Original Article
Protective effect of curcumin against experimentally induced aflatoxicosis on the renal cortex of adult male albino rats: a histological and immunohisochemical study

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Abstract: Background: Aflatoxin contamination of foods is a worldwide problem. Chronic aflatoxin exposure is associated with kidney damage. Curcumin is a herbal agent, used in medicine with a wide range of beneficial therapeutic effects. Objective: to evaluate the effect of curcumin against experimentally induced aflatoxicosis on the renal cortex of adult male albino rats. Materials and methods: Forty adult male rats were included and they were divided equally into 4 groups (10 rats each): Group I (control group), group II (Curcumin group): The rats received curcumin (200 mg/kg b.w.) orally by gastric tube for 5 days/week, group III (Aflatoxin B1 group): The rats received aflatoxin B1 (250 µg/kg b.w./day) orally by gastric tube 5 days/week for 4 weeks, group IV (Aflatoxin B1 and Curcumin group): The rats received aflatoxin and curcumin orally by gastric tube 5 days/week for 4 weeks. Kidney specimens were prepared and sections were stained with hematoxylin and eosin, Masson's trichrome, Periodic acid Schiff, immunohistochemical detection of desmin and Bcl2. Results: The tubules of group III showed degenerative and necrotic changes with disruption of basal lamina. There was a significant decrease Bcl2 expression in the tubules, but the glomeruli showed an enlargement with dilation of their capillaries lumina in some areas, while the other areas showed glomerular atrophy with obliteration of their capillaries lumina. There was a significant increase in desmin expression in the glomerular cells. The interstitium showed hemorrhage and cellular infiltration. Group IV showed improvement of the histological and immunohistochemical changes described before. Conclusion: Aflatoxin B1 has deleterious effects of on the histological structure of the rat's renal cortex and curcumin minimized these effects as it has antioxidant, anti-inflammatory and antiapoptotic activities. We advise eating nutritious diets that contain sufficient amounts of curcumin and regulation must implement to avoid the presence of aflatoxins in high concentrations in human food.

Keywords: Aflatoxin B1, curcumin, kidney, desmin, Bcl2

Introduction

Kidney is an important organ because of its role in getting rid of harmful materials and excretions of drugs and body waste products by highly specialized cells and also, due to its large blood flow [1, 2]. Urinary tract can be affected by bacteria, fungi, and contaminated food [3, 4].

Aflatoxins (AFs) are known as significant mycotoxins produced by Aspergillus flavus, Aspergillus parasiticus, and other Aspergillus species. There are four natural aflatoxins, aflatoxin B1, aflatoxin B2, aflatoxin G1, and aflatoxin G2. Aflatoxin B1 is the most prevalent and toxic with acute toxicity demonstrated in all species of animals [5].

Mycotoxin contamination is a worldwide problem. Up to 25% of the world's food crops and food obtained from animals are significantly contaminated. It is more obvious in developing countries [6].

The consumption of aflatoxins can cause serious health problems such as growth retardation, severe liver, heart and kidney damage, hemorrhage and death [7, 8]. Aflatoxins are of great concern as carcinogenic which classified as group I human carcinogen, mutagenic and immunosuppressive substances [8].

There is recent trend worldwide to go back to traditional medicinal plants, as natural products of herbal origin, such as ginger and curcumin, cinnamon which have antifungal, antiaflatoxigenic and antioxidant activity against the damaging effects of aflatoxin B1 on some organs [9, 10].

Curcumin, a yellow pigment from Curcuma longa, is a major component of turmeric and one member of the ginger family, Zingaberaceae, and is commonly used as a spice and food-coloring material. It has a wide range of pharmacological and physiological actions such as antioxidant, radioprotective, antibacterial, antifungal, antiviral, antiinflammatory, antialfatoxogenic, antiproliferative, proapoptotic and antiatherosclerotic effects. Also it exerting medicinal benefits for nephrotoxicity, arthritis, allergy, asthma, inflammatory bowel disease, psoriasis, diabetes, Alzheimer's disease, multiple sclerosis, cancer, neurodegenerative and cardiovascular disease [11-13].

Aim of this study to evaluate the effect curcumin against experimentally induced aflatoxicosis on the renal cortex of adult male albino rats.

Materials and methods

In this study, 40 adult male rats of average weight 150-250 g were used. The animals were housed in the animal laboratory at the medical research center of Benha faculty of medicine under standard environmental conditions with free access to standard basal diet and liberal supply of tap water. All ethical protocols for animal treatment were followed and were supervised by the animal facilities. The experimental protocol was approved by the Ethical Committee of Benha faculty of medicine.

Used drugs

Aflatoxin was purchased from Sigma Chemical Company (St Louis, Missouri, USA). It is a white to faint yellow odorless powder. It is a light-sensitive mycotoxin, which should be stored at a temperature of 2-8°C in amber glass vials. It should be transferred for weighing with marked caution to prevent dissemination to the surroundings. Aflatoxin B1 was dissolved in olive oil as a vehicle.

Curcumin was purchased from Sigma Company (Cat No. C7727; St Louis, Missouri, USA), it is bright yellow to orange color powder, insoluble in water but soluble in other solvents such corn oil. Curcumin was dissolved in corn oil as a vehicle.

Experimental design

Rats were divided into 4 groups included 10 rats for each.

Group I (Control group): The animals of this group were further subdivided into 2 subgroups each one included 5 rats.

Subgroup IA: Rats received olive oil as a vehicle orally by gastric tube for 4 weeks. They were sacrificed at the same time as the corresponding experimental groups.

Subgroup IB: Rats received corn oil as a vehicle orally by gastric tube daily for 4 weeks. They were sacrificed at the same time as the corresponding experimental groups.

Group II (Curcumin group): Rats received curcumin 200 mg/kg body weight dissolved in corn oil as a vehicle orally by gastric tube 5 days/week for 4 weeks [14-16].

Group III (Aflatoxin B1 group): Rats received 250 μg/kg body weight/day of aflatoxin B1. Aflatoxin B1 dissolved in olive oil as a vehicle by gastric tube 5 days/week for 4 weeks [17].

Group IV (Aflatoxin B1 and Curcumin group): Rats received aflatoxin and curcumin as in the previous groups by gastric tube 5 days/week for 4 weeks.

The blood was drained from the tail vein by using capillary tubes immediately before the end of the experiment into Eppendorf tubes containing heparin (20 ml, 200 IU/ml). The plasma was separated by centrifugation (5000 rpm for 5 min). Biochemical analysis of serum urea, creatinine, uric acid and total protein were carried out using the commercially available standard kits BioAssay System (Hayward, CA94545, USA). All the assays were performed according to the manufacturer's instructions.

At the end of the experiment, the rats were euthanized by cervical decapitation. Portions of kidney tissues were homogenized in a saline solution (0.9%), centrifuged at 3000 rpm for 15 min, and the supernatant was stored at -20°C and used for the detection of lipid perioxidation malondialdehyde (MDA) [18] and antioxidants enzyme as reduced glutathione (GSH) [19].

Table 1. Showing changes in serum urea, creatinine, uric acid and total protein in all experimental groups

	Group I	Group II	Group III	Group IV
Serum urea (mg/dl)	22.04 ± 1.87	24.60 ± 1.82	63.90 ± 5.28**	35.45 ± 2.38*
Serum creatinine (mg/dl)	0.70 ± 0.12	0.73 ± 0.15	2.60 ± 0.13**	0.95 ± 0.05*
Serum uric acid(mg/dl)	1.33 ± 0.048	1.40 ± 0.035	2.83 ± 0.092**	1.57 ± 0.096*
Total protein (mg/dl)	11.83 ± 1.23	10.71 ± 1.20	4.66 ± 0.24**	9.95 ± 0.74*

Values are presented as mean ± SD; Group III compared with group I, **$P < 0.01$; Group IV compared with group III, *$P < 0.05$.

Table 2. Showing changes in the malondialdehyde (MDA) and reduced glutathione in all experimental groups

	Group I	Group II	Group III	Group IV
MDA nmol/g tissue	18.2 ± 1.25	19.7 ± 1.60	36.2 ± 2.37**	15.3 ± 2.23*
GSH µg/g tissue	2.90 ± 0.19	2.85 ± 0.28	1.30 ± 0.15*	2.70 ± 0.28*

Values are presented as mean ± SD; Group III compared with group I, **$P < 0.01$; Group IV compared with group III, *$P < 0.05$.

Specimens of kidney were taken. Paraffin sections were prepared and stained with hematoxylin and eosin (H&E), Masson's trichrome [20], Periodic acid Schiff (PAS) [21] and immunohistochemistery detection of desmin and Bcl2.

Immunohistochemistry study of desmin and Bcl-2

The paraffin-embedded kidney tissue was cut into 5-µm thick sections, deparaffinized, and incubated in 3% H_2O_2 for 10-min to quench endogenous peroxidase activity. After blocking with normal goat serum for 20-min, the sections were stained with a mouse anti desmin monoclonal antibody (Lab Vision Corp, Neomarkers Inc/Lab Vision, Fremont, California, USA) {1:100 dilution} at 4°C overnight and mouse anti Bcl2 monoclonal antibody (Santa Cruz Biotechnology, CA, USA) {1:100 dilution} at 4°C overnight, followed by incubation with a horseradish-peroxidase-conjugated goat anti-mouse antibody at 37°C for 30-min. The antibody binding sites were visualized by incubation with diaminobenzidine (DAB)-H_2O_2 (Dakopatts, Glostrup, Denmark) at room temperature for 10-min. Sections were counterstained with hematoxylin dehydrated, rendered transparent with xylene, mounted and observed under a light microscope. For the negative control the specific primary antibody was replaced by phosphate-buffered saline.

Morphometric study

The mean area percentage (%) of desmin and Bcl2 reaction were quantified in 10 images for each group using Image-Pro Plus program version 6.0 (Media Cybernetics Inc., Bethesda, Maryland, USA). Mean area percentage of desmin and Bcl2 reaction in group III (Aflatoxin B1 group) compared with group I (control group) and group IV (Aflatoxin B1 and Curcumin group) compared with group III (Aflatoxin B1 group) using the t-test, with $P < 0.05$ as the level of statistical significance. Statistical analysis were carried out using IBM SPSS Statistics software for Windows, Version 20 (IBM Corp., Armonk, NY, USA).

Results

Biochemical results

As shown in **Table 1**: serum urea, creatinine and uric acid were highly significantly increased ($P < 0.01$) in group III compared to control group I, but were significantly decreased in group IV compared to group III < 0.01. Total protein was significantly decreased in highly group III compared to control group I, but was significantly increased in group IV compared to group III ($P < 0.05$).

As shown in **Table 2**: The MDA was highly significantly increased < 0.01 in group III compared to control group I, but were significantly decreased in group IV compared to group II ($P < 0.05$). Reduced GSH was significantly decreased in group III compared to control group I, but was significantly increased in group IV compared to group III ($P < 0.05$).

Histological results

Group I: The renal cortex of the control rat showed normal histological appearance of renal corpuscles which formed of tuft of capillaries, and enclosed in a Bowman's capsule.
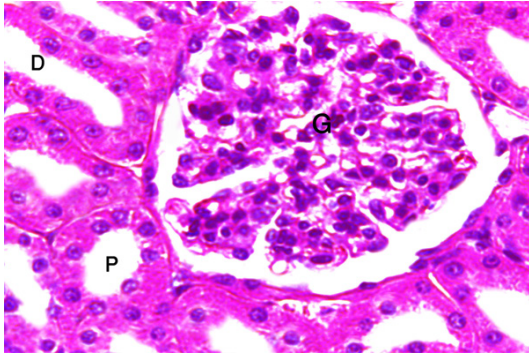
Figure 1. A photomicrograph of a section in the renal cortex from control group showing renal glomeruli (G), proximal convoluted tubules (P) and distal convoluted tubules (D) (H&E × 630).
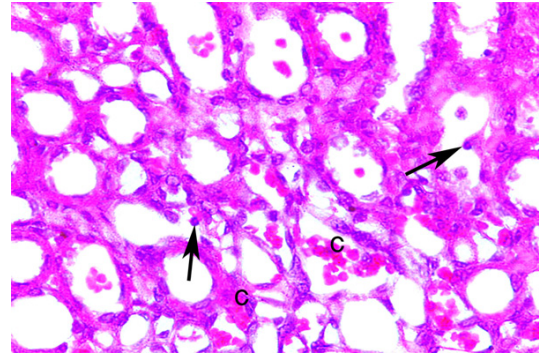

Figure 3. A photomicrograph of a section in the renal cortex from group III showing necrosis of epithelium lining the convoluted tubules with pyknotic nuclei (↑). The lumen of some tubules have hemorrhage and cellular casts (C) (H&E × 630).
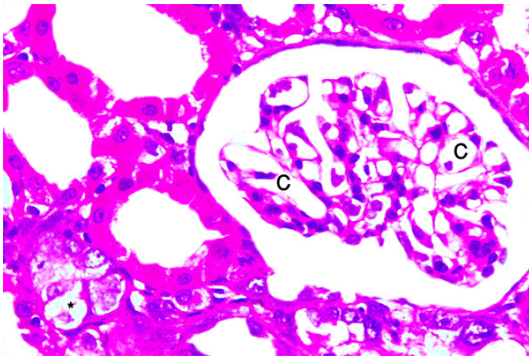

Figure 2. A photomicrograph of a section in the renal cortex from group III showing dilation of the glomerular capillaries (C). Some tubular cells show degenerative changes and karyolytic nuclei (*) (H&E × 630).
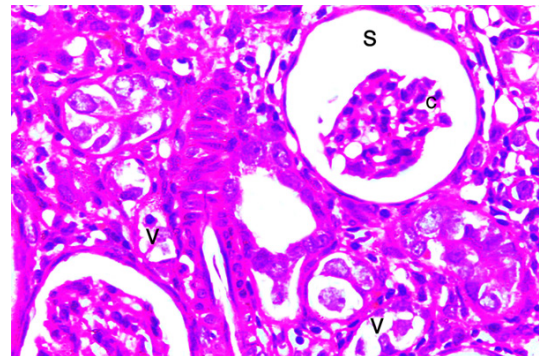

Figure 4. A photomicrograph of a section in the renal cortex from group III showing atrophy of glomerular capillaries (C) with widening subcapsular space of glomeruli (S). While renal tubules were degenerated and had vacuolated cytoplasm (V) (H&E × 630).

The Bowman's capsule is formed of two layers, the inner visceral layer and the outer parietal layer which is formed of squamous layer separated by urinary space Proximal convoluted tubules with narrow lumina were numerous and lined with simple cubical epithelium and basal spherical nuclei. Distal convoluted tubules with wide lumina were less numerous and lined with simple cubical epithelium and basal spherical nuclei (**Figure 1**). Group II no histological changes were detected in the group II as compared to that of the control group.

Group III showed obvious histological changes which appeared in the form of degeneration of the tubules which vary from cloudy swelling in which their cells were enlarged with pale vacuolated cytoplasm. Necrosis of some tubules in which their cells had pyknotic or karyolytic nuclei (**Figures 2**, **3**). The lumen of some tubules

showed hemorrhage and cellular casts (**Figure 3**). Some areas showed an enlargement of the glomeruli with dilation of their capillaries lumina and decreased glomerular cellularity (**Figure 2**). Other areas showed atrophy of glomeruli with obliterating their capillaries lumina (diminution of their size) and widening of subcapsular spaces (**Figure 4**). Other glomeruli showed sclerosis characterized by consolidation (solidification into a firm dense mass) of the glomerular tuft by increased extracellular matrix, obliterating the glomerular capillary lumina with mesangial expansion (**Figure 5**). Interstitial hemorrhage was observed between degenerated convoluted tubules (**Figure 6**). An inflammatory cells infiltration between degenerated tubules and renal corpuscles were seen (**Figure 7**). Group IV showed picture nearly similar to the control group, there were an apparent
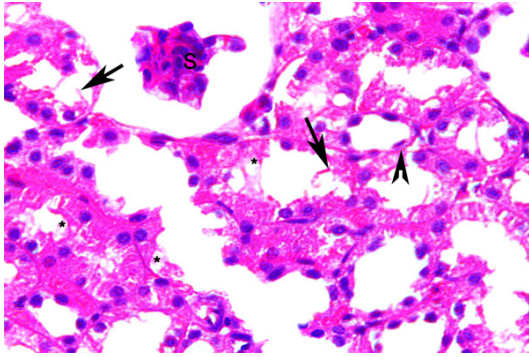
Figure 5. A photomicrograph of a section in the renal cortex from group III showing atrophy and sclerosis of some glomeruli (S). Some renal tubules show epithelial denudate (↑), while others show loss of nuclei, karyolysis (*) and focal epithelial flattening (arrow head ▲) (H&E × 630).
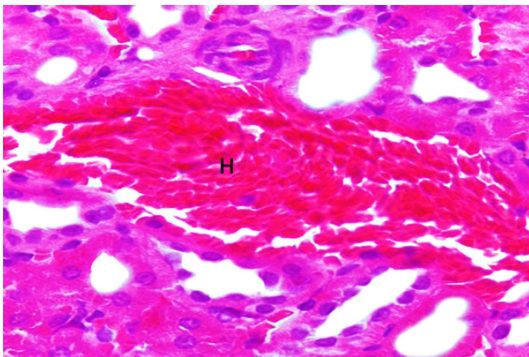

Figure 7. A photomicrograph of a section in the renal cortex from group III showing inflammatory cells infiltration between degenerated tubule and renal corpuscles (I) (H&E × 630).

Figure 6. A photomicrograph of a section in the renal cortex from group III showing interstitial hemorrhage between convoluted tubules (H) (H&E × 630).

Figure 8. A photomicrograph of a section in the renal cortex from group IV showing apparent hypercellularity of glomeruli (G) and regeneration of most convoluted tubules (T). **Notice evidence of mitotic ac**tivity of some tubular cells (↑) (H&E × 630).

hypercellularity of the glomeruli. Most convoluted tubules were regenerated. The evidence of the mitotic activity of tubular cells (many binucleated cells) were seen (**Figure 8**).

Masson's trichrome staining

Group I: The renal cortex of the control rat showed minimal amount of collagen around the renal tubules, Bowman's capsules and capillary tuft of glomeruli (**Figure 9**). Group II no detectable changes were observed in the distribution of collagen fibers in the renal cortex in the group II compared to that of the control group. Group III showed an apparent increase of collagen fibers around the degenerated convoluted tubules, Bowman's capsules and capillary loops of glomeruli (**Figure 10**). Group IV showed apparent scanty collagen fibers around swollen glomeruli and regenerated convoluted tubules (**Figure 11**).
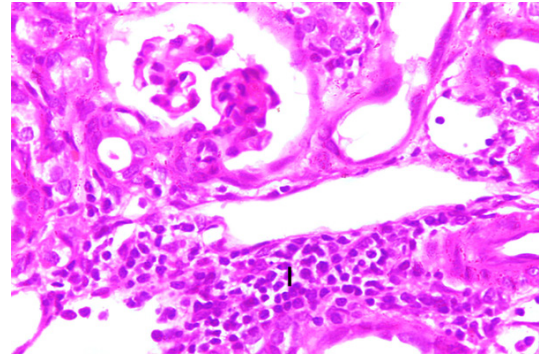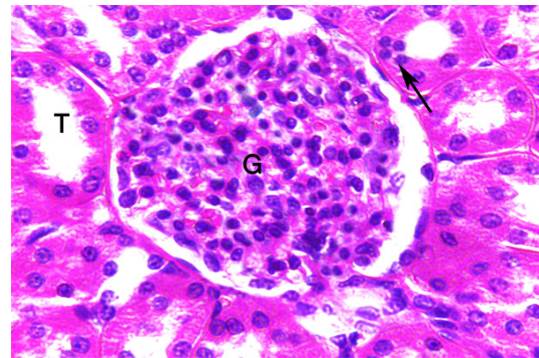
Periodic acid Schiff reaction

Group I: The renal cortex of the control rat showed strong PAS reaction in renal glomeruli as well as basal lamina and brush borders of renal tubules (**Figure 12**). Group II no changes were observed in the intensity of the PAS reaction in the group II compared to that of the control group. Group III showed apparent weak PAS reaction in the disrupted basal luminal, brush borders of renal tubules as well as renal glomeruli (**Figure 13**). Group IV showed an apparent moderate PAS reaction in renal glomeruli as well as basal lamina and brush borders of renal tubules (**Figure 14**).

Immunohisochemical staining of desmin

Group I: The renal cortex of the control rat showed a negative desmin immmostaining in
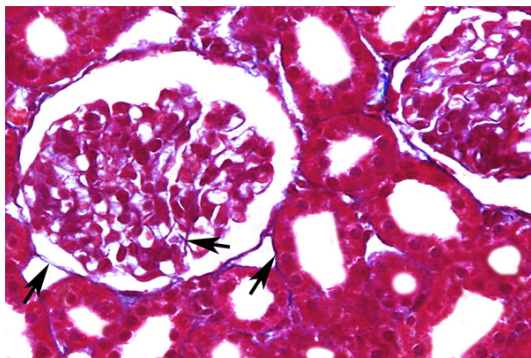
Figure 9. A photomicrograph of a section in the renal cortex from control group showing minimal amount of collagen around renal tubules, Bowman's capsules and capillary tuft of glomeruli (↑) (H&E × 630).
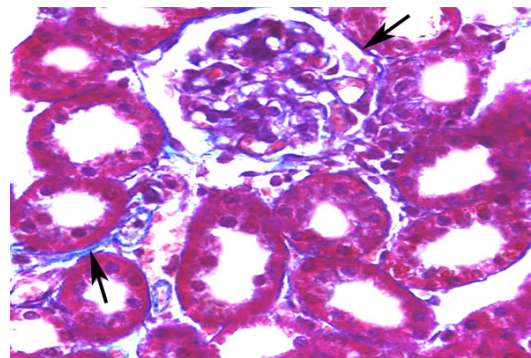

Figure 11. A photomicrograph of a section in the renal cortex from group IV showing apparent scanty collagen fibers around swollen glomeruli and regenerated convoluted tubules (↑) (Masson's Trichrome × 630).
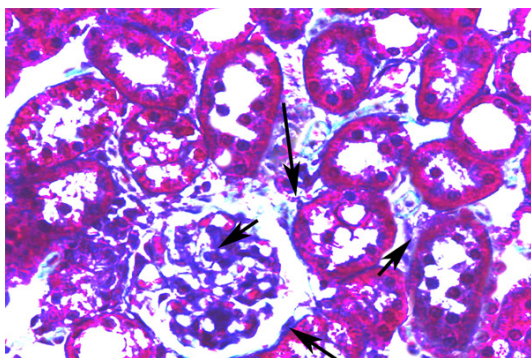

Figure 10. A photomicrograph of a section in the renal cortex from group III showing apparent increase of collagen fibers around degenerated convoluted tubules, Bowman's capsules and capillary loops of glomeruli (↑) (Masson's Trichrome × 630).
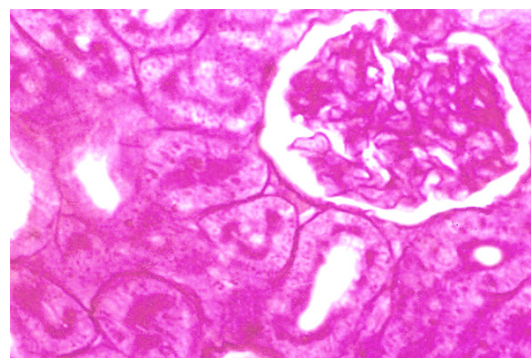

Figure 12. A photomicrograph of a section in the renal cortex from control group showing strong PAS reaction in renal glomeruli as well as basal lamina and brush borders of renal tubules (PAS × 630).

the renal glomerulus, indicating that no podocyte injury (**Figure 15**). Group II no detectable changes were observed to desmin immune reaction in the group II compared to that of the control group. Group III showed positive desmin immunostaining in the renal glomerulus indicating that a remarkable podocyte cytoskeleton injury (**Figures 16**, **17**). Group IV showed decrease of desmin immunostaining in the renal glomerulus (**Figure 18**).

Immunohistochemical staining of Bcl-2

Group I: The renal cortex of the control rat showed intense brown positive Bcl2 immunostaining in the cytoplasm of tubular cells (anti-apoptotic marker) (**Figure 19**). Group II no detectable changes were observed to Bcl2 immune reaction in the group II as compared to the control group. Group III showed weak Bcl2 immunostaining in the cytoplasm of tubular cells. Weak brown color indicating that the presence of apoptosis (**Figure 20**). Group IV showed moderate positive brown Bcl2 immunostaining in the cytoplasm of tubular cells. Increased brown color in group IV compared with group III were observed (**Figure 21**).

Morphometric results

The mean area % of desmin and Bcl2 reaction for all groups were represented in **Tables 3** and **4** and **Figures 22** and **23**. Desmin reaction was significantly increased in group III compared with group I ($P < 0.05$), but was significantly decreased in group IV compared with group III ($P < 0.05$). Bcl2 reaction was significantly decrease in group III compared with group I ($P < 0.05$), but was significantly increased in group IV compared with group III ($P < 0.05$).
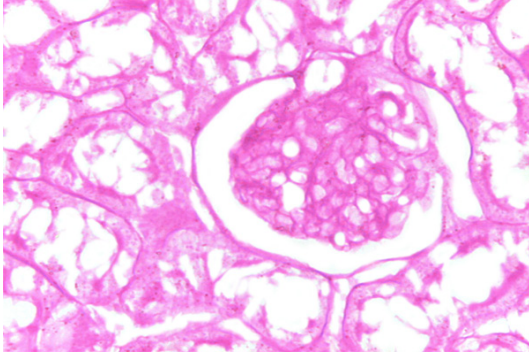
Figure 13. A photomicrograph of a section in the renal cortex from group III showing apparent weak PAS reaction in the disrupted basal lamina, brush borders of renal tubules as well as renal glomeruli (PAS × 630).
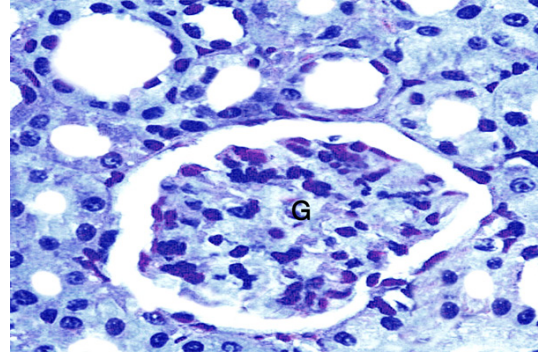

Figure 15. A photomicrograph of a section in the renal cortex from control group showing negative desmin immunostaining in the renal glomerulus (G) (Immunostaining for Desmin × 630).
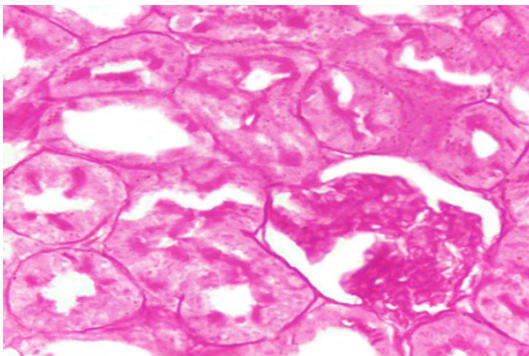

Figure 14. A photomicrograph of a section in the renal cortex from group IV showing apparent moderate PAS reaction in renal glomeruli as well as basal lamina and brush borders of renal tubules (PAS × 630).
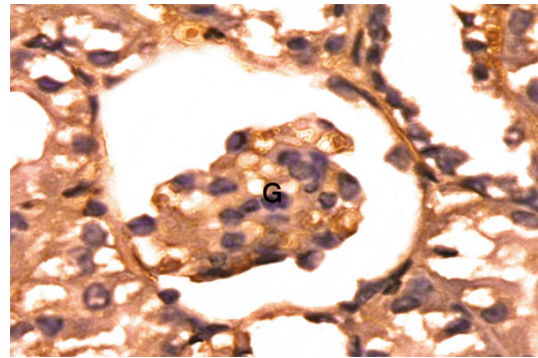

Figure 16. A photomicrograph of a section in the renal cortex from group III showing positive desmin immunostaining in the atrophic renal glomerulus (G) (Immunostaining for Desmin × 630).

Discussion

Aflatoxicosis is the poisoning that results from ingestion of aflatoxins. Two forms of aflatoxicosis have been identified, acute severe intoxication and chronic subsymptomatic exposure associated with various health problems [22].

Our studied group III revealed highly significant increase of serum urea, creatinine and uric acid and a highly significant decrease of total protein as compared with control group. These results were in agreement with the previous studies which stated that the exposure to aflatoxin B1 causing harmful and stressful effect on renal tissue [23-26].

The reduction of total protein could indicate the inhibition of protein synthesis and increase protein catabolism and renal dysfunction [24, 27].

Our studied group III revealed highly significant increase of MDA and a significant decrease of GSH as compared to control group. These results were in agreement with the previous studies [5, 24].

The histological examination of group III revealed that some areas showed enlargement of the glomeruli with dilation of their capillaries lumina. Other glomeruli were atrophied and showed thickness of their wall and obliteration of their capillaries lumina.

These results were in agreement with previous histopathological studies which stated that the exposure to aflatoxin B1 led to sever degenerative renal damage [5, 28, 29].

Some researchers stated that enlargement of the glomeruli reflected function overwork which
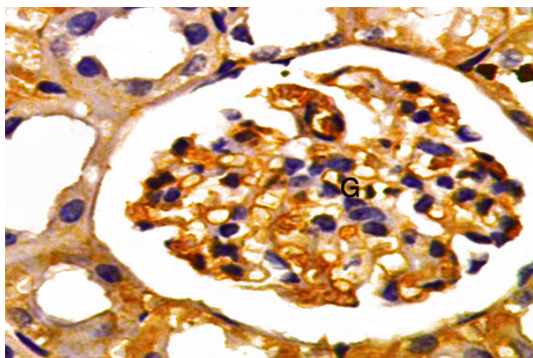
Figure 17. A photomicrograph of a section in the renal cortex from group III showing positive desmin immunostaining in the enlarged renal glomerulus (G) (Immunostaining for Desmin × 630).
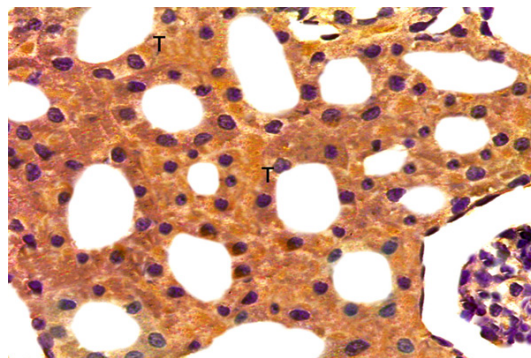

Figure 19. A photomicrograph of a section in the renal cortexfrom control group showing– intense positive brown Bcl2 immunostaining in the cytoplasm of tubular cells (T) (Immunostaining for Bcl2 × 630).
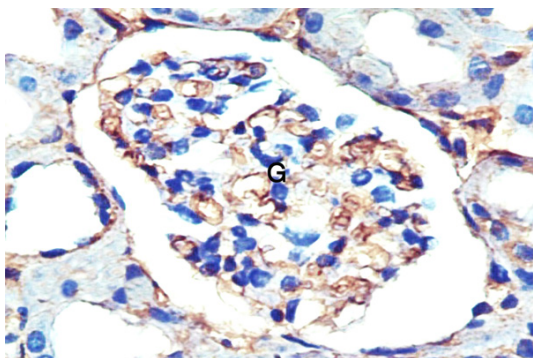

Figure 18. A photomicrograph of a section in the renal cortex from group IV showing decrease of desmin immunostaining in the renal glomerulus (G) (Immunostaining for Desmin × 630).
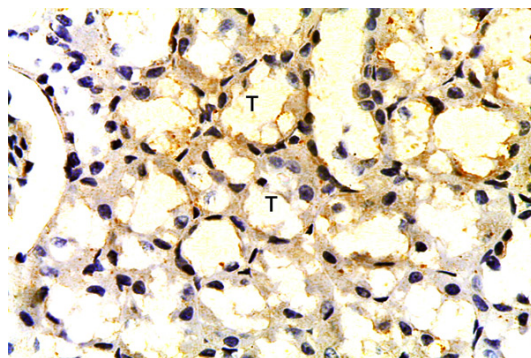

Figure 20. A photomicrograph of a section in the renal cortex from group III showing weak positive brown Bcl2 immunostaining in cytoplasm of tubular cells (T) (Immunostaining for Bcl2 × 630).

placed stress on podocytes because of their limited proliferation capacity [30].

Some investigators reported that podocytes injuries in the form of loss foot processes, vacuolization, retraction and detachment from glomerular basement membrane were common in all types of glomerulosclerosis. They added that alteration nephrin protein which was a major component of slit diaphragm and played critical role in maintaining the permeability of basement membrane. So the glomerular injury may be direct due to injury of podocytes or indirect, secondary to tubular necrosis [31]. This was supported with a significant increase of desmin immunostaining in the renal glomerulus in the present study.

Desmin, an intermediate filament protein, has been suggested as a podocyte injury indicator, the expression of which was often upregulated

in various glomerular diseases in which podocyte damage was involved [32, 33]. Podocyte epithelial dedifferentiation was accompanied by the induction of mesenchymal markers such as desmin [34]. It was hypothesized that podocytes might undergo epithelial dedifferentiation and mesenchymal transition due to upregulation of transforming growth factor-β1 in the diseased kidney and that de novo desmin expression in podocyte could be a reliable relevant marker for podocyte epithelial-to-mesenchymal transition [34].

The present study of group III revealed tubular swelling, degeneration and necrosis. Also rupture of basement membrane of some tubules was occurred. Sloughing of epithelial lining of the tubules resulted in cellular casts. Hemorrhage was present inside the lumen of some tubules.
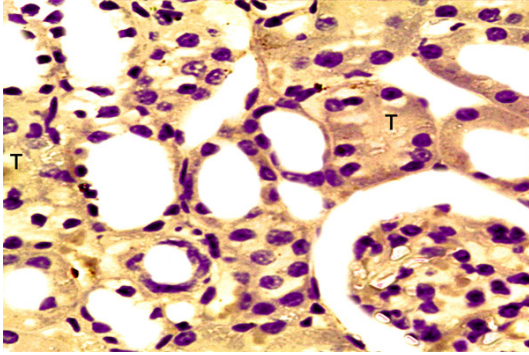
Figure 21. A photomicrograph of a section in the renal cortex from group IV showing moderate positive brown Bcl2 immunostaining in the cytoplasm of tubular cells (T) (Immunostaining for Bcl2 × 630).

Figure 22. Showing the mean area % of desmin expression in all experimental groups.

Table 3. Showing the mean area %, SD of desmin expression in all experimental groups

	Group I	Group II	Group III	Group IV
Mean area %	0%	0%	12.69%	2.29%
± SD	0	0	0.6692	0.6714

SD = Standard deviation; Group III compared with group I (P < 0.05); Group IV compared with group III (P < 0.05).

Table 4. Showing the mean area %, SD of Bcl2 expression in all experimental groups

	Group I	Group II	Group III	Group IV
Mean area %	23.20%	23.04%	1.57%	12.29%
± SD	0.5570	0.5562	0.4071	0.6698

SD = Standard deviation; Group III compared with group I (P < 0.05); Group IV compared with group III (P< 0.05).

Figure 23. Showing the mean area % of Bcl2 expression in all experimental groups.

Some investigators stated that these tubular casts obliterate the lumen of tubules and through tubuloglomerular feedback could result in arteriole vasoconstriction and decrease of glomerular filtration rate filtration pressure [30, 31].

The effect of aflatoxin B1 on the renal tubules was supported with a significant decrease of the antiapoptotic Bcl2 immunostaining in the tubular cells in the present study.

Apoptosis is a complex process that is necessary for regulating cell survival through removal of diseased or damaged cells [35].

The anti-apoptotic Bcl-2 family members such as Bcl-2, plays a pivotal protective role in preserving mitochondrial structure and function,

preventing onset of mitochondrial permeability transition, and finally inhibiting the apoptosis [36].

Some studies reported that AFs induced apoptosis in renal tubular cells and other organs [3, 23, 27, 37].

The present study of group III revealed interstitial hemorrhage, mononuclear inflammatory reaction and fibrosis between degenerated tubules and renal corpuscles.

Some researchers stated that fluid leakage from the tubules lead to edema and cellular infiltrations and also interstitial fibrosis. Most of the fibroblasts originate from tubular epithelium and through epithelial mesenchymal transition and persistent injury or chronic inflammation of the kidney lead to increase of the fibroblast and secrete excess extracellular matrix. The conversion of tubular epithelial into mesenchymal phenotype was due to alteration in

the balance of local cytokines concentration [38-40].

The histological results reported in the current study confirmed the biochemical results and indicated that aflatoxin induced severe histological changes in kidney.

The present study of group III revealed showed apparent decrease of PAS positive reaction in the renal tubules as well as renal glomeruli.

Several studied have shown apparent reduction of tissue glycogen as in liver, kidney, heart, skeletal muscles of rabbits and rats as result of inhibiting of biosynthetic enzymes and stimulating of glycogenolysis enzymes as phosphorylase which help to accelerate breakdown of glycogen [41, 42].

Some researchers stated that the mechanism of action of aflatoxins on the cell was mediated through the production of free radicals and reactive oxygen species (ROS) such as superoxide anion, hydroxyl radical, and hydrogen peroxide. These ROS of oxidative stress were capable of alteration and damaging cell compounds as well as membranes, eventually leading to the impairment of cell functioning, cytolysis and interstitial fibrosis [9, 27, 43].

The histological examination and biochemical parameter of group IV of our study revealed marked improvement of various changes produced by aflatoxin B1. There were apparent hypercellularity of the glomeruli and most convoluted tubules were regenerated, minimal collagen fibers between glomeruli and tubules and moderate PAS reaction. A significant decrease of desmin but significant increase of Bcl2 was detected.

These results were in agreement and confirming the findings of some investigators who reported the protective effect of curcumin against nephrotoxicity [4, 11, 44-46].

Curcumin being active component of turmeric plays efficient role in ameliorating the toxicity induced by aflatoxin in liver and kidney of mice by prevents the formation of ROS and scavenges free radicals, it protects cells from peroxidative stress and increase renal blood flow. Curcumin also enhances the activities of antioxidant enzymes such as superoxide dismutase

(SOD), catalase, reduced GSH and glutathione peroxidase (GSH-Px) [12, 47].

Some researchers reported that curcumin had potent anti-inflammatory which acts through inhibiting neutrophil infiltration, suppression of proinflammatory cytokines in macrophages [48].

Some researcher reported that curcumin had been shown to ameliorate fibrosis due to its the anti-inflammatory property [49]. Other investigators had reported that curcumin might exert a modulatory effect on cytokine transforming growth factor 1 beta induced fibrosis [50].

Curcumin had a potent antiapoptotic effect as decreased the expression of the pro-apoptotic gene and proinflammatory genes as caspase-3 and COX2, whereas it increased the expression of the anti-apoptotic gene Bcl-2 in many organs as kidney, testis and lung [51, 52].

Conclusion

Aflatoxin B1 had a deleterious effect on the histological structure of the kidney. Curcumin had a protective effect against damaging effect of aflatoxcosis on the kidney as it had antioxidant, anti-inflammatory and antiapoptotic activities. We advise eating nutritious diets that contain sufficient amounts of curcumin and regulation must implement to avoid the presence of aflatoxins in high concentrations in human food.

Disclosure of conflict of interest

None.

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