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Some Hepatotoxic Effects of Mercury Chloride on the Liver of Adult Wistar Rats

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Abstract

Mercury is sliver in colour, toxic metallic chemical element that is liquid at room temperature known to be a widespread environmental and industrial pollutant which induced several alternations in the tissues, and produces peripheral neuropathy in experimental animals and human beings. It finds its application in cosmetics manufacturing companies, dental filling, stabilizing agent. The present study investigated the possible adverse effect of mercury chloride on the liver of adult wistar rats. Thirty six adult wistar rates of both sexes weighing between 110 g-300 g were randomly grouped into four groups; group A,B,C,D with each group containing 9 rats and group. A served as the control group maintained daily with feed and water. The other groups B, C and D were given mercury chloride solution orally containing 0.2 g/kg, 0.4 g/kg and 0.5 g/kg body weight of mercury chloride respectively alongside the fed and water for 21 days and weighed weekly after which they were sacrificed using cervical dislocation. Blood was obtained through cardiac puncture for assay of hepatic markers; Alkaline Phosphatase (ALP), Asparatate Transaminase (AST) and Alanine Transaminase (ALT). then liver was removed, weighed and fixed in 10% formol saline and processed for histological studies using Haemotoxylin and Eosin (H and E) staining technique.

Results obtained show as significant ($P<0.05$) decrease in body weights of the treated groups B, C and D as compared with the control group A. There was no significant ($P>0.05$) decrease in organ weights of the wistar rats. Histological observation revealed well preserved histo-architecture in the control group A. however treated group B, C and D showed inflammation of cells, enlargement of the central vein, distorted sinusoids and hemorrhage around the central vein. Biochemical analysis for ALP showed a significant increase in the treated group B, C and D but statistically significant ($P<0.05$) in group C and D only as compared to control group A, AST showed a significant increase in all treated group A, C, and D and all showed statistically significant values ($P<0.05$) as compared to control group A and ALT showed a significant increase in all treated group B, C and D and all showed statistically significant values ($P<0.05$) as compared to control group A. The study then concluded that mercury chloride exposure induced hepatic damage with elevated haptic enzymes, this may ultimately impair hepatic functions in wistar rats.

Keywords: Liver • Mercury chloride • Hepatotoxicity • Hepatic damage • Hepatic enzymes

Introduction

There is an increasing concern that human activities play a major role in polluting the environment by toxic and carcinogenic metal compounds. There are evidences that these metals by accumulating contaminate water sources and food chain with their compounds. Hence, industrial pollution of the environment with metal compounds is becoming a serious problem. Unlike most organic pollutants, heavy metals are not degraded rather accumulate in the environment and food chain [1]. In fact, mercury has proven to be extremely toxic to mankind while their usage in various industries has increased rapidly in this century [2].

Mercury is a slivery colored, toxic metallic chemical element, liquid at room temperature with atomic number 80 and symbol Hg. It is a widespread environmental and industrial pollutant which induces several alterations in the tissues, and produces peripheral neuropathy in experimental animals and human beings. Its poisoning can result from inhalation, ingestion or absorption through the skin. Mercury's wide industry related effects on human and animal Bio systems are well documented by the World Health Organization and general contact with mercury has been shown to be aggravated by contaminated food and water [3,4]. Mercury, identified thousands of years ago is one of the oldest toxicants known [5]. Although in recent years, environmental and occupational exposures to mercury have been greatly reduced, this metal still remains a threat to human health from multiple sources: air, water and food [6]. Previous studies has shown that Mercury Chloride (HgCl) caused histopathological and ultrastructural lesions in the liver proven by periportal fatty degeneration and cell necrosis [7]. Recently, large populations are exposed to relatively low levels of mercury through the use of pesticides for agriculture and the use of fluorescent light bulbs. The liver is the largest single organ in the body next to the skin and it is the body's greatest chemical factory responsible for the breaking down of chemical substances in the body, hence, it is the major site of metabolism for Mercury and it can accumulate in the liver resulting in several hepatic damages [7]. The liver is located in the upper right hand portion of the abdominal cavity, beneath the diaphragm and on top of the stomach, right kidney and intestine. It is a dark reddish-brown organ, cone-like on shape and divided by fissures into four lobes; the right (the largest lobe), left, quadrate and caudate lobes. It weighs approximately 1.5 kg and accounts for approximately 2.5% of the adult body weight [8]. The liver is covered by a capsule (Glisson's capsule) made up of connective tissue. This connective tissue extends into the liver substance through the portal canals (mentioned above) where it surrounds the portal triads. The sinusoids are surrounded by reticular fibers. Connective tissue does not intervene between adjoining liver cells [8].

In the present study, the toxic effect of mercury chloride will be evaluated by assessing changes in the body weight, blood chemistry and histopathology of liver in adult wistar rats

The visceral surface of the liver is covered with peritoneum, except at the fossa for the gallbladder and the porta hepatis-a transverse fissure where the vessels (hepatic portal vein, hepatic artery, and lymphatic vessels), the hepatic nerve plexus, and hepatic ducts that supply and drain the liver enter and leave it. In contrast to the smooth diaphragmatic surface, the visceral surface bears multiple fissures and impressions from contact with other organs [8].

Two sagittally oriented fissures, linked centrally by the transverse porta

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hepatis, form the letter Hon the visceral surface. The right sagittal fissure is the continuous groove formed anteriorly by the fossa for the gallbladder and posteriorly by the groove for the vena cava. The umbilical (left sagittal) fissure is the continuous groove formed anteriorly by the fissure for the round ligament and posteriorly by the fissure for the ligamentum venosum. The round ligament of the liver is the fibrous remnant of the umbilical vein, which carried well-oxygenated and nutrient-rich blood from the placenta to the fetus. The round ligament and small paraumbilical veins course in the free edge of the falciform ligament. The ligamentum venosum is the fibrous remnant of the fetal ductus venosus, which shunted blood from the umbilical vein to the IVC, short-circuiting the liver [8].

The liver is invested by a delicate connective tissue capsule that is continuous with the peritoneum. The capsule contains numerous elastic fibers and is covered by a mesothelium except for a small bare area where the liver abuts the diaphragm.

The liver is composed of epithelial cells, the hepatocytes, arranged in branching and anastomosing plates separated by blood sinusoids. Both form a radial pattern about a central vein that is the smallest tributary of the hepatic vein. The spike like arrangement of hepatic plates about a central vein constitutes the basis of the classic hepatic lobule, which appears somewhat hexagonal in cross section, with a central vein at the center and portal areas at the corners. The liver consists of about 1 million such units. A portal area contains a branch of the portal vein, a branch of the hepatic artery, a bile duct, and a lymphatic channel [9]. All are enclosed in a common investment of connective tissue. Blood passes from small branches of the hepatic artery and portal vein into the sinusoids that lie between plates of hepatocytes. Blood flows slowly through the sinusoids toward the center of the lobule and exits through the central vein. Branches of the hepatic artery carry oxygenated blood and provide about 20% of the blood flow within hepatic sinusoids. In contrast, branches of the portal vein carry nutrient rich blood from the gastrointestinal tract and contribute the remaining 80% of the sinusoidal blood flow [9]. Hepatocytes nearest the branches of the portal vein and hepatic artery-that is, at the periphery of the lobule-receive blood with the highest nutrient and oxygen content. Both diminish as blood flows toward the central vein. Due to this arrangement, three zones can be recognized in a hepatic lobule according to the metabolic activity: a zone of permanent function (zone 1) at the periphery, a zone of intermittent activity (zone 2) near the center of the lobule, and a zone of permanent repose (zone 3) near the central vein. The boundaries of the hepatic lobule can be estimated by observing the central vein and noting the portal areas at the corners of the lobule [9]. Bile canaliculi represent an expansion of the intercellular space, and their walls are formed by the adjacent plasmalemma of two neighboring hepatocytes. Short microvilli extend from the cell membrane into the lumen. At the margins of the canaliculus, the plasma membranes are joined by occluding junctions similar to the zonula occludens of other epithelia. They form an occluding seal to prevent bile from escaping into the intercellular spaces between hepatocytes. Golgi complexes of hepatocytes often lie adjacent to the canaliculi. The bile canaliculi show intermittent contractions. The primary function of the liver is to store carbohydrate in form of glycogen. [9].

Alkaline Phosphatase (ALP); as its name suggest, it is an enzyme responsible for the removal of phosphate group (Dephosphorylation) from molecules, like nucleotides, proteins, and alkaloids [10]. In humans ALP is concentrated in the liver, bile ducts, kidney, bone and placenta. Diseases in afore mentioned areas leads to elevated levels of ALP [11]. Alanine Transaminase (ALT) is a transaminase enzyme associated especially with the liver and commonly used as part of diagnostic evaluation of hepatocellular injury to determine liver health. When used in diagnostics it is almost always measured in international units/liter. Significant elevated levels of ALT often suggests the existence of other medical problems such as viral hepatitis, liver damage, congestive heart failure, diabetes, bile duct problems, infectious mononucleosis or myopathy [12].

Aspartate Transaminase (AST) is an important enzyme in amino acid metabolism and it is found in organs such as liver, heart, skeletal muscle, kidney, brain and red blood cells. It is commonly measured clinically as a marker for liver health. AST is similar to ALT in that both enzymes are associated with liver parenchyma cells. It is raised in acute liver damage, but is also present in the red blood cells, cardiac and skeletal. The ratio of AST to ALT is sometimes useful in differentiating between the causes of liver damage [13]. The toxicity of mercury depends on its chemical form. Various mercury compounds have different toxicities depending on physical and chemical properties that affect absorption, distribution, tissue affinities and stability within the Bio systems. For instance, elemental mercury in the liquid state has unique toxic effects that differ from those of mercury vapor; likewise, organic mercury molecules are toxicologically different from inorganic forms [14].

A decrease in body weight gain, alteration in normal level of various blood chemical parameters accompanied by histopathological necrosis and degenerative changes in the liver tissue were observed as rats were orally exposed to mercury chloride [15].

Liver injury mercury exposure is well established by the elevated levels of serum hepatic marker enzymes indicating the cellular leakage and loss of functional integrity of hepatic membrane architecture. High levels of Aspartate Transaminase (AST) and Alanine Transaminase (ALT) are the crucial parameters to detect liver damage [16].

HgCl2 can inactivate a number of enzymes by blocking the functional sites through binding to sulfhydryl groups, which are part of catalytic or binding domains [17].

Materials and Methods

Terminology of Neuromorphic computing

Thirty-six (36) adult, healthy Wister rats weighing between 110-300 g of either sex were used for the experimental design. They were housed in standard plastic cages and fed with rat chow daily. All the rats were carefully and routinely screened, inspected and confirmed to be healthy during the period of acclimatization.

During acclimatization which lasted for 2 weeks, the animals were fed, monitored and weighed with a weighing scale and the value recorded every week till the end of administration so as to monitor and study the effect of the administration of mercury chloride on the weight of adult wistar rat. The preliminary study of acclimatization and actual animal experiments lasted for six weeks. However, they were acclimatized for 2 weeks and the actual administration of mercury chloride lasted for four weeks.

The study was carried out in a serene and conducive ventilated room of the animal house of the Department of Human Anatomy, Ladoke Akintola University of Technology, Ogbomoso, Oyo State, Nigeria and treated in accordance to the 'Guide for the care and use of animals' prepared and compiled by the National Academy of Science and Published by the National institute of Health 1985.

Reagents

The reagents used are

1. Mercury Chloride obtained from the department of Biochemistry, Ladoke Akintola University of Technology, Ogbomoso.

2. Distilled water obtained from the Department of Food Science and Engineering, Ladoke Akintola University of Technology, Ogbomoso.

3. Formol saline obtained from the Histology Lab, Department of Anatomy, Ladoke Akintola University of Technology, and Ogbomoso.

The rats were separated randomly into four groups of nine animals each. The groups are as follows;

Group A: this group was the control group with nine Wister rats of either

sex and was fed food and water only.

Group B: this group has nine Wistar rats of both sexes and in addition to their food and water; 2 mls of mercury chloride solution containing 0.02 g/kg of mercury chloride was administered for the duration of 28 days.

Group C: this group has nine Wistar rats of either sex and also in addition to the food and water given, 2 mls of mercury chloride containing 0.04 g/kg of mercury chloride administered for the duration 28 days.

Group D: this group also has nine Wistar rats of both sexes and in addition to the food and water that was given 0.05 g/kg of mercury chloride was administered for the duration 28 days.

Mercury chloride was administered orally, using an oral canula every morning between the hours 7 am and 8 am for 28 days. The animals were sacrificed after the last dose of mercury chloride administration by cervical dislocation. Blood was collected from the heart for biochemical analysis and the organ (liver) was harvested immediately and weighed using a sensitive balance. The Liver from each of the group was fixed separately in 10% formol saline for histological analysis.

Statistical analysis

Data was express as mean SEM. The difference was compared for statistical analysis by student's test. Descriptive and inferential statistics was applied to the results. All statistical analysis was performed using SPSS version 6.0.

Results

In Table 1, a significant decrease in body weights of the rats were observed in all treated groups (B,C,D) but statistically significant (P<0.05) in group C and D, as compared to the control group A after the administration of mercury chloride to wistar rats. Significant decrease in body weight was observed in the treated groups. Data were expressed as mean ± SEM (Table 1).

Table 1. Showing mean ± S.E.M of the body weights of wistar rats before and after mercury chloride administration.

Groups	Initial	Final
A	230.9 ± 9.11	209.8 ± 13.90
B	252.7 ± 12.09	200.9 ± 10.17
C	194.0 ± 8.91*	169.7 ± 7.92*
D	173.3 ± 15.95**	138.9 ± 5.50***

Note: P<0.05, values greater than 0.05 were considered insignificant while values less than 0.05 were considered significant (*). Values were expressed as mean ± Standard error of mean.

The organ weights of the liver increased slightly in groups B and C treated with 0.02 g/kg and 0.04 g/kg of mercury chloride respectively and a slight decreased was observed in group D treated with 0.05 g/kg of mercury chloride after administration all with no statistical significance (P>0.05) as compared with the control group A (Table 2) (Figure 1).

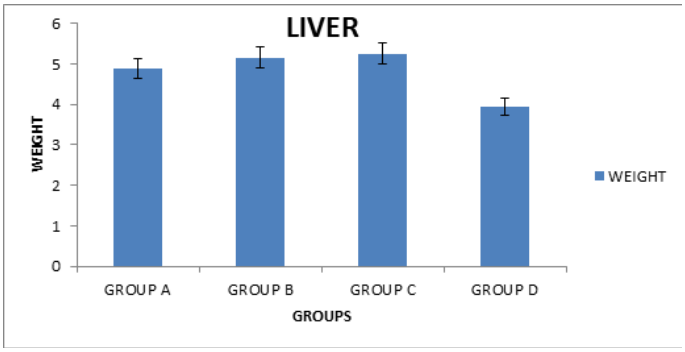

Figure 1. Histogram showing the organ weights of the liver of wistar rats after mercury chloride administration. Data were expressed in Mean ± SEM.

Table 2. Showing mean organ weights (g) ± SEM and relative organ weight after mercury chloride administration.

Groups	Weights (g)	Relative organ weights (%)
A	4.87 ± 0.41	2.32
B	5.15 ± 0.32	2.56
C	5.25 ± 0.31	3.1
D	3.94 ± 0.22	2.84

Biochemical analysis

The table shows the effects of mercury chloride on the levels of liver enzyme markers (AST, ALP and ALT) (Table 3).

Groups	ALP	AST	ALT
A	30.70 ± 2.00	63.27 ± 4.96	25.00 ± 1.64
B	41.99 ± 10.75	84.65 ± 2.82*	32.24 ± 2.21*
C	39.97 ± 1.01*	93.12 ± 3.29*	38.77 ± 2.32*
D	40.32 ± 0.75*	115.4 ± 2.75*	33.33 ± 0.51*

Note: P<0.05, values greater than 0.05 were considered insignificant while values less than 0.05 were considered significant (*). Values are expressed as mean ± Standard error of mean.

Abbreviations: ALP: Alkaline Phosphatase; AST: Asparatate Transaminase; ALT: Alanine Transaminase.

Alkaline Phosphate (ALP): A significant increase in all treated groups B, C and D was observed, but statistically significant (P<0.05) in groups C and D only as compared to control group A.

Aspartate Transaminase (AST): A significant increase was observed in all treated groups B, C and D, and all showed statistically significant (P<0.05) as compared to control group A.

Alanine Transaminase (ALT): A significant increase in all treated groups B, C and D were also observed, and all showed statistically significant (P<0.05) as compared to control group A (Figure 2).

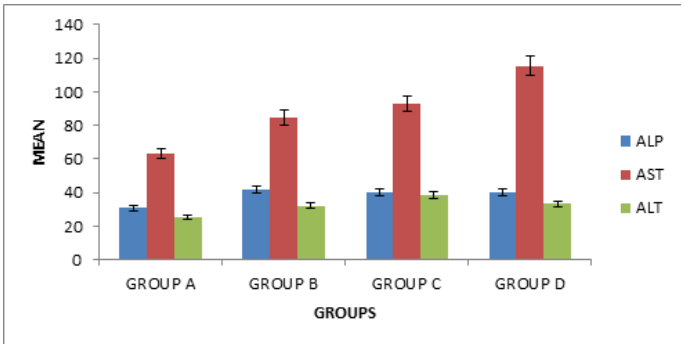

Figure 2. Showing the effect of mercury chloride administration on the levels of ALP, AST and ALT of wistar rats. Note: Significant increase (P<0.05) in levels of ALP, AST and ALT was observed and data were expressed as mean ± SEM.

ALT of wistar rats. Significant increase (P<0.05) in levels of ALP, AST and ALT was observed and data were expressed as mean ± SEM.

Histological observation

Photomicrograph of a normal histology of the liver of the control group showed normal histological features (control) group A (Figures 3 and 4).

Photomicrograph showing a section of a liver of group B treated with 0.2 g/kg body weight of mercury chloride for 21 days with enlarged and inflamed central vein (Red arrow) (Figure 5).

Photomicrograph showing a section of a liver of group B treated with 0.2 g/kg body weight of mercury chloride for 21 days with enlarged central vein (Red arrow) and distorted sinusoids and fibrosis around the Portal Triad (PT) comprising the Hepatic Vein (HV), Hepatic Artery (HA) and the Bile Duct (Bd) (Figure 6).

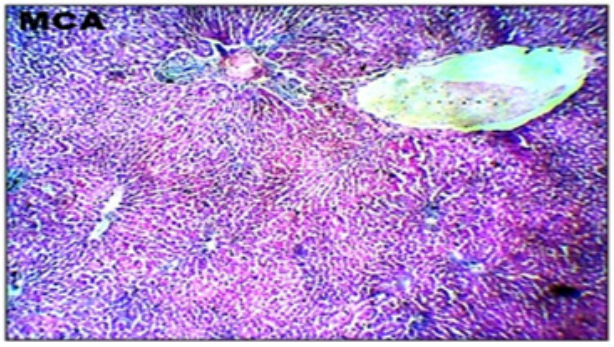

Figure 3. Plate 1a (H and E X40)

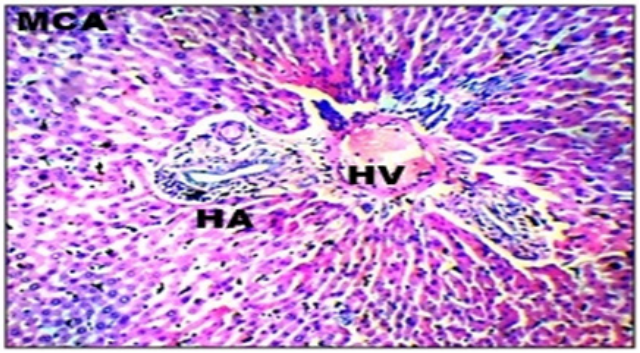

Figure 4. Plate 1b (H and E X100)

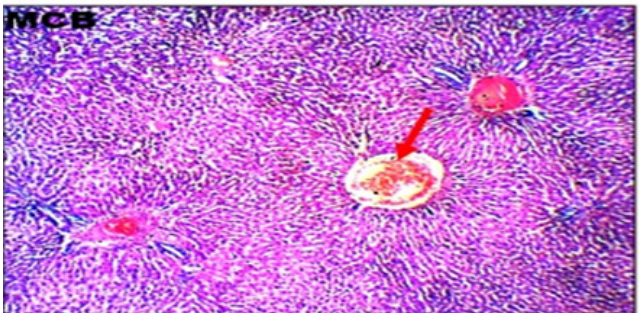

Figure 5. Plate 2a (H and E X40).

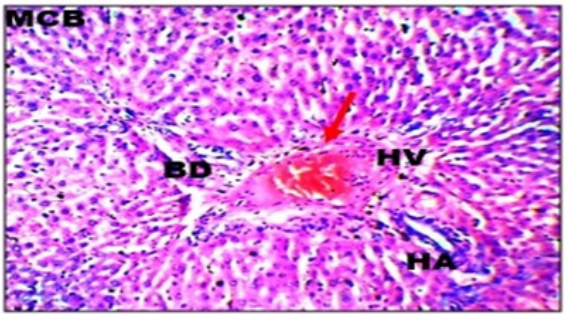

Figure 6. Plate 2b (H and E X100).

Photomicrograph section of a liver of group C treated with 0.4 g/kg body weight of mercury chloride for 21 days revealed inflammation of cells (Red arrow) (Figure 7).

Photomicrograph showed a section of a liver of group C treated with 0.4 g/kg body weight of mercury chloride for 21 days showing inflammation of the central vein and hepatic vein (Red Arrows) (Figure 8).

Photomicrograph section of a liver of group D treated with 0.5 g/kg body weight of mercury chloride for 21 days showed haemorrhage around the enlarged and elongated central vein (Red arrow) and distorted sinusoids (Figure 9).

Photomicrograph section of a liver of group D treated with 0.5 g/kg body weight of mercury chloride for 21 days showed severe hemorrhage and presence of inflammatory red cells as well as fibrosis within and around the portal triad and hepatic sinusoids with elongation of the central vein (Red arrow) (Figure 10).

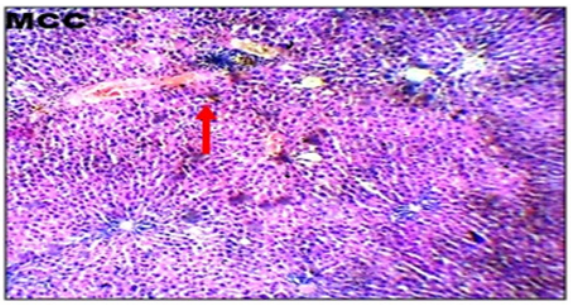

Figure 7. Plate 3a (H and E X40).

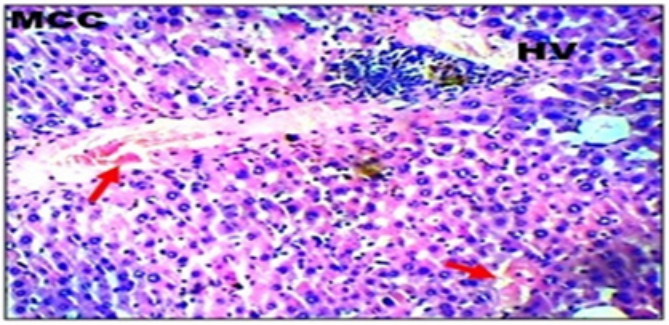

Figure 8. Plate 3b (H and E X100).

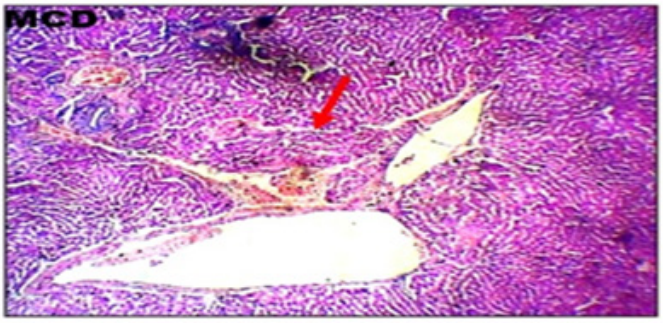

Figure 9. Plate 4a (H and E X40).

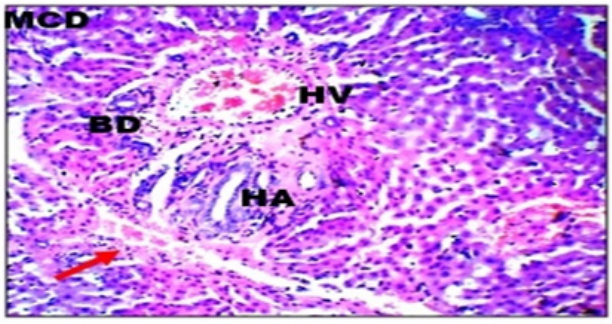

Figure 10. Plate 4b (H and E X100).

Discussion

The present investigation was carried out to determine the effect of daily administration of mercury chloride on the liver of wistar rats. The effect was measured in three ways: on the structure and organ weight of the liver, on the general body weights of wistar rats and on the enzyme activities of the liver. Indeed, the investigation revealed that continuous oral administration of mercury chloride for 21 days on the histology, body weights, organ weights and biochemical parameters by which hepatotoxicity were evaluated.

There was observed body weight loss in the rats during and after mercury chloride administration. The body weights decreased significantly (P<0.05) in the treated groups as compared to the control group during and after mercury chloride exposure and this is consistent with the report given by [15,18,19]. Weight gain depends on availability of nutrients. Therefore, the observed reduction in weight could be due to the decrease in food intake, or due to the overall increased degeneration of lipids and proteins as a result of Mercury chloride toxicity [19].

There was no significant increase (P>0.05) in the organ weights of the treated rats as compared with the control.

The activities of enzymes AST, ALP and ALT in serum were used routinely to assess the functiomal status and hepatic damage of the liver in both clinical and experimental settings. They are useful biomarkers of liver injury in a patient with some degree of intact liver function [10]. These tissue injuries caused functional impairment as evidenced in elevated serum of AST, ALT and ALP activities demonstrated the severity of Mercury (Hg) induced tissue damage [20].

In this present study, we observed elevated levels of AST, ALP and ALT with statistical significance (P<0.05) after daily administration mercury chloride for 21 days. ALP is a well-known indicator of multiple toxicity cases, including those related to hepatic and renal dysfunctions. The enzymatic parameter is thought to be one of the most sensitive markers of Hg toxicity [21]. AST was found to be the most conspicuously increased in the treated group as compared to the control group. The increase in these enzymes may be due to cellular necrosis of hepatocytes, which caused increased permeability of cell resulting in the release of transaminases in the blood stream as reported by previous authors [21-23]. The elevation of AST following mercury chloride exposure was also reported by [20].

The photomicrographs plate of Treated Group A showed normal liver architecture, central veins are not congested or elongated, normal arrangement of sinusoids and no inflammation of hepatocytes [15,24].

However, photomicrograph of the treated groups shows abnormal presentation of the liver structure, hepatic cells across the treated groups showed areas with severe to mild altered histomorphological changes characterized by loss of liver parenchyma, disorganization hepatocellular profile, cell death, dilation of the central vein, severe hemorrhage and presence of inflammatory red cells as well as fibrosis within and around the central vein and hepatic sinusoids also reported conspicuous damage and degenerative and necrotic changes in every area of the mercury exposed liver tissue of wistar rats [25-46].

Conclusion

This study concluded that that mercury chloride induced hepatic damage indicated by distoted hepatic histo-architecture coupled with elevated hepatic enzymes in the mercury-treated rats investigated. The findings from this study may impair hepatic functions; we recommend further studies to corroborate this report.

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