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NUTRACEUTICAL EFFECT OF GINGEROL ON HAEMATOBIOCHEMICAL, LIVER ANTIOXIDANT STATUS AND PATHOLOGICAL CHANGES AGAINST PENICILLIC ACID MYCOTOXICOSIS IN BROILER CHICKENS

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ABSTRACT

To find out the alleviative effect of gingerol on haematobiochemical, organ weight, hepatic lipid peroxidation, antioxidant status and pathological changes against penicillic acid mycotoxicosis in broiler chickens. Out of 80 day-old broiler chicks obtained, 72 were randomly allotted to six groups of 12 chicks each. The birds were fed with diets [(T1-control, T2- Gingerol (0.1%), T3- Gingerol (0.2%), T4- Penicillic acid (15 ppm), T5- Penicillic acid (15 ppm) + gingerol (0.1%), T6- Penicillic acid (15ppm) + gingerol (0.2%)] from 0 to 28 days of age. Remaining eight chicks were used as no toxin, no gingerol and no vaccine group (T7). On the 28th day of trial, remaining birds were sacrificed. The clinical sign observed in the penicillic acid toxin treated birds was depression. The PCV values of T2 and T3 groups decreased significantly ($P<0.05$) when compared to the T5 and T6 groups. The Hb values of T1, T2 and T6 were highly significantly ($P<0.01$) lower than that of T5 group. The nutraceutical effect of gingerol (0.1 and 0.2%) studied against PA toxicity (15 ppm) showed partial alleviation at 0.2 per cent level. Gingerol alone could induce hyperamylaesaemia (gingerol 0.1%), hypercholesterolaemia, hypertriglyceridaemia, increased BUN, creatinine, LDL, VLDL and HDL (gingerol 0.2%). Further gingerol alone induced toxic changes in the liver, kidney, crop, proventriculus. There were mild to moderate improvements in the serum biochemical parameters as well as histological lesions, gingerol at 0.1 per cent could not completely alleviate the toxic effects of penicillic acid (15 ppm). The above results indicated that incorporation of gingerol at 0.2 per cent level could reverse most of the toxic effects of penicillic acid (15 ppm).

KEYWORDS: Gingerol, Penicillic acid toxicity, haematobiochemical alterations, liver antioxidant assays, Pathology,

INTRODUCTION

Penicillic acid (PA), a mycotoxin, was originally isolated from the cultures of *Penicillium puberulum* (Alsberg CL and Black OF, 1913). *P. cyclopium* Westling produced relatively larger amounts of penicillic acid (Bentley R and Keil JG, 1962; Birkinshaw JH et al.1936). The natural occurrence of penicillic acid has been detected in the poultry feed, corn, dried beans, cheese, salami and tobacco products (LeBars J. 1980). The penicillic acid toxins interfered with protein formation by the action on nucleic acids, due to the interaction of penicillic acid with the enzymes of carbohydrate metabolism and affected the lipid metabolism with lowered levels of total lipids (Pandiyan V et al., 1987). The penicillic acid toxin has been shown to have antibacterial, antiviral, antitumour, antidiuretic, cytotoxic, hepatotoxic and carcinogenic properties in the mice and rats (Chan

PK et al., 1980; Kawaski I et al.1972; Phillips TD et al., 1980; Suzuki S et al., 1971). *Zingiber officinale Roscoe* commonly known as ginger (*Zingiberaceae*), a spice and flavouring is acting as carminative, anti-emetic, spasmolytic, peripheral circulatory stimulant, anti-inflammatory (Bradley PR. 1992) and antioxidant (Bhandari U.2003; Jitoe A et al.1992; Krishnakanth TP and Lokesh BR, 1990; Reddy AC and Lokesh BR, 1992). Limited information is available on the hepatoprotective activity of ginger rhizome (Hikino H et al.1985; Shirwaikar A et al. 1992; Sohini YR and Bhatt RM, 1996; Sohini YR et al. 1995). Ginger prevents hepatotoxicity by reducing hepatic injury, exhibiting membrane stabilizing and antioxidant properties (Bhandari U. 2003).

The present study was programmed to find out the alleviative effect of gingerol on haematobiochemical, organ weight, hepatic lipid peroxidation, antioxidant status and pathological changes against penicillic acid mycotoxicosis in broiler chickens

MATERIALS AND METHODS

1. PREPARATION OF FUNGAL CULTURE

The *Penicillium cyclopium* NRRL 1888 culture was obtained from the National Center for Agricultural Utilization Research, Microbial Genomics and Bioprocessing Research Unit, 1815 N University Street, Peoria, Illinois 61604, USA. The *P. cyclopium* NRRL 1888 was subcultured on potato dextrose agar at 10 days interval (Ciegler A et al. 1972). The penicillic acid toxin was produced on maize (LeBars J. 1980). The maize samples were pre-tested for the presence of mycotoxins. The penicillic acid from ground maize culture samples were quantified by using thin layer chromatography at the Central Animal Feed and Food Residue Laboratory, Directorate of Centre for Animal Health Studies, Tamil Nadu Veterinary and Animal Sciences University, Chennai–600 051.

2. PREPARATION OF GINGEROL

Ginger soft SCF (Super Critical Fluid) extracts 20 per cent containing 6-gingerol (65-70%), 8-gingerol (22-25%) and 10-gingerol (7-10%) was obtained from M/s. Sami Labs, 19/1 &19/2, I main, II phase, Peenya Industrial Estate, Bangalore. Powdered maize culture material containing known amounts of penicillic acid was incorporated into the toxin free diet, so that the diet contained 15 ppm of penicillic acid. Gingerol was added at the levels of 0.1 and 0.2 per cent in the diet.

3. EXPERIMENTAL DESIGN

Out of 80 day-old broiler chicks obtained, 72 were randomly allotted to six groups of 12 chicks each. Remaining eight chicks were used as no toxin, no gingerol and no vaccine group. The birds were fed with following diets from 0 to 28 days of age. Two birds from each group were sacrificed on 7^{th}, 14^{th}, 21^{st} and 28^{th} day to study the CMI of the birds. On the 28^{th} day of trial, remaining birds were sacrificed to study the haematobiochemical alternations, pathological changes in different organs and immune status.

Groups	No. of birds
Control	12
Gingerol (0.1%)	12
Gingerol (0.2%)	12
Penicillic acid (15 ppm)	12
Penicillic acid (15 ppm) + gingerol (0.1%)	12
Penicillic acid (15ppm) + gingerol (0.2%)	12
No toxin, no gingerol, no vaccine	8

4. HAEMATOLOGY

Blood samples were collected by intracardiac puncture in Heller and Paul double oxalate anticoagulant mixture. Haematological studies included the estimation of PCV by microhaematocrit method, Hb by acid haematin method and TEC using Hayem's fluid (Coles EH, 1986).

5. SERUM BIOCHEMISTRY

Another set of blood samples collected were allowed to clot and centrifuged at 1500 rpm for 20 min to separate the sera. Serum total protein and albumin were estimated by modified Biuret and Dumas method (Varley H et al. 1980), glucose by glucose oxidase method, total cholesterol (TC) by cholesterol dehydrogenase peroxidase method, AST, ALT and ALP by IFCC (International

Federation of Clinical Chemistry) method, BUN by glutamyl dehydrogenase method, creatinine by Jaffe's kinetic method, uric acid by enzymatic photometric test by IFCC method (Burtis CA and Ashwood ER. 1996), calcium by O-crespophthalein complexone method, phosphorus by modified metol method, sodium and potassium by colorimetric method, amylase by colorimetric method (Coles EH, 1986), serum lipase by turbidimetric UV method (Burtis CA and Ashwood ER. 1996), HDL-Cholesterol by precipitation method and triglycerides (TG) by colorimetric enzymatic method using semi-automatic analyser (Mispa excel, Agappe Diagnostic, India). VLDL was arrived by using the formula TG/5 and LDL using the formula TC-HDL-VLDL and TC/HDL was also found out.

6 LIVER LIPID PEROXIDATION AND ANTIOXIDANTS

6.1. Sample collection

Liver tissue samples collected from control and toxin fed birds were stored at - 20°C till the required assays were carried out.

6.2. Estimation of liver lipid peroxidation

Liver lipid peroxidation was estimated by the formation of thiobarbituric acid (TBARS) following the method of Yagi K. (1976).

6.3. Estimation of liver antioxidants

The protein content was estimated by the method of Lowry OH et al. (1951). Glutathione peroxidase (GPx) was estimated by the method of Rotruck *et al*. (1973), glutathione-S-transferase (GST) by the method of Habig WH et al. (1974), superoxide dismutase (SOD) by the method of Marklund SL and Marklund G, (1974) and catalase (CAT) by the method of Caliborne AL. (1985). Reduced glutathione (GSH) was estimated by the method of Moron MS et al. (1979).

7. LIVER WEIGHT

The liver weight was measured in all the groups of this experimental trial

8. PATHOLOGY

After collection of blood, the birds were sacrificed by cervical dislocation and a detailed post mortem examination was conducted on sacrificed birds. Representative samples of tissues from liver, kidney, crop, proventriculus, gizzard, duodenum, pancreas, spleen, bursa of Fabricius, thymus, caecal tonsils and Harderian gland were collected in 10 per cent formal saline. Paraffin embedded tissues were sectioned to 5 μm thickness and stained by haematoxylin and eosin H&E) for histopathological examination (Bancroft JD and Gamble G. 2008).

9. Statistical analysis

The data generated from different experimental trials were subjected to one-way analysis of variance (ANOVA) using SPSS version 10 software for windows.

RESULTS

CLINICAL SIGNS

Depression was observed in the penicillic acid toxin treated birds.

HAEMATOLOGY

The PCV values of gingerol alone fed groups decreased significantly ($P<0.05$) when compared to the PA+gingerol groups. The Hb values of control, gingerol (0.1%) and PA+gingerol (0.2%) were highly significantly ($P<0.01$) lower than that of PA+gingerol (0.1%) group as shown in Table 1.

Table 1

Mean (± SE) haematological values of broiler chicks fed gingerol against penicillic acid

Groups	PCV (%)	Hb (g/dL)	TEC (millions/cmm)
Control (0 ppm)	$32.00^{ab} \pm 2.93$	$9.87^{yz} \pm 0.29$	2.03 ± 0.13
Gingerol (0.1%)	$25.50^{b} \pm 4.54$	$9.38^{z} \pm 0.29$	2.28 ± 0.22
Gingerol (0.2%)	$25.50^{b} \pm 1.31$	$10.17^{yz} \pm 0.28$	2.56 ± 0.27
PA (15 ppm)	$33.50^{ab} \pm 1.11$	$10.70^{xy} \pm 0.25$	2.31 ± 0.19
PA (15 ppm) + Gingerol (0.1%)	$35.67^{a} \pm 1.76$	$11.37^{x} \pm 0.73$	2.35 ± 0.22
PA (15 ppm) + Gingerol (0.2%)	$34.83^{a} \pm 3.66$	$9.67^{yz} \pm 0.21$	2.20 ± 0.15

Means with same superscripts within a column (a,b/x,y,z) do not differ from each other (P>0.05/P>0.01)

SERUM BIOCHEMISTRY

Highly significant (P<0.01) increase in the total protein and globulin values of gingerol (0.2%) and PA fed groups were observed when compared to other groups as shown in Table.2. The AST values were significantly (P<0.05) lower in the PA+gingerol (0.1 and 0.2%) treated groups (Table.3). The hyperamylaesemia was observed in PA+gingerol (0.1%) group (Table.3). Gingerol (0.2%) group showed elevation of BUN and creatinine values as shown in Table 4. Highly significant (P<0.01) increase in the serum lipid profile values were observed in the gingerol (0.2%) group when compared to the other groups except for HDL-cholesterol which showed significant (P<0.05) increase in the PA+gingerol (0.1%) group as shown in Table 5. Hypercalcaemia was observed in PA+gingerol (0.2%) group as shown in Table 6.

Table 2

Mean (± SE) serum total protein, albumin, globulin (g/dL) and Albumin to globulin ratio of broiler chicks fed gingerol against penicillic acid

Groups	Total protein g/dL)	Albumin g/dL)	Globulin g/dL)	Albumin to globulin
Control (0 ppm)	$.26^{y} \pm 0.23$	$.21 \pm 0.16$	$.06^{ab} \pm 0.24$	0.64 ± 0.12
Gingerol (0.1%)	$.09^{y} \pm 0.25$	$.15 \pm 0.26$	$.48^{a} \pm 0.23$	0.59 ± 0.23
Gingerol (0.2%)	$.16^{x} \pm 0.18$	$.52 \pm 0.18$	$.63^{a} \pm 0.31$	0.61 ± 0.10
PA (15 ppm)	$.57^{x} \pm 0.17$	$.22 \pm 0.20$	$.61^{a} \pm 0.42$	0.53 ± 0.16
PA (15 ppm)+ Gingerol (0.1%)	$.34^{z} \pm 0.31$	$.01 \pm 0.16$	$.47^{b} \pm 0.19$	0.61 ± 0.05
PA (15 ppm)+ Gingerol (0.2%)	$.33^{y} \pm 0.28$	$.25 \pm 0.17$	$.77^{ab} \pm 0.23$	0.74 ± 0.12

Means with same superscripts within a column (a,b/x,y,z) do not differ from each other (P>0.05/P>0.01)

Table 3

Mean (± SE) serum enzyme values (U/L) of broiler chicks fed gingerol against penicillic acid

Groups	ALT (U/L)	AST (U/L)	ALP (U/L)	Amylase (U/L)	Lipase (U/L)
Control (0 ppm)	40.21± 6.82	$199.59^{x} \pm 21.76$	3216.63 ± 917.41	$345.30^{b} \pm 74.90$	24.85 ± 2.81
Gingerol (0.1%)	41.31± 9.29	$168.13^{xy} \pm 7.82$	5477.97 ± 1672.55	$612.41^{ab} \pm 214.46$	22.85 ± 4.12
Gingerol (0.2%)	35.17 ± 2.07	$171.05^{xy} \pm 22.27$	5826.53 ± 1112.51	$693.23^{ab} \pm 81.22$	17.43 ± 0.84
PA (15 ppm)	39.13 ± 4.45	$211.41^{x} \pm 13.49$	5274.02 ± 1928.64	$408.44^{b} \pm 94.56$	19.29 ± 0.53
PA (15 ppm) + Gingerol (0.1%)	17.07 ± 9.61	$129.12^{y} \pm 14.33$	6854.50 ± 2822.75	$968.93^{a} \pm 236.85$	18.32 ± 2.01
PA (15 ppm) + Gingerol (0.2%)	27.68 ± 2.99	$126.78^{y} \pm 8.82$	4168.62 ± 1311.55	$276.65^{b} \pm 55.74$	20.75 ± 1.08

Means with same superscripts within a column (a,b,c/x,y) do not differ from each other (P>0.05/P>0.01)

Table 4

Mean (± SE) BUN and serum creatinine and uric acid values (mg/dL) of broiler chicks fed gingerol against penicillic acid

Groups	BUN (mg/dL)	Creatinine (mg/dL)	Uric acid (mg/dL)
Control (0 ppm)	$4.22^{y} \pm 0.68$	$0.17^{b} \pm 0.03$	6.39 ± 0.83
Gingerol (0.1%)	$3.68^{y} \pm 0.82$	$0.24^{b} \pm 0.05$	6.21 ± 0.91
Gingerol (0.2%)	$7.68^{x} \pm 1.05$	$0.44^{a} \pm 0.09$	5.88 ± 1.07
PA (15 ppm)	$3.50^{y} \pm 0.46$	$0.33^{ab} \pm 0.05$	6.23 ± 0.66
PA (15 ppm) + Gingerol (0.1%)	$3.29^{y} \pm 0.44$	$0.23^{b} \pm 0.07$	6.18 ± 0.29
PA (15 ppm) + Gingerol (0.2%)	$3.19^{y} \pm 0.37$	$0.24^{b} \pm 0.02$	5.38 ± 0.35

Means with same superscripts within a column (a,b,c/x,y) do not differ from each other (P>0.05/P>0.01)

Table 5

Mean (± SE) serum lipid profile values (mg/dL) of broiler chicks fed gingerol against penicillic acid

Groups	Total cholesterol (mg/dL)	Triglycerides (mg/dL)	HDL (mg/dL)	LDL (mg/dL)	VLDL (mg/dL)	TC/HDL ratio
Control (0 ppm)	$74.58^{y} \pm 6.94$	$100.72^{yz} \pm 6.73$	$13.11^{b} \pm 1.19$	$38.83^{y} \pm 6.29$	$20.14^{yz} \pm 3.35$	$5.24^{yz} \pm 0.72$
Gingerol (0.1%)	$66.87^{y} \pm 10.54$	$62.89^{z} \pm 8.13$	$12.74^{b} \pm 0.69$	$41.55^{y} \pm 8.85$	$12.58^{z} \pm 1.63$	$4.94^{yz} \pm 0.92$
Gingerol (0.2%)	$133.86^{x} \pm 8.75$	$214.21^{x} \pm 19.19$	$13.19^{b} \pm 0.78$	$79.83^{x} \pm 5.53$	$42.84^{x} \pm 3.84$	$10.42^{x} \pm 0.75$
PA (15 ppm)	$85.14^{y} \pm 6.62$	$113.85^{y} \pm 13.66$	$13.02^{b} \pm 1.12$	$49.33^{x} \pm 6.21$	$22.78^{y} \pm 2.73$	$6.69^{y} \pm 0.65$
PA (15 ppm)+ Gingerol (0.1%)	$67.35^{y} \pm 7.79$	$70.62^{z} \pm 5.29$	$18.73^{a} \pm 2.05$	$34.69^{y} \pm 7.82$	$13.93^{z} \pm 1.15$	$3.89^{z} \pm 0.65$
PA toxin (15 ppm) + Gingerol (0.2%)	$65.65^{y} \pm 6.18$	$75.77^{yz} \pm 8.67$	$15.53^{ab} \pm 1.81$	$31.56^{y} \pm 4.81$	$13.56^{z} \pm 0.52$	$4.41^{z} \pm 0.48$

Means with same superscripts within a column (a,b/x,y,z) do not differ from each other (P>0.05/P>0.01)

Table 6

Mean (± SE) serum electrolyte (mEq/L) and mineral values (mg/dL) of broiler chicks fed gingerol against penicillic acid

Groups	Total cholesterol (mg/dL)	Triglycerides (mg/dL)	HDL (mg/dL)	LDL (mg/dL)	VLDL (mg/dL)	TC/HDL ratio
Control (0 ppm)	$74.58^{y} \pm 6.94$	$100.72^{yz} \pm 16.73$	$13.11^{b} \pm 1.19$	$38.83^{y} \pm 6.29$	$20.14^{yz} \pm 3.35$	$5.24^{yz} \pm 0.72$
Gingerol (0.1%)	$66.87^{y} \pm 10.54$	$62.89^{z} \pm 8.13$	$12.74^{b} \pm 0.69$	$41.55^{y} \pm 8.85$	$12.58^{z} \pm 1.63$	$4.94^{yz} \pm 0.92$
Gingerol (0.2%)	$133.86^{x} \pm 8.75$	$214.21^{x} \pm 19.19$	$13.19^{b} \pm 0.78$	$79.83^{x} \pm 5.53$	$42.84^{x} \pm 3.84$	$10.42^{x} \pm 0.75$
PA (15 ppm)	$85.14^{y} \pm 6.62$	$113.85^{y} \pm 13.66$	$13.02^{b} \pm 1.12$	$49.33^{x} \pm 6.21$	$22.78^{y} \pm 2.73$	$6.69^{y} \pm 0.65$
PA (15 ppm)+ Gingerol (0.1%)	$67.35^{y} \pm 7.79$	$70.62^{z} \pm 5.29$	$18.73^{a} \pm 2.05$	$34.69^{y} \pm 7.82$	$13.93^{z} \pm 1.15$	$3.89^{z} \pm 0.65$
PA toxin (15 ppm) + Gingerol (0.2%)	$65.65^{y} \pm 6.18$	$75.77^{yz} \pm 8.67$	$15.53^{ab} \pm 1.81$	$31.56^{y} \pm 4.81$	$13.56^{z} \pm 0.52$	$4.41^{z} \pm 0.48$

Means with same superscripts within a column do not differ from each other (P>0.05)

ESTIMATION OF LIVER LIPID PEROXIDATION AND LIVER ANTIOXIDANTS

No significant difference was observed between the control and other groups for liver TBARS and liver antioxidant assay values as shown in Table 7.

Table 7

Mean (± SE) liver TBARS and antioxidant values of broiler chicks fed gingerol against penicillic acid

Groups	TBARS[1]	SOD[2]	CAT[3]	GPx[4]	GST[5]	GSH[6]
Control (0 ppm)	426.70 ± 47.82	0.11 ± 0.01	0.66 ±0.08	525.54 ± 84.65	2.46 ± 0.56	602.37 ± 137.18
Gingerol (0.1%)	589.23 ± 79.29	0.10 ± 0.01	0.54 ±0.08	556.67 ± 64.73	1.58 ± 0.44	480.65 ± 68.26
Gingerol (0.2%)	439.90 ± 75.42	0.09 ±0.01	0.66 ±0.08	564.09 ± 63.78	1.92 ± 0.39	418.25 ± 39.96
PA (15 ppm)	556.47 ± 69.59	0.10 ±0.01	0.55 ±0.07	488.95± 42.06	3.63 ±1.01	252.29 ± 32.31
PA (15 ppm) + Gingerol (0.1%)	607.97 ± 32.92	0.09 ±0.01	0.78 ±0.08	528.29± 51.08	2.04 ± 0.33	269.23 ± 49.96
PA (15 ppm) + Gingerol (0.2%)	509.90 ± 70.21	0.11 ±0.01	0.64 ±0.14	768.15 ± 48.62	2.94 ± 0.72	537.54 ± 144.21

[1] TBARS level in mg/g of tissue

[2] Enzyme required for inhibiting 50% pyrogallol autooxidation/min/mg protein

[3] Catalase required for decomposing µm of H_2O_2/min/mg protein

[4] GPx expressed as µm of glutathione utilized/min/mg protein

[5] GST expressed as µm CDNB–GSH conjugate formed/min/mg protein

[6] GSH level in mg/g of tissue

LIVER WEIGHT

Highly significant (P<0.01) increase in the liver weight of gingerol (0.2%) group was observed when compared to the other groups except for gingerol (0.1%) and PA fed groups as shown in Table 8.

Table 8

Mean (± SE) relative liver weight (g) of broiler chicks fed gingerol against penicillic acid

Groups	Liver weights (g)
Control (0 ppm)	3.07^{y} ± 0.20
Gingerol (0.1%)	3.17^{y} ± 0.13
Gingerol (0.2%)	3.62^{x} ± 0.14
PA (15 ppm)	3.08^{y} ± 0.18
PA (15 ppm) + Gingerol (0.1%)	2.82^{y} ± 0.07
PA (15 ppm) + Gingerol (0.2%)	2.74^{y} ± 0.17

Means with same superscripts within a column do not differ from each other (P>0.01)

PATHOLOGY

Gross Pathology

Grossly, penicillic acid toxin (15ppm) fed birds showed pale and enlarged as shown in Figure 1 or yellowish discolouration of the liver.

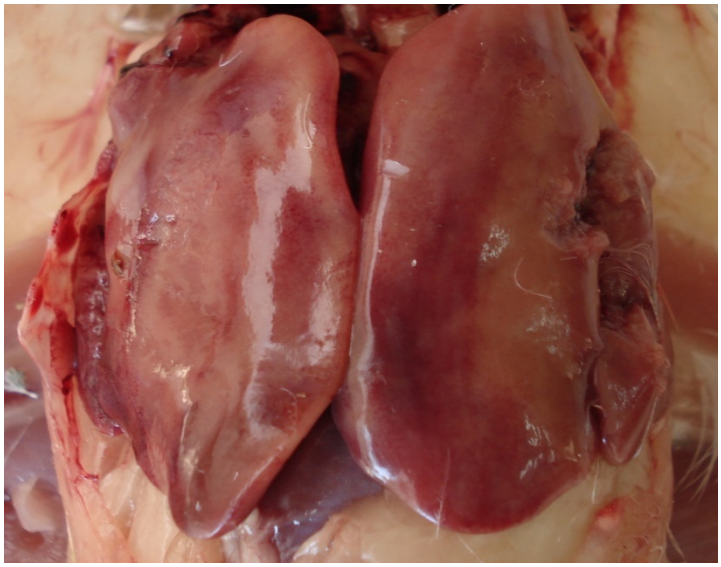

Figure 1
Broiler chicken-Penicillic acid (15 ppm)- Liver-Pale and enlarged

Histopathology

Microscopically, the gingerol (0.1%) fed birds showed diffuse vacuolar degeneration of hepatocytes and microgranulomas in the liver, focal crop mucosal hyperplasia and mild mononuclear cell infiltration and partial mucosal necrosis in the proventricular mucosa. The gingerol (0.2%) fed birds showed microgranuloma and mononuclear cell infiltration in the liver, mild interstitial nephritis and mild hyperplasia of crop mucosal epithelium. The penicillic acid (15 ppm) fed birds showed mild vacuolar degeneration of hepatocytes and focal necrosis with mononuclear cell infiltration in the liver as shown in Figure 2. Bile duct hyperplasia as shown in Figure 3) with periductular mononuclear cell infiltration and necrosis of cholangiolar epithelium were observed in three cases. Lumen of bile duct contained eosinophilic amorphous substances and mild periductular fibrosis was also observed in two cases. Hyperplastic crop mucosa was seen. The PA+gingerol (0.1%) fed birds showed focal necrosis of hepatocytes, microgranulaoma formation as shown in Figure 4 and mild biliary hyperplasia in the liver, mild mononuclear cell infiltration in the interstitium of the kidneys, mononuclear cell infiltration in the proventriculus as shown in Figure 5 and mucosal necrosis with mononuclear cell infiltration in the intestine. The PA+gingerol (0.2%) fed birds showed mononuclear cell infiltration in the proventricular mucosa, focal necrosis and mononuclear cell infiltration in the glandular gizzard.

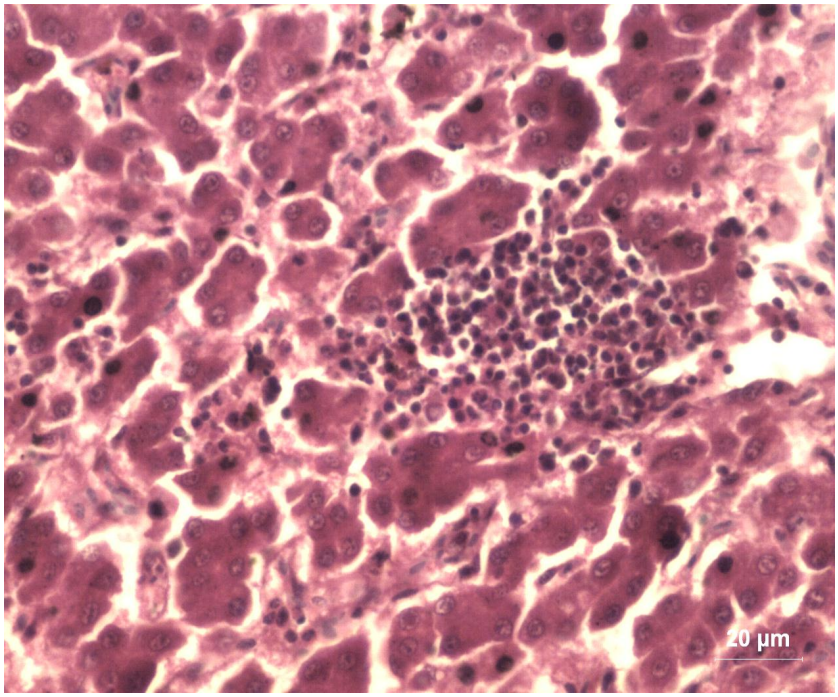

Figure 2
Broiler chicken-Penicillic acid (15 ppm)- Liver- Focal mononuclear cell infiltration H&E scale Bar 10 µm

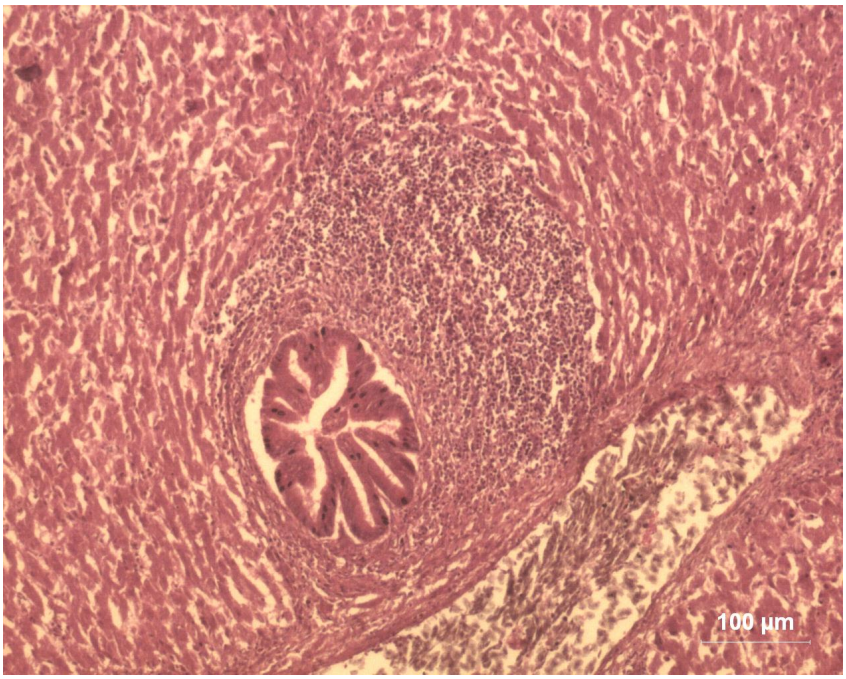

Figure 3
Broiler chicken- Penicillic acid (15 ppm)- Liver- Bile duct hyperplasia H&E scale Bar 100 µm

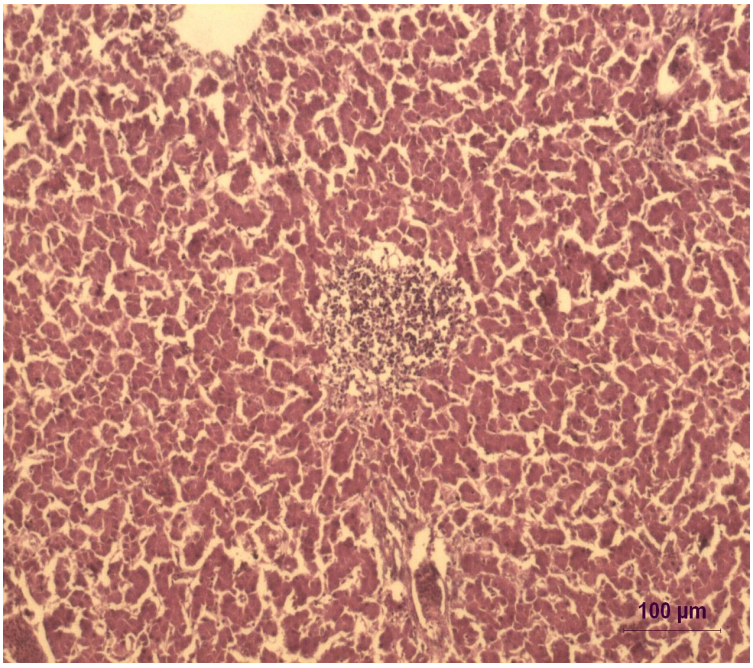

Figure 4
Broiler chicken-PA + Gingerol (0.1%)- Liver –Microgranuloma formation H&E scale Bar 100 µm

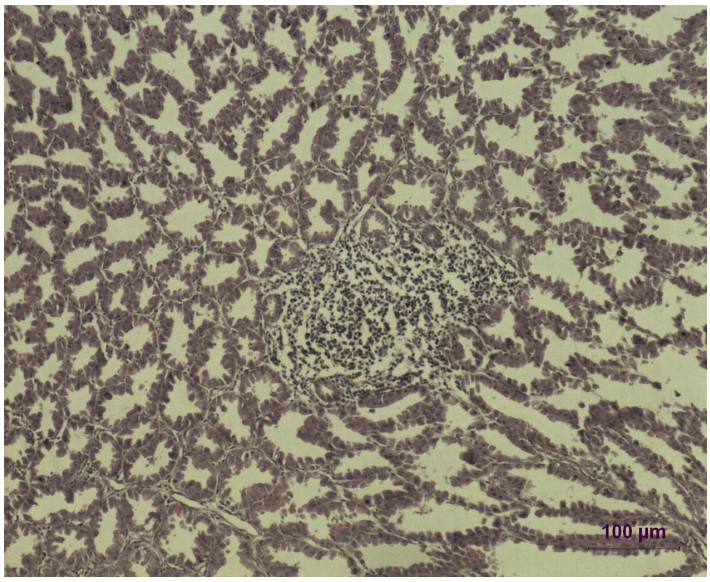

Figure 5
Broiler chicken-Penicillic acid (15ppm) + gingerol (0.1%) –Proventiriculus- Mononuclear cell infiltration H&E scale Bar 100 µm

DISCUSSION

Limited information is available on the hepatoprotective activity of ginger rhizome (Hikino H et al.1985; Shirwaikar A et al. 1992; Sohini YR and Bhatt RM, 1996; Sohini YR et al. 1995). The inclusion of gingerol (0.1%) in the broiler ration resulted in the elevation of amylase and microscopical changes of vacuolar degeneration of hepatocytes and microgranuloma formation in the liver, crop mucosal hyperplasia, proventricular mucosal mononuclear cell infiltration and partial mucosal necrosis. The inclusion of gingerol (0.2%) in the broiler ration resulted in the elevation of BUN and serum creatinine, total cholesterol, triglycerides, LDL, VLDL, TC/HDL ratio, and liver weight and revealed histological changes of microgranuloma

in the liver, mild interstitial nephritis and mild crop mucosal hyperplasia. The toxic changes observed in the penicillic acid toxin fed group viz. anaemic changes could be described to reduced feed intake and malabsorption due to alimentary tract pathology observed in this study, suppressive effect of toxins on the bone marrow (Coles EH 1986) and increased fragility of erythrocytes (Pandiyan V and Shanmugasundaram ERB, 1987). Grossly, penicillic acid toxin (15 ppm) fed birds showed pale and enlarged or yellowish discolouration of the liver which concurred with the findings of Sarmadha (Sarmadha MK. 2003) in experimentally induced PA toxin at the level 50-480 ppm in broiler chicken. The above changes are in accordance with the findings of (Sarmadha MK. 2003) but were reported at higher levels of penicillic acid toxicity (50-480 ppm) in broiler chicken. However, no hepatic pyogranuloma, basement membrane thickening in the glomeruli, ingluvitis and proventricular crypt elongation were observed in this study as reported by (Sarmadha MK. 2003). Probably, it requires higher level of penicillic acid to induce such pathological changes as employed by (Sarmadha MK. 2003).

The penicillic acid (15 ppm) effect on triglycerides, LDL, VLDL and TC/HDL values were reversed by gingerol (0.1%). The PA+gingerol (0.1%) fed broiler chicken showed elevation of serum HDL and amylase values, microscopical changes such as focal necrosis of hepatocytes, microgranuloma formation and mild biliary hyperplasia in the liver, mild mononuclear cell infiltration in the interstitium of tubules of the kidneys, mononuclear cell infiltration in the proventriculus, and mucosal necrosis with mononuclear cell infiltration in the intestine. Hyperproteinaemia, hypoglobulinaemia, increased LDL, VLDL and TC/HDL observed in the penicillic acid toxicity was reversed by dietary inclusion of gingerol at 0.2 per cent level. Microscopically moderate changes observed were mononuclear cell infiltration in the proventriculus and focal mononuclear cell infiltration in glandular gizzard. Hypercalcaemia was observed in this PA+gingerol (0.2%) group could be due to the increased resorption of calcium from the bone by penicillic acid (Pandiyan V. et al.1987).

CONCLUSION

The above results indicated that incorporation of gingerol at 0.2 per cent level could reverse most of the toxic effects of penicillic acid (15 ppm). Though there were mild to moderate improvements in the serum biochemical parameters as well as histological lesions, gingerol at 0.1 per cent could not completely alleviate the toxic effects of penicillic acid (15 ppm). There is no comparable literature available in this study in broiler chickens. Further studies are required to precisely find out the level of incorporation of gingerol and other phytochemicals to completely alleviate the toxic effects of penicillic acid.

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