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PATHOLOGY OF ACUTE EXPERIMENTAL AFLATOXICOSIS IN CHICKEN*

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Aflatoxicosis has been reported in chicken due to feed contamination by several workers. Gardiner and Oldroyed (1965), Kratter *et al.* (1969), Randall and Bird (1979) and Bryden and Cumming (1980) reported hepatic lesions in aflatoxicosis and in broilers Tung *et al.* (1971) reported haemorrhagic lesions in the liver, breast muscles and other organs. Studies on extrahepatic lesions in aflatoxicosis in chicken are scanty. Hence, an experimental work was undertaken to study the pathology of acute experimental aflatoxicosis, including extra hepatic lesions, in chicken.

Materials and Methods

Powdered rice culture of *Aspergillus parasiticus* containing 12.5 ppm of aflatoxin was used to prepare the experimental feed. Sixty, one-week-old White Leghorn chicks were divided into three groups; group I received aflatoxin at a level of 6.25 ppm; Group II received 3.12 ppm of aflatoxin and group III served as control. Two birds from each group were sacrificed at weekly intervals and the tissues were collected in 10% formal saline. The birds which died during the experiment were necropsied. The tissues were routinely processed and stained with H & E for histopathological examination. Special staining methods were used wherever necessary.

Results

The birds which received aflatoxin at the level of 6.25 ppm in feed died within a span of 3 weeks, while the birds which received aflatoxin at the rate of 3.12 ppm developed paralysis after 6 weeks and were sacrificed at weekly intervals. The histopathological changes described in group I were based on spontaneous deaths while the lesions in groups II were based on the birds which were sacrificed at weekly intervals.

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The birds of group I which died of aflatoxicosis revealed haemorrhages in breast and skeletal muscles, or kidneys spleen and brain. Lungs were highly congested. The important microscopic changes in this group in different visceral organs were linear and branching haemorrhages in the cerebrum, cerebellum (Plate: Fig. A) and heart, acute congestion and large areas of haemorrhage in the lungs, intertubular haemorrhages in the kidney and haemorrhages in pancreas; congestion in the sinusoids, large areas of haemorrhage and fatty changes were consistently observed in liver. Intestines revealed acute catarrhal changes with mild lymphoid infiltration.

The birds of group II which were sacrificed at regular intervals revealed significant gross and microscopic lesions. The liver revealed nodular growths of 1 to 5 mm in diameter all over the surfaces. No gross changes could be seen in other organs. Microscopic changes in the liver revealed areas of lipidosis (Plate: Fig B) regenerating hepatic cells which were moderately circumscribed by thin connective tissue and bile duct proliferation. Fat could be demonstrated in frozen sections by oil red 'O' stain. Chronic bronchitis with thickening of pulmonaroy vessels was observed in lungs. The bronchial epithelium was hyperplastic and revealed submucosal infiltration of lymphocytes. Peribronchial lymphoid aggregates were also seen. In kidneys the endothelial cells of glomerular tuft were swollen and vacuolation was seen occasionally. Mild periglomerular and interstitial fibrosis were also observed. Cystic dilatation and enlargement of tubules (Plate: Fig. C) were seen in a few cases. The renal vessels were thickened. The cerebrum and cerebellum revealed degenerative changes in the neurons accompanied by satellitosis and neuronophagia. Hyperplasia of lymphoid tissue was observed in spleen. Spleenic vessels were thickened. Pancreas revealed haemorrhages and cysts filled with homogeneous pinkish exudate. Focal areas of interstitial myocarditis were observed in heart (Plate: Fig D). In degenerating muscle cells enucleation with sarcolysis was observed. Separation and fragmentation of elastic fibres covering the myocardial bundles was constantly recorded. Mild congestion was seen in the intestines.

In the control group significant lesions were not observed in any organs.

Discussion

All the birds in group I which received aflatoxin at the rate of 6.25 ppm died of acute aflatoxicosis whereas the birds which received 3.12 ppm developed paralysis and were sacrificed at weekly intervals. The birds which died spontaneously in group I revealed haemorrhagic lesions in most of the organs. These findings were in accordance with

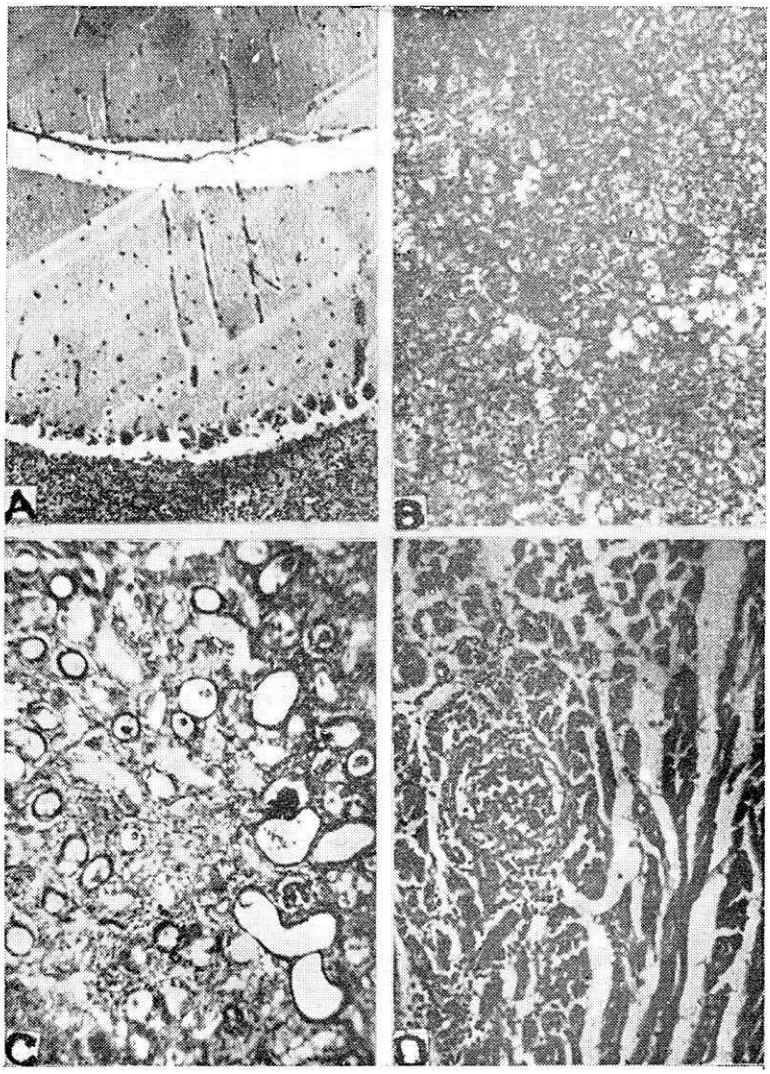

Fig. A Cerebellum
Linear haemorrhages in molecular layer H & E x 150

Fig. B Liver
Areas of lipidosis H & E x 150

Fig. C Kidney
Cystic dilatation and enlargement of tubules H & E x 60

Fig. D Heart
Focal interstitial myocarditis H & E x 100

Devos *et al.* (1965) and Tung *et al.* (1971). The symptoms of incoordination and paralysis in group II may be associated with lesions in brain. The hepatic, pancreatic, renal and intestinal lesions were responsible for loss of body weight due to impaired feed conversion. The nodules observed on the surface of the liver in group II was in accordance with the observations of Asplin and Carnaghan (1961) and Newberne (1973). Although fibrosis and pseudolobulation were reported by Newberne (1973.) extensive fibrosis was not encountered in the present study. The extra hepatic lesions in chicken were not described by earlier workers.

Summary

Gross and microscopic lesions were reported in chicken in acute experimental aflatoxicosis by feeding 2 dose levels of 6.25 ppm and 3.12 ppm of aflatoxin. High mortality with haemorrhagic lesions was observed in chicks which received 6.25 ppm of aflatoxin. Lipidosis, regenerating nodules, bile duct hyperplasia, chronic bronchitis, lymphoid hyperplasia in the spleen mild periglomerular fibrosis, neuronal degeneration or with satellitosis and neuronophagia were recorded in chicks which received 3.12 ppm.

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സംഗ്രഹം

അഫ്ളാടോക്സിൻ കൊടുത്തു കോഴിക്കുഞ്ഞുങ്ങളിൽ ക്ഷതികങ്ങൾ പഠിക്കുകയുണ്ടായി. കൂടുതൽ ഡോസ് കൊടുത്ത ഗ്രൂപ്പിൽ ക്ഷതികങ്ങളും കൂടുതലായിരുന്നു.

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