Predictors of Mortality among Head Trauma Patients Reaching the ICU Alive

MONGY H. BENIAMEN, M.D. 1; NORA I. ABBAS, M.D. 2; KARIM S. MASHHOUR, M.D. 2 and TAREK S. EL-GOHARY, M.D. 2

The Departments of Critical Care, Al-Helal Hospital 1 and Critical Care, Faculty of Medicine, Cairo University 2, Egypt

Abstract

Background: Traumatic brain injury (TBI) is a leading cause of morbidity and mortality worldwide. Ninety percent occurs in low and middle income countries.

Aim of Work: To construct a predictive model for mortality in head injury patients on the basis of easily available parameters.

Methods: Prospective randomized study that included 100 head trauma patients admitted to the ICU in a period of 15 months. Demographic data, diabetes, hypertension and cardiac history were recorded. Admission blood samples were obtained for CBC, coagulation profile, kidney and liver function tests, lactate level and random glucose level. Receiver operating curve (ROC) analysis including the area under the ROC and multivariable logistic regression were used to identify independent mortality predictors of admission parameters to create a prognostic model.

Results: A total of 58 patients died (58%) out of 100 patients included in this study. Multivariate analysis revealed that age >75 years (HR=25.49, 95% CI=2.36 to 275.66), GCS <9 (HR=4.18, 95% CI=1.58 to 11.08), serum creatinine higher than 1.5 mg/ml (HR= 8.75 (95% CI=3.10 to 24.71), PaCO 2 >45 mmHg (HR=14.86 (95% CI=4.85 to 45.49) and history of cardiac disease (HR=0.37, 95% CI=0.15 to 0.91) were associated with high mortality rate.

Conclusion: Age >75 years, GCS <9, PaCO 2 >45 mmHg, serum creatinine exceeding 1.5 mg/ml and cardiac diseases are independent significant predictors of mortality in head trauma patients.

Key Words: Glasgow coma score (GCS) – Renal failure – Cardiac diseases – Hypercapnia – Mortality – Prognosis – Traumatic brain injury (TBI).

Introduction

TRAUMATIC brain injury (TBI) is a leading cause of morbidity and mortality worldwide. Annually, about 1.5 million affected people die while several millions need emergency management [1]. Unfortunately 90% of the burden occurs in low and middle income countries [2]. Head injuries cause immediate death in 25% of acute traumatic injuries [3].

A survey reported that 80% of doctors believed that an accurate assessment of prognosis is important for specific management decisions [4]. Prognostic models are statistical models that combine patients’ data to predict outcome and are likely to be more accurate than simple clinical predictions [5].

Many prognostic scores have been reported but none are widely used. Possible causes may include the small samples, the defective methodology, and that only few prognostic scores were validated in external populations and presented in a clinically practical way. They did not originate from low and middle income countries, where most trauma and related deaths occur [6]. However, the Medical Research Council (MRC) CRASH (corticosteroid randomization after significant head injury) trial which is the largest clinical trial conducted in TBI patients and presents a unique opportunity to develop a prognostic model [7] with high recruitment of patients from low and middle income countries [8].

The traumatized patient outcome is multifactorial through combination of the clinical diagnosis and presence and severity of comorbidities in addition to the amount of monitoring and therapy received [9]. Although decision-making of the traumatized patient is mostly clinically based, it can be fortified by information given by the scoring systems. Decision-making cannot depend on a numerical scale only as the ideal prognostic scale
for the traumatized patient is still far from being reported [10].

This study was designed to determine the various predictors which can affect the outcome of head trauma patients admitted to the intensive care unit (ICU). It aimed at developing and validating a practical prognostic model.

**Patients and Methods**

Our prospective cohort study was conducted in the I.C.U of El-Helal Hospital in Cairo, Egypt from November 2012 to February 2013. The study included all head trauma patients, older than 16 years, admitted to I.C.U from Emergency room. All patients had a Glasgow coma scale of 14 or less, with a positive brain computerized tomography (C.T.) findings, were eligible for inclusion in this study. These patients included isolated head trauma or head trauma associated with major extra-cranial injuries (MEI).

All patients were subjected to full history taking, clinical and radiological examination and laboratory investigations. All cases were evaluated on admission according to Glasgow Coma Score (GCS), the Acute Physiological and Chronic Health Evaluation (APACHE II) scoring system, the Revised Trauma Score (RTS) and the Abbreviated injury score (AIS).

After collecting all data of 100 patients, statistical analysis was done on a computer using Stata© version 11.2 (StataCorp LP, College Station, TX) and IBM© SPSS© Statistics version 21 (IBM© Corp., Armonk, NY).

Data presentation: The D’Agostino-Pearson test was used to test the normality of numerical data distribution. Non-normally distributed numerical data are presented as median and interquartile range. The intergroup differences are compared non-parametrically using the Mann-Whitney U test. Categorical data are presented as number and percentage. Nominal and ordinal data are compared using the Pearson chi square test or the chi square test for trends, respectively.

Multivariable survival analysis: Cox proportional hazard regression was used to determine independent predictors of non-survival by a given time. Modeling the Cox proportional hazards regression model was carried out using a purposeful approach for covariate selection as described by Hosmer et al. [11]. $p$-values are two-tailed. $p<0.05$ was considered statistically significant.

**Results**

Hundred patients were included in this study. There were 92 Males and 8 females. Their age ranged between 17-81 years; mean 39 (± 17.4) and median of 35.5 and IQR (23-52). There were 58 (58%) survivals and 42(42%) non survivals.

The age of the survivals ranged from 17-81 years IQR 25 to 54 with a median age 46 years while the age of non-survivals ranged from 17-78 years IQR 22 to 48 with a median age of 30.5 years. In the non-survival group, there was 90.5% male and 9.5% female. There were significantly more patients in the non-survival group with history of diabetes (DM), hypertension, cardiac comorbidities and drug abuse (Table 1).

There was statistically significant difference between the two groups regarding the injury to hospital time ($p<0.001$). It was more than an hour in 8.6% survivors compared to 0.0% in non-survivors, one to three hours in 67.2% survivors compared to14.3% in non-survivors and more than three hours in 24.1% survivors compared to 85.7% in non-survivors.

<table>
<thead>
<tr>
<th></th>
<th>Survivors</th>
<th>Non survivors</th>
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<tbody>
<tr>
<td>&gt;1 hour</td>
<td>8.6%</td>
<td>0.0%</td>
</tr>
<tr>
<td>1-3 hour</td>
<td>67.2%</td>
<td>14.3%</td>
</tr>
<tr>
<td>&gt;3 hours</td>
<td>24.1%</td>
<td>85.7%</td>
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<tr>
<td>$p$-value</td>
<td>&lt;0.001</td>
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Mechanism of injury ($p=0.015$) was road traffic accident (RTA) in 74.1% of survivors compared to 78.6% of non-survivors, fall from a height in 10.3% of survivors compared to 11.9% of non-survivors, gunshot in 0% in survivors compared to 7.1% of non-survivors, blunt trauma in 13.8% of survivors compared to 0.0% of non-survivors and others modes in 1.7% of survivors compared to 2.4% of non-survivors.

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<thead>
<tr>
<th></th>
<th>Survivors</th>
<th>Non survivors</th>
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<tbody>
<tr>
<td>RTA</td>
<td>74.1%</td>
<td>78.6%</td>
</tr>
<tr>
<td>Fall from a height</td>
<td>10.3%</td>
<td>11.9%</td>
</tr>
<tr>
<td>Gunshot</td>
<td>0%</td>
<td>7.1%</td>
</tr>
<tr>
<td>Blunt trauma</td>
<td>13.8%</td>
<td>0.0%</td>
</tr>
<tr>
<td>Other modes</td>
<td>1.7%</td>
<td>2.4%</td>
</tr>
<tr>
<td>$p$-value</td>
<td>0.015</td>
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</table>
More patients in the non-survivors group had lower SBP (p<0.001) (35.7% in non-survivors vs 0% in survivors), lower DBP (p<0.001) (45.2% vs 12.1%), higher pulse rate (p<0.001) (61.9% vs 13.8%) and higher respiratory rate (78.6% vs 48.3%) (p<0.001).

On presentation, high body temperature, abnormal pupillary reaction and convulsions were significantly more common in the non-survivor group. Both pupils were non-reactive in 66.7% in non survivors compared to 0.0% of survivals. One pupil was reactive in 31% in non survivors compared to 1.7% in survivors (Table 2). More patients in the non-survivor group had acidosis, hypercapnia, hypoxemia, high serum lactate, hypokalemia and high RBS (>200mg%). Regards hematologic variables, significantly more patients in the non-survivor group had low hemoglobin <10g/dl (p-values=0.002), Hematocrit, high leukocytic count, low platelet count and high INR (Table 2).

CT findings including (Obliteration of 3rd ventricle or basal cistern, subarachnoid hemorrhage and midline shift), (p<0.001) were statistically more in non-survivors compared to survivors.

Regarding the scoring systems, the median AIS (p<0.001) was significantly higher in non-survivors than survivors; (6 in non-survivors versus vs 3.5 in survivors) and the median APACHE II score (p<0.001) was significantly higher in non-survivors vs survivors (25 in non-survivors vs 13.5 in survivors). On the other hand, the median RTS (p<0.001) was significantly higher in survivors than non-survivors (4 in survivors compared with 1 in non-survivors) and the median GCS (p<0.001) was significantly higher in survivors than non-survivors (13 in survivors compared with 4.5 in non-survivors).

All five variables included in the final model were independent predictors of survival. Age >75 years was associated with the highest hazard ratio (HR=25.49, 95% CI=2.36 to 275.66, p=0.008). GCS <9 was associated with a hazard ratio of 4.18 (95% CI=1.58 to 11.08, p=0.004). PaCO₂ >45 mmHg was associated with a hazard ratio of 14.86 (95% CI=4.85 to 45.49, p<0.001). Serum creatinine exceeding 1.5mg/ml was associated with a hazard ratio of 8.75 (95% CI=3.10 to 24.71, p<0.001). History of cardiac disease was associated with a reduced risk for survival after adjustment for other covariates (age, GCS, PaCO₂, and serum creatinine) (hazard ratio=0.37, 95% CI=0.15 to 0.91). This decrease was statistically significant (p=0.003). The full model had a log likelihood statistic of ~78.45 which was statistically significant compared with the null model (p<0.0001) denoting adequate model fit.

Table (3): Different factors and their relation with survivor and non survivors.

<table>
<thead>
<tr>
<th></th>
<th>Survivors</th>
<th>Non survivors</th>
<th>p-value</th>
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<tbody>
<tr>
<td>Diabetes</td>
<td>8.6%</td>
<td>42.9%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>12.1%</td>
<td>33.3%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cardiac comorbidities</td>
<td>6.9%</td>
<td>38.1%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Drug abuse</td>
<td>3.4%</td>
<td>40.5%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>High body temperature</td>
<td>10.3%</td>
<td>35.7%</td>
<td>&lt;0.001</td>
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Table (4): Different factor affecting survivor at time of presentation.

<table>
<thead>
<tr>
<th></th>
<th>Survivors</th>
<th>Non survivors</th>
<th>p-value</th>
</tr>
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<tbody>
<tr>
<td>High body temperature</td>
<td>10.3%</td>
<td>35.7%</td>
<td>0.002</td>
</tr>
<tr>
<td>Abnormal papillary reaction</td>
<td>1.7%</td>
<td>97.7%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Both pupils non-reactive</td>
<td>0%</td>
<td>66.7%</td>
<td></td>
</tr>
<tr>
<td>One pupil non-reactive</td>
<td>1.7%</td>
<td>31%</td>
<td></td>
</tr>
<tr>
<td>Convulsions</td>
<td>0%</td>
<td>52.4%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Acidosis (PH &lt;7.35)</td>
<td>1.7%</td>
<td>85.7%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypercapnia (PaCO₂ &gt;45mmHg)</td>
<td>0%</td>
<td>85.7%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypoxia (PaO₂ &lt;60mmHg)</td>
<td>1.7%</td>
<td>78.6%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>High serum lactate (&gt;2mmol/L)</td>
<td>0%</td>
<td>92.9%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypokalemia (K &lt;3.5mmol/L)</td>
<td>20.7%</td>
<td>40.5%</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>High RBS (&gt;200mg%)</td>
<td>8.6%</td>
<td>54.8%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Low hemoglobin (&lt;1 g/dl)</td>
<td>12.1%</td>
<td>38.1%</td>
<td>0.002</td>
</tr>
<tr>
<td>Low hematocrit (&lt;30%)</td>
<td>10.3%</td>
<td>35.7%</td>
<td>0.002</td>
</tr>
<tr>
<td>High leukocytic count (&gt;1 1000/mms)</td>
<td>70.7%</td>
<td>90.5%</td>
<td>0.017</td>
</tr>
<tr>
<td>Low platelet count (&lt;1 50,000/mm³)</td>
<td>0%</td>
<td>31%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>High INR (&gt;1.5)</td>
<td>1.7%</td>
<td>42.9%</td>
<td>&lt;0.001</td>
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</table>
Table (5): Final model produced from previously developed model with transformation of continuous covariates back to categorical variables. All five variables included in the final model were independent predictors of survival.

| Covariate                      | B    | SE  | Hazard ratio | SE  | Lower limit | Upper limit | Z    | P>|z|
|-------------------------------|------|-----|--------------|-----|-------------|-------------|------|-----|
| Age > 75 yr.                  | 3.24 | 1.21| 25.49        | 30.97| 2.36        | 275.66      | 2.67 | 0.008|
| GCS < 9                       | 1.43 | 0.50| 4.18         | 2.08 | 1.58        | 11.08       | 2.88 | 0.004|
| PaCO₂ > 45 mmHg               | 2.70 | 0.57| 14.86        | 8.48 | 4.85        | 45.49       | 4.73 | <0.001|
| Serum creatinine > 1.5 mg/ml  | -0.99| 0.46| 0.37         | 0.17 | 0.15        | 0.91        | -2.17| 0.030|
| Cardiac disease               | -78.45|    |              |     |             |             |      |      |
| Log likelihood                | 79.66|     |              |     |             |             |      |      |
| LR chi square                 | <0.0001|    |              |     |             |             |      |      |
| p>|chi²                        | 0.972|     |              |     |             |             |      |      |
| Area under ROC curve (AUC)    |      |     |              |     |             |             |      |      |
| SE                            | 0.014|     |              |     |             |             |      |      |
| 95% CI                        | 0.945 to 0.998|  |              |     |             |             |      |      |
| p                            | <0.001|     |              |     |             |             |      |      |

B = Regression coefficient. SE = Standard error. 95% CI = 95% confidence interval. Z = z statistic.

**Discussion**

Many variables were studied, but only five of variables were found to be independent predictors of non-survival. These factors are age more than 75, GCS less than 9, PaCO₂ >45mmHg, Serum creatinine exceeding 1.5mg/ml and Cardiac diseases. They were included in the final model.

Age >75 years was associated with the highest hazard ratio (HR=25.49, 95% CI=2.36 to 275.66, \(p=0.008\)). This is because elderly TBI patients usually had a pre-injury medical comorbidity compared with younger adults. This significant increase in comorbidity may be important in exaggerating primary and secondary brain insults added to the less brain physiologic reserve compared to younger TBI patients. The brain trauma foundation [12] published that the chances of survival in patients with intracranial hematomas decrease with advancing age [13-21]. Several authors have identified age as a strong prognostic indicator following injury to the brain [22-30]. Our study is in agreement with (Frankel et al., 2006) [31] who found that adults aged 75 years and older have the highest rates of TBI-related death in sample size (80,000 cases).

Coronado, McGuire et al., 2012 [32] also reported that adults aged 75 years and older have the highest rates of TBI-related hospitalization and death. (Langlois et al., 2004) [33] reported that TBI mortality rate was highest among persons aged 7–75 years, especially those aged 85 years (\(p=0.03\)). Another study performed by Bouras et al., 2007 [34] found that mortality was higher in the elderly in TBI severity subgroups. Young subjects with a GCS <8 tended to benefit from ICU treatment whereas patients 75 and over did not, regardless of their severity of injury. Patients 75 and older were significantly less likely to benefit or survive surgical intervention than younger patients. Regarding patients aged 65-74 years, they may benefit from ICU treatment or surgical intervention. Another study performed by (Martin et al., 2010) [38] reported that differences in the physiologic response to injury and high-risk mechanisms in older adults might partly explain under triage rates in this age group. In patients aged >65 years, occult hypotension (i.e. hypoperfusion that is not evident by standard vital sign criteria) was present in 42% of patients with "normal" vital signs. Spaniolas et al., 2010 [36] reported that older adults might be severely injured in low-energy events (e.g., ground-level falls) that accounted for (34.6%) of all deaths in patients aged >65 years, higher rates of intracranial injury and in-hospital mortality among adults aged >75 years.

The expert Panel strengthens the recommendations regarding older adults. "SBP <110 might represent shock after age 65" and "low-impact mechanisms might result in severe injury" were added under "Older Adults" because under triage of the older adult population is a substantial problem [18,37]. The physiologic parameters used in younger patients might not apply to older adults, occult injury is likely to be greater among older adults and therefore field recognition of serious injury among older adults must be more anticipatory.

GCS is the most widely used system to predict outcome after TBI. TBI patients with GCS <9 have the worst prognosis. About 25-30% of these patients have good long-term outcomes, 17 % have moderate to severe disabilities, and 30 % die [38]. In this
study, GCS <9 was associated with a hazard ratio of 4.18 (95% CI=1.58 to 11.08, p=0.004) and GCC score <9 was independent predictors of mortality.

Randall et al. [12] reported that GCS score has been shown to have a significant correlation with outcome following severe TBI, both as the sum score [39-45] or as just the motor component [40,46-48]. Udekwu et al., [49] reported a good correlation exists between GCS score and FIM (functional independence measure), as determined by rank correlation coefficients, whereas mortality falls steeply between a GCS score of 3-7 followed by a shallow fall.

Narayan et al., [50] found a significant inverse correlation between initial GCS score (obtained 6-12 hours after head trauma) and mortality. Nakamura et al., 2006 reported [51] mortality for TBI patients admitted with GCS <8 after resuscitation may be as high as 50%.

Acute respiratory failure (type II) in TBI patients causes include upper airways obstruction , chest wall trauma, ruptured diaphragm, coma, raised ICP, head injury, opioid and sedative drugs and cerebral cord lesions.

In this study, PaCO2 >45 mmHg was associated with a hazard ratio of 14.86 (95% CI=4.85 to 45.49, p<0.001). It is an independent predictor of mortality. This goes with the result of Miller et al. [52] who found that hypercapnia commonly occur in multiple traumas including TBI. Hypercapnia increases cerebral blood volume and flow by cerebral vasodilatation. This can increase ICP significantly, in the situations of reduced intracranial compliance and adversely reduce cerebral perfusion. In addition, hypercapnia is a well-known cause of secondary insult and mortality [53-56].

Haussmann et al. [57] studied 1693 neurosurgical intubated patients and reported that renal failure worsened prognosis considerably by a mortality of 71-100%. In our study, Serum creatinine exceeding 1.5mg/ml was associated with a hazard ratio of 8.75 (95% CI=3.10 to 24.71, p<0.001) and was an independent predictor of non-survival. Matas AJ et al., found a higher mortality rate in TBI cases associated with renal failure. Moore EM, et al., 2010 [59] found TBI mortality was significantly higher in the renal failure group than control group. Admission levels of urea, creatinine, phosphate and bilirubin were significantly higher in TBI patients with renal failure.

Ala-Kokko, et al., [60] concluded that development of renal failure in TBI patients during the first 24 hours after ICU admission is an independent predictor for hospital death. Even minor insignificant decrease in renal function in severe TBI patients may be associated with worse outcome [61].

In the multivariable model (Table 1), history of cardiac disease was associated with decreased survival, after adjustment for other covariates (age, GCS, PaCO2, and serum creatinine) (hazard ratio =0.37, 95% CI=0.15 to 0.91). This decrease was statistically significant (p=0.003). This association can be attributed to that IHD is worsened by TBI stress which may precipitate MI, central effect of SAH that may cause dysrhythmia in addition anti-platelet and anticoagulant use in cardiac patients can increase the incidence of intra-cerebral hemorrhage. Blunt trauma to the chest in the case of associated extra-cranial injuries may induce arhythmia or aggravate already present one.

Ferraris et al., [62] concluded that pre-injury cardiac risk factors, especially pre-injury warfarin, beta-blocker, clopidogrel, and CHF, are independent multivariate predictors of mortality in significant TBI patients. Nowadays, cardiac patients are living longer, asked to be more physically active and therefore are more prone to falls or injuries.

Conclusion & Recommendations:

Five variables included in the final model (Age >75 years, GCS <9, PaCO2 >45mmHg, Serum creatinine exceeding 1.5mg/ml and Cardiac diseases comorbidity) were independent significant predictors of Non-survival.

The present prognostic model may offer a good potential in clinical practice, research, and policy making, as well as for assessment of the Quality of health-care delivery. Continued development, refinement, and validation are advocated, together with assessment of the clinical impact of prediction models.

Statement about Protection of Human Subjects in Research

We certify that this study involving human subjects is in accordance with the Helsinki declaration of 1975 as revised in 2000 and that it has been approved by the relevant institutional Ethical Committee.

Conflict of interests:

All authors certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers’ bu-
reaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements, or non-financial interest (such as personal or professional relationships, affiliations or beliefs).

References


الملخص العربي

تهدف هذه الدراسة إلى تحديد نماذج تتبع بناء على مجمعات القياس (السيرة الذاتية، والاشتراكات، والخريطة أو مختبرية) التي يمكن أن تؤثر على نتائج المرء في باعات الموتى. أُجريت دراسة مستقلة على وحدة قاعدة المريض على قيد الحياة. تمت تدريج الدفاع والتحقق من صحة نمادج النتيجة العملية. تعتمد نماذج على الإصابات في الدماغ الخفيفة، معتدلة، وشاملة، حيث أن صحة ذلك التقدير في وقت مبكر من تشخيص حالة مرضي. أصابات الرأس هو عامل مهم في اتخاذ قرارات العلاج. ونشق المرضي، أو التواصل مع الأسرة.

من أجل تحسين نتائج العلاج، تقدمت الدراسة في وحدة القاعدة المركزة وتقليل الأمراض والمخاطر. وتغطة دراسة من مريض شرفي يبلغ من العمر سنة عشرة عاماً عا. مع استخدام متعددة على الدماغ إيجابية ومؤشر جلوكو nhiễm من أربعة عشرين المشاكل في دراسة مستقلة، وتشريحة السيرة الذاتية الكامل على البيانات في دراسة. الرقابة على التقدم، والتحق بالنتيجة كنماذج المريض والفحوصات المختبرية، وتحويل المعلومات الإعدادية والفحوصات المختبرية، وتحويل المعلومات الإعدادية والفحوصات المختبرية.

فأظهرت الدراسات ما يلي:

- نسبة أكبر بكثير من المرضى الذين يعانون من تاريخ طبي لأمراض السكري، ارتفاع ضغط الدم، أمراض القلب، وتعاطي المخدرات (الكحول)
- وجود بين الناجين بالمقارنة مع الناجين
- كان هناك تفقذ ذات لائحة إحصائية بين المجموعتين (الناجون وغير الناجين) فيما يتعلق بالوقت المحتسب قبل الوضاع إلى المستشفى والأنجاء.
- عدد مرضى أكبر في المجموعة غير الناجين عناو من انخفاض ضغط الدم الانبساطي، وارتفاع معدل النبض، والندام النقل مقارنة مع مجموعته.
- عدد المصابين باختيار نوعية جزء الجسم، قلة تفاعل بؤرة دقات القلب، والتشنجات أعلى بكثير في مجموعة غير الناجين.
- هناك فرق ذات لائحة إحصائية بين المجموعتين (الناجون وغير الناجين) فيما يتعلق بالنتائج الإجمالية الممتازة.
- وفيما يتعلق بالتحقيقات السريرية والفحوصات الكيميائية، تصل إلى كيف كانت من المرضى المصابين، ارتفاع نسبة ثاني أوكسيد الكربون مع تفسير الكليت في غازات الدم، وارتفاع الكليت في الدم، نقص كهربائيات الدم وارتفاع نسبة السكر الشؤوياتي في الدم كان أعلى في المجموعة غير الناجين.
- وفيما يتعلق بالفحوصات الدموية، وتم نشر الظروف الناجية على مجموعات من غير الناجين. п

تنتهي الملاحظات السريرية، وتوصيات العمل بها باستخدام مراعاة اتفاق متغيرات:

*نسبة 95% CI = 2.36-275.66,*
*HGCI HR = 0.008 = 0.004 = 1.10.88 لي*
*4.18% CI = 1.58 95% CI = 45.49 (0.001 = 3.10 95% CI = 0.37 4.15 0.003)*
*95% CI = 8.75 7.86 ي*