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INFELUENCE OF BENTONITE AND ASCORBIC ACID ON MINIMIZING THE TOXICITY OF AFLATOXIN B_1 IN CHICKS DIETS.

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ABSTRACT : Five experimental groups were used to evaluate the efficiency of sodium bentonite (BNT) and ascorbic acid (AA) in reducing the toxicity of aflatoxin B_1 in El-Salam chicks (Egyptian local chicks) diets. A total number of 450 unsexed one-day old chicks were wing banded, weighted and randomly assigned to 5 groups of five replicates (18 chicks/replicate). The experimental groups were as follow: 1) basal diet (control);(2) control + aflatoxin B_1(1 mg / kg diet); (3) control + aflatoxin B_1 + 0.5% BNT;(4) conrol + aflatoxin B_1 + 500 mg AA/kg diet and (5) control + aflatoxin + BNT + AA.

Aflatoxin B_1 contaminated diet significantly ($P<0.05$) decreased body weight gain, feed intake, feed conversion, survival rate, blood total protein, albumin, globulin. On the other hand, aflatoxin B_1 increased the aspertate aminotransferase (AST), alanine aminotransferase (ALT), creatinine and uric acid. Also, hazard effects on histology of chicks liver and kidneys were associated with aflatoxin B_1. Addition of BNT, AA or BNT + AA to aflatoxin B_1 contaminated diets significantly ($P<0.05$) improved all parameters which were negatively affected by the mechanism of aflatoxin. Most results indicated that addition of BNT + AA were more efficient than BNT or AA alone in minimizing the negative effects of aflatoxin on chicks.

Key Words: Aflatoxin, Bentonite, Ascorbic Acid, Blood, Mortality and Histology..

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INTRODUCTION

Aflatoxins are mycotoxins produced as secondary metabolites by Aspergillus flavus and A. parasiticus (Abdelhamid, 2008). These fungi grow on certain foods and feeds resulting in the production of aflatoxins. The Aflatoxins contaminated-diets lead to many hazard effects on human and animals (death; reduce the production and reproduction; mutagenic, carcinogenic and teratogenic effects and immunotoxicity) (Shehata, 2002). Also, aflatoxins had adverse affect on chick performance (Eckhardt et al., 2014).

Utilization of adsorbents was the most applied method for protected from mycotoxicosis (Zaki et al., 2008). The BNT can be used to reduce toxicity of aflatoxin on broiler (Eckhardt et al., , 2014), promote growth performance, reduce both bacterial colonization of the gut (Tauqir and Nawaz, 2001), adhere to enteric pathogens selectively and exert them, reinforce intestinal mucosal barrier and help in the regeneration of the epithelium (Girardeau 1987). Also, BNT can be used in many medical applications (Veniale et al., 2007). Generally, BNT and tafla clay were better than kaolin in binding of aflatoxins, ochratoxin A and zearalenone (Shehata, 2002).

Immunosuppression enhanced by the consumption of aflatoxins contaminated feed, which is very common in many tropical countries. Practically, it is not possible to destroy the contaminated feed, therefore to avoid the effect of this substance, increasing animal immunity must be made (Zaky et al., 2000). Poultry have the ability to synthesis of AA or vitamin C in their body (McDowell, 2000), but during certain conditions (environmental and pathological stressors and high temperature) are known to alter AA use or synthesis or both in fowl. In these cases, AA supplementation provides benefit in poultry (Pardue and Thaxton, 1986). AA significantly improved weight gain, digestibility of nutrients, carcass traits, bone resistance and immunity of commercial of broilers (Lohakare et al., 2005). Also, AA alleviate the toxic effect of aflatoxin B_1on rabbits (Shehata, 2010).

Using mush method together in detoxification of aflatoxins is scarce such as Zaki et al., (2008), who reported that daily injection of 0.2% Fix in Toxin (kind of BNT) and 1% Nigella sativa (Ns) oil diminished aflatoxicosis in fish.

The present study was carried out to evaluate the protective effects of BNT, AA, BNT + AA on aflatoxins contaminated diet toxicity in El-Salam chicks.

MATERIALS AND METHODS

Five experimental groups were used to evaluate the efficiency of sodium BNT and AA in reducing the toxicity of aflatoxin B_1 in (El-Salam) chicks (Egyptian local chicks) diets. A total number of 450 unsexed one-day old chicks were wing banded, weighted and randomly assigned to 5 groups (nearly similar in average body weight) of five replicates (18 chicks/replicate). The experimental groups were as follow: 1) basal diet (control); (2) control + aflatoxin B_1(1 mg / kg diet); (3) control + aflatoxin B_1 + 0.5% BNT; (4) conrol + aflatoxin B_1 + 500 mg AA/kg diet and 5) control + aflatoxin + 0.5% BNT + 500 mg AA/ kg diet.

Aflatoxin production was carried out according to Davis et al., (1966) using Aspergillus flavus strain NRRL 3145 and liquid yeast medium (2% yeast extract and 20% sucrose). The aflatoxins concentration in liquied media and faeces was determined according to the method of AOAC (2012) using monoclonal antibody columans total aflatoxins (Vicam Science Technology, Watertown, MA USA).

The liquid media contain aflatoxin B_1, B_2, G_1 and G_2 The previous aflatoxins were calculated to equal aflatoxin B_1 toxicity, according to the fowliowing rank order of

toxicity of aflatoxins: B1=M1<G1<M2= B2<G2 in the rations. 1:1:½:¼:¼ intensity (H alzaphel etal.,1966). Then sprayed on diet to obtain AFB_1 required (1 mg/kg diet). Egyptian sodium BNT was provided by Misr Company for bentonites from Alamein area, it contained (%): 51.08 SiO_2; 16.37 Al_2O_3; 0.97 CaO; 2.68 MgO; 1.07 K_2O; 0.84 Na_2O; 9.27 Fe_2O_3, 0.03 MnO, 1.26 TiO_2, 0.13 P_2O_5, 0.017 Cr_2O_3, 0.09 TOT/C, 0.01 TOT/S, 15.80 LOl (Abdel-Motelib et al., 2011). The row BNT was dried in the oven at 80°C over night, milled to less than 300 mesh, added to diet. AA product of United Co. For Chem. 7 Med. Prep., Egypt dissolved in tap water and sprayed on contaminated diet to obtain 500 mg AA / kg contaminated diet. After AA addition the diet was stored in black plastic bags to prevent AA degradation.

The chicks were fed and watered ad libitium. The formula and chemical composition of basal diet (Table 1) was adopted according to AOAC (1990). At the end of the experiment, 15 blood samples of each group were taken at slaughter (3 chicks/replicate). Blood plasma was separated and stored at -20 °C to analysis. Total protein, albumin, AST and ALT, creatinine and uric acid in plasma were analyzed by using commercial kits from Diamond Diagnostics Company, Egypt. The liver and kidneys were removed from the body and kept in formalin solution (10%) for histological study. After fixation, the specimens were dehydrated; cleared; embedded in paraffin was and blocked. Sections of 6 microns thickens were cut using a rotary microtome, mounted and strained by Haematoxylin and Eosin (Carleton et al., 1980).

Data of the experiment were statistically analyzed using the General Linear Model Program of SAS (1996). Significant differences between treatment means were tested by Duncan"s Multiple Range Test (Duncan, 1955).

RESULTS AND DISSCUSION

1- Growth performance:

Aflatoxins contaminated diet decreased ($P<0.05$) live body weight, body weight gain (Table 2), feed intake (Table 3) and feed conversion (Table 4). These results agree with the findings Eckhardt et al., (2014) on broiler fed aflatoxins (3 mg/kg) contaminated diet for 7 weeks. Decrease of body weight gain by aflatoxins may be due to: (1) disturbance of carbohydrate, lipid or protein metabolism in the liver and loss of appetite (Cheeke and Shull, 1985; Marai and Askar, 2008), (2) decrease ($P<0.05$) of feed intake, digestibility of nutrients and disturbance of liver and kidneys functions (Shehata, 2010, 2012 and Amera, 2014). Also, (3) it might be due to detoxification process in the body utilizing glutathione enzymes. Glutathione is the intracellular antioxidant (Deng et al., 2010) and partly composed of methionine and cystein, hence this detoxification process depletes the metabolic availability of methionine leading to poor growth and feed efficiency (Devegowda et al., 1998). Decreasing of feed intake and feed conversion may be due to decreasing metabolism of nutrients and impaired organs functions by aflatoxin (Shehata et al., 2012).

Adding BNT, AA or BNT + AA to aflatoxins contaminated diets significantly improved ($P<0.05$) growth performance in all experimental period (12 weeks). Generally, the best improvement occurred by BNT + AA followed by BNT and AA. The results of BNT agreed with the findings of Arab and Wayatt (1991) who reported that 0.5 and 1% sodium BNT reduced the bad effect of aflatoxin B_1 on growth rate of broiler chickens. Also, Eckhardt et al., (2014) reported that calcium bentonite at 0.25 and 0.5% of aflatoxins contaminated diet improved body weight of chickens at 42 day of age by 13.3 and 22.7%, increased daily feed intake by 9.7 and 24.7%. The beneficial effect of BNT may be due to its ability to

bind aflatoxin in the gastrointestinal tract which decrease aflatoxin uptake and bioavailability (Zaki et al., 2008). The improvement in growth performance by AA agree with those obtained by Salem et al., (2001) on rabbits fed aflatoxins contaminated diet. These results of AA may be due to : 1) increasing feed intake and digestibility of nutrients (Sahin and Kucuk 2001 and Lohakare et al., 2014) of broiler chickens and Japanese quail, respectively. AA had biological role in digestive enzyme biosynthesis and activation (Earp et al., 1970). 2) Also, AA is necessary for bone development as a cofactor for the bioconversion of vitamin D to its active form of 1.25 $(OH)D_3$ (Sergeev et al., 1990). 3) Moreover, AA improve the immunity (Lohakare et al., 2005).

2- Blood parameters:

Some plasma constituents of chicks are shown in Table (5). Total protein, albumin and globulin concentrations were significantly ($P<0.05$) decreased due to aflatoxin effect. These results agree with those reported by (Eckhardt et al., 2014) on broiler. The decrease in total protein and albumin may be attributed to aflatoxin interaction with protein synthesis and cellular integrity in liver (Srivastava, 1984). The activity of AST and ALT enzymes significantly increased in chicks fed aflatoxin B_1 contaminated diet. These results agreed with the findings of Zaki et al., (2000) on ducks and Shehata (2010) on rabbits. Increasing of AST and ALT may be due to hepatocellular necrosis or increasing the permeability of cell membrane (Zaky et al., 2000). The creatinine and uric acid values were higher ($P<0.05$) in of chicks fed aflatoxin contaminated diet, these results may be due to impaired of kidney functions (Cheeke and Shull, 1985).

Addition of BNT, AA or BNT + AA significantly ($P<0.05$) improved blood parameters measured. These results of BNT agree with those obtained by Shehata, (2003) and Eckhardt et al., (2014). The beneficial effect of BNT may be due to its ability to bind aflatoxin in the gastrointestinal tract which decrease aflatoxin uptake and bioavailability (Zaki et al., 2008). The results of AA agreed with the findings of Shehata et al., (2009), who reported that adding 500 mg AA/kg diet fish contaminated with 3 mg aflatoxin B_1 improved total protein, albumin, globulin values and AST and ALT activities in compared with those fed aflatoxin B_1 contaminated alone. The effect of AA may be due to improve the broiler performance, immunity and general health ((Lohakare et al., 2005).

3- Survival rate and aflatoxins excreted in faeces:

The survival rate (Table 6) was reduced in chicks fed aflatoxin B_1 contaminated diet (88.89%) in comparison with control (96.67%). Addition of BNT, AA or BNT + AA increased the survival rate which were 95.56, 94.44 and 95.56, respectively. The incidence of death may be due to the disturbance of organs function (aflatoxicosis caused liver neoplasm, necrosis of hepatocytes and degenerative changes in pancreatic and kidney tissues), immune responsiveness, anaemia and cytotoxic effects (Lovell, 1991 and Verma and Mehta, 1998). The decrease of mortality rate by BNT may be due to decrease aflatoxin uptake, therefore decrease its effect on body organs (Zaki et al., 2008). While, AA improve the broiler performance, immunity and general health ((Lohakare et al., 2005).

The aflatoxin excreted in faeces (Table 6) of chickens fed aflatoxin contaminated diets plus BNT + AA (group 5) and aflatoxins + BNT (group 3) were higher than fed aflatoxin alone or with AA. These results may be due to ability of BNT to bind aflatoxin in the gastrointestinal tract which decrease aflatoxin uptake and bioavailability (Zaki et al., 2008).

4. Histopathological examination:

The histopathological examination on liver reveled that toxic effect of aflatoxin was shown to be focal hepatic hemorrhage (Fig. 1), dispread the hepatocytes faraway from each other and fatty degeneration of hepatocytes with signet ring appearance (Fig. 2 and 3). The present findings are in harmony with those obtained by Amera (2014) who reported that the toxic effect of aflatoxin B_1 contaminated diet (0.25 ppm) on rabbit liver was shown to be focal necrosis and replacement with mononuclear cells and bile ductless. Also, Prabu et al., (2013) showed vacuolar degeneration of hepatocytes, bile duct epithelial hyperplasia and hypertrophy and peribiliary fibrosis were consistently observed. BNT addition to aflatoxins contaminated diet showed portal infiltration with leucocytes and vacuolization of hepatocytes (Fig. 4), whereas, other sections from this group showed apparent normal hepatic parenchyma (Fig. 5). Aflatoxins contaminated diet plus AA showed hydropic degeneration of hepatocytes (Fig. 6 and 7). Addition of BNT + AA, revealed apparent normal hepatocytes (Fig. 8).

Histopathological examination of kidneys of chickens fed aflatoxins contaminated diet revealed fatty degeneration of epithelial lining tubules (Fig. 9, 10, 11) and congestion of inter tubular blood vessels (Fig. 10). These results agree with those obtained by Amera (2014), who reported that aflatoxin B_1 contaminated diet (0.25 ppm) caused necrotic renal tubules together with cystic dilation and hyaline casts. Moreover, addition of BNT showed fatty degeneration of epithelial lining renal tubules (Fig. 12). Addition of AA, revealed slight vacuolations of some renal tubular epithelium (Fig. 13). No histopathological changes were noticed in kidneys of chickens fed aflatoxins plus BNT + AA (Fig. 14 and 15).

6. Economical efficiency:The economical efficiency results indicated that all additions improved the economical efficiency (Table 7). The relative profit of chicks fed aflatoxin contaminated diet was 60.99% in compared with 79.61, 71.84 and 87.54% in chicks fed aflatoxins plus BNT, AA and BNT + AA, respectively and 100% of control group. These results may be due to improvement feed utilization and growth performance.

CONCLUSIONS

Adding 0.5% sodium bentonite (BNT), 500 mg ascorbic acid (AA) / kg feed, or BNT + AA to chicks aflatoxin contaminated diet, minimize aflatoxin toxicity. Addition of BNT + AA was more efficient than BNT or AA in minimizing of chicks aflatoxin contaminated diet

Table (1): formula and chemical composition (%) of the commercial basal diet.

Ingredients	**%**
Yellow corn	63.5
Soybean meal	31.6
Dicalcium phosphate	1.8
Limestone	1.4
Premix (vitamin and mineral mixture)*	0.3
Sodium chloride	0.3
Dl-methionine	0.1
Sand	1
Total	100
Calculated chemical analysis :**	
Crude protein (%)	19.55
Metabolizable energy (kcal/kg)	3835
Calcium concentration (%)	1.01
Available phosphorus (%)	0.485
Methionine (%)	0.43
Methionine + cystine (%)	0.65
Lysine (%)	1.078

*each 3 kg contains: 12000.000 iu vitamin A, 2000.000 iu vitamin D3, 10000 mg vitamin E, 1000 mg vitamin k3, 1000 mg vitamin B1, 5000 mg vitamin B2, 1500 mg vitamin B6, 10000 mg pantothenic acid, 10 mg vitamin B12, 1000 mg folic acid, 3000 mg niacin, 100 mg cobalt, 300 mg iodine, 100 mg selenium, 40000 mg manganese, 3000 mg ethoxyqush.

**according to NRC (1994).

Table(2): Effect of aflatoxin B_1 , sodium bentonite and vitamin C on body weight and body weight gain of El-Salam chicks.

Parameters	Treatments				
	Control	**Aflatoxin**	**Aflatoxin + bentonite**	**Aflatoxin + ascorbic acid**	**Aflatoxin + bentonite + ascorbic acid**
			Body weight (g)		
Initial weight	34 ±1	34 ± 0.9	33.50 ±1	34.50 ±1	34 ± 0.9
2 weeks	$100^{a} \pm 5$	$80^{c} \pm 2$	$95^{ab} \pm 5$	$900^{b} \pm 3$	$95^{ab} \pm 2$
4 weeks	$250^{a} \pm 2$	$230^{d} \pm 5$	$280^{b} \pm 3$	$270^{c} \pm 2$	$280^{b} \pm 5$
6 weeks	$550^{a} \pm 1$	$390^{e} \pm 2$	$490^{c} \pm 2$	$460^{d} \pm 2$	$505^{b} \pm 5$
8 weeks	$800^{a} \pm 2$	$580^{e} \pm 1$	$700^{c} \pm 5$	$650^{d} \pm 5$	$745^{b} \pm 1$
10 weeks	$1005^{a} \pm 2$	$771.33^{e} \pm 7$	$905^{c} \pm 5$	$850^{d} \pm 1$	$950^{b} \pm 3$
12 weeks	$1205^{a} \pm 5$	$900^{e} \pm 1$	$1050^{c} \pm 5$	$1000^{d} \pm 1$	$1100^{b} \pm 5$
			Body weight gain (g)		
2 weeks	66a ±4	$46^{c} \pm 3$	$61.5^{ab} \pm 4$	$55.5^{b} \pm 4$	$61^{ab} \pm 3$
4 weeks	$190^{a} \pm 3$	$150^{c} \pm 3$	$185^{ab} \pm 2$	$180^{b} \pm 5$	$185^{ab} \pm 7$
6 weeks	$260^{a} \pm 3$	$160^{e} \pm 3$	$210^{c} \pm 5$	$190^{d} \pm 4$	$225^{b} \pm 10$
8 weeks	$250^{a} \pm 1$	$190^{e} \pm 3$	$210^{c} \pm 3$	$200^{d} \pm 3$	$240^{b} \pm 4$
10 weeks	$205^{a} \pm 4$	$190^{b} \pm 4$	$205^{a} \pm 2$	$200^{a} \pm 6$	$205^{a} \pm 4$
12 weeks	$200^{a} \pm 3$	$130^{d} \pm 4$	$145^{c} \pm 10$	$150^{c} \pm 1$	$160^{b} \pm 2$
Average	$195.17^{a} \pm 0.7$	$144.33^{e} \pm 0.2$	$169.42^{c} \pm 0.7$	$162.58^{d} \pm 0.2$	$179.33^{b} \pm 0.3$

*Means in the same row bearing different letters differ significantly ($P \leq 0.05$).

Table (3): Effect of aflatoxin B_1 , sodium bentonite and vitamin C on feed intake of El-Salam chicks.

Parameters	Treatments				
	Control	**Aflatoxin**	**Aflatoxin + bentonite**	**Aflatoxin + ascorbic acid**	**Aflatoxin + bentonite + ascorbic acid**
2 weeks	150.00^{a} ±5	135.00^{c} ±10	145.00^{ab} ±10	140.00^{b} ±10	145.00^{ab} ±13
4 weeks	483.33^{a} ± 30	400.00^{c} ± 10	450.00^{b} ± 10	445.00^{b} ± 13	450.00^{b} ± 10
6 weeks	700.00^{a} ± 10	519.67^{c} ± 57	616.00^{b} ± 20	580.00^{b} ± 13	620.00^{b} ± 12
8 weeks	773.33^{a} ± 36	600.00^{e} ± 25	650.00^{c} ± 20	610.00^{d} ± 15	680.00^{b} ± 13
10 weeks	793.33^{a} ± 12	650.00^{d} ± 20	780.00^{b} ± 10	760.00^{c} ± 15	780.00^{b} ± 12
12 weeks	810.00^{a} ± 20	680.00^{e} ± 10	780.00^{c} ± 15	770.00^{d} ± 13	790.00^{b} ± 12
Average	618.33^{a} ± 15	502.50^{e} ± 12	570.17^{c} ± 12	550.83^{d} ± 12	577.50^{b} ± 10

*Means in the same row bearing different letters differ significantly ($P \leq 0.05$).

Table (4): Effect of aflatoxin B_1 , sodium bentonite and vitamin C on feed conversion of El-Salam chicks.

Parameters	Treatments				
	Control	**Aflatoxin**	**Aflatoxin + bentonite**	**Aflatoxin + ascorbic acid**	**Aflatoxin + bentonite + ascorbic acid**
2 weeks	$2.27^{b} \pm 0.07$	$2.95^{a} \pm 0.21$	$2.36^{b} \pm 0.14$	$2.53^{b} \pm 0.2$	$2.38^{b} \pm 0.17$
4 weeks	$2.55^{ab} \pm 0.20$	$2.67^{a} \pm 0.06$	$2.43^{b} \pm 0.08$	2.47ab ± 0.06	$2.43^{b} \pm 0.09$
6 weeks	$2.69^{c} \pm 0.04$	$3.44^{a} \pm 0.03$	$2.93^{b} \pm 0.08$	$3.05^{b} \pm 0.08$	$2.76^{c} \pm 0.11$
8 weeks	$3.09^{ab} \pm 0.02$	$3.16^{a} \pm 0.02$	$3.10^{ab} \pm 0.06$	$3.05^{b} \pm 0.07$	$2.83^{c} \pm 0.06$
10 weeks	$3.87^{a} \pm 0.13$	$3.09^{b} \pm 0.50$	$3.81^{a} \pm 0.01$	$3.80^{a} \pm 0.09$	$3.81^{a} \pm 0.08$
12 weeks	$4.05^{b} \pm 0.05$	$5.23^{b} \pm 0.16$	$5.40^{a} \pm 0.34$	$5.13^{a} \pm 0.02$	$3.61^{a} \pm 0.63$
Average	$3.17^{d} \pm 0.02$	$3.48^{a} \pm 0.01$	$3.37^{b} \pm 0.01$	$3.39^{b} \pm 0.02$	$3.22^{c} \pm 0.02$

*Means in the same row bearing different letters differ significantly ($P \leq 0.05$).

Table (5): Effect of aflatoxin B_1 , sodium bentonite and vitamin C on plasma constituents of El-Salam chicks.

Parameters	Treatments				
	Control	**Aflatoxin**	**Aflatoxin + bentonite**	**Aflatoxin + ascorbic acid**	**Aflatoxin + bentonite + ascorbic acid**
Toatal protein (g/dl)	$4.48^{a} \pm 0.02$	$3.24^{e} \pm 0.02$	$3.77^{d} \pm 0.03$	$3.89^{c} \pm 0.03$	$4.25^{b} \pm 0.02$
Albumin (g/dl)	$3.08^{a} \pm 0.02$	$2.15^{d} \pm 0.01$	$2.60^{c} \pm 0.02$	$2.77^{b} \pm 0.02$	$3.10^{a} \pm 0.02$
Globulin (g/dl)	$1.40^{a} \pm 0.01$	$1.09^{e} \pm 0.01$	$1.19^{b} \pm 0.01$	$1.12^{d} \pm 0.01$	$1.15^{c} \pm 0.01$
Aspertate amino transferase (u/l)	$118.00^{d} \pm 12$	$132.00^{a} \pm 1$	$128.00^{b} \pm 10$	$124.00^{c} \pm 20$	$122.00^{c} \pm 10$
Alanine amino transferase (u/l)	$13.50^{e} \pm 0.1$	$16.20^{a} \pm 0.2$	$15.60^{b} \pm 0.10$	$14.80^{c} \pm 0.1$	$14.40^{d} \pm 0.20$
Creatinine (mg/dl)	$0.22^{c} \pm 0.01$	$0.30^{a} \pm 0.01$	$0.28^{ab} \pm 0.01$	$0.26^{b} \pm 0.03$	$0.25^{b} \pm 0.02$
Uric acid (mg/dl)	$3.94^{e} \pm 0.02$	$5.39^{a} \pm 0.03$	$4.85^{b} \pm 0.03$	$4.38^{c} \pm 0.01$	$4.31^{d} \pm 0.01$

*Means in the same row bearing different letters differ significantly ($P \leq 0.05$).

Table(6): Effect of aflatoxin B_1 , sodium bentonite and vitamin C on mortality rate aflatoxin excreted in feces of El-Salam chicks.

Treatments	Survival rate (%)	Residue of aflatoxins as B1 (ppb)
Control	96.67	-
Aflatoxin	88.89	99.1
Aflatoxin + bentonite	95.56	155.2
Aflatoxin + ascorbic acid	94.44	104.3
Aflatoxin + bentonite + ascorbic acid	95.56	161.9

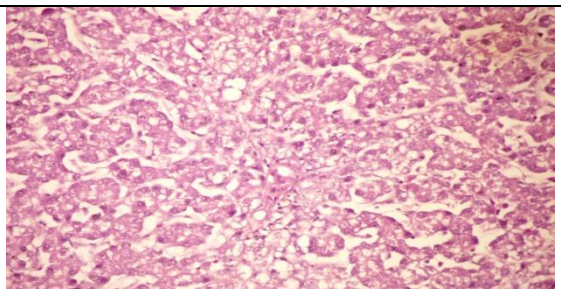

Fig.(1): liver of afl. group, showing focal hepatic heamorrhage (H and E x400)

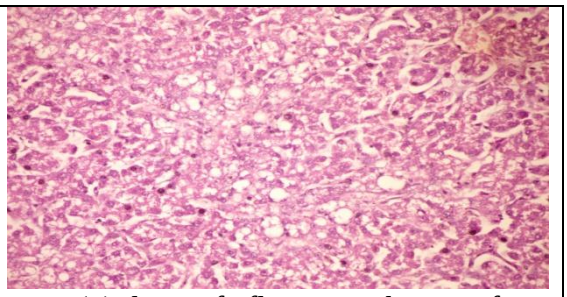

Fig.(2): liver of afl. group, showing fatty degeneration of hepatocytes with signet ring appearance (H and E x400)

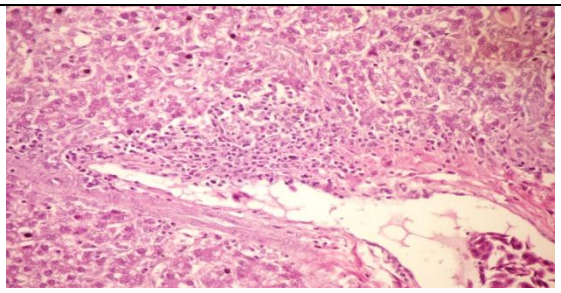

Fig.(3): liver of afl. + bentonite group, showing portal infiltration with leucocytes and vacuolization of hepatocytes (H and E x400)

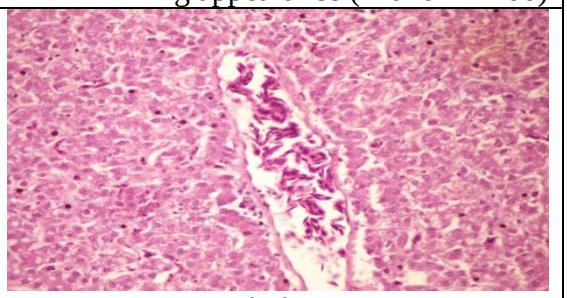

Fig.(4): liver of afl. + bentonite group, showing apparent normal hepatic parenchyma (H and E x400)

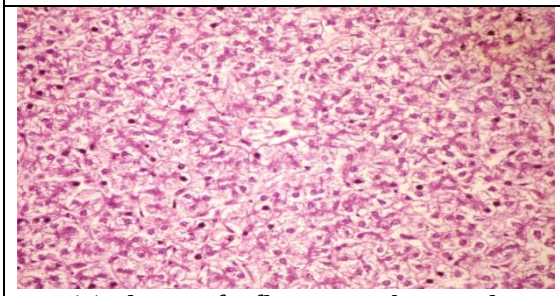

Fig.(5): liver of afl. + ascorbic acid group, showing hydrobic degeneration of hepatocytes (H and E x400)

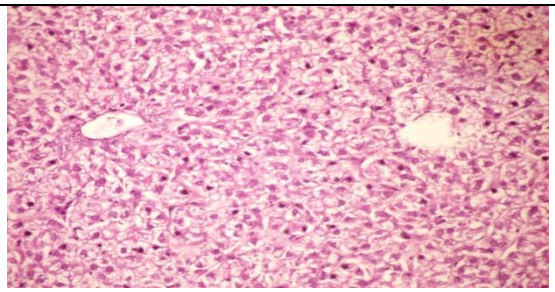

Fig.(6): liver of afl. + ascorbic acid group, hydrobic degeneration of hepatocytes (H and E x400)

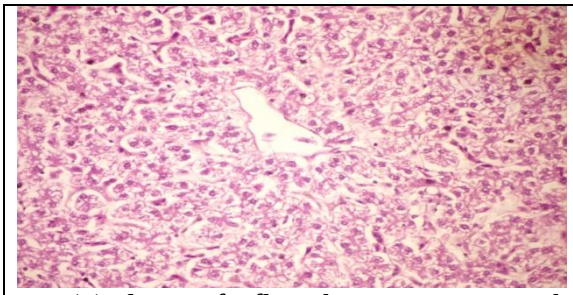

Fig.(7): liver of afl. + bentonite + ascorbic acid , showing small vacuoles in the cytoplasm of hepatocytes (H and E x400)

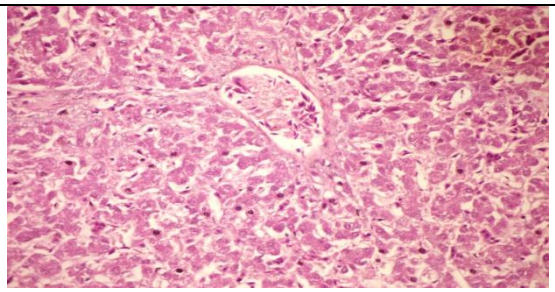

Fig.(8): liver of afl. + bentonite +ascorbic acid, showing apparent normal hepatocytes (H and E x400)

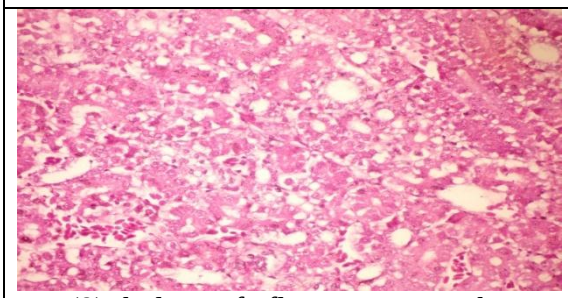

Fig.(9): kidney of aflatoxin group, showing fatty degeneration of epithelial lining renal tubules (H and E x400)

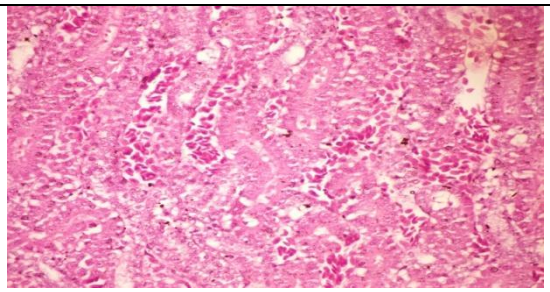

Fig.(10): Kidney of aflatoxin group, showing congestion of intertubular blood vessels and fatty degeneration of epithelial renal tubules
(H and E x400)

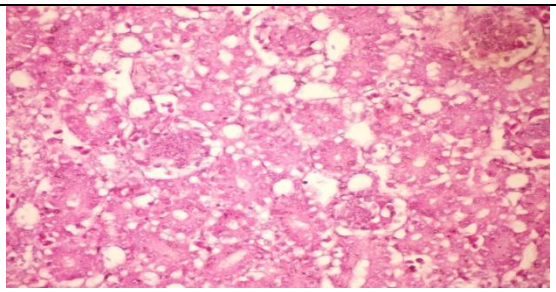

Fig.(11): Kidney of aflatoxin group, showing fatty degeneration of epithelial lining renal tubules (H and E x400)

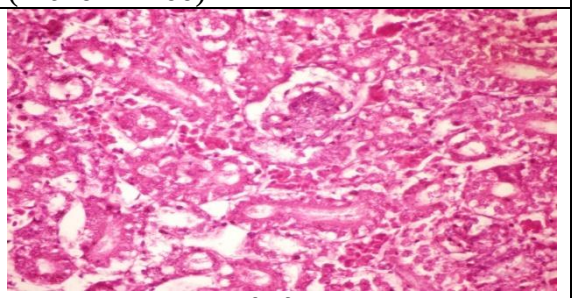

Fig.(12): Kidney of aflatoxin + bentonite group, showing fatty degeneration of epithelial lining renal tubules (H and E x400)

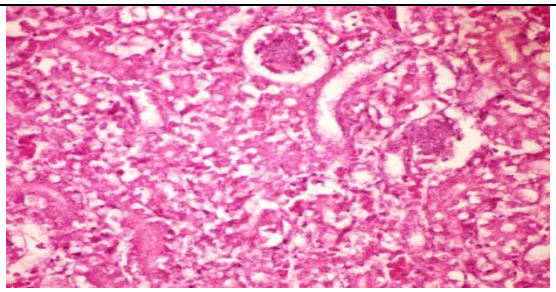

Fig.(13): Kidney of aflatoxin + ascorbic acid group, showing slight vaculations of some renal tubular epithelium (H and E x400)

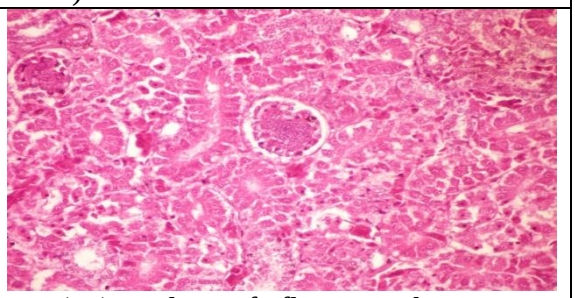

Fig.(14): Kidney of aflatoxin + bentonite + ascorbic acid group, showing no histopathological changes (H and E x400)

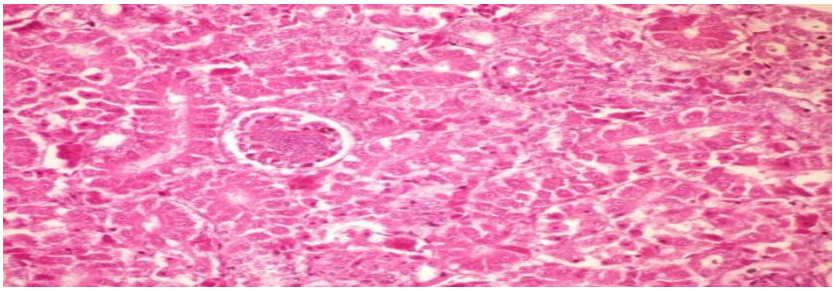

Fig.(15): Kidney of aflatoxin + bentonite + ascorbic acid , showing no histopathological changes (H and E x400)

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الملخص العربى

تأثير البنتونايت وحامض الاسكوربيك على تقليل السمية في علائق الكتاكيت الملوثة بالافلاتوكسين B1

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تم استخدام خمس مجموعات تجريبية لتقييم كفاءة الصوديوم بنتونايت (BNT) وحامض الاسكوربيك (AA) فى تقليل سمية الأفلاتوكسين B1 فى علائق كتاكيت السلام (كتاكيت مصرية محلية). تم توزيع ٤٥٠ كتكوت غير مجنس عمر يوم على خمس مجموعات عشوائيا واحتوت كل مجموعة على خمس مكررات (١٨ كتكوت / مكررة). كانت المعاملات التجريبية كما يلى : ١- عليقة قاعدية (كنترول) ، ٢- كنترول + أفلاتوكسين B1 (١ ملجم / كجم عليقة) ، ٣- كنترول + أفلاتوكسين B1 + BNT ٠,٥% ، ٤- كنترول + أفلاتوكسين B1 + ٥٠٠ ملجم AA/ كجم عليقة ، ٥- كنترول + أفلاتوكسين B1 + BNT + AA.

قللت العليقة الملوثة بالافلاتوكسين B1 معنويا (على مستوى ٥%) الزيادة اليومية فى وزن الجسم ، الغذاء المأكول، كفاءة التحويل الغذائى، معدل الحيوية، البروتين الكلى فى الدم، الألبيومين، الجلوبيولين. على الجانب الاخر أدى الأفلاتوكسين B1 الى ارتفاع الاسبرتيت أمينوترانزفيريز ، الالانين أمينوترانزفيريز، الكرياتينين وحامض اليوريك. حدثت أيضا تغيرات ضارة على كبد وكلى الكتاكيت المصابة الأفلاتوكسين B1. اضافة BNT ، AA ، BNT + AA للعليقة الملوثة بالأفلاتوكسين B1 حسنت معنويا (على مستوى ٥%) كل القياسات التى تأثرت سلبيا بالافلاتوكسين. اشارت معظم النتائج الى ان اضافة BNT + AA كانت اكفأ من اضافة BNT أو AA بمفرده فى تقليل التأثير السلبي للأفلاتكسين في علائق الكتاكيت.