Evaluation serum level of Interleukin 10 with trauma intensity and patient's prognosis

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ABSTRACT
Trauma is among the most important health conditions in the world. It is also an important cause of death and inability particularly in the first four decades of the life of sufferers. Seemingly, it is necessary to prepare a quantitative quantifiable scale for assessing the intensity of trauma for the purpose of further investigations, careful assessment of patients, formulation of preventive programs, enhancement of quality, assessment of the findings of trauma centers and triage.

IL-10 was shown to protect endothelial function after an acute inflammatory stimulus by limiting increases in superoxide generation within the vascular wall. The aim of this study was to analyze the level of Interleukin 10(IL-10) in patients suffering from trauma and also to determine its relationship with short-term and long-term mortality. It was also aimed to study the relationship between trauma and laboratory parameters especially arterial blood gases (ABG) parameters. In a cross-sectional descriptive-analytic study of traumatic patients, the blood levels of IL-10 were measured in traumatic patients upon their admission. Next, the relationship of IL-10 levels with Glasgow Coma Scale (GCS), ABG results, and short-term and long-term mortality was examined. The mean serum level of IL-10 in live patients and the deceased samples was 8.02±1.41 pg/ml and 13.19±2.39 pg/ml, respectively. The serum level of IL-10 was significantly higher in dead patients than alive ones (P<0.001). There exists an important inverse linear relation between serum level of IL-10 and arterial blood Acid-base Scale (PH) and initial amount of GCS in patients, while no significant linear relation exists between serum levels of IL-10 with The Bicarbonate Radical (HCo2) and Carbon Dioxide Partial Pressure (PaC02) of arterial blood.

Introduction

Trauma is among the most important health conditions in the world. It is also an important cause of death and inability particularly in the first four decades of the life of sufferers (1). The most common cause of mortalities below the age of 24 years is brain injury, which impose billion Rials expenses on the Iranian healthcare system (1-3). In 2000, traffic accidents were the second most important causes of premature death and illness, with AIDS
being the first cause of death among men aging from 15 to 24 years.

Accident is a major health problem in the Iranian society while mortalities resulting from accidents are also the third causes of death in humans. In addition, accident is the first cause of death among people aging less than 45 years (4-6).

According to the reports by the Iranian forensics organization, annually more than 14000 death cases result from traffic accidents in Iran. Considering the significant effect of trauma on human and financial resources, study and investigation of trauma is undoubtedly one of the practical needs of modern human (7).

The rate of deaths caused by accident in Iran is at the critical level. Annually, the aforementioned mortalities are increased by 10-15%. Moreover, accident is the second most important cause of death in Iran and the first cause of death in the world (8). Hence, in order to reduce the rate of mortalities and disabilities caused by trauma, early interventions are considered substantial (9).

Some of these interventions include the use of some certain scales for measuring the intensity of damages, determining severity, and determining the condition of patients (static or dynamic). These interventions play a significant role in the selection of the treatment method and the reduction in mortalities (10).

Seemingly, it is necessary to prepare a quantitative quantifiable scale for assessing the intensity of trauma for the purpose of further investigations, careful assessment of patients, formulation of preventive programs, enhancement of quality, assessment of the findings of trauma centers and triage (11).

Since several years ago various scales have been developed for determining the intensity of trauma in injured patients. These scales are also applied to all traumatic patients in some countries.

These scales use anatomic criteria, physiological criteria and a combination of both to determine the intensity of lesions (12-13).

IL-10, which is produced by various inflammatory cells, especially macrophages, 13 is a major inhibitor of cytokine synthesis, suppress macrophage function, and inhibit the production of pro inflammatory cytokines (14-15).

IL-10 expression has been identified within human atherosclerotic plaques (16-17), with high levels of expression being associated with significantly decreased cell death and iNOS expression (17).

Experimentally, IL-10 was shown to protect endothelial function after an acute inflammatory stimulus by limiting increases in superoxide generation within the vascular wall (18). The aim of this study was to analyze the level of IL-10 in patients suffering from trauma and also to determine its relationship with short-term and long-term mortality. It was also aimed to study the relationship between trauma and laboratory parameters especially ABG.

Materials and Methods

In a cross-sectional descriptive-analytic study of traumatic patients, the blood levels of IL-10 were measured in traumatic patients upon their admission. Next, the relationship of IL-10 levels with GCS, ABG results, and short-term and long-term mortality was examined.
In this study, blood samples were taken from traumatic patients upon their admission. The samples were taken to measure IL-10 blood levels. Next, a follow-up was arranged to examine the relationship of IL-10 with levels of PH, Hco3, PC02, and GCS as well as short-term and long-term mortality.

To prevent the creation of the error and elimination of the corrupted parameters such as underlying diseases, patients with diabetes, heart and coronary vessels disease, liver and kidney failure were removed from the study.

**ELISA assay of serum IL-10 level**

Serum IL-10 level was measured by a commercially available ELISA kit with the detection limitation of 20 pg/mL (Human IL-10 DuoSet, R&D Systems, MN, USA) according to the manufacturer’s instructions.

**Ethical considerations**

First of all, the research purposes and its subject were explained to the patients. The patients’ questions - which were in relation to this study - were answered. The informed written consent was obtained from patients after explaining and answering their questions.

The patients were assured that their personal information secret and is not mentioned anywhere and they are completely preserved during the study. All information obtained from patient is confidentiality maintained and they will not be used, except for evaluation of results. These were communicated to the patient so their privacy was preserved. All information, tips and recommendations were provided to all patients at baseline and also further information and advises were available as needed for the patients during the study and thereafter. No costs were paid by the patients for check of IL-10.

**Statistical Analysis**

The collected data were analyzed by SPSS-17 statistical software. The collected data were expressed as percentage and mean ± SD. Continuous (quantitative) variables were compared by Independent samples and Paired t test. Categorical (qualitative) variables were compared by contingency tables and Chi-square test or Fisher's exact test. P-value ≤0.05 was considered statistically significant.

**Result and Discussion**

In this study 32 patients with head trauma were selected and the level of serum IL-10 in these patients was evaluated and the following results were obtained:

There were 30 male and 2 females patients in this study. The mean age of patients was 33.5±15.17 and the mean GCS of patients upon referring was 11.65±2.35.

The mean serum level of IL-10 in live patients and the deceased samples was 8.02±1.41 pg/mL and 13.19±2.39 pg/mL, respectively.

The serum level of IL-10 was significantly higher in dead patients than alive ones (P <0.001).

There exists an important inverse linear relation between serum level of IL-10 and arterial blood PH and initial amount of GCS in patients, while no significant linear relation exists between serum levels of IL-10 with PaCO2 HCo2 of arterial blood.
### Table I Demographics and laboratory findings of patients based on patients gender

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>34.43 ± 17.30</td>
<td>19.50 ± 4.95</td>
<td>33.50 ± 17.15</td>
<td>0.239</td>
</tr>
<tr>
<td>Glasgow Coma Scale</td>
<td>11.57 ± 2.34</td>
<td>13.00 ± 2.83</td>
<td>11.66 ± 2.35</td>
<td>0.413</td>
</tr>
<tr>
<td>Blood Sugar (mg/dl)</td>
<td>177.27 ± 70.14</td>
<td>224.50 ± 85.56</td>
<td>180.22 ± 70.52</td>
<td>0.368</td>
</tr>
<tr>
<td>White Blood Cell</td>
<td>14343.67 ± 3763.92</td>
<td>24550.00 ± 21283.91</td>
<td>14981.56 ± 5845.23</td>
<td>0.621</td>
</tr>
<tr>
<td>Hemoglobin (mg/dl)</td>
<td>12.31 ± 2.42</td>
<td>8.00 ± 6.36</td>
<td>12.04 ± 2.81</td>
<td>0.513</td>
</tr>
<tr>
<td>Platelet</td>
<td>223.03 ± 86.51</td>
<td>249.50 ± 6.36</td>
<td>224.69 ± 83.94</td>
<td>0.673</td>
</tr>
<tr>
<td>Blood Urea Nitrogen (mg/dl)</td>
<td>37.47 ± 17.88</td>
<td>29.00 ± 8.49</td>
<td>36.94 ± 17.49</td>
<td>0.516</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.02 ± 0.33</td>
<td>0.85 ± 0.21</td>
<td>1.01 ± 0.32</td>
<td>0.485</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>4.10 ± 0.49</td>
<td>4.65 ± 0.35</td>
<td>4.14 ± 0.50</td>
<td>0.132</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>143.83 ± 4.27</td>
<td>142.00 ± 0.00</td>
<td>143.72 ± 4.15</td>
<td>0.554</td>
</tr>
<tr>
<td>PH</td>
<td>7.36 ± 0.09</td>
<td>7.36 ± 0.09</td>
<td>7.36 ± 0.09</td>
<td>0.973</td>
</tr>
<tr>
<td>Pa CO2</td>
<td>34.16 ± 9.59</td>
<td>33.50 ± 2.12</td>
<td>34.12 ± 9.29</td>
<td>0.925</td>
</tr>
<tr>
<td>HCO3</td>
<td>19.96 ± 5.93</td>
<td>18.75 ± 3.18</td>
<td>19.88 ± 5.77</td>
<td>0.780</td>
</tr>
<tr>
<td>Pa O2</td>
<td>128.81 ± 46.47</td>
<td>172.50 ± 9.19</td>
<td>131.54 ± 46.24</td>
<td>0.201</td>
</tr>
</tbody>
</table>

### Table II Demographics and laboratory findings of patients based on mortality

<table>
<thead>
<tr>
<th></th>
<th>Alive</th>
<th>Death</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>33.08 ± 17.72</td>
<td>35.33 ± 15.79</td>
<td>0.777</td>
</tr>
<tr>
<td>Glasgow Coma Scale</td>
<td>12.23 ± 2.21</td>
<td>9.17 ± 0.75</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Blood Sugar (mg/dl)</td>
<td>188.73 ± 74.98</td>
<td>143.33 ± 26.80</td>
<td>0.159</td>
</tr>
<tr>
<td>White Blood Cell</td>
<td>15320.77 ± 5965.00</td>
<td>13511.67 ± 5544.04</td>
<td>0.503</td>
</tr>
<tr>
<td>Hemoglobin (mg/dl)</td>
<td>11.60 ± 2.90</td>
<td>13.93 ± 1.32</td>
<td>0.066</td>
</tr>
<tr>
<td>Platelet</td>
<td>220.08 ± 90.85</td>
<td>244.67 ± 42.69</td>
<td>0.527</td>
</tr>
<tr>
<td>Blood Urea Nitrogen (mg/dl)</td>
<td>39.08 ± 18.54</td>
<td>27.67 ± 7.12</td>
<td>0.153</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.02 ± 0.35</td>
<td>0.92 ± 0.16</td>
<td>0.491</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>4.15 ± 0.45</td>
<td>4.07 ± 0.70</td>
<td>0.712</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>143.69 ± 3.97</td>
<td>143.83 ± 5.31</td>
<td>0.942</td>
</tr>
<tr>
<td>PH (mEq/L)</td>
<td>7.37 ± 0.09</td>
<td>7.29 ± 0.09</td>
<td>0.040</td>
</tr>
<tr>
<td>Pa CO2</td>
<td>32.60 ± 8.34</td>
<td>40.70 ± 11.11</td>
<td>0.052</td>
</tr>
<tr>
<td>HCO3</td>
<td>19.94 ± 6.02</td>
<td>19.63 ± 5.00</td>
<td>0.909</td>
</tr>
<tr>
<td>Pa O2</td>
<td>134.85 ± 45.58</td>
<td>117.22 ± 50.66</td>
<td>0.409</td>
</tr>
</tbody>
</table>

### Chart I Distribution of serum Interleukin 10 level based of mortality
Range of levels of IL-10 in patients under study based on the mortality of patients is shown in chart I.

Demographic and experimental findings of studied patients are shown in table I and II on the basis of sex and mortality.

IL-10 is a potent anti-inflammatory cytokine, secreted by lymphocytes of the T-helper type 2 (Th2) subtype and also in large amounts by macrophages. It inhibits many cellular processes that could play an important role in plaque progression, rupture, or thrombosis, including nuclear factor-kB activation (19-20), metalloproteinase production (21), tissue factor (22) and cyclo-oxygenase-2 expression (23), 27 and cell death (24).

Schneider Soares and et al show that Serum IL-10 levels may be a useful marker for severe traumatic brain injury(TBI) prognosis and also, that higher IL-10 levels (more than 90 pg/ml) after TBI were significantly with TBI severity and hospital mortality(25). Maier and et al demonstrated that the plasma IL-10 level was significantly increased above control plasma values in patients with brain trauma and it significant reverse correlated with GCS of patients (26).

In our research, according to the results of the above study, the level of IL-10 in patients with severe trauma was higher and a considerable inverse association between levels of IL-10 and initial GCS in patients was evident.

Kirchhoff and et al demonstrated that the significant increase of IL-10 might indicate a bad outcome of TBI; responsible mechanisms still have to be elucidated (27). Csuka and et al show that IL-10 is predominantly induced intrathecal after severe TBI where it may down regulate inflammatory events following traumatic brain damage (28).

Also, the amount of IL-10 in the dead was higher than live patients significantly indicating the existence of a significant relation between IL-10 and the rate of mortality among the patients.

References

10. Zare M, Kargar S. Evaluation of prehospital care for trauma patients referred to Shahid


