Effect of cadmium and lead exposure and recovery on kidney of fishes juveniles *Carassius carassius*(L.)

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Abstract:

In this study two groups of fishes juveniles *Carassius carasssius* were used.All groups were exposed to two different concentrations of Cd (0.2 &0.5) ppm and Pb (15 & 30) ppm for (LT_{50}) period 8days.first groups histopathological changes in the kidney of fishes were studied .And its include: deformation of brush border , karyolysis , karyohexis , degenration of cell tubules , atrophy of tubules,granuloma and wide necrosis in tubules . But second groups of fishes were transferred in to clean water for recovery period of 8days .And it showed that there was renal damage and no reversal instead .

تاثير التعرض الى الكادميوم والرصاص والاسترداد على نسيج الكلية في يافعات اسماك كارب الكراسين *Carassius carassius*(L.)

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الخلاصة:

وجد في هذه الدراسة عدة تغيرات في الكلية عند تعرض يافعات اسماك الكارب الكراسي الى تراكيز(٢،.و٥،.)وتركيز(١٥و٣٠) جزء بالمليون لكل من الكادميوم والرصاص على التوالي وكلا العنصرين سببا تغيرات نسيجية منها تحطم الانوية وخلايا النبيبات وتحطم واسع في الانابيب الكلوية . وبعد اجراء عملية الاسترداد عند وضع الاسماك في ماء نقي لمدة ٨ ايام فلايوجد تحسن واضح لتلك التغيرات.

Introduction :

Heavy metals are serious pollutants in the water environment and are concentrated by marine organisms (Lloyd, 1992 ; Papathanssion and King , 1993) .The cadmium and lead are one of ahighly toxic to aquatic organisms and is aknown renal toxicant (Singhal and Jain,1997) .Histological analysis appears to be avery sensitive parameter and is crucial in determining celluler changes that may occur in target organs such as kidney , gills and liver (Dutta ,1996).Al-Sudani (1999) and Al-Ali (1999)showed that the lower levels of heavy metals induce physiological and morphological abnormalities in aquatic organisms.

Kidney are one of the most permeable regions of body of fish which are composed of three distinct systems: endocrine hematopoietic, excretory and cortex .Foerlin *et. al,*(1986)observed different responses in the kidney of fishes exposed to cadmium these responses were accompanied by histopathological changes in the proximal renal tubules which are probably associated with the plasma hypocalcaemia response . Durable and Shah (1981) investigated the toxic effects of cadmium kidney of *channa punctatus* .The cadmium induced histopathological changes observed in the kidneys of *Cyprinus carpio* were similar to pathological changes observed in other fishes due to heavy metal toxicity (Singhal and Jain , 1997) . Histopathology has been used by many studies as asublethal test for evaluation toxic effects of water pollutants on fish (Murty, 1986 ; Randy *et. al,* 1996) . The study assess renal damage which caused by exposure to law and high concentrations of both Cd & Pb to study the effect of them and after recovery.

Materials and Methods:

The juveniles of *C. carassius* used for this study were collected from aquiculture of marine science center,Basrah university,Garmat Ali,with weighted 15g.These fish were acclimated to laboratory conditions for seven days prior to use by holding in glass aquaria.The animals were starved for(24)h before use.Three replicates and four individuals were used in this experiment for the Cd and Pb in addition to control.An aqueous stock solution of 1.000part per thuosand was prepared by dissolving (1.9446and1.5985)g from($CdCl_2.2H_2O$and$Pb(NO_3)_2$ respectively in alitter of distilled water. PH(7.8) , Hardness 700mg/l , Oxygen (8.1) mg /l and temperature (25 ±2Ċ) .

Fish exposed to(0.2,0.5) ppm and (15,30) ppm concentration of Cd , Pb respectively for period (LT_{50})for eight days(Al-Mansoori and Saoud ,2002) .Some of this fishes transferd to tap water to study the recovery, kidney samples from dissected fish fixed in boin's solution , dehydrated through an ascending ethanol series , cleared in xylene , and infiltrated with paraffin sectioning of paraffin blocks was done on microtome at 6-7µm and stains with delafield's hematoxylin with eosin (Humason,1971) histological lesions were located light microscopically and photographed with computer camera .

Results:

Histology of kidney:

The fish kidney consist of Bowman's capsules which contain glommeruli,intermediate and distal segments, collecting tubules and interstitial hematopoietic tissue(fig.1).

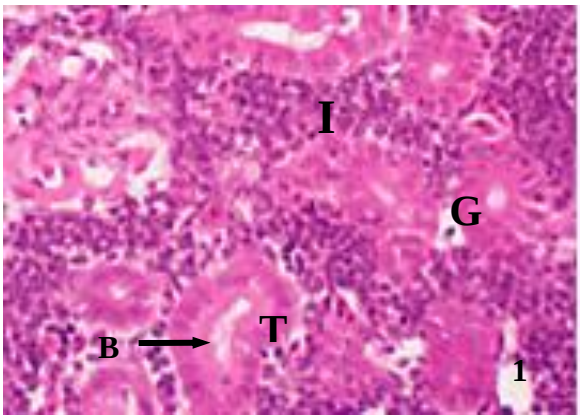

Figure 1. photomicrograph of kidney from acontrol fish ,(B) Bowman's capsules,(G) glommeruli , (T) tubules) ,(I) inter stitial hematopoietic tissue.(H&E. 400X

Histological changes of kidney after exposure to cadmium .

This changes of tubules began by deformation of brush border,gradual atrophy of basal cytoplasm and condensation of nuclear material(fig.2) following by focal necrosis of tubular cells and karyolysis (disintegrating nucleus)and karyohexis(fragmented nucleus)(fig.3,4).Fucal degeneration of tubuler cell was usually followed by more extensive necrosis of the whole nephron ,leukocyte and erythrocyte condensation in the intistenal tissue and tubuler constracyion this found when fish exposed to 0.5 ppm of Cd(fig.5 to 7).

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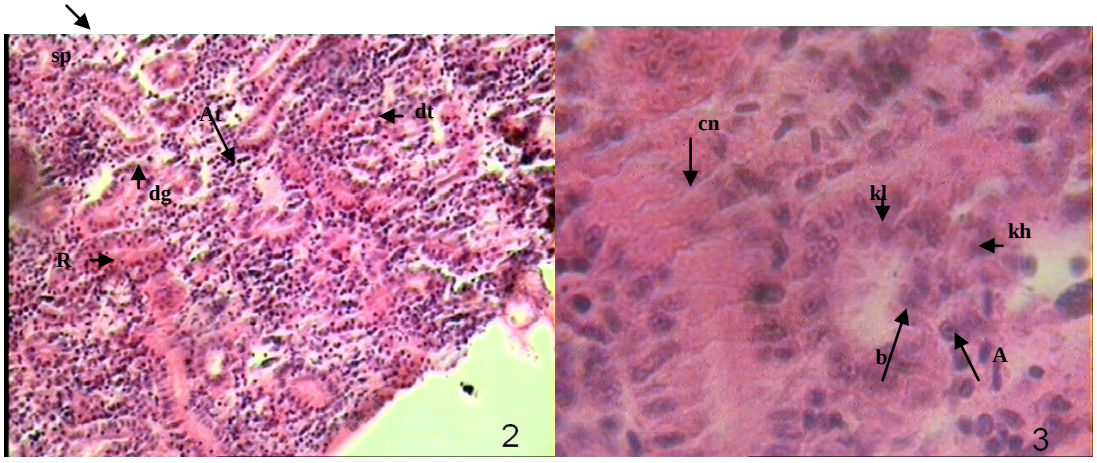

Fig. 2 cross section of kidney of *C. carasius* exposed to (0.2 ppm) of cd for 8 day showing degenration tubules(dt) , atrophy of tubules(At) and defution of tubule, separation of tubule basal lamina from epithelial wall(sp),condensation of red blood cells (R) degeneration granuloma(dg).(H& E 100X)

Fig. 3 cross section of kidney of C. *carassius* exposed to(0.2 ppm) of cd for 8 days showing deformation of brush border (b) , condensation of nuclear materials (cn) , atrophy of tubule cell (A), karyolysis (kl) karyohexis (kh) .(H&E 400X).

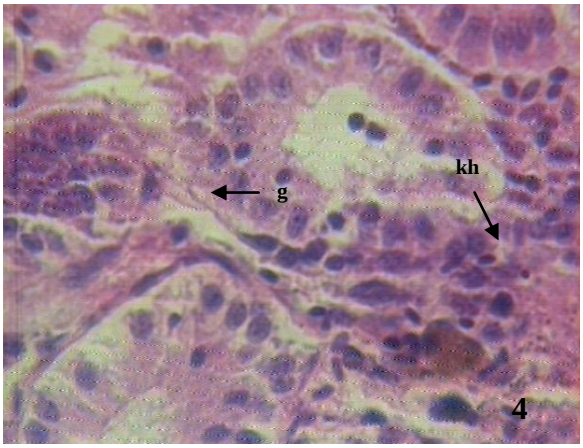

Fig. 4 cross section of kidney of C. carassius exposed to (0.2 ppm) of cd for 8 days showing granuloma (g) and karyohexis (kh). (H&E 400X).

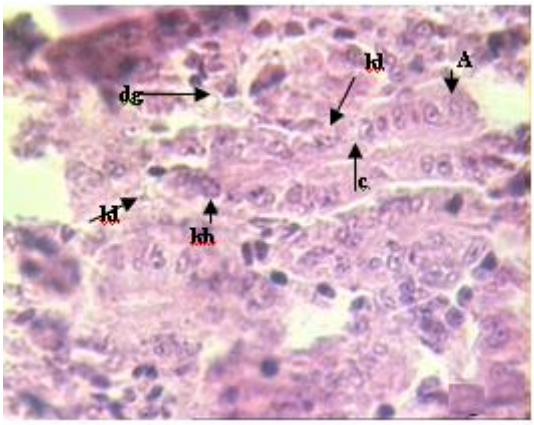

Fig .5 Cross section of kidney of *C. carasius* exposed to (0.5 ppm) of cd for 8 day showing complete disappearance of tubules(d) and loss of nuclei of cells(L) ,distrbution of erythrocyte and leukocyte(db). (H & E 100X)

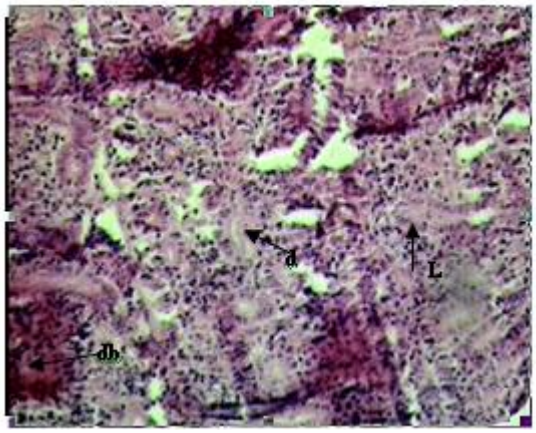

Fig. 6 cross section of kidney of *C. carasius* exposed to (0.5 ppm) of cd for 8 day showing, karyolysis(kl) , karyohexis (kh), atrophy of cell tubules (A)and constraction of tubules(c) degenration tubul cell (D) and degeneration granuloma(dg). (H & E 400X)

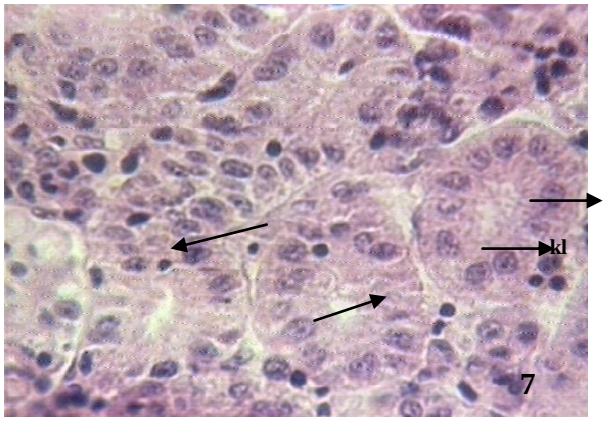

Fig.7 Cross section of kidney *C. carassius* exposed to (0.5 ppm) of cd for 8 days showing karyolysis (kl) , degeneration of cell (d) , shrinking of tubule (s) and necrosis (n) . (H&E 400X)

Histological changes of kidney after exposure to lead.

Similar changes were found by lead but less than cadmium causes karyolysis ,karyohexis, separation of tubules basal lamina from epithelial wall ,concentraction tubules ,cell cytoplasm depresin the lumina atrophy of granuloma, tubule atrophy and distribution of erythrocyte , leukocyte and wide necrosis in tubules (fig.8 to 11).

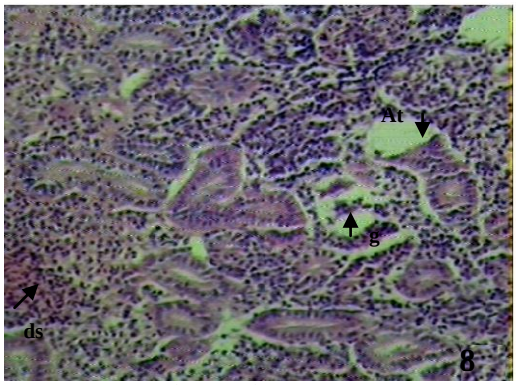

Fig. 8. cross section of kidney of *C. carasius* exposed to (15 ppm) of pb for 8 day showing atrophy of granuloma(g), atrophical tubule (At) ,and distribution of erythrocyte and leukocyte(ds) . (H & E 100X)

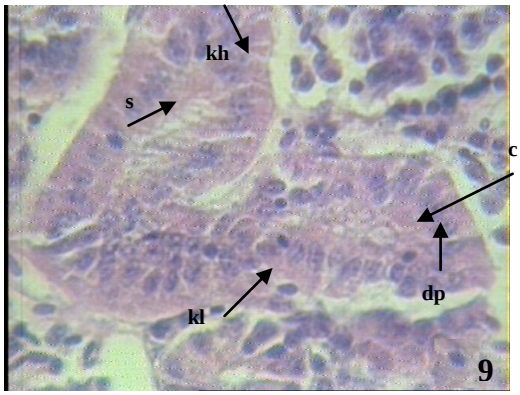

Fig.9. cross section of kidney of *C. carasius* exposed to (15 ppm) of pb for 8 day showing karyolysis(kl) , karyohexis(kh) separation of tubule (s) , concetraction tubules(c) and cell cytoplasm depresin the Lumina(dp). (H & E 400X)

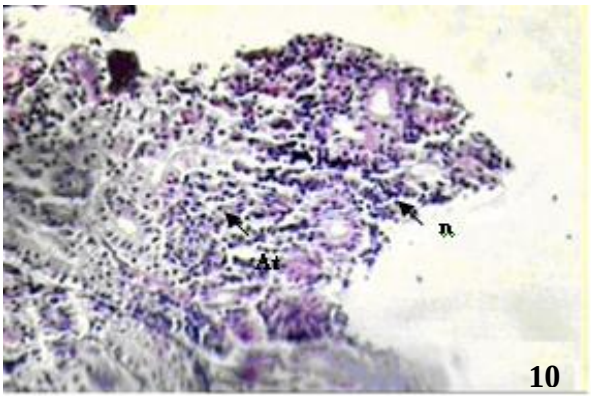

Fig. 10 . Cross section of kidney of *C. carasius* exposed to (30 ppm) of pb for 8 day showing wide necrosis in tubules (nx) and atrophy of tubules (At). (H & E 100X)

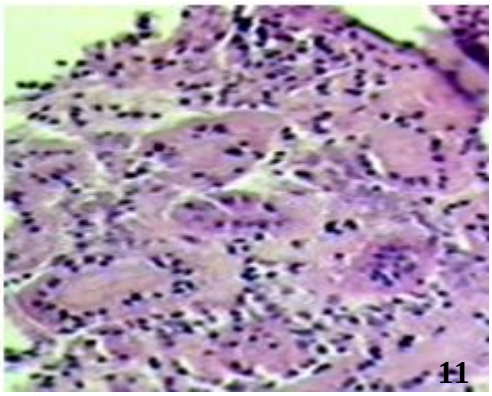

Fig.11 Cross section of kidney of *C. carassius* exposed to (30 ppm) of pb for 8 days showing edema (e) and necrosis (H & E 100X).

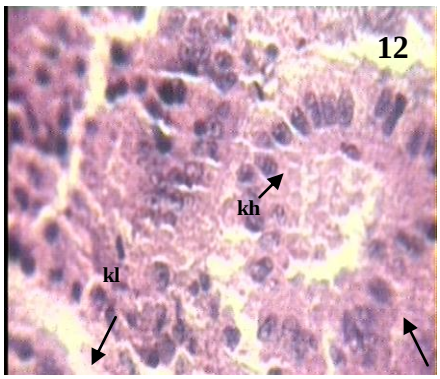

Fig. 12 . Cross section of kidney of *C. carasius* exposed to (30ppm) of pb for 8 day showing karyolysis(kl) , karyohexis(kh) and cells degenration (d) . (H & E 400X).

Histological changes after recovery period for Cd and Pb.

Showed assess renal damage which caused by cadmium and lead ,some of the exposed animals were placed in normal water for 8 days.The histological examination of the kidney of these fish observed that the renal damage continued resulting in greater degeneration changes such as tubule,epithelial wall edema and deformation tubule and atrophy (fig.13-14)for cd and (fig.15-16)for pb.

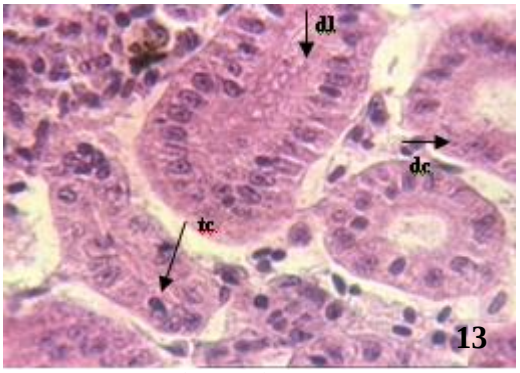

Fig. 13. cross section of kidney of *C. carasius* exposed to (0.2 ppm) of cd for 8 day and revived for the same period in normal water cell depris in the Lumina (dl) , tubules construction (tc)and cell degeneration(dc). (H & E 400X)

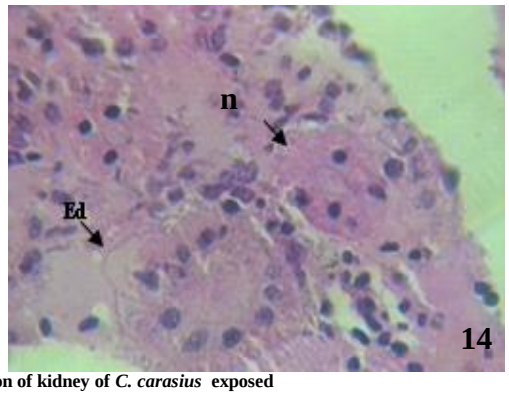

Fig. 14 . cross section of kidney of *C. carasius* exposed to (0.5 ppm) of cd for 8 day and revived for the same period in normal water. this picture shows the edema(ed) and cell necrosis(n). (H & E 400X).

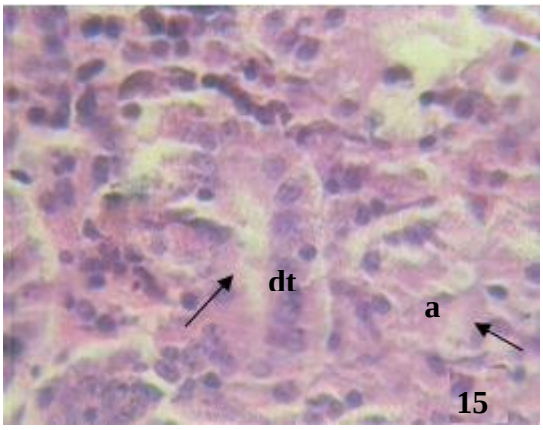

Fig. 15. cross section of kidney of *C. carasius* exposed to (15 ppm) of pb for 8 day and revived for the same period in normal water shows deformation tubules(dt) and atrophy(A). (H & E 400X)

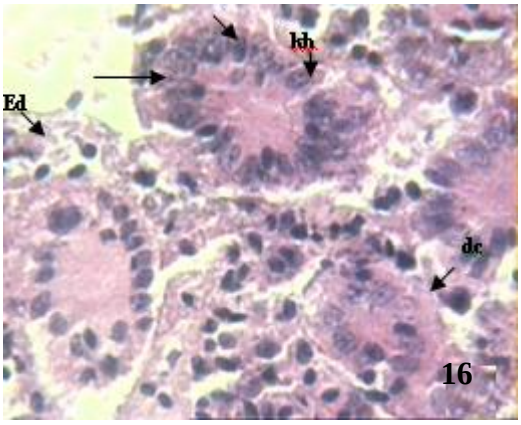

Fig.16. cross section of kidney of *C. carasius* exposed to (30 ppm) of pb for 8 day and revived for the same period in normal water . this picture shows degenration cells(dc) , karyohexis(kh) and karyolysis(kl). (H & E 400X).

Discussion:

Heavy metals are seriouse pollutants of the aquatic environment because of their environmental persistence and ability to be concentrated by aquatic organisms (Veena *et.al*.,1997).Cadmium and lead are non-biodegradable and non-

beneficial heavy metals (Bailey *et.al*.,1999).In this study most pathologies were specific for cadmium and lead in addition the prevalence of pathologies increased with concentration increased.The cadmium and lead induced histopatholoical changes observed in kidney of *C. carassius* were similar to pathological changes observed in to the fishes due to heavy metal toxicity (Singhal & Jain , 1997 ; Ooi & Law ,1989) .All species of experimental animals studied lead has been shown to causes adverse effect in several organs including renal system (Internet-1).

The renal tubular changes in fish might lead to neoplasia and some morphological lesions can be caused physiological effected. The exposure to different concentrations of cadmium and lead induced tubular necrosis and renal dysfunction and since the renal tubular epithelium has its major function in the excration of divalent ions,the pollution with heavy metals can be effect this cells which lead to osmatic and ionic regulation disfunction and this seen by (Al-Mansoori & Saoud,2002)that there are disturbance of ionic balance through of decreas of Na+,K+ concentration in blood plasma of fish exposed to cadmium and lead.Chronic nephropathy occurs after prolonged exposure to lead causes atrophy or hyperplasia of tubular epithelial cell and progressive interstitial and glomerular (Internet-2) and (Internet-3) the observation of lead-induced kidney tumor in rats,conclusive evidence for lead-induced renal cancer(or any other type of cancer)..Roberts(1978) and Schaperclaus *et.al*., (1991) reported the reduced stainability in the hepatic cell of kidney tissues by heavy metal stressed teleosts.Crespo *et. al*.,(1986)showed morphological changes in intestine trout found swellon and detection in epithelial layer extensive in intracellular and degeneration epithelial layer after exposure to Cd and Pb while Latif *et. al*., (1982) found that the Cd and Pb can

accumulate in kidney of *Barbus grypus* and *Barbus belaywim* will Sexena &Parashari (1981) found that the cadmium cause renal tumors in 10 – 30 % fishes . The kidney showed hydropic swelling of tubular cell vaculation and numerous dark granule accumulation in many tubules, tubular degeneration and necrosis were seen in some areas (Thophon *et. al*, 2003) . Whereas Ghosh & Chkrabarti (1993) was exposed to asublethal concentration Cdcl2 57Mg /liter for 30days the kidney exhibited rupture of tubular epithelium including its brush border , degeneration of glomeruli extrusion of cellular material in to the tubular lumen and extensive loss of interstitial hemotopioetic tissue But Rostami – Bashman *et.al*., (2000) observed changes in kidney includes necrosis , hyaline degeneration of tubules and substitution with lymphocytes after exposed to 10 mg/l of Cd for 24h on *Cyprinus carpio*.

Durable & Shah(1981) showed that the cells of proximal tubules of the kidney were the first to be affected soon there after the injury spread to the glomerulus's, hematopoietic tissue of other parts of the tubules . Zike & Osman(2003) Found shrinkage of the glomerular tufts and degeneration of the proximal tubular epithelium in the kidneys of exposed fish were observed rupture of pillar cells and capillaries associated with lamellar telangiectasis were also observed . .

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