International Journal of Medical Science in Clinical Research and Review
Online ISSN: 2581-8945
Available Online at http://www.ijmscrr.in Volume 7|Issue 02 (March-April) |2024 Page: 373-376

Original Research Paper

Management of severe traumatic brain injury in the acute phase

Authors:
S. Sadat[1,3], D. Zeghdoud[1,3], D. Bougdal[2,3], A. Djerbal[1,3]
[1]EHS Salim Zemirli, [2]CHU Mustapha Bacha, [3]Algiers Faculty of Medicine
Corresponding Author:
S. Sadat
EHS Salim Zemirli, Algiers Faculty of Medicine

Article Received: 01-January-2024, Revised: 21-January-2024, Accepted: 11-February-2024

ABSTRACT:
Severe cranial trauma (SCT) is a major public health problem, with a high incidence and a high morbidity and mortality rate, mainly affecting young adults. SCT generally occurs as part of a polytrauma; its causes are still dominated by road accidents; clinically defined by the presence of a Glasgow score of less than or equal to 8. Its initial management is based on appropriate resuscitation, which determines its prognosis. The main objective of this management is to prevent secondary cerebral lesions by controlling secondary cerebral attacks of systemic origin; more specifically, the two major attacks, hypoxia and hypotension. Once the patient has been intubated, ventilated and sedated, a brain scan can be used to determine post-traumatic lesions, in order to rule out a neurosurgical indication. It is therefore essential to refer patients with severe head trauma to an intensive care unit with a neurosurgical unit. Once the patient has been admitted to intensive care, specific monitoring is used to control cerebral and systemic haemodynamics. Treatment consists of preventing secondary cerebral lesions from developing. The outcome of SCT depends on the quality of its management, which must begin at the scene of the accident and be codified in regularly updated "GUIDELINES".

Keywords : Severe head trauma - Neurosurgery - Specific monitoring - Secondary brain injury

INTRODUCTION:

Severe cranial trauma (SCT) is a major public health problem. It is a high-incidence condition, with a high morbidity and mortality rate. It is now the leading cause of death in trauma, ahead of haemorrhage [1]. Clinically defined by the presence of a Glasgow score of less than or equal to 8, after normalisation of haemodynamic and respiratory status. In the United States, where autopsies in cases of violent death are the rule, it is estimated that between 40 and 50% of deaths from traumatic causes are due to severe head trauma [2]. In France, there are currently an estimated 155,000 new cases of head trauma each year, including 30,000 serious cases [3], resulting in almost 8,000 deaths each year [4]. While the overall incidence of TBI is constantly falling in developed countries [2], [5] it is exploding in emerging countries. It is estimated that in these countries, in the medium term, one family in 200 will be affected by a relative who has suffered an SCT [6]. SCT generally occurs as part of a polytrauma. The causes of this pathology remain dominated by public road accidents (50-60%), falls (20-30%), sports and leisure accidents (10-20%) and violence and aggression (10%) [7]. The incidence of age-related SCT mainly concerns young adults, generally in good health, with a clear male predominance [3] [8].however, the incidence of SCT in the elderly is increasing due to falls.

1 Management of severe head injuries :

1.1 Pre-hospital care:

Although it is impossible to intervene in the severity of primary cerebral lesions, which are directly linked to the mechanism responsible for the trauma, the initial management of SCT is based on the prevention of secondary cerebral lesions, which are encouraged by numerous factors described as secondary cerebral aggressions. The two most important of these are hypoxia and hypotension, the existence of which is directly responsible for an increase in mortality. The neurological assessment of SCT is based on an evaluation of the state of consciousness, using the Glasgow score, followed by a search for signs of neurological localisation and trunk reflexes. The search for these reflexes will be limited to the photomotor reflex, as any TC is considered to have cervical spine damage until proven otherwise. Recommendations on the management of SCT recommend tracheal intubation and ventilation, from the pre-hospital phase [9]. This is a procedure with a risk of inhalation, carried out in an emergency, and must therefore comply with the principles of rapid sequence anaesthetic induction (Crush induction).

induction is carried out by injecting a hypnotic, etomidate or ketamine, with a recent trend to prefer ketamine to etomidate due to the absence of interaction with the corticotropic axis [10] , [11], associated with a succinylcholine curare. Non-specific monitoring should be applied, consisting of non-invasive blood pressure measurement, in order to maintain a systolic blood pressure [SBP] level >110 mm Hg [11]. [12]. This target should be maintained for the first 48 hours, particularly in the absence of invasive ICP monitoring [13]. As the main cause of arterial hypotension in the prehospital phase is hypovolaemia most often related to haemorrhage, volume expansion is the first of the treatments to be considered, isotonic saline at 9 ‰ is the main solute recommended [14]. When arterial hypotension persists, catecholamines become necessary; the catecholamine of choice is one with a predominant α-adrenergic action, such as noradrenaline at a dosage of (0.125 - 5 µg.kg-1.mn-1). Respiratory control involves monitoring peripheral oxygen saturation and a capnograph; the aim is to maintain SpO2 ≥ 90% and ETCO2 at 35 mm Hg. Temperature monitoring is also essential, in order to combat hypothermia, which is still common in polytrauma patients. To control cerebral haemodynamics, osmotherapy is recommended as an emergency measure in the event of uni- or bilateral areactive mydriasis and/or deterioration in neurological status not explained by an extracranial cause. The aim of osmotherapy is to "buy time". Through an osmotic effect, it reduces ICP and temporarily restores CPAP. Hypertonic saline 7.5% 2-4 cc/kg or mannitol 20% 0.20-1 g/Kg, i.e. 1-5 ml/Kg are effective immediately and over a 2-6 hour period[14]. When choosing the type of osmotherapy, both mannitol and hypertonic saline (HSS) have comparable efficacy in treating intracranial hypertension [15]. Nevertheless, each of them has side effects that need to be considered before use.

1.2 Treatment in the outpatient department:

A reassessment of the three types of distress is essential as soon as the severe head injury is admitted to the outpatient department, in order to continue the treatment already started in the pre-hospital setting and to determine the state of stabilisation of the various types of distress, which will determine the approach to be taken. treatment. A series of first-line radiological investigations are carried out at this level, including chest X-rays, pelvic X-rays and abdominal and thoracic ultrasound [16]. Transcranial doppler is currently one of these investigations [17]. A secondary radiological work-up consisting of a whole-body CT scan is then carried out in stable or stabilised patients. The most universal protocol involves acquisition of the skull and cervical spine without injection, followed by whole-body acquisition with injection of the contrast product. The purpose of the brain scan is to establish a lesion assessment, to rule out an indication for emergency neurosurgical intervention, to look for a fracture of the cervical spine, and for signs of ICH. The formal neurosurgical indications in the acute phase of SCT are [11], symptomatic extradural haematoma whatever its location, significant subdural haematoma (thickness greater than 5 mm with displacement of the midline greater than 5 mm), trimming and immediate closure of an open embrasure, drainage of acute hydrocephalus.

1.3 Intensive care unit management:

Once the patient has been stabilised and the radiological examination has been carried out, whether or not there is an indication for surgery in the emergency, the patient will be transferred to the intensive care unit. The TCG is installed with the chest raised to 30°, allowing the head to be elevated. This prescription is still accepted as a good method of improving venous return and lowering the ICP level. However, it is only indicated for normovolaemic patients [14]. Monitoring of SCT requires the use of specific and non-specific monitoring. Standard monitoring is provided by an electrocardioscope and invasive and non-invasive blood pressure measurement to ensure a SBP of 110 mmHg. This target should be maintained for the first 48 hours, particularly in the absence of invasive ICP monitoring [11] [15]. venous pressure, diuresis, a saturometer to avoid any desaturation SpO2 <90% during treatment, a capnograph to maintain an expired CO2 target (EtCO2) between 30 and 35 mm Hg and a temperature sensor for targeted temperature control between 35°C and 37°. Biological monitoring for the most appropriate glycaemic control between (1.4-1.8 g/dl), a haemoglobin level above 9-10 g/dl, a platelet count above 100,000 cells/mm3 and a prothrombin level above 70% [11] [15]. The aim of specific monitoring is to control cerebral haemodynamics by monitoring intracranial pressure, cerebral blood flow, cerebral oxygenation, cerebral electrophysiology and its metabolites.

1.3.1 Specific monitoring:

1.3.1.1 Intracranial pressure monitoring:

In SCT , measurement of intracranial pressure (ICP) allows early and reliable diagnosis of intracranial hypertension and its evolution, and guides treatment. The association between increased ICP and poor neurological prognosis has been clearly demonstrated since the 1980s [18]. Two main techniques have been proposed. The first is the ventricular route, which is the "gold standard" reference method, and is the oldest technique, practised for many years. since 1951 [19]. It uses a multi-window catheter, implanted in the frontal horn of a lateral ventricle. This is an external ventricular shunt placed by the neurosurgeon, under aseptic conditions in the operating theatre, in a supine patient. The normal ICP is 11 mm Hg, with the

reference zero located at the level of the external auditory canal (projection of Monro's foramen). Cerebrospinal fluid pressure is transmitted by a fluid column to a transducer. Treatment is indicated as soon as the pressure exceeds 20-25 mm Hg. The main disadvantages of this technique are the risk of infection, the risk of haemorrhage and the mechanical risk of obstruction of the shunt by blood or cerebral matter. Its major advantage is that it allows therapeutic subtraction of cerebrospinal fluid (CSF). The second route, the intraparenchymal route, uses a miniaturised transducer implanted in the brain tissue and located at the end of an optical or electrical fibre. This is an excellent alternative when the ventricular route is not accessible. ICP can be omitted when the initial CT scan is normal in a SCT and there are no clinical or transcranial doppler severity criteria [20].

1.3.1.2 Cerebral blood flow monitoring:

1.3.1.2.1 Transcranial Doppler:

Transcranial doppler(TCD) is a non-invasive, easy, reproducible, simple to learn examination, It studied the velocity of red blood cells in intracranial vessels via a beam of ultrasound. The mean velocity measured by the Doppler is an indirect indicator of cerebral blood flow [21]. Doppler is performed using the three physiological acoustic windows of the skull. The temporal window is the most commonly used; it allows exploration of the middle cerebral artery (sylvian artery), which is the most important from a functional point of view, accounting for 60% of cerebral blood flow [22],[23]. This window is located above the zygomatic process, between the external orbital rim and the tragus. This line is divided into three windows: anterior (A), medial (M), where the insonation angle is close to zero, and posterior (P). In young subjects, the temporal window is wide, whereas in older subjects, only the posterior part of the window is permeable. Finally, in some cases, ultrasound does not penetrate the cranium. This is either due to temporal hyperostosis or advanced osteoporosis. By recording a Doppler signal, we can measure the systolic velocity (Vs) at the time of cardiac systole, which corresponds to the envelope of the spectrum at the systolic peak and is a function of systemic arterial pressure and cardiac output, as well as the diastolic velocity (Vd), which is the envelope of the spectrum at the end of diastole and the area under the curve of the envelope spectrum corresponding to the mean velocity (Vm). By measuring these velocities, the main indices can then be calculated, namely the pulsatility index (PI), the resistance index (RI) and the Aaslid index. The severity thresholds used are a PI > 1.4 and diastolic velocities < 20 cm/s [21] [24].

1.3.1.3 Monitoring cerebral oxygenation:

There are three techniques for monitoring cerebral oxygenation: jugular venous oxygen saturation (SvjO2), cerebral tissue oxygen pressure (PtiO2) and near infrared spectroscopy, the aim of which is to determine whether oxygen supply and demand are in balance in order to diagnose cerebral ischaemia at an early stage. These local or regional cerebral oxygenation monitoring techniques can be used to diagnose early episodes of cerebral ischaemia with normal cerebral perfusion pressure (CPP) and/or ICP(s), which may be unrecognised in up to 10% of cases [25].

1.3.2 Sedation of SCT:

The agents used in neurosedation must allow regular assessment of the neurological state, and the products used must therefore be rapidly eliminated. Benzodiazepines are the most commonly used sedatives, in most cases in combination with other sedatives, particularly morphine. Midazolam is often preferred because it has the shortest half-life; in a strategy of deep sedation, the combination of propofol with midazolam is interesting because their effects can be added together, allowing their dosage to be reduced, but lengthening the recovery time; this combination is mainly used to prevent the risk of "protocol related infusion syndrome" (PRIS); its risk factors are a dosage > 5mg/kg/h for a duration of 48 hours [26]. The main advantage of ketamine is that it maintains the haemodynamic state by secreting endogenous catecholamines. Its use in combination with midazolam has been shown to have no side-effects on cerebral haemodynamics.

CONCLUSION:

The management of severe TC in the acute phase begins with pre-hospital care. It aims to control the factors of secondary cerebral aggression, in order to avoid the development of secondary cerebral lesions. A CT scan of the brain on admission of the STC will rule out a neurosurgical emergency and look for signs of intracranial hypertension; management of the STC in a department specialising in neurotraumatology will ensure specific monitoring to control cerebral haemodynamics.

REFERENCES:

[1] Harrois A : Traumatisme crânien : de la physiologie à la pratique. Conférence d'Essentiel Sfar 2016.

[2] Masson F : Épidémiologie des traumatismes crâniens graves .Ann Fr Anesth Réanim 2000 ; 19 : 261-9

[3] Tentillier E, Comte de Luzy S, Reydy F : La prise en charge du traumatisé crânien grave en préhospitalière. Conférence d'Essentiel Sfar 2015.

[4] Thomas GEERAERTS : Mise au point sur la prise en charge du traumatisé crânien grave:

https://www.sciencedirect.com/science/article/pii/S2352580022001009 © published by Elsevier 2022
[5] Bouzat P, Francony G, Oddo M, Payen J-F : Hypothermie thérapeutique en traumatologie crânienne grave. Ann Fr Anesth Reanim 12(2013)787-791.
[6] Tazarourte K, Gauthier A, Foudi L, Dekadjevi H : Prise en charge initiale des traumatismes crâniens graves et monitorage avant l'arrivée en centre spécialisé .Conférence d'Essentiel Sfar 2010. [7] Virour H, Sirairi M, Lubrano V, Geeraerts T : Traumatismes cranioencephaliques .EMC 2013 [8] Fregse M, Tissier C : Prise en charge du traumatisé grave en phase extrahospitalière EMC médecine d'urgence volume 10 n° 3 septembre 2015.
[9] Hamada S, Harrois A, Laplace C, Duranteau J : L'accueil du polytraumatisé en centre spécialisé. Le Congrès Médecins. Les Essentiels Sfar 2013.
[10] Jabre P, Combes X, Lapostolle F, Dhaouadi M, Collaborative Study Group: Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicentre randomised controlled trial. Lancet 2009, 374:293–300.
[11] Mrozek S, Srairi M, Geeraerts T : Traumatisme crânien grave à la phase aiguë. Le Congrès Médecins. Conférence d'Essentiel Sfar 2016.
[12] Berry C, Ley EJ, Bukur M, Malinoski D, Margulies DR, Mirocha J, et al. Redefining hypotension in traumatic brain injury. Injury 2012;43:1833–7.
https://doi.org/10.1016/j.injury.2011.08.014.
[13] Brenner M, Stein DM, Hu PF, Aarabi B, Sheth K, Scalea TM. Traditional systolic blood pressure targets underestimate hypotension-induced secondary brain injury. J Trauma Acute Care Surg 2012;72:1135–9. https://doi.org/10.1097/TA.0b013e31824af90b.
[14] Tazarourte K, Kleitz O, Laribi S, Vigué B : Prise en charge des traumatisés crâniens graves .EMC 2005.
[15] Recommandations Formalisées d'Experts Actualisation des recommandations : Prise en charge des traumatisés crâniens grave à la phase précoce (24 premières heures) /2016.
[16] Bruder N : Monitorage hémodynamique du traumatisé crânien. MAPAR 1999
[17] Jouffray R, Langeron O, Riou B, Vivien B : Prise en charge hospitalière du traumatisé grave adulte au cours des 24premieres heurs. EMC anésthesie réanimation volume 12 n°4 octobre 2015. [18] Srairi M, Silva S, Lonjaret L, Fourcade O, Geeraerts T : Monitorage du système nerveux central en réanimation Congrès Médecins. Conférence d'Essentiel Sfar 2012.
[19] Ledouxa D, Piret B-S, Boveroux P, Bruno M-A, Vanhaudenhuyse A : Les échelles d'évaluation des états de conscience altérée, P. Boveroux, M-A. Bruno, A. Vanhaudenhuyse, Réanimation (2008) 17, 695—701.
[20]Geeraerts T, Velly L, Abdennour L, Asehnoune K, Audibert G, Bouzat P, et al. Management of severe traumatic brain injury (first 24hours). Anaesth Crit Care Pain Med 2018;37:171–86.
https://doi.org/10.1016/j.accpm.2017.12.001
[21] Geeraerts T, Racta C, Vigué B : Le doppler transcrânien en neurochirurgie. Neurochirurgie (2008) 731–738.
[22] Boishardy N, J .C Granry: Intérêt du doppler transcrànien dans la prise en charge des traumatisés crâniens graves. Ann Fr Anesth Rén, 13: 172-176, 1994 .
[23] Corre M, Sapir D, Tazarourte K : Doppler transcrànien en médecine d'urgence .Le Congrès Médecins. Urgences vitales 2012.
[24] Cattin F, Bonneville J-F : Doppler transcrânien : technique et résultats normaux. EMC Radiologie et imagerie médicale 2009 30-890-A-20.
[25] Minassian A.T : Monitorage du traumatisé crânien grave durant les 24 premières heures. Ann Fr Anesth Réanim 2000 ; 19 : 308-15.
[26] Payen J-F, Francony G : Neurosédation en réanimation. Ann Fr Anesth Reanim 28 (2009) 1015- 1019.