Possible Protective Effect of Vitamin E on The Joined Cardio-Renal Effects of Lead Toxicity and Noise Stress in Rats

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**ABSTRACT:** Pollution is a worldwide problem and its potential to influence the physiological systems of human population is great. Many studies found that some pollutants have detrimental effects on human growth, particularly prenatal growth. Lead is one of the heavy metals commonly found in human populations. Lead toxicity was reported to be related to haemopoietic, hepatic, renal, nervous, gastrointestinal and reproductive disorders in man and animals. Recent studies have reported lead's potential for inducing oxidative stress which plays a role in the pathophysiology of lead toxicity. Noise stress from transportation sources also is related to different physiological effects on human, as well as impairment of auditory function and growth. Studies showed that combined exposure to noise and different pollutants affects heart and other organs. On the other hand, antioxidants were reported to reduce incidence to various physiological alterations accompanied by oxidative stress. Previous studies suggested that antioxidants may play an important role in abating some hazards of lead. The aim of the present work was to evaluate the possible ameliorating effect of vitamin E, as an antioxidant, on some alterations caused by lead or noise alone or in combination on kidney and heart biomarkers in adult male rats. Vitamin E was administered orally in a dose of 200 mg/kg two hours before lead dosage (15 mg/kg, i.p). Animals were exposed to horn noise of about 110 dB for 30 minutes after administration of lead and/or vitamin E. The experiment was conducted for 14 consecutive days. The evaluated parameters included assessment of serum levels of urea, creatinine, Aspartate aminotransferase (AST), creatine kinase (CK) and lactate dehydrogenase (LDH) and levels of reduced glutathione (GSH) and malondialdehyde (MDA) in kidney and heart tissues. The histopathological changes in both kidney and heart were reported. The obtained results revealed that lead and noise either alone or in combination caused statistically changes in the examined parameters with a good potential for vitamin E to protect against these changes.

**Key words:** Lead toxicity- Noise- Vitamin E- Kidney- Heart

**INTRODUCTION:**

Lead (Pd) is ubiquitous and one of the earliest metals discovered by the human. Lead poisoning has been recognized as a major public health risk, particularly in developing countries (Gurer & Ercal, 2000). Though various occupational and public health measures have been undertaken in order to control lead exposure, cases of lead poisoning are still reported. Human exposure to lead occurs through various sources like leaded gasoline, industrial processes such as lead smelting and coal combustion, lead-based paints, lead containing pipes or lead-based solder in water supply systems, battery recycling, grids and bearings, etc. Lead has been shown to affect virtually every organ and system in the body in humans and animals. The most sensitive effects of lead appear to be hepatic, neurological (particularly in children), hematomal, renal and cardiovascular (ATSDR, 2007 and Adeyemi, et al., 2009).

Many epidemiological studies have found an association between exposure to Pb and risk of hypertension. Animal data demonstrate that oral exposure to lead increases blood pressure. At higher levels of exposure in humans, lead produces cardiac lesions and electrocardiographic abnormalities. Other major disorders include ischemic coronary heart disease, cerebrovascular accidents and peripheral vascular disease. Mechanisms by which lead might affect blood pressure include effects on several hormonal and neural regulatory systems, changes in vascular smooth muscle reactivity, cardiac muscle contractility, changes in cell membrane cation transport systems, and possible effects on vascular endothelial cells (Patra & Sarp, 2004 and Navas-Acien et al., 2007).

Chronic nephropathy in humans is associated with blood Pb levels of 40–100 µg/dl. Exposure to high levels of Pb can produce renal tubular damage with glucosuria and aminoaciduria (Gidlow, 2004). Histopathology of kidney of rats exposed to Pb showed kidney tubule lumen dilatation, inflammation and kidney cells degeneration (Aziz et al., 2012). Renal functional abnormalities can be of two types: acute nephropathy and chronic nephropathy. Chronic nephropathy is much more severe than acute one and can lead to irreversible functional and morphological changes. It is characterized by glomerular and tubulointerstitial changes, resulting in renal breakdown, hypertension and hyperuricemia (Flora et al., 2012).

Several mechanisms are proposed for Pb-induced oxidative stress include: 1) direct effect of Pb on cell membrane via generation of reactive oxygen
species. 2) Pb-hemoglobin interaction. 3) aminolevulinic acid-induced generation of reactive oxygen species. 4) direct effect of Pb on the antioxidant defense systems of the cells (Fig. 1) (Ercal et al., 2001 and Flora et al., 2012).

Noise, defined as ‘unwanted sound’, disturbs the human beings physically and physiologically causing environmental pollution and producing reactive oxygen species (ROS) that cause damage to all vital organs (Manikandan et al., 2005). Noise was reported to not only cause hearing loss but also leads to several physiological disorders. Exposure to noise was reported to increase the heart rate and blood pressure, peripheral vasoconstriction and thus increase peripheral vascular resistance (Van Kempen et al., 2002). Aircraft and road traffic noise exposure are associated with psychological symptoms but not with clinically defined psychiatric disorder. In both industrial studies and community studies, noise exposure is related to raised catecholamine secretion. In children, chronic aircraft noise exposure impairs reading comprehension and long-term memory and may be associated with raised blood pressure (Stansfeld & Matheson, 2003).

On the other hand, Vitamin E is the primary liposoluble antioxidant, which may have an important role in scavenging free oxygen radicals and in stabilizing the cell membranes, thus maintaining its permeability. Vitamin E may also affect oxidative changes which occur in other cell organelles. Moreover, it is known that antioxidants, such as Vitamin E, coenzyme Q, vitamin C, glutathione and selenium may act synergically, preventing lipid peroxidation and cell destruction (Rimm et al., 1993; Chaurasia & Kar, 1997 and Valko et al., 2007).

**Abbreviations:** (ROS; reactive oxygen species; GSH: reduced glutathione; GST: glutathione-S-transferase; SOD: superoxide dismutase; GPx: glutathione peroxidase).

**MATERIALS AND METHODS:**

**Animals:**

Adult male albino rats of Sprague-Dawely strain were utilized, weighing about 120±10 gram. They were obtained from the animal house of the National Organization for Drug Control and Research (NODCAR). The animals were caged as eight per cage, and kept under standard laboratory animal housing conditions with water and conventional diet supplied ad libitum. Animals were kept along one week for accommodation before exposure to the treatment.

**Drugs:**

Pb was obtained in the form of lead acetate as a powder from Loba Chemie, India. Pb was prepared just before administration in 0.9% saline solution, which was used as a vehicle, in a dose of 15 mg/kg body weight. Pb was given by intra-peritoneal route daily for fourteen consecutive days.

Vitamin E was obtained in the form of oil from GlaxoSmithKline pharmaceuticals. It was suspended in corn oil just before administration in a dose of 200 mg/kg body weight. It was orally administered for 14 successive days two hours before either Pb dosage or noise exposure.

Animals were exposed to horn noise of about 110 dB for 30 minutes along 14 consecutive days.

**Experimental design:**

Animals were divided into eight groups classified as following:-

<table>
<thead>
<tr>
<th>Subgroups</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>group (1)</td>
<td>Control, received 0.9% saline.</td>
</tr>
<tr>
<td>group (2)</td>
<td>Vehicle, received corn oil and 0.9% saline.</td>
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<tr>
<td>group (3)</td>
<td>Received Lead acetate (15 mg/kg/day, i.p.).</td>
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<tr>
<td>group (4)</td>
<td>Exposed to noise (110 dB for 30 min. /day).</td>
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<tr>
<td>group (5)</td>
<td>Received Vit. E (200 mg/kg/day, P.O.) two hours before lead acetate dosage (15mg/kg/day, i.p.).</td>
</tr>
<tr>
<td>group (6)</td>
<td>Received lead acetate (15 mg/kg/day, P.O.) before exposed to noise (110 dB for 30 min. /day).</td>
</tr>
<tr>
<td>group (7)</td>
<td>Received Vit. E (200 mg/kg/day, P.O.) before exposed to noise (110 dB for 30 min. /day).</td>
</tr>
<tr>
<td>group (8)</td>
<td>Received Vit.E (200 mg/kg/day, P.O.) two hours before lead acetate dosage (15 mg/kg/day, i.p.), then exposed to noise (110 dB for 30 min. /day).</td>
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At the end of the experiment animals were sacrificed. Blood samples of each animal were withdrawn from the retro-orbital plexus 24 hours after the last administered dose (Schermere, 1967).

Blood was subdivided into two portions. The first portion was allowed to coagulate and then centrifuged to obtain serum required for biochemical investigations for kidney and heart enzymes. To assess hematological parameters, the other portion of blood was mixed with disodium salt of ethylene diamine tetra-acetic acid solution.

The needed organs of each animal were removed, washed in saline, dried and weighed to assess their relative weights. Finally, they were divided into two equal groups. The first group was kept in – 80 °C refrigerator in order to prepare tissue homogenates to assess GSH and MDA levels, while the second group
was preserved in 10% formalin solution for the histological assessments. The histological examination was performed by histopathologists in the histology division in the National Organization for Drug Control and research (NODCAR).

**Evaluated parameters**

Estimation of blood urea and creatinine level was evaluated according to the methods of Kaplan et al. (1984) and Murray, (1984), respectively. Serum AST levels were assessed by Reitman & Frankel (1957) method, while CK and LDH were performed using an optimized standard method according to the recommendations of the Deutsche Gesellschaft für Klinische Chemie (DGKC, 1977) and (Weisshaar et al., 1975).

GSH level was measured in kidney and heart tissue homogenates according to Beutler et al., (1963). Determination of MDA was determined according to the method of Buege and Aust (1978) with a slight modification in the incubation period according to Deniz et al., (1997).

Each animal's body weight was recorded before sacrifice and then its heart and kidney were removed and weighed. The mean ratio between each organ and body weight in every group of animals was then calculated. Then, samples from the kidney and heart were removed, rinsed in formalin, dehydrated, cleared, impregnated and embedded in paraffin to facilitate ease of cutting for histological assessment of tissue damage using haematoxylin and eosin.

**RESULTS:**

**Biochemical results:** Treatment with Pb significantly elevated serum levels of Urea, Creatinine, AST, CK and LDH, as compared to control group (Fig. 2). In addition, tissue GSH was significantly decreased in heart, with non-significant reduction in kidney tissue. On the other hand, MDA in the tissue of kidney and heart showed significant increase when compared to control values (Fig. 3).

Noise affected the levels of AST and LDH with a significant increase, while, Urea, Creatinine as well as CK levels were non-significantly increased (Fig. 2). In addition, exposure to noise showed significant reduction in GSH level in both kidney and heart tissues with a concomitant increase in MDA levels, as compared to control group (Fig. 3).

Joined exposure to Pb and noise slightly exaggerate the effects of Pb and noise each alone, as GSH level in kidney showed further decrease upon co-exposure to Pb and noise, as compared to Pb alone. Statistically, Urea, Creatinine, AST, CK and LDH showed significant increase, as compared to control group (Fig. 2).

Moreover, joined exposure to Pb and noise showed significant increase in MDA level in kidney and heart, when compared to control group. Also, co-exposure to Pb and noise significantly increased MDA levels in kidney when compared to noise alone group (Fig. 3).

Administration of vitamin E could ameliorate these effects of both Pb and noise either alone or in combination.

**Relative organs weights:** Treatment with Pb significantly decreased the relative weight of kidney with no apparent effect on heart relative weight. On the other hand, exposure to noise significantly elevated the relative heart weight, as compared to control.

Upon Co-exposure to Pb and noise a significant increase in the relative weight of kidney was observed with no significant change in the relative heart weight, when compared to control. That increase in the relative weight of kidney was significantly higher in combination group than noise alone group.

Administration of vitamin E could not correct the changes reported in the relative weights of kidney and heart either in each group alone or in combination.

**Histological manifestation:** Histological examination of kidney tissue of Pb treated group revealed moderate to marked changes in the form of degeneration in the epithelial lining of the renal tubules. The nuclei appeared pyknotic within the cortico-medullary areas. In addition, congestion of blood capillaries and lymphatic infiltration between renal tubules were observed. Moreover, cellular depress appeared in tubular lumen and vacuolated mesangial cells of glomerular tuft were seen (Fig. 5, 6). While, heart tissue examination showed moderate signs of toxicity where focal areas of necrotic cardiomyocytes together with inter hemorrhagic areas and inter cardiac edema (Fig. 13, 14).

Histopathological changes in kidney sections of animals exposed to noise revealed in the form of hyaline cast formation appeared in most lumen of renal tubules. Moreover, marked congestion in blood capillaries in glomerular tuft together with mild scattered shrinkage of glomerular tuft was also seen. No evidence changes in renal tubules (Fig. 9). While the most observed alteration in cardiac tissue was in the form of hemorrhage, edema. This is beside many cardiomyocytes with clear nuclei (Fig. 19).

In animals exposed to Pb and noise, pathological changes in kidney were marked in the form of eosinophilic material deposited in Bowman's capsule in most glomerular tuft and hyaline cast formation deposited in lumen of renal tubules. The epithelial cells lining were damaged, their nuclei appeared large in size with loss chromatin (faintly stained) in some cells and pyknotic nuclei in other cells. In addition, extravasated RBC’s were seen between tubules (Fig. 7). In addition, co-administration of Pb and noise showed more toxic impacts in cardiac tissues of this group. Perivascular inflammatory reaction and interstitial inflammation could be seen. Mast cell occasionally observed, this is besides apoptotic cardiomyocytes (Fig. 15, 16, 17).

Administration of vitamin E was able to diminish the reported pathological alterations observed either in Pb and noise groups alone or in their combination (Fig. 8, 10, 11, 18, 20, 21, 22).

**DISCUSSION:**

Concerning kidney function, the present results were in agreement with that reported by Hanafy & Soltan, 2004; Flora et al., 2004; Farrag et al., 2007; Ghanem et al., 2009; Abdel-Moneim et al., 2011; Alcaraz-
Conteras et al., 2011; Nishanthi & Anuradha, 2012; Abd El-Kader et al., 2012; Osfor et al., 2012; Ghosh et al., 2012 and Bandyopadhyay et al., 2013b who showed significant elevation in urea, creatinine, uric acid and blood urea nitrogen as well as significant increase in MDA level with a concomitant reduction in GSH, CAT, SOD, GPx, GST and GR activities in renal homogenate of Pb intoxicated experimental animals. These biochemical changes were confirmed with the kidney histological examination observed in the present work.

The kidney is an important target that has been affected by Pb. The increment of urea and creatinine along with significant increase in MDA level in the current study indicated kidney dysfunction. The increase in creatinine concentration might be due to loss of kidney function and considered as a functional evidence of Pb induced nephrotoxicity (Qu et al., 2002). Moreover, Pb was reported to produce oxidative damage in the kidney by enhancing lipid peroxidation (Farrag et al., 2007). Also, it induces oxidative damage to the membranes by accumulation of oxidant metabolites and by direct or indirect inhibition of antioxidant enzymes causing a state of oxidative stress (Abd El-Kader et al., 2012). Some studies showed a linear correlation between creatinine levels and blood Pb levels (Longhman-Adham, 1997).

The current histological changes agree with those of Farrag et al., (2007) and Aziz et al., (2012) who reported congestion and hypercellularity along with degeneration of the renal tubules and hemorrhagic areas in the interstitium. Moreover, inflammatory infiltration and degeneration of renal cells were observed.

Moreover, the presence of Pb was reported to cause impairment of the brush border of epithelial cells and making them impermeable to urea and creatinine thereby causing their elevated levels in the blood (Oloyede et al., 2003). Also, Pb was reported to disrupt the glomerular filtration rate causing electrolyte disturbances which lead to hypertension. These effects could in turn affect the cardiac muscle and probable heart functioning (Vaziri & Gonick, 2008).

In the current work, the degenerative effects of Pb on heart agree with those obtained by Ahmed & Hassanain, (2013) who reported that Pb intake resulted in significant increases in cardiac high-sensitivity C-reactive protein, interleukin-6, E-selectin, troponin I, MDA and serum creatine kinase-MB. In addition, GSH, SOD, GPx and cardiac apelin levels were significantly decreased. Similarly, Gonick et al., (1997) reported a significant increase in plasma and tissue markers of oxidative stress in rats exposed to Pb toxicity.

Results of Ghosh et al., (2012) showed a significant increase in AST levels in rats intoxicated with Pb with significant increase in thiobarbituric acid reactive substance and in contrast to other findings the level of GSH, SOD and CAT was increased.

In addition, several studies on human and experimental animals showed that exposure to Pb induced cardiovascular disorders including ischemic coronary heart disease and hypertension (Ekong et al., 2006; Menke et al., 2006 and Vaziri & Gonick, 2008). Moreover, some cases of myocardial infarction and stroke were associated with low blood Pb levels (Menke et al., 2006 and Nawrot & Staessen, 2006).

The reported elevations in CK, LDH, AST and MDA and reduction of GSH in the present study indicates a destructive effect of Pb on cardiac tissue and a state of oxidative stress.

CK is found in high concentrations in heart and skeletal muscles. Increased serum levels indicates trauma of cells. In addition, its increase reflects electrolyte imbalance. Similarly, LDH is elevated in serum during heart infarction.

Although AST is used as indicator for hepatic degeneration and cirrhosis, it was reported to increase in myocardial infarction (Ghosh et al., 2012). The reported destruction in the heart may also be due to the reported tendency of Pb to cause membrane lipid peroxidation leading to lysis and disintegration of many cells as well as altering the mechanical properties of cells (Hochstein & Jain, 1981).

Population studies have demonstrated a link between Pb exposure and subsequent development of hypertension and cardiovascular disease. In vivo and in vitro studies have shown that chronic Pb exposure causes hypertension and cardiovascular disease by promoting oxidative stress, limiting nitric oxide availability, impairing nitric oxide signaling, augmenting adrenergic activity, increasing endothelial production, altering the rennin-angiotensin system, raising vasoconstrictor prostaglandins, lowering vasodilator prostaglandins, promoting inflammation, disturbing vascular smooth muscle Ca2+ signaling, diminishing endothelium-dependent vasorelaxation and modifying the vascular responses to vasoactive agonists. Moreover, Pb has been shown to cause endothelial injury, impede endothelial repair and reduced endothelial cell growth (Vaziri, 2008 and Vaziri & Gonick, 2008).

The destructive disturbances caused by exposure to noise in the present work could be explained under the basis that acute as well as chronic exposure to noise stress can produce excessive free radicals and other reactive oxygen species. The resultant oxidative stress increases the susceptibility to lipid peroxidation leading to membrane damage and destruction of other biological components of the cell (Endo et al., 2005; Manikandan et al., 2005 and Manikandan & Devi, 2005).

Study made by Demirel and colleagues (2009) showed an elevation in MDA level (an indicator of lipid peroxidation), as well as nitric oxide level and GPx activity by noise exposure. They suggested the presence of oxidative stress which may lead to various degrees of damages in cells, mainly via lipid peroxidation pathway, in the noise exposed animals. The reported oxidative stress accompanying noise exposure was reported to affect various organs such as liver, heart and brain (Zhu et al., 2003; Lenzì et al., 2003; Manikandan et al., 2005 and Al-Naemi & Abdal, 2012).
Co-exposure to Pb and noise showed limited effects on the evaluated biochemical parameters as compared to each effector alone, except for urea and CK levels that were higher in the Pb and noise group than noise group alone.

Furthermore, kidney GSH level in Pb and noise combination group was further decreased as compared to Pb or noise groups alone. In addition, MDA level in heart showed higher increase in Pb and noise group when compared with Pb or noise alone.

The histological disturbances observed due to noise exposure either alone or in combination with Pb on both kidney and heart supports the biochemical findings of the present work and agree with those previously mentioned.

Oral administration of vitamin E in a dose of 200 mg/kg body weight two hours prior to treatment with Pb was able to diminish the elevations observed in AST, urea, creatinine, CK and LDH levels returning them to normal control values. Moreover, GSH as well as MDA levels were normalized in the examined organs.

Treatments of rats with vitamin E before exposure to noise was able to lower serum, AST and CK levels that were elevated due to noise exposure and returning them to normal control values. In addition, GSH level in kidney and heart returned to control level, as compared to noise group but still lower than control level. In parallel, MDA levels were significantly decreased to the normal control level in both kidney and heart tissues.

Vitamin E administration showed the same protective potential when given before joined exposure to Pb and noise showing normal levels of the evaluated biochemical parameters as well as for the oxidative stress biomarkers in all the examined organs.

**Conclusion:** It is concluded from the present findings that presence of Pb exaggerated the biological effects caused by noise alone and that vitamin E has a good ameliorating effect against toxicity of Pb and noise alone or in combination at the biochemical as well as cellular levels of kidney and heart. Previous results of Osfor et al., (2010) support the current findings that vitamin E supplementation was reported to be beneficial in reducing and slowing progressive kidney diseases and may be also effective in reducing cardiovascular disease associated with chronic renal failure and the uremia state. Vitamin E therapy is also considered as a mean of correcting plasma antioxidant status and attenuating the cardiovascular disease that accompanies kidney failure.

![Figure (2):](image-url)

**Figure (2):**
Effect of administration of Pb (15 mg/kg, i.p.), exposure to noise (110 dB for 30 min/day) or their combination, as well as the effect of pretreatment with vitamin E (200mg/kg) on either Pb and noise alone, or their combination for 14 consecutive days on serum levels of urea, creatinine, CK, LDH and AST in male rats.

- a Significant difference from control (P<0.05).
- b Significant difference from lead (P<0.05).
- c Significant difference from noise (P<0.05).
- d Significant difference from lead+noise (P<0.05).
Figure (3): Effect of administration of Pb (15 mg/kg, i.p.), exposure to noise (110 dB for 30 min./day) or their combination, as well as the effect of pretreatment with vitamin E (200mg/kg) on either Pb and noise alone, or their combination for 14 consecutive days on tissue levels of GSH and MDA of kidney and heart in male rats.

- a Significant difference from control (P<0.05).
- b Significant difference from lead (P<0.05).
- c Significant difference from noise (P<0.05).
- d Significant difference from lead+noise (P<0.05).

Fig. (4): Photomicrograph of control kidney showing normal glomerulus with its capillary tuft (GT) surrounded by capsular space and Bowman's capsule (arrow). The proximal (PC) and distal (DC) convoluted tubules were also observed (H&E stain, X 640).

Fig. (5): Photomicrograph of kidney sections of Pb-treated group showing hyaline cast inside lumen of tubules (double head arrow), amorphous eosinophilic material observed in Bowman's capsule (thin arrow) and extravasated RBC's (arrow head) (H&E X 400).

Fig. (6): Photomicrograph in kidney section of animals treated with Pb showing few number of lymphocytes (arrow head), hyaline cast in renal tubules (double head arrow) and degenerative changes in epithelial lining of tubules (triple head arrow) (H&E X 640).
Fig. (7): Photomicrograph in kidney section of animals treated with Pb+noise showing marked amorphous eosinophilic materials inside Bowman’s capsule (long arrow) and hyaline material deposited in tubules (arrow head) and large size with faintly stained nuclei (double head arrow) (H&E X 400).

Fig. (8): Photomicrograph in kidney section in animals treated with Pb+vitamin E showing normal appearance of glomerular tuft (arrow) with focally aggregation of inflammatory cells (arrow head) (H&E X400).

Fig. (9): Photomicrograph in kidney section in animals exposed to noise showing shrinking of glomerular tuft (arrow) and extravasated RBC's between tubules (arrow head) (H&E stain, X 400).

Fig. (10): Photomicrograph in kidney section in animals of noise+vitamin E group showing mild dilated, congested blood vessels (arrow), most renal tubules more or less normal seen in areas (arrow head) (H&E X 400).

Fig. (11): Photomicrograph in kidney section in animals of combination group showing focally amorphous eosinophilic material in glomeruli (arrow) (H&E X 400).

Fig. (12): Photomicrograph of heart control tissue showing normal architecture of intact cardiomyocytes (arrow) (H&E stain, X 400).

Fig (13): Photomicrograph of heart tissue of Pb-treated animals showing atrophied cardiomyocytes with pyknotic nuclei, perivascular edema (arrow head) and congested, dilated cardiac vessel (CV) (H&E stain, X 100).

Fig (14): Photomicrograph of heart tissue of Pb-treated animals showing interstitial hemorrhage (arrow), atrophied cardiomyocytes with pyknotic nuclei (double arrow) (H&E stain, X 400).

Fig (15): Photomicrograph of heart tissue of Pb+noise group showing mast cells (arrow) (H&E stain, X 100).
Fig (16): Photomicrograph of heart tissue of Pb+noise group showing mast cells (arrow) and inflammatory cells (arrow head) (H&E stain, X 400).

Fig (17): Photomicrograph of heart tissue of Pb+noise group showing mast cells (arrow) and inflammatory cells (arrow head) (H&E stain, X 400).

Fig (18): Photomicrograph of heart tissue of Pb+vitamin E group showing many intact cardiomyocytes (arrow head), interstitial edema (arrow) and mild inflammation (double arrow) (H&E stain, X 400).

Fig (19): Photomicrograph of heart tissue of noise group showing pericardium edema (arrow head), interstitial hemorrhage (H) and clear nuclei of cardiomyocytes (arrow) (H&E stain, X 400).

Fig (20): Photomicrograph of heart tissue of noise+vitamin E group showing cardiomyocytes with pyknotic nuclei (arrow) and separated cardiac fiber (arrow head) (H&E stain, X 400).

Fig (21): Photomicrograph of heart tissue of combination treated group showing intact cardiomyocytes (arrow head), inflammatory cells (arrow) and congested cardiac vessel (CV) (H&E stain, X 100).

Fig (22): Photomicrograph of heart tissue of combination treated group showing perivascular edema (arrow) and intact cardiomyocytes (arrow head) (H&E stain, X 400).

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دراسة التأثير الوقائي المحتمل لفيتامين هـ على الآثار الناتجة من تداخل سمية الرصاص مع الضوضاء على
كلاً من القلب والكلى


