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Secondary stroke in patients with polytrauma and traumatic brain injury treated in an Intensive Care Unit, Karlovac General Hospital, Croatia

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ABSTRACT

Traumatic brain injury (TBI) is divided into primary and secondary brain injury. Primary brain injury occurs at the time of injury and is the direct consequence of kinetic energy acting on the brain tissue. Secondary brain injury occurs several hours or days after primary brain injury and is the result of factors including shock, systemic hypotension, hypoxia, hypothermia or hyperthermia, intracranial hypertension, cerebral oedema, intracranial bleeding or inflammation. The aim of this retrospective analysis of a prospective database was to determine the prevalence of secondary stroke and stroke-related mortality, causes of secondary stroke, treatment and length of stay in the ICU and hospital. This study included patients with TBI with or without other injuries who were hospitalised in a general ICU over a five-year period. The following parameters were assessed: demographics (age, sex), scores (Glasgow Coma Score, APACHE II, SOFA), secondary stroke (prevalence, time of occurrence after primary brain injury, causes of stroke and associated mortality), length of stay in the ICU and hospital, vital parameters (state of consciousness, cardiac function, respiration, circulation, thermoregulation, diuresis) and laboratory values (leukocytes, C-reactive protein [CRP], blood glucose, blood gas analysis, urea, creatinine). Medical data were analysed for 306 patients with TBI (median age 56 years, range 18–93 years) who were treated in the general ICU. Secondary stroke occurred in 23 patients (7.5%), 10 of whom died, which gives a mortality rate of 43.4%. Three patients were excluded as the cause of the injury was missile trauma. The study data indicate that inflammation is the most important cause of secondary insults. Levels of CRP were elevated in 65% of patients with secondary brain injury; leukocytosis was present in 87% of these patients, and blood glucose was elevated in 73%. The lungs and urinary tract were the most common sites of infection. In conclusion, elevated inflammatory markers (white blood cell count and CRP) and hyperglycaemia are associated with secondary brain injury. The lack of routine use of intracranial pressure (ICP) monitoring may explain the high mortality rate and the occurrence of secondary stroke in patients with TBI.

Introduction

Traumatic brain injury (TBI) with or without other injuries is one of the most common causes of disability and death. TBI is divided into primary and secondary brain injury. Population-based studies in the United States indicate that the incidence of TBI is between 180 and 250 per 100,000 population per year [1]. Primary brain injury is caused by external forces, whereas secondary brain injury results from arterial hypotension, hypoxaemia, hypercarbia, severe hypocarbia, pyrexia, hyponatraemia, anaemia, diffuse intravascular coagulopathy, intracranial hypertension, brain oedema, intracranial bleeding, cerebral vasospasm, hyperglycaemia, seizures and infection [2,3]. The severity of primary brain injury is the major determinant of outcome in TBI patients [3]. TBI exerts irreversible anatomical damage on the brain; therefore, the focus during resuscitation should be on secondary injury [3]. The aim of

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the present analysis was to determine the prevalence of secondary stroke, causes of secondary stroke and associated mortality, treatment and length of stay in the ICU and hospital.

Materials and methods

This study was a retrospective analysis of a prospective database of patients with brain injury who were hospitalised in the general ICU at Karlovac General Hospital over a five-year period (2008–2013). Clinical and paraclinical parameters and demographics (age, sex) of the patients were evaluated. The clinical parameters included the Glasgow Coma Scale (GCS) as an indicator of the state of consciousness on admission and after the occurrence of secondary insults. The Acute Physiology and Chronic Health Evaluation score (APACHE II) and the Simplified Acute Physiology Score (SAPS II) were used as predictors of mortality. The following parameters were also evaluated: frequency of secondary stroke, time of occurrence after primary brain injury, causes of stroke and associated mortality, and length of stay in the ICU and hospital. The following vital parameters and laboratory values were monitored: electrocardiography (ECG), mean arterial pressure (MAP), pulse oximetry, peripheral oxygen saturation, arterial blood gas analysis, end expiratory CO_2 during mechanical ventilation, blood and skin temperature, haemoglobin, haematocrit, glucose, creatinine, urea and inflammatory markers (leukocytes, C-reactive protein [CRP], and number of non-segmented leukocytes). The Marshall CT classification was used as a predictor of outcome [4]. Descriptive statistics were used to evaluate patient demographics (median age, sex), length of stay in the ICU and hospital, the assessment scales of consciousness and predictive mortality, laboratory values and vital parameters.

Results

The study comprised 306 patients with TBI who were treated in the general ICU. The median age of the patients was 56 years (range 18–93 years) and overall mortality was 29%. Secondary stroke occurred in 23 patients (7.5%), 10 of whom died, which gives a mortality rate of 43.4% in patients with secondary stroke. Three of the patients with secondary brain injury were excluded as the cause of injury was missile trauma. The median age of the patients with secondary stroke was 62 years (range 21–83 years). The mean occurrence of secondary stroke was 8.56 ± 8.16 days after primary brain injury. The length of stay in the ICU was 13.39 ± 15.54 days, and the length of stay in hospital was 29.13 ± 27.16 days. The mean GCS at the time of admission was 7 ± 4, and after the occurrence of secondary stroke was 8 ± 5. The mean predictive mortality at the time of admission to the ICU was 43% according to the APACHE II score and 41% according to the SAPS II score. The mean predictive mortality at the time of secondary stroke was 55% according to the APACHE II score, whereas it was 46% according to the SAPS II score (Table 1). There were signs of inflammation at the time of secondary stroke, with elevated CRP in 65% of patients and elevated leukocytes in 87% of patients. The lungs and urinary tract were the most common sites of infection. Blood glucose was elevated in 74% of patients with secondary stroke. Descriptive statistics for laboratory values at the time of TBI and secondary stroke are shown in Tables 2 and 3, respectively. Eleven of 23 patients with secondary stroke underwent a neurosurgical evacuation of intracranial mass lesions. Based on the Marshall CT classification [4], 63% of the patients were grade IV, and related predictive mortality was 56.2% (Table 4).

Table 1
Descriptive statistics of vital signs rating scales.

Rating scales	$X \pm$ SD
GCS/24 h	7 ± 4
GCS/SS	8 ± 5
APACHE II/24 h	23 ± 11 (*M* 43%)
SAPS II/24 h	48 ± 15 (*M* 41%)
APACHE II/SS	26 ± 9 (*M* 55%)
SAPS II/SS	50 ± 20 (*M* 46%)

GCS—Glasgow Coma Score, APACHE II—the Acute Physiology and Chronic Health Evaluation score, SAPS II—the Simplified Acute Physiology Score, SS—secondary stroke, *M*—mortality, *X*—mean, SD—standard deviation.

Table 2
Descriptive statistics of laboratory values and vital functions at the time of traumatic brain injury.

Laboratory values and parameters of vital functions	$X \pm$ SD
Leukocytes	16.06 ± 15.3
CRP	42 ± 55
Blood glucose	7.75 ± 2.36
PaO_2	112 ± 44
MAP	95 ± 18

CRP—C-reactive protein, PaO_2—partial pressure of oxygen in arterial blood. MAP—mean arterial blood pressure, *X*—mean, SD—standard deviation.

Discussion

The incidence of TBI has been shown in some studies to peak in adolescents and young adults [1]; however, in a prospective population-based study in France, the highest incidence of TBI was found in the elderly (>75 years) [5]. TBI is associated with temporary or permanent limitation of activities and participation, as assessed by the disability rating scale (DRS) [6]. These activities include arousability, awareness, responsivity, cognitive ability for self-care activities, dependence on others, and psychosocial adaptability [6]. Varjačić et al. showed that TBI patients had statistically significant improvement in their activities and progression three to five years after the completion of hospital rehabilitation, as evaluated using the DRS [6]. In the present study, the highest incidence of TBI was found in middle-aged individuals (56 years). Traffic accidents were the most common cause of TBI in adolescents and younger adults in this study, whereas falls were the most common cause in children and elderly individuals. Similar data have been reported by Bruns and Hauser in their review [1]. Fig. 1a shows a CT scan of a patient who developed a subdural haematoma after brain trauma, with brain oedema and midline shift. Fig. 1b shows a CT scan of the same patient after a secondary stroke in the frontal lobe. The study comprised a total of 219 men and 83 women with TBI, which gives a male-to-female

Table 3
Descriptive statistics of laboratory values at the time of secondary stroke.

Laboratory values and parameters of vital functions	$X \pm$ SD
Leukocytes	19.01 ± 14.5
CRP	108 ± 81
Blood glucose	8.92 ± 2.71

CRP—C-reactive protein, *X*—mean, SD—standard deviation.

Table 4
Marshall CT classification of traumatic brain injury.

Grade	*N*	*M*
I	1	9.6%
II	7	13.5%
III	0	34%
IV	15	56.2%

N—number of patients, *M*—predictive mortality.