COPPER DEFICIENCY IN FEEDLOT CATTLE

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Abstract

This study was conducted on 25 animals randomly selected from a herd with 40 feedlot cattle, which had poor growth, rough hair coat, and change in the colour of the hair coat. Blood samples were collected from the vena jugularis into the tubes containing Na₂EDTA for the determination of plasma copper concentration and blood profile. Feed samples were also tested for copper, zinc, and molybdenum content. Ten healthy cattle were used as controls from different herds. In the affected group, mean plasma copper level was found to be at 6.43 µmol/L ± 0.01 and copper, molybdenum, and zinc levels in the diet were 15.55 ppm, 3.85 ppm, and 120 ppm, respectively. The mean plasma copper level in control animals was 13.50 µmol/L ±0.06). The difference was statistically significant (P<0.001). The deficiency of copper content may result from especially high molybdenum and zinc levels in the diet. When any finding indicative of this deficiency is observed in a herd, copper status of the animals should be determined. Then, copper and especially molybdenum and zinc concentration in the diet should be examined.

Key words: cattle, copper, deficiency, molybdenum, zinc, growth inhibition.

Nutrition has a significant impact in animal production. Because trace elements that constitute an important part of animal nutrition have unique roles in mammals, their deficiencies can adversely affect animal health. Copper, one of the trace elements, is involved in numerous physiological functions such as haemoglobin formation, iron metabolism, and connective tissue metabolism (5). Copper deficiency is associated with numerous clinical signs, including anaemia, severe diarrhea, weight loss or diminished weight gain, epiphyseal enlargement, change in hair colour, neonatal ataxia, and infertility (7). There are two types of copper deficiency: primary, resulting from a simple deficiency of copper in the diet; and secondary, resulting from the reduction in copper absorption and/or an increase in excretion by antagonistic effects of molybdenum, sulphur, and zinc (7, 12). In this study, copper deficiency in feedlot cattle showing some clinical sings of the disease was investigated.

Material and Methods

The study was conducted on 25 animals randomly selected from a herd with 40 feedlot cattle in Elazig, Turkey, which were demonstrating clinical abnormalities. The affected cattle were fed straw, barley, wheat bran, wet sugar pulp, and concentrate mixture without any copper supplementation. The injections of vitamin A, D₃, and E were administered to the cattle monthly. An ivermectin preparation was administered to the animals two times at a 15 d interval to control parasites. Blood samples were collected from the vena jugularis into the tubes containing Na₂EDTA for the determination of plasma copper concentration and blood profile. Feed samples were also tested for copper, zinc, and molybdenum concentration. Ten healthy cattle from a different herd were used as a control, and the same procedures were performed. The control cattle were of normal appearance and had a different diet than the affected cattle.

Plasma copper concentrations were determined using an atomic absorption spectrophotometer (Unicam 929 AA). A standard ICP-OES (Perkin-Elmer, Optima 2000 DV) analyser system was used to determine copper, zinc, and molybdenum concentrations in dry matter of feed samples.

Blood profiles of both affected and control groups were determined using an automated blood cell counter (Forcyte, Oxford Science).

Statistical analyses were performed using SPSS for Windows (version 10.0; Microsoft). Group means were compared using Student’s t - test.

Results

Medical history revealed that there was an apparent inhibition in the growth rate of the animals for approximately one month. Clinical examination of the animals showed that all cattle in the herd had rough hair coat (Fig. 1), grey hairs around the eyes (spectacle eye) (Fig. 2), and loss of hair pigmentation. All the animals were sluggish in movement.
Table 1
Mean levels of plasma copper, haemoglobin concentrations, haematocrit, and red blood cell count in 10 healthy cattle (control group) and 25 cattle with copper deficiency (affected group)

<table>
<thead>
<tr>
<th></th>
<th>Affected group</th>
<th>Range</th>
<th>Control group</th>
<th>Range</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma copper (µmol/L)</td>
<td>6.43 ±0.01</td>
<td>3.92 – 10.20</td>
<td>13.50 ±0.06</td>
<td>10.99 – 17.27</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Haemoglobin concentration (g/L)</td>
<td>100.4 ±2.20</td>
<td>78.0 – 119.0</td>
<td>109.3 ±3.30</td>
<td>128.0 – 78.0</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Haematocrit (%)</td>
<td>31.08 ±0.9</td>
<td>15.2 - 37.5</td>
<td>32.4 ±1.10</td>
<td>38.7 – 15.2</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Red blood cell count (x10¹²/L)</td>
<td>6.31 ±0.28</td>
<td>3.19 – 9.15</td>
<td>7.70 ±0.34</td>
<td>9.15 – 3.19</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

± SD
In the affected group, the mean plasma copper level was at .43 μmol/L ±0.01. Mean plasma copper level in control animals was 13.50 μmol/L ±0.06. This indicated that a copper deficiency existed in the affected group compared to the controls (P<0.001). Range and mean plasma copper concentrations, red cell counts, haematocrit values and haemoglobin concentrations of the experimental and control groups and P values are given in Table 1. As seen from the Table, blood haemoglobin and RBC counts were significantly different between the affected and control group. Copper, molybdenum, and zinc level in the diet were 15.55 ppm, 3.85 ppm, and 120 ppm in the affected group, respectively.

Discussion

Copper deficiency is a world-wide problem in cattle (3, 8, 11). It may be due to a shortage of intake in the diet (primary), or due to elements such as sulphur, zinc, and especially molybdenum, which reduce copper absorption (secondary) (4, 7, 9, 12). Copper deficiency is characterised by clinical abnormalities such as poor growth, anaemia, epiphyseal enlargement, depigmentation of hair, and rough hair coat (7, 9, 13). Similarly, poor growth, rough hair coat, loss of the hair pigmentation, and spectacle eye, were found as obvious clinical findings in the examined animals. Although the mean red cell count, haemoglobin concentration, and haematocrit value of the herd were found within reference range, these values were apparently lower in some of the animals in the herd.

Since most clinical signs of copper deficiency are generally non-specific, the diagnosis of copper deficiency usually requires the analysis of copper in blood or liver, and diet (4, 9). The diet samples that contain less than 10 ppm of copper in dry matter are marginally deficient. It has been suggested that copper deficiency especially occurs when molybdenum levels exceed 1-3 ppm or when copper to molybdenum ratio falls below 4.5:1 in the diet (4, 9). Excess dietary molybdenum can lead to the formation of cupric molybdates in the rumen that are not absorbed from the intestine (7). High levels of dietary zinc (100-400 ppm) also interfere with copper absorption and reduce hepatic copper concentration (7, 10). In the present study, although copper concentration in the diet was found to be adequate, molybdenum concentration was greater than 3 ppm, copper: molybdenum ratio was lower than 4.5:1, and zinc level was also high. Therefore, copper deficiency determined in the herd may be related to especially high molybdenum and zinc levels in the diet. Even though excess of sulphur or sulphates in water can also affect adversely copper absorption (4, 7), it is unlikely that sulphur could be a possible reason for the herd’s problems, because sulphate levels of water in this region are too low to induce copper deficiency (1).

The primary site of copper reserves is the liver, and there is an association between blood and liver copper concentrations (6, 10). Although blood copper concentrations may not accurately reflect hepatic stores, it has been reported that lower than normal blood copper concentrations indicate a copper deficiency (2). However, high blood copper concentrations do not necessarily indicate that an animal has a sufficient amount of copper, because liver stores must be depleted before plasma copper falls (10). It has been demonstrated that when plasma copper levels in cattle are below 9.42 μmol/L, liver copper concentrations are claimed as extremely low (8). Koh and Judson (6) reported that liver copper concentration of less than 0.4 mmol/kg dry matter is associated with plasma copper concentration of less than 8 μmol/L. When blood samples are used for copper determination, serum or plasma is preferred, and the plasma copper concentration is usually higher than the serum copper concentration. (7, 10). The normal serum copper concentration range is accepted as 10.99-18.84 μmol/L. Serum or plasma copper concentration of 6.28 μmol/L or less is considered as an evidence of clear deficiency. Values of 6.28 to 10.99 μmol/L are marginal (7). Mortimer et al. (9) suggested that serum copper concentrations less than 10.20 μmol/L indicate a potential deficiency. In the present report, mean plasma copper concentration (6.43 μmol/L), indicates that these animals suffered from a copper deficiency.

The diagnosis of copper deficiency only based on clinical examination is very difficult because clinical findings are non-specific. Consequently, when any suspicious finding of this deficiency is observed in a herd, copper status of the animals should be determined, and copper, and especially molybdenum concentration in the diet, should be investigated. In addition, broad-based survey of serum copper status of cattle must be performed because economic losses related with sub clinical copper deficiency may be wide-spread.

References

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