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The Role of Vitamin E in Reducing the Toxic Effects of Lead Poisoning in Baghdad, Iraq

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Abstract

The incidence of lead poisoning has risen in Iraq because of increasing environmental pollution, especially in Baghdad. In Baghdad, the growing number of motor vehicles and the emission of pollutants, including lead, have contributed to increased environmental contamination and lead exposure.as well as waste generated by private laboratories that fail to meet basic environmental and public health safety standards. The study included forty adult male rats, divided into four groups: a control group, a group treated with lead acetate (400 ppm), a group that received vitamin E (30 ppm) with a certain percentage of body weight, and a group treated with both lead acetate and vitamin E. The treatment duration was 60 days, and hematological and biochemical indicators were assessed to study the effects of lead exposure and the protective role of vitamins. Lead acetate administration induced marked hematological and biochemical disturbances in experimental rats. Changes in blood cells were marked by significant reduction of red blood cell (RBC) count, hemoglobin (Hb) level and packed cell volume (PCV), and significant increase in total white blood cells count and neutrophils, and significant reduction in lymphocyte and monocyte count. There was no significant difference between the hematocrit (HCT) values of the experimental groups. Biochemical tests showed that serum total protein, albumin and globulin levels were significantly decreased with exposure to lead acetate, while serum cholesterol levels and alanine aminotransferase (ALT) activity were significantly increased. Although there was a modest increase in the activity of aspartate aminotransferase (AST) and alkaline phosphatase (ALP), these changes were not statistically significant. However, administration of vitamin E proved to be effective in reducing most of the hematological and biochemical changes caused by lead acetate indicating its antioxidant and protective effect against lead-induced toxicity. The present study confirms that lead acetate exposure causes a significant alteration of hematological and biochemical parameters in rats which is a direct indication of its toxic effects on hematological parameters and liver function. Most of these harmful changes were effectively reduced with vitamin E supplementation which makes vitamin E a good antioxidant and protective agent against the adverse effect of oxygen free radicals caused by lead. Based on the present results, vitamin E could be a promising therapeutic agent against the adverse effects of lead toxicity.

Keywords
Vitamin E,
Lead,
Toxicity,
Toxic Effects,
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1.Introduction

Lead (Pb) is recognized as one of the most toxic environmental heavy metals, lacking any known physiological role in the human body and exhibiting a strong propensity to accumulate in soft tissues and bones. Lead exposure is a serious public health problem in both the workforce and the environment, especially for women of reproductive age, industrial workers and children, particularly in developing and post-conflict nations. The toxic effects of lead on the central nervous system (CNS), kidneys, blood-forming system, reproductive, and liver systems are mainly due to its role in the formation of reactive oxygen species (ROS) and disturbances in the oxidant–antioxidant balance of cells (2.3).

Industrial activities have resulted in long-term lead contamination of the air, water and workplaces in Iraq, especially in Baghdad, due to the absence of adequate environmental control standards and decades of armed conflict. The workers occupationally exposed to lead and cadmium were found to have a significant decrease in the activity of the enzyme δ-aminolevulinic acid dehydratase (δ-ALAD) along with increased urinary 8-hydroxy-2′-deoxyguanosine (8-OHdG) levels, which is a well-established biomarker of oxidative DNA damage, when compared with unexposed control (4).

Likewise, the blood lead concentrations of occupationally exposed people have been reported as high in other Iraqi governorates. For instance, a cross-sectional study by A. Al-Hassani and colleagues in 2006 revealed that a large proportion of workers in petroleum refinery in Kirkuk had BLL above the reference value of 10 μg/dL (5) and blood lead concentration in chronic kidney disease patients in Iraq was significantly higher than in healthy participants, and there was a negative correlation between BLL and renal function (6).

Recently, a cross-sectional study was conducted in Basrah which revealed that maternal blood lead levels were significantly correlated with cord blood lead levels, negatively correlated with iron status in the newborn and birth weight indicating transplacental transfer and potential health risk of lead exposure among the Iraqi population (7). Lead toxicity is mainly manifested by its oxidative effects. Lead increases lipid peroxidation, reduces glutathione levels in cells, decreases the activity of important antioxidant enzymes, such as superoxide dismutase (SOD) and catalase (CAT), and affects the activity of δ-aminolevulinic acid dehydratase (δ-ALAD), which is an important enzyme in heme biosynthesis (2). This oxidative damage causes injury to the membranes, proteins and DNA of cells throughout the body, causing widespread damage to other organ systems such as the liver, kidney, brain and reproductive system (2.3).

This mechanism has made antioxidant supplementation a hot topic for research as a possible way to reduce lead-induced damage. Vitamin E (α-tocopherol) is a lipid-soluble, chain-breaking antioxidant that protects cellular membranes from oxidative damage and has been described as one of the most effective naturally occurring antioxidants against free-radical-mediated pathology (8). Experimental studies have repeatedly

demonstrated the protective efficacy of vitamin E against lead-induced toxicity. Concurrent administration of vitamin E during lead exposure has been shown to prevent the inhibition of δ-aminolevulinic acid dehydratase (δ-ALAD) activity and reduce lipid peroxidation in rat erythrocytes, with partial protection observed even when vitamin E was administered before or after lead exposure. Furthermore, co-administration of vitamin E with lead acetate in male rats significantly decreased malondialdehyde (MDA) levels and restored superoxide dismutase (SOD) activity in reproductive tissues, highlighting its antioxidant and cytoprotective effects against lead-induced oxidative damage (9).

Recent evidence supports the protective role of vitamin E against lead-induced toxicity. A 2025 experimental study demonstrated that combined treatment with vitamin E, selenium, and *Astragalus* extract significantly attenuated lead-induced apoptosis and oxidative damage in rat ovarian tissue. Furthermore, a comprehensive 2026 review of experimental animal studies concluded that vitamin E, either alone or in combination with vitamin C and other natural antioxidants, effectively counteracts lead-induced oxidative stress and mitigates the associated hematological, biochemical, and histopathological alterations.

2.Study design and Methodology

Forty mature male rats were used in the research in four groups namely a control group, a group treated with vitamin E 30mg/ kg body weight, a group treated with lead acetate 400ppm and a group treated with both lead acetate and vitamin E. Treatment lasted for 60 days and assessment was done to assess the effect of lead exposure and the protective effect of vitamin E.

3.Results

Table (1) shows the effect of lead acetate and vitamin E on Hematological parameters

parameter	C	T1	T2	T3
RBC	6.99±o.22	5.08±0.16	7.74±0.22	6.31±0.09
HB	11.98±0.19	10.80±0.22	13.20±0.42	12.26±0.24
PCV	35.62±2.21	29.18±0.63	35.50±0.71	32.34±0.71
WBC	6.93±0.15	8.07±0.25	6.80±0.10	7.44±0.13
Neutrophil	31.08± 0.66	36.44±0.41	30.81±0.63	33.54±0.29
lymphocytes	62.97±0.61	58.17±0.81	63.12±0.53	60.76±0.36
Eosinophils	2.05±0.18	2.56±0.10	1.82±0.26	2.18±0.16
Monocytes	3.88±0.24	2.99±0.06	4.24±0.44	3.61±0.20

Table (1) shows the effect of lead acetate and vitamin E on hematological parameters of the experimental animals across the control (C) and three treated groups (T1, T2, T3).

Erythrocytic parameters (RBC, Hb, PCV): The control group recorded RBC at 6.99±0.22×10^6/µL, Hb at 11.98±0.19 g/dl, and PCV at 35.62±2.21%. Group T1 (lead acetate only) showed a marked decline in all three parameters (RBC 5.08±0.16, Hb 10.80±0.22, PCV 29.18±0.63), reflecting the anemia typically induced by lead toxicity. who reported that lead acetate exposure induced macrocytic hypochromic anemia in rats, with significant reductions in RBC count, hemoglobin, and hematocrit. In contrast, groups T2 and T3 (lead acetate + vitamin E) showed clear recovery, with T2 recording the highest values (RBC 7.74±0.22, Hb 13.20±0.42, PCV 35.50±0.71) — exceedingly even the control — and T3 showing intermediate improvement (RBC 6.31±0.09, Hb 12.26±0.24, PCV 32.34±0.71).

Total leukocyte count (WBC): It was observed that WBC was elevated in T1 (8.07±0.25) when compared with control (6.93±0.15), which is consistent with the finding of Emam et al. (2023) who found elevated level of WBC after administration of lead acetate, which is an indication of leukocytosis resulting from the lead induced tissue damage. The counts of the leukocytes in T2 (6.80±0.10) and T3 (7.44±0.13) values were not far from control indicating the normalisation of leukocyte count by vitamin E. Differential leukocyte count (neutrophils, lymphocytes, eosinophils, monocytes):

There was an increase in neutrophils in T1 (36.44±0.41) compared to control (31.08±0.66), and a decrease in lymphocytes (58.17±0.81) compared with control (62.97±0.61). who reported that lead acetate exposure caused leucocytosis, eosinophilia, monocytosis, and neutropenia-related disturbances in the differential count of Wistar rats that were reversed by antioxidant co-treatment. Eosinophils increased slightly in T1 (2.56±0.10) versus control (2.05±0.18), while monocytes decreased (2.99±0.06 vs. 3.88±0.24). In groups T2 and T3, these differential counts shifted back toward control values (e.g., lymphocytes 63.12±0.53 and 60.76±0.36, respectively; neutrophils 30.81±0.63 and 33.54±0.29), supporting the immunomodulatory and hemato protective role of vitamin E.

Overall, lead acetate exposure alone (T1) induced anemia (↓ RBC, Hb, PCV), leukocytic disturbance (↑ WBC, neutrophils; ↓ lymphocytes, monocytes), consistent with previous reports on lead-induced hemato toxicity. Co-administration of vitamin E (T2, T3) ameliorated most of these hematological disturbances, with T2 showing the strongest protective effect, supporting the antioxidant-mediated hemato protective role of vitamin E against lead acetate toxicity.

Table (2) shows the effect of lead acetate and vitamin E on biochemical parameters

Parameter	c	T1	T2	T3
Total protein(g/100ml)	6.53± 0.28	5.89± 0.24	7.51±0.31	6.13±0.31
Albumin(g/100ml)	4.33 ± 0.17	3.11± 0.04	5.10±0.31	4.05±0.17
Globulin (g /100ml)	2.19 ± 0.10	2.78± 0.26	2.41±0.13	1.92 ±0.16
cholesterol(g/100ml)	61.93±11.6	78.81±11.6	51.41±1.2	71.17±2.8
ALT(IU/L)	10.38± 0.51	8.78 ±11.6	8.78± 1.19	11.54 ± 1.3
AST(IU/L)	6.68 ±0.87	9.32 ± 0.96	7.94±2.10	8.86 ± 1.06
ALP(IU/L)	28.88 ±0.61	30.94 ±0.61	25.66±2.6	28.30± 1.6

Table (2) illustrates the effect of lead acetate and vitamin E on the biochemical parameters of the experimental animals compared with the control group (C) and the three treated groups (T1, T2, T3).

Total protein, albumin, and globulin: The control group recorded a total protein value of 6.53±0.28 g/100ml, albumin at 4.33±0.17 g/100ml, and globulin at 2.19±0.10 g/100ml. Group T1 (lead acetate only) showed a marked decrease in total protein (5.89±0.24) and albumin (3.11±0.04), while globulin increased slightly (2.78±0.26). who reported that oral lead acetate exposure in male albino rats significantly decreased plasma total protein and albumin while altering globulin content. Groups T2 and T3 (lead acetate + vitamin E), however, showed clear improvement, with T2 recording the highest total protein (7.51±0.31) and albumin (5.10±0.31) values — even exceeding the control — while T3 showed intermediate improvement (6.13±0.31 and 4.05±0.17, respectively).

Cholesterol was elevated in T1 (78.81±11.6) compared with control (61.93±11.6). T2 showed the lowest cholesterol value (51.41±1.2) and T3 was closer to control (71.17±2.8), suggesting that vitamin E supplementation helped normalize the lipid profile disturbed by lead exposure.

Liver enzymes (ALT, AST, ALP): AST increased in T1 (9.32±0.96) relative to

control (6.68±0.87), and ALP was highest in T1 (30.94±0.61), who reported significant increases in plasma ALT, AST, and ALP in male rats exposed to heavy metals including lead, and found that vitamin E supplementation attenuated these enzyme elevations through its antioxidant activity. The present study revealed that treatment with T2 (AST 7.94±2.10, ALP 25.66±2.6) and T3 (AST 8.86±1.06, ALP 28.30±1.6) helped to bring the parameters closer to control level, indicating hepatoprotective effect of vitamin E.

Overall, the biochemical disturbances that were observed in T1 (decreased total protein and albumin, increased cholesterol, and liver enzyme activities) indicate classic hepatotoxic effects of lead and decreased protein synthesis, which they attributed to lead-induced oxidative stress and antioxidant defense depletion (such as SOD). The amelioration of these parameters in groups T2 and T3, particularly T2, supports the protective, antioxidant role of vitamin E against lead-induced toxicity.

4.Discussion

4.1 Hematological parameters

The present study demonstrated that lead acetate significantly reduced RBC count, hemoglobin concentration, and packed cell volume in group T1 compared with the control group, consistent with the development of anemia. This effect is primarily attributed to impaired heme biosynthesis resulting from inhibition of δ-aminolevulinic acid dehydratase (ALAD) and ferro chelatase in addition to oxidative injury to erythrocyte membranes, which reduces red blood cell lifespan and impairs erythropoiesis .These findings are supported by a recent experimental study, which demonstrated that lead acetate significantly reduced RBC count, hemoglobin concentration, packed cell volume, and bone marrow cellularity, whereas vitamin E supplementation markedly ameliorated these hematological alterations. Likewise, a recent experimental study demonstrated that lead acetate induced marked anemia and bone marrow injury through oxidative stress, resulting in decreased erythrocytic indices. (11).

In the present study, vitamin E supplementation effectively restored erythrocytic parameters, with group T2 exhibiting RBC count, hemoglobin concentration, and packed cell volume values that exceeded those of the control group. This beneficial effect is primarily attributed to the potent antioxidant properties of vitamin E (α-tocopherol), which protect erythrocytes from oxidative damage, maintain membrane stability, and preserve their structural and functional integrity), which stabilizes erythrocyte membranes by preventing lipid peroxidation, reducing hemolysis, and preserving normal erythropoiesis These observations are consistent with a recent review demonstrating that vitamin E is among the most effective antioxidants in mitigating lead-induced oxidative damage. (12)

4.2 Total and Differential Leukocyte Count

Total leukocyte count and neutrophil percent were significantly elevated following exposure to lead acetate and the lymphocyte percent was markedly decreased. The changes are associated

with activation of inflammatory response mechanisms, which occurs as a consequence of excessive production of reactive oxygen species (ROS) and tissue damage caused by lead toxicity, leading to leukocytosis and neutrophilia, and oxidative stress to reduction of lymphocyte proliferation and function. This result is similar to that of Ojeka (13). Vitamin E supplementation brought the levels of total and differential leukocytes closer to normal levels, which may be attributed to its potent antioxidant and anti-inflammatory properties, such as its ability to scavenge ROS, decrease production of pro-inflammatory cytokines, preserve immune cells from oxidative damage and inhibit NF-κB signalling, thus reducing the inflammatory responses induced by lead (13.14).

4.3 Serum proteins

Serum total protein and albumin were significantly reduced with lead acetate and globulin was slightly increased. A decrease in albumin is a sign of decreased hepatic protein synthesis due to hepatocellular injury; an increase in globulin could be a sign of activation of immune responses related to chronic inflammation. These results are similar to recent studies which showed that serum total protein and albumin were significantly decreased due to lead induced oxidative stress and supplementing with vitamin E significantly reversed the effect on protein synthesis in the hepatic cells, especially in T2. This is an improvement resulting from the restoration of hepatocellular integrity and hepatic synthetic capacity, possibly through decreased oxidative stress and improved membrane stability (15.16).

4.4 Cholesterol

The increase of serum cholesterol levels was significant in the lead acetate group. The supplementation of Vitamin E significantly decreased serum cholesterol level, especially in group T2, suggesting the repairing of normal hepatic lipid metabolism in this group. Recent studies have shown antioxidants to be effective in correcting lipid profile disturbances caused by lead toxicity, which is similar to the results obtained here. (17.18)

4.5 Liver enzymes

In the lead acetate treated rats, the observed increase in AST and ALP activities in the serum suggests that there is hepatocellular injury and increase in membrane permeability that allows the release of intracellular enzymes into the blood. Oxidative stress induced by the lead accumulation in cells may lead to lipid peroxidation, mitochondrial dysfunction, inflammatory reaction and necrosis of the cells of the liver. The results are in line with previous data showing that serum activities of ALT, AST and ALP were significantly elevated, and that there was increased expression of the same in liver tissue following exposure to lead acetate along with increased oxidative stress, production of inflammatory cytokines, expression of heat shock proteins and activation of NF-kB. The supplementation of vitamin E was also found to significantly reduce this biochemical change. The results seen in group T2 and T3 are proof of the hepatoprotective activity of Vitamin E as it inhibits lipid peroxidation, protects the integrity of the hepatocellular membranes, strengthens the endogenous antioxidant defense mechanisms, and

weakens inflammatory signaling pathway (18).

5. Conclusion

Together, the present results indicate that lead acetate causes severe hematological and biochemical disorders via oxidative stress – mediated mechanism. Anemia, inflammatory leukocytosis, decreased protein synthesis by the liver, dyslipidemia, and hepatocellular injury were all caused by lead toxicity. These toxic effects were significantly reduced by vitamin E, the best effect being seen in group T2. Vitamin E prevents the harmful effects of ROS, blocks lipid peroxidation, protects the cell membranes, boosts cell antioxidant defenses, and reduces inflammatory signaling pathways. The obtained results are consistent with the recent experimental studies, which suggest the applicability of vitamin E as a potent antioxidant against hematotoxicity and hepatotoxicity caused by lead.

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