Study of some serum biochemical changes of experimental diabetes mellitus in Donkey

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Abstract

This study was conducted to induce experimental diabetes in donkeys and assess some serum biochemical changes in diabetes mellitus. Fifteen donkeys were used in this experiment and divided into two groups:

A. Treatment group which include (11) donkeys. Diabetic group denoted by Dm.

B. Control group which includes (4) donkeys denoted by Cm.

Animals were housed in field. The animals were left tow weeks for adaptation. Diabetes mellitus was induced by i.v injection of alloxan monohydrate at dose rate 100 mg/kg dissolved in 25 ml of normal saline, while the control groups were injected with 25 ml of normal saline. Blood was collected before injection of alloxan monohydrate and after that (10,20,30,40) days to check fasting and random serum glucose. The laboratory tests including serum glucose, serum urea, serum creatinine, serum cholesterol, serum lactate dehydrogenase. We found out the following result according to the periods of the experiments: Serum glucose level was elevated starting from the 1st 10 days period after induction of diabetes mellitus. It reached its peak levels on day(20,30,40). Serum urea, serum creatinine, serum cholesterol, serum lactate dehydrogenase were elevated in diabetic donkeys, in control groups, but there were no significant difference.

Keywords: Diabetes mellitus, donkeys

Introduction

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both (1). The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels (2). Diabetes as defined in a general term refers to disorders characterized by excessive urine excretion (Polyurea) and derived from the Greek word meaning a siphon. In lay terms diabetes, when used alone, refers to diabetes mellitus. Clinically, diabetes may refer to either diabetes mellitus or diabetes insipidus. (3). Chronic hyperglycemia is associated with micro vascular and cardiovascular complications that increase risk of morbidity and mortality (4). Diabetes is a chronic illness that requires continuing medical care and patient self-management education to prevent acute complications and to reduce the risk of long-term complications (5).

Four major types of diabetes have been defined: type-1 (insulin-dependent diabetes mellitus IDDM), type-2 (non-insulin-dependent diabetes mellitus NIDDM), gestational diabetes mellitus (GDM) and diabetes secondary to other conditions. Type-1 characteristically presents with prominent diabetes symptoms and extreme hyperglycemia. Type II diabetes, which results of insulin resistance, is the most prevalent form of diabetes today. Of individuals diagnosed with diabetes, 90-95% suffers from type II diabetes mellitus. The key objectives in the management of diabetes are optimizing metabolic control, preventing acute and chronic complications and improving quality of life (6). Type1-2 can be diagnosed by the presence of the classical signs and symptoms of diabetes together with unequivocally elevated blood glucose levels; by fasting plasma glucose (FPG) 90 mg/dl.

Aims of study induce of diabetes mellitus in donkey and Measurement of some biochemical parameters
**Material and Method**

Fifteen donkeys were collected in the field, and divided into two groups after two weeks.

**Group A:**

This group includes (11) donkeys aged from (3-7) months, weight was calculated for each animal in order to calculate dose of alloxan for required for the induction of diabetes (DM)

**Group B:**

This group includes four as control group After calculating the live weight of each animal the dose of alloxan were calculated which is given intravenously 100 mg per kg of body eight dose is sufficient for the development of diabetes Alloxan was dissolve material, according to the appropriate dose for each animal, 25 ml of normal saline was injected all test animals, which were numbered from the number (1-11). normal saline were numbered animal (15-19). blood samples were collected from each animal (0'10'20'30'40) day from treatment and control group to estimate the serum biochemical (glucose, urea, creatinine, cholesterol, Lactate dehydrogenase LDH ).

**Results**

Table (1) show blood glucose concentration. At zero time there was no significant differences in the means of serum glucose concentration in fasting (71.3 ± 1.9 VS 80.45±3.6) and random (121.1±9.7VS 144.0±13.1)for treatment and control groups respectively P>0.05. After 10 days experimental the serum glucose concentration was increased in treatment group of fasting blood sample (104.909±1.9 VS 79.79 ± 4.5)there was highly significant differences between two group (p<0.01) on ether hand there was no significant differences in blood glucose concentration of random blood sample (146.9±5.37 VS 158.25± 8.58) for treatment and control group respectively P>0.05. After 20 days experimental diabetes mellitus there was highly significant increased serum glucose concentration of fasting blood sample in the treatment group (125.4±1.3 VS 85.25 ± 1.43) for treatment and control group respectively (p<0.01) on ether hand there was no significant increased blood glucose concentration of random blood sample treatment group (167.56±7.64 VS 131.75 ± 16.94) for treatment and control respectively P<0.05. After 30 days of experimental there was highly significant increased in the serum glucose concentration in the treatment group for fasting (138.4 ±5.77 VS 84.97±3.5) for treatment and control group respectively( p<0.010) on ether hand there was significant increased in the blood glucose concentration for random blood sample in the treatment group (190.2±22.88VS 148.0±12.73) for treatment and control group respectively(P<0.05). After 40 days of experimental there was highly significant increased in the serum glucose concentration for both fasting and random (167.0 ±6.19 VS 84.825±2.65) for treatment and control group respectively in the fasting blood sample (236.875±13.65VS 144.0±6.1) for treatment and control group respectively of random blood sample (p<0.01).
Table (1): Results of serum biochemical values of study donkeys

<table>
<thead>
<tr>
<th>Glucose parameters/fasting</th>
<th>P</th>
<th>Mean± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zero day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>p&gt;0.05</td>
<td>71.3±1.9</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>80.45±3.9</td>
</tr>
<tr>
<td>10 day</td>
<td></td>
<td></td>
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<tr>
<td>Treatment</td>
<td>P&lt;0.01</td>
<td>104.9±1.9</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>79.79±4.5</td>
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<tr>
<td>20 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>P&lt;0.01</td>
<td>125.4±13.66</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>85.25±1.436</td>
</tr>
<tr>
<td>30 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>P&lt;0.01</td>
<td>138.4±5.77</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>84.97±3.5</td>
</tr>
<tr>
<td>40 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>P&lt;0.01</td>
<td>167.00±6.19</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>84.825±2.6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Glucose parameters/Random</th>
<th>P</th>
<th>Mean± SD</th>
</tr>
</thead>
<tbody>
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<td>Zero day</td>
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<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>p&gt;0.05</td>
<td>121.1±9.7</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>144.00±13.1</td>
</tr>
<tr>
<td>10 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>p&gt;0.05</td>
<td>146.9±5.37</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>158.25±8.58</td>
</tr>
<tr>
<td>20 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>P&lt;0.05</td>
<td>167.56±7.64</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>131.75±16.94</td>
</tr>
<tr>
<td>30 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>P&lt;0.05</td>
<td>190.2±22.88</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>148.0±12.73</td>
</tr>
<tr>
<td>40 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>P&lt;0.01</td>
<td>236.875±13.65</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>144.00±6.1</td>
</tr>
</tbody>
</table>

Table (2) shows blood urea concentration. At zero, 10, and 20 days there was no significant differences in the means of serum urea concentration in blood sample (51.95±1.8 VS 54.37±3.2) (57.64±2.64 VS 56.05±3.37) (62.1700±1.9 VS 57.00±3.02) for treatment and control groups respectively p>0.05. After 30 days experimental diabetes mellitus the serum urea concentration was increased in treatment group blood sample (67.36±2.07 VS 57.375±2.99) there was significant differences between two group (p<0.05). After 40 days experimental the serum urea concentration was highly significant increased in treatment group blood sample (70.48±1.93 VS 55.73±3.975) for treatment group (p<0.01).

Table (2): Results of serum biochemical values of study donkeys

<table>
<thead>
<tr>
<th>Urea parameters</th>
<th>P</th>
<th>Mean± sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zero day</td>
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</tr>
<tr>
<td>Treatment</td>
<td>p&gt;0.05</td>
<td>51.95±1.8</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>54.37±3.2</td>
</tr>
<tr>
<td>10 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>p&gt;0.05</td>
<td>57.64±2.64</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>56.05±3.37</td>
</tr>
<tr>
<td>20 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>p&gt;0.05</td>
<td>62.17±1.9</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>57.0±3.02</td>
</tr>
<tr>
<td>30 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>p&gt;0.05</td>
<td>67.36±2.07</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>57.375±2.99</td>
</tr>
<tr>
<td>40 day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>P&lt;0.01</td>
<td>70.48±1.93</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>55.73±3.975</td>
</tr>
</tbody>
</table>
Table (3) show blood creatinine concentration. At zero and 10 days there was no significant differences in the means of serum creatinine concentration in blood sample (1.83 ± 0.06 VS 1.96 ± 0.024) (2.41 ± 0.155 VS 2.04 ± 0.037) for treatment and control groups respectively p > 0.05. After 20, 30, and 40 days experimental diabetes mellitus the serum creatinine concentration was increased in treatment group blood sample (2.68 ± 0.06 VS 2.1 ± 0.057) (3.16 ± 0.11 VS 2.05 ± 0.04) (3.81 ± 0.24 VS 2.085 ± 0.06) there was highly significant differences between two group (p < 0.01).

<table>
<thead>
<tr>
<th>Creatinine parameters</th>
<th>p</th>
<th>Mean ± SD</th>
</tr>
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<tbody>
<tr>
<td>Zero day</td>
<td>Treatment p &gt; 0.05</td>
<td>1.836 ± 0.06</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>1.96 ± 0.024</td>
</tr>
<tr>
<td>10 day</td>
<td>Treatment p &gt; 0.05</td>
<td>2.41 ± 0.155</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>2.04 ± 0.037</td>
</tr>
<tr>
<td>20 day</td>
<td>Treatment P &lt; 0.01</td>
<td>2.68 ± 0.06</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>2.1 ± 0.057</td>
</tr>
<tr>
<td>30 day</td>
<td>Treatment P &lt; 0.01</td>
<td>3.16 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>2.05 ± 0.04</td>
</tr>
<tr>
<td>40 day</td>
<td>Treatment P &lt; 0.01</td>
<td>3.81 ± 0.24</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>2.085 ± 0.06</td>
</tr>
</tbody>
</table>

Table (4) show blood cholesterol concentration. At zero time there was no significant differences in the means of serum cholesterol concentration in blood sample (96.18 ± 4.18 VS 80.5 ± 3.9) for treatment and control groups respectively p > 0.05. After 10 days experimental the serum cholesterol concentration was increased in treatment group blood sample (116.1 ± 9.6 VS 74.00 ± 3.11) there was significant differences between two group (p < 0.05). After 20 days experimental the serum cholesterol concentration there was highly significant increased in treatment group blood sample (124.4 ± 9.66 VS 75.78 ± 2.71) (131.467 ± 4.7 VS 75.5 ± 2.9) (144.94 ± 4.65 VS 78.375 ± 2.87) for treatment group (p < 0.01).

<table>
<thead>
<tr>
<th>Cholesterol parameters</th>
<th>p</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zero day</td>
<td>Treatment p &gt; 0.05</td>
<td>96.18 ± 4.18</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>80.5 ± 3.9</td>
</tr>
<tr>
<td>10 day</td>
<td>Treatment p &lt; 0.05</td>
<td>116.1 ± 9.6</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>74.00 ± 3.11</td>
</tr>
<tr>
<td>20 day</td>
<td>Treatment P &lt; 0.01</td>
<td>124.4 ± 9.66</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>73.78 ± 2.71</td>
</tr>
<tr>
<td>30 day</td>
<td>Treatment P &lt; 0.01</td>
<td>131.467 ± 4.7</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>75.5 ± 2.9</td>
</tr>
<tr>
<td>40 day</td>
<td>Treatment P &lt; 0.01</td>
<td>144.94 ± 4.65</td>
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<td></td>
<td>Control</td>
<td>78.375 ± 2.87</td>
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</tbody>
</table>
Table (5) show blood lactate dehydrogenase (LDH) concentration. At zero, 10, 20, and 30 days there was no significant differences in the means of serum LDH concentration in blood sample (222.27 ± 19.4 VS 250.5±5.9)( 251.9 ± 19.2 VS 258.25±4.0) 256.3 ± 17.5 VS 252.5±1.5) for treatment and control groups respectively p>0.05. After 40 days experimental diabetes mellitus the serum LDH concentration was increased in treatment group blood sample (319.00±18.68 VS 258.75±2.36) there was significant differences between two group (p<0.05).

Table (5): Results of serum biochemical values of study donkeys

<table>
<thead>
<tr>
<th>Lactate dehydrogenase (ldh) parameters</th>
<th>p</th>
<th>Mean± sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zero day</td>
<td>Treatment</td>
<td>p&gt;0.05</td>
</tr>
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<td></td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td>10 day</td>
<td>Treatment</td>
<td>p&gt;0.05</td>
</tr>
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<td></td>
<td>Control</td>
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<tr>
<td>20 day</td>
<td>Treatment</td>
<td>p&gt;0.05</td>
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<td></td>
<td>Control</td>
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</tr>
<tr>
<td>30 day</td>
<td>Treatment</td>
<td>p&gt;0.05</td>
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<tr>
<td></td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td>40 day</td>
<td>Treatment</td>
<td>P&lt;0.05</td>
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<td></td>
<td>Control</td>
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</tbody>
</table>

Discussion

Alloxan induced diabetes mellitus is characterized by a state of hyperglycemia and related clinical signs such as thirst, polyphagia & poly urea. Alloxan (as monohydrate) was used for the induced experimental of diabetes in animals has been use in mouse (7), spiny mouse (8), Rat (9), rabbits (10), feline (11), dogs (12), swine (13), sheep (14, 15) and cattle (16, 17) to induce diabetes mellitus in rabbits. The dose used in this experiment (100 mg /kg) was clinically effective and led to induced hyperglycemia within three days. The results revealed significant differences between diabetic groups and control groups, which are consistent with (18); (19); (20); (21); (22); (23); (24); and (25). The study revealed a significant difference in serum glucose concentrations between the periods of the experiment as there was progressive increase in the serum glucose levels; this is similar to the results of (26); and (27). Alloxan acts on the insulin-producing pancreatic B-cells within islets of Langerhans which are selectively destroyed by the oxidant production (28). In the present study diabetic hyperglycemia induces elevation of plasma levels of urea and creatinine which are considered as significant markers of renal dysfunction (29). Significant increase in the level of plasma urea and creatinine in the diabetic. These results indicated that diabetes could be lead to renal dysfunction. In this study there was an elevation in the levels of the cholesterol that may be due to insulin lack in diabetic animals, which is consistent with the results of (30). On the other hand, a decrease in hepatic triglyceride lipase activity was found in rabbits with diabetes induced by alloxan (31). The behavior of the enzyme is similar to that of adipose tissue lipoprotein lipase, which is under the insulin hormonal regulation (32). Overproduction of very low-density lipoproteins (VLDL) was found to be the main cause of elevated triglyceride levels in type II diabetes.
mellitus (33). The results of the present study indicated that plasma LDH levels in rabbits with diabetes mellitus were significantly higher when compared with the controls. To date, no study examining plasma enzyme in diabetes mellitus has been reported in rabbits. Therefore, we were not able to compare our results with others. Although a number of studies about LDH and GOT levels in diabetes mellitus have been carried out, the results are mostly in conflict. (34), (35) and (36) did not observe any increases in LDH between diabetes and controls; consequently their results are not in accordance with ours. (37), (38), (39), (40), (41), (42) and (43) indicated that LDH levels were higher in patients with diabetes mellitus than those in normal subjects. Consequently, these reports are in accordance with our findings. On the other hand, (44) and (45) observed decreases in LDH in diabetic subjects. These results are in contradiction with our findings and those of other studies. In addition, (46).

References


دراسة لبعض التغييرات للصفات الكيموحيوية لمرض السكري المستحدث في الحمير

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رحمن كاظم محسن
كلية الطب البيطري / جامعة البصرة

عبد الأمير عودة إسماعيل
كلية الطب البيطري / جامعة كربلاء

الخلاصة

أجريت الدراسة الحالية لتقييم بعض الصفات الكيموحيوية المتمثلة بتركيز السكر والكولسترول واليوسيا وأنزيم الكرياتين عند استحداث مرض السكري في الحمير باستخدام مادة الالوكسان. استخدم في التجربة 15 من الحمير والتي قسمت بصورة عشوائية إلى مجموعتين تضمنت المجموعة الأولى (المعالمة) 11 من الحمير والتي حققت مادة الالوكسان بجرعة 100ملغم/كم. أما المجموعة الثانية (سيطرة) والتي تضمنت 4 حمير كانت معيّنة بتوزيع الفضلي بجرعة 25مل/حيوان. تم جمع عينات الدم خمس مرات (قبل الحقن، 40، 30، 20، 10 يوم بعد الحقن). وعلى مرحلتين وهما مرحلة الصوم ومراحل إعطاء الغذاء. أشارت النتائج النتائج إلى ارتفاع عالي المعنوي (p<0.01) لمادة الالوكسان على تركيز السكر عند (10 و20 و30 و40) يوم خلال مرحلة الصوم بينما وجد تأثير معنوي (p<0.05) عند (20 و30) يوم وارتفاع عالي المعنوي (p<0.01) عند 40 يوم خلال مرحلة إعطاء الغذاء. كما تلاحظ ارتفاع معنوي (p<0.05) وارتفاع عالي المعنوي (p<0.01) عند 20 و30 و40 يوم بعد الحقن. أما بالنسبة لمستوى اليوريا فقد أشارت النتائج إلى تأثير عالي المعنوي (p<0.01) عند 40 يوم بعد الحقن. كما أثّرت المعالمة تأثيراً عالياً (p<0.01) على مستوى آنزيم LdH عند 40 يوم بعد الحقن. كما أثّرت المعالمة تأثيراً معنوي (p<0.05) عند 40 يوم بعد الحقن. كما أثّرت المعالمة تأثيراً معنوي (p<0.05) عند 40 يوم بعد الحقن.