

Protective Effect of Turmeric on Liver and Kidney in Chicken Aflatoxicosis

Majid Gholami-Ahangaran, Nader Rangsaz, and Ezatollah Fathi-Hafshejani

Abstract— In this study, 270 broiler chicks randomly divided to six groups. The chicks fed basal diet (as control), Turmeric extract (TE), Mycoad (M), 3ppm productive aflatoxin (AF), AF plus TE, and AF plus M in basal diet, all over growing period. In 28 days old, after euthanization, liver and kidney samples were collected in formalin for pathological examination. Macroscopic evaluation in chickens fed aflatoxin revealed liver and kidney enlargement and discoloration. In chickens fed uncontaminated diet and AF+M or AF+TE no gross lesions were observed. Microscopic examination showed sever congestion, degeneration and necrosis in liver and kidney in chickens received aflatoxin. Supplement of Mycoad^{TR} or turmeric in diet contaminated with aflatoxin could moderate damages in liver and kidney. Based pathological evaluation, it seems that turmeric extract as well as Mycoad can decrease pathological effects of aflatoxin on liver and kidney and can be used as an anti-aflatoxicosis in chickens.

Keywords—Aflatoxicosis, Chicken, Kidney, Liver, Turmeric.

I. INTRODUCTION

AFLATOXINS (AF) as a secondary metabolite of *Aspergillus* species, are carcinogenic, mutagenic, and teratogenic for human and animals [1]. The major source of exposure to AF is via the ingestion of contaminated food and feed.

Chickens are highly sensitive to the adverse effect of AF. Aflatoxicosis is a cause of economic losses in poultry industry [2]. Aflatoxicosis in chickens is characterized by mortality, listlessness, anorexia, decreased growth rates, negative feed conversions, fatty liver, decreased egg production, poor pigmentation, and increased susceptibility to other diseases [2]-[4]. Preventing of mould growth and AF contamination in feed and feedstuffs is very important but when contamination cannot be prevented, decontamination of AF is needed before using these materials. A practical approach to detoxification is the use of sorbents in the diet that adsorb AF in the gastrointestinal tract of poultry and reduce bioavailability and toxicity [4]. Until now, aluminosilicate compounds used as a common commercial anticaking agent for prevention of aflatoxicosis in animal and poultry. The purpose of the study was to evaluate the effectiveness of one antioxidant herbal

Majid Gholami-Ahangaran, Department of Poultry Diseases, Veterinary Medicine Faculty, Shahrekord Branch, Islamic Azad University, Shahrekord, Iran (corresponding author's phone:00989133231388; e-mail:mgholamia1388@yahoo.com).

Nader Rangsaz, Graduated from Veterinary Medicine Faculty, Shahrekord Branch, Islamic Azad University, Shahrekord, Iran (e-mail:n.rangsaz@gmail.com).

Ezatollah Fathi-Hafshejani, Department of Poultry Diseases, Veterinary Medicine Faculty, Shahrekord Branch, Islamic Azad University, Shahrekord, Iran (e-mail:ezzatfathi@yahoo.com).

plant with one reference and standard commercial aluminosilicate (Mycoad, Alteck com., USA) in reducing the negative effects of AF on pathological parameters of liver and kidney in broiler chickens.

II. MATERIALS AND METHODS

A. Aflatoxin Production

Aflatoxin provided by *Aspergillus parasiticus* (PTCC: 1850) belongs to Iranian Scientific and Industrial Researches. Aflatoxin was produced according to the Shotwell method [5] on maize. Productive aflatoxin was assessed by competitive ELISA kit (Romer Co., USA).

B. Preparation of Turmeric Extract

Rhizomes of turmeric (*Curcuma longa L.*) were obtained and alcoholic extract was prepared according to the conventional method [6].

C. Experimental Design

A total of 270 one-day-old broiler chicks (Ross strain) were randomly divided into six groups with three replicates of 15 chicks in each in a separate pen during the 28-day experiment. Treatment groups including: group A; chickens fed basal diet, group B; chickens fed 3 ppm productive aflatoxin in a basal diet, group C; chickens fed basal diet plus 0.05% turmeric extract, group D; chickens fed 3 ppm productive aflatoxin and 0.05% turmeric extract in a basal diet, group E; chickens fed basal diet containing 0.25% Mycoad^{TR} and group F; chickens received basal diet containing 3 ppm productive aflatoxin and 0.25% Mycoad^{TR}.

The maize containing aflatoxin was added to the basal diet to reach an aflatoxin concentration in the experimental diet of 3 ppm. All treatment groups received experimental diets throughout the growing period from hatch till 28 days old.

D. Histopathological Examination

At end of growing period, all chickens were euthanized. Gross lesions in liver and kidney were observed and recorded. For histopathology analysis, liver and kidney of all chickens were fixed in 10% formaline, and after primary preparation, the sections were stained with hematoxylin-eosin (H&E) method [7] for microscopic examination. Sections of liver and kidney with no, mild, moderate, and sever lesions were given scores of 0, 1, 2, and 3, respectively [8].

III. RESULTS

In chickens fed 3 ppm aflatoxin, hepatic enlargement and yellowish discoloration were noted. In some birds, mottling of

liver and distension of gall bladder were seen. The kidneys from the aflatoxin fed chickens were enlarged, pale or congested with a few petechia. In chickens fed uncontaminated diet no gross lesions were observed.

Microscopically, chickens fed 3 ppm aflatoxin exhibited mainly vacuolar and fatty degeneration in hepatocytes. The architecture of the liver was completely changed. Congestion, bile duct hyperplasia, multifocal infiltration of heterophils and mononuclear cells diffused in parenchyma especially in portal area. Perivascular infiltration of mononuclear cells and heterophils and necrosis were also noticed in the livers.

Pathology sections from the kidneys of chickens fed aflatoxin showed congestion, multifocal hemorrhages, and vacuolar degeneration of tubular epithelium. Furthermore, in a few sections increase in thickness of basement membranes were seen. In addition, chickens received aflatoxin alone showed some lymphocytic aggregations in the kidneys.

These changes in liver and kidney in chickens received aflatoxin plus turmeric extract or aflatoxin plus mycoad were much less than chickens fed aflatoxin alone. Control chickens did not show any changes in all sampled tissues. The statistical analysis of pathology results showed there were significant differences in the lesion scores of liver and kidney of chickens receiving aflatoxin alone with other treated chickens. There were no significant differences between chickens fed aflatoxin plus turmeric or aflatoxin plus mycoad (Table 1).

TABLE I
LIVER AND KIDNEY PATHOLOGICAL SCORES IN DIFFERENT TREATMENT GROUPS

Treatments/Organs	Liver	Kidney
control	0.11±0.33 ^a	0.13±0.35 ^a
AF	2.44±0.73 ^c	1.60±0.27 ^c
M	0.22±0.44 ^a	0.30±0.48 ^a
TE	0.44±0.53 ^a	0.30±0.48 ^a
M+AF	1.22±0.66 ^b	1.20±0.78 ^b
TE+AF	1.44±0.53 ^b	1.00±0.47 ^b

* Data presented as Mean ± SD.

^{a,b} Different words in each row represent the existence of significant differences between groups.

IV. DISCUSSION

The liver is considered the main target organ for aflatoxicosis [1]. In present study, chickens received 3 ppm aflatoxin were showed a variety degree of liver damages, comprising congestion of liver parenchyma, vacuolar degeneration of hepatocytes, hepatocyte necrosis and cellular infiltration. Also, aflatoxin intoxication was leaded to congestion of kidney parenchyma, degeneration and necrosis of tubular epithelial cells. Previously reported, the degenerative changes and hepatocellular necrosis following aflatoxicosis can be related to cellular macromolecules damages (Lipid, DNA and protein) that lead to lipid peroxidation and oxidative damage of DNA [9]-[10]. Ozen et al. [11] stated a dose dependent vacular degeneration in liver cells in aflatoxicosis, which was a sign of fat accumulation. In this study, we observed vacular degeneration similar to previous reports [11]-[12]. It was reported that vacular degeneration could be due to impaired lipid transport rather than increases lipid biosynthesis [13]. Supplement of turmeric and Mycoad in diet containing 3 ppm aflatoxin can cause

change of lesions from sever or moderate to moderate until mild. Reduction in severity of lesions in chickens receiving aflatoxin plus Mycoad may be related to neutralization of main part of ingested aflatoxin by silica [8] that available in Mycoad. Therefore, aflatoxin does not reach to liver. Lower severity of lesions in chickens receiving aflatoxin plus turmeric extract can be related to antioxidant property of turmeric.

In present study supplement of turmeric and Mycoad in diet containing aflatoxin could not prevent of aflatoxin induced lesions on kidney and liver, perfectly. Base on available data, there is no scientific evidence in agreement or contrast that it is necessary to further study. In this study, pathological findings obtained from the aflatoxin plus turmeric group are close to the aflatoxin plus mycoad group. These results show that turmeric can be used to prevent aflatoxicosis.

V. CONCLUSION

It was concluded that turmeric extract can decrease adverse effect of aflatoxin on liver and kidney and can be used as a supportive treatment in aflatoxicosis in chickens.

REFERENCES

- [1] H. N. Mishra, and D. Chitrangada, "A review on biological control and metabolism of aflatoxin," *Crit. Rev. Food Sci. Nutr.*, vol. 43, pp. 245–264, 2003.
<http://dx.doi.org/10.1080/10408690390826518>
- [2] D. Tedesco, S. Steidler, S. Galletti, M. Tamani, O. Sonzogni, and L. Ravarotto, "Efficacy of silymarinphospholipid complex in reducing the toxicity of aflatoxin B1 in broiler chicks," *Poultry Sci.*, vol. 83, pp. 1839-1843, 2004.
<http://dx.doi.org/10.1093/ps/83.11.1839>
- [3] N. Rangsaz, and M. Gholami Ahangaran, "Evaluation of turmeric extract on performance indices impressed by induced aflatoxicosis in broiler chickens," *Toxicol. Ind. Health*, vol. 27, pp. 956-960, 2011.
<http://dx.doi.org/10.1177/0748233711401262>
- [4] M. Gholami-Ahangaran, and N. Zia-Jahromi, "Nanosilver effects on growth parameters in experimental aflatoxicosis in broiler chickens," *Toxicol. Ind. Health*, vol. 29, pp. 121-125, 2013.
<http://dx.doi.org/10.1177/0748233711425078>
- [5] L. O. Shotwell, C. W. Hesseltine, R. D. Stubblefield, and W. G. Sorenson, "Production of aflatoxin on rice," *Appl. Mic.*, vol. 14, pp. 425-428, 1966.
- [6] M. Ahmadi, H. R. Rasekh, M. Kamali Nejad, and A. Zare, "The effect of *Curcuma longa* Roxb extract on inflammation of rheumatoid arthritis in male rats," *Herbal Plants*, vol. 7, pp. 21-28, 2007.
- [7] J. D. Bancroft, and A. Stevens, "Theory and practice of histopathological techniques," London: Churchill livingstone, 1996.
- [8] N. K. S. Gowda, D. R. Ledoux, G. E. Rottinghaus, A. J. Bermudez, and Y. C. Chen, "Efficacy of turmeric (*Curcuma longa*), containing a known level of curcumin, and a hydrated sodium calcium aluminosilicate to ameliorate the adverse effects of aflatoxin in broiler chicks," *Poultry Sci.*, vol. 87, pp. 1125-1130, 2008.
<http://dx.doi.org/10.3382/ps.2007-00313>
- [9] M. A. Hashem, and M. H. Mohamed, "Hemato-biochemical and pathological studies on aflatoxicosis and treatment of broiler chicks in Egypt," *Veterinaria Ital.*, vol. 45, pp. 323-327, 2009.
- [10] A. Allameh, A. R. Safamehr, S. A. Mirhadi, S. Mahmoud, M. Razzaghi-Abyaneh, and A. Afshar-Naderi, "Evaluation of biochemical and production parameters of broiler chicks fed ammonia treated aflatoxin contaminated maize grains," *Anim. Feed Sci. Tech.*, vol. 122, pp. 289-301, 2005.
<http://dx.doi.org/10.1016/j.anifeedsci.2005.03.005>
- [11] H. Ozen, M. Karama, Y. Cigremis, M. Tuzcu, K. Ozcan, and D. Eradag, "Effectiveness of melatonin on aflatoxicosis in chicks," *Res. Vet. Sci.*, vol. 86, pp. 485- 489, 2009.

<http://dx.doi.org/10.1016/j.rvsc.2008.09.011>

- [12] M. M. Kiran, O. Demet, M. Ortatatlı, and H. Oguz, "The preventive effect of polyvinylpolypyridone on aflatoxicosis in broilers," *Avian Pathol.*, vol. 27, pp. 250-255, 1998.
<http://dx.doi.org/10.1080/03079459808419332>
- [13] M. McLean, and M. F. Dutton, "Cellular interaction and metabolism of aflatoxin: an update," *Pharmacol. Therapeut.*, vol. 65, pp. 163-192, 1995.
[http://dx.doi.org/10.1016/0163-7258\(94\)00054-7](http://dx.doi.org/10.1016/0163-7258(94)00054-7)