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PATHOLOGY OF AFLATOXICOSIS IN GOATS*

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Aflatoxins are a group of fungal metabolites produced by *Aspergillus flavus, Aspergillus parasiticus* and related fungi. Aflatoxins cause a variety of biological effects including hepatotoxicity, carcinogenicity, teratogenicity and immunosuppression. In Kerala more than 50% of animal feed samples contained sufficient quantity of aflatoxin to cause toxicity (Maryamma *et al.* 1982). Only a few workers have studied the pathology of experimental aflatoxicosis in goats (Maryamma and Sivadas *et al.* 1975, Clark *et al.* 1984 and Miller *et al.* 1984.) So an experiment was designed to study the pathology of aflatoxicosis in goats.

Materials and Methods

Experimental animals: Twelve, clinically healthy, male cross bred kids (2–4 months) were obtained from AICRP on goats, Mannuthy. They were randomly divided into a control group and an experimental group containing equal numbers.

Toxin: Crude aflatoxin was prepared in rice culture (Shotwell *et al.* 1966) using a toxigenic strain of *Aspergillus parasiticus* (obtained from Central Food Technology Research Institute, Mysore). It was powdered and the quantity of toxin present in representative samples were determined (Pons and Goldblatt, 1969). The toxin was blended with jaggery and fed orally to the six randomly selected experimental kids so that every kid receieved 0.25 mg of crude aflatoxin per kilogram body weight, daily. The administration was continued until the goats died.

Postmortem examination: Detailed post-mortem examinations were conducted. Representative tissue samples collected from the liver, spleen, kidney, heart and intestine were processed by routine paraffin embebding technique and sections were stained with Harri's haematoxylin and eosin for microscopic studies.

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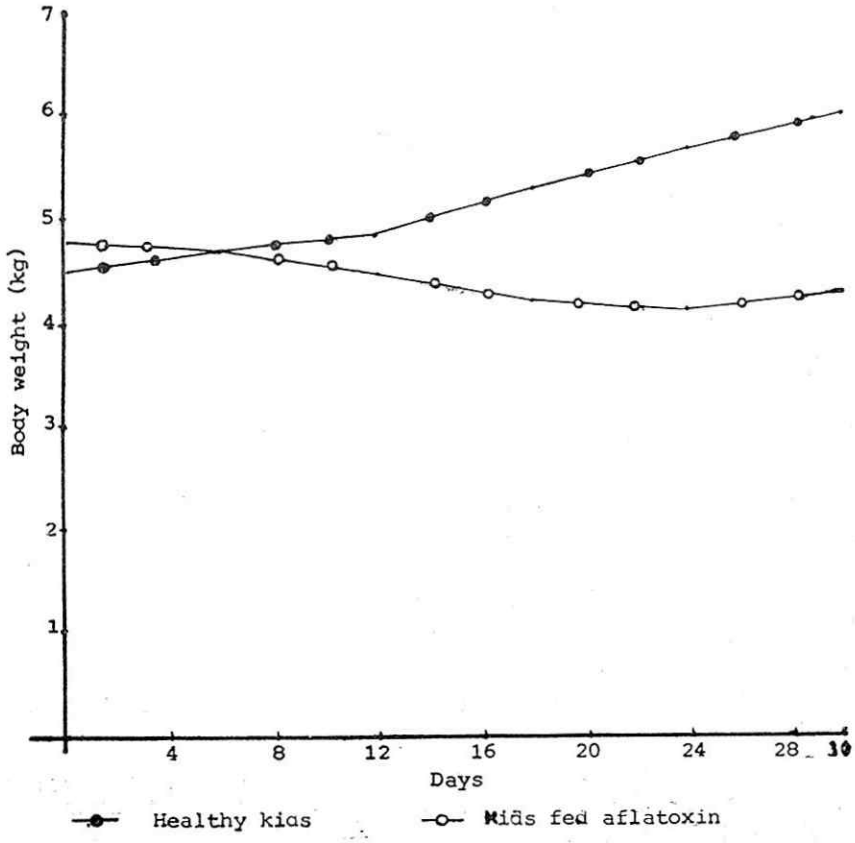
Results

All the kids in the control group remained healthy while all the experimental kids died.

Reduction in feed intake, loss in body weight, unthriftiness, dyspnoea, nasal discharge and respiratory difficulties were shown by the experimental animals. The changes in body weight are shown in the Fig. 1. There was progressive reduction in weight in all the kids dosed with aflatoxin.

Fig. 1. Experimental aflatoxicosis: Mean body weight of kids



Kids dosed with aflatoxin died at varying intervals of time. One kid which consumed 9.3 mg of toxin died on the nineth day. Others consumed 28.68 to 43.28 mg of toxin and lived for a period of thirty to thirty five days.

The post-mortem lesions were similar in all the kids. The liver was slightly enlarged and brownish yellow in colour with rounded borders. Two animals showed petechial haemorrhages on the liver surface. Gall bladder was distended with yellowish green bile. Histologically hepatocytes showed granular degeneration, moderate to severe degree of fatty change and necrosis. Hepatocytomegaly was also a feature. The sinusoids were engorged and there was bile stasis. There were focal areas of haemorrhages in the parenchyma (Fig. 2 to 3). There was slight but perceptible interstitial fibrosis, pseudolobulation and haemosiderosis in two of the kids.

Focal greyish white areas were present uniformly distributed on the surface of the kidney. The cut surface revealed greyish white streaks on the cortex. Histological changes were characteristic of toxic nephrosis. Degeneration and desquamation of tubular epithelial cells, formation of hyalin casts, glomerular oedema and atrophy of glomeruli were seen. Many of the tubules were plugged with degenerated and desquamated epithelial cells. The necrotic debris was seen refluxing into the glomeruli in certain cases. Some of the glomeruli showed haemorrhage in the Bowman's space and was distended (Fig. 4 and 5).

Lesions in the lung were characteristic of acute bronchopneumonia. Apical cardiac and intermediate lobes of both lungs of all animals were greyish white, fleshy and consolidated. In two animals, the changes were also present in the diaphragmatic lobes. Congestion, haemorrhages and infiltration of neutrophils, lymphocytes, macrophages and plasma cells were present in the lung alveoli. There was degeneration of bronchial epithelium and occasional accumulation of lymphocytes in the peribronchial area.

Grossly the bronchial and mediastinal lymph nodes were slightly enlarged, soft and fleshy. Cut surface was juicy. Histologically severe oedema was present in the medulla. Depletion of immunocompetent lymphoid cells was evident in the cortex and paracortex. The number of cortical lymphoid follicles was very few and poorly organised. (Fig. 6) Spleen was congested.

Mild enteritis was seen in the intestine. Mucosa of the abomasum was slightly oedematous.

Bronchopneumonia was the cause of death in all the kids. All the animals in the control group remained healthy throughout the experimental period.

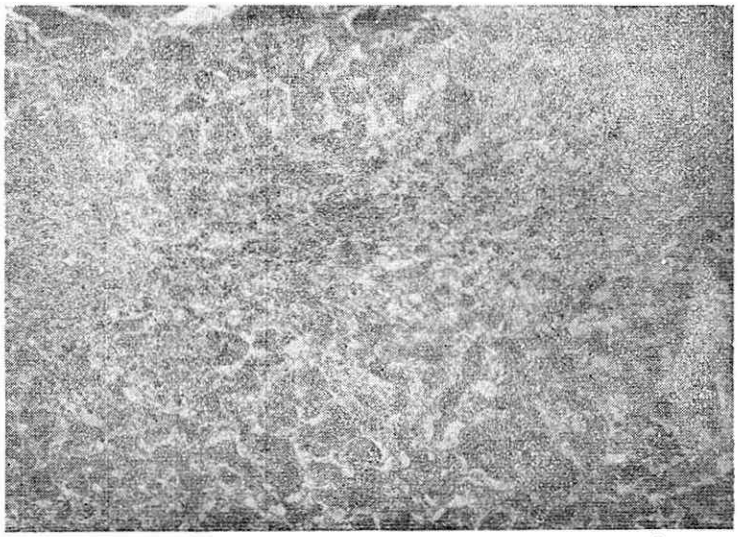


Fig. 2.
Liver: Focal areas of degeneration and necrosis H & E x 250.

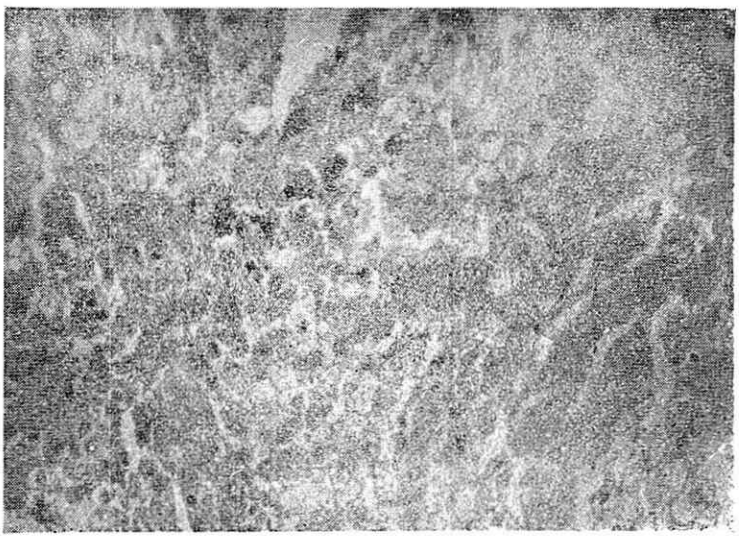


Fig. 3.
Liver: Degeneration, necrosis and hepatocytomegaly. Biliary canaliculi are distended with bile. H & E x 450.

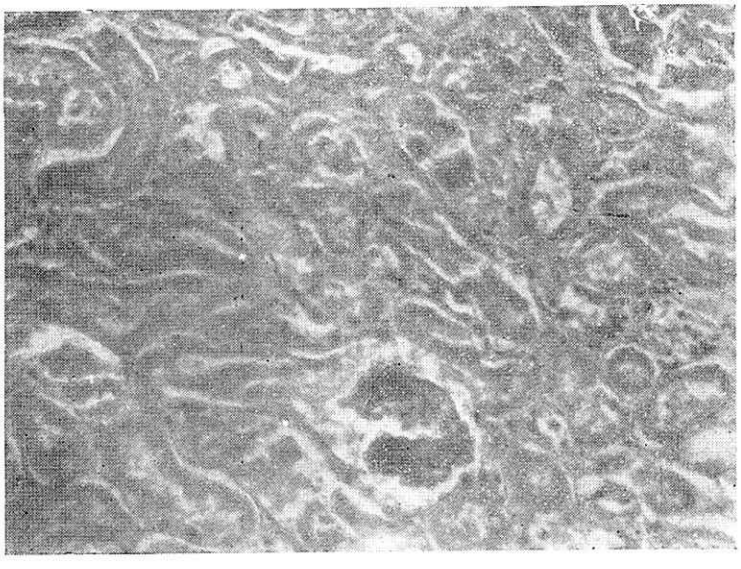


Fig. 4.
Kidney: Haemorrhage in the glomerulus. Degeneration of the tubules. H & E x 250.

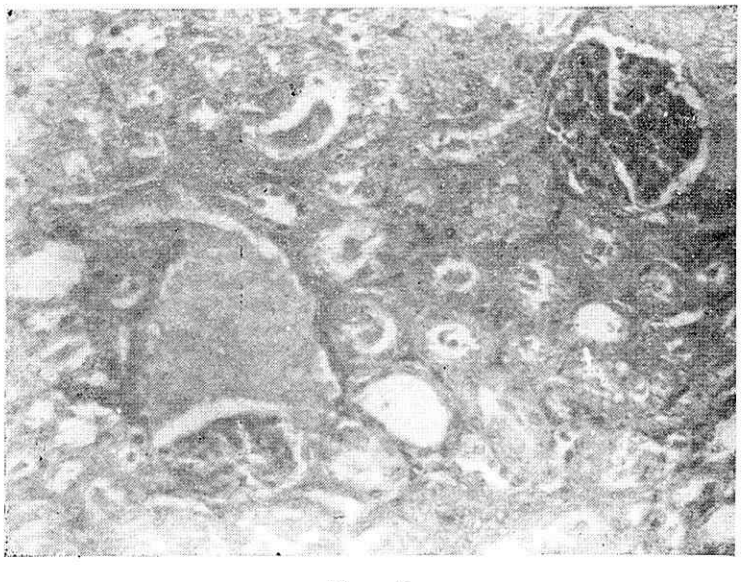


Fig. 5.
Kidney: Glomerular oedema and casts in the tubules H & E x 400

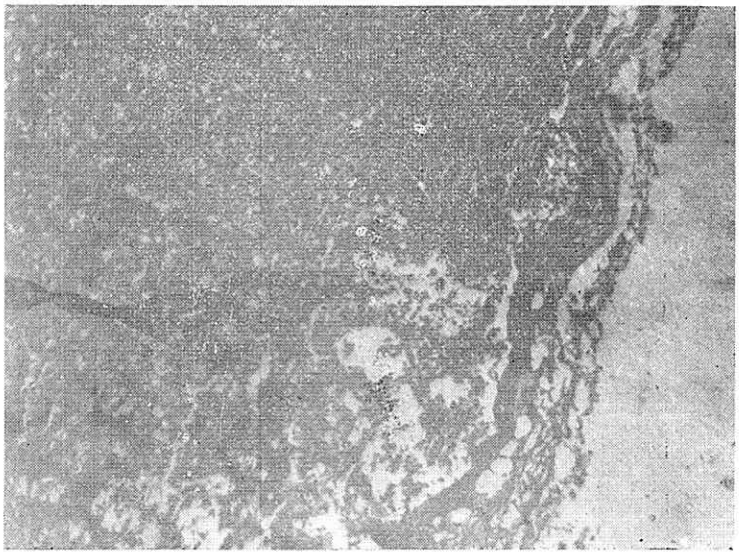


Fig. 6.
Lymphnode: Depletion of lymphocytes in the cortex. H & E x 250

Discussion

Aflatoxin is a potent hepatotoxin. Eventhough, goats are relatively resistant to the toxic effects of the aflatoxin, they too are susceptible to the toxic effects at higher doses of the toxin (Maryamma and Sivadas, 1975). In the present study 0.25 mg/kg body weight of crude aflatoxin was administered and the kids survived for a maximum period of 35 days. Clark *et al.* (1984) fed 0.1, 0.2 and 0.4 mg/kg body weight of toxin and goats survived for a period of eight to thirty days. Reduction in weight gain was a pathognomonic feature in aflatoxicosis.

The gross and microscopic lesions in the liver and kidney were pathognomonic of aflatoxicosis. Gross lesions in liver included brownish yellow discolouration, slight enlargement and petechial haemorrhages. Microscopically there was hepatocytomegaly, haemorrhages, haemosiderosis, degeneration and necrosis, bile duct proliferation, interstitial fibrosis and pseudolobulation. Toxic nephrosis was present in the kidney, and there was degeneration and desquamation of epithelial cells, formation of hyaline casts and tubular epithelial reflux. Similar changes in the liver and kidney were also recorded by Maryamma and Sivadas (1975) and Clark *et al.* (1984)

Extensive pneumonic lesions were observed in all the experimental animals. Bronchopneumonia was the cause of death in all the experimental kids. Miller *et al.* (1984) and Clark *et al.* (1984) also observed pneumonic changes in lungs of goats fed aflatoxin. These workers

explained that aflatoxin caused immuno-suppression, and hence concommittant infections developed in the lung. In the present study also immunosuppression occurred in goats fed aflatoxin, and has already been reported (Anilkumar and Rajan, 1987). Although the toxin caused pathological changes in liver and kidney, the animals died due to pneumonia. The immunosuppressive effect, therefore, appears to have a significant role in making the animals more susceptible for pneumonia The observation made in the study is a proof to clarify the biological effect of aflatoxins. It may also be pointed out that there were pronounced pathological changes in the liver and kidney.

The present study confirmed that goat, like any other species of domestic animal, is susceptible to the toxic effect of aflatoxins. Thare fore, care should be taken to eliminate aflatoxin contaminated feed in the live stock ration. Periodic screening of livestock feed for aflatoxin will help to detect and identify contaminated livestock feed.

Summary

Six goats were fed crude aflatoxin at the rate of 0.25 mg/kg body weight. All the goats died in a period of 9 to 35 days. Bronchopneumonia was the cause of death in all kids. The gross and microscopic lesions in the liver, gall bladder and kidney were pathognomonic of aflatoxicosis.

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