ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 02 | February - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A RETROSPECTIVE STUDY OF ETIOLOGY, CLINICAL AND NEUROIMAGING IN PATIENTS WITH CEREBRAL SINUS VENOUS THROMBOSIS

General Medicine

Dr. V. Anjaneya Prasad Professor & HOD, Department Of Medicine, Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences & Research Foundation.

Dr. A. Santhosh Kumar Assistant Professor, Department Of Medicine,

Dr. P. Valli Krishna Priya* Junior Resident, Department Of Medicine, Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences & Research Foundation. *Corresponding Author

ABSTRACT

Thrombosis of Cerebral vein and dural sinus is a rare multifactorial phenomenon. The incidence is underestimated because it is now known to have a varied clinical spectrum than previously realized and can be more challenging to diagnose. CSVT requires high Suspicious index due to the heterogeneity in etiological factors identified in up to 80% of patients. Neuroimaging is the corner stone in the diagnosis. MRI with MRV is almost 100% Diagnostic.

KEYWORDS

CSVT, Sinuses

INTRODUCTION:

Thrombosis of Cerebral vein and dural sinus is a rare multifactorial phenomenon. The annual incidence is estimated to be 1.5 cases per 100,000 population[1-3] However, the incidence is underestimated because it is now known to have a varied clinical spectrum than previously realized and can be more challenging to diagnose. In the international study of CSVT, mean age was 39 years and more common in females than in males with the ratio of 2.9:1 (Ferro et al., 2004) [1].

CSVT requires high suspicious index due to the heterogeneity in etiological factors identified in up to 80% of patients [4]. It accounts for 10-20 % of the etiology of young strokes in India [5] and is less common than most other types of stroke. Many of the previously held concepts about CSVT are changing with the widespread use of neuroimaging, rising clinical awareness and hematological workup and it's recognized with increasing frequency. [6,7] Current therapeutic options for CSVT include anti-thrombotic therapy with un-fractionated heparin, low-molecular weight heparins (LMWH), oral anticoagulants, intravenous thrombolysis in addition to symptomatic therapy.[2]

Neuroimaging is the corner stone in the diagnosis of cerebral venous sinus thrombosis. Imaging modalities of choice in CSVT and CT scan and MRI with MR venogram. CT scan may be normal in 15-30% cases but MRI with MRV is almost 100% diagnostic[7].

MATERIALS AND METHODS:

45 patients suspected to have CSVT subjected to clinical evaluation were retrospectively studied from June 2021 to July 2022 who were followed until discharge from the hospital. The objective of this study was to evaluate the risk factors, clinical profile, duration of illness, blood investigations and outcome of patients and aims to review current knowledge about CSVT. The diagnosis was confirmed on CT scan brain or MRI brain /MRV. Patients were treated with adjusted dose unfractionated heparin / low molecular weight heparin followed by oral anticoagulation therapy with an INR of 2 – 3.

Laboratory investigations:

1. Complete blood count with peripheral smear (Hb, TC, DC, Platelet counts)
2. ESR
3. RBS
4. Serum urea
5. Serum creatinine
6. FLP, serum electrolytes, HIV
7. Urine routine
8. ECG in all leads
9. CSF analysis (wherever deemed necessary)
10. BT, CT, Prothrombin time with INR, activated partial thromboplastin time.
11. CT scan
12. MRI and MR venogram.
13. Fundus examination.

Inclusion Criteria:

1. Patients with suspected cerebral venous thrombosis by clinical features.
2. Any age group presenting with Cerebral venous thrombosis.
3. Puerperal and non puerperal group.

Diagnostic Criteria:

Patients presenting with history and examination suggestive of cerebral venous thrombosis and confirmed by imaging of brain (CT scan direct and indirect signs, MRI, MRV).

A) Direct signs:
a. Hyper dense sinus on plain CT
b. Cord sign on plain CT
c. Empty delta sign on contrast enhanced CT
d. Dense triangle sign on plain CT.

B) Indirect signs:
a. Cerebral edema
b. Cerebral infarction not confirming to as arterial territory
c. Cerebral hemorrhage.
d. Small ventricles
e. Bilateral signs
f. Gyral enhancement
g. Territorial enhancement
h. Erosion of middle ear structures and changes in mastoid region

Exclusion Criteria:

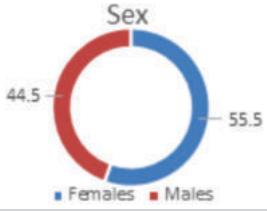
1. Hypertension,
2. Primary seizure disorder,
3. HIV positive patients

Statistical Analysis:

The results were analyzed by calculating percentages, mean values, standard deviation and chi-square test.

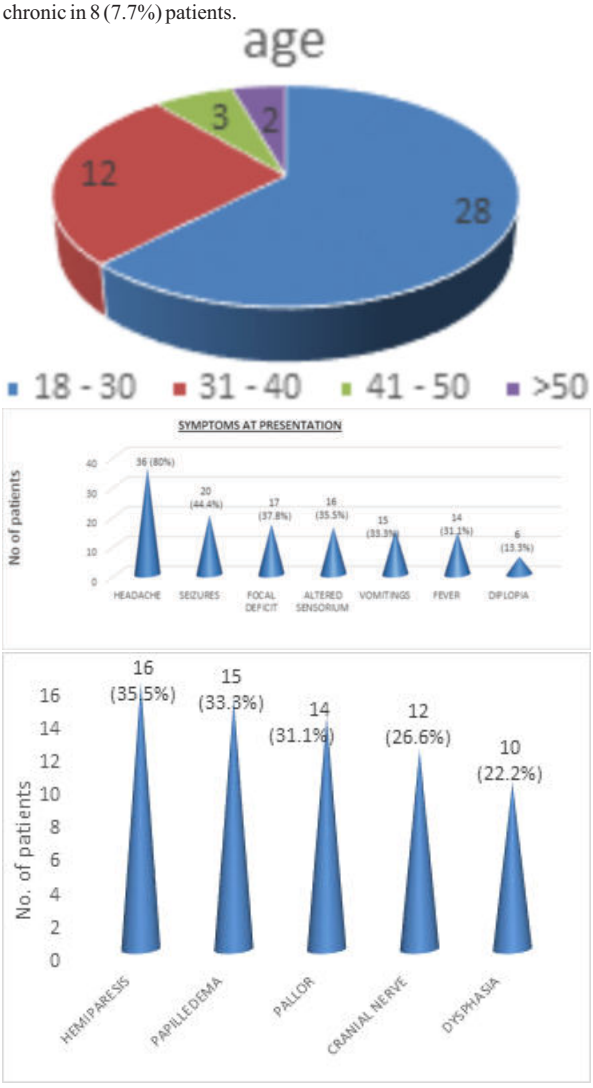
Proportions were compared using chi-square test to study their association and 'P' value of less than 0.05 was considered statistically significant.

RESULTS:



Of the 45 patients, the mean age was 29.7± 8.1 years, with maximum incidence in age group 18-30 years.

Presentation was acute in 20 (44.4%), subacute in 17 (37.8%) and chronic in 8 (7.7%) patients.



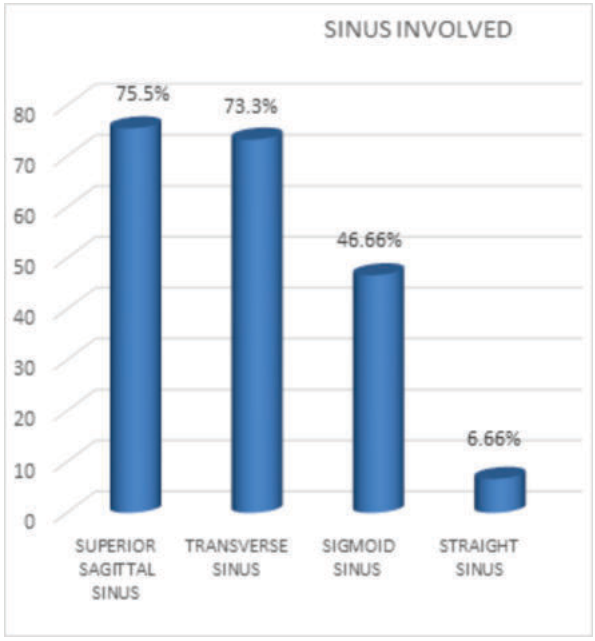
Clinical Signs at Presentation

DISCUSSION:

Previous studies had limitations of having small numbers and incomplete investigations[8,9] Headache associated with raised intracranial pressure and seizures are the common presenting features. Anemia, alcoholism and hyper homocysteinemia were the main risk factors identified. [10]

The predictors of good outcome were high GCS at presentation and superficial venous system involvement. Poor outcome was seen in patients with fever, low GCS at presentation, and focal deficits. In few Asian studies puerperium CSVT is 31–86% as reported[7,11]. Less cases in this study probably represent better women healthcare.

The underlying pathogenesis of CSVT involves two mechanisms. The first includes localized oedema and venous infarction due to cerebral vein occlusion. The second mechanism constitutes the development of raised intracranial pressure due to occlusion of one of the cerebral venous sinuses, since CSF absorbed by arachnoid villi drains into the superior sagittal sinus. The manifestations that indicate the cerebral cortical involvement are convulsions and paralysis, at times seizures are heralding symptoms and should arouse the suspicion of diagnosis. Predisposing risk factors include prothrombotic conditions, head injury, inflammatory disease dehydration and malignancy. Headache was the most common symptom in the present study accounting for 80% of patients. The present study is comparable with Daif [11] et al (1995) 82% and 44.4% of cases had seizures. Anemia has often been noted in 10 (22.2%) of the patients in the present study has been hallmark in puerperal CVT. In the present study, the superior sagittal sinus is most commonly involved accounting for 75.5% followed by transverse sinus with 73.3%.



MODIFIED RANKING SCALE	NO. OF CASES AT DISCHARGE	
0	12	26.6%
1	10	22.2%
2	9	20%
3	8	17.7%
4	2	4.44%
5	1	2.22%
6(DEATHS)	3	6.67%

CONCLUSION:

- The present study emphasizes that CSVT is not an uncommon condition and is one of the treatable, common causes of stroke in young.
- Risk factors like anemia, hyperhomocysteinemia, OCP use, alcoholism and prothrombotic state are being increasingly identified, while conventional risk factors like postpartum state are decreasing.
- MRI was found to be better than CT in identification of cases and MRV is the most sensitive technique to confirm the diagnosis. [9].
- Outcome was good in early detection of CSVT, correcting the underlying cause, effective management with heparin/oral anticoagulants and generally preventing the complications.

REFERENCES:

1. Ferro JM, Correia M, Ponter C, For ISCVT investigators. Prognosis of cerebral venous and dural sinus thrombosis, Results of ISCVT. Stroke2004;35:664-70.
2. Saposnik G, Barinagarrementeria F, Brown RD Jr, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011; 42:1158.
3. Coutinho JM, Zuurbier SM, Aramideh M, Stam J. The incidence of cerebral venous thrombosis: a cross-sectional study. Stroke 2012; 43:3375.
4. Bousser MG, Russell RR: *Cerebral venous thrombosis* London: WB Saunders; 1997.
5. Lath R, Kumar S, Reddy R, Boola GR, Ray A, Prabhakar S, Ranjan A. severe cerebral venous sinus thrombosis. *Neurol India* 2010; 58:392-7.
6. Kalita J, Bandal V, Misra UK, Phadke RV. Cerebral venous sinus thrombosis in a tertiary care setting in India. QJM 2006;99:491-2
7. Nagaraja D, Sarma GR. Treatment of cerebral sinus/venous thrombosis Neurol India 2002;50:114-6.3. Kalita J, Bandal V, Misra UK, Phadke RV. Cerebral venous sinus thrombosis in a tertiary care setting in India. QJM 2006;99:491-2
8. de Veber G, Andrew M. And the Canadian Ischemic Stroke Study Group. outcome of sinovenous thrombosis in patients. N Engl J Med 2001;345:417-23.
9. Anderson CM, Edelman RR, Turski PA. Magnetic resonance venography.Vol. 1. Raven, Philadelphia: Lippincott; 1993. pp. 289
10. van den Bergh WM, van der Schaaf I, van Gijn J. The spectrum of presentations of venous infarction caused by deep cerebral vein thrombosis. Neurology 2005; 65:192
11. Khealani BA, Wasay M, Saadah M, Sultana E, Mustafa S, Shohab Khan F, et al. Cerebral venous thrombosis A descriptive multicenter study of patients in Middle East. Stroke 2008;39:2707-11.
12. Haraidas TT, Taly AB, Veerendra Kumar MM, Subbu Krishna DK. Puerperal cerebral venous thrombosis. Therapeutic benefits of low dose Heparin. Neurol India 1999;47:43-6.