Protective Effect of Vitamin C and Selenium Against the Toxicity Induced by Lead Acetate on Some Physiological Parameters in Blood of Male Albino Rats

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ABSTRACT

The objective of this study was to explore whether Vitamin C or selenium could be protective against the toxic effect of lead acetate. To achieve this purpose, certain hematological and biochemical parameters were studied. Twenty male albino rats (Rattus norvegicus), weighing about 130-150 gram were used. The rats were divided into four groups, each group included five rats. The first group was the control, the second group was administrated orally lead acetate (20 mg / kg of the body weight / day/four weeks), the third group was administrated orally with the same dose of lead acetate plus vitamin C (50 mg/kg body weight/ day/four weeks), the fourth group was given the same dose of lead acetate as the second group and plus sodium selenate in a dose of (0.1 mg /kg body weight/ day/four weeks). Food and water were allowed adlibitum for all the groups. The experimental period was four weeks. The results showed that there was a significant decrease in hematological indices studied in the second group (red blood count, white blood count, and hemoglobin concentration and haematocrit value) after the administration of lead acetate. Moreover, there were higher significantly increase in serum glucose, total lipids, cholesterol, urea and creatinine compared with the control group. The third group showed improvement in the hematological parameters,(red blood& white blood count ,hemoglobin concentration and haematocrit value).Also improvement in the biochemical parameters of serum glucose ,total lipids ,cholesterol ,urea and creatinine compared to second group. The fourth group showed significant improvement compared with the second group. In addition, a significant decrease in serum glucose, total lipids, total cholesterol, urea and creatinine were found of this group. In conclusion, the results of different parameters studied in rats received orally vitamin C or selenium showed improvement compared with the rats orally received lead.
Key words: Vitamin C, Selenium, toxicity, lead acetate, physiological parameters, male albino rat.

INTRODUCTION

Trace elements were known to have a variety of important biological functions and in many instances, they may have adverse effects on biological system [1,2,3&4]. In this respect, lead is a heavy metal of wide occupational and environmental contamination. Lead toxicity is

associated with an increased risk of adverse effect on a variety of target organs, including the central nervous, hematopoietic and renal systems[5&6]. Some studies on workers in lead industries have shown that chronic exposure to lead affect peripheral and central nervous system and reproductive system[7]. Furthermore lead exposure is related to behavioral disorders such as hypo activity and decreased attention [8]. Lead toxicity may depend on many factors such as species differences, life stage ,concentration in food, water and time of exposure [9]. The heavy metals not only affect the hematological and biochemical measurements , but also it leads to metabolic disturbances and disease incidence processes as previously reported [3,4&10]. A wide range of antioxidants, both natural and synthetic ,have been proposed for use in the treatment of many human diseases. However, antioxidant supplementation of diabetics can reduce both oxidative stress[11&12] and protein glycation [13]. Moreover; some antioxidants have the ability to eliminate accumulation of lipid peroxides[14].and may help to reduce the risk of developing diabetic complications[11]. Ascorbic acid is water-soluble vitamin actin as antioxidant to protect cellular components from damage induced by reactive oxygen species[15&16]where it has the potential to scavenge superoxide and hydroxyperoxide radicals[17]. Selenium (Se) is recognized as an essential trace element for humans[18]. Selenium acts as a protective agent against the toxic effects of hydroperoxides[19,120&21].

MATERIALS & METHODS

Experimental animals and design:

The present study was carried out on the albino rats (Rattus norvegicus) weighing about 130-150g . These animals were normal and they were acclimatized to the experimental conditions for two weeks before the onset of the experiment. The considered rats were divided into four groups. The first group was regarded as control. The second group : rats were given a single dose of lead acetate (20 mg/kg. body weight/day/four weeks)using stomach tube .The third group : rats were given the same dose of lead acetate together with vitamin C(50 mg/kg body weight/day/four weeks). The fourth group was orally given a dose of lead acetate as the pervious groups together with sodium selenate(0.1mg/kg body weight/day/four weeks). Food and water were allowed *adlibitum*. The experimental period was four weeks.

Blood sampling:

Blood samples were withdrawn from the retro-orbital sinus of the eye using heparinized micro-haematocrit capillary tube at the end of each week. Two blood samples were collected from each rat, one sample was freshly used for hematological analysis .Serum separated from the second sample was stored at-20 C° and was used for biochemical assays.

A-Blood pictures analysis (Hematological studies):

Complete blood analysis including: Erythrocytes (RBCs) and Leukocytes (WBCs) count were carried out using improved Neubauer Hemocytometer, also Haematocrit

value (Hct) was estimated using micro-capillary technique according the method of Rodak[22]. Hemoglobin concentration was determined according to the method of Drabkin &Austin[23].

B-Biochemical analysis:

Commercial diagnostic kits from Bio-Merieux chemicals were used for the flowing biochemical assays.

The serum glucose (mg/dl) was measured by the enzymatic colorimetric method according to Trinder[24] . Total lipids(mg/dl) were determined according to the method of Knight *et al* [25]. .Serum cholesterol (mg/dl) was estimated by enzymatic – colorimetric methods as described by Thomas[26]. Urea (mg/dl) was estimated using kit supplied the method described by Chaney *et al*., [27]. Serum creatinine (mg/dl) was measured as described by Henery[28].

C-Statistical method:

For comparison of different experiment animal groups the Student t-test was used carried out on the obtained data .Significant differences between the control and treated groups were considered only at (P<0.05)by using the method of Sokal and Rahif, [29]. The data illustrated in tables are the means reading of five-rats± standard error.

Results

A-Effect of administrated lead acetate on some hematological and biochemical studies:

As shown in table(1): Exposure of rats to lead acetate (second group) for four weeks resulted in a significant decrease (p< 0.01)in red and white blood cells count and also a significant decrease in haematocrit value when compared with the corresponding values of control group. Also, the results showed a significant decreased in hemoglobin concentration compared with the control group. The results in table (1) showed a significant increased (p< 0.01) in serum glucose of rats of the second group when compared with control group. Also, in the same group (second) significantly increase of urea and creatinine in comparison with control group (first group). In addition, the results of this group in table (1) showed a significant increased (p <0.01) in serum total lipids and cholesterol compared with the control group.

B-Effect of concomitant administration of Vitamin C and lead acetate on the hematological and

biochemical of blood parameters studied:

The results of third group in which the rats were given orally vitamin C and lead acetate, are shown in table (2). Significant decreases were observed in the total red &white blood cells count, hemoglobin concentration (Hb); and hematocrite (Hct) values compared with the control group. In the same group, serum glucose value of treated rats was significant decrease (p <0.01), while serum urea and creatinine showed a significant increase (P<0.01) compared with the control group. Also in the same group, total lipids and cholesterol showed significant increase (P<0.01) compared with the control group. When comparing the second group (lead acetate group) with the third group (lead acetate &vitamin C group) as shown in table (4), there

was significant increase in hematological parameters of the third group than the second group .On the other hand there was highly significant decrease of all blood biochemical parameters of the third group compared with the second group

C-Effect of sodium selenate and lead acetate on hematological and biochemical of blood rats:

Treatment of rats with sodium selenate and lead acetate as noticed in table (3) showed highly significant decrease in red blood cells ,hemoglobin and hematocrit value, except a higher significant increased in total blood cells comparing with the control group .On the other hand ,significant increase in serum glucose ,total lipids ,urea and creatinine ,while no significant difference was noticed in serum total cholesterol with comparison with the control group .On the other hand when compare the parameters of the second group (lead acetate group) with the fourth group (lead acetate &Selenium)as shown in table (5) indicated that higher significant increased of all hematological parameters in the fourth group which orally selenium& lead than the second which orally lead only. On the other hand the data in table (5) indicated that higher significant decreased of all biochemical parameters of the group fourth in which orally selenium and plus lead than the second group in which the rats orally lead only.

Table (1): Effect of lead acetate (20mg/kg.body weight/day /four weeks) on hematological and biochemical parameters of male rats included in the study.

Groups	Control group First group				Lead acetate group Second group			
Weeks Parameters	First week	Second week	Third week	Fourth week	First week	Second week	Third week	Fourth week
R.B.Csx10^6 cells	6.8±0.04	6.7±0.09	6.7±0.07	6.9±0.02	6.2±0.07 **	5.5±0.03**	5.1±0.05**	4.5± 0.03**
Hb ,g%	13.8±0.05	13.3±0.04	13.0±0.03	12.6±0.02	13.2±0.04**	12.9±0.05**	12.1±0.07**	11.8±0.07**
Hct value	40.3±2.5	40.3±2.5	40.3±2.4	40.8±2.4	36.4±2**	32.9±2**	32.7±1.8**	32.4±1.8**
W.B.Csx10^3 cells	8.3±0.6	8.4±0.6	8.3±0.6	8.3±0.7	6.4±0.7**	6.1±0.7**	5.3± 0.5 **	5.0± 0.4**
Total lipids ,mg/dL	591±0.5	595±0.6	597±0.4	601±0.8	662.6± 0.9**	681.8±0.6**	731.2±0.6**	751.2±0.7**
Total cholesterol ,mg/dL	163±0.8	168±0.5	171±0.6	175±0.4	179.2± 0.6**	189.4±0.5**	197.4± 0.5**	212±0.7**
Glucose mg/dl	131±0.3	130.8±0.7	130.2±0.6	132.2±0.6	152.4±0.5**	161.2± 0.4**	165±0.5**	167.2±0.4**
Urea mg/dl	19.5±0.13	21.5±0.04	22.3±0.07	22.5±0.06	41.3± 0.5**	45.8±0.35**	51.7±0.54**	56.4±0.43**
Creatinine mg/dl	0.94±0.03	0.96.0±0.03	0.98±0.02	0.98±0.02	1.68 ±0.04	1.88 ± 0.02**	2.6± 0.03**	2.78±0.04**

Data were presented as means value ± S.E.　　Significant =　highly significant =***

Table (2): Effect of lead acetate (20mg/kg.body weight/day / four weeks) and vitamin C (50mg/kg.body weight) together on hematological and biochemical parameters of male rats included in the study.

Groups	Control group First group				Lead acetate and vitamin C group Third group			
Weeks Parameters	First week	Second week	Third week	Fourth week	First week	Second week	Third week	Fourth week
R.B.Csx10^6 cells	6.8±0.04	6.7±0.09	6.7±0.07	6.9±0.0.02	6.2±0.04**	6.3±0.04**	6.5±0.04*	6.8±0.04
Hb ,g%	13.8±0.05	13.3±00.4	13.0±1.3	12.6±0.01	12.6±0.12**	13.0±0.04*	13.3±0.1*	12.6±0.1
Hct value	40.3±2.5	40.3±2.5	40.3±2.4	40.8±2.4	38.7±1.1**	39.1±1.1	40.5±1	42.2±0.9
W.B.Csx10^3 cells	8.3±0.6	8.4±0.6	8.3±0.6	8.3±0.6	7.5±0.3**	7.6±0.6**	7.7±0.3**	7.9±0.2**
Total lipids ,mg/dL	591±0.5	595±0.5	597±0.4	601±0.6	652±0.7**	641.4±0.5**	652±0.7**	642.2±0.9**
Total cholesterol ,mg/dL	163±0.8	168±0.5	171.2±0.6	175±.0.4	165±0.7	171.4±0.5**	174.6±0.3**	181.4±0.5**.
Glucose mg/dl	131±0.3.	130.8±0.7	130.2±0.7	132.2±0.4	126.6±0.9**	124.2±0.7**	123.4±0.5**	120±0.2**
Urea mg/dl	19.5±0.13	21.4±0.04	22.3±0.07	22.5±0.06	31.5±0.25**	36±0.35**	41.3±0.53**	47.6±0.51**
Creatinine mg/dl	0.94±.03	0.96±0.03	0.96±0.02	0.98±.0.02	1.24 ±0.03**	1.44±0.03**	1.8±0.03**	1.74±0.03**

Data were presented as means value ± S.E.   Significant =   highly significant =***

Table (3): Effect of lead acetate (20mg/kg.body weight/ day / four weeks) together with Selenium (0.1mg/kg.body Weight) on Hematological and biochemical parameters of male rats included in the study.

Groups	Control group First group				Lead acetate and selenium group Fourth group			
Weeks / Parameters	First week	Second week	Third week	Fourth week	First week	Second week	Third week	Fourth week
R.B.Csx10^6	6.8±0.04	6.7±0.09	6.7±0.07	6.9±0.02	6.5 ±0.09**	6.7 ±0.07**	6.7 ±0.07	6.8±0.02
Hb ,g%	13.8±0.05	13.3±0.04	13.0±0.02	12.6±0.02	13.2±0.04**	13.0±0.05**	13.3±.07**	13.2±0.07**
Hct value	40.3±2.5	40.3±2.5	40.3±2.4	40.8±2.4	38.5±2**	38.7± 2**	38.5± 1.8**	41.9± 1.8**
W.B.Csx10^3	8.3±0.6	8.4±0.6	8.3±0.6	8.3±0.6	9.8± 0.3**	10.3± 0.6**	10.8± 0.3**	10.9 ± 0.2**
Total lipids, mg/dL	591±0.5	595±0.5	597±0.4	601±0.6	601.5±0.6**	610±0.5**	621 ±0.5**	664±0.4**
Total cholesterol ,mg/dL	163±0.8	168±0.5	171.2±0.6	175±0.4	166±0.4	169±0.2	170±0.2	172±0.4
Glucose mg/dl	131.0±0.3	130.8±0.7	130.2±0.6	132.2±0.4	141±0.3**	146±0.7**	152±0.7**	162±0.7**
Urea mg/dl	19.5±0.13	21.4±0.04	22.3±0.07	22.5±0.06	31.2 ±0.37**	32.0±0.31**	37.4±0.4**	38±0.32**
Creatinine mg/dl	0.94±0.03	0.96±0.03	0.98±0.02	0.98±0.02	1.28±0.04**	1.46±0.05**	1.66 ±0.05**	1.76±0.03**

Data were presented as means value ± S.E.   Significant =   highly significant =***

Table (4): Comparison between group received lead acetate(20mg/kg.body weight /day /four weeks)) with group received both lead acetate & vitamin C (20mg lead acetate plus 50 mg vitamin C/kg .body weight/day/ four weeks) on blood parameters of rats.

Groups	Lead acetate group second group				Lead acetate and vitamin C group Third group			
Weeks / Parameters	First week	Second week	Third week	Fourth week	First week	Second week	Third week	Fourth week
R.B.Csx10^6 cells	6.2±0.07	5.5±0.03	5.1±0.05	4.5± 0.03	6.2±0.04	6.3±0.04**	6.5±0.04**	6.8±0.04**
Hb ,g%	13.2±0.04	12.9±0.05	12.1±0.07	11.8±0.07	12.6±0.12**	13.0±0.04**	13.3±0.1**	12.6±0.1**
Hct value	36.4±2	32.9±2	32.7±1.8	32.4±1.8	38.7±1.1**	39.1±1.1**	40.5±1**	42.2±0.9**
W.B.Csx10^3 cells	6.4±0.7	6.1±0.7	5.3± 0.5	5.0± 0.4	7.5±0.3**	7.6±0.6**	7.7±0.3**	7.9±0.2**
Total lipids ,mg/dL	662.6± 0.9	681.8±0.6	731.2±0.6	751.2±0.7	652±0.7**	641.4±0.5**	652±0.7**	642.2±0.9**
Total cholesterol ,mg/dL	179.2± 0.6	189.4±0.5	197.4± 0.5	212±0.7	165±0.7**	171.4±0.5**	174.6±0.3**	181.4±0.5**.
Glucose mg/dl	152.4±0.5	161.2± 0.4	165±0.5	167.2±0.4	126.6±0.9**	124.2±0.7**	123.4±0.5**	120±0.2**
Urea mg/dl	41.3± 0.5	45.8±0.35	51.7±0.54	56.4±0.43	31.5±0.25**	36±0.35**	41.3±0.53**	47.6±0.51**
Creatinine mg/dl	1.68 ±0.04	1.88 ± 0.02	2.6± 0.03	2.78±0.04	1.24 ±0.03**	1.44±0.03**	1.8±0.03**	1.74±0.03**

Data were presented as means value ± S.E.   Significant =   highly significant =***

Table (5): Comparison between lead acetate group (20 mg/kg, body weight/ day /four weeks) with lead acetate &selenium group (20 mg lead acetate Plus o.1mg selenium/kg. body weight/day/four weeks) regarding blood parameters of Rats.

Groups	Lead acetate group second group				Lead acetate and Selenium group Fourth group			
Weeks / Parameters	First week	Second week	Third week	Fourth week	First week	Second week	Third week	Fourth week
R.B.Csx10^6 cells	6.2±0.07	5.5±0.03	5.1±0.05	4.5± 0.03	6.5 ±0.09**	6.7 ±0.07**	6.7 ±0.07**	6.8±0.02**
Hb ,g%	13.2±0.04	12.9±0.05	12.1±0.07	11.8±0.07	13.2±0.04	13.0±0.05**	13.3±.07**	13.2±0.07**
Hct value	36.4±2	32.9±2	32.7±1.8	32.4±1.8	38.5±2**	38.7± 2**	38.5± 1.8**	41.9± 1.8**
W.B.Csx10^3 cells	6.4±0.7	6.1±0.7	5.3± 0.5	5.0± 0.4	9.8± 0.3**	10.3± 0.6**	10.8± 0.3**	10.9 ± 0.2**
Total lipids,mg/dL	662.6± 0.9	681.8±0.6	731.2±0.6	751.2±0.7	601.5±0.6**	610±0.5**	621 ±0.5**	664±0.4**
Total cholesterol ,mg/dL	179.2± 0.6	189.4±0.5	197.4± 0.5	212±0.7	166±0.4**	169±0.2**	170±0.2**	172±0.4**
Glucose mg/dl	152.4±0.5	161.2± 0.4	165±0.5	167.2±0.4	141±0.3**	146±0.7**	152±0.7**	162±0.7**
Urea mg/dl	41.3± 0.5	45.8±0.35	51.7±0.54	56.4±0.43	31.2 ±0.37**	32.0±0.31**	37.4±0.4**	38±0.32**
Creatinine mg/dl	1.68 ±0.04	1.88 ± 0.02	2.6± 0.03	2.78±0.04	1.28±0.04**	1.46±0.05**	1.66 ±0.05**	1.76±0.03**

Data were presented as means value ± S.E.   Significant =   highly significant =***

DISCUSSION

Lead is one of the trace elements essential for animal and human health. These elements, along with amino and fatty acids as well as vitamins, are required for normal metabolic process. However, lead overdose induces hepato-renal dysfunction due to lead-mediated lipid peroxidation [8]. In the present study rats received orally lead acetate in a single dose (20 mg/kg. body weight/day /four weeks) showed a decrease in, hemoglobin content due to, lead toxicity that may be attributed to the disturbance of iron metabolism including absorption, transport and cellular uptake, which led to inhibition of hemoglobin synthesis. On the other, hand, the main effect of lead may be through alteration of enzyme activities responsible for heme synthesis. The toxic lead may affect the production of red blood cells, white blood cells and hemoglobin. In addition, the results (table 1) showed a significant decrease in hematocrit value of rats exposed to lead than the control groups this might be, attributed to dilution of the blood associated with a decrease in the number of RBCs. These results agree with Heibashy and Amer [30] who reported that, the reduction of blood iron and increase in ferritin pointed to the decrease in hemoglobin synthesis. In addition, the present results agree with that of Nissenson et al., [31] .and Hense et al., [32] who interpreted the positive association between low lead levels and hematocrit or hemoglobin as a reflection of the lead- binding capacity of erythrocytes, since more than 95%of blood lead is bound to red cells. Consequently, anemia is a manifestation of lead toxicity, however lead inhibits the body's ability to synthesize hemoglobin by interfering with several enzymatic steps in the hem pathway. Also the present results agree with Widdop [33] who interpreted the decrease of red blood cells and hemoglobin due to lead binding to thiol groups and is thus a potent enzyme inhibitor, particularly those involved in heam synthesis, that results in decreased heam production and accumulation of protoporphyrin in red blood cells. Also the present study in accordance with those obtained by Tepper and Levine [34].; Sinovcic et al., [35] Poulos et al., [36].and Zaahkouk & Abd-El – Reheem [3&4]. They reported that a significant decrease of hematocrit value and hemoglobin concentration in rats exposed to heavy metals ,could be attributed to high blood heavy metals levels which are thought to reflect heme synthesis inhibition or intraheptic and intraspleenic hemorrhage and disturbances of osmotic pressure inside and outside the cell . Moreover, Oski [37] and Booth and Aukett [38].suggested that the hematological changes attributed to lead poisoning may result from coexisting iron deficiency ,since lead poisoning may serve to produce decrease of red blood cells and hemoglobin . The result of the present study (table 1) in which rats orally received lead, showed a highly significant decreased in total leucocytes count, compared with the control group and may be attributed to lead exposure that have effect on hemopoietic cells and on aggravation of the leucopenia.

Lead toxicity caused definite impairment of kidney functions, which is manifested as increase in blood urea and creatinine (Table 1).This dysfunction may be attributed to severe damage in the proximal tubular cells. Renal failure was found to occur when the kidneys are unable to maintain a normal internal environment, early feature by rising plasma urea, this may be due to a decreased glomerular filtration rate(GFR) which may be attributed to decreased renal blood flow (function renal impairment); decreased number of functioning nephrons or obstruction to urine flow. It is generally accepted that the plasma creatinine value is a better indicator of the GFR than urea because the urea is affected by the protein intake ,this is only true when the creatinine value is significantly raised .The results of the present study agree with Grossman[39] and Heibashy and Amer [30] who reported that the increased plasma urea is due to a decreased GFR as a consequence of a decreased renal blood flow .Also our results are in harmony with those reported by Epinel and Gegory, [40] and Champbell and Ofurum, [41] who reported that increased plasma urea and creatinine may be due to the following :(1)decreased renal excretion, (2) increased production of urea due to increased protein catabolism (3)increased creatinine production due to increased dietary intake or of large muscle mass.

The results in table 1in which rats received orally lead showed significant increase in serum total lipids and cholesterol in comparison with control, this might be attributed to lead effect on metabolism of the total lipids. Lead mediated free radicals production could be involved in the increase in lipid peroxidation of lipid moiety of cellular membrane and may distort the structural integrity of these membranes, thereby modifying their functions .One of the most important function of the cell membrane is transport of various molecules into and out of the cells, therefore, increased lipid peroxidation due to lead toxic may be impair the rate of total lipids and fractions transport into the cells. In addition, the results in table1 in which rats orally received lead showed highly significant increase in serum glucose level compare with control group. Which may be either due to decreased glucose utilization or inhibited hepatic glycolysis . This observation was similarly recorded by Mazeaud *et al*.,[42] who reported to an enhanced glycogen breakdown in liver by the accumulated lead.

A wide range of antioxidants, have been proposed for use in the treatment of many human diseases[12]. Antioxidant can improve insulin action and glucose disposal[43]. In the third group of this study in which the rats orally lead & vitamin C the results, showed that improvement in blood pictures. Red &white blood cells count, hemoglobin and haematocrit value showed higher significant increase when compared with the second group in which rats orally lead acetate only. The improvement of these parameters may be, attributed to the antioxidant nature of vitamin C which protects the erythrocytes in the circulation from the oxidative effect of lead toxicity .On the other hand higher significant

decrease was found in serum glucose when compared with the control, while on comparing the glucose level of the third group with the second group it was found that an improvement of glucose may be attributed to the extent of oxidative stress directly by scavenging free radicals and indirectly via their insulinogenic and /or insulin-mimetic effects. Similar results by (Hilton, [44] who reported that vitamin C is a powerful reducing agent capable of reducing certain transition metal ions. On the other hand, the hyperglycemic due to oral lead administration and vitamin C may be antagonized by the inhibitory effect of vitamin C on gluconeogenesis process suggested by Cornel [45]. In addition, antioxidant defenses vitamin C, catalase and reduced glutathione have been reported to be decreased in diabetes[46]. The present results are in accordance with those of previous investigators. Young et al. [11], showed that vitamin C treatment in diabetic rats resulted in a significant reduction in plasma glucose level when compared to untreated diabetic animals. Furthermore, Othman and Momena[47], revealed that treatment of diabetic guinea pigs with ascorbic acid induced marked normalization of plasma glucose level. Otherwise Paolisso et al., [48] (1994) and Fesken et al., [49], reported an increase in insulin in diabetic and non –diabetic animals after vitamin C infusion and this improvement in insulin after vitamin C administration might be attributed to its ability to improve the physical state of plasma membrane and its related to activity as glucose transport through increment of hepatic

reduced gluthathione levels thus interferes with the progression of lipid peroxidation .

In addition, there were improvement in the serum urea and creatinine compared with the second group.

Data in table (4) illustrated an improvement in total lipids and cholesterol in serum of rats orally received lead acetate (20mg/kg.body weight /day /four weeks) together with vitamin C(50 mg/kg body weight /day /four weeks).The decreased level of cholesterol and total lipids in the third group compared with the second group after rats treatment by lead and vitamin C might be attributed to the involvement of vitamin C in the regulation of activity of lipoprotein lipase or in the hydroxylation of cholesterol to bile acids[50&51].

Generally, the treatment of rats with selenium and lead showed significant improvement in hematological parameters except there was a higher significant increased in white blood cells than the first or the second groups table (3&5). This improvement may be related to the ability of selenium which act in different ways to protect against the hepato-nephrotoxicity of lead by direct interference with one or more of subsequent steps of its metabolic activator and selenium can alter lead distribution in target tissues and thereby alter the progress of peroxidative processes. These results agree with Rana and Boora [52]and Rana and Verma, [53]. who reported that an antagonistic interrelationship between heavy metals and selenium, as direct interference of enzyme activity of lipid perodidation, in which

selenium –lead interaction can be brought about by endogenous glutathione that reduce selenite to a selenide compound, the high lipoaffinity of this compound may alter their distribution and toxicity in critical tissues. It is clear from tables(2,3,4&5) that rats exposed to lead with vitamin C or lead with selenium exhibit more or less similarity to the control group in all of the studied parameters .This could be attributed to the ameliorative effects of ascorbic acid and selenium on heavy metals toxicity that may be mediated through their antioxidative actions. Also the present results agree with Ghazaly[54].   and Brake[55]who reported that ascorbic acid act as scavenger of reactive oxygen species and it may protect the lipid from detectable per oxidation damage induced by aqueous free radical,and also added that vitamin C has been shown to enhance the urinary elimination of metals to reduce hepatic and renal burden of metal.

In conclusion the pathobiological events of lead toxicity may be amended by treating the animals with one of these nutrients (vitamin C or selenium) ,and other effect of toxic lead can be improvement by using vitamin C or selenium.

Recommendation: The present study throw lighter on one the most serious phases of lead toxicity which emphasizes the importance of performing more studies to explore all the consequences of heavy metals pollution. This could be agate way to determine means for protection against this pollution by using antioxidants substances.

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التأثير الوقائي لفيتامين ج والسلينيوم ضد السمية المحدثة بخلات لرصاص
على بعض العناصر الفسيولوجية في ذكور الجرذان

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الهدف هو دراسة تأثير سمية الرصاص على العناصر الفسيولوجية في الجرذان ثم علاج هذا التسمم ببعض المواد المؤكسدة مثل فيتامين ج والسيلينيوم. قسمت التجربة إلى أربع مجموعات كل مجموعة بها خمس جرذان المجموعة الأولى هي الضابطة أما المجموعة الثانية فقد تم إعطائها جرعة يومية من خلات الرصاص (٢٠ملجرام لكل كيلو جرام من وزن الجسم) بالتجريع عن طريق الفم باستخدام حقنة مدرجة أما المجموعة الثالثة فقد تم إعطائها نفس الجرعة من خلات الرصاص بالإضافة إلى جرعة من فيتامين ج (٥٠ملجرام لكل كيلوجرام من وزن الجسم) .و المجموعة الرابعة تم إعطائها نفس الجرعة من خلات الرصاص مع جرعة من السلنيوم (١و .لكل كيلو جرام من وزن الجسم) .كانت مدة التجربة أربع أسابيع وأن الأكل والماء كان حرا. وقد أظهرت النتائج التأثير السام للرصاص منفردا في المجموعة الثانية التي أعطيت خلات الرصاص : وجد نقص معنوي في عدد خلايا الدم الحمراء والبيضاء و في تركيز الهيموجلوبين ومستوى الهيماتوكريت مقارنة بالمجموعة الأولى (الضابطة).أما العناصر الفسيولوجية الأخرى فكانت النتائج ايجابية وجدت زيادة معنوية في تركيز   كلا من الدهون الكلية و الكولسترول والجلوكوز وأيضا زيادة معنوية فى تركيز البولينا والكرياتنين مقارنة بالمجموعة الضابطة . وكانت نتائج المجموعة الثالثة والتي أخذت (خلات الرصاص وفيتامين ج معا) أظهر فيتامين ج تأثيرا مضادا ومزيلا لسمية الرصاص وذلك فقد أظهرت النتائج تحسنا واضحا في صورة الدم بالمقارنة بالمجموعة الثانية التي أخذت الرصاص فقط مما يشير إلى التحسن الوظيفي في أجهزة الجسم وخاصة خلايا الكبد ونخاع العظام بعد العلاج بفيتامينُ ج. على الجانب الأخر أظهرت النتائج تحسنا العناصر الفسيولوجية في المجموعة الثالثة في كلا من الدهون   الكلية و الكولسترول والبولينا والكرياتنين و   في تركيز الجلوكوز مقارنة بالمجموعة الثانية . أما المجموعة الرابعة التي قد أعطيت خلات الرصاص و السيلينيوم فقد أظهرت النتائج الآتي:وجد تحسنا في صورة الدم حيث إلا زيادة معنوية في خلايا كرات الدم البيضاء.وعلى الجانب الآخر وجد تحسنا في العناصر الفسيولوجية الأخرى في كلا من الدهون الكلية والجلوكوز و البولينا و الكرياتنين و الكوليستيرول   بالمقارنة بالمجموعة الثانية التي أخذت الرصاص فقط. وقد خلصت الدراسة إلى إثبات التأثير المحسن لإعطاء كلا من فيتامين ج والسيلينيوم على الخلل الوظيفي لأجهزة الجسم الناتجة عن إعطاء الرصاص .

توصية: تلقى الدراسة الحالية الضوء على أحد الجوانب الخطيرة للتسمم بعنصر الرصاص وهو ما يؤكد على ضرورة إجراء المزيد من البحوث للكشف عن جميع جوانب التلوث بالمعادن الثقيلة والذي يفتح المجال إلى التوصل إلى أساليب الوقاية من هذا التسمم باستخدام مضادات الأكسدة.