High Serum Lead Associates Low Hemoglobin among homeless Teenager Females a study in Al bashaier Camp. Sudan 2014

Zainab Basheer Ali, Abdarawouf Ahmed Abbas, Mohammad Abdalsalam Nurein, Mohammad Yousef Alhassan Benyas

Correspondence to Zainab Basheer Ali

ABSTRACT

Background: exposure to lead causes impairment of heme synthesis and affects other heme-dependent processes in the body. Effects in children generally occur at lower lead blood level than in adults.

Objectives: To determine the serum level of lead among teen ager girls in Al Bashaier camp and to correlate the serum lead with Hb level.

Methods: 30 blood samples were collected from adolescence girls of benzene sniffer at Al Bashaer camp as (study group) and 10 were collected as control group from random area of Omdurman in Al khartoum state (un sniffing) girls with the same age range (8 – 20)years. Lead was determined according to NIOSH (National Institute for Occupational Safety and Health) method. Determination of Hb concentration by the Drabikin's method. Analysis data by SPSS by computer.

Results: in the study group, the BLL showed mean of 45.53+/- SD of 20.07. (max – min. was 115-15 µg\100ml respectively). In contrast to the control group in which the mean was 87.8+/- SD of 10.7 (max – min. Was 42 - 22 µg\100ml respectively). This was more than that permitted by WHO estimation (20ugm for children and 25ugm for adults). Hb level was considered as biochemical detector for lead poisoning. Significant low levels of haemoglobin was found in the study group compared to the control group.

Conclusion: lead poisoning is a serious problem among homeless adolescence females in Sudan. It was associates low haemoglobin levels. Health education activities among both the health carers and the homeless individuals should be encouraged to minimise the risk.

Keyword: Pb, lead poisoning, BLL, Hb, benzene sniffing, homeless

INTRODUCTION:

Lead's toxicity was recognized and recorded as early as 2000 BC and the widespread use of lead has been a cause of endemic chronic plumbism in several societies throughout history. Acute and chronic lead poisoning remain problems of enormous importance for child health and development worldwide. Lead has no essential role in the human body, and lead poisoning accounts for about 0.6% of the global burden of disease.^{[1],[2]}

Lead concentration in the environments and its adverse effects on health have been concern by many organization as EPA 2003, NIOSH 1994, and JECFA which reconfirmed the existing tolerable intake of 25 µg/kg body weight per week for infants and children, and extended it to people in all age groups.

The lead uses at many industries ex: paint, to give it longer and cling to surfaces better, in battery of car and solder. Also lead is added to gasoline because of its antiknock properties. High dose of lead cause many diseases ex: damage of N.S, productive system, and damage in liver and kidneys. Also effect in brain of fetus and RBC synthesis which lead to anemia. ^{[4], [5]}

In New York at 1995, screened 518,000 children for lead poisoning which have blood lead level greater than or equal to 20 mg/dl. Also Turkey studied blood level in glue snuffer by Turk bay, sarieci su, and Akay C.1996.. Others were studied in Australian aboriginal communities, studied petrol sniffing by MacLean Sj, d, Abbas ph (1999). Also study in Navajo adolescents about gasoline sniffing and lead toxicity by coulehan jl, Hirsch W and Welty TK at (1983).^[6]

Many studied conducted in Sudan, studies by Abbas A., Mohammed R., Mozzamil Awad. 2006 studied the accumulation of lead in the Omdurman Islamic Printing press workers. Another studied by Gada. T. 1992. was Studied the inorganic lead poisoning in the Printing industry. Othman, Zuhir 2007, Babiker, Fatima 2007. ^{[7], [8], [9], [10]}

Exposure to lead has an effect on RBCs was reported by Rocke feller institute: 1924, that it changes the surface of the RBC and the permeability to water, that they shrink and are incapable of swelling much as normal cells. This concluded to cellular resistance and appear short live of cells, this demonstrated by much more rabid appearance of haemolysis than normal cells, also its effect on physical properties of RBCs is to lose their normal stickiness. ^[11], ^[12].

Lead interferes with the body's ability to make haemoglobin, it stops the pathways of Hb synthesis by inhibiting of deltaaminolevulinic acid dehydrase (ALAD) and ferrochelatase activity which catalyzes the insertion of iron into protoporphyrin (Ix). ^[13], ^[14].

This study aimed to investigate the serum level of lead among teen ager homeless girls in Al Bashaier camp and correlate it with Hb level compared to control matching group.

MATERIAL AND METHODS:

The study Site:

The study was conducted at Bashaer Camp for homeless girls in northern of Khartoum state. The camp is located in a well ventilated area and most of the houses around it are ground floors. Also the area is not jammed by heavy traffic. The camp contained six big rooms, large yards with many huge trees and number of bathrooms. The camp was sponsored by many organizations like the ICRC, red crescent and Ministry of Social Welfare. Various types of food, vegetables and fruits are always available in the camp. The average number of resident girls ranges between 200 to 350 per year.

Samples collection:

A call for participation was made among the girls in the camp. The procedure of blood sampling was explained to them all and they were set free to participate. Those who agreed to participate were selected and 5 cc of venous blood from the antecubital vein in the left forearm was collected using a sterile syringe and heparin containing tube. The number of the participants was 30. Recruitment of the matching control was set in a similar manner.

Ethical clearance and consent:

The study had been approved by the ethical committee of university. Consent was obtained from the camp admin as well as from each volunteer after explanation of the procedure.

Determination of Blood Lead Levels (BLL):

Lead extraction and measuring was determined according to the method of the National Institute for Occupational Safety and Health (NIOSH). (Number 8003):(1994). [15]

The following steps were applied:-

1. 0.2ml of blood was put in a tube

- 2. 1 ml of distilled water was added to it
- 0.5ml of ammonium byrrolidine dithiocrdumat (ABDC), 0.5ml of triton and 0.75ml of methyl isobutyl keton (MIBK) were added simultaneously.

- 4. The mixture was then shaken gently for 10 minutes
- 5. The tube was then put in a centrifuge for another 10 minutes
- 6. The serum was then collected
- read lead concentration by atomic absorption spectrophotometer model 3830
- To convert values resulted to μg/dl, the curve blotted by using the standards values as show in curve (1) and then read lead in (μg/dl). (NIOSH :1994).

Determination of Hb concentration:

- 1. prepare Drabkins solution at suitable degree of concentration.
- 2. Add 0.01 ml of blood to 2ml of Drabkin solution and shaking gently.

3) Read by photoelectric colorimeter. The colorimeter wave length was placed at 540 nm .

(4) By using the factor (6.8) and the concentration of the standard to have Hb concentration percentage should applied the following equation :

Hb = Abs (Sample) x conc [sample] x 6.8

Abs (standard)

Statistical analysis:

Data was statistically analysed by T Student and correlation and aggregation .The program which used to analysis data by SPSS.



RESULTS:

The results of the study were presented in a form of tables and figures.

Table 1: demographic data

Group	Number	Mean Age in years +/- SD	
Study group	30	15.36+/-	
		3.034	
Control	30	18.43 +/-	
group		5.06	
Total	60		

Lead level:

Table (2) shows significant rise in lead among study group versus the control group

Serum level	Control group	Study group	d.f	SE±
Lead	29.9 +/- 8.2	45.53+/- 20.07	38	0.75

(p>0.01).

Haemoglobin concentration:

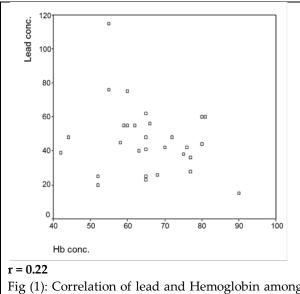
Table 3: shows significant low level of haemoglobin among study group versus control group

Serum level	Control group	Study group	d.f	SE±
Hb	87.8+/- 10.7	65.67+/11.297	38	0.58

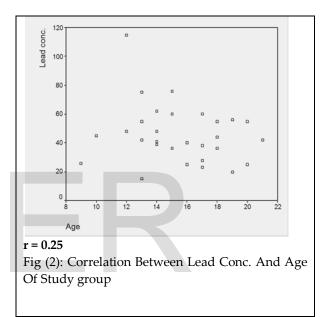
(p>0.01).



In fig (1) The study reflected that there was a negative, weak and insignificant correlationbetween Pb and Hb concentration for the studied sample,



the study group:



Discussion:

The significantly higher lead level in blood of sniffers girls in AlBashaer Camp (study group) (45.53+/-20.07) relation to that of the control groups (29.9 +/- 8.2). The study result were violated the biological standard set by NIOSH (1994) it is up to $22 \mu g/dl$, this finding due to direct gasoline sniffing by girls in the camp. This results agreement with many studies Australia study at 1999, Abbas A.(2006) which found BLL in study

Fig (1): Correlation of lead and Hemoglobin among group (mean 15.66±7.49)., Othman. Z. (2007)

Although the control group selected randomly from Omdurman in Khartoum State and don't use sniffs but there BLL is still high which may reflect air pollution with BLL which released mainly from the cars. This finding support by Ahmed (2007). ^[16] has studied the concentration of BLL from citizens of Khartoum state, mean of blood lead level was $(30.23 \pm 4.79 \ \mu\text{g/dl})$, compared to ($26.6 \pm 3.5 \ \mu\text{g/dl}$) for the control samples, compared to other countries such as USA (10 $\ \mu\text{g}$ /dl) and Germany (15 $\ \mu\text{g/dl})$, therefore , there is a high level of blood lead in population of Khartoum state due to exposure of leaded gasoline.

Significant reduction of Hb concentration among study group may due to the lead poisoning effects, which changes the surface of the RBC and the permeability to water, which it leaded to shrink, also the lead interferes with the body's ability to make haemoglobin. EPA estimated the threshold BLL for a decrease in hemoglobin to be 50 μ g/dL for occupationally exposed adults and approximately 40 μ g/dL for children, although other studies have indicated a lower threshold (*e.g.*, 25 μ g/dL) for children. ^{[17, [18]}.

This finding agree with Ahmed (2007). found that the hemoglobin concentration in study group was (69.0 \pm 8.2 %), which is lower than the control samples (79.3 \pm 7.4 %), The study has referred this decrease in hemoglobin concentration to the elevated concentrations of lead which has negative effect on hemoglobin synthesis pathway. Also agree with Babiker (2007), has studied blood lead level and haemoglobin concentration in study group form school children in three different parts in Khartoum state).

Hb synthesis can affect by high lead level, because all samples which have Hb concentration < 70% they have lead level > 25μ g/dl. but no significant relation between lead concentration and Hb concentration, this for unknown reason.

Conclusion:

This study was performed to measure the level of lead in the blood of benzene sniffers among the homeless adolescence girls at Al Bashaer camp, from the outcome of the study conclude that all sniffer samples were poisoned with high level of lead, and have significant reduction if Hb level contrast to control group but there was that no significant correlation between Hb level not and lead level because of lead metabolism in the body.

References:

[1] WHO (2009). *Global health risks: mortality and burden of disease attributable to selected major risks.* Geneva, World Health Organization.

[2] WHO .(2010). Childhood lead poisoning. World Health Organization, 2A0 Avenue Appia, 1211 Geneva 27,WHO Library Cataloguing-in-Publication Data ISBN 978 92 4 150033 3

[3] JECFA .(1993). Toxicological evaluation of certain food additives and contaminants. Geneva, Joint FAO/WHO Expert Committee on Food Additives (WHO Technical Report Series, No. 837).

[4] Auf der Heide AC, Wittmers LE Jr. :(1992). Selected aspects of the spatial distribution of lead in bone. Neurotoxicology 13:809-20

[5] Needleman, H. 1997, 'Deficits in psychologist and classroom performance of children with elevated Dentine lead levels', New England .J. of medicine.vol.300, pp. 689-695. [6] Agency for Toxic substances and Dieses Registry (ATSDR). (1992), Case studies in Environmental Medicine, lead toxicity. Public health services, Atlanta, GA U.S.A. Department of Health and Human services.

[7] Abbas A., Mohammed R., Mozzamil Awad. 2006, 'Determination of in organic lead in Omdurman Islamic University Printing Press(OIUP)', .Journal of Omdurman Islamic University, vol. 11, pp. 369-379.

[8] Ghada T. Mohammed. (1998). Inorganic lead poisoning in the printing industry. M.Sc. thesis. KhartoumUniversity.

[9] Othman, Zuhir 2007, 'Determination of Blood Lead Levels in Traffic- and Gasoline Exposed Professionals in Khartoum State- Sudan', M. S.c. thesis, O. I.U. Khartoum, Sudan, pp. 22-40.

[10] Babiker, Fatima 2007, 'Assessments of lead levels poisoning among primary schools children in

Khartoum state' M. S.c. thesis, Faculty of Basic Medical Science

[11] Baghurst PA, Robertson EF, McMichael AJ, *et al.* :(1987). The Port Pirie cohort study: lead effects on pregnancy outcome and early childhood development. Neurotoxicology, 8:395-401.

[12] Schwartz J et al. (1990). Lead-induced anemia: dose-response relationships and evidence for a threshold. *American Journal of Public Health*, 80(2):165–168.

[13] WHO .(2010). Childhood lead poisoning. World Health Organization, 20 Avenue Appia, 1211 Geneva 27,WHO Library Cataloguing-in-Publication Data ISBN 978 92 4 150033 3

[14] Agency for Toxic substances and Dieses Registry (ATSDR). (1997). Toxicological profile for lead (Update). Draft of public comment. Public health services, Atlanta, G.A : U.S. Department of Health and Human Service.

[15] National Instute for occupational and safety Health's (NIOSH), :(1994). Manual of analytical metods (NMAM).fourth edition. USA.

[16] Ahmed S. 2007, 'Determination of lead in Air and Blood samples from People in Khartoum State', M. S.c. thesis, Faculty of Basic Medical Science, Omdurman Islamic University. Khartoum, Sudan, pp. 41-43.

[17] EPA (2002). *Child-specific exposure factors handbook (interim report) 2002.* Washington, DC, National Center for Environmental Assessment

[18] US Department of Health and Human Services, Public Health Service: Toxicological Profile for Lead, Agency for Toxic Substances and Disease Registry, Atlanta, Ga, USA, 2000.

¹ WHO (2009). Global health risks: mortality and burden of disease attributable to selected major risks. Geneva, World Health Organization.

² WHO .(2010). Childhood lead poisoning. World Health Organization, 2A0 Avenue Appia, 1211 Geneva 27, WHO Library Cataloguing-in-Publication Data ISBN 978 92 4 150033 3

³ JECFA .(1993). Toxicological evaluation of certain food additives and contaminants. Geneva, Joint FAO/WHO Expert Committee on Food Additives (WHO Technical Report Series, No. 837).

⁴ Auf der Heide AC, Wittmers LE Jr. :(1992). Selected aspects of the spatial distribution of lead in bone. Neurotoxicology 13:809-20

⁵ Needleman, H. 1997, 'Deficits in psychologist and classroom performance of children with elevated Dentine lead levels', New England .J. of medicine.vol.300, pp. 689-695.

⁶ Agency for Toxic substances and Dieses Registry (ATSDR). (1992), Case studies in Environmental Medicine, lead toxicity. Public health services, Atlanta, GA U.S.A. Department of Health and Human services.

⁷ Abbas A., Mohammed R., Mozzamil Awad. 2006, 'Determination of in organic lead in Omdurman Islamic University Printing Press(OIUP)', .Journal of Omdurman Islamic University, vol. 11, pp. 369-379. ⁸ Ghada T. Mohammed. (1998). Inorganic lead poisoning in the printing industry. M.Sc. thesis. KhartoumUniversity.

⁹ Othman, Zuhir 2007, 'Determination of Blood Lead Levels in Traffic- and Gasoline Exposed Professionals in Khartoum State- Sudan', M. S.c. thesis, O. I.U. Khartoum, Sudan, pp. 22-40.

¹⁰ Babiker, Fatima 2007, 'Assessments of lead levels poisoning among primary schools children in Khartoum state' M. S.c. thesis, Faculty of Basic Medical Science

¹¹ Baghurst PA, Robertson EF, McMichael AJ, *et al.* :(1987). The Port Pirie cohort study: lead effects on pregnancy outcome and early childhood development. Neurotoxicology, 8:395-401.

¹² Schwartz J et al. (1990). Lead-induced anemia: dose-response relationships and evidence for a threshold. *American Journal of Public Health*, 80(2):165–168.

¹³ WHO .(2010). Childhood lead poisoning. World Health Organization, 20 Avenue Appia, 1211 Geneva 27, WHO Library Cataloguing-in-Publication Data ISBN 978 92
4 150033 3

¹⁴ Agency for Toxic substances and Dieses Registry (ATSDR). (1997). Toxicological profile for lead (Update). Draft of public comment. Public health services, Atlanta, G.A : U.S. Department of Health and Human Service.

¹⁵ National Instute for occupational and safety Health's (NIOSH), :(1994). Manual of analytical metods (NMAM).fourth edition. USA.

¹⁶ Ahmed S. 2007, 'Determination of lead in Air and Blood samples from People in Khartoum State', M. S.c. thesis, Faculty of Basic Medical Science, Omdurman Islamic University. Khartoum, Sudan, pp. 41-43.

¹⁷ EPA (2002). *Child-specific exposure factors handbook (interim report) 2002.* Washington, DC, National Center for Environmental Assessment

¹⁸ US Department of Health and Human Services, Public Health Service: Toxicological Profile for Lead, Agency for Toxic Substances and Disease Registry, Atlanta, Ga, USA, 2000.