# Posttraumatic cerebral venous sinus thrombosis

- Post-traumatic cerebral venous sinus thrombosis complicated with syndrome of inappropriate antidiuretic hormone secretion: a case report
- Dural Venous Sinus Thrombosis: A Rare Cause of Intracranial Hemorrhage
- Anticoagulation strategies in patients with coexisting traumatic intracranial hematomas and cerebral venous sinus thrombosis: an observational cohort study
- Post-Traumatic Cerebral Venous Sinus Thrombosis (PtCVST) Resulting in Increased Intracranial Pressure during Early Post-Traumatic Brain Injury Period: Case Report and Narrative Literature Review
- Post-traumatic cerebral venous sinus thrombosis in an intensive care unit: A case series of ten patients
- Post-traumatic cerebral venous sinus thrombosis associated with epidural hematoma: A challenging clinical situation
- Cerebral Venous Sinus Thrombosis in an Immunocompetent HIV Patient
- Posttraumatic cerebrovascular injuries in children. A systematic review

see also Dural Venous Sinus Thrombosis following traumatic brain injury.

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Following the TBI, the disruption of blood vessels or other factors related to the injury can predispose the individual to the development of CVST. Patients with dural venous sinus thrombosis (DVST) in select populations following traumatic brain injury (TBI), including those with blunt mechanism or depressed skull fractures, have been shown to have an increased risk of mortality.

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Limited retrospective data suggest that dural venous sinus thrombosis (DVST) in traumatic brain injury (TBI) patients with skull fractures is common and associated with significant morbidity and mortality. Prospective data accurately characterizing the incidence of DVST in patients with high-risk Traumatic Brain Injury are sparse but are needed to develop evidence-based Traumatic Brain Injury management guidelines.

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The trauma may cause damage to the blood vessels in the brain, leading to endothelial injury and a prothrombotic state.

## Clinical features

The patient may experience symptoms of CVST, which can vary depending on the location and extent of the thrombosis. These symptoms may include headaches, visual disturbances, seizures, focal neurological deficits, altered mental status, and signs of increased intracranial pressure.

# Diagnosis

The diagnosis of cerebral venous sinus thrombosis (CVST) following traumatic brain injury involves various imaging techniques and clinical assessment. Here's how it's typically diagnosed:

Clinical Assessment: The patient's medical history, including the details of the traumatic brain injury (TBI), symptoms, and risk factors for thrombosis, are evaluated. Symptoms of CVST may include headaches, focal neurological deficits, seizures, altered mental status, and signs of increased intracranial pressure.

Imaging Studies: Various imaging modalities are used to visualize the brain and detect thrombosis in the cerebral venous sinuses:

Computed Tomography (CT) Scan: This is often the initial imaging modality used to assess traumatic brain injury. In cases of CVST, a CT scan may reveal findings such as hyperdensity in the venous sinuses, parenchymal abnormalities, or evidence of hemorrhage.

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Magnetic Resonance Imaging (MRI): MRI, particularly magnetic resonance venography (MRV), is more sensitive for detecting CVST. It can show direct visualization of the thrombus within the venous sinuses and provide detailed information about the extent of thrombosis and associated complications.

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Cerebral Angiography: In some cases, conventional cerebral angiography or CT angiography (CTA) may be performed to assess the patency of the cerebral venous sinuses and confirm the presence of thrombosis.

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Laboratory Tests: Blood tests may be conducted to assess for prothrombotic conditions or underlying causes of thrombosis, such as coagulation studies, D-dimer levels, and genetic testing for thrombophilia.

# Treatment

Cerebral venous sinus thrombosis treatment

# Systematic reviews

### 2023

Netteland et al. systematically reviewed the currently available evidence on the complications, effect on mortality, and the diagnostic and therapeutic management and follow-up of CVST in the setting of TBI.

Key clinical questions were posed and used to define the scope of the review within the following topics complications; effect on mortality; diagnostics; therapeutics; recanalization and follow-up of CVST in TBI. They searched relevant databases using a structured search strategy. They screened identified records according to eligibility criteria and for information regarding the posed key clinical questions within the defined topics of the review.

From 679 identified records, 21 studies met the eligibility criteria and were included, all of which were observational. Data was deemed insufficiently homogenous to perform meta-analysis and was narratively synthesized. Reported rates of venous infarctions ranged between 7 and 38%. One large registry study reported increased in-hospital mortality in CVSP and TBI compared to a control group with TBI alone in adjusted analyses. Another two studies found midline CVST to be associated with an increased risk of mortality in adjusted analyses. Direct data to inform the optimum diagnostic and therapeutic management of the condition was limited, but some data on the safety, and effect of anticoagulation treatment of CVST in TBI was identified. Systematic data on recanalization rates to guide follow-up was also limited and reported complete recanalization rates ranged between 41 and 86%. In the context of the identified data, they discuss the diagnostic and therapeutic management and follow-up of the condition.

Currently, the available evidence is insufficient for evidence-based treatment of CVST in the setting of TBI. However, there are clear indications in the presently available literature that CVST in TBI is associated with complications and increased mortality, and this indicates that management options for the condition must be considered. Further studies are needed to confirm the effects of CVST on TBI patients and to provide evidence to support management decisions [1)].

### 2020

A systematic review yielded 638 articles, and 13 articles met the inclusion criteria. In patients with skull fractures adjacent to a venous sinus, the prevalence was 26.2% (95% confidence interval = 19.4%-34.4%). This elevated risk was similar between adult (pooled estimate 23.8%; 95% CI = 16.2%-33.5%) and pediatric (pooled estimate 31.3%; 95% CI = 19.1%-46.9%) populations.

They found an unexpectedly high and consistent frequency of DVST among patients with skull fractures regardless of age group or severity of brain injury. These findings are important and highlight the need for further understanding the natural history of DVST and providing better guidelines on its management [2)].

## Retrospective case series

After obtaining institutional approval, 36 adult patients with TBI with skull fractures admitted to an Australian level III adult intensive care unit between April 2022 and January 2023 were prospectively recruited and underwent computed tomography venography or magnetic resonance venography within 72 hours of injury. When available, daily maximum intracranial pressure was recorded.

Dural venous sinus abnormality was common (36.1%, 95% confidence interval 22.5%-52.4%) and

strongly associated with DVST (P = 0.003). The incidence of DVST was 13.9% (95% confidence interval 6.1%-28.7%), which was lower than the incidence reported in previous retrospective studies. Of DVSTs confirmed by computed tomography venography, 80% occurred in patients with extensive skull fractures including temporal or parietal bone fractures in conjunction with occipital bone fractures (P = 0.006). However, dural venous sinus abnormality and DVST were not associated with increased maximum daily intracranial pressure within the first 7 days after injury.

Dural venous sinus abnormality was common in TBI patients with skull fractures requiring intensive care unit admission. DVST was confirmed in more than one-third of these patients, especially patients with concomitant temporal or parietal and occipital bone fractures. Computed tomography venography is recommended for this subgroup of TBI patients [3].

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A case series of 10 patients hospitalized in the intensive care department with post-traumatic cerebral venous thrombosis. Demographic, clinical, and radiological data and their medical management are reported. The incidence of post-traumatic cerebral venous sinus thrombosis in our institution was 4.2%. Cerebral thrombophlebitis was diagnosed incidentally on the initial body scan, on ICU admission in five patients. The left or right lateral sinus was affected in four patients; the sigmoid sinus was affected in 6 patients. Five patients had a thrombosis in the jugular vein. Seven patients had 2 or 3 sites of occlusion. All patients had medical treatment. No hemorrhagic complications were reported. The total duration of anticoagulation was available in 5 cases. A follow-up of MRI or CT scan at 3 months revealed complete sinus recanalization in three patients. Post-traumatic cerebral venous sinus thrombosis in the intensive care department remains underdiagnosed because of the common clinical presentation of traumatic brain injury. Its incidence is increasing because of the increase in high-velocity accidents. And, it seems necessary to conduct prospective studies with a large cohort of patients in the intensive care department [4].

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A case series of five patients admitted for head injury complicated by CVT. The main associated radiological signs were skull fractures crossing the venous sinus and adjacent traumatic hematoma. In four patients, anticoagulation was introduced within 48-72h of CVT diagnosis, with no subsequent hemorrhagic complications. The present report and data from the literature raise the question of systematic additional venoscans when confronted by associated radiological features of post-traumatic CVT [5]

# Case reports

A 66-year-old male who was transferred to the emergency department due to moderate TBI. Initial emergency brain computed tomography (CT) scans revealed certain traumatic lesions, not necessitating any urgent neurosurgical intervention. During his stay in an Intensive Care Unit (ICU), multiple transient episodes of intracranial pressure (ICP) values were managed conservatively, and through placement of an external ventricular drain. Following a series of CT scans, there was a continuous improvement of the initial traumatic hemorrhagic findings despite his worsening clinical condition. This paradox raised suspicion for ptCVST, and a brain CT venography (CTV) was carried out, which showed venous sinus thrombosis close to a concomitant skull fracture. Therapeutic anticoagulant treatment was administered. The patient was discharged with an excellent neurological

status. To date, there are no clearly defined guidelines for medical and/or surgical management of patients presenting with ptCVST. Therapy is mainly based on intracranial hypertension control and the maintenance of normal cerebral perfusion pressure (CCP) in the ICU. The mismatch between clinical and imaging findings in patients with TBI and certain risk factors raises the suspicion of ptCVST [6].

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A 42-year-old man presented with post-traumatic epidural hematoma and rapidly developed transverse sinus thrombosis extending to the internal jugular vein. As the patient was asymptomatic, we decided not to use anticoagulants: close clinical and radiological monitoring was implemented. The hematoma resolved within 2 months, and the CVST diminished by the third month. Such a good outcome is not always the case in ptCVST [7]

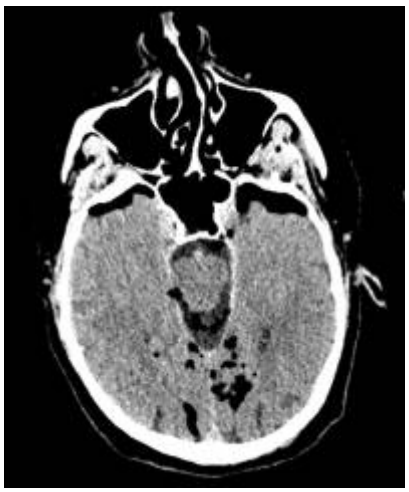
# Case report from the HGUA

## I14431

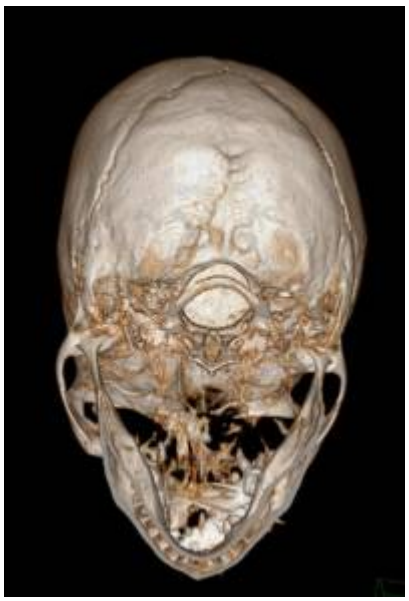
A 61-year-old male suffered an Fall-Related Traumatic Brain Injury from a height of 2m. EMS detects de novo atrial fibrillation. Neurological examination reveals no findings. Hemodynamically stable upon arrival.

CT scan:

Abundant pneumocephalus is identified infra and supratentorial, predominantly bilateral frontal where it reaches 2 cm in thickness and causes mass effect on the adjacent parenchyma with obliteration of the sulci and flattening of the cerebral surface; there is intraventricular extension (lateral and 3rd ventricle), to the basal cisterns and the bilateral parasagittal fronto-parieto-occipital subarachnoid space and cerebellar folia.



Fine subarachnoid hemorrhage in bilateral frontal sulci, left temporal sulci, left Sylvian fissure, and interhemispheric.



Trace of horizontal fracture, slightly oblique in the medial aspect of the left occipital bone, non-displaced, with a fine medial trace extending to the foramen magnum and another fine lateral trace extending to the occipital condyle crossing the condylar foramen, through which an emissary's vein passes to the sigmoid sinus, posing a risk of Cerebral Venous Sinus Thrombosis.

Abundant pneumatization of the petrous apices as an anatomical variant.

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Small intraspinal air bubble partially included at the T3 level, recommending inclusion of the thoracic spine in the next scan unless there is a change in neurological status.

The follow-up CT scan showed evidence of venous thrombosis in the left sigmoid sinus and proximal portion of the left internal jugular vein, anticoagulant therapy with LMWH was initiated.

Stable HD without requiring mechanical ventilation, with good blood pressure control. Pneumatic compression stockings for DVT prophylaxis were removed upon initiation of LMWH. Stable Hb and Ht without thrombocytopenia or coagulopathy. Notable tendency towards hypertension and sinus bradycardia around 45-50 bpm. Initiation of antihypertensive therapy with calcium channel blocker and ACE inhibitor. Eupneic with a good respiratory pattern. Tolerating oral diet albeit very poor appetite, left to demand with prescribed antiemetic.

The patient and family report progressive neurological deterioration over the last 24 hours, with increased drowsiness, bradyphrenia, and decreased speech output. On evaluation by NQx, GCS 12 points were not broken down, and requests for follow-up cranial CT scan revealed:

Subarachnoid hemorrhage in left frontal and temporal intraparenchymal hemorrhage slope with an associated subarachnoid component, not present in the previous study. Likely subarachnoid hemorrhage in the superior vermian cistern of new onset. Disappearance of left pneumocephalus, slight deviation of the midline to the right (2 mm), and subtle decrease in the ipsilateral ambient cistern.

Upon admission to the ICU, continuous hemodynamic monitoring and close NRL surveillance were

initiated.

The patient deteriorated to a GCS of 7 points and intubation and mechanical ventilation were initiated.

The patient shows slow but favorable neurological improvement, is currently aphasic, obeys commands fluctuant, and presents slight right hemiparesis which he mobilizes.

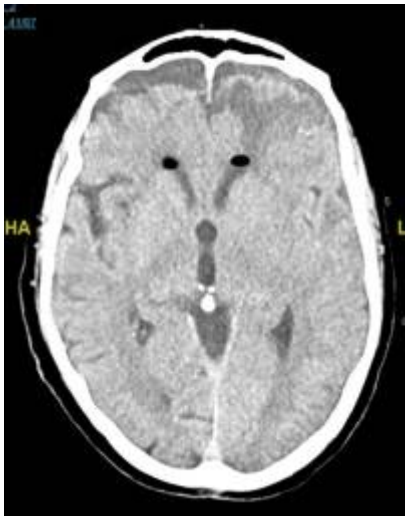
A follow-up contrast CT scan showed a progression of sinus thrombosis, leading to the decision to restart anticoagulation with LMWH.

The patient continued to improve over the following days with eyes opening to call, obeying simple commands fluctuant (language barrier), and improved mobility in the right arm with clear coordinated movements of both arms and legs. Oral diet tolerated with no issues. Mobilized to chair and undergo a rehabilitation program by the Physiotherapy and Rehabilitation Unit.

Cerebral AngioMRI was conducted with an absence of cerebral venous sinus thrombosis and extradural and intraparenchymal lesions in evolution.

The last CT showed

Bilateral frontal hypodensity, as well as hypodensity in the subcortical region of the left temporal pole, related to sequelae of trauma with areas of hemorrhagic contusion, in resorption. Pneumocephalus persists, currently with a bifrontal distribution, smaller than in the previous study.



During his stay in the unit, the patient developed a urinary infection by Proteus Mirabilis and Enterococcus faecalis.

1)

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2)

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