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Histopathological Effect of Arsenic in Drinking Water on Liver and Kidney of Albino Rat: A Light Microscopic Study

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ABSTRACT

Background and aim: Arsenic is a common pollutant of water in developing counties, leads to major health problems, and affects all body organs. The current study aimed to evaluate the histopathological effects of arsenic on the liver and kidney of albino rats.

Materials and methods: A total of 18 Wistar rats were divided into 3 groups (n = 6). The control group received only distilled water, ARS 50 Group received 50ppm of arsenic in drinking water as a daily oral dose for 4 weeks, and ARS 100 group received 100ppm of arsenic in drinking water as a daily oral dose, each day for 4 weeks. After experimentation, the rats were euthanized and tissues procured, treated, slides were prepared, and observational analysis was done.

Results: The liver of rats exposed to low dose showed mild central venous dilatation and congestion, portal haemorrhage, and dilated portal vein. However, the liver of rats exposed to higher doses revealed distortion of tissue architecture, haemorrhage, necrosis, vacuolated cytoplasm, mononuclear infiltrate. These changes were more pronounced in the high dose group, and rats in the control group showed normal tissue architecture. The kidneys of rats exposed to arsenic revealed disrupted architecture, increased or decreased periglomerular space, eosinophilic casts, and mononuclear infiltrate in a dose-dependent manner compared to the control group, which revealed normal renal function architecture.

Conclusion: According to the research results, even smaller doses of 50ppm of arsenic potentially induce pathological effects on the liver and kidney. Hence, the amount of arsenic should be checked in drinking water.

1. Introduction

Heavy metals are inextricably linked to the earth's crust and cannot be degraded or destroyed. Because of their persistent nature, toxicity and tendency to accumulate in organisms and cause food chain amplification are severe biological poisons.[1] Among these metals, arsenic is the most abundant pollutant and a potential human carcinogen with a complex metabolism.[2]Arsenic is one of the most toxic metals found in nature. Natural geological sources of arsenic contamination of drinking water, rather than mining, smelting, or agricultural sources, is the leading cause of human arsenic toxicity (pesticides or fertilizers).[3] In the Indian subcontinent, arsenic contamination of drinking water has become a significant problem.[4] Humans consume arsenic daily, food, water, soil, and air. Skin cancer has been linked to chronic inorganic arsenic exposure, which occurs naturally as a contaminant in drinking water.[5] Other serious health problems include diabetes, liver, kidney, and CNS disorders.[6] It has a slew of other toxic effects.[7] Arsenic accumulates in the liver, kidney, heart, and lungs after chronic arsenic ingestion, with low muscles, neurological system, gastrointestinal tracts, and spleen.[8] Long-term arsenic toxicity causes multisystem disease, with malignancy being the most serious consequence. Individuals, population groups, and geographical areas have different clinical features of arsenic toxicity. It is unclear what factors determine the incidence of a certain clinical manifestation or which body system is targeted. As a result, a wide range of clinical characteristics is typical in people exposed to chronic arsenic poisoning. With symptoms such as abdominal pain, diarrhoea, and a sore throat, the onset is insidious as well as dermatological manifestations.[9]Arsenic usually does not spare any organ in the body. A relationship between chronic arsenic exposure and liver disease and kidney failure has been found in several studies. The relationship between chronic arsenic exposure and the development of specific target organ toxicity, on the other hand, is not universally accepted. Arsenic toxicity causes the greatest human tragedy in developing countries. Many problems must be resolved to address the increasing problem of arsenic contamination and disease. Information is required to determine a threshold for carcinogenic effects to manifest and to define the dose and duration of exposure.[10] Organ-specific

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histology evaluation is now the gold standard for determining the degree of organ injury during chronic heavy metal exposure. As a result, the goal of this study was to design how chronic arsenic exposure affected the histology of some tissue architecture in adult Wistar rats. A better understanding of the effects of arsenic on target organs, focusing on tissue architecture at critical sites, will help define the mechanism(s) of arsenic-induced toxicity in mammals and reduce the level of uncertainty in risk assessment for this metalloid.

2. Materials and methods

This study was carried out with the approval of the ethics committee IAEC/2019/09 and Registration No. 635GORes of Government Medical College's Central Animal House in Jammu relevant officials at this University.

Grouping of animals

For the study, 18 healthy Wistar Albino rats were used, both sexes, weighing between 125 and 160 grams. The rats were procured from the Government Medical College's Central Animal House in Jammu. After a week of acclimatization to laboratory conditions, the rats were randomly assigned to one of three groups: control (C), low dose test (ARS 50), or high dose test (ARS 100), using a block permuted randomization strategy. Each group's rats were given an identification number. The animals were housed in polypropylene cages (4 per cage) with dust-free rice husk as a bedding material under laboratory conditions with control environment of temperature 18 to 29ºC, humidity (30% to 70%) and 12h light/dark cycle (16.00-18.00) as per Committee for Control and Supervision of Experiments on Animals (CPCSEA), India, guidelines which are in accordance with the internationally accepted principles for laboratory animal use and care. Throughout experimentation, the animals were provided standard rodent chow/feed and water ad-libitum. The animals were fasted overnight and weighed before the experiment's initiation using an electronic weighing scale. Arsenic was purchased from Avi Chemicals, New Delhi, under the trade name Arsenic Trioxide.

Experimentation

The rats in the Control Group were given only distilled water and served as a healthy control group. ARS 50 group rats served as the low dose group and were administered 50ppm (50mg/kg) of sodium arsenite in distilled water[13] for 4 weeks and were followed for 28 days. ARS 100 Group rats served as high dose group and were administered 100ppm (100mg/kg) of sodium arsenite[13] in distilled water for 4 weeks and followed for 28 days. Sodium Arsenite solution was prepared freshly every time by dissolving the measured quantity of sodium arsenite in 5 ml of distilled water to obtain desired concentration which was given by gavage feeding in 3-4 divided doses throughout the day. All the animals were observed daily for any physical or behavioral changes throughout the experiment. After the study period was completed, the animals were euthanized 48 hours after the last dose was administered, and tissues from each rat were collected.

Analysis

A naked eye examination was done to see any external changes. After that, the tissues were histologically processed to make 5-6µ thick sections stained with Hematoxylin and Eosin and examined under a microscope by a blind observer. A light microscope was used to analyze at 100X and 400X powers.

3. Results

Microscopic changes of liver

Light microscopic examination of liver sections from Control group rats revealed hexagonal classic hepatic lobules with radiating hepatic cords, central veins in the lobule's center, and portal areas containing portal triad formed by the portal vein, hepatic arteriole, and bile ductule surrounded by connective tissue at 3 to 5 corners of the lobule (Fig. 1). The central veins of the Classical hepatic lobule were found in the lobule's center and had a thin connective tissue wall lined internally by endothelial cells. Hepatocyte cords radiated from the central veins (Fig. 1) at the lobule's periphery, which included the portal areas, and were one cell thick in most places. On the contrary, the liver examination of ARS 50 group rats revealed preservation of basic architectural structure with few histopathological changes in central venous dilatation (Fig. 2) and portal venous dilatation and congestion with few inflammatory cell aggregations (Fig. 3). However, liver tissue of ARS 100 group rats expressed marked histopathological changes in disruption of basic hepatic tissue architecture, markedly dilated central vein, and central venous haemorrhage with focal areas of necroinflammation mononuclear cell infiltrates and evidences of ballooning degeneration (Fig. 4).

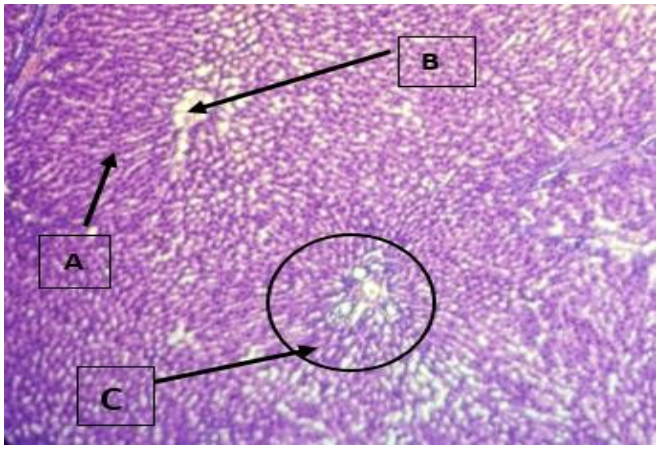

Fig 1. Photomicrograph Of rat liver control group showing radiating cords of hepatocytes (A), central vein (B), and portal triad (C) [control group 10X].

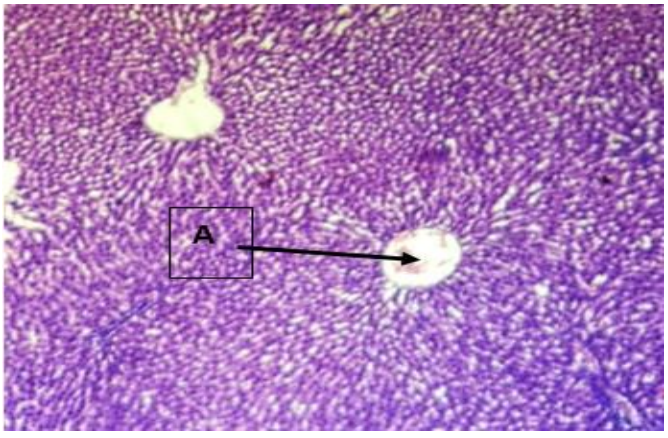

Fig. 2. Photomicrograph Of rat liver showing dilated central vein (A) [ARS 50 group10X].

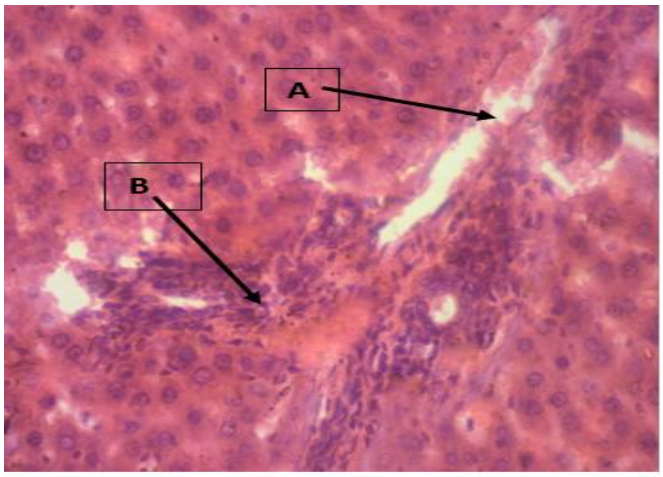

Fig. 3. Photomicrograph of rat liver showing dilated and congested portal vein (A) and inflammatory infiltrates (B) [ARS 50 group 100X].

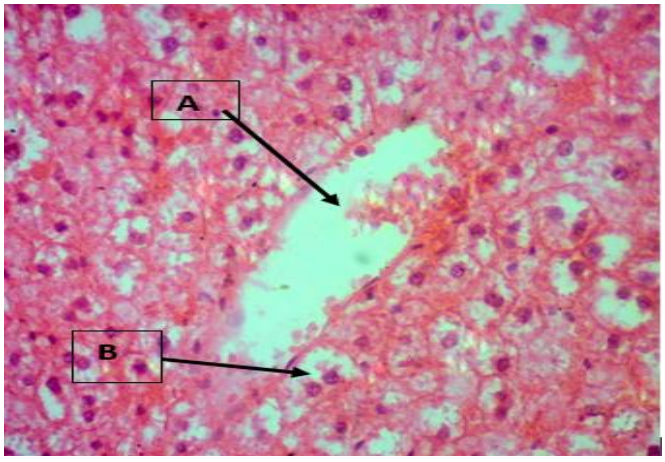

Fig. 4. Photomicrograph of rat liver showing markedly dilated central vein with hemorrhage (A) and balloon degeneration of hepatocytes (B) [ARS 100 group 100X].

Microscopic changes of kidney

The cortex and medulla of the Control group rats' kidneys were normal. The cortex comprised glomeruli, proximal convoluted tubules, distal convoluted tubules, and interlobular blood vessels. Bowman's capsule was shown to contain glomeruli in renal corpuscles. The proximal convoluted tubules outnumbered the distal convoluted tubules (Fig. 5). The kidney sections of ARS 50 group rats revealed that the basic architecture of the cortex and medulla of the kidney was preserved. Diffuse inflammatory cells in the interstitium and interstitial hemorrhage were seen. A decrease in the size of the glomerulus and increased peri-glomerular space was seen(Fig. 6). PCT and DCT appeared normal with no significant changes in the medulla. Further, the kidney sections of ARS 100 group rats revealed marked disruption of the tissue architecture along with extensive interstitial hemorrhage and vascular congestion. In some renal corpuscles, there was shrinkage of glomerular tuft and marked increase in the bowman's space, whereas some of the glomeruli appeared normal or with reduced glomerular space(Fig. 7). The lumen of the PCT and DCT contained eosinophilic material, and cellular cast (cellular fragments), and diffuse inflammatory infiltrates(Fig. 8).

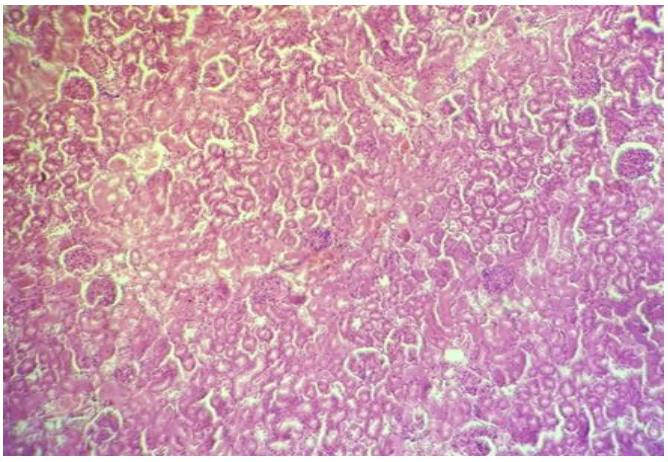

Fig. 5. Photomicrograph of rat kidney showing glomeruli (A) and tubules (B) [Control Group 10 X].

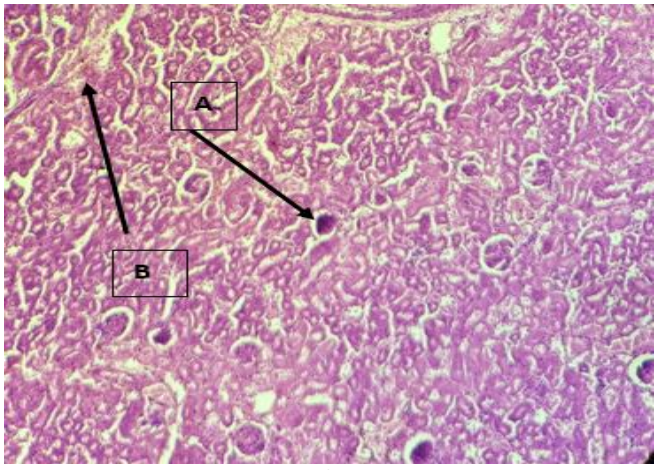

Fig. 6. The photomicrograph of the rat kidney shows a decrease in glomerular size and peri-glomerular space (A) and interstitial hemorrhage [ARS 50 Group 10 X].

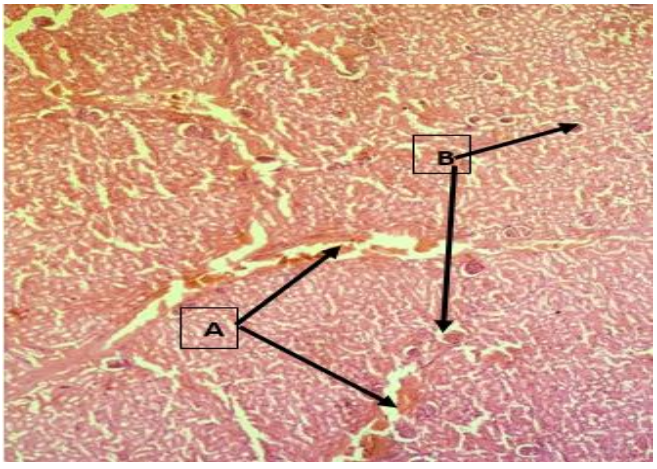

Fig. 7. Photomicrograph of rat kidney showing Extensive interstitial hemorrhage (A) and a marked increase in peri-glomerular space(B) [ARS 100 group 10X].

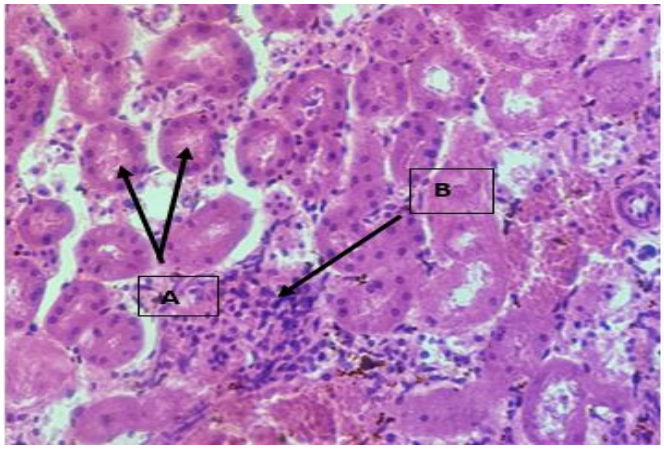

Fig. 8. Photomicrograph of rat kidney showing eosinophilic material in the lumen of convoluted Tubules (A) and diffuse inflammatory infiltrates (B)[ARS 100 group 100X].

4. Discussion

This study focused on depicting the effects of arsenic in drinking water. Even in small doses of 50ppm, the arsenic intake displayed pathological changes in the liver in mild dilatation and congestion of the central vein. Such observations are well documented in various studies of the past.[11, 12] The finding of inflammatory cells was positively supported by past literature, thus emphasizing that arsenic intake is associated with inflammation.[13] Furthermore, it was observed that the high dose group liver tissues displayed necrosis of the liver with tissue disruption and loss of hepatic architecture and normal radiating pattern of hepatic cords, which was also evidenced positively by previously indexed studies.[14, 15]Other changes like mononuclear cells of inflammation seen around the portal vein, sinusoidal dilatation, and areas of tissue haemorrhage were also observed in the test group rats were also in concordance with various studies done in the past.[11, 13, 16] The observations of the present study depicted that kidneys of rats of the test group revealed varied sizes of the glomerulus, i.e., both large and small glomerulus, with either decrease or increase in the periglomerular space, which was more pronounced in the more pronounced high dose group, vascular congestion, inflammatory infiltrate and severe hemorrhage. These changes were positively supported by evidence derived from previous literature.[13, 17]

5. Conclusion

The observations of the study mentioned above hence concluded that arsenic which is present in high concentrations in pesticides and insecticides, forms a contaminant of rivers and other water bodies from where it enters the food chain and even in a small quantity of 50 ppm; can adversely affect the major organs concerned with detoxification of toxins, i.e., liver and kidneys by inducing histopathological changes; thus acting as a cause for major health problems. Henceforth, steps should be undertaken to check the contamination of drinking water with arsenic.

Conflict of Interest

The authors declared that there is no conflict of interest.

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