

HISTOPATHOLOGICAL CHANGES IN THE KIDNEY OF FRESHWATER TELEOSTS, *CIRRHINUS MRIGALA* EXPOSED TO SODIUM FLUORIDE

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ABSTRACT

In the present study *Cirrhinus mrigala* were exposed for the period of 90 days at 5 mg/L, 10 mg/L and 15 mg/L NaF. Histopathological changes observed in kidney showed renal architecture damage in the form of increased capsular space, shrunken glomeruli, shrunken lumen of renal tubules and vacuolated cytoplasm. None of these changes was observed in the control group.

Key words: Fluoride, Fingerlings of *Cirrhinus mrigala*, Kidney histopathology.

INTRODUCTION

The release of fluorides into the environment due to human activities such as mining and processing of phosphate rock and its use as agricultural fertilizer, as well as the manufacture of aluminium. Other fluoride sources include the combustion of coal (Containing fluoride impurities) and other manufacturing processes (steel, copper, nickel, glass, brick, ceramic, glues and adhesives). In addition, the use of fluoride-containing pesticides in agriculture and in drinking water supply also contribute to the release of fluorides in the environment to impart stability to bone and enamel, thereby preventing dental caries and osteoporosis to some extent but its higher concentration is highly to human beings and animals too. The permissible limits of fluorides in drinking water as suggested by Bureau of Indian Standards (BIS, 1983) vary between 0.6 to 1.2 ppm while (WHO, 1984) permits a maximum of 1.5 ppm of it. In addition to its toxic effects on bones and teeth, it also causes serious effects on liver, kidney, and reproductive organs and also has teratogenic effect (Verma, 2004). The kidney is the chief excretory organ of the fish and plays an important and significant role in elimination of any toxic substances in the body. Thus form an important organ for pathological studies.

MATERIALS AND METHODS

The fingerlings (8 cm to 10 cm in length; 10 to 12 mg in weight) were obtained from local sources, acclimatized to the laboratory conditions and used for the experimental purpose. They were exposed to different concentration (5 mg/L, 10 mg/L and 15 mg/L) of sodium fluoride in water for a period of 90 days along with suitable control groups kept in chlorine free water. Fishes were sacrificed after 30, 60 and 90 days of exposure

and kidney were dissected out and processed for paraffin embedding. The paraffin wax section were cut at 6 μ thickness and stained by the standard haematoxylin eosin double staining method. The microphotographs of the slides were obtained using Olympus BX 51 microscope with digital photographic attachment.

RESULTS

The results of the experiment are presented in figure 1-4 of plate records the photomicrographs of transverse section of kidney of control groups fig. 1 shows the transverse section of the kidney of fingerling kept under control condition. In this photomicrograph the histological details of the kidney of *Labeo rohita* are observed. The Bowman's capsule (BC), glomerulus (G), the first (PCT-I) and second (PCT-II) segments of the proximal convoluted tubules can be clearly deciphered. The distal convoluted tubules (DCT) are also discernible in the photomicrograph.

Fig. 2-4 reveal the effect of fluoride toxicity on kidney of treated fingerlings prepared after 30, 60 and 90 days of exposure to 5 ppm, 10 ppm and 15 ppm sodium fluoride respectively. In this case the kidney tissue shows a thick lining of Bowman's capsule, shrunken glomerulus (SG), increased capsular space (ICS) with swelling and sloughing off of the epithelium of capsule cells. The renal tubules shows shrunken lumen (SL), Vacuolated cytoplasm (VC) and finally the normal histology of the lumen is considerably disturbed with the tubular lumen is completely shrunken.

In case of the renal tubules, it is observed that the lumen of the tubules have shrunk (SL) considerably. Large vacuoles (VC) appear in the cells lining the lumen and the nuclei of these cells have been pushed towards the center of the lumen. As is evident from the plate the

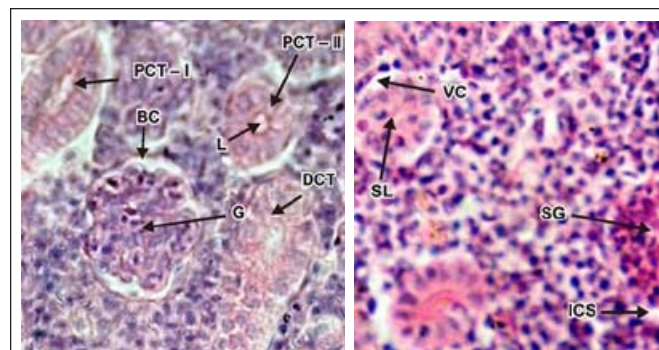


Fig.1: Control after 30 days

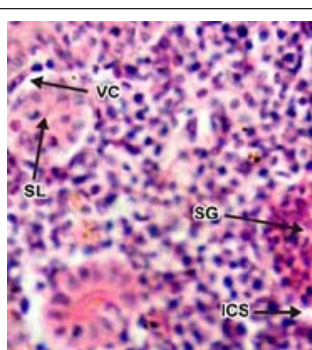


Fig. 2: 5 mg/L NaF after 30 days

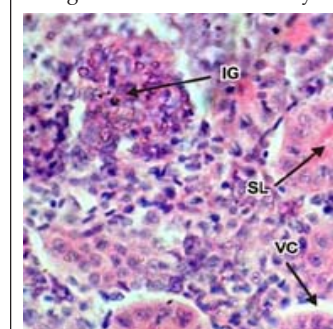


Fig. 3: 10 mg/L NaF after 60 days

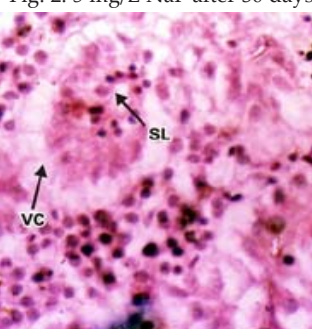


Fig. 4: 15 mg/L NaF after 90 days

Plate: T.S. kidney of *Cirrhinus mrigala* (40×) showing effect of sodium fluoride

lumen appears to be normal even after 30 days of exposure. But with the progressive time period the luminal structure is disrupted. In fig. 3 the nuclei of the cells begin to leave their normal basal position and tend to move towards the lumen (fig. 4) and finally the normal histology of the lumen is considerably disturbed with the tubular lumen is completely shrunken.

DISCUSSIONS

The present study indicates that fluoride can induce histopathological alterations in the kidney of the teleost exposed to various concentration of it. The results of the study also indicate that fluoride toxicity causes many adverse but sublethal effects in the fish. Jankauskas (1974) reported that fluoride is removed from the kidney by glomerular filtration and approximately 1/3rd of ingested fluoride appears in the urine within 24 hours. He also observed that massive doses of fluoride induced tubular necrosis, especially in the convoluted portion, and inflammation of glomeruli. He reports that these changes are associated with clinical finding of impaired kidney functions like polyuria, polydipsia, increased non-protein nitrogen etc. Kessabi *et al.*, (1985) also

observed degeneration and tubular necrosis associated with glomerular inflammation in experimental acute sodium fluoride poisoning in sheep kidney. Marked necrosis of tubular cells, atrophy of the glomeruli and areas of interstitial inflammation of round cells were observed by Kaur and Singh (1980) in mice kidney after sodium fluoride administration. These results are also confirmed in the present study where inflammation in glomerulus, increase in capsular space and shrunken lumen of tubules are observed. Studies have also indicated that sodium fluoride increases intracellular accumulation of calcium (Murao *et al.*, 2000) and inhibits tubular reabsorption-perhaps due to inhibition of the active chloride pump located in the medullary portion of the ascending limb of the Henle loop (Roman *et al.*, 1977). In the present study the severely shrunken lumen of tubules are also suggestive of hindered tubular reabsorption. In addition, clinical indicate that F toxicity may lead to osteosclerosis and end-stage renal failure (Lantz *et al.*, 1987).

In this study, therefore, 15 mg/L sodium fluoride in water caused severe pathological changes in kidney of *Cirrhinus mrigala*, thereby disturbing normal physiology.

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REFERENCE

- BIS (1983): Specifications for drinking water, ISI:1983, Bureau of Standards, New Delhi.
- Jankauskas, J. (1974): Effects of fluoride on the kidney: a review, *Fluoride*, **7**(2): 93-105.
- Kessabi, M.; Hamli, A.; Braun, J. P.; Rico, A. G. (1985): Experimental acute sodium fluoride poisoning in sheep: Renal, hepatic, and metabolic effects, *Fund. Appl. Toxicol.*, **5**(6): 1025-1033.
- Lantz, O.; Jouvin, M. H.; Vernejoul De, M. C.; Druet, P. (1987): Fluoride induced chronic renal failure, *Am. J. Kidney Dis*, **10**(2): 136-139.
- Murao, H.; Sakagami, N.; Iguchi, T.; Murakami, T.; Suketa, Y. (2000): Sodium fluoride increases intracellular calcium in rat renal epithelial cell line NRK-52E, *Biol. Pharm. Bull.*, **35**(5): 581-584.
- Roma, R. J.; Carter, J. R.; North, W. C.; Kauker, M. L. (1977): Renal tubular site of action of fluoride in Fischer 344 rats. *Anesthesiology*, **46**(4): 260-264.
- Verma, R. J. (2004): Impact of fluoride in mammals, *Proc. Nat. Acad. of Sci., India*. **74B**(II): 99-113.
- WHO (1984): Guidelines for drinking water quality. Vol. 1 Recommendations, World Health Organization, Geneva.

