

ROLE OF β 2 MICRO-GLOBULIN IN EARLY ASSESSMENT OF RENAL AFFECTION IN LEAD BATTERY WORKERS-MINIA CITY

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ABSTRACT

Environmental pollution by lead is a major concern for public health. The **aim** of this study was to determine renal parameters which are suitable for early assessment of renal affection in lead battery workers. **Subjects & Methodology:** Sixty subjects (all males) were included in this study, ten volunteer subjects as control (group I) and fifty workers categorized as study subjects who worked on lead battery factories in Minia city-Minia Governorate. The workers included in this study were divided into two group according to their blood lead level (BLL), (group II): included twenty-four workers with blood lead level $< 20 \mu\text{g/dl}$ and (group III): included twenty-six workers with blood lead $\geq 20 \mu\text{g/dl}$. Laboratory investigations were done for all subjects of the study including BLL, urinary delta-Aminolevulinic acid (δ -ALA), renal function indices as blood urea, serum creatinine, serum uric acid, and serum β 2 microglobulin. **Results:** Group II parameters were within normal except β 2 microglobulin which was higher than its normal value. Workers in group III were found to have significantly higher levels of, blood urea, serum creatinine, serum uric acid, β 2 microglobulin and urinary δ -ALA than workers in group II and control group. Although there was significant positive correlation between BLL and all studied parameters, β 2 microglobulin had the highest positivity followed by serum uric acid. **Conclusion:** increase in BLL causes positive renal effects. β 2 microglobulin followed by serum uric acid is good early predictor for renal affection in lead exposed workers.

Keywords: Lead battery workers, renal function, β 2 microglobulin

INTRODUCTION

Lead is a natural element and the most accessible heavy metal in the earth's crust. It is widely used in many industrial processes and domestic purposes (ATSDR, 2005). Its effects appear mainly in the developing countries where 99% of the severest cases of overt lead poisoning occur in them (Venkatesh, 2013).

Lead toxicity is a particularly insidious hazard causing irreversible health effects. It is primarily affecting the central nervous, hematopoietic, hepatic systems and producing serious disorders (Kalia & Flora, 2005). Lead

has been reported to cause nephrotoxicity by several mechanisms, including tubular atrophy and interstitial fibrosis of the kidneys (Karimooy et al., 2010).

There are several sources of lead poisoning have been identified. The most common sources are the leaded gasoline, batteries, paints, building materials, ceramics, and improperly glazed containers (Klaasen, 2001). Retained lead pellets or particles, and variety of folk remedies and cosmetics are also considered important sources of lead toxicity (Meyer et al., 2008). Exposures to lead can occur through

ingestion, inhalation, or occasionally skin contact (**Patrick, 2006**).

Beta-2 microglobulin is a low molecular weight protein that found in the membrane of nucleated cells and in most biological fluids such as serum, urine, and synovial fluids. It is filtrated from glomerular membrane (because of its small size), reabsorped, and catabolized in the proximal tubules (**Chaumont et al., 2012**).

AIM OF THE STUDY

The aim of this study was to estimate the role of some renal parameters for early detection of renal toxicity in lead battery workers.

PATIENTS & METHODS

The current study was cross sectional, and was conducted in Minia city - Minia governorate during the period from February 2016 to September 2016. All study participants signed a written informed consent before performing any step. The samples were taken at work field.

Firstly, the study included 100 males aged 23- 40 years old, but 40 of them were excluded according to the following exclusion criteria to decrease bias and to ensure accurate study.

Exclusion crieteria:

Hypertension, diabetes mellitus, cigarette smoking or alcohol intake, renal disease, use of nephrotoxic drugs, medication for gout, history of any renal operations.

After exclusion, 60 subjects were included in the study: 10 healthy volunteer subjects were chosen as control group or group I with BLL Less than 10 micrograms per deciliter ($\mu\text{g/dL}$) according to **CDC 2014**, and 50 lead battery workers who worked in this field from more than one year. This

50 workers (study subjects) were further subdivided into two groups according to their blood lead level (BLL): $< 20 \mu\text{g/dl}$ (group II), and $\geq 20 \mu\text{g/dl}$ (Group III).

Blood and urine samples were taken from the 60 subjects for assessment of:

1- Blood lead level: determined by electrothermal atomic absorption spectrometry (**Wang & Demshar, 1992**)

2- Urinary delta-Aminolevulinic acid: the steps of the test were performed according to **Tomokuni & Ogata, (1972)**.

3- Renal function tests:

a- Blood urea: The steps of the test were performed by using endpoint BioMed-UREA-S kits purchased from BioMed Company (**Patton & Crouch, 1977**).

b- Serum creatinine: The steps of the test were performed by using fixed rate BioMed-Creatinine kits purchased from BioMed Company (**Bowers & Wong, 1980**).

c- Serum Uric acid: The steps of the test were performed by using Color metric method assay by flexor pro-xl series Netherland (**Hori et al., 1992**).

d- Serum β_2 microglobulin: Immunometric Enzyme Immunoassay (ELISA) for the quantitative determination (**Bjerrum & Birgens, 1986**)

Statistical analysis: it was done using software SPSS version 20. Tests were:

I-One way ANOVA test between the 3 groups followed by Post Hoc test for parametric quantitative data between each 2 groups.

II- Person correlation coefficient

Data were expressed as mean \pm standard deviation (SD). $P < 0.05$ was considered significant.

RESULTS

Serum lead level of workers ranged 15-35 μ g/dl with median equals 20, and so the workers results divided into two groups: group II who had serum lead level $< 20\mu$ g/dl, and group III who had serum lead level $\geq 20\mu$ g/dl (**table 1**).

Table (1): Descriptive analysis of laboratory findings of Lead battery workers and control group

	Control (n=10)	lead battery workers (n=50)	Normal reference range
Blood lead level Range Mean \pm SD	(4-10) 5.8 \pm 1.8	(15-35) 22.82 \pm 7.21 Median=20	$< 10 \mu$ g/dL
Blood lead level < 20 ≥ 20	-----	48(48%) 52(52%)	
Urinary Delta-ALA Range Mean \pm SD	(2-4.7) 3.19 \pm 0.95	(2.7-8.2) 5.41 \pm 1.81	< 6 mg/L
Urea Range Mean \pm SD	(13-32) 22.9 \pm 6.65	(15-50) 29.79 \pm 9.41	13-40 mg/dl
Creatinine Range Mean \pm SD	(0.3-1.3) 0.85 \pm 0.32	(0.33-2) 0.97 \pm 0.38	0.5-1.5 mg/dl
Uric acid Range Mean \pm SD	(1.5-4.2) 2.85 \pm 0.86	(1.9-9.5) 5.47 \pm 2.18	3.5-7.2 mg/dl
β2 microglobulin Range Mean \pm SD	(1-3) 2.14 \pm 0.57	(1.5-7.6) 3.24 \pm 1.68	0-3 μ g/ml

ALA:amino-levulinicacid

SD: standard deviation

Workers having blood lead levels Lead $< 20 \mu$ g/dl (group II) had normal range of all parameters except β 2 microglobulin which is higher than normal. No statistical significance between this group and control group (group I) apart from blood lead levels which was found higher. Workers

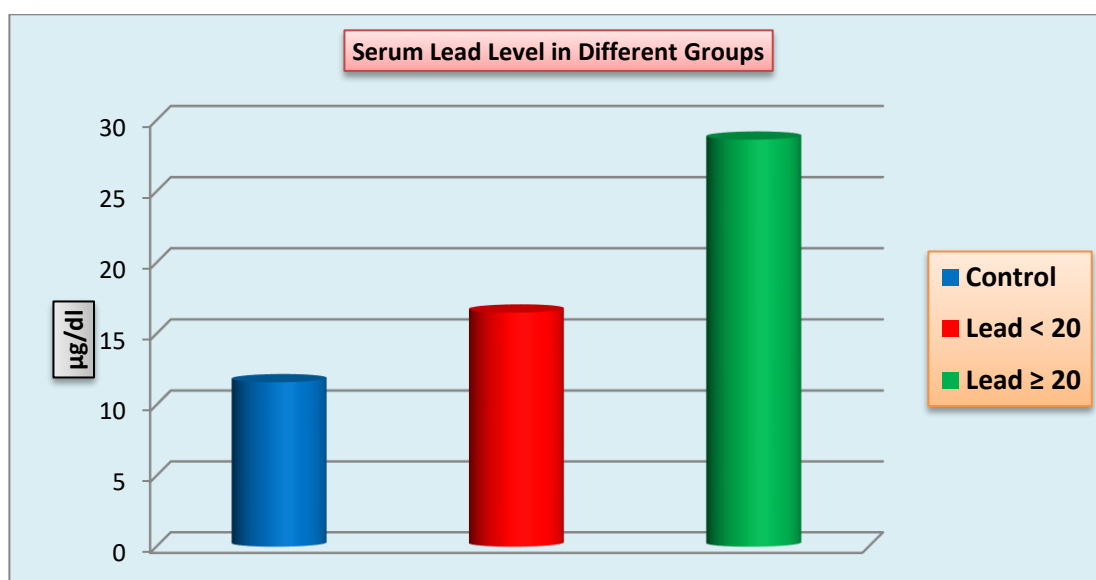
having blood lead levels $\geq 20 \mu$ g/dl (group III), found to have significantly higher levels of urinary delta aminolevulinic acid, blood urea, serum creatinine, serum uric acid and β 2 microglobulin than who having blood lead level $< 20 \mu$ g/dl or control group. (**table 2 and histograms 1-6**).

Table (2): One way ANOVA test for of laboratory data in relation to blood lead level

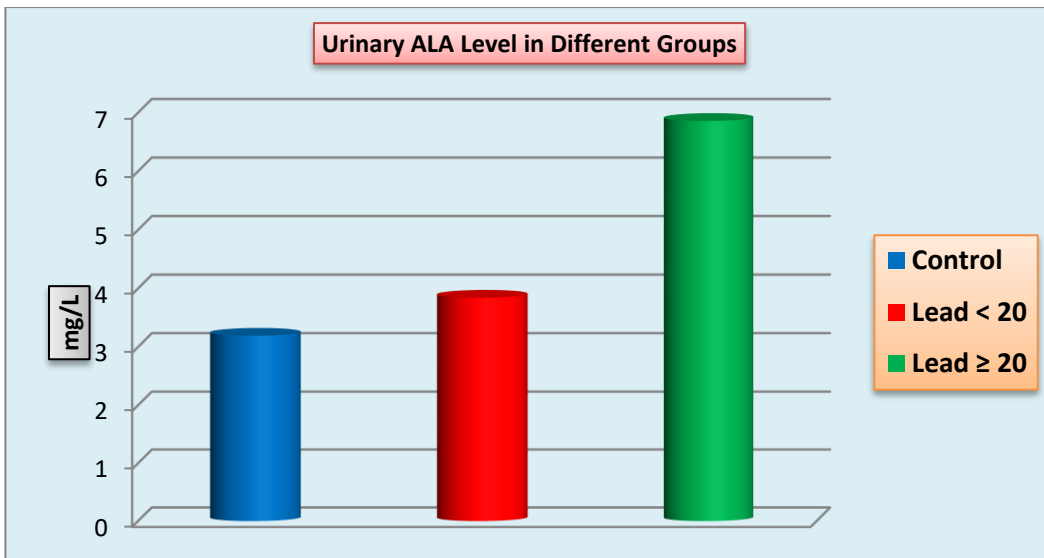
	Group I N=10	Group II N=24	Group III N=26	P value		
Lead level	(4-10)	(15-19)	(20-35)	<0.001*		
Range	5.8	16.5	28.7	IvsII	IvsIII	IIvs III
Mean ± SD	±1.8	±1.44	±5.12	0.003*	<0.001*	<0.001*
Delta ALA	(2-4.7)	(2.7-4.7)	(3.5-8.2)	<0.001*		
Range	3.19	3.84	6.86	I vs II	IvsIII	IIvs III
Mean ± SD	±0.95	±0.46	±1.31	0.196	<0.001*	<0.001*
Urea	(13-32)	(15-36)	(30-50)	<0.001*		
Range	22.9	25.02	34.19	I vs II	I vs III	IIvs III
Mean ± SD	±6.65	±8.32	±8.3	0.765	0.001*	<0.001*
Creatinine	(0.3-1.3)	(0.33-1.4)	(0.5-2)	<0.001*		
Range	0.85	0.77	1.16	I vs II	I vs III	IIvs III
Mean ± SD	±0.32	±0.25	±0.38	0.812	0.031*	<0.001*
Uric acid	(1.5-4.2)	(1.9-6)	(2.7-9.5)	<0.001*		
Range	2.85	4.02	6.81	I vs II	I vs III	IIvs III
Mean ± SD	±0.86	±1.16	±2.05	0.131	<0.001*	<0.001*
β2 micro globulin	(1-3)	(1.5-4.2)	(2.9-7.6)	<0.001*		
Range	2.14	2.15	4.25	I vs II	I vs III	IIvs III
Mean ± SD	±0.57	±0.73	±1.69	0.999	<0.001*	<0.001*

ALA: amino-levulinic acid

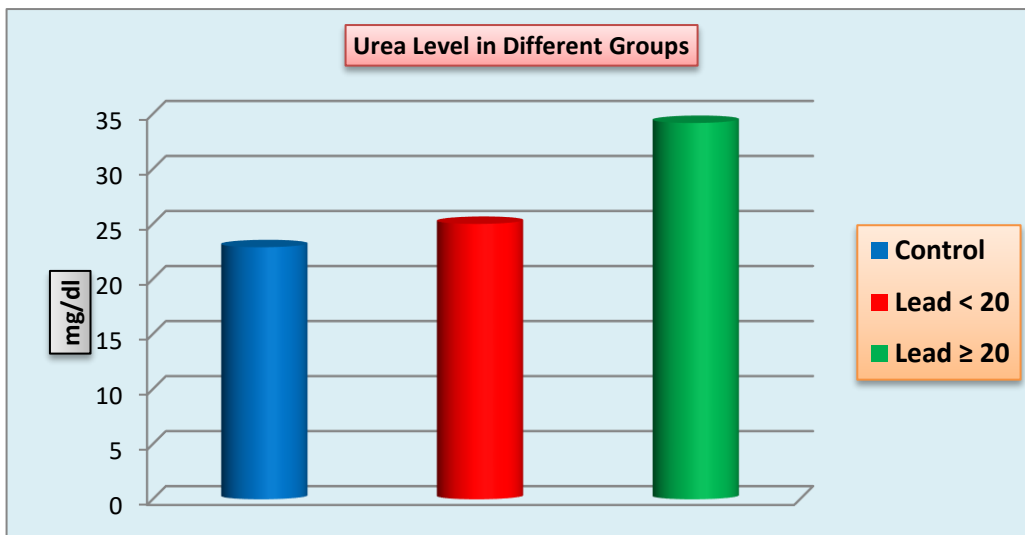
p<0.05 is significant



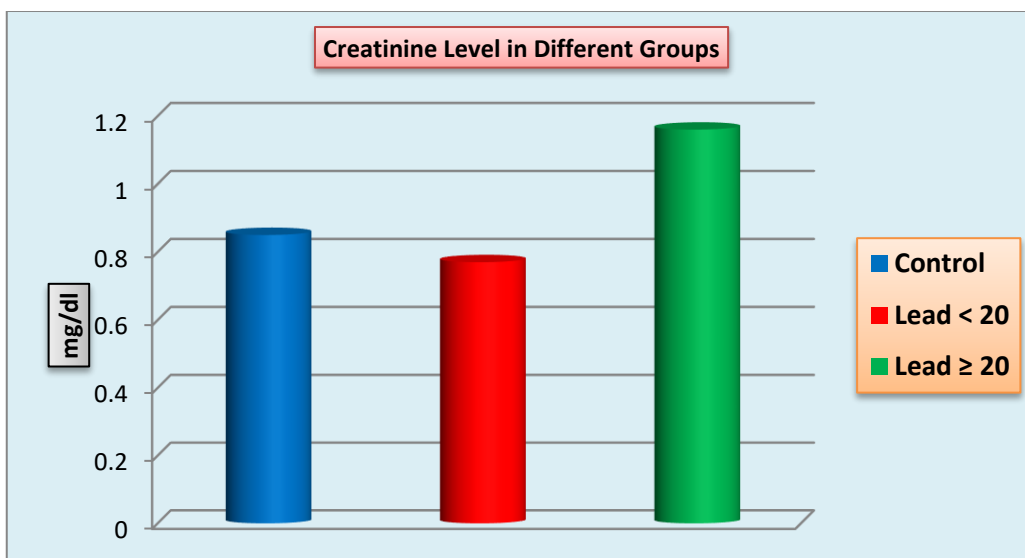
Histogram (1)



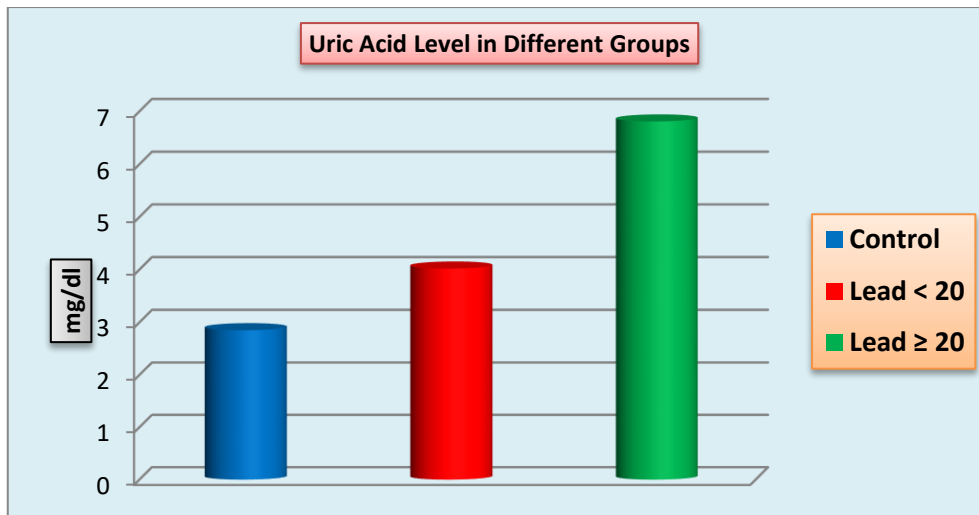
Histogram (2)



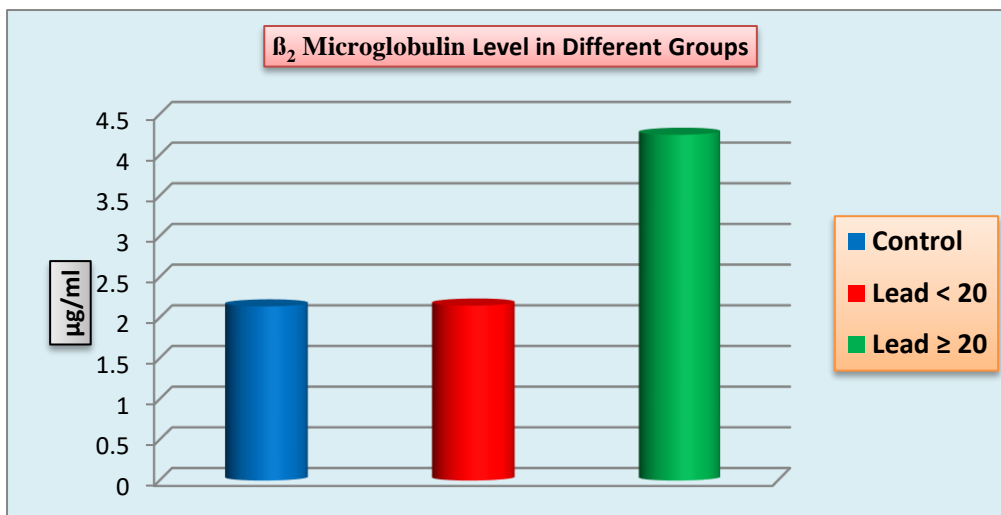
Histogram (3)



Histogram (4)



Histogram (5)



Histogram (6)

Correlation between blood lead level of studied subjects and urinary delta aminolevulinic acid revealed strong correlation coefficient (r=0.957). There was moderate significant correlation between serum lead and

blood urea (r=0.506) or serum creatinine (r=0.663). The correlation between serum lead and serum uric acid (r=0.710) or β₂ microglobulin (r=0.709) was moderate to strong (table 3, fig. 1-5).

Table (3): correlation test between BLL and other laboratory data among lead battery workers

	Blood lead level	
	r	P value
Urinary Delta ALA	0.957	< 0.001*
Urea	0.506	< 0.001*
Creatinine	0.663	< 0.001*
Uric acid	0.710	< 0.001*
β ₂ microglobulin	0.709	< 0.001*

ALA: amino-levulinic acid r: correlation p<0.05 is significant

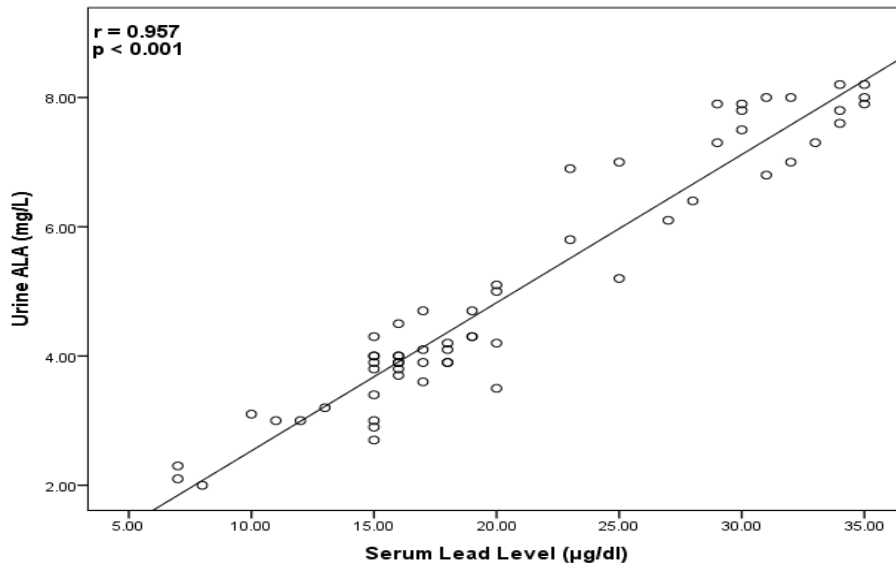


Figure (1): showing correlation between blood lead level and Urinary Delta ALA

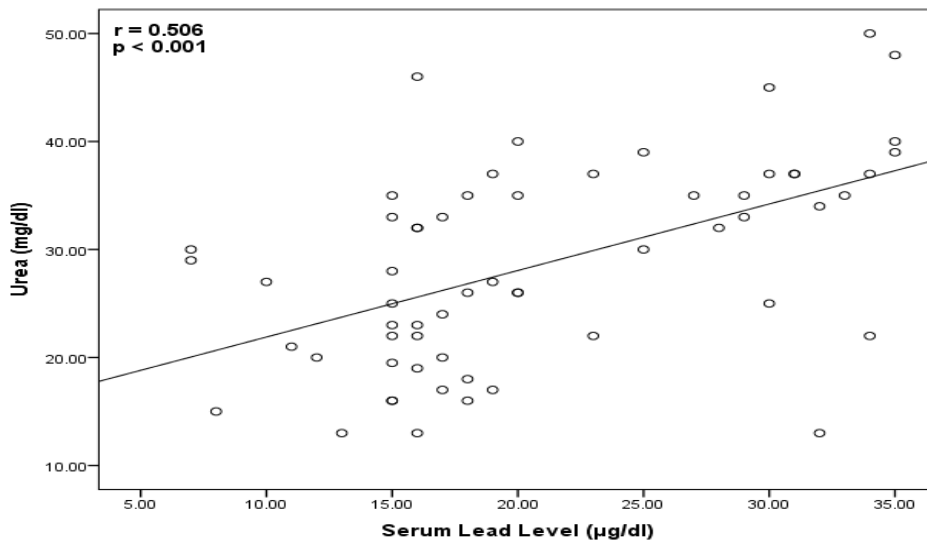


Figure (2) showing correlation between blood lead level and blood urea

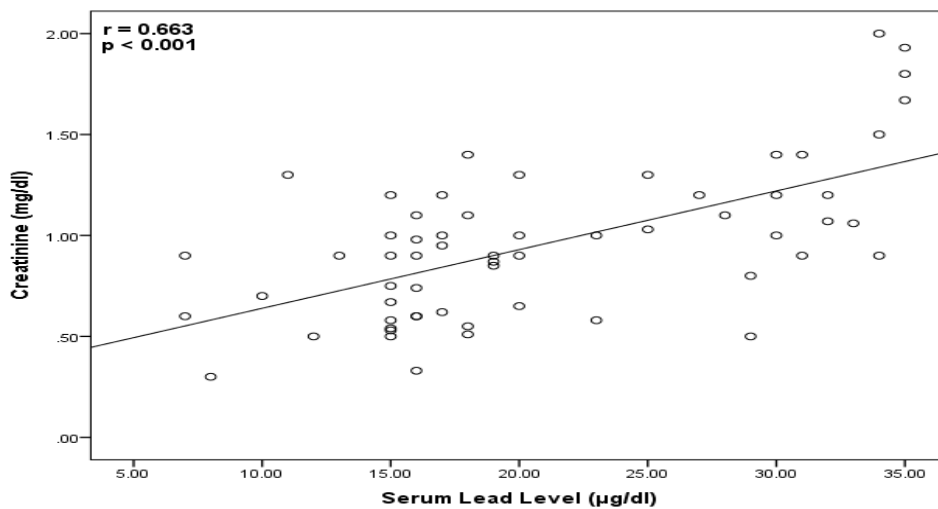


Figure (3) showing correlation between blood lead level and serum creatinine

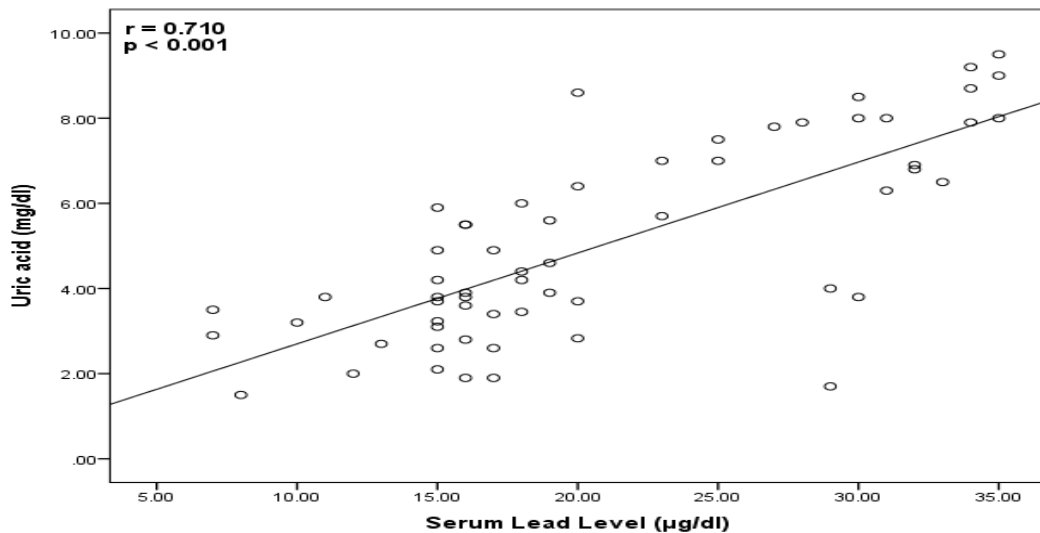


Figure (4) showing correlation between blood lead level and uric acid

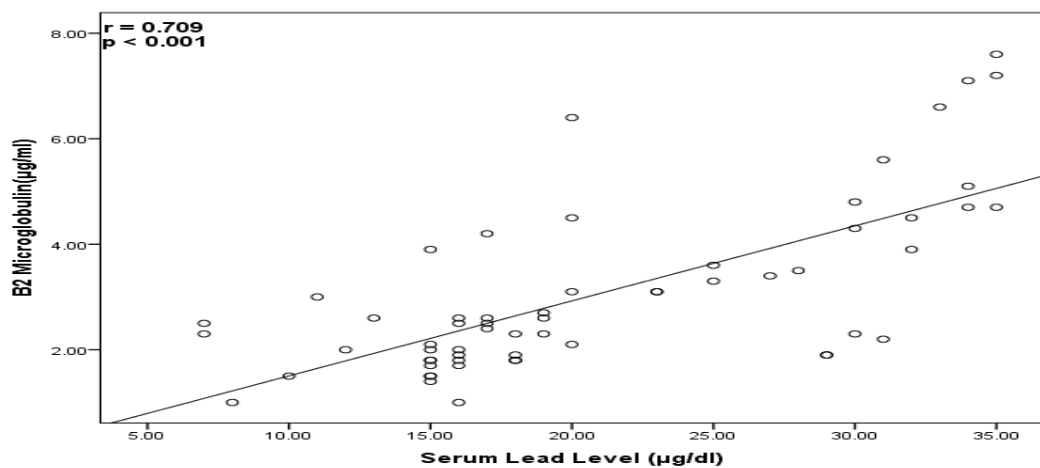


Figure (5) showing correlation between blood lead level and β 2 microglobulin

DISCUSSION

Lead poisoning occurred as a result of exposure to high lead levels which accompanied by severe health effects. Lead toxicity not depends only on exposure levels but also on duration of that exposure. No lead level is surely considered safe or abnormal (**Grant, 2009**).

Regarding blood lead level, the present study showed that mean blood lead level in lead battery workers was 22.82, which has significance increase than control group (5.8 μ g/dL). Such observation was in agreement with **Weaver et al., (2005)**. **Dongre et al., (2010)** reported higher blood lead level

(6.8 μ g/dL) in the control group and this difference may be due to many factors including environment pollution, places of the study whether in mega capital cities or in semi urbanized towns, and variation of socioeconomic status of different societies of above studies.

The present study showed significant elevation of blood urea or serum creatinine levels in lead-exposed workers, and moderate positive correlation of them with BLL. These findings were in agreement with **Ahmed et al., (2008)**; and **Fadrowski et al., (2010)**. Some researches detected that there was an association between

lead exposure in current and even former lead workers and adverse renal function outcomes (**Chowdhury et al., (2015) and Kao & Rusyniak , (2016)**).

Nephrotoxicity occurred by lead because the kidney is the main route of lead elimination. Lead binds to specific lead-binding proteins forming lead-protein complexes which are observed as typical intracellular inclusions in the proximal tubular cells of the renal tubules (**Huang et al., 2013**). Reduction in the glomerular filtration rate and a consequent rise in the blood urea level might be a manifestation of subclinical lead poisoning causing renal insufficiency (**Spector et al., 2011**), and **Alasia et al., (2010)**.

In contrast to current results, **Ekong et al., (2006)**, demonstrated no significant correlations between blood lead level and serum creatinine. Their suggestion may be explained by **Obermayr et al., 2008**, who reported that early stages of lead intoxication associated with tubular dysfunction and so affect tubular parameter (serum uric acid) more than the glomerular parameters (serum creatinine and blood urea) in moderately exposed workers (<60 lg/dl). Another explanation is the hyperuricemia from lead toxicity that causes two mechanisms: the first is endothelial dysfunction which leads to thickening of the afferent arterioles of the glomerulus, and the second is inhibition of nitric oxide release within the vasculature of the kidneys which leads to reduction of renal blood flow and glomerular filtration (**Johnson et al., 2003**); and (**Weaver et al., 2005**).

There was significant increase of serum uric acid level in lead-exposed workers and moderate to strong positive correlation of it with BLL, and this was in accordance with (**Evans et**

al., 2010) and (**Bislimi et al., 2013**). In acute and chronic lead nephropathy, hyperuricemia occurred due to isolated proximal tubular defects that resulted in increased tubular reabsorption and reduced secretion of uric acid. Also lead causes hyperuricemia through inhibition of guanine aminohydrolase and so affection of purine metabolism (**Weaver et al, 2000**).

Urinary δ -ALA level in the present study showed significant elevation in lead-exposed workers and strong positive correlation of it with BLL, and these findings as **Makino et al., (2000)**; and **Fukui et al., (2005)** reported. Urinary concentrations of δ -ALA increased due to inhibition of amino levulinic acid dehydratase (ALAD) (**Yang et al., 2014**). Lead strongly inhibited ALAD activity because it oxidized ALAD's sulfhydryl group and removed zinc from its active site (**Evans et al., 2010**).

The current research revealed significant increase of beta-2 microglobulin level in lead-exposed workers and moderate to strong positive correlation of it with BLL, and these results are in acceptance with (**Lin & Tai-Yi, 2007**) and (**Tian et al., 2005**).

In group II whose BLL <20 μ g/dL, all studied parameters were within normal except β 2 microglobulin which was higher than its normal value. **Aksun et al., 2004** and **Sommar et al., 2013**, suggested that β 2 Microglobulin is more sensitive and accurate parameter for renal functions assessment as it increase more and early before other parameters. β 2 Microglobulin is a preferable biomarker as it is not influenced by age, gender, or muscle mass (**Karimooy et al., 2010**).

CONCLUSION & RECOMMENDATION

All studied parameters of renal affections significantly affected in battery workers in comparison to volunteer subjects living in areas away from industrial cities. β_2 microglobulin has the highest correlation among the studied parameters. Also correlation coefficient of serum uric acid was higher than that of urea and creatinine, but still lower than β_2 microglobulin. So, β_2 microglobulin is the best predictive biomarker in assessment of renal affection in lead exposed workers followed by serum uric acid. Finally, regular periodic measurement of β_2 microglobulin was recommended for all workers exposed to lead.

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التقييم المبكر للتأثر الكلوي بين عمال بطاريات الرصاص - مدينة المنيا

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الملخص العربي

التلوث البيئي بالرصاص هو مصدر قلق كبير للصحة العامة ، ويعتبر الرصاص من الملوثات واسعة الانتشار عالميا وأحد أهم أسباب انتشار الأمراض وتزايدها خاصة في الدول النامية حيث أظهرت بعض الدراسات أن مايقرب من(99٪) من أسوء وأخطر حالات التسمم بالرصاص تحدث في البلدان النامية. لذا فإن الهدف من الرسالة هو تحديد بعض التحاليل الكلوية المناسبة للتقييم المبكر للخلل الكلوي في عمال بطاريات الرصاص.

فقد ضمت هذه الدراسة ستون شخصا جميعهم من الذكور منهم عشرة أشخاص تطوعوا اختياريًا في الدراسة تم إدراجهم كمجموعة 1 ،والخمسون الآخرون من عمال مصانع بطاريات الرصاص بمدينة المنيا تم تقسيمهم لمجموعتين:
المجموعة 2: شملت 24 عامل نسبة الرصاص بالدم كانت أقل من 20 ميكروجرام/ديسيلتر.
المجموعة 3: شملت 26 عامل بلغت نسبة الرصاص بالدم أكثر من 20 ميكروجرام/ديسيلتر.

وقد تم لكل شخص في هذه الدراسة الآتي:

1- استبيان بالتاريخ الشخصي لكل شخص (السن –الحالة الاجتماعية- مدة العمل بالمصنع)
2- قياس: نسبة الرصاص و اليوريا والكرياتينين وحمض اليوريك و (بيتا 2 ميكروجلوبولين) بالدم ونسبة مادة(حمض دلتا أمينو ليفولينك) بالبول.
وقد اظهرت النتائج الآتي:

1- تحاليل المجموعة الثانية كانت في النسب الطبيعية باستثناء بيتا 2 ميكروجلوبولين الذي كان أعلى من قيمته القصوى الطبيعية.

2- عمال مجموعة 3 لديهم نسب أعلى و ذات دلالة احصائية من مجموعة 2 ومجموعة 1 من حيث اليوريا والكرياتينين ونسبة حمض اليوريك و (بيتا 2 ميكروجلوبولين) بالدم ونسبة مادة (حمض دلتا أمينو ليفولينك) بالبول .

3- وجود علاقة توافقية موجبة بين نسبة الرصاص بالدم وكل التحاليل وبالأخص بيتا 2 ميكروجلوبولين وحمض اليوريك بالدم حيث كانت علاقتهما بنسبة الرصاص في الدم اقوى من التحاليل الاخرى.
من النتائج السابقة نستنتج الآتي:

1- زيادة نسبة الرصاص بالدم ذات تأثير سلبي وخطير على الكلي ذلك واضحا في ارتفاع معدلات عوامل تقييم الخلل الكلوي مثل اليوريا، الكرياتينين، حمض اليوريك، و بيتا 2 ميكروجلوبولين بالدم وايضا نسبة دلتا أمينو ليفولينك اسيد بالبول مع ارتفاع نسبة الرصاص بالدم.

2- قيم بيتا 2 ميكروجلوبولين زادت عن معدلها الطبيعي في الوقت اللي لا زالت باقى التحاليل في معدلها الطبيعي، كما كان لديها اعلي علاقة توافقية بين عوامل تقييم الخلل الكلوي. أيضا معامل الارتباط التوافقي لحمض اليوريك في الدم كان أعلى من اليوريا والكرياتينين، ولكن لا يزال أقل من بيتا 2 ميكروجلوبولين. لذلك بيتا 2 ميكروجلوبولين (يليه حمض اليوريك) في الدم تعد أفضل الدلائل المعملية التي يمكن الاعتماد عليها في التنبؤ المبكر للخلل الكلوي في العمال المعرضة للرصاص.