Bioscience Discovery, 8(3): 416-421, July - 2017

Print & Online, Open Access, Research Journal Available on http://jbsd.in
ISSN: 2229-3469 (Print); ISSN: 2231-024X (Online)
Research Article

Efficacy of herbal drug against the histochemical and histological damage due to Mercury in the Kidney of freshwater catfish *Heteropneustes fossilis* (Bloch)

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Article Info

Received: 14-04-2017,
Revised: 31-05-2017,
Accepted: 06-06-2017

Keywords:
H. fossilis, Kidney, Liv_{52}, Mercury, Recovery

Abstract

The protective and curative effect of herbal compound (Liv_{52}), against mercuric chloride (0.1mg/l) induced histochemical and histopathological changes in the Kidney of a freshwater catfish *Heteropneustes fossilis* (Bloch) were studied. Histochemical observations indicated heavy metal accumulation in the Kidney of the Hg exposed fish. The sites of metal accumulation correlated the structural damage of the Kidney. The herbal compound Liv_{52} did not show any protective and curative effects against Hg stress in kidney of freshwater catfish. It had failed to remove the Hg burden from Kidney of freshwater catfish *H. fossilis*.

INTRODUCTION

Toxic contaminants released through industrial, domestic, agricultural wastes and effluents polluting the water resources and badly affecting the aquatic fauna. Heavy metals are considered to be the most important form of pollution to the water bodies due to their toxic nature. The accumulation of the heavy metal in aquatic organisms disturbs their normal life (Pandey *et al.*, 2012). The quantum of these heavy metals in water bodies has increased due to increased demands and rapid developments. Mercury is one of the most dangerous heavy metal among these. Major sources of mercury discharge are the effluents of thermal power stations, paper and pulp industries, electronic wastes, paint industries, batteries, electroplating, metal industries, fossil fuel combustion etc. (Driscoll *et al.*, 2007; Carrie *et al.*, 2010; Yasser and Naser, 2011). Mercury chloride ($HgCl_2$), used in chemical processes, is a white powder, soluble in water, posses carcinogenic characteristics. Mercury enters in the fish body through various routes and accumulates in different organs of the fish body. Hg accumulates due to efficient uptake and comparatively its slower release rate (Dobaradan, 2010).

In fishes, Hg toxicity induces morphological lesions (Chavan, 2015), Physiological disorders (Grosell *et al.*, 2007) and behavioral abnormalities (Ghanbahadur, *et al.*, 2015; Deshmukh, 2016; Bhalerao, 2017).

In past few reports have appeared on histochemical distribution of Hg in fish organs (Bhoraskar and Kothari, 1993; Regine, 2006). Other studies of few species of fishes revealed histopathological changes (Gupta and Kumar, 2006) in vital organs exposed to Hg salts.

Some studies were also conducted to observe the recovery patterns of fishes due to Hg toxicity. An indigenous herbal compound Liv_{52} was tested against Hg toxicity in fishes (Kothari et. al., 1990; Samanta, 2014) and mammalian organs (Rathore and Verma, 1988).

This study was undertaken to understand the protective action of herbal compound Liv_{52} against Hg accumulation and induced tissue damage in kidney of fresh water teleost *H. fossilis*. The role of drug, if any, during the recovery process in the Hg exposed fish kidney was also observed. The same drug was also studied for behavioral recovery in *H. fossilis* (Bhalerao, 2017). Haematological recovery pattern due to endosulfan in *C. striatus* was also reported (Deshmukh, 2016).

MATERIALS AND METHODS

H. fossilis procured from fish farm were acclimatized to laboratory conditions in glass aquaria for seven days. Stock solution of $HgCl_2$ was prepared as per standard guidelines (APHA, 1975). Liv_{52} is an indigenous herbal compound and is known as hepatoprotective drug. Composition of the drug was mentioned in the earlier study (Rathore and Verma, 1988). The experimental concentration of $HgCl_2$ was 0.1 mg/l. Acclimatized fish were divided into groups of 25 each (Table 1.0).

The food and drug were fed at the rate of 30 and 10 mg/day/fish, with few drops of liquid paraffin, to the fishes of all aquaria respectively. On every fourth day water of all aquaria was changed and fresh metal solution was added to experimental groups.

Fishes from groups I, II and III were sacrificed on 30^{th} days while those of recovery experiment were sacrificed on 60^{th} day and accumulation of Hg in fish Kidney was demonstrated by using Sulphide Silver Method for heavy metals (Pearse, 1972). Mercury was localized in tissue section as brownish black deposits of mercury sulphide.

The kidney sections of 5 µ thickness were processed in routine procedure and double stained with Haematoxylin and Eosin for Histopathological studies.

RESULTS AND DISCUSSION

No traces of Hg were observed in group I, i.e., Control group. Histochemical studies (Fig. 1.0) showed that the Hg was accumulated throughout the component tissues of Kidney. The group II fishes exhibited that the Kidney tubules (KT) and haemapoietic tissue were highly loaded with Hg. Histological architecture of Kidney tubules was not visible due to accumulation of mercury (Group II). In group III, the Hg distribution was almost similar to group II. However, overall metal content was lesser than group II. The distal tubules were loaded with Hg, whereas the metal accumulation in proximal tubules was comparatively lesser. During natural recovery in group IV A, reduction in metal content was noticed in Kidney tubules, as against group II and group III. The drug recovery group IV B, showed results almost similar to natural recovery group IVA. This suggested the ineffectiveness of the herbal compound in Hg removal from Kidney.

Histological studies of Kidney (Fig. 2.0), revealed almost similar results as of the histochemical studies. The control group (Group I) fishes showed normal structure. The kidney tubules (KT) were distinct with basally located nuclei and a distinct lumen. Kidney of group II fishes indicated localized histological damage. Vacuolisation due to cytoplasmic degeneration, loss of cellular boundaries and loss or reduction of tubular lumen (RTL), were among the major pathological symptoms in the Kidney. In some cases kidney tubules (KT), were without nuclei indicating cell death. No changes in Hg induced structural damages were observed in group III, i.e., in Liv_{52} fed fish kidney. Pathological lesions in this group were comparable with group II fishes. Decontamination studies in natural recovery fishes (Group IV A), revealed that kidney tubules (KT) were regaining their normal structure after 30 days of removal of Hg stress. There was not any noticeable change in the drug recovery group IV B.

Kidney is an important organ for excretion. It was observed that the Hg accumulates in kidney in two distinctive ways. The first mode of mercury accumulation in the tubular structure of kidney is because of the presence of Hg in the contaminated water and secondly, due to the consumption of Hg contaminated food (Thangam *et al.*, 2014). Filtration of Hg takes place through the glomerulas before excretion. The Hg salts were reported to be slowly transformed in to inorganic salts within the fish body (Mela *et al.*, 2012), resulted in secondary accumulation of Hg by the kidney. Apical presence of Hg in distal tubules pointed to tubular reabsorption of Hg. Tubular excretion had been suggested as a mechanism of handling Hg by the kidney (Gochefeld, 2003; Hajrudin *et al.*, 2010). The fish kidney damages during present investigations were in accordance with the studies conducted in past (Mohan, 2013; Jalaludeen, 2012). The kidney disorders and the pathological damages, due to mercury poisoning varied with the chemical form of Hg and its exposure duration (Pandey *et al.* 2012;

Mela, 2012). Soluble inorganic salts were reported to cause striking pathological changes in the kidney (Thangam, 2014) and other organs with different pollutants as well (Ghanbahadur *et al.*, 2015; Ganeshwade *et al.*, 2016).

Mercury deposition observed in cat fish kidney during simultaneous treatment of the herbal compound was almost similar to the group II. In group III fishes, the histological damage in kidney tubules was comparable with group II, suggesting ineffectiveness of drug against mercury caused lesions in fish kidney. However, it was found effective against Cd induced structural damage in mice kidney (Rathore HS, 1987). The structural recovery in kidney during both the recovery groups was almost similar and no advantage of drug over natural recovery was seen. The natural recovery group (IV A) fishes revealed reduction in Hg burden in cat fish kidney after cessation of mercury exposure and maintenance of fish in Hg free water. This noticeable reduction in Hg content may be either due to excretion or redistribution of toxic mercury. Since no increase in Hg concentration during decontamination phase was noticed in the kidney, it can be concluded that there was no recycling/redistribution of Hg and the observed decrease was presumably due to excretion or elimination of Hg. Slow and efficient reduction in Hg burden was observed in the fish kidney during decontamination phase (Boudou *et al.*, 1991; Palas, 2014).

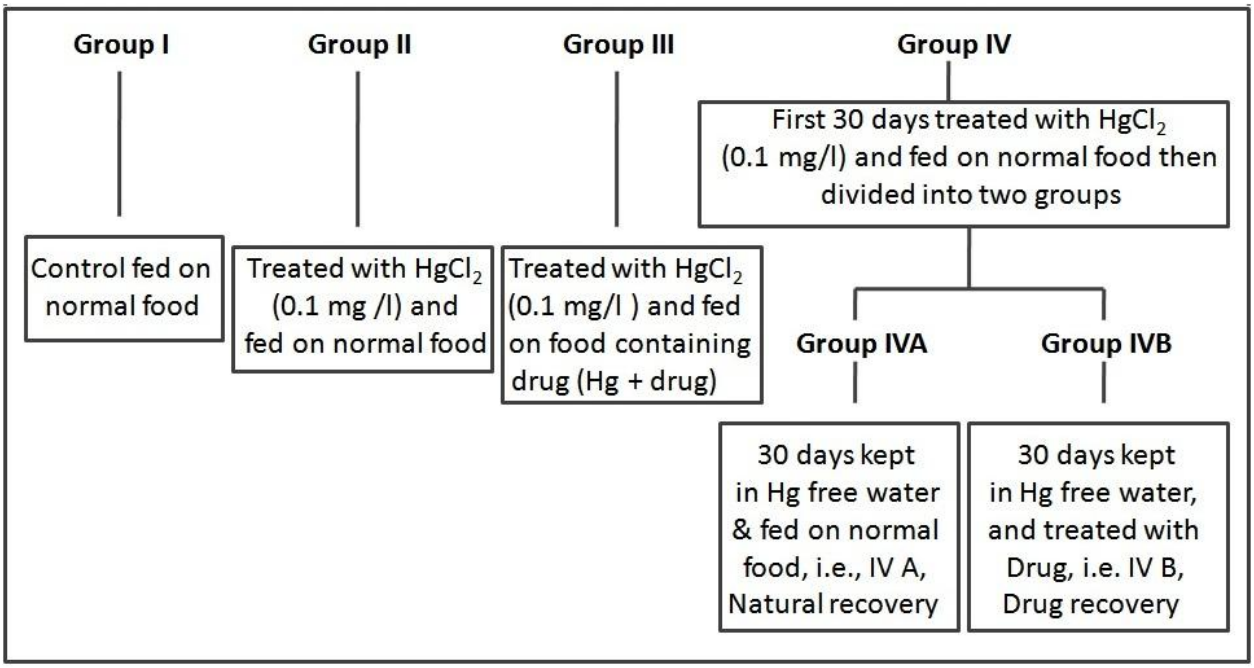

Table 1.0 Experimental Plan

The findings of the study supported by the earlier studies that the kidney accumulates highest concentration of Hg (Ashraj, 2005). The renal cortex accumulates up to 90% of the total body burden of Hg (Mela, 2007). In kidney mercury, bound preliminary to proteins including low molecular weight metal binding protein and kidney lisosomes (Baatrup and Danscher, 1987; Bebianno, 2007).

No remarkable difference in Hg elimination during drug recovery (group IV B) was observed. The present study revealed no remarkable reduction in Hg deposition in kidney of catfish during simultaneous treatment and drug recovery phase.

In the present investigation a visible correlation was observed between metal deposition and tissue damage which indicated the similar sites of active metal deposition and structural damages. No noticeable changes in Hg accumulation were observed in the presence of drug Liv_{52} and during drug recovery phase. The study revealed that in the cat fish kidney the hepatoprotective drug $Liv_{52,}$ was ineffective during simultaneous as well as in post therapy treatment. However, further detail studies are needed to understand the reasons for ineffectiveness of Liv_{52} against toxic action of Hg and also during recovery phase in fish kidney.

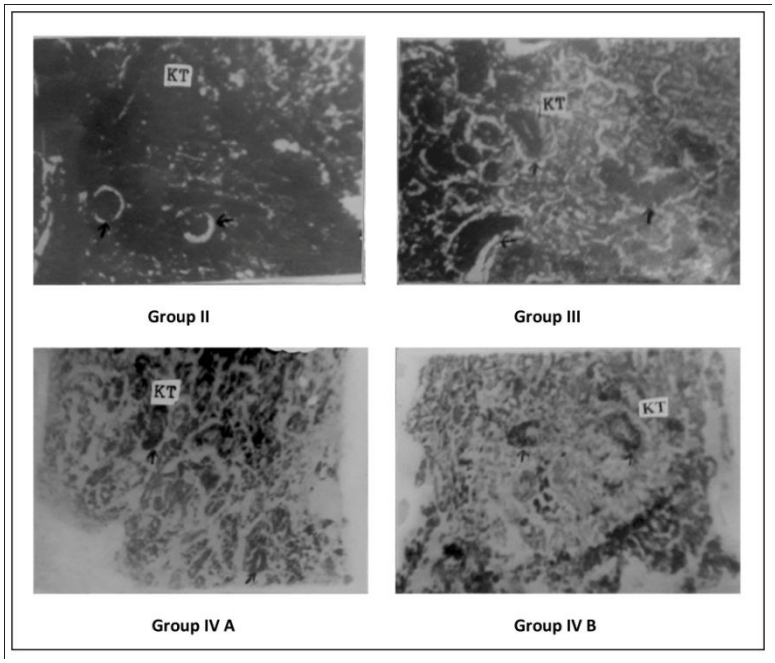

Fig 1.0 Metal accumulation in the kidney of various experimental groups of *H. fossilis* (Bl.)

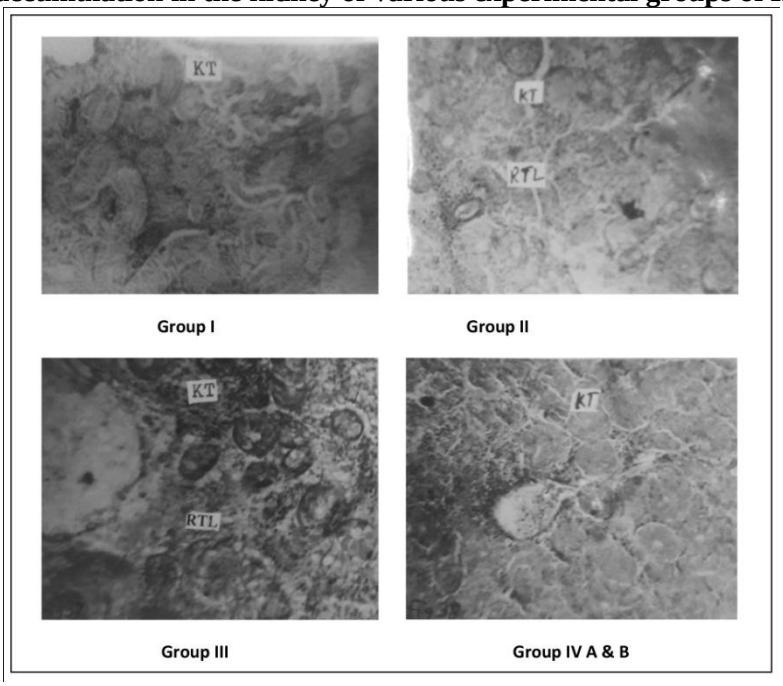

Fig.2.0 Histopathology of kidney of various experimental groups of *H. fossilis* (Bl.)

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How to Cite this Article:

Bhalerao S N, 2017. Efficacy of herbal drug against the histochemical and histological damage due to Mercury in the Kidney of freshwater catfish *Heteropneustes fossilis* (Bloch). *Bioscience Discovery*, **8**(3):416-421.