



Post-Traumatic Cerebral Sinus Thrombosis: A Diagnostic and Therapeutic Challenge in the Emergency Department: Case Report and Literature Review

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Abstract: Post-traumatic cerebral sinus thrombosis (ptCST) is a rare but potentially life-threatening complication of head trauma, accounting for approximately 1% of all cerebral venous thrombosis cases and occurring in around 4% of patients sustaining traumatic brain injury [1,8]. The risk is significantly higher when a skull fracture extends to a dural sinus. We report the case of a 62-year-old male who presented following isolated blunt head trauma and was initially found to have a subarachnoid hemorrhage, a left frontal contusion, and a non-displaced occipital fracture extending to the right sigmoid sinus. Despite initial neurological stability, clinical deterioration occurred within 24 hours, characterized by signs of intracranial hypertension. Contrast-enhanced CT and brain MRI confirmed thrombosis of the right sigmoid sinus [5,6]. Management included symptomatic treatment, corticosteroids, and close neurological monitoring, with anticoagulation deferred given the hemorrhagic risk [2,9]. Favorable clinical evolution was observed at one week. This case highlights the diagnostic difficulty of ptCST, whose clinical presentation is non-specific, the key role of contrast-enhanced neuroimaging in diagnosis, and the ongoing therapeutic challenge posed by the competing risks of thrombosis extension and hemorrhagic complications of anticoagulation [11].

Keywords: post-traumatic cerebral sinus thrombosis, traumatic brain injury, intracranial hypertension, sigmoid sinus, anticoagulation, emergency department

INTRODUCTION

Cerebral venous thrombosis (CVT) is a rare form of stroke with a prevalence of 3 to 5 cases per million persons per year, predominantly affecting women (approximately 75% of cases) [1,3]. The most common etiologies include thrombophilia, malignancy, infections, and the post-partum period [3]. Post-traumatic cerebral sinus thrombosis (ptCST) represents a particularly uncommon subtype, accounting for only 1% of all CVT causes [8]. However, its prevalence reaches approximately 4% in patients with traumatic brain injury, rising to 26% when a skull fracture extends to a cerebral sinus—a configuration that constitutes the primary risk factor for this complication [8].

The pathophysiology of ptCST is rooted in the classic Virchow's triad: endothelial injury following direct vascular trauma, venous stasis secondary to external compression by hematomas or depressed fractures, and a post-traumatic hypercoagulable state driven by thrombin generation and antithrombin dysfunction [8,11]. The clinical presentation is highly heterogeneous and non-specific, ranging from isolated headache to coma, posing a significant diagnostic challenge—particularly in the emergency department setting where head trauma is common [6,11].

This case report presents a patient who developed ptCST following isolated blunt head trauma, with an emphasis on the clinical, radiological, and therapeutic aspects of this entity, supplemented by a focused review of the current literature.

CASE PRESENTATION

Initial Presentation

A 62-year-old male patient with no significant past medical history was brought to the emergency department on August 27, 2025, following isolated blunt head trauma sustained as a pedestrian struck by a motorcycle. The primary impact was on the cranium, and the patient arrived approximately two hours after the incident. History obtained from bystanders noted a witnessed loss of consciousness lasting approximately 30 minutes, followed by three episodes of vomiting and post-traumatic amnesia.

On admission, the patient was conscious and cooperative with a Glasgow Coma Scale (GCS) score of 15/15, hemodynamically stable, and afebrile. Neurological examination revealed no focal deficits and no signs of herniation. Clinical assessment identified right-sided otorrhagia and mild tenderness on palpation of the cervical spine.

Initial Investigations

Non-contrast brain CT demonstrated a subarachnoid hemorrhage along the falx cerebri and several cortical sulci, along with a small left frontal hemorrhagic contusion measuring 7.6 mm. A non-displaced occipital fracture was identified on the right side, extending to the ipsilateral petrous bone and passing through the right sigmoid sinus—a finding that warranted further evaluation with contrast enhancement [5]. Cervical spine radiographs showed no bony abnormalities.

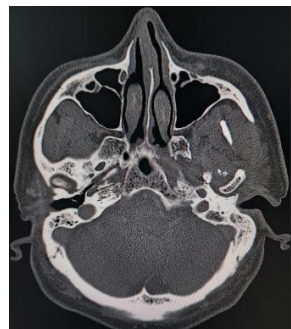


Fig 1: non-displaced fracture line of the right occipital bone radiating towards the ipsilateral petrous bone and passing through the right sigmoid sinus

Clinical Deterioration and Follow-up Imaging

The patient was admitted to the emergency department for 24-hour neurological monitoring as per neurosurgical recommendations. Within 24 hours of admission, mild clinical deterioration was observed, characterized by signs of intracranial hypertension (ICHT): helmet-type headache, vomiting, and mild visual blurring.

Contrast-enhanced brain CT revealed a lacunar filling defect at the level of the right sigmoid sinus, adjacent to the fracture line, consistent with acute venous thrombosis [5]. Brain MRI with MR venography confirmed a thrombus within the right sigmoid sinus on FLAIR sequences, the presence of two frontal hemorrhagic contusion foci, and persistence of the subarachnoid hemorrhage [6].

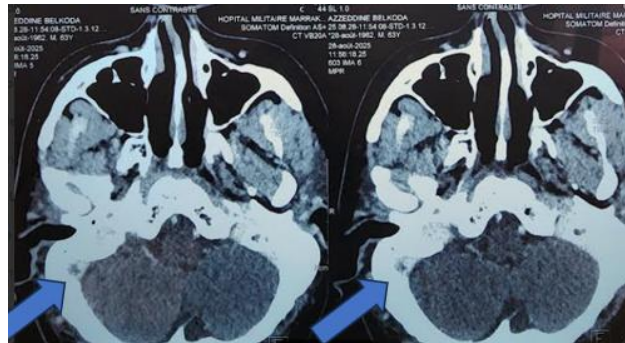


Fig 2: Lacunar image consistent with a thrombus at the level of the right sigmoid sinus opposite the fracture line, consistent with post-traumatic thrombosis.

Management and Outcome

Following multidisciplinary consultation with neurology and neurosurgery, the management strategy included:

- Intravenous hydration with 0.9% normal saline (2 L/24h)
- Intravenous analgesics: paracetamol (given persistent vomiting)
- Intravenous antiemetics: metoclopramide
- Corticosteroids: dexamethasone 8 mg IV every 12 hours for ICHT management
- Anticoagulation: withheld given the hemorrhagic risk (active intracranial hemorrhage) [2,9]
- Close neurological monitoring with serial vital sign assessment

Clinical evolution was favorable. At one week, headache had significantly improved, vomiting had resolved, and visual symptoms had cleared. The patient was discharged with scheduled follow-up MR venography at 15 days to guide the decision regarding anticoagulation [9].

DISCUSSION

Epidemiology

Post-traumatic cerebral sinus thrombosis is an uncommon but clinically significant complication of head trauma. While CVT affects 3-5 persons per million annually—predominantly women [1,3]—ptCST is specifically associated with traumatic brain injury, occurring in approximately 4% of cases [8]. This rate increases dramatically to 26% when the skull fracture extends to a dural sinus, as observed in our patient [8]. ptCST is particularly

rare in children, with a reported incidence of 0.67 per 100,000, accounting for 3-4% of pediatric CVT cases [7].

Pathophysiology

The pathogenesis of ptCST involves all three components of Virchow's triad [11]. First, direct vascular trauma disrupts endothelial architecture, exposing thrombogenic subendothelial tissue and triggering a hypercoagulable state through accelerated thrombin production and antithrombin dysfunction [8]. Second, external venous compression—by hematomas, depressed skull fragments, or compressive fractures adjacent to sinuses—induces venous stasis [8]. Third, elevated intracranial venous pressure eventually compromises blood-brain barrier integrity, leading to both vasogenic and cytotoxic edema. Impaired cerebrospinal fluid resorption through arachnoid granulations projecting into the venous sinuses further aggravates intracranial hypertension [11]. Recent studies have shown that nearly all ptCST cases involve subarachnoid hemorrhage and/or subdural, epidural, or combined contusions, corroborating the central role of local vascular injury [8].

Clinical Features

The clinical presentation of ptCST is notably heterogeneous and non-specific, rendering diagnosis a significant challenge [6,11]. Patients who are initially asymptomatic or present with only mild symptoms may subsequently develop altered mental status, progressive headache, visual disturbances, seizures, and cranial nerve deficits [6]. A change in the character or intensity of headache, or the new onset of signs of intracranial hypertension—as seen in our patient within 24 hours of admission—should prompt urgent neuroimaging [5,6].

Potential complications include subarachnoid or intracranial hemorrhage, venous infarction, cerebral edema, additional CVT, and cerebral herniation [1,11]. The clinical trajectory described by Gowers centuries ago remains applicable: generalized cerebral symptoms including apathy, somnolence, coma, vomiting, and seizures may all herald sinus thrombosis [11].

Diagnosis

The diagnosis of ptCST is primarily radiological. Non-contrast CT, while the standard initial imaging modality for traumatic brain injury in the emergency setting, has a sensitivity of only 20-43% for CVT [5]. Specific CT findings that should raise suspicion for ptCST include skull fractures adjacent to a dural sinus, hyperdense venous sinus, multi-focal hemorrhages, contusions, pneumocephaly, or hematomas near the jugular bulb [5,8].

Contrast-enhanced CT or CT venography can directly visualize the venous clot as a filling defect and is highly reliable for urgent diagnosis [5]. MRI combined with MR venography has largely replaced conventional angiography as the gold standard, offering superior assessment of parenchymal injury and thrombus characterization [6]. In our patient, the sequential use of non-contrast CT, contrast-enhanced CT, and MRI/MR venography allowed for accurate diagnosis and comprehensive lesion characterization.

Treatment

The management of ptCST is guided by four main objectives: prevention of thrombus extension, acceleration of sinus recanalization, prevention of new thrombus formation, and control of intracranial hypertension [2]. Per the American Heart Association/American Stroke Association and European Stroke Organization guidelines, low molecular weight heparin (LMWH) remains the reference treatment for CVT [2,9].

However, ptCST presents a unique dilemma: the risk of hemorrhagic progression from anticoagulation must be weighed against the risk of thrombus extension [9]. To date, no specific evidence-based recommendations exist for this subgroup [9]. The impact of anticoagulation on traumatic brain injury with hemorrhagic complications remains uncertain. Intravenous unfractionated heparin is preferred in severe cases due to its rapid reversibility [2]. A recent study reported that even prophylactic anticoagulation doses may trigger hemorrhagic complications, advocating for early repeat CT within 48 hours of treatment initiation [8].

Emerging evidence supports the potential role of mechanical thrombectomy and endovascular interventions in selected cases with persistent neurological deficits or coma refractory to medical therapy [10]. Nevertheless, conservative approaches have generally demonstrated superior outcomes compared to surgical intervention [10].

Prognosis

The overall prognosis of CVT is guarded: a multicenter study of 624 patients reported a 30-day morbidity rate of approximately 7.7%, with one in five patients experiencing an unfavorable outcome at 6 months [1]. ptCST carries a particularly grave prognosis, with mortality rates ranging from 4.3% to 30% and permanent neurological deficits in 12-25% of survivors [8]. Pediatric patients may benefit from greater vascular plasticity, potentially conferring a more favorable prognosis [7].

CONCLUSION

Post-traumatic cerebral sinus thrombosis is a rare but serious complication of head trauma whose non-specific clinical presentation renders early diagnosis challenging [6,11]. A high index of suspicion is warranted in any patient presenting with head trauma associated with skull fractures adjacent to a dural sinus, multi-focal hemorrhages, or clinical deterioration despite initially reassuring imaging [5,8]. In this context, non-contrast CT is insufficient to exclude the diagnosis, and early recourse to contrast-enhanced CT or MRI/MR venography is essential [5,6].

Treatment remains a significant challenge, as the decision to initiate anticoagulation must balance the competing risks of thrombus extension and hemorrhagic complications—an area in which current evidence and guidelines remain limited [2,9]. Multidisciplinary collaboration between emergency physicians, neurosurgeons, and neurologists is indispensable for individualized therapeutic decision-making and optimal patient outcomes.

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