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CHANGES IN SOME BIOCHEMICAL PARAMETERS AND BODY WEIGHT OF CHICKEN EXPOSED TO CADMIUM

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ABSTRACT

This study was conducted with 6 week old domestic chicken to determine the effect of supplementation of cadmium to dietary. 10 mg/kg Cadmium chloride added to maize- sesame cake meal diet for 4 weeks. The additional cadmium to the diet induced a decreasing body weight and changes in biochemical parameters of chicken. Chicken were divided into two groups. The first group was given a diet containing the concentration of 10 mg cadmium /kg daily for a period of 4 weeks and the second group was given diet without cadmium and used as a control group. The result revealed decrease in the body weight of treated chicken by 12.7% compared to control group, whose body weight increased. The plasma glucose concentration, creatinine, aspartate aminotranseferase (AST), and alanine aminotransferase (ALT) were increased significantly ($P< 0.05$) in Cd treated chicken in comparison to the control group. Cadmium accumulation was observed in the kidney, liver, intestine and muscle. The accumulation of cadmium was markedly higher (3-4 times) in cadmium-treated animals compared to the control.

KEYWORDS: Cadmium, chicken, Body weight and biochemical parameters.

INTRODUCTION

Cadmium (Cd) belongs to the group of highly toxic heavy metals[1], it is found widely in nature and present in air, all soils and aquatic systems. Considerable amounts of Cd get continuously into the environment as a consequence of human activities. The steel industry, waste disposal, volcanic action, zinc refinery, fossil fuels, and traffic are accounted for the largest part of emissions of atmospheric Cd. Raised concentrations of Cd in soil may be found as a result of industrial activities (e.g. mining) or agricultural activities (e.g. sewage sludge, phosphate fertilizers, and pesticides) containing high concentrations of Cd.[2]

Meat, fish, and fruits generally contain up to 50 µg Cd.kg-1 on fresh weight basis, whereas vegetables, potatoes, and grain products may contain up to 150 µg Cd.kg-1 fresh weight. Higher concentrations are found in the kidneys of animals slaughtered for food, in wild mushrooms, and in seafood such as mussels and oysters.[3]

Consumption of contaminated water is the major way by which humans are exposed to Cd[4] and the maximum allowable level in drinking-water is 0.005 mg/dL.[5] Animals can be exposed to Cd pollution by: inhalation of polluted air, ingestion of polluted food and drinking of polluted water.[6,2,7,8] It is absorbed from gastrointestinal tract to blood, and cadmium is taken up from the blood into the tissues. The Cd accumulates in human and animals tissues, especially the liver and kidney, causing their damage.[2] Cadmium induced injury to liver and kidney organs has been attributed to its ability to enhance free radical formation in vivo.[9,10] Cadmium in high doses induces structural and function alterations in various vital organs including liver, kidney, gill and intestine of fishes. Cadmium accumulates in liver of fishes in high concentrations.[11,12] It also induces various pathological changes in liver tissues including engorgement of blood vessels, congestion, vacuolar degeneration of hepatocytes, necrosis of pancreatic cells and fatty changes in the peripancreatic hepatocytes.[13,14] Some biochemical characteristic features are associated with Cd-induced kidney and liver damage. Others are marked increase of plasma creatinine or blood urea nitrogen levels[15] as well as altered plasma glucose level[16], and changes in antioxidant enzymes (GOT and GPT) level, which produced in damaged liver tissues. The objective of this work is to study the effect of cadmium on the changes of weight, and some haematological and biochemical parameters in chicken.

MATERIALS AND METHODS

Six weeks old chickens (n= 16) are collected from their habitat and were housed in cages to acclimatization for one week in laboratory conditions at the room temperature between 28 – 30^0C. After the acclimatization period, chicken were divided into two groups (n=8) randomly and each group was kept in separate cages. The first group was given a maize- sesame cake meal diet containing the concentration of 10 mg cadmium ($Cdcl_2$) /kg and the second group was given maize- sesame cake meal diet without cadmium and used as a control group. The two groups fed ad libitum composition of the diet table (1) for 4 weeks.

Every chicken was weighted before starting the experiment and body weight ranged 250- 300 ± gms, then every chicken was weighted after 4 weeks of experiment period. The blood

samples were collected with heparinized syringe from the wing vein and was used to obtain the plasma by centrifugation (5000 rpm) for 5 min. The plasma glucose, craetinine, ALT and AST were determined using Randox kits.

The animals were sacrificed by cervical dislocation and the kidneys, liver, intestine and muscle were removed, rinsed in ice-cold saline. Tissues were dried and digested with HNO3/H2O2. The Cd content of the homogenates were determined with Atomic absorption spectrophotometer. Cd contents are reported in µg/g dry weight.

Statistical analysis

The data is presented as means ± SD. The mean values of the control and test groups were compared using Student's t-test. The significant level was set at $P < 0.05$.

RESULTS

Body weight gain

The result in table [2] shows the body weight of the control group increased by 37 %, whereas the body weight of Cd treated group decreased by 12.7 % after 4 weeks. The gain in body weight of the treated group were lowered in relation to the control which was reduced to 46% after 4 weeks of treatment.

Biochemical parameters

Table (3) shows the changes of some biochemical parameters in Cd treated chickens compared to control chickens one month of 10mg/kg Cd exposure to diet.

The plasma glucose concentration showed higher significant values ($P< 0.05$) (196 ±24mg/dl) in chickens exposed to Cd for 4 weeks than the control chicken group value (127.3 ±15).

The plasma creatinine increased significantly ($P< 0.05$) in chickens exposed to Cd 4 weeks (0.75 ± 0.16) when compared to the control group (0.42 ± 0.11). The changes in AST and ALT activities are used to check liver function in the Cd treated chicken relative to control group. The Results also revealed that cadmium causes significant ($P< 0.05$) increasing of activity of ALT and AST, as seen in table (3). The plasma ALT and AST level reached (7.7 ± 1.3and 141± 16) in treated chickens comparison to control group (5.6 ± 1and 97 ± 14) respectively.

As can be seen in Table (4), the accumulation of Cd in the kidneys, liver, intestine and muscle, where concentrations of cadmium increased in all tissues.The Cd content reached high level in kidney (186 ± 25 µg/g) and the low level in muscle (18 ± 2.2 µg/g).

Table (1): Composition of the diet

Ingredient	Percentage	
	Control group	Cd-treated group
Sesame seed cake	40	40
Zea mays bran	30	30
Wheat bran	30	30
cadmium ($Cdcl_2$)	-	10 mg/kg

Table (2): Growth performance of chickens exposed to Cd in diet (mean ± SD)

Items	Initial weight	Weight after 4 weeks	Weight gain
Control	276 ± 20.7	390 ± 25.5	114 ± 11.4
Cadmium treated	284 ± 11.9	222 ± 13.5*	− 62 ± 14.8

**: P<0.05 comparing with control*

Table (3): Cadmium effect on blood glucose, AST, ALT and Creatinine in plasma of the chickens (mean ± SD)

Parameter	control	4 weeks
Glucose	127.3 ±15	196 ±24
AST	97 ± 14	141± 16
ALT	5.6 ± 1	7.7 ± 1.3
Creatinine	0.42 ± 0.11	0.75 ± 0.16

**: P<0.05 comparing with control*

Table (4) Cadmium accumulation (µg/g dry weight) in chicken tissues

Items	Liver	Kidney	intestine	Muscle
Control	2.8 ± 0.3	5.4 ± 0.45	2.5 ± 0.25	0.8 ± 1.6
Cadmium treated	90 ± 5	186 ± 25	49 ± 6.5	18 ± 2.2

DISCUSSION

The results showed that the chicken exposed to 10 mg/kg Cd in diet showed a significant lower chicken growth. The final body weight of Cd treated chicken was significantly lower than that of the control group. In young animals [e.g. calf, swine, rabbit, Japanese quail, chicken, rat], the different Cd salts can reduce feed intake and growth rate.[17,18,19]

Blood glucose is a sensitive reliable indicator of environmental stress in chicken. From the results, it is clear that Cd as shown by the elevated blood glucose level affected as a stress of Cd on chicken. Cd induced hyperglycemia with decreased in liver glycogen in catfish,

Heteropneustes fossilis.[20,21],[22] suggested that hyperglycemia occurred in Atlantic salmon *(Salmo salar*) after toxicity with cadmium may be due to changes in liver carbohydrate metabolism (activation of liver glycogenolysis and glycolysis) as well as increased levels of plasma glucose.

The activity of AST and ALT enzymes in blood may also be used as a stress indicator. The significant changes in activities of these enzymes in blood plasma indicates tissue impairment caused by stress.[23,24] In the present study, there were significant changes in AST and ALT activities in plasma of chicken exposed to cadmium compared to the control group. The increase in concentration of AST and ALT in blood plasma indicates impairment of liver. In addition, the increase of plasma AST and ALT may be attributed to the hepatocellular damage or cellular degradation by these heavy metal, perhaps in liver, heart or muscle[25] These results are in agreement with those of[26] who found that sublethal concentration of Cd caused significant increases in AST and ALT of common carp after 7 and 15 days. Also,[27] found that Increased activity of serum enzymes ALT and AST in guinea pigs. Similar result demonstrated by[28] who found that the cadmium exposure induced serum AST, ALT increasing of rat.

In the present study, kidney function tests showed elevation in creatinine in the treated group. The results are in agreement with that reported by[28,29] in guinea pigs and rats.[27], mentioned that the toxic effect of cadmium on the renal tissues was clear as the level of creatinine in serum increased. The elevation in the creatinine was due to nephrotoxic effect of cadmium on renal tubules and glomeruli. Also,[30] found significant elevation in serum creatinine concentration in Cd intoxicated rats. They added that creatinine metabolism was thought to reflect the amount of glomerular filtration.[26], described a significantly increased creatinine level in Cd-treated chickens.

The results obtained in this study show that concentration of Cd in liver and kidneys were significantly increased in animals exposed to Cd ($p<0.05$).[31], found that increased accumulation of Cd in the kidneys, liver and intestine in mice and fish liver.[32,33] found that the diets contained cadium caused accumulation of Cd in liver and kidney of lambs.

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