Dietary Camel Liver Poisoning in Domestic Cats

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Abstract: Camel liver is a a traditional Sudanese treat that occasionally is associated with toxicity when consumed raw. This study is intended to throw light on the possible mechanisms and factors pertaining to such a toxicity. About 18 cats, Felis domestica, free from disease were divided into 6 groups; 5 groups being fed different treatments of camel liver while the control group was fed ox meat. Variations likely to occur due to seasonal differences were probed. Significant blood biochemical changes as well as tissue changes, suggestive of hepatorenal toxicity were observed in the groups fed camel liver of the rainy season and camel liver treated with ox bile. Results open up a vista for further investigation.

Key words: Camel liver, toxicity, mechanisms, cats, meat, blood

INTRODUCTION

The camel in the Sudan is considered an important meat source. On top its liver is traditionally eaten raw without cooking or frying as Mararah. Bovine, caprine and ovine bile is usually added as a condiment to Mararah which consists of raw liver, lung and rumen slices with bile and hot spices. The consumption of camel liver is sometimes associated with toxicity, it is traditionally believed that it may be detrimentally toxic when eaten mixed with the bile of the ox. People in Darfur and Butana avoid eating camel liver during and immediately after the rainy season. They claim it is toxic during this season. This is apart from the effects caused by the consumption of ox bile. In a recent study about the dietary effects of cattle bile juice on broiler chicks performance, a significant drop in the weight gain and food conversion ratio was observed in the broiler chicks, significant pathological changes were also revealed (Ahlam, 2002).

A wide array of new tools is now available for toxicity testing which has the capacity to greatly increase the knowledge of the complex systems under investigation. Alternatives to traditional animal models are not truly competing alternatives but rather additional means for handling toxicology's perplexing problems. The challenge is to use the knowledge and tools wisely as a complement to other approaches (Skalsrew, 1993). The main questions concerning the use of alternatives to traditional animal models are: how do we extrapolate from an in vitro system to an in vivo system (i.e., how do we relate effects in single cells to complex interactions in whole animals)? how do we use available in vitro and in vivo data to design better experimental approaches? and how do we predict potential biological effects from the chemical structure of a substance? (Goldberg, 1993).

In the present investigation cats were chosen as model animals for the dietary testing of camel liver. Mac Donald et al. (2000) reported that of all carnivores, the felids are the most specialized meat eaters, the cat has enhanced or eliminated certain biochemical mechanisms to deal with a diet rich in protein and fat but with little or no carbohydrate. These facts subtracted possible collateral short comings of administering the treatment as the only diet along the course of the experiment and fingered out the afterwards signs as the product of the treatment.

MATERIALS AND METHODS

Animals: About 18 cats, Felis domestica, both sexes, 6-24 months old were used.

Collection of samples
- About 500 g samples of liver, from different lobules were collected from 10 camels in the dry season and 26 camels in the rainy season. Each sample was placed in a clean enumerated plastic bag
- Fresh ox bile was collected from different gall bladders from the slaughterhouse of the Veterinary Meat Processing Centre, Kuku and kept in a freezer
Blood collected from cats by recurrent tarsal vein puncture using disposable syringes
- Into dry clean bottles for enzyme assays and blood chemistry
- Into dry clean bottles containing the anticoagulant Ethylene Diamine Teta Acetic Acid (EDTA) as described by Adam et al. (1974)
- Liver, heart, kidneys intestine and stomach specimens were collected immediately after the slaughter of each cat and fixed into 10% neutral buffered formol-saline in enumerated bottles

Experiments
Experiment 1: Pilot experiment have been carried on monkeys, white lab. rats and cats.

Experiment 2: About 6 groups each of 3 cats were randomly selected. The cats were caged in iron-mesh cages separately. The monitoring period before carrying the experiment was 1 month. The groups I-VI were then fed the following treatments, respectively; camel liver treated with ox bile, camel liver, ox meat treated with ox bile, ox meat, camel liver collected in the rainy season treated with ox bile and camel liver collected in the rainy season. Each cat was fed 150 g days⁻¹ for 10 days.

Haematological methods: RBCs, total WBCs, Hb concentration and PCV were studied according to the method of Schalm (1965).

Chemical methods: Serum samples were analysed for cholesterol (Trinder, 1952), total protein (Weichselbaum, 1946), albumin (Doumas et al., 1971), urea (Evans, 1968), sodium and potassium (Varley, 1976), GOT and GPT (Reitman and Frankel, 1957) and ALP (Varley, 1976).

Histopathological methods: Formaline fixed specimens of liver, kidney, heart, stomach and intestine were embedded in paraffin wax, sectioned at 5 μm and stained with Haematoxylin and Eosin (H and E) (Harvey and Obied, 1974; Adam et al., 1974). The slides were photographed under light microscope, using powers X₁₀, X₀ and X₀, the film developed and clear pictures obtained.

RESULTS AND DISCUSSION

Ante-mortem examination results: At the end of the experiment none of the cats tested died. No significant weight loss or gain was determined, no abnormal clinical signs were seen other than a gradually increasing in appetite. Significant behavioral signs of nervousness, restlessness and circling were observed, deep coarse mewing intorations were also observed.

The results of the post-mortem examination: The cats were slaughtered at 10 days of the experiment. The results of the necropsies performed on the cats from the test Groups I and V have revealed that the livers were enlarged, heavy, pale, gressy, two livers were mottled and one liver was pigmented. The gall bladders were distended with dark green bile, the stomachs were moderately filled with undigested camel liver slices. The kidneys were congested and both kidneys in one cat have shown renal sac haemorrhage. No lesions were observed in the tissues of the cats of the Groups II, III, IV and VI (Table 1). The serum chemistry results are shown in Table 2.

<table>
<thead>
<tr>
<th>Group No.</th>
<th>Group treatments</th>
<th>Hb g/100 mL</th>
<th>Hb (%)</th>
<th>WBC*10³/μm</th>
<th>RBC*10⁶/μm</th>
<th>PCV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Camel liver and ox bile</td>
<td>9.37±0.47</td>
<td>63.67±3.006</td>
<td>9.77±0.75</td>
<td>12.56±1.19</td>
<td>28.00±2.65</td>
</tr>
<tr>
<td>II</td>
<td>Camel liver</td>
<td>8.87±0.25</td>
<td>61.00±1.000</td>
<td>9.75±0.44</td>
<td>12.33±0.35</td>
<td>32.00±2.65*</td>
</tr>
<tr>
<td>III</td>
<td>Ox bile and natural food</td>
<td>8.83±0.32</td>
<td>60.33±1.50</td>
<td>9.37±0.32</td>
<td>11.46±0.33</td>
<td>27.67±1.53</td>
</tr>
<tr>
<td>IV</td>
<td>Natural food</td>
<td>8.33±1.16</td>
<td>57.00±7.816</td>
<td>8.97±1.92</td>
<td>11.77±1.28</td>
<td>24.33±3.79*</td>
</tr>
<tr>
<td>V</td>
<td>Rainy season, camel liver and ox bile</td>
<td>10.03±1.53</td>
<td>68.67±10.26</td>
<td>5.05±2.25*</td>
<td>7.38±0.29*</td>
<td>25.67±3.79</td>
</tr>
<tr>
<td>VI</td>
<td>Rainy season and camel liver</td>
<td>8.70±1.71</td>
<td>59.67±12.06</td>
<td>7.95±3.33</td>
<td>7.50±0.46</td>
<td>24.00±4.36*</td>
</tr>
</tbody>
</table>

Table 2: Some serum constituents in cats fed camel liver and ox bile and in control group

<table>
<thead>
<tr>
<th>Group No.</th>
<th>Group treatments</th>
<th>Potassium mg/L⁻¹</th>
<th>Sodium mg/L⁻¹</th>
<th>Cholesterol mg dl⁻¹</th>
<th>T protein g/100 mL</th>
<th>Albumin g/100 mL</th>
<th>SGOT U/L</th>
<th>SGPT U/L</th>
<th>Alkaline phosphatase R.A.U.</th>
<th>Urea mg/100 mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Camel liver and ox bile</td>
<td>3.32±0.15</td>
<td>109.67±3.86</td>
<td>44.33±12.83</td>
<td>7.77±0.01</td>
<td>3.33±0.12</td>
<td>60.00±5.29</td>
<td>7.67±2.08</td>
<td>8.83±3.15</td>
<td>23.00±1.0</td>
</tr>
<tr>
<td>II</td>
<td>Camel liver</td>
<td>3.17±0.15</td>
<td>113.67±7.51</td>
<td>47.33±7.49</td>
<td>7.77±6.12</td>
<td>3.57±0.77</td>
<td>56.00±6.44</td>
<td>9.67±0.58</td>
<td>86.67±1.53</td>
<td>22.00±2.0</td>
</tr>
<tr>
<td>III</td>
<td>Ox bile and natural food</td>
<td>3.16±0.17</td>
<td>103.67±4.73</td>
<td>52.33±6.04</td>
<td>7.63±6.02</td>
<td>3.60±0.10</td>
<td>50.00±3.61</td>
<td>7.67±0.58</td>
<td>8.83±1.15</td>
<td>25.00±1.0</td>
</tr>
<tr>
<td>IV</td>
<td>Natural food</td>
<td>2.93±0.81</td>
<td>107.00±4.36</td>
<td>87.00±5.35</td>
<td>7.83±6.35</td>
<td>3.80±0.55</td>
<td>46.67±2.13</td>
<td>9.67±1.15</td>
<td>87.33±1.53</td>
<td>19.67±1.0</td>
</tr>
<tr>
<td>V</td>
<td>Rainy season, camel liver and ox bile</td>
<td>2.53±0.32</td>
<td>126.33±9.87*</td>
<td>136.00±2.65</td>
<td>8.10±0.27</td>
<td>4.2±0.17</td>
<td>35.50±5.69</td>
<td>20.00±5.00</td>
<td>83.33±0.58*</td>
<td>98.33±25.11*</td>
</tr>
<tr>
<td>VI</td>
<td>Rainy season and camel liver</td>
<td>2.67±0.67</td>
<td>132.67±8.74*</td>
<td>131.00±17.25</td>
<td>7.97±0.02</td>
<td>4.2±0.17</td>
<td>36.67±5.77</td>
<td>23.00±9.54</td>
<td>83.67±3.21*</td>
<td>108.67±29.77*</td>
</tr>
</tbody>
</table>

Values within a column represent the mean:SEM. *Significant differences (p<0.05) between the test and the control groups.
Histopathological findings: under microscopy the livers of the test groups 1 and 5 exhibited lymphocytic infiltration (Fig. 1) dilated and deforming blood vessels, numerous foci of hepatocellular degeneration and highly fatty cytoplasmic vacuolation of the hepatocytes (Fig. 2) oedema and cytoplasmic vacuolation of the hepatocytes (Fig. 3) and congestion of the hepatic blood vessels, oedema and vacular degeneration of the hepatocytes (Fig. 4). The kidneys of the cats in group 1 and 5 exhibited focal segmental glomerulonephritis (Fig. 5), constricted blood vessels (Fig. 6) constricted blood vessels with dilated tunica musculatis (Fig. 7) and tabular nephrosis (Fig. 8). No such changes were observed in the control groups.

The present results are those of acute hepatorenal toxicity, signs of camel liver toxicity in humans, generally are those of acute abdominal upset Hag (2004). Acute abdominal toxicity is the feature in common in both cases.

Haematological changes in this experiment are similar although, not identical, to those determined in chicks after experimental Ricinus communis poisoning Adam and El Badawi (1992) reported that there were significant differences (p<0.05-0.001) in Hb, PCV and RBC between the test and the control groups. No significant differences in MVC or MCHC were observed between the test and
The present serum chemistry and enzyme assays are similar to those monitored for coumaphos toxicity in Nubian goats, Nassar et al. (1997) observed significant increases in SGPT and ALP and in the concentrations of urea as well as histopathological findings in the liver and kidneys highly suggestive of hepato-renal injury. In a study of Ricinus communis poisoning in chicks Adam and El Badawi (1992) observed significant differences in the value of SGOT and fluctuation of sodium and phosphorous levels within the normal range.

The present histopathological lesions are similar to those observed in chicks poisoned by dietary propoxur (Adam and Osman, 1997), Nubian goats intoxicated with diazinon (Barri et al., 1998) Nubian goats intoxicated with amitraz (Abdalla et al., 1971), chicks after Ricinus communis poisoning although, the chicks showed in addition, the involvement of the intestinal lamina propria (Adam and El Badawi, 1992) and in goats after coumaphos poisoning with the further involvement of the cardiac, stomach intestinal and pulmonary tissues (Nassar et al., 1997).

CONCLUSION

The ingestion of raw camel liver treated with ox bile and raw liver collected in the rainy season induced blood and tissue changes in the cats diagnostically definitive of toxicity, similar changes were not observed in controls. It may be justifiable to conduct future research with a view to further elucidation of the toxicity factors and mechanisms.

REFERENCES


