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Effect of Lead Exposure on Some Biochemical Parameter of Battery Factory and Benzene Fuel Stations Workers

Faisal.H.G.Q.AL-Amier*, Hind S.Abdulhay*, Salah M.Muhsin**
Department of Biology, College of Science, Baghdad University,Baghdad,Iraq.
****Biotechnology Research Center, AL-Nahrain Unversity,Baghadad, Iraq.***
faisalblaizer@yahoo.com
hind442@yahoo.com
alchalabisalah@gmail.com

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Abstract:

Lead pollution is an important environmental problem, despite consciousness of its harmful health effects in the world. This study was an attempt to find the effects on some possible biochemical parameters. The study has been conducted in Baghdad City from March 2017 to April 2018 divided in to two groups exposure 75 workers and non- exposure 10 control. Biochemical tests of liver and kidney Functions showed clear lead effect on them. The average [ALK, GPT, and GOT] in battery factories workers were 75.52 ± 5.47, IU/L 33.54 ± 4.58, 42.64 ± 6.59 IU/L and fuel station workers 62.41 ± 3.70,27.15 ± 2.16,29.42 ± 1.48 IU/L compared with the control group respectively 45,10 ± 6,39, 17,95 ± 2,11 and 28,71 ± 2.65 IU /L the study showed significant increase in the mean liver enzyme of exposed workers, compare with control. The mean of serum urea and creatine level in battery factory workers, fuel station and control. Were 41.16 ± 2.21 mg/dl, 38.04 ± 1, 69 mg/dl and 20, 40 ± 0.54 mg/dl and 0.986 ± 0.06 mg/d, 0.941 ± 0.05 mg/dl, and 0.710 ± 0.06 mg/dl respectively there was significant increase in serum urea and creatinin of the workers compared with control.

Key words: lead, urea, creatine, alkaline phosphates [ALK], Glutamic pyruvic Transaminase [GPT], and Glutamic Oxaloacetic Transaminase [GOT]

تأثيرالتعرض للرصاص على بعض المعايير البايوكميائية من العاملين في مصنع البطاريات ومحطات وقود البنزين

فيصل هادي غني قويزي الامير*
هند سهيل عبد الحي*
صلاح مهدي محسن**
***قسم علوم الحياة،جامعة بغداد ،بغداد،العراق**
****مركز بحوث التقنيات الاحيائية ،جامعة النهرين،بغداد،العراق**

الخلاصة:

يعد تلوث بالرصاص من المشاكل البيئية المهمة رغم الوعي بأثاره الصحية الضارة في العالم. جاءت هذه الدراسة كمحاولة لايجاد التأثيرات البايوكميائية المحتملة لعنصر الرصاص. وقد أجريت الدراسة في مدينة بغداد من أذار 2017 إلى نيسان 2018قسمت العينات الى مجموعتين مجموعة متعرضة عددهم 75 عامل ومجموعة غير معرضة وعددهم 10 السيطره . اظهرت نتائج فحوصات تأثيرالرصاص واضح على وضائف الكبد ، ان متوسط [GOT, GPT, ALK] في عمال مصانع البطاريات عالى التوالي كانت [75,52±5,47]وحدة دولية \التر ،[33,54±4,58] ،[42,44±6,59]وحدة دولية \ لتر وفي عمال محطات الوقود عالى التوالي [62,41 ±3,70] ،[27,15±2,16] ،[29,42±1,48] وحدة دولية \التر وكانت متوسطات نتائج وضائف الكبد في مجموعة السيطرة على التوالي [45,10±6,39] ،[17,95±2,11] ،[28,71±2,65] وحدة دولية \التر وكما اظهرت الدراسة وجود فروق معنويه بين متوسط انزيمات الكبد[للعمال المعرضين مهنين ، غير معرضين] ، وكل مجموعة بين المعرضين. أظهرت النتائج أن متوسط مستويات الرصاص في عمال مصانع صناعة البطاريات كان 28.50 ± 0,52ميكروغرام / ديسيلتر وأقل في عمال محطات الوقود 21.79 ± 0,66 ميكروغرام / ديسيلتر. وكان متوسط مستويات الرصاص في الدم في مجموعة الغير معرضين 8.70 ± 1,09 ميكروغرام / ديسيلتر ،كما اظهرت النتائج وجود فرق معنوي بين متوسط مستويات

الرصاص في الدم وعينة الدراسة المعرضين والسيطر ، وكل مجموعة من المعرضين [عمال مصانع البطاريات ،وعمال محطات الوقود]، أظهرت النتائج فحوصات تأثير واضح على وضائف الكلى في مجموعة عمال مصانع البطاريات متوسط مستوى اليوريا والكرياتين في عمال مصنع البطاريات ومحطة الوقود والتحكم. كان 2,21±41,16 مجم / ديسيلتر ، 1 ± 38.04 ، 69 مغ / ديسيلتر و 20 ، 40 ± 0.54 مغ / ديسيلتر و 0.06 ± 0.986 مغ / دل ، 0.941 ± 0.05 ميلي غرام / ديسيلتر ، و 0.06 ± 0.710 ميلليغرام / ديسيلتر على التوالي كان هناك زيادة كبيرة في اليوريا في الدم والكرياتينين للعمال مقارنة مع السيطرة

مفاتيح الكلمات : الرصاص ،يوريا ،كرياتين ، الغلوتاميك اكسولواستك ترانساميناز،الغلوتاميك بايرفيك ترانساميناز

Introduction:

Lead [pb] toxicity is one of the oldest occupational hazards in the worldwide and it is the one of the most important issues of global health and environmental [1]. Pb reasons a proximal tubular injury with a satisfactory of proximal tubule nuclear inclusion bodies that progress to tubule interstitial illness and fibrosis. Pb piling up in the proximal tubule leads to hyperuricaemia and gout prospect by restraining uric acid secretion and also to dimpled renal clearance, tubular recapture and glomerular filtration rate [2]. Pb concentrations in the blood more than 40 µg/dl are associated with an increased gravity of nephropathy and linked renal failure, lower levels of exposed to pb can act as a cofactor that increases the gravity of renal functional deterioration and the average of functional decline [3]. Pb can cause liver distraction in which free radical reactions are include, pb toxicity generally results from well-known occupational exposed in some condations it may arise from unexpected sources. Pb may cause colic abdominal pain, weight loss and rising in liver function parameters [4]. Over-exposed to pb may also inhibit many enzymatic activities and the liver cannot deal with pb metabolism and elimination this [5]. The absorbed pb is associated with liver, it has been considered the largest main store pb repository and the target organ for its poisoning effects [6–7]. Experimental studies have shown that chronic pb exposed can cause an arising in (AST), (GPT), and (ALK) [8]. In addition, pb stimulates intercellular signaling between hepatocytes and Kupffer cells, which contributes to pb-induced hepatotoxicity [9].

Aims of the study:

Conducting biochemical tests for workers (fuel stations and battery factories) and comparing the results with the control group.

Material and Methods:

The sample included 10 control adult men (nonexposed persons) according to the following criteria: 1-They were free from any disease and not taking any medication.2-Age were ranges between (20- 60) years.

The samples consisted of (75) adult men who were chosen according to the following criteria: 1-All individuals studied [battery factories and gasoline station workers] considered as exposed group to lead poisoning via air polluted by cars exhaust.2-Agewere ranges between (20- 60) years. Serum urea and creatinine were calculated according to the following formula: Serum urea nitrogen Cmmol / l) = (sample absorption) Standard absorbance (x 20) Serum creatinine was determined using the human response and human group (Büssens and Taussky, 1945). The method is based on the reaction of creatinine with bicarbonate under alkaline conditions to form an orange-yellow compound. When acid is added, the color contributed by creatinine is destroyed, while the material produced by non-specific substances remains. The difference in color intensity measured at 500 nm before and after acidification is proportional to creatinine concentration [10]

Liver Enzymes:

Enzymes were measured by using kits provided by Biomerieux - France for the colorimetric determination of Glutamic

Pyruvic Transaminase (GPT) and Glutamic Oxalat Transaminase (GOT) in serum according to the following reaction: NaOH its concentration 0.4 N. For GOT determination, reagents 3 and 4and sodium hydroxide were used, while reagent 2 was replaced with reagent 1 which consists of phosphate buffer pH 7.5, aspartate and ά-ketoglutarate. Samples absorbance (A) was determined by using spectrophotometer on 505 nm. The number of GPT and GOT were measured as units/ml which calculated from applying the absorbance (A) of samples on the standard curves [11].

Alkaline phosphates determination:

Enzymes were measured by using kits provided by Jourilabs -Switzerland for the colorimetric determination of alkaline phosphates readable at a 510 nm absorbance in the spectrophotometer.

Result and Discussion:

Table1. Shows the effect of lead on urea and creatinine parameters, regarding urea, high significant difference **(P<0.01) was found between fuel stations workers 38.04 ± 1.69 mg/dl, batteries factory workers 41.16 ± 2.21 mg/dl compare to control 20.40 ± 0.54 mg/dl, concerning Creatinine, significant difference *(P<0.01) was found between Fuel stations workers 0.941 ± 0.05 mg/dl, batteries factory workers 0.986 ± 0.06 mg/dl compare control 710 ± 0.06 mg/dl. Normal value of urea 18-45mg/dl, normal value of creatinine 0.5-1.1mg/dl. Blood urea and serum creatinine are parameters that can be used to detect the renal effects caused by occupational exposure to Pb. But, when these tests are found abnormal, the nephropathy has already reached the irreversible phase that may lead to renal insufficiency [12].The results showed high significant increase in serum urea in workers compare to the control (p> 0.01) Through these results the probability of injury of workers to renal failure at the long term is greater than the control group, exposure to high level of lead >60 µg/dL may cause renal dysfunction, even a low level of lead ~10 µg/dL may also provide the same problem [13]. The pathological influence of the exposure of lead on the renal systems of workers seems to result to the renal toxicity development under the influence of the oxidative stress that it causes.That such effect only damages kidney in chronic exposure that becomes clinically significant, and that kidney damage does not usually occur in asymptomatic acute cases[14] While in chronic accumulation of lead in the body eventually leads to impairment in renal function, urea and creatinine are a waste product of amino acid metabolism they removed by kidney, oxidative stress appears to be involved in the development of renal toxicity induced by the environmental lead exposure that causes significant pathological lesions on the renal systems of men and animals[15].People with occupational lead exposure are at risk of developing hyperuricmia and renal impairment [16].

Table (1) Show the effect of lead on Urea and Creatinine test in Batteries factory workers Fuel stations workers and control.

Groups	No.	Mean ± SE	
		Urea (mg/dl)	Creatinine (mg/dl)
Fuel stations	25	38.04 ± 1.69 a	0.941 ± 0.05 a
Battery Factories.	50	41.16 ± 2.21 a	0.986 ± 0.06 a
Control	10	20.40 ± 0.54 b	0.710 ± 0.06 b
LSD value	---	7.401 **	0.220 *
* (P<0.05), ** (P<0.01). Means having with the different letters in same column differed significantly.			

As shown in the table 2. ALK-P, GOT and GPT, were highly significant increase fuel station and battery factory in workers compare with control p <0.01was found between fuel stations workers regarding ALK phosphate (62.41 ± 3.70 U/L) , battery factories workers (75.52 ± 5.47 U/L) and control 45.10 ± 6.39 U/L , high significant difference P<0.01 was found between battery factories regarding GOT(42.44 ± 6.59 U/L) and control (28.71 ± 2.65 U/L) and regarding GPT high significant difference **(P<0.01) was found between battery factories workers (33.54 ± 4.58 U/L) and control (17.95 ± 2.11 U/L). Lead can stimulate intercellular signaling between Kupffer cells and hepatocytes, which are enhanced synergistically in the presence of low lipopolysaccharide levels [17]. Higher concentration of lead cause severe periportal inflammation in chicken liver, therefore, it may be assumed that long term lead exposure cause liver damage in human, but low concentration of lead disturbs the normal biochemical process in the hepatobiliary system [18].

Table (2) Show the effect of lead on Liver functions (ALK, GOT, GPT) in Batteries factory workers, Fuel stations workers and control.

The Groups	No.	Mean ± SE		
		[ALK [U/L	[GOT [U/L	[GPT [U/L
Fuel stations	25	62.41 ± 3.70 a	29.42 ± 1.48 b	27.15 ± 2.16 ab
Battery Factory.	50	75.52 ± 5.47 a	42.44 ± 6.59 a	33.54 ± 4.58 a
Control	10	45.10 ± 6.39 b	28.71 ± 2.65 b	17.95 ± 2.11b
LSD value	---	18.569 **	10.187 **	14.467 **
** (P<0.01).				

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