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Case Report
CSVT

A rare presentation of hyperhomocysteinemia and folate deficiency as CSVT in young male

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Introduction: Cerebral sinus venous thrombosis (CSVT) is a relatively rare, potentially fatal neurological condition that can be frequently overlooked due to its vague nature and its varied spectrum of clinical presentation. It's a multifactorial condition with gender-related specific causes. It's a rare form of venous thromboembolism representing almost 0.5-3 % of all types of stroke, affecting predominantly younger people, and females three times more commonly affected than males. Incidents in adults are 3- 4 million. The diagnosis of CSVT is becoming easier now days due to advanced neuroimaging techniques. Abnormality in the thrombophilic profile is associated with enhanced risk of CSVT. It has varied etiologies such as the Hypercoagulable States (inherited) that is hyperhomocysteinemia, protein C and protein S deficiency, Antithrombin-III deficiency, Factor V leiden mutations, autoimmune causes. Other acquired causes like CNS infection, trauma, dehydration, pregnancy, substances abuse and oral contraceptives.

Keywords: Hyperhomocysteinemia, Cerebral sinus venous thrombosis (CSVT)

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Case presentation

A 17-year-old male presented to the emergency department with a history of headache for 15 days, diffuse but mostly in occipital and left temporal region, giddiness, 3- 4 episodes of vomitings in the last three days and one episode of generalised tonic-clonic seizures. There was no history of photo-phobia or aura, no history of trauma.

His past medical history was not suggestive of any major illness/drugs/similar complaints. For headaches, he visited local doctors and had been treated with acetaminophen and multivitamin injections. His family history had no contribution and he is a non-smoker/non-alcoholic.

He was afebrile, heart rate: 71/min, B.P:130/80 mm /Hg.

He was conscious, coherent, oriented to time place and person. Later on the day of admission, he developed weakness and numbness on the left side of his body. On examination power in the left extremities was 4/5 with Basinski's positive. His respiratory, cardiovascular and abdominal examination was unremarkable. Previously CT brain was done in the emergency department, which was inconclusive. Later on, the patient was sent for an MRI brain, where the T2 weighted axial images showed a hyperintense signal in the posterior part of the superior sagittal sinus, extending along the left transverse sinus. Magnetic resonance venogram image showed non-visualisation of

- The entire superior sagittal sinus.
- Left transverse sinus.
- Left sigmoid sinus.

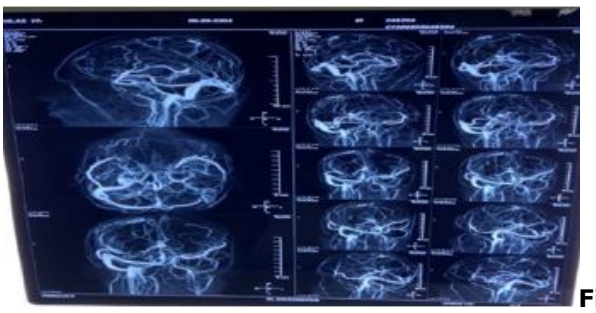

Fig 1: **MRI Brain showing Non visualization of Sinus.**

Diagnosis of CSVT was established and further hematological workup was done for the cause of CSVT. His preliminary blood examination showed Megaloblastic anemia with hemoglobin of 10.2gm/dl. His vitamin B9 levels were 1.8(3.1-20.5). HSCRP was 10.20(<1.0), homocystein level was 34.2(5-15).ANA profile, ANCA, lupus anticoagulant and antiphospholipid antibodies were negative. Thrombophilic work up: protein- C, protein-S and antithrombin-3 were in normal percentage. Factor-V leiden mutation was negative, hence it was concluded that the patient had CSVT due to hyperhomocysteinemia which was most probably due to folate deficiency. The patient was treated with IV levetiracetam500mg twice daily and LMWH(1mg/kg) every 12th hour for 7 days. His symptoms subsided and power gradually improved. He was seizure-free for one week. So discharged on orally administered levetiracetam, warfarin(1mg) and folic acid (5mg). On subsequent follow-up, his INR was monitored and was maintained in the therapeutic range. He showed significant neurological improvement and was symptom-free.

Discussion

This case highlights the rare occurrence of CSVT in young males, with very common symptoms like a headache which can be easily neglected. The prevalence of hyperhomocysteinemia is approximately 1% in the general population. Can be caused by genetic and acquired factors including folate and cobalamin deficiency. B12 act as a cofactor for the enzyme methionine synthase, which forms a part of s- adenosine methionine biosynthesis and regeneration cycle.B12 deficiency prevents 5 -MTHF from being converted into THF. The folate deficiency in this pathway leads to an increase in homocysteine which has a nature of damaging the endothelial cells and further increasing the risk of thrombosis. Homocysteine is a non-protein amino acid synthesized from methionine and either recycled back or converted into cysteine with the help of a B GROUP of vitamins. This conversion back requires active folate (5 MTHF) & B12 whose deficiency leads to hyperhomocysteinemia. From the study conducted by Jayanth k, *et al* it was concluded that hyperhomocysteinemia was an important correctable risk factor of CVST in patients from northern India, and the majority of them had either low vitamin B12 level or *MTHFR* mutation [5].

Conclusion

We conclude that hyperhomocysteinemia is an independent risk factor for CSVT inpatient with the group of vitamin B group deficiency. It can be easily reverted with vitamin supplements. In our view, all the patients with CSVT should be checked for homocysteine level for whom there is no other obvious predisposing factors for venous thrombosis.

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