

Histopathological Changes in the Livers of Broiler Chicken Supplemented with Turmeric (*Curcuma longa*)

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Abstract: Four groups of broiler chicken (A, B, C and D) of 50 birds each, received dietary supplement of turmeric (*Curcuma longa*) at concentrations of 0.0, 2.5, 5.0 and 10% respectively. Liver sections from birds of all groups showed parenchymal and portal infiltration of mononuclear cells and hyperaemia of portal vessels. Dilatation of bile ducts, mild proliferation of biliary epithelium and periportal hepatocyte degeneration was noticed only in groups B, C and D particularly group B birds killed three weeks after treatment. These changes were related to feeding of Turmeric (*Curcuma longa*).

Key words: Chicken; turmeric (*Curcuma longa*); hepatic changes

Introduction

Curcuma longa (turmeric) is a perennial herb, that grows to a height of three to five feet and is cultivated extensively in Asia (India and China) and other countries with a tropical climate. Curcumin, the active ingredient from the spice turmeric is a potent antioxidant and anti-inflammatory agent with hepatoprotective, anticarcinogenic, and antimicrobial properties (Pal *et al.*, 2001). However, Deshpande *et al.* (1998) reported decreased body weights and hepatotoxic effects expressed as focal necrosis, in rats and mice fed turmeric or ethanolic turmeric extract, especially in high doses for prolonged periods. Similarly Kandarkar *et al.* (1998) observed coagulative necrosis together with areas of parenchymal regeneration using whole spice turmeric or ethenolic turmeric extract in doses considered to be cancer protective. On the other hand no toxic effects due to feeding of turmeric (or curcumin) were reported in rats, guinea pigs, monkeys and pigs (Wahlstrom and Blennow, 1978; Bhavanishankar *et al.*, 1980; Bille *et al.*, 1985).

The present study describes for the first time the histopathological changes in the livers of broiler chicken given dietary supplement of *Curcuma longa*.

Materials and Methods

Preparation of diet: Diets containing 2.5, 5 or 10% *Curcuma longa* were prepared by through mixing of 25, 50 or 100 of whole ground rhizomes (source-India) in 1000 kg basic broiler diet using a Hammer mill feed mixer (U.K) for one hour. Chemical analysis of the spice could not be done.

Experimental Design: 200 One day old chicks (Hybrid) were divided into four equal groups (A, B, C and D). They were fed basic diet* supplemented with *Curcuma longa*

in concentrations of 0.0, 2.5, 5.0 and 10.0% for groups A, B, C and D respectively. Half of the chicks were scarified by the end of the 3rd week of the experiment and the other half was scarified after the 6th week (marketing age). The birds were examined postmortem for presence of any gross abnormalities in various organs with particular stress upon the liver. The livers of five chicks selected randomly from each group were fixed in 10% formalin for histopathology.

Histopathological Methods: Liver samples were constantly taken from the dorsal part of the left lobe. These were processed in paraffin and sections 4-6µm thick were prepared and stained with haematoxylin and eosin (H and E).

Results

Postmortem Finding: No salient gross abnormalities could be observed in the various organs examined.

Histopathological Finding:

I. Chicks sacrificed at the end of the 3rd week of the experiment.

Group A : (0% *Curcuma longa* supplement): Liver sections showed hyperaemia of portal vessels with infiltration of lymphocytes and histocyte (plus hetrophils in some sections) in portal areas, and hepatic parenchyma (Fig.1).

Group B: (2.5% *Curcuma longa* supplement):* Asarco company Hyperaemia of portal vessels, dilatation of bile ducts with mild proliferation of bile duct epithelium and degenerative changes in surrounding hepatocytes were observed (Fig. 2, 3 and 4). Mononuclear cell infiltration

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Table 1: Comparison of hepatic lesions in birds supplemented with different concentrations of *Curcuma longa* and killed at the end of 3rd & 6th weeks of the experimented period

	G.A		G.B		G.C		G.D	
	3w	6w	3w	6w	3w	6w	3w	6w
Hyperaemia	+	+	+	++	+	+	+	++_
Mononuclear cell infiltration:Parenchyma	+	+_	+	++	+	++_	+	+
Portal areas	+	+	+	+++	+	++	+	+_
Dilatation of bile ducts	-	-	++	+	+	+_	+	+
Hyperplasia of bile duct epithelium	-	-	+	+	+_	-	+_	-
Periportal hepatocyte degeneration	-	-	++	+	+	-	+	++_

Group A : basic diet + 0% *Curcuma longa*; Group B: basic diet + 2.5% *Curcuma longa*

Group C : basic diet + 5% *Curcuma longa*; Group D : basic diet + 10% *Curcuma longa*

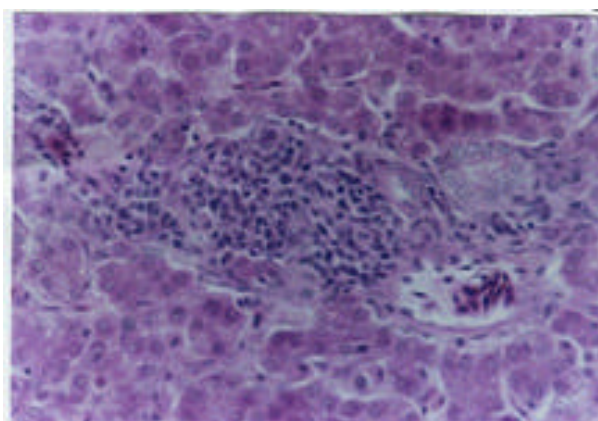


Fig. 1: Liver section (Group A 3 week after treatment) showing section mononuclear cell infiltration in portal area HE x 400

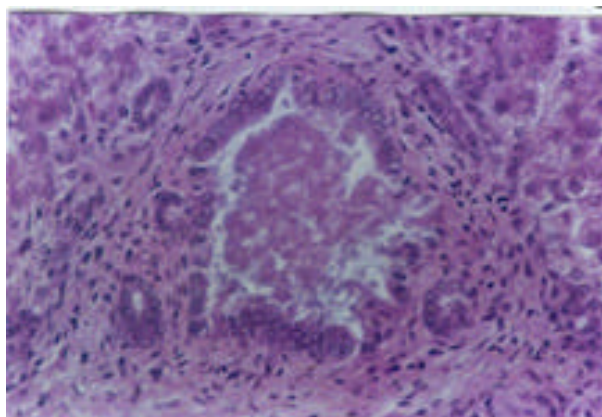


Fig .2: Liver section (group B 3 weeks after treatment) showing dilated bile duct with slight proliferation of lining epithelium HE x 400

(lymphocyte and histiocytes) was seen in parenchyma and portal areas.

Group C: (5% *Curcuma longa* supplement): Dilatation of

bile ducts, proliferation of bile duct epithelium and portal infiltration by mononuclear cells were less evident compared to group B. However, larger foci of mononuclear cell infiltration were seen in the parenchyma (Fig. 5).

Group D: (10% *Curcuma longa* supplement): Hepatic changes were similar to those of group C.

II. Birds scarified by the end of the 6th week of the experiment: In all groups liver sections showed hyperemia of portal vessels which was pronounced in group B-birds. Mononuclear cell infiltration in parenchyma and portal areas was more marked in liver sections of group B and C, particularly the former group. Dilatation of bile ducts, hyperplasia of bile duct epithelium and degeneration of surrounding hepatic cells were not distinct.

In general, chicks in group B and C and D exhibited hepatic changes of variable degrees. Dilatation of bile ducts with degeneration of surrounding hepatocytes were evident in birds of these groups sacrificed by the end of the 3rd week of the experiment especially in group B. On the other hand, mononuclear cell infiltration in hepatic parenchyma and portal areas was more pronounced in birds of group B&C sacrificed at the end of the 6th week of the experiment (Table 1).

Discussion

Tumeric has been used as a spice and therapeutic agent (Govindrajan, 1980; Ammon and Wahl, 1991). Most of its creditable functions are due to its anti-inflammatory and antioxidant properties (Sharma, 1976) as well as to its anticarcinogenic action (Nagabhusan and Bhide, 1987; Huang *et al.*, 1994; Deshpande *et al.*, 1997; Rao *et al.*, 1995). In Saudi Arabia it is widely used as a spice and is believed to be an effective digestive tonic.

Administration of high dose of tumeric or ethanolic tumeric extract for variable periods was found to induce hepatotoxic effects in mice and rats (Deshpande *et al.*, 1998; Kandarkar *et al.*, 1998). This was mostly in the

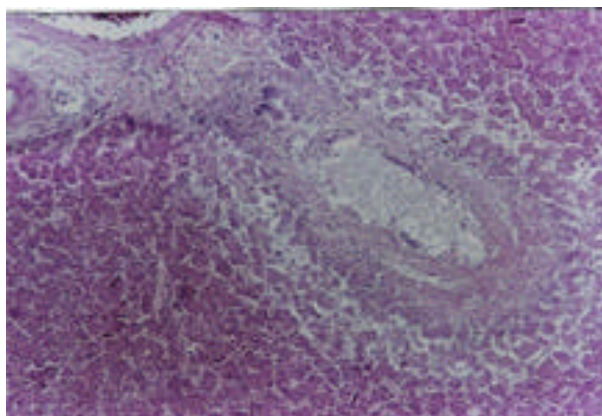


Fig. 3: Liver section (group B 3 weeks after treatment) showing a dilated bile duct with necrosis of surrounding hepatocytes HE x 400

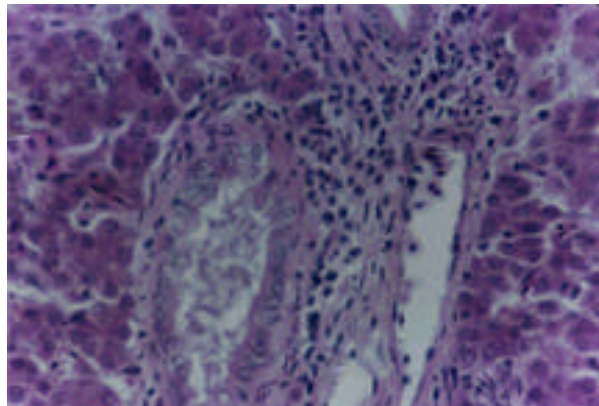


Fig. 5: Liver section (group C 3 weeks after treatment) showing a dilated bile duct with portal infiltration of mononuclear cells He x 400

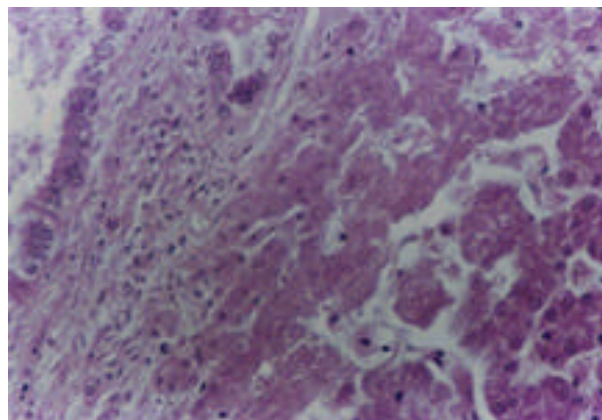


Fig.4: Liver section (group B 3 weeks after treatment). Necrotic changes in hepatocytes around a dilated bile duct HE x 400

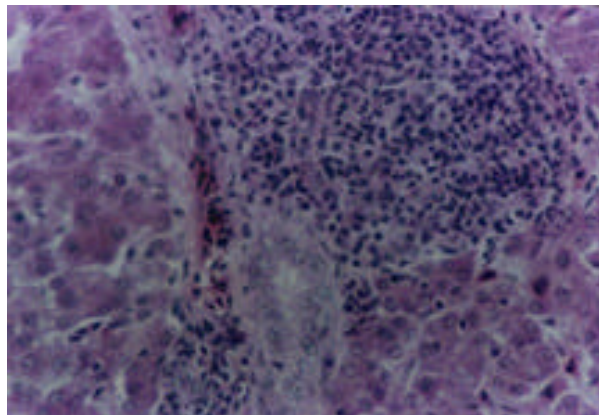


Fig. 6: Liver section (group B, 6 weeks after treatment) showing marked portal infiltration with mononuclear cells. HE x 400

form of focal coagulation necrosis associated in some cases with focal necrotic changes in the spleen and kidney (Deshpande *et al.*, 1998). On the other hand, toxic effects of turmeric or curcumin were not observed by some authors in rats, guinea pigs, monkeys and pigs (Wahlstrom and Blennow, 1978; Bhavanishankar *et al.*, 1980; Bille *et al.*, 1985). The toxic effect of turmeric seems to depend on animal species, dose and duration of treatment.

In the present study hyperaemia and mononuclear cell infiltration in parenchyma and portal areas were seen in liver sections of all groups (including control), especially in group B receiving 2.5% *Curcuma longa* supplement. On the other hand, dilation of bile ducts, hyperplasia of biliary epithelium and periportal hepatocyte degeneration were observed only in birds of group B, C and D fed *Curcuma longa* particularly those of group B receiving 2.5% supplement, killed after the third week of

the experiment. Since these hepatic changes were not seen in control group A (0.0% *Curcuma longa* supplement), they may suggest a role for *Curcuma longa* in bile production and hence fat digestion. Earlier studies in rats (Jentzsch *et al.*, 1959) and dogs (Ramaprasad and Sirsi, 1956) indicated that curcumin had both choleric and cholagogic actions. The periportal hepatocyte degeneration may possibly be due to leakage of bile from the dilated bile ducts. It is worth mentioning that haemorrhage and cholangiolar cell hyperplasia were previously observed in mice fed low doses of turmeric (0.1%) for two weeks but not in mice receiving higher dose (0.5%) this has been related to the anti-inflammatory and anti-proliferative effects of curcuma (Despande *et al.*, 1998).

The present study suggests that feeding *Curcuma longa* (turmeric) to chicken through diet can induce hepatic changes and that these changes are not dose or time

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dependent. No similar studies on birds seem to be available for comparison of results. However, compared to the studies on mice and rats, the hepatic changes in chicken seem to mainly involve the bile ducts rather than the parenchyma, with absence of frank necrotic changes. This may be due to the anti-inflammatory and antioxidant protective properties of *Curcuma longa* (Srimal *et al.*, 1971; Srimal and Dhawan, 1973; Sharma, 1976; Toda *et al.*, 1985; Masuda *et al.*, 1993; 2001; Noguchi *et al.*, 1994).

References

- Ammon, H.P.T. and M.A. Wahl, 1991. Pharmacology of *Curcuma longa*. *Planta Med.*, 57: 1-7.
- Bhavanishankar, T.N., N.V. Shanta, H.P. Ramesh, Indira, A.S. Murthy and V. Sreenivasamurthy, 1980. Toxicity studies on turmeric (*Curcuma longa*): Acute toxicity studies in rats, guinea pigs and monkeys. *Ind. J. Exp. Biol.*, 18: 73-75.
- Bille, N., J.C. Larsen, E.V. Hansen and G. Wurtzen, 1985. Subchronic oral toxicity of turmeric oleoresin in pigs. *Food Chem. Toxicol.*, 23: 967-973.
- Deshpande, S.S., V.S. Lalitha, A.D. Ingle, A.S. Raste, S.G. Garde and G.B. Maru, 1998. Subchronic oral toxicity of turmeric and ethanolic turmeric extract in female mice and rats. *Toxicol. Letters*, 95:183-193.
- Deshpande, S.S., A.D. Ingle and G.B. Maru, 1997. Inhibitory effects of curcumin free aqueous turmeric extract on benzo(a) pyrene-induced forestomach papillomas in mice. *Cancer Lett.*, 118: 79-85.
- Govindrajan, V.S., 1980. Turmeric chemistry, technology and quality. *CRC Crit. Rev. Food Sci.*, 12: 199-301.
- Huang, M.T., Y.R. Lou, W. Ma, H.L. Newmark, K.R. Reuhl and A.H. Conney, 1994. Inhibitory effects of dietary curcumin on forestomach, duodenal and colon carcinogenesis in mice. *Cancer Res.*, 54: 5841-5847.
- Jentzsch, K., T.H. Gonda and H. Holler, 1959. Paper chromatography and pharmacological action of the pigments of Curcumin. *Pharm. Acta Helv.*, 34: 181, (Germin); *Chem. Abstr.*, 53: 17324., 1959.
- Kandarkar, S.V., Sharda S Sawant, A.D. Ingle, S.S. Deshpande and G.B. Maru, 1998, Subchronic oral hepatotoxicity of turmeric in mice - Histopathological and ultrastructural studies., *Ind. J. Exp. Biol.*, Vol. 36, July, pp: 675-679.
- Masuda, T., A. Jitoe, J. Isobe and N. Nakatani, 1993. Antioxidative and anti-inflammatory curcumin related phenolics from rhizomes of *Curcuma domestica*. *Phytochemistry*, 32: 1557-1560.
- Masuda, T., T. Maekawa, K. Hidaka, H. Bando, Y. Takeda and H. Yamaguchi, 2001. Chemical studies on antioxidant mechanism of curcumin: Analysis of oxidative coupling products from curcumin and linoleate. *J. Agri. Food Chem.*, 49: 2539-2547.
- Nagabhushan, M. and S.V. Bhide, 1987. Antimutagenic and anticarcinogenic action of turmeric (*Curcuma longa*). *J. Nutr. Growth Cancer*, 4: 82-89.
- Noguchi, N., E. Komuro, E. Niki and R.L. Willson, 1994. Action of curcumin as an antioxidant against lipid peroxidation. *J. Jpn. Oil Chem. Soc.*, 43: 1045-1051.
- Pal, S., T. Choudhuri, S. Chattopadhyay, A. Bhattacharya, G.K. Datta, T. Das and G. Sa, 2001. Mechanisms of curcumin-induced apoptosis of Ehrlich's ascites carcinoma cells. *Biochem. Biophys. Res. Commun.*, Nov 2; 288: 658-65.
- Ramaprasad, C. and M. Sirsi, 1956. Studies in Indian medical plants; *Curcuma longa*, Linn-Effect of curcumin and essential oil of *C. longa* on bile secretion, *J. Sci. Ind. Res. Sect. C.*, 15: 212.
- Rao, C.V., A. Rivenson, B. Simi and B.S. Reddy, 1995. Chemoprevention of colon carcinogenesis by dietary curcumin, a naturally occurring plant phenolic compound. *Cancer Res.*, 55: 259-266.
- Sharma, O.P., 1976. Antioxidant activity of curcumin and related compounds. *Biochem. Pharmacol.*, 25: 1811-1812.
- Srimal, R.C. and B.N. Dhawan, 1973. Pharmacology of diferuloylmethane (Curcumin), a non-steroidal anti-inflammatory agent. *J. Pharm. Pharmacol.*, 25: 447-452.
- Srimal, R.C., N.M. Khanna and B.N. Dhawan, 1971. A preliminary report on anti-inflammatory activity of curcumin. *Ind. J. Pharm.*, 3: 10.
- Toda, S., T. Miyase, H. Arichi, H. Tanizawa and Y. Takino, 1985. Natural antioxidants III, antioxidative components isolated from rhizomes of *Curcuma longa*. *Chem. Pharm. Bull.*, 33: 1725-1725.
- Wahlstrom, B. and G. Blennow, 1978. A study on the fate of curcumin in the rat. *Acta Pharmcol. Toxicol.*, 43: 86-92.