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Case Report

Cerebral Venous Sinus Thrombosis in a Young Male with Chronic Alcohol Use: A Possible Link to Hyperhomocysteinemia

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Abstract

Cerebral Venous Sinus Thrombosis (CVST) is a rare but significant cause of stroke, predominantly affecting young adults and often presenting with diverse and nonspecific clinical features that can delay diagnosis. We report a case of a 24-year-old male who presented with headache, seizures, and altered sensorium, initially raising consideration of postictal neurological deficits. Subsequent neuroimaging revealed extensive CVST with haemorrhagic venous infarction and mass effect, establishing a structural etiology. The patient had contributory risk factors including chronic alcohol use and hyperhomocysteinemia, highlighting the role of acquired prothrombotic states in individuals outside traditional risk groups. CVST commonly presents with features of raised intracranial pressure, focal neurological deficits, and seizures, and diagnosis relies on a high index of suspicion supported by appropriate neuroimaging, particularly venographic studies. In resource-limited settings, delayed access to advanced imaging and neurosurgical care can adversely affect outcomes. Management is cantered on prompt anticoagulation, even in the presence of haemorrhagic infarction, along with supportive measures and treatment of underlying risk factors. This case underscores the importance of considering CVST in young patients presenting with seizures and focal deficits, even in the absence of classical risk factors, and highlights the need for early recognition and timely intervention to reduce morbidity and improve outcomes.

Introduction

Cerebral Venous Sinus Thrombosis (CVST) is a rare cerebrovascular disorder caused by thrombotic occlusion of the intracranial dural venous sinuses [1]. Population-based studies estimate an incidence of 3–4 per million person-years, increasing to an estimate of 7 per million in young adults, with women affected nearly three times more often than men [1,2]. Peripartum and

postpartum incidence reaches 12 per 100,000 deliveries. This female predominance is attributed to sex-specific risk factors such as oral contraceptive use, pregnancy, puerperium, and hormone replacement therapy [1]. CVST disproportionately affects younger populations, with Asian cohorts showing a mean age of 28–31 years, and 70–80% of cases occurring in women of childbearing age [2]. In contrast, CVST in young males is less common and is often linked to prothrombotic states, alcohol use, or infection rather than hormonal causes [3]. The superior sagittal sinus is most frequently involved, followed by the transverse sinus, leading to impaired venous drainage, raised venous pressure, reduced cerebrospinal fluid absorption, intracranial hypertension, cerebral oedema, and venous infarction [1]. Notably, 30–50% of patients develop haemorrhagic infarcts [3].

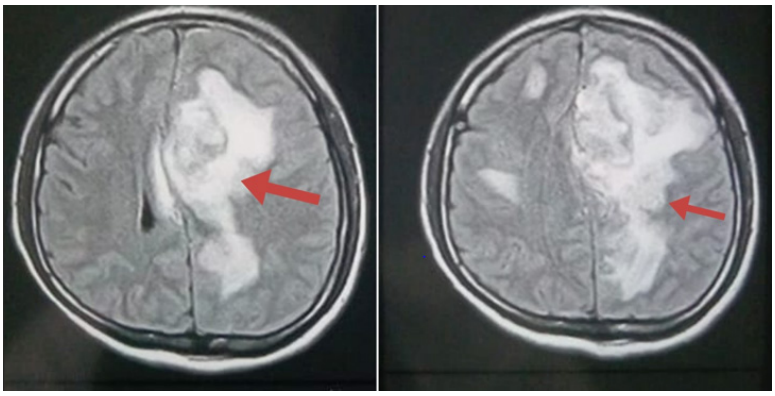
Risk factors include inherited thrombophilia (Factor V, Protein C or S deficiency) and acquired conditions such as malignancy and antiphospholipid syndrome [4], along with systemic contributors like dehydration, alcohol use, and severe anaemia [5]. Clinical presentation is most commonly subacute approximately 60%, as per ISCVT classification, and outcomes are generally favourable, with >80% achieving functional independence [5]. However, morbidity remains significant: up to 90% report persistent headache or fatigue, 44 % have focal deficits, and 9–10% develop seizures [6]. Elevated intracranial pressure is a critical complication, potentially causing visual loss or fatal herniation, with mortality rates of 5–15%, particularly within the first 4 days [6].

We report a case of CVST in a 24-year-old male presenting with headache, seizures, and intraparenchymal haemorrhage. This presentation is atypical given the patient's demographic profile, highlighting that CVST can occur outside traditional risk groups. In this case, chronic alcohol uses likely contributed to a prothrombotic state, illustrating an alternative pathogenic pathway distinct from the more common hormone-related mechanisms. This report emphasizes the need for a high index of suspicion for CVST even in patients without classical risk factors, particularly when clinical features are suggestive. It further underscores the importance of early recognition and timely management to mitigate morbidity and improve outcomes.

Case Description

A 24-year-old right-handed male presented to the emergency department with altered sensorium following a generalized tonic-clonic seizure and subsequent fall. On arrival, he was drowsy with rightward gaze deviation. The seizure episode had occurred one day prior to presentation. He had a history of seizure disorder since early childhood (since 5 years of age), which had was not evaluated and treated. He also had a history of chronic alcohol use for the past 7 years. At presentation, he complained of dizziness, persistent altered sensorium, and diffuse headache. There were no visible external injuries.

On examination, his vital parameters were stable, with a pulse rate of 92 beats per minute and blood pressure of 110/50 mmHg. He was afebrile, and airway, breathing, and circulation were normal. Neurological assessment revealed a Glasgow Coma Scale score of E4V1M5, with reduced verbal response. Prior to presentation at our center, he had been treated at an outside facility with osmotherapy (glycerol), levetiracetam, proton pump inhibitors, antiemetics, intravenous fluids, and was referred to our hospital as symptoms remained unresolved.



Figures 1(a): Axial Fluid-Attenuated Inversion Recovery (FLAIR) magnetic resonance image of the brain demonstrating a large hyperintense intra-parenchymal lesion in the frontoparietal region (arrow), with surrounding vasogenic edema and associated mass effect. Figure 1(b) Axial MRI Brain (FLAIR Sequence) at the level of the Centrum Semiovale demonstrates a large, heterogeously hyperintense intra-parenchymal lesion involving the frontoparietal region (arrow), with surrounding vasogenic edema and associated mass effect, including effacement of adjacent cortical sulci.

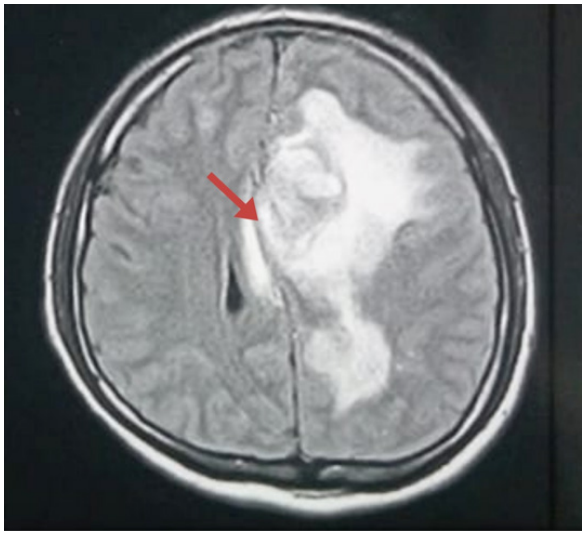


Figure 2: Axial Fluid-Attenuated Inversion Recovery (FLAIR) magnetic resonance image demonstrates marked mass effect with effacement of adjacent cortical sulci and a midline shift of approximately 12mm, as indicated by displacement of the falx cerebri and adjacent midline structures (arrow).

Initial neuroimaging using Non-Contrast Computerized Tomography (NCCT) demonstrated an intraparenchymal haemorrhage with a significant midline shift of approximately 1.2 cm (12 mm). Further evaluation with magnetic resonance imaging (MRI) of the brain and magnetic resonance venography (MRV) revealed multiple diffusion-restricting T2/FLAIR hyperintense lesions in the bilateral fronto-parietal regions, more prominent on the left, with susceptibility blooming consistent with haemorrhagic components and midline shift was confirmed to be 12mm (Figure 1, 2). Multiple intraparenchymal hematomas were identified, the largest in the left fronto-parietal region, contributing to mass effect. MRV demonstrated extensive cerebral venous sinus thrombosis involving the superior sagittal sinus, bilateral transverse sinuses, and right sigmoid sinus, with extension into the right internal jugular vein. Magnetic Resonance Angiography (MRA) showed hypoplasia of the left vertebral artery, while the remaining intracranial arterial system was unremarkable.

Baseline haematological parameters, including haemoglobin, total leukocyte count, and platelet count, were within normal limits. Coagulation profile showed a prothrombin time of 14 seconds, INR of 1.24, and activated partial thromboplastin time of 39 seconds. Renal function and serum electrolytes were within normal limits. Liver function tests revealed mild elevation of aspartate aminotransferase. Inflammatory markers, including C-reactive protein and erythrocyte sedimentation rate, were elevated. Notably, hyperhomocysteinemia was detected, suggesting a potential prothrombotic predisposition contributing to the development of cerebral venous sinus thrombosis.

Given the diagnosis of extensive cerebral venous sinus thrombosis with associated haemorrhagic venous infarction and significant mass effect, the condition was considered a neurosurgical emergency. However, due to lack of immediate neurosurgical availability, the patient was managed conservatively. Anticoagulation therapy with heparin was initiated along with antiepileptic therapy, osmotherapy, and supportive care. The patient showed progressive clinical improvement. Serial neuroimaging demonstrated reduction in cerebral edema and hematoma size, with a corresponding decrease in midline shift from 12 mm to 7 mm. At discharge, the patient was neurologically stable with no new deficits and was given counselling along with referral to alcohol rehabilitation therapy.

Discussion

Cerebral Venous Sinus Thrombosis (CVST) is a rare cerebrovascular disorder accounting for 0.5 to 3 percent of all strokes and predominantly affecting young adults and women of reproductive age [2]. The annual incidence is estimated at 3 to 4 per million, although underdiagnosis is likely due to heterogeneous clinical presentation [7]. In India, CVST contributes to 10 to 20 percent of strokes in young adults. Clinically, it presents with features of raised intracranial pressure, venous infarction, and seizures, with headache being most common, followed by focal neurological deficits, visual symptoms, cranial nerve palsies, and altered consciousness [8,9]. Seizures may be the initial manifestation and can confound diagnosis due to postictal deficits such as Todd's paresis [10]. In this case, a 24-year-old male with untreated seizure disorder and chronic alcohol use presented with a generalized tonic-clonic seizure followed by focal deficits. Although Todd paresis was initially considered, neuroimaging revealed extensive CVST with haemorrhagic venous infarction and midline shift, confirming an underlying structural etiology.

Hyperhomocysteinemia likely contributed significantly, conferring a fourfold increased risk of CVST through endothelial dysfunction, oxidative stress, and impaired fibrinolysis [11,12,13]. This risk is further amplified by vitamin B12 and folate deficiency and chronic alcohol use [14]. Indian registry data identify alcohol as an independent risk factor and suggest a rising burden among young males, aligning with this patient profile [15,16]. Notably, hyperhomocysteinemia is reported in over 50 percent of CVST cases in Northern India [17], which makes this parameter a predictor for hypercoagulable state.

Diagnosis relies on neuroimaging, where NCCT may demonstrate indirect features such as haemorrhagic infarction or edema, while MRV is required for definitive confirmation by demonstrating venous occlusion [9]. However, limited access to advanced imaging and neurosurgical care in resource constrained settings continues to delay diagnosis and worsen outcomes, compounded by broader disparities in neurological healthcare access in India [18]. The differential diagnosis in this case included hypertensive intracerebral haemorrhage, arterial ischemic stroke, herpes simplex encephalitis, and posterior reversible encephalopathy syndrome, which were excluded based on clinical profile, lesion distribution, and imaging findings [19]. Complications of CVST include haemorrhagic infarction, progressive edema, seizures, and life-threatening mass effect [9]. Management is centered on prompt anticoagulation, which remains indicated even in the presence of haemorrhagic infarction, with escalation to decompressive surgery or endovascular therapy in severe or refractory cases [9,18]. Although prognosis has improved with early recognition, severe presentations still carry significant mortality, emphasizing the importance of addressing reversible risk factors such as alcohol use and nutritional deficiencies, along with close follow up.

Conclusion

This case underscores the diagnostic challenge of distinguishing postictal deficits from structural pathology, highlights the importance of early neuroimaging in new focal deficits following seizures, and illustrates the multifactorial risk profile of CVST in young adults, particularly hyperhomocysteinemia and chronic alcohol use, with timely intervention being critical to improving outcomes.

Conflict of Interest

The authors declare no conflict of interest and no funding was received for the study.

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