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AMELIORATIVE EFFECT OF COPPER ALBUMIN COMPLEX AGAINST AFLATOXICOSIS COMPARED WITH CURCUMIN

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In order to assess the potential protective effects of curcumin and the copper albumin complex on aflatoxin B1 (AFB1) toxicity in animal rats, 60 male albino rats were separated into the following four groups: Group 1 serves as the control, Group 2 administers AFB1 orally just three times at a weak dose for five weeks, Group 3 administers the same dose of AFB1 and curcumin (200 mg/kg B.W. diluted in 25 ml maize oil), and Group 4 administers the same dose of AFB1 and the copper albumin complex (0.8 g m/kg B.W.) orally. Following the completion of the experiment, the animals were scarified, blood and tissue samples were taken for biochemical and histological analysis. The findings of this study demonstrated that copper albumin complex had superior protective effects against Aflatoxicosis compared to curcumin. This may be because copper albumin complex has antioxidant properties. According to the findings of this study, the toxicological effects of AFB1 can be avoided by adding copper albumin complex as a food additive

Keywords: *Aflatoxicosis, copper (I) albumin complex, curcumin*

INTRODUCTION

Aflatoxins (AFs) are secondary metabolites that are poisonous to both humans and animals and are produced by the fungi Aspergillus flavus, Aspergillus parasiticus, and Aspergillus nomius. The majority of the time, these fungi attack cereal crops like wheat, walnuts, corn, cotton, peanuts, and tree nuts[1,2] , It can cause a number of consequences, such as hepatotoxicity, teratogenicity, and immunotoxicity, that pose substantial risks to both human and animal health[3,4] AFB1 is categorized as a Group I carcinogen by the International Agency for Research on Cancer (IARC), particularly damaging the liver[5] . In living organisms, AFB1 is subjected to reduction, hydrolysis, and/or oxidation processes mediated by cytochrome P450 (CYP) enzymes[6] Reactive oxygen species (ROS) are created when the most harmful metabolite, AFBO, reacts quickly with DNA, notably in the liver. It can also react covalently with the amino acid lysine to form AFB1-lysine adducts in blood[7]. The routes of AFB1-induced cytotoxicity or cell death have been linked to excessive reactive oxygen species (ROS) generation, DNA damage, oxidative stress, lipid peroxidation, apoptosis, mitochondrial malfunction, necrosis, and inflammatory response[8-10]. The p53 gene's codon 249 is known to be mutated by the AFB1-DNA adduct, which can result in liver cancer in both people and test animals[11]. The liver, which metabolizes a significant portion of AFB1, is an important target organ. As a result, exposure to AFB1 has been associated with the development of hepatocarcinogenesis in humans as well as in a variety of other animals, such as birds, fish, rodents, and nonhuman primates[12].

According to the IARC, AFB1 and AFM1 are, respectively, category 1 and group 2B human carcinogens[13]. AFTs exposure is known to cause hepatocellular carcinoma (HCC), and it has been estimated that between 4.6% and 28.2% of all HCC incidences worldwide are directly correlated with exposure to AFTs[14]. It has been demonstrated that the polyphenolic compound curcumin, also known as turmeric or

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1,7-bis (4-hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione, has anti-inflammatory, anti-cancer, anti-oxidative stress, and immunological regulating properties[15&16]. Curcumin supplementation has been shown to successfully reduce AFB1-induced liver damage, renal dysfunction, and intestinal damage by decreasing oxidative stress, apoptosis, inflammation, necrosis, and CYP450 enzyme expression in animal experimental or in vitro researchs[17-19]. Additionally, multiple studies found that curcumin and black tea could work together to alleviate the liver and renal damage brought on by AFB1[20]. Importantly, research on either humans or animals has demonstrated that curcumin is safe and accepTable[21].

It was determined that several copper complexes showed biological activity, including anti-inflammatory activity, and that some proactive copper complexes typically had more powerful cardinal effects than their original compounds without copper[22-25]. Copper complexes were examined and used well to treat a number of inflammatory disorders. It is possible to employ the copper-albumin chelating complex, which contains copper as one of the copper peptides and retains egg albumin, as an anti-inflammatory and antioxidant substance[26-28]. Additionally, it has a very good tolerance for hepatotoxicity[29]. Particularly for highly efficient copper dependent enzymes like superoxide dismutase (SOD), copper complexes have the ability to alter the biochemistry of cells and alter metabolism. Additionally, it is acknowledged that the removal of superoxide radicals involves the utilization of combinations of biochemically active organic molecules, such as amino acids and peptides, or vitamins that include copper[30]. Because it can live in cells under two different oxidation states, copper is particularly interesting in this sense. Because normal cells cannot reduce Cu(II) to Cu(I), the anoxic nature of cancer cells encourages this process, offering a therapeutic option to target tumors[31]. Cu(I) can catalyze the production of ROS and RNS, which causes an oxidative stress that is pro-apoptotic. In contrast to copper albumin complex, the current study primarily focuses on the preventive benefits of dietary curcumin on the acute liver damage caused by AFB1.

MATERIAL AND METHOD

60 male albino rats (120-150 g B.W.) were placed into 4 groups, with 15 animals in each group. Animals in Groups 1 and 2 were both aflatoxicosed for a period of five weeks without therapy. Animals in Group 3 were aflatoxicosed for five weeks while receiving oral curcumin treatment three times per week (200 mg/kg diluted in 25 cc corn oil). Group 4 received oral treatment with Cu-albumin complex (the complex was diluted in dairy milk at a rate of 0.8 gm/kg body weight) for five weeks while the animals were aflatoxicosed, concurrently with the introduction of aflatoxicosis three times each week. Blood samples from each rat were obtained at the moment of scarification, left at room temperature for 15 minutes to allow for blood coagulation, then centrifuged for 10 minutes at a speed of 3000 rpm. Until analysis, the collected serum was stored at -20°C. Additionally, liver tissue samples are preserved in formalin for histopathological analysis and further frozen at -40°C for tissue biochemical analyses. One gram of liver tissue was homogenized in 9 ml of phosphate buffer (pH 7.4), and the supernatant was recovered after centrifuging the mixture at 3000 rpm for 15 minutes. According to[32&33], the results of the biochemical tests revealed the total protein content and albumin levels. AST, ALT According to the test kit's inscription, manufactured by Spectrum Diagnostics an Egyptian biotechnology business, S.A.E. Superoxide Dismutase (SOD)[34], Total Antioxidant Capacity (TAC)[35] Catalase[36], Ceruloplasmin[37], Glutathione Peroxidase (GPx)[38].

Histological examination

Perls performed a histology analysis that included iron staining Prussian blue response as reported[39]. By Masson's[40] trichrome was completed. The tissue was cut into 3 m thick sections and stained with hematoxylin and eosin[41]. Histopathological analyses were performed using an Olympus CX 41 RF light microscope (Olympus Corporation, Tokyo, Japan).

Statistical analysis

The data was presented as means ± standard error (SE). Students' -t- test was used to determine the significance of the difference between the groups, with $p<0.05$

being considered significant (*), p<0.01 being considered very significant (**), and p<0.001 being considered extremely significant (***).

RESULTS AND DISCUSSION

Results

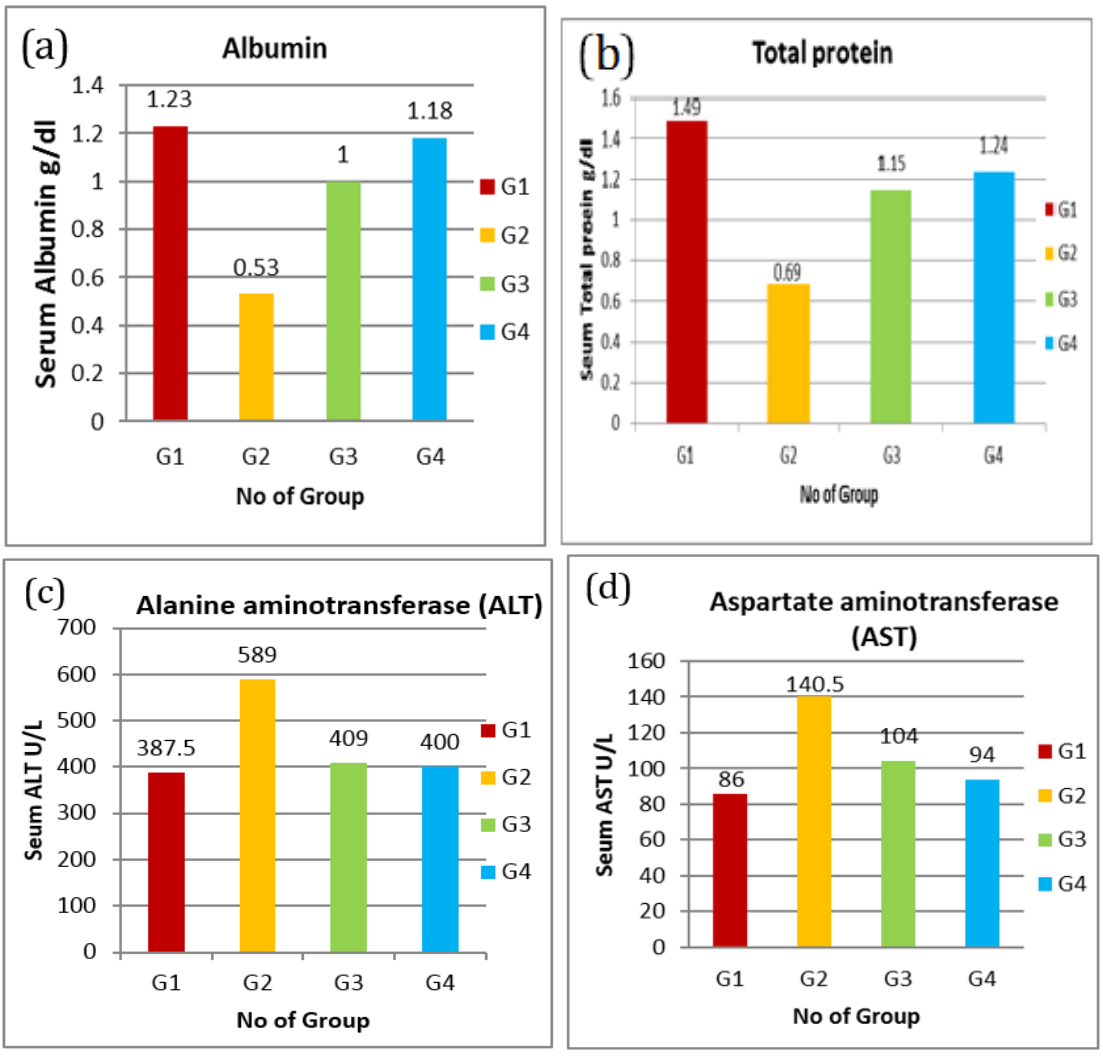
The animals in group (G2) displayed a drastic decrease in serum total proteins, which was highly substantially reduced in comparison to the normal control animals in group (G1) (p<0.001). This was shown in **Table 1** and **Fig. 1(a).** Curcumin co-treatment in (G3) on inebriated rats revealed that was significantly decreased from normal (p<0.01). Similar drunken animals that were also given the Cu-albumin combination in (G4) demonstrated a substantial decrease from normal, (p<0.05).Level of serum albumin (g/dL): The aflatoxicosed animals (G2) are shown in **Table 1, Fig. 1(b)**, with a highly significant reduction from the norm (p<0.001). Curcumin cotreatment in (G3) on atoxicosed rats resulted in significant reductions from the baseline (p<0.05). Similar to inebriated mice, Cu-albumin complex (G4) treatment also revealed that was not significantly different from usual. serum levels of ALT (U/L): **Table 1** and **Fig. 1(c)**, Aflatoxin-exposed animals (G2) increased in comparison to normal control samples (G1), and this increase was significant (p<0.001). In rats that had been aflatoxicosed and received curcumin (G3) treatment concurrently for five weeks, the level of serum ALT was marginally higher than normal. The increment was still significant (p<0.01) in this case. Animals treated simultaneously with C-albumin complex (G4) and aflatoxins showed a modest rise over baseline, where (p<0.05).

Serum AST concentrations (U/L): Aflatoxin-exposed animals (G2) had considerably higher levels than normal control samples (G1), according to **Table 1** and **Fig. 1(d)** (p<0.001). In animals treated with curcumin (G3) after being aflatoxicosed, the level was slightly higher than usual. Similar results were observed in animals that had been aflatoxicosed and were given Cu-albumin complex (G4); these animals' levels were lowered but were still substantially higher than expected (p<0.05). serum ceruloplasmine (g/L): **Table 1** and **Fig. 1(e)** demonstrated that the number of aflatoxicosed animals (G2) compared to the normal control sample (G1) was significantly lower (p<0.001). However, the amount of aflatoxicosed animals treated with curcumin (G3) demonstrated a marginally significant reduction from the norm (p<0.05). The best improvement was seen in atletoxicosed rats of (G4) that were also treated with Cu-albumin complex, and it was within normal limits (non-significant value of p>0.05). serum superoxide dismutase (SOD) activity (U/ml): **Table 1** and **Fig.** 1(f) demonstrated that (G2) for aflatoxicosed animals reduced lower than typical control samples (G1), with a significance level of (p<0.001). In treated aflatoxicosed mice with curcumin (G3), the level of serum SOD increased to be marginally significantly lower than baseline with (p<0.05).

Aflatoxicosed mice received concurrent therapy with the Cu-albumin combination (G4), which resulted in a marginally significant reduction from baseline (p<0.05). tissue glutathione peroxidase (U/gT): **Table 1, Fig. 11(g)**, and (G2) for animals that had been aflatoxicosed showed a considerable decrease from the typical control sample (G1), (p<0.001). However, after receiving curcumin (G3) treatment, the number of aflatoxicosed animals exhibited a substantial decrease compared to the norm (p<0.001). Rats who had been simultaneously treated with Cu-albumin complex and were atoxicoated (G4) showed improvement that was somewhat substantially lower than the norm (p<0.01). serum total antioxidant capacity (mM/L): According to **Table 1, Fig. 1(h)**, and (G2) for animals that had been aflatoxicosed, the normal control sample (G1) was much smaller (p<0.001). However, the number of aflatoxicosed rats treated with curcumin (G3) decreased from the norm in a marginally significant way (p<0.05). The best improvement was seen in rats who were simultaneously treated with Cu-albumin complex and atoxicosted (G4) (non-significant value of p>0.05). serum catalase (U/L): The aflatoxicosed animal sample (G2) in **Table 1, Fig. 1(i),** and (G2) from the normal control sample (G1) was substantially smaller (p<0.001). However, the number of aflatoxicosed mice treated with curcumin (G3) was much lower than expected (p<0.001). Rats that had been simultaneously treated with Cu-albumin complex and were atoxicoated (G4) showed improvement, which was moderately lowered from the norm (p<0.01).

Table 1: Changes in serum total protein concentration (g/dl), serum albumin (g/dl), serum ALT(U/L), serum AST(U/L), serum SOD(U/ml), serum TAC (mM/L), serum catalase (U/L), serum ceruloplasmin (g/L) and tissue GPx(U/gT).

Parameters	Group 1 (Control) N=15	Group 2 (AFB1) N=15	Group 3 (AFB1+curcumin) N=15	Group 4 (AFB1+Cu-albumin) N=15
Total protein (g/dl)	1.49±0.07	0.69±0.08***	1.15±0.02**	1.24±0.08*
Albumin (g/dl)	1.23 ± 0.07	0.53± 0.05***	1.0 ± 0.02*	1.18±0.06 N.S
ALT(U/L)	387.5±4.2	589±39***	409±4.3**	400±0.4*
AST(U/L)	86±2.3	140.5±3.2***	104±4.2**	94±0.3*
SOD (U/mL)	10875±52.6	9372.7±64.7***	10595.8±65.2*	10738±47.6*
TAC (mM/L)	1.36±0.01	0.73±0.02***	1.14±0.07***	1.30±0.09**
Catalase(U/L)	469.3±2.3	414±2.2***	441.5±2.1***	462±1.7**
Ceruloplasmin (g/L)	0.46±0.02	0.24±0.02***	0.38±0.02*	0.43±0.02 N.S
Tissue GPx (U/gT)	62.6 ± 0.49	40.3±0.28***	51.5±0.24***	60.9±0.2**



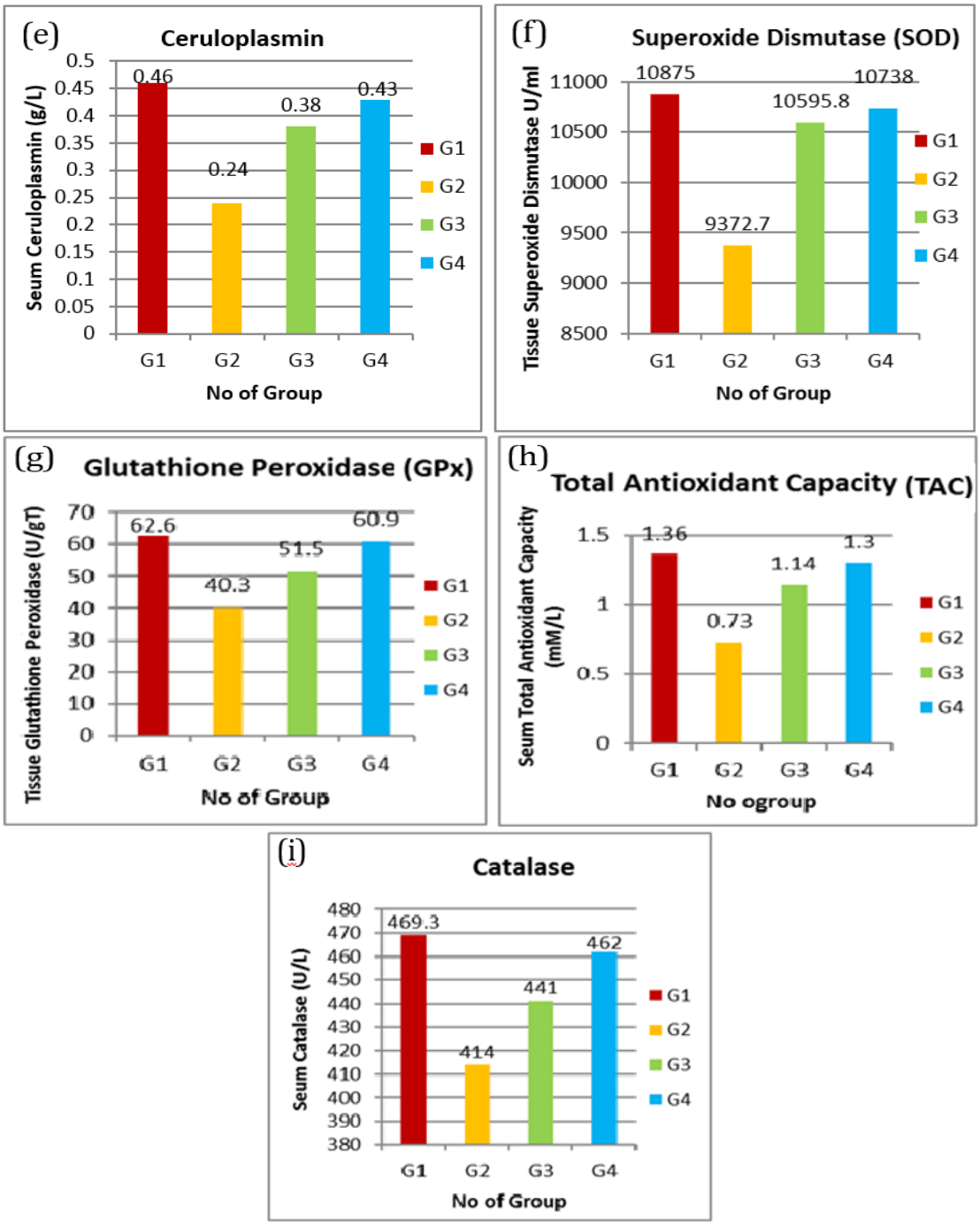


Fig. 1: (a) Shows serum total protein concentration (g/dl) changes with the investigated animals, (b) Shows the concentration of serum albumin (g/dl), (c) Serum ALT activity (U/L), (d) Show that serum AST activity (U/L), (e) Serum ceruloplasmine concentration (g/L), (f) Serum SOD activity (U/ml) in untreated aflatoxicosed animals and treated by curcumin and Cu (I)-albumin complex, (g) Tissue GPx activity (U/gT) in untreated aflatoxicosed animals and treated by curcumin and Cu- albumin complex, (h) Serum total antioxidant capacity (mM/L) in untreated aflatoxicosed animals and treated by curcumin and Cu albumin complex and (i) Serum catalase (U/L) in untreated aflatoxicosed animals and treated by Curcumin and Cu-albumin complex.

Histological examination

The histological characteristics of G1 control mice are shown in **Fig. 2 (a & b)**. A microscopic examination of the aflatoxicosed livers of G2 animals is shown in **Fig. 3 (a)** through (d). The livers of these animals that had been drinking heavily appeared to have noticeably cirrhotic nodules nearby invasive fronts of hepatocellular carcinoma **(Fig. 11(a))**. Marked central vein dilatation **(Fig. 3 (b))**. No signs of kuffer cell hyperplasia were found, only a band of appoptic hepatocytes and marked vacuolar degeneration **(Fig. 3 (c))**.

For the AFB1-only treated rat in **Fig. 3 (d)**, there was noticeable portal inflammation, significant portal vein dilation (Green arrow), and congestion. The normal control sample did not exhibit any induced liver damage **(Fig. 2 (a & b)**. (Group 3, AFB1+ curcumin) **Fig. (4 (a)** through (e)), depict cirrhotic nodules for AFB1 and curcumin-treated rats (green arrows) **(Fig. 4 (a))**, while **Fig. 4 (b)** depicts mild central vein dilatation and congestion for AFB1. The rat treated with AFB1 and curcumin showed substantial (severe) vacuolar degeneration (Green arrow) in **Fig. 4 (c)**, as well as portal tract inflammation, portal vein dilatation, congestion, and mild bile duct hyperplasia **(Fig. 4 (d))**. For AFB1 and curcumin, there is a little kuffer cell hyperplasia **(Fig. 4 (e))**. **Fig. 5 (a)** through (d) (Group 4, AFB1+ copper albumin complex) depict prominent Kuffer cell hyperplasia and mild vacuolar degeneration in the liver of rats treated with AFB1 and copper albumin, respectively. **Fig. 5 (b)** depicts a moderately dilated central vein in the same rats treated with AFB1 and copper albumin. For rats treated with AFB1 and copper albumin, **Fig. 5 (c)** shows prominent bile duct hyperplasia, modest portal tract inflammation, fibrosis, and portal vein dilatation (green arrow), as well as mild hepatocyte vacuolar degeneration **(Fig. 5 (d))**.

Many studies have included the protective effect of fungal poisoning especially aflatoxicosis such as oltipraz[42], Lactic acid Bacteria[43], soybean[44], BHT[45], which can be attributed to their antioxidant and anti-inflammatory properties.

Our research demonstrated that the suppression of protein synthesis in hepatic tissue resulted in significantly lower levels of total protein or serum albumin[46&47], Comparing the curcumin-treated group to the aflatoxicosed group, total protein levels and serum albumin were higher in the curcumin-treated group because curcumin was successful in returning liver enzyme and protein activity, which are utilized as indicators of liver membrane damage, to normal[48], Similar to the results of other research, the total protein levels in the copper complex group increased and were close to those of the control group[49]. According to the blood biochemical data, rats given AF saw a considerable rise in ALT and AST levels, which is consistent with the results of prior studies conducted on poultry[50], Contrary to the aflatoxicosed group, curcumin treatment reduced the activity of the AST and ALT enzymes. This decrease may be explained by a decrease in hepatocellular injury[51], indicating that curcumin may have preventive potential against liver damage brought on by AF, Similar to how copper has been shown to sustain liver function against the most common toxicities as demonstrated by a considerable reduction of transaminases, the copper complex group has lower ALT and AST levels than the aflatoxicosed group[52].

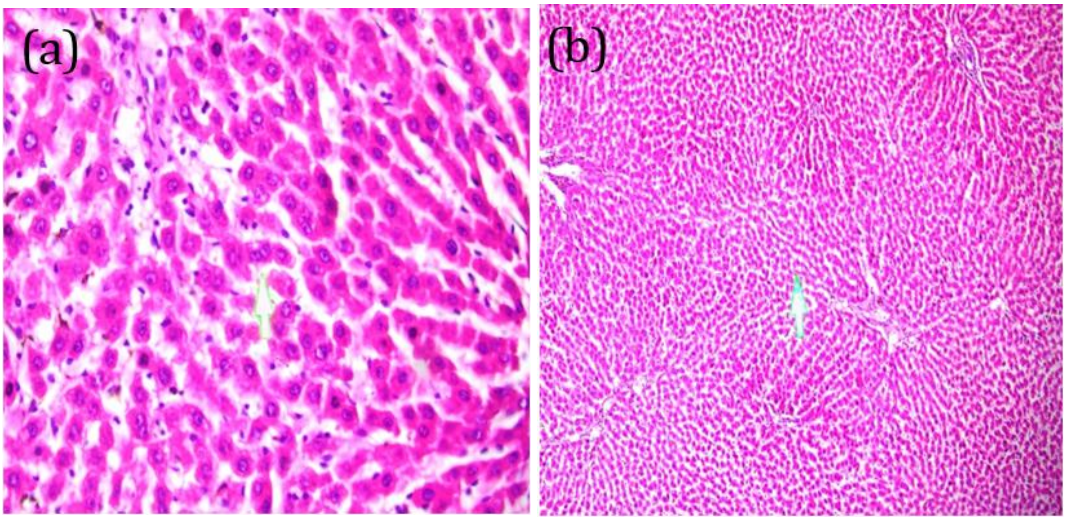


Fig. 2: For cases of normal liver (Group 1), (a) and (b) shows Paraffin section in livers of negative control animals (Hx. & E. ×200).

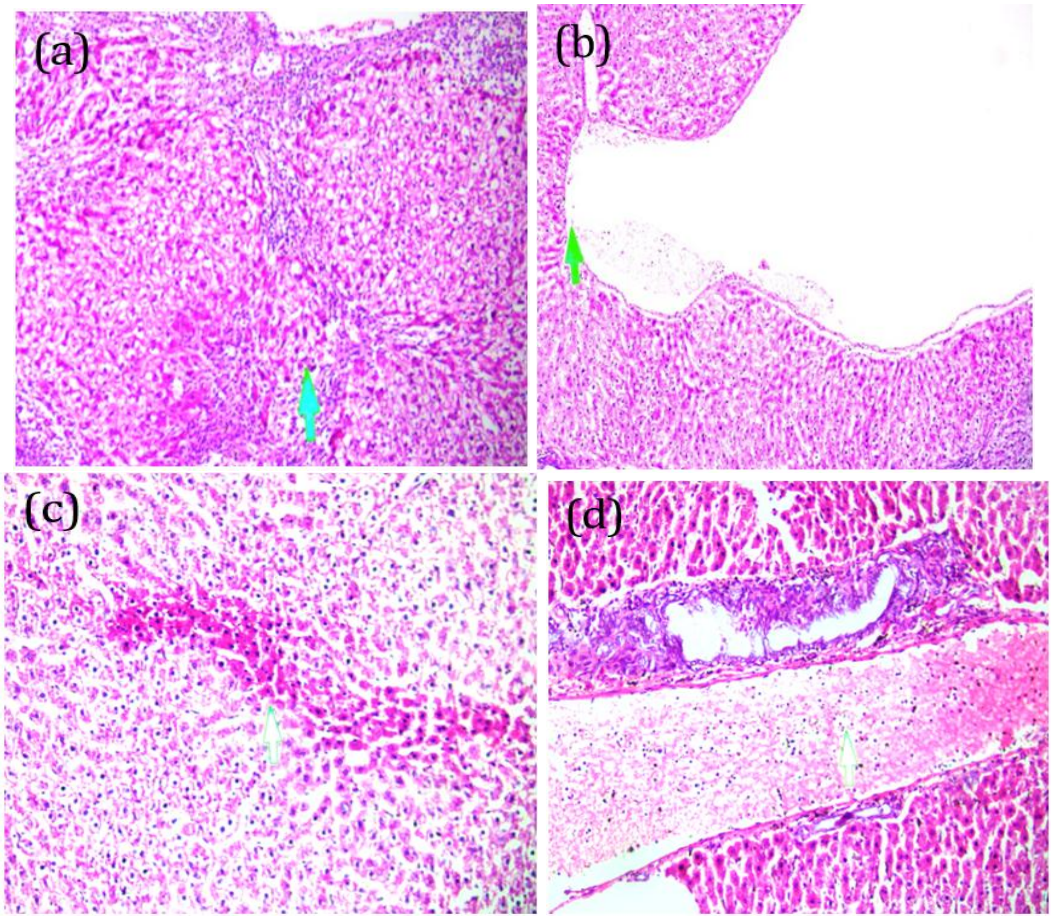


Fig. 3: For cases treated with AFB_1 only (Group 2): (a) Hematoxlin and Eosin stain, x100 power, cirrhotic nodules adjacent to invasive fronts of hepatocellular carcinoma (Green arrow) for AFB_1 only treated rat. (b) Hematoxlin and Eosin stain, x200 power, marked central vein dilatation (Green arrow) for AFB_1 only treated rat. (c) Hematoxlin and Eosin stain, x200 power, marked vacuolar degeneration and band of appoptic hepatocytes, no kuffer cell hyperplasia detected (Green arrow) for AFB_1 only treated rat. (d) Hematoxlin and Eosin stain, x200 power, marked portal vein dilatation (Green arrow) and congestion, mild bile duct hyperplasia and detecTable portal inflammation for AFB_1 only treated rat**.**

Oxidative stress also plays an important role in the toxic effects of AFB1 in broilers, where mycotoxins trigger ROS production and reduce antioxidant capacity in various tissues, such as liver, spleen and heart[52-54]. SOD, CAT and GsPx play important roles. during detoxification by free radicals and they protect cell membranes from peroxidation; however, when the antioxidant defense system is inadequate, there are changes in antioxidant enzyme activity and MDA levels[55&56]. Aflatoxisis was characterized by the presence of oxidative stress markers such as ceruloplasmin, superoxide dismutase (SOD), glutathione peroxidase (Gpx), total antioxidant capacity (TAC), and catalase. Both copper albumin complex and curcumin are effective hepatocellular protecting agents, as shown by the co-treatment that lowered such biochemical markers. According to our findings, the progression of fatty degeneration was blamed for the significant decline in ceruloplasmin levels, Superoxide Dismutase (SOD), Glutathione Peroxidase (GPx), Total Antioxidant Capacity (TAC), and Catalase in the aflatoxicosed group compared to the control group[52-54].

Because curcumin has anti-inflammatory effects, we see an increase in ceruloplasmin levels, (SOD), (GPx), (TAC), and catalase in the curcumin-treated group compared to the aflatoxicosed group[57], The presence of chemical groups like hydroxyl, methoxy, and 1,3-diketone conjugated diene system in the curcumin structure supports its antioxidant property[58&59]. Curcumin may also act by inducing detoxifying enzymes, and these enzymes may detoxify Reactive Oxygen Species (ROS) after the administration of toxicants[60].

The creation of multiple Cu(II) complexes with higher anti-inflammatory action led to a rise in ceruloplasmin levels, (SOD), (GPx), (TAC), and catalase in the case of the cupper complex group compared to the aflatoxicosed group[61], copper is a fundamental element for most aerobic organisms especially human[62], these results in agreement with several studies have shown a change in catalase expression in cancer cells became resistant to chemotherapies[63].

Histological analysis

As shown in **Fig.** 3, we found that rats exposed to AFB1 had damage to their liver cells as well as vacuolar degeneration, portal vein dilation, and congestion. The current investigation showed that after oral treatment of AFB1 for five weeks, hepatocytes experienced vacuolar degeneration and necrosis. The five week therapy group had considerably more vacuolar hepatocellular deterioration. The outcome is in line with earlier research[64,65]. Necrosis has been defined as an uncontrolled kind of cell death, when several biological processes prevent the cell from expanding and the plasma membrane from rupturing[62]. Necrosis is a type of death that takes place when ATP is completely depleted, as can happen during toxic damage and oxidative stress with the production of ROS. It causes alterations in the integrity of cell membranes that result in ion pump degradation, the first step in the development of vacuolar degeneration and cell swelling[63].

We observe minor hyperplasia of the bile duct for AFB1 and substantial central vein dilatation for the curcumin group, as shown in **Fig. 4 (c & a),** respectively. All of these points to the fact that curumin prevents and protects against HCC. However, due to curcumin's limited bioavailability and metabolic instability, medicinal uses of the substance are constrained[64]. Our results support the findings of other research[50] that curcumin may have preventive potential against liver injury. Additionally, we discovered mild kupffer cell hyperplasia for AFB1 in **Fig. 4 (d)**. This is consistent with other studies[65] that claim that kupffer cell hyperplasia observed in the liver is one of the body's defense mechanisms against toxins and contributes to hepatic oxidative stress.

The cupper albumin complex group had modest vacular degeneration and kupffer cell hyperplasia as seen in **Fig. 5 (a)**, also mild vacular degeneration as in **Fig. 5 (d)**, pronounced bile duct hyperplasia as shown in **Fig. 5 (c)**, and moderately dilated central vein as shown in **Fig. 5 (b)**. All of these studies suggest that copper complex functions as a more effective anti-cancer agent than curcumin, which is clear from biochemical indicators. These results are consistent with the results of the earlier study[52].

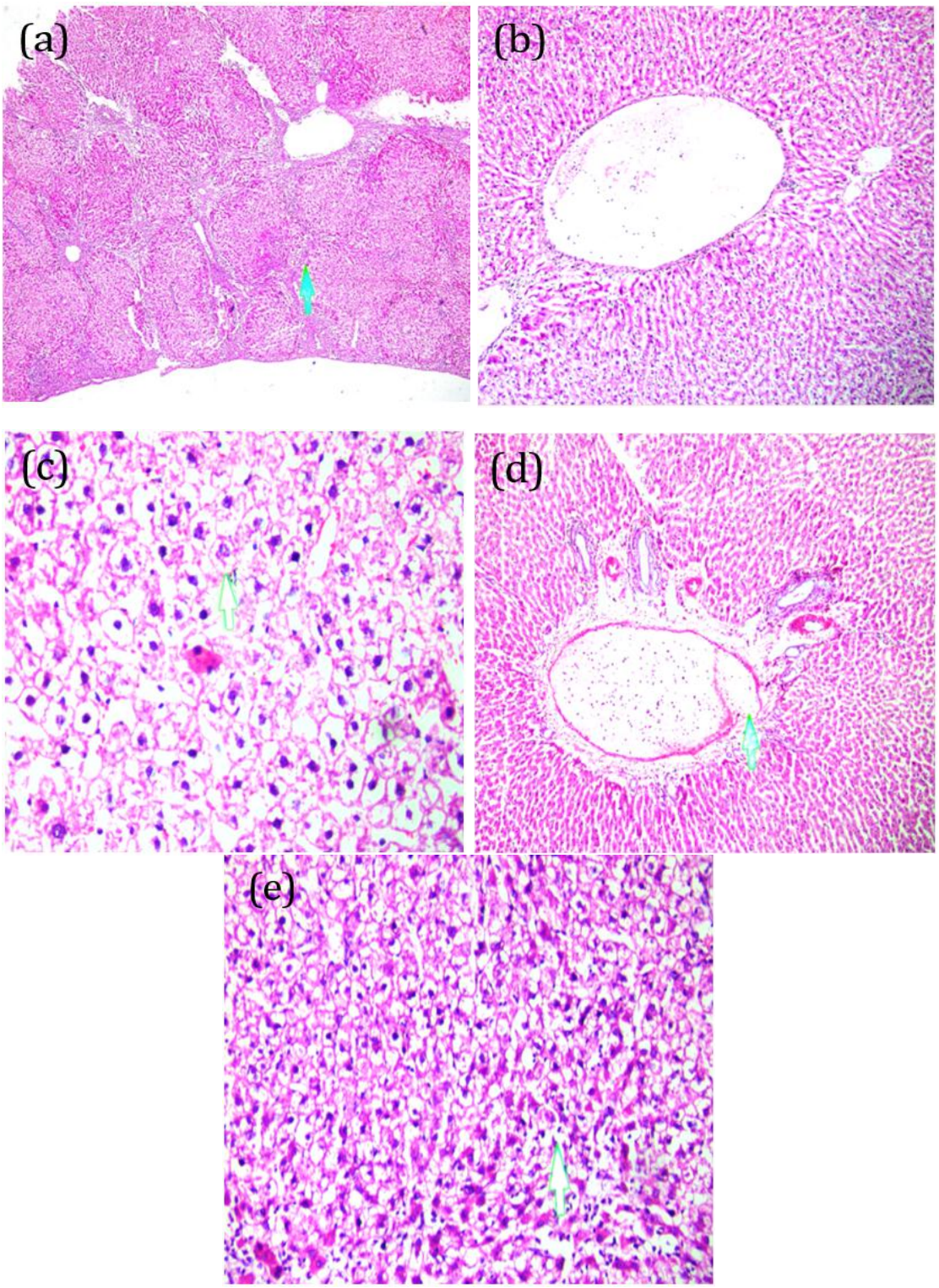


Fig. 4: For cases treated with AFB1 and curcumin (Group 3): (a) Hematoxlin and Eosin stain, x100 power, Cirrhotic nodules for AFB1 and curcumin treated rat (Green arrow). (b) Hematoxlin and Eosin stain, x200 power, moderate central vein dilatation and congestion for AFB1 and curcumin treated. (c) Hematoxlin and Eosin stain, x400 power, prominent (severe) vacuolar degeneration for AFB1 and curcumin treated rat (Green arrow).rat. (d) Hematoxlin and Eosin stain, x200 power, Portal tract inflammation, portal vein dilatation (Green arrow), congestion and mild hyperplasia of bile ducts for AFB1 and curcmin treated rat. (e) Hematoxlin and Eosin stain, x400 power, Mild kuffer cell hyperplasia for AFB1 and curcumin treated rat (Green arrow).

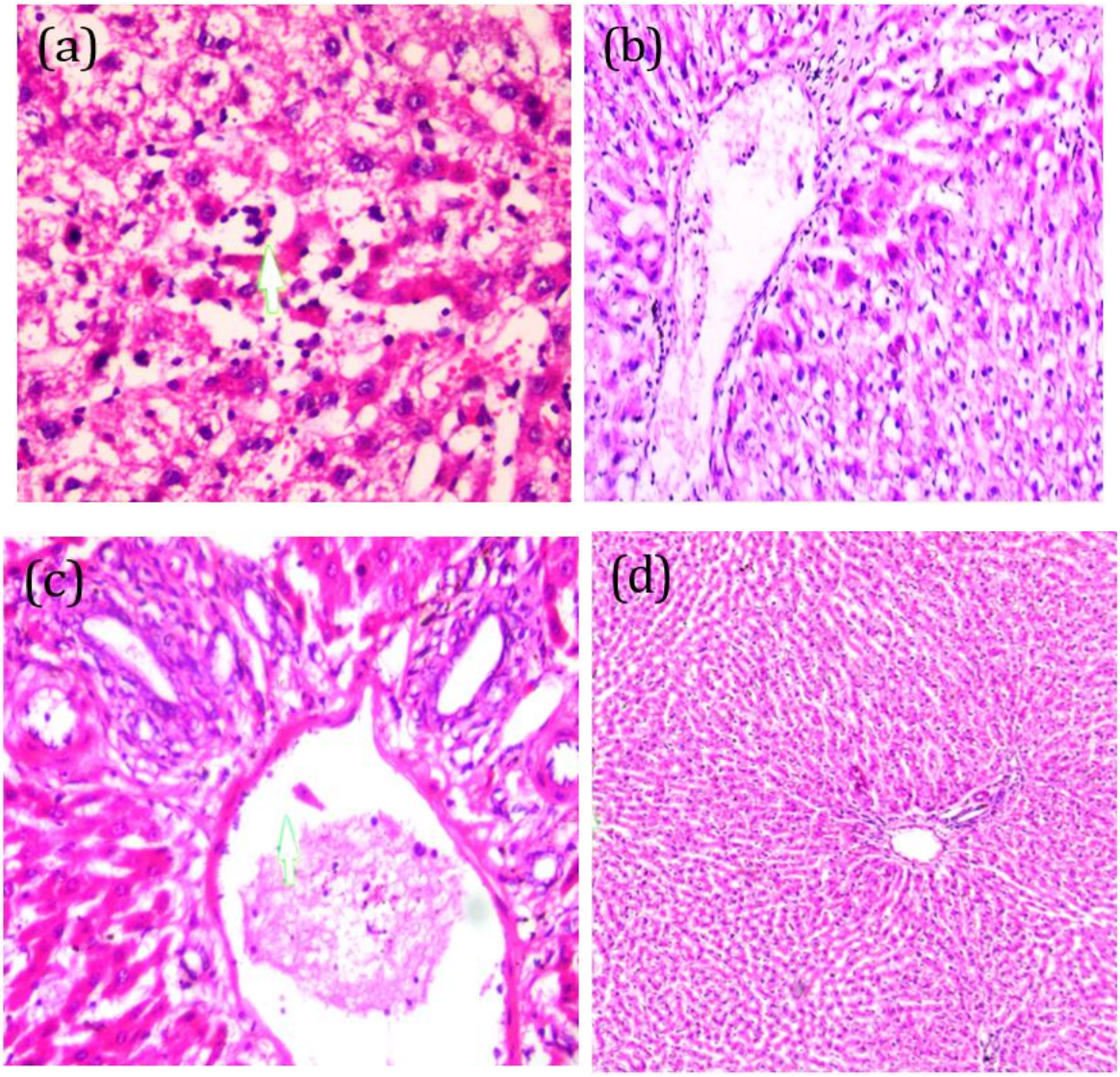


Fig. 5: For cases treated with AFB1 and cupper albumin (Group 4): (a) Hematoxlin and Eosin stain, x400 power. Prominent Kuffer cell hyperplasia and hepatocytes mild vacuolar degeneration for AFB1 and cupper Albumin treated rat. (b) Hematoxlin and Eosin stain, x200 power. Moderate dilated central vein for AFB1 and cupper Albumin treated rat. (c) Hematoxlin and Eosin stain, x400 power. Prominent Bile duct hyperplasia, mild portal tract inflammation, fibrosis and portal vein dilatation (Green arrow) for AFB1 and cupper Albumin treated rat. (d) Mild vacuolar degeneration of hepatocytes.

Conclusions

Copper supplementation may help protect against aflatoxicosis by reducing oxidative stress and liver damage caused by aflatoxin exposure. Generally, these obtained results by using copper albumin complex as a prophylactive and therapeutic agent against aflatoxicosis and it is considered the better anti-cancer agent than curcumin because curcumin is act as only aprophylactive helper agent.

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التأثير التحسينى لمتراكب النحاس مع الالبيومين ضد التسمم بالافلاتوكسين مقارنة بالكركيومين

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من أجل تقييم التأثيرات الوقائية المحتملة للكركمين ومتراكب النحاس مع الالبيومين على سمية الأفلاتوكسين ب١ في الجرذان ، تم تقسيم ٦٠ من ذكور الجرذان البيضاء إلى أربع مجموعات: المجموعة الاولى بمثابة المجموعة الضابطة، المجموعة الثانية جرعت بالافلاتوكسين عن طريق الفم. ثلاث مرات اسبوعيا لمدة خمسة أسابيع، المجموعة الثالثة جرعت بنفس الجرعة من الافلاتوكسين بالاضافة الى الكركمين (٢٠٠ ملجم / كجم من وزن الجسم مذاب في ٢٥ مل من زيت الذرة)، المجموعة الرابعة جرعت بنفس الجرعة من الافلاتوكسين بالاضافة الى متراكب النحاس مع الالبيومين (٠.٨ جم /كجم من وزن الجسم) عن طريق الفم. بعد الانتهاء من التجربة، تم ذبح تلك الجرذان وأخذ عينات من الدم والأنسجة للتحليل البيوكيميائي والهستولوجى. أظهرت نتائج هذه الدراسة أن متراكب النحاس مع الالبيومين له تأثيرات وقائية متفوقة ضد التسمم بالأفلاتوكسين مقارنة بالكركمين. قد يكون هذا بسبب أن هذا المتراكب له خصائص مضادة للأكسدة. ووفقا لنتائج هذه الدراسة، يمكن تجنب التأثيرات السمية للافلاتوكسن عن طريق إضافة متراكب النحاس مع الالبيومين كإضافات طعام.