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**Radiology**

# Fatal Cerebral Trauma: An Imaging Case of Anoxic Ischemia and Jugular Vein Thrombosis

M. Boussif[1*], A. Bouelhaz[1], Ma. Nouri[1], Y. Kherchttou[2], Y. Bouktib[1], A. El Hajjami[1], B. Boutakioute[1], M. Ouali Idrissi[1], N. Cherif Guennouni Idrissi[1]

[1]Department of Radiology, Arrazi Hospital, University Hospital of Mohamed VI, Marrakech, Morocco
[2]Critical Care and Anesthesia Department, University Hospital of Mohamed VI, Marrakech, Morocco

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***Corresponding author:** M. Boussif
Department of Radiology, Arrazi Hospital, University Hospital of Mohamed VI, Marrakech, Morocco

**Abstract** **Case Report**

Post-traumatic cerebral venous thrombosis (PT-CVT) is a rare but serious complication of head trauma, often associated with skull fractures involving the dural venous sinuses or jugular bulb. We report the case of a 25-year-old male admitted after a road traffic accident with vomiting and loss of consciousness. Imaging with contrast-enhanced CT revealed left jugular vein and lateral sinus thrombosis, bilateral frontal and left temporal contusions, and multiple skull fractures. The patient was managed with anticoagulation therapy and close neurological monitoring in the intensive care unit. Despite aggressive management, the patient's condition deteriorated, and he succumbed. PT-CVT is underdiagnosed in traumatic brain injury due to its nonspecific presentation. Imaging, particularly contrast-enhanced CT, is essential for early diagnosis and management. Early detection and anticoagulation therapy are crucial for preventing complications such as venous infarction and hemorrhage, but mortality remains high in severe cases. Our aim is to highlight the importance of imaging in early recognition and prompt management of ptCVT to improve outcomes.

**Keywords:** CT imaging, Post-traumatic cerebral venous thrombosis, road traffic accident, traumatic brain injury.



## INTRODUCTION

Post-traumatic cerebral venous thrombosis (PT CVT) is a rare but potentially severe complication of head trauma, often underdiagnosed due to its nonspecific clinical presentation, which can mimic the symptoms of traumatic brain injury (TBI) [1]. The pathogenesis of post-traumatic CVT implicates extravascular factors, such as direct endothelial injury, venous compression, or a hypercoagulable state. Skull fractures involving the dural venous sinuses or the jugular bulb are commonly identified as contributing factors. [2, 3] The reported incidence of cerebral venous sinus thrombosis (CVST) ranges from 3 to 5 cases per million, whereas ptCVT is observed in approximately 7–35% of TBI cases [4].

Clinically, PT-CVT may present with headache, altered consciousness, or focal neurological deficits, but these symptoms are often masked by the primary traumatic event, leading to delayed diagnosis and increased risk of complications, including elevated intracranial pressure, venous infarctions, and hemorrhage. [5,6] The current literature estimates mortality at around 4–8%. The mismatch between clinical and imaging findings in patients with TBI and certain risk factors should raise suspicion of PT-CVT [7]. Early recognition of this rare and potentially life-threatening complication is crucial for the effective management of severe TBI patients. Imaging, particularly CT scan and magnetic resonance venography (MRV), plays a crucial role in confirming the diagnosis.

This case report aims to highlight the diagnostic and therapeutic challenges of post-traumatic jugular vein and lateral sinus thrombosis, emphasizing the importance of imaging in early diagnosis and CT scan imaging in guiding clinical management.

## CASE PRESENTATION

A 25-year-old male patient with no significant medical history, who presented to the emergency department following a road traffic accident (RTA) with initial vomiting and loss of consciousness. The patient was hemodynamically stable upon arrival, but clinical examination revealed signs of neurological distress.

**A contrast-enhanced CT scan was performed, which revealed the following findings:**

- **Absence of opacification** of the left jugular vein and left lateral sinus, indicating thrombosis. (Figure 1) (Figure 2).
- **Diffuse cerebral edema** with effacement of the cortical sulci and basal cisterns. (Figure 1)
- **Bilateral frontal and left temporal contusions** with minimal subarachnoid hemorrhage visible along the falx cerebri and some cortical sulci.
- **Mass effect** on the midline structures and a **right-left herniation** of 5 mm.
- **Hypodense areas** in the occipital parafalcine and right frontal regions, suggestive of post-traumatic infarction. (Figure 1)
- **Thickening of the intimal wall** of the left internal carotid artery, reducing its luminal diameter in the intracranial portion, though still patent.
- **Fracture** of the left parietotemporal bone with a detached fragment at the petrous apex and double transverse fracture lines, one extending towards the mastoid cells and the other towards the tympanic cavity.
- **Longitudinal transmastoid fracture**, extending through the tympanic membrane and extra-labyrinthine, involving the left carotid canal and extending to the sphenoid body and sphenoid sinus walls. (Figure 3).
- **Undisplaced fracture of the tympanic bone**, with filling of the mastoid cells and the external auditory canal.

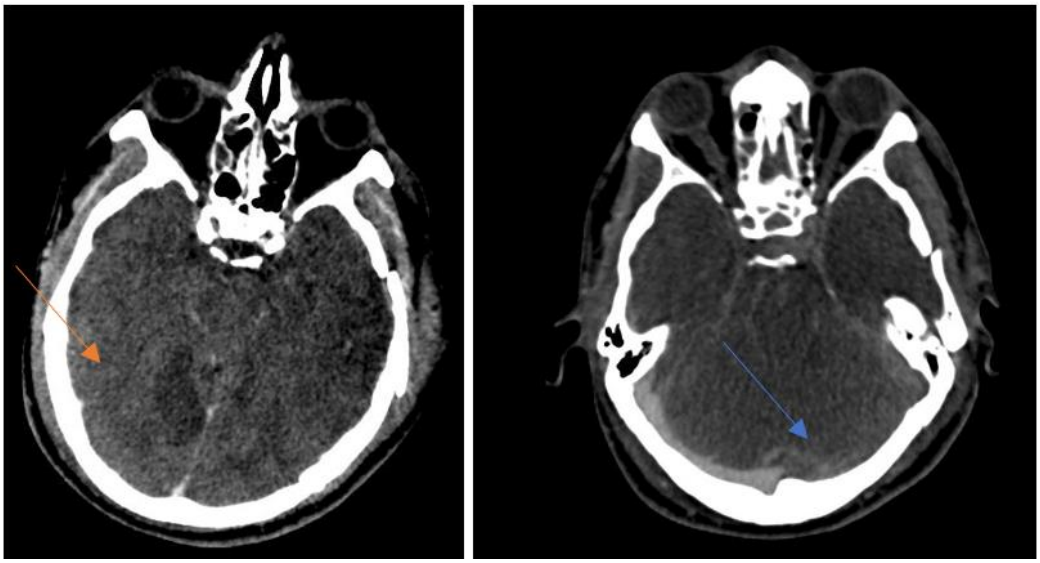

**Figure 1: Head CT, axial with contrast (a, b) showing absence of opacification of the left lateral sinus (blue arrow), diffuse cerebral edema with effacement of the cortical sulci and basal cisterns, and hypodense areas in the occipital parafalcine region (orange arrow)**

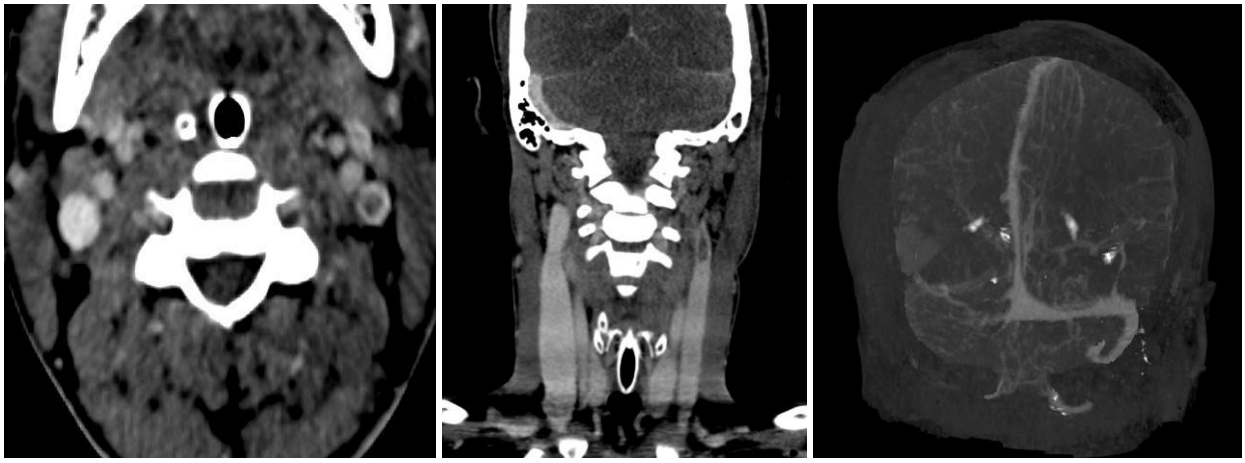

**Figure 2: Head CT with contrast, axial (a) and coronal (b) and head CT venography (c) showing absence of opacification of the left jugular vein (blue arrow) and left lateral sinus (orange arrow)**

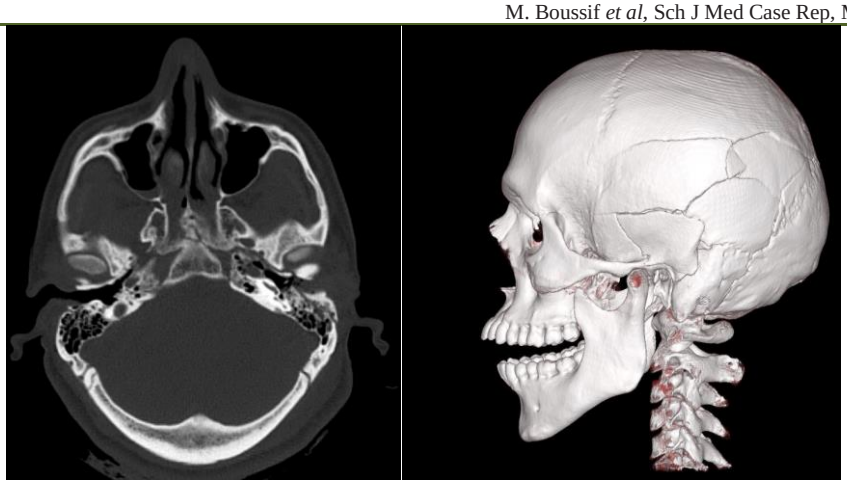

**Figure 3: Head CT, bone window, axial view (a) and 3D reconstruction (b) showing skull fractures**

Following the CT scan, a **transcranial Doppler ultrasound (TCD)** was performed to assess the venous system. The Doppler examination revealed:

- **Complete thrombosis** of the left internal jugular vein, with no detectable flow in the proximal and distal segments. This was consistent with the CT findings of venous occlusion. (Figure 4).
- **Reduced or absent venous flow velocities** in the left lateral sinus, indicating impaired drainage and possible associated thrombosis.
- A **patent right internal jugular vein**, with normal venous flow observed on the contralateral side, suggesting the thrombosis was isolated to the left side.
- **Increased pulsatility** in the left venous system, consistent with raised intracranial pressure, which correlated with the mass effect noted on CT.

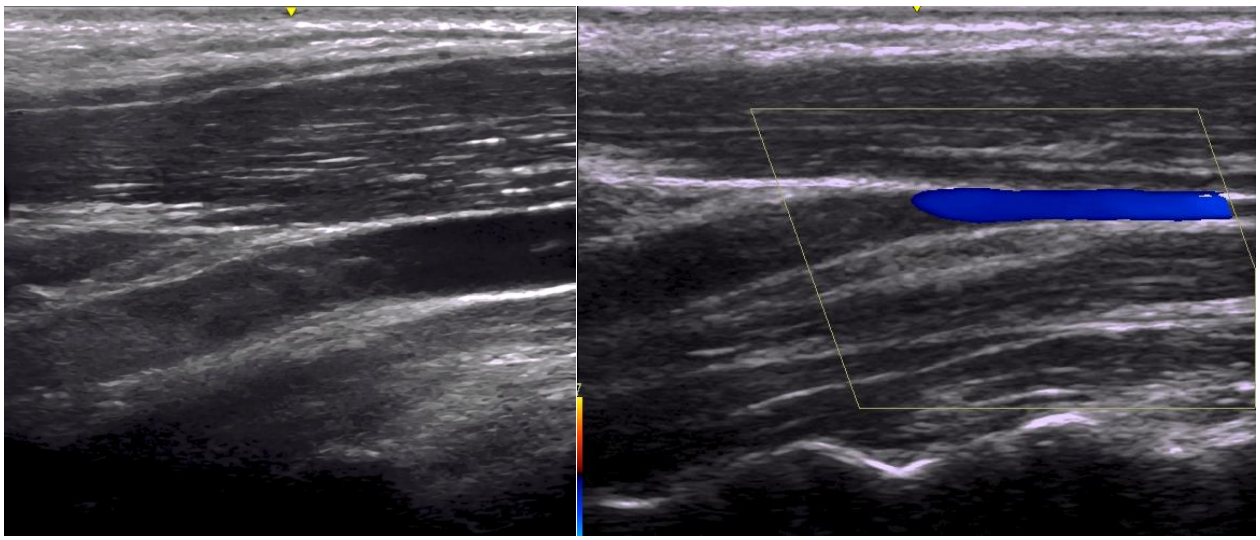

**Figure 4: Ultrasound without Doppler (a) and with color Doppler (b) showing complete thrombosis of the left internal jugular vein (blue arrow), with no detectable flow in the proximal and distal segments and absent venous flow velocities in the left lateral sinus (orrange arrow)**

Based on the imaging findings, the diagnosis of post-traumatic jugular vein and lateral sinus thrombosis was established. The patient was immediately started on appropriate medical management, including anticoagulation therapy, and was closely monitored for neurological deterioration. Follow-up was recommended to assess any changes in the venous system or signs of increased intracranial pressure.

## DISCUSSION

Cerebral venous sinus thrombosis (CVST), whether traumatic or not, is a rare condition, accounting for 0.5–1% of all strokes, with an incidence of 2–5 cases per million people [8,9]. It is more common in younger women and is associated with various risk factors, including thrombophilia, hormonal influences, autoimmune diseases, head and neck infections, non-cerebral malignancies, and hematologic disorders [10 ;

11]. Traumatic brain injury (TBI) is an underdiagnosed cause of CVST (ptCVST), with an estimated prevalence of 32%, particularly in cases with fractures near the affected sinus. [12] The superior sagittal sinus is most frequently involved, and over 50% of ptCVST cases show multiple sinuses affected. [13]

Studies suggest ptCVST is more common in adults than in children, though other research shows similar frequencies in both groups. [14 ; 15] CVST has a 30-day morbidity rate of about 7.7%, with a 6-month prognosis of 20% unfavorable outcomes and a mortality rate of 5%, mainly due to comorbidities or intracranial hemorrhages. In contrast, ptCVST is linked to lower Glasgow Coma Scale scores and higher Injury Severity and Rotterdam CT scores. [16 ; 9] The prognosis of ptCVST is influenced by factors such as age, affected sinuses, and the adaptability of thrombosed sinuses. The severity of ptCVST depends on the location and anatomical structures involved. Notably, pediatric patients may have a better prognosis due to increased sinus plasticity. [6 ; 13]

CVST is driven by Virchow's triad: [17] endothelial injury, hypercoagulability, and blood stasis. Blunt head trauma increases thrombin generation, creating a hypercoagulable state. External factors like hematomas and skull fractures restrict cerebral blood flow, causing venous stasis and increased pressure, leading to cerebral edema, reduced blood flow, and hemorrhage. This disrupts the blood-brain barrier and may cause infarctions or thrombus extension. [13 ; 18]

Clinically, the presentation of post-traumatic CVT can be highly variable, depending on the location and severity of the thrombosis as well as the adequacy of collateral venous circulation. The most common symptom is headache, reported in 75-95% of patients. These headaches may range from mild to severe and are frequently associated with other signs of increased intracranial pressure, including vomiting and visual disturbances. Neurological deficits such as focal deficits, seizures, or altered consciousness may also be present. In the setting of head trauma, these symptoms may be confused with direct trauma effects or post-concussive symptoms, resulting in delays in diagnosis, which can range from three to seven days. [11 ; 13].

Elevated D-dimer levels are often seen in CVT, particularly in cases of traumatic brain injury, though they are nonspecific. D-dimer levels above 500 ng/ml are frequently observed in patients with CVT, but they may be elevated in other conditions such as concurrent hematomas. Notably, a quarter of patients with isolated headaches and CVT may have normal D-dimer levels, which reduces the specificity of this test in the context of TBI.

Imaging plays a pivotal role in the diagnosis of cerebral venous thrombosis (CVT), with various techniques offering valuable insights. Non-contrast CT (NCCT) is commonly used as the initial diagnostic tool for traumatic brain injury (TBI); [8 ; 20] however, its sensitivity for detecting CVST is low, ranging from 20–43%. A normal NCCT scan cannot reliably exclude active post-traumatic CVST (ptCVST) [7]. Despite this, certain imaging findings, such as skull fractures near a dural sinus, hematomas, intraparenchymal bleeding, multiple hemorrhagic foci, contusions, cerebral edema, pneumocephalus, or signs of venous sinus hyperdensity (e.g., the cord sign or delta sign), may raise suspicion for ptCVST. In such cases, early clinical symptoms unresponsive to conservative treatment, in combination with these findings, should prompt further investigation with contrast-enhanced CT venography (CTV) or MRI/MRV for a more detailed assessment of parenchymal defects and venous clots. [17 ; 8 ; 11]

CT venography is useful for revealing a linear hyperdensity along the path of the affected venous sinus or the absence of contrast opacification at the confluence of the sinuses, commonly referred to as the "empty triangle" or "delta sign." However, MRI with venous angiography is considered the gold standard for diagnosing CVT. MRI clearly demonstrates the absence of signal within the thrombotic sinus and can assess the age of the clot based on the breakdown products of hemoglobin in T1- and T2-weighted sequences. CT scans, however, may miss CVT in up to 30% of cases, especially when intravenous contrast is not systematically administered. As a result, MRI remains the preferred modality for detecting CVT. [17 ; 18]

Recent studies suggest that delayed imaging can increase the likelihood of diagnosing ptCVST, even up to 30 days post-trauma. For patients with mild TBI and initial negative imaging, performing a standard CTV within 3–7 days is recommended if there are specific indications. Delayed imaging helps to reassess any previously undetected hemorrhagic lesions or ptCVST signs. [18] Such proactive strategies allow for safe discharge with regular follow-up visits for patients with negative imaging after 3–7 days. Future studies will likely refine patient selection, optimal timing, and the most effective imaging modalities for ptCVST diagnosis. [13 ; 18]

In traumatic cases of CVT, the lateral sinus is most commonly affected, and thrombosis may extend into the jugular vein. The treatment of post-traumatic CVT remains controversial. The decision to initiate anticoagulation therapy, particularly in the acute phase of traumatic CVT, is difficult due to the risk of exacerbating existing hemorrhagic lesions. [15 ; 21] There is a lack of randomized controlled trials providing definitive guidance on the optimal anticoagulation regimen in these cases. In our case, intravenous heparin was initiated following a multidisciplinary discussion between the neurologist and neurosurgeon. Despite intensive care management, the patient's condition

deteriorated, and he ultimately succumbed to complications related to post-traumatic CVT.

The prognosis for post-traumatic CVT has improved with advances in early diagnosis and treatment. However, delays in diagnosis remain common, particularly in cases with atypical clinical presentations. The mortality rate associated with post-traumatic CVT ranges from 4.3% to 30%, and survivors often experience neurological sequelae, particularly when the lateral sinus is involved, as this is typically the dominant hemisphere in most individuals. [22]

## CONCLUSION

In conclusion, while CVT remains a rare complication of TBI, it should be considered in any patient with persistent or worsening neurological symptoms following head trauma, particularly when the clinical presentation includes unexplained headache, focal neurological deficits, or altered mental status. Early diagnosis through appropriate imaging techniques, such as CT venography or, preferably, MRI with venography,and timely initiation of anticoagulant therapy can significantly improve patient outcomes and reduce mortality and morbidity associated with this condition.

**Conflicts of Interest:** The authors declare no conflicts of interest.

## REFERENCES


1. Ferro JM, Canhão P, Stam J, Bousser MG, Barinagarremen-teria F, Investigators. Prognosis of cerebral vein and duralsinus thrombosis: results of the International Study on Cere-bral Vein and Dural Sinus Thrombosis (ISCVT). Stroke 2004;35:664—70.
2. Wang WH, Lin JM, Luo F, Hu LS, Li J, Huang W. Early diagnosis and management of cerebral venous flow obstruction secondary to transsinus fracture after traumatic brain injury. J Clin Neurol 2013;9(4):259–68.
3. Fujii Y, Tasaki O, Yoshiya K, Shiozaki T, Ogura H, Kuwagata Y, et al. Evaluation of posttraumatic venous sinus occlusion with CT venography. J Trauma 2009;66(4):1002–7.
4. Khambholja, J.; Panchal, S.S.; Gami, J.I. A Study of Cerebral Venous Sinus Thrombosis with Special Reference to Newer Risk Predictors. J. Assoc. Physicians 2024, 72, 45–56.
5. Isan, P.; Mondot, L.; Casolla, B.; Fontaine, D.; Almairac, F. Post-traumatic cerebral venous sinus thrombosis associated with epidural hematoma: A challenging clinical situation. Neurochirurgie 2022, 68, e40–e43. [CrossRef] [PubMed]
6. Grangeon, L.; Gilard, V.; Ozkul-Wermester, O.; Lefaucheur, R.; Curey, S.; Gerardin, E.; Derrey, S.; Maltete, D.; Magne, N.; Triquenot, A. Management and outcome of cerebral venous thrombosis after head trauma: A case series. Rev. Neurol. 2017, 173, 411–417. [CrossRef]
7. Netteland, D.F.; Sandset, E.C.; Mejlander-Evjensvold, M.; Aarhus, M.; Jeppesen, E.; Aguiar de Sousa, D.; Helseth, E.; Brommeland, T. Cerebral venous sinus thrombosis in traumatic brain injury: A systematic review of its complications, effect on mortality, diagnostic and therapeutic management, and follow-up. Front. Neurol. 2022, 13, 1079579. [CrossRef] [PubMed]
8. Avsenik, J.; Oblak, J.P.; Popovic, K.S. Non-contrast computed tomography in the diagnosis of cerebral venous sinus thrombosis. Radiol. Oncol. 2016, 50, 263–268. [CrossRef]
9. Theologou, R.; Nteveros, A.; Artemiadis, A.; Faropoulos, K. Rare Causes of Cerebral Venus Sinus Thrombosis: A Systematic Review. Life 2023, 13, 1178. [CrossRef] [PubMed]
10. Coutinho, J.M.; Zuurbier, S.M.; Stam, J. Declining mortality in cerebral venous thrombosis: A systematic review. Stroke 2014, 45, 1338–1341. [CrossRef]
11. Alghamdi, S.R.; Cho, A.; Lam, J.; Al-Saadi, T. Cerebral Venous Sinus Thrombosis in Closed Head Injury: Systematic Review and Meta-analysis. J. Clin. Neurosci. 2022, 98, 254–260. [CrossRef] [PubMed]
12. Meira Goncalves, J.; Carvalho, V.; Cerejo, A.; Polónia, P.; Monteiro, E. Cerebral Venous Thrombosis in Patients with Traumatic Brain Injury: Epidemiology and Outcome. Cureus 2024, 16, e55775. [CrossRef] [PubMed]
13. Harris, L.; Townsend, D.; Ingleton, R.; Kershberg, A.; Uff, C.; O'halloran, P.J.; Offiah, C.; McKenna, G.S. Venous sinus thrombosis in traumatic brain injury: A major trauma centre experience. Acta Neurochir. 2021, 163, 2615–2622. [CrossRef]
14. Dobbs, T.D.; Barber, Z.E.; Squier, W.L.; Green, A.L. Cerebral venous sinus thrombosis complicating traumatic head injury. J Clin. Neurosci. 2012, 19, 1058–1059. [CrossRef] [PubMed]
15. Gong, S.; Hong,W.;Wu, J.; Xu, J.; Zhao, J.; Zhang, X.; Liu, Y.; Yu, R.-G. Cerebral venous sinus thrombosis caused by traumatic brain injury complicating thyroid storm: A case report and discussion. BMC Neurol. 2022, 22, 248. [CrossRef] [PubMed]
16. Netteland, D.F.; Mejlander-Evjensvold, M.; Skaga, N.O.; Sandset, E.C.; Aarhus, M.; Helseth, E. Cerebral venous thrombosis in traumatic brain injury: A cause of secondary insults and added mortality. J. Neurosurg. 2020, 134, 1912–1920. [CrossRef]
17. Bokhari, R.; You, E.; Bakhaidar, M.; Bajunaid, K.; Lasry, O.; Zeiler, F.A.; Marcoux, J.; Baeesa, S. Dural Venous Sinus Thrombosis in Patients Presenting with Blunt Traumatic Brain Injuries and Skull Fractures: A Systematic Review and Meta-

Analysis. World Neurosurg. 2020, 142, 495–505.e3. [CrossRef]
18. Jianu, D.C.; Jianu, S.N.; Dan, T.F.; Munteanu, G.; Copil, A.; Birdac, C.D.; Motoc, A.G.M.; Axelerad, A.D.; Petrica, L.; Arnautu, S.F.; et al. An Integrated Approach on the Diagnosis of Cerebral Veins and Dural Sinuses Thrombosis (a Narrative Review). Life 2022, 12, 717. [CrossRef]
19. Crassard I, Soria C, Tzourio C, et al. A negative D-dimer assay does not rule out cerebral venous thrombosis: a series of seventy-three patients. Stroke 2005 ; 36 : 1716-9.
20. Buyck, P.-J.; Zuurbier, S.M.; Garcia-Esperon, C.; Barboza, M.A.; Costa, P.; Escudero, I.; Renard, D.; Lemmens, R.; Hinteregger, N.; Fazekas, F.; et al. Diagnostic accuracy of noncontrast CT imaging markers in cerebral venous thrombosis. Neurology 2019, 92, e841–e851. [CrossRef]
21. Ochiai, K.; Nishiyama, K. Post-traumatic cerebral sinus thrombosis. BMJ Case Rep. 2021, 14, e239783. [CrossRef]
22. Dany F, El Otmani H, AbdohRafai M, El Moutawakil B, Slassi I. Lateral sinus and jugular vein thrombosis following closed head injury. Afr J Neurol Sci 2014 ; 33(1) : 102-6.