Medical Journal of the
Islamic Republic of Iran

Volume 10
Number 3
Fall 1375
November 1996

LEAD TOXICITY IN EMPLOYEES OF A PAINT FACTORY

MOHAMMAD ABDOLLAHI, Pharm. D., Ph.D., ALI SADEGHI MOJARAD, Pharm.D., AND NASER JALALI*, M.D.

*From the Department of Toxicology, School of Pharmacy, Tehran University of Medical Sciences, Tehran, and the *Loghman Hospital Toxicology Center, Tehran, Islamic Republic of Iran.*

ABSTRACT

Lead-based paint is an important cause of lead poisoning. This report describes our observation of excessive lead absorption among employees in a paint factory in which lead naphthenate and lead oxalate were used as drying agents in paint. In this study, blood levels of lead were compared between nineteen paint factory employees and twenty normal controls.

The nineteen paint factory employees had a mean blood lead concentration of 50.71 μg/dL which was significantly ($P<0.01$) higher than that of controls (20.44 μg/dL). There was a good correlation between blood lead levels and job tenures of the employees [r= 0.55, confidence= 95% (0.13-0.81)]. Lumbar pain (47.37%), abdominal pain (42.1%), renal complications (21%), anxiety (39.1%), nervousness (52.63%), headache (42.4%), peripheral pain (36.8%) and anemia (10.5%) were the chief complaints of these employees. The use of lead naphthenate and lead oxalate in paint is clearly associated with excessive air-borne exposure to lead. Technical and medical protective control of occupational exposures is needed in paint industries.

Keywords: Poisoning, Lead; Paint

MJIRI, Vol. 10, No. 3, 203-206, 1996.

INTRODUCTION

Lead is an ancient metal. Useful physical and chemical properties make a wide spectrum of applications possible for lead and its compounds and alloys. Thus, lead is the most widely used nonferrous metal.[1] Its toxicity to industrial workers has long been recognized. Nevertheless, intense interest continues to surround research in the toxicity of lead.[2,3] Toxic effects of lead are noted in the urinary, nervous, hematopoietic and gastrointestinal systems.[4] Hence, a sensitive analytical system with precise and accurate blood lead measurements is imperative at the lower end of lead exposure.[18] The concentration of lead in blood is one indication of recent absorption of the metal,[5] and is by far the best parameter to use in the biologic monitoring of lead. In a steady state, it reflects the concentration of lead in the rapidly exchanging soft tissue pool.[6] Usually blood samples, obtained either by venipuncture or by finger stick, can be submitted to a clinical laboratory for lead determination.[7] The World Health Organization has pondered the exposure effect and exposure-response relationships of lead and has come to the conclusion that a health-based upper reference limit for men is 1.9 μmol/L (40 μg/L) and for women of child-bearing age, 1.4 μmol/L (30 μg/dL).[6]

Inorganic lead compounds are used most widely in paint

Address for correspondence: Dr. Mohammad Abdollahi, Department of Toxicology, School of Pharmacy, Tehran University of Medical Sciences, Tehran, Islamic Republic of Iran.

and pigment industries.[6] While lead-containing paint has long been known to be a major source of lead poisoning, only a few small epidemiologic studies have attempted to assess directly the relative risk of lead poisoning due to the presence of lead in paint.[8] In order to compare blood levels of lead in paint manufacturing employees and normal controls, we undertook this study of blood lead concentrations in workers exposed to lead compounds in a paint factory.

MATERIALS AND METHODS

Materials

- Triton X-100^R (TX, an alkylphenoxypolyethoxyethanol, Sigma, USA), 5% by volume in demineralized water.

- Ammonium pyrrolidine dithiocarbamate [(1-pyrrolidine carbodithioic acid ammonium salt) (APDC), (Sigma, USA) 2% w/v in demineralized water. This solution, stored in an amber bottle in a refrigerator, remains active for more than a month. It is not necessary to prepare a fresh solution each day. If desired, APDC can be made directly in the 5% Triton-X^R solution.

-Methylisobutylketone (MIBK), water-saturated (Sigma, USA).

-Heparin, sodium salt, 0.2 µg/mL (Leo Pharmaceutical Products, Denmark).[9]

-Lead standards: stock, 1 mg/mL, prepared from reagent grade lead nitrate and demineralized water. The concentrated standard is stable indefinitely.[10]

Method

This study was performed on nineteen male paint industry employees, aged between 26 and 60 years. Job tenures of the employees ranged between three and 23 years. A group of 20 healthy men were used as normal controls who had not worked in a paint factory. A questionnaire about symptoms of lead poisoning was prepared and all persons were asked about their presence.

Whole blood samples were taken by venipuncture in a tube containing sodium heparin as anticoagulant. The samples were stored in a refrigerator as soon as possible until the analysis. Standards were made up in demineralized water. In each of seven 16/130 mm borosilicate test tubes, 10 mL of demineralized water was placed. Tubes 2-7 were treated respectively with 3, 5, 7.5, 10, 15 and 20 µL of stock solution, and the contents thoroughly mixed on a vortex mixer. These standards represented 30, 50, 75, 100, 150 and 200 µg/dL of lead in demineralized water. Tube 1 was the reagent blank. In tubes 1-7, all of the blood lead extraction steps were performed. Into other tubes was pipetted 5 mL of unknown blood. 1 mL of 5% Triton-X^R solution was added to each tube (if this solution already contained APDC, contents were mixed and MIBK added). After mixing on the vortex mixer (rapid hemolysis), 1 mL of the APDC (ammonium pyrrolidine dithiocarbamate) solution was added to each tube, followed again by vortex mixing. Finally, 5 mL of water-saturated MIBK was pipetted into each tube. The tubes were sealed with rubber stoppers, shaken vigorously about 60 times, and centrifuged for 20 minutes at about 3000 rpm. Phase separation was clean and sharp, and the organic supernatant was aspirated directly from the tubes.[10] Unknown samples' concentrations were calculated from standard curves.

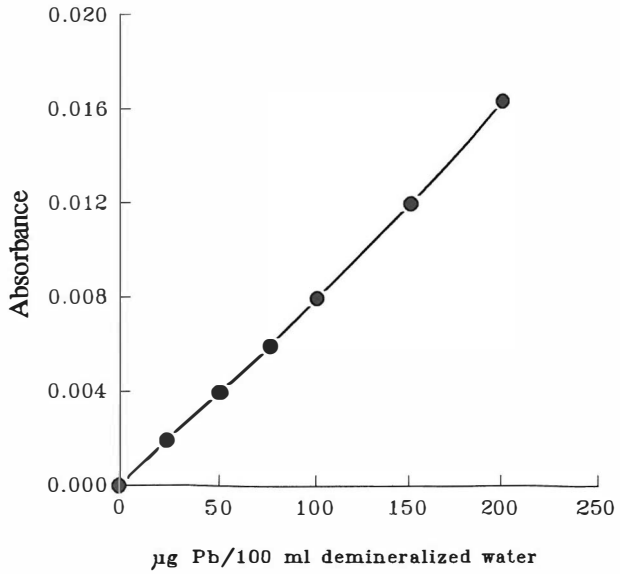

Fig. 1. A typical standard curve for lead.

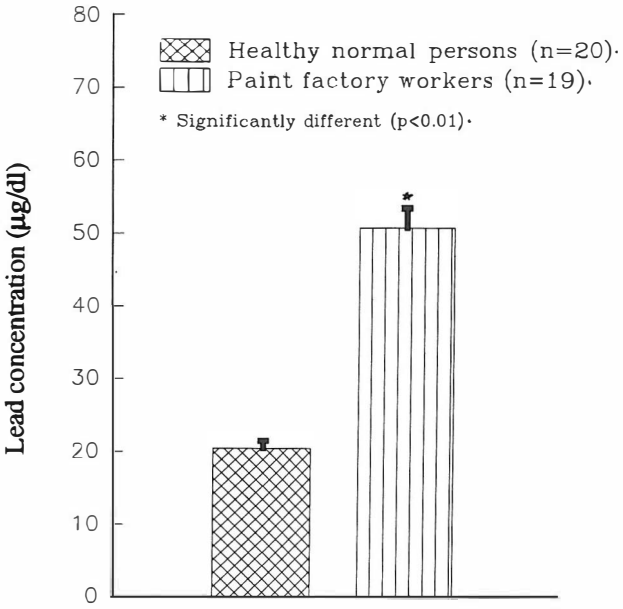

Fig. 2. Comparison between blood lead levels of paint workers and normal controls. Data are expressed as mean ± SE.

Patients were questioned for any previous urine or blood analysis which were noted in their records. Aminoaciduria,

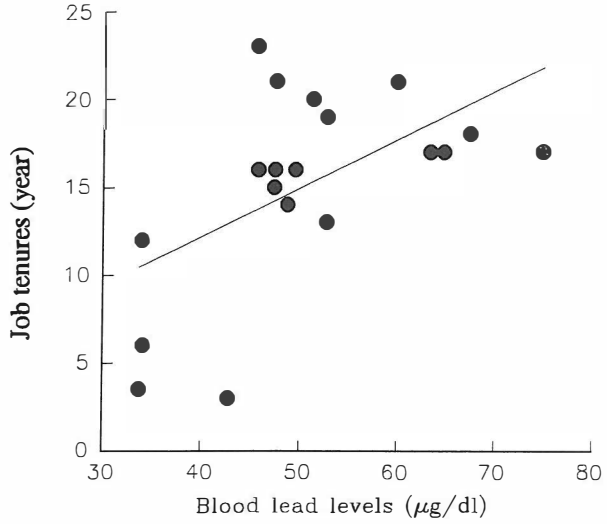

Fig. 3. Job tenures and blood lead levels (μg/dl) of workers in a paint factory (r = 0.55).

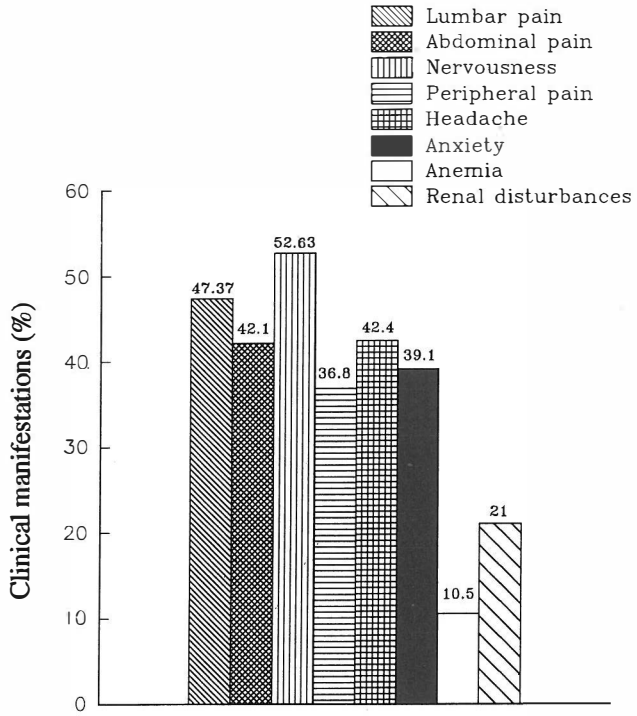

Fig. 4. Percentage of workers' complaints of some symptoms of lead poisoning in a paint factory.

glycosuria, proteinuria and phosphaturia were considered for renal dysfunction.[4,20] Decrease in hematocrit was recorded for anemia determination.[2]

A Shimadzo AA-680 atomic absorption flame emission spectrophotometer was used. The hollow cathode lead lamp was operated at the recommended level of 5 mA. Analyses were performed at 283.3 nm, primarily because of the favorable signal-to-noise ratio, as compared to the 217 nm line. Other settings were as follows: slit 1, scale expansion 3, chart speed 1, flame (Air-C_2H_2), fuel 1.8 L/min, oxidant 8 L/min. Lifting of the flame from the burner head during the adjustments can be remedied by aspirating air for a few seconds.[10] Student's t-test was used to evaluate the differences between control and test group data. Differences with $P \leq 0.05$ were considered significant.

RESULTS

Fig. 1 shows a typical standard curve. In Fig. 2 the blood lead levels (BLLs) of workers and normal controls are compared. The workers had a mean blood lead concentration of 50.71±2.61 μg/dL which was significantly ($P<0.01$) higher than that of controls (20.44±1.06 μg/dL). As shown in this figure, 84.2% of the workers had blood lead concentrations in excess of 40 μg/dL with the highest concentration being 75 μg/dL.

In Fig. 3, the relationship between BLLs and job tenures of the workers is shown. As shown in this figure, there is a direct and imperfect correlation (r=0.55) between blood lead levels and job tenures of the workers, with a confidence interval between 0.12-0.81 (confidence 95%).

The percent of clinical manifestations in these workers is shown in Fig. 4. As shown in this figure, lumbar pain (47.37%), abdominal pain (42.1%), nervousness (52.63%), peripheral pain (36.8%), headache (42.4%) and anxiety (39.1%) were the most common symptoms.

DISCUSSION

In order to evaluate health risks in environmental and occupational exposures, the analysis for trace elements, e.g. lead, in biological fluids, is essential.[19] Atomic absorption spectrophotometry (AAS) is one of the most widely used methods.[11] Lead is best determined by atomic absorption spectrophotometry.[9] This method produces a sensitive, practically noise-free determination with a working curve that is linear at least to 250 μg/dL blood or demineralized water. Blood and aqueous standards (diluted with water) assayed by the flame-AAS demonstrate similar results.[10] In this study aqueous standards were used. The usual cause of poisoning in adults is industrial exposure to inorganic or organic lead compounds.[9] There was a significant difference between blood lead levels of workers and normal controls ($P<0.01$). At a high enough level of lead exposure, i.e. >40 μg/dL virtually all body systems will be injured or have a high risk of injury.[2] Sixteen cases were above this limit in our group, which corresponds to 84.2%. The workers used no respiratory protection, and there was no local exhaust ventilation in the work area. In the present survey, the men worked 42 hours a week. The relationship between BLLs and job tenures of the workers was direct and imper-

fect, because of the existence of abundant ways of lead pollution that prevents an exact survey. Lead absorption may be influenced not only by lead exposure but also by such factors as the subject's respiratory minute volume and the particle size and availability of the lead dust to which he is exposed.[12] Blood lead concentrations can be related to many factors, such as age, smoking and drinking habits.[13]

Symptoms of chronic lead poisoning can be divided into six categories: gastrointestinal, neuromuscular, CNS, hematological, renal and others. The abdominal syndrome is a more common manifestation of a very slowly and insidiously developing poisoning.[5] Symptoms of chronic lead poisoning often begin with general malaise, nausea and vomiting, colicky abdominal pain, constipation,[15] vertigo, fatigue and headache.[16] The main complaints in these employees were lumbar pain (47.37%), nervousness (52.63%), abdominal pain (42.1%), headache (42.4%), peripheral pain (36.8%), anxiety (39.1%), renal complications (21%) and anemia (10.5%). Other symptoms of lead poisoning such as anorexia, malaise, constipation, nausea and vomiting were not mentioned. BLLs, which can be easily monitored, can be reduced by chelation treatments, but the relatively large amounts of lead stored in the bone are subsequently mobilized slowly to raise BLLs even in individuals returned to a lead-free environment.[17] Though lead poisoning can be treated with chelating agents, it appears that prevention of lead toxicity should be the primary goal.[1] A short recapitulation of the main principles follows:

a- Substitution of lead by a less toxic substance.

b- Use of disposable coveralls, shoe covers and gloves.

c- A good quality filter mask is required.

d- There should be no eating, drinking or smoking in the work area.

e- Ventilation is required where there is any chance of lead being vaporized.[6,14,20]

REFERENCES

1. Grandjean PH: Widening perspectives of lead toxicity. Environ Res 17: 303-321, 1978.
2. Landrigan PJ: Toxicity of lead at low dose. Br J Ind Med 46: 593-596, 1989.
3. Landrigan PJ: Current issues in the epidemiology and toxicology of occupational exposure to lead. Toxicol Ind Health 7: 9-14, 1991.
4. Nolan CV, Shaikh ZA: Lead nephrotoxicity and associated disorders: biochemical mechanisms. Toxicology 73: 127-146, 1992.
5. Klaassen CD: Heavy metals and heavy metal antagonists. In: Gilman AG, Rall TW, Nies AS, Taylor P (eds.), The Pharmacological Basis of Therapeutics. 8th ed, New York: MacMillan, pp. 1593-1598, 1990.
6. Herberg S: Lead and its compounds. In: Zenc C (ed), Occupational Medicine—Principles and Practical Applications. 2nd ed, Chicago: Year Book Medical Publishers, pp. 548-549, 1975.
7. Parsons PJ: Monitoring human exposure to lead: an assessment of current laboratory performance for the determination of blood lead. Environ Res 57: 149-162, 1992.
8. Schwartz J, Levin R: The risk of lead toxicity in homes with lead paint hazard. Environ Res 54: 1-7, 1991.
9. Tietz NW: Fundamentals of Clinical Chemistry. 3rd ed, Philadelphia: W.B. Saunders Company, pp. 270, 892-894, 1987.
10. Hessel DW: A simple and rapid quantitative determination of lead in blood. Atomic Abs News (letters) 7: 55-56, 1968.
11. Yeoman WB: Metals and Anions. In: Moffat AC (ed), Clark's Isolation and Identification of Drugs. 2nd ed, London: The Pharmaceutical Press, p. 56, 1986.
12. Williams MK, King E, Walford J: An investigation of lead absorption in an electric accumulator factory with the use of personal samplers. Br J Ind Med 26: 202-216, 1969.
13. Schuhmacher M, Domingo JL, Liobet JM, Corbella J: Lead concentration and δ-aminolevulinic acid dehydratase activity in the blood of the general population of Tarragona province, Spain. The Science of the Total Environ 116: 253-259, 1992.
14. Scott D: Lead intoxication: controlling lead exposure in removal of old paint. Postgraduate Med 74(3): 92-93, 1983.
15. Graham JA, Maxton DG, Twort CH: Painters palsy: a difficult case of lead poisoning. Lancet 2 (8256): 1159-60, 1981.
16. Grandjean PH: Chronic lead poisoning treated with dimercaptosuccinic acid. Pharmacol Toxicol 68: 266-269, 1991.
17. Jones M, Basinger MA, Gale GR, Atkins LM, Smith AB, Stone A: Effect of chelate treatments on kidney, bone and brain lead levels of lead-intoxicated mice. Toxicol 89: 91-100; 1994.
18. Wang ST, Demshar HP: Determination of blood lead in dried-spot specimens by Zeeman-effect background corrected atomic absorption spectrometry. Analyst 117: 959-961, 1992.
19. Tomas J, Anglor B, Christensen M: Comparative study of certified materials and quality control materials for the quality assurance of blood lead determination. Analyst 117: 419-424, 1992.
20. Garrettson LK: Lead. In: Haddad LM, Winchester JF (eds), Clinical Management of Poisoning and Drug Overdose. 2nd ed, Philadelphia: W.B. Saunders Co., pp. 1017-1023, 1992.