The effects of LD\textsubscript{50} of Walterinnesia aegyptia crude venom on blood parameters of male rats

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The effects of an acute LD\textsubscript{50} dose of Walterinnesia aegyptia crude venom was studied in male albino rats over a period of seven days. The following analyses were performed at timed intervals of 1 h, 3 h, 6 h, 12 h, 24 h, 72 h and 7 days: white blood cells (WBC), red blood cells (RBC), Platelets count, hemoglobin content (Hb), hematocrit (Hct) and blood indices [Mean Cell Volume (MCV), mean cell hemoglobin (MCH) and mean cell hemoglobin concentration (MCHC)]. Serum enzymatic activities include alanine transaminase (ALT), aspartate transaminase (AST), γ-glutamyl transferase (γ-GT) and alkaline phosphatase (ALP), with the following metabolites concentrations (glucose, total protein, triglycerides and creatinine). These parameters were found to fluctuate with time, with tendency to regain normal control level within the first 6 h. The 12 to 24 h seems to be crucial for the process of physiological recovery. The process of physiological adaptation and recovery from LD\textsubscript{50} venom dose seems to stabilize after one week, leaving the animal alive with several lesions and disturbed physiological profile.

**Key words:** Blood parameters, crude venom, enzymes, LD\textsubscript{50}, metabolites, Walterinnesia aegyptia.

INTRODUCTION

Annually, there are more than 2.5 million cases of snake bite, mostly in rural tropical areas of which about 100,000 are fatal (White, 2005). Disturbances of haemostasis are among the most severe effects following snake bite by members of several genera from all four families (Ducancel and Goyffon, 2008). Walterinnesia aegyptia is a monotypic elapid snake found in Africa as well as sandy areas of Kuwait, Syria, Saudi Arabia, Lebanon, Jordan, Iran, Iraq, and Egypt (Russell, 1991). Envenomation by W. aegyptia is known to cause rapid deaths and paralysis (Tsai, 2008). The venom of W. aegyptia was found to contain various enzymes including phospholipases A\textsubscript{2} (PLAs) (Simon and Bdolah, 1980). L-amino acid oxidase, and proteolytic enzymes (Gitter and de Vries, 1968). Three-finger toxins, non-enzymatic proteins of 60 to 74 amino acid residues, were thought to be present only in elapid venoms, but their presence was recently demonstrated in colubrid and viperid venoms as well (Doley et al., 2008). Despite their structural similarity, they differ widely in their activities, being mostly cardio and neurotoxic but some of them also act on the haemostatic system (Kini, 2002; Kini and Doley, 2010).

Venom from the W. aegyptia and Pseudocerastes persicus fieldi have been found to have the highest haemolytic activity of the elapids and vipers and also possessed the highest phospholipase A\textsubscript{2} activity (El Hakim et al., 2008). W. aegyptia was reported to induce elevation in serum total proteins, glucose and to alter enzyme activities (Al-Jammaz et al., 1992). Also disorder was seen in liver enzyme activities, total protein and glycogen content. Dramatic disturbance of the kidney glycogen, total protein content and enzyme activities in the kidneys of envenomated animals was recorded (Al-Jammaz et al., 1994). Reduction in serum total albumin, uric acid, cholesterol, phosphorus and calcium levels along with disturbances in serum electrolyte levels (Al-Jammaz, 2001). Metabolic activity of cultured human

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fibroblasts was also affected (Al-Saleh, 1996). This study aims to determine the bio-physiological effects of an acute LD₅₀ dose of *W. aegyptia* crude venom at time intervals of 1, 3, 6, 12, 24 and 72 h as well as a week after envenomation of male rats. This will be achieved through monitoring of changes in certain biochemical parameters.

**MATERIALS AND METHODS**

**Venom collection and preparation**

The venom was obtained from *W. aegyptia*. Specimens kept in serpentina at the Zoology Department, College of Science, King Saud University. Snakes were collected from the central region of Saudi Arabia by a professional hunter. The snakes were kept in large tanks; heat was provided from a 100 watt lamp for a period of 9 h. Water was always available. Venoms was milked from adult snakes, lyophilized and reconstituted in saline solution prior to use in the investigation.

**Determination of LD₅₀ dose**

The LD₅₀ value was determined (Sun, 1963) and obtained from a dose mortality curve set up especially for venom. At the end of the experiment, surviving animals were anesthetized with pentobarbital (60 mg/kg body weight) and whole blood was drawn by heart puncture technique. Dead animals were neglected (Figure 1).

**Experimental animals**

Adult male albino rats (mass range: 200 to 250 g) were divided in two groups:

1. Control group: treated with physiological saline injection (0.2 ml, i.p.);
2. LD₅₀ group: treated with LD₅₀ (0.175 mg/kg, i.p.) of the crude *W. aegyptia* venom.

Scarification of treated animals and two blood samples were collected into heparinized syringe by heart puncture technique at intervals of 1, 3, 6, 12, 24, 72 h and the 7th day. The first sample was collected in a heparinized tube (2.25 µl heparine /5 ml blood) for blood components count analyzed by an XF9030B Haematology Analyzer. The second non-heparinized blood sample was centrifuged (1000g for 10 min), collected in test tubes with screw caps and stored at -20°C until analyzed. In serum, creatinine was estimated according to method of Bartels et al. (1971). Total proteins were measured according to method of Lowry et al. (1951). Triglyceride level was carried out using kits from Boehringer - Mannheim. The level of glucose was determined according to method of Howanitz and Howanitz (1984). Transaminase (ALT and AST), alkaline phosphatase (ALP) and γ-glutamyl transferase (γ-GT) activities were determined according to the recommendations of Scandinavian Committee on Enzymes (SCE) and using Kits from Sera-Pack (Ames Division, Miles Ltd. England).

**Statistical analysis**

In order to compare between the control and LD₅₀ group at different time intervals, a Student T-test was used. The data are presented as means ± SE and statistically analyzed using SPSS 10. Significance was set at the level of P < 0.05.

**RESULTS**

**Effect of an acute dose of LD₅₀ of *W. aegyptia* crude venom on blood elements**

In the present study, the count of WBC was found to have increased after 12 and 24 h (X̄ 6.8 ± 0.6 and X̄ 9.3 ± 0.8, respectively). RBC and Hct values increased above control at the beginning of the experiment reaching maximum values after 12 h (X̄ 6.94 ± 0.28 and X̄ 47.5 ± 2.1, respectively), then begin to decline steadily, till the 7th day where each was significantly found to be decreased (X̄ 2.9 ± 0.2 and 20.0 ± 2.8, respectively). The Mean Cell Volume (MCV) initially increased, reaching maximum value 24 h (X̄ 81.6 ± 3.0) from the beginning of the experiment, then declined to control levels after 72 h (X̄ 65.1 ± 2.8) and continues to drop until the end of the experiment. As for Hb content, it was found to be closely related to the values of MCH and MCHC. Hb content increased significantly after 12 h (X̄ 13.2 ± 0.35), then returns to control after 72 h (X̄ 8.8 ± 0.35) and declined until the 7th day (X̄ 5.5 ± 0.8) (Table 1).

**Effect of an acute dose of LD₅₀ of *W. aegyptia* crude venom on enzymes activities**

In the present study, serum AST increased significantly after 3, 12, 72 h and at the 7th day (X̄ 209 ± 6.1, X̄ 218.3 ± 6.7, X̄ 226.9 ± 8.1 and X̄ 235.2 ± 7.9, respectively). Serum ALT was found to be significantly decreased at 1 h (X̄ 43.7 ± 1.5), followed by some fluctuations in its activity, but was still significantly decreased after 24 and 72 h (X̄ 52.4 ± 2.6 and X̄ 52.4 ± 3.7, respectively) and reached the control level activity at the 7th day (X̄ 140.8 ± 4.1). Serum ALP activity significantly increased at 1, 3 and 24 h (X̄ 551.4 ± 10.9, X̄ 556.1 ± 11.2 and X̄ 385 ± 7.9, respectively). The enzyme activity fluctuated eventually decreased to reach the control activity at the 7th day. Serum γ-GT activity significantly elevated for the first 1 h (X̄ 35.5 ± 1.7), then decreased steadily between 6 h and the 7th days (X̄ 7.1 ± 0.5 and X̄ 7.1 ± 1.39, respectively) (Table 2).
Effect of an acute dose of LD_{50} of *W. aegyptia* crude venom on metabolites' concentrations

Hypoglycaemia was observed after 24 and 72 h (\( \bar{X} = 102.8 \pm 5.6 \) and \( \bar{X} = 91.7 \pm 4.3 \), respectively) with severe hyperglycaemia after 3 and 12 h (\( \bar{X} = 556.1 \pm 12.5 \) and \( \bar{X} = 219.4 \pm 7.5 \) respectively) of rat treatment, returning to control level at the 7th day (\( \bar{X} = 133.3 \pm 5.8 \)). Serum total protein significantly increased at each point of testing in the experiment. Serum triglycerides significantly increased after 1, 3 and 12 h (\( \bar{X} = 279.4 \pm 10.4 \), \( \bar{X} = 202.4 \pm 9.4 \) and \( \bar{X} = 283.4 \pm 9.5 \), respectively), and still increased after 7 days (\( \bar{X} = 275.3 \pm 11.5 \)), with some fluctuations in between. Serum creatinine increased significantly at 3, 6, 12 h and on the 7th day (\( \bar{X} = 2.3 \pm 0.06 \), \( \bar{X} = 3.8 \pm 0.07 \), \( \bar{X} = 3.25 \pm 0.03 \) and \( \bar{X} = 3.0 \pm 0.06 \), respectively) (Table 3).

**DISCUSSION**

*W. aegyptia* venom contains a number of enzymes and proteins that have characteristic effects on the nervous system alongside with other components (Warrell et al., 1989). In addition, some groups have reported that the components of *W. aegyptia* venom have an impact on blood components. Mackay et al. (1969) reported that...
Table 1. Effect of an acute dose of LD$_50$ of $W$. aegyptia crude venom on WBCs (10$^3$/mm$^3$), RBCs (10$^5$ /mm$^3$), platelets (10$^3$/mm$^3$), Hb content (g/dl), Hct value (%), MCV (fl), MCH (pg) and MCHC (g/dl) over 7 days in male rats. Results are represented in form of Mean± SE, where * is significant at < 0.05.

<table>
<thead>
<tr>
<th>Blood parameter</th>
<th>WBCs $10^3$/mm$^3$</th>
<th>RBCs $10^5$/mm$^3$</th>
<th>Hb g/dl</th>
<th>Platelets$^1$ $10^3$/mm$^3$</th>
<th>Hct (%)</th>
<th>MCV (fl)</th>
<th>MCH (pg)</th>
<th>MCHC (g/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>4.1±0.46</td>
<td>4.8±0.29</td>
<td>9.1±0.43</td>
<td>453±39.8</td>
<td>28.9±2.14</td>
<td>60.10±2.9</td>
<td>18.9±0.86</td>
<td>31.5±1.15</td>
</tr>
<tr>
<td>1 h</td>
<td>2.5±0.25$^*$</td>
<td>5.36±0.22</td>
<td>10.8±0.50</td>
<td>561±33</td>
<td>34.2±2.8</td>
<td>63.8±3.2</td>
<td>20.1±0.8</td>
<td>31.6±0.8</td>
</tr>
<tr>
<td>3 h</td>
<td>2.6±0.26$^*$</td>
<td>5.96±0.24</td>
<td>11.2±0.45</td>
<td>647±45$^*$</td>
<td>34.9±2.0</td>
<td>58.6±2.9</td>
<td>18.8±0.72</td>
<td>32.1±0.87</td>
</tr>
<tr>
<td>6 h</td>
<td>2.7±0.29$^*$</td>
<td>5.60±0.23</td>
<td>10.6±0.67</td>
<td>1096±70$^*$</td>
<td>35.8±2.5</td>
<td>63.9±3.4</td>
<td>19.9±0.74</td>
<td>29.6±0.84</td>
</tr>
<tr>
<td>12 h</td>
<td>6.8±0.6$^*$</td>
<td>6.94±0.28$^*$</td>
<td>13.2±0.35</td>
<td>459±45$^*$</td>
<td>47.5±2.1</td>
<td>68.4±3.1</td>
<td>19±0.65</td>
<td>27.8±0.85</td>
</tr>
<tr>
<td>24 h</td>
<td>9.3±0.8$^*$</td>
<td>5.39±0.26</td>
<td>10.6±0.60</td>
<td>428±40$^*$</td>
<td>39.1±2.2</td>
<td>81.6±3.0</td>
<td>18.4±0.7</td>
<td>30.2±0.88</td>
</tr>
<tr>
<td>72 h</td>
<td>4.5±0.42</td>
<td>4.79±0.23</td>
<td>8.8±0.35</td>
<td>252±35$^*$</td>
<td>35.1±1.9</td>
<td>65.10±2.8</td>
<td>19.7±0.69</td>
<td>22.5±0.82$^*$</td>
</tr>
<tr>
<td>7 days</td>
<td>3.5±0.33</td>
<td>2.9±0.20</td>
<td>5.5±0.80</td>
<td>180±40$^*$</td>
<td>20±2.8</td>
<td>58±3.0</td>
<td>17±0.63$^*$</td>
<td>20±0.79$^*$</td>
</tr>
</tbody>
</table>

Control n = 6; all other groups are n = 5

Table 2. Effect of an acute dose of LD$_50$ of $W$. aegyptia crude venom on transaminases (AST, u/l and ALT, u/l), γ-GT (u/l) and ALP (u/l) over 7 days in male rats. Results are represented in form of Mean± SE, where * is significant at < 0.05.

<table>
<thead>
<tr>
<th>Serum enzymes</th>
<th>AST (u/l)</th>
<th>ALT (u/l)</th>
<th>γ -GT (u/l)</th>
<th>ALP (u/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>157.2±5.4</td>
<td>140.4±3</td>
<td>14.2±0.9</td>
<td>186.4±6.8</td>
</tr>
<tr>
<td>1 h</td>
<td>165.9±3.9</td>
<td>43.7±1.5</td>
<td>35.5±1.7</td>
<td>551.4±10.9</td>
</tr>
<tr>
<td>3 h</td>
<td>209.5±26.1</td>
<td>61.1±2.1</td>
<td>14.2±0.8</td>
<td>556.1±11.2</td>
</tr>
<tr>
<td>6 h</td>
<td>148.4±5.4</td>
<td>69.8±3.1</td>
<td>7.1±0.5</td>
<td>287.2±9.8</td>
</tr>
<tr>
<td>12 h</td>
<td>218.3±26.7</td>
<td>113.5±3.4</td>
<td>7.1±0.45</td>
<td>148.8±5.6</td>
</tr>
<tr>
<td>24 h</td>
<td>174.6±5.9</td>
<td>52.4±2.6</td>
<td>10.7±0.60</td>
<td>385±7.9</td>
</tr>
<tr>
<td>72 h</td>
<td>226.9±8.1</td>
<td>52.4±3.7</td>
<td>10.7±0.71</td>
<td>220.8±6.5</td>
</tr>
<tr>
<td>7 days</td>
<td>253.2±7.9</td>
<td>140.8±4.1</td>
<td>7.1±0.39</td>
<td>297.1±8.1</td>
</tr>
</tbody>
</table>

cobra venom from the elapid family cause drop in the number of white blood cells a short period after injection. This is consistent with the results of the present study. Leucopenia may be due to liver failure (Kumar and Clark, 2005) which was observed as a result of envenomation. In the present study, the count of WBC increased after 12 and 24 h and this observation is consistent with observations of Amin et al. (2008) who reported that Bangladesh snake venom caused leucocytosis after a period of injection. Lifshitz et al. (2003) hypothesized that a sympathetic effect, as a result of the stress experienced by the victims, could release temporarily WBCs from the marginal pools. Another possibility is that the venom can release inflammatory cytokines from macrophages. Envenomation, including snake bite could be classified as a type of exposure to stress that leads to disputation in the production of white blood cells and has an impact on different blood components (Maes et al., 1998).

Polycythemia could be a result of kidney or liver failure or an imbalance in the haemoglobin composition leading to a decrease in plasma volume and thus increasing the amount of hematocrit. This lead in turn to a case of hemocoencentration (Chafer, 2004). Causes for a decrease RBCs could be exposure to extreme physiological stress such as envenomation (Benoit et al., 2001). Both RBC and platelets counts fluctuated during the experiment indicating that the process of clotting arose to resist bleeding or haemorrhage and then declined parallel to RBCs. Thrombocytosis is seen in many inflammatory disorders, as well as in acute or chronic blood loss and haemolytic cases. On the other hand, Thrombocytopenia could occur as result of chronic infections and liver disease (Greer et al., 2008).

The present results show that envenomation causes a significant increase in the MCV. This increase reached a maximum after 24 h. This might be explained in term of RBCs trying to carry the largest amount of haemoglobin as a mechanism to counteract hypoxia caused by the venom initial. Increase in size of the RBCs was noticed following by decline to control levels after 72 h. These results are in disagreement with those observed in other snakes from the same family (Nagi and Abdul, 2007). In that study it was concluded that the cobra venom does
not change the shape of RBCs. As for the Hb content, it was found to be closely related to the values of MCH and MCHC. Hb content did not rise significantly after the first hour of the experiment, but the increase became significant after 12 h before dropping to control levels after 72 h and further declining below control value after 7 days. This could be attributed to a physiological mechanism attempting to restore the normal blood composition. It could act by increasing production of these components exceeding their normal level as an overshoot in production, followed by a decrease below the normal level. This wave will repeat until it reaches the normal level (Sejrsen and Nielsen, 2006). The physiological reaction observed in the current experiment, where a high haemoglobin content was observed accompanied by a change in MCH and associated with it, to endorse more oxygen-linking to Haemoglobin to deliver it to vital organs, was not parallel to changes in MCHC. This indicates that the physiological response to envenomation with *W. aegyptia* tends not to be an increase the haemoglobin concentration and prefer to increase the size of the RBCs to carry more haemoglobin. This mechanism may vary depending on the type of venom used, as *Hydrophis spiralis* venom was found to lower haemoglobin, RBCs and WBCs in guinea pigs and rabbits (Karthikeyan et al., 2007).

AST increased with time, significant increase was seen after 3, 12 and 72 h and at the 7th day. On the other hand, serum ALT was found to highly and significantly decreased after the first hour, followed by some fluctuation in its activity, but still significantly low after 24 and 72 h. It reached the control level at the 7th day. The increase in AST activity was persistent in envenomated - experimental animals, while the opposite was observed in ALT enzyme activity. An increase of serum AST and minimal changes in ALT activities after different types of snakes’ envenomation was observed (Assi and Naser, 1999). Symptoms similar to hepatitis, liver cirrhosis and muscular dystrophy were also reported (Porth, 1990).

ALP enzyme activity underwent significant increase at 1, 3 and 24 h. The enzyme activity then fluctuated but eventually decreased to reach the control activity on the 7th day. ALP enzyme is found mainly in the bile ducts of the liver and increase in ALP activity can indicate obstructive or cholestatic liver disease where bile is not properly transported from the liver because of bile duct obstruction. Studies by AL-Jammaz et al. (1992) on *W. aegyptia* venom correlate well with our recent findings. Serum γ-GT enzyme activity was found to be significantly elevated for the first 12 h of the experiment, then decrease steadily from 24 h till the 7th day of the experiment. γ-GT is produced in many tissues as well as the liver. Elevations in serum γ-GT, especially along with elevations in alkaline phosphatase, suggest bile duct disease. An increase occurs in certain enzymes such as ALP and γ-GT which are possible marker for cell damage and degeneration as reported by Mohamed et al. (1978) in an experiment using LD₅₀ of *N. haje* venom. Decrease in these enzymes reduced synthesis, direct inhibition or failure to excrete this is due to cell damage by the toxic venom (Mohamed et al., 1981).

In the present study, hypoglycemia was observed after 3, 24 and 72 h with a severe hyperglycaemia 12 h after envenomation, returning to control level at the 7th day. This reduction of blood glucose level reflects a disturbance in carbohydrate metabolism which could be attributed to some endogenous insulin-releasing effect of the venom components. The stress caused by venom administration might be another factor in reducing the blood glucose level. Such stress might have occurred as direct effect of the venom enhancing the release of insulin, or indirectly by inhibiting catecholamine release due to exhaustion or blocking of the adrenal sympathetic supply (Abu-Sinna et al., 1993). Moreover, hypoglycaemia following *W. aegyptia* venom injection has been interpreted to be due to either an increased out-pouring of insulin, or to an inhibition of the diabogenic factors of the anterior pituitary and suprarenal cortex (Mohamed et al., 1965). The hyperglycaemia observed is in accordance with Fahim (1998). Several explanations were given to the hyperglycaemia produced after snakes or scorpion envenomation such as altered neurotrans-

### Table 3. Effect of an acute dose of LD₅₀ of *W. aegyptia* crude venom on total protein (g/dl), Creatinine (mg/dl) and Triglycerides (mg/dl) glucose (g/dl) over 7 days in male rats. Results are represented in form of Mean± SE, where * is significant at < 0.05.

<table>
<thead>
<tr>
<th>Metabolites</th>
<th>Glucose (mg/dl)</th>
<th>Total proteins (g/dl)</th>
<th>Triglycerides (g/dl)</th>
<th>Creatinine mg/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>141.7±4.6</td>
<td>6.9±1.3</td>
<td>129.6±6.5</td>
<td>1.3±0.05</td>
</tr>
<tr>
<td>1 h</td>
<td>130.6±5.1</td>
<td>7.1±2.3</td>
<td>279.4±10.4</td>
<td>2.2±0.06</td>
</tr>
<tr>
<td>3 h</td>
<td>556.1±12.5</td>
<td>6.8±1.2</td>
<td>202.4±9.4</td>
<td>2.3±0.06</td>
</tr>
<tr>
<td>6 h</td>
<td>130.6±6.2</td>
<td>6.6±1.4</td>
<td>153.8±5.7</td>
<td>3.8±0.07</td>
</tr>
<tr>
<td>12 h</td>
<td>219.4±7.5</td>
<td>6.6±1.6</td>
<td>283.4±9.5</td>
<td>3.25±0.03</td>
</tr>
<tr>
<td>24 h</td>
<td>102.8±5.6</td>
<td>6.3±1.5</td>
<td>174.1±8.5</td>
<td>1.9±0.04</td>
</tr>
<tr>
<td>72 h</td>
<td>91.7±4.3</td>
<td>6.7±1.1</td>
<td>186.2±7.4</td>
<td>1.9±0.07</td>
</tr>
<tr>
<td>7 days</td>
<td>133.3±5.8</td>
<td>7.3±1.4</td>
<td>275.3±11.5</td>
<td>3.0±0.06</td>
</tr>
</tbody>
</table>
mission of adrenergic mechanism (Freire-Maia and Ferreira, 1961), β -receptor activation (Corrado et al., 1968) or release of tissue and medullary catecholamines (El-Asmar et al., 1974). Some components of the venom itself could induce hyperglycaemia, and metabolic dysfunction, as inhibition of glucose uptake by skeletal muscle, inhibition of insulin release or stimulation of glucagon secretion (Johnson and Ensinck, 1976), glycogenolysis and/or retarded glucose utilization at the peripheral tissues (Mohamed et al., 1965). Even a role of hypothalamo-pituitary adrenal axis -HPA- (El-Fiky, 1999) was suggested as possible mechanisms for the increased serum glucose level. An interesting observation in this experiment is that the hyperglycaemia declined steadily with time. It is then possible that there is an endogenous insulin releasing effect of one or more venom components; in addition, it is possible that venom components themselves may possess insulin - like effect as proposed by Abu-Sinna et al. (1993). Serum total protein maintained its significant increase all over the period of the experiment. The ability of W. aegyptia crude venom to disturb protein metabolism was previously observed by Al-Saleh (1996) . The increased serum level could be due to tissue destruction (Moustafa et al., 1974) or due to Hb concentration (Ismail and Osman, 1973), as well as to the action of protease inhibitor activities present in the venom Yang (1999). Total protein is increased in inflammatory conditions such as chronic or severe infections, while low total protein can result from protein loss, as occurs in haemorrhage, glomerulonephritis, nephrosis and chronic liver disease. This could be explained on the basis of the venom components such as cytotoxins, neurotoxins, myotoxins and phospholipases A2. Serum triglycerides were found to be highly and significantly increase after 1 and 12 h and still increased after seven days, with some fluctuation during the time of experiment. This disturbance indicate that plasma lipids are possible targets for the W. aegyptia venom. Phospholipases A2 (PLA2) specifically catalyzes hydrolysis of the sn-2 ester bond in glycerophospholipids to give lysophospholipids and fatty acids. SPLA2s in snake venoms are either GI or GII (Valentin and Lambeau, 2000).GI s SPLA2s are found in venoms of Elapidae (Sajevic et al., 2011). The increases in serum triglycerides levels in envenomated rats observed in the present study could be due to hepatocyte damage rendering them unable to phosphorylate the increasing amounts of fatty acids, hence leading to fatty liver and alteration of cell membranes of tissue(s). Serum creatinine level increased during the period of the experiment. The increase was found to be significant after 3 and 12 h and on the 7th day, while the increase was highly significant after 6 h. This rise indicates impairment of renal function and nephrotoxicity. Similar observations were reported in rats following administration of various snake venoms (Rahmy et al., 1995, Omran et al., 1997). The tissue distribution of the venom showed the highest uptake in the kidney (Ismail et al., 1996).

**Conclusion**

It appears that LD_{50} of W. aegyptia crude venom might have produced tissues injuries, especially in liver, heart, kidney and muscles and those injuries appeared to be time dependent.

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