JOURNAL OF CARDIOVASCULAR, NEUROVASCULAR & STROKE

Contents

Volume 6 **Number 3** **Sept 2024**

No. ***Title*** ***Page***

Case Reports

EDITORIAL

I am deeply honoured to assume the role of Editor-in-Chief for the Journal of Cardiovascular, Neurovascular, and Stroke (CVNS). It is with great respect for the foundation built by my predecessor that I step into this position, committed to upholding the values that define our journal. As we look toward the future, I would like to share my vision for CVNS and highlight the exciting path ahead.

In today's expansive landscape of medical publications, CVNS must continuously demonstrate its relevance and importance. Our unique mission positions us to do just that. We have established ourselves as a crucial platform, particularly for researchers and clinicians who encounter barriers when submitting their work to more established European or American journals. CVNS has become a beacon for authors, especially those from Southeast Asia, offering them the opportunity to showcase their research on an international stage. This inclusivity and dedication to diversity remain central to our purpose.

Moreover, we recognize the profound value that case reports contribute to medical science. While many established journals have moved away from accepting such reports, we embrace them. These anecdotal studies often offer the first glimpse of new diagnoses, treatments, or complications, enriching the broader medical knowledge in ways that larger studies may overlook. Our commitment to providing a platform for these educational and insightful pieces will remain a distinctive feature of CVNS.

Looking ahead, my vision for CVNS is to strengthen our foundation while expanding into new frontiers. I aspire for our journal to become a leading forum for ground-breaking research in cardiovascular, neurovascular, and stroke disciplines. This includes maintaining our commitment to timely and rigorous peer review and encouraging submissions that challenge the boundaries of current knowledge. We will continue to uphold the highest standards of scientific integrity and foster an inclusive platform for researchers around the world.

To the members of our editorial board, I look forward to your continued collaboration, insights, and dedication to advancing medical science. Together, we will ensure that CVNS remains a leading voice in academic discourse, contributing meaningfully to both the medical community and patient care.

Thank you for your unwavering dedication and support. I am excited to embark on this journey with you all and look forward to the future we will shape together.

Warm regards,

Assoc. Prof. Dr. Khairul Azmi Abdul Kadir
Editor-in-Chief
Journal of Cardiovascular, Neurovascular, and Stroke (CVNS)

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.

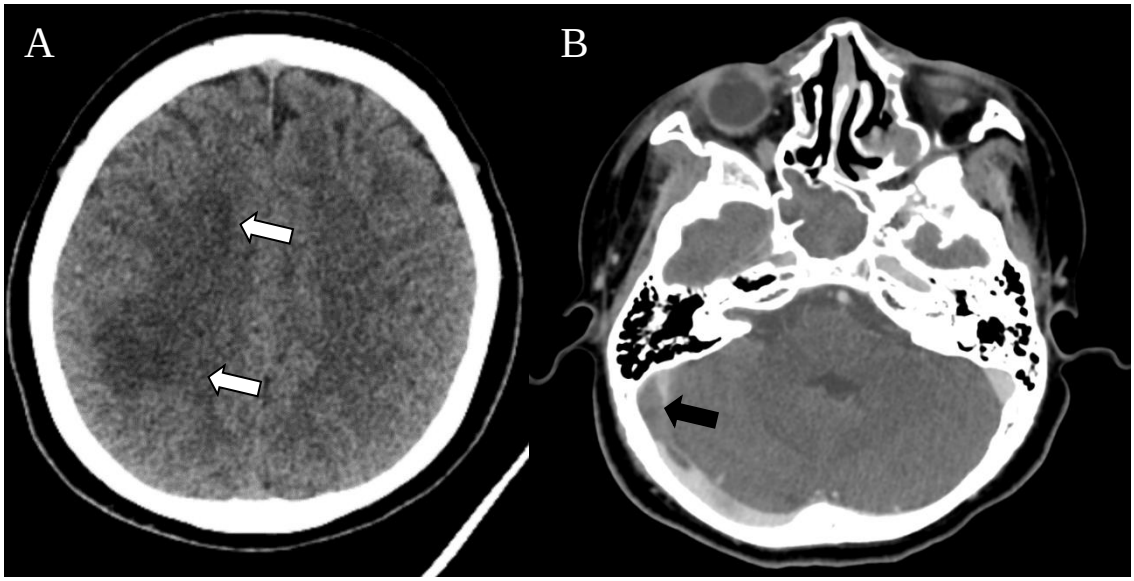

Figure 2: CT Brain on Day 13 of trauma showed new ill-defined hypodensities at right frontal and parietal lobes (white arrows) suggestive of recent infractions with suspicious hypodensity in right transverse and sigmoid sinuses (not shown) (A). CT venography brain performed confirmed the right transverse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

IATROGENIC SMALL BOWEL HEMORRHAGE TREATED WITH TRANSCATHETER ARTERIAL EMBOLIZATION

[1*]Nguyen Vu Dang, [1]Bui Tien Si

[1]Department of Radiology, Can Tho University of Medicine and Pharmacy, Ho Chi Minh, Vietnam

***Corresponding author**:
Nguyen Vu Dang, Department of Radiology, Can Tho University of Medicine and Pharmacy, Ho Chi Minh, Vietnam.

Email: nvdang@ctump.edu.vn

DOI: https://doi.org/10.32896/cvns.v6n3.10-18
Received: 07.06.2024
Revised: 24.09.2024
Accepted: 27.09.2024
Published: 30.09.2024

ABSTRACT:

Background:
Postsurgical iatrogenic gastrointestinal hemorrhage is an emergency which could carry high mortality rate and cause prolonging the hospitalization time. Endoscopic intervention plays a main role, although not always successful meanwhile reoperation is often difficult and dilematic. Endovascular intervention is minimally invasive and could be an effective management for this urgency.
Case Report:
A 74 years-old male patient with no significant pathology history had an abdominal pain at the right upper quadrant extending to the gastric areas. He was diagnosed with cholangitis and biliary obstruction due to pacreatic head tumor. Subsequently, he experienced a Whipple surgery for the pancreatic head tumor and D2 duodenal removal accompanied by a choledochojujenal anastomosis. Since the 9th day post-surgery, he has a suspicious gastrointestinal hemorrhage. On the day 11th after the surgery, he had a hypovolemic shock with a hemoperitoneum. The suspicion of the bleeding causes from the resected arteries of the sutured sites had been risen. An abdominal computed tomography angiography (CTA) was performed and the hemorrhagic pseudoaneurysmal artery was identified. After a discussion, he was immediately sent to the angio-suite for the interventional embolization. Check angiogram showed complete occlusion of the pseudoaneurysm without any complications.
Conclusion:
Transcatheter arterial embolization had shown its effectiveness in the treatment of the postoperative iatrogenic gastrointestinal bleeding. Its technical simplicity makes it more advantageous outweighing a repeat of a surgery especially in the high-risk patients with multiple comorbidities.
Keywords:
Iatrogenic gastrointestinal hemorrhage, Transcatheter arterial embolization, Therapeutic endoscopy, Pseudoaneurysm

INTRODUCTION:

Post-operative gastrointestinal bleeding is a complication that can lead to severe consequences. It could prolong hospital stay or increases the mortality risk. Some lethal cases even necessitate emergency rescue intervention. Generally, the cause of gastrointestinal bleeding originates from peptic ulcers. Less common causes include benign and malignant conditions such as gastrointestinal angiodysplasia, portal hypertension, trauma, and medical procedures. The incidence of post-operative gastrointestinal bleeding is relatively low, ranging from 0.4% to 16% depending on the different studies [1,2]. One of the rare causes is visceral artery pseudoaneurysm following abdominal surgeries [3,4]. The management for this entity needs prompt, accurate, and highly effective diagnostic and therapeutic measures. The first common treatment option for this complication is endoscopic hemostasis which is a preferred method in most patients if the patient's hemodynamics are stable. However, in many cases, endoscopy may be challenging due to the limited visualization and localization of the bleeding site due to a large amount of intraluminal blood. Some other cases manifest with intra-abdominal hemorrhage which is not controllable endoscopically. Under these certain circumstances, a repeated surgery is hard to obtain since it could dramatize the existing injuries and it is difficult to identify the bleeding vessels inside the previously operated structures due to post-operative anatomical distortion, inflammation, adhesion. whilst being given the unstable hemodynamics, a rapid hemostasis intervention is timely required to save the patient's life. In such situations, an endovascular embolization for hemostases is a highly recommended option.

CASE REPORT:

A 74-year-old male patient was admitted to the university hospital due to an abdominal pain. He has a medical history of hypertension and type 2 diabetes. After the examination, the patient was diagnosed with suspected cholecystitis due to distal common bile duct choledocholelithiasis. There was an associated suspicious pancreatic head tumor, adjacent to the second part of duodenum. The patient was scheduled for exploratory surgery and treatment. Upon the surgery, a tumor mass was found in the pancreatic head, with the characteristics corresponding to the computed tomography (CT) images. A Whipple surgery was performed accordingly. The patient tolerated quite well post-surgically. However, on the 9th postoperative day, the patient began to have minimal black blood-stained stools, indicating a gastrointestinal hemorrhage with suspicious bleeding from the operation sites. The patient was managed conservatively, being administered with fluids, receiving medicines, and being closely monitored. On the 11th postoperative day, the patient became lethargic and tachypneic with SpO2 fluctuating between 88 to 90%. He also had a hypotension with blood pressure of 80/50 mmHg and a hemoglobin drop to 10g/dl, indicating an acute blood loss with unknown causes. A multidisciplinary discussion was made by the surgery, the intensive care and the radiology teams leading to an initial diagnose which was hemorrhagic shock due to massive gastrointestinal bleeding and/or possible internal bleeding. The patient was resuscitated with intravenous fluid and blood transfusions to maintain the vital signs. He then was sent for urgent radiological screening, keeping in view an emergency intervention for urgent hemostasis. A contrast-enhanced abdominal CT angiography (CTA) had revealed the contrast extravasation adjacent to the lower part of the left liver, suspecting a bleeding site originating from the operated arteries in Figure 1. A swift discussion between the surgical and the interventional radiology teams lead to the unison decision for endovascular

intervention accordance to the relative consent. Subsequently, the patient was transferred immediately to the angio-suite for the urgent embolization. Diagnostic run was performed using a Yashiro catheter showing a pseudoaneurysm arising from a branch of the right hepatic artery. Selective catheterization and angiogram were done followed by coiling embolization of the bleeding artery as shown in Figure 2. Complete occlusion of the pseudoaneurysm as well as the the feeding artery was obtained which was demonstrated on the post embolisation images in Figure 3.

The day right after the embolization of the bleeding artery, the patient's clinical condition significantly improved in the post procedure room. His blood pressure returned to normal ranges, and it was stable at 100/60 mmHg. His SpO2 level was up to 96%. He was conscious and even being able to sit up. He was then transferred to the ward for close monitoring of the vital signs and further treatment. After a few days operatively, he was doing well and discharged subsequently without any recorded post-intervention complications.

DISCUSSION:

In the past decades, transcatheter arterial embolization (TAE) has developed and emerged as the first-line treatment for gastrointestinal bleeding when endoscopic therapy is ineffective. Interventional radiology offers rapid, safe, and minimally invasive hemostasis, also serving as a substitute for surgery. With various modern techniques and increasing experience of interventional physicians, it also has become a worthy consideration when facing the cases with recurrent bleeding after previous endoscopic or surgical interventions.

This TAE technique is suitable for the patients with coagulation disorders, with a shorter preparation time and being easier to identify the bleeding vessels compared to open surgery. Particularly, it can be repeated multiple times due to minimal invasiveness, which is a significant advantage over surgery. By far, the advanced embolization materials combining with the skills of interventional physicians, significantly reduces the complications of intestinal ischemia compared to earlier periods.

Its indications could include massive bleeding (requiring transfusion of at least 4 units of blood within 24 hours), unstable hemodynamics or hemorrhagic shock (hypotension with systolic blood pressure < 100 mmHg and heart rate > 100 beats per minute), acute status unmanageable conservatively, failure of endoscopic hemostasis on two occasions or high-risk patients, recurrent bleeding after surgery and those with associated intra-abdominal hemorrhage [2, 6].

This TAE procedures should be performed as early as possible right after the diagnostic suspicion of hemorrhagic emergency. The causative blood vessels can be accurately localized using CT or pre-procedure endoscopy. Other centers require the CTA to be performed routinely to identify the bleeding arteries [5]. However, in cases of profuse and massive bleeding, this step may be skipped, and the patient should be promptly transferred to the interventional centers. Computed tomography angiography is currently useful and has high sensitivity in detecting the bleeding vessels. [6, 8]

Regarding the technique, firstly the culprit blood vessel is often identified via endoscopy, the blood clotting clips or through a CTA. Therefore, performing the angiography allows a rapid access to the causative blood vessels. Visualization of contrast agent extravasation into the bowel lumen or the creation of pseudoaneurysms serves as indications for the culprit blood vessel. The interventional embolization may need to be performed simultaneously with supportive measures for blood clotting. The femoral artery approach is commonly used, utilizing a 5F or 6F sheath for femoral artery access. Various types of guiding catheters and microcatheters with small diameters are used for visceral artery

accesses. The commonly used ones include Cobra, Yashiro, and Sidewinder 4F catheters utilizing as the diagnostic as well as the guiding catheters. After safe access is properly achieved, background-subtracted angiography is performed to identify the hemorrhagic blood vessels. It is essential to use guiding catheters for small blood vessels and super-selective catheters to avoid artery constriction. After identifying the bleeding arteries, microcatheters are used to access and occlude it using microcoils or glue (N-butyl cyanoacrylate and lipiodol). Using coils ensures quick and safer occlusion for proximal arterial branches, preventing bowel infarction but still being relatively effective. Cyanoacrylate glue typically facilitates rapid and effective occlusion, being relatively safe provided the operating physician is well-trained and being experienced.

Regarding the effectiveness, the transcatheter arterial embolization (TAE) might have high clinical success rates which were reported in the majority of studies. They can achieve the effective hemostasis in up to 98% of gastrointestinal bleeding [1, 2, 7, 9]. In a meta-analysis conducted by Loffroy et al, the technical success rate reached 93% [7], meanwhile in another study by Chun-Gao Zhou et al with 26 patients undergoing intravascular embolization, the technical success rate was 95% and the clinical success rate was 82% [1].

The recurrent bleeding which range from 9 to 47% [10] which could cause another emerging dilemmatic situation. For that, a consideration of another intervention should be carefully discussed and selected. One more disadvantage was that, if the patient was agitated and not well-cooperative, the patient's breathing or movement could cause degradation of the image during diagnostic and embolization processes. For that, general anesthesia with intermittent pauses could reduce the image artefacts.

Complication of the TEA are not common, one of the most concern complications are bowel ischemia which is, however, reported with a minimal incidence [9, 11]. They are related to the usage of penetrating embolic materials including polyvinyl particles, liquid N-butyl cyanoacrylate. Using coils as oen of the embolic agents seem to be safer but some time the coils could not reach to the bleeding sites. However, they are quite effective in big hemorrhagic vessels with good control of this embolic material placement.

In the above illustrated clinical case, the patient had undergone a major surgery with Whipple procedure, rapidly progressed to the hemorrhagic shock. In this difficult situation, the decision for a repeat of an operation was difficult to be made due to high risks of unsuccessfulness, being unable to identify the bleeding sites, worsening the patient status by exerting another burden to the preexisting injury. Similarly, gastroenteroscopy also was not preferable because the hemorrhage may extent to the peritoneal cavity whereby the endoscopy machine could not approach. After all, the choice of intravascular embolization was made with good results, timely saving the patient without complication and avoiding prolong hospitalization time. This case gives another illustration of the endovascular benefit outweighing the other modalities in the management of the acute gastrointestinal hemorrhage. This is also sharing the common points with other reported studies in the advantages of the TAE for the management of emergent gastrointestinal hemorrhage especially in those who were unable to control surgically or endoscopically.

CONCLUSION:

Postoperative gastrointestinal bleeding poses challenges in the selection of treatment options due to multiple risk factors and technical difficulties. With the

recent advancement of medical gadgets and the interventional techniques, arterial embolization has become the preferred modality of choice in such emergency dilema, maximizing the outcome effectiveness, minimizing excessive cost and possible complications. Beside the interventional embolization, multidisciplinary collaboration between critical care, internal medicine management, surgery, and radiological specialties is essential to offer the most beneficial treatment for the patients. However, with the diferences of the current reported results in effectiveness, bleeding recurrences and complications, further improvement of embolic materials, protocols, technical gagets are still needed to be considered.

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. Chun-Gao Zhou, Hai-Bin Shi, Sheng Liu, Zheng-Qiang Yang, Lin-Bo Zhao, Jin-Guo Xia, Wei-Zhong Zhou, Lin-Sun Li. Transarterial embolization for massive gastrointestinal hemorrhage following abdominal surgery. World J Gastroenterol, (2013). 19(40), pp 6869-6875.

2. Maciej Pech, Zbigniew Serafin, Frank Fischbach, Robert Damm, Tomasz Jargiełło, Max Seidensticker, Maciej Powerski. Transarterial embolization of acute iatrogenic hemorrhages: predictive factors for mortality and outcome. Br J Radiol. (2020) 93;(1110). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10993211/

3. Loffroy R, Favelier S, Pottecher P, Estivalet L, Genson P.Y, Gehin S, Cercueil J.P, Krausé D. Transcatheter arterial embolization for acute nonvariceal upper gastrointestinal bleeding: Indications, techniques and outcomes, Diagnostic and Interventional Imaging, (2015). 96(7–8), pp 731-744.

4. T. Christensen,1 L. Matsuoka,1 G. Heestand,1 S. Palmer,2 R. Mateo,1 Y. Genyk,1 et al. Iatrogenic Pseudoaneurysms of the Extrahepatic Arterial Vasculature: Management and Outcome. 2006. 8(6). Pg 458-464. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2020760/

5.TalM. Hörer, AnnaMaria Ierardi, Serena Carriero, Carolina Lanza, Gianpaolo Carrafiello, David T. McGreevy. Emergent vessel embolization for major traumatic and non-traumatic hemorrhage: Indications, tools and outcomes. Seminar in vascular Surg. (2023). Volume 36, Issue 02, Pg 283-299. https://www.sciencedirect.com/science/article/pii/S0895796723000297

6. Miller Jr M, Smith TP. Angiographic diagnosis and endovascular management of nonvariceal gastrointestinal hemorrhage. Gastroenterol Clin North Am, (2005). 34, pp 735-752.

7. Loffroy R, Rao P, Ota S, De Lin M, Kwak BK, Geschwind JF. Embolization of acute nonvariceal upper gastrointestinal hemorrhage resistant to endoscopic treatment: results and predictors of recurrent bleeding. Cardiovasc Intervent Radiol, (2010). 33, pp 1088- 1100.

8. Weida Wu, Jianyang Peng, Guohui Zhou, Chunsheng Zhang, Yuanzhen Lin, Zhicheng Weng. Efficacy of transcatheter arterial embolization in treating nonvariceal gastric remnant bleeding: a retrospective 5-year study. BMC Gastroenterology. (2024). 24, Article: 92.

https://bmcgastroenterol.biomedcentral.com/articles/10.1186/s12876-024-03179-x
9. Charbel Ishak, Haider Ghazanfar, Sameer Kandhi, Ahmed Alemam, Hafsa Abbas, Harish Patel et al. Role of Transcatheter Arterial Embolization in Acute Refractory Non-variceal Upper Gastrointestinal Bleeding Not Controlled by Endoscopy: A Single-Center Experience and a Literature Review. Cureus. (2022). 14(10). DOI: 10.7759/cureus.29962.
10. Shin JH Refractory gastrointestinal bleeding: role of angiographic intervention. Clin Endosc. 2013, 46:486-91. 10.5946/ce.2013.46.5.486.
11. Song SY, Chung JW, Yin YH, et al. Celiac axis and common hepatic artery variations in 5002 patients: systematic analysis with spiral CT and DSA. Radiology. 2010, 255:278-88. 10.1148/radiol.09090389

FIGURE LEGEND:

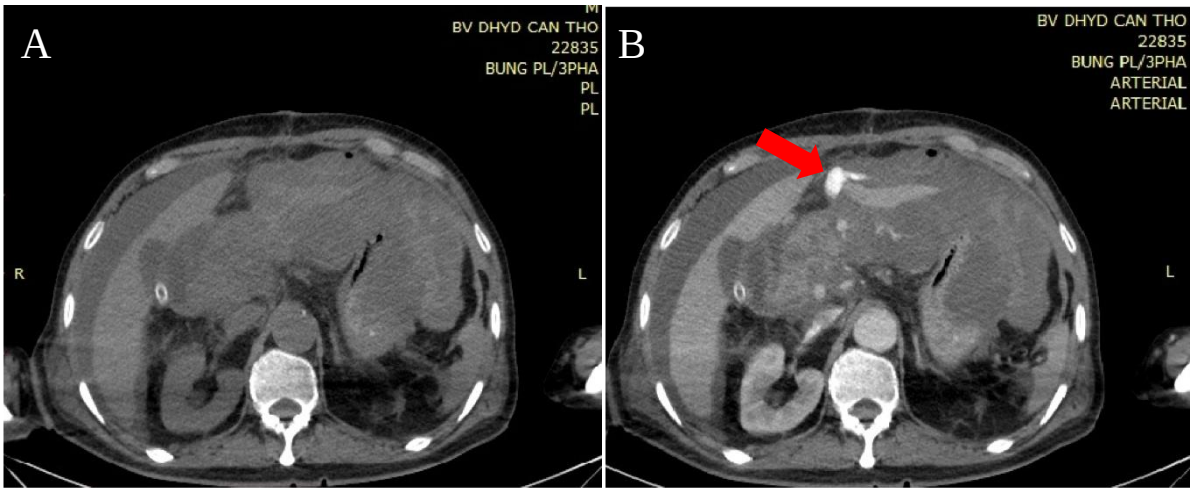

Figure 1: Plain abdominal CT image (A). CT angiography image, showing contrast extravasation, indicating active bleeding (Red arrow, B)

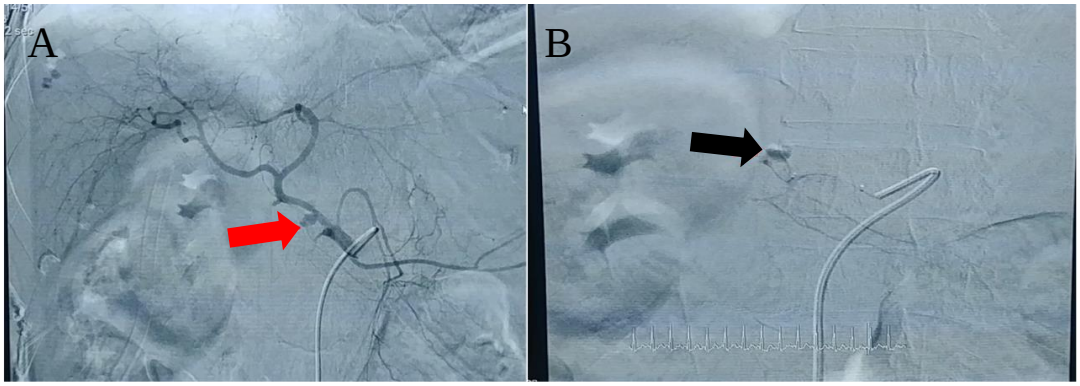

Figure 2: The digital subtraction angiography (DSA) revealed the presence of a pseudoaneurysm originating from a branch of the right hepatic artery (Red arrow, A). Selective angiogram of the bleeding artery with selective microcatheterization (Black arrow, B).

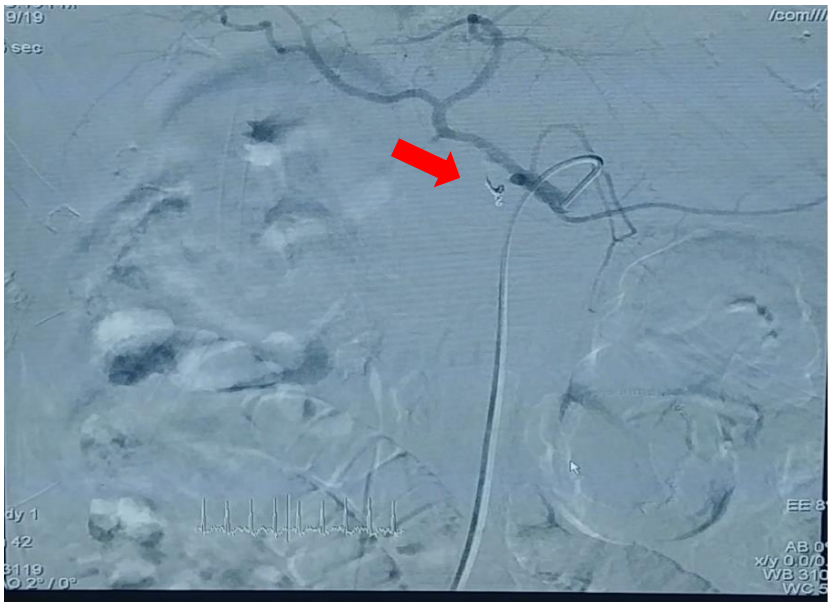

Figure 3: There was no contrast opacifying the pseudoaneurysm after coiling (Red arrow). The bleeding artery is completely occluded.