0021-7557/10/86-01/73
Jornal de Pediatria

Original Article

Factors associated with intracranial hypertension in children and teenagers who suffered severe head injuries

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Abstract

Objective: To analyze factors associated with intracranial hypertension in pediatric patients who suffered severe head injuries.

Methods: Retrospective cohort study, with data collected from September 1998 through August 2003, including patients aged 0 to 16 who suffered severe head injuries, Glasgow score < 9, and submitted to intracranial pressure (ICP) monitoring (n = 132). Intracranial hypertension (IH) was defined as an episode of ICP > 20 mmHg requiring treatment, while refractory IH was ICP over 25 mmHg requiring barbiturates or decompressive craniectomy. Univariate analysis was followed by multivariate analysis; variables were considered significant if p < 0.05.

Results: Ages ranged from 2 months to 16 years, median age 9.7 (6.0-2.3) years. Glasgow scores ranged from 3 to 8, median 6 (4-7). Traffic accidents were responsible for 79.5% of events. Monitoring devices were installed, on average, 14 hours after trauma, median time 24 hours. One hundred and three patients (78%) had IH, while 57 (43.2%) had refractory IH. In multivariate analysis, younger age ranges were associated with IH [relative risk = 1.67 (1.03-2.72); p = 0.037], and abnormal postures were associated with refractory IH [relative risk = 2.25 (1.06-4.78)]. The group mortality rate was 51.5%; it was correlated with use of barbiturates in refractory IH and low cerebral perfusion pressure at the intensive care unit.

Conclusions: IH and refractory IH were frequent events in pediatric patients who suffered severe head injuries. The younger the patient, the greater the chance of developing IH. The presence of abnormal postures was found to be a risk factor for refractory IH.

J Pediatr (Rio J). 2010;86(1):73-79: Pediatric head injuries, pediatric traumatic brain injury, pediatric brain injury, intracranial hypertension.

Introduction

Every year, trauma kills 22 thousand Brazilian children and teenagers.[1] Eighty five percent of victims of severe trauma suffer associated head injury (HI), the injury responsible for most deaths.[2,3] The number of survivors with impairments ranges from 3 to 31 for each death.[3]

After a HI, events such as hypoxemia, intracranial hypotension and hypertension (IH) cause secondary damage to the encephalon and are strongly associated with poor results.[4] Patient interventions focus on preventing and correcting such factors, with the goal of ensuring the

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This study was performed in association with the Graduate Program in Health Sciences, Pediatric and Adolescent Health, of Universidade Federal de Minas Gerais (UFMG) School of Medicine, Belo Horizonte, MG, Brazil, and with Hospital João XXIII, Fundação Hospitalar do Estado de Minas Gerais (FHEMIG), Belo Horizonte, MG, Brazil.

No conflicts of interest declared concerning the publication of this article.

Suggested citation: Guerra SD, Carvalho LF, Affonseca CA, Ferreira AR, Freire HB. Factors associated with intracranial hypertension in children and teenagers who suffered severe head injuries. J Pediatr (Rio J). 2010;86(1):73-79.

Manuscript submitted Jun 10 2009, accepted for publication Oct 28 2009.

doi:10.2223/JPED.1960

supply of oxygen to the encephalon. Measures taken to that end include controlling intracranial pressure (ICP) and maintaining cerebral pressure perfusion (CPP).

Intracranial hypertension can lead to decreased pressure perfusion, limiting or stopping blood flow and causing neuronal death.[5] It can also kill through herniation of intracranial structures. Clinical evaluations have been known to be an unreliable parameter for IH detection for over 80 years.[6] Using only this criterion, IH is only detected in advanced stages, when there is already intense suffering or when herniation and death are inevitable.

Several authors associate IH with poor neurological outcomes or higher mortality rates.[7,8] Monitoring ICP and aggressive treatment for IH have also been associated with better outcomes in the literature about pediatric patients with severe head injuries.[9,10]

However, pediatric guidelines recommend monitoring ICP in children and teenagers with severe head injuries in cases of class III evidence, uncertain level of clinical safety or therapeutic option.[11]

Studies have sought factors related to intracranial hypertension, but mostly among adults or in samples with few children.[4,12] We need more pediatric studies to determine factors for identifying which patients might benefit from ICP monitoring and which could be spared of its risks, complications, and expenses.

The objective of this study was to analyze factors associated with IH in children and teenagers who suffered severe head injuries, hospitalized at intensive care units.

Methods

This was a retrospective cohort study, with data collected from September 1998 through August 2003. It included patients aged 0 to 16 admitted to the pediatric intensive care unit (ICU) of Hospital João XXIII (HJXXIII) because of severe blunt trauma to the head (score below 9 in the Glasgow coma scale) and submitted to ICP monitoring. Exclusion criteria included penetrating injuries caused by firearms, blades or other objects, as well as teenagers with severe HI admitted to the adult ICU, due to differences in behavior and protocols.

The following devices were used, depending on hospital availability and the preferences of the neurosurgeon: Richmond subarachnoid screw, Codman intraparenchymal catheter tip sensor,® Camino fiber optics,® and intraventricular external shunt catheter.

HJXXIII is a public school hospital located in Belo Horizonte, capital city of Minas Gerais, a state in Southeastern Brazil. The metropolitan region is home to approximately 5 million inhabitants. The hospital belongs to Fundação Hospitalar do Estado (State Hospital Foundation); it features 400 beds, and is considered a reference for urgent and emergency cases. On average, it treats 13 thousand patients per month, 4 thousand of which are children and teenagers (33%). Of those, 310 are admitted to the hospital every month, and 250 are admitted to the pediatric ICU every year.

The treatment of pediatric ICU patients uniformly followed a protocol based on the 1995 guidelines for treatment of severe head injuries.[13] The protocol was published in a review article[14] and reviewed when the guidelines were published in 2000.[15]

The study was approved by the Research Ethics Committee of Universidade Federal de Minas Gerais (UFMG), Belo Horizonte (Minas Gerais, Brazil), under protocol ETIC 420/04.

The data were collected from patients' records at the hospital's Medical Archive and Statistics Service for the period between September 1998 and February 2002. Since then, the data were collected at the bedside, after obtaining free and informed consent.

Intracranial hypertension was defined as an episode of ICP above 20 mmHg and requiring treatment; patients were treated if ICP remained at least 5 minutes above said value.[16]

Refractory intracranial hypertension was defined as an episode of ICP sustainably above 25 mmHg and requiring barbituric coma or craniectomy after ruling out mass lesions with surgical indication.[11]

Head injury severity was measured according to the Glasgow Coma Scale (GCS).[17] Children under the age of 4 were assessed by verbal and motor response adapted to their age.[3,18] Patients were considered victims of severe HI if they still scored 8 or less in the GCS 6 hours after the trauma.

Patients with abnormal flexion or extension postures were grouped for an analysis of correlation with occurrence intracranial hypertension.[12]

Tomographic findings were collected in a single group of traumatic brain injuries with greater probability of developing IH (hemorrhages, hematomas, injuries, edema, edema and swelling, cistern compression or obliteration) and another with lower probability [normal tomography or tomography showing diffuse axonal injury (DAI)].[12]

Circulatory shock upon first admission was defined as presence of systolic blood pressure below the fifth percentile for the relevant age, or a weak pulse and capillary ballooning above 2 seconds, as long as there are corrective measures, such as volume replacement or use of vasoactive medication.

Trauma severity was assessed by the Pediatric trauma scale (PTS).[19] The following associated injuries were considered: injuries to the spinal column and medulla, thorax, abdomen and musculoskeletal system.

Infections to the central nervous system were considered if diagnosed and treated by attending physicians based on clinical suspicion and whatever complementary test they requested.

The data was analyzed using the computer application Epi-Info. Comparisons between proportions used the chi-square test, and Fisher's exact test when one or more quads had an expected value below 5. The linear trend chi-square test was used in situations featuring progression. Relative risk (RR) and its 95% confidence interval were also calculated. RR is noted as a value followed by confidence interval in parentheses.

Variance analysis was used to compare means for data with normal distribution, but the Kruskal-Wallis nonparametric test was used in cases in which variances were not homogeneous and were expressed in terms of medians and 25-75% inter quartile range (25-75 IQ). Variables with $p < 0.25$ in univariate analysis were analyzed simultaneously by logistic regression used for multivariate analysis, using computer application MULTLR. The final model considered as significant all remaining variables with value $p < 0.05$.

Results

Between 1998 and 2003, the pediatric ICU had eight beds and received circa 1,000 patients; 315 had blunt head injuries and 191 had GCS scores below 9. Of those, 132 received ICP monitoring and were included in this study.

Patient age ranged from 2 months to 16 years, with median age 9.7 (6.0-12.3) years. The most common trauma mechanisms were being run over by a car (51.5%), injuries in automobile passengers (18.2%), to bicycle riders (9.8%), falls (13.6%), assault (1.5%), and others or not informed (3%).

Most patients had multiple intracranial lesions (330 lesions in 132 patients). The most common were swelling, found in 74 patients (56.1%); DAI, found in 56 (42.4%); contusion or intraparenchymal hemorrhages, found in 46 (34.8%); and subarachnoid hemorrhages, found in 41 (31.1%).

Subdural hematomas (20 patients, 15.2%), intraventricular hemorrhages (15 patients, 11.4%), and extradural hematomas (14 patients, 10.6%) were less frequent.

GCS scores ranged from 3 to 8, with a median of 6 (4-7). PTS scores ranged from -3 to 11, with a median of 4. One hundred and twelve major associated injuries were diagnosed: 48 thoracic, 37 musculoskeletal, 21 abdominal, and 6 of the spinal column and medulla.

IH and associated factors

One hundred and three patients monitored (78%) had at least one IH episode requiring treatment, and 57 (43.2%) had refractory IH episodes.

In univariate analysis, younger patients suffered IH more frequently than older ones (Table 1).

Table 2 shows the univariate analysis of refractory IH frequency. In this analysis, the factors sex, age and presence of abnormal postures were associated with the occurrence of events, but they were then submitted to multivariate analysis to verify the association.

The correlation between age and occurrence of IH and refractory IH was analyzed using the linear trend chi-square test. Table 3 shows that, compared to teenagers, the younger the patient, the greater the chance of IH. The same analysis was performed for refractory IH without finding significant statistical differences (Table 4).

No kind of associated injury showed greater risk of developing IH.

Multivariate analysis

Multivariate analysis demonstrated that younger age was related to higher probability of IH [RR = 1.67 (1.03-2.72); $p = 0.037$]. All children younger than 1 year old suffered IH. The presence of tomographic changes was not significantly associated with IH [$p = 0.08$; RR = 2.78 (0.88-8.76)].

In multivariate analysis, the presence of abnormal postures was related to higher probability of refractory IH [$p = 0.03$; RR = 2.25 (1.06-4.78)]. Being younger was not statistically significant [RR = 1.36 (0.95-1.96); $p = 0.092$].

Characteristics and complications of monitoring

On average, ICP monitoring devices were installed 14 hours after trauma, but 50 percent of patients were monitored after more than 24 hours, while 5% were monitored after more than 48 hours.

One hundred and five (79.5%) patients were monitored using a subdural or subarachnoid Richmond screw, 19 (14.4%) with intraparenchymal fiber optics, 6 (4.6%) with intraventricular catheters, and 2 (1.5%) with subdural catheters.

After finding normal ICP, the subdural screw malfunctioned in three patients. Based on clinical and tomographic assessments, monitoring was withdrawn.

Eleven of 132 patients (8.3%) under monitoring suffered central nervous system (CNS) infections, versus five among 58 unmonitored patients (8.5%). The difference was not statistically significant [$p = 0.8$; odds ratio = 0.98 (0.36-2.70)].

We were unable to compare the various monitoring devices in terms of event frequency due to the small number of patients who did suffer CNS infections.

Table 1 - Factors associated with the occurrence of intracranial hypertension in patients with severe head injuries submitted to intracranial pressure monitoring at the Hospital João XXIII pediatric intensive care unit between September 1998 and August 2003 (n = 132)

	IH			
Factor	**Yes, n (%)**	**No, n (%)**	**RR (95%CI)**	**p**
Sex			0.90 (0.76-1.06)	0.36
Male	69 (77.5)	20 (22.5)		
Female	37 (86.0)	6 (14.0)		
Age*	8.7±4.2	10.5±3.6		0.04
GCS†	6 (4-7)	6 (4-7)		0.86
PTS†	4 (2-5)	4 (2-5)		0.84
Abnormal postures			1.02 (0.85-1.22)	0.99
Yes	35 (81.4)	8 (18.6)		
No	71 (79.8)	18 (20.2)		
TBI			1.32 (0.90-1.95)	0.09
Yes	96 (82.8)	20 (17.2)		
No	10 (62.5)	6 (37.5)		
Circulatory shock			1.07 (0.91-1.27)	0.59
Yes	37(84.1)	7 (15.9)		
No	69 (78.4)	19 (21.6)		
Total	106 (80.3)	26 (19.7)		

25-75 IQ = 25-75% inter quartile range; 95%CI = 95% confidence interval; GCS = Glasgow coma scale; IH = intracranial hypertension; PTS = pediatric trauma scale; RR = relative risk; TBI = traumatic brain injury.
* Mean ± standard deviation.
† Median (25-75 IQ).

Table 2 - Factors associated with the occurrence of refractory intracranial hypertension in patients with severe head injuries submitted to intracranial pressure monitoring at the Hospital João XXIII pediatric intensive care unit between September 1998 and August 2003 (n = 132)

	Refractory IH			
Factor	**Yes, n (%)**	**No, n (%)**	**RR (95%CI)**	**p**
Sex			0.71 (0.49-1.05)	0.01
Male	34 (38.2)	55 (61.8)		
Female	23 (53.5)	20 (46.5)		
Age*	8.3±4.6	9.6±3.7		0.08
GCS†	6 (4-7)	6 (4-7)		0.82
PTS†	4 (2-5)	4 (2-5)		0.82
Abnormal postures			1.51 (1.03-2.20)	0.06
Yes	24 (55.8)	19 (44.2)		
No	33 (37.1)	56 (62.9)		
TBI			1.17 (0.60-2.28)	0.83
Yes	51 (44.0)	65 (56.0)		
No	6 (37.5)	10 (62.5)		
Circulatory shock			1.00 (0.66-1.51)	0.85
Yes	19 (43.2)	25 (56.8)		
No	38 (43.2)	50 (56.8)		
Total	57 (43.2)	75 (56.8)		

25-75 IQ = 25-75% inter quartile range; 95%CI = 95% confidence interval; GCS = Glasgow coma scale; IH = intracranial hypertension; PTS = pediatric trauma scale; RR = relative risk; TBI = traumatic brain injury.
* Mean ± standard deviation.
† Median (25-75 IQ).

Table 3 - Correlation between age range and occurrence of intracranial hypertension in patients with severe head injuries admitted to the Hospital João XXIII pediatric intensive care unit between September 1998 and August 2003 (n = 132)

	IH		
Age (years)	**Yes, n (%)**	**No, n (%)**	**RR (95%CI)**
15 to 16	10 (66.7)	5 (33.3)	1.00
10 to 14	35 (76.1)	11 (23.9)	1.14 (0.77-1.69)
5 to 9	38 (82.6)	8 (17.4)	1.24 (0.85-1.81)
1 to 4	18 (90.0)	2 (10.0)	1.35 (0.92-1.99)
0 to < 1	5 (100.0)	0 (0)	1.50 (1.05-2.15)
Total	106 (80.3)	26 (19.7)	

95%CI = 95% confidence interval; IH = intracranial hypertension; RR = relative risk.
Linear trend chi-square test = 4.769.
p = 0.03.

Table 4 - Correlation between age range and occurrence of refractory intracranial hypertension in patients with severe head injuries admitted to the Hospital João XXIII pediatric intensive care unit between September 1998 and August 2003 (n = 132)

	Refractory IH		
Age (years)	**Yes, n (%)**	**No, n (%)**	**RR (95%CI)**
15 to 16	6 (40.0)	9 (60.0)	1.00
10 to 14	19 (41.3)	27 (58.7)	1.03 (0.51-2.10)
5 to 9	16 (34.8)	30 (65.2)	0.87 (0.42-1.81)
1 to 4	12 (60.0)	8 (40.0)	1.50 (0.73-3.07)
0 to < 1	4 (80.0)	1 (20.0)	2.00 (0.94-4.27)
Total	57 (43.2)	75 (56.8)	

95%CI = 95% confidence interval; IH = intracranial hypertension; RR = relative risk.
Linear trend chi-square test = 2.445.
p = 0.12.

No patient required surgery because of hemorrhagic complications secondary to installing the ICP monitoring device.

The group's mortality rate was 51.5 percent. It was associated with use of barbiturates, low cerebral perfusion pressure, sodium disturbances, swelling, and acute respiratory distress syndrome.

Discussion

Patients were predominantly male, aged 7 to 9, victims of traffic accidents, and suffering multiple traumatic brain injuries. Swelling and hematomas occurred well above expected rates.[7,9] The profile might explain the high percentage of patients with IH, and especially refractory IH.

Almost 80 percent of patients had at least one IH episode. This piece of data is of questionable relevance, since there is no evidence that ICP slightly above 20 mmHg is more harmful than some of the measures used to control it.[20]

However, after observing that over 40 percent of patients had refractory IH, excluding those with surgical indication for mass effect lesions, the number is worrisome. The literature recognizes that morbidity and mortality are very high for refractory IH patients,[11,21] but also that the benefits of the measures used to control it are even more questionable.[11,22,23]

Thus, in light of current knowledge, having children and teenagers with severe head injuries exposed to risk of more than 40 percent of developing undocumented refractory IH is unacceptable, but so is instituting aggressive treatments without their need being certain.[11]

In this study, half of all patients were monitored more than 24 hours after the injury. This may have contributed to the high rates of IH, since we know that sustained IH leads to a vicious circle of ischemia, edema and worsened IH.[24]

The study showed higher rates of IH in younger children. Despite the small number of children younger than 1, they had the highest rates of IH. The data reinforce the importance of monitoring in this age range and counter the argument that such patients are under lower risk of developing IH because of their open fontanelles and unconsolidated sutures.[11]

The importance of monitoring small children increases with reports that children younger than 4 who suffer head injuries have worse prognoses than older ones.[7] This study is one of the few to document the occurrence of refractory IH in infants and small children.[10]

Patients who suffered severe head injuries and have tomographic changes also had high rates of IH, as expected.[2] However, unlike what has been described for adults, the group with isolated DAI also had high rates of IH. The results were similar to Esparza et al.'s pediatric study.[25,26]

The difference in event rates in children may be explained by the higher ratio of cranial content and higher frequency of swelling compared to adults.[9]

The study's methodological limitations may have influenced the results. Diagnoses of tomographic lesions made by the neurosurgeons at the emergency service may be inaccurate. This is even more relevant when considering tomography in children. Retrospective data collection was another study limitation.

Circulatory shock did not influence the occurrence of IH or refractory IH, unlike the findings from Narayan et al., in which the association of hypotension with being 40 years old or more or to abnormal postures was associated with higher rates of IH among adults who suffered head injuries.[12] The result may be attributed to the criteria used in this study for including patients with signs of shock, but not hypotension; this would lead to the cerebral blood flow being less compromised and, consequently, to less ischemia and swelling.

The presence of abnormal postures was correlated to the occurrence of refractory IH, even after multivariate analysis. The result is incredibly important, since it means ICP monitoring is recommended for pediatric patients with abnormal postures even with normal tomography and hemodynamic stability, contrary to what is recommended for adult patients.[12] Bruce et al. are among the few authors to correlate IH in children with the presence of abnormal postures.[27]

Less than 8 percent of monitored patients had CNS infections; all of them had good outcomes, as the literature would lead us to expect.[12] No prophylactic antibiotics were used while the devices were installed, and there was no difference in rates of infection for patients with and without monitoring devices.[11]

Three quarters of patients were monitored using a Richmond screw. Subarachnoid monitoring, either by screw or by catheter, seems to be a cheap option, with easy installation and accessible in locations with fewer resources,[28] especially considering how hard it is to install ventricular catheters in trauma patients and how expensive fiber optics and catheter tip sensors can be.

In three cases in this study, the screw initially showed normal ICP and then stopped working. Physicians chose to keep those patients unmonitored from then on based on clinical and tomographic assessments. In this analysis, those three patients are listed as having no ICP increases. However, this study limitation does not bias it towards higher rates of IH.

The absence of hemorrhagic complications secondary to ICP monitoring requiring surgical interventions matches the literature. Significant hematomas requiring surgical removal were found in only 0.5 percent of patients. Clearly, there is a greater risk of coagulation conditions.[29]

In 2005, Carvalho studied the group's elevated mortality rate,[30] which may have been secondary to the high frequency of refractory IH and the use of barbiturates at the time.

Conclusions

IH and refractory IH were frequent events in pediatric patients who suffered severe head injuries, independently of the presence of risk factors accepted for adult patients.

The younger the patient, the greater the chance of developing IH. The presence of abnormal postures in pediatric patients who suffered severe head injuries was found to be a risk factor for refractory IH.

Criteria for ICP monitoring in Pediatrics should be different than those used for adults. The infectious and hemorrhagic complications of monitoring have little clinical significance, and they are at least five times less risky than refractory IH.

Acknowledgements

We would like to thank the professionals at Hospital João XXIII and at the UFMG School of Medicine Graduate Program; and professors Maria do Carmo Barros de Melo (UFMG) and Eduardo Juan Troster (USP) for their valuable suggestions and corrections.

References

1. DataSUS. [website] Brasil, Ministério da Saúde. http://w3.datasus.gov.br/datasus/index.php. Access: 10/02/2009.

2. Orliaguet GA, Meyer PG, Blanot S, Jarreau MM, Charron B, Buisson C, et al. Predictive factors of outcome in severely traumatized children. Anesth Analg. 1998;87:537-42.

3. American College of Surgeons – Committee on Trauma. Advanced trauma life support program for doctors. 7th ed. Chicago: ACS; 2004.

4. Chesnut RM, Marshall LF, Klauber MR, Blunt BA, Baldwin N, Eisenberg HM, et al. The role of secondary brain injury in determining outcome from severe head injury. J Trauma. 1993;34:216-22.

5. Chesnut RM. Secondary brain insults after head injury: clinical perspectives. New Horiz. 1995;3:366-75.

6. Browder J, Meyers R. Observations of the behavior of the systemic blood pressure pulse, and spinal fluid pressure following craniocerebral injury. Am J Surg. 1936;31:403-26.

7. Alberico AM, Ward JD, Choi SC, Marmarou A, Young HF. Outcome after severe head injury. Relationship to mass lesions, diffuse injury, and ICP course in pediatric and adult patients. J Neurosurg. 1997;67:648-56.

8. Downard C, Hulka F, Mullins RJ, Piatt J, Chesnut R, Quint P, et al. Relationship of cerebral perfusion pressure and survival in pediatric brain injured patients. J Trauma. 2000;49:654-8.

9. Bruce DA, Raphaely RC, Goldberg AI, Zimmerman RA, Bilaniuk LT, Schut L, et al. Pathophysiology, treatment and outcome following severe head injuries in children. Childs Brain. 1979;5:174-91.

10. Cho DY, Wang YC, Chi CS. Decompressive craniotomy for acute shaken/impact baby syndrome. Pediatr Neurosurg. 1995;23:192-8.

11. Adelson PD, Bratton SL, Carney NA, Chesnut RM, du Coudray HE, Goldstein B, et al. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. Chapter 19. The role of anti-seizure prophylaxis following severe pediatric traumatic brain injury. Pediatr Crit Care Med. 2003;4: S72-5.

12. Narayan RK, Kishore PR, Becker DP, Ward JD, Enas GG, Greenberg RP, et al. Intracranial pressure: to monitor or not to monitor? A review of our experience with severe head injury. J Neurosurg. 1982;56:650-9.

13. Guidelines for the management of severe head injury. Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care. J Neurotrauma. 1996:13:641-734.

14. Guerra SD, Jannuzzi MA, Moura AD. Pediatric head injury. J Pediatr (Rio J). 1999;75:S279-93.

15. Bullock MR, Chesnut RM, Clifton GL, The Brain Trauma Foundation, The American Association of Neurological Surgeons, The Joint Section on Neurotrauma and Critical Care. Guidelines for the management of severe traumatic brain injury. Brain Trauma Foundation. J Neurotrauma. 2000:17:449-554.

16. The Brain Trauma Foundation. The American Association of Neurological Surgeons. The Joint Section on Neurotrauma and Critical Care. Intracranial pressure treatment threshold. J Neurotrauma. 2000;17:493-5.

17. Teasdale G, Jennett B. Assessment of coma and impaired consciousness – A practical scale. Lancet. 1974;2:81-4.

18. James HE. Neurologic evaluation and support in the child with an acute brain insult. Pediatr Ann. 1986:15:16-22.

19. Tepas JJ 3rd, Ramenofsky ML, Mollitt DL, Gans BM, DiScala C. The Pediatric Trauma Score as a predictor of injury severity: an objective assessment. J Trauma. 1988:28:425-9.

20. Kaufmann AM, Cardoso, ER. Aggravation of vasogenic cerebral edema by multiple-dose mannitol. J Neurosurg. 1992;77:584-9.

21. Pfenninger J, Kaiser G, Lütschg J, Sutter M. Treatment and outcome of the severely head injured child. Intensive Care Med. 1983;9:13-6.

22. Bárcena JP, Centelles JM, Milla PM, Juvé JI. Indicación del coma barbitúrico em el traumatismo craneoencefálico grave. Med Intensiva. 2002;26:407-412.

23. Muizelaar JP, Marmarou A, Ward JD, Kontos HA, Choi SC, Becker DP, et al. Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. J Neurosurg. 1991;75:731-9.

24. Sharples P, Stuart AG, Matthews DS, Aynsley-Green A, Eyre JA. Cerebral blood flow and metabolism in children with severe head injury. Part 1: Relation to age, Glasgow coma score, outcome, intracranial pressure, and time after injury. J Neurol Neurosurg Psychiatry. 1995;58:145-52.

25. Lee TT, Galarza M, Villanueva PA. Diffuse axonal injury (DAI) is not associated with elevated intracranial pressure (ICP). Acta Neurochir (Wien). 1998;140:41-6.

26. Esparza J, M-Portillo J, Sarabia M, Yuste JA, Roger R, Lamas E. Outcome in children with severe head injuries. Childs Nerv Syst. 1985;1:109-14.

27. Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzzell B. Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema". J Neurosurg. 1981;54:170-8.

28. Dantas Filho VP, Falcão AL, Sardinha LA, Facure JJ, Araújo S, Terzi RG. Aspectos técnicos da monitorização da pressão intracraniana pelo método subaracnóideo no traumatismo crânio-encefálico grave. Arq Neuropsiquiatr. 2001;59:895-900.

29. Affonseca CA, Carvalho LF, Guerra SD, Ferreira AR, Goulart EM. Coagulation disorder in children and adolescents with moderate to severe traumatic brain injury. J. Pediatr (Rio J). 2007;83:274-82.

30. Carvalho LF. Estudo dos fatores relacionados com a mortalidade de crianças e adolescentes com traumatismo crânio-encefálico internados em unidade de terapia intensiva terciária [dissertação]. Belo Horizonte: Universidade Federal de Minas Gerais; 2005.

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