THE RELATIONSHIP BETWEEN SELENIUM DEFICIENCY AND THE DEVELOPMENT OF PULMONARY AND SUBCUTANEOUS EMPHYSEMA IN BOVINE EPHEMERAL FEVER VIRUS-INFECTED CATTLE

G. O. ODIAWO, Department of Clinical Veterinary Studies, Faculty of Veterinary Science, University of Zimbabwe, P.O. Box MP 167, Mount Pleasant, Harare, Zimbabwe

ABSTRACT

Bovine ephemeral fever (BEF) was diagnosed on several commercial farms around Harare, Zimbabwe. The affected animals showed signs of fever (40-41.5 °C), depression, ruminal stasis, lameness and recumbency. Eight of those attended had severe respiratory distress and subcutaneous emphysema. Haematological and biochemical results indicated leucopenia with an attending lymphopenia. Selenium deficiency was detected only in those animals which showed respiratory embarrassment and subcutaneous emphysema.

INTRODUCTION

Bovine ephemeral fever (BEF) is a non-contagious viral disease of cattle caused by a pleomorphic virus of the rhabdovirus group (Heuschele, 1970; St. George, 1981). The disease is characterised clinically by an acute onset of fever, depression, salivation, ruminal stasis, muscle fasciculations, lameness and recumbency (Mackerras, Mackerras & Burnett, 1940; Basson, Pleniar & Van der Westhuizen, 1970; Burgess & Spradrow, 1977). An uncharacteristic clinical sign of severe respiratory distress characterised by a forced expiratory grunt and the development of pulmonary and subcutaneous emphysema has been described only in a small percentage of clinically affected cattle (Sen, 1931; Mackerras et al., 1940; MacFarlane & Haig, 1955; Burgess & Spradrow, 1977; Theodoridis & Coetzer, 1979). Most of these succumb to the disease with high mortality.

Mulhearn (1937) described this condition in Australian cattle which were affected with BEF. His opinion was that secondary bacterial infection may have accounted for the respiratory signs. Basson et al., (1970) described histopathological lesions in such animals dying of the disease. They observed focal, pale circumscribed areas in skeletal muscles consisting of hyaline necrosis of muscle fibres. Theodoridis & Coetzer (1979) described in detail these changes as pulmonary emphysema resulting from bronchiolar obliteration with cellular debris and necrosis. Other histological findings included Zenker's degeneration of muscular layer and thrombosis of pulmonary vessels.

They suggested that forced movement during the clinical bout could precipitate pulmonary distress. Because only a small percentage of clinically affected animals showed this syndrome, they suggested that studies be directed to the microbiology, blood physiology and nutrition of such animals.

Lober, Thurston, Gander & Goodale (1971) and Stoner (1972) were, however, unable to attribute the pulmonary pathology in such cases to the direct viral activity because BEF virus is non-cytolytic. Cowie & Dear (1976) observed an increase in the incidence of respiratory embarrassment in cattle with clinical selenium deficiency. Their findings, however, did not involve control animals. Blood, Radostits & Henderson (1985) reported that secondary pneumonia seems to be sequela to clinical selenium deficiency in cattle where muscles of the throat and chest are affected. They described hyaline degeneration and coagulative necrosis of the muscle layer of the bronchioles. Cowie, Dear, Blood and others, therefore, believe that impaired respiratory muscle function might contribute to the increased incidence of pneumonia in cases of responsive nutritional myopathy.

However, Philippo, Arthur, Price & Halliday (1987) reported that the incidence of pneumonia in housed calves was not related to selenium deficiency but to herd size and building design. This paper describes the clinical, haematological and selenium status of eight cases of BEF with pulmonary and subcutaneous emphysema studied in Zimbabwe between January 1987 and April 1988.

MATERIALS AND METHODS

Animals

A severe outbreak of BEF occurred in several commercial farms around the capital, Harare, during the early months (January to April) of 1986, 1987 and 1988. Those of 1986 have been described (Hill, MacKenzie & Honhold, 1988, in press) while the 1987 ones were sporadic and less severe than those of 1986 and 1988. The cases described fell under three main clinical groups:

(1) Those which showed transient fever, less severe clinical signs and quick recovery.

(2) Severely affected animals with pyrexia, salivation, ruminal stasis, knuckling, muscle fasciculations, lameness and recumbency, and the course of illness lasted for about 3-5 days with subsequent recovery.

(3) A few cases (8 attended) which had similar clinical signs like those in (2) but in addition had very severe respiratory distress characterised by a forced expiratory grunt and on chest auscultation, dry crackling rales were detected suggesting pulmonary emphysema. Subcutaneous emphysema was evident along the trunk, neck region and the hind limbs extending up to the hock joints. All these cases were attended in the fields except for case number 242 which ended up in the University Veterinary Hospital.

Blood and serum sample analysis

Blood samples were taken by venipuncture at the height or near peak temperature reaction into plastic tubes containing dipotassium diamino-ethane-tetra-acetate (EDTA) and stored at -20 °C until analysed or analysed within hours of collection. Blood for serum was collected in plain plastic tubes and kept at

Received 3 January 1989—Editor
Seemed to have resolved the respiratory crisis since sema were seen only in animals which had low GSHPx activity, except in animal number 71 which on the dead animal (15) as death was reported two days later. The animals had an uneventful recovery.

Glutathione peroxidase activity was low in all the animals with respiratory distress and subcutaneous emphysema, except for animal 71 which was low on selenium but never showed any respiratory problems. Some of the animals (52, 61, 109, 15, 242, 4*, 71, 70, 89, 66*) had low calcium levels but never had any respiratory problems. Some of them (numbers 71, 109, and 89 may have been related to the time when the blood samples were taken.

Glutathione peroxidase is a selenoenzyme (Rotruck, Pope, Ganther, Swanson, Hafeman & Hoekstra, 1973) and its activity in blood is related to blood selenium concentration and dietary intake. It had therefore, been considered a sensitive indicator for selenium status in rats, sheep and cattle (Hoekstra, 1973) and its activity in blood is related to the time when the blood samples were taken.

RESULTS

Most animals showed pyrexia (40-41.5°C). There was a general tendency to leukocytosis with a shift to the left and an accompanying lymphopaenia (Table 1). The red blood cell counts of the affected animals were within normal range. Hypocalcaemia was detected in those animals which were bled at the height of the temperature reaction; however, some of them (numbers 71, 109 & 89) had normal serum calcium levels but never showed any respiratory problems. Some of the animals (52 & 61) had low calcium levels but adequate selenium and never showed any respiratory embarrassment. No autopsy was performed on the dead animal (15) as death was reported two days later.

The regimen of treatment described above seemed to have resolved the respiratory crisis since the animals had an uneventful recovery.

DISCUSSION

Respiratory distress and subcutaneous emphysema were seen only in animals which had low GSHPx activity, except in animal number 71 which had low blood selenium levels but without the development of respiratory distress. The leukocytosis with an accompanying leukopaenia and hypocalcaemia reported here are similar to those reported by previous authors (Mackerras et al., 1940; Basson et al., 1970; Burgess & Spradbrow, 1977; Young & Spradbrow, 1988; Uren & Murphy, 1985). Normal calcium levels in animal numbers 71, 109 and 89 may have been related to the time when the blood samples were taken.

Glutathione peroxidase activity is related to selenium deficiency and the development of emphysema. The regimen of treatment described above seemed to have resolved the respiratory crisis since the animals had an uneventful recovery.

### TABLE 1 Glutathione peroxidase activity, haematological and biochemical results of five animals from group (2) and eight animals from group (3)

<table>
<thead>
<tr>
<th>Animal No.</th>
<th>rbc</th>
<th>wbc</th>
<th>seg</th>
<th>Lymph</th>
<th>Ca</th>
<th>Mg</th>
<th>Inorg. phos.</th>
<th>Glutathione peroxidase activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>71**</td>
<td>6.31</td>
<td>12.64</td>
<td>18</td>
<td>79</td>
<td>2.26</td>
<td>1.0</td>
<td>ND</td>
<td>Deficient</td>
</tr>
<tr>
<td>52</td>
<td>6.40</td>
<td>15.44</td>
<td>18</td>
<td>82</td>
<td>1.87</td>
<td>0.9</td>
<td>0.50</td>
<td>Adequate</td>
</tr>
<tr>
<td>61</td>
<td>5.40</td>
<td>12.31</td>
<td>18</td>
<td>82</td>
<td>1.84</td>
<td>1.9</td>
<td>1.0</td>
<td>Adequate</td>
</tr>
<tr>
<td>346*</td>
<td>5.54</td>
<td>15.67</td>
<td>63</td>
<td>21</td>
<td>1.60</td>
<td>1.1</td>
<td>1.00</td>
<td>Adequate</td>
</tr>
<tr>
<td>109</td>
<td>7.72</td>
<td>8.50</td>
<td>42</td>
<td>41</td>
<td>2.40</td>
<td>1.0</td>
<td>1.20</td>
<td>Adequate</td>
</tr>
<tr>
<td>15*</td>
<td>5.99</td>
<td>18.73</td>
<td>70</td>
<td>29</td>
<td>1.93</td>
<td>ND</td>
<td>2.10</td>
<td>Deficient</td>
</tr>
<tr>
<td>242*</td>
<td>9.67</td>
<td>21.02</td>
<td>75</td>
<td>24</td>
<td>1.82</td>
<td>ND</td>
<td>1.0</td>
<td>Adequate</td>
</tr>
<tr>
<td>4*</td>
<td>8.99</td>
<td>8.54</td>
<td>51</td>
<td>47</td>
<td>1.10</td>
<td>ND</td>
<td>4.30</td>
<td>Deficient</td>
</tr>
<tr>
<td>21*</td>
<td>8.69</td>
<td>23.76</td>
<td>50</td>
<td>50</td>
<td>1.90</td>
<td>1.2</td>
<td>1.00</td>
<td>Deficient</td>
</tr>
<tr>
<td>70</td>
<td>6.82</td>
<td>13.66</td>
<td>59</td>
<td>33</td>
<td>1.86</td>
<td>ND</td>
<td>3.10</td>
<td>Adequate</td>
</tr>
<tr>
<td>89</td>
<td>6.80</td>
<td>16.12</td>
<td>50</td>
<td>30</td>
<td>1.89</td>
<td>1.12</td>
<td>2.10</td>
<td>Deficient</td>
</tr>
<tr>
<td>66*</td>
<td>5.85</td>
<td>13.31</td>
<td>59</td>
<td>36</td>
<td>1.78</td>
<td>0.7</td>
<td>1.40</td>
<td>Deficient</td>
</tr>
</tbody>
</table>

* Animals which showed pulmonary distress with subcutaneous emphysema
** Animals with low blood selenium levels but never showed any respiratory distress
ND = Not done

4°C, centrifuged and serum obtained. Blood cell counts were determined using Coulter Counter machine (Coulter Counter®, ZM). Blood selenium levels were determined and later showing any respiratory distress. No autopsy was performed except for animal 71 which was low on selenium but never showed any respiratory distress. Some of the animals (52 & 61) had low calcium levels but never had any respiratory problems. Some of them (numbers 71, 109 & 89) had normal serum calcium levels but never showed any respiratory problems. Some of the animals (52 & 61) had low calcium levels but adequate selenium and never showed any respiratory embarrassment. No autopsy was performed on the dead animal (15) as death was reported two days later.

The regimen of treatment described above seemed to have resolved the respiratory crisis since the animals had an uneventful recovery.
The pathogenesis of pulmonary emphysema has been described (Spencer, 1985) as resulting from obliteration and destruction of respiratory bronchioles causing 'air-trapping' and also due to inflammatory weakening of the walls of either respiratory bronchioles or more distally situated structures including peribronchial alveolar walls.

The histological findings described by the above author are similar to those described for BEF by Basson et al. (1970) and Theodoridis & Coetzee (1979). They described bronchial obliteration with inflammatory cell infiltrates and cellular debris, destruction of respiratory bronchioles and thrombus formation in pulmonary blood vessels of BEF cases with pulmonary and subcutaneous emphysema.

The findings reported here would suggest that selenium deficiency in cattle infected with BEF virus is a factor in the pathogenesis of pulmonary distress in cattle. It is the author's opinion that the occurrence of respiratory distress and subcutaneous emphysema in these animals was related to reduced GSHPx activity (selenium deficiency) and that the infection was superimposed on this nutritional stress. Work is in progress to verify this hypothesis.

ACKNOWLEDGEMENTS

The author would like to thank Mr Ishmael Mutema of Biochemistry Department, Veterinary Research Laboratory, Causeway, Harare for his services in estimating the enzyme activity. He also would like to thank the technical staff in the Clinical Laboratory of the Department of Clinical Veterinary Studies, Faculty of Veterinary Science, University of Zimbabwe for their valuable work which helped in the preparation of this paper.

REFERENCES


