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SEVERE BRAIN INJURIES: CORRELATION BETWEEN SURVIVAL AND INTRACRANIAL HYPERTENSION

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There are several reasons of intracranial pressure (ICP) increase in the brain trauma. Brain edema, due to the brain-blood bariere injury, contusion of brain tissue and intracranial hematomas that represent mass lesion, cerebrovascular autoregulation failure which leads to hemodinamic disorder, and traumatic subarchnoid haemorrhagae that is commonly associated with CSF flow disturbances are the main causes. The aim of our study was to examine the survival of patients with severe brain trauma in the presence of different values of ICP. This prospective study included 32 patients with intracranial pressure monitored, and appropriate treatment undertaken. Twenty-two patients (68.75%) had elevated ICP, and in 10 patients (31,25%) there were no criteria of intracranial hypertnesion (ICHTN). The results of our study showed that absolute lethal value of ICHTN is 50mmHg and over – none of the injured survived such ICP if lasted more than two hours, because of inevitable brain and brainstem ischemia and failure of the vital functions. The relatively lethal values of ICP ranged from 40 to 50mmHg, in the case of which we menaged to prevent a fatal outcome in one out of five cases. *Acta Medica Medianae 2011;50(3):10-15.*

Key words: *traumatic brain injury, intracranial monitoring, intracranial hypertension*

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Introduction

Intracranial pressure (ICP) is the pressure inside the skull, which is defined as inflexible and completely closed system, that is equally distributed within its entire volume. This applies to intact brain, while in the brain trauma or lesion of other origin, in most of the cases, it is possible to record the presence of the supratentorial pressure gradient (1).

The sum of volumes of blood, brain and cerebrospinal fluid is constant and that is the very essence of the Munro-Kelly`s doctrine. This doctrine directly applies to ICP because the existence of dynamic equilibrium of these three elements ensures the ICP stability.

Brain edema (due to brain-blood bariere injury), contusion of brain tissue and intracranial hematomas (that represent mass lesion), cerebrovascular autoregulation failure (which leads to hemodinamic disorder) and traumatic subarchnoid haemorrhage (that is commonly associated with CSF flow disturbances) are the main reasons for the occurrence of ICHTN in brain trauma patients.

Normal ICP values depend on age, and for infants it is 1.5 to 6 mmHg, for younger children 3-7 mmHg, and for older children and adults it is <10-15 mmHg (2).

Physiological increases of the ICP can occur in healthy people during everyday activities during coughing, sneezing, defecation, etc. Although values of ICP in such cases are measured to 50 mmHg, and more, they are clinically insignificant because they last only for a few seconds. Most clinicians define ICHTN as values of ICP exceeding 20 mmHg, in a time period longer than two hours.

Indications for the monitoring of ICP in brain trauma cases are:

- Patients with a Glasgow Coma Score (GCS) ≤8 and with abnormal CT findings.
- Patients with GCS≤8 with normal CT findings, which have two of the following three: systolic blood pressure <90 mmHg, age over 40, present decerebrations or decortications.
- Patients with systemic injuries and alterations of consciousness, where therapy is linked with excessive intravenous fluid intake.
- After the surgical removal of an intracranial mass.

It is widely believed that ICP will always be lowered after the evacuation of large intracranial hematoma. On the contrary, intracranial hypertension was diagnosed in more than 50% of patients with severe brain injuries in which the intracranial mass was evacuated (3).

This may be caused by diffuse cerebral edema, blending of separate focal areas of brain

contusion, postoperative hematoma at the site of operations or in other brain areas, and a variety of systemic complications (4).

There are two contraindications for invasive ICP monitoring: conscious patients and coagulopathy.

Patients and methods

The study included 32 patients, 29 male and 3 female, who were monitored and treated during the period February 16, 2008 to February 15, 2010. The study was prospective. Seventeen patients had ICP monitor implanted subdurally, 11 intraparenhimally and 4 intraventricularly. During ICP monitoring, (control) native brain CT scan was performed in cases when in the course of two hours the values of ICP continuously remained higher than 20 mmHg. Mass effects shown on CT associated with shifting the midline structures for more than 5mm were surgically treated (outer or inner decompression). Patients with CT findings without mass effect of existing brain lesions and with ICHTA were treated conservatively.

During the ICP monitoring (Codman, ICP Express) of patients each day, every hour, the ICP values were recorded and if three consecutive values were above 20 mmHg, the ICHTN was defined in specific patient. Patients who suffered ICHTN were divided in separate groups depending on the maximal values of ICP during a two-hour measurement. Neurological assessments were quantified in Glasgow Coma Score (GCS) in time interval of four hours, or 30 min in case of ICHTN increase. ICP monitoring is performed in patients with ICHTN until the values of ICP become normal and after that for further 48 to 72 hours, and in cases resistant to therapy usually until death occurs. In patients with no ICHTN for the first two days after the injury, ICP monitoring was performed in the next 48 hours or longer.

ICP values in patients in whom monitoring was done were described as normal or hypertensive, and later were classified successively growing for further processing. All patients were initially treated with antiedematous therapy, which in patients with ICHTN was intensified, prolonged, and sustained by mechanical ventilation support, sedation, analgesia and relaxation. Such therapy was applied in the control group according to the findings on CT of the brain, additional radiological and laboratory tests, and consciousness of the patient.

Quantitative statistical analysis was conducted using the computer. For entering, ranking, clustering, tabular and graphical representations of data we used the Excel program from Microsoft Office 2003 software package. Calculations were carried out using the SPSS software, version 10.0. In all analyses as the limit of statistical significance the error estimate of 0.05 or 5% was taken. Comparison of representation of certain modalities of attribute features of the two groups of patients was conducted by Fisher's exact probability test (Fisher exact test) in cases where some of the expected frequencies was less than 5.

Results

In the study group, 22 patients (68.75%) had elevated ICP values, and in 10 patients (31.25%) there were no criteria of intracranial hypertension (Figures 1 and 2).

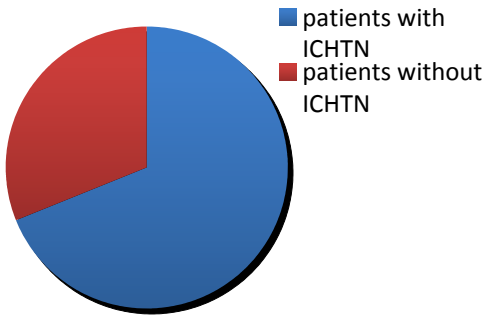

Figure 1. Correlation between number of patients with and without ICHTN

Table 1. Presentation of patients who died and survived in the study group by the presence of ICHTN

Monitoring of ICP	N	Deceased (%)	Survived (%)
With ICHTN	22	13 (59,10%)	9 (40,90%)
Without ICHTN	10	2 (20,00%)	8 (80,00%)
Σ	32	15 (46,875%)	17 (53,125%)

In the group with ICHTN, the survival was lower than in the group without ICHTN, with survival rate of 41% and 80%, respectively (Table 1).

Fisher's test showed that the percentage of survival was significantly lower in the group of patients with the presence of ICHTN ($p=0.04$, $p<0.05$).

In the subgroup with ICHNT (18 patients) who had the ICP values above 30 mmHg in continuity longer than two hours, the death rate was 66.66% (Figure 3). In 10 patients with ICHNT who had the ICP values above 40 mmHg continuously for longer than two hours the mortality was 90.00% (Figure 4). One patient that had the ICP values up to 45 mmHg during three successive measurement survived after internal decompression of brain contusion focus, with reclining ICP below 30 mmHg after the intervention. In five patients with ICP values over 50 mmHg, continuously over 2 hours, there was a fatal outcome without exception. In two patients, ICP values were recorded over 80 mmHg in continuity (Figure 2). Based on these data it is possible to demonstrate the relationship between the values of ICP and mortality of patients with severe brain trauma (Figure 5).

In two patients with ICHTN in whom mean values of ICP did not exceed 20-30 mmHg (Figure 2), successfully controlled ICHTN led to favorable treatment outcomes.

Patient	Died	ICHTN recorded	30 do 40	40 do 50	50 do 60	60 do 70	70 do 80	80 do 90	>90
1	**Yes**	yes							
2	**Yes**	yes							
3	No	no							
4	**Yes**	yes							
5	**Yes**	yes							
6	No	yes							
7	No	yes							
8	No	no							
9	No	yes							
10	No	no							
11	No	no							
12	**Yes**	yes							
13	No	yes							
14	**Yes**	yes							
15	**Yes**	yes							
16	No	yes							
17	**Yes**	yes							
18	No	no							
19	**Yes**	yes							
20	No	yes							
21	No	no							
22	**Yes**	yes							
23	**Yes**	yes							
24	No	yes							
25	No	no							
26	No	yes							
27	**Yes**	no							
28	**Yes**	yes							
29	No	yes							
30	No	no							
31	**Yes**	no							
32	**Yes**	yes							

Figure 2. Presentation of survival of patients with and without ICHTN

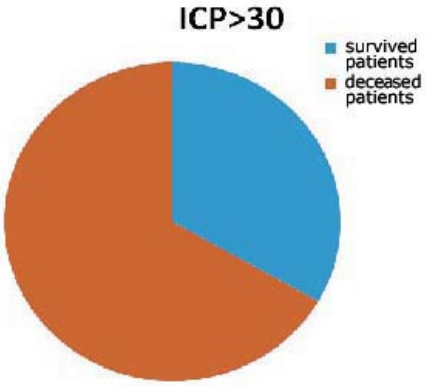

Figure 3. In the subgroup of patients with ICHNT who had ICP values above 30 mmHg in continuity longer than two hours the mortality rate reached 66.66%

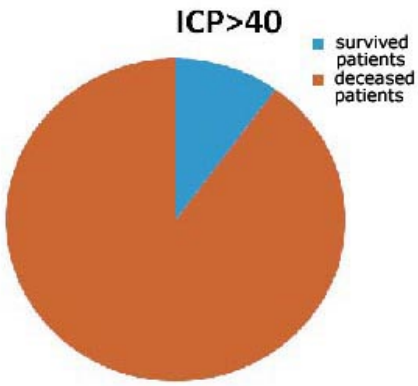

Figure 4. In the subgroup of patients with ICHNT who had ICP values above 40 mmHg in continuity longer than two hours the mortality was 90.00%

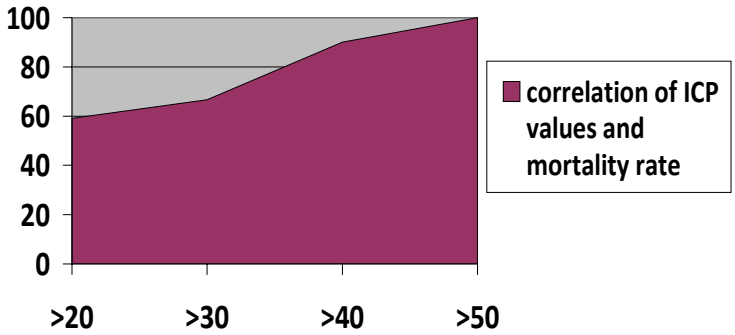

Figure 5. The correlation of ICP values in the continuity for two hours and the percentage of mortality of ICHTN patients

Discussion

Since 1996, there is an official recommendation of the Brain Trauma Foundation for routine ICP monitoring after severe craniocerebral injury. ICP monitoring can: help early detection of mass lesions, restrict the use of some interventions that may harm the patient (such as hyperventilation), reduce ICP by drainage of CSF and thereby increase the cerebral perfusion, assist in determining the prognosis and improving the outcome.

During brain injury, a number of secondary ischemic insults "enrich" the initial brain injury in the upcoming hours and days. A significant reduction in mortality and morbidity can be achieved in patients with traumatic brain injuries using management protocols. These protocols emphasize the early intubation, rapid tranfer to intensive care unit (ICU), prompt resuscitation, early CT brain imaging, urgent evacuation of the intracranial traumatic expansive lesion, followed by appropriate treatment in ICU.

Applicability of the mechanisms of tissue damage during stroke to tissue damage after severe brain injuries (SBI) is questionable. Pathogenesis of acute stroke is well described and it is known that vascular occlusion leads to ischemia due to interruption of tissue perfusion, which often results in topographically well-limited ischemic core and the surrounding penumbra. Pathophysiological mechanisms responsible for neuronal death in SBI are not completely understood. Earlier studies have emphasized the reduced perfusion to explain SBI after ischemia, and used the classical mechanisms of stroke for the explanation of irreversible tissue damage. However, cerebral metabolism is often reduced after SBI (5), because of the trauma itself (6, 7) but also because of the additional application of sedatives. In SBI, cerebral blood flow, that is necessary for the preservation of tissue, must be higher than in stroke, in order to overcome metabolic vulnerability of brain tissue. It is controversial but possible that excitotoxicity can lead to an increase of cerebral metabolism which is not supported by adequate levels of cerebral blood flow (CBF), which leads to earlier death of nerve cells. Furthermore, the effects of trauma on cerebral physiology show striking spatial (8) and temporal (9) heterogeneity, both among patients and in patients. It is not possible yet to determine a universal threshold of sensitivity of the tissue that could be applicable for all patients, or in different brain regions of a patient (10).

According to reports from the trauma data bank, which is based on 654 patients with SBI, in 72% of patients the value of ICP>20 mmHg was found (11). This is similar to the results of our study where ICHTN was found in 69% of patients.

The association between the intensity of ICHTN and poor outcome after severe head injury is well known. In one of the studies, 77% patients with ICP below 15 mmHg had a favorable outcome, which was reported in only 43% patients with ICP above 15 mmHg (12). In our study, the ratio of mortality in patients who had ICP continuously above 20mmHg with those who hadn`t was 59% and 20%, respectively. A significant difference in survival was found comparing the patients without ICHTN to patients with ICHTN ($p=0.04$, $p<0.05$). In the study of Miller et al. (13), the ratio was 92% to 18%, in favor of non-ICHTN patients. The high percentage of mortality of patients with ICHTN in this study could be explained in less efficient diagnostic and therapeutic options about 30 years ago. In the study by Saul (14), the ratio of mortality was 69% to 15% in favor of patients with ICP lower than 25 mmHg in continuity, compared to patients that had ICP higher than 25 mmHg.

The results of our study showed that the absolute letal values of ICHTN are 50 mmHg and more. There were no patients who have survived such ICP for a period longer than two hours. Such high ICHTN leads to diffuse ischemia of the brain and brain stem with the failure of vital functions. (15) Relatively lethal values were above 40 mmHg, in the case of which we managed to prevent a fatal outcome in one patient. In 5 cases ICP became uncontrolled and resistant to all treatment measures undertaken, with values that ranged over 50 mmHg. It is possible to find in the literature some examples of therapy-resistant ICP values, which some authors try to solve, at least temporarily, by administrating hyperosmolar NaCl solution (16), or introducing a patient into a barbiturat coma (17).

The importance of average ICP values on survival was found in the papers of Nga (18) and McGraw (19). McGraw et al. found that the primary factor in the survival is average ICP in a group of 293 patients with SBI while using the method of stepped linear regression analysis. Study conducted by Sahipaul et al. (20) clearly reveals the level of confidence of the clinician towards the ICP monitoring as a diagnostic and therapeutic procedure. That study included more than 100 neurosurgeons from many medical centers in Canada, and it showed that 23.3% of them have low, 56.3% medium and 20.4% a high level of confidence in this method. Therefore, it is obvious that the majority of neurosurgeons are aware that ICP monitoring is not a miraculous method which is capable of removing terrible consequences of SBI.

Monitoring and treatment of SBI is much more complex and depends on many factors and parameters so that any single method could be of exclusive significance for saving the severely injured. However, the insight into the values of ICP and therefore in to the value of cerebral perfusion pressure can significantly aid in the diagnosing, treatment and prognosis in patients with SBI.

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TEŠKE KRANIOCEREBRALNE POVREDE: PREŽIVLJAVANJE BOLESNIKA U ODNOSU NA PRISUSTVO I VREDNOSTI INTRAKRANIJALNE HIPERTENZIJE

Aleksandar Kostić, Ivan Stefanović, Vesna Novak, Aleksandar Igić, Boban Jelenković i Goran Ivanov

Razlozi porasta intrakranijalnog pritiska u traumatskoj moždanoj povredi su multipni. Važne pojave su: edem mozga, koji se razvija zbog oštećenja nervnih ćelija i krvno moždane barijere, kontuzije mozga i intrakranijalni hematomi, koji se ponašaju kao mas lezije, slom cerebrovaskularne regulacije (dovodi do porasta volumena krvi) i traumatska subarahnoidalna hemoragija (zbog poremećaja toka likvora). Cilj našeg istraživanja bio je da ispitamo preživljavanje bolesnika sa teškim moždanim traumama, u svetlu prisustva intrakranijalnog pritiska i određenih njegovih vrednosti. U ovu prospektivnu studiju uključeno je 32 bolesnika kojima je praćen intrakranijalni pritisak i preduzimana odgovarajuća terapija. Dvadeset dva bolesnika (68,75%) imala su povišene vrednosti ICP, a kod deset bolesnika (31,25%) nije bilo kriterijuma intrakranijalne hipertenzije. Rezultati naše studije pokazali su da su apsolutno smrtonosne vrednosti ICHTN 50mmHg. Nije bilo bolesnika koji su preživeli toliki ICP u trajanju dužem od 2 sata. Prilikom tolikog ICP dolazi neminovno do difuzne ishemije mozga i moždanog stabla sa zatajivanjem vitalnih funkcija. Relativno smrtonosne vrednosti bile su preko 40mmHg, gde smo samo kod jednog bolesnika uspeli pravovremenom operativnom reakcijom da sprečimo smrtni ishod. *Acta Medica Medianae 2011;50(3):10-15.*

***Ključne reči**: traumatska moždana povreda, intrakranijalni monitoring, intrakranijalna hipertenzija*