Serobiochemical Alterations in Subclinically Affected Dromedary Camels with \textit{Trypanosoma \textit{evansi} in Iran

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\textbf{A B S T R A C T}

The aim of this study was to investigate the effects of subclinical \textit{Trypanosoma \textit{evansi}} infection on serum biochemical parameters of dromedary camels. Jugular vein blood samples were taken weekly for three successive weeks from 110 apparently healthy male camels and examined for the presence of trypomastigote form of \textit{T. \textit{evansi}} in blood smears. The parasite was seen in 17 (15.45\%) blood smears. Various serum biochemical parameters i.e., glucose, urea, cholesterol, triglyceride, calcium, magnesium, sodium, potassium, phosphorus, total protein, albumin, triiodothyronine (T\textsubscript{3}), thyroxine (T\textsubscript{4}), cortisol, and activities of aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH) and creatine kinase (CK) were determined. Infected camels had significantly lower serum glucose, T\textsubscript{3} and T\textsubscript{4} concentrations (P<0.05), and significantly higher triglycerides concentration and ALT activity (P<0.05). It was concluded that subclinical infection of camels with \textit{T. \textit{evansi}} can also affect some biochemical parameters.

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\textbf{INTRODUCTION}

Camel is one of the best adopted animals of the desert, a source of milk, meat and wool and is used for transportation and racing (Kamal, 2008; Meiloud \textit{et al}., 2011). Camel trypanosomiasis, also known as Surra, is a disease caused by \textit{Trypanosoma \textit{evansi}}. This parasite affects many species of mammals in Africa, Asia and Latin America (Omer \textit{et al}., 2007). Camels are most often affected in the Middle East and Africa (Brun \textit{et al}., 1998). Surra is the most economically important disease of camel herds with morbidity of up to 30\% and mortality of around 3\% (Enwezor and Sackey, 2005). The disease may occur in both acute and chronic forms, but generally the chronic form is more common. The acute form of the disease is usually fatal within a few weeks, but the chronic form lasts for years and is characterized by anemia, emaciation, recurrent fever, edema, conjunctivitis, lacrimation, enlargement of the lymph nodes, and abortions (Gutierrez \textit{et al}., 2005). Many investigators have shown alterations in blood constituents and tissue lesions in infected camels (Chaudhary and Iqbal, 2000; Al–Qarawi \textit{et al}., 2001; Saleh \textit{et al}., 2009). The aim of this study was to investigate the effects of subclinical \textit{T. \textit{evansi}} infection on serum biochemical parameters of dromedary camels.

\textbf{MATERIALS AND METHODS}

The research was carried out in the Yazd province, an arid region in center of Iran having minimum and maximum summer temperature 14.50 and 45.50\(\)C, respectively. This study was performed on 110 apparently healthy one-humped male 5 to 10 years old camels kept by local farmers and were fed low quality diets contained mainly straw, barley and wilted grass. Repeated jugular vein thin and thick blood smears were prepared weekly for three successive weeks during summer season (23th June to 22nd September 2008) to confirm parasites microscopically. Giemsa staining of slides revealed trypomastigote form of parasite in the blood of infected animals (Soulsby, 1982). Non infected animals were considered as control. Jugular vein blood samples were also collected without any anticoagulant and were
subjected to extraction of serum and stored at –20°C till analysis.

Serum glucose was measured by enzymatic (GOD-PAP) colorimetric method, urea by enzymatic (Urease) colorimetric, cholesterol by enzymatic (CHOD-PAP) colorimetric, triglyceride by enzymatic (GPO-PAP) colorimetric, calcium by orthocresolphthalein complex, magnesium by xylidyl blue, phosphorus by ammonium molybdate, albumin by bromcresol green (BCG), total protein by biuret method. Activity of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were determined by enzymatic (IFCC) colorimetric method, alkaline phosphatase (ALP) and lactate dehydrogenase (LDH) by enzymatic (DGKC) colorimetric and creatine kinase (CK) by enzymatic (IFCC/DGKC) colorimetric method (Shimadzu spectrophotometer, model AA200, Tokyo, Japan). Estimation of triiodothyronine (T3), thyroxine (T4) and cortisol levels in serum were made by ELISA method. Sodium and potassium concentrations were determined by flame photometry method (digital flame analyzer, model 2655–00 Cole–Pulmer Instrument Company, Chicago, IL, USA) (Latimer et al., 2003). The data were analyzed by SPSS software ver. 16, with Independent Student’s t–test.

RESULTS

Thin and thick blood smears revealed slender and flagellated trypomastigote forms that were morphologically compatible with T. evansi. The parasite was seen in 17 (15.45%) blood smears. As the area of sampling was out of Tsetse belt zone and according to the morphological and morphometrical indices, the parasites were diagnosed as T. evansi. The mean (±SE) values of biochemical parameters in dromedary camels naturally infected with T. evansi and non–infected camels are presented in Table 1. A significant (P<0.05) reduction was seen in the values of glucose, T3 and T4 whereas values of triglyceride and ALT were significantly elevated in infected camels in comparison with non–infected group (Table 1). The concentrations of serum urea, cholesterol, calcium, magnesium, sodium, potassium, phosphorus, albumin, total protein, cortisol and activity of AST, ALP, LDH and CK differed non–significantly between infected and non infected groups.

DISCUSSION

In the present study, a significant (P<0.05) reduction was seen in the values of T3 and T4 whereas values of triglyceride and ALT were significantly elevated in infected camels in comparison with non–infected group. Serum glucose concentration had a significant decrease in the infected subjects. Similar results were documented by Raisinghani and Lodha (1980) in camel. This is a common finding in trypanosomiasis, and is reported due to excessive utilization of blood glucose by the parasites for their metabolism (Anosa, 1988). It has also been shown that parasite count is inversely proportional to glucose concentration (Jatkar and Singh, 1974). Increased metabolic rate caused by fever and hepatocyte degeneration could also be a reason for hypoglycemia in trypanosomiasis (Cadioli et al., 2006). In contrast, Hilali et al. (2006) reported no significant changes in the mean serum glucose of buffalo calves.

Table 1: The mean (±SE) serum biochemical parameters of infected and non–infected dromedary camels with Trypanosoma evansi

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Units</th>
<th>Infected camels (n=17)</th>
<th>Non infected camels (n=93)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>mmol/L</td>
<td>2.21±0.39*</td>
<td>3.60±0.13</td>
</tr>
<tr>
<td>Urea</td>
<td>mmol/L</td>
<td>10.08±0.95</td>
<td>9.61±0.35</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>mmol/L</td>
<td>0.79±0.10</td>
<td>0.89±0.04</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>mmol/L</td>
<td>1.02±0.34*</td>
<td>0.44±0.03</td>
</tr>
<tr>
<td>ALT</td>
<td>IU/L</td>
<td>21.06±1.34*</td>
<td>16.71±0.78</td>
</tr>
<tr>
<td>AST</td>
<td>IU/L</td>
<td>81.12±5.52</td>
<td>116.60±8.44</td>
</tr>
<tr>
<td>ALP</td>
<td>IU/L</td>
<td>94.12±7.18</td>
<td>118.19±8.89</td>
</tr>
<tr>
<td>LDH</td>
<td>IU/L</td>
<td>479.65±45.98</td>
<td>505.45±40.18</td>
</tr>
<tr>
<td>CK</td>
<td>IU/L</td>
<td>149.94±69.48</td>
<td>92.38±13.18</td>
</tr>
<tr>
<td>Calcium</td>
<td>mmol/L</td>
<td>2.44±0.10</td>
<td>2.55±0.03</td>
</tr>
<tr>
<td>Magnesium</td>
<td>mmol/L</td>
<td>1.08±0.03</td>
<td>1.08±0.02</td>
</tr>
<tr>
<td>Sodium</td>
<td>mEq/L</td>
<td>165.06±3.46</td>
<td>167.85±1.28</td>
</tr>
<tr>
<td>Potassium</td>
<td>mEq/L</td>
<td>6.58±0.27</td>
<td>6.27±0.11</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>mmol/L</td>
<td>2.16±0.13</td>
<td>1.96±0.5</td>
</tr>
<tr>
<td>Albumin</td>
<td>g/dL</td>
<td>3.60±0.16</td>
<td>3.75±0.05</td>
</tr>
<tr>
<td>Total protein</td>
<td>g/dL</td>
<td>7.36±0.18</td>
<td>7.44±0.08</td>
</tr>
<tr>
<td>T3</td>
<td>nmol/L</td>
<td>3.73±0.022</td>
<td>5.37±0.37</td>
</tr>
<tr>
<td>T4</td>
<td>nmol/L</td>
<td>135.14±9.31*</td>
<td>159.06±4.29</td>
</tr>
<tr>
<td>Cortisol</td>
<td>nmol/L</td>
<td>11.12±1.87</td>
<td>10.16±0.99</td>
</tr>
</tbody>
</table>

Values bearing asterisk in a row differ significantly (P<0.05).

The results of enzyme assays showed elevation of ALT activity in the infected animals, while no changes were noted in the ALP, AST, CK and LDH activity in the present study. Increase in AST and ALT activity has been reported in T. evansi infected camels (De La Rue et al., 1997). The rise in AST activity can be attributed partly to cellular damage caused by the trypanosomes lysis, while the increase in ALT activity probably results from host cellular damage caused by the trypanosomes lysis, while the increase in ALT activity probably results from host destruction of trypanosomes (Enwezor and Sacky, 2005). Compared with the previous report in which ALP, AST and LDH enzymes displayed increase during trypanosomiasis (Rahman, 1992), in the present study, no changes were observed in these enzymes of infected camels. There is possibility that vital organs (liver and kidneys) could have not been affected at the time of examination. Our findings are in accordance with the works of Chaudhary and Iqbal (2000) and Gutierrez et al. (2005).

There was no significant difference in the serum albumin and total protein of infected animals when compared with healthy group. It has been shown a significant increase in total protein and globulins of rabbits infected with T. brucei (Orhue et al., 2005) and camels and buffalo calves infected with T. evansi (Gutierrez et al., 2005; Hilali et al., 2006). The increase in serum total protein may have been due to increased release of tissue specific enzymes and other intracellular proteins secondary to parasite–induced cell membrane disruption (Orhue et al., 2005). It was also shown that increase of total protein and globulins could be due to elevation in the gammaglobulin, as immunological response against the parasite (Orhue et al., 2005; Hilali et al., 2006). Orhue et al. (2005) indicated that serum albumin levels decreased in trypanosomiasis. The edema reported in the dependent parts of the body during the
chronic stage could be due to a significant decrease in the albumin levels that possibly indicates great liver damage (Enwezor and Sacky, 2005).

In the present study, subclinical infection with *T. evansi* was associated with significant reduction in the concentrations of T3 and T4. It was shown that trypanosomal infection rapidly impairs function of the thyroid gland in goats experimentally infected with *T. congolense* (Mutayoba et al., 1988) and induces hypothyroidism in cattle (Abebe and Eley, 1992). Also in camel, *T. evansi* can induce a significant reduction in serum T3 and T4 levels associated with parallel decrease in the blood concentration of TSH (Al–Qarawi et al., 2002). Mechanisms by which trypanosoma affect the thyroid functions are not completely understood. However, in the infected calves release of cytokines could inhibit the thyroid hormone production (Kahl et al., 2002). Bartalena et al. (1998) indicated that all steps of thyroid hormones synthesis may negatively be affected by cytokines.

Increased serum lipid, hypertriglyceridemia and hypercholesterolemia were reported in experimentally infected rats with *T. brucei* (Igbokwe et al., 2009). Rouzer and Cerami (1980) showed that rabbits infected with *T. brucei* developed a hypertriglyceridemia which could be due to defective plasma triglyceride degradation that probably cause free fatty acid unavailable for importation into hepatocytes despite of serum triglyceride elevation. While, Adamu et al. (2009) showed that *T. brucei* in pigs significantly reduced the serum levels of cholesterol and triglycerides. The results of the present study showed a significant increase in the serum triglyceride in the infected camels but no change was observed in the serum cholesterol concentration. From the present study, it may be concluded that subclinical infection of camels with *T. evansi* can also affect some biochemical parameters, but these variations were very limited compared with clinical cases.

Acknowledgments

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REFERENCES


