenchymal lesions and sinus involvement is summarized in Table 3.

Sixty-one (32.2%) of patients received Low molecular Weight Heparin (LMWH) while twenty-nine (32.2%) patients received IV unfractionated heparin. One patient underwent decompressive craniotomy with subsequent use of LMWH. All patients received oral anticoagulant thereafter.

Minimum follow up period was for three months. The follow up MRV was done in seventy-six patients. Fifty-six (62.2%) patients had complete recanalization, while nineteen (21.1%) had partial recanalization. One patient had

incomplete recanalization. Majority of the patients (49.54%) had baseline MRS score of 1-2, with follow up MRS showing a significant decline with maximum (62;68.8%) patients having a MRS score of zero. Five (5.5%) patients had MRS score of above 2 after 6 months. The outcome in the study population was good with 93% survival rate. Six (7%) patients died due to the episode.

Table 2: Symptoms and signs at the time of presentation

Symptoms & Signs	Frequency	%
Headache	78	86.7
Fever	10	11.1
Focal seizures	11	12.2
GTCS	42	46.7
Coma	13	14.4
Hemiplegia	20	22.2
Aphasia	17	18.9
Cerebellar signs	2	2.2
Papilloedema	62	68.9
Cranial nerve 6 involvement	14	15.6
Cranial nerve 7 involvement	9	10.0

Fifty eight (64.4%) patients had complete recovery on subsequent follow up. Twenty five (27.8%) patients had residual deficits in the form of hemiparesis in twelve (48%) patients, seizures in nine (36%) patients, visual field defects in three (12%) and ataxia in one patient.

We identified factors associated with poor prognosis at the time of hospital admission which were, low level of consciousness including stupor or coma (p=0.000), intra parenchymal haemorrhage (p=0.005), infarct (p=0.01), presence of midline shift (p=0.000), parietal lobe involvement (p=0.005), higher basal midline score (p=0.07). Six (7%) patients died during hospitalization, with no death occurring in patients who survived the acute episode.(Table 4)

At the end of three months follow up, factors predicting persistent disability included low

Glasgow coma scale (GCS) and coma at admission(p=0.000), presence of hemiparesis(p=0.000) and aphasia(p=0.000), generalised tonic clonic seizures at presentation(p=0.03%).At the end of 6 months, similar factors were responsible for the persistent disability(p=0.000) (Table 5)

Table 3: CT Brain and MRI Brain findings

CT/MRI Findings	Frequency	Percent
Infarct	46	51.1
Hemorrhage	39	43.3
Frontal lobe	29	32.2
Parietal lobe	39	43.3
Temporal lobe	16	17.8
Occipital lobe	4	4.4
Cerebellum	2	2.2
SINUSES INVOLVED		
Superior sagittal sinus	65	72.2
Transverse sinus	61	67.8
Sigmoid sinus	41	45.6
Straight sinus	15	16.7
Internal jugular vein	11	12.2
Cortical vein	39	43.3
Deep vein	8	8.9
Inferior sagittal sinus	5	5.6
SSS& TS	20	40
SSS,TS,SS,Straight sinus,cortical vein, IJV	5	10
SSS, TS, SS	15	30
SSS, TS, Deep veins	2	4
SSS, SS, Deep veins	2	4
SSS,Cortical veins	27	54
SSS,TS,SS, Deep vein	2	4
SSS, TS, SS, Straight sinus	8	16
TS,SS	15	30
TS,SS,Deep vein	2	4
SS,TS, IJV	5	10
TS, SS,IJV	5	10

There was a statistically significant greater LVEF (%) in group I, as compared to group II (p-value < 0.0001). However, compared to group I, group II showed significantly higher mean HR (p-value < 0.0001). Further analysis revealed no statistically significant difference in both the groups with respect to mean SBP (p-value = 0.1231), DBP (p-value = 0.1404), and MBP (p-value = 0.1072).

Table 4: Prognostic factors on admission

Predictors	Total No (N=90)	Died (N=6)	Survived (N=84)	P VALUE
Med Age ≤32	48	3	45	.865
Gender - Male	51	3	48	.733
Anaemia - Present	26	2	24	.804
GCS <10	19	6	13	.000
Headache	78	4	74	.136
Fever	10	0	10	.370
Focal seizures	11	0	11	.344
GTCS	42	5	37	.060
Coma	13	6	7	.000
Hemiparesis	20	3	17	.090
Aphasia	17	3	14	.040
Papilloedema	62	6	56	.080
Midline shift	13	5	8	.000
MRS Basal Score > 2	41	6	35	.007

DISCUSSION

Overall outcome in our study population was good, with independent survival in 93% of the patients. It is clear that the majority of our patients included younger age group. Cerebral venous sinus thrombosis is known to affect young individuals. Several other Indian studies have also found that CVST is more common in the younger age group. A prospective, clinical study conducted on forty patients found that majority of the patients were in the age group of 15-35 years.⁶

In our study population more number of males were affected compared to that of females. The high frequency of CVST in men in our study could be probably due to the rising consumption of alcohol by men, improvement in obstetric care, and higher level of clinical suspicion and detection of CVST at an early stage. Similar results were also obtained from a study analysing the long term outcome of patients with CVST, where the incidence of CVST was more among men compared to those of women.⁷

Among the etiological factors, the most common risk factors identified was related to pregnancy and puerperium, followed by alcohol consumption and hyperhomocystenemia. Puerperium and pregnancy, as predisposing factors for CVST, are well known. Most of the pregnancy-related CVST occurs during the third trimester or puerperium. Pregnancy induces several prothrombotic changes in the coagulation system that also persists during early puerperium. Hypercoagulability worsens after delivery due to volume depletion and trauma. Our study is consistent with most of the earlier case series of CVST reported from India which had very high proportions of puerperal CVST.⁸ However, there has been a decline in CVST related to pregnancy, as described in several studies.⁷ The increased incidence of puerperal CVST in our study calls for better obstetric care and clinical suspicion in the present population.

Alcoholism and hyperhomocystenemia were the main risk factors in men. Alcohol contributes to thrombosis by dehydration, hypercoagulability, and reactive thrombocytosis, with resultant hyperviscosity of blood.⁷ A case of CVST following binge drinking has been reported in literature.⁹ The increased consumption of alcohol in our place of study could attribute to the increased incidence of alcohol induced CVST. Hence, this calls for a better awareness of the same and more stringent de-addiction measures.

Oral Contraceptive Pills use as a risk factor was seen in eight patients. Oral contraceptives have a strong cause of CVST due to its estrogenic

component because the estrogens increase the levels of coagulation factors and decrease the levels of anticoagulant proteins such as antithrombin.¹⁰ Hence, the use of OCPs should be controlled, and only used when absolutely necessary. The prothrombotic mutations strongly enhance the risk of oral contraceptives causing venous thrombosis. Thus, screening for prothrombotic defects might be useful in women considering taking contraceptive hormones.¹¹ With regard to the type of OCPs, there is no difference in the incidence of CVST with the use of third generation or previous generation OCPs.¹² Thus, the results of our study suggests better screening of patients before starting OCPs.

The other etiological factors in our cohort were polycythemia. The cause of polycythemia could not be determined in our patient. Infectious causes were TB meningitis, mastoiditis and HIV. Dehydration and trauma were other uncommon etiologies in our population. Hypercoagulable state like protein C deficiency and S deficiency were also detected. Malignancy was found in one patient. One patient had primary antiphospholipid antibody syndrome (APLS). Thus, our present study found a wide range of etiological disorders as the cause of CVT. This suggests a detailed search for the causative factors, which could enable treating the specific causes and hence the disease.

The major clinical features at presentation were headache with or without features of raised intracranial tension, focal and generalised tonic clonic seizures, fever and altered sensorium which is comparable with previous studies.⁴ Papilloedema was the most frequent sign. This was followed by focal deficits like hemiplegia, aphasia, coma, cerebellar signs and sixth and seventh cranial nerve involvement. We identified distinct clinical syndrome that included stroke like presentation, encephalopathy, seizures and Idiopathic Intracranial Hypertension like presentation. Similar findings have been seen in another Indian study by Narayan et al, where papilloedema was the most frequent sign at presentation.⁷ In the NIVSR cohort, a stroke-like presentation was present in 28.5% patients, isolated seizures in 29.4%,

idiopathic intracranial hypertension-like presentation in 18.2%, encephalopathy in 25.2%, and psychosis was observed in 1.8% patients.

The present study assessed the MRS score of the patients at baseline and on subsequent follow up. Attempt was made to correlate the MRS score with the outcome. MRS score was recorded on follow up which showed improved scores on subsequent visit, suggesting good outcome of majority of the patients in this study. Our cohort had good outcome which was reflected in the improvement in the MRS scores at 3 and 6 months. In a cohort of forty-one CVST patients, 92% of the patients had good outcome with MRS score between 0-3 at the end of one year.¹³

We also tried to find factors that predicted the outcome in our patients. During the acute phase, low GCS, coma at presentation, presence of haemorrhage and midline shift were associated with a worse MRS score. This is comparable with another study by Girot M et al which concluded that the presence of intracerebral haemorrhage, older age group, men, deep vein thrombosis was associated with higher mortality and disability at 6 months.¹⁴ Patients admitted for CVST with intracerebral bleed and coma should be considered as high risk for death and dependency and should be given close monitoring. At the end of 3 months the factors associated with disability included low GCS at admission, GTCS, presence of hemiplegia and aphasia. Similar results were obtained at 6 months follow up. Thus, according to our study, factors associated with poor outcome included low GCS at presentation, presence of intracerebral haemorrhage, midline shift, GTCS, focal deficits including hemiparesis and aphasia. This is comparable with another Indian study where it was seen that the predictors of poor outcome included presence of fever, deep venous thrombosis, seizures, focal neurological deficits, and unconsciousness.⁷ Another study found that focal neurologic deficit and severely disabled patients with MRS score of 3-5 on admission were independent predictors of dependency or death in patients with CVST.¹⁵ Assessment of prognostic

indicators of CVST in a Chinese cohort during pregnancy also found the presence of infection, seizures and ICH to be significantly associated with mortality¹⁶

Our patients were also assessed for the presence of disabilities at the end of 6 months. Majority of the patients had no disability on subsequent follow up. 27.8% of patients had residual deficits, among which, the most common was hemiparesis, followed by seizures, visual field defects and ataxia. Similar long term sequelae has also been seen in another study, with long term sequelae in the form of epilepsy, headaches, visual loss, pyramidal deficits and cognitive impairment¹⁷

We conclude that the overall prognosis of CVST is good. Coma, intracerebral haemorrhage, infarcts, presence of midline shift are independent predictors for poor outcome. Factors responsible for long term sequelae include Coma, intracerebral haemorrhage, infarcts and generalised tonic clonic seizures on admission. Thus, a knowledge of these factors would enable us to predict the sequel following admission, facilitating towards better overall care of the patient.

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Table 5: Prognostic factors on follow

3 months follow-up						
Predictors for poor & good outcome		Total No (N=84)	GOOD OUTCOME (N=75)	POOR OUTCOME (N=9)	Chi Square / Fisher's Exact Test & P Values	
Age	≤ 32	44	40	4	.338	.561
	> 32	36	31	5		
Gender	Male	48	40	8	4.150	.040
	Female	36	35	1		
Anaemia	Present	24	20	4	1.244	.265
	Absent	60	55	5		
Aphasia	Present	14	7	7	27.010	.000
	Absent	70	68	2		
Papilloedema	Present	56	49	7	.560	.454
	Absent	28	26	2		
Coma	Yes	7	3	4	17.210	.000
	No	77	72	5		

Table 6: Prognostic factors at 6 months follow

6 months follow-up						
Predictors for poor & good outcome		Total No (N=80)	GOOD OUTCOME (N=74)	POOR OUTCOME (N=6)	Chi Square / Fisher's Exact Test & P Values	
Age	≤ 32	43	40	3	.030	.848
	> 32	37	34	3		
Gender	Male	47	41	6	4.550	.030
	Female	33	33	0		
Anaemia	Present	24	22	2	.030	.587
	Absent	56	52	4		
Aphasia	Present	14	10	4	10.860	.001
	Absent	66	64	2		
Papilloedema	Present	54	50	4	.002	.640
	Absent	26	24	2		
Coma	Yes	7	4	3	13.820	.000
	No	73	70	3		

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