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Delayed Presentation of Acute Extensive Cerebral Venous Sinus Thrombosis Following Non-Penetrating, Closed Head Injury: A Case Report

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Abstract

Cerebral Venous Sinus Thrombosis (CVST) is a potentially life-threatening condition associated with high mortality and morbidity. We present a unique case of a young man who presented with acute worsening headache ten days after a non-penetrating head injury, and subsequent neuroimaging revealed extensive CVST without underlying skull fractures. The patient was successfully managed with Low Molecular Weight Heparin (LMWH) followed by a three-month course of Warfarin. This case highlights the importance of repeated neuroimaging in patients with post-traumatic headache, regardless of bony injury. Early identification and appropriate treatment are crucial in preventing fatal complications and neurological impairment. To the best of our knowledge, this is the first reported case of delayed presentation of extensive CVST without skull fractures or significant neurological dysfunction.

Introduction

Thrombosis of the cerebral dural venous sinuses is a rare condition that can occur in both traumatic and non-traumatic situations. It can lead to various complications, such as elevated cerebrospinal fluid pressure, venous infarcts, and hemorrhages, resulting in chronic neurological deficits or even mortality. The mortality rate associated with cerebral venous sinus thrombosis (CVST) ranges from 5.5% to 30%, with approximately 15.5% to 25.5% of survivors experiencing residual neurological deficits [1,2]. Certain factors like age over 37 years, male gender, intracerebral hemorrhage, involvement of deep cerebral veins, low Glasgow Coma Scale (GCS) score, concomitant malignancy, and CNS infection are associated with a poor prognosis in non-traumatic CVST cases [1].

Clinical suspicion plays a crucial role in diagnosing CVST, and early initiation of anticoagulant therapy is vital to prevent serious complications. We present a case of a young adult male who presented with a headache, without focal neurology, altered sensorium, seizures, or evidence of intracranial hypertension, 10 days after a non-penetrating closed head injury. Subsequent diagnosis revealed extensive CVST. This case underscores the importance of not overlooking post-traumatic headaches, no matter how trivial they may appear, and highlights the need for timely evaluation and appropriate management.

Case Presentation

A 29-year-old Sri Lankan man presented to our facility with an acute worsening headache persisting for 12 hours. Ten days earlier, he had a history of sustaining a severe blow to the head with an iron rod resulting in a transient loss of consciousness. Initial evaluation on the day of the injury, including a non-contrast CT brain scan, was unremarkable. The patient was discharged with analgesics but returned after eight days with

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worsening vertex and occipital headache, which was unresponsive to medication. The headache did not vary with changes in posture, activity, or time of day. It was not affected by movements, such as stooping or valsalva maneuver, and it did not worsen in the early morning. The patient did not experience neck stiffness, photophobia, or phonophobia. Further imaging revealed a small (5.5 mm) subdural hematoma over the right occipital area appearing to extend towards the falx medially. He was sent home with reassurance. He readmits 2 days later with persistent headache unresponsive to analgesics. At this presentation, his Glasgow Coma Scale (GCS) was 15/15, and body temperature, haemodynamic and respiratory parameters were also normal. He had no signs of focal neurology or meningism. A new NCCT brain revealed a para-falcian hyper-dense strip at vertex (Figure *1*). A CT venogram was requested due to a high degree of suspicion of superior sagittal sinus thrombosis. It revealed large filling defects suggestive for extensive Cerebral Venous Sinus Thrombosis (CVST) involving the superior sagittal sinus, torcula, bilateral transverse sinuses and proximal portions of bilateral sigmoid sinus (Figure *2*). Deep cerebral veins were prominent. 3-dimensional reconstruction of the skull did not reveal any fractures in the vault or base. Treatment was initiated with Low Molecular Weight Heparin (LMWH) as bridging therapy for Warfarin. The patient remained on Warfarin for three months, targeting an International Normalized Ratio (INR) of 2 to 3. He experienced gradual symptomatic improvement without neurological deterioration during subsequent follow-up.

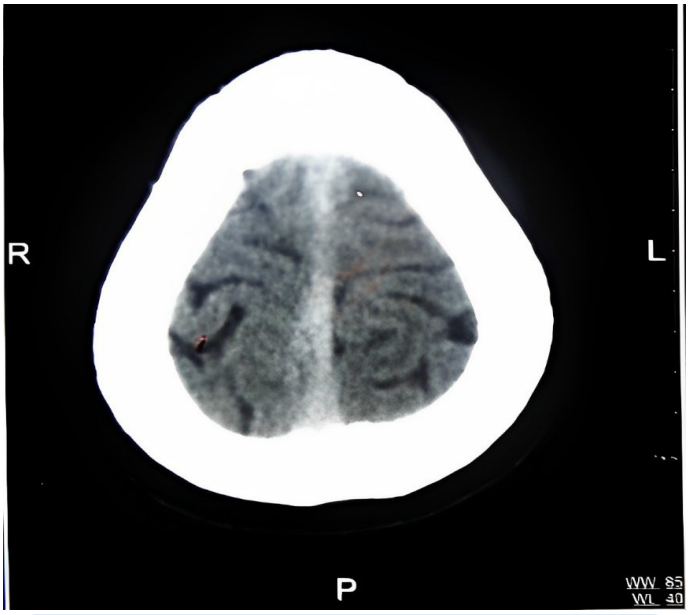

Figure 1: Axial, noncontrast computed tomograph of head, taken ten days after head injury, showing hyperdensity along superior sagittal sinus.

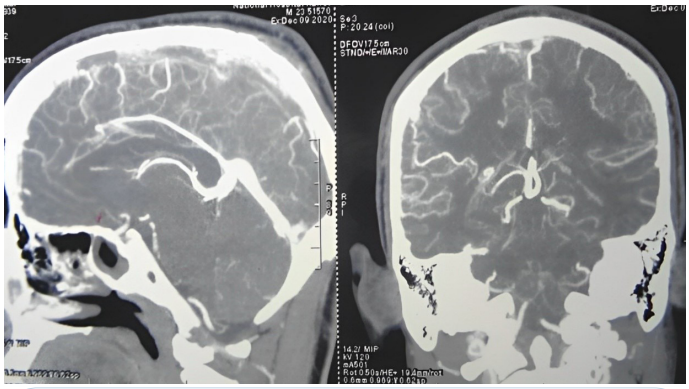

Figure 2: CT venogram of cerebral veins, sagittal view (left panel), and coronal view (right panel), depicting extensive filling defects in the superior sagittal sinus.

Discussion

CVST itself does not present with specific clinical symptoms, and it is often overlooked as a cause of post-traumatic headache unless it is suspected. In this reported case, the patient presented with chronic progressive headache following a recent non-penetrating head injury. Without fractures or significant hemorrhages observed in the initial non-contrast CT scan, it could have been easily misdiagnosed as post-concussion headache or early-stage chronic post-traumatic headache [3]. A higher level of suspicion for CVST would have been warranted if there were conventional risk factors such as hypercoagulable states, pregnancy, oral contraceptive pill usage, or if the headache was associated with reduced Glasgow Coma Scale (GCS), seizures, papilledema, or focal neurology. In the presented case, delayed onset of symptoms, lack of response to initial analgesics and initial negative imaging findings highlight the importance of considering CVST as a potential complication, even in the absence of immediate neurological deficits or underlying fractures. The prompt diagnosis with suitable neuroimaging techniques and subsequent initiation of anticoagulation treatment were possible because of high level of suspicion of the condition, even before neurological deterioration occurred.

Non-contrast CT brain imaging is a useful and readily available preliminary tool for evaluating CVST, as shown in this case. Radiological findings such as the dense delta sign or hyper-dense cord sign in non-contrast CT brain can suggest thrombosed dural sinuses or cortical veins. Cerebral venography is necessary for definitive diagnosis, with Magnetic Resonance Venography (MRV) being the most suitable modality, as it can detect non-filling thrombosed veins. However, MRV may be non-diagnostic in acute CVST and limited by availability and time [4]. CT venography is equally sensitive in detecting thrombosis through the visualization of filling defects [4]. A retrospective study recommended the use of post-traumatic cerebral venography only when fractures are present overlying the dural sinuses or jugular bulb [5]. Contradicting this, the reported case highlights the necessity of suspecting post-traumatic CVST even in the absence of skull fractures.

Post-traumatic CVST is a rarely described entity in the literature. Direct endothelial injury may account for CVST in penetrating head injuries. Cases of post-traumatic CVST without underlying fractures have not been previously reported in the adult population, although a few cases have been documented in pediatric patients [6,7]. Depressed skull fractures and tangential mechanical force applied to the superior sagittal sinus or its bridging veins may contribute to CVST in non-penetrating head injuries. The mere shear stress from the blow, even without dural wall rupture, the extension of a thrombus from an injured emissary vein, or external compression of the dural sinus from a depressed fracture or expanding hematoma, could all be potential pathogenic factors for CVST in closed head injuries. Post-traumatic coagulopathy and dehydration may also play contributory roles. Preexisting coagulopathy, whether inherited or acquired, further increases the risk of post-traumatic CVST.

Conclusions

This case underscores the need for vigilance and a high index of suspicion in patients with post-traumatic headaches. Healthcare professionals should be vigilant of the possibility of post-

traumatic CVST, even in the absence of classical risk factors or skull fractures. Timely diagnosis and treatment are imperative to ensure the best possible patient outcomes. Further studies are warranted to better understand the pathophysiology, risk factors, and optimal management strategies for traumatic CVST.

Ethics Approval and consent to participate

It was not applicable, as this is a case report. However, written and signed informed consent from patient's legal guardian was obtained for publishing the case report.

Consent for Publication

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

Availability of data and materials

The details of this patient's reports and images are with corresponding author.

Competing interests: The author does not have any competing interests.

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Author's contributions: The corresponding author was involved in managing the patients at the emergency department, follow up of the patient and preparation of the entire manuscript.

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