The Egyptian Journal of Hospital Medicine (April 2023) Vol. 91, Page 4117-4123

Clinical Diagnosis and Management of Traumatic Acute Intracranial Subdural Hematoma: Review Article

Omar Mohammed Omar Darah*, Mustafa Abd-Elsalam Mustafa El-Zohiery, Magdy El-Sayed Hassan Rashed, Wael Abd-Elrahman El-Mesallamy
Department of Neurosurgery, Faculty of Medicine, Zagazig University, Egypt
***Corresponding author:** Omar Mohammed Omar Darah, **Mobile:** (+20) 01021825006, **E-mail: Omardrah89@gmail.com**

ABSTRACT
Traumatic brain injury (TBI) is the leading cause of death and disabling conditions in persons. The frequency and severity of head injuries have both decreased as a result of numerous industrial safety improvements, the installation of airbags in automobiles, and the enforcement of speed restrictions. TBI survivorship has increased due to advancements in emergency response times and acute care, but this has also increased the need for accurate ways to detect individuals at risk of subsequent diseases. Pupil reactivity, the Glasgow Coma Scale, and head computed tomography are currently the main clinical signs indicating the existence of brain damage. The current review will cover the clinical presentation, diagnosis, and treatment of TBI,
Keywords: Traumatic brain injury, Glasgow Coma Scale, Head injuries, Review, Zagazig University.

INTRODUCTION
Brain damage caused by an external mechanical force, such as impact, rapid acceleration or deceleration, blast waves, or projectile penetration, is known as traumatic brain injury (TBI). With current technology, brain structural damage may or may not be detectable, and brain function may be temporarily or permanently impaired [(1)].

Based on severity, anatomical aspects of the damage, and the mechanism, TBI is often categorized into closed and piercing head injuries (the causative factors). When there is a closed injury, also known as a non-penetrating or blunt injury, the brain is not exposed. A penetrating, or open, head injury happens when something pierces the skull and pierces the dura mater, the outermost membrane enclosing the brain [(2)].

DIAGNOSIS
TBI patients are often treated and initially diagnosed in the emergency room of a hospital. The process of determining the severity of brain injury starts after vital signs are evaluated, stabilized and other life-threatening injuries are discovered and treated [(3)].

CLINICAL DIAGNOSIS
Initial Clinical Evaluation:
Since its invention in 1974 by Teasdale and Jennet, the Glasgow Coma Scale (GCS) has served as the benchmark for impartially evaluating patients with catastrophic brain injuries. The GCS offers a reliable, tracking a patient's neurological condition over time is a relatively simple and objective way to evaluate neurological health, which are its two key benefits [(4)].

With brain injuries, there are three degrees of severity: mild, moderate, and severe. The Glasgow Coma Scale (GCS), the most popular tool for classifying TBI severity, rates a subject's level of awareness on a range
from 3 to 15 based on how they react to verbal, motor, and eye-opening stimuli [(5)].

The Glasgow Coma Scale is a neurological tool designed to record a person's level of consciousness in a reliable, impartial manner for both initial and follow-up examination.

A patient's score is determined by comparing their condition to the requirements of the scale, and the results range from 3, which denotes profound unconsciousness, to either the old scale of 14 or the new scale of 15 (The more widely used modified or revised scale). The GCS is now used by clinicians and first responders to assess all acute medical and trauma patients. It was originally used to gauge a patient's level of consciousness after suffering a head injury.

In hospitals, it is also utilized to keep track of chronic patients receiving critical care. Professors of neurosurgery at the University of Glasgow, Graham Teasdale and Bryan Jennet, published the scale in 1974. Three tests make up the scale: verbal, motor, and eye responses.

Both the individual three values and their aggregate are taken into account. Death or a deep coma is represented by a GCS of 3, while 15 is the maximum (a fully conscious individual) [(5)].

Best eye response (E):
The four ratings are, from most severe to least:
1. No eye movement.
2. If there is pressure on the patient's fingernail bed, the eye may open in response to pain fails to elicit a response, pressure or rub may be applied to the supraorbital and sternal areas).

Received: 01/11/2022
Accepted: 03/01/2023

3. Opening the eyes on speaker (not to be confused with an awaking of a sleeping person; such patients receive a score of 4 not 3).
4. Sudden opening of the eyes.

Best verbal response (V):
There are 5 levels, with the worst being grade being the highest:
1. There is no spoken reply.
2. Sounds that are unclear (Moaning but no words).
3. Unacceptable language (Random or exclamatory articulated speech, but no conversational exchange).
4. Perplexed (The patient answers inquiries coherently, but there are some signs of confusion and disorientation).
5. Oriented (Patient provides accurate and clear responses) to inquiries about name, age, reason, day of the week, etc.).

Best motor response (M):
There are 6 levels, with the most severe being degree
1. No motion is elicited in (1).
2. Including discomfort (shoulder internal rotation, arm abduction). (Forearm pronation, wrist extension, and decerebrate reaction).
3. abnormal flexion pain (adduction of arm, internal rotation of shoulder, pronation of forearm, flexion of wrist, decorticate response).
4. Flexibility/Retreat from Pain (flexion of elbow, supination of forearm, flexion of wrist when supra-orbital pressure applied; pulls part of body away when nail bed pinched).
5. Emphasizes the pain (Purposeful movements towards painful stimuli; e.g., hand crosses mid-line and gets above clavicle when supra orbital pressure applied).
6. Obeys commands (The patient complies with requests for easy tasks) [(5)].

Interpretation:
- Both the individual components and the overall score are crucial.
- As a result, the score is written as "GCS 9 = E2 V4 M3 examination."
- Severe brain injury has a GCS of 8 or low. Mild brain injury has a GCS of 9 to 12 or below [(5)].

For young children, there are comparable systems; nevertheless, the GCS grading system has a limited capacity to predict outcomes. As a result, additional classification schemes, like the one in **Table 1**, are also employed to determine severity. The duration of post-traumatic amnesia (PTA), loss of consciousness, and all three criteria are used in a current model created by the American Departments of Defense and Veterans Affairs (LOC) [(5)].

Table (1): Severity of traumatic brain injury.

Severity	GCS	Post traumatic amnesia	Loss of conscious
Mild	13-15	Less than 1 day	0-30 minutes
Moderate	9-12	1 day-7 days	30 minutes to 24 hours
Severe	3-8	More than 7days	More than 24 hours

Basic brainstem reflexes should also be tested in patients. The evaluation of the pupillary reflex, corneal reflex, gag/cough reflex, oculocephalic reflex, vestibuloocular reflex, and spontaneous breathing are all included in this assessment. Greater than 1 mm of anisocoria or asymmetry in the pupillaries (up to 1 mm may be physiological). Must be attributed to an intracranial lesion until proven otherwise) [(5)].

CLINICAL PRESENTATION
- The degree of parenchymal brain injury and the size of the hematoma determine how clinically an acute SDH presents in a patient.
- Headache, nausea, disorientation, personality changes, lower level of consciousness, speech difficulties, various changes in mental status, impaired eyesight or double vision, and weakness are some symptoms of acute SDH. Of fact, neurological abnormalities connected to acute SDH could possibly be the source of these symptoms.

Hemiparesis contralateral to the hematoma; altered degree dilated or unresponsive pupil ipsilateral to the hematoma; consciousness (or earlier: a pupil with a more constrained range of reaction).

IMAGING STUDIES
Imaging is a crucial clinical technique for treating patients with brain injuries [(6)].

Plain x-ray: However, it is When evaluating penetrating head trauma, it is occasionally used to quickly determine the level of foreign body penetration and to check for bullet fragments that have been retained inside the brain after a gunshot wound. In patients with closed head traumas, CT scans are used more frequently than plain x-rays [(6)].

CT scan: All patients with acute head injuries who need to be admitted and monitored in the hospital are routinely assessed using CT scans. The use of CT scans facilitates the detection of fractures in the bone windows and the existence of contusions, intraparenchymal hematomas, or extra-axial hematomas in the tissue windows. Among the disorders that are looked for during CT scans include pneumocephalus, hydrocephalus, cerebral edema, midline shift, and compression of the subarachnoid cisterns near the base of the brain [(6)].

Table (2): Advantages and disadvantages of CT scan in the TBI [6].

Advantages	Disadvantages
Non-invasive and rapid	Traumatic vascular lesions may be missed
Very sensitive for acute hemorrhage	Diffuse axonal injury is likely to be missed
Defines nature of ICH (i.e.,SDH, SAH)	Motion artifact may limit study
Defines anatomical location of lesion	Posterior fossa lesions are poorly depicted
Identifies fractures of the cranium	Depressed skull fractures at the vertex (or along the plane of an axial scan) are poorly depicted
Sensitive to detecting intracranial air	The scanner has a weight limit, and a patient may be too heavy
Sensitive in identifying foreign objects	A patient may decompensate while in the scanner

Imaging of Subdural Hematoma

Acute SDHs are less than 72 hours old and show up on a CT scan as being denser than the brain. Compared to the brain, acute SDHs are isodense or hypodense and range in age from 3 to 20 days.

Chronic SDHs are hypodense in comparison to the brain and have a lifespan of at least 21 days (3 weeks). SDHs can, however, have mixed characteristics, as in the case of acute bleeding into a chronic SDH [7].

Essentially a radiographic diagnostic, acute subdural hematoma (SDH) presents as a crescent-shaped, pan-hemispheric blood accumulation (Figure 1). Nonetheless, some SDH have lentiform features that resemble acute epidural hematomas (EDH). Acute SDH is shown on a CT scan as a crescent-shaped lesion collecting between the dura and arachnoid membrane. They most typically appear after tearing of the cortical bridging veins. Blood to flow through the continuous subdural area to freely move between the brain hemispheres. The primary radiological characteristic that sets acute EDH apart from it is its focused nature. Many acute SDH do not exhibit the conventional crescent shape. Biconvex margins, which resemble conventional EDH, are seen in about 8–14% of these atypical SDH [7].

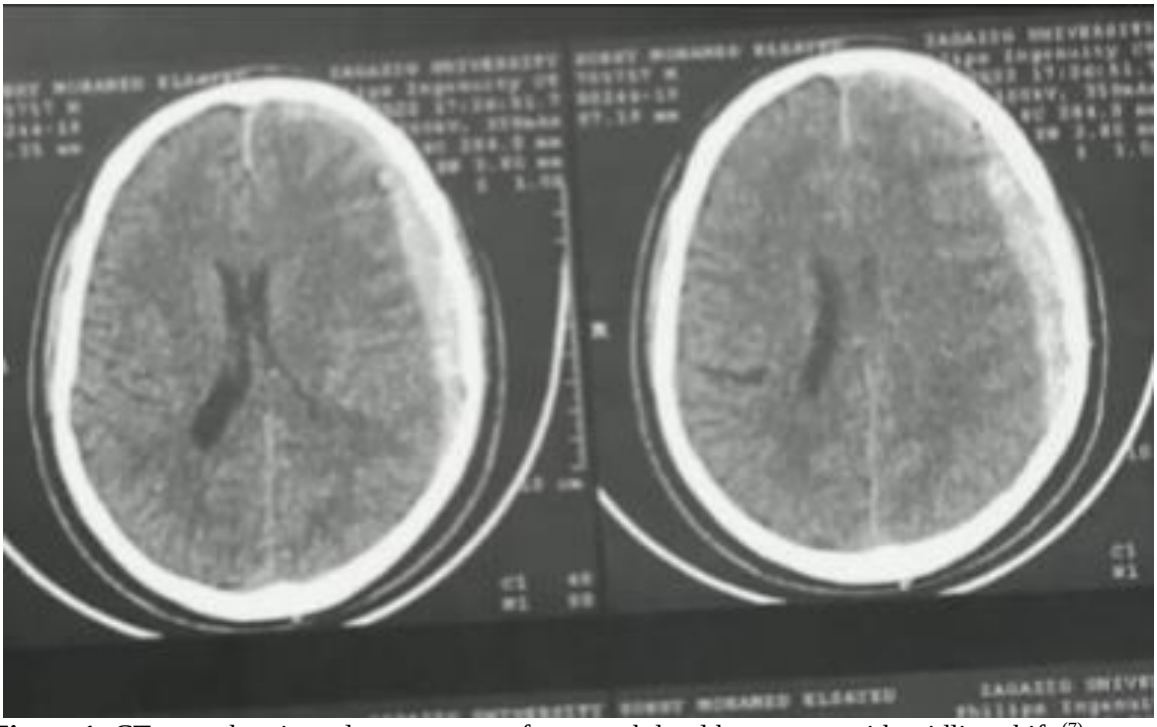

Figure 1: CT scan showing a large amount of acute subdural hematoma with midline shift [7].

Atypical SDH appearances on CT scans

Because there are no restrictions in the subdural space, which has promise. Lesions with a crescent shape and a panhemispheric appearance are typical of acute SDH. In CT scans, it is typically simple to distinguish between acute SDH and EDH [8,9].

Nonetheless, CT has identified a number of SDH subtypes. These unusual SDH characteristics are frequently described in the literature. A 39% incidence of unusual findings on CT scans was reported by In a study of 71 cases of acute SDH that were assessed by CT, Reed et al. One-fourth of their SDH had convex borders, simulating the characteristics of acute EDH. This could easily result in a wrong diagnosis [10].

On radiographs, it was On the basis of shape alone, it is challenging to distinguish acute lentiform SDH from EDH. Thanks to several additional CT features that were very helpful in making the correct diagnosis, four differential features could be found that were in favor of SDH. A dural line, a crescentic tail, and an acute angle were present above the hematoma boundary, and a direct link to the underneath intracerebral hematomas [10].

The main 4 differential features are (Figure 2):

- **Su and Colleagues** described the hematoma's crescentic tail, which was concave in the direction of the brain, near its edge. Another comparable discovery was that hematomas in the outermost serial sections gradually take on their crescent shape. This was thought to be a reliable indicator of acute SDH [10].
- The second characteristic was the acute angle between the skull bone and the inner hematoma border. Deep depression of the brain surface led to obtuse angles. Given that the arachnoid membrane and brain parenchyma were both present, obtuse angles at the margin may be a key sign of the subdural site of hematomas easier to indent than the dura mater. In terms of radiology, this characteristic resembled the hyperconvexity described by [10].
- The third clue for a differential diagnosis was the appearance of dural lines on CT scans. When using non-enhanced CT scans, dura can occasionally be seen, notably the artificial dura that is frequently utilized in neurosurgery treatments. The connection between hematomas and dural lines should be carefully observed to determine the proper diagnosis of SDH [10].

The identification of related ICH was the final feature. A further hint for localization was if a dural line connected the biconvex hematoma to the ICH. The subdural space is where the hematoma was detected if it was found linking directly to the intra-parenchymal part of the hematoma [10].

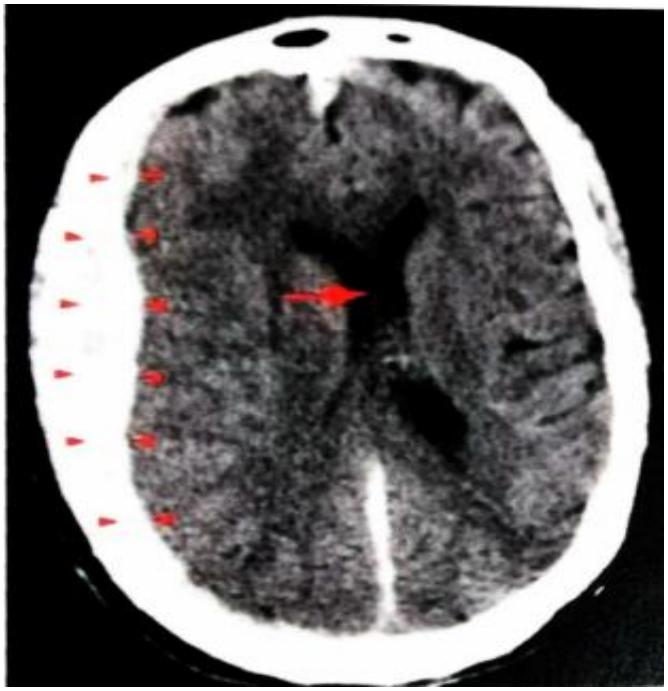

Figure 2: CT scan shows features of acute subdural hematoma.

Magnetic resonance imaging (MRI)

Acute brain injury evaluation with MRI is not widely used. Although MRI offers remarkable anatomical details, it is not frequently utilized to assess severe head injuries due to its lengthy acquisition periods and difficulty getting an MRI in critically unwell patients [7].

MANAGEMENT

Emergency medical staff should ideally begin stabilizing the head injury sufferer at the scene of the accident. Preventing hypoxia, maintaining a healthy blood pressure, and identifying and treating medically correctable cerebral abnormalities are the first steps in treating any patient with a TBI [11].

The advanced trauma life support (ATLS) approach, which focuses on maintaining good oxygenation and tissue perfusion while keeping airway patency, is used to assess the head-injured patient first. The purpose of this is to stop the emergence of second-degree brain damage. Just by opening and maintaining the airway and giving these patients respiration at the scene of the accident, the horrifying morbidity and mortality of severe brain damage might be significantly decreased. The 90% death rate for these patients was drastically reduced to 40% when this strategy was implemented. While prehospital care has improved over the past ten years, morbidity and mortality in these patients have decreased. Also, this

makes prompt medical and surgical intervention possible [9].

Pre-hospital Management

If the damage is penetrating, one should try to halt the bleeding by packing the wound with sterile material or placing a compressive bandage. To establish whether the patient has had blunt or penetrating head trauma, we must first evaluate the severity of the injury. In unconscious patients, neck collars for spinal protection are required [9].

Whether a severe TBI occurs as a single head injury or in conjunction with other injuries, Hypotension and hypoxia (arterial oxygen saturation of 90% and systolic blood pressure 90 mm Hg) are frequently present. Both conditions require prompt treatment. Hypotension can occur in as many as 16% of patients when they first arrive at the hospital and has been linked to significantly higher morbidity and death in patients with serious head injuries. Moreover, studies have shown that up to 50% of patients with head injuries have hypoxia at some point between the time of the event and when they arrive at the hospital. 100% inspired oxygen is initially used in conjunction with endotracheal intubation to treat and prevent hypoxia when airway protection is compromised or as a result of chest or facial trauma [9].

ABCs of trauma resuscitation:

To stop secondary worsening from hypoxia or hypotension, stabilizing an airway, breathing, and circulation (ABC) is the top priority [12].

Airway and breathing:

Pre-hospital intubation may be beneficial for patients with isolated severe TBIs, however some centers routinely intubate patients if their GCS is below 9. Monitoring the end-tidal CO2 can help prevent complications including unrecognized esophageal intubation and unintentional hyperventilation. Another element that could have a negative impact on a patient's success is the additional delay in transferring them for treatment that will last. A "scoop and run" policy may be desirable if the projected Short arrival time at the hospital (e.g., less than 10 minutes). [12].

Indications of intubation:

1) Coma (GCS8),
2) A lack of protective reflexes,
3) Insufficient ventilation,
4) Spontaneous hyperventilation,
5) Bilateral fracture mandible,
6) Significant oral bleeding
7) Pulmonary edema, and
8) Seizure.

The efforts at intubation must be preceded by the administration of O2 by mask or intravenous infusion of sterile saline using a cannula with a large diameter [12].

The severely head injured patient is ventilated to normocapnia unless there is evidence of tentorial herniation (fixed, dilated or asymmetric pupils with extensor posturing) after being intubated. Mild hyperventilation can then be used to temporarily lower intracranial pressure (ICP). The hypocapnia-induced vasoconstriction will lower the ICP and, as a result, the cerebral blood volume (CBV), but it may also lower the pressure at a period when the post-injury CBF is already decreased, cerebral blood flow (CBF). Hence, frequently recommending hyperventilation prevention in TBI patients runs the risk of making the brain ischemic and escalating subsequent brain injury [12].

If there are overt signs of a brain herniation, mannitol or hypertonic saline can also be used to quickly lower the ICP. It may also be necessary to be transported to a neighboring trauma center or hospital that offers a bed in an intensive care unit, 24-hour computed tomography (CT) service, and neurosurgical services have an impact on a patient's prognosis [12].

Serious trauma patients are more likely to survive if they are taken straight to a trauma center than receiving care at a nearby hospital first. The way emergency medical services are currently delivered the majority of trauma patients in the UK (75%) are subsequently transported to a trauma center after receiving sufficient resuscitation and stabilization. An organized emergency trauma system should include the transportation of critically ill patients to the most suitable resource facility. It has been demonstrated that the establishment of such a system in Canada and the United States improves the prognosis for trauma victims, including TBI [13].

Circulation

Fluid resuscitation is necessary for patients with a systolic blood pressure below 110 mmHg. Little volume resuscitation (250 ml) using hypertonic saline solution looks to be quite promising in this case, despite the fact that lactated ringer's solution is typically advised [14].

Lactated Ringer's solution or regular saline solution is advised for volume resuscitation in head injured patients, the brain trauma foundation's recommendations for the treatment of severe head injury. Hypotonic solutions shouldn't be used because they'll make cerebral edema worse [14].

Crystalloid, colloid, and occasionally packed red blood cells should be used in aggressive fluid resuscitation. In head-injured patients, hypertonic saline solution expands intravascular volume, draws water from

the intracellular space, lowers ICP, and increases heart contractility, among other positive benefits [14].

Steroids

Steroids have no place in TBI, according to the guidelines of the Brain Trauma Foundation, as they have not yet proved to lower ICP or enhance patient outcomes. As a result, current recommendations do not suggest using steroids to enhance outcomes or lower patient's intracranial pressure after severe head injuries [15].

Seizure prophylaxis

Between 4%-53% of patients with TBI have at least one seizure [16]. GCS below 10, a penetrating head injury, cortical contusions, a fractured, depressed skull, epidural, subdural, or cerebral hematoma, or a seizure within 24 hours of the injury are only a few factors that have been linked to an elevated risk of seizures. Seizures boost neurotransmitter release, increase brain metabolic rate, and are linked to increases in ICP. Anti-epileptic medications (phenytoin or carbamazepine) are effective at avoiding seizures, according to a meta-analysis of clinical studies, but they have no long-term effects on mortality or the incidence of seizures [9].

Only the first seven days after an accident do the present studies suggest the use of phynetin or carbamazepine to prevent seizures [9].

General Patient Management

Once hemodynamic stability has been attained, examine the patient for other injuries such as chest, pelvic, long bone fractures, include abdominal injuries. Long bone fractures that require early splinting minimize tissue damage and bleeding. Elevating the damaged extremity, while using a tight compressive dressing, is recommended. When auscultation reveals diminished breath sounds, a hemopneumothorax should be suspected, and thoracocentesis may be performed. As 10% of patients with severe head injuries also have concurrent spine abnormalities, it is vitally crucial to examine the spine in patients with TBI. Many of these wounds involve the cervical spine [14]. Meningitis, ventriculitis, and cerebral abscess have a higher risk of occurring in TBI patients. When transventricular or midline injuries, air sinus wounds, leaks of cerebrospinal fluid, or those occur, infectious consequences are more common [14].

Neurological Assessment

The neurologic evaluation of individuals with TBI needs to take the following into account besides figuring out the GCS score: examinations of the brainstem, pupils, the corneal reflex, the gag reflex, and the motor evaluation of reflexes and the senses [17].

Acute post-traumatic subdural hematoma management

The attending neurosurgeon will decide whether to evacuate the hematomas or treat them non-operatively once ASDH has been diagnosed on a CT scan. If patients are conscious when they are admitted and do not worsen afterward, they are treated conservatively, and had an ASDH that was only a few millimeters thick. The surgical strategy is left up to the attending neurosurgeon, who should carry out the operation he believes is optimal for each specific patient. The methods include decompressive craniectomy, a standard craniotomy with hemorrhage irrigation, and hemorrhage evacuation [1].

When both parenchymal injury and secondary insult occur, the incidence of cerebral edema is significantly higher than in either situation on its own are present. Hematoma thickness does not correlate with the occurrence of brain edema [1].

The management's objectives are to maintain cerebral blood flow (CBF), cerebral perfusion pressure (CPP), and intracranial pressure (ICP) in order to prevent cerebral ischemia. Some publications have reported a steady decrease in mortality and an increase in the proportion of patients who have a favorable result following a decompressive craniotomy or craniectomy. According to some writers, surgical decompression may improve clinical results by increasing cerebral compliance, CPP, and CBF, reducing edema, and improving oxygen flow to brain tissue. Although the surgical procedure is quite simple, postoperative complications can significantly impair clinical success [18].

Prior to DC, medical care was given in accordance with the Brain Trauma Foundation's (BTF) Head Injury Guidelines. In addition to sedation (short-acting sedatives such as midazolam 0,03- mg/kg/h or propofol 0.3-4 mg/kg/h) and narcotics (fentayl 0.045-0.3 mg kg/min), medical therapy comprised intubation, ventilation, oxygenation, head elevation fluid resuscitation, and sedation. The following stage was mild hyperventilation (PaC02 32-36 mmHg), followed by hyperosmolar/diuretic therapy with a target serum osmolarity of 300-310 mosmol (using mannitol 0.25-1 g/kg bolus, furosemide 20 mg bolus, or 250 ml hypertonic saline 3% or 5%). Muscle relaxants with a limited half-life were only applied briefly [18].

Supporting and sponsoring financially: Nil.
Competing interests: Nil.

REFERENCES

1. **Chabok Y (2011):** Acute subdural Hematoma. Neurosurgery Quartely, 21(2):103-6.
2. **Leitgeb J, Mauritz W, Brazinova A *et al*. (2012):** Outcome after severe brain trauma due to acute subdural hematoma. Journal of neurosurgery, 117(2):324-33.
3. **Starr F (2015):** Initial Trauma Assessment and Resuscitation. In Common Surgical Diseases: An Algorithmic Approach to Problem Solving (pp. 25-28). New York, NY: Springer New York. https://link.springer.com/chapter/10.1007/978-1-4939-1565-1_6
4. **Zuercher M, Ummenhofer W, Baltussen A *et al*. (2009):** The use of Glasgow Coma Scale in injury assessment: a critical review. Brain Injury, 23(5):371-84.
5. **Jain S, Iverson M (2020):** Glasgow Coma Scale. StatPearls. Treasure Island (FL)., StatPearls Publishing .
6. **Zakharova N, Kornienko V, Potapov A *et al*. (2014):** Neuroimaging of traumatic brain injury. Springer. https://books.google.com.eg/books?hl=ar&lr=&id=rzW6BQAAQBAJ&oi=fnd&pg=PR5&dq=6.%09Zakharova+N,+Kornienko+V,+Potapov+A+et+al.+(2014):+Neuroimaging+of+traumatic+brain+injury.+&ots=zzBZjnhn6U&sig=-6eCFlRd0Vba2IXYVbA5FYk4jvg&redir_esc=y#v=onepage&q=6.%09Zakharova%20N%2C%20Kornienko%20V%2C%20Potapov%20A%20et%20al.%20(2014)%3A%20Neuroimaging%20of%20traumatic%20brain%20injury.&f=false
7. **Dekeyzer S, Luc H, Thijs V (2019)**: Clinical Neuroradiology. Traumatic Brain Injury: Imaging Strategy, 2(1):55-9.
8. **Tehli O, Kazanc B, Türkoglu E *et al.* (2011):** Subdural hematomas and emergency management in infancy and childhood: a single institution's experience. Pediatric Emergency Care, 27(9):834-6.
9. **Gerard C, Busl M (2014):** Treatment of acute subdural hematoma. Current Treatment Options in Neurology, 16(1):211-9.
10. **Su C, Wang C, Huang H *et al.* (2010):** Differential CT features of acute lentiform subdural hematoma and epidural hematoma. Clinical Neurology and Neurosurgery, 112(7):552-6.
11. **Friess H, Kilbaugh J, Huh J (2012):** Traumatic brain injury. Pediatric Neurocritical Care, 4(2):103-9.
12. **Kovacs G, Sowers N (2018):** Airway management in trauma. Emergency Medicine Clinics, 36(1):61-84**.**
13. **Walcott P, Khanna A, Kwon S *et al*. (2014):** Time interval to surgery and outcomes following the surgical treatment of acute traumatic subdural hematoma. Journal of Clinical Neuroscience, 21(12):2107-11.
14. **D'Amours K, Sugrue M, Deane A (2002):** Initial management of the poly-trauma patient: A practical approach in an Australian major trauma service. Scandinavian Journal of Surgery, 91(1):23-33.
15. **Sawauchi S, Murakami S, Ogawa T *et al.* (2007):** Acute subdural hematoma associated with diffuse brain injury: analysis of 526 cases in Japan neurotrauma data bank. No Shinkei geka. Neurological Surgery, 35(1):43-51.
16. **Frey L (2013):** Seizures and. Management of Adults With Traumatic Brain Injury. bing.com/images
17. **McHugh S, Engel C, Butcher I *et al.* (2007):** Prognostic value of secondary insults in traumatic brain injury: results from the IMPACT study. Journal of Neurotrauma, 24(2):287-93.
18. **Faleiro M, Faleiro M, Caetano E *et al*. (2008).** Decompressive craniotomy: prognostic factors and complications in 89 patients. Arquivos de Neuro-psiquiatria, 66:369-73.