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Manganese Induced Histopathological Changes in Digestive Tract, Liver and Kidney of A Fresh Water Snakehead Fish *Channa Punctatus*

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Abstract

Fresh water fish *Channa (Ophiocephalus) punctatus* was exposed to manganese chloride ($MnCl_2$) in water at a concentration of 120 mg/l for a period of 1 and 2 months. Manganese produced a number of histopathological changes in stomach, intestine, liver and kidney. In stomach gastric glands were affected. Some vacuoles were noted in the submucosa of stomach of treated fishes while they were not present in the control fishes. Intestine and pyloric caeca also showed almost similar histopathological changes. At 30 days treatment, some of the goblet cells which are situated at the tips of villi got ruptured, nuclei were enlarged and got accumulated in the submucosa. The cytoplasm of mucosal epithelial layer was degenerated at various points in 60 days treated fishes. Some of the villi were also distended at this stage. In liver cytoplasm of the hepatocytes was vacuolated and by 60 days necrosis has also been reported. In kidney necrosis and enlargement of bowman's capsule were reported. The brush border of the uriniferous tubules started degenerating and nephrocytes seems to be loosely arranged. With lapse of time the damage was more as most of the cells were degenerated while the nuclei got accumulated in the hematopoietic tissue. The remaining nephrocytes were vacuolated.

Keywords: *Ophiocephalus Punctatus,* Manganese, Histopathology.

Introduction

Since fishes are the main source of human diet, the presence of elements in the water bodies and its accumulation in the body of fish is a matter of great concern (Erdogrul and Erbilir,2007). Pollutants, heavy metals pose a serious water pollution problem due to their toxic properties and adverse effect on aquatic life (Malik, 2010). Heavy metals are known to produce toxic effects on the different tissues of various terrestrial and aquatic animals. Some of these are highly toxic at even very low concentrations and alter the cellular architecture. Poisonous effects of manganese among industrial workers are well recognized (Tolonen, 1972). Manganese interferes with the physiological functions as a few reports have shown the presence of hypoglycemia in manganese exposed workers (Rubenstein et al.1962; Hassanein et al.1968). Manganese is known to produce neurological disorders when inhaled by miners and the lungs has long been thought as the main route of the entry of this metal (Rodier, 1955; Tanaka and Lieben,1969).Increase number and amount of industrial, agricultural and commercial discharge into the aquatic resources have led to various deleterious effects on the aqua fauna (Van der Oost et al.., 2003). The small doses of manganese chloride have been reported to increase oxygen uptake in mammals, whereas large doses had the reverse effect (Deysach and Ray, 1949; Michel et al., 1969). However numerous studies have been conducted on pathogenesis of manganese toxicity in mammals (Pentschew et al., 1963; Chandra, 1972) but a little work has been done on this aspect in the teleost fishes. In the present report histopathological lesions have been worked out in manganese intoxicated teleost fish, *Channa punctatus.*

Materials and Methods

The fish, *Channa punctatus* (50-70 gm in weight and 14-16cm in length) were collected from the water resources and were acclimatized in the laboratory for a week. Then fishes were randomized in three groups; each group contains two fishes. Out of three groups one was of control fishes and two were of treated fishes. The treatment was given in the

surrounding water of fishes in aquarium for 30 and 60 days with a sub lethal dose (120 mg/liter) of manganese chloride. On completion of treatment, the fishes were dissected along with the controls to fix kidney, liver,stomach,pyloric caeca and intestine in neutral formalin. Then paraffin sections of 6 µm were cut and stained with haematoxylin-eosin to study the histological alterations produced by manganese chloride in the treated fishes.

Results and Discussion

Stomach

At 30 days treatment no significant histopathological changes were observed in the histological architecture of stomach while at 60 days stage some of the cells of gastric glands were damaged and vacuoles appeared in some mucosal cells. At a few places corrosion in gastric lining was observed.

Intestine and Pyloric Caeca

In manganese exposed fish for 30 days, many histopathological changes were observed in intestine and pyloric caeca . In both these tissues, a few of the goblet cells especially at the tips of villi got ruptured due to which clumping of nuclei was observed in the submucosa. Mucosal lining was ruptured at few places and vacuolation also started at 60 days stage. Even the corrosion in the mucosal epithelial layer was noted in chronic stages while muscularis and serosa depicted on histopathological changes.

Liver

Liver is the most affected organ by contaminants in water which has main role in the process of detoxification and biotransformation (Camargo and Martinez, 2007). In prolonged treatment with manganese the pathological lesions were more acute without any hepatocytic regeneration. After 30 days exposure cellular disorganization and nuclear hypertrophy were observed and the hepatocytes were vacuolated. Many hepatocytes were seen undergoing hydropic degeneration and only a few nuclei were pycnotic. Such changes were more conspicuous in the centrolobular area in comparison to the perilobular one. While at 60 days treatment stage most of the hepatocytes of centrolobular and perilobular areas lost their cell organization as their cell membranes were ruptured and their cellular contents were infiltered, leading to submassive necrosis. The scars of the infiltered cells along with the clumped degenerating nuclei were visible. Dilation of the sinusoids was also observed. Nuclear hypertrophy and dilation of the sinusoids were also observed by the Butchiram et al. (2013) in the liver of fish under the stress of phenol which supports our findings. These damages in the liver probably diminish its detoxification process (Kolanczyk and Schmieder, 2002; Solen et al., 2003).

Figures (1-11): Showing the Effect of Manganese in Different Tissues of Channa Punctatus

Control Two Month Treatment

Fig.1, Stomach-100x

Fig.2, Stomach-100x

Fig.3, Intestine-400x

Fig.4, Intestine-400x

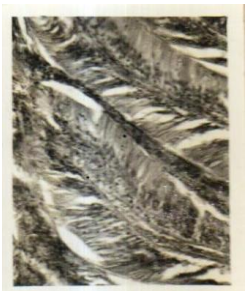

Fig.6, Pyloric Caeca-400x

Fig.7, Pyloric Caeca-400x

Fig.8, Liver-400x

Fig.9, Liver-600x

Fig.10, Kidney-600x

Fig.11, Kidney-400x

Fig.5, Intestine-400x
(One month treatment)

Kidney

At 30 days treatment stage necrosis started and the space in malpighian capsular body was widened due to the shrinkage of glomeruli. At several places coalescing and later on dissolution of basement membranes of the two adjacent renal tubules was observed. The connective tissues got disorganized and due to the degeneration of brush border of uriniferous tubules, the nephrocytes seems to be loosely arranged. While at 60 days stage most of the cells were necrosed leaving no scares even. The nuclei got accumulated in hematopoietic tissue and nephrocytes if remained unruptured were vacuolated. Thus almost complete damage of most of the renal tubules took place.

The present observations reveal the high toxicity of manganese to *Channa punctatus.* Metal ions reach the alimentary tract along with the food due to which stomach and intestine both were affected by manganese. Mena et al. (1969) have shown that the inhaled manganese find its way in the gastrointestinal tract from where it is either absorbed or eliminated. Chandra and Imam (1973) reported decrease in the number of pepsinogen granules in the stomach of guinea pigs treated with manganese. We have reported that the corrosion of the mucosa and gastric glands, one of the important toxic stress of manganese which support the finding of Vonoettingen (1935). In kidney proximal convoluted tubules showed vacuolation, loss of brush border and degeneration of epithelium. Carone and Spector (1960) reported necrosis in proximal convoluted tubules under the toxic stress of bismuth which also support our findings. Vacuolation in the renal tubular epithelial cells has been reported by Bencosme et al. (1960). According to the Lindquist and fellers (1966) it is also possible that heavy metals are found at membrane site, as a consequence the reabsorption mechanism may be blocked due to which pathological changes develop in the tissue. Manganese chloride produced significant changes in histological picture of liver which become more severe with increase in the duration of treatment. It produces degenerative changes including enlargement of liver cells and vacuolation in the cytoplasm while in further exposure hepatocytes lost their architecture. The nuclei showed increase in size and hypertrophy in the beginning but in later stage clumping of degenerating nuclei was observed. Schaffner (1957), Sastry and Sharma (1977) and Goel et al. (1979) reported similar histopathological changes in liver.

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