

Positive Outcome in Traumatic Brain Injury with Haematoma and Fracture: A Case Report

ANJALI KHOTLE<sup>1</sup>, AKHILESH SINGH PARIHAR<sup>2</sup>, IBRAHIM BOMBAYWALA<sup>3</sup>, CHARUTA GADKARI<sup>4</sup>

(CC) BY-NC-ND

# **ABSTRACT**

Traumatic Brain Injury (TBI) is described as the break in natural functioning of the brain caused due to any kind of trauma to the head. The injury to head can cause distinctive types of haemorrhages namely, Epidural Haematoma (EDH), subdural haematoma (SDH), Subarachnoid Haematoma (SAH) and Intra-Parenchymal Haematoma (IPH), SAH and SDH being the more common ones. Acute subdural haematomas often occur in patients suffering from falls and motor vehicle crashes, resulting in compression, and swelling of the brain that increases the intracranial pressure. When both compression and swelling occur, mortality and morbidity can be high. In the following case, the patient presented with both intracranial as well as extracranial haematomas in moderation and got cured during a week of hospital stay with almost minor functional and neurological disability as indicated by the Glasgow Coma Scale (GCS) and other variables. As per current evidence the best way to minimise long-term disability in moderate and severe TBI is extensive multidisciplinary post TBI rehabilitation.

Keywords: Epidural haematoma, Glasgow coma scale, Subarachnoid haematoma, Subdural haematoma

## **CASE REPORT**

A 24-years-old patient with alleged history of fall from bike 24 hours ago, sustaining injury to the head was brought to emergency medicine department. Initially, he was taken to a local hospital where his GCS was 11 (Eye response was 2; Verbal response was 4; Motor response was 5) from where he was referred to the hospital for further management. The severity of his TBI was classified as 'moderate'.

He presented with history of bleed from right ear along with nasal and oral bleed. There was history of projectile vomiting along with confusion after fall. There was no history of loss of consciousness, seizures or any comorbidities.

On examination: Airway was patent but not protected as the patient was drowsy. C spine stabilisation was done along with Philadelphia (hard) collar. Breathing rate was 26 breaths/min, SpO2- was 90% on room air, hence patient was started on four litre of oxygen through Hudson mask. Pulse was 78 beats/min and BP was 90/60 mmHg. Two large bore i.v. lines were secured, and i.v. crystalloid were started. The GCS of the patient came down to 8 (E2; V3; M5) after he presented in the hospital. The patient was conscious but disoriented to time place and person. Neurological examination of the patient was done which is as shown in the table [Table/Fig-1].

|   | Right                        | Left                         |
|---|------------------------------|------------------------------|
| Pupils  | Sluggishly reactive to light | Sluggishly reactive to light |
| Power: Upper limbs                                    | 3/5                          | 5/5                          |
| Power: Lower limbs                                    | 3/5                          | 5/5                          |
| Tone  | Flaccid                      | Flaccid                      |
| Deep tendon reflexes                                  | -1                           | 0                            |
| Plantar reflex  | Extensor                     | Flexor                       |
| [Table/Fig-1]: Representing neurological examination. |                              |                              |

During chest auscultation bilateral air entry was found to be equal as well as S1, S2 was heard normally with no murmurs. On palpation abdomen was soft and non tender. Local examination revealed a lacerated wound of size 7×1 cm over right temporoparietal region, an abrasion over left dorsal lumbar region of size 4×2 cm, an abrasion over right dorsal side of elbow of size 3×1 cm, and an

abrasion over right thigh of size 2×2 cm. Pelvic compression and chest compression tests were found to be negative.

Patient was advised Non Contrast Computed Tomography (NCCT) brain plain, chest X-ray, along with an ENT call for nasal, oral, and ear bleed, along with other routine investigations. He was admitted under neurosurgery ICU in view of extensive head trauma.

All routine investigations were within normal limits. ECG was done and was normal. NCCT brain plain was suggestive of haemorrhagic contusions in the right temporal lobe [Table/Fig-2], extradural haemorrhage in the right temporoparietal region [Table/Fig-2,3], subdural haemorrhage in the left temporal region along with haemosinus [Table/Fig-3], minimal subarachnoid haemorrhage along the sulcal spaces of left frontal, left temporoparietal lobes and left sylvian fissure [Table/Fig-3], linear undisplaced fracture of the right parietal bone with extension upto the right temporal bone [Table/ Fig-4]. Haemomastoideum and haemotympanicum were also present.



[Table/Fig-2]: Representing EDH and Haemosinus.



[Table/Fig-3]: Representing EDH, SDH and SAH.



Acutely given treatment was as follows: Inj. Hydrocortisone 100 mg i.v. stat, Inj. Mannitol 100 mL i.v. stat was administered as antioedema measure, Inj. Levetiracetam 1 gm i.v. stat was given as anti-convulsant. Inj. Paracetamol 100 mL i.v. stat and Inj. Diclofenac 75 mg deep Intramuscular (IM) stat were administered to provide analgesia. Inj. Pantoprazole 40 mg i.v. stat, Inj. Ondansetron 4 mg i.v. stat, Inj. Tetanus toxoid 0.5 mg IM stat were also administered. Patient was kept nil by mouth till further orders. After stabilisation he was admitted under neurosurgery-intensive care and the same treatment was continued along with addition of Inj. Ceftriaxone 1 gm i.v. twice daily, Inj. B-complex 1 AMP i.v. once daily.

The patient's GCS improved to 12 (E3;V3;M6) and also neurological improvement; hence he was shifted from ICU to ward in a course of 5 days and was started on oral medications like Tab. Methylprednisolone in tapering dose, Tab. Ceftriaxone 500 mg twice daily, Tab. Furosemide 20 mg twice daily, Tab. Pantoprazole 40 mg once daily, Tab. Ondansetron 4 mg thrice daily, Tab. Paracetamol 650 mg thrice daily. The patient showed progressive improvement and was discharged after 7 days with a GCS of 15 (E4;V5;M6). Patient was advised to follow-up after eight days. He reported to neurosurgery outpatient set-up and was showing good neurological recovery.

## DISCUSSION

The patient presented with EDH, SDH as well as minimal SAH to the emergency room. His GCS on arrival was eight along with some degree of neurological deficit. Patient was classified as a case of moderate TBI and was managed conservatively. During his seven days hospital stay patient showed progressive improvement and was discharged with a GCS of 15. He was advised rehabilitation therapy for muscle strengthening and power loss.

TBI is described as any interruption in natural brain functioning, or other brain related pathologies, caused due to trauma to the head. Approximately 50 million cases are reported worldwide on a yearly basis [1]. TBI remains the most prevalent reason of morbidity and mortality in individuals younger than 40 years [2]. In all age groups, TBI accounts for 30 to 40 % of all the trauma related deaths and neurological injuries. TBI is expected to be the most important cause of disability due to neurological diseases (2-3 times higher than that for Alzheimer disease or cerebrovascular disorders) till 2030. The epidemiology of TBI is different in different countries such as, in high-income countries the most commonly affected individuals are elderly, mainly due to fall, but in the low- and middleincome countries the affected individuals are mainly middle aged (to be specific 20-40 years of age) and the cause is mainly road traffic accidents [3], as it was in this case. Males are more prone to TBI as compared to females. There is a trimodal distribution of age, with spikes at 0 to 4, 15 to 24, and >75 years of age. As the age at time of injury increases so does the chances of mortality increases [4].

Being a diverse entity, TBI reflects various perceptible forms of injury (such as, extrinsic compression from mass lesion, contusion,

Diffuse Axonal Injury (DAI)} which in combination with a range of mechanisms {such as, ischaemia, apoptosis, mitochondrial dysfunction, Cortical Spreading Depression (CSD), and microvascular thrombosis} inflicts neuronal injury in differing proportions which results in different clinical outcomes [2,5,6].

On the basis of GCS, TBI can be categorised into three categories. Mild TBI that is GCS 14 to 15, also known as "concussion" accounts for approximately 80% of head injuries. The label of mild however is a misnomer. It can result in serious, exhausting short- and long-term sequelae. Moderate TBI (mTBI) that is GCS 9 to 13 accounts for roughly 10% of all head injuries. Although the mortality rate of mTBI is <20%, however long-term disability can be greater. There was abnormal CT findings in over 40% of the patients suffering from mTBI from which 8% might need a neurosurgical intervention. Patients corresponding to a GCS of three to eight have a mortality rate of 40% and are classified under severe TBI. The first 48 hours after the injury are the most crucial ones with most deaths occurring in that period of time. In cases of severe TBI, good recovery can be expected in less than 10% of patients [4].

According to studies, cases have been reported either with extracranial bleed or intracranial bleed or with both bleeds. Amongst which the most commonly occurred type was SDH, present in 30% of the patients. EDH, IPH and SAH were seen amongst 22% patients each [7,8]. Moreover, to the best of our knowledge the literature search revealed that the incidences of all the haematomas occurring together are fewer as compared to cases presenting with a single haematoma.

In recent times, TBI is gaining more attention, reason being the complications both acute and chronic that lead to permanent disabilities resulting in decreased life expectancy [9]. According to studies, every year in India an approximate of 1.5 to 2 million persons suffer from TBI, of which approximately 1 million succumb to death. The leading cause of TBI is road traffic injuries (60%) followed by falls (20%-25%) and violence (10%). Alcohol intoxication is present amongst 15%-20% of TBI patients at the time of trauma [10,11]. In cases with both extracranial and intracranial bleeding the prognosis of the patient is usually very poor but, in this case the patient survived and showed good clinical as well as neurological improvement.

In TBI the most common tool for assessing degree of brain injury and the prognosis of patient is a detailed clinical examination and GCS [12,13]. During the hospital stay which was almost seven days, the patient's neurological condition improved gradually and the GCS improved from 8 (E2;V3;M5) to 15 (E4;V5;M6).

The most common investigative modalities use to diagnose TBI are Computed tomography (CT) scan and Magnetic Resonance Imaging (MRI). CT scan can best visualise fractures and reveal indication of brain haemorrhage, haematomas, contused brain tissue, and brain tissue oedema. If the person's health has stabilised or if the CT findings are ambiguous, an MRI may be employed [14,15].

One of the primordial aims of medical management in EMD is to prevent any secondary injury to the brain while providing the optimum conditions for recovery from any reversible deficits. This is done in accordance with the protocol provided by brain trauma foundation. This entails establishing and ensuring clean and clear airway with sufficient oxygenation and delivering adequate replacement fluid to provide appropriate peripheral circulation and blood volume along with proper anti-oedema measures [16].

Treatment for moderate or severe TBI mainly includes surgical treatment which may include decompressive craniectomy. But some cases of moderate TBI can also be treated conservatively. Medical management of moderate TBI includes corticosteroids, tranexamic acid, anti-inflammatory drugs, etc. Corticosteroids remain the first line of management [4,17]. As in this case, hydrocortisone was used as the first line of treatment, and patient was managed conservatively.

# **CONCLUSION(S)**

TBI is linked to both short-term and long-term disabilities, being a major issue for public health and rehabilitation specialists. To summarise, the patient presented with a poor functional and cognitive state and has shown a progressive recovery overall. But not every moderate TBI is associated with better prognosis and recovery many cases can result in clinical deterioration leading to fatal outcome. With proper assessment and multidisciplinary management, recovery is good and usually results in a successful outcome.

## REFERENCES

- [1] Faul M, Wald MM, Xu L, Coronado VG. Traumatic brain injury in the United States: Emergency department visits, hospitalizations, and deaths, 2002-2006 [Internet]. 2010 [cited 2023 Apr 15]. Available from: https://stacks.cdc.gov/view/ cdc/5571
- [2] Khellaf A, Khan DZ, Helmy A. Recent advances in traumatic brain injury. J Neurol, 2019:266(11):2878-89. Available from: https://pubmed.ncbi.nlm.nih. gov/31563989/.
- [3] Maas AIR, Menon DK, David Adelson PD, Andelic N, Bell MJ, Belli A, et al. Traumatic brain injury: Integrated approaches to improve prevention, clinical care, and research. Lancet Neurol. 2017;16(12):987-1048. Available from: https://pubmed.ncbi.nlm.nih.gov/29122524/.
- [4] Wright DW, Merck LH, Head Trauma. Tintinalli's Emergency Medicine: A Comprehensive Study Guide. 9th ed. New York, NY: McGraw-Hill Education; 2020
- Schwarzmaier SM, De Chaumont C, Balbi M, Terpolilli NA, Kleinschnitz C, [5] Gruber A, et al. The formation of microthrombi in parenchymal microvessels after traumatic brain injury is independent of coagulation factor XI. J Neurotrauma. 2016;33(17):1634-44. Available from: /pmc/articles/PMC5011628/.
- Kuru Bektaşoğlu P, Koyuncuoğlu T, Akbulut S, Akakın D, Eyüboğlu İP, Erzik C, et al. Neuroprotective effect of plasminogen activator inhibitor-1 antagonist in the rat model of mild traumatic brain injury. Inflammation. 2021;44(6):2499-17.

- [7] Tong WS, Zheng P, Zeng JS, Guo YJ, Yang WJ, Li GY, et al. Prognosis analysis and risk factors related to progressive intracranial haemorrhage in patients with acute traumatic brain injury. Brain Inj. 2012;26(9):1136-42. Doi: 10.3109/02699052.2012.666437.
- Perel P, Roberts I, Bouamra O, Woodford M, Mooney J, Lecky F. Intracranial [8] bleeding in patients with traumatic brain injury: A prognostic study. BMC Emerg Med. 2009;9(1):01-08. Available from: https://bmcemergmed.biomedcentral. com/articles/10.1186/1471-227X-9-15.
- Huie JR, Nielson JL, Wolfsbane J, Andersen CR, Spratt HM, DeWitt DS, et al. [9] Data-driven approach to integrating genomic and behavioral preclinical traumatic brain injury research. Front Bioeng Biotechnol [Internet]. 2023 Jan 10 [cited 2023 Apr 15];10. Available from: https://pubmed.ncbi.nlm.nih.gov/36704298/.
- [10] Goldman L, Siddiqui EM, Khan A, Jahan S, Rehman MU, Mehan S, et al. Understanding acquired brain injury: A review. Biomedicines. 2022;10(9):2167. Available from: https://www.mdpi.com/2227-9059/10/9/2167/html.
- [11] Gururaj G. Epidemiology of traumatic brain injuries: Indian scenario. Neurol Res. 2002;24(1):24-8. Doi: 10.1179/016164102101199503. Available from: https:// pubmed.ncbi.nlm.nih.gov/11783750/.
- [12] Al-Hassani A, Strandvik G, El-Menyar A, Dhumale A, Asim M, Ajaj A, et al. Functional outcomes in moderate-to-severe traumatic brain injury survivors. J Emerg Trauma Shock. 2018;11(3):197-04. Available from: /pmc/articles/ PMC6182963/.
- [13] Hartmann A, Kegelmeyer D, Kloos A. Use of an errorless learning approach in a person with concomitant traumatic spinal cord injury and brain injury: A case report. J Neurol Phys Ther. 2018;42(2):102-09. Available from: https://pubmed. ncbi.nlm.nih.gov/29547485/.
- [14] Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GWJ, Bell MJ, et al. Guidelines for the management of severe traumatic brain injury, fourth edition. Neurosurgery. 2017;80(1):06-15. Available from: https://pubmed.ncbi.nlm.nih. gov/27654000/
- [15] Mendez MF. What is the relationship of traumatic brain injury to dementia? J Alsheimers Dis. 2017;57(3):667-81. Available from: https://pubmed.ncbi.nlm. nih.gov/28269777/.
- [16] Dash HH, Chavali S. Management of traumatic brain injury patients. Korean J Anesthesiol. 2018;71(1):12. Available from: /pmc/articles/PMC5809702/.
- Papa L, Goldberg SA. Head Trauma- Rosen's Emergency Medicine: Concepts [17] and Clinical Practice, 10th ed, Elsevier, 2022.

### PARTICULARS OF CONTRIBUTORS:

- Intern, Department of Emergency Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Sawangi (Meghe), Maharashtra, India. Senior Resident, Department of Emergency Medicine, Acharya Vinoba Bhave Rural Hospital, Datta Meghe Institute of Higher Education and Research, Sawangi (Meghe), 2. Maharashtra, India
- 3. Intern, Department of Emergency Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Sawangi (Meghe), Maharashtra, India.
- Professor, Department of Emergency Medicine, Acharya Vinoba Bhave Rural Hospital, Datta Meghe Institute of Higher Education and Research, Sawangi (Meghe), 4.
- Maharashtra, India.

#### NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR: Anjali Khotle

Intern, Department of Emergency Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Sawangi (Meghe), Maharashtra, India.

E-mail: anjalikhotle1998@gmail.com.

#### AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- · Was informed consent obtained from the subjects involved in the study? No
- For any images presented appropriate consent has been obtained from the subjects. No

### PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Mar 04, 2023
- Manual Googling: Apr 28, 2023
- iThenticate Software: May 01, 2023 (8%)

EMENDATIONS: 6

Date of Submission: Feb 23, 2023 Date of Peer Review: Apr 12, 2023 Date of Acceptance: May 02, 2023 Date of Publishing: Jul 01, 2023

ETYMOLOGY: Author Origin