FURTHER STUDIES ON BOVINE LIPOMATOSIS AS ENZOOTIC HERD PROBLEM IN EGYPT

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Introduction

Bovine lipomatosis abdominal fat necrosis in cattle generally characterised by
the presence of hard irrugular masses of necrotic fat tissue in abdominal cavity,
specially in mesenteric, pirenal and intestine. The disease has been reported from
several countries (10, 3, 4, 15, 20).

A review of literature on he aetiology and pathogenesis of fat necrosis are
incompletly understood. Genetic influences were suggested (7), disturbance of
circulation(14) and hyperthermia was reported as a possible cause of fat necrosis
(1). Additional causes were mentioned, hromonal disturbence (6), pressure on the
tissue (12) and composition of fatty acids and disturbence in its metabolism (18).

Fat necrosis is thought to initiate a change in the composition of fat within
normal fat cells, fat composition can change as a result of number of factors
pancreatitis (18), feeding long chain saturated fatty acids (19) and depression of
thyroid function (8). Liberation of glycerol which is soluble and carried the short
chain saturated fatty acids and unsaturated fatty acids are liquid at body tem­
perature, while longer chain saturated fatty acids are solid (9). Yellowish color
assocaited with fat necrosis is due to polymerization of unsaturated fatty acids
(9). Pathogenesis of fat necrosis is closely related to Vit. E, selenium deficiency
(10). Success trials for treatment of fat necrosis were applied using
isoprothialane (17).

The purpose of this study is to carry out further investigation on the biochemi­
cal aspect of bovine fat necrosis and determination the composition of fatty acids
in necrosed fat as well. Experimental diagnostic therapy using Natrium selenite for
treatment of clinical cases of fat necrosis is also aimed.

Materials and Methods

Dairy herd: A herd of 250 local and cross-breed cows were kept for milk production in
Al-Awamer dairy farm. This farm is located 10 Km east of Assiut in a new cultivated
land. Individuals of the herd showed signs of fat necrosis in various degrees and
forms.

Study design:
A. Clinical and biochemical study: Sixty four cow were selected from the herd for
this purpose, and classified according to their clinical status into the following
groups:
  Group I (Healthy cows): Ten healthy cows were included in this group. All cows
  were clinically healthy without any signs of bovine lipomatosis.
  Group II (Early stage of fat necrosis): A group of 39 cows showed signs of fat
  necrosis in early stage.
  Group III (Advanced stage of fat necrosis): A total number of 18 cows were
  included in this group.
  Group IV (Chronic form of fat necrosis): Seven aged cows were involved in this
group. All cows were emaciated and had a history of fat necrosis.

B. Experimental diagnostic therapy using natrium selenite: This part of study
includes 5 cows in the early stage of the fat necrosis which selected from Group II,
in addition to other 5 cows selected from Group VI, those resembling the chronic
form of fat necrosis. Natrium selenite was used as diagnostic therapy and given
orally mixed with ration to diseases cows. Natrium selenite administered orally to provide the cows a dose 5 mg/cow/day over a period of 6 months. Close observation and clinical examination were carried out.

C. Blood samples and biochemical analysis: Blood samples were collected, and blood serum was separated according to the ordinary methods of hematology. Biochemical essay includes the estimation of potassium (K), sodium (Na), chloride (Cl), Magenessium (Mag), Calcium (Ca), phosphorus (P), glucose, amylase, albumin, triglycerides, total protein, total lipids and selenium. All above mentioned parameters were determined using diagnostic kits supplied by Boehringer Mannheim, Germany. Biochemical analysis were carried out in the diagnostic laboratories of Klinik fur Klauentiere-krankheiten und fortpflanzungskunde der Freie Universital, Berlin, Germany.

D. Tissue samples: Samples of necrotic fat were collected from slaughter animals. Tissue samples were taken from intestinal, mesentric and perirenal fat, and used for fatty acids estimation. Hplc - chromatography was used for this purpose, and the analysis was carried out in Institute fur Biochemie der Freie, Universtat Berlin, Germany.

E. Clinical examination: All cows were subjected to close clinical examination which includes appetite, defecation urination, milk yield, body condition score and rectal examination.

Results

Herd history and clinical findings: Bovine fat necrosis as a herd problem was investigated over a period of 4 years. The ratio of diseased cows to total numbers of the herd was about 30%. Aged cows were more susceptible than young ones, especially in chronic form of fat necrosis. The most affected cows were over 7 years old. Neither heifirs nor bulls were involved in the record of such herd as cases of fat necrosis. Individuals of such herd were related to local and cross breed cattle. Ration provided to cows was concentrate and rice straw in summer and alfa-alfa as green feeder in winter. Water supply was ground water. Diseases cows were examined clinically, selected according to their clinical status, and classified into three groups according to associated clinical signs of fat necrosis.

In Table (1) the common clinical findings of examined groups were summerised. Post-slaughter findings revealed a characteristic lesions in each stage of fat necrosis. Early stage of the disease was characterised by the presence of well circumscribed irregular areas of necrosis. Some time much more denser than normal opaque grayish-red colour, surrounded by a well demarkated white zone containing chalk-like calcification.

Results of experimental diagnostic therapy with Na. selenite: Cows in the early stage of fat necrosis showed marked improve in their condition after 6 weeks from start with therapy. Body condition score and milk production showed no alteration. In the last two months of the experiment there was a marked softening of subcutaneous fat tissue specially at areas of back and base of the tail. Two cows of a treated groups showed complete disappearance of subcutaneous nodules, the rest of cows showed no recognisable changes. Rectal examination revealed a marked reduction in the size of masses present in the abdominal cavity with detectable softening on palpation. Three cows showed these findings but the rest indicated no visible alteration in the size and consistency of masses.

Results of biochemical essay: Results of biochemical analysis were illustrated in Table (2). The data indicated no significant changes in the mean values of all estimated parameters in between the examined groups, with exception the mean values of Triglycerides, total lipids and phospholipids which markedly increased in all

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Results of fatty acids essay: The values in healthy and necrotic fat for each fatty acid were as follows: myristic acid (14:0) was 3.2% and 2.2%, palmitic acid 24.8% and 22.5%, palmitoleic acid 2.3% and 0.6%, Margaric acid 0 and 1.4%, stearic acid 29.4% and 34.1%, olic acid 35.5% and 28.6%, linolic acid 2.0% and 2.3% and linolenic acid 0 and 0.5% respectively.

Discussion

Bovine fat necrosis was investigated as an enzootic herd problem in local and cross-breed cows at Assiut-Egypt (8, 9, 13). Throughout long standing clinical observations of affected herd, bovine fat necrosis was classified according to the related clinical signs into early, advanced and chronic stages, no absolute clinical lines of demarcation between these stages, but clinical diagnosis and rectal examination could be help in identification of each stage specially between early and chronic form of the disease. General signs of fat necrosis which recorded in this study were similar to the published findings (14, 16, 5, 4, 11, 17, 9).

The analysis of fat samples in the present study indicates that fatty acids composition in necrotic fat were characterised by a marked increase in stearic acid (C:18:0) concentration, a significant decrease in olic acid (C:18:1) level in addition to a slight increase in the level of palmitic acid (C:16:1). Normally mesenteric and perirenal fat tissue contain a higher proportion of saturated fatty acids particularly stearic acid. This degree of saturation in mesentric and perirenal fat depots could explain why a greater portion of necrotic fat lesion were found in mesenteric and perirenal fat depots. However, the compositional changes causes the affected tissue to be recognized as a foreign body, that the appearance of fibroblasia and collagen associated with necrotic fat. Similar observation were previously reported (1, 18, 19). Biochemical analysis of blood serum in healthy and diseased groups revealed a significant increase in mean values of triglycerides, total lipides and phospholipids specially in cows showed the signs of chronic fat necrosis, these findings incitaded a presence of disturbance of fat metabolism in cows affected with fat necrosis. Thus an abnormality of lipid metabolism may responsible for the occurrence of fat necrosis. Such findings were agreed with other results on fat necrosis (18, 19, 17).

Results of selenium analysis indicated a slight drop in selenium level in cows of Group III (advanced fat necrosis), while a significant drop of selenium value in cows of chronic stage of the disease. No changes were observed in the early stage of the disease, if compared with the healthy group. Marked drop in selenium level in cows showing chronic fat necrosis could be interpret as selenium deficiency, however selenium is considered a component of enzyme-glutathione peroxidase and its role in the protection of cells by destroying oxidizing agent such as hydrogen peroxide and lipid peroxidase, thus inadequate level of selenium or selenium deficiency resulting in degeneration and necrosis of fat cells (2, 10).

Experimental diagnostic therapy using natrium-selenite: Oral administration of natrium-selenite mixed with ration for cows in early and chronic stage of fat necrosis showed variable degree of response. A marked improvement was observed in 60% of cows in early stage of the disease, improvement was in form of decrease in size of fat masses in the abdominal cavity and subcutaneous depots as well. Palpable softening of hard masses was detected, subcutaneous fat nodules were disappeared partially or completly.

On the other hand cows in chronic fat necrosis showed no response to natrium selenite therapy throughout the period of treatment. Variations in response to natrium selenite do not exactly understood, but results could be declaired that the role of selenium in the early stage of the disease can protect the fat cells from further oxidative processes that finally followed by degeneration and necrosis of
fat cells. However the already degenerated and necrotic fat cells (chronic stage) did not respond totally to natrium therapy.

Summary

Bovine lipomatisos as enzootic problem was observed in cows of a native breed herd. The clinical signs were in the form of obesity, body condition score (4-6); drop in milk yield; marked deposition of subcutaneous fat at the base of the tail and the entrance of chest. Chronic cases were characterized by severe emaciation; loss of appetite; long standing diarrhea; recurrent tympany. A marked hyperlipemia were observed in all groups of animals. Phosphlipids levels were elevated in cows in the advanced stage of the disease and also in chronic cases. Triglycerides levels were only increased in chronic cases. Amylase enzyme-level was drop in chronic cases of bovine lipomatosis. Both groups of advanced and chronic lipomatosis showed a marked decrease in selenium level. Fatty acids determination in necrosed fatty tissues indicated a marked increase in the concentration of stearic acid (18:0) 34%; while olic acid (18:1) showed decrease in its level 28.6%. Diagnostic therapy using Natrium selenite in dose of 5 mg/cow/day was supplied to the animals with the ration over a period of 6 months. In 70% of treated cows in the early stage of the disease were improved and no hard masses were palpated rectally, while the rest of cows in this stage showed variable degree of necrosis. On the other hand no detectable changes were observed in cows treated in the chronic phase (fat necrosis) of bovine lipomatosis.

Zusammenfassung

In einer Rinderherde in Neulandgebiet obergypertens traten über mehrere Jahre hinweg gehauft fettgewebenekrosen am intraabdominalen (Gekrose-) fett auf. Da bei waren fast ausschließlich tiere einer boden standigen rasse (native breed) und misch Rasse (cross breed) betroffen, während die unter gleich Bedingungen gehaltenen schwarzbunten Rinder nicht erkrankten. Die unfangrichten klinisch-chemischen und morphologischen untersuchungen deuten auf eine storung des fettsaurenmusters des fettgewebs hin. Wahren auf selen substitution im futter (Na-Selenite) eine gewiss Besserung und Abnahme der Krankheitsfrequence eintrat.

Resume

La maladie"Fat Necrosis" est apparue chez les bovins locaux a Assiout (Egypte). Les symptomes de cette maladie se sont caracterises par l’apparition de masses grasses a l’interieur de l’abdomen autour du rein et des intestins. L’analyse due sang a decele une presence d’anomalies dans la composition et le metabolisme des acides gras. Le diagnostic therapeutique a base SELENIUM pur le cheptel malade, a revele que ce remede a joue un role dans la cause de la maladie "Fat Necrosis" chez les bovins.

References


Table (1) Clinical status and associated clinical findings of examined groups of bovine fat necrosis

<table>
<thead>
<tr>
<th>Animal's group</th>
<th>N</th>
<th>Clinical status</th>
<th>Body condition</th>
<th>Appetite</th>
<th>Milk production</th>
<th>Other associated clinical signs and rectal findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I 10</td>
<td>Healthy cows</td>
<td>2.5-4</td>
<td>Normal</td>
<td>Normal</td>
<td>All cows were clinically healthy without apparent signs indicating fat necrosis.</td>
<td></td>
</tr>
<tr>
<td>Group II 39</td>
<td>Cows in early stage of the disease</td>
<td>5-7</td>
<td>Normal</td>
<td>Decrease</td>
<td>Marked deposition of subcutaneous fat tissue in back chest and around the tail. Rectal examination revealed large amount of soft fat.</td>
<td></td>
</tr>
<tr>
<td>Group III 18</td>
<td>Cows in advance stage of disease</td>
<td>3-5</td>
<td>Depressed</td>
<td>Marked drop</td>
<td>Slight reduction subcutaneous fat. Rectal examination revealed the presence of moderate sized hard masses.</td>
<td></td>
</tr>
<tr>
<td>Group IV 7</td>
<td>Chronic form of fat necrosis</td>
<td>1.5-3</td>
<td>Inappetence</td>
<td>No milk production</td>
<td>General weakness, marked emaciation cows until to move, constant diarrhea tympany. Rectal examination revealed large, hard and irregular masses in abdominal cavity.</td>
<td></td>
</tr>
</tbody>
</table>

N = number of case
### Table (2) Mean values of measured parameters for biochemical essay in healthy and diseased groups

<table>
<thead>
<tr>
<th>Animals groups</th>
<th>N</th>
<th>K (mmol/L)</th>
<th>Na (mmol/L)</th>
<th>Cl (mmol/L)</th>
<th>Mag. (mmol/L)</th>
<th>Ca. (mmol/L)</th>
<th>P (mmol/L)</th>
<th>Glucose (mmol/L)</th>
<th>Amylase (U/L)</th>
<th>Albumin (g/L)</th>
<th>Trigl. (mmol/L)</th>
<th>T. protein (g/L)</th>
<th>T. lipids (mmol/L)</th>
<th>Phosp. (mmol/L)</th>
<th>Sei (umg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Healthy</td>
<td>10</td>
<td>4.625</td>
<td>148.7</td>
<td>103.42</td>
<td>0.371</td>
<td>2.185</td>
<td>1.371</td>
<td>2.228</td>
<td>54.63</td>
<td>33.83</td>
<td>3.2</td>
<td>73.14</td>
<td>3.361</td>
<td>2.461</td>
<td>54.7</td>
</tr>
<tr>
<td>S.E</td>
<td></td>
<td>±0.248</td>
<td>±3.26</td>
<td>±14.58</td>
<td>±0.373</td>
<td>±0.146</td>
<td>±0.399</td>
<td>±13.03</td>
<td>±3.42</td>
<td>±0.720</td>
<td>±3.52</td>
<td>±1.061</td>
<td>±0.355</td>
<td>±2.30</td>
<td></td>
</tr>
<tr>
<td>Group II</td>
<td>39</td>
<td>5.022</td>
<td>148.13</td>
<td>79.72</td>
<td>0.811</td>
<td>1.861</td>
<td>2.075</td>
<td>1.315</td>
<td>40.15</td>
<td>34.78</td>
<td>1.225</td>
<td>81.28</td>
<td>7.044</td>
<td>2.902</td>
<td>45.8</td>
</tr>
<tr>
<td>Early stage</td>
<td></td>
<td>±0.5322</td>
<td>±5.91</td>
<td>±5.22</td>
<td>±0.143</td>
<td>±0.556</td>
<td>±0.449</td>
<td>±1.780</td>
<td>±12.62</td>
<td>±3.58</td>
<td>±0.179</td>
<td>±6.17</td>
<td>±1.99</td>
<td>±1.535</td>
<td>±1.63</td>
</tr>
<tr>
<td>Group III</td>
<td>18</td>
<td>5.233</td>
<td>148.13</td>
<td>94.41</td>
<td>0.959</td>
<td>2.08</td>
<td>2.233</td>
<td>0.638</td>
<td>65.04</td>
<td>36.18</td>
<td>2.562</td>
<td>81.44</td>
<td>8.578</td>
<td>4.558</td>
<td>41.7</td>
</tr>
<tr>
<td>Advanced</td>
<td></td>
<td>±0.720</td>
<td>±8.19</td>
<td>±2.12</td>
<td>±0.174</td>
<td>±0.619</td>
<td>±0.501</td>
<td>±13.61</td>
<td>±3.44</td>
<td>±1.62</td>
<td>±5.59</td>
<td>±2.213</td>
<td>±3.642</td>
<td>±2.83</td>
<td></td>
</tr>
<tr>
<td>Group IV</td>
<td>7</td>
<td>5.213</td>
<td>145.17</td>
<td>95.78</td>
<td>0.604</td>
<td>1.67</td>
<td>2.62</td>
<td>0.842</td>
<td>19.60</td>
<td>32.88</td>
<td>12.62</td>
<td>70.93</td>
<td>5.66</td>
<td>12.80</td>
<td>38.1</td>
</tr>
<tr>
<td>Chronic</td>
<td></td>
<td>±0.462</td>
<td>±7.28</td>
<td>±4.19</td>
<td>±0.167</td>
<td>±0.61</td>
<td>±0.71</td>
<td>±0.210</td>
<td>±8.33</td>
<td>±4.10</td>
<td>±8.47</td>
<td>±2.75</td>
<td>±1.43</td>
<td>±2.63</td>
<td>±2.04</td>
</tr>
</tbody>
</table>

N = number of animals
X = Mean
S.E = Standard errors