PEDIATRIC TRAUMATIC BRAIN INJURY: PATHOPHYSIOLOGY, TREATMENT AND CASE REPORTS

VICENȚIU SĂCELEANU[1], COSMIN CÎNDEA[2], ADRIANA SĂCELEANU[3]

[1,3] "Lucian Blaga" University of Sibiu, [1,2] Clinical Emergency Hospital Sibiu

Keywords: *pediatric trauma, traumatic brain injury, TBI, epidural hematoma, diffuse axonal injury, decompressive craniectomy,*

Abstract: *Traumatic brain injuries are the leading causes of morbidity and mortality in children, most frequently caused by car crushes and accidental falls. The treatment focuses on limiting the primary lesions and minimizing secondary lesions; haemorrhagic shock may rapidly occur in infants and young children. A Paediatric Glasgow Come Score lower than 9 points suggests a severe brain injury, therefore accurate and rapid clinical and neuro-imagistic diagnosis is the key of success. The extradural hematoma and scalp hematoma are sufficient enough to produce anaemia and shock as the total blood volume is lower in infants. So they need early surgical intervention for extradural hematoma and blood volume replacement. Tight dural closure is the key step in surgical management. Duraplasty can be done with a patch of pericranium / periosteum. Cranioplasty is not indicated in infants. The Paediatric Neurosurgical Department and Paediatric Intensive Care Unit represent also a vital necessity, as long time follow–up is mandatory.*

INTRODUCTION

Traumatic brain injuries are among the main causes of morbidity and mortality in children. The most common unintended causes are road accidents and falls, aggressions and abuses. Statistics show that boys seem to be affected twice by the rate of girls.(1)

The therapeutic target in the management of these lesions focuses on limiting the progression of primary lesions and minimizing secondary lesions.

Pathophysiology

Primary brain lesions:

1. Appear as an immediate consequence of a traumatic agent.
2. Linear forces generate focal lesions such as intracranial haemorrhages and contusions (brain parenchyma strikes skeletal prominences resulting in lesions at the site of the stroke but also counter-kick injuries on the opposite site)
3. The clinical symptomatology of traumatic brain injuries is closely related to the location and severity of the trauma.
4. Acceleration-deceleration lesions can also generate angular forces, resulting in axonal shear (primary axotomy). These lesions can be suspected in children whose neurological deficit is not correlated with the apparently minor lesions seen on computed tomography (CT) Scan.(2)

Secondary brain damage

- Appear immediately or late, in two forms:

First form includes metabolic alterations such as hypoxemia, hypotension, increased intracranial pressure (ICP) and Intracranial hypertension, (ICH), hypercapnia or hypercapnia, hyper / hypoglycaemia, electrolyte imbalances, expanded hematoma, coagulopathy, epileptic seizures, and hyperthermia.

This form can be avoided by treatment. It is currently the main target in the acute management of traumatic brain injuries.

The second form includes the sequence of cellular events that lead to neurodegeneration and ultimately to neuronal death. The involved mechanisms are: cerebrovascular barrier alteration, diffuse axonal lesions, necrosis, apoptosis, and inflammation. Although serious research is being undertaken in this area, there is no current treatment for this form of secondary injury.

The Monro-Kellie Doctrine:

The intracranial volume is inextensible, being composed of a fixed volume of brain, cerebrospinal fluid (CSF) and blood. Any increase of a compartment must be offset by the decrease of another compartment.

Secondary cerebral lesions cause cerebral edema, immediate compensatory mechanism is deployment of CSF in the spinal canal and removal of venous drainage into jugular veins. These two reactions decrease ICP but once surpassed, even a small increase in cerebral edema or intracranial volume causes a serious increase in ICP, which compromises cerebral perfusion, causing ischemia, which in turn will increase brain edema, culminating in cerebral herniation and death.

Clinical and Imagistic Aspects

Patients with intracranial lesions, with ICP and ICH, may initially present headaches and vomiting symptoms but with rapid alteration of mental status and obnubilation. A GCS (Glasgow Coma Score) of less than 9 points suggests a severe brain injury.(2) GCS >12 minor, 9-12 moderate <9 severe.

Table no. 1. Paediatric Glasgow Coma Scale (3)

Eye Opening	Best Verbal Response	Best Motor Response
4 Spontaneous	5 Coos, babbles	6 Normal Spontaneous Movement
3 To speech	4 Irritable, cries	5 Withdraws to touch
2 To pain	3 Cries to pain	4 Withdraws to pain
1 None	2 Moans to pain	3 Abnormal flexion
	1 None	2 Abnormal extension
		1 None

If ICP continues to grow, cerebral herniation syndrome may occur: Cushing triad: irregular breathing,

[1]Corresponding author: Adriana Săceleanu, Str. Corneliu Coposu, Nr. .2-4, Sibiu, România, E-mail: vicentiu.saceleanu@gmail.com, Phone: +40700 22931

Article received on 17.06.2018 and accepted for publication on 31.08.2018
ACTA MEDICA TRANSILVANICA September 2018;23(3):48-51

bradycardia, hypertension; epileptic seizures and cranial nerve damage may also occur as a consequence of brainstem compression.(3)

ICP imaging signs include change in the position of the median line and diminishing the size of the ventricular system (CT).(4,5)

Magnetic resonance imaging (MRI) can provide useful information only after the patient is stable.

Treatment and Management

The first intention is to identify primary lesions and prevent or treat secondary lesions. There are a number of treatable parameters that can exacerbate secondary cerebral lesions:(6)

- Hypoxemia
- Hypotension
- ICP / ICH
- Hyper / hypocapnia
- Hydroelectrolytic abnormalities
- Growing hematomas
- Seizures
- Hyperthermia

Primary interventions:

Stabilization begins with the application of basic resuscitation measures: airway security, oxygen therapy and adequate ventilation, rapid prevention or treatment of hypotension.

Hypercapnia and hypoxia should be avoided - they are brain vasodilators that increase cerebral blood flow and thus intracranial volume, increasing ICP and ICH. Orotracheal intubation helps not only to protect the airways but also to the better control of ventilation and oxygenation.

All efforts must be made to avoid hypotension because it has been shown to increase mortality and morbidity.

Raising the head to 30 degrees to lower venous obstruction can help control ICP.

Post-traumatic hyperthermia is not uncommon in patients with Traumatic Brain Injury (TBI). Fever increases the brain metabolism and oxygen need, promoting ICH. Besides, fever lowers the epileptic trigger.

Sedation and analgesia is also important for minimizing ICP. Stress and painful stimuli increase blood pressure and thus ICP.

The first CT should be done after initial resuscitation, but it should also be used whenever neurological damage and increase of ICP that is not related to medical treatment appears.(7)

Intracranial pressure monitoring: The general consensus is that a ICP of less than 20mmHg and a cerebral perfusion pressure (CPP) of 40mmHg should be maintained.

CSF drainage: generally used in case of hydrocephalus, it is used in this case for ICP monitoring; CSF elimination reduces total intracranial volume, which lowers ICP and improves CPP.(3)

Neuromuscular blockade: if initial manoeuvres are unsuccessful. It works by lowering metabolic and oxygen demand, promoting cerebral venous return by decreasing intrathoracic pressure, facilitating ventilation and oxygenation, eliminating respiratory-ventilator asynchrony.

Hyperosmolar therapy: Hypertonic saline solutions have been shown to be useful in the treatment of ICH in children with traumatic brain injury. The minimum dose required maintaining ICP below 20mmHg and serum osmolarity below 360mOsm / L should be used. Hypertonic saline solutions have an advantage over mannitol in hypovolemic patients.(4)

Hyperventilation: Reduces ICH by reflex vasoconstriction in the presence of hypercapnia. Vasoconstriction decreases cerebral blood flow, thus lowering ICP. It is one of the fastest methods of lowering the ICP for the child in imminent herniation. A potential danger associated with hyperventilation is cerebral ischemia. Avoidance of hyperventilation prophylaxis below 30mmHg should be considered in the first 48h.

Barbiturates: in high doses are used for refractory ICH. It lowers cerebral metabolism, lowers blood brain volume and inhibits exotoxicity. The minimum dose required to control ICH should be used. Disadvantages would be the impossibility of evaluating the neurological patient.

Temperature control: It is known that hyperthermia exacerbates neuronal damage. Therefore, in many clinical trials, the effects of therapeutic induced hypothermia have been investigated in these patients. One of the latest, "The cool kids trial", a multicentre international study, concluded that moderate hypothermia (32-33, Celsius) applied in the first 8h after a severe cerebral injury, up to 48h may help reduce ICH. Careful! The warming can be done only with a slower increase of 0.5 degrees Celsius per hour to prevent possible complications: arrhythmias, electrolyte abnormalities, haemorrhages and increased risk of infection or sepsis.

Decompressive craniectomy: It is always accompanied by duroplasty with the removal of the bone flap, and it may be considered in patients who show signs of neurological deterioration, herniation, or refractory to the medical treatment of ICH. After the failure of conservative therapy, bilateral decompressive craniectomy may be needed as the ultimate resort treatment strategy. In children, decompressive craniectomy improves the neurological outcome and diminishes death risk. The need for decompressive craniectomy and duraplasty is particularly important in children due to the lack of response to conventional therapies in the treatment of intracranial hypertension. The surgical technique for diffuse brain swelling: wide bilateral hemicraniectomy, dural opening in a stellate fashion, dural graft to increase the available volume before closing, and finally wound closure.(8)

Prophylaxis of epileptic seizures: it is widely accepted that all post-traumatic epileptic seizures should be treated as they contribute to hyperthermia and ICH. A therapeutic option is to administer the prophylaxis of Phenitoin to children who have suffered severe brain injury.(9)

CASE REPORT

Epidural Haematoma in a 7-month-old child

Patient R. I. 7 months, hospitalized after falling of the bed (approx. 1m height) with irritability, bulging fontanel, pallor, Paediatric GCS= 8 points.

Ex CT Scan cranio-cerebral highlights:

- Right F-T-P epidural hematoma
- Right T linear fracture

Treatment

Neurosurgical emergency evacuation, due to the CT appearance and the fact that the pGCS (Pediatric GCS) was lower with 2 points in 2 hours.

- Right T-P Flap.
- Evacuation of the F-T-P right extradural hematoma
- Haemostasis, identifying and stopping the bleeding source.
- Levelling the fracture.
- The bone flap was put in place

Suture of all the anatomical layers and placing a sub fascial drainage tube

Postoperative

- Very good neurological evolution.

Postoperative CT scan highlighted evacuation of epidural collection F-T-P right and the levelling of the fracture.

Figure no. 1. CT Scan in ED

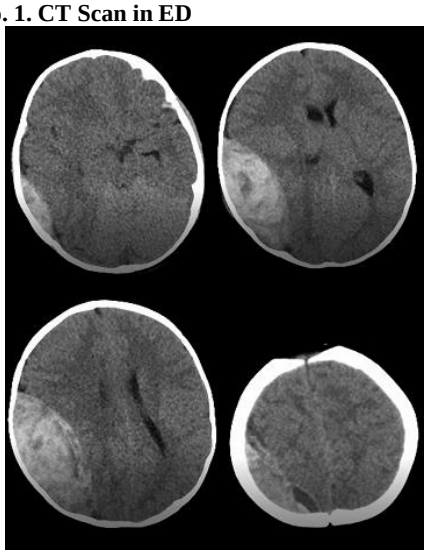


Figure no. 2. Hematoma evacuation surgery

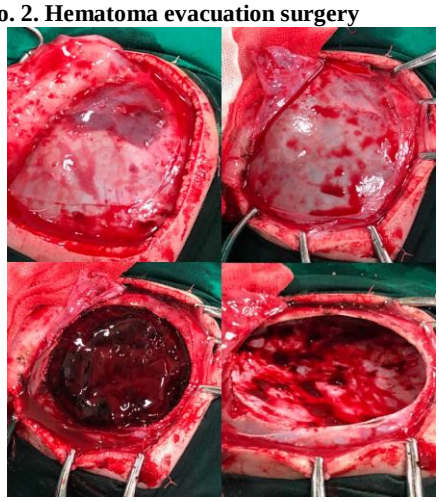


Figure no. 3. CT Scan post evacuation

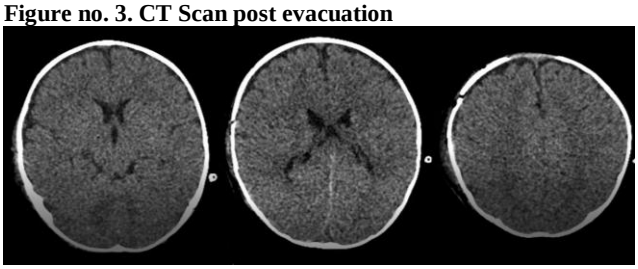
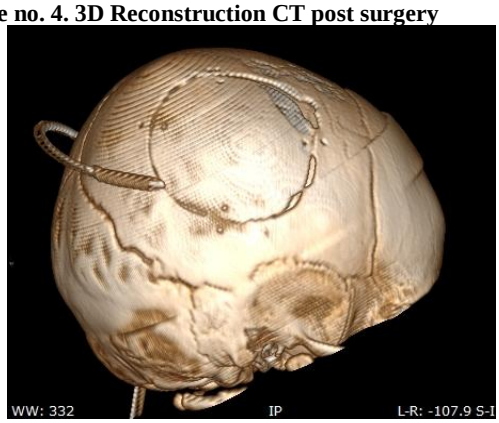


Figure no. 4. 3D Reconstruction CT post surgery



- The patient was discharged conscious, without intracranial hypertension syndrome, without any complaints of painful nature, no motor or other deficits.

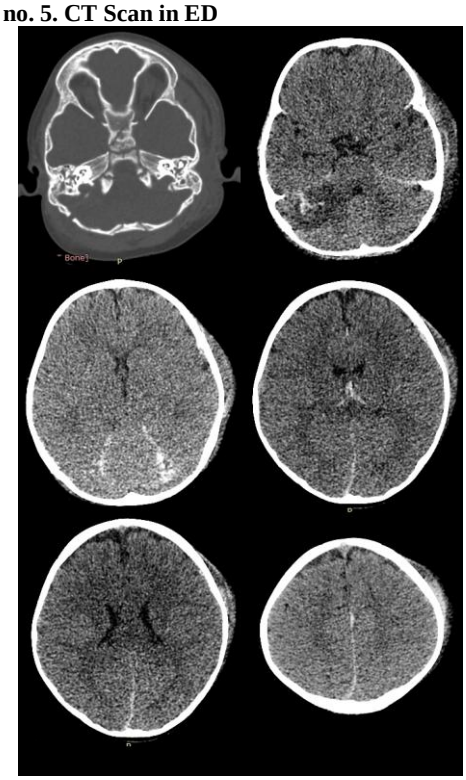
Multiple Severe Brain Trauma in a 2-Year-Old Child

The present report focuses on a case of a 2-year-old girl, who was victim of a car accident. The neurological evaluation in the Emergency Department found the child in deep coma (pGCS 6points) and she was sent to ICU (intensive care unit).

CT Scan in emergency showed:

- subdural right FTP haematoma
- small left frontal epidural haematoma
- subarachnoidian haemorage intra and supratentorial
- intraventricular haemorage
- important brain edema
- multiple skull fractures.

Figure no. 5. CT Scan in ED



Three days later, CT Scan examination showed a favourable evolution of brain lesions, maintaining the cerebral concussions and subarachnoidian haemorrhage. Despite the positive imagistic evolution, the patient's neurologic status was slowing improving. The coma and tracheal intubation was maintained.

The patient was discharged in the department of Infantile Surgery where, for the next two weeks, her neurological deficit reduced, being conscious, able to eat by herself and capable to start the programme of neurorehabilitation.

DISCUSSIONS

The most important therapeutic resource in the treatment of paediatric patients with traumatic brain injury should be to stop the progression of the primary lesion and minimize the secondary lesions, all applied in a shorter time sequence.(10)

To quantify the degree of cerebral damage, pGCS is

used, a score below 9 points suggesting a severe cerebral lesion. To gain time, a very important aspect is the urgent use of CT examination; the MRI being kept only for stable patients.

In the supportive treatment of intensive care, attention is focused on the correction of hypoxemia, hypotension, decrease of ICP, correction of hypo/hyperglycemia, hypo/hipercapnia, electrolyte imbalances, prophylaxis of epilepsy seizures and hyperthermia.

Other recent promising research (11) focused on the role of thrombin, iron or TNF-α in the pathogenesis of secondary lesions in children with TBI. It has been shown that large amounts of thrombin released by cerebral haemorrhage have neurotoxic effects and thrombin at low levels would have beneficial effects in neuroregeneration and neuroplasticity. Iron resulting from hematoma lysis increases cerebral edema and is neurotoxic. Inhibition of TNF-α may have beneficial effects in the neuroreabilitation of the paediatric patient with TBI.(12)

Due to our experience, focal cerebral lesions, after removal of the causal factor, often have a spectacular evolution, with rapid improvement in neurological status. This is due to the elasticity of the child skull and high neuroplasticity capacity at this age. Evolution of diffuse brain lesions is slower, often with an echo on the child's intellectual development

CONCLUSIONS

Children aged 0-3 years have a very different pathological evolution compare with adults and the therapeutic management is different. Children with head injuries must be transferred directly to a paediatric department of neurosurgery and paediatric intensive care unit. As in any other medical situation, the prophylaxis is the most vital factor in the improvement of the outcome.

Studies have shown that dysautonomia affects about one third of patients after moderate to serve TBI in the first few weeks after brain injuries.

It has been proven that the key to success is the accurate and rapid clinical and neuro-imagistic diagnosis. Nevertheless, long-time follow–up is also mandatory for a better recovery. The Paediatric Neurosurgical Department and Paediatric Intensive Care Unit represent a crucial necessity in any hospital.

CT–scan represents the crucial and most important diagnosis tool and investigation procedure that must be performed to all children with TBI, in the first three hours.

Considering the risk of radiation in infants, difficulty in taking the history and a much varied symptomatology than the adults, the clinician is always in a dilemma whether or not to perform a CT scan to all infants with head injury.

The EDH (extradural hematoma) and scalp hematoma are sufficient enough to produce anaemia and shock, because the total blood volume is less in infants than in adults. Early surgical intervention for EDH and blood volume replacement have been proven to be very important for young patients, due to the haemorrhagic shock that can rapidly occur in infants and young children.

Children with minor head traumas may develop rare complications of head injuries such as extradural hematomas.

Most of the extradural hematoma in infants are asymptomatic, even though they might be of extended volume, which can definately endanger the life of the patient. Hence, any head injury in infants needs special attention and a high level of suspicion, especially when the infant has a scalp hematoma or pallor. Early detection and proper management can reduce the mortality rate of extradural hematoma in infants with good results.

There are promising research on neuroinflammation in a cerebral traumatic process. However, time is required to be inserted into the treatment guidelines and clinical practice.

Grow skull fracture is more common in infants and young children due to the thickness of the skull and its low degree of mineralization. In this case, surgery is required to prevent any complications.

Tight dural closure is a high necessity in the proper and successful surgical management. Duroplasty can be done using a patch of pericranium / periosteum. Cranioplasty is not indicated in infants.

REFERENCES

1. Ciurea A. Tratat de Neurochirurgie. Bucuresti: Editura Medicala; 2011.
2. Ciurea A, Gorgan M. TBI in infants and toddlers, 0-3 years old. Journal Medical Life; 2011. p. 234-243.
3. Farrel C, et al. Management of the paediatric patient with acute head trauma. Pediatric Child Health; 2013.
4. Saceleanu V. Imaging aspects in intracerebral hemorrhage. Acta Medica Transilvanica. 2010;15(1):215-218.
5. Saceleanu V, Pereanu M. Neurochirurgie Clinică. Sibiu: Editura Univeristatii Lucian Blaga din Sibiu; 2014.
6. Gardner MAM. Pediatric TBI: an Update on Management. Current Pediatrics Reports; 2017. p. 213-219.
7. Saceleanu V, et al. Cerebral Vascular Malformation - an incidental discovery after a traumatic brain injury. Acta Medica Transilvanica. 2017;22(3):31-33.
8. Popermack ML, et al, NG. Moderate-to-Severe Traumatic Brain Injury in Children: Complications and Rehabilitation Strategies. J Pediatric Healthcare. 2015 May;e1-e7.
9. Rasmussen L, et al. Neurocritical care for Severe Pediatric Traumatic Brain Injury. Mescape; 2018.
10. TM O. Standardizing ICU management of pediatric traumatic brain injury is associated with improved outcomes at discharge. Journal of Neurosurgery Pediatrics; 2016.
11. Krenzlin H, Lorez V, Danckwardt S, Kempski O, Alessandri B. The importance of thrombin in cerebral injury and disease. International Journal of Molecular Sciences; 2016.
12. Hua Y, Keep RF, Gu Y, Xi G. Thrombin and brain recovery after intracerebral hemorrhage. Stroke; 2009.