Case Report

Chronic CVT in stroke in young

Sandeep Yamsani[1*], K Siddappa[2]

[1]Final Year Postgraduate Student, [2]Professor, Department of General Medicine, SS Institute of Medical Sciences and Research Centre, Davangere, Karnataka, INDIA.
Email: sandeep.10010@gmail.com

Abstract

Cerebral sinus venous thrombosis (CSVT) is a rare phenomenon that can be seen with some frequency in young patients. CSVT is a multifactorial condition with gender-related specific causes, with a wide clinical presentation, the leading causes differ between developed and developing countries, converting CSVT in a condition characterized by a highly variable clinical spectra, difficult diagnosis, variable etiologies and prognosis that requires fine medical skills and a high suspicious index. Patients who presents with CSVT should underwent to CT-scan venography (CVT) and to the proper inquiry of the generating cause. This disease can affect the cerebral venous drainage and related anatomical structure. The symptoms may appear in relation to increased intracranial pressure imitating a pseudotumorcerebri. Prognosis depends on the early detection. Correcting the cause, generally the complications can be prevented. Mortality trends have diminished, and with the new technologies, surely it will continue. This work aims to review current knowledge about CSVT including its pathogenesis, etiology, clinical manifestations, diagnosis, and treatment.[1]

Key Words: Brain, cerebral embolism and thrombosis, cerebral sinus venous thrombosis, cerebrovascular disease, neurosurgery, sinus thrombosis.

[*]**Address for Correspondence:**
Dr. Sandeep Yamsani, Final Year Postgraduate Student, Department of General Medicine, SS Institute of Medical Sciences and Research Centre, Davangere, Karnataka, INDIA.
Email: sandeep.10010@gmail.com

Received Date: 20/07/2017 Revised Date: 08/08/2017 Accepted Date: 02/09/2017
DOI: https://doi.org/10.26611/1021331

Access this article online	
Quick Response Code:	Website: www.medpulse.in
	Accessed Date: 06 September 2017

INTRODUCTION

Puerperal cerebral vein thrombosis (CVT) may account for 15-20% of stroke in the young. However it is the commonest cause of stroke in young women in India. In western countries the incidence of puerperal CVT ranges from 1 in 1666-10000 pregnancies. Bansal *et al*[2], reported a higher incidence of these disorders in North India, with a frequency of 4.5 per 1000 obstetric admissions.[2] In Saudi Arabia the incidence was 7 per 1,00,000 patients with the relative frequency in comparison to arterial strokes being 1:62.5.[3] The annual incidence of CVT is estimated to be 3 to 4 per million population of which 75% are women[8]. The reported mortality of 5-30%, will decline with early detection and better treatment.[4]

CASE REPORT

A 27 year old male patient Saleem, a resident of Davangere who was apparently normal 3 months back, developed weakness of left upper and left lower limb around evening. Then patient had loss of consciousness along with weakness, sudden in onset, non-progressive, no aggravating or relieving factors. Patient was shifted to CG hospital where CT scan was done and was told normal. After 1 month, patient started improving. Distal muscle weakness was still remaining. Patient was able to walk with support till the previous day afternoon. Later patient developed headache, sudden in onset, involving whole head, no aggravating and relieving factors associated with vomiting since Monday, sudden in onset watery, foul smelling non blood tinged, no other aggravating or relieving factors, NO history of chest pain, breathlessness. No history of syncopal attacks in past; History of loss of speech, sudden in onset, patient was able to understand questions and respond to commands, but unable to speak. He can perform YES/NO comments with left hand when asked to show left. History of

weakness of lower limb, increased following loss of speech, sudden in onset, patient unable to lift upper and lower limb. No history of diplopia, deviation of angle of mouth, vision loss, sensory disturbances of face; Patient is able to swallow food and water. Patient was able to get sensation of full bladder and void urine. Patient was able to sense hot and cold sensations. No history of neck rigidity, back pain.

Past History: Patient was admitted to CG hospital 4 months back with left hemiplegia; No history of Hypertension, Diabetes mellitus, Cardiac diseases. On clinical Examination the patient was afebrile, with a pulse rate of 66 beats per minute, blood pressure- 160/100 mm of Hg and no pedal oedema. Central Nervous System Examination revealed that the patient was conscious, with loss of speech and able to understand speech indicating motor aphasia. Cranial Nerve Examination was normal. Motor Examination revealed flexed attitude of both the upper limbs. Hematologic studies revealed elevated Prothrombin time (PT = 66.7sec) and Activated Partial Thromboplastin time (APTT = 37.9sec). Lipid profile showed reduced levels of Very Low Density Lipoprotein (VLDL = 9.90 mg/dl). Serological studies (Enzyme Linked Immuno Sorbent Assay) has not revealed any elevation of Anti Cardiolipin Antibody (IgG: 0.6 GPLU/ml, IgM: 1.00 MPLU/ml). MDCT Brain showed gliotic area involving right insular lobe cortex, right temporal lobe cortex and right periventricular white matter. Superior sagittal, Straight sinus and Superficial cortical veins appear hyperdense. Electron Encephalogram (EEG) was abnormal in the form of focal slowing across right frontotemporal lobe region.

DISCUSSION

The causes and association of CVT are similar to those of venous thrombosis elsewhere in the body. To name a few are endocrinological disturbances like oral contraceptive pills, pregnancy, immunological disturbances like anti phospholipid syndrome, connective tissue disorders like bechet's syndrome, sarcoidosis and others. The postulated mechanisms include thrombosis of cerebral veins which cause effects due to venous obstruction, localized cytotoxic cerebral oedema and venous infarction. It may start as ischemic neuronal damage petechiae later merging into large hematomas. Cerebral oedema damages the intracellular membrane pumps leading to intracranial swelling and vasogenic oedema. CVT results in intracranial hypertension due to obstruction of cerebrospinal drainage in the end, therefore ventricles do not dilate. Intracranial hypertension is seen in 20% cases of CVT. Clinical features:- CVT can present in protean ways with a variable mode of onset and wide spectrum of signs. With the growing usage of non-invasive imaging it is apparent that 35% of patients with benign intra cranial hypertension may have underlying CVT. There is slight female preponderance of 1.3:1 (probably reflecting oral contraceptive usage and pregnancy). Its usual onset is in 3rd or 4th decade of life.

Table 1: CVT: Neurological features

Neurological signs and symptoms	Frequency (%)
Recent Headache	95
Focal Deficits	46
Papilledema	41
Paresis	40
Impaired Consciousness (GCS<14)	39
General seizures	37
Dysphasia	22
Isolated intracranial hypertension	20
Coma (GCS<5)	15
Transient Neurological Symptoms	14
Cerebellar signs	10

Management: Treatment of dural sinus thrombosis is influenced by its etiology and the clinical features. In patients with a pure benign intracranial hypertension-like syndrome, diuretics may be sufficient to control the raised intracranial pressure. If these measures fail and visual fields/acuity deteriorate, optic nerve sheath fenestrationor lumbo-peritoneal shunt may be required. Anecdotal reports say that anticoagulation with intravenous heparin may be of benefit[9,11]. There is preliminary evidence to show that heparin improves outcome and can be administered in the presence of hemorrhagic venous infarction. The dramatic response to heparinisation is due to limitation of further thrombogenesis within the superficial cortical veins. In a recent Cochrane review, the authors concluded that anticoagulant treatment for cerebral sinus thrombosis appeared to be safe and was associated with a potentially important reduction in the risk of death or dependency which did not reach statistical significance. The multicentric Dutch-European Cerebral Sinus Thrombosis Trial (DECST) evaluated anticoagulation and benefit with low molecular weight heparin (LMWH) and warfarin and recommended initial therapy with LMWH for 5-7 day, followed by at least three months of warfarin, or more, in cases with an underlying prothrombotic state. Though fibrinolysis or local administration of thrombolytic agents via catheter should be beneficial in patients with CVT, there is insufficient evidence to support its widespread use. An earlier study group has recommended that in view of its invasive and potentially dangerous nature their use should be restricted to cases with a poor prognosis. As per the recent Cochrane review, there is currently no available evidence from randomised controlled trials regarding the efficacy orsafety of thrombolytic therapy in dural sinus thrombosis. In a landmark study on the causes and

predictors of mortality the authors reached two conclusions. First, although CVT has a low case fatality, it is possible to predict some patients who are at increased risk of death. These patients should be closely monitored, and worsening of their clinical condition should be regarded as an indication for more aggressive treatment. Second, given the potential for neurologic recovery after CVT, there is a case for assessing decompressive craniectomy.

REFERENCES

1. Srinivasan K. Cerebral venous thrombosis. In: JS Chopra,IMS Sawhney, eds. Neurology in Tropics. 1st Ed New Delhi: BI Churchill Livingstone Pvt Ltd, 1999: 497-505.
2. Bansal BC, Gupta RR, Prakash C. Stroke during puerparium in young females below yhe age of 40 years as a result of cerebral venous/venous sinus thrombosis. Jpn Heart J 1980; 21: 171-83.
3. Srinivasan K. Cerebral venous and arterial thrombosis in pregnancy and puerparium. Angiology 1983; 134: 731-46.
4. Lamy C, Hamon JB, Coste J, MasJL.Ischemicstrokeinyoung women: Risk of recurrence during subsequent pregnancies. Neurology2000; 55; 269-74.
5. KittnerSJ,SternBJ, Feeser BR, HebelJR, NageyDA, BuchholzDW, et al. Pregnancy and the Riskof Stroke.NEnglJMed1996; 335: 768-74.
6. Lanska DJ, Kryscio RJ. Risk factors for puerparium, postpartum stroke and intracranial venous thrombosis. Stroke2000; 31: 1274-82.
7. Daif, et al. Cerebralvenousthrombo is in adults. Astudy of 40 cases from SaudiArabia.Stroke1995; 26:1193-5.
8. StamJ.ThrombosisoftheCerebralVeinsandSinuses.NEnglJ Med2005; 352:1791-8.
9. Bousser MG. Cerebralvenousthrombosis:diagnosisand management.JNeurol2000;247:252-8.
10. Allogren H, AbbotRJ.Cerebralvenousthrombosis.PostgradMedJ2000; 76:5-12.
11. DeBruijn SF, deHaanRJ, StamJ. Clinicalfeaturesand prognosticfactorsofCVTinaprospectiveseriesof59patients. JNeurolNeurosurgPsychiatry2001; 70:105-8.
12. FeroJM, CanhaoP, StamJ, Bousser MG, Barinagarrementeria F.Prognosisofcerebralvein and duralsinusthrombosis: results of the International Studyon Cerebral Vein and DuralSinus Thrombosis (ISCVT). Stroke 2004; 35: 664-70.
13. De Bruijn SFTM, Stam J, Kappelle LJ. Thunderclap headache as first symptomofcerebralvenoussinusthrombosis. Lancet 1996; 348: 1623-5.
14. JacobsK, MoulinT, BogousslavskyJ, WoimantF, DehaeneI, TatuL, et al. The stroke syndrome of corticalveinthrombosis. Neurology1996; 47:376-82.

Source of Support: None Declared
Conflict of Interest: None Declared