Aflatoxicosis

EDWARD S. MURRAY, *D.V.M.*
Spur, Texas

Aflatoxicosis is a noninfectious, noncontagious intoxication of an animal with a metabolite produced by a toxic mold. It is characterized in the acute phase by death loss and in the chronic cases by consumption decrease and death loss due to hepatic and renal insufficiency. Morbidity and mortality rates range from 5 to 55%.

The etiological agents most commonly incriminated are *Aspergillis flavus* and *Penicillium rubum*. The literature states that other molds are also capable of producing toxic metabolites. As research continues and need arises, I feel sure that more molds will be found that produce toxic metabolites.

A review of the available literature on Aflatoxicosis indicates that most of the work was published from 1950 through 1969. The majority of the literature deals with this condition in swine, while little mention is made of aflatoxicosis as an entity in the bovine.

Aflatoxins are classified according to the color of florescence under ultraviolet light. The toxic aflatoxin seen at the lowest levels is B_1 (blue), and following in order are: G_1, B_2, G_2, and M. Toxin B_1, the most toxic aflatoxin, is also that which is produced in the largest quantity.

As one might suspect, the weather conditions surrounding an outbreak of aflatoxicosis are paramount, i.e., sufficient moisture must be present to allow proliferation of mold colonies. Moisture content must exceed 15% to harbor toxic molds, while ideal relative humidities range from 70 to 99%. Aflatoxins were produced at the highest rates when environmental temperatures were around 30 degrees centigrade. Another fact worthy of note is that as the CO_2 level increases, the production of aflatoxin decreases, so that when silage is not properly packed or when the ensilation process is incomplete, aflatoxins are more readily produced.

The chemical configuration of the aflatoxins was elucidated in England and Holland in 1962. The structure of these metabolities closely resembles cumarin. This family is well known to most for its anticoagulant and hepatic toxin characteristics.

From a clinical standpoint, the history of the individual pens of cattle is necessary. As I previously stated, weather conditions are paramount. The length of time cattle have been on feed, the weight of the cattle upon entry, the daily rate of gain, their consumption, conversion and the death loss, aid in determining the problem involved.

Let us now examine the clinical aspects of aflatoxicosis. Peracute and acute syndromes vary slightly. Some of the animals are found dead in the lot while others exhibit signs of pulmonary emphysema. Animals stand with the head extended, mouth open, viscid saliva hanging from the commisures of the mouth and when approached the affected animals are reluctant to move. Still others are reluctant to rise and when left alone return to sternal recumbency. A small percentage of the animals attempt to walk on their knees without placing their feet on the ground as if they had a severe laminitis. Ten to thirty-five percent of the animals are affected in this manner.

Upon examination, the body temperature rarely exceeds 104°F, while most range from 102-103°F. The mucous membranes appear anemic and in severe cases are icteric. A marked loss in body condition is evident. Blood-tinged diarrhea is inconsistent but present in numerous cases. Auscultation of the lungs reveals pneumonic sounds in the lower one-third of the lung fields and emphysematous sounds in the dorsal and posterior portions. As a general rule death occurs in 25 to 50% of the affected animals.

The animals chronically affected with varying degrees of aflatoxicosis are unthrifty for they continue to consume but convert at uneconomical rates. A marked amount of body condition is lost during the acute phase of the disease and the animal appears unable to regain the weight loss. Hooves commonly grow long and turn up at the ends, as in cases of chronic laminitis.

Laboratory tests showed total white count elevated to 12,000 and 13,000 with a moderate "shift to the left." Prothrombin time and SGOT increased. BUN and SGPT and the icterus index also increased but not as consistently as the SGOT and Prothrombin.

Gross postmortem findings can be described as follows: As an average carcass was opened one found moderate numbers of subcutaneous petechiae and 2 to 10 very large, i.e., 5 to 15 cm., hemorrhagic areas in the subcutaneous tissue. In severe cases, icterus was plainly visible, as was the ventral edema. Edema was confined to the brisket, axillary and prefemoral regions.

The pleural and peritoneal cavities had large amounts (three to ten gallons) of sero-sanguinous fluid.

Large hemorrhages, 1 to 5 cm., were noted on the pleural and peritoneum.

The liver was large, distinctly lobulated, extremely friable and bright yellow in color on the cut surface. In other cases the liver was distended, pale, and showed what grossly would appear to be anemic infarcts.

The spleen was engorged and covered with ecchymoses, while the

kidneys were enlarged and pulpy and exhibited subcapsular petchiae.

The intestinal wall was thickened due to the edema and the lumen contained a moderate amount of hemorrhagic material with an occasional cast-like blood clot.

The lungs showed a moderate to severe interstitial emphysema in the dorsal and posterior half, while pneumonia in varying degrees was evident, with red and gray hepatization. In the peracute deaths, emphysema was quite marked and the bronchi, trachea, and pharynx contained varying amounts of viscid, frothy substance, white in color and usually blood tinged.

Histopathological reports from the laboratories are somewhat limited though fairly consistent in their findings. Hepatic changes were as follows: focal centrolobular degeneration; focal necrosis with infiltration of eosinophils; severe fatty metamorphosis with marked bile stasis; focal necrosis, slight leukocytosis; portal fibrosis with infiltration of eosinophils; proliferation of the epithelium lining bile ducts.

Renal histopathology indicated a mild toxic tubular nephrosis and subacute interstitial nephritis. Histopathology of the lung revealed a marked proliferative pneumonitis with interstitial emphysema. Splenic changes showed a mineralization of splenic trabeculae, congestion and leukocytosis.

Comments were consistently made by the pathologists concerning the toxic hepatosis, toxic nephritis, and nephrosis. One gets the impression of a severe toxicosis but no specific toxin can be incriminated.

Bacterial culturing was performed on most animals and all indicated negative results for *Clostridium spp.* The organisms most frequently isolated were *E. coli* and *B. subtilis.*

Animals in this particular feedlot were being fed a high silage-low grain ration. Death losses were excessive in all ages of cattle and in all stages of finishing. Losses were very sporadic as there would be numerous deaths for several days followed by a period when there were almost no losses.

Harvesting conditions on the high plains of Texas in September and October, 1970, were anything but ideal due to sporadic rain for a period of approximately 45 days. During this time silage was cut on to trucks and was not unloaded into the pit for 48 to 72 hours. Proper silage packing was difficult and at times impossible. For these reasons collection of samples was directed at the silage rather than other foodstuffs.

Numerous samples of silage were collected and sent to various laboratories. At long last a toxic mold, *Aspergillis flavus*, was isolated and a short time later the aflatoxin was incriminated.

At the present time I know of no successful treatment for aflatoxicosis other than removing the source of the toxin and using supportive treatment to allow time for as much hepatic and renal regeneration as possible. Control, likewise, must be directed at removing the aflatoxin.