CEREBRAL VENOUS SINUS THROMBOSIS: UNUSUAL CLOT-FORMING PHENOMENON POST HEAD TRAUMA

[1*]Siew Im Khoo, [1]Mohamed Azlam B. Mohamed Micdhadhu, [2]Seu Kean Tan, [1]Irene Looi

[1]Department of Neurology, Hospital Sebarang Jaya, Jln Tun Hussein Onn, Seberang Jaya, 13700 Perai, Pulau Pinang
[2]Department of Radiology, Hospital Sebarang Jaya, Jln Tun Hussein Onn, Seberang Jaya, 13700 Perai, Pulau Pinang

***Corresponding author**:
Siew Im Khoo, Department of Neurology, Hospital Sebarang Jaya, Jln Tun Hussein Onn, Seberang Jaya, 13700 Perai, Pulau Pinang.

Email: siewimkhoo@gmail.com

DOI: https://doi.org/10.32896/cvns.v6n3.1-9
Received: 13.05.2024
Revised: 13.08.2024
Accepted: 26.08.2024
Published: 30.09.2024

ABSTRACT:

Background:
Cerebral venous sinus thrombosis (CVST) is commonly seen in daily practice, especially in those with predisposing factors. Clinicians might however miss the diagnosis if there is a low index of suspicion during clinical assessment of head injury cases, particularly in those without risk factors.
Case Report:
We report a case of a post-traumatic head injury with skull fracture and subarachnoid haemorrhage complicated by delayed, countercoup cerebral venous sinus thrombosis in the context of no predisposing factor for thrombosis. He was started on anticoagulant therapy and achieved clinical improvement within a week.
Discussion:
CVST is seen in post head trauma patients regardless of severity of the injury. Presence of neurological deficit post head injury like altered mental status, headache, vomiting or focal neurological signs like hemiparesis raised the suspicion of CVST. Occurrence of CVST can be delayed by up to more than 10 days post trauma and it can happen at the opposite site of the trauma.
Conclusion:
High index of suspicion is needed to diagnose CVST early and subsequently early intervention or treatment could improve clinical outcome and prognosis of the patients.
Keywords:
Cerebral Venous Sinus Thrombosis, Clot, Post-Trauma, Subarachnoid haemorrhage, Countercoup injury

INTRODUCTION:

Cerebral venous sinus thrombosis (CVST) is commonly seen during daily general practice [1]. It is potentially fatal if not recognized earlier and treated in time [1, 2]. Several predisposing factors place one at a higher risk of CVST, including hereditary thrombophilia, pregnancy and puerperium, post-operative state, and use of oral contraceptive pills (OCP) [1]. This condition is usually overlooked given the nonspecific symptoms [1], low index of suspicion, and unawareness about its association with a head injury [3], particularly in uncommon background [4]. Challenges arise as there is difficulty in diagnosing CVST using non-contrast computed tomography (NCCT) [3]. Fortunately, with the advancements in imaging protocols, the recognition of CVST cases during the last two decades seemed to be increasing [2]. Commonly, patients with CVST presented with altered mental status [2, 5, 6], headache [1, 2, 7], vomiting [2, 7], and focal neurological signs [1, 2, 5]. The aetiology of CVST can be broadly divided into primary hypercoagulable states, secondary hypercoagulable states (drug-induced and pregnancy-induced), post-surgical, and post-traumatic. The brain is rich in tissue factor, a lipoprotein that plays a significant role in the coagulation cascade following injury. Typically expressed in vascular walls and astrocytes, stimulated and activated following injury [8]. The mainstay of treatment for CVST cases, the anti-coagulant therapy, not only improves the prognosis of the patients but clinical recovery [2, 6].

CASE REPORT:

This case was about a healthy 19-year-old man who was involved in a motor vehicle accident while riding a motorbike. He could not recall the mechanism of the accident. He was found unconscious and vomited fresh blood at the scene. When he was brought into Hospital Sungai Bakap (a district hospital), Glasgow Coma Scale (GCS) was full; his vitals were stable, blood pressure was 155/61, heart rate 75 beats per minute, saturation of oxygen was recorded as 97% at room air. His left pupil was 3mm briskly reactive to light, while his right pupil was 4mm sluggish. He sustained a right periorbital hematoma, subconjunctival haemorrhage, and laceration wound over the right eyebrow. Other neurological examinations revealed normal findings. All four limbs' power was 5/5. His computed tomography (CT) brain showed multiple skull vault and facial bone fractures, including bilateral frontal bone extending to the left vertex, superior wall of bilateral ethmoidal sinuses, superior, lateral, and inferior walls of sphenoid sinus, all walls of bilateral orbits, nasal bone, nasal septum, left greater wing of sphenoid extending to the anterior wall of the left external auricular canal, and mastoid part left temporal bone and right mandible. There were multiple intracranial haemorrhages (Figure 1) with generalized subarachnoid haemorrhage (SAH), intraventricular haemorrhage (IVH), and pneumocranium. CT angiography brain was done the next day and showed no arterial aneurysm. Instead, there was SAH, slightly reduced but worsening right frontal peri-haemorrhagic oedema, hydrocephalus, and generalized cerebral oedema.

The patient was kept in the tertiary hospital to monitor his neurological function closely. His blood pressure and heart rate were stable, and he had no neurological deficit. However, he had persistent fever in the ward. He was given intravenous Cefuroxime for one week and later on upgraded to intravenous piperacillin-tazobactam when the temperature increased to 40 degrees Celsius. Clinically, he was not septic, GCS was full, no sign of meningism, and the septic parameter was reducing in trend. Serum C-reactive protein (CRP) level dropped from 65mg/L to 15mg/L, white blood cell count 22x103 to 7.3x103. The antibiotics were completed for a total of 2 weeks. Serum lupus anti-coagulant was not

detected. The oral-maxillofacial surgery and ophthalmology teams planned for open reduction and internal fixation (ORIF) surgery later after their assessment.

On day 13 of trauma, the patient developed left-sided weakness with the power of 3/5. A plain CT brain showed new hypodensities at the right frontal and parietal lobes (Figure 2A) and suspicious hypodensity within the right transverse and sigmoid sinuses. The right SAH was however resolving. With the suspicion of cerebral venous sinus thrombosis, a CT venogram was performed and showed hypodense filling defects in the right transverse and sigmoid sinuses in keeping with dural sinus thrombosis (Figure 2B). Subcutaneous enoxaparin 80mg (1mg/kg) was started twice daily.

On day 20 of trauma, the patient vomited despite being stable neurologically. CT brain proceeded and showed a slight increase in SAH of the right suprasellar cistern. Subsequent CT brain on Day 22 of trauma and Day 27 showed no interval change, and his vomiting resolved. Upon discharge on Day 31, his GCS was full. His power over the left was 3/5 proximally and 4/5 distally. He was able to ambulate with minimal assistance. He was discharged with subcutaneous enoxaparin, given the possibility of facial surgery on the recent date.

His neurological status did not show significant change six weeks post-trauma when he was assessed in the outpatient clinic, and the repeated CT brain showed no significant increment of bleeding. As there was no plan for any surgical intervention from the dental and ophthalmology team, his anticoagulation was thus changed from subcutaneous enoxaparin to oral dabigatran 150mg twice daily.

DISCUSSION:

Prevalence

Trauma-induced CVST is relatively uncommon and therefore poses diagnostic and management challenges. In a literature review of 846 paediatric CVST cases, only 28 patients (3%) were reported in the context of head trauma [6]. National Trauma Data Bank (NTDB) - the most extensive trauma database of around 900 trauma centres in the United States, documented around 453,775 head and neck injury patients for a duration of one year (2009–2010) [9]. Among them, 76 patients had cerebral venous sinus injuries. Meta-analysis and systemic review of 638 articles conducted by Bokhari R et al. concluded that skull fractures abutting sinuses are responsible for CVST – in 26.2% of cases [10]. In a paediatric study conducted by Hersh et al. of 2224 patients with a head injury and skull fractures, among 41 patients who underwent venous imaging, 20% of patients had intrinsic sinus thrombosis, and 34% had extrinsic venous compression [11]. In the study conducted by Fujii Yoshiyuki et al. of 97 patients with a head injury and skull fractures, the incidence of venous sinus thrombosis was 22.4%, mortality around 50%, and lethal intracranial hypertension in 40.9% [12]. A systemic PubMed search of records (Nader Hejrati study) related to paediatric TBI until June 2019 stated that 38.2% of CVST cases are attributed to TBI [13].

The severity of head injury (HI)

To diagnose CVST in patients with head injuries (HI), especially patients without risk factors, clinicians need to have a high level of suspicion because it can occur even in mild head injury cases. A paediatric case of post-HI CVST presented with progressive headache and vomiting following a minor fall [7]. In an institutional study and literature review, patients who improve clinically are not subjected to contrast-enhanced CT brain. On the other hand, patients with clinical suspicion are subjected to CT brain with contrast irrespective of either mild, moderate, or severe head injury. Four patients presented with mild HI but developed persistent headache/ vomiting, and their imaging showed CVST [2]. Our case presented with severe HI and extensive

subarachnoid haemorrhage, but there was no suspicion of CVST on admission. Only when he developed neurological deterioration later in the ward, we proceeded for contrasted imaging.

Signs of suspicion

The common signs for post-head trauma are altered mental status [2, 5, 6], headache [1, 2, 7], vomiting [2, 7], or focal neurological signs like hemiparesis. [1, 2, 5] In mild HI with CVST, the patient presents with an increase in the severity of headaches associated with vomiting. In moderate head injury patients, in addition to the symptoms mentioned above, the patient may have worsening of the sensorium, drowsiness, and altered mental status. There may be cranial nerve palsies depending upon the location of CVST [2]. A case reported a young male admitted for horizontal diplopia and headache with clinical findings of bilateral sixth cranial nerve palsy, and fundoscopy revealed papilledema and retinal haemorrhage. His magnetic resonance imaging (MRI) brain showed a right frontoparietal cortex contusion and left transverse sinus thrombosis [1]. In severe head injury patients, they have worsening cerebral oedema, venous congestion, and deterioration of GCS [2]. Our case presented with sudden unilateral weakness, which raised the suspicion for us to repeat the CT brain. The new cerebral oedema resulting from the CVST over the right transverse sinus and sigmoid sinus explained the newly developed left hemiparesis.

Duration from trauma

In one institutional study, four mild HI patients developed suspicious symptoms after 1, 2, 5, and 7 days post-trauma respectively [2]. A paediatric case presented within 24 hours post-trauma [7]. Our patient had clinical deterioration 12 days post-trauma, with imaging showing CVST. This is similar to a case reported by Marjan et al., who was presented to the hospital 12 days post-trauma [1].

Countercoup CVST

Countercoup brain injury involves a contusion remote from, and classically opposite to, the original site of impact to the head [14]. Our case had multiple skull vault fractures, particularly involving the left side, but the CVST occurred on the contralateral side. This could be explained by countercoup sinus thrombosis. 2 cases with right temporal bone fracture were complicated with left-sided sinus thrombosis. In contrast, another patient with left mastoid and temporal bone fracture was complicated with right-sided sinus thrombosis [2]. Another case reported right frontoparietal cerebral contusion was complicated with left transverse sinus thrombosis [1].

Thrombosis location

The involvement of sinus thrombosis is related to the location of the fracture. Fractures of the petrous temporal bone have the highest chance of injury to the transverse sinus, sigmoid sinuses, and jugular bulb. In contrast, the association of occipital bone fractures is higher with thrombosis of the superior sagittal sinus [15]. Lashkar B reported a case of a 9-year-old boy with minor HI who had sutural diastasis of the right lambdoid suture and thrombosis of the right sigmoid sinus [7]. Ragurajaprakash K's study reported cases of fracture extending to the inferior aspect of the right jugular foramen with right sigmoid sinus thrombosis, and another case involved left lambdoid suture, and left sigmoid sinus, transverse sinus, internal jugular vein [2]. Matsushige et al. showed that the most commonly involved sinus is the posterior part of the superior sagittal sinus; however, sigmoid sinus thrombosis is the most common site, according to Dalgiçet et al. [16, 17].

Treatment and outcome

There is some concern about the use of anticoagulants in head trauma patients. Hence, the role of hyperosmolar therapies (mannitol and hypertonic saline) has been advocated as first line treatment to lower intracranial pressure. If the clinical condition worsens, systemic anticoagulation, surgical decompression, or endovascular therapies such as chemical thrombolysis can be tried [5]. In the literature of posttraumatic paediatric CVST, among 28 cases reported, three did not specify the treatment received, 23 received no intervention, and the remaining 3 cases received anticoagulant, all of which achieved complete recovery of symptoms [6]. Short-term parenteral anticoagulant (subcutaneous enoxaparin) was given during hospitalization in a study, 10–20 days in severe HI cases, 7–10 days in moderate HI cases, and 5-7 days in mild cases until thrombosis resolved completely. All ten patients with CVST were not discharged with oral anticoagulant as thrombosis resolved completely, and patients recovered rapidly in a few days. During follow-up, all patients recovered completely [2]. Enoxaparin 40 mg subcutaneously twice a day can be started if there is no new bleeding after 48 hours. When associated with CVST, head injury patients take a long duration for recovery and cognitive impairment. Prompt and timely diagnosis of CVST is essential, which helps in proper early management with anticoagulants (after 48 hours of trauma) and faster recovery [2].

Asadollahi M et al. reported a case who was treated with intravenous heparin (80 IU/kg bolus, and 18IU/kg/hour continuous infusion) and added warfarin daily after 24 hours, whereby patient's symptoms resolved entirely within two weeks [1]. Our patient initially started with a parenteral anticoagulant and later switched to an oral anticoagulant during follow up 6 weeks post-trauma. He was planned for a total of 3 months of the anticoagulant in view of provoked thrombosis.

CONCLUSION:

Early clinical diagnosis and early institution of therapy, particularly heparin or thrombolysis followed by oral anticoagulants, have improved the prognosis of CVST [4]. Therefore, careful evaluation of cerebral venous sinuses in the diagnostic checklist when looking at admission CT in all cranial trauma cases is critical, particularly in patients with fractures or hematomas near venous sinuses, e.g., basilar skull fractures, which are associated with transverse/sigmoid sinus thrombosis [5, 16]. Similarly, not all extra-axial hyperdense haemorrhagic collections are subdural hematomas located in the posterior fossa and along the interhemispheric fissure and deserve scrutiny to exclude or confirm associated with dural sinus injury. Second, the persistence or emergence of raised intracranial pressure features in the delayed or subacute period, e.g., persistent headache, seizure, papilledema, etc., with CT showing unexplained cerebral oedema or delayed ICH, should be promptly investigated with appropriate vascular imaging (CT venography/Magnetic resonance venography, MRV) [3]. Our case interestingly reported that post-traumatic head injury could be complicated with CVST as long as 12 days from the trauma. It can occur at the opposite side of the fracture site (countercoup thrombosis). Prompt initiation of anticoagulants and physiotherapy aids in rapid recovery of clinical symptoms.

DATA AVAILABILITY:

Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable request.

FUNDING:
This work received no external funding.

CONFLICT OF INTEREST:
The authors have no conflicts of interest to declare and are in agreement with the contents of the manuscript.

REFERENCES

1. Asadollahi M, Ramezani M, Sakhaii E, Karimialavijeh E. A Case of Post-traumatic Cerebral Venous Sinus Thrombosis. Journal of Neurology and Neuroscience. 2016;7(4):0-.
2. Ragurajaprakash K, Senthilkumar R, Prabu SS, Madeswaran K, Kiruthika P. Post-traumatic cerebral venous sinus thrombosis–Institutional study and literature review. Interdisciplinary Neurosurgery. 2022 Mar 1;27:101398.
3. Mandeep Singh Ghuman, Pravin Salunke, Sushanta K. Sahoo, and Shabdeep Kaur. Cerebral venous sinus thrombosis in closed head trauma: A call to look beyond fractures and hematomas! J Emerg Trauma Shock. 2016 Jan-Mar; 9(1): 37–38. doi: 10.4103/0974-2700.173865
4. Chiewvit P, Piyapittayanan S, Poungvarin N. Cerebral venous thrombosis: diagnosis dilemma. Neurology international. 2011 Nov;3(3): e13.
5. Hsu PJ, Lee CW, Tang SC, Jeng JS. Pearls & Oy-sters: Delayed traumatic intracerebral hemorrhage caused by cerebral venous sinus thrombosis. Neurology. 2014 Sep 30;83(14): e135-7.
6. Awad AW, Bhardwaj R. Acute posttraumatic pediatric cerebral venous thrombosis: case report and review of literature. Surgical Neurology International. 2014;5.
7. Lakhkar B, Lakhkar B, Singh BR, Agrawal A. Traumatic dural sinus thrombosis causing persistent headache in a child. Journal of Emergencies, Trauma and Shock. 2010 Jan;3(1):73.
8. Giesen PL, Rauch U, Bohrmann B, Kling D, Roqué M, Fallon JT, Badimon JJ, Himber J, Riederer MA, Nemerson Y. Blood-borne tissue factor: another view of thrombosis. Proceedings of the National Academy of Sciences. 1999 Mar 2;96(5):2311-5.
9. Qureshi AI, Sahito S, Liaqat J, Chandrasekaran PN, Siddiq F. Traumatic injury of major cerebral venous sinuses associated with traumatic brain injury or head and neck trauma: analysis of national trauma data bank. Journal of Vascular and Interventional Neurology. 2020 Jan;11(1):27.
10. Bokhari R, You E, Bakhaidar M, Bajunaid K, Lasry O, Zeiler FA, 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 Neurosurgery. 2020 Oct 1;142:495-505.
11. Hersh DS, Shimony N, Groves ML, Tuite GF, Jallo GI, Liu A, Garzon-Muvdi T, Huisman TA, Felling RJ, Kufera JA, Ahn ES. Pediatric cerebral venous sinus thrombosis or compression in the setting of skull fractures from blunt head trauma. Journal of Neurosurgery: Pediatrics. 2018 Mar 1;21(3):258-69.
12. Fujii Y, Tasaki O, Yoshiya K, Shiozaki T, Ogura H, Kuwagata Y, Sugimoto H, Sumi Y. Evaluation of posttraumatic venous sinus occlusion with CT venography. Journal of Trauma and Acute Care Surgery. 2009 Apr 1;66(4):1002-7.
13. Hejrati N, Ebel F, Guzman R, Soleman J. Posttraumatic cerebrovascular injuries in children. A systematic review. Child's nervous system. 2020 Feb;36(2):251-62.
14. Payne WN, De Jesus O, Payne AN. Contrecoup Brain Injury. 2022 May 29. In: StatPearls [Internet]. Treasure Island

(FL): StatPearls Publishing; 2022 Jan–. PMID: 30725650.

15. Delgado Almandoz JE, Kelly HR, Schaefer PW, Lev MH, Gonzalez RG, Romero JM. Prevalence of traumatic dural venous sinus thrombosis in high-risk acute blunt head trauma patients evaluated with multidetector CT venography. Radiology. 2010 May;255(2):570-7.
16. Dalgiç A, Secer M, Ergüngör F, Önder OK, AKDAĞ R, Ciliz D. Dural sinus thrombosis following head injury: report of two cases and review of the literature. Turkish Neurosurgery. 2008 Jan 1;18(1).
17. Matsushige T, Nakaoka M, Kiya K, Takeda T, Kurisu K. Cerebral sinovenous thrombosis after closed head injury. Journal of Trauma and Acute Care Surgery. 2009 Jun 1;66(6):1599-604.

FIGURE LEGEND:

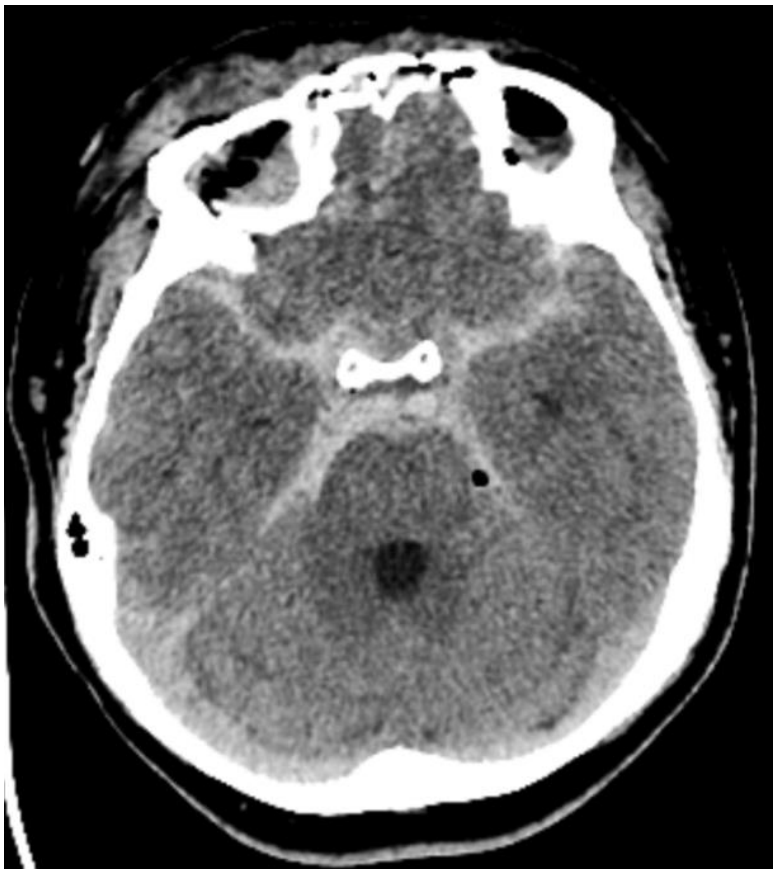

Figure 1: CT Brain on admission post trauma noted multiple intracranial haemorrhages with acute generalised subarachnoid bleeds, pneumocranium and multiple bones fractures.

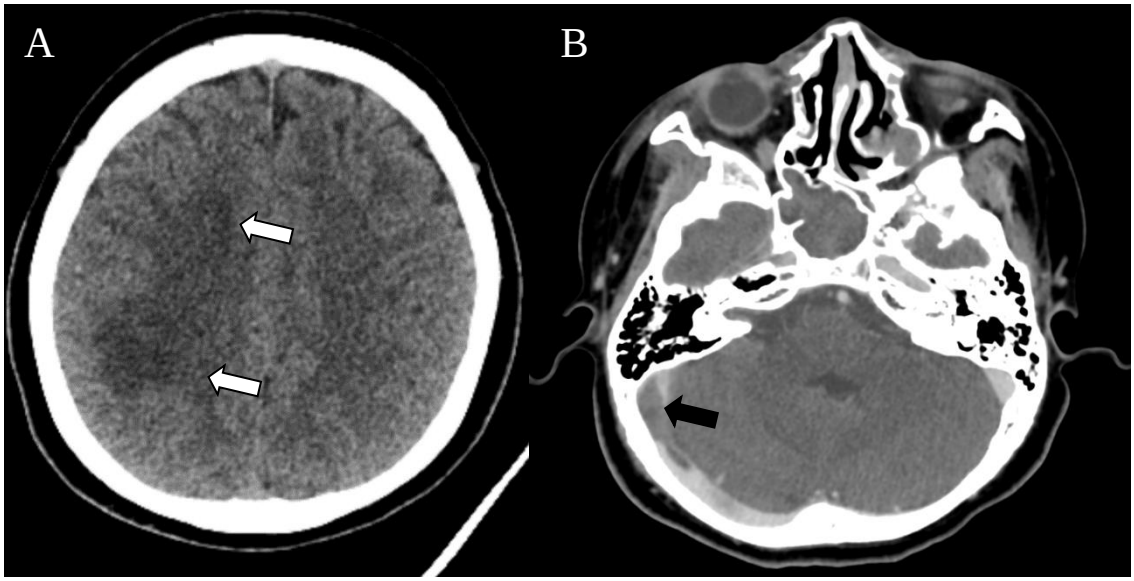

Figure 2: CT Brain on Day 13 of trauma showed new ill-defined hypodensities at right frontal and parietal lobes (white arrows) suggestive of recent infractions with suspicious hypodensity in right transverse and sigmoid sinuses (not shown) (A). CT venography brain performed confirmed the right transverse and sigmoid sinuses thrombosis (Black arrow, B).