



Severe Traumatic Brain Injury

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Coming back from the weekly visit to his parents, FD, a 34 year old male while driving on the highway, lost control of his car and crashed against the guard-rail. The left side of the car was completely destroyed. In less than 14 minutes he was rescued by an advanced life ambulance team with physician on board. He was comatose, the airway was open, respiratory rate 32/m, heart rate 135 bpm, systolic blood pressure 85 mmHg; the first Glasgow Coma Score was 6 (V1; E1; M4). Unequal pupils (left > right pupil) were detected; his abdomen appeared distended and an fracture of the left leg was evident. He was suddenly intubated and ventilated after administration of midazolam (0.1 mg/kg). A colloid infusion was started; a cervical collar was placed on his neck and he was blocked on a pneumatic mattress and the left leg was splitted.

On arrival at the ED, his neurologic condition was the same but he showed a downward and external rotation of the left ocular bulb with a palpebral contusion. Haemodynamic status deteriorated with a BP of 73/43, Hr 135 bpm; SpO₂ was 100% and Hb on the coxymeter 4.5g/dl. A chest X ray was positive for multiple chest rib fractures. Urgent abdominal US detected the presence of a free blood collection. The patient was immediately transferred to the OR where the fractured spleen was removed. At the end of the operation, after multiple hemotransfusions and cristalloid infusion, haemodynamics condition improved to a BP 125/76mmHg, Hr 95 bpm, SpO₂ 100% at a FiO₂ .4 and Hb 9,5 g/dl and remained stable. His neurological condition did not improved after stabilization (GCS = 6; best mor response a coordinate flexion to pain). His was then transferred to Radiology: the first Ct scan showed only a modest brain swelling with no intracerebral and/or extraassial blood collection.

He was then transferred to ICU, sedated with remifentanyl (0.05 mcg/kg/m) and propofol (2.5 mg/kg/h). An ICP monitoring through a intraparenchymal transducer tipped catheter was placed and the first value detected was 15 mmHg. Twelve hours later ICP increased to 25 mmHg refractory to a heavier sedation and mannitol. A second CT showed a bilateral intraparenchymal, subcortical blood collections that were considered as the expression of a diffuse brain injury. There was no shift of the midline and no indication for neurosurgical operation was given by the specialist.

Key Clinical questions:

What are the priorities in the treatment of severe head injuries?

- the role and prevention of cerebral ischemia
- the ABCs:

What about sedation in severe HI?

How much to rely on the first CT for further evolution and prognosis?

Key Learning Points

- Brain ischemia is the main cerebral blood flow pattern complicating head injury
- Head injured patients require aggressive approach in the acute phase for prevention of secondary insults; hypoxia and hypotension are the most frequent, important complications that should be prevented
- Referral to hospital with neurological facilities should be the gold standard where surveillance, diagnosis and prompt surgical intervention could be provided in case of detection of mass lesion.

Introduction

Traumatic brain injury (TBI) represents a relevant pathology in the ED and still remains the leading cause of death among people younger than 25 years of age (1) and the main single factor in determining prognosis in the polytraumatised patient. Approximately 200 patients/100.000 inhabitants/year are admitted in the ED for a TBI; among them 15-20 are severe TBI (STBI) patients defined as patients scored with a GCS < 9.

What are the priorities in the treatment of severe head injuries?

-the role and prevention of cerebral ischemia

Prognosis in head injury has been strictly correlated with the degree and duration of ischemia and more than 90% of autopsies on patients dead after a head injury showed ischemic lesions of different severity (2). Many causes have been advocated for posttraumatic cerebral ischemia such as intracranial hypertension, arterial hypotension, brain edema and swelling, focal tissue compression from intracranial hematomas and involvement of microcirculation and vasospasm of large cerebral arteries. Many in vivo studies have confirmed that ischemia is the predominant cerebral blood flow pattern in the early posttraumatic phase in head trauma patients (3). In addition, early ischemia has been found to correlate with poor outcome and early mortality. The correlation between CBF and mortality is lost after this early period whereas it remains valid for cerebral metabolic rate for oxygen (4). This pattern of early ischemia cannot be attributed only to abnormally low cerebral perfusion pressure, excessive hyperventilation or vasospasm, because it is still present even after normalisation of the haemodynamic and respiratory parameters, suggesting the presence of an increased distal vascular resistance due to different factors (extrinsic microvascular compression by damaged and edematous astrocytic processes, active muscular contraction of the resistance arterioles caused by the trauma induced release of vasoactive substances such as calcium, catecholamines, prostaglandins, haemoglobin, neuropeptides and intravascular thrombosis)(5).

This pattern was demonstrated even with monitoring of brain parenchyma oxygen saturation (6) and the measurement of glutamate and lactate concentration in interstitial and CSF fluid (7,8).

-the ABCs

The cornerstones of emergency treatment of SHI must be aimed at the assessment of the ABC's and the simultaneous resuscitation in the primary survey to obtain a normal oxygenation (arterial haemoglobin oxygen saturation > 95%), the maintenance of CO₂ values at 30-35 mmHg (avoiding hyperventilation with resulting hypocarbia and subsequent hypoperfusion), the rigorous maintenance of normal systemic arterial pressure values (avoiding even short episodes of arterial hypotension).

A=Airway patency

All comatose patients (GCS < 9) should be intubated under sedation and analgesia (9, 10). The indications for an early endotracheal intubation are the following: any case of airway obstruction, the maintenance of an adequate oxygenation and ventilation, the prevention of hyper and

hypocapnia, the protection from airways obstruction, the prevention of neurological deterioration in hostile environments (transport, radiological procedures etc). Oratracheal intubation is usually preferred. Blind nasotracheal intubation should be avoided in any case of suspected basal or maxillary fracture; moreover it needs the patient breathing spontaneously, there is an high percentage of failures and it could give nasal bleeding.

A cervical spine lesion must ever be suspected in all comatose patient; he/she should be treated as having a spine injury until demonstrated otherwise.

But looking at daily clinical behaviour, do we all act according to clinical guidelines? A survey study performed in Italy on 1278 patients showed that about 12% in GCS 7 and 33% of GCS 8 were not intubated in the preH setting (11).

Is prehospital endotracheal intubation a gold priority in the management of the STBI?

Some papers were recently published discussing the key role of prehospital intubation of STBI. Murray (12) showed that prehospital intubation did not demonstrate any improvement in survival, Bochicchio (13) demonstrated that prehospital intubation in patients who survived more than 48 hours after the trauma was associated with a significant increase in morbidity and mortality. The conclusions of these studies must take into consideration the different organisation of EMS and the technical skills of the first aid.

B = Breathing

All intubated patients must be *ventilated*, to obtain:

- a) adequate oxygenation ($paO_2 >90$ mmHg, $SaO_2 >95\%$)
- b) prevention of hyper- or hypocapnia, with $PaCO_2$ of 35 mmHg.

Hypercapnia is avoidable as a factor aggravating brain injury and must absolutely be corrected if not prevented. Cerebral acidosis or vasodilatation can cause intracranial hypertension and secondary brain damage.

Why not to hyperventilate?

Hyperventilation, which may lead to hypocapnia, is not advisable because cerebral vasoconstriction induced by the drop in blood CO_2 may cause cerebral hypoperfusion; this worsens the already critical situation of diminished blood flow or inadequate oxygen transport.

C = Circulation

Prehospital hypotension, defined as a single observation of a systolic blood pressure < 90 mmHg, has been found to be an independent predictor of outcome (14). In the NeuroLink study (11) patients who did not show episodes of hypoxia and hypertension had 52% of good outcomes with a mortality rate of 19%; patients who simultaneously presented either hypoxia and hypotension had 51% mortality and 18% good outcomes.

What is the optimal target for BP?

The value of 90 mmHg to delineate the threshold for hypotension has arisen arbitrarily and is more statistical than a physiologic parameter (15). In facts no studies have been performed to

corroborate this. In the Italian Guidelines for the treatment of the STBI a systolic pressure higher than 110 mmHg was indicated as the target (10)

Is mean arterial blood pressure a better endpoint than systolic blood pressure?

It may be valuable to maintain mean arterial pressure above those represented by systolic blood pressure (15)

What about sedation of the STBI?

Sedation and analgesia is indicated for STBI. Hypnotics such as propofol and midazolam are probably the most used drugs. Midazolam has a dose related effect (16) and propofol can cause hypotension when administered in boluses in the hypovolemic patient. To avoid these effects older drugs with a lesser effect on hemodynamics are now coming back to a daily use such as etomidate mostly in US and less in Europe (17) and Ketamine whose effects on cerebral blood flow when used alone restricted his role in TBI.

Intravenous lidocaine (1.5 mg/kg) is useful in blunting the increase of ICP with endotracheal intubation (18) but no study demonstrates that this was helpful in making patients' outcome better.

Fentanyl or morphine are the most used drugs for analgesia.

Muscle relaxation could be used to facilitate endotracheal intubation; suxamethonium, rocuronium or vecuronium are the most used drugs.

Sedation/analgesia should be continued using short-acting drugs so that neurological assessments can be made at regular intervals in the ED. Muscle relaxing drugs should be avoided if possible.

How much to rely on the first CT for further evolution and prognosis?

Notwithstanding all these assumptions, prompt diagnosis and early surgical treatment of intracranial masses still remains the central point for the management of TBI. For this reason cerebral CT scan should be performed following protocols that could allow the detection of expanding masses. Servadei (19) has demonstrated that even patients with normal CT scan have a 4% possibility to be complicated by a mass lesion; this percentage increases to a 20% with a diffuse injury IV.

The Italian guidelines for the care of the STBI advise to perform the first CT scan as soon as possible and the second before 12 hours after the trauma if the first CT was done within 3 hours or within 24 hours if done later (10). In STBI an abnormal CT scan has a risk for raised ICP of 50-60% (9).

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Conclusions / Recommendations:

- From the pathophysiological point of view, in STBI brain ischemia is the most relevant pattern. This is especially true in the first 24 following trauma and is not necessarily correlated to cerebral perfusion pressure even if lower systemic arterial blood pressure will worsen the damage.
- STBI patients require aggressive approach in the acute phase for the prevention of secondary insults. Hypoxia and hypotension are the most frequent, important (an preventable) complications.
- Referral to hospitals with neurosurgical facilities should be the gold standard where surveillance, diagnosis and prompt surgical intervention could be provided in case of detection of mass lesion.

Annotated Bibliography

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The paper defined the role of ischemia in the first 24 hours after severe TBI. The mean cerebral blood flow of 186 patients was 22.5ml/100gr/min in the first 6 hours but increased significantly during the first 24 hours. One third of patients in the first 6 hours most of patients had a cerebral blood flow lower than the ischemic threshold of 18 ml/100g/min. The severity of ischemia was correlated with outcome but only in the first 24 hours.

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An italian prospective database collecting data from patients admitted in four hospitals in the area of Milan. The number of patients is relevant and higher than that published by the TCDB. The results are of this study are of the greatest interest.

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Even if published in 1993, this paper defines for the first time the role of hypoxia and hypotension in the determination of prognosis of the severe head injured patient. One of most frequently cited paper in neurotraumatology.

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Even if published in 1999, they represent the most comprehensive document on Guidelines for treatment of the severe head injured patient in the prehospital phase. Free download from the Internet.

Questions

1. In the STBI endotracheal intubation:

- is considered indicated in all patients with a GCS < 9
- the nasotracheal approach is to be preferred because it does not need neck extension
- is indicated in all patients with GCS \leq 13 even if they are able to maintain a normal oxygenation
- is not indicated until a cervical lesion is excluded in the comatose patient

2. The appearance of unequal pupils in an unconscious patient may allow:

- to exclude the presence of a subdural hematoma
- to confirm a lesion of the 5th pair of cranial nerves
- to indicate an immediate CT scan to detect an intracerebral hematoma
- to diagnose an hypoxic cerebral lesion

3. If the first CT of a STBI patient has been performed one hour after the trauma and it was negative and patient anamnesis did not show any risk factor:

- It is not necessary to schedule a new CT scan until the next 24 hours
- A CT scan should be done within the 12 hours after the trauma
- There is no reason to continue sedation and analgesia and to schedule another CT scan
- The program should be based on ICP values trend

4. Brain ischemia after a STBI is always related to:

- Reduction of cerebral perfusion pressure
- To arterial blood hypoxia
- To arterial blood hypercapnia
- To a complex network of pathophysiological chain reaction triggered by the impact

5. All STBI patients should be intubated and ventilated. Which should be the target for paCO_2 ?

- PaCO_2 less than 30 mmHg
- PaCO_2 35 mmHg
- $\text{PaCO}_2 > 40$ mmHg
- PaCO_2 is not important in cerebral flow regulation, the only important target should be PaO_2 control

Answers

1. Answer a.

Endotracheal intubation, if available, is the most effective procedure to maintain the airways that should be secured in all patients with GCS < 9, when there is the inability to maintain an adequate airway or when hypoxemia is not corrected by supplemental oxygen.

2. Answer b.

An unequal pupil is due to the compression of the III cranial nerve. It appears more rapidly and more frequently than signs of involvement of the motor nerves. It could be due also to a peripheral paresis/palsy of the III cranial nerve or to a direct contusion of the ocular bulb but in the comatose patient it should indicate the suspect of an intracranial expanding mass until proven otherwise.

3. Answer b.

It was demonstrated that even if the first CT in a STBI is normal, a percentage of 4% of these patients could develop an intracranial surgical mass that should be evacuated. This percentage increases with worsening of the initial CT

4. Answer d.

Ischemia is the main pathophysiological multifactorial consequence of a STBI especially in the first 24 hours after the trauma. Ischemic patterns of flow are not strictly correlated to the cerebral perfusion and could be present even if arterial pressure is maintained in normal value. If arterial drops the cerebral ischemic damage will obviously deteriorate.

5. Answer b.

PaCO₂ should be maintained around 35 mmHg. Hyperventilation will lower paCO₂ due to cerebral arteriolar vasoconstriction resulting in further worsening of cerebral ischemia.