

# Screening for Chronic Lead Poisoning in Lead Factory Workers

Pages with reference to book, From 239 To 241

Dilshad Ahmad Khan ( Armed Forces institute of Pathology, Rawalpindi. )

Iftikhar Ahmed Malik, Muhammad Saleem, Rizwan Hashim, Rashid Bashir ( Armed Forces Institute of Pathology, Rawalpindi. )

## Abstract

One hundred and forty-nine lead factory workers comprising of 46 fume exposed, 78 handling lead materials and 25 controls were screened for chronic lead poisoning. Blood lead level was determined by atomic absorption spectrometry and urinary ALA by ion-exchange chromatography. Fume exposed workers had significantly higher ( $P < 0.01$ ) blood lead (median 61.20 ug/dl, range 21.20 - 171.10 ug/dl) and urinary ALA levels (median 410 mg/I, range 01.0-22.9 mg/I) than workers handling lead materials and controls. Urinary ALA was found to be a more sensitive and specific test for lead poisoning than estimation of blood lead levels (JPMA 44:239,1994).

## Introduction

Lead is cumulatively toxic and one of the serious metallic poisons<sup>1</sup>. Its toxicity in occupationally exposed individuals is well recognized in smelting operations in lead furnaces, battery repairing, ship breaking, glass manufacturing, printing, painting and radiator repairing. Various studies carried out in these vocations showed lead toxicity in the range of 39%-47%<sup>2-5</sup>. Lead inhibits the heme synthetic pathway by affecting 5-amino levulinic acid (ALA) dehydrase enzyme and increasing ALA levels in plasma to the extent that ALA starts to get excreted in urine. Urinary ALA is used for screening of lead poisoning in exposed worker<sup>6</sup> and has been found to correlate with serum lead levels<sup>7</sup>. Persistent increase in urinary ALA 5-amino levulinic acid due to lead exposure is also advantageous for testing. As lead leaves the blood to be stored in the skeletal system, a random blood sample may show a misleading normal level. In contrast urinary ALA remains increased for longer duration in lead exposed worker<sup>8</sup>. The condition of lead exposed industrial worker in Pakistan is worse due to ignorance, lack of safety measures, poor industrial hygiene and deficient resources. This study was planned to screen for chronic lead poisoning in different groups of adult males exposed to lead fumes and lead material in workers of a factory using lead metal.

## Patients and Methods

The study was carried out from July, to December, 1993 at the Armed Forces Institute of Pathology, Rawalpindi. A total of 149 subjects aged 18-59 years were included in the study. They were grouped in three categories.

1. Lead Fume Exposed: Forty six lead furnace workers of a factory were studied. They were exposed to lead fumes released from a smelting operation. Work duration was 8-12 hours per day with the length of service ranging from 2 to 38 years. These were further sub-grouped into less than 10 years and more than 10 years of service.
2. Lead Handlers: Seventy eight workers of a factory handling lead pellets who were not exposed to fumes were included. Their length of service varied from 2-28 years. These were further sub grouped to less than 10 or more than 10 years of service.
3. Controls: Twenty five healthy, age and service matched men not exposed to any known source of lead were also included in this study.

A) Blood: Ten ml blood was collected for lead estimation in heparinized tubes from each individual away from their place of work. Blood lead levels were estimated by Hitachi AAS-180-80 atomic absorption spectrometer with Zeeman correction mode using acetylene flame as atomizer<sup>9</sup>.

B) Urine: Ten ml of mid-thy urine samples were collected in amber bottles, containing 0.1 ml glacial acetic acid. The urine samples were stored in refrigerator at 4°C. Urinary ALA estimation was done using ALA kit (Boehringer Mannheim). Urinary ALA elution was carried out by ion exchange chromatography column test and eluted ALA was measured by colorimetric method<sup>7</sup>. The median and range of blood lead and urinary ALA in controls and different groups of industrial workers were calculated. Wilcoxon Rank sum test was applied for statistical analysis of that.

## Results

Exposed workers were grouped into three categories according to blood levels. They were considered as normal if levels were less than 50 µg/dl; acceptable to continue work with necessary precaution if blood lead levels were between 50-70 µg/dl and unacceptable if levels were more than 70 µg/dl<sup>11</sup>. The relationship between lead exposure and urinary ALA levels was considered according to de Bruin and workers and were grouped as: normal with urinary ALA levels of 0-4.5 mg/dl, mild toxicity at a range of 4.6-10 mg/dl, and moderate to severe when levels were more than 10 mg/dl. Lead fume exposed workers were found to have significantly higher blood lead (median 61.2 µg/dl) and the urinary ALA levels (median 4.1 mg/l) as compared to controls (Table I).

**Table I. Comparison of urinary ALA in lead fume exposed workers with control subjects.**

Parameter	Control (n=25)		Lead fume exposed workers (n=46)		P
	Median	Range	Median	Range	
Age (years)	33	(22-59)	46	(22-55)	NS
Service (years)	15	(02-30)	30	(02-38)	NS
Urinary ALA (mg/l)	2.50	(0.82-04.20)	04.10	(01-22.90)	<0.01
Blood Lead Level (µg/dl)	23.80	(10.20-44.10)	61.20	(21.20- 171.10)	<0.01

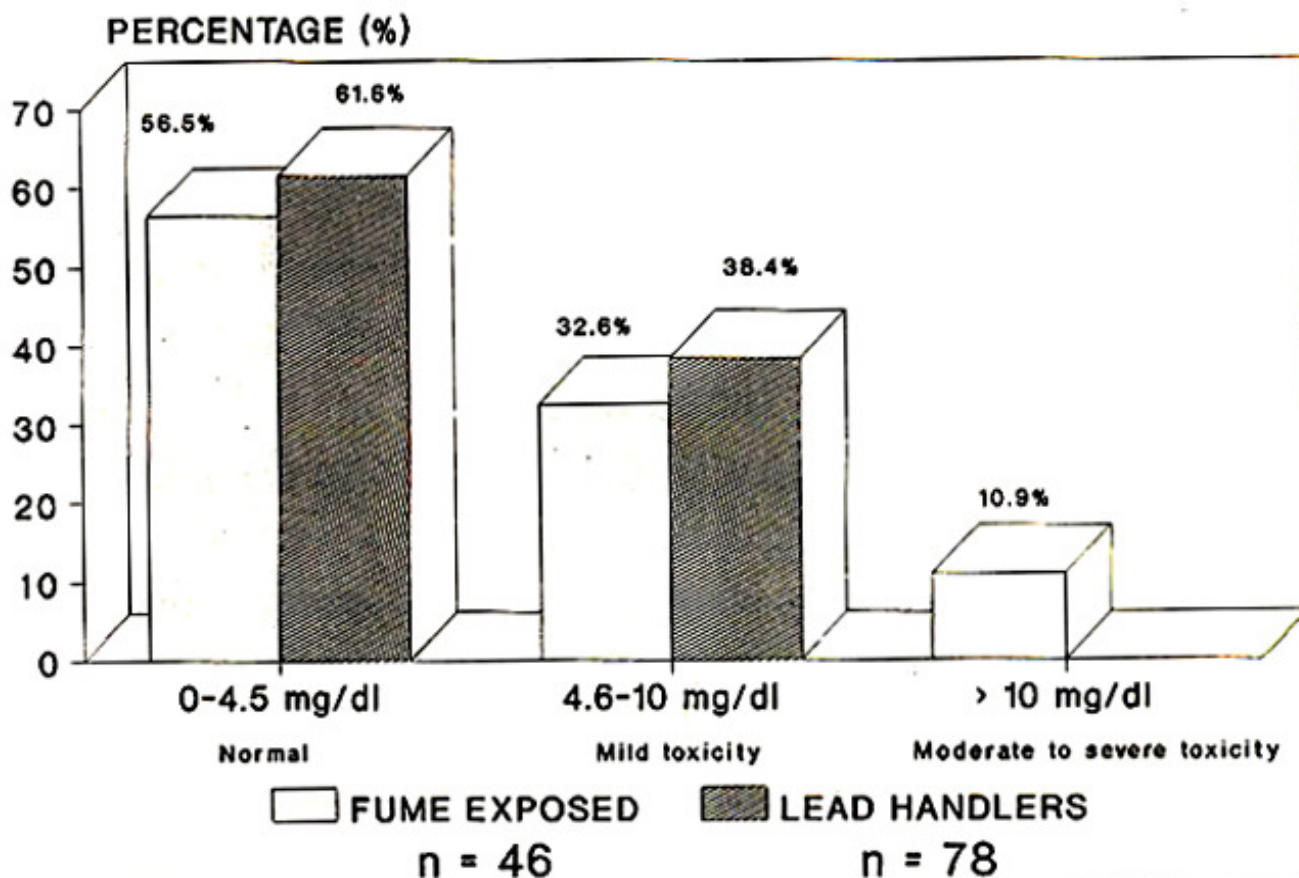
Mild lead toxicity was found in 32.6% and moderate to severe in 10.8% of fume exposed workers. Urinary ALA estimation revealed that 15 (32.6%) had mild (urinary ALA level <10 mg/l) 5 (10.9%) had moderate to severe (more than 10mg/l) lead toxicity (Figure).

Workers handling lead pellets had moderately raised urinary ALA (median 3.9 mg/l) and blood lead levels (median 42 µg/dl) compared with controls (Table II).

**Table II. Comparison of urinary ALA in workers handling lead with control subjects.**

Parameter	Control (n=25)		Workers handling lead (n=78)		P
	Median	Range	Median	Range	
Age(years)	33	(22-59)	43	(24-56)	NS
Service (years)	15	(02-30)	16	(02-28)	NS
Urinary ALA (mg/l)	2.50	(0.82-04.20)	03.90	(0.82-07.60)	<0.01
Blood Lead Level (ug/dl)	23.80	(10.20-44.10)	42.50	(08.50- 130.60)	<0.01

In lead handlers 38.4% had mild toxicity and the rest no toxicity (Figure).



**Figure. Screening of lead toxicity in fume exposed and lead handlers by urinary ALA estimation (n=124).**

Lead fumes exposed workers of more than 10 years of service had significantly elevated urinary ALA levels as compared to those having less duration of service (Table III).

**Table III. Comparison of urinary ALA in lead fume exposed workers having <10 years of service with >10 years of service.**

Parameter	<10 years of service (n=23)		>10 years of service (n=23)		P
	Median	Range	Median	Range	
Age (years)	44	(23-53)	46	(29-55)	NS
Service (years)	4	(2-8)	18	(10-38)	<0.01
Urinary ALA (mg/l)	3.50	(1.10-11.70)	4.40	(1.10-22.90)	<0.05
Blood lead level (ug/dl)	58.40	(21.20-164.40)	63.40	(24.30-171.10)	NS

Similar findings were also observed in lead handlers. However, the blood lead levels did not show any significant change with the duration of exposure in both groups.

#### Discussion

The present study was carried out in workers exposed to lead fumes and those handling lead pellets showed that the former had a higher urinary ALA (median 4.1) and blood lead levels (median 61.1) as compared to controls (Table IV).

**Table IV. Comparison of urinary ALA in workers handling lead having <10 years of service with >10 years of service.**

Parameter	< 10 years of service (n=29)		> 10 years of service (n=49)		P
	Median	Range	Median	Range	
Age (years)	26	(15-54)	46	(27-59)	<0.01
Service (years)	5	(2-9)	15	(10-28)	<0.01
Urinary ALA (mg/l)	3.60	(0.82-05.60)	04.20	(0.91-07.60)	<0.05
Blood lead level (ug/dl)	39.40	(08.50-97.60)	42.30	(18.90-130.60)	NS

In a study from Karachi moderately high blood lead levels were reported in traffic policemen due to exposure to lead fumes in low concentrations<sup>12</sup>. Mild lead toxicity in 32.6% and moderate to severe in 10.8% cases of those exposed to lead fumes might be due to direct absorption of lead from the lungs. Sarah et al.<sup>13</sup> determined blood lead levels in indoor firing range users exposed to lead fumes. Their blood lead levels ranged from 6.4 to 51.1 ug/dl and these levels were low when compared to lead

furnace shops workers. Lead is absorbed from the alveoli of lung with nearly 90% efficiency<sup>14</sup> and high blood levels in lead fume exposed workers in our study was probably due to poor working conditions, lack of knowledge and absence of preventive measures for personal protection. Workers handling lead pellets with bare hands had moderately raised blood lead levels (median 42 ug/dl) and 38.4% had mild lead toxicity. These results were consistent with studies conducted on occupationally exposed lead battery workers and ship scrapping employees whose blood lead levels were over 60 ug/dl<sup>2,4</sup>. Automobile radiator mechanics handling lead materials also had elevated levels ranging from 16-73 ug/dl with 39% mechanics showing levels greater than 40 ug/dl<sup>3</sup>. Workers exposed to lead fumes and lead handlers showed significant correlation between the duration of exposure and their urinary ALA levels but no significant change was noted in their blood lead levels. Similar findings were reported by others<sup>3,15</sup>. Unless blood lead values are determined within a relatively short time following exposure to lead, they may be of little help in any screening programme designed for detecting lead exposure in asymptomatic individuals. Measurement of urinary ALA values completely eliminates this disadvantage since urinary ALA levels remain increased for prolonged periods following removal of the individual from the source of lead exposure<sup>7</sup>. Urinary ALA test has shown closest correlation with the clinical picture of lead poisoning when compared to all other currently available major laboratory tests<sup>16-19</sup>. It is concluded that chronic lead poisoning was more common in workers who were exposed to lead fumes because they had significantly higher blood lead and urinary ALA levels as compared to lead handlers and unexposed healthy adults. The urinary ALA estimation is a more sensitive and specific test than blood lead levels for testing for lead toxicity.

## References

1. Blanke, R.V. and Decker, W.J. Text Book of clinical chemistry, 2nd ed. Philadelphia, WB. Saunders Company, 1986, p.1712.
2. Matte, D., Figueroe, J.P., Burr, G., et al. Lead exposure among lead-acid battery workers in Jamaica. *Am.J.Ind.Med.*, 1989; 16:167-77.
3. Goldman, R.H., Baker, E.L., Hannan, M., et al. Lead poisoning in automobile radiator mechanics. *N.Engl.J.Med.*, 1957;31 7:214-8.
4. Chiang, H.C. and Chang, R.Y. Lead intoxication in shipscrapping employees in Taiwan. *Kao haiung I.Hsueh Ko. Hsueh Tsa. Chih*, 1989;5:284-90.
5. Pollock, C.A. and Ibela, L.S. Lead intoxication in Sydney Harbour bridge workers. *Aust N.Z.J.Med.*, 1988;18:46-52.
6. Hudak, A. and Kiss, G. Improved method for the adjustment of urinary delta-aminolevulinic acid concentration. *Am.J.Ind.Med.*, 1991;19:59-65.
7. Davis, J.R., Abrahams, R.H., Fishbein, W.I., et al. Urinary delta aminolevulinic acid (ALA) levels in lead poisoning. *Arch Environ Health*, 1981;1 7:164-71.
8. Davis, J.R. and Andelenan, S.L. Urinary delta aminolevulinic acid (ALA) levels in lead poisoning. *Arch Environ Health*, 1967;15:53- 59.
9. Wang, S.T., Pizzolsto, S. and Peter, P. Microsampling technique and determination of blood lead by Zeeman atomic absorption spectrophotometry. *Sci Total Environ*, 1988;71 :37-43.
10. Selander, S. and Cramer, K. Inter relationships between lead in blood, lead in urine and ALA in urine during lead work. *Br.J.Ind. Med.*, 1970;27:28.
11. de Bruin, A., Hoorboom, H. Early signs of lead exposure, a comparative study of laboratory tests. *Br.J.Ind. Med.*, 1967;24:203.
12. Manser, W.W. Sunna - A toxic cosmetic, *J. Pak. Med. Assoc.*, 1989;39:196.
13. Sarah, E.V., John, W.M., Jeffrey, K.M., Magdalena, et al. Lead absorption in indoor firing range

users. Am.J.Public Health, 1989;79,8:1029-32.

14. Gross, SB. Human oral and inhalation exposure to lead: summary of Kehoe balance experiments. 3. Toxicol Environ Health, 1981 ;8:333.

15. Yassi, A., Cheang, M. and Tenebheim, M. An analysis of occupational blood lead trends in Manitoba 1979 through 1987, Am. J.Public Health, 1991;81;736-40.

16. Haeger-Aronson, B. Studies on urinary excretion of delta aminolevulinic acid and other haem precursors in lead workers and lead-intoxicated Rabbits. Scand. J.Clin. Lab. Invest 1960;12 (Sops 47):1.

17. Cramer, K. and Selander, S. Detection of industrial lead poisoning. The Lancet, 1966;5:545.

18. Mauzerall, D. and Granick, S. The occurrence and determination of delta aminolevulinic acid and porphobilinogen in urine. J.Biol.Chem., 1956;219:435-46.

19. Griggs, ItS. Lead poisoning: Haemstologic aspect Prog. Hematol., 1964;4: 117.