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Case Report

Dural Venous Sinus Thrombosis in a Young Patient with Atypical Symptoms

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ABSTRACT

Introduction: Dural venous sinus thrombosis (DVST) is a rare but potentially life-threatening condition characterized by thrombus formation in the dural venous sinuses. Typically presenting with headache, seizures, or focal neurological deficits, DVST can be challenging to diagnose when symptoms are atypical.

Case report: This case report describes a 24-year-old male patient presenting with atypical symptoms of persistent fatigue, multiple episodes of vomiting, intermittent visual disturbances and mild cognitive impairment, ultimately diagnosed with DVST. The report discusses the diagnostic approach, management, and outcomes, emphasizing the importance of considering DVST in young patients with non-specific neurological symptoms. The case highlights the role of advanced imaging and multidisciplinary care in achieving a favourable outcome.

Conclusion: Clinicians should maintain a low threshold for suspecting DVST in young patients with neurological symptoms and prothrombotic risk factors, as early diagnosis and treatment are critical for preventing complications.

KEYWORDS

• DVST • Headache • Focal seizures • Multi-disciplinary care • Prothrombotic factors

Key Message:

- DVST is often underdiagnosed due to its variable presentation.

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- This case underscores the importance of maintaining a high index of suspicion for DVST in young patients with risk factors, even when symptoms are atypical.

INTRODUCTION

Dural venous sinus thrombosis is an uncommon cerebrovascular disorder with an estimated incidence of 1.3–1.6 per 100,000 individuals annually.[1] It predominantly affects younger individuals, with risk factors including hypercoagulable states, pregnancy, oral contraceptive use, and infections. Classic symptoms include headache (90% of cases), seizures, and focal neurological deficits, but atypical presentations can delay diagnosis, increasing the risk of complications such as cerebral oedema or haemorrhage.[1]

Case Presentation

A 28-year-old male presented to the emergency department with a 3-week history of persistent fatigue, multiple episodes of vomiting,intermittent visual disturbances described as "flashing lights" in the peripheral vision and mild cognitive impairment including difficulty concentrating and short-term memory lapses.

His medical history was unremarkable and he had no history of smoking, alcohol use, or illicit drug use. Family history was negative for thrombotic disorders.

Clinical Findings: On examination, the patient was alert and oriented.

Vital signs were within normal limits with a blood pressure 118/76 mmHg, heart rate 72 beats per minute and a temperature of 98.06°F.

Neurological examination revealed no focal deficits, normal cranial nerve function, and intact motor and sensory systems.

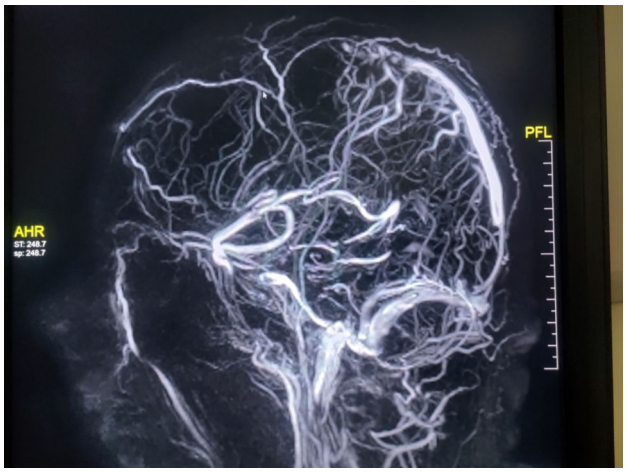
Fundoscopic examination showed no papilledema.

Visual field testing was normal, and no nystagmus was observed.

Diagnostic Assessment: Initial differential diagnoses included migraine with aura and transient ischemic attack.

Routine blood tests, including complete blood count, electrolytes, and thyroid function, were normal.

A brain magnetic resonance imaging (MRI) scan with venography (MRV) was performed, revealing a filling defect in the superior sagittal sinus thrombosis. T2-weighted MRI showed no evidence of cerebral oedema or infarction.



A thrombophilia workup was initiated, including tests for protein C, protein S, antithrombin III, factor V Leiden, and antiphospholipid antibodies, all of which were negative.

Management: The patient was admitted to the neurology ward and started on therapeutic anticoagulation with low-molecular-weight heparin (LMWH; enoxaparin 1 mg/kg subcutaneously twice daily).

After 48 hours, he was transitioned to a direct oral anticoagulant (DOAC; apixaban 10 mg twice daily for 7 days, followed by 5 mg twice daily).

The patient was counselled on the importance of medication adherence and the need for follow-up imaging.

Supportive care included cognitive rehabilitation exercises to address mild cognitive deficits.

Follow-Up and Outcomes: At the 1-month follow-up, the patient reported significant improvement in fatigue and cognitive function. Visual disturbances had resolved completely.

Apixaban was continued, with a plan for reassessment at 6 months to determine the duration of anticoagulation.

Due to financial constraints, patient refused for follow up scans in view of improving symptoms.

At the 6-month follow-up, the patient remained symptom-free. Apixaban was discontinued, and the patient was prescribed low-dose aspirin for secondary prophylaxis.

DISCUSSION

DVST is often underdiagnosed due to its variable presentation. In this case, the absence of classic symptoms such as headache or seizures posed a diagnostic challenge. The patient's symptoms fatigue, visual disturbances, and cognitive impairment are non-specific and overlap with conditions like migraine, transient ischemic attack or chronic fatigue syndrome. This case underscores the importance of maintaining a high index of suspicion for DVST in young patients, even when symptoms are atypical.

DVST results from an imbalance in the haemostatic system, leading to thrombus formation in the dural sinuses.

Common risk factors include inherited thrombophilia (e.g., prothrombin G20210A mutation, factor V Leiden), acquired conditions (e.g., pregnancy, COC use), and local factors (e.g., head trauma, infections).[4]

The pathophysiology of DVST involves obstruction of venous drainage, leading to increased intracranial pressure and, in severe cases, cerebral oedema or haemorrhage. Atypical symptoms, as seen in this case, may reflect early or partial sinus occlusion, where compensatory venous collaterals mitigate severe neurological deficits.[5]

MRV is the gold standard for diagnosing DVST, with a sensitivity and specificity exceeding 90%. In this case, MRV confirmed the diagnosis by demonstrating a filling defect in the superior sagittal sinuses.[6] Computed tomography (CT) venography is an alternative when MRV is unavailable, but it carries the risk of radiation exposure, particularly in younger patients.[7]

Management and Anticoagulation: Anticoagulation is the cornerstone of DVST management, aiming to prevent thrombus propagation and promote recanalization. Guidelines from the American Heart Association/American Stroke Association recommend LMWH or unfractionated heparin initially, followed by oral anticoagulants for 3–6 months in provoked DVST or longer in unprovoked cases or those with persistent risk factors.[8]

Prognosis: The prognosis of DVST is generally favourable with timely diagnosis and treatment, with mortality rates below 5%. Recanalization occurs in 80–90% of patients within 6–12 months. However, recurrence risk is significant (2–3% per year), particularly in patients with thrombophilia.[8]

CONCLUSION

This case report illustrates the diagnostic and therapeutic challenges of DVST in a young patient with atypical symptoms. Persistent fatigue, visual disturbances, and mild cognitive impairment, while non-specific, warranted thorough investigation, leading to the I dentification of DVST through MRV. Prompt anticoagulation and risk factor modification resulted in an excellent outcome. Clinicians should maintain a low threshold for suspecting DVST in young patients with neurological symptoms and prothrombotic risk factors, as early diagnosis and treatment are critical for preventing complications.

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