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Effect of Occupational Exposure to Lead on Liver Function Parameters

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Abstract: Lead a divalent cation has long been recognized as an ubiquitous pollutant of the eco-system. Occupational exposure to lead is the most common cause of elevated blood lead levels, (BLL). Occupational exposure to lead has been associated globally with adverse health effects. This study was designed to ascertain if some occupational exposure to lead in Nigeria results in elevated blood lead levels and impairment of liver function. A total of eighty –six (86) adult Nigerians who were occupationally exposed to lead were used in the study. They consist of thirty- two (32), mechanics, thirty (30) panel beaters and twenty-four (24) mixed population comprising of petrol attendants, battery chargers and motorcycle riders grouped as "others". The control subjects, who were not in any way occupationally exposed to lead were (30) thirty in number. The BLL were assayed using atomic absorption spectrophotometer (AAS). The biochemical indices for liver function were assayed by standard methods. The results obtained show that occupationally exposed subjects had significantly elevated BLL compared to controls (Test subjects 69 .79 + 62.30µg/dl, control: 1.98 + 3.85µg/dl). The occupationally exposed subjects had significantly higher prevalence of lead toxicity, (57%), against the controls (0%; P= 0.05). The highest prevalence of lead toxicity was found in the test subjects grouped. "others". The activity of alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP) in mechanics were 48.80±24.0, 69.40±30.90 and 57.70±28.0i.U/L respectively), panel beaters, (43.80±17.50, 72.50±29.0 and 75.0±27.80i.U/L respectively) and "others", (32.80±26.20, 38.50±27.10, and31.10±17.0i.U/L respectively) and were significantly elevated compared to controls (19.0±4.90, 23.40±7.10 and25.40±11.50i.U/L respectively ; $p<0.0001$). There were no mean significant changes in the serum levels of total protein, albumin, conjugated and total bilirubin in exposed subjects compared to controls ($p>0.05$). From these findings it is concluded that occupational exposure to lead in Nigeria is associated with significant elevation of BLL, increased prevalence of lead toxicity and liver dysfunction.

Key words: Lead · Occupational exposure · Liver Function Status

INTRODUCTION

Lead a divalent metal is an ubiquitous pollutant of the eco-system. It occurs naturally in the earth crust as inorganic or organic compound [1]. It is soft malleable and grey in colour. Lead has been persistently used by man for various purposes because of its peculiar chemical properties coupled with its poor ability to conduct heat and electricity [2]. Lead is used in water pipes, cosmetic industries and as anti knock in petroleum [3]. The metal enters the environment through soil, food, water and air [4, 5]. Lead poisoning is a global health problem but it is unrecognized as such in a number of African countries. Lead poisoning as indicated by elevated blood lead levels (BLL) have been observed in the general population in some parts of Nigeria [6 and 8]. In adults, occupational exposure to lead is the most common cause of lead poisoning. This has been associated with adverse effect on the kidney, cardiovascular, haemopoietic and hepatic systems in humans [9, 10, 11]. The liver is a vital organ in the human body which is involved in detoxification and excretion of and product of metabolism glycogenesis etc. Occupational exposure to lead has been associated with abnormal liver function. There is therefore need to assess the BLL in occupationally exposed individuals and also to evaluate the effect of such BLL on the liver.

This study was aimed at determining the blood lead levels of occupationally exposed individuals (mechanics, panel beaters, others such as petrol attendants, auto electricians etc) and to assess the effect of the observed BLL on the liver function indices in these subjects.

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MATERIALS AND METHODS

Area of Study: This study was carried out in Benin City, Edo State, South-South Nigeria. The study was carried out across the three Local Government Areas in Benin City, which are; Egor, Oredo and Ikpoba-Okha.

Study Population: Eighty six (86) apparently healthy adult Nigerians (83 males and 3 females, age range 15 – 50yrs) were recruited for the study. Informed consent was obtained from them. A questionnaire was also administered to all subjects. They were all apparently healthy and must have spent at least six(6) months on the job. These occupationally exposed subjects were made up of thirty-two (32) auto-mechanics, thirty (30) panel beaters and another group of twenty four (24) subjects comprising petrol attendants (7), battery charges (3), vulcanizers (02), auto-electricians (03) spray painters (04) and auto electricians (05).

Control Subjects: This comprised thirty (30) apparently healthy Nigerians. (Twenty eight (28) males and two (2) females, age range 20 – 40yrs). They were all students of University of Benin, Benin City, Nigeria who were not in any way occupationally exposed to lead.

Sample Collection: Ten (10)ml of whole blood were obtained by venepuncture from each subject. Five (5)ml were dispensed into labeled ethylenediamine tetra-acetic acid (EDTA) containers. The remaining five (5.0)ml were put into labelled plain containers. The EDTA blood samples were used for the determination of blood lead levels (BLL) by graphic furnace atomic absorption spectroscopy. The serum obtained from the plain containers was used for the assay of liver function indices/parameters.

Serum total protein levels was determined using the Biuret method as reported by Cheesbrough, (1999) [12]. Serum albumin levels were determined using the dye-binding method (BCG) as reported by Cheesbrough (1999) [12]. Serum total and conjugated bilirubin were determined by the Evelyn and Malloy's method as described by Cheesbrough (1999) [12].

Serum alkaline phosphatase (ALP) activity was assayed by modified king and Armstrong method as described by Tietz et al, (1996) [13] and serum alanine aminostransferase (ALT) and aspartate aminotransferase (AST) activity by Reitman and Frankel (1957) [14] method using commercial kit supplied by Randox laboratories Ltd, UK.

Statistical Analysis: The results obtained were analysed using student "t" test, anova and correlation analysis.

RESULTS

The results obtained in this study shows that individuals occupationally exposed to lead have significantly elevated whole blood lead levels (BLL) when compared to controls. The prevalence of lead toxicity in occupationally exposed subjects was 57% with no toxicity found in the controls (Table 1). Amongst the occupationally exposed subjects, varying prevalence of lead toxicity were observed. The highest prevalence of lead toxicity (64%) was found in the group "others". Furthermore, in all the test subjects the serum activity of the liver enzymes ALP, ALT and AST were significantly elevated when compared to controls (Table 2). There were no significant correlation between BLL and the activities of ALT, AST and ALP in all the test subjects, (Tables 3-5). However, in the test subjects "others" AST activity showed a significant negative correlation with BLL. (Table 5). The serum concentration of total protein, albumin, conjugated and total bilirubin in test subjects were not significantly altered when compared to control subjects.

Table 1: Prevalence of lead toxicity among the study population

	Test subjects (n = 86)	Control (n = 30)	P - value
Blood lead level (BLL) µg/dl	69.79 ± 62.50	1.98 ± 3.85	P < 0.001
No. with BLL above 40µg/dl	49 (57%)	0(0%)	P < 0.05
No with BLL below 40 µg/dl	37(43%)	30(100%)	

Values represent mean ± standard deviation (S.D)

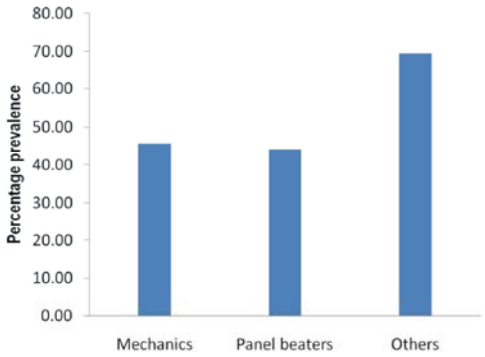

Fig. 1: Prevalence of lead toxicity in occupationally exposed subjects

Table 2: Biochemical profile of mechanics, panel beaters and others compared to urban controls

Departments	Urban control (n = 24)	Mechanics (n = 32)	Panel beaters (n = 30)	Others (n= 24)
Lead level (µg/dl)	1.98 ± 3.85[a]	57.80 ± 45.70[d]	59.40 ± 44.1[d]	69.50 ± 37.0[d]
Total protein (g/dl)	7.07 ± 0.71[a]	7.26 ± 0.56[a]	7.02 ± 0.62[a]	6.85 ±0.53[a]
Albumin (g/dl)	4.37 ± 0.92[a]	4.84 ± 0.98[a]	4.29 ± 0.92[a]	4.38 ± 0.82[a]
Total bilirubin (mg/dl)	0.79 ± 0.25[a]	0.79 ± 0.37[a]	0.98 ± 0.49[a]	0.72 ± 0.32[a]
Conjugated bilirubin (mg/dl)	0.46 ± 0.17[a]	0.44 ± 0.24[a]	0.54 ± 0.28[a]	0.39 ± 0.19[a]
Alkaline phosphatase (i.U/L)	25.40 ± 11.5[a]	57.7 ± 28.0[d]	75.0 ± 27.8[d]	31.10 ± 17.0[a]
ALT (i.U/L)	19.0 ± 4.90[a]	48.8 ± 24.0[d]	43.8 ± 17.5[d]	32.80 ±26.2[c]
AST (i.U/L)	23.4 ± 7.10[a]	68.3 ± 31.8[d]	72.5 ± 29.0[d]	38.50 ± 27.1[c]

Values represent mean ± standard deviation
Key:
a = Not significant
b = P < 0.05
c = P < 0.01
d = P < 0.001
e = P < 0.0001
Values with different superscript alphabets within the same row differ significantly (P < 0.05)

Table 3: The effect of whole blood lead levels (BLL) on liver function status in panel beaters

Parameters	Urban control (n = 30) A	Panel beaters (n = 30) B	P – value A/B	Correlation coefficient (r) with BLL
BLL (µg/dl)	1.98 ± 3.85	59.40 ± 44.10	e	
Total protein (g/dl)	7.07 ± 0.71	7.02 ± 0.62	a	-0.066[a]
Albumin (g/dl)	4.37 ± 0.92	4.29 ± 0.92	a	-0.216[a]
Total bilirubin (mg/dl)	0.79 ± 0.25	0.98 ± 0.49	a	-0.289[a]
Conjugated bilirubin (mg/dl)	0.46 ± 0.17	0.54 ± 0.28	a	-0.262[a]
Alkaline phosphatase (i.U/L)	25.40 ± 11.50	75.0 ± 27.80	e	-0.203[a]
ALT (i.U/L)	19.0 ± 4.90	43.80 ± 17.50	e	0.025[a]
AST (i.U/L)	23.40 ± 7.10	72.50 ± 29.0	e	-0.186[a]

Values represent mean ± standard deviation
Key
a = Not significant
b = P < 0.05
c = P < 0.01
d = P < 0.001
e = p < 0.0001

Table 4: The effect of whole blood lead levels (BLL) on liver function status of "others"

Parameters	Urban control (n =30) A	Others (n =24) B	P – value A/B	Correlation coefficient (r) with BLL
BLL(µg/dl)	1.98 ± 3.85	68.50 ± 36.50	e	
Total protein (g/dl)	7.07 ± 0.71	6.88 ± 0.50	a	- 0.277 [a]
Albumin (g/dl)	4.37 ± 0.92	4.38 ± 0.82	a	- 0.161 [a]
Total bilirubin (mg/dl)	0.79 ± 0.25	0.73 ± 0.32	a	0.250[a]
Conjugated bilirubin (mg/dl)	0.46 ± 0.17	0.38 ± 0.21	a	0.269[a]
Alkaline phosphatase (i.U/L)	25.40 ± 11.50	31.10 ± 17.0	a	0.119[a]
ALT (i.U/L)	19.0 ± 4.90	32.80 ± 26.20	d	- 0.347[a]
AST (.U/L)	23.40 ± 7.10	38.50 ± 27.10	d	- 0.500[b]

DISCUSSION

The results obtained in this study show that subjects who were occupationally exposed to lead have significantly higher blood lead levels when compared to control. This findings corroborates earlier reports of Ogunsola *et al*. [15], Supplido and Ong, [16], Bener *et al*. [17] and Anetor *et al*. [18]. This elevated BLL may be attributed to the presence of lead in petrol, its use in acid battery and its high content in paints sold in Nigeria [19]. The mean BLL of the test subjects (69.79 + 62.50µg/dl) is suggestive of severe plumbism which is indicated by BLL of 50 - 60µg/dl and above [20]. At this level the affected employee is expected to be removed from the source of exposure to lead. The BLL of all occupationally exposed subjects was higher than the recommended acceptable maximum limit for occupational exposure to lead of 30µg/dl for females and 40µg/dl in males [21].

The prevalence of lead toxicity in exposed subjects was 57% while none was found in the controls. Statistical analysis revealed a significantly higher prevalence of lead toxicity in occupationally exposed subjects. Similar findings have been observed by Berner *et al.* [17] in United Arab Emirate, Paul *et al.* [11] and Whitaker *et al.* [22] in United States of America. This high prevalence of lead toxicity may be due to continuous persistent use of leaded petrol in Nigeria which accounts for 20-30 % of BLL of city dwellers [15]. Another factor is lack of awareness of the effect of lead on human health by those who are occupationally exposed to the metal. Furthermore, increased absorption of lead can result in elevated BLL and lead toxicity. Mehdi *et al.* [23] reported increased absorption of lead in exposed subjects compared to unexposed controls.

The liver function was assessed using serum total protein, albumin, conjugated and total bilirubin, ALP, ALT and AST activities. The results obtained show that there was no statistically significant changes in serum total protein, albumin and total bilirubin of all lead exposed subjects compared to controls. This findings is in agreement with the earlier results of Wachukwu *et al.* [24] on experimental animals. The activity of the hepatic enzymes ALP, ALT and AST were significantly increased in lead exposed subjects compared to controls. This agrees with earlier reports of Wachukwu *et al.* [24]. These hepatic enzymes are markers of hepatocyte organelle or billary duct. They can leak into the circulation as a response to heaptocyte injury caused by reactive metabolism in the liver. Hence serum activity of these enzymes is a reflection of the physiological state of the liver and their activity in the blood depends on the severity of the cellular damage. The effect of the elevated BLL on the liver function of occupationally exposed subjects shows that in mechanics, panel beaters and others, elevated BLL was associated with hepatocellular toxicity. This findings suggest that in lead exposed subjects the metal may elicit hepatotoxicity resulting in injury/damage to the hepatic cells. This is in agreement with previous reports (Wachukwu *et al.,* 2004) [24]. The BLL however showed no significant correlation with liver function parameters in test subjects.

CONCLUSION

These findings reveal that occupational exposure to lead in Benin City, Nigeria is associated with significant elevation of BLL, lead toxicity and liver dysfunction.

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REFERENCES

1. World Health Organization, (WHO), (2001). Lead Air Quality Guidelines, 2nd edn, WHO Regional Office for Europe, Copentagem, Denmark.
2. Xinteras, C., 1992. Impact of lead contaminated soil on public health, science corner. Agency for Toxic Substance and Disease (ASDR) Registry, U.S. Department of Health and Human Services Atlanta, Georgia, pp: 1-27.
3. Goyer, R.A., 1993. Lead toxicity: current concerns. Environmental Health Perspective 100: 177-187.
4. Enuneku, A.A., 2010. Toxicological and biochemical alterations in adult amphibians *(Hoplobatrachus occipitalis* and *Bufo maculates)* exposed to cadmium and lead. Ph.D Thesis, University of Benin, Nigeria.
5. Haji, A. and H. Amir, 2012. The effect of occupational exposure to lead on blood hemoglobin concentration of workers in Kermanshah oil refinery. Iranian Journal of Toxicology, 19: 766-770.
6. Phitzner, M.A., D.T. Thacher, J.M. Pettifor, A.I. Zoakah, O. Lawson and P. Fischer, 2001. Prevalence of elevated blood lead levels in Nigerian children. Ambulatory Child Health, 6(2): 115-126.
7. Wright, N.J., D. Thachert, A. Pfitznerm, P.R. Fisher and J.M. Pettifor, 2005. Causes of lead toxicity in a Nigerian city. Archives of Disease in Childhood., 90: 262-265.
8. Ademuyiwa, O., R.N. Ugbaja, F. Idumebor and O. Adebawo, 2005. Plasma lipid profiles and risk of cardiovascular disease in occupational lead exposure in Abeokuta, Nigeria. Lipids Health Disease., 4: 19.
9. Adeniyi, T.T., G.O. Ajayi, M.A. Sado and H.J. Olopade, 2012. Vitamin C and garlic *(Allium sativum*) ameliorate nephrotoxicity and biochemical alterations induced in lead – exposed rats. Journal of Medicine and Medical Sciences, 3(5): 273-280.
10. Bartemaeus, E.S. and M.J. Jacobs, 2002. The effect of exposure to petroleum products on some renal function parameter of motor mechanics in Port-Harcourt metropolis in Nigeria. Global Journal of Pure and Applied Sciences, 9(1): 1-7.
11. Paul, M.D., B. Kristal, B.J. Estella, R. Ashtanazi and J. Ribak, 1999. Lead exposure in battery factory worker is not associated with anemia. Journal of Occupational and Environmental Medicine, 41(2): 120-123.
12. Cheesbrough, M., 1999. District Laboratory Practice in Tropical Countries. Part I. Cambridge University Press (USA) pp: 454.
13. Tietz, N.W., L.E. Pruden and A. Ole-Siggard, 1996. Electrolytes In: Fundamentals of Clinical Chemistry. Carl, A.B. and Edward E.A. (eds). Saunders W.B. (USA), pp: 497-520.
14. Reitman, S. and S. Frankel, 1957. A colorimetric method for determination of serum glutamate oxaloacetate and glutamic pyruvate transaminase. American Journal Clinical Pathology, 28: 56-58.
15. Ogunsola, O.J., A.F. Oluwole, O.I. Asubiojo, M.A. Durosinmi, A.O. Fausi and W. Ruck, 1994. Environmental impact of vehicular traffic in Nigeria: Health aspects. The Science of The Total Environment, 146/147: 114-116.
16. Suplido, M.L. and C.N. Ong, 2000. Lead exposure among small – scale battery recyclers, automobile mechanics and their children in Manilla. The Philipines Environmental Research, 82(3): 231-238.
17. Bener, A., A.M. Almehdi, R. Alwash and F.R.M. Al-Neamy, 2001. A pilot survey of blood lead level in various types of workers in the United Arab Emirates. Environmental International, 27(4): 311-314.
18. Anetor, J.I., O.O. Babalola, F.A.A. Adeniyi and T.S. Akingbola, 2002. Observations on the haemopoietic systems in tropical lead poisoning. Nigerian Journal of Physiological Sciences, 17(1-2): 9-15.
19. Adebamawo and O.A. Agbede, 2006. An evaluation of lead levels in paints for residential use sold in the Nigerian market. Indoor and Bult Environmental Sage Publications, 15(b): 551-554.
20. Gordon, J.N., A. Taylor and P.N. Bennett, 2002. Lead poisoning case studies. Clinical Pharmacology, 53: 451-458.
21. The National Institute of Occupational Safety and Health. (NIOSH), (1992). Preventing Lead Poisoning in Construction Workers, pp: 91-116.
22. Whitaker, S., 2003. Lead exposure in radiator workers. A survey of Washington state radiator repair shops and review of occupational lead exposure registry data. Journal of Occupational and Environmental Medicine, 45: 724-725.
23. Mehdi, J.K., F.J.M. Al-immariah and A.A. Al-Suhail 2000. Levels of some trace metals and related enzymes in workers at storage – battery factory, Iraq. Eastern Med. Health Journal, 6(1): 66-82.
24. Wachukwu, C.K., E.B. Dede, C.C. Ozoemena and E. Amalas, 2001. Effect of gasoline on blood cells and liver function of albino rats (*Rattus-rattus*). Journal of Medical Laboratory Sciences, 13(1): 24-27.