Histological Study for Some Organ of Albino Mice Exposed to Heavy metals Copper Sulphate and Lead Nitrate

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Abstract

The present study was carried out the histological changes induced by lead nitrate [Pb (NO3)2] and Copper Sulphate (CuSO4.5H2O) toxicity in 48 mature Albino Mice were divided into 7 groups each with 5 mice. Group 1 served as control received tap water, group 2, 3, 4 received (2,4,6 mg /kg of body weight) from lead nitrate, while group 5, 6, 7 received (50,100,150 mg /kg of body weight) from Copper Sulphate respectively in drinking water for 6 weeks. After end of treatment with heavy metals the mice were sacrificed. The lesions were characterized by degeneration, necrosis, and fibrosis, cellular and vascular changes in the heart, lung and spleen. The intensity and distribution of such lesions found more severe in mice treated with highest dose from both elements.

Key words: Lead, Copper, Histological changes, lung, heart and spleen.

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INTRODUCTION

Heavy metals are chemical elements occur naturally in the environment with specific gravity that is at least five times the specific gravity of water (1). Some metals are no degradable and their presence in food chain may be the main reason for bioaccumulation in different organs (2). Among heavy metals lead is widespread and insidious environmental toxins and is known as sever and aggressive contaminant on human and animals due to their vast natural availability and widespread industrial and daily usage (3, 4). Lead can induced many biochemical and physiological dysfunctions by affecting on many organs such as kidney, liver, spleen and testes (5). Many Previous studies have established the effects of lead exposure on urinary systems and gastrointestinal (6). It also can causes hematological disorders when interfere with heme synthesis, lead can be encephalopathy immunosuppression, mutagenic, teratogenic and carcinogenic effects (1). Copper is an essential trace element for normal growth and metabolism to maintain the functioning of living organisms by many vital roles in the cell and function as cofactors for over 30 different enzymes such as peroxidases, catalase and cytochrome oxidase (7). Deficiency of this element can causes many physiological disturbances such as depressed in reproductive function and growth, decrease in some organs weight, anemia, cardiac and vascular disorders, new born ataxia and bowing of legs, while increased levels of copper become toxic to lives (8). High concentrations of Cu may cause increased oxidative damage to lipids, proteins, and DNA (9). Different research of patients with Wilson's disease and some genetic defect resulted from copper accumulation in the tissues (7). Study in male rats copper ingested found harmfully effect on sperm quality and weight of testis (10). Pathological study of toxic copper for different organ includes liver, kidney, lunge, spleen and intestine (11).

MATERIAL AND METHODS

Animals and doses preparation: Healthy 35 mature Albino Mice were used in this study. Animals were bred in the animal house of Pharmacy College, Karbala University. The mice

were housed in plastic cages measuring 30×12×11 cm. The experiment starts from February to March 2016. Thirty five mature mice (20-25g) (10 weeks old) were divided randomly into 7 groups each with 5 mice. Group 1 served as control received tap water, group 2, 3, 4 received (2,4,6 mg /kg of body weight) from lead nitrate[Pb (NO3)2], while group 5, 6, 7 received (50,100,150 mg /kg of body weight) from Copper Sulphate (CuSO4.5H2O) respectively, all groups received doses orally for 6 weeks, these doses are calculated according to the body weights by methods of (12).

Histological study: at the end of experiment the animals were sacrificed lungs, hearts and spleen organs of the control and treated groups was removed, they were dissected and washed then kept in 10% formalin immediately for 24 hour. The samples were processed for routine histological evaluation by (13).

RESULTS

The histopathological study in control group showed the lung have normal intact tissue and the bronchiole lined by pseudostratified columnar epithelia and alveoli have regular normal thickness walls, and normal inter alveolar septa connecting the alveoli together (Fig.1), the heart muscle showed normal histological structure of the fibers and intact cardiac muscle (Fig.6), and the spleen of the control group reveled intact tissue and normal both red and white pulp (Fig.10).

The lung treated with pb revealed the histopathological changed according to the following:

In group 2 the animal treated with 2 mg pb showed alveolar edema and chronic inflammatory cells infiltration (Fig.2).

Group 3 that treated with 4 mg pb showed interstitial and alveolar edema, infiltrations of inflammatory cells, focal reactive lymphoid follicles, and alveolar septal mild fibrosis (Fig.3).

Group 4 that treated with 6 mg pb showed few RBCs in alveoli, alveolar and interstitial edema, reactive chronic inflammation, septal fibrosis, vascular wall contraction, and hemosiderin deposition (Fig.4, 5).

The heart muscle showed the histopathological changed according to the following:

In group 2 the animal treated with 2 mg pb showed there are no remarkable pathological changes seen (Fig. 7).

Group 3 that treated with 4 mg pb showed slight degeneration of myocardial cells, dilated interstitial space (Fig.8).

Group 4 that treated with 6 mg pb showed hypertrophic cardiomyocytes, and focal degenerative changes with vacoulation (Fig.9).

The spleen showed the histopathological changed according to the following:

In group 2 the animal treated with 2 mg pb showed increased red pulp area and there is no significant changes (Fig.11).

In group 3 the animal treated with 4 mg pb showed red pulp congestion, reactive hyperplasia, and deposition of hemosiderin in white pulp (Fig.12).

In group 4 that treated with 6 mg pb the red pulp showed congestion, deposition of hemosiderin, while in white pulp degenerative changes and increased vascularity (Fig.13, 14).

The lung treated with cu revealed the histopathological changed according to the following:

In group 5 the animal treated with 50 mg cu showed mild interstitial edema and focal lymphatic infiltration in the septa (Fig.15).

In group 6 the animal treated with 100 mg cu showed mild interstitial edema, and infiltration of lymphatic cells in the septa (Fig.16).

In group 7 the animal treated with 150 mg cu showed interstitial edema, and infiltration of lymphatic cells in the septa, focal RBCs in the alveolar space, and thick alveolar wall (Fig.17).

The heart treated with cu revealed the histopathological changed according to the following:

In group 5 the animal treated with 50 mg cu there is no significant changes (Fig.18).

In group 6 the animal treated with 100 mg cu mild interstitial edema and focal degenerative of the myofibers (Fig.19).

In group 7 the animal treated with 150 mg cu interstitial edema and focal degenerative of the myofibers more frequent (Fig.20).

The spleen treated with cu revealed the histopathological changed according to the following:

In group 5the animal treated with 50 mg cu there is no significant change (Fig.21).

In group 6 the animal treated with 100 mg cu red pulp hyperplasia, congestion, and hemosiderin deposition (Fig.22).

In group 7 the animal treated with 150 mg cu there is white pulp degeneration, vascular hyalinization; frequent a nucleated cells, and congestion (Fig.23, 24).

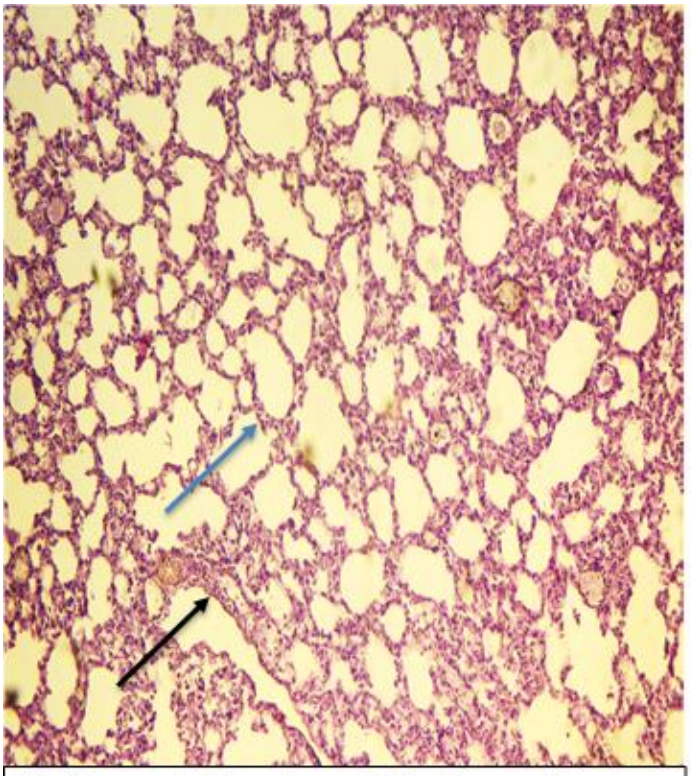

Fig. 1 lung control showed normal intact tissue and the bronchiole lined by pseudostratified columnar epithelia (black arrow) and alveoli have regular normal thickness walls(red arrow) X100 H&E

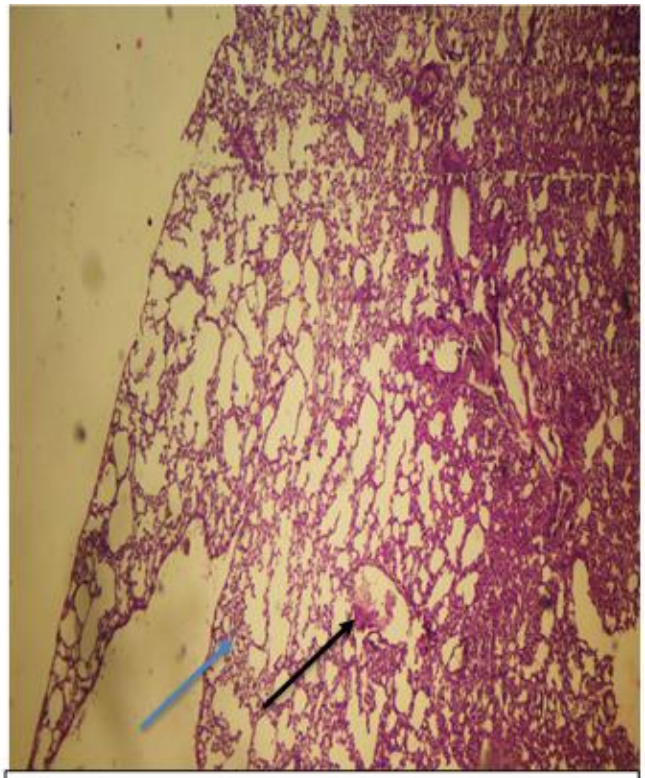

Fig. 2 lung 2mg pb showed alveolar edema(black arrow) and chronic inflammatory cells infiltration(red arrow) X100 H&E

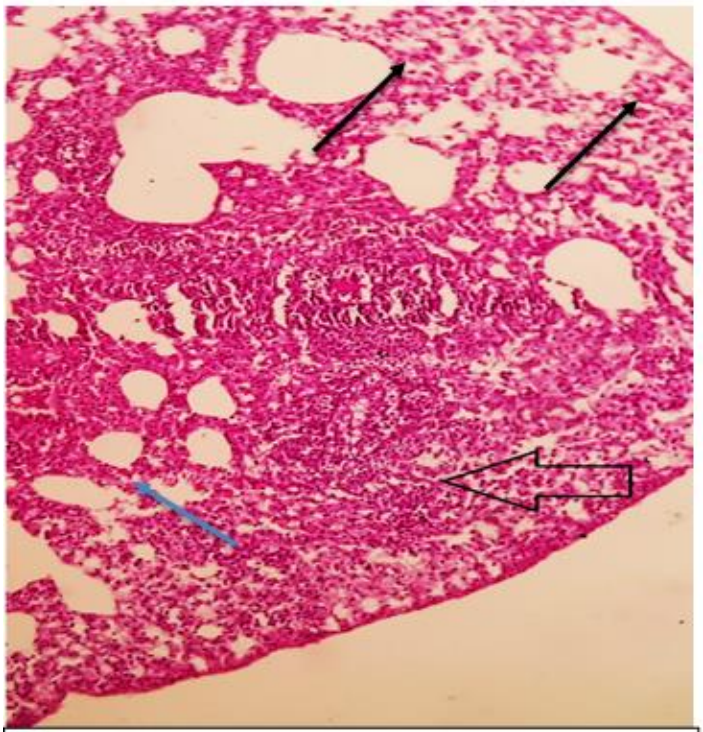

Fig. 3 lung 4mg pb showed infiltrations of inflammatory cells (black arrow), rapture of alveoli (blue arrow), and alveolar septal mild fibrosis (large arrow) X400 H&E

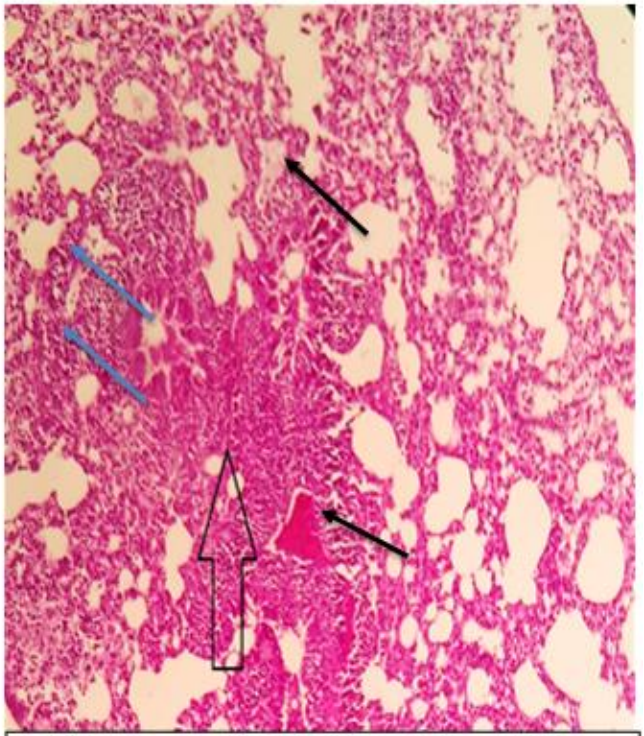

Fig. 4 lung 6 mg pb showed alveolar and interstitial edema (black arrow)septal fibrosis(large arrow), and hemosiderin deposition(blue arrow) X400 H&E

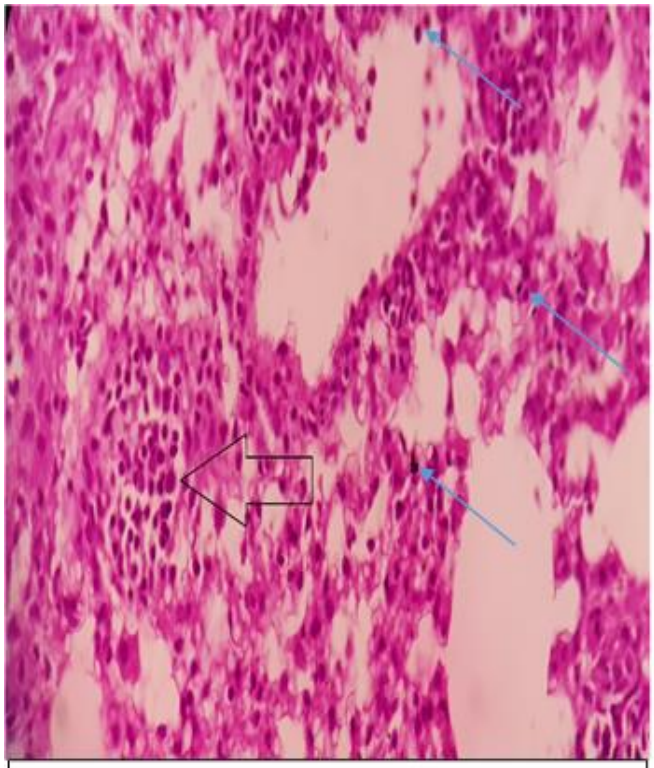

Fig. 5 lung 6 mg pb showed septal fibrosis (large arrow), and hemosiderin deposition (blue arrow) X100 H&E

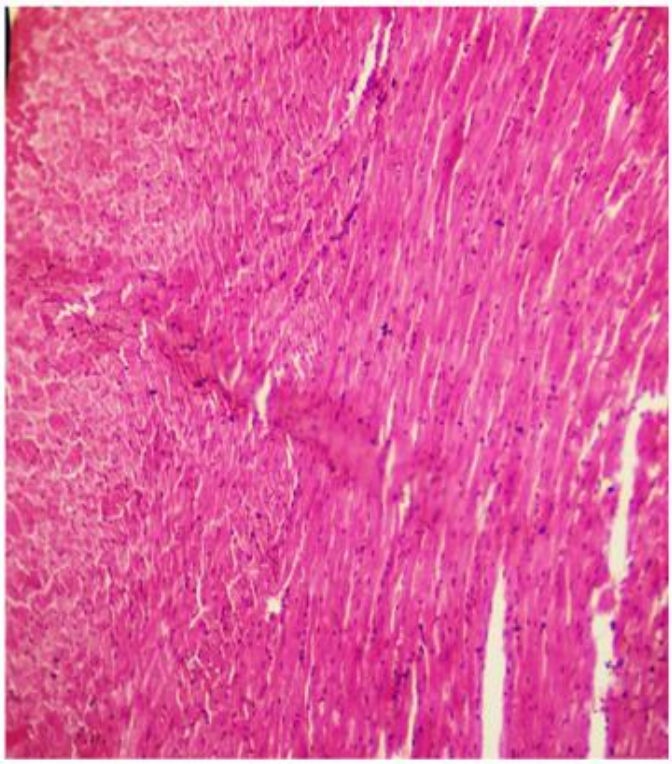

Fig. 6 heart muscle showed normal histological structure of the fibers and intact cardiac muscle 1000x

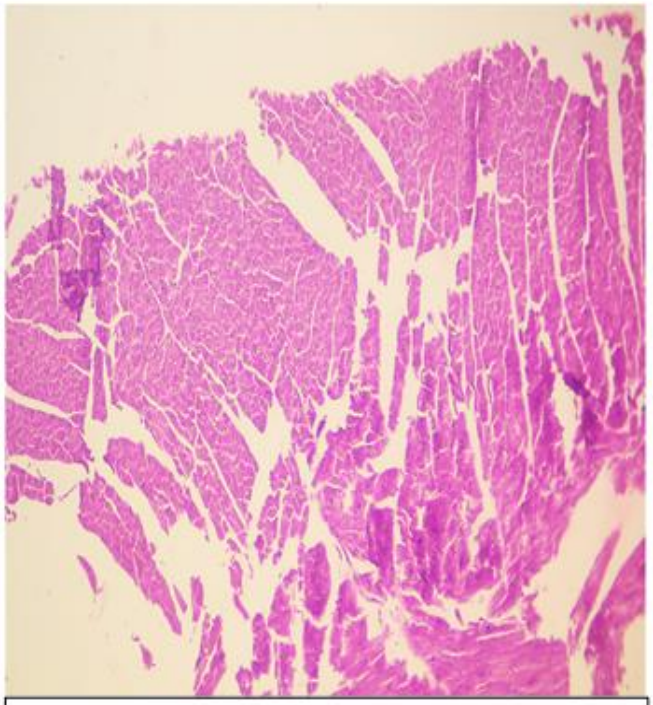

Fig. 7 group 2 treated with 2 mg pb showed there are no remarkable pathological changes seen H&E 100x

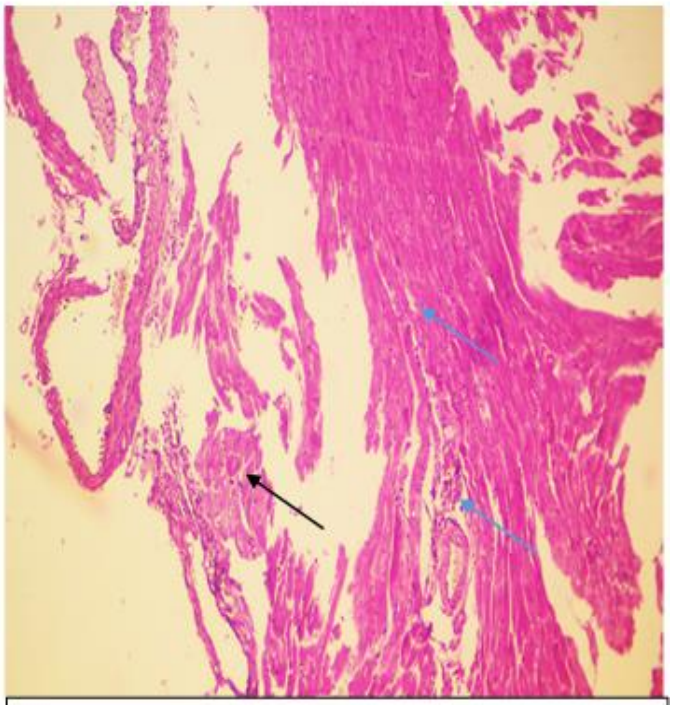

Fig. 8 group 3 treated with 4 mg pb showed slight degeneration of myocardial cells (black arrow), dilated interstitial space(blue arrow) H&E 100x

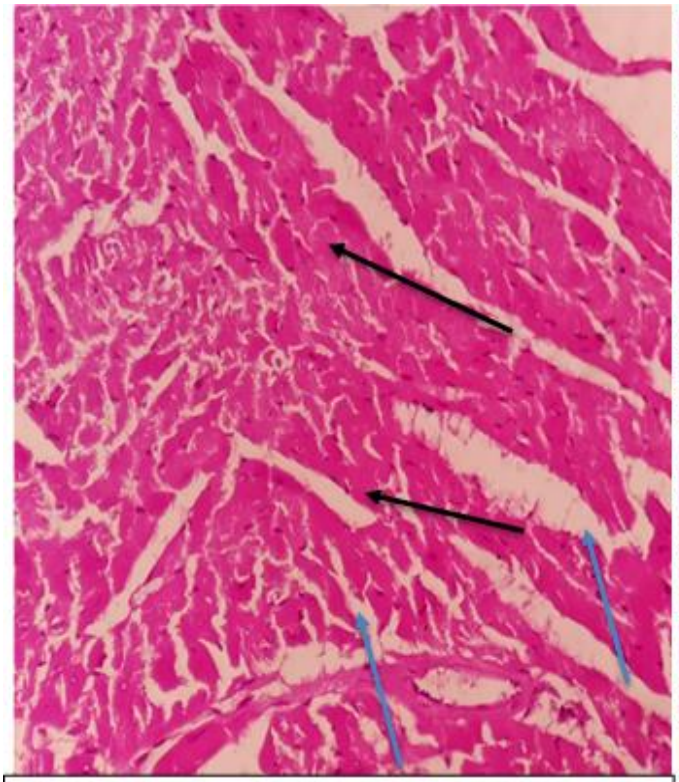

Fig. 9 showed hypertrophic cardiomyocytes (black arrow), and focal degenerative changes with vacoulation (blue arrow) H&E 1000x

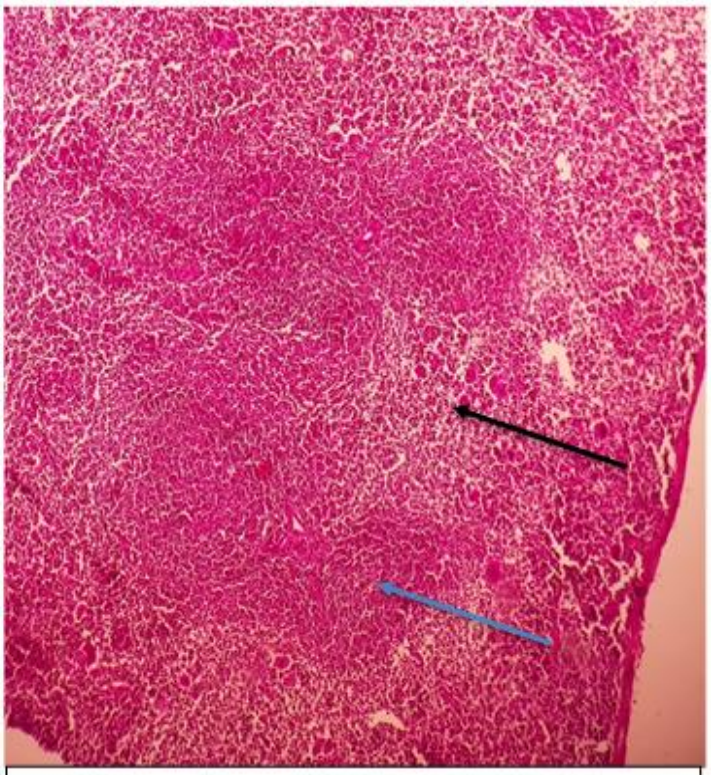

Fig. 10 spleen of the control group reveled intact tissue and normal both red pulp (black arrow) and white pulp (blue arrow) H&E 100x

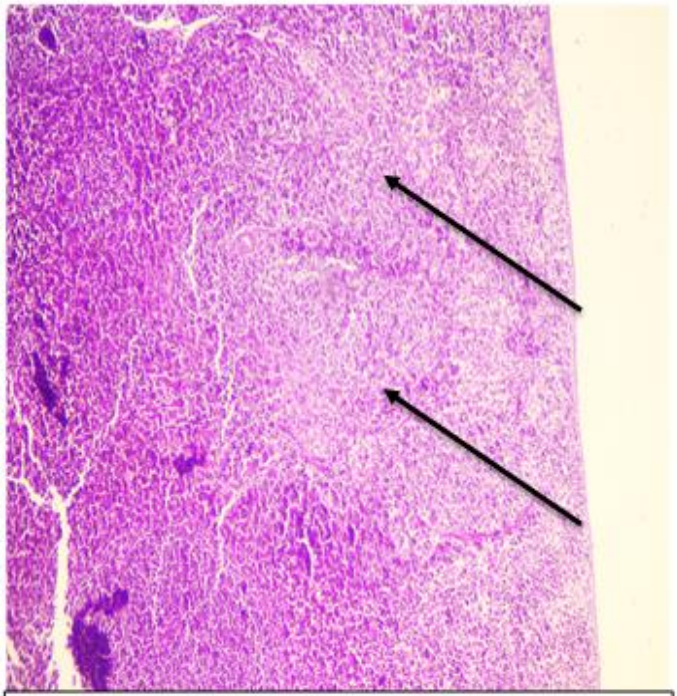

Fig. 11 showed increased red pulp area (black arrow) and there is no significant changes 1000x

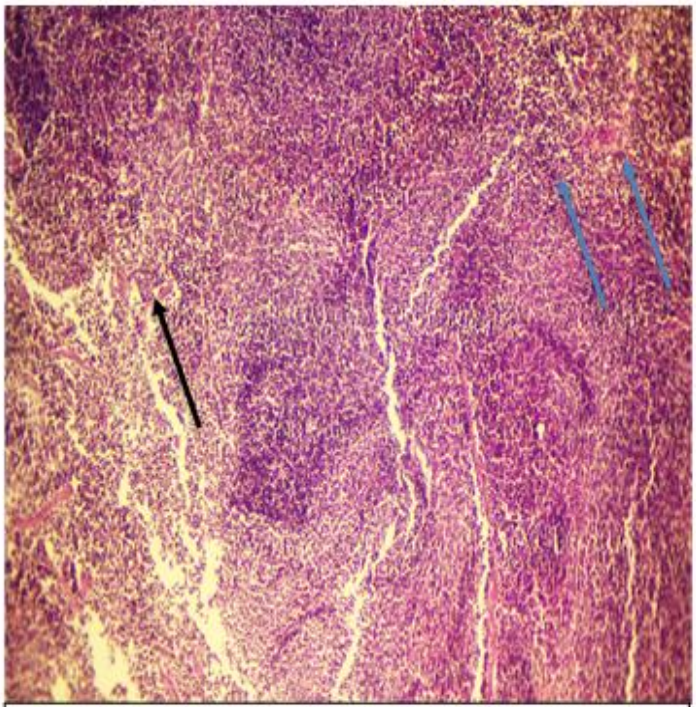

Fig. 12 showed red pulp congestion (black arrow), reactive hyperplasia, and deposition of hemosiderin in white pulp (blue arrow) H&E 1000x

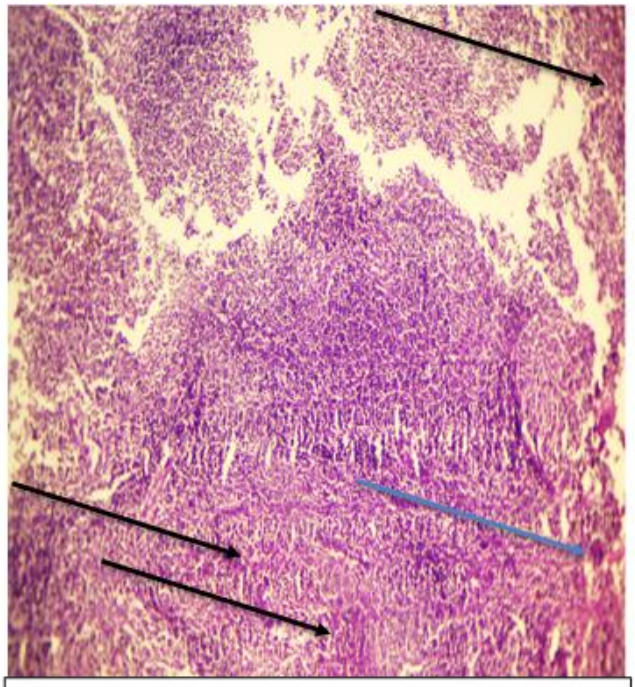

Fig. 13 showed congestion (black arrow), and increased vascularity (blue arrow) H&E 100x

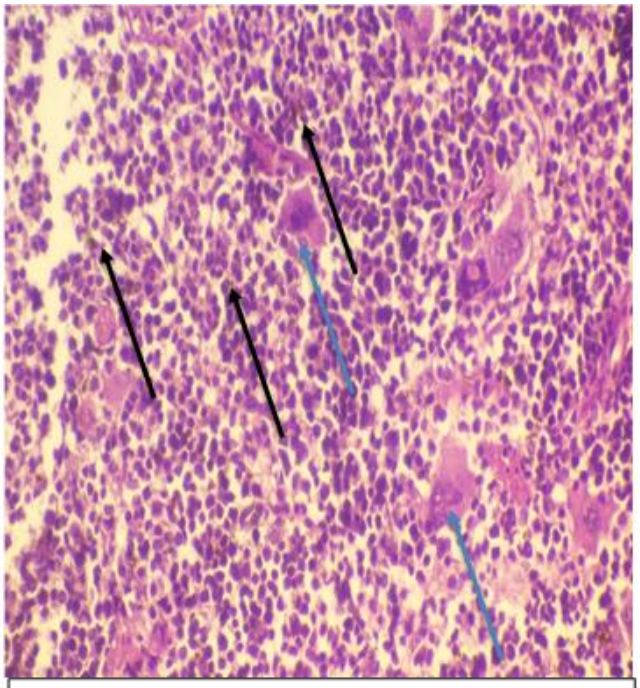

Fig. 14 deposition of hemosiderin (black arrow), presence of megakaryocytes (blue arrow) H&E 1000x

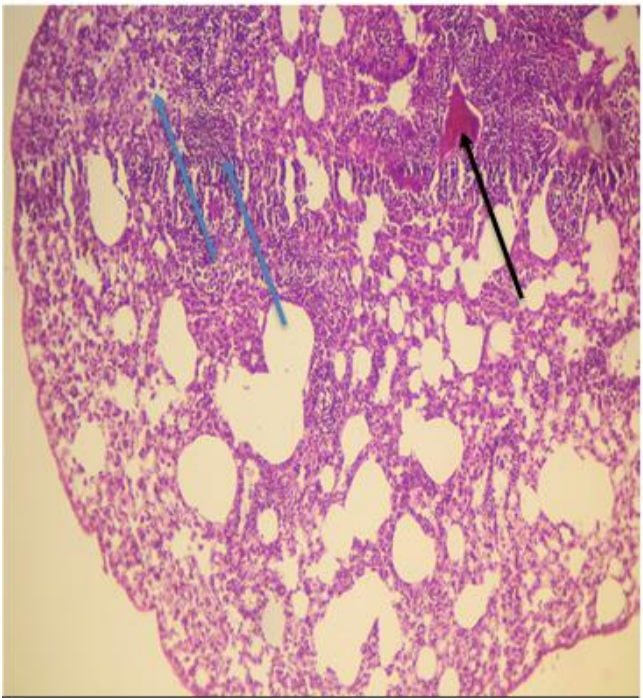

Fig. 15 showed mild interstitial edema (black arrow), and focal lymphatic infiltration in the septa (blue arrow) H&E 1000x

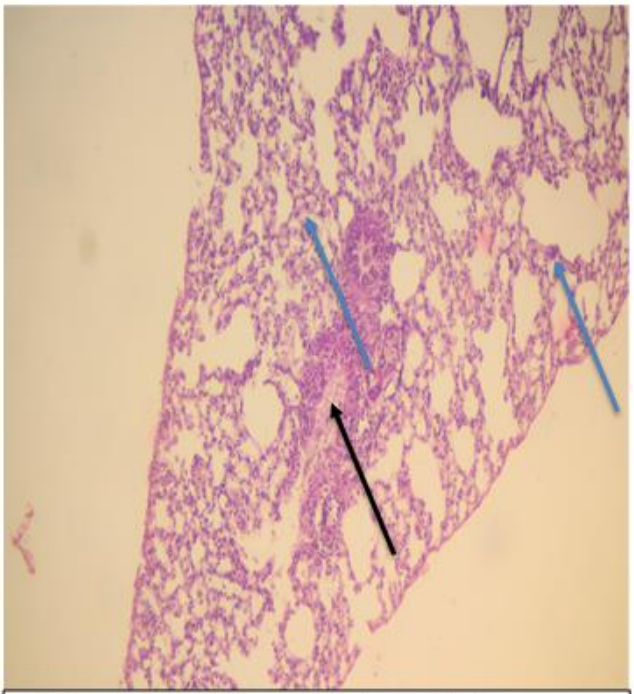

Fig. 16 showed mild interstitial edema (black arrow), and infiltration of lymphatic cells in the septa (blue arrow) H&E 100x

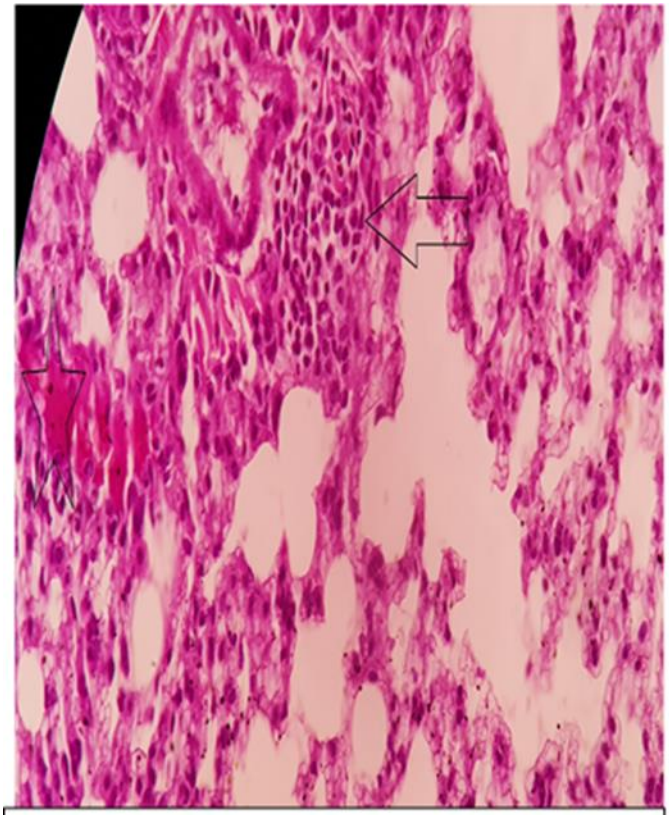

Fig. 17 showed interstitial edema (star), and infiltration of lymphatic cells in the septa (large arrow) H&E 1000x

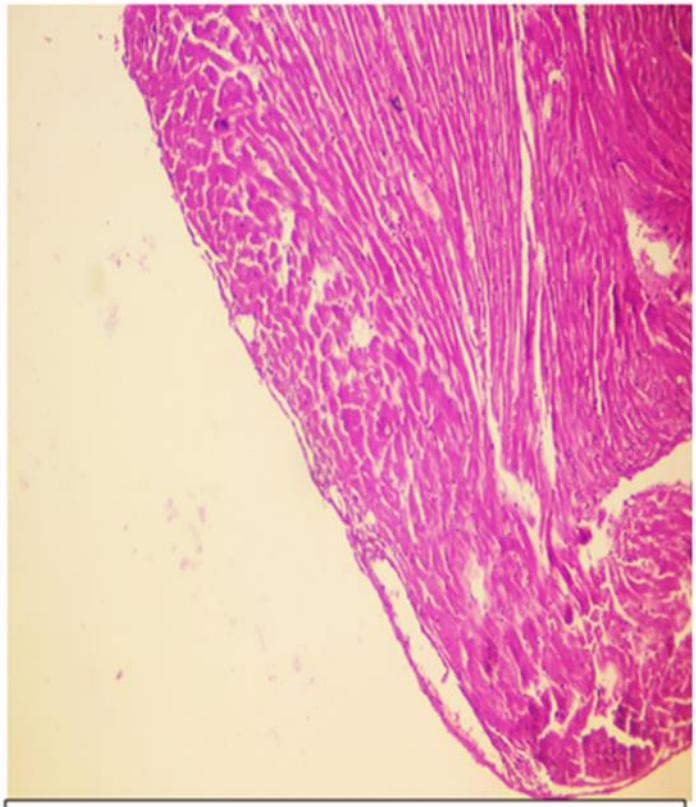

Fig. 18 showed no significant changes H&E 100x

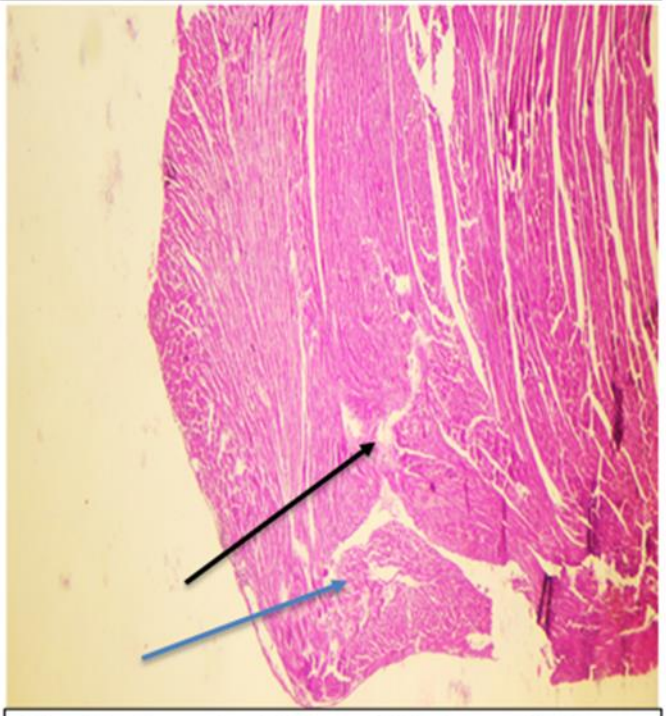

Fig. 19 showed mild interstitial edema (black arrow) and focal degenerative of the myofibers (blue arrow) H&E 100x

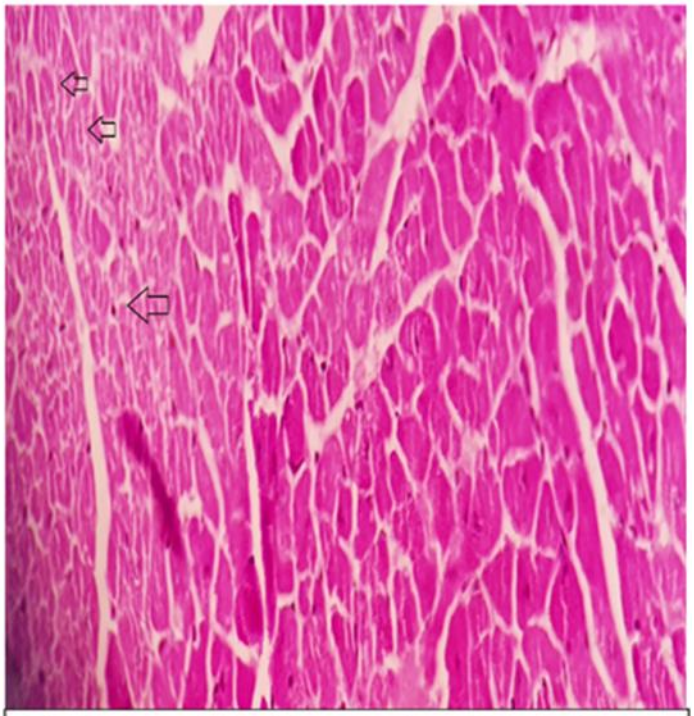

Fig. 20 showed focal degenerative of the myofibers (arrow) H&E 1000x

DISCUSSION

It well knows that heavy metal have harmfully effect on both human and animals' health and harmfully effect on the environment. In the present study Histopathologic evaluation of exposed mice to 6 weeks administration from Copper Sulphate and lead nitrate, this demonstrated progression of lungs, hearts and spleen organs lesions similar to those described in other studies (6) . the present study in agreement with what found by(14) who demonstrated dramatic lesion in the spleen tissues of male rat treated with nickel and chrome represented by lymphatic necrosis of some lymphoid follicles, presence of megakaryocytes and thickening of splenic capsule. These morphological changes can be attributed to the rapid accumulation of heavy metals in the cytosolic of epithelium, as well as lead can causes damage in the cell membrane by changes enzyme activity then changes sodium-potassium pump subsequently causes cell injury (15). Different study showed that heavy metals cause (ROS) reactive oxygen species which convoluted to cell damages (16). In this pathway can forming the hydroxyl radical (OH^-) by react heavy metals with H2O2 via Fenton-type or Haber-Weiss-type causing tissue injury and DNA damage by apoptosis and finally necrosis. Other research found histopathological changes of heavy metals resulted from high level of MDA while low level from GSH and SOD as indicator for lipid layer and cell destroyed from oxidative stress (17). (18) showed Histopathological examination for lung, kidney, liver and heart of male rats exposed to heavy metal characterized by diffuse haemosiderosis pigment, thickened of the alveolar wall, increased alveolar macrophages, diffused red blood cells and edema within lung section, while in heart found interstitial edema, myofibres degeneration. As well as present result in agreement with what found by (19) in which demonstrated deleterious effects on the lung tissue of male rat treated with lead include disruption of the alveoli, fibrosis and damage to the lung tissues, airway epithelial damage or infiltration of inflammatory cells he also found inhalation of lead or other heavy metals for a long time can cause pneumonia or asthma. On other hand (6, 3, 20) found necrosis, Hyperplasia, degenerative changes, lymphocytes infiltration and alveolar fibrosis in the pulmonary, kidney, liver and spleen tissues of rat exposed to heavy metal. Histopathological of present study in agreement with (4) who revealed Histopathologic assessment of lead acetate on the spleen of rats. (2) also observed Hepatocytes vacuolization, necrosis and different spleen change when treated with concentrations of CuSo4.

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