Microscopic Evaluation for the Protective Role of Ascorbic Acid against Lead Toxicosis in Male Rats

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ABSTRACT

The present study was carried out to evaluate the microscopic changes related to the protective effect of supplementation with two levels (2000 and 4000 ppm in drinking water) of ascorbic acid (AA), against lead (Pb) toxicosis (150 ppm in drinking water) in rats. One hundred and eighty adult male Sprague-Dawley rats were used. The concurrent daily administration for each of the two levels of AA with the toxic dose of Pb acetate was continued allover the 3 months of the experiment after which necropsy and histopathologic examination were done. The histopathologic examination in the first trial for treatment with the low level of AA revealed no or less protective effect where some mild to moderate changes of Pb toxicosis in some organs were seen. The histopathologic examination in the second trial for treatment with the high level of AA revealed an obvious alleviating effect on various body organs. In conclusion for the present study the supplementation with AA resulted in an appreciable improvement in the architecture of body organs, especially at levels of 4000 ppm.

Key Words: Antioxidant, Lead acetate, Lead poisoning, Toxicosis, Vitamin C.

INTRODUCTION

Lead is considered a common environmental pollutant (Ragan and Turner, 2009 and Grant, 2009). Lead poisoning (also known as plumbism) is a medical condition caused by increased levels of lead in the blood. It is caused by exposure to lead by contact in air, household dust, soil, water, and commercial products (Rossi, 2008). No level of lead in the body below which no harm occurs has been discovered (Flora, 2008). Lead may impair development and have harmful health effects even at lower levels, and there is no known safe exposure level (Rossi, 2008). Lead poisoning may be acute (from intense exposure of short duration) or chronic (from repeated low-level exposure over a prolonged period), but the latter is much more common (Trevor *et al*. 2007). Acute lead poisoning usually lead to hemolysis followed by anemia and hemoglobinuria with damage to the kidney (nephropathy) and causes changes in urination such as decrease urine output (Brunton *et al*. 2007 and Rubin and Strayer, 2008). Chronic lead poisoning usually presents with symptoms affecting multiple systems (Kosnett, 2005), but it is usually associated with three main types of symptoms: gastrointestinal, neuromuscular, and neurological (Pearce, 2007). Central nervous system and neuromuscular symptoms usually result from intense exposure, while gastrointestinal symptoms usually result from exposure over longer periods (Brunton *et al*. 2007). The brain is the organ most sensitive to lead exposure (Cecil *et al*., 2008). Lead poisoning was also reported to affect both male and female reproductive systems. In man, when blood lead levels exceed 40 μg/dL, sperm count is reduced and changes occur in volume of ejaculate, sperm motility, and their morphology (Grant, 2009).

The toxic manifestations of the heavy metals are caused primarily due to their oxidative stress. The Long term exposure to these metals could lead to apoptosis (Flora *et al*., 2008). The oxidative stress induced by lead had a crucial role in cholestasis, apoptotic/ necrotic hepatocellular damage, and the impairment in liver transport function (Gonzalez *et al*., 2007). The treatment against metal poisoning with chelating agents is compromised with a number of serious side-effects. Supplementation of antioxidants

along-with a chelating agent proved to be a better treatment regimen than monotherapy with chelating agents (Flora *et al*., 2008). Recently the consumption of antioxidants is increasing day after day. Khanna and Nehru (2007) found that the isolated glial cells and neurons from rat cerebral cortex showed a varied pattern of important antioxidant enzymes and glial cells are more capable of handling the oxidative stress conditions induced by lead intoxication. Vitamin E, as one of the powerful antioxidants was found to counteract the harmful effects of lead not only by preventing free-radical formation but also by favoring lead disposal (Gonzalez *et al*., 2007). Ascorbic acid is also known to be one of the powerful antioxidants and considered as an essential micronutrient and powerful antioxidant (Griffiths and Lunec, 2001). Numerous studies were done to support the antioxidant and protective effects of AA (Sebastian *et al*., 2003, Mehri *et al*., 2005, Oyinbo *et al*., 2006, and Li and Schellhorn 2007). In other studies, vitamin C supplementation was found to produce reductions in lead retention during the consumption of a lead-containing drink (Dawson and Harris, 1997, and El-Neweshy and El-Sayed, 2010). Objective: To carryout the histopathologic evaluation for the effect of daily supplementation with ascorbic acid (AA) against lead toxicosis in rats.

MATERIALS AND METHODS

Laboratory animals: One hundred and eighty adult male Sprague-Dawley rats (of about 2 months old and of 150-200 g body weight), were obtained from The animal House of The College of Veterinary Medicine and Animal Resources, King Faisal University. The rats were kept for one week of acclimatization at standard hygienic conditions, fed on standard pelleted rat feed and water (free from any sources of lead contaminant) ad libitum.

Chemicals: L-Ascorbic Acid "AA" (Sigma) and lead acetate "Pb acetate" (Fisher Chemical, Fairlawn, NJ)

Experimental design: The rats were randomly subdivided into 6 groups (each of 30 rats) caged in a metal cages, fed on standard pelleted rat feed and received the following treatments in drinking water:

- Group 1: Received drinking water (free from any chemicals) ad libitum, allover the experiment, and maintained as a negative control for other groups.
- Group 2: Received daily dose of AA (2000 ppm) in drinking water and maintained as a positive control for the low level of AA.
- Group 3: Received daily dose of AA (4000 ppm) in drinking water and maintained as a positive control for the high level of AA.
- Group 4: Received daily dose of Pb acetate (150 ppm in drinking water), that achieved and maintain target Blood lead levels of 35–40 µg/dL (Virgolini *et al*., 2008) allover the experiment and maintained as a positive control for the lead intoxication. Pb acetate administered in drinking solutions was dissolved in distilled de-ionized water and prepared fresh on a weekly basis.
- Group 5: Received daily doses of only low level of AA (2000 ppm) in drinking water for one week previous to intoxication, then continued with concurrent daily doses of 150 ppm Pb acetate in drinking water, allover the 3 months of the experiment. This group was considered the first experimental group for evaluation of the first trial of protection by low level of AA.
- Group 6: Received daily doses of only high level of AA (4000 ppm) in drinking water for one week previous to intoxication, then continued with concurrent daily doses of 150 ppm Pb acetate in drinking water, allover the 3 months of the experiment. This group was considered the second experimental group for evaluation of the first trial of protection by high level of AA.

The treated animals of all groups were observed for recording any clinical signs or mortalities throughout the experiment. At the end of the experiment (3 months), the all rats of each group were euthanized and subjected to necropsy. Tissue specimens were collected from the brain and other

internal organs (male sex organs, kidneys, liver, stomach, intestine, heart, lungs and spleen) for histopathologic examination.
Histopathologic techniques: The collected tissue specimens of all rats were fixed in 10% neutral buffered formalin solution then passed automatically through the routine paraffin-wax embedding technique, microtomy and staining with hematoxylin and eosin (H&E) (Drury and Wallington, 1980) and then studied under the the light microscope.

RESULTS

Animal of all groups have not exhibited any abnormal clinical signs and were apparently healthy during the first month of the experiment. The intoxicated rats of the group four of Pb intoxication exhibited some mild nervous signs of restlessness, fairness with some excitations, especially from end of the second month till the end of the third month of Pb intoxication, while no mortalities occurred. Similar signs were observed in rats of the group 5 but during the third month of concurrent administration with low level of AA. At necropsy, moderate to severe degree of congestion and hemorrhages of the superficial vasculatures were seen grossly in the brain and other internal organs of intoxicated rats of group 4, and somewhat in group 5 (first trial of protection). The organs of the rats in other groups were apparently normal.

The microscopic findings in the organs of the positive control group for Pb intoxication (group 4) revealed moderate to severe changes in body organs. These changes includes congestion and hemorrhages from the meningeal and deep blood vessels of the brain tissue, focal to diffuse gliosis and neuronal degeneration, especially at the cerebrum, cerebellum and midbrain in addition to degeneration with nuclear pyknosis of the cerebellar Purkinji cells. The testis showed spermatogonial degeneration and necrosis, congestion and interstitial edema and mild degeneration of the Leydig cells. The epididymal tubules at the head and tail regions also showed degenerated epithelium, interstitial congestion and edema with some luminal contents of immature spermatogonial cells. The prostate gland and seminal vesicle showed areas of cystic dilatation and other areas of epithelial papillary proliferations with interstitial congestion and edema. The kidney showed vascular congestion, hemorrhages and hemolysis in addition to excess of intravascular and interstitial mononuclear cell aggregation. The renal tubular epithelium also showed degeneration, numerous formations of nuclear eosinophilic lead inclusion bodies with epithelial necrosis and desquamation. The liver showed hepatocellular degeneration and necrosis, multiple foci of mononuclear cell aggregation in addition to newly formed bile ductules at the portal areas. The stomach showed severe mucosal changes of epithelial degeneration, necrosis and desquamation with cystic dilatation in some of the deep crypts of the gastric glands in addition to some degrees of congestion with eosinophilic and mononuclear cell infiltration and aggregation allover the wall of the stomach. The intestinal mucosa also showed some degrees of epithelial degeneration, necrosis and desquamation, congestion and mononuclear cell infiltration. The lungs showed variable changes of bronchopneumonia with common perialveolar and peribronchial vascular congestion and mononuclear infiltration and aggregation. The cardiac muscle suffered some necrobiotic changes with numerous areas of intermuscular congestion and hemorrhages and excess of intravascular and intermuscular mononuclear cells. The spleen showed small lymph follicles with depletion of the lymphocytes.

The microscopic examination of the organs of the group 5 rats revealed the following main findings: In the brain tissue some moderate degrees of meningeal congestion with cerebral changes of focal gliosis, neuronal degeneration and neuronophagia (Fig. 1).

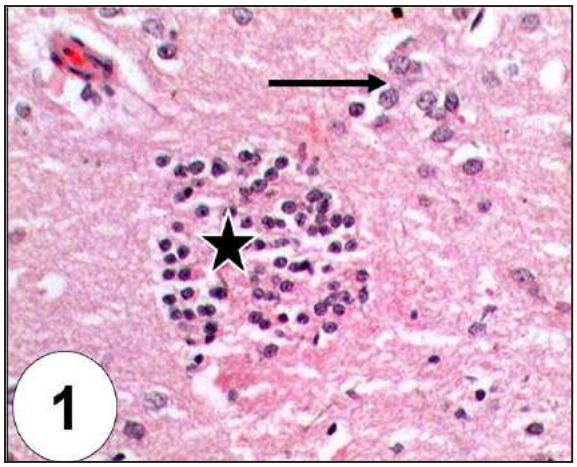

Fig.1: Cerebral cortex of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: focal gliosis (Black asterisk) and neuronophagia (Arrow). H and E. X 400.

The cerebellar vasculatures were congested and were accompanied by nuclear pyknosis and degeneration of the Purkinji cells. Some of the semineferous tubules of the testes were degenerated, necrotic and separated by areas of interstitial edema (Fig.2).

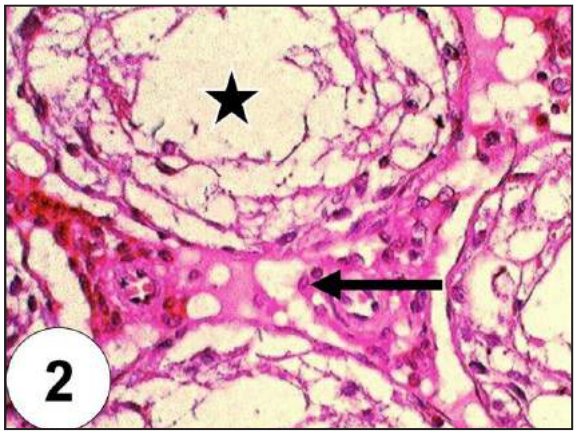

Fig.2: Testis of rat received 2000 ppm AA + 150 ppm Pb acetatee in drinking water: Interstitial edema (Arrow) and degenerated and necrotic semineferous tubule (Black asterisk). H and E. X 400.

The Cauda epididymis contained few luminal contents of degenerated immature spermatogonial cells (Fig.3), while the tubules in some areas were widely separated by interstitial edematous fluids mixed with an excess of the congested blood capillaries and mononuclear cell infiltration (Fig.4).

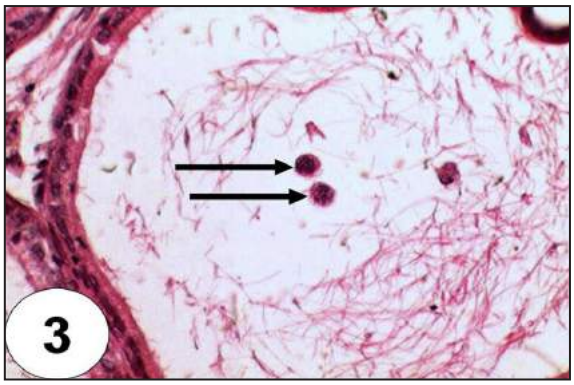

Fig.3: Cauda epididymis of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Few luminal contents of degenerated immature spermatogonial cells (Arrows). H and E. X 400.

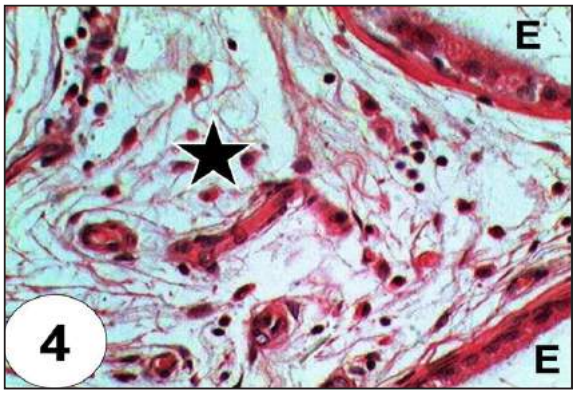

Fig.4: Cauda epididymis of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Epididymal tubules (E) widely separated by interstitial edema (Black asterisk) with excess of congested blood capillaries and mononuclear cell infiltration. H and E. X 400.

The secretory acini of the prostate gland were highly active with cystic dilatation and also widely separated with the edematous fluids, and congested blood capillaries (Fig.5).

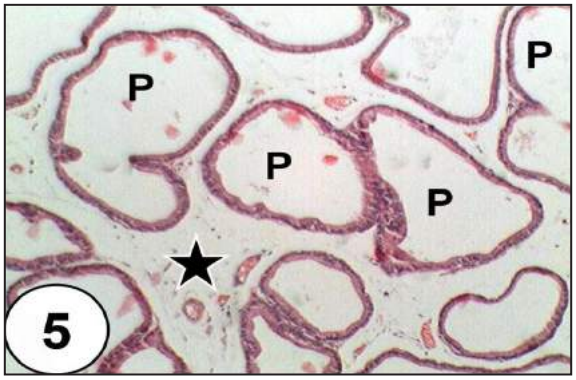

Fig.5: Prostate gland of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Cystic dilated Prostatic acini (P) Widely separated by edema (Black asterisk). H and E. X 160.

The kidneys revealed common changes of glomerular and intertubular congestion, while the renal tubules showed some degrees of degenerative changes with nuclear pyknosis or nuclear contents of the eosinophilic lead inclusion bodies (Figs. 6 and 7).

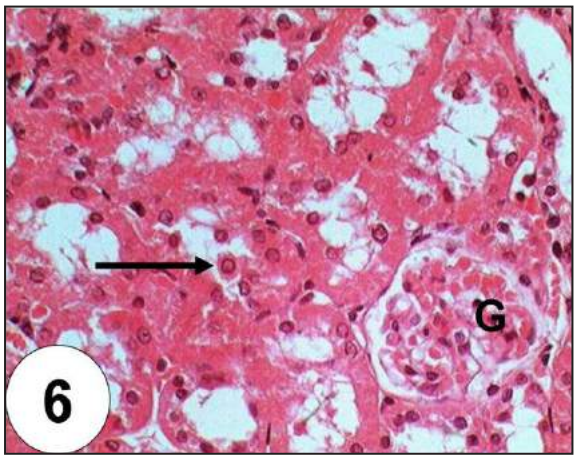

Fig.6: Kidney of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Congested glomerulus (G) and degenerated tubular epithelium with intranuclear eosinophilic lead inclusion bodies (Arrow). H and E. X 250.

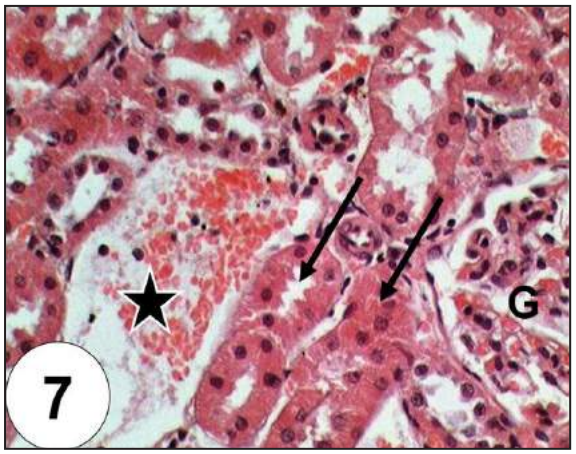

Fig.7: Kidney of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Glomerular congestion (G), degenerated epithelium of the proximal tubules with nuclear pyknosis (Arrows) and intertubular congestion and hemorrhage (Black asterisk). H and E. X 400.

The liver showed some degrees of hepato-cellular degeneration and necrosis while the portal areas contained large number of mononuclear cell aggregation and newly formed bile ductules (Fig.8).

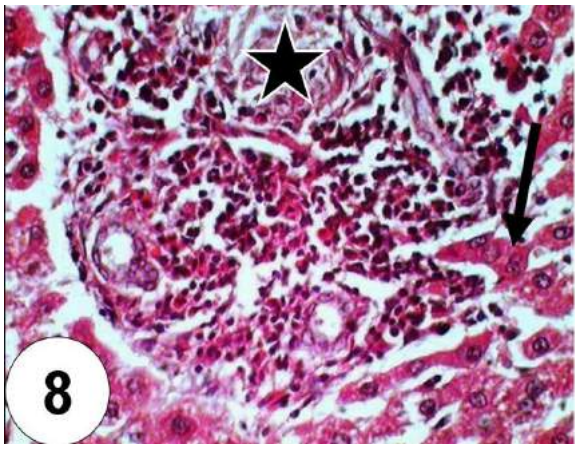

Fig.8: Liver of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Degenerated and necrotic hepatic cells (Arrows) with large numbers of mononuclear cell aggregation and small newly formed bile ductules (Black asterisk). H and E. X 250.

The stomach showed mucosal changes of epithelial degeneration, with cystic dilatation in some of the deep crypts of the gastric glands in addition to mild degrees of congestion and few eosinophilic mononuclear cell infiltration. The small intestine, especially the ileum was affected with an excess of the epithelial vacuolation particularly in the crypts of the intestinal glands in addition to submucosal edema, congested capillaries and eosinophilic cell infiltration (Fig. 9).

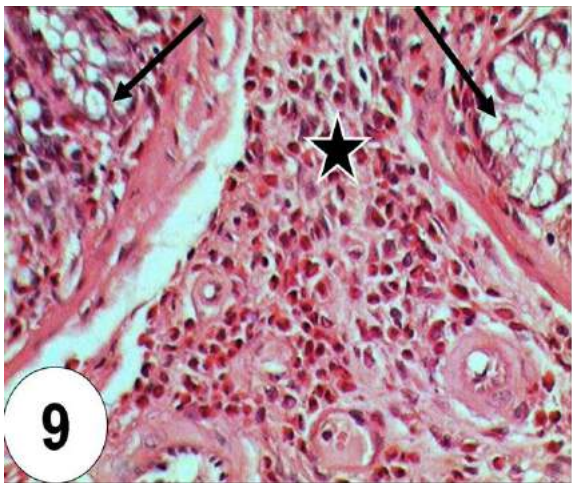

Fig.9: Intestine (Ileum) of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Vacuolar degenerated epithelium of the glandular crypts (Arrows) with submucosal edema, congested capillaries and eosinophilic cell infiltrations (Black asterisk. H and E. X 400.

The lungs showed peribronchiolar changes of congested blood vessels and some mononuclear cell infiltration (Fig. 10).

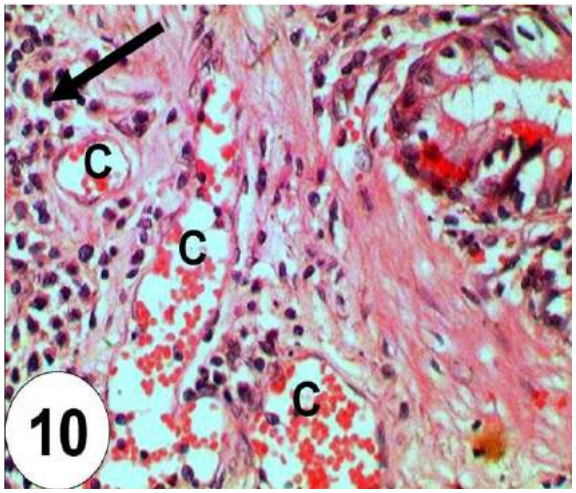

Fig.10: Lung of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Excess of peribronchial congested blood capillaries (C) and mononuclear cell infiltration (Arrows). H and E. X 400.

The cardiac muscle revealed active and hypertrophied nuclei in addition to severely congested intermuscular blood vessels (Fig. 11).

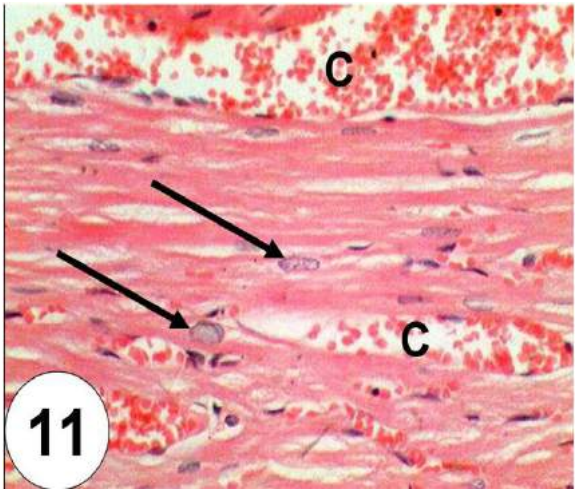

Fig.11: Heart of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Active and hypertrophied nuclei of the cardiac muscle (Arrows) with severely congested intermuscular blood capillaries (C). H and E. X 400.

The spleen was commonly congested, while the lymph follicles were small and atrophied and appeared with depleted lymphocytes (Fig 12).

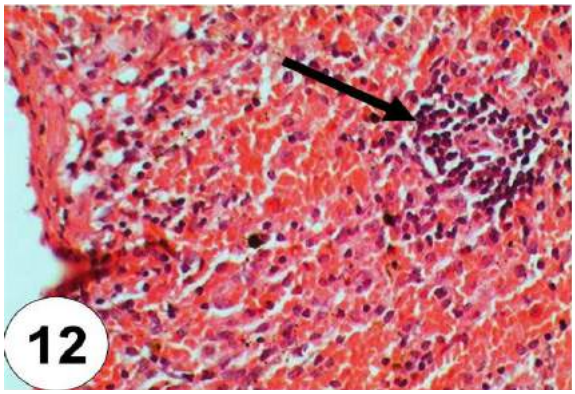

Fig.12: Spleen of rat received 2000 ppm AA + 150 ppm Pb acetate in drinking water: Small atrophied lymph follicle with depletion of the lymphocytes (Arrow) with congestion. H and E. X 250.

The microscopic examination of the organs of the group 6 revealed the following main findings. The brain tissue contained large numbers of the active cerebral astrocytic cell (Fig. 13) in addition to active and healthy cerebellar Purkinji cells (Fig. 14).

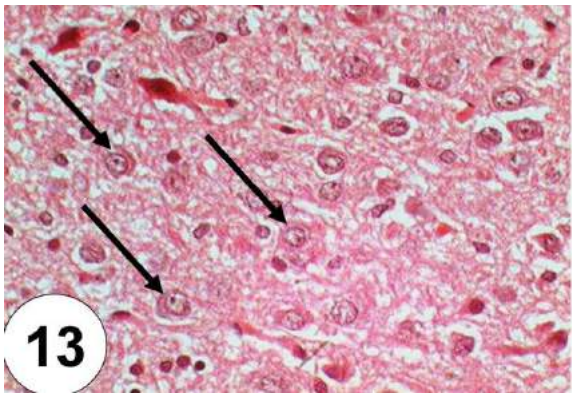

Fig.13: Cerebral cortex of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Excessive numbers of the active astrocytes (Arrows). H and E. X 400.

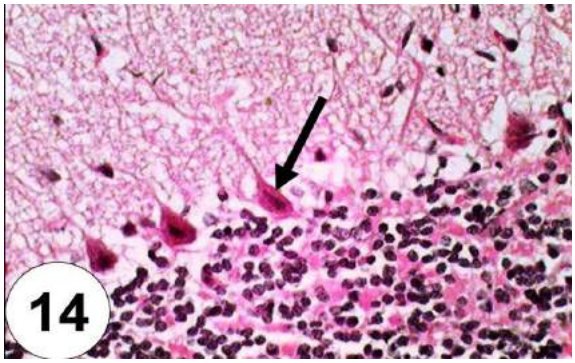

Fig.14: Cerebellum of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Active and apparently healthy Purkinji cells (Arrow).H and E. X 400.

The testes showed normal seminiferous tubules, active spermatogenesis and normal interstitial contents of the Leydig cells (Fig. 15).

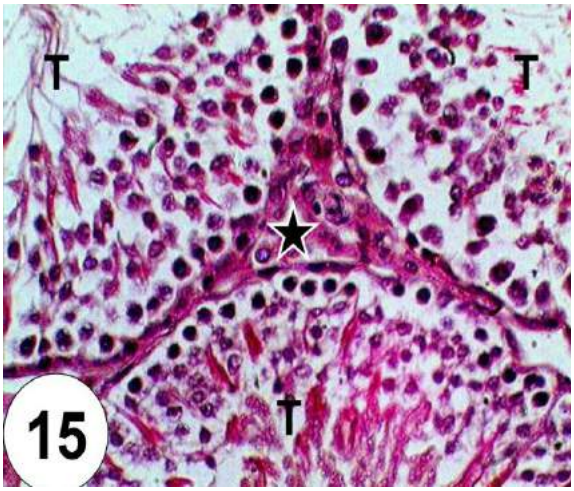

Fig.15: Testis of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Apparently normal semineferous tubules with active spermatogenesis (T) with normally present interstitial Leydig cells (Black asterisk). H and E. X 400.

The tubules at the caput and cauda epididymis appeared normal and filled with seminal contents (Fig. 16).

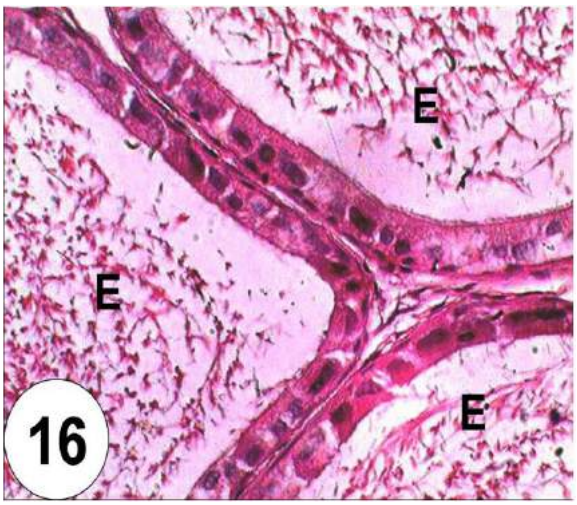

Fig.16: Cauda epididymis of rat received4000 ppm AA + 150 ppm Pb acetate in drinking water: Normal Epididymal tubules (E) filled with seminal contents. H and E. X 400.

The secretory acini of the prostate glands and seminal vesicles were active and accompanied in some areas by interstitial edema and congested capillaries (Figs. 17 and 18).

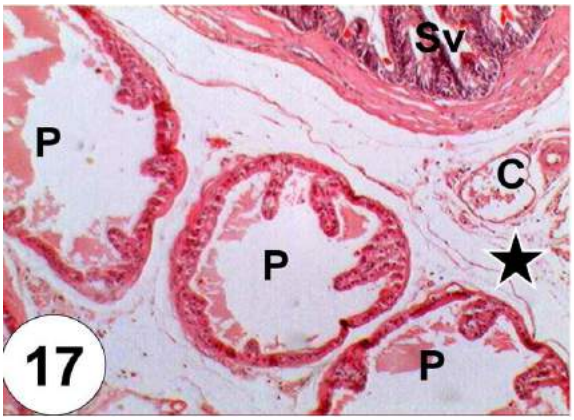

Fig.17: Prostate gland and seminal vesicle of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Nearly normal acini of the prostate gland (P) and seminal vesicle (Sv) with mild interstitial edema (Black asterisk) and congested blood capillaries (C). H and E. X 160.

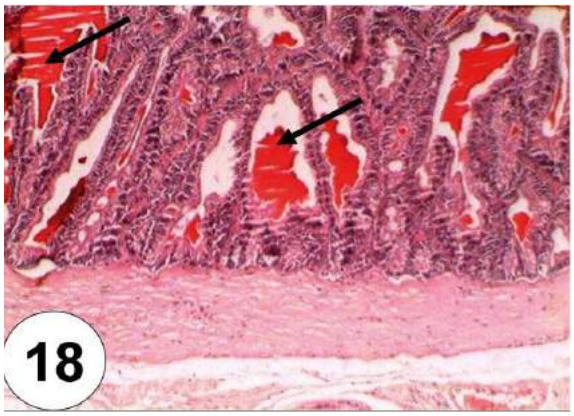

Fig.18: Seminal vesicle of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Active secretory acini with an excess of secretions (Arrows). H and E. X 250.

The kidneys contained nearly normal proximal tubules and with somewhat enlarged and congested glomeruli (Fig. 19).

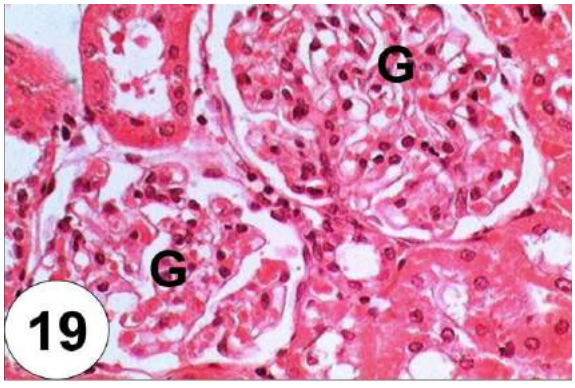

Fig.19: Kidney of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Enlarged and congested glomeruli (G) with apparently normal proximal tubules. H and E. X 400.

The Liver showed mild hepatocytic degeneration, while some of the portal areas appeared contained dilated veins and some newly formed bile ductules (Fig. 20).

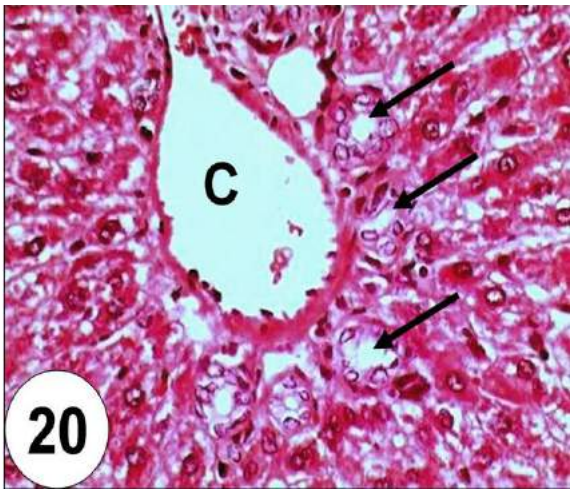

Fig.20: Liver of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Dilated portal vein with an excess of newly formed bile ductules (Arrows) with mild hepatocytic degeneration. H and E. X 400.

The intestinal mucosa was nearly normal with a normal and intact epithelium (Fig. 21).

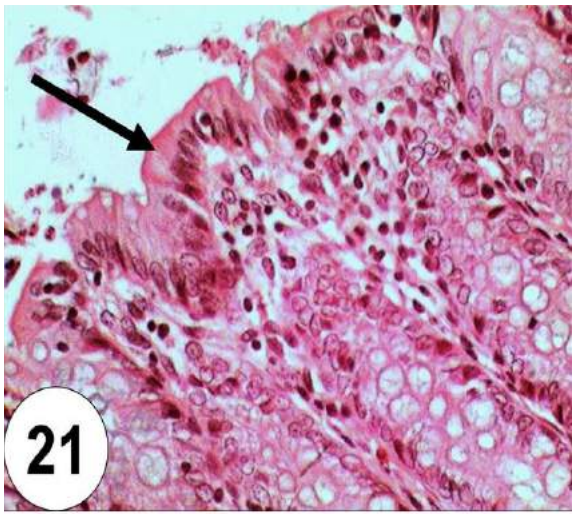

Fig.21: Intestine (Ileum) of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Intestinal mucosa with normal and intact covering epithelium (Arrow). H and E. X 400.

The pulmonary tissue contained normal bronchioles but the surrounding blood vessels were congested (Fig. 22).

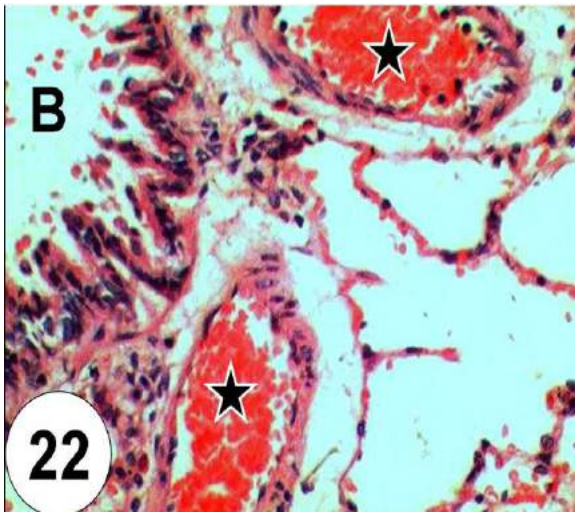

Fig.22: Lung of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Bronchiole (B) with surrounding congested arterioles (Black asterisks). H and E. X 400.

The myocardial muscle was normal with minute glycogenic vacuolations (Fig. 23).

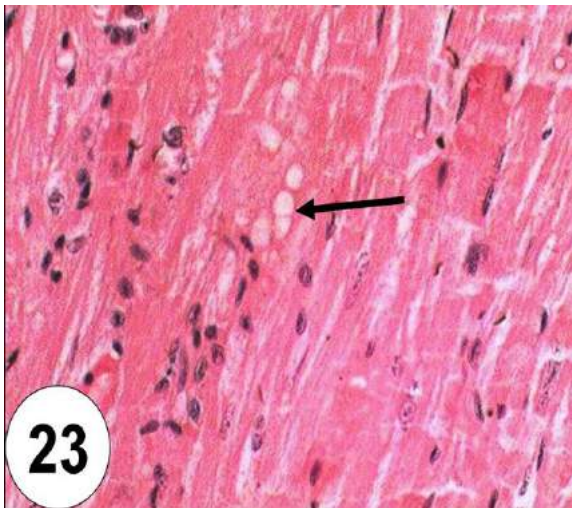

Fig.23: Heart of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Normal myocardial muscle with minute glycogenic vacuolations (Arrow). H and E. X 400.

The spleen was slightly enlarged and contained hyperplastic lymph follicle with large thick walled follicular arterioles (Fig. 24).

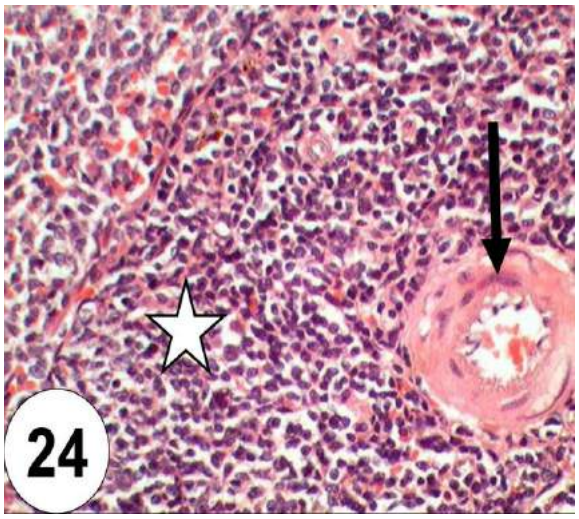

Fig.24: Spleen of rat received 4000 ppm AA + 150 ppm Pb acetate in drinking water: Enlarged and hyperplastic lymph follicle (White asterisk) with large thick walled follicular arteriole (Arrow). H and E. X 400.

DISCUSSION

The present experimental study was carried out to evaluate the histopathologic changes related to the protective effect of supplementation with AA, against intoxication with Pb acetate in male rats. The experiment extended for 3 months during which a concurrent daily supplementation for each of two levels of AA with Pb acetate. Necropsy and histopathologic examination for the organs of the rats of all groups was done. The detected lesions in the organs of Pb intoxicated groups were similar to those described by Kosnett (2005), Pearce (2007) and Trevor *et al*. (2007), for chronic Pb toxicosis.

In the first protective trial with the low level of AA (2000 ppm) in rats of group 5, the microscopic examination revealed less or somewhat no protective and alleviating effect. Some mild to moderate changes of Pb toxicosis in some organs were seen, comparable to the findings in the control group. The detected lesions in brain tissue (meningeal congestion with cerebral focal gliosis and neuronophagia, cerebellar congestion with nuclear pyknosis and degeneration of the Purkinji cells) were indicative for the less alleviating effect of the low level of AA. These findings in brain tissue are mainly due to the fact that the brain is considered the most sensitive organ to lead exposure (Cecil, *et al*., 2008). The testes also showed some changes (degenerative and necrotic changes in semineferous tubules and interstitial edema. The Cauda epididymis contained some degenerated and immature spermatogonial cells, interstitial edema with congestion and mononuclear cell infiltration. The prostate gland contained highly active and dilated acini, congestion and edema. In the kidneys, glomerular and intertubular congestion with tubular degeneration and nuclear contents of the eosinophilic lead inclusion bodies were also seen. The livers showed some degrees of hepato-cellular degeneration and necrosis, mononuclear cell aggregation and small newly formed bile ductules at the portal area. The intestinal lesions were in the form of epithelial vacuolation submucosal edema, congested capillaries and infiltration with eosinophils. The lungs showed peribronchiolar changes of congested blood vessels and some mononuclear cell infiltration. The myocardial muscle showed congested intermuscular blood vessels and capillaries. The splenic follicles were small, atrophied and accompanied by depletion of the lymphocytes. The detected adverse effects in some of the examined organs are in agreement to some extent with the report of Patra *et al*., (2001) who stated that rats treated with AA did not reduce lead burden in the liver, kidney, brain, and blood. These negative results may be attributed to the less or no effect of treatment with the low levels of AA. Although it is biologically plausible that AA may affect lead absorption and excretion, Flora *et al*. (2008) reported that the effect is more obvious in low-exposed subjects with higher AA supplementation.

In the second protective trial with the high level of AA the microscopic examination revealed an obvious alleviating effect comparable to the other experimental group of treatment with the low level of AA and other

groups. This alleviating effect in the nervous tissue was manifested by cerebral activation with astrocytic glial cell reaction and normal cerebellar Purkinje cells. In addition to this alleviating antioxidant effect of the high level of AA, these findings support the report of Khanna and Nehru (2007) who mentioned that the cerebral glial cells and neuron are capable, to some extent of handling the oxidative stress due to the exposure to Pb. The testes, epididymis, prostate glands and seminal vesicles appeared normal and associated with changes of active secretory functions in these organs. These finding in the male genital organs are indicative for the ameliorating effect of this high level of AA on the known testicular and male reproductive lesions for lead poisoning (Grant, 2009). The renal glomeruli were also active and apparently enlarged and congested, while the proximal tubules were with nearly normal epithelium. The intestinal mucosa was apparently normal with intact covering epithelium. In the liver, some reactions of mild hepatocytic degeneration, dilated veins and some newly formed bile ductules were seen. The pulmonary bronchioles were normal but surrounded by congested blood vessels. The spleen contained enlarged and hyperplastic lymph follicle with large and thick walled follicular arterioles. These obtained results are partially in agreement with the findings of El-Neweshy and El-Sayed (2010). They mentioned that the trial for treatment of Pb poisoning (20 mg/kg.b.w.) by supplementation with low level of Vitamin C (20 mg/kg.b.w.) diminished the severity of pathological changes and reduced the number of affected organs comparable to lead-intoxicated rats. These findings of the alleviating effect on various body organs are mainly related to the effect of the higher level of AA on lead absorption and excretion (Flora *et al*., 2008). In conclusion, the supplementation with AA in this study was found to produce an appreciable improvement in the architecture of body organs, especially in case of treatment with high levels.

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التقييم المجهرى لدور حمض الأسكوربيك الوقائى ضد التغيرات التسممية للرصاص فى ذكور الجرذان

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الملخص

أجريت هذه الدراسة بهدف فحص وتوضيح التغيرات المجهرية المتعلقة بالتأثير الوقائى لإضافات حمض الأسكوربيك (فيتامين ج) بمعدلين (2000 و 4000 جزء فى المليون فى ماء الشرب) وذلك على التأثير التسممي لخلات الرصاص على أعضاء الجرذان. ولقد استخدم فى هذه الدراسة عدد (180) من ذكور الجرذان البالغة التي تم توزيعها عشوائيا على 6 مجاميع بكل منها عدد 30 جرذًا. ولقد استمر التعاطي المتزامن لكلٍ من معدلي حمض الأسكوربيك مع التسمم بالرصاص وكذا باقي المعاملات لمدة 3 شهور؛ حيث تم فى نهاية التجربة إجراء التشريح المرضي وجمع عينات الأنسجة والفحوصات المجهرية. ولقد دل الفحص المجهرى لمجموعة المحاولة الأولى لعلاج آثار التسمم بالرصاص (بالمعدل المنخفض لحمض للأسكوربيك) على وجود تأثير وقائى بسيط أو منعدم، حيث لوحظ وجود تغيرات تسممية بسيطة أو متوسطة ببعض الأعضاء. أما بخصوص الفحص المجهري لمجموعة المحاولة الثانية لعلاج آثار التسمم بالرصاص (بالمعدل المرتفع لحمض الأسكوربيك) على وجود تأثير وقائي وعلاجي واضح بمختلف أعضاء الجسم. ولقد خلصت هذه الدراسة إلى أن حمض الأسكوربيك له تأثير واضح فى تحسين الحالة التركيبية والوظيفية لأعضاء الجسم، وخاصة فى حالة المعدلات المرتفعة (التي تصل إلى معدل 4000 جزء في المليون أو أكثر بماء الشرب) على التسمم بالرصاص.

الكلمات المفتاحية: التسمم بالرصاص، خلات الرصاص، السمية، فيتامين "C"، مضادات الأكسدة.