Blood Lead Level among Fuel Stations Workers and its correlation with Liver, Kidney Dysfunction in Damascus City (Syria)

Abstract

Chronic exposure to lead is known to cause adverse health effects. Workers at gas stations are exposed to high concentrations of lead during filling cars and through cars' emissions and being in contact with contaminated hands, food, water and clothing. This study was designed to find blood lead level and their adverse effects on kidney and liver function among gas station workers. Forty fuel station workers (exposed group) and thirty apparently healthy subjects (non-exposed group) in Damascus were randomly selected for the study. Blood lead levels were determined using Atomic absorption spectrometry after microwave-assisted acid digestion. Serum concentration of creatinine, uric acid and urea values were recorded to assess kidney function, whereas AST and ALT serum concentrations were used to evaluate liver function. The results showed a non-significant elevation of blood lead level in the exposed group (11.04 ± 10.36 µg/dl) compared to the non- exposed group (8.1± 2.97 µg/dl). Serum concentration of creatinine and uric acid were significantly elevated in the exposed group, but there was no change in AST and ALT serum levels . It is concluded that blood lead levels of fuel station workers don't exceed the threshold that may cause kidney or liver dysfunctions.

Key words: Occupational lead exposure, Lead nephrotoxicity, Lead hepatotoxicity

Introduction

Human exposure to heavy metals such as lead could cause serious adverse health effectsespecially among occupationally-exposed workers. Workers at fuel stations are heavily exposed to lead[1], as gasoline contains an organic form of lead (tetraethyl lead) which is used as an antiknock agent[2].Lead poisoning is associated with blood lead levels above the accepted blood level of less than 10 µg/dl as determined by the world health organization (WHO)[3]. Heavy exposure to lead has been associated with kidney and liver toxicity[4,5]. The kidney is often targeted by heavy metals due as it reabsorbs and concentrates divalent ions and metals during excretion. The severity of kidney impairment caused by lead depends on the type, dose, and duration of exposure[6].Acute lead exposure causes impairment of the proximal tubular architecture and induces histological changes such as eosinophilic intra-nuclear inclusions of lead-protein complexes in tubular cells and mitochondrial swelling. On the other hand, chronic exposure to lead may damage kidneys as manifested by increased urate secretion, vasoconstriction of renal blood vessels and consequent glomerulosclerosis, hypertension and interstitial fibrosis[7,8].

Lead exposure may also causes liver toxicity associated with changes in liver structure[9,10].Lead in the body is conjugated in the liver with glutathione so that part of it and its conjugate are accumulated in hepatic tissues [11,12,13]leading to depletion of glutathione and increased oxidative stress, which aggravates liver impairment[14,15,16,17].

To the best of the authors' knowledge, no past studies have evaluated the blood lead level, and its correlation with liver and kidney function among fuel station workers as a result of their workplace in the city of Damascus (Syria).So the objective of this study was to evaluate blood

lead levels and its correlation with kidney and liver dysfunctions among fuel station workers in Damascus.

Materials and Method

This Comparative cross-sectional study was carried out on seventy subjects with the ages ranging from 19 to 61 years old in Damascus city during the period March-April 2019. A total of40 human volunteers, who work in fuel stations in Damascus (Exposed group) were selected for the study. Another group of 30 apparently healthy subjects who were not engaged in activities that predisposed them to direct contact with gasoline were recruited as controls (non-exposed group). Exclusion criteria included subjects with hepatitis B or C, cirrhosis, diabetes mellitus and malignancy. The volunteers participating in this study after filling a consent form. All participants were given clear explanation regarding the methodology of the research. The present study was approved by the Institutional Ethical Committee, Faculty of Pharmacy (Al-Kalamoon University). A questionnaire for all volunteers was filled including the subject's personal and clinical information such as age, gender, occupation, no. of years working in a fuel station and daily hours spent on duty.

Samples collection and preparation

Five ml blood sample was taken from each volunteer through venipuncture using sterile needle attached to syringe (5ml).The sample was divided into two halves: one for assessing kidney function (creatinine, uric acid and urea) and liver function (AST, ALT) analysis and the other half was treated with EDTA anticoagulant materials for determination of blood lead levels.

Detection of blood lead concentration

Lead concentration in blood was determined by atomic absorption spectrometry (AAS) (ZEE nit 700P,Germany).Blood samples weredigested by the microwave induced acid digestionmethod by adopting the method of Kazi et al.and Yahaya et al.[18,19]. Three milliliters offreshly prepared mixture of concentrated nitric acidand hydrogen peroxide (2:1V/V)were added to o.5 ml of whole blood sample and stood for 10 minutes.Then heated in microwave oven (Berghof 'speedwave® four' microwave) withmaximum heating power of 800 W for 3 minutes.After this the digestion flasks were cooled. Theresulting solutions were evaporated to semidriedmass to remove excess acid, and diluted with 0.1 M nitric acid .A blank extraction (without sample) was carried out through the completeprocedure. Quantification of lead levels was then performed using an atomic absorption spectrophotometer.

Biochemical assays

Serum levels of AST, ALT, creatinine, uric acid and urea were determined using a spectrophotometer (Photometer 5010, Germany) and a commercially available kit according to the manufacturer's instructions(EltechGroup,). Hematocrit and hemoglobin were determined by an automated complete blood count (CBC) analyzer (Nihon Kohden, Japan).

Data Analysis.

Statistical Package for Social Sciences (SPSS)version 23 was used to analyze the data. Numbers and percentages were used to describe demographic characters (Qualitative data). Kolmogorov-Smirnov test was used to test the normal distribution of data. Quantitative data

weredescribed as means ± standard deviation (SD) or medians (range), depending on normality of data. Independent sample t-test and ANOVA were used to assess normally distributed data while Mann Whitney test and Kruskal Wallis Test were used for non-normally distributed data. A p value of ≤ 0.05 was considered statistically significant.

Results

Table 1 showed the demographic characteristics of exposed and non-exposed groups. All subjects in this study were male and their ages ranged between 19 to 61 years. In the exposed group, 60% were aged19-45 years, 27.5% were aged 46-60, and 12.5% were over 61 years old. In the non-exposed group, 96.7% were aged 19-45 years, while 3.3% were over61 years old. 67% of exposed group were smokers, whereas the percentage in the non-exposed group was 53.3 %. In the exposed group, the duration spent on duty 50% of them have been working in fuel stations for1-10 years,. Regarding hours spent daily on duty, 47.5 % indicated a 4-6 hrs daily work shift.

The findings show that there was no significant differences in Hb, HCT % and BMI between the exposed group and the non-exposed group (table 2).

Interestingly, when comparing blood lead levels in the exposed and control groups table (3), there was no significant elevation in the exposed group, compared to the non-exposed group (P = 0.29).

As showed in table 4, liver function determined by AST and ALT serum concentrations were normal in the exposed group compared to the non-exposed group (P = 0.83, 0.75 for AST and ALT respectively). However, the results showed a significant elevation in serum creatinine and uric acid in the exposed group compared to non-exposed group ($p < 0.05$).

Mann-Whitney Testwas performed to assess effect of smoking on blood lead level on attendant fuel station workers (exposed group). The results showed a non-significant difference (P = 0.42) between blood lead level in the smoker compared to the non-smoker subjects (table 5).

Pearson's correlation coefficient was performed to assess the impact of age, years and hours spent in job on blood lead concentration of exposed groups. As shown in table 6, the analysis reported a significant (moderate) positive correlation between blood lead concentration and age ($r = 0.312$, $p = 0.05$), and a significant positive moderate correlation between blood lead concentration and hours spent on job ($r = 0.483$, $p = 0.002$).

Table 7 show blood lead level, serum concentration of AST and ALT according to age, years and daily hours spent on job. The results show that blood lead level was higher in subjects aged over 61 year but this elevation was not significant compared to other age intervals.The blood lead concentration also increased with the increase of years and hours of works spent in fuel stations,but this elevation was not statistically significant. In addition, working years and hours on duty did not significantly affect serum lead levels .

Regarding kidney function, , there were no significant differences in serum creatinine, uric acid and urea in the different age groups nor in the groups with different working years and working hours (table 8).

Discussion

Leaded gasoline has been identified as a significant predictor for increased blood leadlevels in fuel station workers. No previous studies have been determinedblood lead concentrations of fuel station workers in Syria and their toxicity on kidney and liver. The findings of the present study unexpectedly showed that the mean blood lead levels of fuel station workers was not

significantly different from non-exposed group. The mean lead concentration in the exposed group was 11.04 µg/dl (the highest concentration found was 66.50 µg/dl and the lowest was 8.10 µg/dl). This finding is in agreement with a previous study that took place in Gaza Strip[20],which found that11.4 µg/dl was the mean blood lead concentration in fuel station personnel. However, other studies performed in other countries have reported higher blood level concentrations. The means were 45.43 µg/dl in Abuja, Nigeria [21],15.11 µg/dl in Nnewi, Nigeria [22],14.1µg/dl in Basrah, Iraq[23], 33.6 µg/dl in Khartoum, Sudan[24] and 30.05 µg/dl in Iran[25]. On the other hand, there are other studies that reported lead concentrations that are lowers than found in the present work. For instance, the mean lead concentration was reported 3.5 µg/dl in Denmark[26],8.6 µg/dl in Ghana[27], and 5.6 µg/dl in Greece[28].Low blood lead level in these three countries may be due to implementing effective contamination prevention and intervention programs regarding lead chronic intoxication as well as to the presence of effective surveillance of the health status of all fuel station workers.

According to the WHO, the acceptable blood level of lead in adult humans is less than 10 µg/dl, [3]. Based on this criteria, 77.5% of the fuel station workers had acceptable blood lead levels (see table-9), and 22.5% had higher than acceptable level .

The data also show considerable variance in blood lead levels among fuel station workers which may be ascribed to variance in years and daily hours spent on duty. Table -10 indicates an in increase in blood lead level in those with longer years and hours spent working. The data showed a significant positive correlation between blood lead level and hours spent on job ($r = 0.483$, $p = 0.002$) (see table 6). In accordance with our findings, Nurjazuli*et al.,*[29],have demonstrated in 2003 that work duration was a dominant factor for high blood lead levels, and Stoleski*et al.,*[30] revealed a positive correlation between blood lead level and work duration and occupational exposure..

Occupational lead exposure has also long been linked to the development of renal dysfunction [31].In the present study, there was a significant elevation in serum concentrations of creatinine and uric acid in the exposed group comparing to the non-exposed group, but their serum concentration were still within the normal physiological range. The results are in agreement with a previous work which showed that lead-exposed workers with low blood lead level of less than 7 µg/dl did not suffer from lead-related kidney dysfunction[32].Also, there was no change in serum creatinine and uric acid in men with high blood lead levels of 36 µg/dl.The renal dysfunction seems to develop only when blood lead levels exceeds a threshold of 60 µg/dl[8].

The liver function of the exposed group was assessed using serum AST and ALT activities .The results showed no significant differences in serum concentration of serum AST and ALT between the exposed group and non-exposed group. In the same manner, Allouche and his colleagues[33] reported no disturbance in biochemical liver parameters in people with previous long term-exposure to low or moderate lead concentrations. Another study on two occupationally lead-exposed groups with moderately elevated blood lead levels, showed a non-significant differences in serum levels of AST and ALT compared to the non-exposed group[34], and that only those with exceedingly high lead levels had hepatic dysfunction [35].Therefore, the normal serum concentration of AST and ALT of the exposed group in the present study may be due to blood lead level that is below the threshold that causes liver dysfunction .

Conclusion

The present study confirms that fuel station workers in Damascus city (Syria) had a mean blood lead level that is not statistically different from the rest of the society. The normality of blood

lead level may be due to short occupational exposure to lead in the working place. In addition, these workers did not suffer from work-related kidney or liver impairment, possibly due to the blood lead level being below the threshold value that causes renal/hepatic disturbance. The findings support the notion that policies controlling lead exposure in the working place dictates the health outcomes in lead-exposed personnel.

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Conflict of Interests

No conflict of interest associated with this work

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Table 1: Demographic characteristics of study subjects.

	Exposed		Non-exposed	
Variables	N	%	N	%
Gender				
Male	40	100	30	100
Female	0	0	0	0
Total	40	100	30	100
Age				
19-45	24	60	29	96.7
46-60	11	27.5	0	0
> 61	5	12.5	1	3.3
Total	40	100	30	100
Occupation				
Fuel station	40	100	0	0
Total	40	100	0	0
Cigarette smoking				
Yes	27	67.5	16	53.3
No	13	32.5	14	46.7
Total	40	100	30	100
Years spent in job				
< 1 year	6	15	0	0
1-10 years	20	50	0	0
11-20 years	4	10	0	0
21-30 years	6	15	0	0
> 30 years	4	10	0	0
Total	40	100	0	0
Hours spent on duty				
1-3 hr	5	12.5	0	0
4-6 hr	19	47.5	0	0
7-10 hr	9	22.5	0	0
> 10 hr	7	17.5	0	0
Total	40	100	0	0

Table 2: Comparison of haematological parameters and BMI in the exposed and non-exposed groups

Variables	Non-Exposed (n=30)	Exposed (n=40)	t value	P value
HB	14.79 ± 1.06	14.6 ± 0.87	0.809	0.421
HCT%	46 ± 3.3	45.42 ± 2.70	0.800	0.426
BMI	25.2 ± 4.75	25.21 ± 5.13		0.950

Data are expressed as Means ± Standard Deviations

Table 3:Blood lead concentration (µg/dl) in the exposed and non-exposed groups

	Non-Exposed(n=30)	Exposed (n=40)	P value
Lead (µg/dl)	8.15 (3.10 - 15.80)	8.50 (5.40 - 66.50)	0.29
	8.1 ± 2.97	11.04 ± 10.36	

Data are expressed as median (range), mean ± SD

Table 4: Comparison of AST, ALT, creatinine, uric acid and urea concentrations (mg/dl) in the exposed and non-exposed groups

Variables	Non-Exposed (n=30)	Exposed (n=40)	P value
AST (mg/dl)	19.4 (6.7-40.5)	25.1 (6.7-56.2)	0.83
ALT (mg/dl)	18.55 (6.1-65.2)	25.7 (5.4-79.16)	0.75
Creatinine (mg/dl)	0.65 (0.51-1.02)	0.94 (0.8-1.16)*	0.000
Uric acid (mg/dl)	5.5 (3.5-8.1)	6.67 (3.9-9.75)*	0.000
Urea (mg/dl)	25.6 (18-42)	27.05 (16-51)	0.054

Data are expressed as median (range).*= P ≤ 0.05 compared to non-exposed group

Table 5: Blood lead level in smokers and non-smokers of the exposed group

	Smoker	Non-smoker	P value
Pb (µg/dl)	8.6 (5.4-26.4)	8.4 (6.1-66.5)	0.42

Data are expressed as median (range)

Table 6: **Pearson's correlation coefficients (r) between Pb blood level and age ,Years spent on job, hours spent on job in the exposed group**

Variables	r	P value
Age	0.312	0.05*
Years spent on job	0.271	0.9
Daily hours spent on job	0.483	0.002**

*** = Correlation is significant at the 0.05 level**

**** = Correlation is significant at the 0.01 level**

Table 7: blood concentration of lead, serum concentration of AST and ALT in the exposed group according to age working years and working hours.

		Pb (µg/dl)	P value	AST (mg/dl)	P value	ALT (mg/dl)	P value
Age	19-45	8.25 (5.7-14.2)	0.174	16.65 (6.1-54)	0.298	25.8 (10.1-79.16)	0.531
	46-60	8.2 (5.4-26.4)		25.7 (10.1-65.2)		22.5 (5.4-70.8)	
	> 61	15 (7.3-66.5)		20.2 (12.3-52.8)		20.2 (12.3-31.5)	
Years spent on job	< 1 year	8.65 (6.5-14.2)	0.388	15.35 (6.1-18)	0.149	24.15 (11.14 -37.1)	0.946
	1-10 year	8.75 (5.7-32.7)		20.2 (10.1-54)		26.4 (5.4-79.16)	
	11-20 year	6.95 (5.4-8.2)		30.8 (14.6-65.2)		20.15 (16.8-49.5)	
	21-30 year	9.2 (6.2-26.4)		18.5 (16.8-42.67)		29.25 (10.1-36)	
	> 30 year	8.25 (7.3-66.5)		22.95 (12.3-25.8)		21.35 (12.3-70.80	
Daily hours spent on duty	1-3 hr	8.7 (6.2-14.2)	0.367	16.8 (15-35.1)	0.113	36 (11.2-79.16)	0.476
	4-6 hr	8.1 (5.7-11.2)		16 (6.1-43.8)		22.5 (5.4-52.8)	
	7-10 hr	8.2 (6.8-26.4)		16.8 (10.1-54)		27 (10.1-70.8)	
	> 10 hr	10.1 (5.4-66.5)		32.4 (12.3-65.2)		22.5 (15.1-49.5)	

Kruskal Wallis Test, P < 0.05, Data represented as median (range)

Table 8: serum concentration of creatinine, uric acid and urea in the exposed group according to age working years and working hours.

		Creatinine (mg/dl)	P value	Uric acid (mg/dl)	P value	Urea (mg/dl)	P value
Age	19-45	0.94 (0.8-.16)	0.95	6.77 ± 1.07	0.071	28.6 ± 8.38	0.186
	46-60	1.015		6.50 ± 1.13		27.64 ± 5.07	

		(0.87-1.09)					
	> 61	0.94 (0.87-1.09)		7.91 ± 1.24		34.9 ±7.11	
Years spent on job	< 1 year	0.94 (0.87-1.09)	0.377	6.70 ± 0.60	0.188	27.35 ± 4.49	0.39
	1-10 year	1.01 (0.80-1.16)		6.92 ± 1.33		27.65 ± 8.64	
	11-20 year	0.94 (0.87-1.02)		5.81 ± 0.45		29.22 ± 8.18	
	21-30 year	0.94 (0.87-1.02)		6.75 ± 1.26		31.75 ± 6.59	
	> 30 year	0.97 (0.94-1.09)		7.81 ± 0.24		35.1 ± 5.41	
Daily hours spent on job	1-3 hr	1.01 (0.87-1.02)	0.649	7.69 ± 0.93	0.248	29.62 ± 8.24	0.996
	4-6 hr	0.94 (0.8-1.16)		6.68± 0.94		29.17 ± 8.29	
	7-10 hr	0.94 (0.87-1.09)		6.5 ± 1.10		29.2 ± 4.97	
	> 10 hr	1.01 (0.94-1.09)		7.11 ± 1.71		28.54 ± 9.67	

Kruskal Wallis Test, P < 0.05, Data represented as median (range) ,ANOVA test, P < 0.05, Data represented as mean ± SD

Table 9: Numbers and % of subjects in the exposed groups whose blood lead concentration were < or ≥ 10 µg/l

	Number	%	Pb (µg/dl) Mean	Pb (µg/dl) Median (range)	P- value
< 10 µg/l	31	77.5	7.79	7.9 (5.4-9.9)	6×10^{-6}
≥ 10 µg/l	9	22.5	22.24	14.2(10.1-66.5)*	

Data are expressed as median (range). *

P-value is significant at the level of < 0.05.

Table 10: Blood lead level according to years an daily hours spent in work in the exposed group

		Pb µg/l (mean)
Duration (years)	< 1 year	8.9

	1-10 years	9.83
	11-20 years	6.87
	21-30 years	12.3
	> 30 years	22.57
Time (hrs)	1-3 hrs	9.04
	4-6 hrs	8.05
	7-10 hrs	11.46
	> 10 hrs	20.04