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Case Report

Cerebral venous sinus thrombosis with hemorrhagic infarct: A rare presentation in a risk-defying male patient ☆

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ABSTRACT

Cerebral venous sinus thrombosis (CVST) is a rare but critical cerebrovascular condition characterized by clot formation in cerebral veins or sinuses. We present a case of a 30-year-old male with CVST, an atypical presentation of right-sided weakness and sudden loss of consciousness. While CVST typically manifests as severe headaches and neurological deficits, our patient's unique symptoms pose diagnostic challenges. Advanced imaging techniques, including MRI with venography, played a pivotal role in confirming the diagnosis. Treatment involved anticoagulation therapy and resulted in a favorable outcome. This case highlights the importance of considering CVST in patients with unusual neurological symptoms and the crucial role of early diagnosis and intervention. Advances in diagnostic modalities and treatment options have significantly improved outcomes in CVST patients, emphasizing the need for timely recognition and management.

Background

Cerebral venous sinus thrombosis (CVST) is a rare yet serious cerebrovascular disease where clots form in cerebral veins or sinuses. It occurs in 1%-4% of cerebral venous thrombosis cases, comprising 0.5% of all strokes. Unlike arterial strokes, it affects brain-draining veins, causing varied clinical symptoms like headache, seizures, altered mental status, and focal neurological deficits. [1–3]

Previously, diagnosing CVST was challenging, but advancements in magnetic resonance imaging (MRI) and venography (MRV) have made it more reliably detectable. Unlike our case, this condition is more common in females and often linked

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to factors like surgery, trauma, sepsis, cancer, thrombophilia, antiphospholipid syndrome, pregnancy, and the postpartum period responsible for creating an imbalance between coagulation and anti-coagulation system [4].

Increased pressure in tiny blood vessels due to the obstruction can cause bleeding, which happens more frequently in CVST compared to other types of strokes. Fortunately, prompt treatment with heparin, the primary therapy, halts clot formation and restores normal blood flow through the body's natural anticoagulation mechanisms. With modern treatments, most patients with CVST have favorable outcomes, with low mortality rates (8%-14%) and a small percentage (8.7%) experiencing lasting neurological deficits [5].

Case details

A 30-year-old male, standing at a height of 160 cm and weighing 60 kg, presented to the emergency department with a sudden onset of right-sided weakness. He had been experiencing severe headaches for the past 5 days, accompanied by profound nausea and vomiting. The patient had no history of prior medication usage or similar episodes and denied any underlying chronic medical conditions, such as diabetes, hypertension, or malignancy. There was no reported family history of venous thrombosis or stroke.

Upon initial examination, the patient had a normal body temperature, with a blood pressure reading of 130/80 mm Hg and a heart rate of 64 beats per minute. No abnormalities were detected during the assessment of his respiratory and cardiovascular systems. However, the neurological examination revealed a reduction in muscle strength in the right upper and lower extremities. Additionally, the patient reported paresthetic sensations on the right side of his body and had difficulty comprehending language.

Laboratory findings at admission showed normal results for metabolic profiles, including liver and renal function tests. Homocysteine levels, platelet count, bleeding time, and prothrombin counts were within the normal range. However, the patient exhibited an elevated D-Dimer level of 1200 ng/mL (normal range: 200-500 ng/mL). The biochemical panel indicated an elevated leukocyte count of 12,000 (normal range: 4000-11,000) and a hemoglobin level of 12.6 g/dL. A slightly elevated erythrocyte sedimentation rate (ESR) of 30 mm/h (normal value: <15 mm/h) was noted. A noncontrast CT scan of the head revealed a hypodense lesion in the left parietal lobe with areas of hemorrhage within it, causing a mass effect by effacing adjacent sulci. Hyperdense content was also observed in the superior sagittal sinus and cortical veins in the left parietal lobe region. Venous hemorrhagic infarction was suspected, leading to the recommendation for magnetic resonance imaging (MRI) with venography (MRV). The MRV confirmed thrombosis in the superior sagittal sinus, left vein of Trolard, and parietal cortical veins, along with hemorrhagic infarction in the left parietal lobe (Figs. 1A–D and Figs. 2A–C). Notably, no features of vascular malformation were detected.

The patient was started on low molecular weight heparin, adjusted based on his weight, for a 10-day duration, followed by oral vitamin antagonists. He reported improvement in his clinical symptoms and regained motor function. Subsequently, he was discharged with oral anticoagulants, and although he still exhibited minimal residual symptoms, he was advised to undergo physiotherapy. The patient returned for a follow-up appointment after 2 weeks, reporting continued resolution of his symptoms and a good recovery. His protein C and protein S levels were within the normal range during follow-up. Although a follow-up MRI after three months was recommended, the patient declined due to economic reasons, as he was feeling well.

Discussion

In this case, sudden loss of consciousness and one-sided weakness occurred. CVST's typical clinical presentation involves acute to subacute onset of severe headache, vision impairments, seizures, altered consciousness, and varying symptoms. Acute secondary headache with change in intensity, course, and neurological deficits is the most common manifestation and usually has a good prognosis when the patient presents exclusively with this symptom [4].

Neurological deficits like weakness, cranial nerve palsy, cortical blindness, aphasia, neuro-ophthalmological symptoms motor and sensory changes pose diagnostic challenges, particularly in young patients. Motor deficits are frequently seen in patients with cerebral venous thrombosis (CVT), occurring in 19.1%-39% of cases. These deficits are more prevalent when CVT involves specific venous structures like the superior sagittal sinus, cortical veins, and cerebral deep venous system. Early diagnosis and intervention are crucial to mitigate morbidity and mortality risks [6]. We present a young patient with motor weakness, sensory involvement, and difficulty in comprehension.

CVST's pathophysiology includes hemostasis, vessel wall changes, and blood composition alterations. Infections or noninfectious factors in the head and neck area can trigger it, especially with thrombophilic conditions. Clots impede blood flow, raise venous pressure, hinder drainage, and reduce brain cell oxygen. Severe cases may lead to venous hemorrhagic infarction due to inadequate drainage [1,7].

The primary risk factors for cerebral vein and dural sinus thrombosis (CVT) encompass hormonal changes induced by factors like oral contraceptives, pregnancy, steroids, or hypothyroidism. Additionally, abnormalities of the skull, skull trauma, mastoid sinus infections, systemic illnesses such as dehydration, sepsis, and connective tissue disorders, as well as conditions like cancer, iron deficiency anemia, and folic acid deficiency, play significant roles in increasing the susceptibility to CVT [8,9]. However, In our study patient had none of those risk factors.

Imaging, notably CT or MRI with venography, is pivotal for confirming CVST diagnosis and guiding management. Unenhanced MR is more sensitive for the demonstration of the venous thrombus and the occluded dural sinus or vein when compared to unenhanced CT [4]. It offers detailed cerebral vein visualization, ruling out alternate causes like bleeding or tumors. Conveying imaging's importance to requesting physicians is vital, facilitating early intervention and improved out-

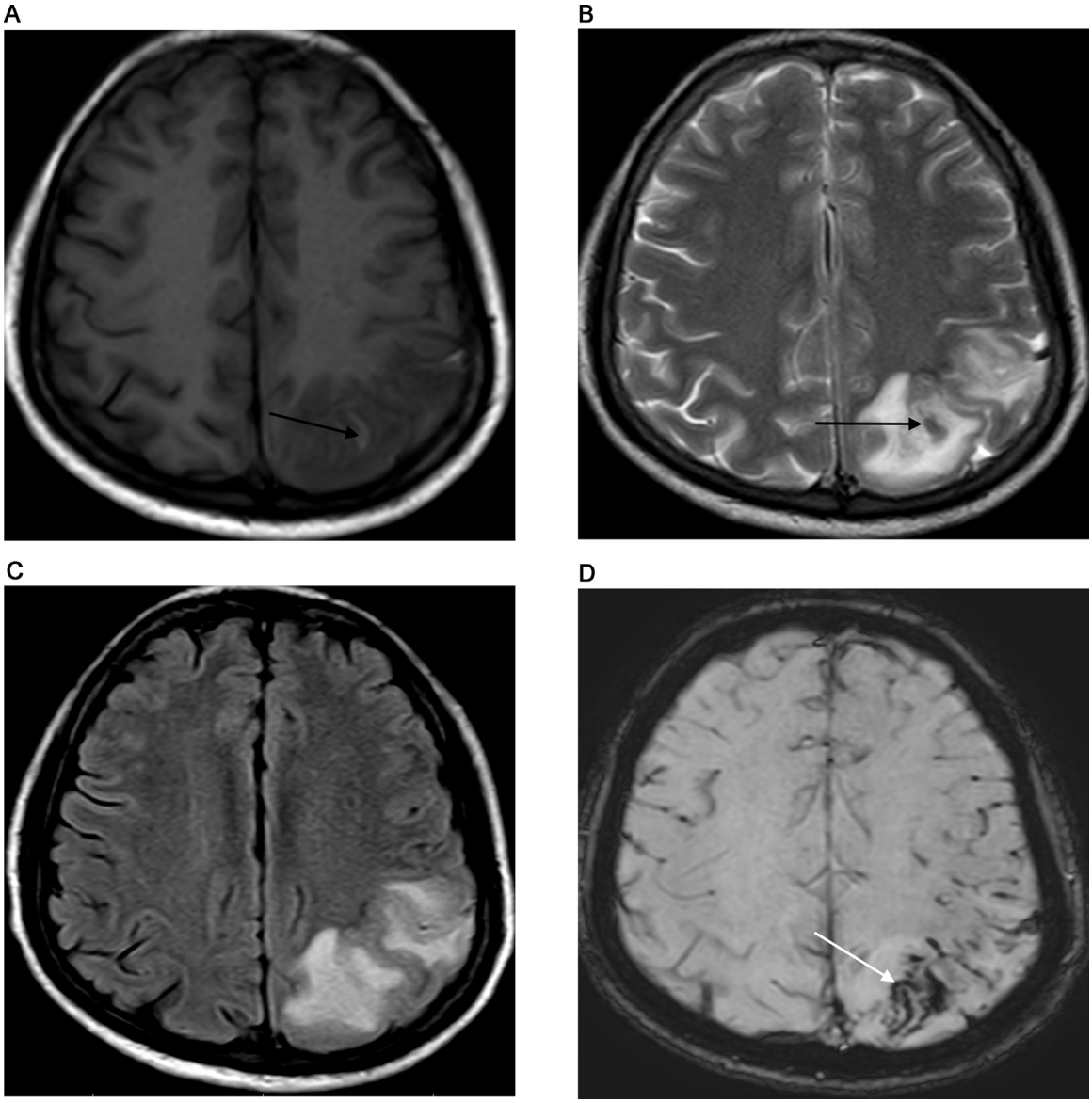

Fig. 1 – (A) T1 weighted axial MRI image at the level of centrum semiovale showing the ill-defined area of T1 low signal intensity in the left parietal lobe with a patchy area of T1 high signal intensity within (black arrow). (B) T2 weighted axial MRI image of the level of centrum semiovale showing a relatively well-defined area of high signal intensity with a patchy area of low signal intensity within (black arrow) in the left parietal lobe involving both gray and white matter. (C) FLAIR weighted axial MRI image of the level of centrum semiovale showing a relatively well-defined area of high signal intensity in the left parietal lobe involving both gray and white matter. (D) SWI weighted axial MRI image of the level of centrum semiovale showing low signal intensity area of blooming in the left parietal lobe (white arrow).

comes through timely detection [10,6]. In our study, MRV was very crucial for confirming venous sinus thrombosis.

Lumbar puncture results commonly reveal increased opening pressure, along with cerebrospinal fluid (CSF) irregularities, such as elevated cell counts and elevated protein levels. In cases where there is a strong clinical suspicion of CVST, it's important to note that a normal D-dimer level should not discourage further diagnostic evaluation. Although fibrin split products, often measured by D-dimer levels, can be supportive in diagnosing CVST, they should not be the sole determinant in deciding whether to pursue additional testing [4,6]. In our study, the patient exhibited elevated levels of D-Dimer.

CVST imaging's diagnostic pearls involve detecting thrombi/clots in cerebral veins/sinuses as filling defects on contrast-enhanced images. Notable findings include vein distension, collateral vessels, empty delta sign in the superior sagittal sinus, and hemorrhagic infarction areas [11]. CT venography and MRI with venography have high sensitivity and specificity for CVST detection. MRI's "cord sign," a hyperintense signal resembling a cord in the sinus, is distinctive.

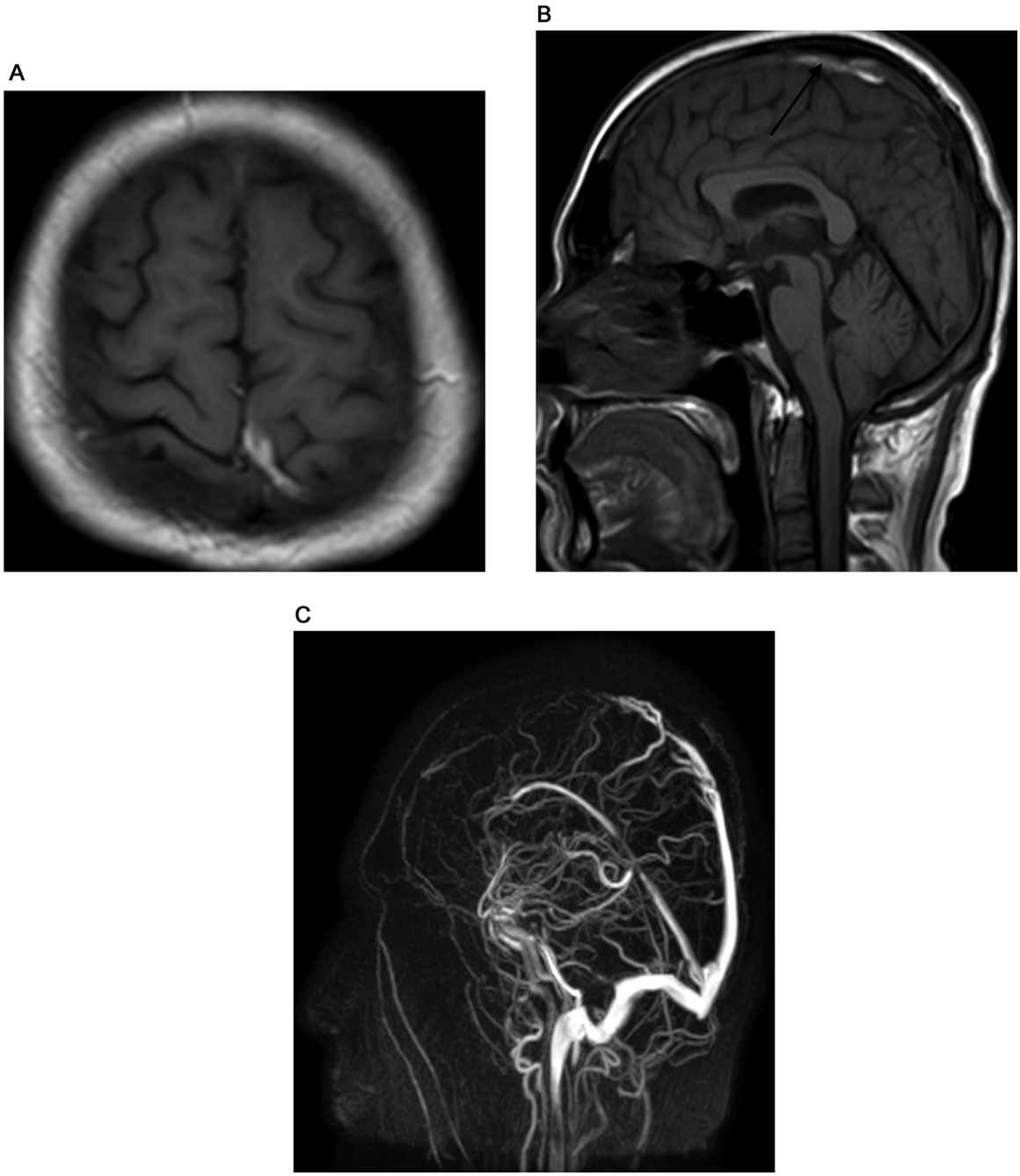

Fig. 2 – (A) Axial T1 weighted MRI image showing high signal intensity in the left vein of Trolard in the left high parietal lobe. (B) Midsagittal T1 weighted MRI image showing high signal intensity in superior sagittal sinus in the region of parietal convexity with loss of flow void (black arrow). (C) Sagittal 3D reconstruction of MRV image showing nonvisualization of left vein of Trolard and anterior aspect of the superior sagittal sinus with an irregular outline in the parietal region (black arrow).

CVST diagnosis combines imaging, clinical presentation, and lab results. Thrombi in cerebral venous structures, correlated with symptoms and risks, confirm the diagnosis [12].

Therapeutic strategies for CVST involve anticoagulation, preferably using low molecular weight heparin (LMWH) or unfractionated heparin (UFH), to impede clot progression. Severe cases with venous hemorrhagic infarction demand aggressive management, addressing intracranial pressure, and providing support care. Decompressive craniectomy is helpful for critically ill CVT patients displaying brainstem dysfunction, refractory intracranial hypertension, and mass effect on radiological and clinical assessment. Prompt imaging-based diagnosis shapes treatment approaches, impacting outcomes [9,5].

Unlike in arterial stroke mortality rate has significantly decreased due to recent advances in diagnostic modalities, and a shift in risk factors and treatments in patients with CVST. The prognosis depends on clot extent, hemorrhagic infarction, and causes. Early intervention heightens recovery

chances. Imaging guides treatment, reducing complications and mortality risk [6].

Previous research has employed multivariate approaches to examine the long-term prognostic factors associated with CVT. The International Study on CVT identified several significant factors contributing to unfavorable outcomes, including coma, cerebral hemorrhage, and malignancy. Furthermore, male gender, age exceeding 37 years, mental health disorders, thrombosis affecting the deep cerebral venous system, and central nervous system infections were recognized as risk factors associated with adverse consequences. Seizures (10%) and the occurrence of new thrombotic events (4%) were common complications observed during the follow-up period. While CVT recurrence and severe visual impairment were infrequent, they represented severe and potentially avoidable outcomes [8]. It is noteworthy that, despite being male, our patient did not exhibit any of these additional risk factors, with a positive recovery following treatment.

Conclusion

In this case of CVST, atypical clinical presentation underscored the importance of a high index of suspicion and advanced imaging for accurate diagnosis. Timely intervention with anticoagulation therapy yielded a favorable outcome, highlighting the critical role of early recognition and treatment in CVST management. Recent advancements in diagnostic techniques and therapeutic strategies have improved prognosis in CVST patients. This case reinforces the need for clinicians to consider CVST in cases of unexplained neurological symptoms, ensuring that appropriate measures are taken promptly to mitigate morbidity and mortality associated with this rare cerebrovascular condition.

Author contributions

Shailendra Katwal: Conceptualization, as mentor and reviewer for this case report and for data interpretation. Sundar Suwal: Contributed in performing literature review and editing. Suman Lamichhane: Contributed in writing the paper and reviewer for this case. Amrit Bhusal: Contributed in writing the paper. Tek Nath Yogi: Contributed in writing the paper. All authors have read and approved the manuscript.

Ethical approval

This case report did not require review by the ethical committee.

Registration of research studies

Not applicable

Provenance and peer review

Not commissioned, externally peer reviewed

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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