Detection of Subclinical Neuropathies in Some Egyptian Workers Exposed To Lead (Cross-Sectional Study)

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Abstract Chronic lead toxicity is more common and serious for multiple organs. The objective of the present study was designed to detect subclinical neuropathies in Egyptian workers exposed to lead. A cross sectional study was carried out on sixty male Egyptian individuals; fifty workers were chronically occupationally exposed to lead and ten apparently healthy male volunteers not exposed to lead in their occupation served as a control group. All cases were subjected to clinical examination for lead toxicity. Neurological and nerve conduction study were done for both motor and sensory nerves. Estimation of blood and urine lead levels for all participants was done. A laboratory assessment was carried out for evaluation of hemoglobin level, reticulocytic count and basophilic stippling. The result of this study demonstrated elevation of both blood and urine lead levels of all workers. Increased in distal motor latency with slow conduction velocity and decreased amplitude of nerve conduction were observed in the studied nerves of all workers exposed to lead. Also, slow conduction velocity and decreased amplitude were the earliest and most sensitive finding of sensory conduction study of all workers exposed to lead. There were a significant correlation between blood and urine lead level, duration of exposure to lead and different parameters of nerve conduction. The present study concluded that nerve conduction either sensory or motor can give a clear image about chronic lead poisoning even with absence of any clinical findings (sub-clinical neuropathy). This study recommend periodic examination of workers exposed to lead for early detection and after recognition of these affected workers we must advise them improve their hygiene, use personal protective equipments or try to change their work and to notify industrial safety about them.

Keywords lead, nerve conduction, neuropathy

Introduction

ead is a part of our world today. It is found in the dust, soil, water, air, food and paint of some homes or buildings built before 1978. These type of exposures affect the entire population, and occur primarily by ingestion or inhalation (Ragan & Turner, 2009).

Although lead is extremely useful in improving the performance of paint, gasoline, and plumbing, it is also dangerous (Bellinger, 2005). In industrial societies, sources of lead exposure include: the smelting and refining of lead, the production and disposal of storage batteries, combustion of leaded fuels, production of iron and steel, manufacture of leaded paints, burning of lead-painted surfaces, application of pesticides containing lead arsenate, and incineration of leaded plastics (Ye & Wong 2006). Lead toxicity is classified into acute and chronic. Chronic lead toxicity affects all body systems including the nervous system (Phipps et al., 2012).

Nerve conduction study is an essential element in the evaluation of peripheral neuropathies by clarifying the distribution and extent of involvement, type and time course of nerve damage. When these data are combined with clinical information, a full characterization of the neuropathy is possible (Bromberg, 2013).

Nerve conduction study is minimally invasive test that is widely available and standardized and is primarily requested in order to investigate sensory or motor dysfunction in the limbs (Vollans & Hasan, 2011). The compound muscle action potential is a biphasic potential with an initial negative or upward deflection from the baseline. For each stimulation site, the latency and amplitude are measured as shown in **Figure (1).** Motor conduction study is monitored by the amplitude of compound muscle action potential, distal motor latency and the measured motor conduction velocities. (Soliman & Kothari 2002). This work was designed to detect subclinical neuropathies in Egyptian workers exposed to lead.

Subjects and methods

This cross sectional study was carried out on sixty male Egyptian individuals; fifty workers out of them were chronically occupationally exposed to lead, collected from automechanic workshops (welders, battery recyclers & chargers, and car painters).

The study was performed following approval of the Medical Research Ethical Committee of Faculty of Medicine, Tanta University. Workers and control group signed a written informed consent before starting the study and after complete description of the study. Privacy of the patient and confidentiality of the data and results of investigation will be maintained by using coding number. After recognition of these affected workers we must advise them improve their hygiene, use personal protective equipments or try to change their work.

The selected subjects were divided into four groups according to kind of their works:

► Group I: Ten male volunteers served as control group working in hospital (workers and male nurses) were included. They were apparently healthy persons of comparable age, socioeconomic life style and smoking habits compared to other groups and with no history of occupational lead exposure.

► **Group II:** Included sixteen welders.

► **Group III:** Included sixteen battery recyclers and chargers.

► Group IV: Included eighteen car painters.

All workers were chosen as moderate smokers with smoking index= 101-300 (smoking index=number of cigarettes per day X total duration by years). By history, all diabetics, cancer patients, alcohol consumers and addicts were excluded from this study.

Each one of the groups II, III and IV was subdivided into 3sub- groups according to the duration of exposure to lead (less than 10 years, 10-20 years and more than 20 years).

Subjects were interviewed for sociodemographic data & occupational history and subjected to clinical examination for lead toxicity, neurological examination, and electrophysiological study using the Neuropack II- NEM- 7102/K.O Nihon Kohden apparatus. Room temperature was adjusted at 25 °C (Edward et al., 2008).

Half ml blood without any preservative was used after digestion and 100ml urine was obtained without digestion for estimation of lead levels for all participants by using an inductively coupled plasma optical emission spectrometry according to Granadillo et al., (1994). Laboratory assessments was carried out on (2.5ml blood) added to EDTA tubes for evaluation of hemoglobin level by ERMA PCE210, reticulocytic count by method of Davis & Bigelow., (1989) and basophilic stippling according to Munoz & Guo, (2011).

Electrophysiological study

This study was done in the right side only of upper and lower extremities.

1- Motor conduction study for median, radial and common peroneal nerves was illustrated in Fig. 2 , 3 & 4 (respectively).

2- Sensory conduction study using antidromic technique for median, radial and sural nerves was illustrated in Fig.5, 6 & 7 (respectively).

Statistical study

Statistical presentation and analysis of the results of this study were conducted, using the mean, standard student t- test, Chi-square, Tukey's test, Linear Correlation Coefficient, and Analysis of variance [ANOVA] tests by SPSS Version18. **Results**

All workers and control participants were matched with comparable sociodemographic data with no significant differences between all studied groups as regard to age, marital state, residence and educational level. Also, no significant differences in occupational history of the studied groups of workers as regard to duration of exposure (Table 1). All workers in all studied groups did not use any protective equipment at all.

General manifestation and clinical examination (Table 2):

In the present study 75 % of welders, 93.75 % of battery recyclers & chargers and 22.22 % of car painters workers suffered from headache. While, abdominal pain (43.75 % of welders, 87.5 % of battery recyclers & chargers and 50 % of car painters) and constipation (37.5%, 62.5 % and 33.33 % in group II, III and IV respectively) were represented the only gastrointestinal manifestations. Moreover, dental caries was observed in 18.75 % and 5.56 % of battery recyclers & chargers and car painters respectively. There were significant differences between all studied groups as regard to headache and abdominal pain.

In this study, no motor manifestations were detected in all subjects after meticulous clinical examination of muscle status (normal, wasted or hypertrophied), observation of fasciculation or involuntary movements, muscle tone examination (normal, hypotonic or hypertonic) and lastly muscle power assessment. There was statistically significant difference between all studied groups as regards the presence of abnormal superficial sensation including pain, touch and temperature ($X^2 = 12.445$ and P = 0.002).

Laboratory investigation

As demonstrated in **Table (3)**, the highest mean of blood lead level was observed in group III (71.001 \pm 5.991µg/dl), followed by group IV and II (55.138 \pm 6.172 and 46.111 \pm 6.641 µg/dl respectively). Also, the highest mean urine lead level was 25.850 \pm 3.592 µg/dl in group III. Significant differences were detected between all studied groups by ANOVA test (**F** for blood lead level 121.190 and **P= 0.000**, while for urine lead level 146.86 and **P= 0.000**).

By Tukey's test, there were significant difference between each two groups for both blood and

urine lead level (**P**= 0.000) except between group III & IV in urine lead level.

► Hemoglobin level, basophilic stippling & reticulocytosis:

There was only one subject in group III suffered a decrease in hemoglobin level (Hb=10.6 gm/dl). No red blood corpuscle abnormalities neither basophilic stippling nor reticulocytosis were detected.

<u>Electrophysiological changes of the</u> <u>studied groups:</u>

<u>Motor electrophysiological changes</u> of the studied groups:

Median nerve compound muscle action potential was illustrated in **Table** (4). By ANOVA test, there was significant increase in distal motor latency between workers and control group ($\mathbf{F}=17.282$ and $\mathbf{P}=0.000$). Regarding to amplitude and motor conduction velocity of median nerve, there were significant decrease in all workers compared to control group ($\mathbf{F}=96.077$ & $\mathbf{F}=16.843$ and $\mathbf{P}=0.000$) respectively.

In radial compound muscle action potential, the mean of distal motor latency was 3.210 ± 0.197 ms in group I, 3.479 ± 0.588 (ms) in group II, $6.028 \pm$ 1.598 ms in group III, and 3.837 ± 0.256 ms in group IV. While, amplitude and conduction velocity were significantly decreased in all groups of workers (**Table 5**).

Table (6) showed the muscle action potential of the common peroneal nerve, the highest mean with significance of distal motor latency was for group II and group III (6.920 ± 0.978 and 6.843 ± 0.518 (ms) respectively). The mean values of the amplitude were diminished in all workers. Additionally, the worst mean of conduction velocity was observed in group III 39.488 \pm 3.964 (M/S) and the best one was groupI 51.640 \pm 0.341(M/S).

By ANOVA test, there were significant differences between all studied groups as regard to motor compound action potential of median, radial and common peroneal nerve (Table 4, 5 & 6). While by Tukey's test, there were significant differences between each two group except between group III versus group IV of distal motor latency and motor conduction velocity and between group I versus II in amplitude and motor conduction velocity of median nerve (Table 4).

In radial nerve and by Tukey's test, there were significant differences between group I versus III, II & III and III & IV as regard distal motor latency, while there were significant differences between group I & II, I & III and I & IV of amplitude and motor conduction velocity (Table 5).

Table 6 demonstrated significant difference between each two groups as regard to all motor compound action potential of common peroneal nerve except between II versus III and III versus IV of distal motor latency and amplitude respectively.

<u>Sensory electrophysiological changes of the</u> <u>studied groups:</u>

As regard amplitude of median sensory nerve action potential, there was a significant difference between all groups (**P**= 0.000). According to Tukey's test there were significant differences between each two groups except group III versus IV. The best conduction velocity for median sensory action potential was 42.320 ± 0.496 (M/S) for control group, decreasing gradually through car painters and welders groups till been worst in battery recyclers and chargers reaching group 31.036 ± 2.978 (M/S) (**Table 7**). According to Tukey's test there were significant differences between each two groups except group II & IV as regard sensory conduction velocity.

In this study, radial sensory nerve action potential, the best mean value of the amplitude was observed in group I ($13.640 \pm 0.608 \mu v$) decreasing gradually till reaching the worst value ($1.548 \pm 0.526 \mu v$) in group III. The mean values of the conduction velocity of the radial nerve decreased evidently in group III followed by group IV (**Table 8**).

In this study, the lowest amplitude of sural nerve was $3.033 \pm 0.710 \ (\mu v)$ for battery recyclers & chargers group and the highest one was $28.450 \pm 0.525 \ (\mu v)$ for control group (**Table 9**).

<u>Correlations of all parameters of the</u> present study with blood lead level:

There was a significant positive correlation between blood lead level and duration of exposure in all workers exposed to lead (r= 0.493) with linear relationship as shown in **Graph 1**.

In all workers exposed to lead, there was a positive correlation between blood lead level and distal motor latency of median, radial and common peroneal nerves. There were also, a negative correlation between blood lead level and both amplitude & motor conduction velocity of the same previous nerves (Table 10, 11 & 12). Furthermore, in all workers exposed to lead sensory conduction velocities and amplitude of the median, radial and sural sensory nerve action potential were in a negative linear correlation with blood lead level (Table 13, 14 & 15). While, the simple regression analysis between dependable factor (blood lead level) and independable one (duration of exposure and urine lead level) are done as illustrated in Table (16 & 17). This equation is used to predict dependable factor from independable one, in the current study the prediction of blood lead level is possible from duration of exposure by 22.7 % and from urine lead level by 54.619 % (**P-value= 0.000**).

There was a significant positive correlation between blood lead level and urine lead level in all workers (r=0.869) with linear relationship (Graph 2).

Group											Chi-Square	
		Grou	ıp II	Grou	ıp III	Grou	ıp IV	Tota	ıl			
		N(16)	%	N(16)	%	N(18)	%	N(50)	%	X ²	P-value	
u	<10 years	4	25.00	4	25.00	4	22.22	12	24	2.567	0.633	
ttio	10-20 years	8	50.00	6	37.50	5	27.78	19	38			
Dura	>20 years	4	25.00	6	37.50	9	50.00	19	38			

Table (1) Duration of exposure to lead in all studied workers

N: number, G I: control, G II: welders, G III: battery recyclers & chargers, G IV: car painters.

Table (2): Chi square analysis for general manifestations and clinical examination of chronic lead poisoning in all studied workers and controls

Group								Chi-Square					
		Gro	oup I	Gı	coup II	Gr	oup III	Gr	roup IV	To wo expose	tal of rkers ed to lead		
		N (10)	%	N (16)	%	N (16)	%	N (18)	%	N (50)	%	X ²	P-value
ys		0											
eneral ations 3da ago	Headache		0.00	12	75.00	15	93.75	4	22.22	31	62	20.082	< 0.001*
	Abdominal pain	0	0.00	7	43.75	14	87.50	9	50.00	30	60	7.552	0.022*
G nifest	Constipation	0	0.00	6	37.50	10	62.50	6	33.33	22	44	3.328	0.189
ma	Dental caries	0	0.00	0	0.00	3	18.75	1	5.56	4	8	4.05	0.132
tio	Pain	0	0.00	5	31.25	11	68.75	16	88.89	32	64	12.445	0.002*
iical ninat	Touch	0	0.00	5	31.25	11	68.75	16	88.89	32	64	12.445	0.002*
Clir exal n	Temperature	0	0.00	5	31.25	11	68.75	16	88.89	32	64	12.445	0.002*

N: number, GI: control, GII: welders, GIII: battery recyclers & chargers, GIV: car painters, *: significant

Table (3): ANOA one way statistical analysis followed by	tukey`s for blood and urine	lead level in all studied
workers		

	Blood lead leve	el (µg/dl)	Uriı	ne lead level(µg/	dl)	ANOVA					
	Mean ±	SD	Mea	an ± SD		F	F1	Р	& P1-value		
Group I	28.460 ±0.825		8.014±0.600			121.190	146.86		0.000*		
N(10)											
Group II	46.111±6.641		20.608±1.459								
N(16)											
Group III	71.001 ± 5.991	25.850 ±3.592									
N(16)											
Group IV	55.138 ± 6.172		24.734 ±1.964								
N(18)											
				Tukey`s test							
Blood	I& II	I& II	Ι	I& IV		II& III	II&	IV	III & IV		
	0.000*	0.000	*	0.000*		0.000*	0.00)0*	0.000*		
Urine	I& II	I& II	Ι	I& IV		II& III	II&	IV	III & IV		
	0.000*	0.000	*	0.000*		0.000*	0.00)0*	0.495		

N: number, G I: control, G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant F ANOVA for blood lead level, F1 ANOVA for urine lead level, P Significance for blood lead level, P1 Significance for urine lead level

	Distal motor latency of the median nerve in (ms)		Amplitude of the median nerve (mv)	co vo th ne	Motor onduction elocity of e median rve (M/S)		ANOVA			
	Mean ± SD		Mean± SD	Me	ean± SD	F	F1	F2	P, P1 & P2-value	
Group I N(10)	4.012±0.112	3.465±0.287	48.	182±0.443	17.2 82	96.0 77	16.84 3	0.000*		
Group II N(16)	4.814±0.411	3.216±0.755	47.	213±2.051						
Group III N(16)	5.516±0.723		2.514±0.286	42.608±3.928						
Group IV N(18)	5.628±0.845		0.976±0.227	42.901±2.309						
			Tukey`s test							
Distal motor	I& II	I& III	I& IV		II& III		П& Г	V	III & IV	
latency	0.014*	0.000*	0.000*	:	0.015*		0.002	*	0.956	
Amplitude	0.523	0.000*	0.000*	:	0.000*		0.000	*	0.000*	
Motor conduction velocity	0.797	0.000*	0.000*	< 0.000*			0.000	*	0.988	

Table (4): ANOA one way statistical analysis followed by tukey's for compound muscle action potential of	the
median nerve in all studied workers and control group	

N: number, G I: control, G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant P-value for Distal motor latency *F* ANOVA for Distal motor latency F1 ANOVA for Amplitude

F2 ANOVA for Motor conduction velocity

ms: millisecond P1-value for Amplitude mv: millivolt P2-value for Motor conduction velocity M/S: Meter/Second

Table (5): ANOA one way statistical analysis followed	l by tukey`s for compound muscle action potential of the
radial nerve in all studied workers and control group	

	Distal motor latency of the radial nerve in m	Amplitu the rac s nerv	de of dial re	Mo condu velocit radial	otor uction y of the nerve		ANOVA				
	Mean ± SD	Mean±	Mean± SD		Mean± SD		F1	F2	P & P1- value	P2- val ue	
Group I N(10)	3.210±0.197	1.890±0	.137	48.760±	0.241	30.6 93	11.6 73	5.0 48	0.000*	0.00 4*	
Group II N(16)	3.479±0.588	1.318±0	0.551	46.256	46.256±3.197						
Group III N(16)	6.028±1.598	1.121±0	.281	45.954±1.968							
Group IV N(18)	3.837±0.256	1.122±0	0.287	45.463±	45.463±1.955						
		,	Tukey`:	s test							
Distal motor	I& II	I& III	Ið	& IV	II&	III	П	& IV	Ш &	IV	
latency	0.879	0.000*	0	.297	0.00	0*	0.	653	0.00	0*	
Amplitude	0.001*	0.000*	0.	*000	0.4	17	0.399		1.00	00	
Motor conduction velocity	0.035*	0.014*	0.	002*	0.93	80	0.728		0.9	0.918	

N: number, G I: control, G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant

F ANOVA for Distal motor latency

F1 ANOVA for Amplitude

P-value for Distal motor latency ms: millisecond P1-value for Amplitude mv: millivolt

F2 ANOVA for Motor conduction velocity

P2-value for Motor conduction velocity M/S: Meter/Second

	Distal motor latency of the common peroneal nerve(ms)		An th	nplitude of ne common peroneal nerve(mv)	ve ve	Motor conduction clocity of the common peroneal nerve(M/S)	ANOVA				
	Mean ± SD		Me	ean±SD	Mean±SD		F	F1	F2	P, P1 & P2-value	
Group I N(10)	4.709±0.366		5.7	/80±0.457	51	.640±0.341	22.4 90	42.5 11	61.5 85	0.000*	
Group II N(16)	6.920±0.978		3.6	99±1.371	47.870±1.645						
Group III N(16)	6.843±0.854		2.0	011±0.822	39.488±3.964						
Group IV N(18)	6.031±0.518		1.9	958±0.856	43.056±1.935						
				Tukey`s test							
Distal motor	I& II	I& III		I& IV		II& III		II& IV		III & IV	
latency	0.000*	0.000*		0.000*		0.991		0.005*		0.013*	
Amplitude	0.000*	0.000*		0.000*		0.000*		0.000*		0.999	
Motor conduction velocity	0.002*	0.000*		0.000*		0.000*		0.000*		0.001*	

Table (6): ANOA one way statistical analysis followed by tukey's for compound muscle action p	potential of the
common peroneal nerve in all studied workers and control group	

N: number, G I: control, G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant *F* ANOVA for Distal motor latency *P-value for Distal motor latency* F1 ANOVA for Amplitude mv: millivolt

F2 ANOVA for Motor conduction velocity

ms: millisecond *P1-value for Amplitude* P2-value for Motor conduction velocity M/S: Meter/Second

Table (7): ANOA one way statistical analysis followed by tukey's for sensory action potential of the media
sensory nerve in all studied workers and control group

	Amplitude nerve actio	of median sensor on potential (µv)	ry Senso velocity o	ry conduction f median sensory (M/S)		ANOVA	
	M	ean ± SD	Ν	Iean± SD	F	F1	& P1 – value
Group I N(10)	19.850 ± 0.58	3	42.320 ± 0).496	24.386	29.868	0.000*
Group II N(16)	15.653 ± 3.97	7	34.689 ± 3	3.565			
Group III N(16)	12.290 ± 2.38	7	31.036 ± 2	2.978			
Group IV N(18)	11.841 ± 1.58	6	35.400 ± 3	3.139			
		,	Tukey`s test				
Amplitude	I& II	I& III	I& IV	II& III	II& IV	II	I & IV
	0.002*	0.000*	0.000*	0.003*	0.000*		0.957
Sensory conduction velocity	0.000* 0.000* (0.000*	0.005*	0.898	().000*

N: number, G I: control, G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant

F ANOVA for Distal motor latency

F1 ANOVA for Amplitude

P-value for Distal motor latency ms: millisecond mv: millivolt P1-value for Amplitude

F2 ANOVA for Motor conduction velocity

P2-value for Motor conduction velocity M/S: Meter/Second

	Amplitude of radial sensory nerve action potential (μv)		Sensor veloc sen	ry conduction ity of radial sory (M/S)	ANOVA			
	Mean ± SD			Mean ±	SD	F	F1	P & P1- value
Group I N(10)	13.640 ± 0.608		46.780 ± 0.892			115.151	96.0 21	0.000*
Group II N(16)	6.318 ± 2.621	1		40.379 ± 4.068				
Group III N(16)	1.548 ± 0.526			27.455 ±	1.834			
Group IV N(18)	6.480 ± 1.248			35.989 ±	3.300			
			Tuk	ey`s test				
Amplitude	I& II	I& III	Ι	& IV	II& III	II& IV	Ι	II & IV
	0.000*	0.000*	0.000*		0.000*	0.992		0.000*
Sensory conduction velocity	0.000*	0.000*	0	.000*	0.000*	0.001*		0.000*

Table (8): ANOA	one way statistical analysis foll	lowed by tukey`s fo	or sensory actio	on potential of radial sensory
nerve in all studi	ed workers and control group			

N: number, G I: control, G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant F ANOVA for Distal motor latency P-value for Distal motor latency ms: milliseco

F1 ANOVA for Amplitude

F2 ANOVA for Motor conduction velocity

P-value for Distal motor latencyms: millisecondP1-value for Amplitudemv: millivoltP2-value for Motor conduction velocityM/S: Meter/Second

Table (9): ANOA one way statistical analysis followed by tukey's for sensory action potential of sural sensory nerve in all studied workers and control group

	Amplitude o po	of sural nerve act tential (µv)	tion Se cond velocit nerv	nsory luction y of sural re(M/S)	ANOVA			
	Ν	Iean ± SD	Mea	n ± SD	F	F1	P & P1- value	
Group I N(10)	28.450 ± 0.52	25	44.370 ±	0.834	670.539	108.292	0.000*	
Group II N(16)	7.829 ± 1.06	29 ± 1.065		1.130				
Group III N(16)	3.033 ± 0.710	3.033 ± 0.710		0.939				
Group IV N(18)	5.453 ± 2.467	7	36.457 ±	2.068				
			Tukey`s test					
Amplitude	I& II	I& III	I& IV	II& III	II& IV		I & IV	
	0.000*	0.000*	0.000*	0.000*	0.001*	ć C	.000*	
Sensory conduction velocity	0.000*	0.000*	0.000*	0.000*	0.001*	:	0.089	

N: number, G I: control, G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significantFANOVA for Distal motor latencyP-value for Distal motor latencyms: millisecond

F1 ANOVA for Amplitude

F2 ANOVA for Motor conduction velocity

P-value for Distal motor latencyms: millisecondP1-value for Amplitudemv: millivoltP2-value for Motor conduction velocityM/S: Meter/Second

Motor parameters of median nerve in workers exposed to lead			Blood lead level		
		(r)	P-value		
Distal motor latency	Group II	0.834	< 0.001*		
	Group III	0.877	< 0.001*		
	Group IV	0.661	0.003*		
	Total	0.758	< 0.001*		
Amplitude	Group II	-0.615	0.011*		
	Group III	-0.887	< 0.001*		
	Group IV	-0.864	< 0.001*		
	Total	-0.471	< 0.001*		
Motor conduction velocity	Group II	-0.330	0.212		
	Group III	-0.919	<0.001*		
	Group IV	-0.732	0.001*		
	Total	-0.741	< 0.001*		

Table (10): Spearman correlation between blood lead level and different motor parameters of the median nerve in workers exposed to lead

G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant

Table (11): Spearman correlation between blood lead level and different motor parameters of the radial nerve in workers exposed to lead

Motor parameters of rad	lial nerve	Blood lead level			
in workers exposed to	o lead	(r)	P-value		
Distal motor latency	Group II	0.632	0.009*		
	Group III	0.903	< 0.001*		
	Group IV	0.691	0.002*		
	Total	0.795	< 0.001*		
Amplitude	Group II	-0.883	< 0.001*		
	Group III	-0.966	< 0.001*		
	Group IV	-0.808	< 0.001*		
	Total	-0.747	< 0.001*		
Motor conduction velocity	Group II	-0.721	0.002*		
	Group III	-0.819	< 0.001*		
	Group IV	-0.714	0.001*		
	Total	-0.566	< 0.001*		

G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant

Table (12): Spearman correlation between blood lead level and different motor parameters of the comm	non
peroneal nerve in workers exposed to lead	

Motor parameters of common peroneal nerve in v	Blood lead level		
		(r)	P-value
Distal motor latency	Group II	0.902	< 0.001*
	Group III	0.924	< 0.001*
	Group IV	0.628	0.005*
	Total	0.679	< 0.001*
Amplitude	Group II	-0.758	0.001*
	Group III	-0.921	< 0.001*
	Group IV	-0.949	< 0.001*
	Total	-0.867	< 0.001*
Conduction velocity	Group II	-0.861	< 0.001*
	Group III	-0.989	< 0.001*
	Group IV	-0.758	< 0.001*
	Total	-0.945	< 0.001*

G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant

Sensory parameters of m	edian nerve	Blood lead level			
in workers exposed	to lead	(r)	P-value		
Amplitude	Group II	-0.710	0.002*		
	Group III	-0.971	< 0.001*		
	Group IV	-0.885	< 0.001*		
	Total	-0.803	< 0.001*		
Conduction velocity	Group II	-0.905	< 0.001*		
	Group III	-0.992	< 0.001*		
	Group IV	-0.774	< 0.001*		
	Total	-0.883	< 0.001*		

Table (13): Spearman correlation between blood lead level and different sensory parameters of the median nerve in workers exposed to lead

G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant

Table (14): Spearman correlation between blood lead level and different sensory parameters of the radial nerve in workers exposed to lead

Sensory parameters of ra	adial nerve	Blood lead level			
in workers exposed	to lead	(r)	P-value		
Amplitude	Group II	-0.928	< 0.001*		
	Group III	-0.935	< 0.001*		
	Group IV	-0.923	< 0.001*		
	Total	-0.944	< 0.001*		
Conduction velocity	Group II	-0.807	< 0.001*		
	Group III	-0.915	< 0.001*		
	Group IV	-0.947	< 0.001*		
	Total	-0.974	< 0.001*		

G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant

Table (15): Spearman	correlation	between	blood	lead	level	and	different	sensory	parameters	of sur	al nerv	e in
workers exposed to lea	d											

Sensory parameters of sural nerve in workers exposed to lead			Blood lead level		
		(r)	P-value		
Amplitude	Group II	-0.946	< 0.001*		
	Group III	-0.912	< 0.001*		
	Group IV	-0.941	< 0.001*		
	Total	-0.849	< 0.001*		
Conduction velocity	Group II	-0.922	< 0.001*		
	Group III	-0.951	< 0.001*		
	Group IV	-0.846	< 0.001*		
	Total	-0.937	<0.001*		

G II: welders, G III: battery recyclers & chargers, G IV: car painters, *: significant

Table (16): Simple regression analysis between blood lead level (Dependable variable) and duration of exposure (Independable variable)

Linear regression	Unstandard	lized Coefficients	Standardized Coefficients	Т	P-value	R ² %			
	В	Std. Error	Beta						
(Constant)	47.495	2.910		16.320	0.000	22.7%			
Duration of exposure	0.623	0.159	0.493	3.923	0.000				
Dependent Variable: blood lead level =47.498+0.6233*Duration									

	Unstandardized Coefficients		Standardized Coefficients	Т	P-value	R ² %
	В	Std. Error	Beta			
(Constant)	-6.361	8.301		-0.766	0.447	54.619%
Urine lead level	2.679	0.346	0.745	7.744	0.000	
Dependent Variable: blood lead level = -6.361 + 2.679* ULL						

Table (17): Simple regression analysis between blood lead level (Dependable variable) and urine lead level (Independable variable)



Fig (1): Compound muscle action potential (Preston & Shapiro 2007).



Fig. (2): Standard technique of motor conduction study for median nerve (Weber 1988).



Fig. (3): Standard technique of motor conduction study for radial nerve (Papanas et al., 2010).



Fig. (4): Standard technique of motor conduction study for common peroneal nerve (Weber 1988).







Fig.(6):Standard technique of sensory conduction study for radial nerve (Papanas et al., 2010).



Fig.(7):Standard technique of sensory conduction study for sural nerve (Weber 1988).



Graph (1): Correlation between blood lead level and Duration of exposure in workers exposed to lead



Graph (2): Correlation between blood lead level and urine lead level in workers exposed to lead. *: significant



Fig (8): Motor conduction study of the median nerve of a case study from welders (group II) showing delayed distal motor latency, low amplitude and slightly reduced motor conduction velocity in the compound muscle action potential in the median nerve.



Fig (9): Motor conduction study of the radial nerve of a case study from welders (group II) showing low amplitude and reduced motor conduction velocity of the radial nerve.



Fig (10): Motor conduction study of the common peroneal nerve of a case study from welders (group II) showing low amplitude and reduced motor conduction velocity of the common peroneal nerve.

DISCUSSION

Lead is a cumulative metallic poison that causes both acute and chronic intoxication. Chronic poisoning is more common and serious (Al-Rudainy 2010). Longterm lead exposure can result in lead neuropathy (Sadeghniiat-Haghighi et al., 2013). The classic form of lead neuropathy is a motor one, but sensory involvement may occur also (Thomson & Parry, 2006).

According to Gharaibeh et al., (2014), automechanic (welders, battery recyclers and chargers, and car painters) are at high risk for lead exposure so why the choice of these occupations was done in the current study. In Egypt, there are many small automechanic workshops, where workers exposed to lead by inhalation. So, this study was designed to detect subclinical neuropathies in Egyptian workers exposed to lead. In the current study, all workers did not use any protective equipment. This finding might be attributed to negligence using of protective equipments is out of mind in difficult socioeconomic level population, so the high blood and urine lead level are expected outcome. Lormphongs et al., (2003) and Centers for Disease Control and Prevention, Morbidity and Mortality (2013) found that, blood lead level in occupational workers who used cotton masks were lower than workers who never used any protective masks.

In the current study, the mean blood lead level was higher $(71.001 \pm 5.991 \ \mu g/dl)$ in battery chargers and recyclers than other groups. Blood lead level has been accepted as the most reliable biomarker for lead toxicity (Al-Rudainy, 2010). Ahmed et al., (1987)

reported that the acceptable blood lead level in Egypt is 30μ g/dl. While the acceptable blood lead level in Tahran and in America are 9.33μ g/d and 10μ g/dl respectively (Herman et al., 2007 and Sadeghniiat-Haghighi et al., 2013).

In the current study, the mean blood lead level in control group was 28.460 ± 0.825 . This high level could be attributed to many causes such as air pollution which is a serious problem in the major cities in Egypt (Ahmed et al., 1987). Eating fish from River Nile showing increased lead level from contamination by drainage of industrial discharges in it, as reported by El-Kattan et al., (2008). Also, water is a source of lead poisoning from lead water pipe (Woolf et al., 2007, El-Kattan et al., 2008 and Ismail, 2011).

In this study, there was a significant positive correlation between blood lead level and the duration of exposure in all workers exposed to lead with linear relationship. Similar result was noticed in Oman by Al-Rudainy, (2010). This result could be explained by lead is a cumulative poison, when inhaled daily; it mounts up in the tissues by time (Gulson & Salome, 2012). In addition, the most highly significant correlation with duration of exposure was group III (battery recyclers and chargers). A study done in India by Patil et al., (2006) who found that workers in battery manufactures and recyclers had the worse blood lead level and clinical manifestations. Moreovere, Gharaibeh et al., (2014) denoting that, working or living near battery factories usually associated with high blood lead concentrations.

In this study, the highest mean of urine lead level was 25.850 μ g/dL in group III (battery recyclers and chargers) with significant differences between all studied groups by Tukey's test. Furthermore, there was significant positive correlation between blood lead level and urine lead level in all workers. This was in accordance with, Moreira& Neves (2008) and Latif et al., (2013). So, urine can be used to replace blood for the assessment of occupational exposure to lead to avoid the invasive blood sampling. This result could be due to that, most of the lead absorbed into the body is excreted by the kidney (approximately 65%) (Abdel-Maaboud et al., 2005).

In the present study, the general manifestations from chronic lead poisoning represented by headache 62%, abdominal pain (60 %) and constipation (44 %). The majority of them were in group III. Dental caries was found in 8% of workers exposed to lead. Baker et al., (1979) reported that, no toxic effects could be detected below 40 µg/dl. Also, California Department of Public Health Occupational Lead Poisoning Preventive Program (2009) claimed that, the non specific symptoms "constipation and headache" appeared at the range of 40-79 µg/dl. In contrast, Gharaibeh et al., (2014) found that abdominal colic, constipation and headache starting to appear at $10 \,\mu g/dl$.

In the current study, there was only one subject in group III suffered decrease in hemoglobin level (Hb=10.6 g/dl). This result coincided with Lidsky & Schneider (2003) and Herman et al., (2007) who found that, lead can produce anemia both by interfering

with heme synthesis and by decreasing iron absorption from gut. Kwong et al., (2004) found a relationship between iron and lead. Iron deficiency anemia may increase the risk of lead poisoning.

The present study revealed no clinical abnormalities in red blood corpuscle such as basophilic stippling or reticulocytosis. Moreover, Fonte et al., (2007) and Gunturu et al., (2011) studied cases with occupational lead poisoning, high blood lead level, hemoglobin level decreases, basophilic stippling and reticulocytosis become positive. On other hand, Shobha et al., (2009) studied another cases with occupational lead poisoning with no one had basophilic stippling inspite that their blood lead level were elevated 4-12 times above normal.

Basophilic stippling represents aggregation of ribosomal RNA in the cytoplasm of the red blood cell especially in the peripheral blood smear (Valentine et al., 1976 and Phipps et al., 2012). It was considered since 1899 as a classic laboratory sign of lead poisoning, now it considered a non specific finding for lead poisoning as it found in 27 % of internal medicine patients "malignant, rheumatologic, hematologic, cardiovascular and other diseases". Also, basophilic stippling is present in a small percentage of normal people (Cheson et al., 1984).

In the present study, there were no clinical motor manifestations detected in all subjects of the studied groups. While the superficial sensation including pain, touch and temperature in group II, III and IV were decreased significantly. However, Thomson & Parry (2006) found that, both motor and sensory manifestations for lead poisoning occurred at 80 µg/dL but motor manifestation may appear above 100 µg/dL. Also, Dsouza et al., (2009) observed that, wrist drop of lead poisoning occurred when blood lead above 106.5 µg/dL. Another study had been done by Shobha et al., (2009) who found that both motor and sensory manifestations may appear even below the level of 70 μ g/dL. This result may be attributed to the effects of lead on both central and peripheral nervous systems. Central nervous system is more affected in children but peripheral nervous system is more in adult (Bellinger, 2004). Lead interferes with the ability of calcium to trigger exocytosis of neurotransmitters in neuronal cells suggesting that lead might generally target proteins involved in calcium-mediated signal transduction (Goldwin, 2001).

Sadeghniiat-Haghighi et al., (2013) found that, no significant differences in nerve conduction indices between the two sides (right and left extremities) among all participants. So this study was done in the right side only. In this study, there were significant differences between all four studied groups as regards distal motor latency, amplitude and motor conduction velocity of median, radial and common peroneal nerves. This result was parallel to that obtained by Gidlow (2004) and Thomson and Parry (2006), who found prolongation of distal motor latency values than normal in workers excessively exposed to lead.

These findings could be explained by Bleecker et al., (2005) who found that, lead directly

damages the peripheral nerves through axonal degeneration and demyelination.

The median nerve appears to be more susceptible to lead effects than the ulnar nerve and distal motor latency is considered sensitive and early indicator for motor conduction study abnormalities by axonal degeneration and segmental demylination" (Chia et al., 1996). So, the present study preferred the median nerve than the ulnar nerve.

Additionally, Bilińska et al., (2004) found lowered amplitude of motor nerves "radial and common peroneal nerves" than the control group even if there were no clinical manifestations which is called the subclinical neuropathy, which coincide with results of the current study.

In this study, the worst sensory conduction velocity for the median nerve was obvious in group III (battery recyclers and chargers). These results were in accordance with those of Chia et al., (1996) who found changes in sensory nerve parameters compared with controls with a significant difference in both groups. Bilińska et al., (2005) observed a significant reduction in the conduction velocity for the sural nerve in workers exposed to lead. This result could provide an explanation that sensory nerve action potential measures the integrity of the fastest conducting myleinated fibers Rubens et al., (2001).

The present study concluded that both blood and urine lead level and nerve conduction either sensory or motor can give a clear image about chronic lead poisoning even with absence of any clinical findings (sub-clinical neuropathy).

Recommendation

The OSHA guidelines should be followed for safety of workers health and environment.

• Workers hygiene should be improved through awareness about the nature and hazards of this work.

• The personal protective equipments should be available for workers, who must be educated about the importance of avoidances or minimization of exposure to lead.

• Pre- employment and periodic medical examination including blood and urine lead levels and nerve conduction assessments should be performed to welder, battery recycler and charger and car painter workers. This screening protects workers from developing sensory or motor nerve affection by allowing early recognition of these affected workers who must try to change their work.

• The head of workshop and industrial safety must be notified about any case detected.

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Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

Limitations

This study limited by the small sample size to the extent that it is not possible to make any strong conclusions. Prospective and controlled studies

involving larger numbers of chronic lead exposed workers involving other different types of occupational workshops in different regional area to evaluate the magnitude of the problem in Egypt are needed. Further work should include determination of blood lead level in relations to nutrition and level of trace elements such as zinc, iron, calcium and magnesium and the relationship between blood lead level and different antioxidants and if they have protective role for electrophysiological changes in workers chronically exposed to lead.

References

- Abdel-Maaboud RM, El-Attar MM, Mohamad NA, et al. (2005). Lead toxicity in some rural communities in Assiut Governorate. Assiut University Bulletin for Environmental Researches 8 (2): 325-332.
- Ahmed NS, El-Cendy KS, El-Refaie AK, et al. (1987). Assessment of lead toxicity in traffic controllers of Alexandria, Egypt, Road instructions. Archives of Environmental Health: An International Journal 42(2): 92-95.
- Al-Rudainy LA (2010): Blood lead level among Fuel Station Workers. Oman Medical Journal 25(3): 208-211.
- Baker EL, Landrigan PJ, Barbour AG, et al. (1979). Occupational lead poisoning in the United States: clinical and biochemical findings related to blood lead levels. British Journal of Industrial Medicine 36: 314-322.
- Bellinger DC. (2004). Lead. Pediatrics 113(4): 1016-1022.
- Bellinger M. (2005). Teratogen update: lead and pregnancy. Clinical and Molecular Teratology 73(6):409-420.
- Bilińska M, Brzezowska D, Koszewicz M, et al. (2004). Subclinical lead neuropathy. Pol Merkur Lekarski 17(99): 244-247.
- Bilińska M, Antonowicz-Juchniewicz J, Koszewicz M, et al. (2005). Distribution of conduction velocity in the ulnar nerve among lead exposed workers. Medycyna Pracy 56(2): 139-146.
- Bleecker ML, Ford DP, Vaughan CG, et al. (2005). Effect of lead exposure and ergonomic stressors on peripheral nerve function. Environmental Health Perspectives 113(12): 1730-1734.
- Bromberg MB. (2013). Electrodiagnostic approach to the evaluation of peripheral neuropathies. Physical Medicine and Rehabilitation Clinics of North America 24(1):153-68.
- California Department of Public Health Occupational Lead Poisoning Preventive Program (2009). Medical Guidelines for the Lead-Exposed Worker. [On line] <u>http://www.cdph.ca.gov/programs/olppp/Docu</u> <u>ments/medgdln.pdf. Updated. April 2009. Pp.</u> <u>1-11.</u>
- Centers for Disease Control and Prevention, Morbidity and Mortality Weekly Report (2013). Very high blood lead levels among adults-United States 2002-2011 62(47): 967-971.

- Cheson BD, Rom WN, Webber RC. (1984). Basophilic stippling of red blood cells: A nonspecific finding of multiple etiology. American Journal of Industrial Medicine 5(4): 327-334.
- Chia SE, Chia HP, Ong CN, et al. (1996). Cumulative blood lead levels and nerve conduction parameters. Occupational Medicine 46(1): 59-64.
- Davis BH, Bigelow NC. (1989). Flow cytometric reticulocyte quantification using thiazole orange provides clinically useful reticulocyte maturity index. Archives of Pathology & Laboratory Medicine 113:682–689
- Dsouza HS, Dsouza SA, Menezes G, et al. (2009). Evaluation and treatment of wrist drop in a patient due to lead poisoning: case report. Industrial Health 47: 677-680.
- Edward F, Krieg Jr, David W, et al. (2008). A metaanalysis of studies investigating the effects of lead exposure on nerve conduction. Archives of Toxicology 82: 531–542.
- El-Kattan YA, Abo-El Roos A. (2008). Levels of some heavy metals in River Nile water and Oreochromis niloticus fish at Mwnoufia Governorate. Egyptian Journal of Comparative Pathology & Clinical Pathology 21(1): 64-75.
- Fonte R, Agostia A, scafa F, et al. (2007). Anemia and abdominal pain due to occupational lead poisoning. Haematologica 92 (2): 13-14.
- Gharaibeh MY, Alzoubi KH, Khabour OF, et al. (2014). Lead exposure among five distinct occupational groups: A comparative study. Pakistan Journal of Pharmaceutical Sciences 27(1): 39-43.
- Gidlow DA. (2004). Lead toxicity. Occupational Medicine 54: 76-81.
- Goldwin HA. (2001). Current Opinion in Chemical Biology. The Biological Chemistry of Lead 5 (2): 223-227.
- Granadillo VA, Parra LI, Romero RA. (1994). Total chromium in whole blood, blood components, bone and urine determined by fast furnace program electrothermal atomization AAS and using neither analyte isoformation nor background correction. Analytical Chemistry 66: 3624-3631.
- Gulson P, Salome F. (2012). Distribution and Effects of Lead. Summer Hill –Australia. <u>http://www.lead.org.au/lanv3n3/lanv3n3-</u> <u>12.html</u> (June.14.2014)
- Gunturu KS, Nagarajan P, McPhedran P, et al. (2011). Ayurvedic herbal medicine and lead poisoning. Journal of Hemotology & Oncology 4(1): 51.
- Herman DS, Geraldine M, Venkatesh T. (2007). Evaluation, diagnosis and treatment of lead poisoning in a patient with occupational lead exposure: a case presentation. Journal of Occupational Medicine and Toxicology 2(7).
- Ismail AI. (2011): Study the impact of lead poisoning on children at the city of Tanta. MS in Physics, Faculty of Science, Tanta University, P: 56.
- Kwong WT, Friello P, Semba RD. (2004). Interactions between iron deficiency and lead poisoning:

epidemiology and pathogenesis. Science of the total Environment 330(1-3): 21-37.

- Latif A, Osman M, El-Tallawy HN, et al.(2013). Prevalence of lead toxicity among secondary school students in Sohag city (Upper Egypt) and its impact on cognitive functions. Egyption Journal of Neurology and Neurosurgery 24(2): 505-515.
- Lidsky TI, Schneider JS. (2003). Lead neurotoxicity in children: basic mechanisms and clinical correlates. Brain 126: 5–19.
- Lormphongs S, Miyashita K, Morioka I, et al. (2003). Lead exposure and blood lead level of workers in a battery manufacturing plant in Thiland. Industrial Health 41(4): 348-353.
- Miles JD. (2009). Median Sensory Nerve- thumb (Antidromic). Frontal Cortex Medical Software.http://frontalcortex.com/?page=help& topic=help&qid=h0. 11/01/2014.
- Moreira MF, Neves EB. (2008). Use of urine lead level as an exposure indicator and its relationship to blood lead. Cadernos De Saúde Publica 24(9): 2151-2159.
- Munoz J, Guo Y. (2011). Basophillic stiplling: a lead to the diagnosis. Blood 118 (2).
- Papanas N, Trypsianis G, Giassaks G, et al. (2010). The sural sensory/ radial motor amplitude ratio for the diagnosis of peripheral neuropathy in type 2 diabetic patients. Hippokratia 14 (3): 198-202.
- Patil AJ, Bhagwat VR, Patil JA. (2006). Effect of lead (Pb) exposure on the activity of superoxide dismutase and catalase in battery manufacturing workers (BMW) of Western Maharashtra (India) with reference to heme biosynthesis. International Journal of Environmental Research and Public Health 3(4): 329-37.
- Phipps A, Fets H, Mackenzie B. (2012). Lead poisoning due to geographia: the consumption of miniature pottery. Open Journal of Pediatrics 60-66.
- Preston DC, Shapiro BE. (2007). Basic nerve conduction studies. Electromyography and neuromuscular disorders. Clinicalelectrophysiologic correlations. 2nd edition. Elsevier Philadelphia, Pennsylvanea: 25-45.
- Ragan P, Turner T. (2009). Working to prevent lead poisoning in children: getting the lead out. Official Journal of the American Academy of physician Assistants 22(7):40-45.
- Rubens O, Logina I, Kravale I, et al. (2001). Peripheral neuropathy in chronic occupational inorganic lead exposure: a clinical and electrophysiological study. Journal of Neurology, Neurosurgery and Psychiatry 71: 200–204.
- Sadeghniiat-Haghighi K, Saraie M, Ghasemi M, et al. (2013). Assessement of peripheral neuropathy in male hospitalized patients with lead toxicity in Iran. Jornal of Reseach in Medical Sciences 18(1): 6-9.

- Shobha N, Taly AB, Sinha S, et al. (2009) Radial neuropathy due to occupational lead exposure: Phenotypic and electrophysioloical characteristics of five patients. Annals of Indian Academy of Neurology 12(2): 111-115.
- Soliman E, Kothari MJ. (2002). Diabetic neuropathy. In www.emidicine.com last update march 29.
- Thomson RM, Parry GJ. (2006). Neuropathies associated with excessive exposure to lead. Muscle Nerve 33(6): 732 -41.
- Valentine WN, Paglia DE, Fink K, et al. (1976). Association with haemolytic anemia, basoplilic stippling, erythrocyte pyrimidine 5' – nucleotidase deficiency, and intraerythrocytic accumulation of pyrimidines. The Journal of Clinical Investigation 58: 926-932.
- Vollans SR, Hasan SS. (2011): The basic science and clinical applications of neurophysiological investigation. Orthopaedics and Trauma 25(1): 7.
- Weber RJ. (1988): Motor and sensory conduction and entrapment syndroms. In Practical electromyography edited by Johnson EW. 2nd edition. Williams and Wilkins, Baltimore Hong Kong London Sydney Pp: 92-186.
- Woolf AD, Goldman R, Bellinger DC. (2007). Update on the clinical management of childhood lead poisoning. Pediatric Clincs of North America 54 (2): 271-294.
- Ye X, Wong O. (2006). Lead exposure, lead poisoning, and lead regulatory standards in China 1990– 2005. Regulatory Toxicology and Pharmacology 46 (2): 157–162.

الملخص العربي

الاكتشاف تحت الاكلينيكي لاعتلال الاعصاب الطرفية في بعض العمال المصريين المتعرضين مهنيا للرصاص (دراسه عينة احصائية)

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التسمم المزمن بالرصاص هو الأكثر شيوعا وخطورة مما يسببه من اعتلال في وظيفة العديد من أجهزة الجسم. وتحدف هذه الدراسة لتحديد كيفية اكتشاف اعتلال الاعصاب الطرفية تحت الإكلينيكية في العمال المصريين المتعرضين مهنيا للرصاص. و قد أجريت دراسة عينه إحصائيه على ستين من الأفراد الذكور المصر.يين. خمسون منهم كانوا عمالا متعرضيين مهنيا للرصاص بشكل مزمن وقد تم جمعهم من ورش العمل (عمال اللحام، إعادة تدوير البطاريات وأجهزة الشحن ودهان السيارات).

في حين تم اخذ عشرة متطوعين من الذكور الأصحاء ظاهريا لم يتعرضوا للرصاص في وظائفهم كمجموعة ضابطه. وقد خضعت جميع الحالات للفحص السريري لاعراض التسمم بالرصاص، والفحص العصبي ودراسة التوصيل العصبي للجهاز (الحسي والحركي). وقد تم تقدير مستويات الرصاص في الدم والبول لجميع المشاركين. وأجري تقييم المختبر من أجل تقييم مستوى الهيموجلوبين، وعدد الخلايا الشبكية ومستقعد التنقير. وقد اظهرت نتائج هذه الدراسة ارتفاع كل من مستويات الرصاص في الدم والبول في العاملين المتعرضين للرصاص. وكشفت هذه الدراسة وجود زيادة في طور الخفاء القاصي الحركي مع بطء وانخفاض واضح في السعة للتوصيل العصبي وذلك لكل الفئات المعرضة للرصاص. أيضا، كانت السعة بطيئة والتي تعد من اوائل التغيرات والأكثر حساسية في دراسة التوصيل العصبي وذلك لكل الفئات المعرضة للرصاص. ولقد لوحظ ارتباط كبير بين كل من مستوى الرصاص في الدم والبول ومدة التعرض للرصاص والعاير طبيعي لجميع العاملين المعرضين للرصاص. ولقد الوحظ ارتباط كبير والتي تعد من اوائل التغيرات والأكثر حساسية في دراسة التوصيل الحسبي المعلي ولذي لكل الفئات المعرضين للرصاص. ولقد لوحظ ارتباط كبير بين كل من مستوى الرصاص في الدم والبول ومدة التعرض للرصاص والمعاير المحتلفة من التوصيل العصبي. وخلصت هذه الدراسة الى التوصيل العربي تعد من اوائل التغيرات والأكثر حساسية في دراسة التوصيل الحسي الخبي المختلفة من التوصيل العصبي. وخلصت هذه الدراسة الى أن التوصيل بين كل من مستوى الرصاص في الدم والبول ومدة التعرض للرصاص والمعاير المختلفة من التوصيل العصبي. وخلصت هذه الدراسة الى أن التوصيل العصبي الحسي أو الحركي يمكن أن يعطي صورة واضحة عن التسمم بالرصاص المزمن حتى مع عدم وجود أي نتائج سريرية (الاعتلال العصبي تحت الاكلينيكي). وتوصي هذه الدراسه ب الفحص الدوري للعمال المعرضين للرصاص للكشف المبكر و بعد التعرف علي هؤالا العضري على الاكلينيكي). وتوصي معرف الموال المن المن المن علين الرصان المنا المنعاعي، والن التصررين علينا أن ننصحهم بتحسين النظافة الشخصية، واستخدام معدات الوقاية الشخصية أو مواولة تغيير عملهم وابلاغ الامن الصنعاعي بذلك.

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