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Hematological and biochemical study on effect of protective agents against lead toxicity in albino rats

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Abstract: *In The present work investigated some parameters in blood serum of albino rats. The daily oral administration of lead acetate at a dose of 20 mg/kg for 20 days caused significant increase in mean corpuscular hemoglobin (MCH) white blood cell count (WBC) and blood platelets (PLT). In contrast significant decrease in red blood cell count (RBC), hemoglobin (HB), hematocrit (HCT),mean corpuscular volume (MCV), mean corpuscular hemoglobin concentrations (MCHC),was recorded during all the experimental periods. In addition, the daily oral administration of lead acetate for 20 days caused general increase of albumin, urea, uric acid , creatinine, cholesterol and triglycerides in rat's blood serum compared to the control level. Moreover, a significant decrease in total protein, globulin, ALT and AST activity was observed after 20 days of lead acetate. Signs of improvements in the previous hematological parameters were noticed after oral administration and biochemical of vitamin C or vitamin E with/without zinc sulphate during lead acetate administration.*

Keywords*: Lead acetate – vitamin C - vitamin E - hematological and biochemical parameters – albino rats.*

Introduction

Lead is a dangerous heavy metal which is widely spread in the environment. Lead content in the air, food and tap water has increased several folds during recent years due to extensive use of this metal in petrol, paints, battery and other industries (Tuormaa, 1995). Despite of attempts for reducing the exposure to this metal, there are still some reports of cases with severe lead toxicity (Hershko et al, 2005).

On the other hand, chronic lead poisoning is a problem which threats mankind's life and seems to be an unknown reason for some diseases during aging (Coyle *et al 2005,* and Vig & HU, 2000).

The toxic effects of lead on blood indices are well known. Significant decrease in RBC count, hematocrit (Hct) and hemoglobin (Hb) were seen in rats and human with high blood lead levels. (Hofmann & Segewitz, 1975; Petering, 1977 Alexa *et al.*, 2002; ; Noori *et al*., 2003 and Klauder; &Othman *et al*., 2004; Toplan *et al*., 2004).

Lead is considered as pathogenic factor of atherosclerosis, arterial hypertension and may cause an anemia (Tendon et al., (1992; Lima magi and Kassik, 1995; Belacy et al,. 1996.) reported that lead induced inhibition of renal and hepatic transaminase and alkaline phosphates, on the other hand several studies have led to reports that lead has effects on glucose utilization at low levels resulting in disturbed acetylcholine synthesis and energy metabolism, Yun et al .,2000).

On the other hand Gupta et al,.(1995) stated that the body attempts to regulate the pb toxicity by promoting self defense by enhanced production of thiol compounds such as glutathione. However, many efforts have been devoted to protect both human and laboratory animals form leading toxicity using both natural and synthetic organic comound beside some minerals.

Vitamin B6, natural sulfer compounds garlic oil, lipoic acid, calcium and selenium are some of these compounds that were used in the treatment of lead poisoning (Flora et al, 1984) Attia and Ali, (1993)Ballew and Bowman,(2002).

Ascorbic acid resulted to lower concentration of lead in the blood and restored the levels of iron, calcium and zinc in the blood as well as the lipid balance (Kamalakkannan etal.,2005)

Ascorbic acid administration offered protection to the cell from expansion or abnormalities in their structural features.

It is concluded that ascorbic acid not only confers protection against lead toxicity but it can also perform therapeutic role against toxicity (Bhattacharjee et al., 2003). Some researchers have reported impairment of learning and memory in cases of lead poisoning. They

also found progressive deterioration in mental capacity in workers with many years of excessive industrial lead exposure (Marchlewicz et al., 2006).

There is a preventive and curative effect of combined supplementation of Garlic and vitamin B-complex against lead toxicity in albino mice. Evaluation of the liver functions in three experimental groups has proved the potency of supplemented Garlic and vitamin B-complex to keep more or less normal liver functions as manifested by levels of serum and hepatic activity of transaminase (GOT and GPT), alkaline phosphatase (ALP), acid phosphatase (ACP), lactate dehydrogenase (LDH) and total protein which had been distributed under chronic lead toxicity.

These results indicated the protective role of combined zinc and Garlic supplementation against chronic lead toxicity on the liver function (Zayat *et al.,* 1996).

Many scientists studied the efficacy of thiamine, ascorbic acid, and Becozinc (a pharmacological preparation containing vitamins of the B complex group, garlic, and zinc) in enhancing excretion and reducing tissue burden of lead and reversing lead-sensitive bio-chemical alterations was investigated in lead pre-exposed-mice (Tandon and Singh, 2000).

These vitamins were effective in mobilizing lead from blood, liver, and kidney into urine and or faeces and in restoring partially blood zinc protoporphyrin level (Tandon *et al.,* 2000).

Many researchers showed and examined that the effect of treatment with Garlic not only confer protection against lead toxicity but it can also perform therapeutical role against toxicity (Bhattacharjee *et al.,* 2003).

The aim of the present study was to investigate the possible hematological and biochemical changes that might occur in albino rat's after lead toxicity, and to evaluate the role of vitamin C or vitamin E with/without zinc sulphate as protective agents.

Materials and Methods

1- Experimental animals:

Male albino rats weighing about 100-120 gm, were used throughout this study . the diet and tab water were offered *adlibitum* all over the experimental period. The animals were divided into six subgroups.

The first group served as control. Lead acetate (20mg/kg.b.wt) was orally administrated daily by means of stomach tube to the rats in groups 2,3,4,5 and 6 for 20 days.

On the other hand groups 3,and 4 were also given daily, vitamin C (150mg/kg) (El-Nahas et al.,1993) and vitamin E respectively all over the experimental period.

The rats in subgroups 5 and 6 were also given daily zinc sulphate (50mg/kg) in addition to vitamin C or vitamin E all over the experimental period.

Vitamin E was dissolved with olive oil and administrated orally in a dose of 150 mg/kg. according to El-Nahas et al.,(1993).

At the end of experiment the animals were scarified and blood samples were collected from jugular vein for hematological examination (Jain,1986). Clear serum samples were separated by centrifugation at 3000 r.p.m. for 20 min and then collected and stored in a deep freeze at 20˚c for different biochemical analysis .

2- Biochemical analysis :

Serum samples were analyzed for triglycerides, and total cholesterol by the methods described by Fossati and Prencipe (19.82) and Allain (1974), respectively. Non protein nitrogen constituent were determined by the methods of Mackay (1980) for uric acid and Bart'els and Bohmer(1972) for creatinine. Serum total protein was determined according to the methods described by Weichselbaum(1946). Serum albumin was determined using bromocresol green method according to Doumas et al,.(1971). Serum transaminase were measured as recommended by Reitman and Frankel (1979).

3- Hematological parameters:

Determination of hematological parameters was carried out using a 18 automated parameters hematology analyzer, ABC Micors 60 from Horiba ABX, France .

4- Statistical analysis:

The statistical analysis for T-test was performed by using SPSS.

Result and discussion

Data obtained under the present investigation generally indicated that blood parameter of male albino rats have been affected by daily oral administration of lead acetate and treatment by zinc sulphate in addition to vitamin C or zinc sulphate in addition to vitamin E.

The effect of lead acetate administration on hematological parameters.

The effect of lead acetate administration on rats on hematological parameters is demonstrated in table 1. In groups 2,3,4,5 and 6 the total RBCs counts was significantly decreased to the extent 27%, 16.8%, 15.7%, 14.8% respectively compared to the control groups. However, in group 2, the decrease was noticed after 20 days of administration with a value of 20.4%.

On detecting the blood hemoglobin level, the data are given in table (1). After 20 days of lead acetate oral administration there was a general decrease in hemoglobin concentration. The percentage decrease was 24.50%comared to control group.

On the other hand, rats treated with lead acetate plus vitamin E, showed a general decrease in the same parameter recording 23.4% as compared to control animals.

While lead acetate administration in addition to vitamin C or vitamin E was decreased by 22.55% and 23.43% respectively compared to the control.

The present result also showed significant decrease in hematocrit value in treated rats as compared to control ones. The decrease was

obvious at the 20th day of lead acetate administration with value of 11.5% compared to control level. On the other hand, rats treated with lead acetate and zinc sulphate plus vitamin C or Zinc sulphate plus vitamin E, the results showed a general decrease in same parameters recording 5.4% and 2.1% respectively as compared to normal control animals. While lead acetate administration in addition to vitamin C or vitamin E was decreased by 5.6% and 3.0% respectively compared to the control.

On the other hand, the effect of lead acetate administration on rats on hematological parameters is demonstrated in table 1. In groups 2,3,4,5 and 6 the total MCV value was generally decreased to the extent 5.6%, 6.3%, 9.2%, 8.1% respectively compared to the control groups. However, in group 2, the decrease was noticed after 20 days of administration with a value of 5.2% but MCH was increased compared to control level (table 1).

The decrease in RBC could be explained on the basis of inhibitory effected of lead on histogenesis. Changes in hemoglobin can be explained due to changes in RBC size impaired biosynthesis of heme in bone marrow .

Although not statistically significant, hemoglobin and hematocrit level also decreased in the test group ($p>0.05$), which in some reports has been attributed to decrease in copper metabolism and iron consumption (Klauder et al 1977;Gautam et al 1987; A.T.S.D.R. et al,1993).

Lowered RBC count, decreased MCHC and MCV are other concordant hematological change were found in the group which lead acetate was administrated (Yagminas et al 1990;Falke et al 1990). Anemia was in the form of microcytic and hypochromic. This might be due to effects of lead in cell metabolism, alteration of the enzyme activity, and interaction with reactions in which calcium is their secondary mediator. Lead induced inhibitory effects on the erythrocyte enzymes GA3PD and G6PD have already been proved (Calderon et al 1993).

It may be due to low hemoglobin production because of lead induced disturbance of heme biosynthesis (Wildman *et al.,* 1976).

In our study, may be bone marrow could overcome lead toxicity because of subchronic exposure which was not at high dose, but it suppressed the production of Hb. Low Hb level might result in reduced oxygen transfer by RBCs, which was compensated by increased number of these cells. Also, tissue hypoxia is a possible mechanism for high production of RBCs in moderate lead poisoning. MCV and MCHC levels were decreased after lead intoxication in our study that it was in agreement with several studies (Noori et al.; Falke & Zwennis, 1990; Antonowicz et al., 1991).

In this study, lead intoxication had the same effects on blood indices that were observed previously in other researches (Hofmann & Segewitz 1975; Wildman *et al*. 1976; Falke & Zwennis and Antonowicz *et al.*1990; Alexa *et al. 2002*; Noori *et al*.2003; Othman *et al*.2004; Toplan *et al*. 2004;).

In contrast to the RBCs , the white blood cell count showed general increased level upon lead acetate administration all over the experimental periods examined .

However the increase in WBC count was more pronounced at the 20^{th} day of lead acetate administration with a value of 63.09% as compared to control group .

On the other hand in rats treated lead acetate and zinc sulphate plus vitamin C or zinc sulphate plus vitamin E, the result showed a general increase in the same parameters recoding 76% and 75.85 % respectively as compared to the control group.

While, lead acetate administration in addition to vitamin C or vitamin E was increased by 68.4% and 70.20% respectively as compared to the control .

The result are in agreement with the findings given by kollar and Roan, (1980), who reported that this induction of W.B.C is a positive response for survival due to cell mediated immune response of animals. leukocytosis has been attributed to the lead-induced inflammation (Koller and Roan, 1980).

In general the blood platelet count was increased in response to administration of lead acetate at the different intervals of the experiment.

Lead acetate administration gave rise to a maximal effect after 20 days of pb administration with value 81.27% compared to the control level.

On the other hand, in rats treated with lead acetate and zinc sulphate in addition vitamin C or zinc sulphate in addition to vitamin E, the results showed a general increase in the same parameter recording ٨٩,١٨% and 90.76% respectively as compared to control animals. While lead acetate administration in addition to vitamin C or vitamin E was increased by 83.09% and ٨٦,٥٤% respectively as compared to the control.

Platelet count showed considerable increase compared to the control groups ($p<0.001$). In the previous studies, some cases of thrombocytopenia after lead intoxication (Sudakova et al 1983) followed by thrombocytosis have been reported (Yagminas et al 1990; Sudakova et al 1983), which is consistent with the findings of this study which was conducted over a long period of 12 weeks.

Oral administration of lead acetate showed significant decrease in serum total protein level as compared to control group reaching a percentage decrease of 25.63% by the end of the experimental duration . However, lead acetate administration and treatment by zinc sulphate in addition to vitamin C or zinc sulphate in addition to vitamin E was decreased by 23.48% and 21.9%, respectively compared to the control. Lead acetate administration and treatment by vitamin C or vitamin E was decreased by 24.04% and 22.70% respectively as compared to the control.

The observed decrease in total serum protein may be attributed to activation of anabolism of protein due to lead toxicity which affected on the enzyme of anabolism process (Attia,1993). So the body produces higher level of protein to bind with lead on and thus prevent lead toxicity.

The observed decrease in total serum protein agreed with Sauk etal, 1993 and Abdel Aziz (2002).

Lead acetate experimental rats showed a general increase in serum albumin level as compared to control animals with percentage increased (87%) through the end of experimental duration . On the other hand there was general increase in the serum albumin concentration in rats treated with lead acetate plus zinc sulphate in addition to vitamin C or zinc sulphare in addition to vitamin E reaching a percentage increase of 90% and 89.33% respectively.

In contrast serum globulin showed significant decrease ($p<0.05$) compared to the control at the different time intervals studied (table2).

How ever lead acetate administration and treatment by vitamin C or vitamin E also have been increased reaching percentage increase of 93% and 89% respectively as compared to the control at the different time intervals studied .

In view of consideration that the liver is the site of albumin production (Rossan, 1960) the formation or derangement synthesis of albumin is due to a damaged liver.

Results of the present investigation were in agreement with findings given by Attia (1993) and Abdel Aziz (2002) who reported that levels of serum albumin and globulin and consequently total protein were affected due to lead toxicity .

Table 2 demonstrate the mean value of serum total cholesterol concentration in both control and experimental group . The higher increase was observed after 20 days of lead acetate administration , where reached 76.43% as compared to the control group .

However, lead acetate administration and treatment by zinc sulphate in addition to vitamin E or zinc sulphate in addition to vitamin E or zinc sulphate in addition to vitamin E was increased by 81.57% and 80% respectively as compared to the control , lead acetate administration and treatment by vitamin C or vitamin E was increased by 81.17% and 79.77% respectively as compared to the control.

The increment in cholesterol content agreed with that reported by Ashour (2002) in response to 20 day feeding of lead acetate in rabbits.

Serum triglecerides showed significant increase where it reached 67.68% as compared to the control group by 20th day of experimentation . However, lead acetate administration and treatment by zinc sulphate in addition to vitamin C or zinc sulphate in addition to vitamin E was increased by 80.56% and 76.44% respectively as compared to the control . Lead acetate administration and treatment by vitamin C or vitamin E was increased by 77.5% and 76.1% respectively as compared to the control .

(Table 3) showed significant decrease in ALT activity among lead acetate treated rats as compared to the control ones . The percentage decrease among individuals at the last duration was 31.1% as compared to control group . On the other hand, lead acetate administration and treatment by zinc sulphate in addition to vitamin C zinc sulphate in addition to vitamin E was decreased by 18.92% and 9.76% respectively as compared to the control. Lead acetate administration and treatment by vitamin C or vitamin E was decreased by 22.9% and 13.4% respectively as compared to the control.

Repeated doses of lead acetate produced significant decrease in AST activity. This decrease reached 12.25% at the end of the experiment. On the other hand, lead acetate administration and treatment by zinc sulphate in addition to vitamin C or zinc sulphate in addition to vitamin E was increased by 5.81% and 4.39% respectively as compared to the control . Lead acetate administration and treatment by vitamin C or vitamin E was decreased by 7.39% and 4.9% respectively as compared to the control.

The present result are in accordance with Abdel Aziz (2002) who reported inhibition of serum ALT and AST in treated with 40mg/kg lead acetate .

The daily oral administration of lead acetate for 20 days increased significantly the concentration of uric acid in the serum of treated rats at 20th day of treatment (table 4) this increase reached 77.19% as compared to the control. However, lead acetate administration and treatment by zinc sulphate in addition to vitamin C or zinc sulphate in addition to vitamin E was increased by 92.9% and 94.74% respectively as compared to the control . Meanwhile, lead acetate administration and treatment by vitamin C or vitamin E. was

increased by 87.72% and 91.22% respectively as compared to the control.

The elevation of uric acid may be due to degradation of purines and pyrimidines or to an inability of excretion (Wolf et at, 1972) the elevation of uric acid in response to lead administration agreed with Ankrah et al, 1996, MC-Bride et al, 1998 and Ashour (2002).

Urea concentration in rat's treated with lead acetate for 20 days was increased by 80.80% as compared to the control level (table 4), however , lead acetate administration and treatment by zinc sulphate in addition to vitamin C or zinc sulphate in addition to vitamin E was increased by 84.95% and 88.49% respectively as compared to the control .

Meanwhile lead acetate administration and treatment by vitamin C or vitamin E was increased by 84.62% and 86.87% respectively at the end of the experiment as compared to the control .

Enhanced protein catabolism and accelerated rated amino acid domination for gluconeogensis is probably and acceptable postulate to interpret the elevated level of urea . On the other hand , the elevated serum urea levels may be due to the destruction of red cells during pb administration. The presence of some toxic compounds might increase blood urea and decrease plasma protein (Varely,1976) .

In general, the concentration of creatinine in rat's blood serum treated with lead acetate for 20 days was increased by 93.55% as compared to control group. However, lead acetate administration and treatment by zinc sulphate in addition to vitamin C or zinc sulphate in addition to vitamin E was increased by 96.8% and 90% respectively as compared to the control. Lead acetate administration and treatment by vitamin C or vitamin E was increased by 95.70% and 96.77% respectively as compared to the control. This amelioration may be due to the improvement in liver detoxification function (Stojko et al.,1985).

The elevation of serum creatinine and uric acid concentration in treated rats indicating impaired renal function and kidney damage which may be attributed to the nephrotoxic effect of lead acetate.

Creatinine increment caused by lead agreed which Khalil – Manesh et al., (1992) and Ashour (2002).

An advantage of Zn – salts nay be a good to tolerance in humans (Tschumi and Floersheim, 1981). Zn is known to be an important component of over 100 enzymes, including several involved in DNA synthesis and their repair. It also a component of super oxide dismutase , claimed to protect against free radical which revealed the improvement of some previous result in hematology and biochemistry .

Vitamin C is a water soluble antioxidant which can directly scavenge super oxide and hydroxyl 1 radicals. Many vitamins trials have focused on single antioxidants (pacht et al, 1986 and Schectman et al, 1991). Some antioxidants such as vitamin E are lipid-soluble and therefore primarily active in the lipid functions of membranes, cells and tissues.

Further, The various antioxidants serve to regenerate each other when vitamin E interacts with a lipid radical to donate an electron, a tocopherol radical is formed which can be generated through the action of vitamin C (langseth, 1995). The action of vitamin E and vitamin C may explain the improved hematological and biochemical parameters in lead acetate treated rats.

Severe changes in blood indices by lead that were found in the present investigation and other studies indicate the necessity of even more concerns about the bio-environment pollution of lead. Designation and provision of the health programs to limit causal exposure to this toxic element is highly important for our health.

In conclusion, zinc sulphate and vitamin C or vitamin E improved the expected hematological and biochemical parameters changes in lead intoxicated rats. So we suggest to give the people exposed to lead compounds doses of vitamin C and E in addition to zinc sulphate.

Table (1): the effect of oral administration of lead acetate and treatment by vitamin C or vitamin E with/without zinc sulphate on blood indices of albino rats .

Parameters	Sampling Date	Experimental groups									
		control	Lead acetate	% of change	Lead acetate + Vit.c	% of change	Lead acetate + Zinc+vit.c	% of change	Lead acetate + Vit.E	% of change	Lead acetate + Zinc+vit.E	% of change
RBC count (×10 cell/u1)	Zero time	5.65 ± 0.19	5.51 ± 0.15	-2.47	5.55 ± 0.15	-1.76	5.56 ± 0.16	-1.59	5.56 ± 0.14	-1.59	5.58 ± 0.13	-1.23
	5days	5.70 ± 0.61	5.43 ± 0.16	-4.73	5.54 ± 0.17	-2.80	5.58 ± 0.14	-2.10	5.59 ± 0.13	-1.92	5.60 ± 0.17	-1.75
	10 days	5.72 ± 0.17	5.16 ± 0.15	-9.79	5.22 ± 0.16	-8.74	5.31 ± 0.18	-7.16	5.39 ± 0.19	-5.76	5.42 ± 0.16	-5.24
	20 days	5.73 ± 0.16	4.56 ± 0.11	-20.41	4.17 ± 0.15	-27.22	4.77 ± 0.11	-16.75	4.83 ± 0.12	-15.70	4.88 ±0.13	-14.83
Hb (g/d1)	Zero time	11.30 ± 0.16	10.10 ± 0.21	-10.61	10.60 ± 0.17	-6.19	10.62 ± 0.19	-6.01	10.71 ± 0.16	5.22	10.59 ± 0.17	-6.28
	5days	11.10 ± 0.13	9.16 ± 0.22	-17.47	9.90 ± 0.14	-10.8	9.35 ± 0.18	-15.76	9.96 ± 0.15	-11.08	9.87 ± 0.16	-11.08
	10 days	10.91 ± 0.14	8.17 ± 0.14	-25.11	7.71 ± 0.13	-29.33	7.82 ± 0.21	-28.32	7.80 ± 0.23	-28.50	7.91 ± 0.14	-27.49
	20 days	10.20 ± 0.18	7.70 ± 0.23	-24.5	7.90 ± 0.26	-22.5	7.96 ± 0.25	-21.96	7.81 ± 0.24	-23.43	7.98 ± 0.21	-21.76
HCT(%)	Zero time	34.16 ± 0.40	34.10 ± 0.35	-0.17	34.13 ± 0.30	-0.08	34.14 ± 0.31	-0.05	34.15 ± 0.32	-0.02	34.15 ± 0.31	-0.02
	5days	34.11 ± 0.41	33.91 ± 0.4	-0.58	34.0 ± 0.31	-0.32	33.95 ± 0.31	-0.46	34.19 ± 0.33	-0.23	33.98 ± 0.40	-0.38
	10 days	33.18 ± 0.43	30.30 ± 0.3	-8.67	32.15 ± 0.33	-3.10	32.80 ± 0.32	-1.14	32.30 ± 0.3	-2.65	32.88 ± 0.21	-0.90
	20 days	33.00 ± 0.19	29.19 ±0.36	-11.54	31.13 ± 0.27	-5.12	32.0 ± 0.26	-3.03	31.23 ± 0.25	-5.36	32.30 ± 0.32	-2.12
MCH(%)	Zero time	60.64 ± 0.11	61.88±0.12	2.04	61.49 ±0.11	+1.40	61.40 ± 0.13	1.25	61.42 ± 0.11	1.28	61.20 ± 0.13	0.92
	5days	59.84 ± 0.12	62.44*0.13*	4.34	61.37 ±0.12*	2.55	60.84 ± 0.12	1.67	61.16 ± 0.11*	2.21	60.67 ± 0.12	1.38
	10 days	58.0 ± 0.11	58.72±0.11	1.24	61.59 ±0.12*	6.18	61.77 ± 0.11*	6.5	59.92 ± 0.10	3.31	60.66 ± 0.11*	4.59
	20 days	57.59 ± 0.12	64.01±0.10*	11.14	66.23 ±0.11*	15.002	67.08 ± 0.10*	16.47	64.65 ± 0.11*	12.26	66.18 ± 0.10*	14.92

All values expressed as mean ± S.E. differences at P≤ 0.05 .

Continuation of table 1.

Parameters	Sampling Date	Experimental groups										
		control	Lead acetate	% of change	Lead acetate + Vit.c	% of change	Lead acetate + Zinc+vit.c	% of change	Lead acetate + Vit.E	% of change	Lead acetate + Zinc+vit.E	% of change
MCV(%)	Zero time	20.32±0.11	18.33± 0.12*	-9.79	19.09 ± 0.11	-2.06	19.10 ± 0.12	-6.003	19.26 ±0.11	-5.21	18.97 ± 0.12*	-6,64
	5 days	19.47±0.12	16.86±0.13*	-13.40	17.87 ± 0.12*	-8.21	17.65 ± 0.13*	-9.34	17.81± 0.14*	-8.52	17.62 ± 0.11 *	-9.50
	10 days	19.07±0.11	15.83± 0.10 *	-16.99	14.77 ± 0.13 *	-22.54	14.72 ± 0.12*	-22.81	14.47 ±0.12*	-24.12	14.59 ± 0.11*	-23.49
	days 20	17.80±0.15	16.88± 0.11	-5.16	16.80 ± 0.14	-5.61	16.68 ± 0.10	-6.29	16.16 ±0.15	-9.21	16.35 ± 0.16	-8.14
MCHC(%)	Zero time	33.10±0.13	26.61±0.12*	-19.60	31.08± 0.11*	-6.10	31.10± 0.10 *	-6.04	31.36± 0.11*	-5.25	31.01± 0.11*	-6.31
	5 days	32.54±0.11	29.01±0.13*	-10.84	29.11± 0.10 *	-10.54	29.01± 0.14*	-10.84	29.13± 0.16*	-10.47	29.04± 0.12*	-10.75
	10 days	32.88±0.14	26.96±0.12*	-18.004	23.98± 0.11*	-27.06	23.84± 0.12*	-27.49	24.14± 0.12*	-26.58	24.05± 0.13*	-26.85
	days 20	30.90±0.11	26.63±0.12*	-13.81	25.37± 0.10 *	-17.89	24.87±0.13 *	-19.61	25.00± 0.13*	-19.09	24.70± 0.14*	-20.06
WBC count (×10 cell/u1)	Zero time	8.60 ± 0.21	8.77 ± 0.30	1.97	8.75 ± 0.31	1.74	8.76 ± 0.31	1.86	8.77 ± 0.25	1.97	8.78 ± 0.21	2.09
	5 days	8.70 ± 0.22	8.99 ± 0.21	1.03	8.92 ± 0.26	2.52	8.90 ± 0.33	2.29	8.91 ± 0.65	2.41	8.86 ± 0.23	1.83
	days 10	8.80 ± 0.22	9.91 ± 0.30	12.61	8.90 ± 0.24	1.13	8.88 ± 0.29	0.90	8.85 ± 0.25	0.56	8.81 ± 0.27	0.11
	days 20	8.86 ± 0.25	12.13 ± 0.11*	36.90	11.66 ± 0.12*	31.60	11.5 ± 0.20*	29.79	11.11 ± 0.21*	25.39	11.00 ± 0.20*	24.15
PLTS (×10 cell/u1)	Zero time	315 ±20.15	3.39 ±11.11	-98.92	329 ± 10.20	4.4	333 ± 10.30	5.71	325 ± 11.21	3.17	336 ± 11.23	6.66
	5 days	360 ±22.12	376 ± 16.90	4.4	371 ± 15.15	3.05	380 ± 15.17	5.55	368 ± 16.11	2.2	365 ± 16.20	1.83
	days 10	370 ±17.18	399 ± 14.13	7.83	391 ± 16.16	5.67	380 ± 17.20	2.70	386 ± 17.30	4.32	383 ± 17.20	3.51
	days 20	379 ± 16.3	450 ± 16.3*	18.73	440 ± 16.15*	16.09	420 ± 16.15*	10.81	430 ± 21.4*	13.45	414 ± 20.9*	9.23

All values expressed as mean ± S.E. differences at P≤ 0.05 .

Significant *

Table (2): The effect of oral administration of lead acetate and treatment by vitamin C or vitamin E or some biochemical Parameters in albino rats serum .

Parameters	Sampling Date	Experimental groups									
		control	Lead acetate	% of change	Lead acetate + Vit.c	% of change	Lead acetate + Zinc+vit.c	% of change	Lead acetate + Vit.E	% of change	Lead acetate + Zinc+vit.E	% of change
Total protein (gm/d1)	Zero time	0.20±5.25	0.11±5.13	-2.28	0.16±5.15	-1.90	0.21±5.20	-0.95	0.21± 5.16	-1.71	0.17± 5.22	-0.57
	5days	0.25±5.32	0.15±5.00	-6.01	0.14±5.10	-4.14	0.21± 5.11	-3.95	0.21±5.15	-3.19	0.16±5.17	-2.82
	10 days	0.19±5.19	0.22±4.95	-4.62	0.15±4.96	-4.43	0.19± 4.98	-4.05	0.18±4.97	-4.24	0.17±5.10	-1.73
	days 20	0.23±5.11	0.25*±3.80	-25.64	0.14*± 3.88	-24.07	0.20*±3.91	-23.48	0.16*±3.95	-22.70	0.14*±3.99	-21.92
Albumin (gm/d1)	Zero time	0.29±3.11	0.16±3.19	2.57	0.10±3.18	2.25	0.11±3.15	1.29	0.14±3.14	0.96	0.14±3.12	0.32
	5days	0.16±3.16	0.18±3.30	4.43	0.25±3.25	2.85	0.22± 3.12	-1.26	0.11± 3.25	2.85	0.10±3.24	2.53
	days 10	0.12±3.10	0.17 3.36±	8.39	0.17±3.35	8.06	0.17± 3.33	7.49	0.16±3.34	7.74	0.19± 3.30	6.45
	days 20	0.11±3.00	0.20±3.39	13	0.20±3.21	7	0.18±3.30	10	0.17± 3.33	11	0.16±3.32	10.66
Globulin (gm/d1)	Zero time	±0.202.14	*±0.211.94	-9.35	*±0.161.97	-7.94	±0.142.05	-4.20	±0.112.02	-5.61	±0.162.10	-1.87
	5days	±0.192.16	*±0.181.70	-21.29	0.17*±1.85	-14.35	*±0.151.99	-7.87	*±0.151.90	-12.04	*±0.171.93	-10.65
	days 10	±0.162.09	*±0.171.59	-23.92	*1.61±0.18	-22.97	*±0.161.65	-21.05	*±0.181.63	-22.01	*±0.151.80	-13.87
	days 20	±0.172.11	*±0.110.41	-80.57	*±0.130.67	-68.25	*±0.140.61	-71.09	*±0.160.62	-70.62	*±0.140.67	-68.25
Cholesterol (mg/d1)	Zero time	3.13±64.50	3.16±67.11	4.05	3.16±66.76	3.50	3.19±66.11	2.49	3.15±66.90	3.72	3.14±66.76	3.50
	5days	3.16±68.00	*3.18±75.15	10.51	3.19±73.17	7.60	3.16±72.16	6.11	3.16 *±74.19	9.10	2.21± 73.00	7.35
	days 10	3.20±69.19	4.16*81.00±	17.07	4.15±79.19	14.45	4.16±78.00	12.73	4.17±79.90	15.47	3.19±79.60	15.04
	days 20	3.30±70.0	3.19*±86.50	23.57	2.30 *±83.18	18.83	3.16*±82.90	18.42	2.51*±84.16	20.22	2.19 *±84.00	20
Triglycerides (mg/d1)	Zero time	7.11±133.10	6.90±139.11	4.52	5.11±139.00	4.43	6.17±138.22	3.84	5.17±138.55	4.09	5.51±198.16	48.88
	5days	9.22±135.60	±6.14170.11	25.45	6.11±165.13	21.87	6.15±160.11	18.07	6.18±161.61	19.18	5.14±161.00	18.73
	days 10	6.30 ±136.9	±6.83176.60	28.99	6.32±168.16	22.83	6.41±166.0	21.23	4.16±169.93	24.12	3.11±169.70	23.95
	days 20	5.16±138.15	±5.18182.80	32.32	3.17±169.17	22.45	4.14±165.0	19.43	4.19±171.16	23.89	5.10±170.70	23.56

All values expressed as mean ± S.E. differences at P≤ 0.05 .

Significant

Table (3): The effect of oral administration of lead acetate and treatment by vitamin C or vitamin E with / without zinc sulphate on biochemical parameters in rat's serum.

Parameters	sampling date	Experimental groups									
		Control	Lead acetate	% of change	Lead acetate + vit.c	% of change	Lead acetate + zinc + vit.C	% of change	Lead acetate + vit.E	% of change	Lead acetate + zinc + vit.E	% of change
Urea (Mg/dl)	Zero time	47.11 ±3.11	49.46 ± 2.17	4.99	49.30 ± 2.15	4.65	48.96 ± 3.15	3.92	49.10 ± 2.75	4.22	48.15 ± 2.95	2.21
	5 days	48.18 ± 2.85	51.16 ± 3.13 *	6.18	49.90 ± 2.66	3.57	49.70 ± 2.17	3.15	49.50 ± 2.19	2.74	49.19 ± 1.30	2.09
	10 days	50.15 ± 2.11	55.17 ± 4.15*	10.009	53.51 ± 3.17*	6.69	53.11 ± 3.13*	5.90	52.81 ± 2.15	5.30	51.77 ± 2.16	3.23
	20 days	52.15 ± 2.19	62.16 ± 4.16*	19.19	60.17 ± 3.11*	15.37	60.00 ± 4.19*	15.05	59.17 ± 4.11*	13.46	58.15 ± 3.30*	11.50
Uric acids (gm/dl)	Zero time	0.53 ± 0.06	0.57 ± 0.05	11.76	0.56 ± 0.01	9.80	0.55 ± 0.02	7.84	0.56 ± 0.03	9.80	0.54 ± 0.01	5.88
	5 days	0.54 ± 0.03	0.5 ± 0.03	-5.66	0.58 ± 0.02	9.43	0.55 ± 0.03	3.77	0.54 ± 0.03	1.88	0.53 ± 0.02	0
	10 days	0.57 ± 0.02	0.61 ± 0.04	12.96	0.59 ± 0.04	9.25	0.85 ± 0.02	57.40	0.57 ± 0.01	5.55	0.56 ± 0.01	3.70
	20 days	0.90 ± 0.01	0.70 ± 0.04*	22.80	0.64 ± 0.03	12.28	0.61 ± 0.02	7.01	0.62 ± 0.02	8.77	0.60 ± 0.03	5.26
Creatinine (mg/dl)	Zero time	0.90 ± 0.01	0.93 ± 0.01	3.33	0.92 ± 0.01	2.22	0.91 ± 0.01	1.11	0.91 ± 0.02	1.11	0.92 ± 0.01	2.22
	5 days	0.91 ± 0.02	0.94 ± 0.02	3.29	0.93 ± 0.02	2.19	0.93 ± 0.03	2.19	0.93 ±0.01	2.19	0.93 ± 0.02	2.19
	10 days	0.92 ± 0.02	0.95 ± 0.03	3.26	0.94 ± 0.01	2.17	0.93 ± 0.01	1.08	0.94 ± 0.02	2.17	0.93 ± 0.02	1.08
	20 days	0.93 ± 0.01	0.99 ± 0.03	6.45	0.97 ± 0.02	4.30	0.96 ± 0.01	3.22	0.96 ± 0.03	3.225	0.94 ± 0.03	1.07
ALT (1u/ml)	Zero time	55.9 ± 2.13	51.17 ± 2.15	-8.46	52.0 ± 1.99	-6.97	53.11 ± 2.20	-4.99	52.11 ± 2.15	-6.77	53.0 ± 1.19	-5.18
	5 days	58.11 ± 3.11	50.50 ± 2.13	-13.09	52.19 ± 2.30	-10.18	53.50 ± 2.18	-7.93	53.50 ± 2.17	-7.93	53.90 ± 2.19	-7.24
	10 days	60.60 ± 2.95	48.14 ± 3.19*	-20.56	49.9 ± 2.15*	-17.52	52.56 ± 2.16*	-13.26	55.11 ± 2.16*	-9.05	56.20 ± 2.17*	-7.26
	20 days	65.50 ± 3.00	44.60 ± 4.11*	-31.90	50.50 ± 2.45*	-22.90	53.11 ± 2.33*	-18.91	56.70 ± 2.11*	-13.43	59.11 ± 2.16*	-9.75
AST (1u/m1)	Zero time	51.11 ± 2.11	48.0± 1.90	-6.08	48.90 ± 2.10	-4.32	49.88 ± 1.93	-2.40	49.97 ±1.95	-2.23	48.0 ± 1.90	-6.08
	5 days	47.70 ± 2.19	45.15 ± 2.13	-5.34	45.29 ± 2.22	-5.05	45.44 ± 2.16	-4.73	45.77 ± 2.00	-4.04	46.0 ± 2.11	-3.56
	10 days	46.21 ± 2.0	41.17 ± 2.12	-10.90	42.17 ± 2.17	-8.74	42.90 ± 2.50	-7.16	43.11 ± 2.21	-6.70	43.91 ± 2.2	-4.39
	20 days	44.39 ± 4.11	38.95 ± 3.11	-12.25	41.11 ± 3.70	-7.38	41.77 ± 4.20	-5.90	42.2 ± 3.19	-4.93	42.44 ± 3.13	-4.39

All values expressed as mean ± S.E differences at $p \leq 0.05$.

* Significant

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