Clinical, Hematological and Biochemical Studies on Wool Eating Syndrome in Sheep

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ABSTRACT:

Key words: Wool eating syndrome, trace elements deficiencies, pica, alopecia.

This study was carried out on 20 balady sheep belonged to a governmental farm in Alexandria governorate. Ten sheep were suffered from eating of each other's wool (wool eating habit), weight loss, apparent parakeratosis and different degrees of alopecia. The other 10 sheep were clinically healthy and used as control group. Skin scraping and fecal examination showed that the affected sheep were free from mite infestation and internal parasites. Hematological examination of wool eating sheep showed a significant decrease in hemoglobin and hematocrit without significant changes in erythrocytic count, total leucocytic count, MCV, MCH and MCHC than in healthy animals. Biochemical analysis of serum of affected animals showed significant decrease in serum copper, zinc and iron, total protein and a significant increase in ALP activity than in healthy animals. Non-significant changes in serum calcium, phosphorus, albumin, glucose, urea, creatinine, ALT and AST activity between diseased and healthy animals. It could be concluded that syndrome of wool eating in sheep is caused mainly by multiple trace element deficiencies particularly copper, iron and zinc.

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1. INTRODUCTION

Pica or altrophagia is a metabolic disorder characterized by different symptoms such as surface licking, soil and non-food object eating (wool, hair, bone, wall, etc) (Abdelrahman et al., 1998). Wool eating or wool plucking as a type of altrophagia occurs in sheep, especially in lambs. It is considered that a deficiency of minerals, such as calcium, phosphorus, sodium chloride, copper, zinc, manganese, cobalt, as well as vitamin or protein deficiency might be the cause of the disease (Aytekin and Kalinbacak 2008; Youde, 2002). Nelson et. al., (1984) reported that obvious parakeratosis and hyperkeratosis in animal skin, alopecia and severe wool eating habits were present in two sheep flocks and one goat flock with zinc deficiency which quickly cause alopecia or brittle wool after seventeen days of the experiment, while Akgül et al (2000) showed that clinical signs and abnormalities present in lambs with wool eating disease include growth retardation, diarrhea, poor appetite, salivation, abnormal hooves, swollen joints, stiff gait, hair loss, parakeratosis and compromised disease resistance. The clinical signs varied with age of the animal, the younger sheep and lambs are more affected while the adults are less affected (Suliman et al., 1988).

Trace elements deficiency, in particular copper, zinc and cobalt have been incriminated in etiology of alopecia and wool eating habit in sheep (Meyer and Losh, 2002). Fahmy et al. (1980) determined that serum copper, zinc, manganese and iron levels significantly decreased in sheep which had alopecia and wool eating habit while Baysu et al. (1973) who examined inorganic phosphorus, calcium, potassium, sodium and chloride in blood serum of lambs with pica in Sivrihisar and found that inorganic phosphorus values in the diseased lambs were low.

Akgül et al (2000) reported that serum iron, manganese, phosphorus, calcium, sodium and chloride values of diseased sheep were not significantly different in diseased animals and the control animals. On the other hand, serum zinc and copper values of the diseased animals were significantly lower than the controls.

Suliman et al (1988) reported that serum globulin, total protein, ALP, zinc, copper levels were decreased in wool eating sheep and lambs than in normal ones, while LDH level was elevated and albumin level was normal.

Youde (2002) found that serum sulfur and copper levels decreased, whereas serum Ca, P, Fe, Mn, Zn, Co, Mo, and Se levels were at normal levels in their research on wool eating sheep and goats in China.

So the aim of this study is to reach a correct diagnosis of the syndrome of wool eating in sheep.
depending on the clinical, hematological and biochemical examination of the affected animals.

2- MATERIAL and METHODS:

2.1. Animals:
The present investigation was carried out on ten diseased Balady sheep (2-3 years old) from a flock of sheep belonged to a governmental farm at Alexandria Governorate. Diseased individuals showed signs of eating each other wool (wool eating habit), decreased body weight, parakeratosis and various degrees of alopecia. Another ten apparently clinically healthy Balady sheep related to another flock at the same locality were used as a control group.

2.2. Fecal examination and skin scraping:
Fecal examination and skin scraping were preformed according to method described by (Kelly, 1984) to ensure absence of mite and internal parasites infestation.

2.3. Hematological analysis:
One ml of blood was withdrawn from jugular vein into heparinized test tubes. Hemoglobin concentration (Hb), erythrocytic (RBCs) and total leucocytic (WBCs) counts and packed cell volume were estimated by using of fully automated veterinary hematology analyzer, Exigo, Boule medical AB., Sweden in the central laboratory Faculty of veterinary medicine, Alexandria University

2.4. Biochemical analysis:
Five ml of blood were withdrawn by puncturing the jugular vein, into test tube without anticoagulant, left to clot and centrifuged for serum separation. Commercially available diagnostic kits were used for colorimetric determination of serum calcium (Glider and King, 1972), inorganic phosphorus (El-Merzabani et al., 1977), total proteins (Doumas et al., 1981), albumin (Gindler and westgard,1973), glucose (Trinder,1969 ), ALT and AST (Reitman and Frankel, 1957) , ALP (Berth and Delanghe, 2004), urea (young,2001), creatinine (Newman and price,1999). Serum iron, zinc and copper were estimated by atomic absorption spectrophotometer AAS, N as described by Meret and Henkin (1971).

2.5. Statistical Analysis:
Data collected were subjected to analysis by T-independent student test to assess significant differences between groups with the aid of SAS (2004).

3. RESULTS:
Clinical examination of diseased animals showed wool eating habit, unthriftiness, wool abnormalities including steely wool appearance, parakeratosis and some degree of alopecia. In addition to poor productivity, pale mucous membranes and depraved appetite (Fig.1 and 2).
Fecal examination and skin scraping of affected animals revealed absence of any internal or external parasitic infestations. Hematological analysis of blood samples of affected sheep showed a significant decrease in hemoglobin (Hb) and PCV values (P<0.05) than the normal ones but there were no significant changes in erythrocytes and total leucocytes counts, MCV, MCH and MCHC between the diseased and the normal animals (Table 1).

Biochemical analysis of serum of diseased sheep showed a significant decrease in serum copper, zinc, iron and total protein as compared with control ones. There was a significant increase in ALP activity without significant changes in serum levels of ALT and AST. Serum levels of urea, creatinine, calcium, inorganic phosphorus, albumin and glucose showed non-significant changes between affected and control animals (Table 2, 3).

Fig. 1. Showing affected sheep with wool eating syndrome.
Fig. 2. A Sheep showing wool abnormalities as steely wool and wool eating syndrome.

### Table (1): Hematological results in wool eating and control sheep (mean± SE).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control group</th>
<th>Diseased group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>11.13±0.17</td>
<td>9.15±0.18*</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>32.33±0.30</td>
<td>28.39±0.51*</td>
</tr>
<tr>
<td>RBCs (*10⁶/mm³)</td>
<td>10.2±0.23</td>
<td>9.64±0.25</td>
</tr>
<tr>
<td>WBCs (*10³/mm³)</td>
<td>8.97±0.17</td>
<td>8.89±0.17</td>
</tr>
<tr>
<td>MCV(fl)</td>
<td>31.86±0.86</td>
<td>29.56±0.73</td>
</tr>
<tr>
<td>MCH(pg)</td>
<td>10.96±0.31</td>
<td>9.55±0.32</td>
</tr>
<tr>
<td>MCHC(g/dl)</td>
<td>34.42±0.33</td>
<td>32.38±1.05</td>
</tr>
</tbody>
</table>

Symbol * denotes Significant difference from control at (P<0.05)

### Table (2): Serum minerals in wool eating and control sheep (mean± SE):

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Diseased</th>
</tr>
</thead>
<tbody>
<tr>
<td>Copper (µg/ml)</td>
<td>1.77±0.06</td>
<td>0.81±0.03*</td>
</tr>
<tr>
<td>Zinc (µg/ml)</td>
<td>1.14±0.10</td>
<td>0.64±0.04*</td>
</tr>
<tr>
<td>Iron (µg/ml)</td>
<td>132.12±6.58</td>
<td>87.48±3.11*</td>
</tr>
<tr>
<td>Calcium (mg/dl)</td>
<td>8.64±0.20</td>
<td>8.37±0.21</td>
</tr>
<tr>
<td>Phosphorous (mg/dl)</td>
<td>6.39±0.08</td>
<td>6.02±0.13</td>
</tr>
</tbody>
</table>

Symbol * denotes Significant difference from control at (P<0.05)

### Table (3): Some biochemical parameters in wool eating and control sheep: (mean± SE).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control group</th>
<th>Diseased group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total protein (g/dl)</td>
<td>6.42±0.08</td>
<td>5.34±0.08*</td>
</tr>
<tr>
<td>Albumen (g/dl)</td>
<td>3.48±0.12</td>
<td>3.23±0.11</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>68.17±3.11</td>
<td>69.81±3.13</td>
</tr>
<tr>
<td>AST (U/l)</td>
<td>71.93±1.51</td>
<td>75.07±3.77</td>
</tr>
<tr>
<td>ALT (U/l)</td>
<td>27.04±0.99</td>
<td>26.97±1.14</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/l)</td>
<td>118.47±6.43</td>
<td>332.94±21.55*</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>22.36±1.16</td>
<td>24.03±1.12</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.40±0.04</td>
<td>1.36±0.06</td>
</tr>
</tbody>
</table>

Symbol * denotes Significant difference from control at (P<0.05)
4. DISCUSSION

Faulty feeding and trace element deficiencies are the common causes of wool eating in lambs, sheep and goat that leads to economic loss (Akgül et al, 2000). In our study, the clinical signs of affected sheep with wool eating were unthriftiness, parakeratosis, wool abnormalities and different degrees of alopecia. Similar clinical findings were observed by other authors (Ott et al.,1982; Youde et al., 2002). Wool abnormalities usually related to copper and zinc deficiency(Church and Pond,1988) . There were other signs including pale mucous membrane, inappetance and decreased growth rate which similar to those recorded by Radostits et al. (2000); Abd El-Raof; Ghanem (2006).
Results of skin scraping and fecal examination revealed absence of internal and external parasites in the affected animals which agreed with Abd El-Raof and Ghanem (2006) who found absence of internal and external parasites in alopecic sheep. Hematological examination of blood of diseased animals (table 1) showed that HB and PCV values were significantly decreased in diseased animals than control ones, which indicates affection with anemia in wool eating sheep which may be attributed to disturbance in iron metabolism as copper deficiency decreases the absorption of iron, releasing of iron from body stores and utilization in hemoglobin synthesis( Church and Pond,1988) also it agreed with Suliman et al (1988) who reported that HB and PCV values were statistically different before and after treatment in zinc deficiency lambs with wool eating habit. There were non-significant changes in RBCs and WBCs counts between diseased and control animals which agreed Akgül et al (2000) and Hasan et al (2008). There were non-significant changes in MCV, MCH and MCHC levels between healthy and affected sheep which suggested that wool eating sheep were suffered from normocytic normochromic anemia which agreed with Al-Saad et al. (2010) who reported normocytic normochromic anemia in sheep suffering from zinc deficiency.

Biochemical findings of serum minerals of diseased sheep (Table 2) showed significant decrease in level of copper, zinc and iron than normal ones. Those results were similar to those recorded by Fahmy et al (1980) who reported a significant decrease in copper, zinc and iron in sheep with alopecia and wool eating, and Ali (2000) who recorded a significant decrease in levels of serum copper, zinc and iron in sheep showing alopecia. While Akgül et al (2000) found non-significant changes in iron level in diseased and normal sheep. While there was non-significant changes in serum calcium and phosphorus between wool eating and control sheep which agreed with Akgül et al (2000) and Hasan et al (2008).
Regarding to serum total protein and albumin levels (Table3), serum total protein was significantly decreased in affected sheep without significant changes in albumin level which may be attributed to inappetence that affect the diseased sheep. Those results agreed with Akgül et al (2000) and Nelson et al(1984) while it disagree with Abd El-Raof and Ghanem (2006) who showed significant decrease in level of total protein and albumin in sheep suffered from alopecia.
Our results also showed non-significant changes in levels of serum glucose, urea and creatinine in sheep affected with wool eating and control ones. Those results agreed with Akgül et al(2000) and Hasan et al (2008) (Table3)
Liver enzymes examination revealed that Serum level of ALP in diseased sheep was significantly increased while there were non-significant changes in ALT and AST levels. Those results agreed with Hasan et al (2008) who mentioned high ALP values in young and rickety lambs, but this disagree with Abd El-Raof and Ghanem (2006) who reported significant increase in AST and ALT in alopecic sheep as well as with Al-Saad et al (2010) who reported significant increase in AST and ALT in zinc deficient sheep.

5. CONCLUSION
Multiple trace elements deficiencies particularly copper, zinc and iron are the common cause of the wool eating syndrome in sheep and sheep
breeders must pay attention for supplementing such elements in animal ration to avoid occurrence of such condition.

6. REFERENCES


Al-Saad, K.M, AL-Sadi, H.I. , Abdul-Majeed, M.O. 2010. Clinical, Hematological and pathological studies on zinc deficiency (Hypozincemia) in sheep. Veterinary research 3(2) :14-20


