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Research Article

CVT IN YOUNG ADULTS; STUDY FROM TERTIARY CARE TEACHING HOSPITAL IN CHENNAI, INDIA

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ABSTRACT

Background and Purpose: We undertook this study to determine the frequency, clinical patterns, and etiologies of cerebral venous thrombosis in young adults in tertiary care teaching hospital.
Methods: Records of all adult patients admitted with documented diagnosis of cerebral venous thrombosis from Jan 2011 through march 2015 in Department of Neurology, hospital.
Results: A total of 351(228 males and 123 females) cases of stroke in young were admitted to the Department of Neurology between Jan 2011 to march 2015. Cerebral venous thrombosis accounted for 18.80 %(66/351), Ischemic stroke 60.11 %(211/351) and 21.08 %(74/351) had spontaneous intracerebral haemorrhage (ICH).Among CVT patients (37 men, 29 women) aged 15 to 45 years were identified. Fifty two cases (78.8%) had a clinical picture of headache. Twenty eight cases (42.43%) had seizures. Protein C and S deficiency was present in 8(12.12%), Behçet's disease in 4 cases 6.06%), antiphospholipid antibodies in 4(6..06%), oral contraceptives and hormonal pills(OCP) in 10(15.16%), postpartum in 3(4.54%), systemic lupus erythematosus in 2(3.03%) , PCOD(Polycystic ovarian disease) in 2(4.54%) and factor V leiden in one case . Alcohol intake in 30 (45.46%) and smoking in 32(48.49%)
Conclusions: Cerebral venous thrombosis in young adults is not uncommon. Alcoholism, OCP, coagulation disorder, postpartum state, Behçet's disease are common aetiologies of CVT.All patients with a headache/stroke should undergo MRI/MRV/MRA of brain as a routine protocol before being labelled idiopathic.

INTRODUCTION

Thrombosis of the cerebral venous channels in the brain is an uncommon cause of cerebral infarction relative to arterial thrombosis but is an important consideration because of its potential morbidity and mortality. The symptoms and signs of cerebral venous thrombosis (CVT) are nonspecific so it may be difficult to diagnose clinically and is often underdiagnosed/misdiagnosed. Headache, papilledema, vomiting, seizures, impaired consciousness and focal neurological deficits could be its presenting manifestations (Bousser Barnett, 1992; Bousser *et al*., 1985). Newer imaging modalities have led to easier recognition of venous sinus thrombosis, offering the opportunity for early therapeutic measures. Venous sinus thrombosis also may be associated with other medical complications that require therapeutic intervention.

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In developing countries, hypercoagulable states around the puerperium as well as infectious diseases are believed to be the major causes (Southwick *et al*., 1986; Srinavasan, 1984), but in western countries these are less significant (Bousser and Barnett, 1992). In this article, 66 cases of CVT are reported from a Tertiary care teaching institute. These were encountered over a 4-year period in the one of centre of South India. In this paper, we discuss the frequency, clinical patterns, and etiologies of cerebral venous sinus thrombosis.

Anatomy Overview

The cerebral venous system is formed by deep and superficial veins that drain into the dural sinuses before returning the blood to the systemic circulation. The superficial veins include the superior anastomotic vein of Trolard lying across the parietal lobe and the inferior anastomotic vein of Labbe lying on the surface of the temporal lobe.

Both veins drain the cortex and underlying white matter into the superior and inferior sagittal sinuses. The deep cerebral veins (the basal or Rosenthal vein and deep internal cerebral veins) drain the deeper white matter, the basal ganglia and the diencephalon. Many deep cerebral veins drain into the great cerebral vein of Galen. The latter together with the inferior sagittal sinus returns the blood to the straight sinus. The straight and superior sagittal sinuses together form the two transverse sinuses that drain into the sigmoid sinuses returning the blood to the internal jugular veins.

Venous drainage occlusion in the brain and subsequent venous congestion will have different manifestations depending on the venous system involved, the extent of the venous collaterals, the extent of the thrombosis and its duration (Walter, 2005). The occlusion of a major venous sinus can impair the cerebral spinal fluid (CSF) resorption in the arachnoid villi sites resulting in high intracranial pressure. Because the obstruction is in the arachnoid villi sites, no gradient pressure will form, hence the absence of hydrocephalus on imaging. Second, venous congestion can lead to parenchymal edema:

Following venous thrombosis, the high venous pressure results in an increase in the net capillary filtration with the development of a vasogenic edema in the interstitial tissue drained by the thrombosed veins. Neurological signs secondary to that type of edema can be reversed if the veins flow is restored (e.g. spontaneously or following anti thrombotic drugs administration). However, if the venous flow is not restored on time, ischemia will result and a cytotoxic edema will develop. This type of edema, secondary to sodium/potassium pump dysfunction in the cell membrane, is similar to the changes following an arterial infarct; however it is less severe and can be reversed (Makkat *et al*., 2003; Lövblad *et al*., 2000).

Venous infarct can then result in small hemorrhages that may become confluent forming an intracerebral hematoma (ICH). The hematoma location is different from that of an arterial ICH and should raise the concern of CVST.

MATERIALS AND METHODS

The records of all patients with CVT seen in two large hospitals from Jan2011 through March 2015 were reviewed. Both hospitals caters urban and rural population of southern India. Data regarding clinical features, risk factors and diagnostic test were evaluated in details. The diagnosis of CVT was based on a partial or complete absence of filling of at least one dural sinus using MRI and MRV imaging. Our studies include all patient with CVT in age group of 15-45 years of age. All patients were evaluated and treated as per standard protocol for treatment of CVT. Neurological examination was done for all patients including ophthalmoscopy. A detailed past medical history obtained for each case. The following investigations were performed in all cases: complete blood count, erythrocyte sedimentation rate, basic blood biochemistry, chest Xray, ECG, prothrombin time (PT), activated partial thromboplastin time (PTT), prothrombotic profile, vasculitic panel, urinalysis, cerebral MRI/ MRV. Prothrombotic profile include levels of antithrombin III, protein C, protein S, factor V Leiden and antiphospholipid antibodies. Vasculitic profile include ANA,P-ANCA, C-ANCA.

RESULTS

Clinical characteristics: There were 37 males and 29 females (M:F 1.27:1) with a mean age of 28.8 years (range 15 to 45 years). Fifty two patients (78.8%) presented with headache. The cardinal manifestations of the 66 patients are presented in Table 1.

Table 1. Symptoms and signs of CVT cases

Symptoms/Signs	Number of cases (%)
Headache	52(78.8%)
Focal Neurological deficits	44(66.7%)
Seizures	28(42.43%)
Drowsy/Stupor/Coma	13(19.6%)
Papilledema	14(21.22%)
Cranial Nerve Palsy	13(19.6%)

Table 2. The occluded sinuses according MRI and MRV studies

Occluded sinus	Number of cases (%)
Superior sagital sinus alone	15(22.72%)
Superior sagital sinus+others	43(65.15%)
Lateral sinus	3(4.45%)
Lateral sinus+straight sinus	5(7.57%)

Headache was presentation in 52 patients (78.8%). Papilledema in 14 patients (21.22%).Focal neurological deficits were found in 44(66.7%) . Cranial nerve involvement was seen in 13 patients (19.6%) and they were as: sixth nerve involvement in 4 (6%), seventh nerve in 9 (13.63%). Twenty eight patients (42.43%) presented with seizures (focal in 10 cases and generalized in 18 patients). 13(19.6%) patient presented with impaired consciousness in form of drowsiness, stuper and coma.

Neuroimaging findings: Brain MRI/MRV was standard modality of choice for neuroimaging. Hemorrhagic infarction was observed in 25 patients (37.87 %), and ischemic infarct in 15(22.7%). Brain MRI and MRV was abnormal in all cases. The most common involvement is superior sinus plus other sinus involvement in 43(65.15%), superior sagital sinus involvement alone in 15(22.72%), lateral sinus plus straight sinus in 5(7.57%) and lateral sinus in 3(4.45%) in that order as depicted in Table 2.

The etiology varied as shown in table 3, In 30 cases(45.45%), alcohol had been used and 10(15.16%) cases were associated with OCP/hormone pill containing low dose estrogen (LD) pills had been used. In overall, 12.13% of cases had a coagulation disorder in hematology studies and the most common coagulopathy was protein C/S deficiency. Among less common causes were APLA in 4 (6.06%), Behcets disease in 4 (6.06%) cases, postpartum state in 3 (4.54%), polycythemia in 2 (4.54%), SLE in 2 (3.03%) cases, PCOD in 2 (3.03%) and Factor V Leiden in 1(1.51%) case.

DISCUSSION

The true incidence of CVT in India and most regions of the world is still unknown. Kalbag and Woolf indicated that CVT was the principal cause of death in 1 per 2 million persons per year in England and Wales between 1953 and 1961 (Kalbag and Woolf, 1992).

Table 3. Etiology of venous sinus thrombosis

	Alcohol	Protein c/s deficiency	APLA	Bahcets disease	OCP/hormonal pills	Post partum state	Polycythemia	SLE	PCOD
No	30	8	4	4	10	3	2	2	2
%	45.45	12.13	6.06	6.06	15.16	4.54	3.03%	3.03	3.03

APLA-Antiphospholipid antibody, OCP-oral contraceptive pill, SLE-Systemic lupus erythematous, PCOD-polycystic ovarian disease

In our study total of 351 (228 males and 123 females) cases of stroke in young were admitted to the Department of Neurology between Jan 2011 to march 2015. Cerebral venous thrombosis accounted for 18.80 %(66/351), Ischemic stroke 60.11 % (211/351) and 21.08 %(74/351) had spontaneous intracerebral haemorrhage (ICH). Incidence of venous stroke is lesser than arterial stroke in a ratio of 1:4.31. Headache, the most frequent and often the earliest symptom, was encountered in 52 patients (78.8%) of our cases. It was present in 75% of the 110 cases reported by Ameri and Bousser (Ameri and Bousser, 1992) and in 41% to 74% in other McLean series (McLean, 1991). Headache and papilledema mimicking idiopathic intracranial hypertension was encountered in 14 patients (21.22%) of our cases. It was reported in 40% of cases in a series from Saudi Arabia (Abdulkader, 1995).

About 56% of our patients were males and alcohol was etiologically related in 45.45% of cases. These data don't match with other studies and probably associated with rise in alcoholism in this part of country. We think this disorder is an important cause of morbidity and mortality in males with habit of alcoholism in this population. According to the etiologic factors of our study we concluded that hormonal changes (from external or internal sources) had a strong phathophysiologic role. We found that oral contraceptive pills/hormonal pills use was the second most common aetiology after alcohol. In our study, cases of CVT due to infectious causes were not recorded. It was the cause in 16% and 17% of the cases reported by Bousser *et al*. (1985) and Shell and Rathe, respectively (Shell 1988).

It is believed that puerperium underlie the development of CVT mainly in developing countries countries. Puerperal CVT, for example, was reported to be responsible for 25% of maternal deaths in India and to complicate 4.5 of 1000 obstetrical admissions (Srinavasan, 1984). Three cases (4.54%) of our patients were in puerperium period and this is less than what recorded in previous studies. This finding shows success of national woman and child programs in recent decade. Routine screening for protein S, protein C, antithrombin III deficiencies, and antiphospholipid antibodies is very important when CVT has no apparent cause. These coagulopathies may have been implicated in many earlier reported cases of idiopathic CVT. We could found a definite cause for all patients of CVT. In many reports about 20% to 30% of the CVT cases had no clear etiology (Bousser and Barnett, 1992; Abdulkader Daif, 1995).

In conclusion, CVT is not uncommon in India among young population; infectious causes are no more common in adults. Alcohol is most common etiological factor of CVT in our study, followed by OCP, protein C/S deficiency, APLA, Behcets disease, post partum state, polycythemia, SLE,PCOD and factor five Leiden mutation in that order.

Seventy eight percent cases presented with headache so brain MRI and MRV should be performed as part of the diagnostic workup in all patients presenting with headache, pseudotumor cerebri,paplledema and other features of CVT. Assessment of coagulation factors such as protein C, protein S, antithrombin III, and antiphospholipid antibodies should also become a routine investigation in every patient who has CVT without apparent cause before labeling it as cryptogenic.

REFERENCES

Bousser, M.G. and Barnett, H.J.M. 1992. Cerebral venous thrombosis. In: Barnett HJM, Mohr JP, Stein BM, Yatsu FM. Stroke Pathophysiology, Diagnosis and Management. 2nd ed. New York, NY: Churchill Livingstone Inc.

Bousser, M.G., Chiras, J., Sauron, B. and Castaigne, P. Cerebral venous thrombosis: a review of 38 cases. Stroke. 1985;16:199-213.

Southwick, F.S., Richardson, E.P. Jr, Swartz, M.N. 1986. Septic thrombosis of the dural venous sinuses. Medicine (Baltimore)., 65:82-106.

Srinavasan, K. 1984. Ischemic cerebrovascular disease in the young: two common causes in India. Stroke. 15:733-735.

Kalbag, R.M., Woolf, A.L. 1967. Cerebral Venous Thrombosis, Vol 1. London, England: University Press.

Ameri, A. and Bousser, M.G. 1992. Cerebral venous thrombosis. Neurol Clin.,10:87-111.

McLean, B. 1991. Dural sinus thrombosis. Br J Hosp Med., 5:226-231.

Abdulkader Daif, Adnan Awada, Saad Al-Rajeh, Mohammed Abduljabbar *et al*. Cerebral Venous Thrombosis in Adults, A Study of 40 Cases From Saudi Arabia. Stroke. 1995;26:1193-1195

Shell, C.L. and Rathe, R.J. 1988. Superior sagittal sinus thrombosis still a killer. West J Med.,149:304-307.

Walter, M. Jul 2005. van den Bergh, Irene van der Schaaf, and Jan van Gijn The spectrum of presentations of venous infarction caused by deep cerebral vein thrombosis Neurology, 65: 192 - 196.

Makkat, S., Tadeusz Stadnik, Els Peeters, Michel Osteaux. May-June 2003. Pathogenesis of venous stroke: evaluation with diffusion- and perfusion-weighted MRI Journal of Stroke and Cerebrovascular Diseases, Volume 12, Issue 3, Pages 132-136.

Lövblad, K.O., Bassetti, C., Schneider, J., Ozdoba, C., Remonda, L. and Schroth, G. 2000. Diffusion-weighted MRI suggests the coexistence of cytotoxic and vasogenic oedema in a case of deep cerebral venous thrombosis. Neuroradiologyni1>rqni2>-l0>. Oct; 42(10):728-31.

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