

SERUM LEAD LEVELS AND ITS HEMATOLOGICAL EFFECTS IN MALE CAR PAINTERS OF LAHORE

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ABSTRACT

Background: Lead is a poisonous metal and is widely used in daily life. Increased use of lead in industry, and its excessive inhalation and ingestion can adversely affect the major biological functions in human body. This study was conducted to determine the serum lead levels and its toxic effects on hematological indices.

Material & Methods: This descriptive study was carried out in 70 male car painters working in Lahore. Blood lead concentrations was determined by atomic absorption spectrometer. Serum lead levels were observed and hematological changes were also checked. Data analysis was done by SPSS version 20.

Results: The 70 car painters were divided on basis of exposure into two groups; one with <10 and other with >10 years. The age range from 25 to 45 years with a mean of 30.41 ± 4.99 . The serum lead levels ranged from 10.40 µg/dL to 24.40 µg/dL with mean 15.34 ± 3.44 . The difference in means of the two groups was statistically insignificant ($p=0.25$). RBC count ranged from $4.07 \times 10^6/\mu\text{L}$ to $5.85 \times 10^6/\mu\text{L}$ with mean 5.00 ± 0.36 . The difference in means of two groups was statistically insignificant ($p=0.13$). Hb of car painters ranged from 9.0 g/dL to 17.40 g/dL with mean 14.34 ± 1.13 . The difference in means of the two groups was statistically insignificant (p value 0.18). HCT ranged from 36.1% to 48.40% with a mean of 41.74 ± 2.38 . The difference in means of the two groups was statistically significant ($p=0.003$). MCV ranged from 78.4fL to 97.40fL with a mean of 84.72 ± 4.10 ($p=0.51$). MCH ranged from 18.0 to 33.70 with a mean of 28.90 ± 2.18 . The difference of means in two groups was statistically insignificant (p value 0.86). MCHC of the subjects ranged from 19.7 to 36.7 with a mean of 33.56 ± 2.09 . The difference in means of MCHC in two groups was statistically insignificant ($p=0.32$). The mean values of Red Blood Cell count among the two groups was 4.951 and 5.082 respectively but were statistically insignificant with a p value of 0.139.

Conclusion: Serum lead levels were below the safety limits in all the subjects. The duration of exposure had no effects on call the hematological indices except hematocrit.

KEY WORDS: Reactive Oxygen Species; Lead; Hemoglobin.

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INTRODUCTION

Worldwide Lead is a commonly used metal due to its industrial use, yet is among the most toxic ones.¹ Lead is well known for its deleterious occupational and environmental hazards and is considered to be a ubiquitous global environmental pollutant.

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The quantity of lead consumption in the 20th century is far more than the total consumption in all previous eras, mainly because of its industrial applications.² The most common way for lead to get into soil is from exterior house lead paint. Due to natural weathering, paint dust and chips fall to the ground and abrade off the old paint causing soil contamination. Lead deposited into dust and soil remains a long-term source of lead exposure for children when they ingest the soil, play in it or when people bring soil into the house on their shoes.³ The chief source of lead contamination of drinking water is the distribution system of water. Lead based water pipes which

are in use for centuries, is the major route through which lead enters drinking water and thus contaminating it.⁴

Airborne lead comes from lead painted wood works in homes, gasoline fumes, and factory emissions. This lead, in high concentrations present in the atmosphere, settles down on to the dirt and dust particles and is inhaled, contributing to the major cause of lead exposure in individuals particularly children.⁵⁻⁸

The heavy use of lead in industries has caused high levels in water and urban polluted air causing global contamination of air, water and soil. Multiple researchers have found that lead can cause neurological, hematological, gastrointestinal, reproductive, circulatory and immunological pathologies.⁹ A high body lead level results in decreased excretion of urinary urate and is connected with clinical gout.¹⁰ It has been shown in later surveys that even below these levels some toxic manifestations may occur, mainly due to recent exposure.¹¹

Some of the lead compounds are used extensively in paints because of being colorful. Nevertheless, most of the lead found in food, paint and majority of lead containing consumer products is inorganic in nature. It is absorbed via skin in minimal amounts. Lead exposure occurs mainly through the respiratory and gastrointestinal (GI) tracts by inhalation or ingestion of lead respectively. Bloodstream absorbs roughly 30-40% of inhaled lead. The particle size of airborne lead is mostly too large to be inhaled in community environments. For this reason, inhalation of airborne lead is a major source of exposure for occupationally exposed adults but not for children.¹²

It has been shown in numerous studies that lead causes anemia by inhibiting heme and globin synthesis.¹³ Lead is well-known for its effects on different systems of human body in occupational workers, nevertheless, the effects of lead in Pakistani car painters have not been studied. For that reason we aimed to study various blood parameters in car painters because of their exposure to high leaded paints and the effect of lead on hematological system.¹⁴⁻¹⁹ The aim of this study was to ascertain the serum lead levels with various hematologic indices in car painters of Lahore.

MATERIAL AND METHODS

This descriptive study was carried out in 70 car painters from Lahore over a period of 6 months. Blood samples were taken from all the 70 patients after an informed consent. The total sample size was 70 car painters. Car painters from Lahore of age ranging between 25-50 years were included. Car painters with prolonged history of infections / illness were excluded. Blood samples were collected

from 70 car painters after an informed consent and fulfilling the inclusion and exclusion criteria, under aseptic conditions from the median cubital vein from anterior aspect of forearm. Five ml blood was obtained and divided into two tubes. The sample was transported to University of Health Sciences in a cool box containing ice bags. Hematologic indices were determined by an automated analyzer (Sysmex XT 1800i) on the same day.

Among the groups independent sample t test was applied for normally distributed parameters i.e. red blood cell count and hematocrit. Mann-Whitney test was applied for not normally distributed parameters i.e. serum lead level, Hb, MCV, MCHC and MCH. The data were entered and analyzed using SPSS 20. Mean \pm S.D was given for quantitative variables (Lead level, hematological indices and age). Distribution of variables was checked using the Shapiro-Wilk test. A p-value of ≤ 0.05 was considered to be statistically significant.

RESULTS

The 70 Car painters were divided on the basis of exposure into two groups; one with experience of < 10 years and other with experience of > 10 years. The age range of car painters was from 25 to 45 years with a mean of 30.41 ± 4.99 . The experience in years ranged from 5 to 20 years with mean 10.32 ± 4.72 .

The serum lead levels ranged from a minimum of $10.40 \mu\text{g/dL}$ to $24.40 \mu\text{g/dL}$ with mean 15.34 ± 3.44 . The mean values of serum lead levels among the two groups were 14.84 and 16.08 respectively with a p value of 0.25. (Table 1)

Regarding the hematological parameters of the two groups, the RBC count ranged from $4.07 \times 10^6/\mu\text{L}$ to $5.85 \times 10^6/\mu\text{L}$ with mean 5.00 ± 0.36 (p value 0.13). The Hb of car painters ranged from a minimum value of 9.0 g/dL to a maximum of 17.40 g/dL with mean 14.34 ± 1.13 (p value 0.18). The HCT ranged from 36.1% to 48.40% with a mean of 41.74 ± 2.38 (p value 0.003). MCV ranged from a lowest value of 78.4 fL to a highest value of 97.40 fL with a mean of 84.72 ± 4.10 (p value 0.51). MCH ranged from 18.0 to 33.70 with a mean of 28.90 ± 2.18 (p value 0.86). MCHC of the subjects ranged from 19.7 to 36.7 with a mean of 33.56 ± 2.09 (p value 0.32). (Table 2)

DISCUSSION

Lead is widely utilized in manufacturing of paints because it possesses anticorrosive and other metallic properties. Paints used for commercial and industrial purposes contain high lead content. During their work, painters are constantly exposed to these leaded paints.²⁰ Lead has been a known poisonous metal for thousands of years, and it remains a persistent environmental health threat. Exposure to

Table 1: The mean and standard deviation of variables.

Parameters	Mean	Std. Deviation
Age in years	30.41	±4.988
Experience in year	10.321	±4.725
Serum lead levels in $\mu\text{g}/\text{dL}$	15.342	±3.440
Red blood cells $\times 10^6 /\mu\text{L}$	5.003	±0.361
Hemoglobin in g/dL	14.340	±1.125
Hematocrit in %	41.735	±2.378
Mean corpuscular volume in fL	84.723	±4.104
Mean corpuscular Hb in pg	28.904	±2.179
Mean corpuscular Hb concentration	33.562	±2.089

Table 2: Comparison of hematological parameter between the group exposed to lead with <10 years and >10 years.

Parameters	Group	N	Mean value	P-value
Red blood cells $\times 10^6 /\mu\text{L}$	Less than or equal to 10 years	42	4.951	0.139
	More than 10 years	28	5.082	
Hematocrit in percentage	Less than or equal to 10 years	42	41.06	0.003
	More than 10 years	28	42.74	
Serum lead level in micrograms /deciliter	Less than or equal to 10 years	42	14.84	0.259
	More than 10 years	28	16.08	
Hemoglobin in grams /deciliter	Less than or equal to 10 years	42	14.21	0.18
	More than 10 years	28	14.52	
Mean corpuscular volume in fL	Less than equal to 10 years	42	84.59	0.513
	More than 10 years	28	84.91	
Mean corpuscular Hemoglobin in pg	Less than or equal to 10 years	42	29.00	0.86
	More than 10 years	28	28.75	
Mean corpuscular Hb concentration in g/dl	Less than or equal to 10 years	42	33.61	0.32
	More than 10 years	28	33.48	

lead can result in significant adverse health effects to multiple organ systems including the nervous, hematologic, renal, and reproductive systems. The different sources of lead and its unknown threshold of subclinical toxicity continue to make lead a matter of public health concern. Occupational exposure to lead is most often faced at battery manufacturing facilities and lead smelters, as well as in renovating houses in which workers inhale and ingest lead-contaminated fumes and dust from lead-based paint. Blood lead level ($>40 \mu\text{g}/\text{dl}$) is most often used by health care providers as an indicator of recent lead exposure.²¹

In Pakistan work regarding the lead levels in occupationally exposed individuals has not been done particularly in commercial painters. However few researches in other occupationally exposed lead workers has been done like the traffic constables

of Karachi and Islamabad. The mean age range of our subjects was 30.41 ± 4.988 years. The mean age range in traffic constables of Islamabad and Karachi was 21-45 and 20-55 years respectively, not comparable with our study. The duration of service /exposure in our subjects ranged from minimum of 5 to 20 years whereas in their study it ranged from 3 months to 18 years, not as in our study. In their study blood lead levels, Hb, copper and manganese levels were determined. The blood lead levels ranged from 7.6 -108.8 $\mu\text{g}/\text{dl}$ with a mean value $27.27 \pm 4.04 \mu\text{g}/\text{dl}$. 46% constables had lead level upto 20 $\mu\text{g}/\text{dl}$, 19% had lead level ranging from 20-25 $\mu\text{g}/\text{dl}$, 21% had levels above 25 $\mu\text{g}/\text{dl}$ and 13% above safety limit 40 $\mu\text{g}/\text{dl}$. Controls had a low mean blood lead level as compared to cases but there were no controls in our study. No significant difference was found in mean lead level at various age groups in the constables of

Islamabad, however the levels were significantly high ($p < 0.001$) at all age groups in traffic constables of Karachi. There was no correlation between lead level and length of service as compared to our study but in our study the painters hematocrit mean was 41.06 in the group with less than 10 year exposure and 42.74 in the group with more than 10 years exposure, with a p-value of 0.003 that was statistically significant. Hb in their study was found to be normal in all age groups as compared to our study.²² Muhammad et al in another study reported oxidative stress parameters and lead levels in painters of Lucknow, India in which 35 painters aged 25-50 years were selected as in our study. The mean Blood lead level (BLL < 400 micrograms) were selected from 56 male painters that were initially screened for blood lead. Controls were also taken. Association was made between low level lead exposure and antioxidant status. It was found that BLL in painters were 219.2 ± 61.9 $\mu\text{g}/\text{dl}$, approximately seven times higher than controls (30.6 ± 10.1) $\mu\text{g}/\text{dl}$. The painters exhibited a significant decrease in antioxidant enzyme catalase (56.77 ± 11.11) versus controls (230.30 ± 42.55) and SOD (0.64 ± 0.19) compared to controls (2.68 ± 0.62). Painters duration of exposure was from 5 to 10 years and in our study from 5 to 20 years but in our study lead levels were below safety limits and not significantly high as in their study. This may indicate that the paint used in India is more hazardous than the one used in Pakistan but still the quantity of white lead in lead based paints made in our industries should be measured for the better health assurance of paint users.²³ In our study it has been shown that painters are constantly exposed to lead toxicity because commercially available paints contain a high percentage of lead in it. Also, it is seen that even low level lead exposure can cause significant genotoxicity, oxidative stress and cellular level damage than affecting the blood indices, or other blood tests like liver function tests and renal function tests. For these variables to become significant in the blood a longer duration of exposure and lead levels more than safety limits are required.

CONCLUSION

Serum lead levels were below the safety limits in all the subjects. The duration of exposure had no effects on call the hematological indices except hematocrit.

REFERENCES

1. Shotyk W, Le-Roux G. Bio geochemistry and cycling of lead. *Met Ions Biol Syst* 2005; 43:239-75.
2. Xu J, Lian L, Wu C, Wang X, Fu W, Xu L. Lead induces oxidative stress, DNA damage and alteration of p53, Bax and Bcl-2 expressions in mice. *Food Chem Toxicol* 2008; 46:1488-94.
3. Mielke HW, Gonzales CR, Powell E, Jartun M, Mielke PW. Nonlinear association between soil lead and blood lead of children in metropolitan New Orleans, Louisiana: 2000-2005. *Sci Total Environ* 2007; 388:43-53.
4. Maas RP, Patch SC, Parker AF. An assessment of lead exposure potential from residential cutoff valves. *J Environ Health* 2002; 65:9-14,28.
5. Jacobs DE, Clickner RP, Zhou JY, et al. The prevalence of lead-based paint hazards in U.S. housing. *Environ Health Perspect* 2002; 110: A599-A606.
6. Lanphear BP, Hornung R, Ho M, Howard CR, Eberly S, Knauf K. Environmental lead exposure during early childhood. *J Pediatr* 2002; 140:40-7.
7. Nichan V, Li WI, Smith MA, Noonan G, Kulkarni M, Kodavor M, et al. Blood lead levels in children after phase-out of leaded gasoline in Bombay, India. *Sci Total Environ* 2006; 363:95-106.
8. Storch HV, Costa-Cabral M, Hagner C, Feser F, Pacyna J, Pacyna E, et al. Four decades of gasoline lead emissions and control policies in Europe: a retrospective assessment. *Sci Total Environ* 2003; 311:151-76.
9. Gomez JM, Vargas GG, Carrillo LL, Emma Aranda ES, Gomez A, et al. Genotoxic effects of environmental exposure to Arsenic and Lead on children in region Lagunera, Mexico. *Ann NY Acad Sci* 2008; 1140:358-67.
10. Rosin A. The Long-term Consequences of Exposure to Lead. *Isr Med Assoc J* 2009; 11:689-94.
11. Wright RO, Tsaih SW, Schwartz J, Spiro A 3rd, McDonald K, Weiss ST, et al. Lead exposure biomarkers and minimal status exam scores in older men. *Epidemiology* 2003; 14: 713-8.
12. ATSDR. Toxicological Profile for Lead. U.S. Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry, 2007.
13. Jain NB, Laden F, Guller U, Shankar A, Kazani S, Garshick E. Relation between Blood Lead Levels and Childhood Anemia in India. *Am J Epidemiol* 2005; 161: 968-73.
14. Schauder A, Avital A, Malik Z. Regulation and gene expression of heme synthesis under heavy metal exposure review. *J Environ Pathol Toxicol Oncol* 2010; 29:137-58.
15. Flora SJS, Mittal M, Mehta A. Heavy metal induced oxidative stress and its possible reversal by chelation therapy. *Indian J Med Res* 2008; 128:501-23.
16. Flora SJS, Mittal M, Mehta A. Heavy metal induced oxidative stress and its possible reversal by chelation therapy. *Indian J Med Res* 2008; 128:501-23.

17. Chiba M, Shinohara A, Matsushita K, Watanabe H, Inaba Y. Indices of lead exposure in blood and urine of lead exposed workers and concentration of major and trace element and activities of SOD, GSH-Px and catalase in their blood. *Tohoku J Exp Med* 1996; 178:49-62.
18. Valko M, Leibfritz D, Moncol J, Cronin MT, Mazur M, Telser J. Free radicals and antioxidants in normal physiological functions and human disease. *Int J Biochem Cell Biol* 2007; 39:44-84.
19. Han SG, Kim Y, Kashon ML, et al. Correlates of oxidative stress and free-radical activity in serum from asymptomatic shipyard welders. *Am J Respir Crit Care Med* 2005; 172: 1541-8.
20. Khan MI, Ahmad I, Mahdi AA, Akhtar MJ, Islam N, Ashquin M, et al. Elevated blood lead levels and cytogenetic markers in buccal epithelial cells of painters in India: genotoxicity in painters exposed to lead containing paints. *Environ Sci Pollut Res Int* 2010; 17: 1347-54.
21. Kelada SN, Shelton E, Kaufmann RB, Khoury MJ. Delta-aminolevulinic acid dehydratase genotype and lead toxicity: a HuGE review. *Am J Epidemiol* 2001; 154:1-13.
22. Agha F, Sadaruddin A, Khatoon N. Effect of Environmental lead pollution on Blood Lead Levels in Traffic Police Constables in Islamabad, Pakistan. *J Pak Med Assoc* 2005; 55:410.
23. Mohammad IK, Mahdi AA, Raviraja A, Najmul I, Iqbal A, Thuppil V. Lead-induced oxidative stress in painters. *Arh Hig Rada Toksikol* 2008; 59:161-9.

CONFLICT OF INTEREST
Authors declare no conflict of interest.
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None declared.