

Excellent Outcome Following Very Severe Traumatic Brain Injury with Bilateral Acute Traumatic Intracranial Hematomas: A Case Report

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Abstract

Traumatic Brain Injury (TBI) is a significant global burden in terms of mortality and disability. The Glasgow Coma Scale (GCS) is used to determine the severity of TBI, categorizing cases as mild (GCS 13-15), moderate (GCS 8-12), or severe (GCS 3-7). Good recovery from very severe TBI (GCS 3-5) is exceedingly rare. Here, we present a case exhibiting an excellent outcome following a very severe TBI. A 46-year-old man experienced severe head trauma after falling. On admission CT scan revealed a left temporal epidural hematoma, accompanied by a contralateral right subdural hematoma with contusional hemorrhage. The patient's consciousness rapidly declined to 4, leading to surgery for the evacuation of bilateral hematomas via bilateral craniotomies, along with the removal of the right temporal bone. Post-surgery, the patient received comprehensive management to address brain edema, transient hypotension, hyperthermia, hyperglycemia, respiratory dysfunction, liver dysfunction, and infection. Gradual improvement in the patient's GCS score from 4 to 15 was observed, accompanied by the resolution of general conditions, brain edema, and left hemiparesis. The ophthalmological examination revealed bilateral upper left quarteranopsia. By day 140 after the injury, the patient had completely returned to work. Despite the high mortality and morbidity with very severe TBI featuring bilateral intracranial hematomas, appropriate surgical intervention and systemic management can rescue patients without neurological deterioration.

Keywords: Very severe traumatic brain injury; Excellent outcome; Bilateral intracranial hematoma.

Introduction

Traumatic Brain Injury (TBI) is a significant global burden in terms of mortality and disability. TBI is a significant global burden in terms of mortality and disability. TBI is a type of brain injury caused by motor vehicle accidents, falls, violence, sports activities, and other factors. TBIs can be classified into different types, including skull fractures, cerebral concussions, subarachnoid

hemorrhages, subdural hematoma, epidural hematoma, and cerebral contusion. The severity of TBI is often determined using the Glasgow Coma Scale (GCS), which classifies TBIs as mild (GCS 13-15), moderate (GCS 8-12), or severe (GCS 3-7) [1]. Furthermore, very severe TBI is defined as GCS 3-5. [2]. The all TBI cases are mostly classified as mild (GCS 13-15), while approximately 0.6% are severe TBI (GCS 3-7) and 0.3 % are very severe TBI [2]. Regarding recovery outcomes, approximately 8% of severe TBI cases

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achieve good recovery (Glasgow Outcome Scale, GOS 5), while good recovery from very severe TBI (GCS 3-5) is rare [3]. Furthermore, good recovery from severe TBI with bilateral traumatic hematomas ranges from 0 to 3% [4], while good recovery from very severe TBI with bilateral intracranial hematomas is extremely rare.

Optimal treatment for bilateral traumatic intracranial hematomas remain largely unknown. When it comes to surgical treatment for bilateral traumatic intracranial hematomas, there is still controversy regarding the most appropriate approach. The options under debate include bilateral craniectomies, bilateral craniotomies, unilateral craniectomy, or unilateral craniotomy [5,6]. Furthermore, there is a scarcity of detailed reports on the course of very severe TBI cases with excellent outcomes. In this study, we present a case of a very severe TBI with an excellent outcome following evacuation and craniectomy for acute epidural hematoma and contra-lateral subdural hematoma associated with intracerebral contusional hemorrhage. Subsequently, we discuss the optimal treatment options for similar cases.

Case presentation

A 46-year-old man experienced a fall from a height of 2 meters while at work, resulting in a blow to his left temporal head. He was promptly transported to our hospital via emergency car. A CT scan revealed the presence of a left temporal epidural hematoma accompanied by a temporal bone fracture, as well as a contra-lateral acute subdural hematoma and contusional hemorrhage in the temporal and frontal lobes. Upon admission, his level of consciousness was assessed using the Glasgow Coma Scale (GCS) and scored 13 points. However, within two hours, his consciousness rapidly declined to a GCS score of 4 points. Bilateral light reflexes became significantly diminished and dilated, with the pupils exhibiting dilation and slight anisocoria (right>left). A follow-up CT scan indicated the enlargement of bilateral hematomas, prompting the decision to proceed with emergency surgery. The initial step involved performing a right temporal craniectomy, during which a remarkably thin right temporal bone with a thickness of 1.8 mm was removed. Subsequently, durotomy was conducted, and subdural and subcortical hematomas were evacuated to the maximum extent possible. Following hematoma evacuation, a Gore-Tex seal was placed on the brain surface without dura mater plasty, while the removed skull bone was positioned beneath the abdominal subcutaneous tissues. In the subsequent stage, a left temporal craniotomy was performed to fully evacuate the epidural hematoma. The left temporal bone was also found to be exceptionally thin, measuring 1.8 mm in thickness. Following the operation, upon the patient's return to the intensive care unit, the pupils exhibited isocoria and the light reflexes were prompt. The GCS score improved to 8. postoperative CT scans confirmed decompression on both sides of the brain. The episodic hypotension (<90 mmHg) was recognized and dopamine was administered (Figure 1). Although left brain swelling resolved after the surgery, right brain swelling persisted, and the patient's GCS remained at a low level between 6-8 points. On day 3 after the injury, the patient developed left hemiparesis in the upper and lower limbs. CT scans indicated an increase in right brain swelling. The patient's body temperature exceeded 39°C, and acetaminophen was administered to reduce the fever (Figure 2). On day 4 after the injury, the patient experienced acute respiratory distress due to airway narrowing. Oxygen saturation in the blood dropped below 85%,

and arterial blood gas analysis revealed decreased O₂ pressure and increased CO₂ pressure. Subsequently, trans-oral tracheal intubation was performed with spontaneous respiration. However, hyper-ventilation exceeding 25 breaths per minute and tachycardia over 120 beats per minute persisted. Oxygen saturation in the blood occasionally decreased. Consequently, on day 6 after the injury, the patient was transitioned to artificial ventilation using an artificial ventilator with synchronized intermittent mandatory ventilation (SIMV) mode. This intervention improved the patient's respiratory status, with blood oxygen saturation exceeding 98%. The pupils exhibited isocoria, and the light reflex became prompt (Figure 3). However, the level of consciousness remained at a low level between GCS 8 and 9. Intravenous infusion of glycerol at a rate of 800 mL/day, prednisolone at a dose of 20 mg/day, and flocemide at a dose of 20 mg/day were continued. On day 9 after the injury, the ventilation mode was changed from SIMV mode to continuous positive airway pressure (CPAP) mode. On day 11 after the injury, the tracheal tube was removed, and oxygen was supplied at a rate of 3 L/minute via a facial mask. The patient maintained a blood oxygen saturation level of over 96%. The patient responded to commands, opened his eyes, and moved his hands and legs, albeit with left hemiparesis (MMT: upper limb 3/5, lower limb 4/5).

From day 12 to 14 after the injury, the patient's condition remained relatively stable. The level of consciousness ranged between GCS 10-11, and left motor weakness persisted (MMT: upper limb 3/5, lower limb 4/5). On day 15 after the injury, the level of consciousness improved to GCS 12, and the left motor weakness showed improvement (MMT: upper limb 4/5, lower limb 4/5) (Figure 4). Oxygen supply was discontinued as blood oxygen saturation reached 98%. The patient began receiving soft foods due to mild swallowing difficulties. On day 18 after the injury, the level of consciousness further improved to GCS 13. On day 21 after the injury, the level of consciousness reached GCS 14, and left hemiparesis improved to MMT (upper limb 4/5, lower limb 4/5). The patient was able to stand and walk with the support of parallel bars. A follow-up head CT scan indicated a reduction in swelling in the right hemisphere and the disappearance of the intracerebral hematoma. On day 24 after the injury, the level of consciousness reached GCS 15, and the left motor weakness disappeared (Figure 4). On day 28 after the injury, the patient contracted COVID-19 and was quarantined in the hospital for 10 days. After the COVID-19 quarantine period, the patient continued to experience further improvement in higher brain function and limb functions. On day 41 after the injury, an electroencephalography showed occasional paroxysmal discharges in the right temporal lobe. Another head CT scan demonstrated further reduction in swelling in the right hemisphere and slight atrophy of that hemisphere (Figure 5). At this stage, the patient had fully recovered but required rehabilitation to address disuse-related issues. On day 63 after the injury, the patient underwent cranioplasty using his own temporal bone, which was buried under the abdominal skin. By 70 days after the injury, mild recent memory disturbance and attention disorder were observed in the patient's higher brain functions. On day 76 after the injury, the patient was discharged home. On day 132 after hospital discharge, the patient underwent an ophthalmological examination due to visual disturbances, which revealed bilateral upper left quarter anopsia (Figure 6). By day 140 after the injury, the patient had completely returned to work.

Written informed consent for publication of the research details and clinical images were obtained from the patient. The patient provided informed consent after the study. This study was approved by the Institutional Review Board of Asahi Hospital (No. 2023-1).

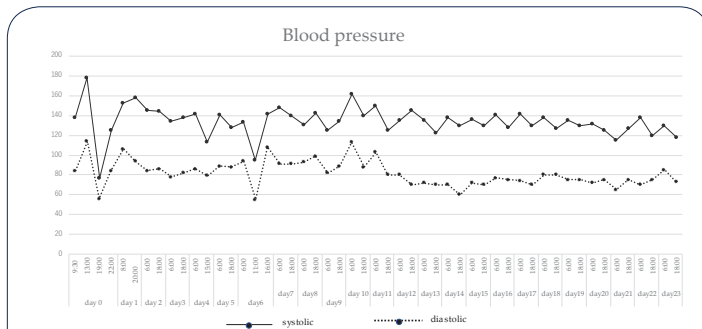


Figure 1: Image showing retrograde pyelography. Ureter appeared to be dilated with? vaju.

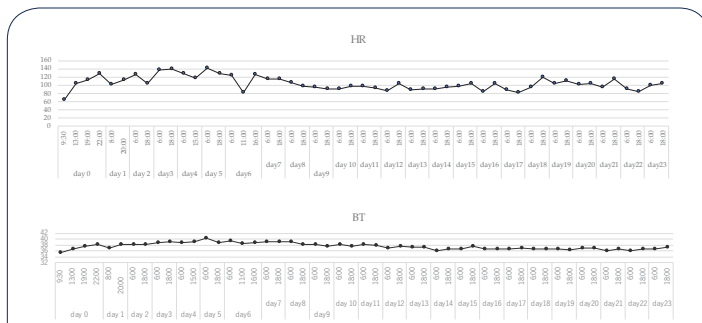


Figure 2: Change of blood pressure during day 0 to 23 following hospital admission. injury. Systolic: systolic blood pressure. Diastolic: diastolic blood pressure.

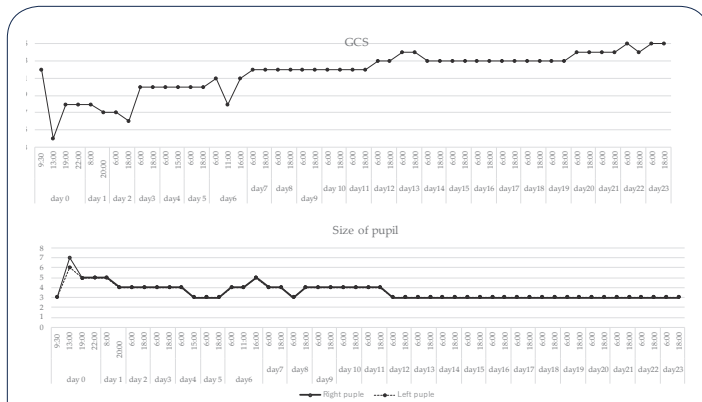


Figure 3: Changes of GCS and size of pupil during day 0 to 23 following hospital admission. GCS: Glasgow Coma Scale.

Discussion

Traumatic acute extra-axial hematomas, including acute epidural hematomas (AEDHs) and acute subdural hematomas (ASDHs), often occur in conjunction with intracerebral contusional hemorrhage. AEDHs typically result from a direct blow to the temporal region, occasionally causing a skull fracture and subsequent rupture of the middle meningeal artery. These hematomas rapidly

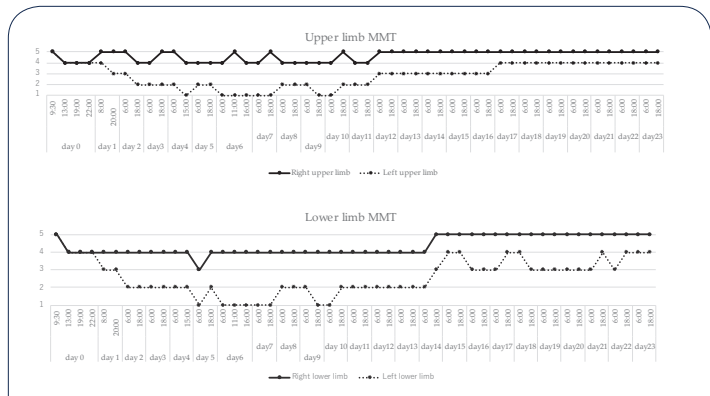


Figure 4: Changes of MMT of upper limbs and lower limbs. MMT: manual muscle test.

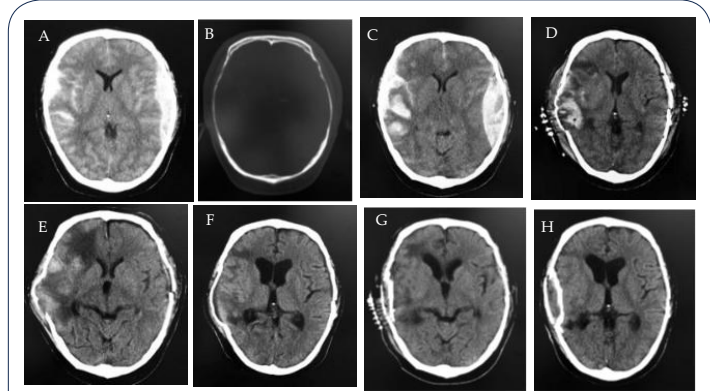


Figure 5: Head CT images from day 0 to 75 following hospital admission. A: CT immediately after hospital admission. B: Bone window of CT immediately after hospital admission showing very thin bilateral temporal bones. C: CT before operation at 3 hours following hospital admission. D: CT on day 1 following hospital admission. E: CT on day 10 following hospital admission F: CT on day 40 following hospital admission. G: CT after cranioplasty on day 60 following hospital admission. H: CT on day 75 following hospital admission.

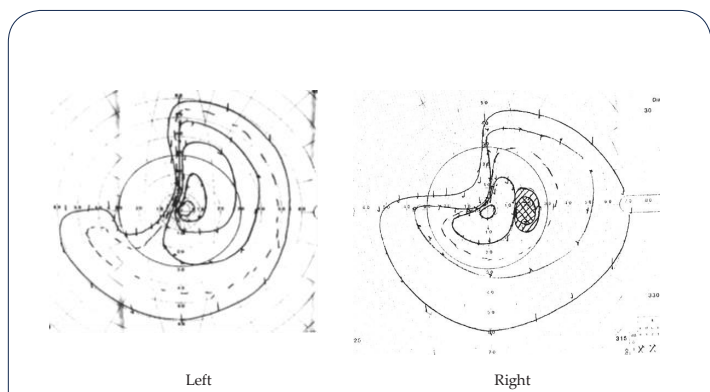


Figure 6: Visual fields showing left quadrant hemianopsia. Left: visual field of left eye. Right: visual field of right eye.

increase in size and can lead to herniation syndromes once a critical intracranial pressure threshold is reached [7]. In some cases, AEDHs can cause contralateral subdural hematomas and contusions, as observed in the present case. On the other hand, ASDHs commonly result from deceleration forces that cause the brain's surface to shear against the undersurface of the skull, leading to injury of the bridging veins [8].

Currently, guidelines for the surgical management of traumatic brain injury recommend the following [9]. Surgical evacuation for AEDHs should be performed for hematomas larger than 30 cm³, irrespective of the Glasgow Coma Scale (GCS) score. However, AEDHs measuring less than 30 cm³ with a GCS score greater than 8 can be managed without surgery, with close neurological observation. Similarly, surgical evacuation for ASDHs is recommended for patients with a hematoma thickness greater than 10 mm or midline shift exceeding 5 mm if the GCS score rapidly declines to less than 9 and the pupils show asymmetry or dilation. Craniotomy or craniectomy with bone flap removal and dura mater plasty are the preferred surgical techniques for evacuation. Furthermore, surgical intervention for traumatic contusional parenchymal hemorrhage (TCPH) is recommended for patients exhibiting progressive neurological deterioration and refractory intracranial hypertension. Additionally, patients with parenchymal hemorrhage larger than 50 cm³ are recommended to undergo operative treatment. Conversely, patients without neurological deficits and no significant signs of mass effect can be managed without surgery but require intensive monitoring and serial imaging. Regarding bilateral traumatic intracranial hematoma, the surgical indications are as follows: 1) signs of brain herniation; 2) clinical deterioration of consciousness with hemorrhagic progression; 3) evident mass-occupying effect and compression of basal cisterns. The decision on whether to perform unilateral or bilateral surgery should be based on clinical, radiological, and ICP findings, with neurosurgeons making the final determination. Surgical operations can be performed simultaneously or separately if both lesions require surgical intervention [5,6].

In the present case, a coup epidural hematoma, contralateral subdural hematoma, and contusional parenchymal hemorrhage were observed. Bilateral craniotomies with unilateral bone flap removal were performed to address the bilateral intracranial hematomas. In addition, the patient exhibited cerebral edema, respiratory dysfunction, transient hypotension, anemia, infection, hyperthermia, tachycardia with mild heart failure, hyperglycemia, pulmonary infection, and liver dysfunction. The patient was mechanically ventilated and underwent tracheal intubation in the Neurological Intensive Care Unit (NICU). Several guidelines for traumatic brain injury provide recommendations for managing these conditions [10-13].

Transient hypotension (systolic blood pressure <90 mmHg) lasting at least 5 minutes has been associated with a significant increase in neurological morbidity and mortality. Therefore, blood pressure should be closely monitored, and hypotension should be avoided. Rapid correction of hypotension can be achieved using vasopressor drugs such as phenylephrine, norepinephrine, and catecholamines. The duration of hypoxemic episodes (< SaO₂ 90%) is a crucial predictor of mortality. Oxygenation levels should be monitored, and hypoxia (PaO₂ < 60 mmHg or O₂ saturation < 90%) should be prevented [10]. Stress-related hyperglycemia in TBI is induced by counter-regulatory hormones and/or insulin resistance, further damaging the injured brain tissue. Hyperglycemia after TBI is associated with an increased risk of mortality and poor neurological outcomes. Therefore, maintaining serum glucose concentrations between 8 mmol/L (1.4 g/L) and 10-11 mmol/L (1.8-2 g/L) is recommended for patients with severe TBI [11]. To prevent infection with intubation, administration of peri-procedural antibiotics is recommended to reduce the incidence of pneumonia.

Early tracheostomy should be considered to reduce the duration of mechanical ventilation. However, for antiseizure prophylaxis, the routine use of phenytoin or valproate is not recommended for preventing late posttraumatic seizures (PTS). Hyperventilation is recommended as a temporary measure to reduce elevated intracranial pressure (ICP), but prophylactic hyperventilation (PaCO₂ of 25 mmHg or less) is not recommended [12]. Osmotherapy is commonly employed to control cerebral edema. Hypertonic solutions, such as mannitol and glycerol, are used in osmotherapy. It is recommended to administer mannitol 20%, glycerol 10%, or hypertonic saline solution (at 250 mOsm) via infusion over 15-20 minutes to manage threatened intracranial hypertension or signs of brain herniation. Compared to mannitol, glycerol has similar effectiveness. Glycerol is associated with a significantly lower risk of acute kidney injury, electrolyte disturbances, and rebound effects [13]. On the other hand, high-dose glucocorticoids are not recommended for improving outcomes or reducing ICP. High-dose methylprednisolone is associated with increased mortality and is contraindicated [9]. We presented an exceptional outcome in a case of very severe TBI with a GCS score of 4. The complete recovery in this case is likely attributed not only to surgical treatment but also to systemic management, including therapies for hyperthermia, hyperglycemia, respiratory dysfunction, and episodic hypotension. Hyperthermia, hyperglycemia, and respiratory dysfunction were speculated to be associated with brain edema affecting the hypothalamus and midbrain. These pathological conditions are interconnected, emphasizing the importance of integrated medical treatment in the ICU. In this case, glycerol 10% was used for two weeks to reduce brain edema. Although mannitol 20% can rapidly reduce brain edema, it can sometimes lead to acute renal failure. Therefore, complications associated with the use of glycerol were not observed. Dopamine hydrochloride was used for episodic hypotension, sliding-scale insulin for hyperglycemia, tracheal intubation and the supply of high-concentration oxygen for respiratory dysfunction and decreased blood oxygen saturation, followed by mechanical ventilation. Antibiotics were administered to control respiratory system infections. Body cooling and acetaminophen were employed to manage hyperthermia. The combination of monoammonium glycyrrhizinate and cysteine hydrochloride (MG-CH) was used to treat liver dysfunction [14].

The patient contracted COVID-19 on day 27 after the injury and received a 5-day course of the anti-SARS-CoV-2 agent Molnupiravir without developing respiratory symptoms. Cognitive impairments have been observed in patients recovering from COVID-19 [15]. However, the patient did not show cognitive deterioration after COVID-19. The absence of cognitive deterioration may be attributed to either being in the postinjury convalescent period during the COVID-19 infection or the lack of significant changes due to the effect of Molnupiravir. The patient's motor and cognitive functions had almost fully recovered by the time of the cranioplasty operation. At discharge, the patient exhibited only left quadrant hemianopsia. This rare case of a patient with very severe TBI achieved an excellent outcome and was able to return to work. We provided appropriate treatment to the patient, closely adhering to the guidelines for severe TBI. It is crucial to thoroughly understand the individual conditions of patients with severe TBI and provide them with the appropriate treatments. Without such comprehensive and tailored care, patients with severe TBI may not be able to be saved or recover without disability.

Conclusion

In conclusion, we presented an extremely rare case of a patient who achieved an excellent outcome following a very severe traumatic brain injury, which included an acute epidural hematoma, contralateral subdural hematoma, and intracerebral contusional hemorrhage. Despite the high mortality and morbidity associated with such cases, it is possible to rescue some patients, and rare cases may achieve full recovery if the individual conditions of patients with severe TBI are thoroughly understood and appropriate treatments are provided.

Declarations

Ethical statement: This study was approved by the Institutional Review Board of Asahi Hospital (No. 2023-1). The patient provided informed consent after the study. Written informed consent for publication of the research details and clinical images were obtained from the patient.

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Conflicts of interest: The authors have no conflicting financial interest.

References

1. Stocchetti N, Carbonara M, Citerio G, Ercole A, Skrifvars MB, et al. Severe traumatic brain injury: targeted management in the intensive care unit. *Lancet Neurol.* 2017; 16: 452-464.
2. Van Dijck JT, Reith FC, van Erp IA, et al. Decision making in very severe traumatic brain injury (Glasgow Coma Scale 3-5): a literature review of acute neurosurgical management. *J Neurosurg Sci.* 2018; 62: 153-177.
3. Van Dijck JTJM, Mostert CQB, Greeven APA, et al. Functional outcome, in-hospital healthcare consumption and in-hospital costs for hospitalised traumatic brain injury patients: a Dutch prospective multicentre study. *Acta Neurochir (Wien).* 2020; 162: 1607-1618.
4. Pandey S, Sharma V, Singh K, et al. Bilateral Traumatic Intracranial Hematomas and its Outcome: a Retrospective Study. *Indian J Surg.* 2017; 79: 19-23.
5. Hu Y, Sun H, Yuan Y, Li Q, et al. Acute bilateral mass-occupying lesions in non-penetrating traumatic brain injury: a retrospective study. *BMC Surgery.* 2015; 15: 6.
6. Pandey S, Sharma V, Singh K, et al. Bilateral traumatic intracranial hematomas and its outcome: a retrospective study. *Indian J Surg.* 79: 19-23.
7. Bullock MR, Chesnut R, Ghajar J, et al. Surgical management of Traumatic Brain Injury Author Group. Surgical management of acute epidural hematomas. *Neurosurgery.* 2006; 58(3): S7-15.
8. Karibe H, Hayashi T, Hirano T, Kameyama M, Nakagawa A, Tominaga T. Surgical management of traumatic acute subdural hematoma in adults: a review. *Neurol Med Chir.* 2014; 54: 887-894.
9. Hawryluk GWJ, Rubiano AM, Totten AM, et al. Guidelines for the management of severe traumatic brain injury: 2020 Update of the decompressive craniectomy recommendations. *Neurosurgery.* 2020; 87: 427-434.
10. The Brain Trauma Foundation. The American Association of Neurological Surgeons. The Joint Section on Neurotrauma and Critical Care. Resuscitation of blood pressure and oxygenation. *J Neurotrauma.* 2000; 17(6-7): 471-478.
11. Shi J, Dong B, Mao Y, Guan W, Cao J, Zhu R, Wang S. Review: Traumatic brain injury and hyperglycemia, a potentially modifiable risk factor. *Oncotarget.* 2016; 7: 71052-1061.
12. Carney N, Totten AM, O'Reilly C, et al. Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. *Neurosurgery.* 2017; 80: 6-15.
13. Wang J, Ren Y, Zhou LJ, Kan LD, Fan H, Fang HM. Glycerol infusion versus mannitol for cerebral edema: A systematic review and meta-analysis. *Clin Ther.* 2021; 43: 637e649.
14. Chu S, Niu Z, Guo Q, et al. Combination of monoammonium glycyrrhizinate and cysteine hydrochloride ameliorated lipopolysaccharide/galactosamine-induced acute liver injury through Nrf2/ARE pathway. *Eur J Pharmacol.* 2020; 882: 173258.
15. Tavares-Júnior JWL, de Souza ACC, Borges JWP, Oliveira DN, Siqueira-Neto JI, Sobreira-Neto MA, Braga-Neto P. COVID-19 associated cognitive impairment: A systematic review. *Cortex.* 2022; 152: 77-97.