A FIELD OUTBREAK OF CHRONIC AFlatoxicosis IN DAIRY CALVES IN THE WESTERN CAPE PROVINCE

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ABSTRACT
An outbreak of mortality in Friesland dairy calves in which 7 out of 25 calves died in the western Cape Province, Republic of South Africa is described. Clinical signs included a loss in body mass, staring hair coat, diarrhoea and rectal prolapse. Histopathological changes in the liver were characterised by severe portal fibrosis with bile duct proliferation and mild portal round cell infiltration. The calves were fed a ration containing locally-produced maize. The implicated maize was infected with Aspergillus flavus and contained aflatoxins B, B, G, and G, with total aflatoxin levels as high as 11 790 ng g⁻¹. This is the first report of a field outbreak of bovine aflatoxicosis in South Africa.

Keywords: Aspergillus flavus, bovine

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INTRODUCTION
Aflatoxicosis is a mycotoxicosis caused by aflatoxins, hepatotoxic metabolites of Aspergillus flavus Link and A. parasiticus Speare produced in a variety of cereals and other foodsstuffs, particularly groundnuts (Arachis hypogaea L) and maize (Zea mays L) 5 6 10. A shipment of contaminated Brazilian groundnut meal which caused a massive outbreak of acute aflatoxicosis in turkeys in the United Kingdom in 1960, also affected cattle 7.

Outbreaks of acute aflatoxicosis in turkeys have been reported from Australia 8, India 9, Thailand 12, and the United States of America 3 14 in South Africa, outbreaks of aflatoxicosis have been confirmed only in pigs and dogs 9.

In this paper we report on a field outbreak of aflatoxicosis in Friesland calves in the western Cape Province following the consumption of rations containing locally-produced, aflatoxin-contaminated maize.

MATERIALS AND METHODS

History
The maize involved in this outbreak was produced by Farmer A in the Moorreesburg district, Cape Province, who had a total harvest of ca 58 tons. The maize was harvested at a very high moisture content (estimated by Farmer A to be at 18 to 19%) in early May 1986 during dry weather and was stored on the floor of a neighbour's shed, where it was said to have been turned daily. One section, where the maize had been piled high, was not turned regularly and was found to be hot, due to fermentation. According to Farmer A, this portion was sold last. Farmer B, on whose farm the problem occurred, bought 100 bags (each 75 kg) of maize from Farmer A at the end of May or beginning of June, and a further batch of 60 bags from Farmer A via Farmer C (who fed the maize in small quantities to his own cattle and sheep without apparent problems) at the end of May 1989. Farmer A claims that he fed the maize to his own cattle without ill effects. Maize was sold to 6 different farmers of whom only 2 reported problems: Farmer B and another unconfirmed case where apparently 15 out of 54 calves died. The maize bought by Farmer B was only fed to his calves. Approximately 25 Friesland calves varying in age from 1.5 to 9 months were kept in pens and supplied with the following ration ad lib: 2 bags millet maize, 2 bags second grade wheat, 1 bag mollase urea mixture, 1 bag high protein concentrate; 2 bags bran and 4 bags coarsely milled wheat straw.

The first calf died in early August with clinical signs of a loss in body mass, staring hair coat, diarrhoea and rectal prolapse. Two further cases with similar clinical signs occurred and the private veterinarian was summoned for an autopsy. Organ samples were collected from one of these calves (Calf 1) and placed in formalin while a live calf, in extremis, was submitted to the Regional Veterinary Laboratory, Stellenbosch on September 25, 1989 (Calf 2). The farmer immediately stopped feeding the ration, but lost a further 3 calves with similar clinical signs. Many of the surviving calves were stunted and exhibited poor weight gains.

Materials and methods

Organ samples in 10% formalin of Calf 1 were received for histopathology. A live, 5-month-old bull calf in very poor condition and with obvious diarrhoea was submitted for a post-mortem examination (Calf 2). Heparinated blood was collected from the anterior vena cava for clinical pathology and the animal was euthanased for autopsy. Organ samples were collected in 10% formalin for histopathology and appropriate samples were collected for bacteriological and parasitological examination.

Maize samples

Maize samples (1 kg) were collected from each of 4 bags of maize from Farmer B where the outbreak occurred and from 3 bags each from Farmers A and C. maize samples were examined mycologically by plating kernels directly on Aspergillus flavus and parafusus agar (AFPA) 13, and analysed chemically for aflatoxins as follows: extracts of dried and ground maize samples were prepared and purified using the CB procedure described by the Association of Official Analytical Chemists 14. Quantification of individual aflatoxins was done by reverse-phase HPLC and fluorescence detection incorporating post-column derivatisation with iodine 15.

RESULTS

Macroscopic pathology

The carcass of Calf 2 was very cachectic and showed marked hydrothorax, hydropericardium ascites. The kidneys were slightly enlarged and pale. The liver was very shrunken and fibrotic. There were no signs of icterus.

Microscopic pathology

The changes in the livers of Calves 1 and 2 were characterised by severe fibrosis, bile duct proliferation and mild round cell infiltration in the portal triads. In the kidneys, diffuse mild interstitial fibroplasia (Calf 2) and focal interstitial lymphocyte infiltration (Calf 1) were observed. Demyelination in the medulla oblongata and mild neutrophil infiltration of the lamina propria in the intestine (Calf 2) were the only other lesions recorded.

Clinical pathology

A marked anaemia with moderately increased total bilirubin levels was present in Calf 2, but other serum data were within the normal range: total plasma proteins 62 g l⁻¹, albumin 22 g l⁻¹, globulin 40 g l⁻¹, haemoglobin 5.26 g d⁻¹, haematocrit 0.18, gammaglutamyl-
transferase 17 U L⁻¹; aspartate transami
nase 44 U L⁻¹; and total serum bilirubin
9.3 μmol L⁻¹. Bacteriological isolation
yielded Escherichia coli from the intestines
only and the faeces was negative for
helminth eggs.

**Myotoxicology**

Aspergillus flavus was isolated from 75 to
100% of the maize kernels (not surface
disinfected) in all samples. All the maize
samples also contained aflatoxins (Table
1). Marked variation occurred in the
levels of aflatoxins B₁, B₂, G₁, and G₂ in the
samples obtained from the 10 different
bags examined e.g. aflatoxin B₁ levels
ranged from 1.7 to 14 000 ng g⁻¹ and
total aflatoxins from 2.2 to 15 930 ng g⁻¹.

**DISCUSSION**

This is the first reported field outbreak of
aflatoxicosis in cattle in South Africa⁵. The
clinical signs and pathological changes
were consistent with those described for
bovine aflatoxicosis¹ 3 5 6 8 11, while ex
tremely high levels of the aflatoxins were
detected in the mouldy maize (Table 1).

Due to the increases in the purchasing
price and transportation cost of maize,
more farmers in the western Cape are
harvesting early in order to prepare the
lands for wheat planting. The maize is
usually harvested at a high moisture
level associated with consumption of aflatoxin-
contaminated peanuts. Journal of the
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187: 636-637


whereas the maximum level allowed in
feed for lactating cows and calves in
South Africa is 20 ng g⁻¹ in terms of the
Fertilizers, Farm Feeds, Agricultural
Remedies and Stock Remedies Act⁹.

The wide variation in aflatoxin concen-
tration in different bags of maize from the
same farm also shows that different
amounts of aflatoxin can be produced in
"hot spots" in stored maize under these
conditions. In view of these conditions, it
can be expected that further field out-
breaks of aflatoxicosis will occur in the
western Cape.

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Fig. 1: Portal fibrosis and bile duct proliferation with mild portal mononuclear cell infiltration. The connective tissue has extended into the parenchyma surrounding small groups of hepatocytes

Table 1: Concentrations of aflatoxins in samples of maize implicated in a field outbreak of aflatoxicosis in dairy calves

<table>
<thead>
<tr>
<th>Farm No</th>
<th>Sample No</th>
<th>Aflatoxin concentration ng g⁻¹</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Farm where maize produced</td>
<td>1</td>
<td>12</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1.7</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>14 000</td>
<td>730</td>
</tr>
<tr>
<td>B. Farm where outbreak occurred</td>
<td>1</td>
<td>1 880</td>
<td>280</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>5 810</td>
<td>930</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>830</td>
<td>170</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>9 800</td>
<td>730</td>
</tr>
<tr>
<td>C. Farm where no cases were recorded</td>
<td>1</td>
<td>580</td>
<td>120</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1 400</td>
<td>280</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>900</td>
<td>160</td>
</tr>
</tbody>
</table>

ND Not detected. Detection limit = 0.1 ng g⁻¹