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CT Follow-Up and Clinical Outcome in Severe Traumatic Injury Patients

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A B S T R A C T

Determining a patient's prognosis after severe traumatic brain injury remains difficult and complex. The purpose of the present study was following up patients with severe traumatic brain injury by correlating their clinical outcome and sequential computer tomography (CT) findings. We investigated 51 patients who survived the first year following an accident. All patients underwent successive CT examinations within a maximum period of 2 years. The patients' outcomes depended on the underlying brain damage and are presented by the Glasgow Outcome Scale. Based on the investigated data we concluded that the worst outcomes were experienced by patients with initial massive cerebral edema, extensive subdural hematoma and intraventricular hemorrhage, followed by stroke as subacute CT finding and cerebral atrophy as chronic finding visible at follow-up CT scans. The majority of lesions identified by CT scan were found in the frontal lobes, basal ganglia, and temporal lobes. We suggest that CT examination still represents a simple and useful tool in attempting to predict the clinical outcome in patients with severe traumatic brain injury.

Key words: *traumatic brain injury, computer tomography, clinical outcome*

Introduction

Traumatic brain injury (TBI) represents a major public health problem, especially among adolescent/young males and among elderly people of both sexes. Estimates of traumatic brain injury (TBI) incidence, severity, and cost reflect the enormous losses those injuries incur to individuals, their families, and society on the whole. Recent epidemiological data suggest that in the United States there are about 100–300 cases of traumatic brain injury (TBI) *per* 100,000 population *per* year[1,2]. Severe TBI accounts for 5–25% of that number, respectively[1–3].

The most common cerebral lesions related to TBI are contusions, intracerebral hemorrhage and subarachnoid hemorrhage (SAH). The latter two lesions may be seen in conjunction with cerebral vasospasm and hydrocephalus. TBI can produce diffuse brain injuries scattered throughout the brain, which may or, more often, may not be seen on CT scans, and which can occur with or without an associated mass lesion. It has been shown that ischemia as a variant of diffuse injury may also commonly occur in a significant number of TBI patients. Most linear skull fractures are not of prognostic importance; of greater concern is the possibility that forces strong enough to cause a skull fracture may also have caused some damage to the underlying brain. Fractures of the base of the skull are problematic since they open the path to intracranial infection, or cause injury to nerves, arteries, or other structures[4].

Despite extensive investigations it is still difficult to accurately predict the long-term outcome in the first weeks following traumatic brain injury[5–7]. The heterogeneity of the patients' premorbid health status, the nature and severity of the injury, the comorbidity, the interval from injury to initial treatment, the acute interventions, and the differences in follow-up protocols all create difficulties in the scoring system for head injury severity, since all these factors might affect the outcome. Some evidence suggests a correlation between a post-traumatic vegetative outcome and the presence of ventilatory dysfunction, decorticate posturing, and extraneural trauma[8], or age, pupillary abnormalities[9], and a low score on a test of motor responses[10]. The authors hypothesized that patients presenting with a GCS of 3 accompanied by fixed and dilated pupils have severe primary or secondary

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brain injury and no reasonable chance of functional recovery[8–10]. Patients who developed persistent vegetative state had a significantly higher frequency of MRI (magnetic resonance imaging)-detected lesions in the corpus callosum and corona radiata. Patients in persistent vegetative state also had significantly more injuries to the dorsolateral upper brain stem than patients who recovered[11].

In acute settings, CT still represents a vital tool in the assessment of patients with serious head injury. It remains the investigation of choice even following the advent of MRI, due to both the ease of monitoring of injured patients and the better demonstration of acute bleeding and bony injury. However, there are papers comparing CT and MR in detection of SAH giving the edge to MRI[12].

The purpose of the present study was to investigate the outcome of severe brain injury patients while following them up for more than one year after the accident. We have evaluated sequential CT findings and outcome/clinical state.

Patients and Methods

The study group consisted of 51 patients referred to the Coma Unit of the Hospital for Medical Rehabilitation, Krapinske Toplice, Croatia, following severe blunt head trauma priorly treated at neurosurgical departments in different institutions throughout the country. The group included 38 males and 13 females, ranging in age from 15 to 61 years. Patients eligible to participate were first hospitalized between January 2003 and December 2004, having survived a severe TBI defined as a GCS score of 8 and no spontaneous movement for 72 hours. All were reported to have been neurologically normal prior to injury.

Participants underwent CT examinations 3–5 times within the initial period, and follow-up scanning 2–4 times in the period from the following two weeks to 6 months later, as well as 2–4 scans from 6 months to a maximum period of 2 years. The CT examinations in acute settings were performed at different radiological (neurosurgical) centers, while follow-up scanning was performed on a Siemens Somatom Emotion 16 MSCT scanner at the Hospital for Medical Rehabilitation, Krapinske Toplice. After the period of one year following the traumatic event, the patients were assessed by neurologists at the rehabilitation center using the Glasgow Outcome Scale (GOS)[13].

Statistics

We performed descriptive statistics in order to establish the main features of the data. Moreover, nonparametric Spearman rank correlation was performed to evaluate the relationship between the variables (age, gender, GCS, cerebral edema, cerebral contusions, intracerebral hemorrhage, subarachnoid hemorrhage, intraventricular hemorrhage, subdural hemorrhage, epidural hemorrhage, hygroma, hydrocephalus, stroke, encephalomalacia, cerebral atrophy, pneumocephalus, abscess, meningitis, epilepsy, diffuse axonal injury, hemorrhagic shock, impressive fracture, as well as the mechanisms of accident including fall, pedestrian accident, bicycle/motorcycle or vehicle/car accident, and outcome expressed as GOS score). The levels of $p<0.01$ and $p<0.05$ were considered statistically significant. Discriminant analysis was used for building a predictive model of group membership based on the observed characteristics of each case. All continuous values are presented as mean ± standard error or as mean with a 95% confidence interval (CI) unless otherwise stated. All calculations were performed using SPSS for MS Windows, ver. 11.5[14] (http://spss-for-windows.software.informer. com/11.5/).

Results

Table 1 shows the frequency of different mechanisms of injury within patient groups. The mechanisms of injury were presented according to gender. The most frequent mechanism of injury was a vehicle/car accident (28/51, 54.9%), followed by falls. Mechanism of injury *vs.* gender crosstabulation reveals male predominance in the vehicle/car accidents (23/28, 82.1%) and falls (8/9, 88.9%). A general male dominance was found in all the investigated accident groups (38/51, 74.5%). Distribution of accidents according to age is shown in Figure 1. Adolescents and young adult men were the most represented group among our patients. Figure 2 shows the distribution of mechanisms of injury according to age. Younger

TABLE 1
MECHANISM OF INJURY ACCORDING TO GENDER CROSSTABULATION

Mechanism		Gender		Total
		Female	Male	
Fall	Count	1	8	9
	% within mechanism	11.1	88.9	17.6
	% within gender	7.7	21.1	
Pedestrian	Count	5	23	28
	% within mechanism	17.9	82.1	54.9
	% within gender	38.5	60.5	
Bicycle/ Motocycle	Count	2	3	5
	% within mechanism	40.0	60.0	9.8
	% within gender	15.4	7.9	
Vehicle/car	Count	5	23	28
	% within mechanism	17.9	82.1	54.9
	% within gender	38.5	60.5	
Explosion	Count	0	1	1
	% within mechanism	0.0	100.0	2.0
	% within gender	0.0	2.6	
Total	Count	13	38	51
	% within mechanism	25.5	74.5	

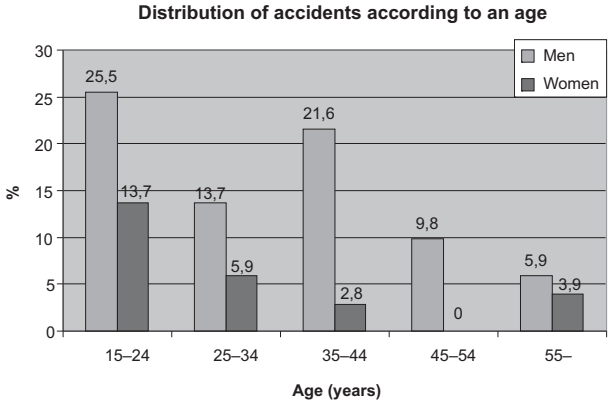

Fig. 1. Age according to mechanisms of injury. Box-plot presents medians, quartiles, minimum and maximum age within each group.

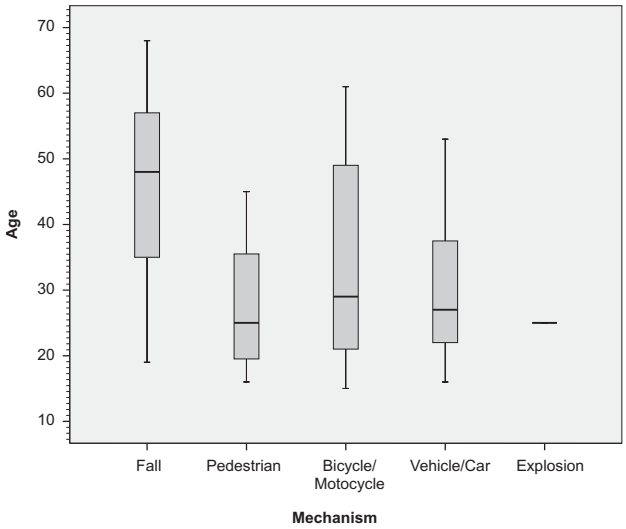

Fig. 2. Distribution of patients according to age and gender.

people mostly suffered injuries in traffic accidents, while older people sustained them in falls.

In patients with PVS, initial CT showed widespread lesions in the brain (Table 2), classified as contusion, contusion-hemorrhage and ischemia. These lesions were most frequently found in the frontal and temporal lobes. The majority of the contusions/hemorrhages visible by CT were found in the frontal, parietal and temporal lobes, and 9 were located in the pons. Secondary ischemic lesions were located in the occipital lobe, 2 unilaterally and 2 bilaterally, 3 in the pons, 2 in the frontal lobe, 3 unilaterally temporoparietally and 6 in the basal ganglia. In 6 patients signs of cerebral hemispheral or bilateral hypoxia were described.

17 (33.3%) of our patients remained in persistent vegetative state after the period of one year. Severe disability was observed in 18 (35.2%) of those who recovered from vegetative state. Moderate disability was present in 11 (21.5%) patients, 2 (3.9%) showed good recovery and 3 (5.88%) patients died before the period of one-year follow up. One patient died 3 months after the traumatic event due to infectious complications. Furthermore, one patient died 8 months after the incident from a massive pulmonary embolism. These causes of death are considered standard complications in severely ill, bed-ridden patients.

TABLE 2
PATHOLOGICAL FINDINGS (CONTUSION, HEMORRHAGE AND ISCHEMIA) REPORTED IN CT OF INPATIENTS WITH PSV

Site of lesion	%
Frontal lobe	52%
Temporal lobe	37%
Parietal lobe	17%
Occipital lobe	15%
Hippocampus/ Parahippocampal gyrus	4%
Basal ganglia	40%
Thalamus	16%
Pons	15%
Cerebellum	7%
Corpus callosum/Corona radiata	15%

The Spearman analysis shows that good recovery is in significant negative correlation with cerebral edema ($p<0.01$) and cerebral atrophy ($p<0.05$). Moderate disability outcome also shows a significant negative correlation with cerebral atrophy ($p<0.01$). Severe disability has a significant positive correlation with intraventricular hemorrhage ($p<0.05$). A significant positive correlation was seen between persistent vegetative state and cerebral edema ($p<0.01$), subdural hematoma ($p<0.01$), stroke ($p<0.05$), and cerebral atrophy ($p<0.01$), and a negative correlation was established with GCS ($p<0.01$). Other correlations considering outcome and CT finding were not statistically significant.

The Spearman correlation analysis reveals that GCS is in significant negative correlation with cerebral edema ($p<0.01$), intracerebral hemorrhage ($p<0.05$), and diffuse axonal injury (DAI) ($p<0.05$).

Cerebral contusions often coexist (in a statistically significant correlation) with intracerebral hemorrhage ($p<0.01$) and SAH ($p<0.05$), as well as with, in later CT scans, cerebral atrophy ($p<0.05$) and encephalomalacia ($p<0.01$). Intracerebral hemorrhage is in positive correlation with pneumocephalon ($p<0.05$) and cerebral atrophy ($p<0.05$). Intraventricular hemorrhage often appears with pneumocephalon ($p<0.05$). Subdural hemorrhage positively correlates with cerebral abscess ($p<0.05$). Cerebral edema is in significant correlation with meningitis ($p<0.05$), subdural hematoma ($p<0.05$), and DAI ($p<0.05$). Stroke correlates positively with subdural hematoma ($p<0.05$). Cerebral atrophy and encephalomalacia coexist ($p<0.01$). Pneumocephalon is in strong positive correlation with abscess ($p<0.01$).

Epilepsy is more frequent with advanced age ($p < 0.01$). According to our statistics, hemorrhagic shock more frequently occurs in younger age groups ($p < 0.05$).

Fall as mechanism of accident is more frequent in older patients ($p < 0.01$). In falls, cerebral abscess ($p < 0.01$) and impressive fractures ($p < 0.05$) are frequently found. Subdural hematoma is frequent in pedestrian accidents ($p < 0.01$).

Other examined correlations were not statistically significant.

Discussion and Conclusion

Prognosis in TBI can vary from essentially minor disability to severe damage of mental and/or motor functions and lethal exit. In respect to severe TBI, as estimated, the outcome is particularly discouraging. According to the data presented in a large European Brain Injury Consortium survey comprising more than 10,000 patients, the injury proved fatal in 31 percent, 3 percent remained in persistent vegetative state, and 16 percent were severely neurologically disabled[15]. Other similar investigations show comparable results[16–20]. As our patients were selected from those continuing their rehabilitation program at the Croatian Referral Coma Unit, and participation in this national program depends on the Croatian Institute for Health Insurance, our epidemiological data differ to a certain extent. The majority of our examined persons were younger severely disabled patients who had survived the first 3 months after a traumatic event. As many as 33.3% of the patients remained in persistent vegetative state after a period of one year, suggesting extensive brain damage as the cause of the definite clinical state.

According to the data comprised in our investigation, males are over three times more likely than females to experience TBI. The highest incidence is among persons 15 to 44 years of age (particularly in the 15–24 group). The most common cause of severe traumatic head injury was motor vehicle accident, with falls being the next most common cause; one investigated patient underwent an occupational accident. Motor vehicle accidents represent the main cause of TBI in the younger group of patients, while falls are the most common cause in the older group.

The outcome of head injuries largely depends on the extent and nature of primary damage and the effectiveness of therapy in preventing, or limiting, secondary brain damage. The worst prognosis was experienced by patients with initial massive cerebral edema (particularly malignant edema), extensive subdural hematoma and intraventricular hemorrhage, stroke as a sign of secondary brain lesions, and substantial cerebral atrophy visible at follow-up CT scans. The majority of lesions identified by this method were found in the frontal lobe, basal ganglia and temporal lobes.

Kampfl et al.[11,21] reported DAI with lesions in the corpus callosum and dorsolateral brainstem to be the key site in posttraumatic PVS visible by MR imaging. They also proved the basal ganglia to be the second most common site of injury in patients with PVS. Our results established by CT scans revealed 40% basal ganglia and only 15% corpus callosum/corona radiata lesions, with the majority of lesions visible in the frontal lobe (52%). The appearance/description of DAI as an independent radiological entity is too low on CT findings to be of statistical importance in this study. In spite of PVS being a well-known and common entity, not much literature is available concerning CT findings in PVS. However, in the study by Levin et al. brainstem injury was uncommon[18]. MRI is far superior to CT scan in detecting DAI, lesions of the corpus callosum and brainstem lesions. Nevertheless, CT is still a vital tool in the assessment of patients with serious head injury. It remains the investigation of choice even following the advent of MRI, due both to the ease of monitoring of injured patients and the better demonstration of fresh bleeding and bony injury.

Our patients with a favorable outcome had significantly fewer secondary insults than the patients with an unfavorable outcome. The injured brain is exceptionally vulnerable to secondary ischemic insults and their occurrence is associated with a poor neurological outcome after severe TBI[22]. Several factors may contribute to posttraumatic cerebral ischemia, including systemic arterial hypotension, increased ICP, cerebral edema, focal tissue compression from hematomas, and microvascular disease[23–26]. As a consequence of traumatic brain injury, excitatory amino acids are released which, in turn, generate an overproduction of second messengers[27,28]. Cerebral ischemia is one of the most important causes of secondary brain injury in TBI. For example, 90% of patients with TBI with a fatal injury showed regional and global cerebral ischemia on autopsy[29]. Our investigation shows a prevalence of occipital lobe and pons ischemic infarction. The posterior circulation involvement could be explained by the compression of the vessels in the ambient cistern secondary to the midline shift. The most common localization of ischemic infarct is the basal ganglia. The anterior stretch of the lateral branch of the perforator of the middle cerebral artery plays a major role in its pathogenesis[30]. More importantly, patients with multiple trauma are particularly susceptible to such a secondary insult because of hemorrhagic shock and/or concomitant lung injury[31]. The common association of subarachnoid hemorrhage should be mentioned as well, suggesting that vasospasm could also be responsible for ischemic posttraumatic lesion. In particular, hypoperfusion can occur in the brain, surrounding a focal contusion or underlying a subdural hematoma[32].

According to studies conducted during the first several months after a traumatic or nontraumatic brain injury, patients in persistent vegetative state remain more severely disabled if serial neuroimaging scans are normal than if they are abnormal. Serial scanning usually documents progressive brain atrophy, which reduces the likelihood of neurological recovery.

A computed tomography scan still represents the gold standard for the radiological assessment of a TBI patient. A CT scan is easy to perform and it is an excellent test for detecting the presence of blood and fractures, which are the most crucial lesions to identify in medical trauma cases. MRI is not commonly used for acute head injury because it takes longer to perform MRI than CT scanning, and it is sometimes difficult to transport an acutely-injured patient from the emergency room to the MRI scanner. However, after a patient has been stabilized, MRI may demonstrate the existence of lesions that were not detected on the CT scan.

With regard to this data severe TBI represents a major public health problem, often associated with certain ethical and legal dilemmas. Traumatic brain injury can cause death, but it can also cause disabling injury requiring extensive and costly rehabilitation. For many people, a brain injury triggers a significant change in lifestyle and function. Predicting which patients with severe brain damage will progress to persistent vegetative state is extremely important, as crucial decisions about life support, resuscitation, tube feeding and other issues have to be faced. Although numerous studies have examined a wide variety of clinical and laboratory variables, no well-established criteria applied during the period of coma can predict a vegetative outcome with certainty.

It is becoming increasingly clear that the management of patients with multiple traumas and severe TBI is a very complex task. Diagnosis and therapy need to be primarily directed at preventing multiorgan failure and minimizing secondary brain injury. Thus, early control of hemorrhage, stabilization of circulation and tissue oxygenation, optimization of cerebral perfusion pressure and cerebral blood flow, as well as adequate treatment of increased intracranial pressure are mandatory.

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PRAĆENJE TEMELJEM KOMPJUTERIZIRANE TOMOGRAFIJE I KLINIČKI ISHOD U BOLESNIKA S TEŠKOM TRAUMATSKOM OZLJEDOM GLAVE

S A Ž E T A K

Klinički ishod bolesnika nakon teške traumatske ozljede glave je teško predvidiv. Svrha ovog istraživanja je bila praćenje bolesnika s teškom traumatskom ozljedom glave i usporedba kliničkog ishoda bolesnika i nalaza sekvencijske kompjuterizirane tomografije (CT) glave. U istraživanje je uključen 51 preživjeli bolesnik nakon godinu dana od traumatskog događaja. Svi bolesnici su uzastopno praćeni nalazima CT glave u maksimalnom vremenskom odmaku od 2 godine. Klinički ishod bolesnika koji je u ovisnosti o morfološkom oštećenju mozga je prezentiran pomoću Glasgow Outcome skale. Na temelju ispitanih podataka smo zaključili da su bolesnici s najtežim ishodom trpjeli početni opsežni moždani edem, veliki subduralni hematom i intraventrikularno krvarenje, nakon čega slijedi moždani udar kao znak sekundarne lezije mozga i moždana atrofija kao kronični nalaz vidljivi na uzastopnim nalazima CT glave. Većina lezija mozga identificiranih temeljem CT nalaza su dokazane u čeonim režnjevima, bazalnim ganglijima i sljepoočnom režnju. Naša je sugestija da kompjuterizirana tomografija još uvijek predstavlja jednostavno i korisno dijagnostičko sredstvo temeljem kojeg je moguće predvidjeti klinički ishod u bolesnika s teškom traumatskom ozljedom glave.