SOME TOXICOLOGICAL STUDIES ON EFFECT OF SOME WATER POLLUTANTS (HEAVY METALS) IN ALBINO RATS

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A B S T R A C T

The toxicological studies of cadmium (Cd), lead (Pb) and manganese (Mn), were carried out on albino rat. Twenty five rat classified to five equal groups. First group was given water contain Cd, Pb and Mn, as (*0.54g /L ,0.73g L 0.54g /L)respectively* and kept as positive control. Second, third and forth groups were given water (orally by stomach tube 1ml/rat/day for 2 month) contain some pollutant as first group but after treatment with alum, ferric chloride and *Pseudomonas* bacteria respectively . Fifth group administrated distal water and kept as control negative. At the end of experiment serum sample were collected for estimation of Alanine amino transferase (ALT), Aspatrate amino transferase (AST) and electrolytes (Na, Ca, Ph and K), liver samples collected for determination of oxidative cascade and samples from liver and kidney were collected for residues and histopathological examination. The result indicated that the forth group (given water treated with *Pseudomonas* bacteria) showed less toxic effect than other groups (second and third) in which water was treated by alum and ferric chloride respectively. We can concluded that removal of heavy metal from water by bacteria was more effective than removal by ferric chloride which was more effective than removal by alum.

Key words: Heavy metal, oxidative stress, residues

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1. INTRODUCTION

Environmental pollution is one of the major problems of the world and it is increasing day by day due to urbanization and industrialization (Mustafa et al 2010). Water used in industries creates a waste that has potential hazards for our environment due to the introduction of various contaminants such as heavy metal (Prabavathy and De 2010). These pollutants find their way to aquatic ecosystem such as river, ponds and lakes which cause risk to the health of human and ecosystem (Rehman Anjum and Reddy 2013). Heavy metals are toxic at relatively low concentration (Raymond and Felix 2011). Heavy metals exert their toxic effect by generating reactive oxygen species (ROS) such as O^{2-}, H_2O_2, and .OH causing oxidative stress(Shaista et al., 2010, Chezhian et al., 2011, Mehmet et al., 2011). Foyer and Noctor 2002 reported that reactive oxygen species will affect the regular metabolism by damaging the cellular components. Lead can cause adverse effect on heart, brain, kidney, reproductive tract and also cause chromosomal abnormalities (Hao et al., 2002) also cadmium cause poisoning in various tissues (liver, kidney and testes) of humans and animals (Stohs et al., 2000) while Lu et al., 2005 reported that although manganese has less poisoning effects on human comparing with other metals but chronic entering to body more than usual amount has pathologic effect called Magnesia. Cadmium, lead and manganese have the ability to induce and synergize significant imbalance in plasma electrolytes in mice in a short period of time and therefore can be used as biomarker of heavy metal pollution as well as deterioration (Osuala et al 2013). For all the above many

trails were performed for treatment of polluted water such trails include bioremediation and coagulation with aluminum or iron salts. In bioremediation a biological agents, mainly microorganisms, e.g. yeast, fungi or bacteria to clean up contaminated soil and water (Strong and Burgess, 2008). Coagulation is one of the most important physicochemical operations used in water treatment (Pernitsky and Edzwald 2006). The most common coagulation process is by alum while coagulation with iron salt produce good result when conditions are too acidic and give chemical sludge that difficult to removed (Tripathy and De 2006). This study aimed to investigate the toxic effect of heavy metals (in polluted water before and after treatment) on rats through, detect the residues of heavy metal in liver and kidneys , determine liver oxidative cascade (GSH, GST and MDA),analysis of serum electrolytes(Ca, Na, Ph and K) and serum level of ALT and AST activity.

2. MATERIAL AND METHOD

2.1 Experimental animals.

Twenty five apparently healthy albino rats were obtained from Veterinary Serum And Vaccine Research Institute, Abbasia, Cairo, Egypt. The animals housed in stainless steel wire bottom cages and kept under constant environmental conditions and fed on fresh standard pellet and given tap water ad libitum throughout the study. All animals were acclimatized for 1 week before the beginning of the experiment.

2.2 Experimental design.

In this study twenty five albino rats were divided in to five groups each one five rats. First Group administrated water polluted with cadmium (*0.54g cadmium sulphate /1L distal water),* lead *(0.73g lead acetate /1L distal water*) and manganese *(0.54g manganese sulphate / 1Ldistal water)* and kept as positive control. Second Group administrated water after treatment with conventional method by Alum. Third Group administrated water after treatment with chemical method by ferric chloride. Forth Group administrated water after treatment with bioremediation by *Pseudomonas* bacteria. Fifth Group administrated distal water and kept as negative control. 1ml water daily / rat, orally by stomach tube for 2 months, kept under observation all over the experimental period.

2.3 Detection of trace element residue in liver and kidney.

Determinations of trace element were performed according to (Al Ghais 1995).

2.4 Detection of liver oxidative stress parameters.

Determination of GSH, GST and MDA in liver tissue homogenate were performed according to (Beutler E., et al 1963; Habig *et al.,* 1974 and Satoh K., 1978) respectively.

2.5 Biochemical analysis.

ALT and AST in serum samples according to (Schumann and Klauke 2003) while Potassium ,Phosphorous, Sodium and Calcium levels in serum samples were detected according to (Sunderman and Sunderman, 1985, El-Merzabani et al., 1977, Trinder, 1951 and Grindler and King, 1972) respectively .

2.6 Histopathological examination.

Autopsy samples were taken from liver and kidney in different group of rat for histopathological examinations according to Banchroft et al, 1996.

2.7 Statistical analysis.

The data were analyzed for obtaining mean, standard deviation (SD) and statistical comparisons between means of different groups. The statistical analyses were done by one way

ANOVA and DUNCAN test using SPSS program version 16. (Kirk 1982). *P* value < 0.05 was assumed for statistical significance.

3. RESULT

3.1 Heavy metal residues in liver and kidney.

Table (1) illustrated that Regarding cadmium residue in liver and kidney of rats in control positive group first group was significant increase when compared with control negative group fifth group while after water treatment it was significant increased in second and third group however in between 3 groups of treatment there were significant increase in second and third group in compared with forth group . Lead and manganese residue in liver and kidney of rats in control positive group showed significant increase in compared with control negative group while after water treatment there were significant increase in second, third and forth group in compared with control negative group and in compared in between.

3.2 Liver oxidative stress parameters.

Concerning to level of GSH highly significant decreased in control positive group (first group) in comparison to control negative group (fifth group). Second and third group showed significant decrease in GSH when compared with control negative group, while forth group cleared no significant decreased in compared with control negative group. Concerning to level of GST and MDA were significant increased in control positive group (first group) were recorded in comparison to control negative group (fifth group). groups (second and third)taken water after treatment by Alum and ferric chloride there were significant increase in GST and MDA when compared with control negative group, while after treatment of water by pseudomonas bacteria there were no significant increase in compared with control negative group as cleared in table (2).

3.3 Biochemical analysis.

3.3.1 ALT and AST level: Concerning to ALT level significant increase in control positive group (first group) in comparison to control negative group (fifth group). groups given water after treatment by Alum and ferric chloride(second and third) there were significant increase when compared with control negative group, while group given water treated by bioremediation(forth group) there were no significant increase in compared with control negative group. Concerning to AST level significant increase in control positive group was observed. After treatment of water by Alum and ferric chloride there were significant increase recorded in second and third group when compared with control negative group, while after treatment of water by bioremediation(forth group) there were no significant increase in compared with control negative group as showed in table (3).

3.3.2 Electrolytes levels in serum .Regarding to the effect of heavy metal on the mineral (calcium, phosphorous, sodium and potassium) there were significant increase at groups first, second, third and forth in comparison to control negative group (fifth group) as clear in table (4)

3.4 Histopathological examination.

Concerning to histopathological finding on liver and kidney. Liver first group showed focal inflammatory cells aggregation was detected in the portal

Table (1). Residue of polluted surface water before and after treatment on liver and kidney of albino rats.

Item	WHO/ FAO 2007 mg/kg	Group I Control positive	Group II P.S.W. treated Alum	Group III P.S.W treated Ferric chloride	Group IV P.S.W. treated Bioremediation	Group V Control Negative
Cd Liver	----	0.556 ± 0.01[a]	0.151± 0.009[b]	0.068± 0.015[c]	0.027 ±0.015[d]	0.001± 0.001[d]
Cd Kidney	1	2.56 ± 0.19 [a]	1.64 ± 0.07 [b]	1.22 ±0.01 [c]	0.15± 0.04[d]	0.020± 0.01 [d]
Pb Liver	0.1-0.5	0.78 ± 0.05 [a]	0.18 ± 0.03 [b]	0.17± 0.02 [b]	0.09± 0.01[c]	0.046 ± 0.006 [d]
Pb Kidney	0.1-0.5	0.71 ± 0.05 [a]	0.19 ± 0.02 [b]	0.11± 0.01 [c]	0.09± 0.03 [c]	0.044 ± 0.01 [d]
Mn Liver	0.5	0.577± 0.02 [a]	0.200 ± 0.02 [b]	0.178± 0.106 [b]	0.088± 0.008[c]	0.03 ± 0.007[d]
Mn Kidney	0.5	0.55 ± 0.008 [a]	0.108± 0.013 [b]	0.074 ±0.01 [c]	0.042± 0.003[c]	0.015 ± 0.025 [d]

Mean with different letters at the same raw differ significant (*P < 0.05*).

Table (2). Effect of polluted surface water before and after treatment on oxidative stress parameters on liver homogenate of albino rats.

Groups	GSH mmol/g. tissue	GST U/ g. tissue	MDA nmol/g. tissue
Group I. (Positive Control) P. S.W. wit3h Cd, Pb and Mn	5.77± 0.81[c]	1.5 ± 0.02[a]	12.47 ±0.74[a]
Group II. P. S.W. treated with Alum	7.91± 0.22 [b]	1.29 ±0.21 [a]	8.88 ±0.46 [b]
Group III. P. S.W. treated with ferric chloride	9.67± 1.19 [b]	1.14 ± 0.22 [a]	8.06± 0.64 [b]
Group IV. P. S.W. treated with Bioremedation	11.94± 0.53 [a]	1.0 5 ± 0.2 [a]	7.53 ±1.01 [b]
Group V. (Negative Control) Distal water	14.08± 0.25 [a]	0.84 ±0.21 [b]	6.25 ±0.72 [c]

Mean with different letters at the same raw differ significant *(P < 0.05).*

Table (3). Effect of polluted surface water before and after treatment on ALT & AST level of serum of albino rats.

Groups	ALT U/L	AST U / L
Group I. (Positive Control) P. S.W. with Cd, Pb and Mn	49.98± 3.11 [a]	90.4 ± 4.7 [a]
Group II. P. S.W. treated with Alum	32.6 ± 1.50 [b]	65.7± 3.9 [b]
Group III. P. S.W. treated with ferric chloride	20.16± 4.2 [c]	53.0± 4.7 [c]
Group IV. P. S.W. treated with Bioremediation	17.44± 2.4 [d]	48.1± 2.9 [d]
Group V. (Negative Control) Distal water	16.98± 5.9 [d]	45.61± 4.0 [e]

Mean with different letters at the same raw differ significant *(P < 0.05)*.

Table (4). Effect of polluted surface water before and after treatment on electrolytes level of serum of albino rats.

Mean with different letters at the same raw differ significant *(P < 0.05)*.

Groups	Sodium mEq / L	Potassi mEq / L	Calcium mg/dl	Phosph mg/dl
Group I. (Positive Control) P. S.W. with Cd, Pb and Mn	187.20± 1.85[a]	15.14± 1.72[a]	20.07 ±0.03[a]	19.88 ± 0.007[a]
Group II. P. S.W. treated with Alum	175.02± 1.19[a]	9.50 ±0.09 [b]	15.92± 0.08[b]	13.78 ± 0.01[b]
Group III. P. S.W. treated with ferric chloride	169.99± 0.92[a]	9.58± 0.24 [c]	14.72± 0.07[c]	13.66 ± 0.04[c]
Group IV. P. S.W. treated with Bioremedation	160.62± 2.1[b]	8.61 ±0.03 [d]	14.53± 0.02[d]	12.49 ± 0.02[d]
Group V. (Negative Control) Distal water	156.68± 0.44[c]	7.58 ±0.11[d]	13.49± 0.01[e]	11.40 ± 0.007[e]

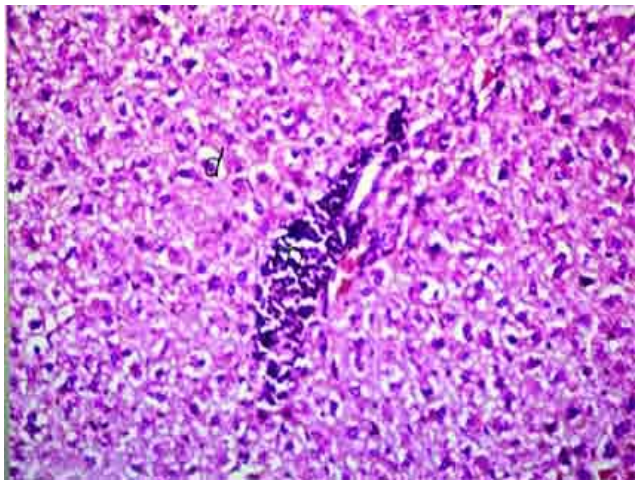


Fig (1). Liver of rat in group I showing degenerative changes in the hepatocytes (d) with inflammatory cells aggregation (m) in portal area. H & E stain x400.

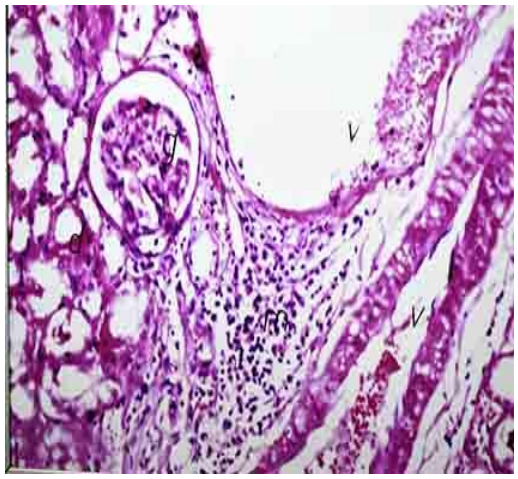


Fig (2). Kidney of rat in group I showing perivascular and periglomerular inflammatory cells infiltration (m) with congestion in blood vessels (v). H & E stain x 400.

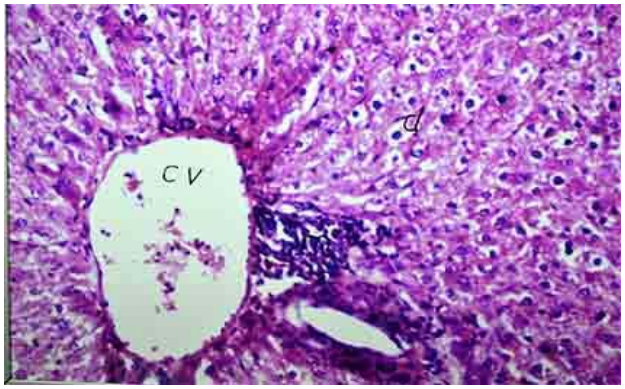


Fig (3). liver of rat in group II showing dilation in portal vein (Pv) with inflammatory cells aggregation in portal areas (m) as well as dilatation in bile duct (bd) and degeneration in hepatocytes (d). H&E stain x 400.

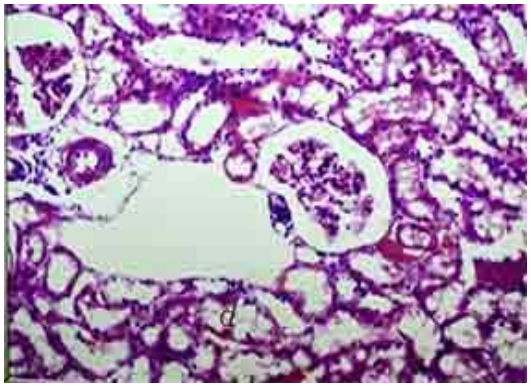
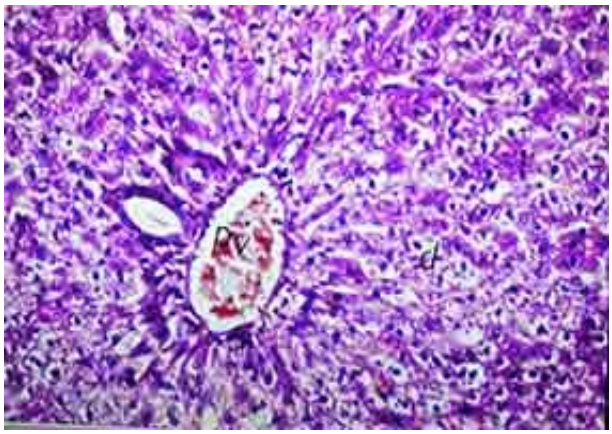


Fig (4) kidney of rat in group II showing degeneration in lining epithelium of the tubules (d) H &E stain x 400



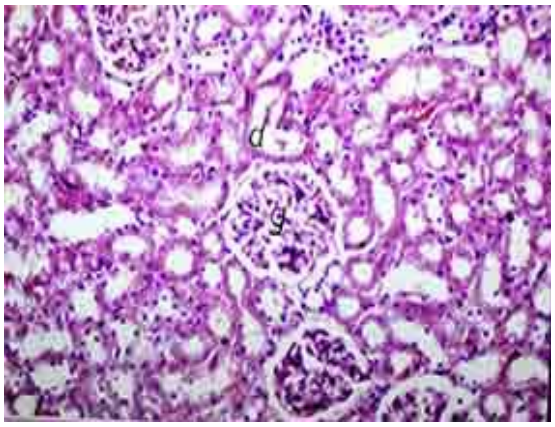


Fig (5). Liver of rat in group III showing congestion in portal vein (pv) with degeneration in the hepatocytes (d). H & E stain x 400.

Fig (6). Kidney of rat in group III showing mild congestion in glomerular tuft (g). H & E stain x 400.

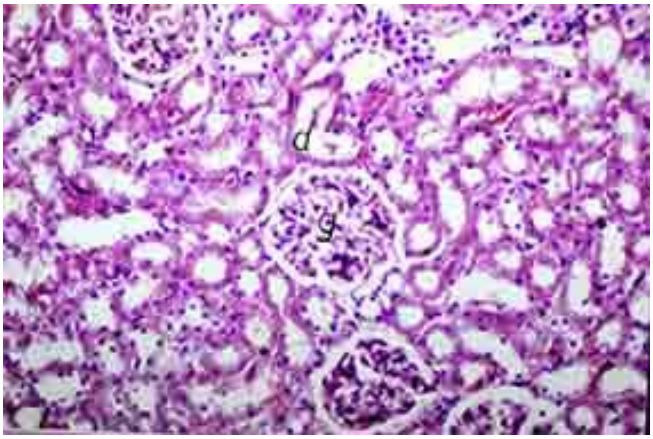


Fig (7). Liver of rat in group IV showing normal histological structure. H & E stain x 400

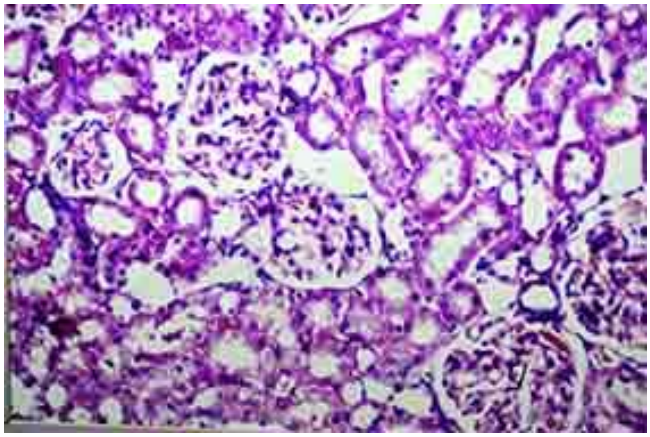


Fig (8) kidney of rat in group IV showing mild congestion in glomerular tuft (g) H & E stain x 400.

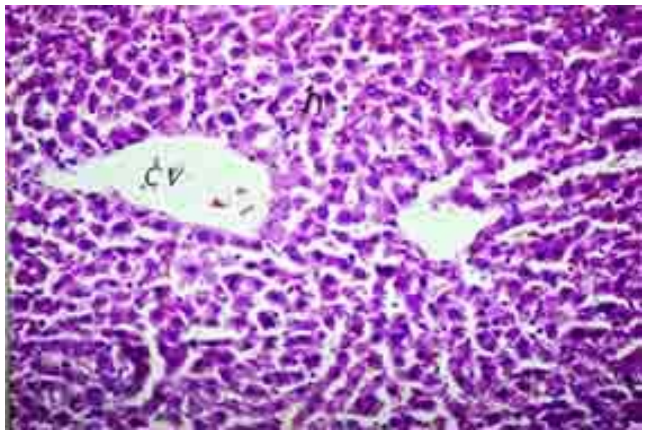


Fig 9. Liver of rat in group V showing n normal histological structure of the central vein (CV) and surrounding hepatocytes (h) H&E stain x 400.

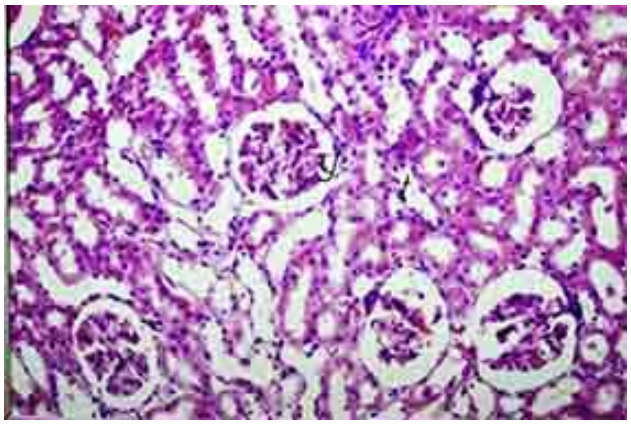


Fig 10. Kidney of rat in group V showing normal histological structure of the glomeruli (g) and tubules (t) of the cortex .H & E stain x 400.

area associated with degenerative change in the hepatocytes. The portal areas showed sever congestion in the portal vein as well as dilatation in the bile ducts (Fig.1). Kidney showed focal inflammatory cells infiltration was observed in the perivascular and in the periglomerular tissues, as well as in between the degenerated and necrosis tubules at the cortex. The corticomedullary portion showed degenerative change in the tubules (Fig.2). Liver of second group showed inflammatory cells aggregation in portal area as well as dilatation in the portal veins and bile ducts (Fig.3). While kidney showed degeneration in the lining epithelium of the tubules (Fig.4). Liver of third group showed congestion in the portal vein associated with degeneration in the heopatocytes (Fig.5). While kidney showed mild congestion in the glomerular tufts (Fig.6).Liver of forth group showed no histopathological alterations (Fig.7). While kidney showed mild congestion was noticed in the glomerular tufts (Fig.8). All these results in comparison to control negative group as clear in (Fig 9&10).

4. DISCUSSION

Results of Heavy metal residues in liver and kidney before water treatment were similar to (Bala et al 2012 and Bala et al 2013) who also reported high concentration of lead in liver than in kidney which may be as a result of detoxification of toxic substance by the liver, while concentration found in kidney may be as a result of excretory function of the kidney, in which some toxic substance are mobilized from the body tissues and are send to the kidney for excretion. This result was in line with those reported by (Doyle and Spaulding 1978 and Stabel-Tancher et al 1975). Highest level of cadmium which found in kidney samples and more than in the liver are agreed with (Gasparik et al., 2004). The higher concentration of cadmium in the kidney tissue is due to detoxification function of the organ where these metals accumulated (Stoyke et al., 1995). Animals exposed to cadmium accumulate it in their livers and kidneys as their free protein thiol group content which leads to strong fixation of heavy metals. The concentration of manganese in the liver is significantly higher than that of kidneys. Our results are in line with (Razavian and Rabiee 2014). Concentration of manganese in liver was higher than that of kidney may be due to Mn is repelled by faces, urine, milk and sweat, but the main place to filtrate Mn is the liver (99%) so that higher concentration of manganese makes some disorders on fatty acids elongation and saturation in the liver (Senturk and Oner 1996). Our results supported by histopathological changes on liver (Fig. 1).

Our result of Liver oxidative stress parameters in group taken water before treatment was agreed with (El-Sayed 2008) which may be due to heavy metal induced oxidative damage by increased lipid peroxidation and inhibitions of enzymes required to prevent such oxidative damage. Similar results showed by (Kelley *et al*., 1999).The damages caused by heavy metal were clear in the histological examination in the form of vacuolar, hydropic as well as fatty changes of the hepatocytes, renal tubular and testicular cells that finally showed sloughed. These results were coincide with those reported by many investigators (Gunn *et al*., 1963, Gibbiani, 1966, WHO, 1992, Karl *et al*., 2005). A possible explanations for heavy metal induced damage via production of free radicals that alter mitochondrial activity and genetic information. The cellular damage showed in liver, kidneys and testes in the present study results from cadmium binding to SH –group in tissues, the production of lipid peroxides and the depletion of the

glutathione besides inhibition of the activity of antioxidant enzymes (Sarkar *et al*., 1995). The another explanation is that heavy metal detoxified in the liver through formation of metallothionein complex, which is slowly released from that organ and causing congestion, hemorrhage, apoptosis and necrosis (Brazoska *et al*., 2003). Finally, the renal damaged in the present study may be due to that heavy metal induced impairment to the glomerular infiltrations in rats causing renal tubular degeneration, necrosis and fibrosis (Uriu *et al*., 1998).this result confirmed with the histopathological change in fig (1) as there were Focal inflammatory cells aggregation was detected in the portal area associated with degenerative change in the hepatocytes (fatty change and vacuolar degeneration) in liver and there were Focal inflammatory cells infiltration was observed in the perivascular and in the periglomerular tissues as well as in between the degenerated and necrosis tubules at the cortex. The corticomedullary portion showed degenerative change in the tubules (Fig 2) in kidney.

Our result of ALT and AST before treatment similar to result obtained by (Nabil, et al 2012 and Shi and Mao 1994). The significant increase on ALT and AST activities throughout the experimental period is directly related to progressive liver damage and necrosis leading to liberation of these enzymes or due to extensive break down of body tissue (George et al 1963). Furthermore may be due to free radicals generating during thiol autoxidation (Thiol and oxygen radicals) may be the primary sources of oxidants that may contribute to the heavy metals induced high liver microsomal membrane fluidity, free radical generation and alteration in the liver tissue histogram (Abdou, et al 2007). Our results supported by histopathological changes on liver fig (1)).

Our result of electrolytes level in serum before treatment agreed with result obtained by (Sheikh et al 2011). This result may be due to trace elements cause alteration on channel properties it has been showed that membrane proteins including ion channels are responsive to redox state (Bertl and Slayman 1990). Changes in the redox state of amino acid residues in channel proteins may lead to a conformational change and alterations of channel activity (Ruppersberg et al 1991). that may contributed by heavy metal have the ability to induce and synergise significant imbalance in plasma electrolyte in mice in short period of time and therefore can be used as a biomarker of heavy metal pollution as well as as early warning signal of cell deterioration (Osuala, et al 2013). Our result confirmed by histopathological finding on liver and kidney of treated groups our result agreed with Vinoth Kumar *et al* 2010

Our result after polluted water treatment may be attributed to treatment by bioremediation occurred as the bacterial cell wall is the first component that comes into contact with metal ions where the solutes can be deposited on the surface or within the cell wall structure. Since the mode of solute uptake by dead/inactive cells is extracellular, the chemical functional groups of the cell wall play vital roles in biosorption (Doyle et al., 1980). Treatment by Alum and Ferric chloride occurred as they form coagulant with heavy metal that can be easily removed (Citulski et al 2009).

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بعض الدراسات السمية على تأثير بعض ملوثات المياه (المعادن الثقيلة) على الفئران البيضاء

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اجريت الدراسة لمعرفة التأثير السمي للكادميوم والرصاص والمنجنيز على الفئران البيضاء. تم تقسيم خمس وعشرون فأر على خمس مجموعات يعنى كل مجموعة بها خمسة فئران. المجموعة الأولى. تم اعطائها مياه تحتوي على عنصر الكادميوم والرصاص والمنجنيز بالكميات التالية 0.54 جم / لتر من الكادميوم و0.54 جم/ لتر من المنجنيز و0.74 جم/ لتر من الرصاص. المجموعة الثانية. تم اعطائها مياه تحتوي على عنصر الكادميوم والرصاص والمنجنيز معالجة بالشبة. المجموعة الثالثة. تم اعطائها مياه تحتوي على عنصر الكادميوم والرصاص والمنجنيز معالجة بكلوريد الحديديك. المجموعة الرابعة. تم اعطائها مياه تحتوي على عنصر الكادميوم والرصاص والمنجنيز معالجة ببكتيريا السيدومونس. المجموعة الخامسة. تم اعطائها مياه مقطر فقط. استمرت التجربة لمدة شهرين وتم أخذ عينات بعد الذبح بعد الشهرين. تم اخذ السيرم لقياس انزيمات الكبد وهي الألانين امينو ترانس فيرز والاسبرتيت امينو ترانس فيرز ولقياس الالكتروليت ايضا مثل الصوديوم و البوتاسيوم و الفوسفور و الكالسيوم. تم اخذ الكبد والكلي لقياس المتبقيات من المعادن فيهم وايضا لعمل الفحص الهيسوباثولوجى.تم اخذ الكبد ايضا لقياس مضادات الأكسدة وتشمل الجلوتاثيون المختزل والجلوتاثيون اس ترانس فيرزو المالونالدهيد.وأظهرت النتائج انه عند قياس وظائف الكبد أنزيمات الألانين امينو ترانس فيرز والاسبرتيت امينو ترانس فيرز حيث أظهرت النتائج زيادة معنوية في نشاط هذه الانزيمات خلال فترة التجربة. وجد عند قياس الالكتروليت ان نسبة الصوديوم والبوتاسيوم والفوسفور والكالسيوم تزيد زيادة معنويه في المجموعة الاولي وتقل في المجموعات المعالجة حت تصل في المجموعة الرابعة الى نسب تكاد تشابه هذه النسب للمجموعه الخامسة او المجموعة الضابطة. وجد عند قياس مضادات الأكسدة في كبد الفئران التى تضمنت قياس الجلوتاثيون المختزل والجلوتاثيون اس ترانس فيرز حيث اوضحت النتائج انخفاضا معنويا في المجموعات التى تم اعطائها المياه المعالج بينما اظهر قياس المالونداي الداهيد ارتفاعا معنويا في ذلك المجموعات مقارنة بالمجموعة الضابطة.الفحص الهستوباثولوجى اسفر عن ان الكبد في المجموعة يحتوى علي تغيرات فسادية لخلايا الكبد لوحظ تراكم من الخلايا الالتهابية مع عدم ترتيب خلايا الكبد وفرط التهيج للخلايا المبطنة للقنوات المرارية كما شوهد تغيرات فسادية للخلايا المبطنة للأنابيب البولية في الكلى وكانت هذه التغييرات شديدة في المجموعة الاولى اما المجموعة الثانية والثالثة فهى تقل أما المجموعة الرابعة فهى تكاد تشبه المجموعة الخامسة او المجموعة الضابطة. بعد العلاج بالطرق المختلفة وجد ان العلاج بالشبة اقل تاثيرا من العلاج بكلوريد الحديدك اقل تأثيرا من العلاج بالبكتيريا.

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