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HAEMATOLOGICAL ASSESSMENT OF OCCUPATIONAL EXPOSURE TO LEAD HANDLERS IN ENUGU URBAN, ENUGU STATE, NIGERIA

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ABSTRACT

Objective: To determine blood levels of lead and its effects on haematological parameters among occupational lead handlers in Enugu, Enugu State, Nigeria. In developing countries, rapid industrialisation has lead to an alarming demand for lead. Furthermore, the burden of lead toxicity is greatly underestimated. Hence, the need to assess the unavoidable toxic effects of lead as done in this study.

Methods: Blood lead levels were measured by atomic absorption spectrometry (AAS) in eighty one (81) male subjects from three manufacturing companies, all located in Enugu metropolis, Nigeria. Thirty (30) staff of the industries not directly involved in lead handling served as control group I, while twenty (20) apparently healthy individuals from within the same locality not involved in lead handling served as control group II. Haematological values, blood lead levels and blood pressure (BP) were established using standard procedures. Statistical Analysis System (SAS) software was used to analyze the results. P value of < 0.05 was taken as significant.

Results: Mean blood levels were 7.00 ± 0.07 µg/dl in test subjects; 3.00 ± 0.19 µg/dl in control group I and 2.00 ± 0.04 µg/dl in the control group II. There were significant statistical differences ($p< 0.05$ for each) in haemoglobin (Hb), packed cell volume (PCV), reticulocyte, total white blood cell (WBC), monocyte, autohaemolysis without glucose, and systolic and diastolic pressure between subjects and control group I. There were also significant differences ($p < 0.05$ for each) in the mean levels of Hb, PCV, reticulocyte, eosinophil, monocytes and systolic and diastolic pressures between the test subjects and control group II. There were however, no statistically significant differences ($p>0.05$) in the means of other parameters. Basophilic stipplings were not observed in the red cells of those directly exposed to lead.

Conclusions: It is suggested, therefore, that comprehensive and preventive measures towards exposure to lead in work places, and routine haemotological investigations be included in the bio-monitoring of the health status of lead workers.

Key Words: Occupational Exposures, Haemotological Assessment, Lead. *(Accepted 19 February 2008)*

INTRODUCTION

With global increase in high level of industrialisation, there has been an increase in the demand for lead (Pb) and lead related products.[1] The consequence of uncontrolled use of this toxic metal in pure or alloyed form among others, include pollution of the environment especially at the site of the industries, homes, etc.[1,2] Lead has been known to be toxic to most living things at high doses.[3] The burden of lead toxicity is greatly underestimated[4,5] because most of the Pb poisonings are clinically overt[2,6,7]. Furthermore, there is a paucity of information on the blood lead levels in the developing countries[8] including Nigeria[9]. The suspectedly increasing menace in our environment and its unavoidable toxic effects to the organs has provoked an assessment of the effect of Pb on the haemopoietic system.

SUBJECTS AND METHODS

Eighty-one apparently healthy male subjects and fifty controls (aged between 16 and 65 years) were recruited for this study. The duration of exposure of each subject in the job was between 6 months and 20 years and only those regularly exposed to the lead dust and fumes were considered fit for the study. Nine of the subjects who had some obvious clinical features of lead poisoning such as wrist-drop, insomnia, Burton's blue line at the gingival margine etc, were excluded.

After obtaining informed consent from each of the subjects and pre-test counselling given, 4mls of blood were obtained by venepunture. All blood counts were

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done by standard haematological methods: blood lead level was estimated by atomic absorption spectrometry (Model 306, Graphite Furnace AAS).[10] Care was taken to prevent contamination from the environment of blood specimens during sample preparation and analysis. The instrument was adjusted as follows: wavelength () of 217nm, band pass of 1.0mm, reciprocal sensitivity of 0.06 µg/dl, detection limit of 0.05 µg/dl, optimum working range 2.5, flame type air acetylene oxidation (AA)), hollow cathode lamp (HCL). Haematological parameters were investigated as described by Dacie and Lewis.[11] Statistical Analysis System (SAS) software was used to analyse the results. P value of < 0.05 was taken as significant.

RESULTS

Table I shows blood lead levels and haematological values in the different study groups. There were significant differences in the mean levels of blood lead (Pb-B) ($p<0.001$), Hb ($p<0.0001$), PVC ($p<0.05$), reticulocyte ($p<0.05$), total WBC ($p<0.0001$), monocyte ($p<0.05$) counts, percentage (%) lysis without glucose ($p<0.05$), systolic pressure ($p<0.0001$) and diastolic pressure ($p<0.001$) between subject and control group I. There were also significant differences in Pb-B levels ($p<0.05$), Hb ($p<0.001$), PCV ($p<0.05$), reticulocyte ($p<0.05$), eosinophil ($p<0.05$), monocyte ($p<0.05$) counts, systolic pressure ($p<0.0001$) and diastolic pressure ($p<0.0001$) between the subjects and control group II. There were however no significant differences ($p>0.05$) in the means of other parameters. Mean cell haemoglobin concentration (MCHC), neutrophil, lymphocyte, eosinophil, basophil counts, % lysis with added glucose (+G) between the subjects and control group I; and MCHC, total WBC, neutrophil, lymphocyte, basophil counts, % lysis (+G), % lysis without glucose (-G) between subjects and control group II ($p>0.05$). Blood films were normocytic and normochromic. No basophilic stippling was observed.

Table II represents a comparison of Pb-B levels and haematological values in test subjects and control group I with respect to duration of exposure. There were significant differences in Pb-B level ($p<0.0001$) in the range 6 10 years and MCHC ($p<0.005$), systolic and diastolic pressures ($p<0.005$) in the range 0 5 years only.

Table III describes the results of blood lead levels and haematological values with respect to age in the different study groups. There were significant differences in % lysis (+G) ($p<0.01$) and systolic pressure ($p<0.01$) between subject and control groups age range < 30 years. There were no significant differences ($p>0.05$) between subject and the control group I, within this age range. Thirty to Forty years: systolic pressure between subject and control group I, monocyte ($p>0.05$), between subject and control group II. There was no significant differences ($p>0.05$) between subject and control groups I within < 30 and > 40 years.

Table I: **Represents Blood Lead Levels (PB-B) and Haematological Values in the Different Group.**

	Subjects (N = 81)	Control groupI (N = 30)	Control groupII (N = 20)
Pb-B(µg/dl)	7.00 ± 0.07	3.00 ± 0.19*	2.00 ± 0.04*
Haemoglobin (g/dl)	12.05 ± 1.62	12.96 ± 0.089**	13.25 ± 1.01**
Packed cell volume (%)	37.97 ± 5.15	39.73 ± 0.24*	40.65 ± 3.63*
Mean cell haemoglobin concentration (g/l)	32.58 ± 1.32	32.94 ± 1.87	32.66 ± 1.44
Reticulocytes (%)	3.00 ± 1.42	2.03 ± 1.81**	2.10 ± 1.93*
White blood cell (total) (c.u.mm)	6509.67 ± 1215.67	6153.33 ± 1251.77***	6200 ± 1405**
Neutrophils (%)	52.22 ± 5.63	53.47 ± 4.92	53.60 ± 3.97
Lymphocytes (%)	45.01 ± 7.77	46.20 ± 4.52	43.55 ± 4.01
Eosinophils (%)	1.71 ± 0.90	1.93 ± 0.98	2.20 ± 1.28*
Basophils (%)	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Monocytes (%)	0.94 ± 0.74	0.63 ± 0.65*	0.50 ± 0.51*
Percentage lysis (with glucose) + G(%)	0.17 ± 0.17	0.15 ± 0.16	0.17 ± 0.20
Percentage lsysis (without glucose) – G (%)	0.74 ± 0.35	0.91 ± 0.35*	0.79 ± 0.51
Systolic psressure (mmHg)	133.57 ± 10.62	117.33 ± 10.12***	155.50 ± 11.46***
Diastolic pressure (mmHg)	82.63 ± 6.18	75.42 ± 5.61**	75.00 ± 6.070***

P Value compared to control groups: *<0.05, **<0.001, *<0.0001**

Key: ***SD:*** Standard deviation; ***N:*** Number of ssubjects; ***Pb-B:*** Serum lead concentration; ***Subject:*** Lead hsandlers (Workers)

Control groupI: Staff of the Industry not directly involved in the lead handling.Control group II:individuals not involve in lead handling in any form

Table II: **A Comparison of the Mean Pb-B Levels and Haematological Values in Subject and Control Group I With Respect To Duration of Exposure.**

	6 MONTHS – 5 YEARS		6 – 10 YEARS		> 10 YEARS	
	Subjects (N = 51)	Control group I (N= 20)	Subject (N = 20)	Control group I (N = 10)	Subject (N = 9)	Control group I
Parameters	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**
Pb-B(μg/dl)	7.70 ± 0.08	4.00 ± 0.075	6.00 ± 0.08	3.00 ± 0.05**	1.00 ± 0.01	N = 0
Haemoglobin (g/dl)	13.24 ± 0.98	13.47 ± 1.18	12.95 ± 1.87	13.64 ± 0.78	10.84 ± 2.88	N = 0
Packed cell volume (%)	40.84 ± 0.98	41 .25 ± 4.10	39.85 ± 5.50	41.00 ± 4.24	33.22 ± 10.13	N = 0
Mean cell haemoglobin csoncentration (g/l)	32.47 ± 1.48	33.72 ± 3.38*	32.46 ± 1.07	33.35 ± 2.84	32.95 ± 1.68	N = 0
Reticulocytes (%)	1.66 ± 1.89	2.325 ± 2.19	2.85	2.54 ± 2.21	1.58 ± 1.67	N = 0
White blood cell (total) (c.u.mm)	6233 ± 1508	6510 ± 1524	6040 ± 1169	6620 ± 1720	7311 ± 1495	N = 0
Neutrophils (%)	52.27 ± 5.91	52.80 ± 4.43	52.20 ± 6.73	53.60 ± 4.93	50.56 ± 4.42	N = 0
Lymphocytes (%)	45.14 ± 6.2	44.40 ± 3.56	44.45 ± 6.51	44.10 ± 3.67	45.78 ± 3.93	N = 0
Eosinophils (%)	1.63 ± 0.92	1.95 ± 0.95	2.20 ± 0.95	2.10 ± 1.52	2.00 ± 1.32	N = 0
Basophils (%)	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	N = 0
Monocytes (%)	0.84 ± 0.78	0.80 ± 0.70	1.25 ± 0.72	0.70 ± 0.95	1.22 ± 0.97	N = 0
Percentage lysis (with gslucose) + G (%)	0.22 ± 0.18	0.17 ± 0.18	0.17 ± 0.18	0.11 ± 0.13	0.12 ± 0.15	N=0
Percentage lysis (without glucose) – G (%)	0.80 ± 0.41	0.71 ± 0.55	0.72 ± 0.36	0.64 ± 0.652	0.77 ± 0.44	N = 0
Systolic pressure (mmHg)	124.4 ± 12.40	115.3 ± 8.81*	124.8 ± 14.19	123.00 ± 7.89	134.40 ± 14.88	N = 0
Diastolic pressure (mmHg)	77.75 ± 8.14	73.50 ± 5.87*	79.25 ± 7.83	74.50 ± 4.92	82.78 ± 9.72	N = 0

P Value compared to control groups: *<0.005, **<0.0001

Table IV: **Lead Concentrations in Water from Different Sources Consumed by Lead Workers and their Control GroupI in the Different Companies Studied.**

Company	Lead Concentration (μg/dl)
First	0.10(μg/dl)
Second	0.40(μg/dl)
Third	0.20(μg/dl)
Public Water Supply (WATER BOARD)	0.00(μg/dl)

Table III: **Comparison of the Mean PB-B Levels and Haematological Values With Respect To Age in the Different Study Groups.**

	<30 (Years)			30 – 40 (Years)			>40 (Years)		
	Subjects (N = 26)	Control group I(N= 5)	Control group II (N = 20)	Subject (N = 44)	Control groupI (N = 17)	Control groupII(N = 20)	Subject (N = 10)	Control groupI(N = 8)	Control
Parameters	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**N = 0**
Pb-B(μg/dl)	6.00 ± 0.08	2.00 ± 0.05	0.00 ± 0.00	5.00 ± 0.08	5.00 ± 0.07	3.00 ± 0.07	9.00 ± 0.88	1.00 ± 0.04	N = 0
Haemoglobin (g/dl)	13.04 ± 1.70	13.46 ± 1.27	13.16 ± 1.15	12.67 ± 2.19	13.5 ± 1.16	13.31 ± 0.95	12.31 ± 2.14	13.70 ± 0.82	N = 0
Packed csell volume (%)	40.81 ± 5.22	40.20 ± 4.87	40.75 ± 3.99	39.82 ± 53.83	41.53 ± 0.26	40.58 ± 3.55	37.60 ± 7.00	42.13 ± 2.85	N = 0
Mean cell hsaemoglobin csoncentration (g/l)	32.26 ± 1.04	32.50 ± 0.561	32.50 ± 1.52	40.58 ± 53.83	56.17 ± 97.06	33.35 ± 2.84	32.76 ± 0.51	32.25 ± 0.90	N = 0
Reticulocytes (%)	1.62 ± 1.62	2.08 ± 1.80	1.20 ± 0.95	1.86 ± 2.11	2.27 ± 2.21	2.20 ± 1.98	2.93 ±2.77	1.05 ± 1.61	N = 0
White blood cell (total) (c.u.mm)	6092 ± 1482	6480 ± 1890	6400 ± 1451	6307± 1211	6300 ± 1277	60667 ± 1422	7230 ± 1454	6763 ± 1497	N = 0
Neutrophils (%)	52.38 ± 5.75	53.00 ± 5.15	53.75 ± 3.50	51.45 ± 6.23	53.35 ± 5.41	53.92 ± 4.27	53.50 ± 6.64	52.13 ± 4.82	N = 0
Lymphocytes (%)	44.62 ± 5.63	44.40 ± 4.93	44.00 ± 3.74	44.45 ± 6.32	44.12 ± 5.05	43.25 4.31	42.20 ± 5.60	45.25 ± 4.57	N = 0
Eosinophils (%)	1.81 ± 0.94	2.00 ± 0.71	1.75 ± 0.46	1.89 ± 0.99	2.00 ± 1.70	2.42 ± 1.56	1.90 ± 1.29	1.88 ± 1.25	N = 0
Basophils (%)	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	N = 0
Monocytes (%)	0.92 ± 0.74	0.60 ± 0.55	0.63 ± 0.52	1.09 ± 0.77	0.77 ± 0.75	0.42 ± 0.52**	0.90 ± 0.88	0.88 ± 0.64	N = 0
Percentage lysis (with glucose) + G (%)	0.22 ± 0.10	0.12 ± 21.00	0.11 ± 0.16*	0.17 ± 0.17	0.81 ± 0.16	0.16 ± 0.21	0.21 ± 0.16	0.11 ± 0.14	N =0
Percentage lysis (without glucose) – G (%)	0.78 ± 0.44	0.86 ± 0.68	0.74 ± 0.52	0.71 ± 0.41	0.73 ± 0.50	0.73 ± 0.52	0.89 ± 0.39	0.56 ± 0.41	N = 0
Systolic pressure (mmHg)	123.7 ± 11	120.00 ± 0.00	113.8 ± 13.02*	127.40± 14.16	116.80 ± 10.73	116.8 ± 11.58*	129.0 ± 18.38	121.3 ± 6.41	N = 0
Diastolic pressure (mmHg)	78.27 ± 8.60	76.00 ± 5.48	72.50 ± 4.63	79.20 ± 9.27	75.29 ± 6.24	75.00 ± 522	76.50 ± 5.60	73.13 ± 4.58	N = 0

P Value compared to control groups: *<0.01, **<0.005

DISCUSSION

Data on blood lead concentrations and haematological parameters determined in this study provide the first population studied for lead in workers in Enugu, Eastern Nigeria, despite long-term use of lead materials in this area. This is unlike other continents where many studies had been undertaken,[4,12].

In interpreting associations between blood lead levels and some haematological values observed in this study, it is pertinent to recognise that the estimates of environmental lead exposure were not included, though better results may be obtained when evaluation of the risk of occupational exposure to lead integrate total airborne lead concentration.[13] Accordingly, differences in blood lead levels observed between the different study groups may reflect different degrees of lead exposure, variation in lead absorption or in the metabolic response to lead, or a combination of these factors. The current Centres for Disease Control (CDC) recommends a cut-off value of 10 µg/dl for diagnosing lead poisoning and a value of 20 µg/dl necessitating medical attention.[14,15]

The mean blood lead level in our test subjects (7.00µg/dl) is below the cut-off value for lead poisoning so far reported. It contrasts with 40 60 µg/dl in automobile radiator mechanics in England[4] and was greater than 60 µg/dl in battery factory workers in the United States of America.[16] However, individual Pb-B levels in this study ranged between 0 and 30 µg/dl. The high values did not show any clinical or laboratory evidence of lead poisoning.

The mean results from the test subjects (7.0 ± 0.07 µg/dl) differs significantly from that of the control group I (3.0 ± 0.009 µg/dl; $p<0.05$) and control group II (2.0 ± 0.4 µg/dl; $p<0.05$). This can be attributed to unprotected and/or excessive exposure, coupled with poor nutritional status consequent to feeding difficulties.[2] Moreover, deficiencies of iron, zinc and calcium have been reported to enhance lead absorption from the intestine.[17,18] Hence this is of grave concern in the studied areas since nutritional deficiency anaemia are common.[19,20]

In addition to the reasons given above, the first manufacturing company whose staff had the highest mean blood lead level (14.00 ± 0.09 µg/dl) were completely exposed to lead fumes and dust. Besides they have very poor ventilation and hygiene, with no medical facility. Futhermore, they used old lead battery casings for collection and storage of drinking water. This is supported by the higher water lead concentrations 0.04 parts per million (ppm) observed in them (Table IV). This is higher than the maximum permissible level (0.10ppm) recommended by the World Health Organisation (WHO) and is in contrast to the second company with well-ventilated workshops, with mean blood lead level of 5.70 ± 0.08 µg/dl.

In this second company, their workers receive at least a tin of milk at the end of the day (though some workers preferred selling theirs to consuming them). It is possible that milk consumption has a decreasing (inhibitory) effect on lead absorption.[3] Furthermore, the workers maintained healthy hygiene, which helped to decrease exposure of their family and the general population to lead.[21,22] In addition, they have good medical facilities, though they were screened for lead poisoning. Their drinking water had a lead concentration of 0.10ppm. Subjects from the third manufacturing company had the lowest levels (0.80 ± 0.03 µg/dl). This may have been due to well-ventilated and less crowded workshops. The lead concentration of drinking water was 0.20ppm, and must have contributed greatly to their low Pb-B level.

Generally, the significant higher Pb-B levels in the test subjects when compared with the control groups which was within the duration of exposure 6 10 years, is in line with other studies which confirmed that an increase in the duration of exposure is directly proportional to an increase in the Pb-B levels.[23] Strikingly, there seems to be a decrease in Pb-B level at duration greater than 10 years. This is in contrast to the results from other studies.[9] The reason may be that, most staff who have been exposed for a period of 10 years, become promoted to a rank of supervisor and consequently, no longer directly involved in lead handling. However, higher but non-significant Pb-B levels ($p>0.05$) were observed in the first and third manufacturing companies when compared with the control group I.

More recently, the most likely explanation is that, the subjects and control group I share the same source of drinking water which has been found in this study to contain high lead concentration; and in addition were mostly motor mechanics who handle gasoline and its related products which contain lead.[24]

Furthermore, higher but non-significant Pb-B levels were observed in subject in the third manufacturing company. This might be because the test subjects and control group I shared the same source of drinking water, which was most likely to be the major source of lead exposure in that locality.

Also, significantly high Pb-B levels were found in subjects when compared with the control groups, with respect to age in the three companies. This agrees with other studies, which showed that age is inversely proportional to incidence of lead poisoning.[2] In contrast, the general results with respect to age did not show significant differences ($p>0.05$) with age. It is possible that this may be due to the population size studied in each company.

Significant differences in haematological values between the test subjects and controls in the different study groups point to a relationship between haematological values, nutritional status and Pb-B levels. In addition, although these values are within the normal range in the Nigerian community,[25,26] the high lead level and normal haematological values are similar to previous findings in radiator mecanics.[4] However, the results of the present study contrasted with findings in other lead exposed workers in Denmark.[12]

Although significant decreases in Hb, PCV and MCHC levels were observed in test subjects rather than in the controls, they appear in the same range as in the lower limits for normal healthy Nigerians.[26,27] The lower Hb, PCV and MCHC levels in the test subjects than in controls can be attributed to some individual physiological variations, Pb-B levels and probably difference in feeding patterns.[9,20] Decrease in Hb concentration (anaemia) is a late feature of chronic lead poisoning, unless exposure is extreme and involves extensive haemolysis. Anaemia is usually of moderate severity, and the red cells are variable hypochromic and microcytic.[28]

The absence of basophilic stippling and polychromasia in the peripheral blood films (which are known features of lead poisoning)[9] shows that, although the lead levels were relatively high in the subjects, they had not attained toxic levels.

The safety standard for blood lead concentrations in occupationally exposed adults is <50 µg/dl.[12] Although this has generally been accepted, the results of the present study have shown that, there is need for a downward revision to <7 µg/dl. For instance, previous studies in the United States of America have shown that blood lead levels of 10 µg/dl have an effect on the red cells.[2]

There was no significant difference ($p>0.05$) between the test subjects and control groups I and II in MCHC values, all were within the normal range. This is in line with precious study in adult Nigerians.[25] However, there were significant differences ($p<0.05$) between subject and control groupII which might have resulted from changes in both Hb and PCV levels.

Inspite of a significant increase in the recticulocyte levels in test subjects more than in controls, the results could not be readily compared with any other work on lead workers as none, to the best of our knowledge, existed. However, the most probable explanation for this reticulocytosis may be due to bone marrow compensating for the decrease in red cell mass.

A slightly higher mean value for total leucocyte count was obtained in the test subjects than in the controls. This higher value might be attributed to latent infection by both bacteria and viruses, and malnutrition. The differential leucocyte count appeared within the published normal range among healthy adult Nigerians.[19,20] Lead handling/poisoning therefore appears to alter the total leucocyte counts more than the differential counts.

The results of this study show a marked statistically significant difference in both diastolic and systolic pressures between the test subjects and the controls ($p<0.0001$), though the mean and standard deviation (SD) were within the normal range. This result is in contrast to earlier studies on blood pressures (BP) and lead in a survey in Wales, which showed that there was no evidence of association between blood lead and either resting, BP or the rise in BP during cold pressure,[29,30] but agrees with the results of recent studies.[31] The most likely explanation is the interference of lead with the kallikrenkinin systems and impairment of renal function.[31,32] From this study, oral interviews of most subjects with high BP revealed that socio-economic problems were contributing factors.

From this study, Pb-B level as low as 7 µg/dl has been shown to affect haematological parameters, and blood pressure. However, these effects may be attributed to lead toxicities only after other contributing factors are excluded. This investigation has therefore highlighted several important public health issues that need urgent attention.

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