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Toxicological Pathology of Aflatoxn B1 on Liver and Kidney in Broiler Chicks for Short Term

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ABSTRACT

A short term toxicological pathology study was done for six weeks on broiler chicks by feeding them on contaminated diets with aflatoxin B1, the birds were 14 days old at the start of the study there were macroscopic changes as enlarged and pale yellow liver, enlarged and hemorrhage of kidneys in most of the treated especially at the high dose level, histopathological changes were vacuolated hepatocytes and subcapsular region with areas of liver cell necrosis and areas of hemorrhage,periportal fibrosis and congestion of portal vein, septal fibrosis and mononeuclear cells also renal lesions with tubular necrosis, congestion, atrophy glomerular and degenerate vacuolate corticle tubules. In addition, there was hepatic bile duct proliferation with foci of hepatic oval cell proliferation too, those could reflect the carcinogenic effect of aflatoxin B1, as it could induced malignant liver cell tumor if the study continued for longer period.

Keywords- *Aflatoxin, liver, kidney, broiler chicks.*

INTRODUCTION

Aflatoxins are a group of compounds whose metabolites have been demonstrated to exert carcinogenic, immunosuppressive, hepatotoxic and other pathological effects [1].An outbreak of liver cell carcinoma rainbow trout salmo gairdneri Richardson in UK studied by [2].[3] studied the histopathological changes in broiler chickens fed aflatoxin and cyclopiazonic acid. [4] reported Acute and Chronic Effects of Aflatoxin on the Liver of Domestic and Laboratory Animals, While [5] found duration-dependent histopathological and histomtric changes in the testis of aflatoxin B1-treated mice, by indicated intraperitoneal route to 90 day old Swiss mouse at a daily dose of 50mg/kg body weight for 7, 15, 35, 45 days. In the recovery group the mice were kept, after cessation of the treatment, under observation for 35 or 70 days. [6] reported an article on Acute effects of aflatoxin on northern bobwhite (Colinus Virginianus) treated with 0, 100,

500, 1000 and 2000 parts per billion (ppb), orally once per week for 4 weeks, mortality in all treatment groups except in the 100 ppb, There was Immunosuppression, reduction in gamma-globulin, glucose, and gamma-glutamyltransferase blood levels, and abnormal liver histology. [7] studied the histopathology of the liver affected with Aflatoxins in broiler chicks of 42 days of age, the criteria of diagnosis of aflatoxin depends upon clinical signs, mortality rate and postmortem examination. [8] did evaluation of pathological changes in broilers during chronic aflatoxin (50 and 100 ppb) and clinoptilolite exposure, the study was on one day old chicks until 42 days of age.[9]studied Histopathological alterations in Aflatoxicity and its amelioration with herbomineral toxin binder in broilers of sixty day old of either sex twenty birds treated with aflatoxin B1 with 1ppm, 40% of birds were sacrificed at sixth week for necropsy examination.[10]reported production of aflatoxin from Aspergillus flavus and acute aflatoxicosis in young broiler chicks.the study was on 7 days old chicks offered feed containing 0, 1600, 3200 and 6400 µg/ kg aflatoxin B1 for 7 days. Clinical signs were depression, ruffled feathers, decreased interest in feed, increased water intake and soft to watery feces. A significant decrease in body weight especially at6400 µg/kg aflatoxins B1 in feed. Pathological lesions pallor discoloration of liver and enlargement of liver and kidneys, Hemorrhages on different parts of the body.[11]studied the effect of ozone-treated aflatoxin contaminated diets on DNA damage, expression of androgen and androgen receptor genes and histopathological changes in japanese quail, sixty, three-week old male quail divided randomly into 6 experimental groups (10 birds per group), one group fed diet contaminated with aflatoxin B1, 1mg/kg diet, those birds showed significantly higher DNA damage(micro nucleated poly chromatic erythrocytes formation and DNA fragmentation and lower expression pattern of androgen gene and its receptor.[12] White Leghorn layer breeder hens, 30 weeks of age, the study was divided into 12 groups ,one group was offered feed supplemented with 100,500,2500,5000 and 10000 µg/Kg aflatoxin B1 (AFB1), the experimental feeds for three weeks with two weeks recovary, body weight and relative weights of liver and kidneys of aflatoxin fed birds were significantly higher than control group.[13] Aflatoxins in wild bird foods, as background, they adviced that aflatoxins are naturally-occurring toxicants produced by certain moulds on food and feed commodities grown in warm, humid conditions Aflatoxin B1, the most toxic aflatoxin, is a potent carcinogen and there is evidence that it is a genotoxic human carcinogen (ie a chemical agent that adversely modifies DNA).[14] studied the Reduction of Toxic Effects of Aflatoxin B1 by Using Baker Yeast (Saccharomyces cerevisiae) in Growing Broiler Chicks Diets, using 200 ng/g of alatoxin B1, the study was on five groups of 20 chicks each, one group only treated with aflatoxin, the above group showed reduced food consumption and body weight and with hisopathological changes in the liver.[15] studied the Effects of Aflatoxin B1 and Fumonisin B1 on Blood Biochemical Parameters in Broilers.

MATERIALS AND METHODS

Experimental design

Forty chick broiler of 14 days of mixed sexes were obtained from the local market of basrah city in Iraq, individually weighted, divided in four groups of 10 birds per group, housed in experiment room and continuous lighting. The birds were randomly assigned to the following treatment groups.the first group, control diet without additive treatment, group two as low dose group treated with diet contaminated with aflatoxin B1 at 3.25 ml of 1 part per million(1ppm) for every 250gm of feed, group three as intermediate dose group fed on contaminated diet as aflatoxin B1 at 6.5ml of 1 part per million(1ppm) mixed with 250 gm of feed and group four as high dose group were fed 13 ml of 1 part per million(1ppm) of aflatoxin B1 in 250 gm of feed, feed and water were provided for ad libitam consumption. Chicks were reared in wire cages per group for six weeks and fed a typical broiler diet. Diets were designed to satisfy the bird needs feed consumption efficiency and body weight were weekly determined. Dead birds were daily recorded. All chicks were sacrified at the end of six weeks of treatment. After sacrifice, there will be microscopic examination for all visceral organs especially liver and kidneys and any signs of hemorrhage on surface of any organ, after that samples will be taken from visceral organs including liver and kidney, those will be fixed in 10% neutral buffered formalin after fixation for at least one weeks, those sample will be embedded in liquid be made, those will cut on rotary microtome slides will be prepared and cut at 5 µm, then they will be stained with hematoxylin and eosin and histopathological changes will be reported for every organ and any abnormalites.

RESULTS

Histopathological changes of the study were revealed septal fibrosis in liver of high group as shown in figure (1) and (2), vacuolated hepatocyte and subcapsular region of liver of high dose group as shown in figure (3), liver congestion of portal vein and periportal fibrosis of high dose group as shown in figure (4) and(5), diffuse vacuolation and congestion of high dose group in figuer (6) and (7), while intermediate group vacuolated arround central vein and focus mononeuclear cells as shown in figure (8) and bile duct proliferation of liver in figure (9) and(10). In kidney, vacuolation of renal corticle tubules and atrophy glomerular were also noticed of high dose group in figure (11), congestion, necrosis and degenerate/vacuolate corticle tubules were shown in figure (12),(13) and (14), several atrophic glomeruli as show in figure(15), whereas necrosis of corticle tubules and atrophic glomeruli as show in figure (16).

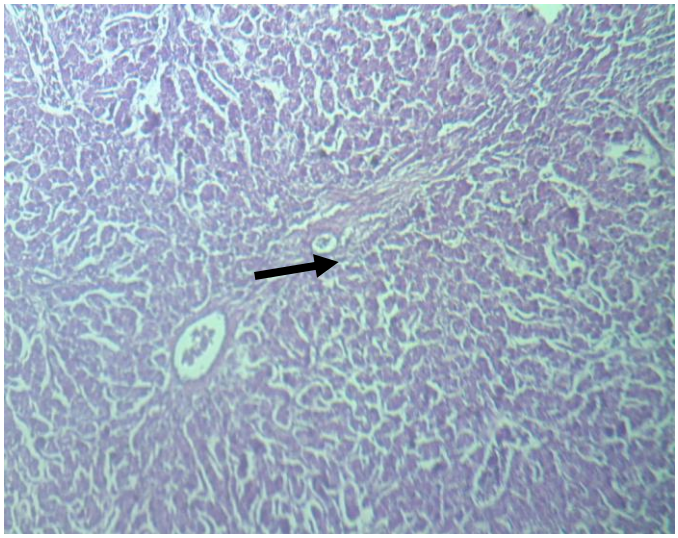

Fig.(1): liver of bird with high dose group show septal fiborosis. (arrows) (H&E stain) (10x).

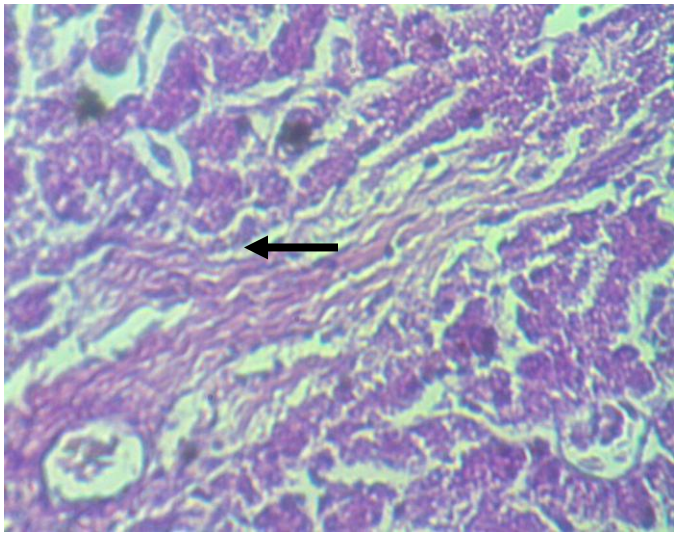

Fig. (2): liver of bird with high dose group show septal fiborosis. (arrows) (H&E stain) (40x).

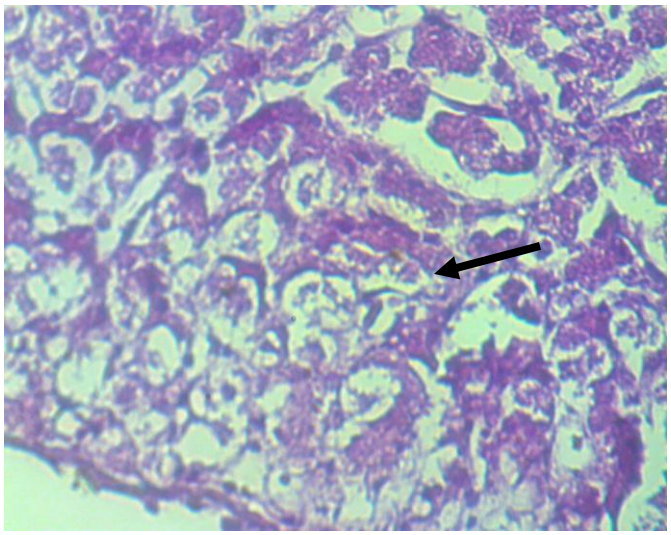

Fig.(3): liver of bird with high dose group show vacuolated hepatocyte and subcapsular region**.** (arrows)(H&E stain) (40x).

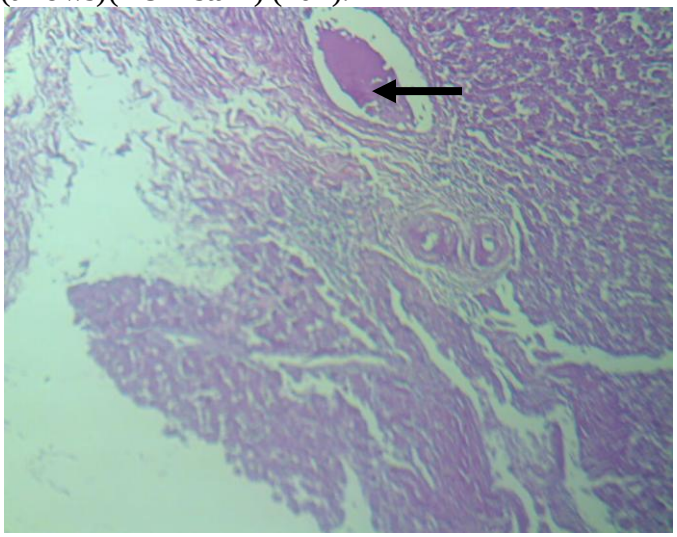

Fig.(4): liver of bird with high dose group show periportal fibrosis and congestion of portal vein. .(arrow) (H&E stain) (10x).

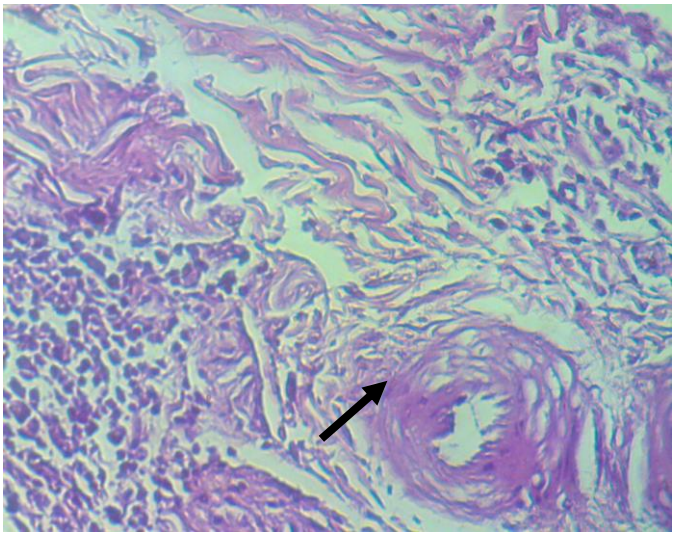

Fig.(5): liver of bird with high dose group show periportal fibrosis and congestion of portal vein.(arrows)(H&E stain) (40x).

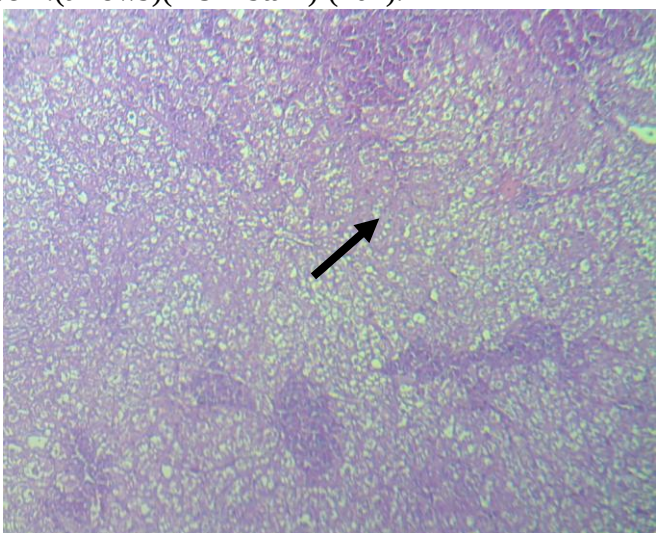

Fig.(6): liver of bird with high dose group show diffuse vacuolation and congestion.(arrows)(H&E stain) (4x).

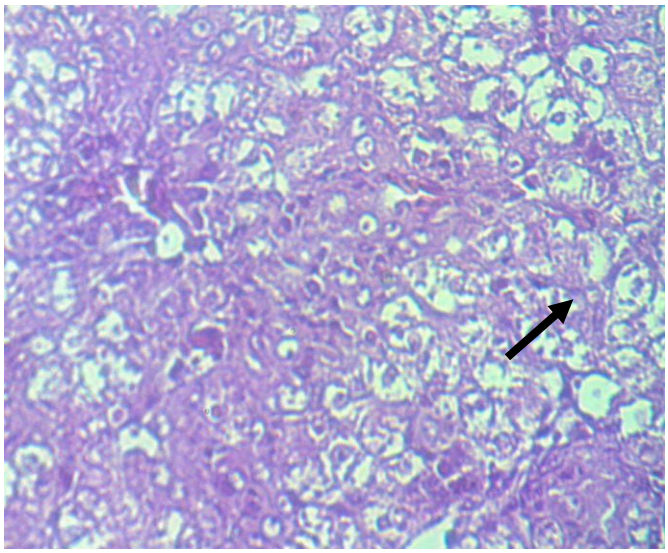

Fig.(7): liver of bird with high dose group show diffuse vacuolation and congestion.(arrows)(H&E stain) (10x).

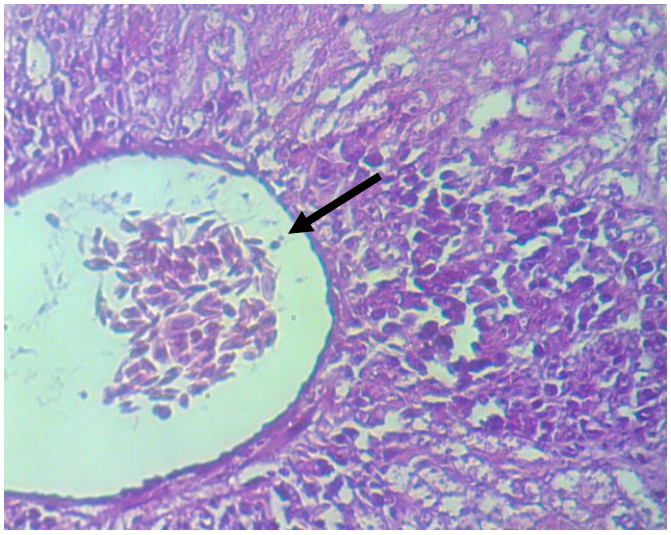

Fig.(8): liver of bird with intermediate dose group show vacuolation arround central vein and focus of mononuclear cells.(arrows)(H&E stain) (40x).

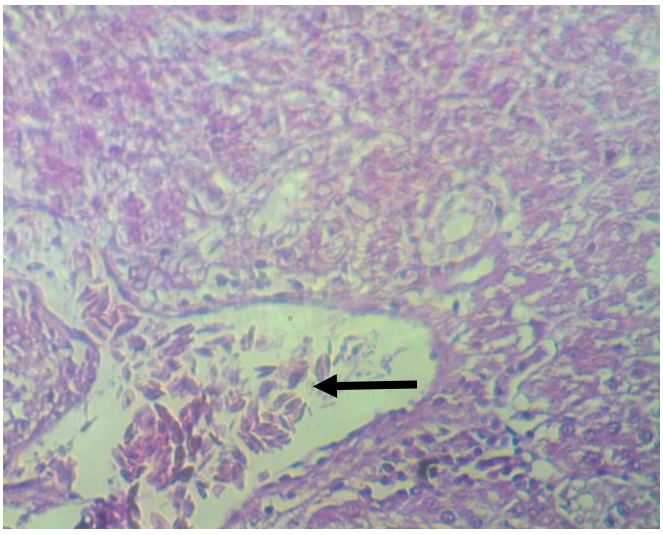

Fig.(9): liver of bird with intermediate dose group show bile duct proliferation.(arrows)(H&E stain) (10x).

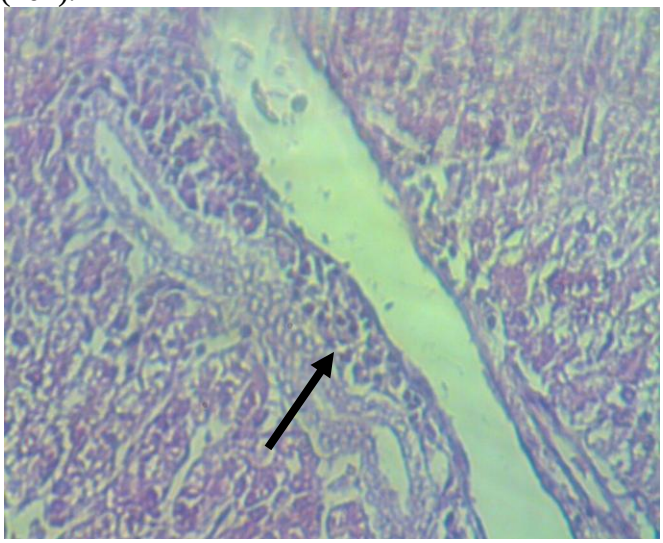

Fig.(10): liver of bird with intermediate dose group show bile duct proliferation.(arrows)(H&E stain) (40x).

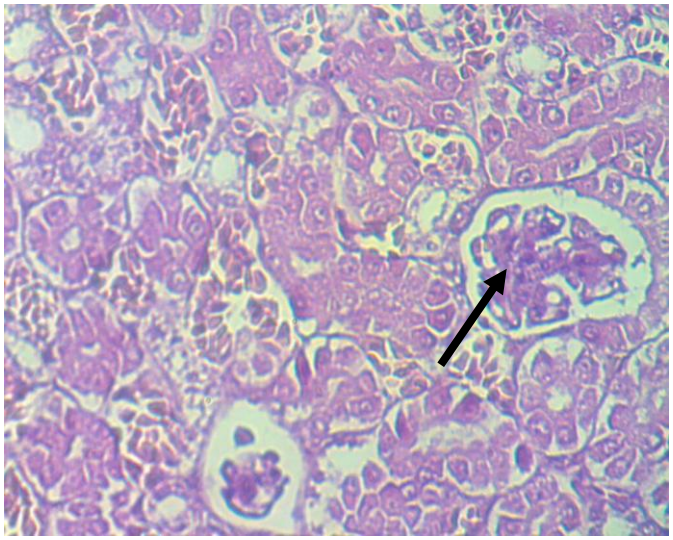

Fig.(11): Kidney of bird with high dose show atrophy of glomerular and vacuolation of renal cortical tubules.(arrows)(H&E stain) (40x).

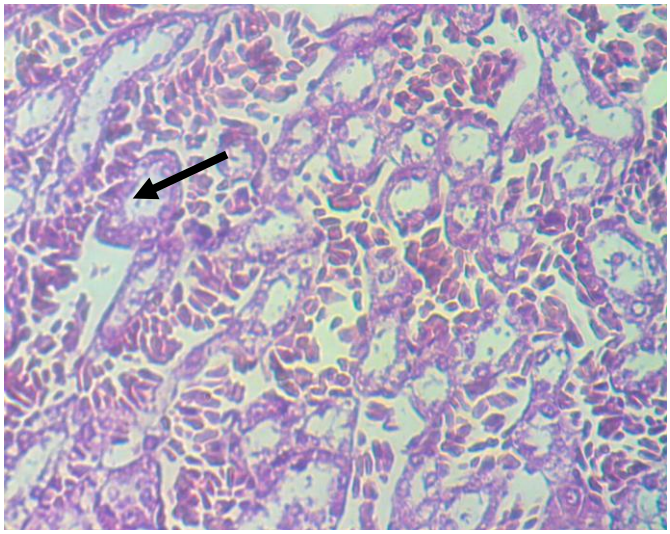

Fig.(12): kidney of bird with high dose group show congestion and degeneration of corticle tubules. (arrow) (H&E stain) (40x).

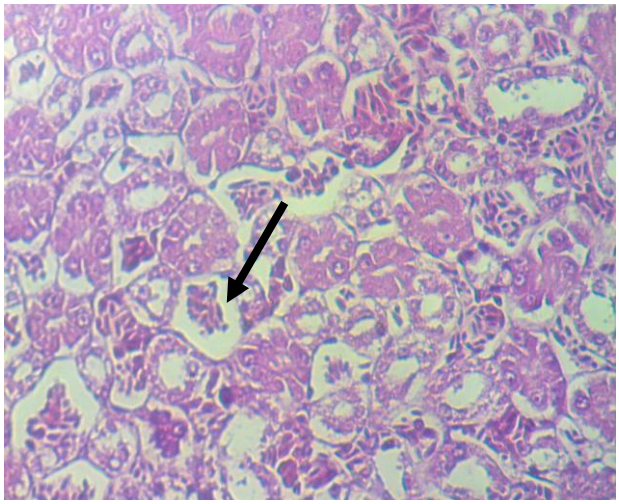

Fig.(13): kidney of bird with high dose group show necrosis and degeneration of corticle tubules. (arrow) (H&E stain) (40x).

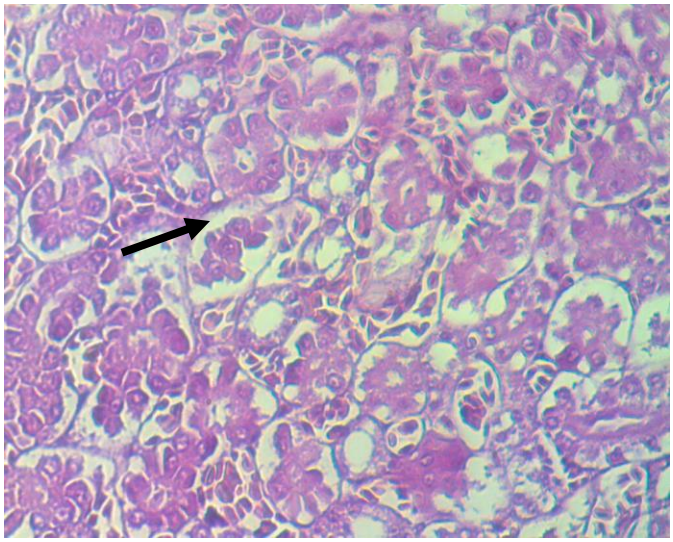

Fig.(14): kidney of bird with high dose show degenerate/vacuolate of corticle tubules. (arrow) (H&E stain) (40x).

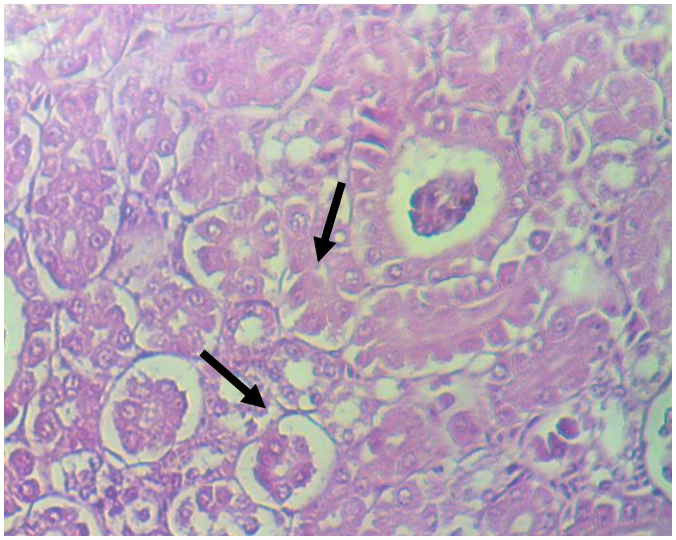

Fig.(15): kidney of bird with high dose group show several atrophic glomeruli. (arrow) (H&E stain) (40x).

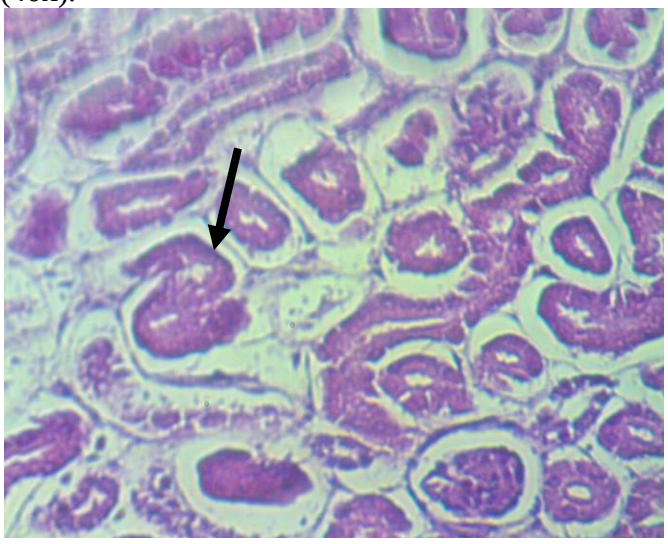

Fig.(16): kidney of bird with high dose group show necrosis of corticle tubules and atrophic glomeruli. (arrow) (H&E stain) (40x).

DISCUSSION

The present study showed toxic effects of aflatoxin B1 on liver and kidney to broiler chiks when exposure in diet for short time. the above was supported by[1],where they found of hepatic aflatoxin residues in British wild birds: two passerine species, the house sparrow (Passer domesticus) and greenfinch (Carduelis chloris). [2] reported an outbreak of liver cell carcinoma in rainbow trout salmo gairdneri Richardon their study agreed with the present study that aflatoxin B1was hepatic toxic associated with affected liver, were enlarged with hemorrhage and necrosis with varying degrees of involvment by liver cell carcinoma with local invasion , necrosis and hemorrhage, metastasis of liver cell carcinoma in occasional fish to spleen and kidney[3]showed enlarged yellow discoloration of liver, also thickening of crop and necrosis and thickening of proventricular mucosa, histopatho-logycally, degeneration and necrosis were seen in liver, kidneys, intestine, pancreas, heart, pectoral muscle, spleen and bursa of fabricius of all toxin fed birds. The above change agreed with the present toxicity study as their was fatty changes of hepatocytes with areas of liver cell necrosis and areas of hemorrhage, also renal lesions with tubular necrosis mostly of proximal convoluted tubules. [4] showed the pathologic changes observed in the livers of different species after a single dose and after continuous administration of aflatoxin. The above change agreed with the present toxicity study as their was showed pathologic changes observed in the livers of broiler chicks after a single dose and after continuous administration of aflatoxin.[5] reported that The various changes are discussed in

relation to the possible effects of AFB1 on treated animals. the above agreed with the present paper that their was effect of AFB1 by various changes on many organs of the bird. [6]showed fatty changes as fatty liver with some hemorrhage and necrosis, while those of 1000 and 2000 ppb groups showed bile ducts proliferation and severe necrosis, the number of birds displaying necrosis of liver increased with increasing level of aflatoxin and that will support the findings in the present paper the effects of aflatoxin B1 as a hepatic toxic associated with affected liver, were enlarged with hemorrhage and necrosis.[7]in their studies on liver histopathology affected with aflatoxin in broiler chicks showed grossly liver was enlarged with congestion and pale patches microscopically hyperplasia, congestion, necrosis, cerhosis and peri central vein of RBCs and inflammatory cells. The above paper supported the findings the present paper, which showed histopathological changes. [8] reported histopathologically changes ranged from slight to moderate hydropic degeneration and /or fatty change, bile duct hyperplasia and periportal septal fibrosis found in chicks fed 100 ppb aflatoxin- containning diet and that could support the liver induced by aflatoxin B1 in the present paper.[9]reported histotoxicologic alteration induced by aflatoxin and that would support the histopathologic changes induced by aflatoxin B1 in the present paper.[10] reported microscopically, congestion of liver parenchyma, cytoplasmic vaculation/fatty change of hepatocytes, necrosis of hepatocytes, newly formed bile ducts, mononuclear and hetrophilic cell infiltration ,Kidneys of aflatoxins B1 intoxicated birds were enlarged and microscopically revealed degeneration and necrosis of tubular epithelial cells, congestion and hemorrhages of the parenchyma and clinical, gross and histopathological lesions were dose related. those were in agreement with the toxic findings with aflatoxin B1 on the liver and kidney of the present paper.[11]showed histopathologically revealed severe lesions of liver and testis, the reduction in expression of androgen and its receptor was associated with reduction testis weight. the above agreed with the present paper that their was effect of AFB1 on liver only with out testis.[12] showed Pathological lesions in aflatoxin (AF) fed birds included enlarged, pale and friable liver, swollen kidneys andhemorrhages on different organs. Histopathological lesions in liver included fatty change, congestion and hemorrhages, while in kidneys tubular necrosis, cellular infiltration, congestion and hemorrhages were found in groups fed AFB1 at 500 µg/Kg and higher doses. In AF fed hens, no significant ameliorative effects of vitamin E could be observed upon AF induced decrease in feed intake, gross pathology and histopathological alterations and organ weight except body weights. The above paper supported the findings the present paper, which showed histopathological lesions in liver and kidney.[13] showed that birds such as chickens, ducks and turkeys are more susceptible than mammals to adverse effects from aflatoxins. The above paper supported the findings the present paper, which showed effect of aflatoxin B1 on broiler chicks.[14]showed hemorrhage in liver, macroscopically liver was enlarged with yellow discolaration. their study agreed with the present study that aflatoxin B1was hepatic toxic associated

with affected liver, were enlarged with hemorrhage.[15] showed that aflatoxin B1 caused liver damage. The above paper supported the findings the present paper, which showed that aflatoxin B1 caused liver damage.

CONCLUSIONS

THE OUTBREAK OF hepatic toxic suggesting strong circumstantial evidence for diet being a factor in outbreak and increased suseptibility of birds to aflatoxin B1 induced hepatic toxic suggesting contamination of the diet by aflatoxin B1.

REFRENCES

1. B. Lawson , S. MacDonald, T. Howard, S.K. Macgregor and A.A. Cunningham (2006). Exposure of garden birds to aflatoxins in Britain. Sci. of the Total Enviro, 361: 124–131.
2. S.K.Majeed, D.W.Jolly and C.G. opinath(1984). An outbreak of liver cell carcinoma in rainbow trout salmo gairdneri Richardson in UK. J. Of fish dis., 71:165-168.
3. R. Kumar, and Ch. Balachandran (2009). Histopathological changes in broiler chickens fed aflatoxin and cyclopiazonic acid. Veterinarski Arhiv, 79, (1): 31-40.
4. P.M. Newberneand and W.H. Butler (1969). Acute and Chronic Effects of Aflatoxin on the Liver of Domestic and Laboratory Animals. Cancer Research, 29:236-250.
5. A. Faridha, K. Faisal, and M.A. Akbarsha (2006).Duration-dependent histopathological and histomtric changes in the testis of aflatoxin B1-treated mice. J. Endocrinol Reprod, 10, (2): 117-133.
6. D. L. Moore, S. E. Henke, A. M. Fedynich, J.C. Laurenz and R. Morgan(2013). Acute effects of aflatoxin on northern bobwhite(Colinus Virginianus). J. of Wildlife Dis., 49,(3): 568–578.
7. S. A. Lafi, N. A. Taha and S. M.H. Al-Genabi (2010). Histopathology of the liver affectedWith Aflatoxins in broiler chicks. Al- Anbar J. Vet. Sci., 3, (1):115-119.
8. A. M. Ortatatli, B. H. Ogˇuz, A. F. Hatipogˇlu And M. Karaman, (2005). Evaluation of pathological changes in broilers during chronic aflatoxin (50 and 100 ppb) and clinoptilolite exposure. Research in Veter. Sci., 78 : 61–68.
9. M.A.E. Ahmed, K. Ravikanth, D.S. Rekhe and S. Maini (2009). Histopathological alterations in Aflatoxicity & its amelioration with herbomineral toxin binder in broilers. Veterinary World, 2,(10):390-392.
10. Z. Hussain, M. Z. Khan and Z. Al Hassan (2008). production of aflatoxin from Aspergillus flavus and acute aflatoxicosis in young broiler chicks. Pak. J. Agri. Sci., 45,(1):95-102.
11. G. E. Mariam, M. D. Mohamed, H. F. Abdel Razik, M. F. Ibrahim and K. R. S. Farid (2013). Effect of ozone-treated aflatoxin contaminated diets on DNA damage, expression of androgen and androgen receptor genes and histopathological changes in japanese quail. Global Veterinaria, 11, (1): 01-13

12. A.Wajid, K. M. Zargham, K.Ahrar and H. Iftikhar, (2010). Pathological Effects of Aflatoxin and Their Amelioration by Vitamin E in White Leghorn Layers. Pak Vet. J., 30,(3): 155-162.
13. Advisory Committee on animal feedingstuff. ACAF (2001) . Aflatoxin in wild birds foods. Twelfth Meeting of ACAF on 5 December – Agenda Item 6.
14. K. ÇELÝK, M. DENLÝ and T. SAVAS (2003). Reduction of Toxic Effects of Aflatoxin B1 by Using Baker Yeast (Saccharomyces cerevisiae) in Growing Broiler Chicks Diets. R. Bras. Zootec, 32,(3):615-619.
15. E. N. C. Tessari, E. Kobashigawa, A.S. P. Cardoso, D. R. Ledoux, G. E. Rottinghaus and C. A. F. Oliveira (2010). Effects of Aflatoxin B1 and Fumonisin B1 on Blood Biochemical Parameters in Broilers. Toxins , 2:453-460.