Original Research Article

Coagulation profile tests as a predictor for adult trauma patients’ mortality

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ABSTRACT

Background: Coagulopathy is commonly observed in poly-traumatized patients and is a known contributor to trauma mortality. Although, the incidence of coagulopathy is strongly associated with the severity of the injury, coagulopathy itself exerts an independent factor on mortality.

Methods: This is a prospective, observational study on 100 trauma patients. All patients were evaluated using the modified shock index (MSI). Coagulation profile tests including platelet count, prothrombin time (PT), partial thromboplastin time (PTT), D-dimer and fibrinogen/fibrin degradation products (FDPs) were performed for all patients on admission and at 12 hours intervals. Statistically, a logistic regression analysis was performed of coagulation profile tests to determine the incidence of trauma induced coagulopathy (TIC) and its impact on 24 hours mortality. Correlation between clinical and laboratory status was done.

Results: There was a statistically significant difference between the dead and the survived patients in the coagulation profile tests and MSI. The best cut-off point of each parameter of coagulation profile tests (PLT count, PT, PTT, d-dimer, FDPs) and MSI was calculated using receiver operating characteristic curve and were <173 × 10⁹/l, >18.7 s, >31 s, >5 mg/l, >321.5 mg/l and 1.6 respectively. Trauma induced coagulopathy in our study was defined by more than 2 of the following: PLT <173 × 10⁹/l, PT >18.7 s, activated partial thromboplastin time (APTT) >31 s, D-dimer >5 mg/l and FDPs>321.5 mg/l with a p value 0.001 and associated with increased mortality.

Conclusions: The incidence of trauma induced coagulopathy early after trauma is high and its severity is related to the injury itself. It is independent predictor of mortality. TIC was developed with presence of more than 2 of the coagulopathy parameters.

Keywords: Coagulopathy, Trauma patients, platelets, D-Dimer, MSI

INTRODUCTION

It is a well-known fact that trauma is a universal phenomenon and a major cause of morbidity and mortality throughout the world. It is the disease of young and the leading cause of death in the first four decades of life. Coagulopathy is present immediately at admission in 25% of trauma patients and is associated with a 5-fold increase in mortality. Uncontrolled haemorrhage from coagulation dysfunction is one of the main potentially preventable causes of the mortality in both civilian and military settings. Trauma induced coagulopathy is caused by multiple factors, such as anaemia, heamodilution, hypothermia, acidosis, shock, and serious trauma itself which affects patient’s outcome due to critical bleeding requiring massive transfusion.
The reported incidence of trauma-associated coagulopathy ranges from 10 to 87.5%. The wide range of values reflects the lack of a standard definition for coagulopathy. Other factors contributing to the variability in incidence rates include differences in the patient populations evaluated, blood sampled at different time points, and the use of various coagulation assays. Moreover, studies investigating the same coagulation marker may use different cutoff values or sensitivity levels, thereby limiting generalizability. A meta-analysis of 22 studies found the overall incidence of trauma-associated coagulopathy to be 35.2%.

Standard laboratory tests used to measure hemostasis and bleeding risk in patients with trauma include the international normalized ratio (INR), prothrombin time (PT), activated partial thromboplastin time (APTT), and platelet counts (PLT). D-dimer and fibrinogen (FIB) levels may provide additional useful data; however, their use is not routine. PT and APTT were originally developed to measure the in vivo activity of specific coagulation factors; however, they are currently used to predict the bleeding risk in perioperative poly traumatized patients. The coagulation panel and PLT may also be used to predict the bleeding risk.

The aim of the study was to determine the incidence of trauma induced coagulopathy in poly trauma patients who attended emergency department (ED), Menoufia University Hospital and also to evaluate the prognostic value of coagulation tests in prediction of 24hrs mortality.

**METHODS**

One hundred consecutive poly-trauma patients who attended the emergency department (ED), Menoufia University Hospital between December 2017 and June 2019 were prospectively collected in this study. They were observed for the first 24 hours after the occurrence of trauma.

**Inclusion criteria**

All patients exposed to trauma and presented to our emergency department whom age is 18 years old or more were included in the study.

**Exclusion criteria**

Patients who arrived to our emergency department in a cardio pulmonary arrest, patient who died within the first six hours of arrival, those who had reference from another hospital after the first aid or any resuscitation, those who received blood transfusion, patients with isolated traumatic brain injury, patients with history of any bleeding or coagulation disorders, anticoagulant and haemostatic drug history and pregnant women were excluded.

The course of the study was authorized from the Ethical Committee of our institution.

All patients were subjected to the following such as: history taking including information about mode of trauma, complete full physical examination in the form of primary and secondary survey. The data of patients collected at the time of admission were age of patients, gender, heart rate, systolic blood pressure, diastolic blood pressure and modified shock index and level of consciousness.

Coagulation tests including platelet count, INR, PT, APTT, fibrinogen/fibrin degradation products (FDPs) and D-dimer were performed for all patients at admission and at 12 hours intervals for the first 24 hours of injury and assessed at the Menoufia University Hospital clinical chemistry laboratory using routine laboratory assays. Statistically, a logistic regression analysis was performed of coagulation profile tests to determine the incidence of trauma induced coagulopathy and its impact on 24 hours mortality. To obtain comparable odds ratios for the relationships, we rescaled each variable as follows PLT <173 × 10^9/L, PT>18.7 s, APTT >31s, D-dimer level <5 or >5 mg/L and FDPs >321.5 mg/l. In our study coagulopathy was defined as more than 2 of the following: PLT<173 × 10^9/L, PT>18.7 s, and APTT>31 s, d-dimer>5 mg/l, FDPs>321.5 mg/l.

Correlation between clinical and laboratory status of the included patients were done using coagulation profile tests and modified shock index (MSI).

MSI was calculated for each patient according to vital signs at the time of admission and at 6 hours intervals by this equation,

MSI=HR/MAP

Where, HR is heart rate, MAP is mean arterial blood pressure.

Furthermore, hemoglobin (Hb), hematocrit (HCT) and glucose levels were measured and recorded.

**Statistical analysis**

Data were statistically analyzed using Statistical Package of Social Science (SPSS). Quantitative data were expressed as a mean±standard deviation (SD) while qualitative data were expressed as frequency and percentages. Qualitative variables were compared using a chi-square test while Quantitative continuous data were compared using the Mann- Whitney test. The area under the receiver operating characteristic (ROC) curve for each scale was used to compare the accuracy of the studied models.

A p value less than 0.05 was considered statistically significant. A univariant analysis with non-linear
correlation (cubic spline functions) was used to evaluate the shape of the relationship between the continuous variables and outcome.

**RESULTS**

In this study, data of 100 adult trauma patients presented to ED, Menoufia University Hospital were evaluated. Out of these 100 patients 22 pts. (22%) died. The mean age of patients was (44.5±20.6) among the dead patients and (35.6±27.3) among the survived patients. The study included 82 male patients and 18 female patients between them 20 male patients and 2 female patients died with ratio 90.9% and 9.1% respectively. The most common modes of trauma associated with mortality were varied from road traffic accidents by 76% and falling from heights by 15% to others as violence and falling heavy object on the patient by 9% (Table 1).

Thirty-two of survived trauma patients was admitted ICU (32%), 28 patients were admitted in the ward (28%) and 18 patients underwent operations (18%) (Figure 1).

![Figure 1: Final destination of the patients over 24 hours.](image)

**Table 1: Socio-demographic data and mode of trauma affecting 24 hours mortality.**

<table>
<thead>
<tr>
<th>Age in years</th>
<th>No. of dead (%)</th>
<th>No. of survived (%)</th>
<th>U</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean±SD</td>
<td>44.5±20.6</td>
<td>35.6±27.3</td>
<td>1.4</td>
<td>0.149</td>
</tr>
<tr>
<td>Range</td>
<td>18-75</td>
<td>18-75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>20 (90.9)</td>
<td>62 (79.5)</td>
<td>1.5</td>
<td>0.218</td>
</tr>
<tr>
<td>Female</td>
<td>2 (9.1)</td>
<td>16 (20.5)</td>
<td>16.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Mode of trauma (n=100)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RTA (n=76)</td>
<td>16 (72.7)</td>
<td>60 (76.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FFH (n=15)</td>
<td>2 (9.1)</td>
<td>13 (16.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assault (n=4)</td>
<td>4 (18.2)</td>
<td>0 (0.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Falling heavy object (n=5)</td>
<td>0 (0.0)</td>
<td>5 (6.4)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The characteristics of the dead and the survived patients according to the coagulation profile tests including the PLT count, PT, APTT, INR, FDPs and MSI were statistically significant different between them on the admission time and after 12 hours up follow. The mean of the PLT count regarding the dead patients versus the survived ones was 174.2±1632.6 and 23.9±7.7 per liter respectively with p value 0.032 at the admission time and after 12 hours was 145.6×10^3±69.4 per liter respectively with a p value 0.117.

PT results for the dead patients compared to the survived ones was 23.9±9.07 and 16.7±4.7 seconds respectively with p value 0.001and after 12 hours was 22.9±7.2 and 16.7±3.6 seconds respectively with p value 0.008.

INR results of the dead patients versus the survived ones was 1.4±0.18 and 1.23±0.21 respectively with p value 0.001 and after 12 hours was 1.5±0.40 and 1.27±0.19 respectively with p value 0.003.

APTT results of the dead patients versus the survived ones was 40.5±9.7 and 23.0±7.9 seconds respectively with p value 0.001 and after 12 hours was 46.5±13.5 and 23.7±7.7 seconds respectively with p value 0.001.

FDPs for the dead patients versus the survived ones was 1632±1454 and 404.2±829 mg/l respectively with p value 0.001 and after 12 hours was 2023.7±1627.6 and 399.1±773.2 mg/l respectively with p value 0.001.

Finally, MSI for the dead patients compared to the survived ones was 2.8±1.06 and 1.11±0.32 respectively with p value 0.001 and after 12 hours was 0.042±0.17 and 1.11±0.32 respectively with p value 0.001 (Table 2).

There was a statistically significant difference between the dead and the survived patients regarding occurrence of trauma induced coagulopathy using the laboratory parameters of coagulopathy. Ninety percentage of the dead patient had developed trauma induced coagulopathy (TIC) with more than 2 laboratory parameters of coagulopathy and only 20.5% of the survived patients had developed TIC with more than 2 parameters with a p value 0.001 while 9.1% of the dead patient had developed TIC with only 2 laboratory parameters of coagulopathy and 26.9% of the survived patients had developed TIC with only 2 laboratory parameters of coagulopathy with a p value 0.142. We observed that all the dead patients developed coagulopathy with more than 2 laboratory parameters with a highly significant p value (Table 3).
The ROC curve was used and evaluated for each parameter of coagulation profile tests (PLT count, PT, APTT, D-dimer and FDPs) and MSI independently at the time of admission to assess the validity of these tests in prediction of 24 hours mortality associated with trauma induced coagulopathy. The best cutoff point for each parameter of coagulation profile tests (PLT count, PT, APTT, FDPs and D-dimer) and MSI were calculated using the ROC curve and were ROC curve (<173 × 10⁹/l, >18.7 s, >31 s, >321.5 mg/l and >5 mg/l and 1.6) respectively (Table 4) (Figure 2).

In our study, coagulopathy was defined as more than 2 of the following: PLT <173 × 10⁹/l, PT>18.7 s and APTT >31 s, D-dimer >5 mg/l and FDPs >321.5 mg/l). These results were statistically significant related to increased 24hrs mortality according to results we obtained from our ROC curve (Table 3).

Area under the ROC curve (AUC) was calculated for each parameter in the coagulation profile tests (PLT, PT, APTT, FDPs, D-dimer) and MSI and was ROC curve (0.650, 0.793, 0.913, 0.877, 0.99 and 0.914) respectively.

### Table 2: Comparison between the dead and the survived patients according to coagulation profile and in different times over 24 hours.

<table>
<thead>
<tr>
<th>Coagulation profile</th>
<th>Dead (n=22)</th>
<th>Survived (n=78)</th>
<th>U</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Platelets</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>174.2×10⁹/l±41.5, 100-248</td>
<td>218.3×10⁹/l±75.4, 91-383</td>
<td>2.1</td>
<td>0.032</td>
</tr>
<tr>
<td>Mean±SD, range</td>
<td>12 hours follow up</td>
<td>145.6×10⁹/l±59.5, 71.0-234</td>
<td>188.2×10⁹/l±69.4, 77-350</td>
<td>1.56</td>
</tr>
<tr>
<td><strong>PT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>23.9±9.07 s, 15.7 s-47.5 s</td>
<td>16.7±4.7 s, 12.0 s-35.2 s</td>
<td>4.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean±SD, range</td>
<td>12 hours follow up</td>
<td>22.9±7.2 s, 8.0 s-28.5 s</td>
<td>16.7±3.6 s, 12.1 s-25.0 s</td>
<td>2.64</td>
</tr>
<tr>
<td><strong>INR</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>1.4±0.18, 1.18-1.80</td>
<td>1.23±0.21, 1.0-1.8</td>
<td>4.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean±SD, range</td>
<td>12 hours follow up follow up</td>
<td>1.5±0.40, 0.65-1.9</td>
<td>1.27±0.19, 1.0-1.78</td>
<td>2.98</td>
</tr>
<tr>
<td><strong>APTT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>40.5±9.7 s, 20 s±55 s</td>
<td>23.0±8.9 s, 14.5 s-32 s</td>
<td>5.9</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean±SD, range</td>
<td>12 hours follow up</td>
<td>46.5±13.5 s, 17.5 s-60.0 s</td>
<td>23.7±7.7 s, 14.0 s-38.3 s</td>
<td>3.2</td>
</tr>
<tr>
<td><strong>FDPs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>1632±1454 mg/l, 100-5000 mg/l</td>
<td>404.2±829 mg/l, 20-3400 mg/l</td>
<td>5.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean±SD, range</td>
<td>12 hours follow up</td>
<td>2023.7±1627.6 mg/l, 600-4532 mg/l</td>
<td>399.1±773.2 mg/l, 33-3156 mg/l</td>
<td>3.6</td>
</tr>
<tr>
<td><strong>MSI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>2.8±1.06, 1.4-4.4</td>
<td>1.4±0.42, 0.65-2.48</td>
<td>5.9</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean±SD, range</td>
<td>12 hours follow up</td>
<td>0.04±0.17, 0.0-0.72</td>
<td>1.11±0.32, 0.0-2.12</td>
<td>6.3</td>
</tr>
</tbody>
</table>

### Table 3: Comparison between died and survived patients regarding occurrence of TIC.

<table>
<thead>
<tr>
<th>Laboratory* parameters indicating TIC</th>
<th>No. of Dead (%)</th>
<th>No. of Survived (%)</th>
<th>Z-test</th>
<th>P value</th>
<th>Chi-square</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No factor</td>
<td>0 (0.0)</td>
<td>17 (21.8)</td>
<td>2.08</td>
<td>0.037</td>
<td></td>
<td></td>
</tr>
<tr>
<td>One factor</td>
<td>0 (0.0)</td>
<td>24 (30.8)</td>
<td>2.7</td>
<td>0.007</td>
<td>37.56</td>
<td>0.001</td>
</tr>
<tr>
<td>2 factors</td>
<td>2 (9.1)</td>
<td>21 (26.9)</td>
<td>1.47</td>
<td>0.142</td>
<td></td>
<td></td>
</tr>
<tr>
<td>More than 2 factors</td>
<td>20 (90.0)</td>
<td>16 (20.5)</td>
<td>5.82</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*: Laboratory parameters indicating; TIC: (PLT <173 × 10⁹/l, PT>18.7, PTT>31, D-dimer >5, FDPs >321.5).
Statistically the value of AUC of 0.5 suggests no discrimination and that between 0.5-0.6 could be considered as a fair test in prediction of mortality while the value of AUC between 0.6-0.7 could be considered as an acceptable test. The value of AUC between 0.7-0.8 could be considered as a good test in prediction of mortality. The value of AUC between 0.8-0.9 could be considered as an excellent test in prediction of mortality. Our ROC curve results showed that the ROC curve for base line platelet count and 24 hrs mortality was 0.650 which could be considered as an acceptable test but with low values of sensitivity, specificity and accuracy which were 50%, 66.7% and 63% respectively, so it could be considered as an acceptable test in prediction of 24 hrs mortality associated with TIC (Table 4, Figure 2A). While the ROC curve for base line PT and 24 hrs mortality was 0.793 which could be considered as a good test in prediction of 24 hrs mortality associated with TIC with good values of sensitivity, specificity and accuracy which were 86.4%, 64% and 70% respectively, so it could be considered as a good prognostic test (Table 4, Figure 2B). Also the ROC curve for base line PTT and 24 hrs mortality was 0.913 which could be considered as an excellent test in prediction of 24 hrs mortality associated with TIC with very good values of sensitivity, specificity and accuracy which were 81.8%, 87% and 87% respectively, so it could be considered as an excellent prognostic test (Table 4, Figure 2C). Whereas the ROC curve for base line FDPs and 24 hrs mortality was 0.877 which could be considered as an excellent test in prediction of 24 hrs mortality associated with TIC with very good values of sensitivity, specificity and accuracy which were 86.4%, 83% and 85% respectively, so it could be considered as an excellent prognostic test (Table 4, Figure 2D). Finally the AUC for D-dimer and MSI were 0.99 and 0.914 respectively this makes them as an excellent tests in prediction of 24 hrs mortality associated with TIC. The sensitivity of D-dimer and MSI were 100% and 90.9% respectively while their specificity were 70% and 74.4% respectively and their accuracy were 74% and 78% respectively. From these results we can consider also the D-dimer and MSI as very reliable and excellent prognostic tests (Table 4).

We can use our investigated parameters to rule out or rule in the occurrence of the traumatic induced coagulopathy and mortality according to the sensitivity and specificity of each one. From these results we can consider the MSI, PT, APTT, D-dimer and FDPs as significant prognostic tests more than PLT count in detection of trauma induced coagulopathy and in prediction of 24 hours mortality. This can be explained by the higher values of the AUC, sensitivity, specificity and accuracy of the previous parameters except for the PLT count. Also we can use MSI as a strong predictor of mortality as it was of high sensitivity (Table 4, Figure 2).

Table 4: Validity of platelets, PT, PTT, D-dimer and FDPs in prediction 24 hours hospital mortality using the ROC curve.

<table>
<thead>
<tr>
<th>Best cut off</th>
<th>AUC</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
<th>Accuracy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelets</td>
<td>&lt;173 × 10^9/l</td>
<td>0.650</td>
<td>50.0</td>
<td>66.7</td>
<td>29.7</td>
<td>82.5</td>
</tr>
<tr>
<td>PT</td>
<td>&gt;18.7 s</td>
<td>0.793</td>
<td>86.4</td>
<td>64</td>
<td>40</td>
<td>94</td>
</tr>
<tr>
<td>APTT</td>
<td>&gt;31 s</td>
<td>0.913</td>
<td>81.8</td>
<td>87</td>
<td>64</td>
<td>94</td>
</tr>
<tr>
<td>D-dimer</td>
<td>&gt;5 mg/l</td>
<td>0.99</td>
<td>100</td>
<td>70</td>
<td>48</td>
<td>100</td>
</tr>
<tr>
<td>FDPs</td>
<td>&gt;321.5 mg/l</td>
<td>0.877</td>
<td>86.4</td>
<td>83</td>
<td>60</td>
<td>97</td>
</tr>
<tr>
<td>MSI</td>
<td>1.6</td>
<td>0.914</td>
<td>90.9</td>
<td>74.4</td>
<td>50.0</td>
<td>97</td>
</tr>
</tbody>
</table>

There was also a statistically significant correlation between the modified shock index and the coagulation profile tests (PLT count, PT, APTT, FDPs). The value of R (correlation coefficient) between PLT count and modified shock index (MSI) was 0.075 which a weak correlation with a non-significant p value 0.460 while there were more significant correlations between PT, INR, APTT, FDPs and MSI as the correlation coefficient.
(R) value were (0.617, 0.447, 0.684 and 0.615) respectively with highly significant p value for all these correlations as the p value was 0.001.

Table 5: Correlation between coagulation profile and MSI.

<table>
<thead>
<tr>
<th>Coagulation profile</th>
<th>Modified shock index</th>
<th>R*</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT</td>
<td></td>
<td>0.617</td>
<td>0.001</td>
</tr>
<tr>
<td>INR</td>
<td></td>
<td>0.447</td>
<td>0.001</td>
</tr>
<tr>
<td>PTT</td>
<td></td>
<td>0.684</td>
<td>0.001</td>
</tr>
<tr>
<td>PLT count</td>
<td></td>
<td>0.075</td>
<td>0.460</td>
</tr>
<tr>
<td>FDPs</td>
<td></td>
<td>0.615</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*R: correlation coefficient (0-1).

These direct relations indicate higher mortality and bad prognosis with higher values of MSI and abnormal coagulation profile as it correlated clinical and laboratory status of the patient. The study found that when MSI was 1.6 or more, the coagulation tests were abnormal which indicates occurrence of trauma induced coagulopathy and increased 24 hours mortality (Table 5).

DISCUSSION

This study examined the prognostic value of the initial coagulation tests with regard to 24 hours in-hospital mortality after trauma and developed a series of prognostic models to detect the occurrence of trauma induced coagulopathy and its impact on the mortality.

This is evident in our study that the mean age of the patients included was (44.5±20.6) and (35.6±27.3) between died and survived patients. In the present study, the majority of patients were males by 82% and females were only (18%).

These results were near to those reported by Yadav and Agarwal who found that most of the studied patients were men (88%) and female only (12%) with majority of patients are young population (20-50) years old. These results were also close to a study done by Albatanony et al who found that most patients were male (74%) and 26% were female with mean age of 37.9±5.9 years.

These results also near to those reported by Mazandarani et al, who found that, most of the studied patients were males (78.94%) and females only (21.05%), with mean age (37.09±14.60) years.

In the present study, road traffic accident was responsible for most of cases (76%). These results confirmed the report published by World Health Organization, 2016 stated that approximately 90% of the world’s fatalities occur on the roads in low and middle income countries. This result also is very near to those reported by Mazandarani et al who found that the most common cause of trauma was traffic accidents (78.7%).

Our result agrees with a study done by Albatanony et al, who found that the road traffic accident was responsible for most of the cases (71%).

Present study found that a statistically significant difference in the coagulation profile including (PLT count, PT, INR, APTT, FDPs) between dead and survived patients that the initial abnormal coagulation profile tests associated with occurrence of trauma induced coagulopathy and increased mortality.

This can be explained with that coagulopathy is present immediately at admission in 25% of trauma patients and is associated with a 5-fold increase in mortality.

As clinical standard plasma tests of prothrombin time (PT) and activated partial thromboplastin time (APTT) reflect the overall enzyme activities involved in the extrinsic and intrinsic pathways, it is normal to have abnormal coagulation profile values in died people who were developed trauma induced coagulopathy.

Previous studies have shown that the most consistent coagulation abnormality is PT. PT reflects the activation time of the extrinsic, or tissue factor, pathway based on the cascade model of hemostasis. Most previous investigations of trauma associated coagulopathy focused on PT or INR abnormalities. The international mission on prognosis and analysis of clinical trials (IMPACT) in traumatic brain injury (TBI) study found that PT prolongation on admission was present in 221 of 850 patients (26%) and was associated with a 64% increase in mortality risk. APTT reflects the activation time of the intrinsic, or contact activation, pathway and is particularly sensitive to deficiencies in coagulation factors IX, XI, and VIII. Although affected less often than the PT, APTT is more highly correlated with poor outcome and mortality than are other markers of coagulation.

Also, three epidemiological studies from America and Europe have analyzed trauma registry data to describe post trauma coagulopathy from injury event were agreed with our results as we had 22% of the study population died within the first 24 hours after trauma and associated with trauma induced coagulopathy. The first study, from a level I trauma center in Miami, Florida, showed that up to 25% of trauma patients on arrival at a hospital already have an abnormal coagulation profile. It further correlated this finding of early post trauma coagulopathy with a lower survival.

A similar analysis was performed in 2 further studies, one in England and the other in Germany. All 3 studies correlated initial or early coagulation profile with patient outcome. In Germany in 2001, Rixen et al reviewed 2069 patients and found that PT was statistically significantly associated with outcome in a multiple linear regression model controlling for other known prognostic factors. The third study, by Brohi et al published in 2003, showed very similar results, with the incidence of an abnormal...
first PT or APTT being independently associated with the outcome of death, even controlling for crystalloid or colloid administration. The results of our study revealed the best cutoff points of coagulation profile tests (PT, APTT, INR, PLT count, FDPs, D-dimer) and MSI at which coagulopathy associated after the injury occurred and which also related to high incidence of 24 hours mortality using ROC curve. The best cutoff point for each were (>18.7 s, >41 s, >1.36, <173×10^9/l, 321.5 mg/l, >5 mg/l, and 1.6) respectively.

Previous studies have shown that the coagulation abnormality were associated with PT >18 s and APTT >60 s which is closely related to our results regarding PT which was >18.7 s and not with APTT which was >31 s. Our results also agrees with a study done by Yuan et al, who founded that multivariate logistic regression analysis revealed that INR >1.25, and APTT >36 s were independently associated with in-hospital mortality. These variables can be readily obtained on admission to the ED and are consistent with prior studies of prognostic predictors. These values are very near to our results which were INR >1.36 and APTT >31 s.

A previous study showed results close to ours as it founded that the prevalence of acute traumatic coagulopathy is associated with the severity of the injury. The percentage of platelet count <100×10^9/l, international normalized ratio INR >1.25, the prothrombin time PT >14s, activated partial thromboplastin time APTT >36 s, D-dimer >5 mg/l and fibrinogen (FIB) <1.5 g/l was also closely related to the severity of the injury and higher mortality rates.

Thrombocytopenia on admission is a complication of trauma in fewer than 10% of cases. In a study done at 2010, 10.7% of patients had a PLT <100×10^9/l. Thus, coagulation tests may provide more useful information on mortality after poly traumatized patients included traumatic brain injury than do the standard admission variables. This result is not similar to our result where the PLT was <173×10^9/l associated with increased mortality.

In a study in 2016, the outcome of patients with high D-dimer/low fibrinogen was poorest among the severe trauma patients. Moreover, mortality was significantly higher in patients with high D-dimer levels than in those with low D-dimer levels among patients without fibrinogen deficiency on arrival at the ED.

This study showed there was a statistically significant difference between died and survived patients regarding occurrence of trauma induced coagulopathy with a p 0.001 using the laboratory parameters of coagulopathy that 90% of the dead patients developed coagulopathy with more than 2 parameters. This result agrees with a study done by Brohi et al, who couldn’t determine when he correlated high injury severity, shock, and increased mortality with the early coagulopathy of trauma. Whether the coagulopathy is secondary to hemorrhage resulting in loss of coagulation factors, tissue hypo-perfusion, or extensive tissue injury resulting in the release of tissue factors causing the coagulopathy.

Our results agree with a study done by Niles et al, whose results clearly demonstrates that coagulopathy, independent of demographics, injury severity, urgent surgery or blood and blood product transfusion, was strongly associated with early death post major trauma.

In a previous study done by Singh et al. observed that among traditionally used predictors SBP <90 mmHg had the maximum odds of mortality however, both cut-offs of MSI <0.7 and >1.3, had higher odds of mortality.

Our study also found a statistically significant correlation between coagulation profile tests (PLT count, PT, APTT, FDPs) and modified shock index with a highly significant p value for all parameters as the p value was 0.001. This relation indicates higher mortality and bad prognosis with higher values of MSI and abnormal coagulation profile as it correlated clinical and laboratory status of the patient. The study found that when MSI was 1.6 or more, the coagulation tests were abnormal with prolonged values which indicate occurrence of trauma induced coagulopathy and increased 24 hours mortality.

From another point of view, a previous study founded that standard coagulation tests are performed using plasma in the absence of blood cells (these are removed by centrifugation). Also, these tests are stopped upon formation of the first fibrin strands, when only 5% of the total thrombin has been generated. Moreover, these tests do not assess the quality/the strength of the clot. Hyper fibrinolysis is recognized as a potential contributor to mortality in trauma, and this aspect is not assessed by standard coagulation tests. Another study done by Johansson showed that recent data suggest that whole blood viscoelastic tests, such as thromboelastometry (ROTEM) or thrombelastography (TEG) portray trauma induced coagulopathy (TIC) more accurately and substantially faster than standard coagulation tests. So it is considered a significant limitation of our study that these new techniques used to assess trauma induced coagulopathy as (ROTEM) and (TEG) were not available in our hospital and cannot be compared with the standard coagulation profile tests in detection TIC and evaluation of mortality.

**CONCLUSION**

The incidence of trauma induced coagulopathy early after trauma is high and its severity is related to the injury itself. It is independent predictor of mortality even in the presence of other risk factors and our results confirmed this where 90% of the dead patients had developed trauma induced coagulopathy (TIC) with more than 2
parameters of coagulopathy and 9.1% of the dead patient had developed TIC with only 2 parameters. We can also consider the development of trauma induced coagulopathy as a leading cause of death in the severely poly traumatized patients and from our results we can suspect its occurrence in our patients with more than 2 of the following laboratory parameters of coagulopathy: PLT<173 × 10^9/L, PT>18.7 s, APTT>31 s, D-dimer>5 mg/l and FDPs >321.5 mg/l.

The incidence of in-hospital mortality due to trauma induced coagulopathy was about 22% of the study population. The early coagulopathy of trauma was rapidly diagnosed in the ED using standard coagulation profile tests including PLT count, PT, PTT, INR, FDPs, D-dimer which was of statistically significant difference between the dead and the survived patients and with good sensitivity and accuracy in detecting the TIC and also in predicting of 24 hours mortality.

The present study results also show that MSI, as a potential marker for predicting the mortality rate and is significantly better than HR, SBP and DBP alone as the optimal level at which MSI was found to be highly correlated with mortality was 1.6 and above. There was a statistically significant correlation between coagulation profile tests and modified shock index as it correlated the clinical and laboratory status of the patients and its impact on increased 24 hours mortality.

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