A Rare Case of Alcohol Intoxication Masquerading Cerebral Venous Sinus Thrombosis

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ABSTRACT

Cerebral venous sinus thrombosis remains a diagnostic challenge courtesy to its variable degree of presentation and low level of clinical suspicion. We report a rare case of cerebral venous sinus thrombosis presenting as a complication of alcohol intoxication. Symptoms of cerebral venous sinus thrombosis are likely to remain camouflaged in alcoholics making the task for the clinician more so difficult. Strong suspicion should remain in any alcoholics with persistent neurologic features. A 52-year-old gentleman presenting with delirium, seizure, and a persistent headache was found to have multiple cerebral venous sinus thrombosis. After ruling out all the possible risk factors eventually we came into the conclusion of the role of alcohol inducing this catastrophic condition. He was treated with anticoagulant resulting in prompt resolution of his symptoms. Anticoagulation was discontinued after 3 months and the patient continues to remain asymptomatic after 6 months of regular follow-up.

Keywords: Cerebral venous sinus thrombosis, alcohol intoxication, anticoagulation, thrombophilia, headache, seizure.

I. CASE PRESENTATION

A 52-year-old gentleman was brought into the emergency unit by the paramedics as found to be less responsive and delirious. On arrival, he had repeated episodes of generalized tonic-clonic seizures each lasting for about 2 to 3 minutes requiring anti-epileptic medication to terminate. He had a strong alcohol odor of his breath. He is a chronic alcoholic with previous multiple admissions to the hospital with alcohol intoxication and once with a traumatic sub-dural hematoma. As his orientation improved, he complained of constant headaches. On examination, he was malnourished, hemodynamically stable. He was responding to verbal commands with initial AMT score 2 out of 10 which improved gradually to 8 out of 10. He had no motor or sensory deficits and a bilaterally equally reactive pupil of 3 mm size. Fundoscopy was normal. Planters were bilaterally downgoing. In view of likely alcohol intoxication, he was initially treated with fluids containing dextrose and intravenous vitamin supplementation. He continued to suffer from headaches despite being given repeated analgesics warranting the need for a brain scan. Despite CT head ruling out a new stroke or a brain bleed likely association of a venous sinus thrombosis prompted the treating team to perform MRV which confirmed the presence of multiple venous sinus thrombosis. After ruling out all the possible risk factors eventually we came into the conclusion of the role of alcohol inducing this catastrophic condition.

II. DIFFERENTIAL DIAGNOSES AND LABS

Routine labs including full blood count, renal function tests and thyroid function test, clotting screen, serum electrolytes, serum homocysteine, folate, and vitamin B12 levels all were normal. Liver function tests revealed elevated serum glutamic-oxaloacetic transaminase levels (87 U/L). Alcohol levels came back at 4300 mg/L. Toxicology screen was negative. Liver ultrasound remained unremarkable. MRV confirmed the presence of diffuse thrombosis of right transverse and sigmoid sinus and partial thrombosis of the superior sagittal sinus. A thrombophilia screening (anti-thrombin 3, protein C, protein S, factor 5 Leiden) was planned after 3 months which came back as normal along with negative anti-phospholipid antibodies.

III. TREATMENT

He was treated with subcutaneous low molecular weight heparin (enoxaparin) and antiepileptic (levetiracetam) along with intravenous dextrose fluid infusion and vitamin supplementation. He was discharged after 5 days hospital course with significant improvement of cognition. He was
referred to the alcohol rehabilitation clinic, anti-coagulation clinic, stroke clinic for further follow-up. He was planned for thrombophilia screening three months after discharge from the hospital which ruled out any possibility of thrombophilia.

IV. BACKGROUND

Cerebral venous sinus thrombosis accounts for a small percentage of all ischemic events but unfortunately is associated with a variable range of neurological deterioration.[1] Most alarmingly could be missed due to its variable range of presentation, therefore; early diagnosis of a CVST can be a grueling task and often misleading. Although alcoholics are predisposed to cerebrovascular accidents yet alcohol related CVST remains an elusive diagnosis [2]. A holistic approach and high index of clinical suspicion are required to identify this entity to ensure prompt treatment with anti-coagulants which often can result in complete neurological recovery and saving the patient from devastating neurological complications. We present a rare case of CVST in a chronic alcoholic who presented with delirium and seizure.

V. DISCUSSION

With advancements in the fields of medicine and technology, arriving at a diagnosis seems to be easier for many previously known complex medical conditions. However Cerebral Venous Sinus Thrombosis (CVST) remains an elusive diagnosis for many clinicians due to its broad spectrum of presentations alongside variability in radiologic appearances and often poses a quagmire of diagnostic challenges for the treating clinician [1]. The first detailed description of CVST dates back to 1825, though the findings were noted in autopsy specimens and not in the living [1]. In recent times however, due to the advancements in imaging modalities, more cases are being diagnosed and treated, rescuing patients from life-threatening neuropathological complications.

Although in 25% of cases the etiology remains elusive, the usual risk factors of CVST are meningitis, parameningeal infections, facial and paranasal sinus infections, thrombophilia, antiphospholipid syndrome, homocystinuria, pregnancy, alcoholism, dehydration, malignancies, paroxysmal nocturnal hemoglobinuria, and contraceptive medications [3]. 25% of cases etiology remains elusive [3]. Superior sagittal sinus remains the most common site for thrombus formation (72%), followed by lateral sinus (70%) [3]. At times more than one sinus is involved (30-40%) [3].

It is to be noted rarely alcohol intoxication or binge drinking, leads to CVST [4]. It is also interesting to note that chronic heavy drinking is a risk factor for cerebral infarction whilst moderate drinking has a protective effect [4]. In an Indian study, male alcoholism has been found to be linked with CVST [1].

The pathophysiology for chronic alcoholism contributing to cerebral venous sinus thrombosis is not yet clear but it is suggested that dehydration and hyperviscosity associated with alcoholism is the likely cause [2]. Virchow triad (Stasis, hypercoagulability, endothelial dysfunction) are known to be the contributing factor for development of any venous thrombosis; therefor, another suggested mechanism for alcohol induced thrombosis formation is endothelial dysfunction [5]. Alcohol causes endothelial dysfunction through Nitric Oxide (NO) pathway as long-term alcohol consumption interferes with NO production or endothelial release of NO [5]. In addition, the proapoptotic caspase pathway is activated by high ethanol levels in the blood [5]. Alcohol consumption also predisposes to thrombus formation by influencing on coagulation, fibrinolysis and platelet activities [6], [7]. Acute alcohol intoxication can cause transient thrombexane-mediated platelet activation and hypercoagulability [8],[9]. Liver dysfunction secondary to alcoholism results in decreased hepatic synthesis of anticoagulant thrombotic factor [10]. Inflammatory response related to metabolic effects of ethanol on the liver activates coagulation [11], [12]. All such alcohol associated hypercoagulability state increases risk of development of venous thrombosis.

Headache remains the most common clinical presentation (75-90%) whereas nausea, vomiting, visual problems, papilledema, delirium, and focal neurological lesions have all been reported [1], [3]. Seizures are more common in men than in women [1]. Altered mental status with persistent headache, seizure, or any other focal neurological deterioration in alcoholics should prompt a clinician to consider investigations to explore for a possible CVST.

Due to advancements in the field of neuroimaging, a condition that eluded diagnosis in a person who is alive, has now a real possibility of diagnosis and treatment leading to full recovery. Nowadays following a clinical suspicion, a CT brain, MRI brain, and MR venogram aid in diagnosis [3]. Although in 15-30% of cases a CT scan may be normal but MRI with MRV is more often than not, confirmatory [1]. The most common findings in a CT scan are infarcts which do not correlate with the specific arterial territory associated with other findings such as, cerebral oedema, tentorial enhancement, thrombosed cortical vein (popularly known as the Cord Sign) [1]. MRI with MRV usually reveals the absence of flow in the thrombosed sinuses [1]. In addition to MRV, more advanced imaging addendums such as 2D time-of-flight and the phase-contrast MRV can achieve higher sensitivity in diagnosing venous sinus thrombosis [1].

Treatment of CVST is with anticoagulants. Low Molecular Weight Heparin followed by oral anticoagulants remain the cornerstone of treatment [1], [2]. Usual protocols of anticoagulation should be ensured, i.e. oral anticoagulants should be overlapped with heparin till INR is 2-3 after which oral medication is continued for 3 to 6months (in case of no procoagulant state) [1]. Lifelong anticoagulation may be required in presence of a procoagulant condition [1]. Thrombolysis although can be effective but presents with the risk of a hemorrhagic transformation of the infarct [1].

Follow-up screening for thrombophilia should be performed 8-12 weeks after the acute insult to avoid false interpretation [1]. It is to be noted that the results will also be affected by anticoagulation, therefore; it is paramount to ensure that anticoagulants are withheld for at least 2 weeks prior to testing [1].

Prompt identification and early anticoagulation may be lifesaving in CVST.

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VI. LEARNING POINTS

- Persistent neurologic symptoms in a case of alcohol intoxication or binge drinking should prompt in thinking of cavernous sinus thrombosis.
- Thrombophilia test should be carried 8-12 weeks after the acute insult to avoid false interpretation.
- Anti-coagulants should be withheld for 2 weeks prior to thrombophilia testing to avoid any interaction of the anti-coagulants with the test results.

VII. CONCLUSION

It requires a strong clinical suspicion in alcoholics with persistent neurologic features to rule out cerebral venous sinus thrombosis as it often remains camouflaged beneath the initial presentation of alcohol intoxication.

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REFERENCES


Dr. Saquib Navid Siddiqui completed MBBS in 2012 in Dhaka, Bangladesh. During his MBBS journey he obtained highest marks in all 3 professional examinations from his medical college remaining within top 15% of the total marks. Since then he underwent training in various subspecialties of medicine in various tertiary level hospitals in Bangladesh. He completed MRCP (UK) in 2016 (One of the top scorer in MRCP paces exams from Bangladesh) and joined NHS in 2019 as trust grade specialist registrar in general medicine. He has recently been successful in attaining a specialty registrar training post in respiratory medicine to be commencing from March, 2021. He is the recipient of 2 prestigious awards in his current working trust (Covid 19 recognition award and Epic award). He has 2 other publications as the lead and corresponding author to his credit (‘A Case Of Massive Pulmonary Embolism in Covid-19 Pneumonitis’ published in IOSR journal on 19/06/2020 and ‘A Diagnostic Road To Damascus: A Case of Conversion to Pontine Infarct’ published in IOSR journal on 10/07/2020). He is an active contributor in providing education to the junior doctors of his trust by regularly participating in delivering various teaching sessions. He is also the initiator of a weekly teaching session in his trust in elderly care. He has completed formal course in teaching to improve his teaching skills. He has completed two qualitative improvement projects in his current trust which has had profound influence in improving quality of providing health care service in the respective area (Quality improvement project on ‘Evidence based clinical guidelines for the management of Covid-19 on the Oxford Ward High Dependency Unit’ in 2020 and Quality improvement project on ‘Prescription Of Therapeutic Oxygen As Drug’ in 2020). He has had 3 poster presentations in Society Of Acute Medicine conference held in Glasgow, 2020. He has participated and completed various clinical courses and skill development programmes since starting his career in NHS 2019. In addition, he is actively working as associate regional advisor for the Royal College of Physicians of Edinburgh since May 2020.

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