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Alcohol a Provocating Factor in Male Cerebral Venous Thrombos is a Prospective Analysis in Western Rajasthan

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Abstract

Cerebral Venous Thrombosis (CVT) is a rare form of cerebrovascular disease with variable manifestations like headache, focal neurological deficit, seizure and altered sensorium thus making the diagnosis difficult. Indian studies revealed that CVT contribute to 10% of all stroke. Earlier studies from India showed an increase incidence of CVT in women due to puerperium, Oral contraceptive pills and hormone replacement therapy.

Keywords: Prothrombotic • Hyperviscosity • Alcoholism • Performance • CVT

Introduction

Cerebral Venous Thrombosis (CVT) is a rare form of cerebrovascular disease with variable manifestations like headache, focal neurological deficit, seizure and altered sensorium thus making the diagnosis difficult. Indian studies revealed that CVT contribute to 10% of all stroke. Earlier studies from India showed an increase incidence of CVT in women due to puerperium, Oral contraceptive pills and hormone replacement therapy. However recent studies showed that males are equally or more affected by CVT compared to females [1,2]. Hereditary and acquired prothrombotic conditions are a major risk factor in male CVT [3,4]. Among males, alcoholism is a major cause to predispose CVT by causing a state of dehydration, hyperviscosity, and increased platelet reactivity leading to an acquired prothrombotic state. Thus a high index of suspicion is needed to make a diagnosis and prevent further complications [5,6].

Materials and Methods

Twenty five male patients diagnosed to have CVT and proven by MRI were selected after taking informed written consent and ethical approval from outpatient clinic of neurology department at Dr. S N medical college from the period from September, 2014 to August, 2018. Their ages ranged from 10 to 70 years. Detailed addiction [7,8]. All patients were subjected to detailed neurological and systemic examination for any evidence of anemia, dehydration, DVT of leg. Etiological work up was done to identify the cause and risk factors like malignancy, systemic disease, hyperhomocystenemia, prothrombotic conditions Genetic studies for mutations were, however not done due to economic limitations Hyperhomocysteinemia was defined as serum homocysteine level more than 15 mg/100 ml in less than 60 years old patients and more than 20 mg/100 ml in more than 60 years old patients. Anemia was defined as hemoglobin level of less than 13 gm/dl in men and 12 gm/dl in women as per World Health Organisation (WHO) guidelines. MRI and MRV was done in all patients. Patients with indefinite evidence of CVT on imaging and presence of hypertensive haemorrhage, arterial stroke, metabolic encephalopathy and presence of space occupying lesions on imaging are excluded. Statistical Analysis: The data were collected. P-value less than 0.05 were considered statistically significant. All data were expressed as mean or patient's Number (n) and percentage (%) as appropriate [9,10].

Results

history was taken with respect to clinical features, substance abuse and alcohol Twenty five male CVT patients were diagnosed and included in our study. Their ages ranged from 10 to 70 years with mean age of onset 31 yrs. Majority were in 20-30 s age group (Figure 1).

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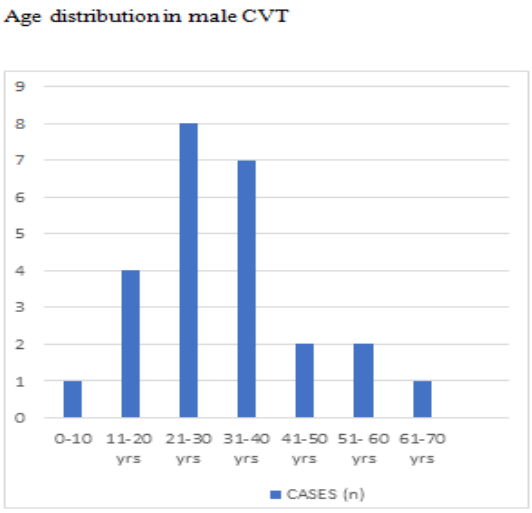

Figure 1. Age distribution in male CVT.

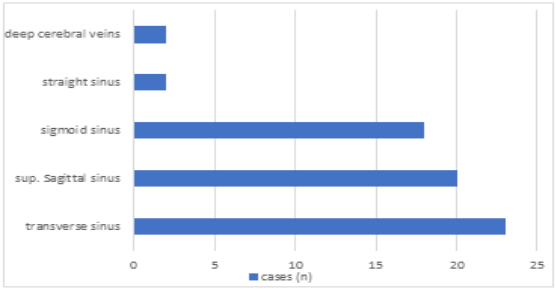

Figure 2. Venous system involvement in CVT.

The most common clinical presentation was headache seen in all patients (100%) with vomiting in 24 (96%) patients. Majority had a sub-acute onset of symptoms [n=14, (56%)], acute in [n=4(16%)] and chronic in [n=7(28%)]. Seizures was observed in 40% (n=10) of the patients. Focal seizures with secondarily generalisation was the most common semiology observed in [n=7 (70%)]. 3 patients had Generalised Tonic Clonic Seizures [11,12]. Motor weakness was observed in 5 patients (20%) and all had hemiparesis. 4 patients had altered sensorium at the time of presentation. Papilledema was observed in 21 patients (84%) out of which majority had grade 4 degree of papilledema (44%). Fever was absent in all. Cranial Nerve involvement was noted in 16%. Most common cranial nerve affected was unilateral sixth Nerve (8%) and facial (8%) nerve [13,14].

Addictions were observed in 10 patients (40%) out of which [n=8 (80%) were chronic alcoholics. [n=2 (20%)] of the patients were addicted to cigarette smoking/tobacco chewing. All Alcoholics in our study were heavy drinkers. Mild Anaemia was observed in 20% of the patients all patients had venous sinus thrombosis in MR venography. Many patients had multiple venous sinus involvement and transverse sinus was noted to be the most common sinus involved [n=23 (92%)] followed by Superior sagittal sinus [n=20(80%)]. The major risk factors identified were alcoholism [n=8(32%)], hyperhomocystenemia [n=6(24%)], Factor V mutation, behcet's disease, malignancy, dehydration all contributed equally to 4%. No specific etiological diagnosis could be found in majority of the patients [n=5 (20%)]. All patients were treated with anticoagulants and followed up to six months. 3 left against medical advice [15,16]. 2 died and rest became asymptomatic. Clinical Profile and venous system involvement of CVST is summarised in Table 1 and Figure 2 respectively [17,18].

Parameters	No of cases (%)
Sample size	25
Mean age (yrs.)	31 yrs.
Age range (yrs.)	25842
Onset	
1. Acute	4 (16%)
2. Sub-acute	14 (56%)
3. Chronic	7 (28%)
Symptoms and signs	
1. Headache	25 (100%)
2. Vomiting	24 (96%)
3. Seizures	10 (40%)
a. GTCS	3 (30%)
b. Focal	7 (70%)
4. Weakness	5 (20%)
a. Hemiparesis	5 (20%)
5. Altered sensorium	4 (16%)

6. Fever	0
7. Crania nerve palsy	4 (16%)
8. papilloedema	21(84%)
9. diplopia	2 (8%)
Risk factors	
Hyperhomocystenemia	6 (24%)
Alcohol	8 (32%)
Anemia	5 (20%)
infection	0
Fa V (leiden) mutation	1 (4%)
Malignancy	1 (4%)
Behcet	1 (4%)
Polycythemia	2 (8%)
Dehydration	1 (4%)
idiopathic	5 (20%)
Drug abuse	0 (0%)
Protein s deficiency	0
Protein c deficiency	0

Table 1. Clinical profile of male CVT patients.

Discussion

Cerebral venous thrombosis is a diagnostic challenge for clinicians because of variable pattern of presentation. Many studies has beendone on puerperal CVT. Recently the scenario of CVT is changing. Modern imaging technique like MRI and MRV and good obstetric care has led to decreased incidence of CVT in females [19,20]. Nowadays there is a male dominance seen by various Indian studies. Although various Indian and western studies have been done on CVT (Table 2).

	Indian studies				**Western studies**	
Series	Narayan	**Pai**	**Aneesh**	**Jeyaram**	**Wassay**	**Ferro**
Study design	retrospective	retrospective	Ambispective	prospective	ambispective	prospective
Sample size	428	612	116	203(all males)	182	624
Mean Age (μ=yrs)	31.3 yrs.	31.9 yrs.	35.21 yrs.	38+6.9 yrs.	38 yrs.	39.1 yrs.
Age range (yrs)	8-65 yrs.	[Pubmed][Pubmed]-	21-30 yrs.	[Pubmed][Pubmed]–	13-42 yrs.	16-86 yrs.
Gender ratio (M/F)	1.16:1	0.12639	1.3:1	All males	0.08681	0.04444
MC symptom	Seizures>paresis	Headache>paresis	Men (Sz>Headache) Females (headache>vomiting)	Headache>seizures	Headache>paresis	Headache>seizures
Risk factor	Anemia=hyperhomocystenemia>alcohol	Infection>Genetic thrombophilia	Males (Alcohol) Females (puerperium>OCP)	Alcohol>cigarette	Genetic thrombophilia	OCPs>Genetic thrombophilia
MC sinus involved	Sup. Sagittal sinus>transverse sinus	Sup. Sagittal sinus>cortical venous thrombosis	Sup. Sagittal sinus>transverse sinus	Sup. Sagittal sinus>transverse sinus	Sup. Sagittal sinus	Sup. Sagittal sinus>transverse sinus

Table 2. Comparison between series of patients with cerebral venous thrombosis.

A systematic study was lacking from western India, Rajasthan. Hence a study was conducted focusing on predisposing factors of CVT in males. In our study, majority had CVT in 20-30 s age groups. Mean age is 31 yrs. In comparison, the mean age was 31 yrs. in

study by whereas in western studies ICVST the mean age is 39 yrs. Headache was most common and early detected symptom in our study. Headache with or without vomiting was found in 100%patients. The possible hypothesis explained was stretching of the nerve fibers in the walls of the occluded sinus and local inflammation as suggested by the evidence of contrast enhancement of the sinus wall surrounding the clot [21,22]. Headache was most commonly sub-acute in onset in [n=14 (56%)], a cute in [n=4 (16%)], and chronic in [n=7 (28%)] patients. Stroke like presentation was seen in 20% [n=5] patients in which all had hemiparesis.

Encephalopathy was seen in 16% [n=4]. Cranial nerve involvement seen in [n=4 (16%)] Seizures occurred in [n=10 (40%)]. 70% (n=7) had focal seizures and 30% (n=3) had generalized seizures. In a study by, seizures was the most common symptom followed by stroke while in, most common symptom was headache followed by seizures similar to our study. In our study, Papilloedema was seen in [n=21 (84%)] patients in which Grade IV papilloedema was most common sign contributing to 44% (n=11). This is high which may be due to delayed presentation of most cases. On MRV, most common sinus involved was transverse sinus [n=23(92%)], followed by superior sagittal sinus [n=20(80%)]. In contrast, found superior sagittal sinus involvement was most common followed by transverse sinus.

In our study, the predisposing risk factors observed in males were alcoholism [n=8(32%)], hyperhomocystenemia [n=6 (24%)], mild anemia [n=5 (20%)], dehydration [n=1 (4%)], protein c and s deficiency [n=0 (0%)], behcet disease [n=1 (4%)], polycythemia vera [n=2 (8%)], factor V mutation [n=1 (4%)], malignancy [n=1 (4%)] and idiopathic in[n=5(20%)]. All alcoholics were chronic heavy drinkers. In our study, alcoholism and hyperhomocystenemia were the most common risk factors found in men. Narayan et al found that anemia and alcoholism were most common risk factors and among prothrombotic conditions, hyperhomocystenemia followed by protein S deficiency. Heavy alcohol consumption may be an independent risk factor for endothelial dysfunction. Through the Nitric Oxide (NO) pathway.

There is an interference with NO production or release from endothelial cells by chronic alcohol consumption; especially, high concentrations of alcohol reduce NO synthesis and endothelial proliferation. Further ore, high concentrations of ethanol activates the proapoptotic caspase pathway. Thus heavy alcohol consumption causes higher procoagulant state and an impaired fibrinolytic potential activities which can predispose individuals to thrombosis. Acute ingestion of large but tolerable dose of alcohol transiently increases thromboxane-mediated platelet activation, and hyperagreegation is observed after acute alcohol consumption. Moreover, long term alcohol causes chronic liver damage causing a state of overall decreased synthesis of anticoagulant thrombotic factors. Virchow triad (triad of Stasis, hypercoagulability, and endothelial dysfunction) is believed to contribute to thrombosis. Our study emphasized that increase consumption of alcohol has led to high incidence of CVT in males and most of the alcoholics presented with raised intracranial pressure. Thus whenever heavy alcoholics present with raised intracranial pressure, CVT must be excluded. All patients were treated with anticoagulants and followed up to six months. 3 left against medical advice. One died and the outcome was favourable in rest of them.

Conclusion

CVT is uncommon but treatable disease. Alcoholism is strongly associated with male CVT. Prognosis is good if treated early. High index of suspicion of CVT is indicated if chronic alcoholic presenting with headache with raised intracranial pressure.

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