PATHOLOGICAL STUDIES ON EXPERIMENTAL MERCURIC CHLORIDE TOXICITY IN BROILER CHICKEN

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ABSTRACT

The present study was undertaken to examine the toxic effects of mercury on gross and histopathological changes in visceral organs of broiler chickens. Twenty four birds of either sex were evenly divided into two groups of twelve birds each. Group I birds were kept on standard feed and water and served as control, whereas Group II birds were given mercuric chloride @ 50 ppm/bird/day mixed with standard feed. After 45 days, all the birds were sacrificed and visceral organs collected for gross and histopathological examination. Group II birds fed with mercuric chloride revealed marked enlargement and haemorrhage in liver with dilatation of sinusoids, focal areas of fatty degeneration and necrosis of hepatocytes. Kidney showed swollen glomerular tuft and detachment of tubular epithelium. Cardiac lesions included severe haemorrhage, hydropericardium and necrosis. Lungs showed reddish brown discoloration, infilteration of mononuclear cells and thickening of alveolar septa. Histopathological section of the brain revealed congestion, vacuolar degeneration, perivascular cuffing and gliosis. In view of toxic effects induced by mercury, it is essential to periodically monitor the level of this heavy metal in poultry feed and water.

Key words: Chicken, Histopathology, Mercuric Chloride, Toxicity

INTRODUCTION

Mercury is a naturally occurring element and is used in wide variety of products and processing industries, including paints, pharmaceuticals and production of different chemicals. It exists in environment as elemental mercury, inorganic mercury and organic mercury. Most forms are transformed into methyl mercury which is highly toxic (Gupta, 2012). Poultry industry is quite vulnerable to mercury toxicity due to ingestion of this heavy metal through contaminated feed, water, drug over-dose, spraying and cleaning of sheds involving variety of chemicals. Once in blood circulation, the main target organs of mercury are kidney, liver, neural tissues and other organs (Opinion of the Scientific Pannel on Contaminants, 2008). Inhalation of mercury vapour may result in pneumonitis and respiratory poisoining (Glezos *et. al.,* 2006) leading to gastrointestinal irritation and failure of renal and peripheral nervous system. There are not many studies on toxic effects of mercury on various vital organs of poultry. The present study was therefore, designed to examine the effect of mercuric chloride on gross and histopathological changes in visceral organs of broiler chicken.

MATERIALS AND METHODS

Day old, healthy, commercial broiler chicks of either sex were procured from

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Phoenix Poultry Farm, and were reared in battery brooders under standard hygienic and management conditions. These were offered pre starter, starter and finisher ration based on standard feed formulations. After acclimatization period of 7 days, twenty four birds were evenly divided into two groups of twelve birds each as described below. To overcome any mortality within the groups, replicate groups were reared concurrently under identical hygienic and management conditions. The experiment lasted for 45 days after which all the birds were sacrificed by exanguination.

Group I (n=6+6) Given standard feed and water *ad libitum*.

Group II (n=6+6) Given mercuric chloride, (technical grade, Qualigens) @ 50 ppm mixed with standard feed per bird per day throughout the experiment.

Detailed necropsy examination was conducted on the birds and gross pathological lesions were recorded. The representative tissue samples of liver, kidney, heart, lungs and brain were collected and preserved in 10% buffer formalin solution for fixation. After fixation for 3-4 days, the tissues were processed by paraffin embedding technique. Tissue sections were cut at 5 µm thickness and stained with routine Hematoxylin and Eosin stain for detailed histopathological studies (Gridley, 1960).

RESULTS AND DISCUSSION

The present study showed that ingestion of mercuric chloride caused serious histopathological changes and derrangements in kidney, liver, heart and in various other organs, whereas, the birds of control group maintained on standard feed and water revealed normal morphological appearance.

There was marked enlargement of the liver grossly with rounded borders and echymotic haemorrhages. Histopathologically, congestion, marked dilatation of sinusoids, kupffer cell prominence and haemorrhages were noticed. Focal areas of fatty changes and necrosis of hepatocytes (Fig. I), multifocal necrosis of hepatocytes and biliary hyperplasia were also noticed (Fig. II). Similar lesions have been described by earlier workers in experimental toxicity induced by mercuric chloride. Stoev and Lazarova (1998) reported granular or hyaline degeneration and fatty change, necrosis, prominence of kupffer cells, hyperaemia and perivascular edema in the liver of sheep treated with mercuric chloride.

Section of kidney revealed swollen glomerular tuft, intertubular haemorrhage and necrosis. Kidney revealed swelling and detachment of tubular epithelium from basement membrane and obliteration of tubular lumen. At few places, total loss of tubular epithelium was also evident (Fig. III). Bano and Hasan (1990) also observed disintegration of renal epithelium along with displacement of nuclei, shrinkage of glomeruli, breakdown of Bowman's capsule and heavy infiltration of inflammatory cells in kidneys of cat fish (*heteropneustes fossilis*) fed with mercuric chloride. Mercury is believed to cause hepato-renal toxicity by binding with covalant bonds like thiol and carboxylic groups, thereby blocking the function of several enzymes (Franco *et. al.*, 2009). In addition, mercury toxicity leads to excessive generation of reactive oxygen which seriously harms the body tissues (Farina *et. al*., 2011).

Cardiac lesions included morphological changes like misshapen heart with severe haemorrhages in the epicardium. Hydropericardium was noticed in fewer birds. Microscopic study revealed thickening and degeneration of blood vessels, necrosis of cardiac muscle fibers, degenerative changes in myocardium (Fig. IV) with infiltration of mononuclear cells. Mercury has been shown to produce adverse effects on cardiovascular system. A decrease in heart rate and other cardiac

disorders were observed in male rats given a dose of 2 mg mercury per Kg body weight as methyl mercuric chloride (Arito and Takahashi, 1991). There is scarcity of literature on the effect of mercury on cardiac tissue of chicken. However, Brake *et al*. (1976) reported myocardial changes characterized by myocarditis, infiltration of polymophonuclear cells and lymphocytes and fatty degeneration in juvenile chickens exposed to mercury in drinking water. These investigators also stated that mercury caused cardiovascular disturbances in chicken despite being administered at dosage that otherwise did not inhibit the normal growth.

Birds fed mercuric chloride revealed lungs with reddish brown discolouration and severe congestion. Section of lungs from these birds showed areas of congestion, haemorrhages and consolidation with infiltration of mononuclear cells (Fig. V). Thickening and rupture of alveolar septa and emphysema were also observed (Fig. VI). In addition, areas of haemorrhage, edema, degeneration and desquamation of epithelial cells in the alveolar lumen were noticed. Goyer (1991) reported corrosive bronchitis, interstitial pneumonia following exposure to high concentration of metallic mercury vapour. Exudative pneumonia in chronic mercury toxicity in goats was also reported by Pathak and Bhowmik (1998).

In the present study, the birds showed congestion in the brain grossly, while histopathological sections revealed congestion, vacuolar degeneration, perivascular cuffing and gliosis (Fig. VII). Neuronal degeneration and satellitosis (Fig. VIII) was also noticed in few birds. Marilyn *et al.,* (2000) also studied the effect of methyl mercury on egret birds and reported very severe damage to neural tissue including axon swelling, missing of axon tissue, loss of myelin and degeneration of sciatic nerve. Pathak and Bhowmik (1998) observed congestion, edema and vacuolation in the brain in chronic toxicity of inorganic mercury in goats. It is known that methyl mercury crosses the blood brain barrier as against inorganic mercury. The comparative damage to the neural tissue could be due to different kinetics of the mercury compounds.

In view of the severe toxic effects of mercury to vital organs and the tendency of the metal to accumulate in the kidney and other tissues (Ana *et al.,* 2005; Hussain *et. al.,* 2013), it is essential to periodically monitor the mercury levels in water and feed of poultry, especially the farms which are located in the vicinity of industrial areas.

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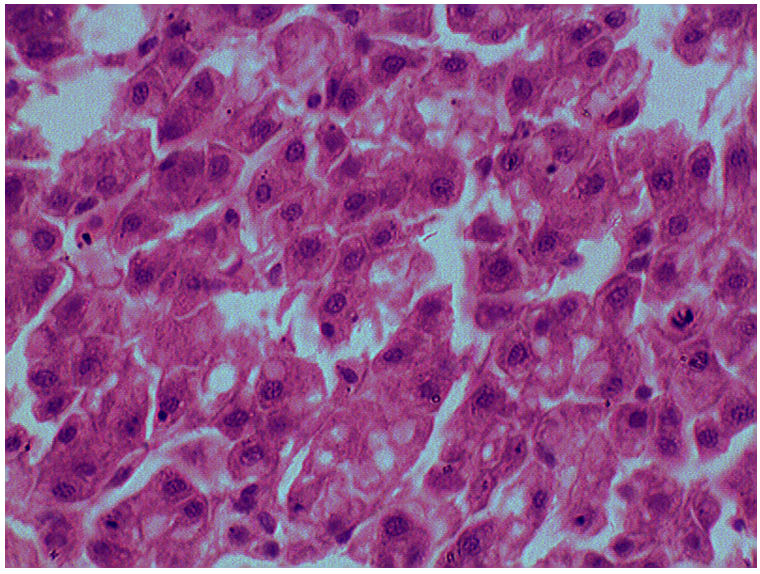

Fig. I: Focal areas of fatty degeneration and necrosis of hepatocytes in liver. (H&E X400)

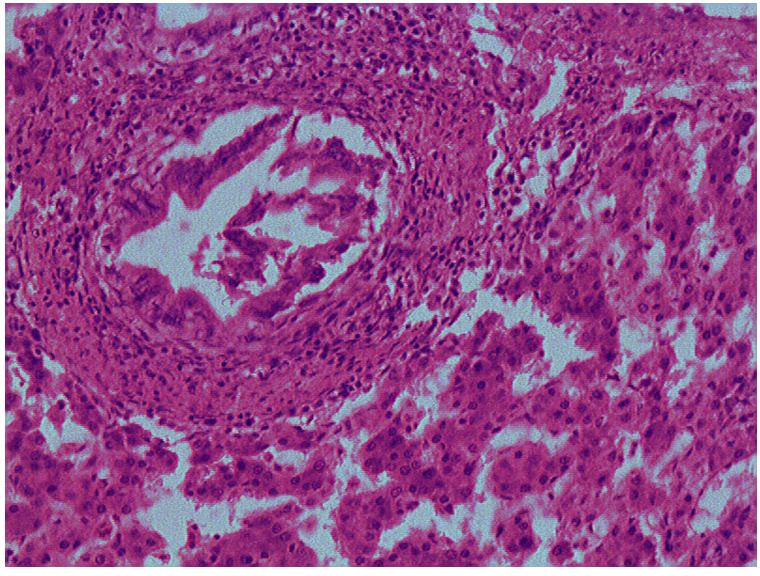

Fig. II: Section of liver showing biliary hyperplasia and multifocal necrosis. (H&E X400)

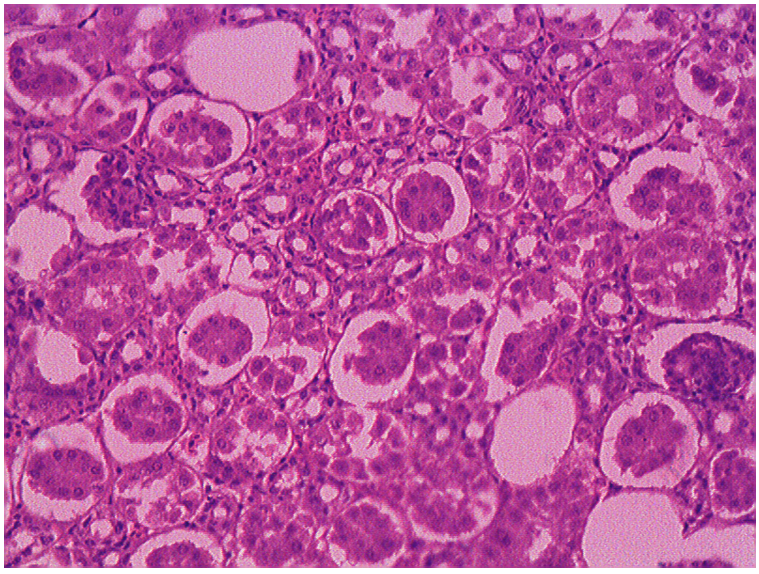

Fig. III. Swelling and detachment of tubular epithelium and denudation in kidney. (H&E X400)

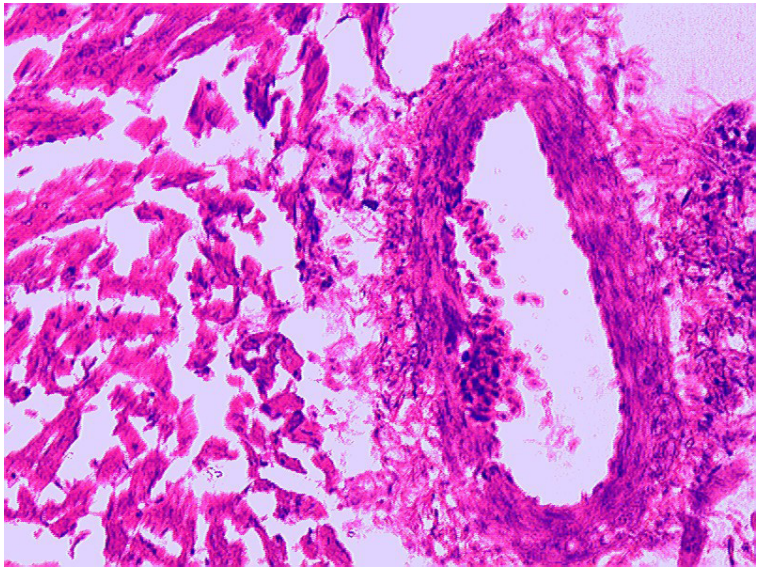

Fig. IV. Thickening of blood vessel and degeneration of myocytes heart. (H&E X400)

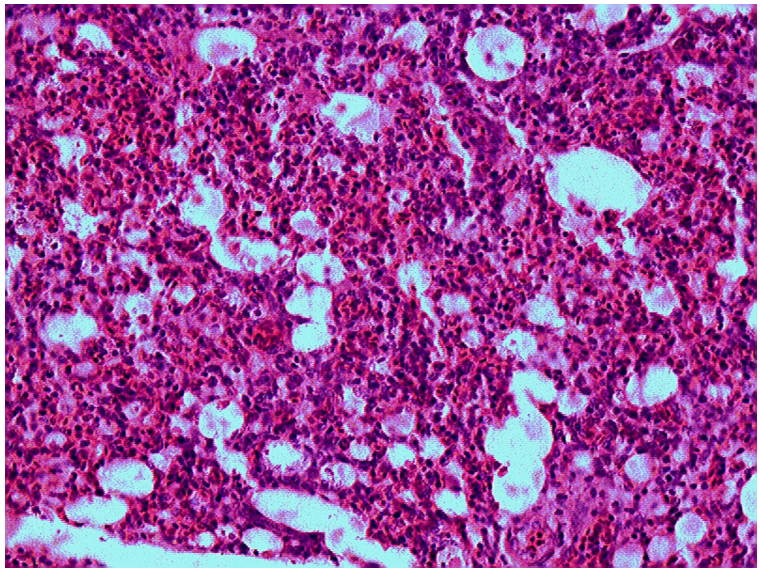

Fig. V. Lung showing congestion, haemorrhage and mononuclear cell infiltration. (H&E X400)

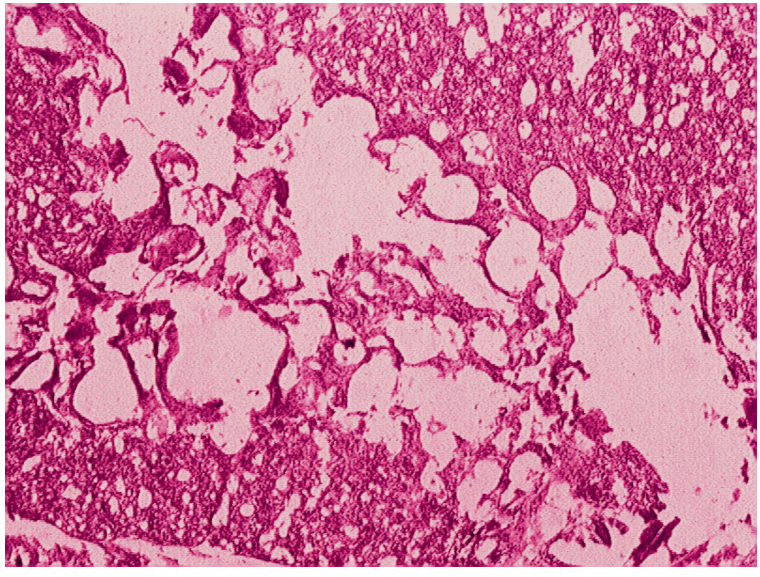

Fig. VI. Section of lung showing broken alveolar septa and emphysema. (H&E X400)

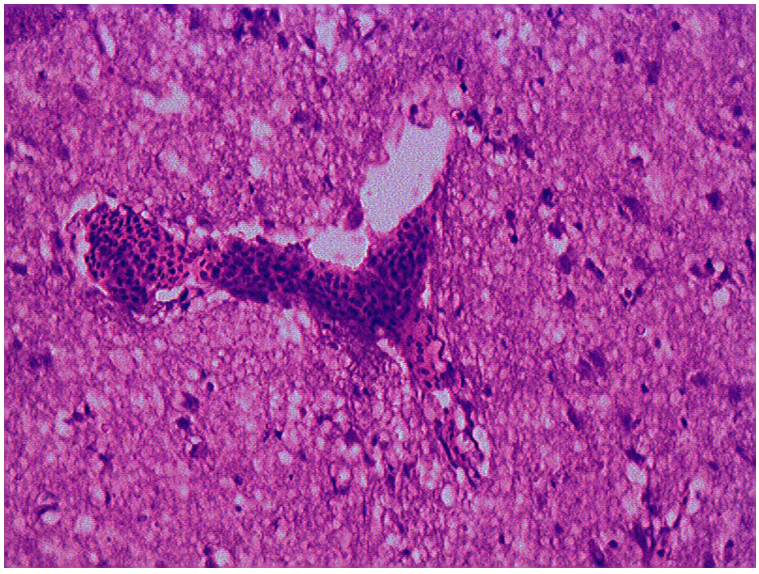

Fig. VII. Section of brain showing congestion, vacuolar degeneration and perivascular cuffing. (H&E X400)

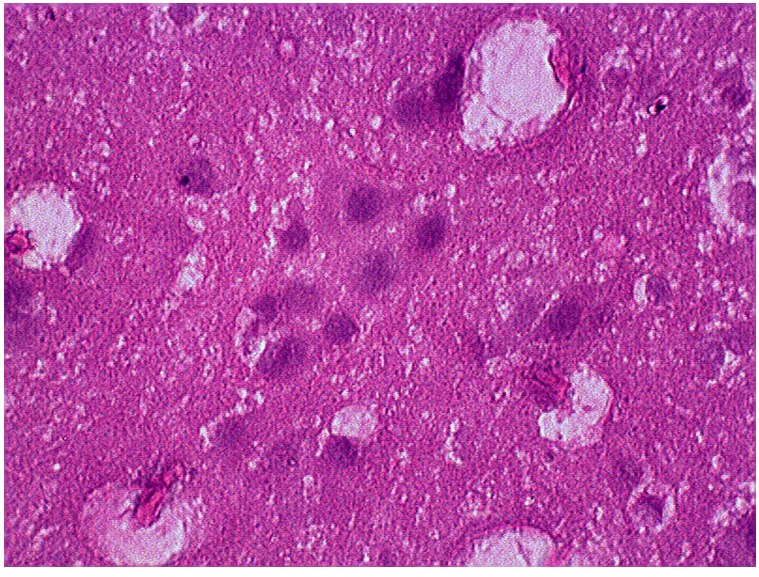

Fig. VIII. Section of brain showing neuronal degeneration and satellitosis. (H&E X400)