Hematological and Biochemical Studies for Gasoline Toxicity Among Gasoline Workers In Gaza Strip.

Prof. Ismail I. Abdel Aziz*
Dr. Salam Zakaria Al Agha **
Dr. Ossama ahmed shehwan ***

الملخص

دراسة دموية وبيوكيميائية لتأثير التعرض للجازولين المخلوط بالرصاص على العاملين في محطات الوقود بقطاع غزة

•يتضمن البحث دراسة تأثير الجازولين المخلوط بالرصاص على الأشخاص الذين يعملون في محطـات البنزين . وقد اشتملت الدراسة على دراسة علم الدم ودراسة بيوكيميائية الدم للأشخاص المعرضين للبنزين المخلوط بالرصاص .

•وقد تبين من خلال النتائج مدى خطورة التعرض مباشرة للبنزين المخلوط بالرصاص خـصوصا علـى المعايير الدراسية المختلفة ، حيث يناسب هذا طرديا مع زيادة التعرض للبنزين المخلوط بالرصاص .

•حيث أظهرت الدراسة أن هناك نقصا ملحوظا في نسبة الهيموجلوبين كما سجلت نقصا ملحوظا في عدد كريات الدم الحمراء ، كما أظهرت الدراسة نقصا في معدل الهيماتوكريت، بينما لوحظ أن هناك زيادة في عدد كريات الدم البيضاء وعدد الصفائح الدموية .

•كذلك أظهرت الدراسة زيادة في معدل فحص اليوريا والكرياتنين وحمض البوليك ونسبة البروتين الكلى حيث لوحظ أنها في زيادة لنستدل من ذلك على خلل وظيفي في الكلى عند هؤلاء الأفراد .

•كذلك أوضحت الدراسة أن هناك زيادة واضحة في نشاط الأنزيمات الناقلة ALT ، AST وكذلك أنزيم الفوسفاتيز القلوي ، حيث تدل هذه النتائج على وجود خلل في عمل وظائف الكبد وذلك عند زيـادة نـسبة الرصاص في أجسادهم زيادة مزمنة .

•لذا فإنه من الملاحظ أن هذه الدراسة قامت بتسليط الضوء على أهم عوامل المؤثرات التي تـؤثر علـى صحة الإنسان والتي تتطلب إستراتيجية من شأنها لعب دور مهم في الوقاية من التعرض للرصاص. لذلك هناك حاجة ماسة لجهود كبيرة لتطبيق برامج تدريبية وتثقيفية وصحية والتوعية الكاملة لاسـتخدام أدوات الوقاية والسلامة.

* Department of comparative anatomy, Faculty of Science, Islamic university of Gaza, Palestine.
** Department of Biology, Faculty of Science, Al.Aqsa University, Gaza, Palestine.
***El Manar laboratory center , Khanyuonis, Gaza, Palestine.

ABSTRACT

The present work is aimed to show the induced toxic effect of leaded gasoline toxicity among gasoline worker in Gaza Strip.

The following parameters were estimated. Hematological parameters including: Complete blood cell count: Hb., RBC, HCT, WBC and platelets.

Biochemical Parameters Including:

1. Kidney function tests which are composed urea, createinen and uric acid.
2. Liver function tests which are composed of ALT , AST, Alkaline Phosphates and total protein.
3. Measurement of levels of lead in serum of gasoline workers by atomic absorption.

- Hemoglobin content and hematocrit value were decrease in parallel manner with decrease in red blood cell count. , in contrast, the WBC count was increased as compared to the control group.
- The present result also showed general increase in urea , creatinine , and uric acid.
- In the period of study showed a general increase in alkaline phosphatase , ALT and AST activity as compared to control group, at high level of lead intoxication.

Keywords: Leaded gasoline, Gasoline workers, Hematological Parameters, liver function, kidney function.

INTRODUCTION:

Lead is one of the oldest chemical toxins, lead fumes and dioxide were probably amongst the first workplace health and safety issues.

The effect of lead exposure on various organs and systems is discussed pathologically in term of dose effects and environmentally as a dose-response.

These hemopoietic change are mainly secondary to the effect of lead on the normal pathway of hemoglobin system synthesis (Cramer *et al*., 1975).

Ong and Lee (1980) stated that lead has a high affinity for intracellular constituents of RBC and less for the stoma membrane.

Abdel Aziz (2002) noticed that lead acetate administration revealed that levels of serum albumin , total proteins , lactate dehydrogenase (LDH) activity, aspartate aminotransferase (AST) and acid phosphates were affected due to lead toxicity.

Lead poisoning may affects numerous organ systems and is associated with anumber of morphological and biochemical changes, including

kidney dysfunction, rate of reproduction , abnormal glucose metabolism, nervous system , bone metabolism, impairment of liver function and hematological disorders (Al-Saleh 1994; Dos-Santos et al., 1994; Gupta et al., 1995; Belacy et al., 1996; Yun and Hoyer 2000; Dowd et al., 2001; Lavicoli et al., 2003 and Sivaprasad et al., 2003).

Review of Literature:

Lead serves no useful purpose in animal or human body and its presence in the body can lead to toxicity regardless of the exposure pathway. Its toxicity can affect every organ system. The proposed mechanisms for toxicity could involve the fundamental physiological and biochemical processes.

The anemia that occurs in lead poisoning results from two basic defects:

1. Shortened erythrocyte life span .
2. Impairment of heme synthesis .

Shortened of life span of RBC is thought to be due to increased mechanical fragility of cell membrane. Hernberge et al. 1967.

Gautam and Chowdhury (1987), noticed that erythropoietic alteration in normal and splenectomized mature male rats treated with aqueous lead acetate intraperitoneally at dosages of 4 and 6 mg/kg body weight over a period of 30 days.

Changes in the morphology of Erythrocytes revealed that lead might increased the development of irregularly shaped blood cell, and the development of anemia resumed by a decrease in Hb, Hematocrit and red cell count (Bazzaz et al., 1989).

Bazzaz et al. 1989, noticed that the change in the morphology of erythrocytes revealed that lead might increased the development of irregularly shaped blood cell and the development of anemia.

Bazzaz et al. 1989, reported that there is significant reduction of hemoglobin, packed cell volume, number of erythrocytes and leucocytes associated with significant increase in the number of monocytes were observed in case of lead intoxication.

Exposure to lead significantly decrease red blood cell counts, hemoglobin level and haematocrit value of rats (Terayama 1993).

Goyer (1996), stated that the effects of lead over exposure on heme synthesis have been throughly investigated and there is a consensus that adverse effects on Hb are associated with BLL values of 50 µg/dL in adults.

Anemia can result from both shortened red cells life span and impairment of heme synthesis.

Kim et al. (2003), noticed that anemia accompanies lead poisoning is in part the result of various inhibitory effects of lead on heme biosynthesis. Lead also increase the rate of red blood cell destruction due to the profoundly depressed activities of erythrocyte pyrimidine 5-nucleotidase activities.

Exposure to lead at different doses in drinking water significantly decrease red blood cells count, hemoglobin concentration and haematocrit value of rabbits (Bersenyi, et al., 2003).

Payton et al. (1994), found that early kidney damage is difficult to detect. However, a 10mg/dL increase in BLL has been associated with a 10.4 ml/minute decrease in renal creatinine clearance rate.

Hogan et al. (1992), pointed out that renal parameters showed modifications of blood urea nitrogen levels for both oral and intraperitoneal administered male wistered rats with lead acetate for 4 weeks which can indicate a prerenal uremia. This was supported by the significant increase of creatinine in the first week of lead treatment by intraperitoneal injection.

Daily oral administration of lead acetate at dose of 40 mg/kg body weight caused significant increase in serum urea, uric acid, and creatinine of rabbits Ashour (2002).

Speich et al. (1983), noticed that elevation of amino transferases SGOT and SGPT in serum of experimental animals. In human cases both positive

and negative finding have been reported by (Waldron 1975, Tola and Nordmen 1977).

Abdel Aziz (2002) identified the biochemical changes in adult male domesticated rabbits following oral administration of lead acetate at dose of 40 mg/kg body weight daily for 20 days. Data revealed that levels of serum AST and ALP were significantly increased.

Sivaprasad et al. (2003), found that the activities of serum SGOT and SGPT were elevated in rats administered 2% lead acetate drinking water for 5 weeks.

MATERIAL AND METHODS:

The material of this work consisted of 80 workers occupationally exposed to leaded gasoline and a control group of (18) healthy workers who have never been exposed to leaded gasoline.

1- The exposed group was divided into five sub-groups according to their blood lead levels:

- 1st 30-40 µg/dL
- 2nd 40-50 µg/dL
- 3rd 50-60 µg/dL
- 4th 70-80 µg/dL
- 5th > 90 µg/dL

1. Blood Sampling:

Blood sample about 8 ml was down from each individual of gasoline workers group and non exposed group to leaded gazoline. The blood samples were divided into two tube , 2ml on EDTA tube for complete blood count including Hb , WBCs , Haematocrit , RBCs and platelets.

The other 6 ml of blood were left short time to allow blood to clot then clear serum sample was obtained by centrifugation at 3000 rpm to measure chemical tests:

2. Chemical Analysis:

- Serum total protein , albumin , urea , and creatinine were estimated by method of Mackary and Mackary (1927).
- Serum uric acid was determined by using the technique specified by Fossati etal, 1980 .
- On the other hand alkaline phosphates activity , SGOT , and SGPT were determined to the method of Trendier (1969).

3. Statistical Analysis

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

RESULTS:

• Hematological Studies:

1. Blood hemoglobin level (Hb):

The mean value of blood hemoglobin level in control and the studied groups were shown in table (1). Blood hemoglobin level in control group exhibited value of (14.5 g/dl). In studying groups blood hemoglobin level was decreased to (13.4, 12.7, 12.0, 10.2 and 8.8 g/dl) with percentage decreases of (7.59, 12.41, 17.24, 29.66 and 39.31 %) compared to the control group.

2. White blood cell count :

On detecting the white blood cell count the data are given in table (2). In contrast to the red blood cell the white blood cell count showed a general increase as compared to the control group. However, the increase in white blood cell count was significant in the third, fourth and fifth group with value of (9.1), (10.2) and (10.8) respectively compared with control group. With percentage increase of (12.35%), (25.93%) and (33.33%) as compared to control group.

3. Hematocrit value (PCV) or (HCT) :

On detecting the hematocrit value the data are given in table (3) . In the studied groups, the result showed a general decrease in hematocrit value as compared to the control group. However the decrease in hematocrit (PCV)

percentage was highly significant in the fifth group, with a value of (31.40%) of the control level.

4. Red blood cell count (RBC):

The data shown in table (4) demonstrated the mean value of red blood cell count of both control and studied groups. Red blood cell count showed a significant decrease in total red blood cell count in all over the studies of group examined in studying groups red blood cell count were decreased to " 3.7 , 3.2 , 3.0 , 2.9 and 2.8 " ($x10^6$ cell/ µl) with a percentage decreases of (19.57 , 30.43 , 34.78 , 36.96 and 39.13 %) compared to control group.

5. Platelets count :

On detecting the blood platelet count (PLT), the data are given in table (5). The present result also showed a general increase in platelet count as compared to the control group. However, the significant increase in platelet count was observed in the group four and group five with a value of ({243.3 and 252.2} x 10^3 cells µ/l) respectively of the control level.

Table 1. Average Hb Content Level In Both Normal And Studies Groups of Gasoline Workers

	control group	**studies group**				
	lead con. >30 n=18	**group 1 (30-40)**	**group 2 (40-50)**	**group 3 (60-70)**	**group 4 (70-80)**	**group 5 (>90)**
Mean ± S.E	14.5 ± 0.8	13.4 ± 0.9	12.7 ± 0.9	12.0 ± 0.6	10.2 ± 0.9	8.8 ± 0.9
S.D	0.8	0.6	0.9	0.4	0.4	0.5
P. value		0.95	0.52	0.68	1.69	1.06
level of significant		Non significant	significant	significant	significant	significant
% of change		-7.59	-12.41	-17.24	-29.66	-39.31

Hb. Level showed significant reduction in the studied groups in comparison to the control group .

Table 2. Average Of WBCs Count In Both Normal And Studies Groups Of Gasoline Workers

	control group	**studies group**				
	lead con. >30 n=18	**group 1 (30-40)**	**group 2 (40-50)**	**group 3 (60-70)**	**group 4 (70-80)**	**group 5 (>90)**
mean ± S.E	8.1 ± 0.7	8.1 ± 0.7	8.6 ± 0.6	9.1± 0.7	10.2± 0.7	10.8± 0.7
S.D	0.7	0.2	0.6	0.4	0.3	0.4
P. value		-0.07	-0.55	-0.49	-1.17	-0.56
level of significant		non significant	non significant	significant	significant	significant

% of change		0.00	6.17	12.35	25.93	33.33

WBCs count showed non significant increase in the first (2) groups . the remaining groups showed significant increase

Table 3. Average HCT Value Among The Gazoline Workers And The Control Groups

	control group	studies group				
	lead con. >30 n=18	group 1 (30-40)	group 2 (40-50)	group 3 (60-70)	group 4 (70-80)	group 5 (>90)
mean ± S.E	43.0 ± 2.1	40.2 ± 2.3	38.2± 2.3	36.2 ± 2.4	33.1 ± 2.4	29.5 ± 2.4
S.D	2.2	1.6	2.3	1.3	1.3	1.3
P. value		0.89	0.63	0.59	0.93	1.07
level of significant		non significant	significant	significant	significant	significant
% of change		-6.51	-11.16	-15.81	-23.02	-31.40

Table (3) Showed reduction in HCT value in the studied groups with high significant reduction in the group (5)

Table 4. Average Of RBCs Count In Both Normal And Studied Groups Of Gasoline Workers

	control group	studies group				
	lead con. >30 n=18	group 1 (30-40)	group 2 (40-50)	group 3 (60-70)	group 4 (70-80)	group 5 (>90)
mean ± S.E	4.6 ± 0.2	3.7 ± 0.3	3.2± 0.4	3.0 ± 0.3	2.9 ± 0.3	2.8 ± 0.3
S.D	0.3	0.5	0.4	0.2	0.2	0.2
P. value		23.4	0.99	0.35	0.28	0.29
level of significant		significant	significant	significant	significant	significant
% of change		-19.57	-30.43	-34.78	-36.96	-39.13

RBCs count was significantly decreased in all the studied groups in comparison to the control.

Table 5. Average Platelet Count In Both Normal And Studied Groups Of Gasoline Workers

	control group	studies group				
	lead con. >30 n=18	group 1 (30-40)	group 2 (40-50)	group 3 (60-70)	group 4 (70-80)	group 5 (>90)
mean± S.E	211.8±7.3	216.7±13.1	224.0±15.4	231.9± 16.0	243.3± 16.0	252.2± 16.0
S.D	15.2	14.5	15.4	11.2	8.9	10.0
P. value		-0.32	-0.36	-0.35	-0.50	-0.39

level of significant		non significant	non significant	non significant	significant	significant
% of change		2.31	5.76	9.49	14.87	19.07

Platelet count showed non significant increase in the first (3) groups . the remaining groups showed significant increase

• Biochemical Studies :

1) Serum urea level :

The mean value of serum urea concentration in control and studied groups is shown in table (6). urea concentration in control group exhibited value of (27.4 mg/dl). In the studied groups serum urea concentration was increased to (30.0 , 33.3 , 36.0 , 38.3 and 43.0 mg/dl) with a percentage increases of (10.18%) , (21.53 %), (31.39 %) , (39.78 %) and (56.93 %) as compared to control group .

2) Serum creatinine level :

The mean values of serum creatinen concentration in control and studied groups are shown in table (7). Creatinen concentration in control group exhibited a value of (0.9 mg/dl). In the studied groups serum creatinen concentration was increased to (1.0, 1.1, 1.2, 1.3 and 1.5 mg/dl) with percentage increase of (11.11%), (22.22%), (33.33%), (44.44%) and (66.67%) as compared to control group.

3) Serum uric acid level :

The data of blood serum uric acid level under investigation is illustrated in table (8). Uric acid concentration in control group exhibited value of (3.7 mg/dl). In the studied groups, serum uric acid concentration was increased to (4.0, 4.3, 4.6, 5.0 and 5.2 mg/dl) with percentage increases of (8.11%), (16.22%), (24.32%), (35.14%) and (40.54 %) as compared to control group.

4) Serum total protein concentration :

The data of blood serum total protein level under investigation is illustrated in table (9) demonstrate the mean value of serum total protein concentration level of both control and studied groups. However the increase in the level was more pronounced in the fifth group with value of (8.42 mg/dl) of the control level.

5) Serum alanine amino transferase activity (ALT) :

The mean value of serum alanine amino transferase activity in control and studied group during the study are presented in table (10). Normal Serum alanine amino transferase activity was (28.6 u/l). Upon studied group, the ALT activity was increase throughout the study. However the increase in

alanine amino transferase activity was more obvious in the fifth group with a percentage of (23.43%) compared to control group.

6) Serum aspartate amino transferase (AST) :

Data recorded for serum AST activity was presented in table (11). Serum aspartate amino transferase activity was (27.4 u/l). Upon studied group, the aspartate amino transferase activity was increase throughout the study. However the increase in aspartate amino transferase activity was more obvious in the fifth group with a percentage of (27.37 %) compared to control group.

7) Serum alkaline phosphates :

The data shown in table (12) , showed serum alkaline phosphates activity in control a studied groups. The normal alkaline phosphates activity was (80.4 ul). Upon studied groups, alkaline phosphates activity was increased through out the study. However the highest change in alkaline phosphatase activity was observed in the fifth group with a percentage increase of (15.67%) compared to the control group.

Table 6. Average Of Urea Concentration In Both Normal And Studies Groups Of Gasoline Workers

	control group	**studies group**				
	lead con. >30 n=18	**group 1 (30-40)**	**group 2 (40-50)**	**group 3 (60-70)**	**group 4 (70-80)**	**group 5 (>90)**
mean ±S.E	27.4 ± 2.5	30.19 ± 3.0	33.3± 3.3	36.0± 3.4	38.3± 3.4	43.0± 3.4
S.D	3.3	2.6	3.3	2.9	2.3	3.7
P. value		-0.68	-0.74	-0.58	-0.48	-0.97
level of significant		significant	significant	significant	significant	significant
% of change		10.18	21.53	31.39	39.78	56.93

Table (6) Showed significant increase of urea level in the studied groups of gasoline workers in comparison to the control.

Table 7. Average Of Creatinine Concentration In Both Normal And Studies Groups Of Gasoline Workers

	control group	**studies group**				
	lead con. >30 n=18	**group 1 (30-40)**	**group 2 (40-50)**	**group 3 (60-70)**	**group 4 (70-80)**	**group 5 (>90)**
mean ± S.E	0.9 ± 0.1	1.0 ± 0.1	1.1± 0.1	1.2± 0.1	1.3± 0.1	1.5± 0.1
S.D	0.1	0.1	0.1	0.1	0.1	0.1
P. value		-0.71	-0.73	-0.83	-0.69	-0.84
level of significant		significant	significant	significant	significant	significant
% of change		11.11	22.22	33.33	44.44	66.67

There is significant increase in serum creatinine level among the studied groups in comparison to the control .

Table 8. Average Of Uric Acid Concentration In Both Normal And Studies Groups Of Gasoline Workers

	control group	studies group				
	lead con. >30 n=18	group 1 (30-40)	group 2 (40-50)	group 3 (60-70)	group 4 (70-80)	group 5 (>90)
mean ± S.E	3.7 ± 0.4	4.0 ± 0.4	4.3± 0.5	4.6± 0.5	5.0± 0.5	5.2± 0.5
S.D	0.5	0.3	0.5	0.3	0.2	0.2
P. value		-0.49	-0.48	-0.54	-0.62	-0.24
level of significant		non significant	significant	significant	significant	significant
% of change		8.11	16.22	24.32	35.14	40.54

Serum uric acid level showed significant increase among the studied groups of gasoline workers expect group (1) in comparison to the control.

Table 9. Average Of Total Protein Concentration In Both Normal and Studies Groups of Gasoline Workers

	control group	studies group				
	lead con. >30 n=18	group 1 (30-40)	group 2 (40-50)	group 3 (60-70)	group 4 (70-80)	group 5 (>90)
mean ±S.E	6.9± 0.3	7.4 ± 0.4	7.6± 0.4	8.0± 0.4	8.2± 0.4	8.42± 0.4
S.D	0.4	0.5	0.4	0.4	0.4	0.3
P. value		-1.10	-0.32	-0.79	-0.29	-0.39
level of significant		non significant	significant	significant	significant	significant
% of change		7.25	10.14	15.94	18.84	22.03

Serum total protein showed significant increase in all studied groups of gasoline workers expect group (1) in comparison to the control.

Table 10. Average Of ALT Activity Level In Both Normal And Studies Groups Of Gasoline Workers

	control group	studies group				
	lead con. >30 n=18	group 1 (30-40)	group 2 (40-50)	group 3 (60-70)	group 4 (70-80)	group 5 (>90)
mean ± S.E	28.6 ± 3.0	30.1 ± 3.1	31.6± 3.4	32.8± 3.4	33.8± 4.1	35.3± 4.1
S.D	4.3	3.9	3.4	3.5	3.5	3.2
P. value		-0. 35	-0. 33	-0.26	-0.19	-0.26
level of significant		non significant	significant	significant	significant	significant
% of change		5.24	10.49	14.69	18.18	23.43

There was a significant increase in ALT in the studied groups except group (1) compared to control group.

Table 11. Average Of AST Activity Level In Both Normal And Studies Groups Of Gasoline Workers

	control group	studies group				
	lead con. >30 n=18	group 1 (30-40)	group 2 (40-50)	group 3 (60-70)	group 4 (70-80)	group 5 (>90)
mean ± S.E	27.4 ± 0.9	28.9 ± 1.4	30.2± 1.7	31.8± 4.2	33.6± 4.2	34.9± 4.2
S.D	4.2	4.2	1.7	3.7	3.1	2.9
P. value		-0. 87	-0. 59	-0.35	-0.30	-0.23
level of significant		non significant	significant	significant	significant	significant
% of change		5.47	10.22	16.06	22.63	27.37

There was a significant increase in AST in the studied groups except group (1) compared to control group.

Table 12. Average of Alkaline Phosphates (ALP) Activity In Both Normal and Studies Groups of Gasoline Workers

	control group	studies group				
	lead con. >30 n=18	group 1 (30-40)	group 2 (40-50)	group 3 (60-70)	group 4 (70-80)	group 5 (>90)
mean ± S.E	80.4 ± 1.8	82.1 ± 2.6	83.5± 3.0	84.6± 3.3	86.8± 3.1	93.0± 3.1
S.D	3.3	2.4	3.0	2.4	3.4	7.7
P. value		-0.54	-0.35	-0.25	-0.48	-1.42
level of significant		non significant	non significant	non significant	non significant	significant
% of change		2.11	3.86	5.22	7.96	15.67

Serum ALP showed non significant increase in the studied groups compared to the control.

- All values are expressed as Mean ± SE .
- P value > 0.05 non significant .
  < 0.05 significant .
  < 0.01 highly significant .
  < 0.001 very highly significant .

DISCUSSION:

The present work was carried out in Gaza strip which suffers from several environmental problems. One of these problems in concern resulted from leaded gasoline toxicity.

The problem was identified as a major environmental health problem in Gaza strip.

Human exposure occurs primarily through diet, air, drinking water and ingestion of dirt and chips (U.S. Environmental Protect Agency, EPA 1989 and Agency for toxic substances and disease registry ATSDR 1993).

The efficiency of lead absorption depends on the route of exposure, age and nutritional status (EPA 1986a).

Lead is known to have toxic effect on several biologic systems in particular nervous system, kidney, hematological and reproductive systems. (CDC 1991, and Ghorbe et al., 2001)

Our results revealed inverse significant relationship between lead and hematological parameters. Anemia can results from both shortened red cells life span and impairment of heme synthesis. (Goyer 1996).

These result were supported by previous studies. Litis et al. (1978). The observed decrease in hemoglobin content may be attributed to decrease red blood cells or impaired biosynthesis of heme in bone marrow. (Zayed et al., 1993)

Decreased hemoglobin and red blood cell could also be attributed to insufficiency of protein synthesis that mainly induces decrease of essential amino acids and short age of energy source of protein synthesis incorporated in hemoglobin production. The decrease in red blood cell count observed here is in agreement with that record by Gautam and Chowdhary (1987); Solliway et al.1996; Bersenyi et al. 2003 and Lavicoli et al. 2003). A shortening of erythrocyte survival time was observed in the rats exposed to lead (Terayama, 1993).

On the other hand, white blood cell (WBC) generally increases as compared to the control level. The dramatic increase in white blood cell count indicates the activation of defense mechanism and immune system of gasoline workers. Similar results have been also reported by (Whitby ,1980).

The kidney is one of the main organs of the body that is imbedded in per renal fat and other supporting structures. Each kidney is composed of the functional filtering units, the nephrons.

This in turn is composed of the renal corpuscles and the urineferous tubule both of which are engaged in active physiological functions of the kidney.

Data present here showed elevation in serum urea concentration on gasoline worker with long term exposure to lead. The observed increase in

serum urea is in agreement with the increased level of blood urea concentration with an increased kidney weight that were encountered by Schraishuhu et al. (1992).

Urea is the principal end product of protein catabolism enhanced protein catabolism and accelerated amino acid deamination for gluconeogenesis is probably an acceptable postulate to interpret the elevated levels of urea. On the other hand, the elevated serum urea levels may be due to the destruction of red blood cell during inoculation.

Similar results have been also reported by Khalil-Manesh et al. (1992) delineated that an increase in serum urea nitrogen in rats exposed to lead acetate as compared to controls.

Uric acid is the end product of the catabolism of tissue nucleic acid i.e. purine and pyrimidine bases metabolism (Wolf et al,1972).

The elevation of uric acid in response to lead intoxication coincides with what reported by An Krah et al. (1996) and Mc Bride et al. (1998).

In the present work, the serum uric acid level exhibited significant increment in the gasoline worker, which may be due to degradation of purines and pyrimidines or to an increase of uric acid level by either over production or inability of excretion Wolf et al. (1972).

Total protein biosynthesis was gradually increased due to the lead ion concentration and in the case of low concentration the long period of treatment induced this effect. The observed increase in total serum protein also agreed with Sauk et al. (1992) and Attia (1993).

Thus compensatory mechanisms operate to overcome the toxicity of ingested lead by maintaining a high concentration of glutathione in liver and kidney as shown by increasing total protein level after 60 days of lead administration.

The liver plays a key role in many processes of intermediary metabolism. It is also an important organ in the detoxication of drugs and carcinogens and affects the excretion of a wide range of compounds into bile. This organ is composed almost exclusively of a single type of cells, the hepatocytes or parenchyma cells about 60% of the cells consist of parenchyma cell and 30% consist of endothelial cell (Kupffer cells) which line the hepatic sinusoids. The rest of cells include vascular and supporting tissue and bile ducts (Whitby et al., 1980).

High activities of aminotransferase (ALT & AST) and alkaline phosphates are detected when lead is above 80 ug/dl. These activities are increase due to hepatocellular damage by hepatotoxic drugs infection, hepatitis and primary and secondary liver cancer (Wilkinson ,1977***)***

Transaminase enzymes are synthesized mainly in the liver and their levels in serum are low in normal subjects. Lead may accumulate in liver and exert its toxic effect via per oxidative damage to hepatic cell membranes causing transaminases to liberate into the serum**.** (Sivaprasad et al., 2003).

Serum alkaline phosphates is known to be increased due to over production or release of the enzyme by liver in response to diverse stimuli as hepatocellular injury, increased intraductile pressure inflammatory.

Diseases of the ducts and expanding lesions compressing parenchyma and ducts of the biliary system (Harrison et al., 1964).

The increase activity of alkaline phosphatase in sera of lead exposed workers may provide one of the few dues to the presence of neoplastic growth in liver. (Hawki and Oser ,1965)**.**

Similar results have been also reported by Gupta et al. (1995). They reported that a high alkaline phosphates activity is known to occur in cell or tissue exhibiting either a high turn over or a high anabolic rate.

In conclusion: Our results showed that gasoline workers must be considered as risky personnel as leaded gasoline toxicity affects many systems in the body: liver ,kidney, bone marrow and may lead to organ damage. So, hematological and biochemical assessment are mandatory for those workers periodically.

REFERENCES:

1. Abdel Aziz, I. I. (2002). Biological study on rabbits after administration of lead acetate and treatment by Nigella Saliva oil or olive oil or Garlic lobes. Al Azhar Univ. Gaza, 5: 27-40.

2. Al-Saleh, I. (1994). Biochemical and clinical consequences of lead poisoning. Med. Res. Rev., 14: 415-486.

3. Attia ,H. M.(1993). Protective effect on onion, propiylethiole and systiein against changes of serum protein and enzyme activity induced by lead poisoning. The New Egyp. Journal of Medicine ,8(6):1618-1622.

4. Ashour, A. (2002). Can garlic lobes, olive oil or black seed oil after protection for some serum biochemical constituents against lead toxicity in rabbits. Al-Agsa Univ. J., 9 (1): 74-95.

5. Bazzaz, A. A.; Muhsin, S. S. and Sulaiman, N. M. (1989). Haematological changes in blood of mice induced by subchronic ingestion of lead monoxide. Biol. Sci. Res., 20 (1): 31-40.

6. Belacy, N.; Shata, H.; Edress, G. and El-Arbagy, R. R. (1996). The contribution of heavy metals in kidney dysfunction. J. Union. Arab. Biol. S., (A) Zoology, (147-155).
7. Bersenyi, A.; Fekete, S.; Szoes, Z. and Be'rta, E. (2003). Effect of ingested heavy metals (Ed. Ph. And Hg) on haematology and serum biochemistry in rabbits acta vet. Hung, 51 (3): 297-304.
8. Bersenyi, A.; Fekete, S.; Szoes, Z. and Be'rta, E. (2003). Effect of ingested heavy metals (Ed. Ph. And Hg) on haematology and serum biochemistry in rabbits acta vet. Hung, 51 (3): 297-304.
9. Cramer, R.; Goyer, R. A.; Jayenbury, R.; and Wilsen, M. H. (1975). Renal ultrastructure function and parameters of lead toxicity in workers with different period of lead exposure. Brit. J. Ind. Med., 31: 113-127.
10. Dos-Santos, A. C.; Colacciopo, S.; Dal-Bo, C. M. and Dos-Santos, N. A. (1994). Occupational exposure to lead kidney function tests and blood pressure. Am. J. Med., 26 (5): 635-643.
11. Dowd, T. L.; Rosen, J. F.; Mints, L. and Gundberg, G. M. (2001). The effect of Pb^{2+} on the structure and hydroxyapatite binding properties of osteocakin. Biochem. Biophys. Acta. Molec. Basis of disease, 1535 (2): 153-163.
12. Fossati, P.; Prencipe, L. and Berti, G. (1980). Clin. Chem., 227-262.
13. Gautam, A. K. and Chowdhury, A. R. (1987). Effect of lead on erythropoietic system of intact and splenectomized rats. Indian J. Physiol. Pharmacol., 31 (2): 117-124.
14. Ghorbe, F.; Boujelbene, M.; Makni-Ayadi, F.; Guermazi, F.; Kammoun, A.; Murat, J.; Groute, F.; Soleihavoup, J. P. and Elfeki, A. (2001). Effect of chronic lead exposure on kidney function in male and female rats, determination of lead exposure biomarker. Archphysiol. Biochem., 109(5): 457-463.
15. Goyer, R. A. (1996). Toxic effects of metals in casarett and Doull's. Toxicology: The basic science of poisons 5^{th} ed. C.D. Kleassen, Ed MC Graw-Hill New York.
16. Gupta, G. S.; Singh, H. and Parkash, P. (1995). Renal toxicity after oral administration of lead acetate during pre- and post implantation periods: effects on trace metal composition metallo-enzymes and glutathione. Pharmacol. Toxicol., 76 (3): 206-211.
17. Harrison, J. H., Drum, E. D., Bethune, J. L., and Vallee ,B. L. (1967).Structural and functional of Zink in horse liver alcohol dehydrogenase .Proc. Natt. Acad Sa. ;57(5) 1434-1440.
18. Hawki, P. B. and Oser, B. C. (1965). Hawks physiological chemistry 4th edition, the Blakiston Co. Inc. New York, p. 174.

19. Henderson, D. A. (1955). Chronic nephritis in Queensland Aust. Ann. Med., 4: 163-177.
20. Hernberg, S. Nurminen, M. and Hasan, J. (1967). Non random shortening of red cells survival times in men exposed to lead. Environ. Res., 1: 247.
21. Kim, R.; Rotnitzky, A.; Sparrow, D.; Weiss, S. T.; Wager, C. and Hu, H. (2003). A longitudinal study of low-level lead exposure and impairment of renal function: JAMA, 275 (15): 1177-1181.
22. Kim, Y.; Yoo, C. L.; Lee, C. R.; Lee, J. H.; Lee, H.; Kim, S. R.; Change, S. H.; Lee, W. J.; Hwang, G. H. and Lee, Y. H. (2002). Evaluation of activity of erythrocyte-5-nucleotidase (P5N). In lead exposure workers, with focus on the effect on hemoglobin. Ind. Health, 40 (1): 23-27.
23. Lavicoli, I.; Cavelli, G.; Stanek, E. J.; Castellino, N. and Calabrese, E. J. (2003). Effect of low doses of dietary lead on red blood cell production in male and female mice. Toxicol. Lett., 3: 137(3): 193-199.
24. Mackay, E. M. and Mackay, L. L. (1927). The concentration of urea in blood of normal individuals. Clin. Invest., 4: 295.
25. McBride,M.B.;Rigden,S;Haycock,G.;Dalton,N.;and Simmonds,H.A. (1998).Presymptomatic detection of familial juvenile hyper uric gemic nephropathy in children radiator.Nephro.,12;357-364.
26. Ong, C. N.; Lee, W. R. (1980). Distribution of lead 203 in human peripheral blood Br. J. Ind. Med.,37: 70-77.
27. Payton, M. G. Hu,; Sparrow, D.; Weiss, S. T. (1994). Low-level lead exposure and renal function in the normative aging study. Am. JEP., 140(19): 821-829.
28. Schraishuhn, J.; Kaufer Weiss, I. and Weiss, E. (1992). Light and electron subtoxic lead level. Berl Munch. Tieraztl Wochenschr, 105(9): 290-293.
29. Sivaprasad, R.; Nagaraj, M. and Voralakshmi, P. (2003). Combined efficacies of lipoic acid and meso-2,3-dimercaptosuccinic acid on lead induced erythrocyte membrane lipid peroxidation and antioxidant status in rats. Hum. Exp. Toxicol., 22(4): 183-192.
30. Solliway, B. M.; Schaffer, A.; Prott, H.; and Yannai, S. (1996). Effects of exposure to lead on selected biochemical and haematological variables pharmacol. Toxicol., 78(1): 18-22.
31. Speich, M.; Metayer, G.; Around P. and Nguyen, V.; Bousguet, B. (1983). Low lead doses and atherogenic. Diet in rabbits Biochemical results in blood. Ann. Nutre. Metab., 27(6): 521-530.

32. Terayama, K. (1993). Effects of lead on electrophoretic mobility, membrane sialic acid, deformability and survival of rat erythrocytes. Ind. Health, 31(3): 113-126.
33. Terayama, K.; Machara, N.; Muratsugu, M.; Makine, M. and Yamamura, K. (1986). Effect of lead on electrophoretic mobility of rat erythrocytes. Toxicology, 40(3): 259-265.
34. Tola, S. and Nordmen, C. H. (1977). Serum creatinine concentration, AST and ALT and lead exposure Int. Arch. Occup. Environ. Health, 39(1): 37.
35. Waldron, H. A. (1975). Lead levels in blood of residents near the M6-A38 (M) interchange. Birmingham Nature (lond) 253: 345-346.
36. Whitby, L. G.; Rercy-Robb, I. W. and Smith, A. F. (1980). Lecture Notes on Clinical Chemistry Blackwell Scientific Publications Oxford London Edinburgh Melbourne 2nd ed Chapter 9: 167-187.
37. Wilkinson, J. O. H. (1977). By J. Hospt. Med., 18: 343.
38. Yun, S. W. and Hoyer, S. (2000). Effects of low level lead on glycolytic enzymes and pyruvate dehydrogenase of rat brain in vitro: relevance to sporadic. Al Zheimer's disease. J. Neural. Transm., 107(3): 355-368.
39. Zayed, S. M. A.; Amer, H. A.; Fahmy, M. A.; Nahdy, F. (1993). Toxicological potential of malathion residues in stored soybean seeds. Journal of Environmental Science and Health Part B: pesticides, food contaminants and Agriculture Wastes, 28: 711-792.