Study of kidney functions on occupationally lead exposure in some jobs in Al-Shati canyon area

Amna Basher Faraj[1], Mohamed Abd Alrhman Basher[2] ,Ali Farag Hawad[1], Abdulhakim A.M. Elwafa[1] and Albashir M. A. yhmed[1].

[1]Faculty of engineering and technology, [2]Faculty of medicine, Sebha University

E-mail: Hawad_a @yahoo.com

Abstract:

Background: Kidney disease is receiving increased global public health concern because of a significant increase in the prevalence of the disease. A high occupational lead exposure is well documented to be able to induce nephropathy, chronic exposure to lead may cause lead nephrotoxicity characterized by glomerular sclerosis, interstitial fibrosis and proximal tubular nephropathy. Lead-induced renal damage also occurs in the absence of acute intoxication so that occult lead nephropathy may not be recognized.
Objective: This study aims to evaluate blood lead level and the effect of lead on kidneys function in some occupationally lead exposed workers.
Methods: This study was carried out on subjects whom resident in Al-Shati canyon area, which located on south of Libya. Total of 166 subjects were participated in this study, they were all males, from which 60 subjects were not occupationally lead exposure, none of them were smokers or working on jobs, exposing them to lead, they were employed as control, 106 were considered as occupationally lead exposure.
Results: Lead, concentrations in blood of occupationally lead exposed groups (fuel station workers, car maintenance workshop workers, soldering workers, painting workers) and none occupationally lead exposed workers groups were 2.65±1.41, 1.97±1.47, 2.09± 1.06, 2.216±0.423, 2.07± 1.38, respectively, these values of lead concentration in blood were not statistically significantly different as compared with control. The creatinine in the blood of occupationally lead exposed workers groups were all within normal range. The levels of the creatinine for groups of occupationally lead exposed workers (fuel station workers, soldering workers and Paint workers) were statistically significantly different compared to the control. The levels of uric acid for groups of occupationally lead exposed workers (fuel station workers, car maintenance workshop workers and soldering workers) were not statistically significantly different compared to the control.
Conclusion: In conclusion these results show effect of lead on kidneys in most occupationally lead exposure; in additions even exposing to low lead levels can cause kidney dysfunction.

Keywords: lead, creatinine, uric acid, fuel station, car maintenance workshop, soldering workers and painter.

Introduction:

Research conducted in recent years has increased public health concern about the toxicity of lead at low dose and has supported a reappraisal of the levels of lead exposure that may be safely tolerated in the workplace. It has become increasingly evident that low-level lead exposure resulting in blood lead levels between 10 and 15 pg/dl (0.48-0.72 pmol/l) can lead to deleterious effects, particularly in infants and young children (Goyer, 1993). Lead is one of the most useful elements in industry but serves no useful function in the human body (Mahmoud Loghman-Adham, 1997).Exposure to the lead is public health problem and threat to environment with proven harmful impact on human, including industrial workers and general population. (Cabaravdic, et al., 2011). Lead is a toxic metal whose widespread use has caused extensive environmental contamination and health problems in many parts of the world. It is a cumulative toxicant that affects multiple body systems, including the neurological, haematological, gastrointestinal, cardiovascular and renal systems (World Health Organization, 2010). Children are particularly vulnerable to the neurotoxic effects of lead, and even relatively low levels of exposure can cause serious and, in some cases, irreversible neurological damage (Fewtrell, et al., 2003and IPCS, 1995). A multitude of studies have documented an association between chronic occupational lead exposure and impairment of renal function (Pollock and Ibels, 1988; Pollock Ibels 1986; Baker, et al., 1979; Pinto, et al. 1987 and Hong, et al.1980). Lead has been reported to cause nephrotoxicity by several mechanisms, although it is not known, which of these pathways, are predominant (Nolan and Shaikh 1992; Sanchez-Fructuoso, et al. 2002 and Vaziri 2002). Several adverse renal and vascular outcomes have been reported of low-level hyperuricemia, including hypertension and tubulointerstitial fibrosis (Mazzali, et al., 2001a), renal afferent arteriolopathy (Mazzali et al., 2002), glomerular hypertrophy, glomerulosclerosis (Nakagawa et al., 2003), and glomerular hypertension (Sanchez-Lozada et al., 2002). Lead poisoning results from the interaction of the metal with biological electron –donor groups, such as the sulfhydryl groups, which interferes with a multitude of enzymatic processes. Lead also interacts with essential cations, particularly calcium, iron, and zinc; it interferes with the sodium-potassium-adenosine triphosphate (Na-k-ATP) pump; and it alters cellular and mitochondrial membranes, thereby increasing cellular fragility. Additionally, lead inhibits pyrimidine-5-nucleotidase and alters other nucleotide functions (Habal, 2006). In some analyses reports of data for evaluations of longitudinal study of the health effects of inorganic lead exposure in 803 current and former lead workers (Weaver et al., 2003) found associations between lead exposure and dose measures and adverse renal function outcomes. Lead measures were associated with decreased renal function, primarily in the oldest tertile of workers (> 46 years of age). Lead is classically a chronic or cumulative toxin; hence, acute adverse effects are usually observed only following short-term exposures to high concentrations. Acute exposures to lead may cause gastrointestinal disturbances (anorexia, nausea, vomiting and abdominal pain), hepatic and renal damage, hypertension and neurological effects (malaise, drowsiness, and encephalopathy) that may lead to convulsions and death (IPCS, 1995). Long-term exposure to lead at levels less than those required to cause acute symptoms can result in weakness in the fingers, wrists and ankles, general fatigue, headaches,

anaemia, small increases in blood pressure, and reduced kidney function. There is a paucity of studies examining past lead exposure and health outcomes over long periods of time in adults with most research focusing on possible effects of lead on cardiovascular health, a recent review of observational studies by (Navas-Acien et al., 2007) concluded that while the apparent effect of lead on blood pressure was modest, the association could be causal, but there was not sufficient evidence to infer a causal relationship of lead exposure with clinical cardiovascular outcomes (Soderland, et al.,2010). Lead accumulates slowly in the body and even low doses can eventually lead to poisoning. 95% of lead in body is deposited in the bones and teeth while 99% of lead in blood is associated with erythrocytes. Lead poisoning can cause nervous system toxicity and renal tubular dysfunction leading to irreversible interstitial nephrosis with progressive renal impairment and hypertension. Lead also depresses haem synthesis and shortens the life span of erythrocytes, causing a hypochromic microcytic anaemia. One study showed altered hippocampal volume and brain metabolites in workers occupationally exposed to lead (Jiang, et al.,2008). Another showed a significant increase in the frequency of chromosomal aberrations in workers exposed to lead compared to the controls (Madhavi, et al., 2008). This study aims to evaluate blood lead level and the effect of lead on kidneys functions in some residents whom occupationally exposed to lead in Alshati valley area.

Material and Methods:

This study was carried out on subjects whom resident in Al-Shati canyon area, which located on south of Libya. Total of 166 subjects were participated in this study, they were all males, from which 60 subjects were not occupationally lead exposure none of them were smokers or working on job exposing to lead, they were employed as control, 106 were considered as occupationally lead exposure (they were work in different, occupationally lead exposure jobs, painter, Fuel station, Car maintenance workshop and Soldering). Their ages were ranging from 21 to 60 years. Blood samples were collected from subjects and the samples delivered into tubes contain no anticoagulant for determination of creatinine and uric acid, it was kept for 60-90 minutes at room temperature to clot then centrifuged at 3000 rpm for 10 minutes then the serum was separated. Serum samples were stored at–20^{o}c until determinations. The kits for these analyses were supplied by Biocon, a German company, and 5ml of blood delivered into tubes contain heparin as anticoagulant used for lead determination. Creatinine was determined by spectrophotometer following the method by (Taursky, 1956). Uric acid was determined by the method of (Henry, et al. 1957). Lead in blood was determined by atomic absorption spectrometry (AAS), Using–a Perkin-Elmer Model 2380 atomic absorption spectrometer, as described by the method of Hassel(1968). Statistical analysis was carried out using minitab statistical program in all analysis. Data was expressed as mean ± standard deviation. Level of statistical significance was at $p<0.05$.

Results:

Table: (1) shows the characteristics of the study groups, the means of work durations for the four groups were 9.2800, 19.543, 11.095, and 10.920 respectively. Number of the workers in the four groups were smokers, 13(52%), 14(40%), 8(38%) and 9(36%) respectively. None of the workers in the four groups were engaged in other jobs that may expose them to lead poisoning.

Table: (1) Characteristics of the study worker groups.

Characteristics	Fuel station N=25		Car maintenance workshop N=35		Soldering N=21		Painter N=25	
	Range	$\bar{X}$	Range	$\bar{X}$	Range	$\bar{X}$	Range	$\bar{X}$
Age (years)	21-58	35.360	25-60	40.200	25-49	36.762	28-55	37.320
Duration of Work (years)	1-22	9.2800	4-35	19.543	2-24	11.095	5-34	10.920
Worker number and % of smokers	13(52%)		14(40%)		8(38%)		9(36%)	

N=number $\bar{X}$=mean

Table (2) and fig (1) shows means± standard deviation of lead concentrations in blood of occupationally lead exposed groups (fuel station workers, car maintenance workshop workers, soldering workers, Painting workers) and none occupationally lead exposed workers group. The values for blood lead concentrations were 2.65±1.41, 1.97±1.47, 2.09± 1.06, 2.216±0.423, 2.07± 1.38, respectively.

These values of lead concentration in blood for occupationally lead exposed workers groups (fuel station workers, car maintenance workshop workers, soldering workers, Painter workers) were not statistically significantly different as compared with that of none occupationally lead exposed group. Whereas, the p value for lead, concentrations were 0.086, 0.744 0.949 and 0.455 respectively ($p<0.05$). The lead concentrations for all groups were less than that concentration can cause effect

Table: (2) Concentration of lead in none occupationally and occupationally lead exposed.

parameter	None occupational (N=60)	Occupational lead exposure							
		Fuel station (N=25)		Car maintenance workshop (N=35)		Soldering (N=21)		Painter (N=25)	
	$\bar{X}$± SD	$\bar{X}$± SD	p	$\bar{X}$± SD	p	$\bar{X}$± SD	p	$\bar{X}$± SD	p
Lead µg/dL	2.07±1.38	2.65±1.41	0.086 (N.S)	1.97±1.47	0.744 (N.S)	2.09±1.06	0.949 (N.S)	2.216± 0.423	0.455 (N.S)

N=Number, $\bar{X}$ ± SD =means ±standard deviations and N.S =Not significant at $P > 0.05$

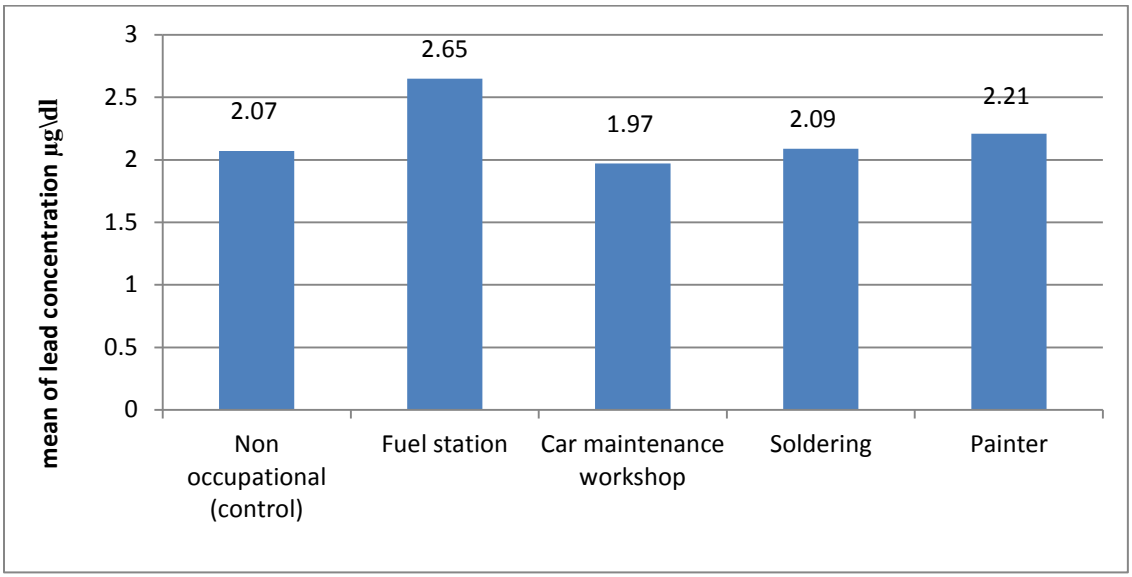

Fig: (1) Concentration of lead in none occupationally and occupationally lead exposed.

Table (3) and Figure (2) show means and standard deviation of Creatinine and Uric acid in blood of none occupationally and occupationally leads exposed workers groups (fuel station workers, car maintenance workshop workers, soldering workers and Paint workers). The values for Creatinine were 0.97±0.31, 1.24±0.43, 1.07±0.29, 1.12±0.37, and 1.32±0.47 respectively, these values for Creatinine in the blood of occupationally lead exposed workers groups were all within normal range. The mean ± standard deviation Creatinine in the car maintenance workshop workers was not statistically significantly different when p values was1. 127 ($p>0.05$).The levels of the Creatinine for groups of occupationally lead exposed workers(fuel station workers, soldering workers and Paint workers)were statistically significantly different compared to that of non occupationally lead exposed where the p values were 0.010 , 0.011 and 0.002, respectively,($p< 0.05$). The values for Uric acid for none occupationally and occupationally lead exposed groups were 7.12±2.22, 6.28±2.03, 6.94±2.70, 6.52±2.42, 5.40±1.41 respectively. The levels of Uric acid for groups of occupationally lead exposed workers (fuel station workers, car maintenance workshop workers and soldering workers) were not statistically significantly different compared to that of non occupationally lead exposed where the p values were 0.099, 0.748 and0.331 respectively,($p> 0.05$), whereas that for the Paint workers group was significantly different where the p value was 0.000,(< 0.05).

Table: (3) concentration of creatinine and uric acid in none occupationally and occupationally lead exposure.

parameter	Non occupational (N=60)	Occupational lead exposure							
		Fuel station (N=25)		Car maintenance workshop (N=35)		Soldering (N=21)		Painter (N=25)	
	$\bar{X}$±SD	$\bar{X}$± SD	p	$\bar{X}$± SD	p	$\bar{X}$± SD	p	$\bar{X}$±SD	p
Creatinine mg/dl	0.97 ±0.31	1.24 ± 0.43	0,010 (S)	1.07 ± 0.29	1.127 (N.S)	1.12 ± 0.37	0.011(S)	1.32 ± 0.47	0.002 (S)
Uric acid mg/dl	7.12 ±2.22	6.28 ± 2.03	0.099 (N.S)	6.94 ± 2.70	0.748 (N.S)	6.52 ± 2.42	0.331(N.S)	5.40 ± 1.41	0.000 (S)

N=Number, $\bar{X}$± SD =means ±standard deviations, N.S =Not significant at P > 0.05 and S = significant at P < 0.05

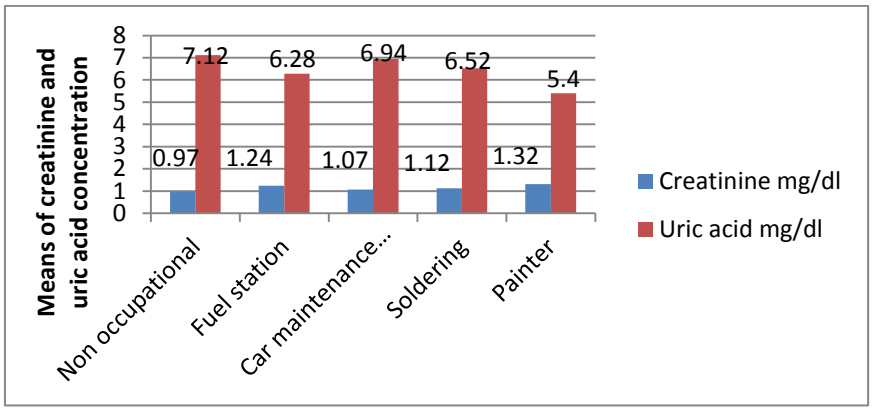

Fig: (2) concentration of Creatinine and Uric acid in none occupationally and occupationally lead exposure.

Discussion:

Kidney disease is receiving increased global public health attention because of a significant increase in the prevalence of the disease, the huge cost of treatment, and the appreciation of its role as a risk factor for cardiovascular disease (Levey, et al., 2009). Lead circulates in the blood, and can either be excreted by the kidneys or can deposit in the bone. The half-life of lead is approximately 35 days in the blood and 10 to 30 years in the bone. Accordingly, blood lead levels may denote both acute exposure and may also reflect equilibration with the bone pool, whereas bone stores reflect the cumulative or chronic exposure and account for 95% of total body lead burden (Barry and Mossman, 1970).In this study the low blood levels may indicated low occupationally lead exposure in all studies groups. Several studies have indicated a strong association between blood lead levels (BLL) and age related decline in renal function of the general population (Kim. Et al. 1996 and Staessen, et al., 1992). Exposure to lead in adults, shows symptoms at levels above 40 µg/dL, but are more likely to occur only above 50–60 µg/dL(Karri, et al.,2008). Symptoms begin to appear in children generally at around 60 µg/dL (Needleman, 2004). However, the lead levels at which symptoms appear vary widely depending on unknown characteristics of each individual (Bellinger, 2004). The results of this investigation shows increased levels of the Creatinine for groups of occupationally lead exposed workers in fuel station workers, soldering workers and Paint workers, this results may be reflect the chronic occupational exposure to lead. These results are not agree with that of (Dioka, et al., 2004), whom stated that occupational lead exposure did not have any effect on creatinine. Clinical manifestations of renal impairment do not become evident until more than 50% of the nephrons have been destroyed (Goodman, 1985). Lead-induced renal damage also occurs in the absence of acute intoxication so that occult lead nephropathy may not be recognized as such (Restek-Samarzija et al., 1997). Delay, in the onset of adverse effect of lead poisoning on kidney function is known to occur in previous lead workers (Restek-Samarzija et al., 1996, Restek-Samarzija and Momcilovic, 1992). Chronic accumulation of lead in the body eventually leads to impairment in renal function (Restek-Samarzija et al., 1996). In this study the levels of uric acid for occupationally lead exposed for the Paint workers group were significantly raised compared with controls, this result is consistent with of (Dioka, et al., 2004). Chronic lead nephropathy can be missed in its early stages because changes induced by chronic low-level lead exposure are subtle and not reflected by changes in routine renal function tests such as blood urine nitrogen (BUN) and serum creatinine concentrations. More than 50% of kidney function may be lost before significant changes in serum creatinine are detected. Recent studies have focused on the use of more sensitive tests to detect lead-induced renal dysfunction in its early stages, when such changes might still be reversible by treatment and abatement of lead sources (Mahmoud Loghman-Adham, 1997). Those parameters which are used in this study may are not specific for assessing kidney functions and are not sensitive enough for detecting early effect of occupationally lead exposure. This result shows effect of lead on kidneys in most occupationally lead exposure; in additions even exposing to low lead levels can cause kidney dysfunction.

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الخلاصة:

يلقى الاعتلال الكلوي اهتماما عالميا متزيدا بسبب الانتشار الكبير للمرض. و تؤكد الوثائق العلمية أن التعرض المهني الشديد للرصاص قادر علي التسبب في اعتلال الكلي، والتعرض المزمن للرصاص ربما يؤدي ألي التسمم الكلوي، الذي يتميز بتصلب النسيج الكُبيبي، إلتهاب النسيج الليفي البيني و الاعتلال الكلوي الناتج عن عطب الأنبوب القريب، الضرر الكلوي الناتج عن الرصاص أيضا يظهر في غياب التسمم الحاد ولذلك الاعتلال الكلوي الخفي بسبب الرصاص ربما لا يتم التعرف عليه. تهدف هذه الدراسة ألي تقدير مستوي الرصاص في الدم وتأثير الرصاص علي وظائف الكلي في بعض الأشخاص المعرضين مهنينا له في منطقة وادي الشاطئ .أجريت هذه الدراسة على 166 شخص، يسكنون منطقة وادي الشاطئ، جميعهم رجال، منهم 106 شخص معرضين مهنيا للرصاص وستون شخص غير معرضين مهنيا للرصاص، جميعهم غير مدخنين، استخدموا كعينة ضابطة، تراوحت أعمارهم. بين 21 -61 سنة. مستوي الرصاص في دم المعرضين مهنيين للرصاص كان مخفض حيت كان في كل من المهن الآتية محطات الوقود، وصيانة السيارات، عمال اللحام و عمال الطلاء ، 2.65 ±0.423 2.216 ± 1.06, 2.09 ±1.47, 1.97 ±1.41, علي التوالي أما في العينة الضابطة فقد كان 2.07 ± 1.38. هذه القيم لم تكن ذات اختلاف معنوي عند مقارنتها بالعينات الضابطة . مستوي تراكيز الكريتينين في دم المعرضين مهنيين للرصاص كان ضمن ألمدي الطبيعي، وعند مقارنة مستوي الرصاص عند العاملين بمحطات الوقود والعاملين بمهنة اللحام وعمال الطلاء بالعينة الضابطة تبين و جود فروق معنوية، أما مستوي حمض اليوريك في المعرضين مهنيا من خلال العمل في كل من محطات الوقود و ورش صيانة السيارات وإعمال اللحام لم تكن ذات فروق معنوية عند مقارنتها بالعينات الضابطة،أما العاملين في مهنة الطلاء فقد كانت ذات فروق معنوية. تبين هذه النتائج تأثير الرصاص علي وظائف الكلي حتى حينما يكون تركيز الرصاص منخفض.

الكلمات المفتاحية: الاعتلال، الكلوي، التعرض، المهني، للرصاص.