

Blood Lead Levels and Anemia in Lead Exposed Workers

Pages with reference to book, From 64 To 66

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Abstract

The effects of lead on haematological parameters were studied in 51 occupationally exposed individuals comprising of 27 lead furnace workers, 24 lead pellet handlers and 20 healthy age and service matched controls. Blood lead levels were estimated by atomic absorption spectrometer AAS-180-80 and haematological parameters by Technicon H.1TM system. The lead furnace workers had highest blood lead levels (median 71.20 ug/dl, range 21.2- 171.10 ug/dl) and low Hb (median 106 g/l, range 73-144 g/l) as compared to healthy subjects (median lead levels 29.80 ug/dl, range 10.20-54.10 ug/dl and Hb median 135 g/l, range 101-153 g/l). The workers handling pellets had moderately increased blood lead levels (median 45.50 ug/dl, range 8.50-130.6 ug/dl) and low Hb (median 114 g/l, range 74-158 g/l). The furnace exposed workers had higher blood lead levels and low Hb compared to the lead pellet handlers. TRBC, Hct, MCV, MCH and MCHC did not reveal any significant difference in all groups. It is concluded that chronic lead exposure causes nonnucleated normochromic anaemia and shows a dose response relationship between lead levels and severity of anemia (JPMA 45: 64, 1995).

Introduction

Lead is a cumulative toxic metal and is known for its toxicity since time immemorial. It is rapidly and efficiently absorbed from respiratory tract (nearly 90%) as well as from gastrointestinal tract (10-40%)¹. It acts at various steps interfering with the production of heme, causing anaemia. There are conflicting views on the type of anaemia encountered in chronic lead intoxication. Some believe that it is of hypochromic microcytic while others maintain that it is normocytic normochromic¹⁻³. To assess that increasing exposure to lead causes increasing anaemia, Schwartz et al⁴ conducted an epidemiological study and concluded that there is dose related depression of hematocrit in their subjects. Similarly Veemla and Noah⁵ in their study of 58 patients of lead poisoning, observed that those with lower haemoglobin values had relatively higher blood lead levels. There have been very few studies on lead intoxication in Pakistan, though millions are exposed to lead. This study was done on lead exposed workers, to evaluate the degree of their exposure to lead, by determining blood lead levels and to see the degree and type of anaemia caused by chronic lead exposure.

Material and Methods

The study was conducted from January to May, 1993 in a factory, where lead is extensively used in the manufacturing of pellets and other material. A total of 51 workers were divided into two groups according to the risk involved. A control group was also included.

High Risk- Group I

Twenty-seven workers, ages ranging 22-55 years exposed to lead fumes while working on lead melting furnaces 8-12 hours/day with no safety measures were included in this group. The duration of exposure was from into 28 years.

Moderate Risk-Group II

Twenty workers ages ranging 24-56 years, who handled lead pellets and bullets with bare hands,

without any safety precautions, were included in this group. The duration of exposure was from 5 to 35 years.

Controls

Twenty healthy, asymptomatic men from a similar factory who were not exposed to any known source of lead were in this group. Their ages ranged from 22 to 59 years and service duration from 2 to 30 years.

Blood Sampling and Tests

Subjects were sampled away from their place of work, to avoid contamination. Antecubital vein was used for standardization. Heparin was used as anticoagulant. Blood lead levels were determined on Hitachi Atomic Absorption Spectrometer-AAS-180-80 with Zeeman correction mode, using acetylene flame as atomizer. Complete blood counts were done at Armed Forces Institute of Pathology, Rawalpindi on Technicon HI System to obtain Hb, Hct, TRBC, MCV, MCH and MCHC.

Statistical Analysis

The sample size did not follow the Gaussian distribution, therefore Wilcoxon Rank Sum test was used for comparison.

Results

Both the high risk and moderate risk groups had significantly elevated blood lead levels as compared to controls (Tables I and II). Fume exposed workers (Table I) had higher blood lead levels ranging from 21.2 to 171.1 ug/dl (median 71.2), compared to 10.2 to 54.1 ug/dl (median 29.8) in controls. Significant anaemia was also seen in both exposed groups with median Hb levels of 106 g/l in high risk and 114 g/l in moderate risk, as compared to 135 g/l in control group. Anaemia was more marked in high risk group as compared to moderate risk but the difference is not statistically significant (Table II). However in all three groups when compared (Tables I, II and III), there was no difference in the values of MCV, MCH and MCHC, showing that the anaemia was of normochromic, normocytic type. When blood lead levels were correlated to haemoglobin (Figure), there was significant ($P < 0.01$) regression correlation between the two. That is, as the blood lead level increased the haemoglobin concentration fell.

Discussion

We have found a few peculiar results in our study. The control group values of blood lead concentration, haemoglobin and other parameters were within normal limits. The median blood lead level was 29.8 ug/dl, while 40 ug/dl is the WHO recommended upper acceptable limit in adult male workers⁶. However, in USA, the Centers for Disease Control in 1985 stated a level of 25 ug/dl to be considered as elevated⁷.

Anaemia (Tables I and II)

Table I. Comparison of different parameters in lead fume exposed workers with control subjects (Results are compared by applying Wilcoxon Rank Sum Test).

Parameters	Control (n=20)		Lead fume exposed workers (n=27)		P
	Median	Range	Median	Range	
Age (Years)	33.00	22.00-59.00	46.00	22.00-55.00	NS
Service (Years)	15.00	2.00-30.00	10.00	1.00-28.00	NS
Blood Lead level(ug/dl)	29.80	10.20-54.10	71.20	21.20-171.10	<0.01
Haemoglobin(g/l)	135.00	101.00-153.00	106.00	73.00-144.00	<0.01
TRBC ($10^{12}/l$)	4.67	3.35-5.23	3.69	2.49-5.00	<0.01
Hct	0.42	0.30-0.46	0.32	0.22-0.45	<0.01
MCV (fl)	88.9	85.0-96.0	85.0	81.5-108.2	NS
MCH (pg)	29.2	26.8-34.8	28.6	27.0-35.5	NS
MCHC (g/l)	328	301-352	332	318-356	NS

Table II. Comparison of different parameters in workers handling lead pellets with control subjects (Results are compared by applying Wilcoxon Rank Sum Test).

Parameters	Control (n=20)		Workers Handling lead pellets (n=24)		P
	Median	Range	Median	Range	
Age (Years)	33.00	22.00-59.00	43.00	24.00-56.00	NS
Service (Years)	15.00	2.00-30.00	16.00	5.00-35.00	NS
Blood lead level (ug/dl)	29.80	10.20-54.10	45.50	8.50-130.60	<0.01
Haemoglobin (g/l)	135.00	101.00-153.00	114.00	74.00-158.00	<0.01
TRBC ($10^{12}/l$)	4.67	3.35-5.23	3.79	1.72-5.23	<0.01
Hct	0.42	0.30-0.46	0.36	0.23-0.46	<0.01
MCV (fl)	88.9	85.0-96.0	90.2	77.8-96.0	NS
MCH (pg)	29.2	26.8-34.8	29.3	24.5-30.7	NS
MCHC (g/l)	328	301-352	329	316-338	NS

is the main finding in this study, with Hb, TRBC and Hct below the lower normal limits. This is consistent with all reported studies^{3,8,9}. Anaemia due to lead poisoning is of hypochromic, microcytic type. However, a few authors believe it to be normochromic, normocytic^{2,3}. We have found a normocytic, normochromic anaemia (Table I and II), with MCV, MCH and MCHC no different from the normal values. The causes of anaemia in chronic lead exposure are many. Lead suppresses many enzymes in the process of heme synthesis¹, decreases life span of red cells and delays regeneration of red cells¹⁰. Lead affects red cell membrane enzymes and proteins^{3,11-13} and increases hemolysis¹⁴. The microcytic anemia usually found in lead exposed individuals may be due to co-existing iron deficiency.

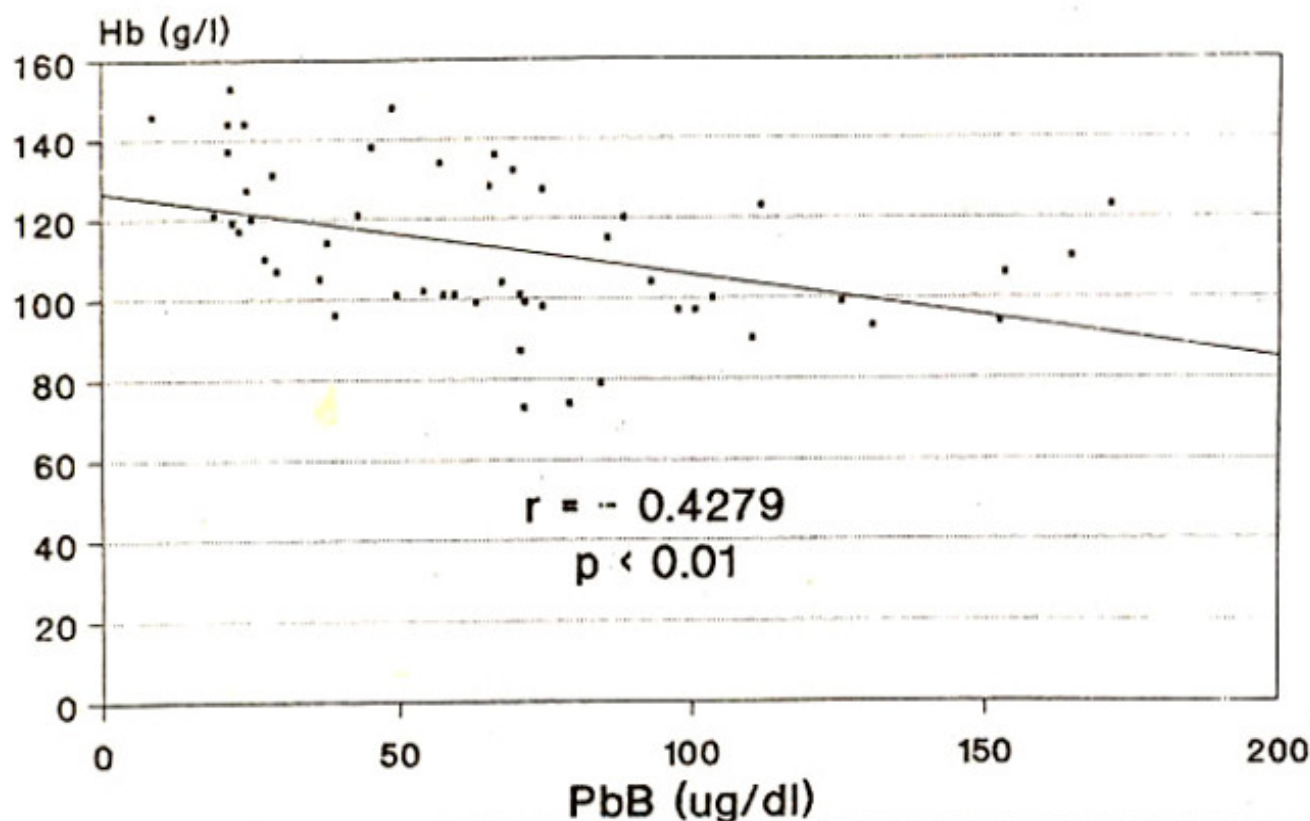


Figure. Correlation analysis between Hb and PbB in lead exposed workers.

The results shown in Figure are similar to other studies^{4,5}. A strong dose response relationship is shown between anaemia and blood lead levels. Decreasing haemoglobin and haematocrit are seen with increasing blood lead levels. However, duration of exposure to lead is an important factor in the causation of anaemia¹⁵. Acute exposure does not cause anemia, it has to be chronic. Occupational exposure to lead in Pakistan is still unchecked, causing high blood lead levels and significant anaemia

of nonnucleated normochromic type. Furthermore, there is a dose response relationship between lead levels and severity of anaemia.

References

1. Klassen, C.D. Toxicology. In: Goodman, G.A., Theodore, W.R. eds. Pharmacological basis of therapeutics. 8th ed., New York. Maxwell McMillan Int., 1991, pp.1593-8.
2. Lewis, R. Metals. In: Joseph LaDou ed. Occupational Medicine. Philadelphia, WB. Saunders Co., 1990, pp.306-11.
3. Firkin, F., Chesterman, C. Penington, D., Rush, B. eds. de gruchy's clinical haematology in medical practice. 5th ed., London, Blackwell Scientific Publications, 1989, p.210.
4. Schwartz, J., Landrigan, P.J., Baker, E.L. et al. Lead induced anemia, dose-response relationships and evidence for a threshold. *Am. J. Public Health*, 1990;80: 165.8.
5. Veerula, G.R. and Noah, P.K. Clinical manifestations of childhood lead poisoning. *J. Trop. Med. Hyg.*, 1990;93:170-7.
6. Phoon, W.H., Lee, L.S. and Ho, C.K. Biological monitoring of workers exposed to inorganic lead in Singapore. *Singapore Med. J.*, 1 990;3 1:127-30.
7. Manser, W.W.T. Plumbum! Karachi, Quo Vadis ?. Editorial. *J. Pak. Med. Assoc.*, 1988;38:227-8.
8. Debaun, M.R. and Sox, H.C. Setting the optimal erythrocyte protoporphyrin screening decision threshold for lead poisoning: A decision analytic approach. *Pediatrics*, 1991;88:121-31.
9. Hemberg, S. Lead. In Zinz C (ed). *Occupational Medicine; Principles and practical applications*. Chicago, New Year Publications, 1977, pp.673.84.
10. Grandjean, P., Jensen, B.M., Sando, S.R. et al. Delayed blood regeneration in lead exposure, an effect on reserve capacity. *Am. J. Public Health*, 1989;79: 1385.8.
11. Apostoli, P., Romeo, L., De Matteis, et al. Effects of lead on red blood cell membrane proteins. *Int. Arch. Occup. Environ. Health*, 1 988;6 1:71-5.
12. Moore, M.R., Meredith, P.A., Goldberg, A. Lead and heme synthesis, In: Singhal, R.L., Thomas J.A. eds. *Lead Toxicity*, Baltimore, Urban and Schwarzenberg, 1980, pp.79-117.
13. Ali, M.A.M. and Quinlan, A. Effect of lead on globin synthesis in vitro. *Am J Clin. Pathol.*, 1977;67:77-9.
14. Sugawara, E., Nakamura, K., Lukumura, A. et al. Uptake of lead by human red blood cells and intracellular distribution. *Kitasato Arch. Exp. Med.*, 1 990;63: 15-23.
15. Hryhorczuk, D.O., Hogan, M.M. and Katherine, M. The fall of zinc protoporphyrin levels in workers treated for chronic lead intoxication. *3. Occup. Med.*, 1985;27:816.