THE MANAGEMENT OF CHILD WITH SEVERE HEAD INJURY: OUR EXPERIENCE

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Objectives: To analyze the children admitted to our Pediatric Intensive Care Unit (PICU) for severe neurotrauma and describe the management algorithms adopted by us for pediatric head injury. Methods: All the children affected by severe head injury and admitted to PICU since November 1992 to November 2000 have been examined. Injury severity has been classified using the Glasgow Coma Score (GCS), while the long term neurological outcome with the Glasgow Outcome Score (GOS). We have described the clinical presentation, the kind and dynamics of injury, the management prior to our admission, the presence of post-traumatic seizures and, finally, the clinical outcome one month after discharge. Results: 122 children with head injury have been identified. The most frequent cause of injury has been represented by car accidents and motorbike or bicycle falls. Patients with severe head injury have presented the following scores: GOS 1 (dead children) 14.7%, GOS 2 (persistent vegetative state) 1.6%, GOS 3 (severe disability) 22.2%, GOS 4 (mild disability) and GOS 5 (no disability) 61.5%. Conclusion: A correct management of children with head injury helps clinicians to improve outcome and to reduce mortality. Therapeutics algorithms suggested by us could be useful for the management of this kind of patients.

Key words: severe head injury - childhood - therapeutics algorithms

Introduction

Head injury is the leading cause of death among children older than one year of age (1,2). Mortality, due to head injury, has been reported to be approximately 15% among children in the United Kingdom and 33-45% in the United States (3,4). In Italy, 1 in 600 children die from head injury (2,5). Mortality has also been reported to be higher among male, with respect to female children (4), with the reported male to female ratio ranging from 2:1 to 4:1 (4). The most common cause of head injury, in pediatric age, is car accidents (4,6), while child abuse and falls constitute other important causes, especially among children less than 5 years of age (4,7). Head injury is responsible for both primary and secondary brain damage, and long-term complications can be causes of invalidity. The outcome of these patients greatly depends on the extent and nature of primary damage and on the effectiveness of therapy for preventing, or limiting, secondary brain damage (8-11).

Hypoventilation and hypotension are major early complications of aggravation of secondary brain damage, that are typically accessible to emergency maneuvers at the scene. Hypoventilation is noted in 50% of comatose patients and will still be present upon admission in 40% of the children who will not benefit from adequate scene management, by emergency tracheal intubation (12). The other major point of concern during the initial management is the prevention or stabilization of hypotension, frequent and multifactorial in these patients. Whatever the cause of hypotension, it must be corrected as soon as possible, by isotonic glucose-free fluids, since it has major noxious consequences for cerebral perfusion pressure and for avoiding the cerebral metabolic acidosis due to post-traumatic hyperglycaemia (13).

The fear of such complications has led to a general policy of "scoop and run" transport to the next available hospital, with 50-75% of the children not being intubated until they reach the hospital. Thus, it is crucial that patients with head injury must be treated by specialized personnel and facilities. With the overwhelming evidence that shock dramatically increases incidence of brain swelling and increased mortality, a causal link between systemic insult and the progression of brain injury was established for the first time. In this report we have described our experience on the management of the children with severe head injury and proposed therapeutics algorithms for these patients.
Materials and Methods

In recent years the Brain Trauma Foundation, in cooperation with the American Association of Neurological Surgeons, developed a set of guidelines for the treatment of neurotrauma (14-16). Guidelines 1 to 11 list the priorities established in the guidelines. Guidelines 1 and 2 delineate appropriate principles of resuscitation. Guidelines 3 and 4 describe indications for ICP monitoring and their significance in the rational management of head-injured patients. The importance of intracranial hypertension in determining the outcome of head-injured patients was demonstrated in a recent clinical trial (17). Patients with lesions that are contiguous to the brain-stem are at risk from ICP levels lower than 20 mmHg. The treatment of ICP should be based not only on frequent clinical examinations and cerebral perfusion pressure (CPP) data, but also on the results of computed tomographic scanning. Guideline 5 shows the possible way for the management of CPP. The appropriate guideline for maintenance of CPP is controversial. In children CPP levels range from 45 to 65 mmHg. Guideline 6, on the use of hyperventilation, is perhaps the most controversial. The guideline well states that an arterial carbon dioxide pressure of 3.3 kPa should be avoided after severe brain injury. The problem comes with more moderate levels of hyperventilation. The measurements of brain jugular oxygen tension and arteriovenous differences in oxygen corroborate the potential risk of chronic hyperventilation. A recent work demonstrates a severe blunting of the response to hyperventilation of the injured brain and this finding suggests that moderate hyperventilation, in situations in which intracranial hypertension is difficult to treat, might be appropriate and relatively risk-free (18). Thus, the role of more moderate hyperventilation (4.4-4.7 kPa PaCO2) needs further delineation. Guidelines 7 to 9 show the use and the role of mannitol, barbiturates and glucocorticoids in the treatment of severe brain injury. Guideline 10, on the nutritional support of brain-injured patients, is very important. There is the strong support for the statement that feeding must be instituted by the end of the first week, if at all possible. Guideline 11, on the role of the drugs for prophylaxis of the post-traumatic seizures, remains an area of continuing discussion, although no contrary evidence is in the summary statement. In our institution for the prophylaxis of the early post-traumatic seizures phenytoin is used according to the protocol showed in Guideline 12. Based on these guidelines and the new international recommendations, the treatment of severe pediatric brain injury in our PICU follow the flow chart showed in Guidelines 13 and 14. All patients admitted in our PICU with severe brain injury were analyzed. The severity of brain injury was assessed by Glasgow Coma Score (GCS). One month after the injury, the outcomes were evaluated with the Glasgow Outcome Score (GOS): GOS 1 (dead children), GOS 2 (persistent vegetative state), GOS 3 (severe disability), GOS 4 (mild disability) and GOS 5 (no disability). Age, gender, modality and type of injury, type of medical care received at peripheral hospital and post-traumatic seizure incidence were reported.

Guideline 1

The integration of brain-specific treatments into the initial resuscitation of the patient with severe head injury

The fundamental goals of resuscitation of the head-injured patient are the restoration of circulating volume, blood pressure, oxygenation and ventilation. The physician should initiate maneuvers that serve to lower ICP and do not interfere with these aims as early as possible during resuscitation of any patient with a head injury. Treatment modalities, such as hyperventilation and mannitol administration, that have the potential of exacerbating intracranial ischemia or interfering with resuscitation should be reserved for patients who show signs of intracranial hypertension, such as evidence of herniation or neurological deterioration.

Guideline 2

Resuscitation of blood pressure and oxygenation

Early post-injury of hypotension or hypoxia greatly increase morbidity and mortality from severe head injury. At present, the proper definition of hypotension and hypoxia is unclear in these patients. However, ample Class II evidence exists regarding hypotension, defined as a single observation of a systolic blood pressure lower than the fifth percentile by age for at least 15 minutes, to warrant the formation of guidelines stating that these values must be avoided if possible, or rapidly corrected in severe head injury patients. Strong Class II evidence suggests that raising the blood pressure in hypotensive severe head injury patients improves outcome in proportion to the efficacy of the resuscitation. During resuscitation and the early hours of stabilization in children with severe head injury isoflurane glucose-free solutions are recommended to avoid the worsening of cerebral metabolic acidosis.

Guideline 3

Indications for intracranial pressure (ICP) monitoring

Guideline: ICP monitoring is appropriate in patients with severe head injury with an abnormal admission CT scan. Severe head injury is defined as a GCS score of 3-8 after cardiopulmonary resuscitation. An abnormal CT scan of the head is one
that reveals hematomas, contusion, edema or compressed basal cisterns. ICP monitoring is appropriate in patients with severe head injury with a normal CT scan if two or more of the following features are noted at admission: age < 2 years, unilateral or bilateral motor posturing, systolic blood pressure < than the fifth percentile by age for at least 15 minutes. ICP monitoring is not routinely indicated in patients with mild or moderate head injury. However, a physician may choose to monitor ICP in certain conscious patients with a traumatic mass.

Summary: ICP monitoring per se has never been subjected to a prospective, randomized clinical trial to establish its efficacy in improving outcome from severe head injury. Hence, there are insufficient data to support its use as a standard. However, there is a large body of published clinical experience that indicates that ICP monitoring: 1) helps in the earlier detection of intracranial mass lesions; 2) can limit the indiscriminate use of therapies to control ICP, which themselves can be potentially harmful; 3) can reduce ICP by cerebrospinal fluid drainage and thus improve cerebral perfusion; 4) helps in determining prognosis; and 5) may improve outcome. ICP monitoring is accepted as a relatively low-risk, high-yield, modest-cost intervention.

Guideline 4

Intracranial pressure treatment threshold

Guideline: ICP treatment should be initiated at an upper threshold of 2.7 kPa.

Options: Interpretation and treatment of ICP based on any threshold should be corroborated by frequent clinical examination and CPP (MAP – ICP) data.

Guideline 5

Guidelines for Cerebral Perfusion Pressure (CPP)

Guideline: There are insufficient data to support treatment guidelines for this topic.

Options: CPP, in children, should be maintained at values 5.3-8.6 kPa.

Summary: Maintenance of a CPP > 9.3 kPa is a therapeutic option that may be associated with a substantial reduction in mortality and improvement in quality of survival and is likely to enhance perfusion to ischemic regions of the brain after severe traumatic brain injury. No study has demonstrated that the incidence of intracranial hypertension, morbidity or mortality is increased by the active maintenance of CPP above 9.3 kPa, even if this means normalizing the intravascular volume or inducing systemic hypertension.

Guideline 6

The use of hyperventilation in the acute management of severe brain injury

Standards: In the absence of ICP, chronic prolonged hyperventilation therapy (PaCO₂ of ≤ 3.3 kPa) should be avoided after severe brain injury.

Guidelines: The use of prophylactic hyperventilation (PaCO₂ ≤ 4.7 kPa) therapy during the first 24 h after severe brain injury should be avoided because it can compromise cerebral perfusion during a time when CBF is reduced.

Options: Hyperventilation therapy may be necessary for brief periods when there is acute neurological deterioration or for longer periods if there is intracranial hypertension refractory to sedation, paralysis, cerebrospinal fluid drainage, and osmotic diuretic. SjO₂, AvdO₂ and CBF monitoring may help to identify cerebral ischemia if hyperventilation, resulting in PaCO₂ values of < 4 kPa, is necessary.

Summary: Chronic prophylactic hyperventilation therapy should be avoided during the first 5 days after severe brain injury, particularly during the first 24 h. CBF measurements in children with severe brain injury demonstrate that blood flow early after injury is low and strongly suggest that in the first few hours after injury the absolute value approach those consistent with ischemia. Hyperventilation will reduce CBF values even further but will not consistently cause a reduction of ICP and may cause loss of auto regulation. The cerebrovascular response to hypocapnia is reduced in those with the most severe injuries (subdural hematomas and diffuse axonal injury), and there is substantial local variability in perfusion. Although the CBF level at which irreversible ischemia occurs has not been clearly established, ischemic cell change has been demonstrated in 90% of those who die after brain injury, and there is positron emission tomographic evidence that such damage is likely to occur when CBF drops below 15-20 ml/100 g/min. A prospective randomized clinical trial has determined that outcomes are worse when traumatic brain injuries patients are treated with chronic prophylactic hyperventilation therapy.
Guideline 7  
**The use of mannitol in severe head injury**

Guideline: Mannitol is effective for control of raised ICP after severe head injury. Limited data suggest that intermittent boluses may be more effective than continuous infusion. Effective doses range from 0.25 to 1 g/kg body weight, in 15/20 minutes infusion.

Options: 1) Indications for use of mannitol before ICP monitoring are signs of transtentorial herniation or progressive neurological deterioration not attributable to systemic pathology; 2) serum osmolarity should be kept below 320 mOsm/L and na-triemia should be maintained at values of 140-145 mEq/L and 3) eu-volemia should be maintained by adequate fluid replacement. A Foley catheter is essential in these patients.

Summary: Mannitol is effective in reducing ICP, and we recommend its use in the management of traumatic intracranial hypertension. Serum osmolalities of > 320 mOsm/L and hypovolemia should be avoided. Some data suggest that bolus administration is preferable to continuous infusion.

ICP and decreasing mortality in the setting of uncontrollable ICP refractory to all other conventional medical and surgical ICP-lowering treatments. The use of barbiturates for the prophylactic treatment of ICP is not indicated. The potential complications attendant on this form of therapy mandate that its use be limited to critical care providers and that appropriate systemic monitoring (i.e. transcranial doppler sonography) be undertaken to avoid or treat any hemodynamic instability.

Guideline 8  
**The use of barbiturates in the control of intracranial hypertension**

Guidelines: High-dose barbiturate therapy may be considered in hemodynamically stable, salvageable severe head injury patients with intracranial hypertension refractory to maximal medical and surgical ICP-lowering therapy.

Summary: High-dose barbiturate therapy is efficacious in lowering any method of feeding is better than another or that early feeding before 7 days improves outcome. Based on the level of nitrogen wasting documented in head-injured patients and the sparing effect of feeding, it is a guideline that full nutritional replacement be instituted by the 7th day.

Guideline 9  
**The role of glucocorticoids in the treatment of severe head injury**

Standards: The use of glucocorticoids is not recommended for improving outcome or reducing ICP in patients with severe head injury.

Summary: The majority of available evidence indicates that glucocorticoids do not lower ICP or improve outcome in patients with severe head injuries. The routine use of glucocorticoids is not recommended for these purposes.

Guideline 10  
**Nutritional support of brain-injured patients**

Summary: Data show that starved head-injured patients lose sufficient nitrogen to reduce weight by 15% per week. Data in patients who are not head-injured show that a 30% weight loss increases the mortality rate. The data strongly support feeding, at least by the end of the 1st week. It has not been established that any method of feeding is better than another or that early feeding before 7 days improves outcome. Based on the level of nitrogen wasting documented in head-injured patients and the sparing effect of feeding, it is a guideline that full nutritional replacement be instituted by the 7th day.

Guideline 11  
**The role of antiseizure prophylaxis after head injury.**

Standards: Prophylactic use of phenytoin, carbamazepine or phenobarbital is not recommended for preventing late post-traumatic seizures. On the other hand, there are few data that deal with the prophylaxis of the early post-traumatic seizures.

Summary: The majority of studies do not support the use of the prophylactic anticonvulsants studied thus far for the prevention of late post-traumatic seizures. Routine seizure prophylaxis later than 1 week after head injury is therefore not recommended.

Guideline 12  
**Use of phenytoin for prophylaxis of early post-traumatic seizures.**

In all children initial bolus of 15 mg/kg in 30 minutes.

Phenytoin doses:
- Child 0-6 years 10 mg/kg in 4 daily doses for 1 week
- Child 6-14 years 6 mg/kg in 4 daily doses for 1 week

During infusion of phenytoin monitoring heart rate and blood pressure.
Results

During eight years of clinical activity we had 122 children with severe brain injury (GCS ≤ 8), 77 males and 45 females. The mean age was 7.8 ± 6.2 years. The more frequent etiology of brain injury were: car accidents (56 cases), motorbike accidents (22 cases), bike accidents (14 cases), pedestrian accidents (8 cases), domestic accidents (12 cases), falls and sports trauma (7 cases) and child abuse (3 cases). Pervious hospitals attempted endotracheal intubation in 65% of children while only 12% were transported to our unit with a correct cervical spine stabilization. The main factors that influence the children’s outcome were early complications, such as hypoxia and hypotension. Multivariate analysis showed that these complications, independently to early GCS (severity of the trauma), were statistically significantly associated to outcome. In children with poor outcome (GOS ≤ 3), the incidence of early complications was 68% (86% in deceased children), in children with GOS 4-5 (good outcome) the incidence of early complications was only 14%. Epidemiological analysis showed that only 3 over 56 children injured in car accidents, were placed in safe belt seats, and 10 over 22 children injured in motorbike accidents, wore helmets. In the bike accidents none of the children injured had a helmet. The more frequent brain lesions were: mild to severe cerebral edema (52 children), diffuse axonal injury (6 children), epidural/subdural hematoma (33 children), subarachnoid haemorrhage (9 children), intraparenchimal haemorrhage (22 children). Fracture of cranial vault were assessed in 67 children: 88% were linear fractures and the other 12% were depressed fractures. The eighteen children (14.7%) with early seizures showed the worst outcome: 53% had a GOS ≤ 3, in comparison with 19.1% of children without seizures (p<0.01). The outcome of all studied patients was: GOS 1 14.7% (18/122); GOS 2 1.6% (2/122); GOS 3 22.2% (27/122). The 61.5% (75/122) reached a GOS of 4-5.

Discussion

Pediatric head injury data are often heterogeneous due to the few trials and the different modality of treatment. Our data show that head injury is more frequent in males than...
in females (63.80% vs. 36.20%), although no differences in outcome has been found. Car, motorbike and bike accidents are the most frequent causes of trauma, while in comparison to other studies, child abuse was rare (only 3 cases over 122) (4,7). In the United States the prevention programs adopted in the last years obtained a standardized management of neurotrauma patients, and a significantly lower head injury mortality was recorded (1). Our data showed that protection devices (belt seat, helmet etc.) are still less used, because only a small percentage (respectively 3 and 10) of children wore these devices. This undermarks how important would be a specific primary prevention in our country. Also the initial assistance and treatment of neurotrauma patients showed incorrect approaches: only 65% of these children have been stabilized and intubated before their arrival in PICU. Early complications, such as post-traumatic hypoxia and hypotension significantly worsened outcome, because 68% (86% in deceased children) of patients with GOS ≤ 3 had these complications in comparison with 14% of children with GOS 4-5. This association has been well documented in adults (20-22), while few data exist in pediatrics (23,24). Sharples and coll. recently highlighted that hypotension, associated to hypoxia, triple and quadruple, respectively, mortality of head injured children (25). Confirming this finding, our results underline the importance of focusing many efforts in the early and accurate treatment of these complications. The 14.7% of children had early post-traumatic seizures. Incidence of these in literature ranges between 9 and 15% (26,27). Post-traumatic seizures were associated to GCS, to type of brain lesions and to children age (19) and worsened secondary brain damage (28).

In our study children with post-traumatic seizures had worse outcome in comparison with children without seizures. The 53% of children with seizures showed a poor outcome (GOS ≤ 3), while only 19.1% of children without seizures had a poor outcome (p < 0.01). We believe that management of children with severe brain injury (GCS ≤ 8, depressed fractures of cerebral vault, blood in cerebral parenchyma) should include prophylaxis of early post-traumatic seizures, as represented in Guideline 12 (29).

In conclusion, evaluation of final outcome in our patients showed good results: percentage of good outcome was 61.5% (GOS 4-5), only 16.3% of children (GOS 1 and 2) deceased or survived with persistent vegetative state, while 22.2% showed severe neurological impairment (GOS 3). According to international literature, our results were obtained following therapeutic algorithms and standardized guidelines. A correct stabilization and sedation, an accurate monitoring of intracranial pressure, a prophylaxis of early post-traumatic seizures, a corrected use of osmotic diuretics, a constant monitoring of body temperature, a rigid metabolic and glycemic control are all procedures able to ameliorate outcome of children with neurotrauma, either as mortality either as neurologic impairment.

Although in recent years many new therapeutical approaches have been developing to treat children with neuro-trauma (cerebral microdialysis, O2 free radicals "scavengers", cerebral hypothermia etc.), the application of accurate therapeutic and diagnostic flow-chart could improve management and outcome of these patients, decreasing their neurologic impairments and social cost.
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LJIEČENJE DIJETETA S TEŠKOM OZJEDOM GLAVE: NAŠE ISKUSTVO

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