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CASE REPORT
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Deep Cerebral Venous Sinus Thrombosis in an Alcohol Dependent Male: An Important Differential for Wernicke's Encephalopathy

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Abstract

Wernicke's encephalopathy (WE) is suspected in patients with chronic alcoholism with poor nutrition presenting with walking difficulty, ocular problems and fluctuating orientation. However, one must quickly consider other possibilities if typical radiological features are absent or unexplained symptoms are present. A man in his mid-40s with an alcohol intake of ten years presented with walking difficulty, nystagmus, bilateral lateral rectus palsy and fluctuating orientation for ten days, along with deficits in recent memory. He had a history of poor nutrition for three months, diarrhoea for one month, and severe diffuse headache and slurred speech for ten days. Plain MRI-Brain revealed an acute non-haemorrhagic left thalamic infarct and chronic infarct/early gliosis in the left cerebellar hemisphere and left temporal lobe. Contrast-enhanced MR-Venography revealed cerebral venous sinus thrombosis. The patient showed rapid improvement with high-dose parenteral thiamine and low molecular weight heparin and is currently maintaining well on 3 milligrams of warfarin.

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Introduction

Cerebral venous sinus thrombosis is a rare condition that includes thrombosis in cerebral veins or the dural sinuses. It has a highly variable presentation, including headaches, seizures and altered mental states. Any pro-thrombotic state, such as pregnancy, puerperium, oral contraceptive intake, or hormone replacement therapy, may precipitate the condition. Because of these gender-specific risk factors, the prevalence is three times higher among females compared to males.[1] Multiple studies have reported that alcoholism leads to dehydration and blood hyperviscosity, thereby becoming a unique risk factor for the condition among males with chronic alcoholism.[2-5] In any case of chronic alcoholism, Wernicke's encephalopathy (WE) is a severe but easily treatable metabolic disorder that must be looked for, since up to 80% of cases go undiagnosed and may lead to irreversible memory impairment if untreated, and the classic triad of symptoms appears in only 16–33% of cases.[6,7]

This case report focuses on the importance of considering these two entities and differentiating between them while evaluating patients presenting with chronic alcohol consumption.

Case Presentation

A man in his mid-40s presented to the psychiatry OPD along with his wife with complaints of difficulty walking, blurred vision and fluctuating orientation for 10 days associated with on-and-off severe diffuse headache. He also had a history of poor oral intake and frequent diarrhoea for one month. Ten days prior, he had two episodes of blood-tinged vomiting and agitation. The patient had a history of intake of alcohol intake for 10 years, with average consumption being 90 mL of Indian-made foreign liquor 2 to 3 times per week. The drinking became regular 5 years back, consuming 180 ml of whiskey every evening with friends due to an irresistible desire, especially after work. For the last two years, he also reported withdrawal symptoms. For the past three months, he had been trying to quit alcohol. At the time of presentation, he had been off alcohol for at least 10 days. The patient's past medical or psychiatric history was insignificant.

On admission, the patient's vital signs were within normal limits, and blood oxygen saturation was well above 97% in room air. His BMI was 24.6 kg/m^2. He was oriented to time, place and person but could not recollect events of the recent past. On mental status examination, the patient was apathetic, had slurred speech, mildly irritable affect, no thought/perceptual abnormalities, good insight into his illness and had a contemplation stage of motivation for alcohol use.

He was responding normally to verbal commands. He had horizontal nystagmus in both eyes, along with bilateral lateral rectus palsy. There was a mild blurring of disc margins on fundus examination. Power examination revealed Medical Research Council (MRC) grade 5/5 in all four limbs. All deep tendon reflexes were brisk. Bilateral plantar was equivocal. He was unable to walk on his own on the day of admission, but was able to do so with support.

Assessment for alcohol dependence and withdrawal: At the time of admission, he scored 8 (Anxiety-3; Headache-5) on the CIWA-Ar (Clinical Institute Withdrawal Assessment-Alcohol Revised) scale. The patient also scored 15 (mild dependence) on the Severity of Alcohol Dependence Questionnaire (SAD-Q) applied on the 4th day post-admission.

Investigations

Routine investigations showed raised MCV (107.5fL), raised TSH (5.8 mIU/L) and raised FBS (149.7 mg/dl), PPBS (314.5 mg/dl) and HbA1C (7.70%), and low vitamin D3 (8.88 ng/mL). Liver function tests were grossly within normal limits. Serum magnesium was normal (1.95 mg/dL). Serum homocysteine level was raised (28.30 µmol/L). MRI brain (plain) was done, which revealed an acute non-haemorrhagic infarct in the left thalamus (Figure 1C & 1D), chronic infarct/early gliosis with hemosiderin staining in the left cerebellar hemisphere and the left temporal lobe (Figure 1A), and loss of flow void in bilateral sigmoid sinuses and left transverse sinus. Due to the presence of a few red flags such history of headache and vomiting suggesting features of raised intracranial pressure, and the presence of apathetic attitude, slurred speech and mildly irritable affect which could not be explained by Wernicke's Encephalopathy alone, MR-venography with contrast was done which revealed attenuation of flow-related enhancement in the left transverse sinus, sigmoid sinus and internal jugular vein and a patchy filling defect in the anterior aspect of the superior sagittal sinus suggestive of venous thrombosis (Figure 1B).

Neuropsychological Assessment

A neuropsychological assessment of the patient was done using the NIMHANS (National Institute of Mental Health and Neurosciences) Neuropsychological battery on day four of admission. The patient scored low on verbal fluency (6.33 on the Controlled Oral Word Association Test) and categorical fluency (08 words on the animal naming test). The patient's scores on other domains of the test were within normal limits.

Diagnosis

Initially, the presence of a classic triad of gait disturbance, eye signs and fluctuating orientation against the backdrop of chronic alcohol intake and

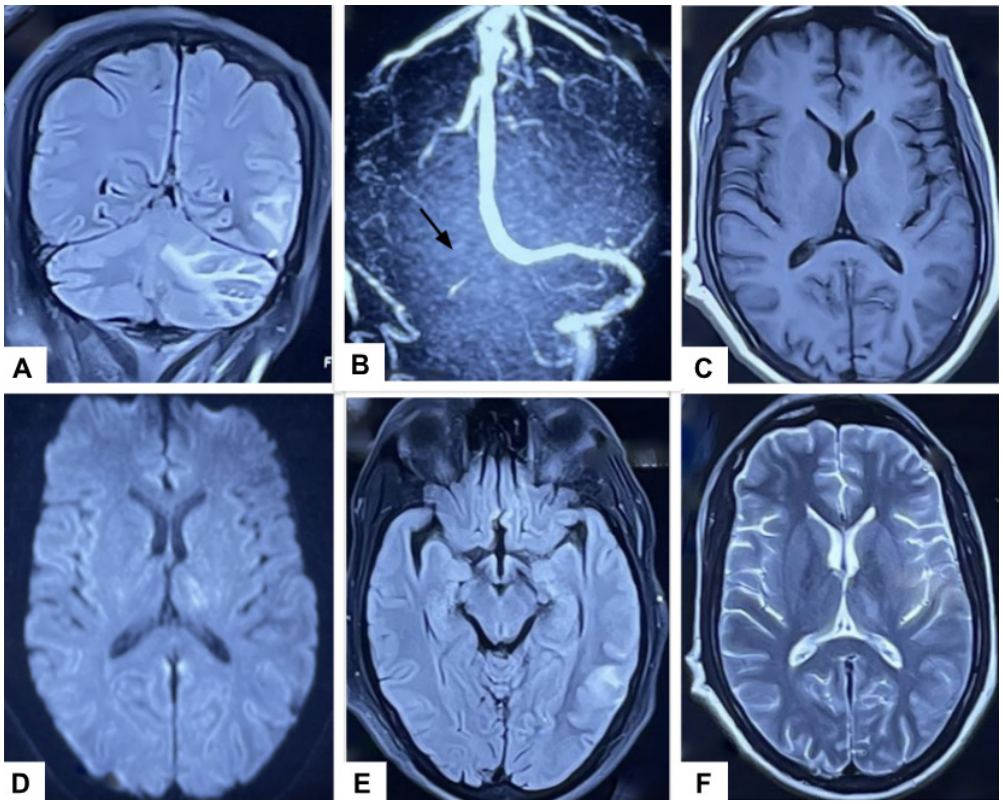

Figure 1: MRI Brain showing an acute non-haemorrhagic infarct in the left thalamus (C, D), chronic infarct/early gliosis with hemosiderin staining in the left cerebellar hemisphere and the left temporal lobe (A), and loss of flow void in bilateral sigmoid sinuses and left transverse sinus. Contrast-enhanced MR-Venography showing attenuation of flow-related enhancement in the left transverse sinus, sigmoid sinus and internal jugular vein and a patchy filling defect in the anterior aspect of the superior sagittal sinus, suggestive of venous thrombosis (B).

poor nutrition was highly suggestive of Wernicke's encephalopathy as per Caine's criteria. So, a provisional diagnosis of alcohol dependence syndrome and Wernicke's encephalopathy was made. However, after radiological evidence failed to show Wernicke's encephalopathy lesions, but instead showed venous sinus thrombosis and thalamic infarct, the diagnosis was appropriately revised to alcohol dependence syndrome and Cerebral venous sinus thrombosis (CVST) and Thalamic Infarct.

Treatment

The patient was started on high-dose parenteral thiamine 1500 mg/day, along with an injection of pantoprazole. Benzodiazepine was added on prn basis for mild anxiety and sleep. Following neurology referral and diagnosis of cerebral venous thrombosis, he was started on injection low molecular weight heparin (LMWH) 0.6 mL twice daily along with tablet warfarin 2 mg in the evening. The dose of tablet warfarin was optimised until the target INR was achieved, and LMWH was stopped after five days. The patient's INR was maintained on 3 milligrams of tablet warfarin. After the initial 5 days, the dose of parenteral thiamine was decreased to 750 mg/day for another five days, after which it was switched to oral formulation, 300 mg/day. Tab levothyroxine 25 mcg and tab metformin 500 + Glimepiride 1mg were started to manage his hyperglycemia and hypothyroidism. Vitamin D3 supplementation was also given. The headache subsided from the second day of admission, while his ocular signs almost completely resolved by the 5th day. From the 3rd day, he could walk on his own but had a prominent, wide-based ataxic gait with a tendency to sway on the left side, which also gradually resolved completely in 2 weeks. He was discharged after 2 weeks with residual symptoms of complaints of intermittent headache on the right temporal area lasting for 1 to 2 hours per day, mild in intensity.

Outcome and Follow-up

The patient was discharged on day 14 of admission. His only lingering symptom was a mild intermittent headache. The patient's warfarin was maintained at 3 mg with an INR of 2.9. Thiamine was continued, and so were the medications for diabetes and hypothyroidism. The patient has been regular with his follow-up for the last two months and has been abstinent from alcohol use.

Discussion

At face value, the patient had all the risk factors for developing Wernicke's Encephalopathy: chronic alcohol use, poor nutrition, and recurrent diarrhoea. Despite lacking typical radiological features, the history and symptoms still warranted high-dose thiamine (1500 mg/day for five days), which was duly given to the patient with no delays, as an untreated deficiency could lead to severe cellular damage resulting in permanent Korsakoff's syndrome or even death.[8] This is because thiamine is an essential cofactor in the TCA cycle and pentose-phosphate pathway, and its deficiency impairs ATP, DNA/RNA, and NADPH synthesis, leading to toxic metabolite accumulation.[9]

Our patient had cerebral venous sinus thrombosis and a left thalamic infarct. Nystagmus and gait disturbance could have resulted from the cerebellar involvement. The decreased verbal and categorical fluency test scores, the apathetic attitude and recent memory impairment were typical of left-sided thalamic infarct.[10] The involvement of multiple brain regions could explain all the symptoms in the patient, but the important question remains: what could have been primarily responsible for the cerebrovascular event? Especially given the fact that the patient had no history of any significant medical condition.

Elevated homocysteine level in the patient points to a pro-thrombotic state.[11] From as early as 1983, alcohol intake has been shown to increase blood viscosity.[2] A more recent study was conducted by Saribal D. 2019 among 20 males with chronic alcohol consumption and 20 age- and sex-matched controls.[4] He found that the mean blood viscosity of the study group was significantly higher than the mean viscosity of the control group. Alcohol causes blood hyperviscosity by increasing serum osmolality, which results in the shrinkage of red blood cells. Kumari and colleagues (2005) suggested that decreased blood fibrinogen causes blood hyperviscosity, while Mukamal and colleagues. 2001 claimed that alcohol can decrease blood fibrinogen levels.[12,13] Other mechanisms, such as decreased synthesis of thromboxane A2 and increased lipids & cholesterol by alcohol, also play a role.[14,15]

In 2014, a case of Wernicke's Encephalopathy mimicking stroke was reported by Bhan and colleagues. Our case seems to be the exact reverse of the above case report: a patient with chronic alcoholism presenting with a clinical history pointing strongly towards Wernicke's encephalopathy but no radiological evidence to support it, and instead showed evidence of stroke. Nonetheless, the case offers a unique learning point: how alcohol uniquely predisposes a patient with chronic alcoholism to develop cerebral venous sinus thrombosis by way of dehydration and increased blood viscosity, and as such, the possibility of comorbid cerebral venous thrombosis must always be entertained in chronic alcoholic patients.

Patient's Perspective

I was having a headache when I was brought to the hospital, and did not feel like talking with anyone. But the headache went away after a couple of days. I used to drink alcohol every day for the last 5 years or so. I was not drinking too much, only about 180 ml of whiskey in the evening. But I was largely unable to control myself from drinking, as I used to drink in the company of colleagues at the workplace. I was working as a supervisor for cooks at a canteen of an engineering college in Gorakhpur, while my family was back home in Deoria. I had not been eating well for the last 3 months, and also had frequent loose stools for 1 month. But I did not think it was anything serious, and as my family was not with me, I did not care to visit the hospital sooner. Then I think it was about 2 weeks back, one night I had a terrible headache and vomited as well. And the vomit even contained some blood on it. Thereafter, what happened after that is hazy in my memory. But I remember being brought here, the hospital.

I could not walk on my own, and my vision was all blurred. But with treatment, I gradually recovered. First thing, my headache subsided after a couple of days, and after a few more days, my vision improved. And towards the time of discharge, I was able to walk steadily on my own. I feel like I have been brought back from the depths of despair. I agree that alcohol played a major role in this illness, and from now I give my word that I will never touch alcohol again. I will also change my profession. I will stay at home with my family in Deoria and look for another job. I can open up a roadside food truck.

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