Risk Factors for Mortality in Head-Injured Patients with Probability of Survival Greater Than 0.5

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Background: *Traumatic brain injury is one of the major problems and leading cause of death worldwide. The present study was aimed to identify factors responsible for mortality by comparing survivors and nonsurvivors in patients that had a low probability of mortality.*

Material and Method: *A nested case-control study was conducted at Sawanpracharak Hospital from January 1, 2007 to December 31, 2007. All head injury deaths that had TRISS-PS greater than 0.5 were enrolled as the "case" patients. Head injured patients with TRISS-PS greater than 0.5 and that survived were chosen as the "control" patients. The number of controls per case was 2:1. Patients with ages < 15 were excluded from the present study.*

Results: *Six factors associated with increased mortality are age ≥ 45 years (OR = 1.61, 95% CI = 1.1-2.3), alcohol intoxication (OR = 3.11, 95% CI = 1.4-6.9), admission GCS 3-8 (OR = 4.16, 95% CI = 2.4-7.2), arrival GCS-M ≤ 4 (OR = 1.46, 95% CI = 1.0-2.0), Head-AIS ≥ 4 (OR = 3.31, 95% CI = 1.3-8.3), and admission SBP < 90 mmHg (OR = 13.36, 95% CI = 3.2-56.3).*

Conclusion: *Head injury continues to be a substantial public health problem. Deaths that met criteria for low probability of mortality, especially in those without associated risk factors should be analyzed for errors that may be preventable.*

Keywords: *Traumatic brain injury, TRISS-derived Probability of survival, Risk factor, Mortality*

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Traumatic brain injury (TBI), with an estimated 10 million cases a year, is one of the major problems and a leading cause of death worldwide[1]. The annual incidence of head injuries has been estimated at 150 to 300 cases per 100,000 population[2-4]. The management of severe and moderate traumatic brain injuries is very labor-intensive and costly for the institution and the families involved. Factors such as mechanism of injury, intoxication level, intubation status, pupil reactivity, hypotension and hypoxia were reported in several studies to be significant in the assessment of head injury severity and outcome[5-8].

During 2006, 18,168 trauma patients attended the emergency department (ED) at Sawanpracharak Hospital and 2,458 patients were hospitalized. Overall mortality rate was 9.6%. Of the 2,458 that were hospitalized, 59.9% were head injured patients. Of all traumatic death, 89.1% died from head injury, and 47.1% of which had more than 50% probability of survival.

The aim of the present study was to identify factors responsible for mortality by comparing survivors and nonsurvivors in patients that had a low probability of mortality.

Material and Method

A nested case-control study was conducted at Sawanpracharak Hospital from January 1 to December 31, 2007. In this period, all head injury deaths that had more than 50% probability of survival at the time of admission, as determined by the Trauma Injury Severity Score (TRISS-PS) were identified and enrolled as the "case" patients. Other head injured and survived patients with more than 50% probability of survival were chosen at random from the same day of admission and became the "control" patients. The number of controls per case was 2:1. Patients who sustained head injuries and died at the scene or en route to the hospital were excluded from the present study. Patients with ages < 15 were also excluded.

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Data were obtained from medical record, imaging reports, operating room reports and outpatient reports. Data collected include demographic and clinical characteristics (age, gender, alcohol intoxication, underlying disease), injury mechanism, type of injury, sites of injury, direct transport or interhospital transfer, endotracheal intubation prior to hospital arrival or in the ED, arrival systolic blood pressure(SBP), arrival Glasgow Coma Scale (GCS), oxygen saturation (SpO_2), admission SBP, admission GCS, pupil reactivity, hematocrit (Hct), head Abbreviated Injury Scale (AIS), Injury Severity Score (ISS), computed tomography (CT), surgical interventions, hospital complications, transfer times, and length of stay.

The χ^2 test and Fisher's exact test were used to compare categorical qualitative variables. Student's t-test was used for comparison of continuous quantitative variables. The association between the groups was measured using the odds ratio with 95% confidence intervals for every prognostic factor. Only variables with a p-value ≤ 0.05 in the separately analyses were selected and studied in the logistic regression analyses. For all statistical tests used, a value of $p < 0.05$ was considered statistically significant. Analysis was performed using STATA 10SE.

Results

Demographics and patient characteristics

During the one-year study period, the final case-control study of 333 adult patients with head injury, of which met low mortality risks (TRISS-PS > 0.5), were enrolled into the present study. Some prehospital variables were excluded from the analysis because of inadequate data. One hundred eleven that died represent the case group and 222 that survived represent the control group. Demographic and clinical data are demonstrated in Table 1.

Univariate and multivariate analysis of mortality

Univariate analysis showed no significant differences in gender, underlying disease, injury mechanism, transfer time, site of injury, skull fracture, and epidural hematoma between the two groups. There was a statistically significant difference between the case group and the control group, regarding age ($p = 0.002$), alcohol intoxication ($p < 0.001$), traffic accidents ($p = 0.022$) and hospital transfer ($p = 0.002$). There was also a statistically significant difference between the two groups in regard to TRISS-PS ($p < 0.001$), head AIS score ($p < 0.001$), ISS score ($p < 0.001$), arrival GCS ($p < 0.001$), arrival GCS motor component (GCS-M) ($p < 0.001$), $SpO_2 < 92\%$ ($p = 0.001$), arrival SBP < 90 mmHg ($p = 0.001$), pupil reactivity ($p < 0.001$), admission GCS ($p < 0.001$), admission SBP < 90 mmHg ($p < 0.001$), Hct < 30% ($p = 0.001$), subdural hematoma ($p < 0.001$), brain contusion ($p = 0.022$), intraparenchyma hematoma ($p < 0.001$) and brain edema ($p = 0.005$) (Table 1).

Eighteen variables associated with an increased mortality met criteria for inclusion in multivariate analysis (Table 2). Only six variables, age 45 years or older (OR1.6, 95% CI 1.1-2.3, $p = 0.012$), alcohol intoxication (OR3.1, 95% CI 1.4-6.9, $p = 0.005$), admission GCS score 3-8 (OR4.2, 95% CI 2.4-7.2, $p < 0.001$), arrival GCS-M ≤ 4 (OR1.5, 95% CI 1.0-2.0, $p = 0.025$), head AIS score ≥ 4 (OR3.3, 95% CI 1.3-8.3, $p = 0.011$) and admission SBP < 90 mmHg (OR13.4, 95% CI 3.2-56.3, $p < 0.01$) were independent predictors of mortality (Table 3).

Discussion

Road traffic crashes were the most common cause of injury (71.5%) and 78.2% of them had been injured in motorcycle accidents. The head and extremities were the most frequently injured organs. The mortality of the elderly trauma population varies between 10% and 34%[9,10]. Several investigators have stated that age is a good indicator of mortality in cases of traumatic head injury, especially for those in the extreme age groups[8,11,12]. Some authors have suggested a critical age threshold for worsening prognosis at 55 to 60 years of age[8,13]. Some studies have found stepwise thresholds for risk, particularly with age > 65[14]. The present study showed that 54.1% of all deceased patients occurred in age 45 years or older, and 25.2% occurred in patients who were 60 years or older. The OR varied with age: 1.0 for age 15 to 29 years, 1.1 for age 30 to 44 years, 2.2 for age 45 to 59 years and 2.8 for age ≥ 60 years. The mortality for patients age ≥ 45 years was 54.1%, compared with 32.9% for patients age 15-44 years. Patients age 45 years or older increased odds of death 1.6 compared with age 15-44 years in multivariate analysis.

Alcohol consumption is known to increase the overall risk of injury[15], but decreases the likelihood of death when injury severity and all other significant factors are accounted for. Rutledge and Messick reported that alcohol intoxication was associated with between 14% and 64% of fatal injuries in North Carolina, and found the strong association of alcohol in trauma-related deaths[16]. In the current study, 42.0% were under the influence of alcohol. The

Table 1. Demographic and clinical characteristics: comparison of case and control group

Variable	Cases (n = 111)	Controls (n = 222)	p-value
Age (yr)			
15-29	29 (26.1%)	87 (39.2%)	0.002
30-44	22 (19.8%)	62 (27.9%)	
45-59	32 (28.9%)	43 (19.5%)	
$\geq$ 60	28 (25.2%)	30 (13.5%)	
Mean $\pm$ SD	45.2 $\pm$ 18.9	37.9 $\pm$ 17.7	
Male:Female	86:25 (77.5%:22.5%)	167:55 (75.2%:24.8%)	0.650
Underlying disease	29 (26.1%)	50 (22.5%)	0.466
Alcohol intoxication	60 (70.6%)	80 (38.5%)	<0.001
Hospital transfer	85 (76.6%)	132 (59.5%)	0.002
Intubated at ED	61 (55%)	15 (6.8%)	
Blunt:Penetrating	108:3 (97.3%:2.7%)	212:10 (95.5%:4.5%)	0.424
Traffic accident	89 (80.2%)	149 (67.1%)	0.022
Motorcycle accident	63 (70.8%)	123 (82.6%)	
Other	26 (29.2%)	26 (17.4%)	
Transfer time (min)	433.4 $\pm$ 922.8	858.6 $\pm$ 1437.7	0.342
TRISS-PS			
0.51-0.60	12 (10.8%)	0	<0.001
0.61-0.70	17 (15.4%)	2 (0.9%)	
0.71-0.80	14 (12.6%)	1 (0.5%)	
0.81-0.90	23 (20.7%)	8 (3.6%)	
0.91-1.00	45 (40.5%)	211 (95.0%)	
Mean $\pm$ SD	0.82 $\pm$ 0.14	0.97 $\pm$ 0.80	
Head AIS score			
1	0	2 (0.9%)	<0.001
2	1 (0.9%)	85 (38.3%)	
3	26 (23.4%)	68 (30.6%)	
4	73 (65.8%)	52 (23.4%)	
5	11 (9.9%)	15 (6.8%)	
Mean $\pm$ SD	3.8 $\pm$ 0.6	2.9 $\pm$ 0.9	
ISS score			
1-14	19 (17.1%)	156 (70.3%)	<0.001
$\geq$ 15	92 (82.9%)	66 (29.7%)	
Mean $\pm$ SD	17.39 $\pm$ 5.22	10.55 $\pm$ 6.55	
Arrival GCS			
3-8	86 (77.5%)	37 (16.7%)	<0.001
9-12	16 (14.4%)	26 (11.7%)	
13-15	9 (8.1%)	159 (71.6%)	
Mean $\pm$ SD	6.6 $\pm$ 3.2	12.9 $\pm$ 3.2	
Arrival GCS-M			
1	15 (13.5%)	0 (0)	<0.001
2	20 (18.0%)	2 (0.9%)	
3	11 (9.9%)	2 (0.9%)	
4	22 (19.8%)	9 (4.1%)	
5	31 (27.9%)	41 (18.5%)	
6	12 (10.8%)	168 (75.7%)	
SpO_2 (%)			
< 92	16 (17.6%)	4 (3.8%)	0.001
$\geq$ 92	75 (82.4%)	101 (96.2%)	
Arrival SBP (mmHg)			
< 90	8 (7.2%)	2 (0.9%)	0.001
$\geq$ 90	103 (92.8%)	220 (99.1%)	
Mean $\pm$ SD	138 $\pm$ 34.5	132 $\pm$ 23.0	

Table 1. (Cont.)

Variable	Cases (n = 111)	Controls (n = 222)	p-value
Pupil reaction			
Normal pupils	49 (44.1%)	200 (90.1%)	<0.001
Unilateral dilated pupils	22 (19.8%)	18 (8.1%)	
Bilateral dilated pupils	40 (36.1%)	4 (1.8%)	
Admission SBP (mmHg)			
< 90	18 (16.2%)	2 (0.9%)	<0.001
$\geq$ 90	93 (83.8%)	220 (99.1%)	
Mean $\pm$ SD	123.7 $\pm$ 41.9	130.2 $\pm$ 23.8	
Hct (%)			
< 30	25 (22.9%)	20 (9.4%)	0.001
$\geq$ 30	84 (77.1%)	192 (90.6%)	
Admission GCS			
3-8	91 (81.98%)	36 (16.2%)	<0.001
9-12	12 (10.8%)	21 (9.5%)	
13-15	8 (7.2%)	165 (74.3%)	
Mean $\pm$ SD	6.2 $\pm$ 3.1	13.0 $\pm$ 3.2	
Skull fracture	24 (21.6%)	62 (27.9%)	0.215
Epidural hematoma	7 (6.3%)	22 (9.9%)	0.272
Subdural hematoma	27 (24.3%)	23 (10.4%)	<0.001
Cerebral contusion	13 (11.7%)	49 (22.1%)	0.022
Intraparenchymal hematoma	33 (29.7%)	30 (13.5%)	<0.001
Cerebral edema	22 (19.8%)	20 (9.0%)	0.005

TRISS-PS = trauma injury severity score-derived probablility of survival; Transfer time = time from injury to arrival at ED; ED = emergency department; GCS = Glasgow Coma Scale; GCS-M = motor component of GCS; AIS = abbreviated injury scale; ISS = Injury Severity Score; SpO_2 = oxygen saturation; SBP = systolic blood pressure

mortality for patients without alcohol was 38.5%, compared with 70.6% for patients with alcohol. Alcohol used significantly increases the odds of mortality to 3.1 after TBI in multivariate analysis.

Secondary brain injury is the leading cause of in-hospital deaths after TBI[17,18]. Multiple studies documented the adverse effects of prehospital hypoxia on outcome in patients with severe traumatic brain injury[19,20]. Intubation in unconscious, severely injured patients is a life-saving intervention. Two-thirds of the patients who were in coma when transferred to the neurosurgical unit arrived without an endotracheal tube in place[21]. In the current study, SpO_2 lower than 92% correlated with mortality in the univariate analysis (p = 0.001). The transferred group had a higher mortality than direct admission group (76.6% vs. 59.5%). Fifty five percent of patients in the case group were intubated at ED, compared with 6.8% in the control group. Almost all that were intubated arrived unconscious with severe head injuries. The reason for the lower incidence of intubation is not clear. The lack of trauma protocols and guidelines makes the stabilization and transfer of injured patients an inefficient process that may contribute to mortality.

Hypotension occurs in about one-third of patients with severe traumatic brain injury and is one of the most important predictors of outcome. In the prehospital setting, nearly 40% of patients with traumatic brain injury sustain a secondary insult. Hypotension accounts for 11% and both hypoxia and hypotension for 24%[20]. In the current study, arrival SBP < 90 mmHg and Hct < 30 mg% were correlated with mortality only in the univariate analysis, whereas admission SBP < 90 mmHg appeared to be the most significant factor in multivariate analysis with an odds ratio of 13.4 (95% CI 3.2-56.3, p < 0.01). Prompt and appropriate resuscitation is, therefore, an essential part of the supportive care provided for these patients with brain injury during all aspects of transport and resuscitation. Patients that did not respond to resuscitation at ED had a poor prognosis.

In a prospective study, Ritter et al[22] found that there was a significant relation between pupil reactivity and cerebral blood flow, and when the GCS

Table 2. Univariate analysis of predicting mortality/risk factors for death with trauma brain

Variables	OR	95% CI	p-value
Age (y)			
15-29	1.0	-	
30-44	1.1	0.6-2.0	<0.001
45-59	2.2	1.2-4.1	
≥ 60	2.8	1.5-5.4	
Alcohol intoxication	3.8	2.2-6.9	<0.001
Hospital transfer	2.2	1.3-3.9	0.002
TRISS-PS			
0.51-0.9	28.1	13.3-63.1	
0.91-1.00	1.0	-	<0.001
Head AIS score			
≥ 4	7.2	4.2-12.6	<0.001
1-3	1.0	-	
Injury Severity Score			
≥ 15	11.4	6.3-21.4	<0.001
1-14	1.0	-	
Arrival GCS			
3-8	41.1	21.9-77.0	<0.001
9-12	10.9	4.9-24.2	
13-15	1.0	-	
Arrival GCS-M			
2	77.0	5.4-20.6	<0.001
3	140.0	27.8-213.3	
4	34.2	15.8-74.0	
5	10.5	54.1-362.2	
6	1.0	-	
$Sp0_2$(%)			
< 92	5.4	1.6-22.9	0.001
≥ 92	1.0	-	
Arrival SBP (mmHg)			
< 90	8.5	1.7-83.4	0.001
≥ 90	1.0	-	
Pupil reaction			
Bilateral dilated	40.8	18.9-88.1	<0.001
Unilateral dilated	4.98	2.6-9.6	
Normal	1.0	-	
Admission GCS			
3-8	52.1	27.6-98.5	<0.001
9-12	11.8	5.0-27.8	
13-15	1.0	-	
Admission SBP (mmHg)			
< 90	21.3	4.9-191.2	<0.001
≥ 90	1.0	-	
Hct (%)			
< 30	2.9	1.4-5.7	0.001
≥ 30	1.0	-	
Subdural hematoma	2.8	1.4-5.4	0.001
Cerebral contusion	0.5	0.2-0.9	0.022
ICH	2.7	1.5-4.9	0.004
Cerebral edema	2.5	1.2-5.1	0.005

OR, odd ratio;

Table 3. Multivariate analysis of predicting mortality

Variable	Odd ratio	95% CI	p-value
Age ≥ 45 yr	1.6	1.1-2.3	0.012
Alcohol intoxication	3.1	1.4-6.9	0.005
Admission GCS 3-8	4.2	2.4-7.2	<0.001
Head-AIS ≥ 4	3.3	1.3-8.3	0.011
Arrival GCS-M ≤ 4	1.5	1.0-2.0	0.025
Admission SBP < 90 mmHg	13.4	3.2-56.3	<0.001

score was less than 8, death or a vegetative state was the outcome for 39% of the patients with two reactive pupils, 66% of those with only one reactive pupil and in 85% of those whose pupils both were nonreactive. In the current study, unilateral dilation was observed in 19.8% in the case group compared with 8.1% in the control group, and bilateral dilation in 36.1% in the case group compared with 1.8% in the control group. Pupil reactivity was correlated ($p < 0.001$) with mortality only in the univariate analysis.

A combination of both the anatomic and physiologic measures of injury severity would be better predictors than either measure alone. TRISS is the most widely used combined scoring system. It has remained the gold standard for comparing outcomes from different hospitals. Comparison between the two groups in the current study demonstrated that the injury severity (TRISS-PS, head AIS score, ISS score) were more severe in the case group, but in multiple regression analysis only head AIS ≥ 4 was statistically significantly. Field GCS and arrival GCS, which are purely physiologic measure of injury severity, were predictive of survival[23]. The ability to obtain an accurate initial GCS score in the ED may be compromised by prehospital secondary insults. Approximately 30% of patients admitted to hospital with moderate and severe head injury will ultimately die[24]. Mortality for those with GCS ≤ 8 after resuscitation may be as high as 50%[11,25,26]. The univariate analysis in the current study found that arrival GCS score 3 to 8 ($p < 0.001$), arrival GCS-M ≤ 4 ($p < 0.001$) and admission GCS score 3-8 ($p < 0.001$) correlates with mortality, whereas the multivariate analysis found significance only arrival GCS-M ≤ 4 (OR1.5, 95% CI 1.0-2.0, $p = 0.025$) and admission GCS score 3-8 (OR4.2, 95% CI 2.4-7.2 $p < 0.001$).

The seriousness of TBI reflected by the CT scan is assumed to predict the clinical course and outcome. The influence of the type of cerebral lesion on cranial pressure and mortality has been evaluated variously in the literature[27-29]. In the current study, subdural hematoma (p < 0.001), brain contusion (p = 0.022), intraparenchyma hematoma (p < 0.001) and brain edema (p = 0.005) were correlated with death only in the univariate analysis.

Mortality risks in low probability of mortality differed from those in high probability of mortality. Deaths that met criteria for low probability of mortality were defined as TRISS unexpected deaths. When a patient has not sustained overwhelming impact damage to the brain but dies, it is reasonable to assume that death might have been avoided. Since the classic report in 1979 by West and Trunkey[30] documented that one third of central nervous system-related deaths were preventable in a county without a trauma center. This compared with a 1% preventable death rate in a county with an established trauma center. The rate of preventable trauma deaths in the literature is 30% in nontrauma hospital and 1-5% in trauma centers[31]. In Sawanpracharak Hospital, 47.1% of head injury deaths were TRISS unexpected death. These unexpected outcomes should serve as filters for peer review to identify preventable death.

In conclusion, TBI continues to be a substantial public health problem. Despite its retrospective nature, the present study was able to define six risk factors indicative significantly associated with survival in patients with head injury and had TRISS-PS > 0.5. Age $\geq$ 45 years and GCS-M $\leq$ 4 at ED increased risk of death by 50% (OR 1.6, 95% CI 1.1-2.3, and OR 1.5, 95% CI 1.0-2.0). Alcohol intoxication, head AIS $\geq$4, and admission GCS score 3 to 8 had a 3 to 4 fold increased odds of death (OR 3.1, 95% CI 1.4-6.9, OR 3.3, 95% CI 1.3-8.3 and OR 4,2, 95% CI 2.4-7.2). There is more than tenfold greater odds of death for admission SBP < 90 mmHg (OR 13.4, 95% CI 3.2-56.3). Deaths that met criteria for low probability of mortality, especially in those without associated risk factors should be analyzed for errors that were preventable.

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Potential conflicts of interest

None.

References

1. Alexander E Jr. Global Spine and Head Injury Prevention Project (SHIP). Surg Neurol 1992; 38: 478-9.
2. Diamond PT. Brain injury in the Commonwealth of Virginia: an analysis of Central Registry data, 1988-1993. Brain Inj 1996; 10: 413-9.
3. Kraus JF, McArthur DL. Epidemiologic aspects of brain injury. Neurol Clin 1996; 14: 435-50.
4. Tiret L, Hausherr E, Thicoipe M, Garros B, Maurette P, Castel JP, et al. The epidemiology of head trauma in Aquitaine (France), 1986: a community-based study of hospital admissions and deaths. Int J Epidemiol 1990; 19: 133-40.
5. Signorini DF, Andrews PJ, Jones PA, Wardlaw JM, Miller JD. Predicting survival using simple clinical variables: a case study in traumatic brain injury. J Neurol Neurosurg Psychiatry 1999; 66: 20-5.
6. Rovlias A, Kotsou S. Classification and regression tree for prediction of outcome after severe head injury using simple clinical and laboratory variables. J Neurotrauma 2004; 21: 886-93.
7. Fearnside MR, Cook RJ, McDougall P, McNeil RJ. The Westmead Head Injury Project outcome in severe head injury. A comparative analysis of pre-hospital, clinical and CT variables. Br J Neurosurg 1993; 7: 267-79.
8. Andrews PJ, Sleeman DH, Statham PF, McQuatt A, Corruble V, Jones PA, et al. Predicting recovery in patients suffering from traumatic brain injury by using admission variables and physiological data: a comparison between decision tree analysis and logistic regression. J Neurosurg 2002; 97: 326-36.
9. Day RJ, Vinen J, Hewitt-Falls E. Major trauma outcomes in the elderly. Med J Aust 1994; 160: 675-8.
10. Richmond TS, Kauder D, Strumpf N, Meredith T. Characteristics and outcomes of serious traumatic injury in older adults. J Am Geriatr Soc 2002; 50: 215-22.
11. Hukkelhoven CW, Steyerberg EW, Rampen AJ, Farace E, Habbema JD, Marshall LF, et al. Patient age and outcome following severe traumatic brain injury: an analysis of 5600 patients. J Neurosurg 2003; 99: 666-73.
12. Luerssen TG, Klauber MR, Marshall LF. Outcome from head injury related to patient's age. A longitudinal prospective study of adult and pediatric head injury. J Neurosurg 1988; 68: 409-16.
13. Braakman R, Gelpke GJ, Habbema JD, Maas AI, Minderhoud JM. Systematic selection of

prognostic features in patients with severe head injury. Neurosurgery 1980; 6: 362-70.
14. Boto GR, Gomez PA, De La CJ, Lobato RD. Severe head injury and the risk of early death. J Neurol Neurosurg Psychiatry 2006; 77: 1054-9.
15. Treno AJ, Gruenewald PJ, Ponicki WR. The contribution of drinking patterns to the relative risk of injury in six communities: a self-report based probability approach. J Stud Alcohol 1997; 58: 372-81.
16. Rutledge R, Messick WJ. The association of trauma death and alcohol use in a rural state. J Trauma 1992; 33: 737-42.
17. Ghajar J. Traumatic brain injury. Lancet 2000; 356: 923-9.
18. Signorini DF, Andrews PJ, Jones PA, Wardlaw JM, Miller JD. Adding insult to injury: the prognostic value of early secondary insults for survival after traumatic brain injury. J Neurol Neurosurg Psychiatry 1999; 66: 26-31.
19. 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.
20. Chi JH, Knudson MM, Vassar MJ, McCarthy MC, Shapiro MB, Mallet S, et al. Prehospital hypoxia affects outcome in patients with traumatic brain injury: a prospective multicenter study. J Trauma 2006; 61: 1134-41.
21. Rose J, Valtonen S, Jennett B. Avoidable factors contributing to death after head injury. Br Med J 1977; 2: 615-8.
22. Ritter AM, Muizelaar JP, Barnes T, Choi S, Fatouros P, Ward J, et al. Brain stem blood flow, pupillary response, and outcome in patients with severe head injuries. Neurosurgery 1999; 44: 941-8.
23. Davis DP, Serrano JA, Vilke GM, Sise MJ, Kennedy F, Eastman AB, et al. The predictive value of field versus arrival Glasgow Coma Scale score and TRISS calculations in moderate-to-severe traumatic brain injury. J Trauma 2006; 60: 985-90.
24. Thornhill S, Teasdale GM, Murray GD, McEwen J, Roy CW, Penny KI. Disability in young people and adults one year after head injury: prospective cohort study. BMJ 2000; 320: 1631-5.
25. Jennett B, Teasdale G, Galbraith S, Pickard J, Grant H, Braakman R, et al. Severe head injuries in three countries. J Neurol Neurosurg Psychiatry 1977; 40: 291-8.
26. Moppett IK. Traumatic brain injury: assessment, resuscitation and early management. Br J Anaesth 2007; 99: 18-31.
27. Marshall LF, Marshall SB, Klauber MR, Van Berkum CM, Eisenberg H, Jane JA, et al. The diagnosis of head injury requires a classification based on computed axial tomography. J Neurotrauma 1992; 9 Suppl 1: S287-S292.
28. Marshall LF, Toole BM, Bowers SA. The National Traumatic Coma Data Bank. Part 2: Patients who talk and deteriorate: implications for treatment. J Neurosurg 1983; 59: 285-8.
29. Tabaddor K, Danziger A, Wisoff HS. Estimation of intracranial pressure by CT scan in closed head trauma. Surg Neurol 1982; 18: 212-5.
30. West JG, Trunkey DD, Lim RC. Systems of trauma care. A study of two counties. Arch Surg 1979; 114: 455-60.
31. Llullaku SS, Hyseni NS, Bytyci CI, Rexhepi SK. Evaluation of trauma care using TRISS method: the role of adjusted misclassification rate and adjusted w-statistic. World J Emerg Surg 2009; 4: 2.

ลักษณะเสี่ยงต่อการเสียชีวิตในผู้บาดเจ็บศีรษะที่มีโอกาสรอดชีวิตมากกว่าครึ่งในโรงพยาบาลสวรรค์ประชารักษ์

สุชาติ วิภาสกรวราวุธ, พรจันทร์ สุวรรณมนตรี, ศิริลักษณ์ โถแก้ว

***ภูมิหลัง**: การบาดเจ็บจากสาเหตุต่าง ๆ มีแนวโน้มเพิ่มขึ้นทั่วโลก การศึกษานี้จึงมีวัตถุประสงค์เพื่อหาลักษณะเสี่ยงต่อการเสียชีวิตของผู้บาดเจ็บศีรษะที่มีโอกาสรอดชีวิตมากกว่าครึ่ง*

***การออกแบบ**: การศึกษาวิเคราะห์แบบย้อนหลังเปรียบเทียบโดยสุ่มตัวอย่างกลุ่มควบคุม*

***วัสดุและวิธีการ**: กลุ่มศึกษาเป็นผู้ที่เสียชีวิตเนื่องจากบาดเจ็บศีรษะที่มีค่า TRISS-PS มากกว่า 0.5 ในระหว่างวันที่ 1 มกราคม พ.ศ. 2550 ถึง วันที่ 31 ธันวาคม พ.ศ. 2550 กลุ่มควบคุมเป็นผู้รอดชีวิตที่บาดเจ็บศรีษะที่มีค่า TRISS-PS มากกว่า 0.5 และได้รับการสุ่มโดยการจับฉลากผู้ที่รอดชีวิตที่นอนโรงพยาบาลวันเดียวกันในอัตราส่วน สองต่อหนึ่ง ผู้บาดเจ็บที่อายุต่ำกว่า 15 ปี และผู้ที่เสียชีวิตก่อนถึงห้องฉุกเฉิน ไม่รวมอยู่ในการศึกษาครั้งนี้ รวบรวมข้อมูลทั่วไป ข้อมูลทางคลินิก ผลการรักษา วิเคราะห์ข้อมูลด้วยสถิติ odds ratio และวิธีถดถอยพหุแบบ logistic ในกรณีศึกษาหลายปัจจัยร่วมกัน กำหนดค่าช่วงความเชื่อมั่น (confidence interval, CI) ร้อยละ 95*

***ผลการศึกษา**: กลุ่มศึกษา 111 ราย กลุ่มควบคุม 222 ราย ลักษณะทั่วไปที่ไม่แตกต่าง ได้แก่ เพศ ประวัติ โรคประจำตัว กลไกการบาดเจ็บ สาเหตุการบาดเจ็บและพาหนะของผู้ป่วย จากการวิเคราะห์ถดถอยพหุเปรียบเทียบระหว่างกลุ่มเสียชีวิตกับกลุ่มรอดชีวิต พบว่าอายุมากกว่าหรือเท่ากับ 45 ปี มีความเสี่ยง 1.6 เท่า (95% CI = 1.1-2.3) ประวัติการดื่มแอลกอฮอล์เสี่ยง 3.1 เท่า (95% CI = 1.4-6.9) GCS แรกรับหอผู้ป่วยที่อยู่ระหว่าง 3-8 เสี่ยง 4.2 เท่า (95% CI = 2.4-7.2) ระดับความรุนแรงการบาดเจ็บศีรษะ (AIS) ที่มากกว่าหรือเท่ากับ 4 เสี่ยง 3.3 เท่า (95% CI = 1.3-8.3) ปฏิกิริยาตอบสนองของกล้ามเนื้อแขนขาแรกรับห้องฉุกเฉินที่น้อยกว่าหรือเท่ากับ 4 เสี่ยง 1.5 เท่า (95% CI = 1.0-2.0) และ SBP หอผู้ป่วยน้อยกว่า 90 มิลลเมตรปรอท เสี่ยง 13.3 เท่า (95% CI = 3.2-56.2)*

***สรุป**: ผู้เสียชีวิตที่ได้รับบาดเจ็บศีรษะที่มีโอกาสรอดชีวิตมากกว่าครึ่งควรได้รับการทบทวนวิเคราะห์เพื่อค้นหาข้อผิดพลาดที่อาจเป็นสาเหตุของการเสียชีวิต โดยเฉพาะอย่างยิ่งในผู้ป่วยที่ไม่มีลักษณะเสี่ยงทั้ง 6 ข้อ ดังกล่าว*