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In Vivo Study of Physiological, and Histological Effects of Different Doses of Mercury Oxide on Liver and Kidney in Male Wistar Rats

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Abstract

MERCURY accumulation affects the gastrointestinal, and renal systems. In this study, we aimed to study the physiological, and histological effects of mercury oxide on the liver and kidney in male Wistar rats. During 22 days, we divided 25 rats into 5 groups. The control group is placed first, followed by vinegar, low, medium, and high dose mercury groups. The control group was given only water. The vinegar-only group was given only vinegar. Mercury oxide-treated (HgO) group was given HgO 0.375 mg/kg/day. Mercury oxide treated group given HgO 1.5 mg/kg/day. Mercury oxide-treated (HgO) group was given HgO 4.5 mg/kg/day. We studied the levels of ALP, LDH, AST, ALT, albumin, creatinine, and urea. Histopathology of the liver and kidney were also studied. The result of this study was hepatic sinusoid dilation, renal tubule degeneration, and glomerulus shrinkage. This study showed non-significant differences among groups in terms of renal glomerulus diameter. The results showed that HgO at dose (1.5 mg/kg/day) had significantly higher levels of LDH, ALT, and AST enzymes when compared to the control group. While at the highest dose of mercury oxide (4.5 mg/kg/day), LDH, ALT, and AST enzyme levels decreased when compared to the lower doses. Our results showed a non-significant increase in urea level. Consequently, our investigation demonstrated that exposure to mercury oxide after therapy may result in toxicity to the kidneys and liver.

Keywords: Mercury oxide, Toxicity, Histopathology, Liver, Kidney.

Introduction

Mercury oxide (HgO) is an inorganic compound that consists mainly of one atom of oxygen (O) and one atom of mercury (Hg). In nature, there are two main forms of mercury oxide, red and yellow. The most important component of the red form of mercury oxide is mercury. Mercury oxide is a very toxic heavy metal [1, 2] and it is known to pose a critical environmental hazard [3, 4]. This compound has countless industrial applications. It is used in medical measurement instruments, pesticides, dyes and fertilizers [5], cosmetics, glass modifiers, antiseptic compounds, [6], and batteries production [7, 8, 9].

In the year 2013, an agreement was signed among 147 countries around the world to regulate mercury global releases. Despite this, globally, minor emissions and pollution of mercury take place [10]. Mercury has been shown to cause a genotoxicity effect, due to its ability to bind sulfhydryl groups [11]. In many countries, as a result of its serious toxicity, its usage has been restricted.

Mercury is much more widely distributed than other heavy metals because of its high mobility [12, 13]. However, its effects on human health and the environment have long been documented. Because of its extensive usage, it had a noteworthy influence on human health [11]. Many ways mercury-contaminated humans and one of the most important ways is via inhaling the vapor of mercury from gold mining, forest fires, and volcanic eruptions [14, 15, 16, 17]. Another way of mercury contamination is via consumption of contaminated fish [1, 2], which can cause life-threatening health problems, involving nephrotoxic [18], pneumotoxic [19, 20, 21], hepatotoxicity [19, 21, 22], cardiovascular and digestive systems toxicity [23, 25, 26, 27, 28]. Although, to date, there is doubt related to the relationship between cancer development and

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mercury exposure [11]. Mercury oxide like mercury, is extremely toxic and considered a worldwide transported pollutant. While there are many studies on mercury toxicity in human health, to our knowledge, there has been limited research on the *in vivo* effect of mercury oxide on the liver and kidney. So, the main aim of this study is to measure the physiological, and histopathology effects of different doses of mercury oxide on the liver and kidney of male albino rats.

Material and Methods

Animals

For this study, male healthy Wister rats (*Rattus norvegicus*) weighing 200 g – 300 g were used. Animals were kept under standard laboratory conditions of 12/12 hours dark/light cycle and room temperature (25 ^{o}C) with free access to water and food in clean cages in the Animal House of the University of Zakho.

Preparation of Mercury Oxide Doses

Mercury oxide was dissolved in vinegar. For preparation doses of mercury oxide, we dissolved, 0.1125 mg of mercury oxide in 0.5ml of vinegar to obtain the first dose. For the second dose, we dissolved 0.45 mg of mercury oxide in 0.5 ml vinegar. For the third dose, 1.35 mg of mercury oxide was dissolved in 0.5 ml vinegar.

Experimental Design

Each group with five rats a total of 25 rats were divided into 5 groups. Group 1 which is the untreated control group, was gavaged pure water. Group 2 which is the vinegar-treated group, was gavaged pure vinegar. Group 3, was gavaged the first dose of mercury oxide (0.375 mg/kg/day). Group 4, was gavaged the second dose of mercury oxide (1.5 mg/kg/day). Group 5, was gavaged the third dose of mercury oxide (4.5 mg/kg/day) for 22 days.

Physiological and Histopathological Studies

After obtaining approval from the University of Zakho Animal Research Ethics Committees. Chloroform was used to anesthetize all rats. All rats were dissected to take out directly 5 ml of fresh non-coagulated blood from the hearts by using 5 ml medical disposable syringes. Immediately, collected blood samples were divided into two types of blood tubes, EDTA and plane tube. Then, all collected blood samples were sent to the laboratory. At that moment, the liver and right kidneys of all rats were removed, cleaned with distilled water, and weighed. Subsequently, they fixed in 10% of neutral buffered formalin, ascending grade of ethyl alcohol was used to dehydrate kidneys, cleared in xylene, and fixed in paraffin wax. They sectioned at a size of 5 µm and hematoxylin and eosin stain were used to stain them. Sections were examined at 400x magnification using a compound microscope and a Dino-Eye microscopic camera was used to measure the diameter of 10 randomly chosen glomeruli in the cortex of examined kidneys of each rat. In the same way, the pathology of the liver was examined.

Statistical analysis

For statistical analysis, GraphPad Prism Version 9 software was used. One-way analysis of variance (ANOVA) and the Dunnett test were used to compare variances among groups. A $P\text{-}value \leq 0.05$ is considered significant among groups. Results are presented as means ± standard error [24].

Results

The Body Weight

Our results reveal that body weight rates of all treated groups of rats significantly decreased after 22 days of exposure to mercury oxide compared to the control group (Fig.1). I.e., when the rats were gavaged with mercury oxide at a dose of 1.5 mg/kg/day, the body weight rates decreased to 238.5 ± 9.8 g, compared to 287.7± 85.2 g for the control group.

The Enzymes Activities of the Kidneys

A minor increase occurred in urea levels in the kidneys of rats given large doses of mercury oxide compared to the control group (Table 1). In contrast, the levels of urea were decreased when low dosages of mercury oxide 0.375 mg/kg/day were given. Nevertheless, when compared to the higher dose and control, the changes are not significant. Compared to the control group, the creatinine levels remained fairly higher at various mercury oxide dosages. I.e., creatinine levels of the control group 0.3 ± 0.01 U/l were raised to 0.4 ± 0.051 U/l at dose 0.375 mg/kg/day and reached 0.35 ± 0.03 U/l at dose 4.5 mg/kg/day. That is, the changes are not significant.

The Enzymes Activities of the Livers

Our data suggest that there are significant differences in AST, ALT, ALP, and LDH, (*P-value* 0.0092, 0.0476, 0.0034, and 0.0194, respectively) levels between the control and mercury oxide-treated groups (Table 2). In contrast, our results show that there is a non-significant difference in GGT, albumin, and protein levels (*P-value* 0.121, 0.056, and 0.218 respectively) between the control and treated groups. When we compared the lower dose (0.375 mg/kg/day) to the higher dose (4.5 mg/kg/day) treated mercury oxide groups, the maximum dose showed a decrease in the AST, ALT, ALP, and LDH levels. In the mercury oxide treated (1.5 mg/kg/day) group, the AST, ALT, ALP, and LDH enzyme levels were significantly increased compared to the higher dose (4.5 mg/kg/day). Our data shows significant differences when we compared albumin levels of the control group, to the dose 1.5 mg/kg/day.

Histopathology of Kidneys

When the control and vinegar-treated kidneys of rats were examined (Figures 2 A and B), the renal glomeruli were found to have normal Bowman's space, normal capsules, and normal mesangial cells, proximal and distal convoluted tubules. Mercury oxide administration caused various observed effects on the renal morphology including, glomeruli size reduction and fragmentation into bifurcated components as well as renal tubules and glomeruli degeneration and blood extravasation in the medulla (Figures 2 C and D, and Figure 3). However, we did not observe any significant differences in the weights of kidneys and the diameter of glomeruli among the control and the experimental groups (Figure 4 and Figure 5).

Histopathology of Liver

The liver of the control and vinegar-treated groups showed a normal histological structure of the hepatocytic plate, hepatic sinusoid, and central vein (Figures 6 A and B). Although the hepatocyte structures are normal, mercury oxide administration in group 3 and group 4 resulted in noticeable alterations in the liver structure. These changes included the dilation of a hepatic sinusoid (Figures 6 C and D). We observed a significant difference in liver weights between the control and treated groups (Figure 7).

Discussion

Globally heavy metals are accumulated in the environment. Especially, highly toxic mercury. Its accumulation in the environment increased gradually because it was not banned from use in industries (29). Studies showed that humans around the world are exposed to mercury (30). In a study on animals, mercury exposure caused loss of appetite and severe weight loss. This harmful effect may inhibit several vital metabolic processes within the body and eventually may lead to delays in development and growth (31).

There is a strong link between environmental metal exposure and chronic kidney diseases. As kidneys are very susceptible to the toxic effects of metals (32). Mercury exposure may cause adverse effects, such as nephrotoxicity, hepatotoxicity, neurotoxicity, immunotoxicity, teratogenicity, and cardiovascular and endocrine toxicity (33-34).

To date, the exact mechanism by which mercury oxide may cause nephrotoxicity is not clear. However, studies indicate that the reactive oxygen species (ROS) act as an important kidney disease mediator. Metabolism of mercury oxide in cells produced ROS such as hydrogen peroxide (H2O2), singlet oxygen (1O2), hydroxyl radical (.OH), superoxide anion (O2.-), and peroxyl radicals (HOO. -). ROS toxicity is caused by antioxidant defense system disruption which can lead to damage to cellular DNA, proteins, and lipids (35).

According to studies heavy metals have serious side effects on mammalian organs (36). Our data indicated that there are significant increases in serum AST, ALT, ALP, and LDH levels. This data reinforces the data in the study done by Zaki *et al*. in 2011, which found significant increases in serum AST, ALT, and ALP levels in mercury oxide-treated catfish. The cytotoxic effects of mercury oxide may be attributed to glutathione, metallothionein, and protease activity alteration. Moreover, it is known that mercury oxide can produce ROS that can cause an increase in lipid peroxidation, which sequentially leads to a reduction of cell membrane integrity and eventually, cell death. Also, cell death may be caused by failure of DNA repair systems (37). Mercury oxide can disturb cellular growth, proliferation, and differentiation processes and may cause some enzymes to be inactivated, and others like caspase to be activated and may cause alterations in the ultrastructure of hepatocytes (33). Mercury oxide-induced apoptosis in the liver may be caused by epigenetic mechanisms (34). Additionally, mercury oxide-induced behavior changes may occur in the brain due to neurotransmitter modulation including serotonin and dopamine (38). Currently, we discussed some mechanisms of mercury oxide-induced toxicities, but many are still far from being clearly understood.

Conclusion

From the results we obtained from this study, we conclude that mercury oxide should be considered a major relevant risk factor for kidney and liver diseases. One potential limitation of our work as we only studied two organs, the kidney and liver of rats, and the other limitation was we only used biochemical and histological approaches. So, our recommendation for future work is to include different types of approaches for different types of organs of different types of animal models to better understand the exact mechanism (s) of mercury oxide toxicity.

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Declaration of Conflict of Interest

The authors declare that there is no conflict of interest.

Funding statement

This study didn't receive any funding support.

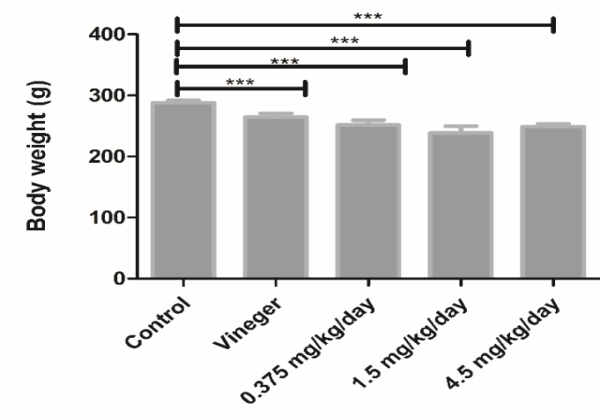

Fig. 1. Mercury oxide effects on the body weight.

TABLE 1. Effects of mercury oxide on kidney function markers (urea and creatinine) in Wistar rats

Kidney Function Test	Control	Vinegar	0.375 Mg	1.5 Mg	4.5 Mg	P-Value
Urea	43.1± 2.3	45.1 ± 1.8	39.7 ± 4.9	44.86 ± 2.12	45.6 ± 14.1	0.186
Creatinine	0.3 ± 0.01	0.4 ± 0.05	0.4± 0.05	0.4 ± 0.034	0.35 ± 0.03	0.104

TABLE 2. Effects of mercury oxide on liver function markers (AST, ALT, ALP, GGT, Albumin, Protein, and LDH) in Wistar rats

Function Liver Tests	**Control**	**Vinegar**	**0.375 Mg**	**1.5 Mg**	**4.5 Mg**	***P-Value***
AST	160.5±68.9	158.6 ± 21.2	215.5±58.1	248.4±49.2 *	123.1±35.3	0.0092**
ALT	56.1 ±21.9	65.1±10.6	63.0±8.2	77.7±25.2	42.9±3.6	0.0476*
ALP	426.4 ±154.4	435.6±147.7	240.0±56.9 *	350.7±103.9	149.0±32.6**	0.0034**
GGT	-1.4 ±1.2	-1.0±1.7	0.0±0.0	0.0±0.0	0.5±0.9	0.1213
Albumin	4.2 ± 1.2	3.9±0.3	4.0±0.1	3.3±0.3*	3.5±0.5	0.0562
Protein	6.7 ±2.1	6.7±0.3	6.6±0.5	6.4±0.3	6.2±0.2	0.2186
LDH	1350.2± 50.6	684.5±337.3	1052.6±505.4	1662.7±498.1	427.8±225.0 *	0.0194*

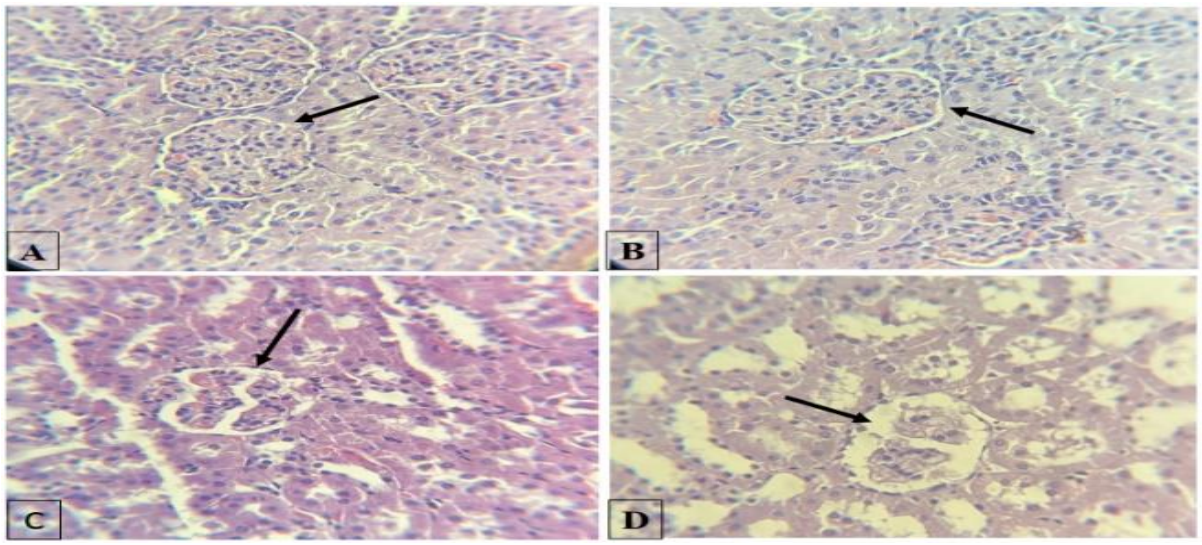

Fig. 2. Transverse section of kidneys showing: (A and B) normal architecture of renal corpuscle and renal tubules in control and vinegar groups respectively. (C and D) showing the breakdown of the glomerulus into two parts (arrow) in groups 4 and 5 respectively (A, B, C, and D 400x).

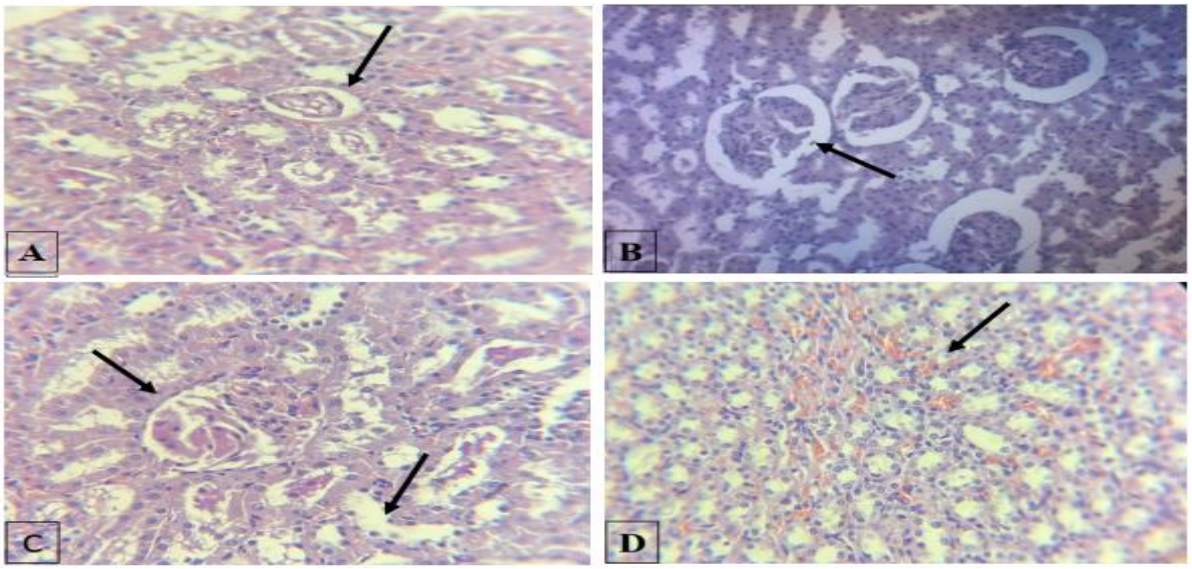

Fig 3. Transverse section of the kidney showing: (A and B) shrinkage of glomeruli in group 3 and group 4 respectively. (C) Reveals degeneration of glomeruli and renal tubules (arrow) in group 3. (D) Highlighting extravasation of blood in medulla group 3 (A 100x. B, C and D 400x).

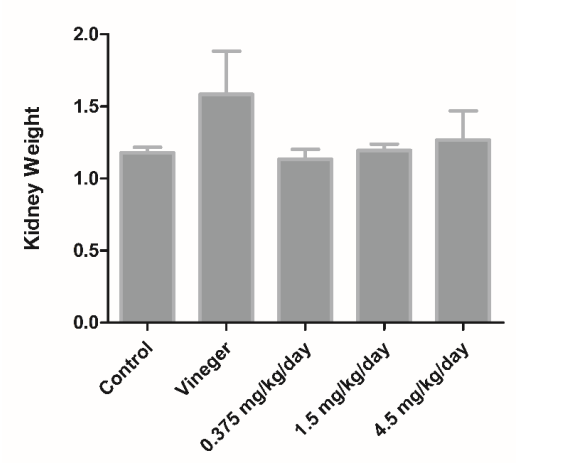

Fig. 4. Illustrates the weights of kidneys (g) in both the control and experimental groups

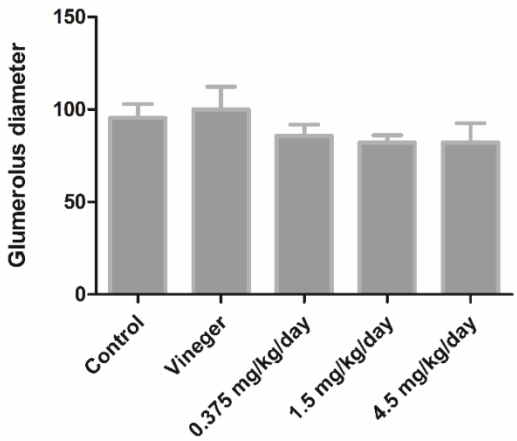

Fig. 5. Illustrates the diameter of glomeruli (µm) in both the control and experimental groups

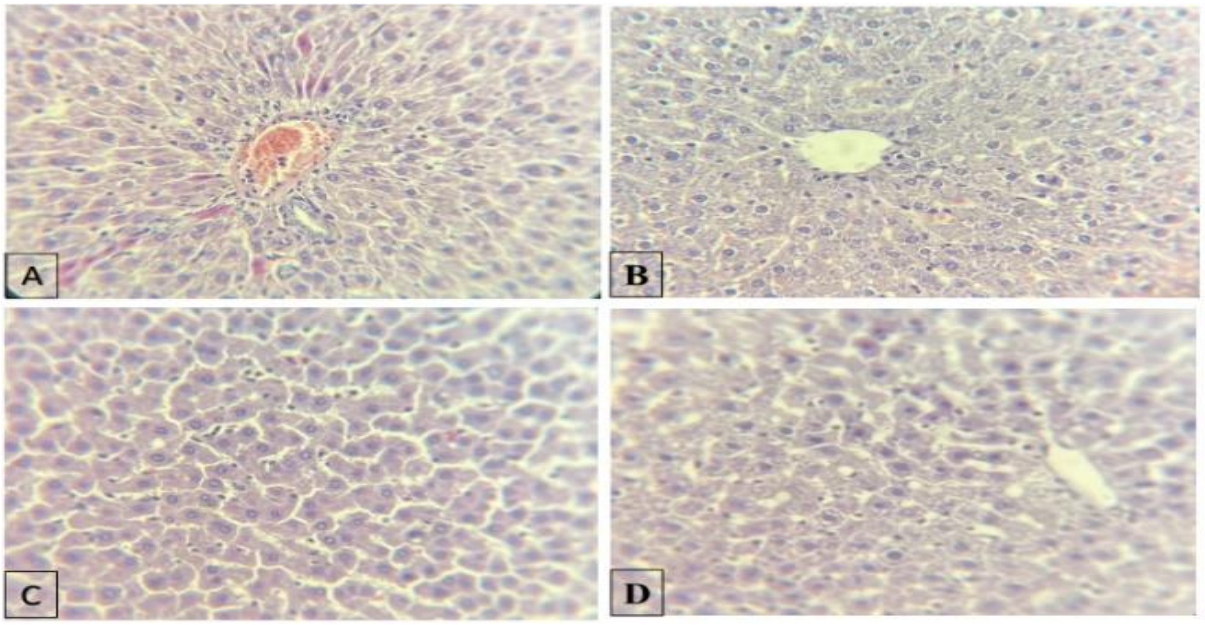

Fig. 6. Transverse section of the liver showing: (A and B) normal histological structures of the hepatocytic plate, hepatic sinusoid, and central vein in control and vinegar groups respectively. (C and D) dilation of a hepatic sinusoid with normal hepatocyte structure in groups 3 and 4 respectively (A, B, C, and D 400x).

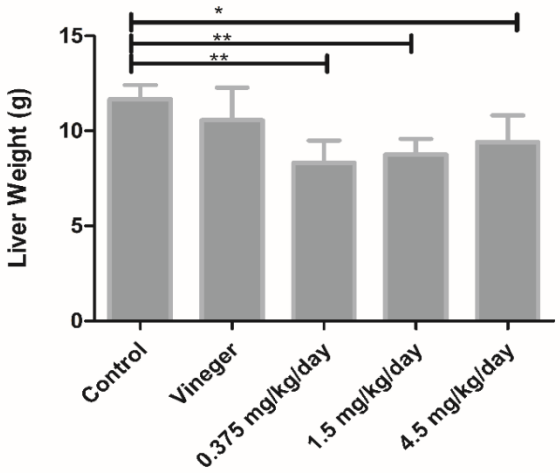

Fig. 7. Illustrates the liver weight (g) in both the control and experimental groups.

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دراسة في الجسم الحي للتأثيرات الفسيولوجية والنسيجية لجرعات مختلفة من أكسيد الزئبق على الكبد والكلى في ذكور جرذان ويستار

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[2] قسم الفسيولوجيا الطبية والصيدلة، كلية الطب، جامعة دهوك، دهوك، إقليم كردستان، العراق.

الملخص

يؤثر تراكم الزئبق على الجهاز الهضمي، والكلى. هدفنا في هذه الدراسة هي دراسة التأثيرات الفسيولوجية والنسيجية لأكسيد الزئبق على الكبد والكلى في ذكور جرذان ويستار. خلال 22 يومًا، قمنا بتقسيم 25 جرذن إلى 5 مجموعات. أعطيت المجموعة الضابطة الماء فقط. المجموعة التي تناولت الخل فقط أعطيت الخل فقط. أعطيت المجموعة المعالجة بأكسيد الزئبق 0.375 (HgO) ملجم/كجم/يوم من الزئبق. المجموعة المعالجة بأكسيد الزئبق تعطى 1.5 ملغم/كغم/يوم من الزئبق. أعطيت المجموعة المعالجة بأكسيد الزئبق 4.5 (HgO) ملجم/كجم/يوم من الزئبق. قمنا بقياس مستويات ALP، LDH، AST، ALT، الألبومين، الكرياتينين، واليوريا. كما تمت دراسة التشريح المرضي للكبد والكلى. لاحظنا تمدد الجيوب الكبدية ، و تلف النبيبات الكلوية، وانكماش الكبيبة. أظهرت هذه الدراسة اختلافات غير كبيرة بين المجموعات من حيث قطر الكبيبة الكلوية. أظهرت النتائج أن الزئبق بجرعة (1.5 ملغم/كغم/يوم) كان لديه مستويات أعلى بكثير من إنزيمات LDH، ALT، وAST بالمقارنة مع مجموعة السيطرة. أثناء تناول أعلى جرعة من أكسيد الزئبق (4.5 ملجم/كجم/يوم)، انخفضت مستويات إنزيم LDH وALT وAST بالمقارنة مع الجرعات الأقل. أظهرت نتائجنا زيادة غير مهمة في مستوى اليوريا. وبناء على ذلك، أظهرت هذه الدراسة أن تعرض لأكسيد الزئبق يمكن أن يسبب تسمم الكبد والكلى.

الكلمات الدالة: أكسيد الزئبق، علم وظائف الأعضاء، الأنسجة، الكبد، الكلى.

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