HISTOPATHOLOGIC STUDY OF THE HEPATIC AND RENAL LESIONS INDUCED BY EXPERIMENTAL TOXIC DIETARY APPLICATION OF AFLATOXIN B1 IN BROILER CHICKS.

Ena'am Bader Falih
Department of pathology and Poultry, Veterinary Medicine College, Baghdad University, Baghdad.
(Received 16 March 2005, Accepted 28 June 2005)
Keywords: AFLATOXIN, PATTY DEGENERATION, NECROSIS

ABSTRACT

The present study investigated the toxicologic histopathologic effect of aflatoxin B1, 60 broiler chicks of one-day age divided randomly and equally into three groups, for dietary study, using one group as untreated control, while the other groups were given aflatoxin B1 mixed with diet in concentration of 0.5 and 1.5 ppm respectively, for 30 days, the study showed fatty degeneration of liver associated with bile duct proliferation, accompanied by infiltration of mononuclear cells and lymphocytes. Also there was degeneration in proximal convoluted tubules mostly as hyaline degeneration especially at 0.5 ppm dose level. Those at 1.5 ppm dose level, there was focal liver cell necrosis associated with fibrosis and enlarged proliferated bile ducts, serer vacuolation and necrosis of epithelial of renal proximal convoluted tubules with thickening of the glomerular basement membrane.

INTRODUCTION

Aflatoxins considered the most dangerous mycotoxin which contaminate food and animal diet, they have carcinogenic effects in man and animals, in addition to the histopathological changes which they induced (1).

Those include aflatoxin B1, B2, G1, G2 and the second generation metabolites M1 and M2 according to their light reflection of ultra violet light 365 NM (2). Aflatoxin B1, considered one of the most effective mycotoxin as carcinogenic and was put in grade 2 between 300 toxic carcinogenic substances world wide with concentration of part of billion (3). Because of the dangerous contamination of food and animal diet, some restrictions were put by world organization for the permitted percentages of aflatoxin in food materials as (5-20) ppm (4). Histologic evidence of continued low-level aflatoxin consumption in poultry is seen in 2 immunologically active organs. Typical changes in the liver include fat accumulation in the hepatocytes and bile duct proliferation (5), also with portal fibrosis which is a common lesion associated with chronic poisoning by aflatoxin in the chicken (6). The present study intended to relink to light the macroscopic and microscopic histopathological changes, which were caused by those toxins in the livers and kidneys of broiler chicken in particular, they were more prominent as the birds become more susceptible as they grow older.

Material and Methods
1-Chicks of meat broiler of fawpro of one-day, supplied by the Hatchery of Al-Saqker company.
2-Chick commercial feed as starter supplied from Al-Saqker company.
3-Chick feed containing aflatoxin B1 with 2 concentrations of 0.5 and 1.5 ppm (part per million) was supplied from Agriculture College/ Preventive Department. The concentration of the aflatoxin was checked by High Performance Liquid Chromatography (HPLC). Also recording the quantity of aflatoxin by HPLC method in Veterinary Central Laboratories.
4-10% Neutral buffered formalin for fixation of histopathological specimens.
5-Hematoxylin and eosin stain with periodic acid schiff (P.A.S.) stain.

Experiment design
For the present study, 60 chicks were used. Those were randomly divided equally into three groups, 20 chicks for every group. The first group of (20) chicks (the control) were given chick feed without any aflatoxins, the control chick feed was also checked as clean, by taking sample, and examined by High Preformance Liquid Chromatography to make sure that it is negative from any aflatoxin.

While group 2 and 3 were given two concentration of aflatoxin as 0.5 and 1.5 ppm respectively. The experimental study project started on day one old chicks continued for thirty days. Chicks from each group were killed by cervical dislocation when they were 30 days aged.

During the treatment period, clinical signs, and macroscopic pathological changes were recorded. Sections of livers and kidneys were taken from all treated birds, fixed in 10% neutral buffered formalin and histopathological sections were prepared.

RESULTS

Clinical Signs
Clinical signs were recorded during the 30 days of treatment with two concentration of aflatoxin and compared with control birds. Chicks of second group (feed diet with 0.5 ppm) showed rough feathers, poor appetite, reduced food consumption in comparison with the control untreated birds. While the third group (feed diet with 1.5 ppm) appeared with poor condition, slow growth with the same main festation of second group but with more in severity, 4 chicks of this group died a bout 3 weeks after treatment. The dead birds were characterized by loss of body weight and retarded growth in comparison with the untreated controls.

Macroscopic findings
Chicks treated with 0.5 ppm concentration of aflatoxins B₁ after 30 days of treatment, there was congestion of internal organs in general, further more, there was enlarged pale livers, with evidence of small focal haemorrhages on its surface, some birds showed reduced size of liver with firm consistency, also enlarged gall bladder and glandular proventiculas with petechial haemorrhages on their external surface. Kidneys were enlarged, pale with petechial haemorrhage on their cortex. While striking necropy finding of third group (treated with 1.5 ppm) have been observed, those were restricted to the liver, which appeared friable with petechial hemorrhages, some chicks showed moderate nodularity of their liver surface with fatty change. Others demonstrated presence of small foci of necrosis and liver enlargement. Changes in the kidneys of this group were the same as those mentioned in previous one, but they were higher in severity.

Microscopical observations
The hepatic histopathological changes of the second group (0.5 ppm concentration), were characterized by fatty degeneration of hepatocytes in liver parenchyma (Fig. 1), associated with congestion of portal and central veins, in addition to sinusoidal congestion (which were filled with RBC) (Fig. 2).

Some chicks showed presence of infiltration of mononuclear cells, specially, lymphocytes and plasma cells in liver parenchyma and periportal regions, accompanied by bile duct proliferation (Fig. 3), associated with liver cell atrophy in the adjacent areas.
Renal histopathological changes showed degeneration of proximal convoluted tubules characterized by swelling epithelial lining of some tubules and vacuolar degeneration of the other tubules associated with infiltration of mononuclear cells and lymphocyte between tubules (fig 4 and 5) also some tubules appeared with hyaline degeneration (fig-6). Further more some of the proximal convoluted tubules were dilated.

Samples taken from the liver of third treated group (1.5 ppm), showed severe fatty degeneration particularly in periportal area (fig 7) also there was loss of hepatic parenchyma, accompanied by focal liver cell necrosis (fig 8).

Also there was pyknotic nuclei and deep acidophilic cytoplasm (fig-9) associated with marked bile duct hyperplasia and portal fibrosis (fig-10).

The changes in the kidneys were characterized by presence of degeneration of proximal convoluted tubules, associated with necrosis and exfoliation of epithelial lining in some of those tubules, disturbance and irregularities of the epithelium (fig-11) and evidence of tubular regeneration supported by presence of cortical tubular basophilia (fig-12).

Further more, there was thickening of the glomerular basement membrane accompanied by increase glomerular cellularity and presence of periodic acid shift P.A.S. positive homogenous substances (fig-13) 30 days after experimental intoxication with aflatoxin B1.

Figure-1: Microscopic section of the liver in one of the aflatoxin treated (0.5ppm) chicks, showing focal parenchymal cell hyperplasia and fatty degeneration with congestion of sinuoids, after 30 days of intoxication (40X, Ha E stain).
Figure 2: Microscopic section of the liver in one of the aflatoxin treated (0.5 ppm) chicks, showing congestion of portal vein and sinusoids with slight infiltration of mononuclear cells between hepatocytes, after 30 days of intoxication (10X, H&E stain).

Figure 3: Microscopic section of the liver in one of the aflatoxin treated (0.5 ppm) chicks, showing marked infiltration of mononuclear cells and bile duct proliferation in perportal zone, after 30 days of intoxication (40X, H&E stain).
Figure 4: Microscopic section of the kidney in one of the aflatoxin treated (0.5ppm) chicks, showing vacuolar degenerating and cellular swelling of epithelial tubular lining, after 30 days of intoxication (20X, H&E stain).

Figure 5: Microscopic section of the kidney in one of the aflatoxin treated (0.5ppm) chicks, showing infiltration of mononuclear cells and congestion between renal tubules, after 30 days of intoxication (10X, H&E stain).
Figure 6- Microscopic section of the kidney in one of the aflatoxin treated (0.5ppm) chicks, showing hyaline degeneration and vacuolation of epithelial lining tubules with loss of renal parenchyma, after 30 days of intoxication (20X. HaE stain).

Figure 7- Microscopic section of the liver in one of the aflatoxin treated (1.5ppm) chicks, showing marked fatty degeneration in peri-portal zone, after 30 days of intoxication (10X. HaE stain).
Figure 8: Microscopic section of the liver in one of the aflatoxin treated (1.5ppm) chicks, showing focal liver cell necrosis associated with infiltration of mononuclear cells, after 30 days of intoxication (10X, H&E stain).

Figure 9: Microscopic section of the liver in one of the aflatoxin treated (1.5ppm) chicks, showing loss of hepatic parenchyma associated with pyknotic nuclei and deep acidophilic cytoplasm of necrotic cell parenchyma, after 30 days of intoxication (40X, H&E stain).
Figure 10: Microscopic section of the liver in one of the aflatoxin treated (1.5ppm) chicks, showing marked bile duct hyperplasia enclosed by fibrosis, after 30 days of intoxication (40X. H&E stain).

Figure 11: Microscopic section of the kidney in one of the aflatoxin treated (1.5ppm) chicks, showing advanced degenerated tubules associated with necrosis and exfoliation of epithelial lining in some of the tubules, with diffuse mononuclear cell infiltration, after 30 days of intoxication (40X. H&E stain).
Figure 12. Microscopic section of the kidney in one of the aflatoxin treated (1.5 ppm) chicks, showing basophilic cortical tubules associated with infiltration of lymphocytes, after 30 days of intoxication (40X. H&E stain).

Figure 13. Microscopic section of the kidney in one of the aflatoxin treated (1.5 ppm) chicks, showing presence of PAS positive homogenous acidophilic substance in glomerular tuft, after 30 days of intoxication (20X. PAS stain).
درسة مرضية نموذجية للأفراد الكندي والكلوية الناجمة عن التسمم التجبري بذيلان الأفلاكونكس

في أفراوح اللحم

أعلى ذيل

فرع الأركان والكتالج، كلية الطب البيطري، جامعة بغداد، بغداد، العراق

الخليفة

لمعرفة التغيرات المرضية النموذجية للأفراد الكندي التسمم بذيلان الأفلاكونكس B1، ا슴 شرخ 60 أفراوح بعمر يوم واحد

استخدموا 60 أفراوح بعمر يوم واحد

أستخدموا في ثلاث مجموعات: انيث الإسلا (0.5) prompt، Grupo الإسلا (0.5) prompt، Grupo الإسلا (1.5) prompt

تم في اليوم الأول والثاني والثالث من تسمم الافلاكونكس، و كشفت النتائج

هناك تأثيرات هامة تختلف مع العلاجات المختلفة، حيث تظهر الأفراوح تأثيرات نموذجية

تتمحور بالتحري ككلوية ضخمة مع فرق تسمم الفلاكونكس وانتشار خلايا

58
REFERENCES


