

The Effect of Occupational Exposure to Lead on Blood Hemoglobin Concentration in Workers of Kermanshah Oil Refinery

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ABSTRACT

Background: Blood hemoglobin can be damaged by toxins such as lead. Thus, this study was carried out to assess its blood hemoglobin concentration in the staff of Kermanshah Oil Refinery that had been exposed to lead components.

Methods: In this study, 150 workers at Kermanshah Oil Refinery were selected as the case group and 70 workers of textile industry factory were chosen as the control group. Informed written consent was obtained from them for participation in the study and blood sampling. Blood hemoglobin level was assessed by SAHLI method and serum lead concentration was assayed by atomic absorption spectrophotometry at 217.4 nm. The results were analyzed by statistical procedures, such as correlation test, and P-values less than 0.05 were considered significant.

Results: The results showed that the presence of a statistically significant difference between hemoglobin levels ($P < 0.05$). Mean hemoglobin level was 15.96gr/100cc in the control group and 12.51gr/100cc in the case group. There was a significant relationship between lead exposure and hemoglobin level.

Conclusion: Lead is a toxin that its chronic exposure decreases hemoglobin level. Protective measures should be anticipated to save the workers from harms of lead exposure.

Keywords: Hemoglobin, Lead, Oil Refinery.

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INTRODUCTION

Hemoglobin is a main component of red blood cells that has a vital role in transportation of oxygen and carbon dioxide. It can hold 1.34cc O₂ per gram in the fully saturated form (1). Lead is one of the toxins that can inhibit hemoglobin synthesis as a result of its influence on erythroblast growth and interference with hemoglobin production (2).

Many studies have shown that lead can inhibit enzymes that take part in hemoglobin synthesis. Lead and its related compounds are toxic materials that have various physiologic unfavorable effects on human including hematopoietic system (3). Hippocrates was the first person that described the characteristics of lead toxicity. more than one thousand years later Nickander performed a research on saturnism (lead poisoning). Martine

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published a book about saturnism in 1700 A.C (4).

There are many services in the health and treatment centers for detection and prevention of lead poisoning including blood lead measurements, lead excretion from urine before or after ingestion of chelators, lead assessment from bones, teeth, and soft tissues, determination of ALAD activities in blood, examination of purphyrine metabolism disorders indices, measurement of ALAD or CP rate in urine, hematologic indices such as basophilic stippling and pointed red blood cells, measurement of hemoglobin level, early laboratory diagnosis of kidney and neurotic involvement, and many other health services (5).

In the places that vapors of lead and its components are propagated in the air, they can enter into body by many ways such as oral, dermal, and inhalational routes (6). In Kermanshah Oil Refinery, TEL (tetra ethyl lead) is used and the workers are at risk of lead exposure. Tetra ethyl lead is a toxic material that was added to gasoline to amplify its octane degree. However, it results in lead intoxication for any person exposed to it. Many studies have been shown that the staff who are working in such places must be checked up carefully for lead exposure (7-9). As Poncka (10), Torra (11) and many other researchers have recommended not only the workers but also all of the people who are directly or indirectly exposed to lead components must be checked as well.

In the present study, blood lead and hemoglobin concentrations in workers of a local oil refinery were measured. The objective was to investigate the relationship between blood lead and hemoglobin concentrations.

MATERIALS AND METHODS

In this study, 150 workers at Kermanshah Oil Refinery were selected randomly and assigned to the case group.

All of them were working in units that had lead exposure such as reservoir and oil transportation unit, refinery operation unit, refinery maintenance unit and tin-smither and tin-filler unit. Also, 70 workers at Kermanshah West Textile Factory who had no occupational lead exposure were randomly selected as the control group.

Demographic and work information of all of the workers were collected. Their blood samples were taken from antecubital fossa for measurement of hemoglobin and lead concentrations.

A.P.D.C (Ammonium pirolidine dithiocarbamate) and M.I.B.K (methyl isobotile keton) were added to the blood samples. Assessment of lead concentration was performed using AAS (atomic absorption spectrophotometry) with flame. The apparatus was Smith-Hafity model at 217.4 nm and accuracy of 0.05 (12). Hemoglobin concentration was measured by SAHLI method (1).

HCl (hydrochloric acid) was added to the samples to hemolyze red blood cells (RBCs) and change hemoglobin to hematine with brown discoloration. The brown color was measured by spectrophotometry. The results were analyzed by statistical testes such as correlation test and P-values less than 0.05 were considered significant.

RESULTS

Hemoglobin (gr/dl) and lead ($\mu\text{gr/dl}$) concentrations are shown in Table 1. Mean hemoglobin concentration was lower in workers of tin-filler unit compared to others while their mean lead concentration was higher. Mean hemoglobin concentration was 12.51 ± 0.23 in the case group, whereas their mean lead concentration was 35.30 ± 6.68 . The corresponding values were 15.96 ± 0.84 and 19.70 ± 3.91 for the control group. The differences between the two groups were statistically significant ($P < 0.0001$).

Table 1. Mean (\pm SD) hemoglobin and lead concentrations in the case and control groups

Group name	numbers	Blood lead μ gr/100 ^{cc}	Hemoglobin gr/100 ^{cc}
Cases groups	Reservoir unit	08	29.17 \pm 2.31
	Refinery operationunit	38	31.11 \pm 5.35
	Refinery maintenance unit	52	33.54 \pm 6.16
	Tin-filler unit	52	41.08 \pm 7.74
Total cases groups	150	35.30 \pm 6.68	12.51 \pm 0.23
Control group	70	19.70 \pm 3.91	15.96 \pm 0.84

DISCUSSION

The findings of this study confirmed the presence of a significant relationship between occupational exposure to lead and blood hemoglobin concentration. Many researchers have described the relationship between occupational lead exposure and blood lead levels clearly (6, 8, 9). This relationship is proportional in nature since more lead exposure results in higher blood lead concentrations. The results of the present study also corroborated this fact.

In workers in the case group, blood lead level was higher while their hemoglobin level was lower. This shows the effect of this toxin on hemoglobin. Many researchers have notified the effect of lead on blood and they believe that this toxin can cause anemia. Tolla and his colleagues observed that lead can reduce hemoglobin level (5). They showed the effects of lead on hemoglobin synthesis in animal models as well as human.

Studies have demonstrated that anemia is one of the earliest manifestations of lead intoxication, after its effect on hemoglobin synthesis (9). Nevertheless, there is no threshold limit value (T.L.V.) for this toxin. William and his colleagues stated that hemoglobin concentration depended on blood lead concentration in occupations that have lead exposure (10), whereas Sacatra *et al.*, reported no decline in hemoglobin level or drop in erythrocyte production in workers with blood lead concentrations less than 50 μ gr/100^{cc} (12).

Cremer *et al.*, did not observe hemoglobin reduction or eritrosite deduction in workers that their blood lead was 50 μ gr/100^{cc} (6). Lilis showed that there is a significant prevalence of low hemoglobin levels (less than 14 g/100 ml) among smelter workers (13), whereas Hawad *et al.*, did find a significant difference between lead exposure and no exposure and hemoglobin background (14). Ribarov *et al.*, showed that lead significantly increases the rate of hemoglobin-catalyzed lipid peroxidation (15). Hu *et al.*, studied the relationship between bone lead and hemoglobin and conclude that patella bone lead levels are associated with decreased hematocrit and hemoglobin levels despite the presence of low blood lead levels. This finding may reflect the subclinical effect of bone lead stores on hematopoiesis and is the first epidemiological evidence that bone lead may be an important biological marker of ongoing chronic toxicity (16).

Yartireh in a study on the workers of Kermanshah Oil Refinery showed that the workers exposed to lead components had higher blood and urine lead level compared to workers with no exposure (17). Aminipour *et al.*, showed that workplaces are the main factor for increasing the blood lead and blood factors change and concluded that occupational health experts should pay attention to this matter (18). Sohrabi *et al.*, obtained similar results in their study on the role of cigarette smoking in blood lead level increase and hemoglobin decrease (19). Measurement of

blood lead and hemoglobin in societies that they are exposed to lead is more sensitive than hemoglobin determination alone because through this method, the relationship between hemoglobin and blood lead and their internal reaction can be illustrated more clearly and with greater certainty. As Richter *et al.*, predicted this matter in their study entitled "*Lead exposure: effects in Israel*". They studied blood lead levels and parallel ambient lead exposure levels in selected Israeli population groups. These studies were prompted by newly emerging findings on subtle renal, hematologic, and neurobehavioral effects of low levels of exposure to lead in both children and adults. There was a high correlation ($r = 0.89$) between individual blood lead levels in the groups studied and free erythrotoporphyrin, a measure of the toxic effect of lead on hemoglobin synthesis. Hemoglobin depression was weakly associated ($r = 0.66$) with increases in blood lead levels (20).

CONCLUSION

Concomitant measurement of blood lead and hemoglobin concentrations is recommended in situations that cause lead exposure. This is more sensitive than hemoglobin determination alone because the relationship between hemoglobin and lead concentrations can be defined. In this way, potential hazard of lead exposure can be depicted more clearly.

If workers have lead exposure in their workplaces, regular measurement of their hemoglobin level and monitor its alterations is strongly recommended. Measurement of lead concentrations in urine samples is valuable as well. Also, using protective measures can reduce lead exposure in workers who are at risk.

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REFERENCES

1. Javaheri H. Principles of Hematological Procedures. 1 ed. Tehran: Golgasht publication; 1992.p.49-62.
2. Ghazi S. Work disease and toils. Tehran University. 1993.p. 59-82.
3. Lotfi S. Lead metabolism and its intoxications. Teacher training university. 1993.p.79-97.
4. Sanaii Gh. Industrial toxicology. Tehran University.1991.p.180-214.
5. WHO Task Group on Environmental Health Criteria. Lead-environmental aspects.: Geneva : World Health Organization; 1989.
6. Cremer JE. A study of action lead – alkyls in the solvents. Industrial Med. 1989; 81(6):325-30.
7. Oil ministry. Kermanshah oil refinery. Tehran oil ministry. 1991.p.9-13.
8. Angerer J, Schaller KH. Analyses of Hazardous Substances in Biological Materials.Wiley-VCH. 1996;1.p.118-23.
9. Frenz P. Chronic exposure to industrial lead in adults. Rev Med Adult. 1997;121(9):38-47.
10. Torra M, Rodamilans M, Montero F, Farré C, Corbella J. Exposure to lead among the population of Barcelona: chronologic trends from 1984 to 1995]. Medicina clínica. 1997;108(16):601-3.
11. Pönkä A. Lead in the ambient air and blood of children in Helsinki. Science of the total environment. 1998;219(1):1-5.
12. Katz M. Methods of air sampling and analysis. 2 ed. USA: American Public Health association; 1993.P. 850-61.
13. Lilis R, Eisinger J, Blumberg W, Fischbein A, Selikoff I. Hemoglobin, serum iron, and zinc protoporphyrin in lead-exposed workers. Environmental health perspectives. 1978;25:97-102.
14. Hawad AF, Eman MA, Amnia B, Elssaidi MA. Lead and Hemoglobin Level in Blood of Occupationally Exposed Workers at Brack Alshatti Area, Libya.Available from:

- <http://www.scribd.com/doc/9664718/Lead-and-Hemoglobin-Level-in-Blood-of-Occupationally>.
15. Ribarov SR, Benov LC, Benchev IC. The effect of lead on hemoglobin-catalyzed lipid peroxidation. *Biochimica et Biophysica Acta (BBA)-Lipids and Lipid Metabolism*. 1981;664(3):453-9.
 16. Hu H, Watanabe H, Payton M, Korrick S, Rotnitzky A. The relationship between bone lead and hemoglobin. *Journal of the American Medical Association*. 1994;272(19):1512-7.
 17. Yartireh HA. Assessment of urine and blood lead in workers of Kermanshah oil refinery. *Jundishapur Scientific Medical Journal*. 2002; 31: 60-5.
 18. Aminipour M, Barkhordari A, Ehrampoush M, Hakimian A. Blood lead levels in workers at Kooshk lead and zinc mine. *Journal of Shahid Sadoughi University of Medical Sciences* 2008; 16(2):24-30.
 19. Shahrabi J, Dorosti A. Study of blood lead levels, hemoglobin & plasma ascorbic acid in a car company welders. *Iran Occupational Health journal*.2006; 3(1): 43-49.
 20. Richter E, Neiman S, Yaffe Y, Gruener N. Lead exposure: effects in Israel. *Israel J Med Sci*. 1980;16(2):89-95.