Case Report

DOI: http://dx.doi.org/10.18203/2349-2902.isj20174638

Delayed acute onset post traumatic sigmoid sinus/cerebral venous thrombosis

Bopaiah K. S., Shreykumar P. Shah*

Department of Neurosurgery, Apollo BGS Hospital, Mysore, Karnataka, India

Received: 20 September 2017 **Accepted:** 27 September 2017

*Correspondence: Dr. Shreykumar P. Shah, E-mail: shrey161@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Post traumatic Sinus/cerebral venous thrombosis is a rare entity. A high index of suspicion is required as the prognosis is poor in this clinical setting, in view of the effects of the head injury. We report 2 cases of the patient worsening acutely after initial recovery from trauma. The etiology and pathogenesis in such cases are varied and hypothetical with no definite underlying cause identified. Cerebral venous thrombosis (CVT) as an etiology of headache is not always easy to diagnose unless suspected, and in any patient with head injury and delayed deterioration after initial recovery, post traumatic Cerebral venous thrombosis should be kept as a differential diagnosis.

Keywords: Delayed acute onset, Post traumatic, Sinus thrombosis

INTRODUCTION

Cerebral Venous Thrombosis was first described in early part of 18th century by Ribes 1825 and Abercrombie 1828. The exact incidence is not known because of its rarity and non-specific presentation. The etiology is diverse with a hypercoagulable state or a direct cause being identifiable in about 85% of cases. In cases of Head Injury development of CVT may be overlooked due to other factors (difficulty in working up in an already unstable patient, non-specific presentation of CVT and may simulate Diffuse Axonal Injury).

Thrombosis occurring in the cerebral veins / sinuses leads to stagnation of blood and venous hypertension and secondary damage as a result of hypoxia/ischaemia. The mechanism of venous thrombosis in penetrating injuries is probably secondary to direct injury. Sinus dissection, sinus distortion and flow impediment by bone fragments can create venous flow obstructions, which in turn induce thrombosis. Intramural hemorrhage secondary to rupture

of sinusoids at the site of entry of draining veins, and extension of thrombus from injured scalp and emissary veins and release of thromboplastin is also believed to contribute to a locally procoagulant state, which may predispose to venous thrombosis.^{2,3}

The clinical presentation of venous thrombosis is sub-acute and clinical features depend on the involvement of the sinus or cortical veins. Sinus involvement usually presents with features of raised intracranial pressure whereas cortical vein thrombosis may present with focal neurological deficits or seizures.¹

In the clinical setting of trauma and the patient deteriorating rapidly other causes have to be ruled out and therefore radiology plays an important role in early diagnosis and management of CVT. On CT Imaging various abnormalities which may directly/indirectly imply the development of CVT may be seen. They include Dense triangle sign because of thrombosis in the sinus, Cerebral oedema (localized/generalized),

Hemorrhagic infarction / intracerebral hemorrhage, Cord sign – an irregular and high-density lesion located in the superficial aspect of the cerebral hemispheres which represents thrombosed cortical veins and Delta sign or empty triangle sign which is demonstrated on enhanced CT as a filling defect in the posterior part of the thrombosed superior sagittal sinus.

MRI is more sensitive in picking up the thrombus and is therefore the imaging modality of choice along with MR Venography in diagnosis of CVT.⁴ MRV can show the non-filling of thrombosed veins. However early in the course of the disease MRI may be non-diagnostic.

CT Venography is another technique for delineation of venous anatomy, and is equally sensitive for demonstrating thrombosis. A high index of suspicion is therefore required to diagnose it as imaging information may be non-diagnostic.⁵

The management guidelines of CVT in the setting of trauma have not been standardized unlike in the case of Non-traumatic CVT. In our case due to thrombosis and Intracranial contusions multiple approaches with antioedema measures, steroids, and unfractionated / fractionated heparin were used.

The mortality rate of sinus thrombosis ranges from 5.5 % -30% and out of those who survive 15.5%-25.5% are left with residual deficits. GCS <8 and presence of intracerebral hemorrhage have been shown to be significant and independent predictors of poor outcome in non-traumatic sinus thrombosis.⁶

Whether these observations can be applied to traumatic sinus thrombosis remains to be proven. Studies have also shown that most cases of the traumatic sinus thrombosis have been reported in patients with minor head injury, or in those who did not require surgical intervention for traumatic intracerebral hematoma.^{7,8}

CASE REPORT

Case 1

44 year old female was admitted following a Road Traffic Accident. On admission patient was conscious with a GCS 13/15(E3M6V4) with symmetrically reacting pupils. CT Imaging revealed left temporal hemorrhagic contusions with surrounding oedema. She was managed with antioedema measures (Mannitol), analgesics, antibiotics and IV Fluids.

Patient's condition improved, however on the 3rd day following the incident she developed severe headache and disorientation. Her GCS was 12/15 (E2M6V4). Repeat CT brain revealed hemorrhagic contusions in left tempero-parietal region with peri-lesional oedema and mass effect. Due to persistent headache an MRI with MR Venogram was done which revealed large filling defect in

left transverse and sigmoid sinus and jugular vein suggestive of thrombosis.

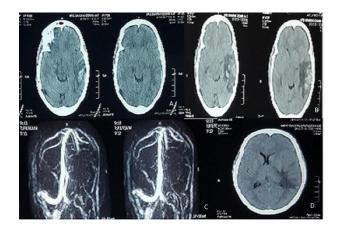


Figure 1: A) CT Brain on the day of admission showing left temporal hemorrhagic contusions with surrounding edema. B) CT brain on post admission day 2 showing. C) MR Venogram (on post admission day 4) showing complete occlusion of left sigmoid and transverse sinus. D) CT Brain on day of discharge showing.

She was started on anti- coagulative therapy with low molecular weight heparin. Dose adjusted with regular PT/INR monitoring. She gradually improved symptomatically and was discharged on oral anti-coagulants.

Case 2

36 year old male was admitted following a Road Traffic Accident. On admission patient was semi-conscious with a GCS 9/15(E2M5V2) with symmetrically reacting pupils. CT Imaging revealed Right Fronto-Temporal thin SDH, Bilateral basifrontal contusions, fracture occipital bone, Diffuse subarachnoid haemorrhage and cerebral oedema. He was managed with ventilator support and antioedema measures (Mannitol/Glycerol/3% saline). Serum osmolarity was closely monitored and maintained below 320mmol/lit.

Patients condition gradually improved over a period of one week to GCS 14/15 (E4M6V4). On the 10th day following the incident he developed sudden onset motor aphasia and progressively deteriorated over a period of 24 hrs to GCS-4/15(E1M2V1) with symmetrical reacting pupils.

He was put on ventilatory support and was worked up for seizures, metabolic abnormalities, meningitis, sepsis and delayed bleed during this period.

All the above-mentioned investigations were negative and an MRI with MR Venogram was done which revealed Complete Superior Sagittal Sinus (up to torcula) and cortical vein thrombosis.

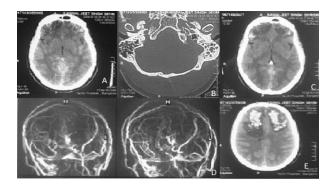


Figure 2: A) CT Brain 12 hrs post trauma showing diffuse sub arachnoid haemorrhage and cerebral oedema. B) CT brain on the of admission showing fracture occipital bone. C) CT brain 36 hrs post trauma. D) MR Venogram (on post admission day 11) showing complete occlusion of superior sagittal sinus. E) CT Brain on 11th day post trauma.

Repeat CT imaging done prior to starting thrombolytic therapy revealed Bifrontal ICH. He underwent Emergency Decompressive surgery but his condition deteriorated further.

DISCUSSION

Cerebral venous thrombosis (CVT) as an etiology of headache is not always easy to diagnose unless suspected. The clinical manifestations are non-specific, and overall, it is still not a common diagnosis. Sinus thrombosis is not classically thought of as being associated with closed head injury. Rather, the classic aetiologies are neoplasm's or hypercoagulable states such as pregnancy.

This condition should be suspected in a patient with head injury who does not improve, has plateauing of neurological recovery, develops seizures or fresh neurological deficits, has persistently raised ICP with papilledema, intractable fever with leukocytosis8 or as in our case where in there was delayed deterioration after initial recovery. Aggressive management with antioedema measures may contribute to a hypercoaguable state and predispose to development of CVT indirectly in a small group of patients.

ACKNOWLEDGEMENTS

Authors would like to thank the support extended by Apollo BGS Hospital, study subjects and their family members.

Funding: No funding sources Conflict of interest: None declared Ethical approval: Not required

REFERENCES

- Stam J. Thrombosis of the cerebral veins and sinuses. N Eng J Med. 2005;352:1791-8.
- 2. Meena AK, Jayalaxmi SS, Murthy JM. Cortical sinovenous thrombosis following closed head injury. Neurol India. 2000;48:195-6.
- 3. Pathak A, Dutta S, Marwaha N, Singh D, Varma N, Mathuriya SN. Change in tissue thromboplastin content of brain following trauma. Neurol India. 2005;53:178-82.
- 4. Lee S, Brugge K G. Cerebral venous thrombosis in adults: The role of imaging evaluation and management. Neuroimag Clin N Am 2003;13:139-52.
- 5. Moiyadi AV, Devi BI. Post-traumatic non-sinus cerebral venous thrombosis. Indian Journal of Neurotrauma. 2006;3(2):143-6.
- de Bruijn SF, de Haan RJ, Stam J. Clinical features and prognostic factors of cerebral venous sinus thrombosis in a prospective series of 59 patients. J Neurol Neurosurg Psychiat. 2001;70:105-8.
- 7. Kumar GSS, Chacko AG, Joseph M. Superior sagittal sinus and torcula thrombosis in minor head injury. Neurol India. 2004;52:123-4.
- 8. D'Alise MD, Frank FF, Horowitz. Sagittal sinus thrombosis following minor head injury treated with continuous urokinase infusion. Surg Neurol. 1998:49:430-5.
- 9. Provenzale JM, Kranz PG. Dural sinus thrombosis: sources of error in image interpretation. AJR Am J Roentgenol. 2011;196(1):23-31.
- 10. Wiggins AC. Transverse and sigmoid sinus thrombosis after traumatic brain injury. IJCRI. 2013;4(11):660-2.

Cite this article as: Bopaiah KS, Shah SP. Delayed acute onset post traumatic sigmoid sinus/cerebral venous thrombosis. Int Surg J 2017;4:3765-7.